THE COMORBIDITY OF INTERNALISING DISORDERS ON ATTENTION DEFICIT HYPERACTIVITY DISORDER IN PRIMARY SCHOOLS IN LEPELLE-NKUMPI MUNICIPALITY, LIMPOPO PROVINCE.

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DECLARATION

I, MORONGWA CAROLINE TAKALANI, hereby declare that the thesis for the Doctor of Philosophy degree at the University of Venda, hereby submitted by me, has not previously been submitted for a degree at this or any other university, and that it is my own work in design and execution and that all reference material contained therein has been duly acknowledged.

Signature M.C Takalani Date 10.09.2020

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DEDICATION

I dedicate this work to my wonderful husband Dr James Takalani. I have grown through your support. It has been your desire to see me obtain this degree, and through your support and guidance I finally made it.

ABSTRACT

Attention Deficit Hyperactivity Disorder (ADHD) is the most common diagnosed neuro-cognitive behavioural developmental disorder among school going–age children. ADHD in most instances is found to comorbid with internalising disorders such as anxiety and depression, therefore causing impairment on behaviour, emotions, academic, social and many areas of functioning. The existence of comorbid internalising disorders complicates the presentation of ADHD symptoms than if it was pure ADHD. The aim of the study was to explore the comorbidity of internalising disorders and ADHD symptoms. The study investigated whether children with ADHD have more symptoms of anxiety and depression than children without ADHD. The study went further to investigate whether children with ADHD have low self-esteem than children without ADHD. Also, that internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who have more externalising comorbidities which are Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) and anger issues. The null hypotheses further showed that, children with ADHD do not have more symptoms of anxiety and depression than children without ADHD. Moreover, children with ADHD have no lower self-esteem than children without ADHD. Also, internalising disorders (anxiety and depression) are not prevalent in girls than in boys who do not exhibit more externalising disorders which are Oppositional Defiant Disorder (ODD), Conduct Disorder (CD) and anger issues. The study was conducted in Lepelle-Nkumpi Municipality in Lebowakgomo sub circuit.

A total of 250 children (94 boys and 156 girls, aged 6 – 12) who were purposively selected participated in the study. A total of 125 clinically diagnosed ADHD were matched to 125 control group for age and gender. Participants were assessed on a battery consisting of Parent/Teacher Disruptive Behavioural Rating Scale (DBD), which was used to assess the presence and degree of ADHD- related symptoms (inattention and hyperactive/ impulsive); Oppositional Defiant Disorder and Conduct Disorder and Beck Youth Inventory Second Edition (BYI II), which was used to assess emotional and psychological issues that children experience. ADHD scores obtained on the DBD scale were correlated with scores obtained on BYI II scale. The study only focused on learners from primary schools in Lepelle-Nkumpi in Lebowakgomo sub circuit. In this research study, children who were reported to have a history of neurological problems such as head injurie,

epilepsy, cerebral palsy or severe psychiatric disorders were excluded from the study.

The results were analysed using the analysis of variance (ANOVA) and post hoc benferoni analysis to determine the significant difference within ADHD subtypes. Results of the study showed that children with ADHD had comorbid internalising disorders (anxiety and depression) when compared to their neurotypical group. Both boys and girls with a clinical diagnosis of ADHD showed symptoms of anxiety and depression. More symptoms of anxiety and depression were observed within the ADHD-HI, ADHD-PI and ADHD-C subtypes. Comorbid internalising disorders (anxiety and depression) were also associated with impairments with ADHD symptom, in particular inattention.

Children with ADHD also showed more symptoms of low self-esteem when compared to their neurotypical group. Both boys and girls showed significant difference level of low self-esteem in all ADHD subtypes (ADHD-HI, ADHD-PI and ADHD-PI). The results further revealed that both boys and girls equally showed symptoms of comorbid internalising disorders (anxiety and depression), whereas girls showed significantly more symptoms of externalising comorbidities such as ODD, CD and anger issues.

In conclusion, the findings suggest that the comorbidity of internalising disorders in a clinically diagnosed child with ADHD worsens or complicates the child's expected normal functioning unlike when the child was presenting with only ADHD. The results further suggest that a child with clinical diagnosis has double impairment as a result of many impairments which are occurring at the same time. Moreover, the existence of externalising comorbidities which are salient in girls with ADHD may require proper screening and assessment.

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LIST OF ABBREVIATIONS AND ACRONYMS:

ADHD	:	Attention Deficit Hyperactivity Disorder
ADHD-C	:	Attention Deficit Hyperactivity Disorder- Combined type
ADHD-HI	:	Attention Deficit Hyperactivity Disorder Predominantly Hyperactive/ Impulsive type
ADHD-PI	:	Attention Deficit Hyperactivity Disorder- predominantly inattentive type
ANOVA	:	Analysis of Variance
APA	:	American Psychiatric Association
BLL	:	Blood Lead Level
BYI	:	Beck Youth Inventories
CBCL	:	Child Behaviour Check List
CD	:	Conduct Disorder
CPT	:	Conners Continuous Performance Test
DA	:	Dopamine
DBD	:	Disruptive Behavioural Disorders
DDT	:	Dynamic Developmental Theory
DMDD	:	Disruptive Mood Dysregulation Disorder
DNA	:	Deoxyribonucleic acid
DSM III	:	Diagnostic Statistical Manual of Mental Illness Third Version
DSM IV-TR	:	Diagnostic Statistical Manual of Mental illness Fourth Edition- Text Revised
DSM-5	:	Diagnostic Statistical Manual of Mental Illness Fifth Edition
DZ	:	Dizygotic
EF	:	Executive Function Classification of Disorder

GDG	:	Guideline Development Group
GRADE	:	Grading of Recommendations Assessment Development and Evaluation
GRC	:	Guideline Review Committee
ICD	:	International Classification of Disorders
MMI	:	Mild Moderate Intellectual
MRI	:	Magnetic Resonance Imaging
MZ	:	Monozygotic
ODD	:	Oppositional Defiant Disorder
PET	:	Positron emission tomography
PFC	:	Prefrontal cortex
PICO	:	Population Intervention Comparator and Outcomes
RPC	:	Research and Publication Committee
SCT	:	Sluggish Cognitive Tempo
SGB	:	School Governing Body
TCV	:	Total Cerebellum Volume
UHDC	:	University Higher Degree Committee
WHO	:	World Health Organization

CHAPTER ONE

INTRODUCTION

1.1 Introduction

In this chapter, a background to the study, including the aim, problem statement, hypotheses, and objectives of the study, significance of the study and definitions of concepts that are used is outlined. The study explores the comorbidity of internalising disorders and attention deficit hyperactivity disorder (ADHD). Moreover, the study wants to show how the existence of these comorbid internalising disorders can go further to impair children's normal functioning unlike they suffered pure ADHD. Attention deficit hyperactivity disorder is a persistent, clinically heterogenous disorder that puts a huge weight on society in terms of financial cost, stress to familes, and adverse academic and vocational outcomes (Biederman & Faraone, 2004). ADHD is a neuropsychiatric developmental disorder originating from childhood and commonly characterised by extreme levels of inattentiveness, impulsiveness, and hyperactivity (American Psychiatric Association, 2013; Van der Oord, Prins, Oosterlaan & Emmelkamp, 2006). Other disorders are further also linked to ADHD that is continuing to cause even more impairments.

Additionally, childhood ADHD is associated with impairment in academic achievement, family interaction, peer relationship, self-esteem, and health related quality of life (Harpin, Mazzone, Raynaud, Kahle, & Hodgkins, 2016). As they develop into adolescence, children's impairments become complex, and are at the risk of engaging in risk taking behaviours. ADHD often comorbids with externalising and internalising disorders (Jensen, Hinshaw, Kraemer, Lenora, Newcorn, Abikoff, & Vitiello, 2001), which often result into severe negative long-term consequences (Harris, Stoppelbein, Greening, Becker, Luebbe & Fite, 2013). Psychiatric disorders in children and adolescents may cause psychosocial impairments, and if not treated predicts increased risk for other mental disorders as well as substance use problems in adulthood (Castagna, Calamia & Davi, 2017). This behaviours as a result can be observed as being influenced.

Most behaviors seen among these children can therefore be seen as a result of multiple disorders coexisting with ADHD, causing multiple behavioural impairments and developing other mental disorders as they enter different developmental phases in their lives. Although internalising disorders are under researched, a study by Hahlweg, Heinrichs, Kuschel, Bertram and Naumann (2010) found that 20% of children experience either internalising or externalising disorders and its prevalence rate cannot be reduced through treatment only. Effective preventive interventions are therefore urgently needed for the well-functioning of these children. Although the current study is more focused on the comorbidity of internalising disorders, externalising disorders have been well explored and observed with its influences on the disorder ADHD.

Externalising disorders in a form of conduct disorder and oppositional defiant disorders in existence with ADHD are among those which complicate the presentation of ADHD. Outrageous behaviors such as anger, temperament behaviour and disruptive behaviors can be seen as having a link to ADHD symptoms. Studies continued to show that these externalising disorders were more noticeable in males than in females which made them to be easily referred and access intervention (Skogli, Teicher, Andersen, Hovik & Oie, 2013). On the other hand, internalising disorders were regarded as more intense and were more prevalent in females (Quinn & Madhoo, 2014). Children with ADHD in nature have low frustration tolerance and recurrent temper tantrums (Connor, Chartier, Preen & Kaplan, 2010) hence the acting out behaviours. Most behaviors observed as a result of internalising and externalising disorders are due to inattention and hyperactivity as key symptoms of ADHD (Carre, McCornick & Hariri, 2011; Lonigan & Phillips, 2001). Following is the background information about ADHD and the influence that other comorbid disorders has on the disorder.

1.2 Background to the study

Attention deficit hyperactivity disorder (ADHD) is regarded as one of the most common diagnosed neurodevelopmental disorders among school going–age children (Leirbakk, Clench-Aas & Raanaas, 2015). The disorder is mainly characterised by problems in allocating attention, regulating motor activity and controlling behavioural impulses (Schellack & Meyer, 2012; Muller, Aaserson, Banaschweski, Buitelaar, Ebstein, Eisenberg & Roeyers, 2011). Although the disorder is known to continue into adolescence, the onset of the symptoms is observed during early childhood on

average, between 3 and 6 years of age (Barkley & Murphy, 2006). Attention deficit hyperactivity disorder is a disorder of the brain affecting behavior mostly displayed by children, and therefore having a serious impact on academic achievement and low social competence.

ADHD is a prevalent childhood disorder, with most individuals affected having psychiatric disorders associated with ADHD (Masi & Gignac, 2017). ADHD, depression and anxiety are often comorbid in children, often resulting in more damaging ADHD consequences (Leirbakk, Clench-Aas & Raanaas, 2015). The presence of both ADHD, anxiety and depression is associated with major impairments, and may have negative impact on the child's functioning, as well as future outcomes. These disorders, anxiety and depression are classified as internalising disorders, whereas ADHD is considered an externalising disorder (In- Albon, 2012). Both these disorders have major defects from as early as childhood and through adolescence and adulthood.

According to Carter, Wagmiller, Gray, McCarthy, Horwitz and Briggs-Gowan (2010), children from as early as 6 years and older already meet the criteria for mental disorders having impairments with the prevalence of 21.6%. This shows that it is not unusual that mental disorders are uncommon among children, meaning that children as early as elementary phase are already prone to psychiatric disorders. Biederman (2005) and Kessler, Adler, Barkley, Biederman, Conners, Demler, Faraone, Greenhill, Howes, Secnik, Spencer, Ustun, Walters and Zaslavsky (2006) state that 60% to 100% of patients diagnosed with ADHD exhibit one or more psychiatric disorders such as anxiety and depression. ADHD in most instances is found to co-exist with internalising disorders (that is, anxiety and depression, causing impairment on behaviour) (Johansen, Aase, Meyer & Sagvolden, 2002; Sagvolden, Johansen, Aase & Russel, 2005). These internalising disorders are said to be repeatedly reported than externalising disorders and in particular anxiety disorders being the most common mental disorders in children followed by ADHD and aggressive behavioural disorders (In-Albon, 2012). Quite often internalising disorders may be overlooked yet causing more serious damage to the individual.

Children presenting with mental illness continue to suffer in silence for long, especially that these disorders do not disappear over a period of time (In-Albon, 2012). Lack of immediate intervention in this regard may lead to a possibility of developing other mental disorders when entering adolescence and into adulthood. Although internalising disorders are underresearched, a study by Hahlweg, Heinrichs, Kuschel, Bertram and Naumann (2010) found that about 20% of children experience either internalising or externalising disorders and the prevalence rate cannot be reduced through treatment only. Effective preventive interventions are therefore urgently needed for the well-functioning of these children. However, strengthening intervention strategies is quite significant beacuase of the nature of the presentation of both disorders which may create confusion in differentiating the symptoms.

Both ADHD and internalising disorders have the tendency to display overlapping symptoms (Baldwin & Dadds, 2008), which become a challenge in assessing and treating these individuals. A study conducted by Karustis, Power, Rescorla, Eirald and Gallagher (2000) found that internalising symptoms in children with ADHD were associated with impairment on various measures of social and academic competence. Loe and Feldman (2007) observed that children with ADHD have persistent academic difficulties as compared to children without ADHD. These children are typically and often mistakenly regarded as having low intellectual functioning hence they are referred for further assessment or scholastic placement by teachers.

It is estimated that between 13% and 51% of children diagnosed with ADHD suffer from internalising disorders such as anxiety (Manassis, Tannock, Young & Francis-John, 2007; Bauermeister, Shrout, Ramirez, Bravo, Alegria, Martinez-Taboas, Chavez & Canino (2007); Gillberg, Gillberg, Rasmussen, Kadesjo, Rastam, Johnson, Rothenberger & Nikiasson, 2004). Of these children diagnosed with ADHD, up to 5.7% to 17.7% of them, anxiety disorder was found to be among the most common psychiatric disorder (Yorbik & Birmaher, 2003). In a study conducted in Germany, it was found that between 0.5% and 9.4% of children with ADHD were observed to have more internalising disorders more than externalising disorders. This shows that internalising disorders are prevalent and more alarming although research has shown that children with externalising disorders are easily been referred for further management (In-Albon, 2012). It can be understood that, since externalising behaviors

are easily noticed, more support in a form of educating these individuals about internalising disorders may appear to be beneficial to them and significant others. More impairments associated with ADHD are therefore outlined.

ADHD causes impairments in many areas of life such as poor peer relationships, aggression and learning problems. All these impairments are associated with poor academic achievement and high risk for developing low self-esteem (Biederman, 2005). Most individuals with ADHD as explained by Schatz and Rostain (2006), are observed to have anxiety as a common comorbid disorder. Anxiety as a comorbid disorder has a tendency to escalate among ADHD suffers and as a result majority of them continue to display other forms of anxiety disorders (Dan & Raz, 2015). A study by Lonigan and Phillips (2001) was able to link anxiety disorders to biological defects, while other studies showed some similarities of key symptoms with ADHD (Brown, 2000). Both ADHD and anxiety seem to have overlapping symptoms which to some extend may confuse the real manifestation of the disorder. Lonigan and Phillips (2001) further elaborated that anxiety disorders are as a result of temperament of behaviour inhibition which is a key symptom of ADHD i.e. inattention. Children with anxiety approach situations with restraint withdrawal, avoidance or distress. Moreover, they are shy, withdrawn and fearful, and they tend to avoid challenges.

Additionally, children with ADHD and anxiety were observed to be impaired and yet less impulsive, and inattentive (Pliszka, Carlson & Swanson, 1999). Thus, they may have a huge impact on their attitude especially towards themselves as individuals and refrain to take part in activities. On the other hand, Youngstrom, Loeber and Stouthamer-Loeber (2000) as cited in Van der Oord, Prins, Oosterlaan and Emmelkamp (2006) found that parental factors such as family history of alcohol use disorder, parental inconsistency, harsh parenting, family discord and parent psychopathology (parental substance abuse) may aggravate the level of internalising disorders and ADHD symptoms. Other researchers managed to outline more challenges in association with internalsing disorders.

In a study conducted by Palaniappan (2016) in India, the prevalence of ADHD in children was found to range from 1.6% to 14%. Of this figure, about 60% was observed to be having academic problems, whereas 75% of them experienced peer related problems (Malhi & Singhi, 2001). Palaniappan (2016) further observed that early age

onset of ADHD in children was strongly linked to comorbid internalising disorders resulting in lower social and global domains of self-esteem. Having childhood ADHD can be a risk factor for developing psychiatric disorders. Children diagnosed with ADHD and internalising disorders face multiple challenges and those with anxiety were observed to display challenges with the ability to concentrate and finish schoolwork (Baldwin & Dadds, 2008). Other challenges experienced by these children are peer rejection and academic incompetence (Keilley, Lofhouse, Bates, Dodge & Pettit, 2003), which will continue to put children at risk of developing negative outcomes. Anxiety in these children may further lead to withdrawal in most activities due to negative reaction from peers and leading to fear of future participation (Skinner & Piek, 2001).

The existence of anxiety partially inhibits impulsivity and response inhibition deficits seen in ADHD, i.e. making working memory and other cognitive deficits worse (Brown, 2000). These individuals in particular are less likely to display hyperactive behavior as compared to those with pure ADHD (Pliszka, Carlson & Swanson, 1999). Moreover, extreme challenges in tasks that require working memory and serial additions (Brown, 2000) were common, which means they are likely to make more errors, indicating more impairments of working memory (Schatz & Rostain, 2006). Although anxiety can be seen as a remedy to ADHD symptomatology in particular hyperactivity, it continues to cause more impairments in the cognitive functioning among these individuals. Moreover, comorbid intenalising disorder such as depression seem to be influencial in one's ability to function.

Children with ADHD were also said to be having comorbid internalising disorder i.e. depression and that was estimated between 15% and 75% (Spencer, 2000). Depression in children diagnosed with ADHD was linked with poor long-term outcomes and greater psychiatric problems such as aggression and suicide (Connor, Glatt, Lopez, Jackson & Mellow, 2002). Severe symptoms of ADHD and high rates of aggression were said to be more prevalent in children diagnosed with ADHD and depression (Connor, Edwards, Fletcher, Baird, Barkley, & Steingard, 2003). These children were further observed to be more depressed and anxious than children with only ADHD (Connor et al., 2003). Children diagnosed with ADHD and depression will appear to have emotional challenges as compared to their normal counterparts with

only ADHD and without depression.

It was further observed that children with ADHD and comorbid depression often display impairment in areas of social and academic functioning as compared to their normal counterparts (Blackman, Ostrander & Herman, 2005). They will continue to experience challenges in areas that will require learning, planning and decision making and for this reason their cognitive functioning becomes impaired. Moreover, socially, children with ADHD and comorbid depression suffer rejection by their normal peers. Therefore, due to the deficit in their social and academic functioning, these children are more likely to exhibit comorbid conduct problems (Jensen, Martin, & Cantwell, 1997) as an externalising behaviour. The coexistence of ADHD and depression then can be observed as a gateway to a diagnosis of externalising disorders at a later stage. Although internalising disorders like anxiety and depression may not disappear over time, they can be a risk factor for the development of further mental disorders in adulthood (In-Albon, 2012).

Above all internalising disorders impair the normal functioning in ADHD sufferers because they may influence severity, daily functioning, treatment and prognosis (Brown 2000). Children diagnosed with ADHD and internalising disorders such as anxiety and depression may have the tendency of low Sluggish Cognitive Tempo (SCT) such as confusion and mental alertness (Todd, Rasmussen, Wood, Levy & Hay, 2004). The existence of internalising disorders and ADHD may then impact on the emotional, behavioural and cognitive functioning in these children.

ADHD and comorbid disorders in other countries

ADHD and internalising disorders in general were underresearched, however few studies conducted, managed to observe the difference between African American and European American school going children. Studies emphasised that the prevalence of ADHD among male children is somehow disproportionately diagnosed with an estimated rate of 5.65%, 4. 3% for Hispanics, 3% for Whites, and 1.77% for females in all races (Bailey & Ofoemezie, 2013). This can be viewed by the fact that males are often easily diagnosed and more referred for further management than females.

ADHD in the past was predominantly known to be common among children in Western countries. However, studies have also found ADHD to be a prevalent psychiatric

disorder in the African continent (Meyer, Eilertsen, Sundet, Tshifularo, & Sagvolden., 2004; Ofovwe, Ofovwe, & Meyer, 2006). For instance, in a study conducted in South Africa, Gauteng, Mako (2002) observed a significant correlation in the prevalence of ADHD symptoms in those children who were referred to a clinic for diagnosis and treatment and those in the community, except for the older girls (> 10 years of age). This shows that ADHD is not a culturally influenced disorder; rather it affects individuals across all spectrums. Dwivedi and Banhatti (2005) argued that cross-cultural studies need to be based on other better means of identifying the disorder rather than using the clinical diagnosis. In order to understand the phenomenon of ADHD, it is important to explore the complex interaction between organic, environmental, and cultural factors. Therefore, it seems that ADHD-like behaviour is caused by the same fundamental neurobiological processes, probably caused by genetic factors expressed independently of cultural differences (Johansen et al., 2002; Sagvolden, Johansen, Aase & Russel, 2005).

However, cultural differences may affect the performance on psychometric measures. Therefore, there is a need for assessment methods that are culturally sensitive and valid for different ethnic groups. Thus, systematic research to identify and develop the instruments required to assess ADHD symptomatology are greatly needed in developing countries.

1.3 Problem statement

ADHD is a neurobehavioural disorder commonly affecting children and is clearly observed among school going children. There are many challeges experienced by school going children and many of these challenges are associated with the disorder ADHD in co-existence with internalising disorders such as anxiety and depression. Those challenges include poor behavioural planning, poor scholastic performance, and low self-esteem and anger issues. These may have a negative impact impact on their behaviour and their quality of life socially and academically.

Children with ADHD and anxiety were observed to be struggling with inattention than impulsivity compared to those with ADHD only (Tannock, 2009). Problems related to ADHD and comorbid anxiety were problems in performing cognitive complex tasks such as those involving working memory, aggressive symptoms and low self –esteem

(Tannock, 2000). Children with ADHD and depression on the other hand were found to have inattention symptoms than hyperactivity which causes disruption of interpersonal functioning. (Blackman, Ostrander & Herman, 2005; Humphreys, Katz, Lee, Hammen, Brennan & Najman, 2013). Problems such as severe ADHD symptoms and high rates of aggression and severe psychiatric problems are found to be common among children with ADHD and depression (Blackman, Ostrander & Herman, 2005). Having ADHD and internalising disorders may only worsen the situation these children already found themselves and that is causing double impairment.

The development of internalising disorders at an early age may result in more serious impairments and worse developmental outcomes throughout childhood and adolescence (Seymourm Chronis-Tuscano, Iwamoto, Kurdziel & MacPherson, 2014). Both ADHD and internalising disorders cause major impairments on inattention which is a key symptom of ADHD. These disorders make children to experience multiple impairments because they are cognitively impulsive with lack of planning, organisatoion and failure to remember important things. These impairments are therefore essential in areas that require learning.

The researcher has observed an influx of learners referred from various schools in Lepelle-Nkumpi district as a result of poor scholastic performance and impaired behaviour not clearly understood by teachers. Most referrals by the school teachers came with a recommendation that these learners must be placed in a special school because they do not behave in a normal way as compared to other learners of their age and as a result they do not fit in mainstream. These children were also often mistakenly perceived by their educators as having mental impairment as they did not see them fitting in like the rest of the other children. Furthermoe, it appears that when comorbid symptoms are present, teachers and parents report more concern for these symptoms which are observed in a form of poor scholastic achievement and behavioural problems. Children presenting with ADHD and comorbid disorders continue to suffer in silence as a result of multiple disorders, since teachers and parents are not expected in screening behavioural and emotional disorders. The study therefore seeks to contribute towards addressing this gap by developing guidelines which will assist both techers and parents so that they can be able to pay attention to those signs with the aim that children receive early identification so that they are

refereed properly for eary intervention.

1.4 Aim of the study

The aim of the study was to explore the comorbidity of internalising disorders such as anxiety and depression on attention deficit hyperactivity disorder symptoms (that is, inattention, hyperactivity/impulsiveness) in primary schools in Lepelle- Nkumpi municipality, Limpopo Province.

1.5 Objectives of the study

The objectives of the study were as follows:

- To explore whether children with ADHD have comorbid internalising comorbidities (anxiety and depression).
- To explore whether children with ADHD have lower self -esteem than children without ADHD.
- To explore whether internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who have more externalising comorbidities (Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) and anger issues.
- The study further made an attempt to develop guidelines, which will serve as a tool that teachers and parents will use in screening and ensuring proper referral for the child with the hope that at the end the child receives proper intervention.

1.6 Hypotheses and null hypotheses of the study

The main hypotheses of the study were:

- Children with ADHD have more symptoms of anxiety than children without ADHD.
- Chuldren with ADHD have more symptoms of depression than children without ADHD.

- Children with ADHD have more symptoms of low self-esteem than children without ADHD.
- Internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who have more externalising comorbidities (oppositional defiant disorder (ODD), conduct disorder (CD), anger issues and disruptive behaviours.

The main Null hypotheses of the study were as follows:

Research hypothesis 1:

Children with ADHD have more internalising disorders than children without ADHD

Null hypothesis 1(a)

Children with ADHD have no more anxiety disorders than children without ADHD

Alternative hypothesis 1(a)

Children with ADHD have more anxiety disorders than children without ADHD.

Null hypothesis 1(b)

Children with ADHD have no more depression symptoms than children without ADHD

Alternative hypothesis 1(b)

Children with ADHD have more depression symptoms than children without ADHD.

Research hypothesis 2

Children with ADHD, because of their comorbid internalising disorders, have a lower self-esteem than children without ADHD.

Null hypothesis 2

Children with ADHD have no lower self-esteem than children without ADHD.

Alternative hypothesis 2

Children with ADHD have a lower self-esteem than children without ADHD.

Research hypothesis 3

Internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who exhibit more externalising comorbidities (ODD), (CD), anger issues, and disruptive behaviours

Null hypothesis 3(a)

Girls do not have more anxiety symptoms than boys.

Alternative hypothesis 3(a)

Girls have more anxiety symptoms than boys.

Null hypothesis 3(b)

Girls do not have more depression symptoms than boys.

Alternative hypothesis 3(b)

Girls have more depression symptoms than boys.

Null hypothesis 3(c)

Boys have no more symptoms of ODD than girls.

Alternative hypothesis 3(c)

Boys have more symptoms of ODD than girls.

Null hypothesis 3(d)

Boys have no more symptoms of CD than girls.

Alternative hypothesis 3(d)

Boys have more symptoms of CD than girls.

Null hypothesis 3(e)

Boys express no more anger than girls.

Alternative hypothesis 3(e)

Boys express more anger than girls.

Null hypothesis 3(f)

Boys show no more disruptive behaviours than girls.

Alternative hypothesis 3(f)

Boys show more disruptive behaviours than girls.

1.7 The rationale for the study

Quite often children with ADHD are mistakenly classified as having mental retardation by school teachers and they are further referred for assessment for placement in special schools. Improper diagnosis of a disorder by clinicians may therefore result into wrong management to the diagnosed i.e. the end user being wrongly labelled, dependency to medication instead of encouraging other effective therapeutic interventions, improper school placement and to some individuals they may be rejected by their peers or engage in risk taking behaviours such as cigarette smoking, alcohol abuse, sexual promiscuity, suicide etc. It then becomes imperative that a proper diagnosis of the disorder as well as other coexisting disorders is properly done in order to have a proper diagnosis and treatment plan. According to Miller, Johnston, Klassen, Fine, and Papsdorf (2005), treating ADHD may be important since it may decrease symptoms, enhance functionality, and improve well-being for the individual and his or her close contacts.

Internalising disorders such as anxiety and depression are among the most common comorbid psychological disorders of childhood and adolescence (Castello, Mustillo, Erkanl, Keeler, & Angold, 2003). However, there is still no enough research work done on the comorbidity of internalising disorders on ADHD symptoms. The existence of other comorbid disorders has devastating future outcomes on the life of an individual

suffering from these conditions at once. A body of research on ADHD has previously focused the prevalence, gender and risk-taking behaviours associated with ADHD. Studies conducted previously on ADHD and comorbid psychopathology (Elia, Ambrosisni, & Berrettin, 2008; Pliszka, 2000) focused more on male participants, whereas the current study will focus on comorbid psychopathology and ADHD on both male and female participants.

In a study conducted by Tung, Li, Meza, Jezior, Kianmahd, Hentschel, O'Neil and Lee (2016), on patterns of comorbidity among girls showed that girls with ADHD frequently exhibit comorbid internalising and externalising disorders. The study paid attention to girls, hence it is for this reason that the resesrcher in the the current study wants to look at the comorbid internalising and externalising disorders in both genders. Research on comorbid disorders among girls with ADHD showed that girls with ADHD experience internalising problems (Biederman et al., 2008; Biederman et al., 2010), hence the current study attempts to bridge the gap by exploring comorbid disorders across gender if boys also may suffer internalising disorders. A study done on internalising problems and Attention Deficit Hyperactiviyt Disorded, showed a posistive association between ADHD and internalising problems in both boys and girls (Norén, Molero, Lichtenstein, Anckarsäter, Lundström, Bottai & Hellner, 2016). The study was a longitudinal study conducted among Sewdish twins. It is for this reason that the current study is attempting to do similar research but in the area of Limpopo among Africans to assess if the same research could yield similar results.

Although there was one study conducted in Limpopo among rural primary school children in Limpopo Province, on the comorbidity of ADHD and major depression (Mokobane, Pillay & Meyer, 2017). The study was conducted in the Capricorn District and focused only on depression, while the current study will assess both anxiety and depression among primary schools in Lepelle-Nkumpi municipality within the Capricorn District.

1.8 Significance of the study

The comorbidity of internalising disorders poses as a risk factor to individuals or suffers of ADHD by compromising their quality of life while worsening symptoms of ADHD i.e. inattention, hyperactivity and impulsivity. Sayal, Daley, James, Yang, Batty, Taylor,

Pass, Sampson, Sellman, Valentine and Hollis (2012) argued that early intervention for childhood behavioural problems may help improve health and educational outcomes in affected children and therefore reduce the likelihood of developing additional difficulties. Comorbid internalising disorders along with ADHD compromise the success of children especially in important areas such as academic achievement, family interaction, peer relationships, self-esteem and health related quality of life (Harpin, Mazzone, Raynaud, Kahle & Hodgkins, 2016). These children may be assisted so that they do not have to deal with multiple impairments which compromise their ability to succeed academically, socially and have good interpersonal skills. The National Institute for Health and Clinical Excellence (2008) suggested that stepped care approach for the identification and management of these common childhood behavioural disorders should be observed. Moreover, providing more information to stakeholders such as policy makers and educational ministry may strengthen more support between parents, teachers and children who are already at risk of behavioural impairments.

Parents and educators at large are overwhelmed by what they see as a result of this impairing disorder seen through behaviour as well as emotional impairments which may complicate the existing disorder. The current study went futher to investigate the rate at which these comorbid internalising disorders have on ADHD symptoms. This was done with a view of developing an appropriate guideline which may guide different teachers and parents towards assisting children at risk of behavioural disorders. It is trusted that a study of this nature could be useful to the following stakeholders: policy makers; educaton ministry; educators; parents; teachers; clinicians; ADHD children and scholars.

1.8.1 Policy makers

- The study could provide useful information that can assist policy makers in implementing constructive policy acts which recognize mental health disorders (anxiety and depression) and behavioural disorders (ADHD) among school children in South Africa in particular Limpopo province. Moreover, provide policies which do not discriminate against children diagnosed with ADHD from normal children, while on the other hand protecting the rights of those children. This may

also be beneficial to policy makers because children may no longer be discriminated or deprived of education as a result of their psychiatric condition which can be managed.

- The findings of the study could also assist in developing culturally appropriate guidelines that may be added to the already existing guidelines that are used in the identification and intervention of at risk children with behavioural and comorbid internalising disorders.

1.8.2 Education ministry

- The study coud provide useful information that can assist Limpopo Department of Education that from the drafted policies, be able to create a protocol that may provide support to teachers as early as elementary phase by ensuring that workshops on disorders that are prevalent among school going children will be frequently conducted. The department as a result may benefit from having teachers who will be well informed and creative in handling these children.

- The department may further benefit as teachers may be upfront in terms of screening and referring these children further to the hospital for further management before the problem becomes severe. Moreover, the department may benefit by creating more posts for psychologists to provide psychological services to several schools at district levels.

1.8.3 Educators

- The results of the study could help educators to know how to identify and screen children who display inappropriate levels of inattention and hyperactivity in classrooms during lessons. Moreover, making teachers aware of comorbid internalising disorders (anxiety and depression) which also influence children's functioning.

1.8.4 Parents

- The results of the study could help parents of children with high levels of hyperactivity and inattention to benefit from intervention programs which involve behaviour modification and educational approaches.

1.8.5 Teachers

- To continue to strengthen such intervention programs by providing training and feedback to teachers about strategies discussed with parents. This may further reinforce a reciprocal approach between parents and teachers tohelp at risk children.

1.8.6 Clinicians

- To bring awareness to clinicians who are being refereed these children for further management so that they are more aware of other possible disorders and the role they play on the child's functioning. This may benefit psychologists who give intervention to be vigilant of other existing disorders then make proper assessment, diagnoses and further make proper treatment options.
- The study may further emphasise on the rule which governs psychologist to always administer Disruptive Behavioural Disorder scale (DBD) along with Beck Youth Inventories (BYI-II) scale. This approach may benefit psychologist to arrive at the actual diagnoses and then provide proper intervention.

1.8.7 ADHD children

- The results of the study may encourage children who experience high levels of hyperactivity, impulsivity and inattention as well as internalising disorders (anxiety and depression) to speak openly about their feelings, emotions and difficulties without being stigmatised or labelled regardless of their condition/ diagnosis.

- Above all providing a conducive environment which enables learning and success is essential.

1.8.8 Scholars

- Moreover, scholars who are interested in behavioral and emotional disorders may have more knowledge on how to identifying and screen for learners at risk for behavioural and emotional disorders in primary schools, more especially parents and teachers who interact with these children from time to time.

- The more people are knowledgeable with how ADHD and comorbid internalising disorders affect areas of learning and behaviour, the more likely it is that children who are at risk will be identified earlier and therefore receive immediate intervention for effective functioning. According to Jensen, Hinshaw, Kraemer, Lenora, Newcorn, Abikoff, March, Arnold, Cantwell, Conners and Elliott (2001), clinical and epidemiological studies in most instances have shown that as many as one-third of children with ADHD have co-occurring anxiety and depression. Generally, ADHD in existence comorbid alongside with other disorders impact negatively on children's normal functioning.

1.9 Delimitation of the study

The study focused mainly on school going children attending primary schools. Children with a medical history of neurological problems (e.g. epilepsy, head injuries, cerebral palsy, or cerebral malaria) were excluded from the research project. None of the participants was under the use of psycho-stimulant medication at the time of testing. The study further looked at children diagnosed with Attention Deficit Hyperactivity Disorder (ADHD) and were compared to those without ADHD. These children were further assessed for comorbid internalising disorders i.e. anxiety and depression. Participants were school children from Lepelle Nkumpi Municipality who formed part of the research study.

1.10 Expected outcomes

After the results have been obtained and analysed, the researcher shared the findings with the Department of Education and requested for a collaboration with the department wherein workshops on ADHD may be conducted within the district. The aim of the workshops was based on the findings of the study, that ADHD is persistent

and prevalent among school going children. Moreover, the workshop was intended to inform the educators about the co-existence of ADHD with other psychiatric disorders such as anxiety and depression which may worsen the condition.

Other expected outcomes were to share light on how internalising disorders affect self-esteem, emotions and ademic performance. Most children who are school going experience challenges which later compromise the quality of their education and ability to learn. Therefore, the researcher further requested the school principal from the participating school to educate teachers about ADHD and how to assess it. This also enabled the researcher to create open channels between the school and hospital for further referral whereby identified children can be sent to the hospital for further management. Furthermore, other expected outcomes such as publication of articles and book chapters where information can be obtained will be done.

1.11 Definition of concepts

Attention hyperactivity deficit disorder (ADHD): This is a behavioural disorder common among children and adolescents. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM5), ADHD is a persistent pattern of inattention and/or hyperactivity- impulsivity that interferes with functioning or development with symptoms presenting two or more settings (e.g. at home or school, with friends) and negatively impacting directly on social, academic or occupational functioning (American Psychiatric Association, 2013). The core features of the disorder which are hyperactivity, impulsivity and /or inattention are inappropriate for developmental stage, moreover, ADHD is accompanied by comorbid disorders. In the context of the current study, ADHD is a disorder of the brain which causes harm to a child's behaviour and other areas of functioning which may lead to a child's negative outcomes.

Comorbidity: This refers to the co-existence of more than one disorder at once, other than the existing disorder (Vella, Aragona & Alliani, 2000). As indicated by Angold, Costello and Erkanli (1999), true comorbidity occurs as a result of overlapping symptomatology, that is, one disorder manifesting itself as an earlier form of the other, or from shared risk factors. In the current study, comorbidity is understood as the existing of many other mental disorders in an individual whereas there is already an existing disorder. Comorbidity as being understood in the study is having multiple

disorders at a time, which is overwhelming for an individual to handle.

Internalising disorders: This refers to disorders of emotions often caused by difficulties regulating negative emotions (Terzian, Hamilton & Ericson, 2011). These disorders are often not easily observed or diagnosed, however, manifested themselves through acting out behaviour which is more external such as anxiety and depression, also known as common mental disorders in children and adolescents (In-Albon, 2012). Children usually maniefest anxiety as a sense of being overwhelmed and not calm most of the time. Anxiety in particular is accompanied by a feeling of restlessness and not feeling at ease. Moreover, depression is accompanied by feelings of sadness and emotional withdrawal. Internalising disorders are therefore observed as silent disorders which do not disappear over a period of time while on the other hand having a major impact on behaviour resulting into negative outcomes. In view of the current study, internalising disorders are always not known to the sufferers and those who are in contact with them. Therefore, the intention of the study was to give a broader perspective on the impact of internalising disorders on an individual's functioning.

1.12 Delineation of the study

This thesis is structured into 9 chapters. In the introductory chapter, the aim was to provide background information about ADHD and to present the problem statement regarding the study. In the same chapter, the significance and rationale behind the study were clearly outlined, then followed by the objectives and hypothesis towards the study. In chapter two, various theoretical frameworks/ perspectives about ADHD and comorbid internalising disorders were presented. The chapter also provided a detailed theoretical framework driving the current study.

In chapter three, related literature is reviewed and well presented. General introduction to ADHD, diagnostic criteria, symptoms, and the aetiology of the disorder, and treatment strategies were presented in detail in this chapter. In the same chapter, a detailed description of comorbid disorders and differential diagnosis was clearly outlined. In chapter four, the comorbidity of internalising disorders (anxiety and depression) on ADHD symptoms were highlighted. Chapter five outlines the methodological aspects used in the study. The research design that guided the present study and the approach are also discussed. Data collection and analysis are carefully

outlined and discussed in detail.

Chapter six provides a presentation of results from the participants. In this chapter, presented results are from the ADHD and non- ADHD group. Chapter 7 discusses results in the context of existing literature. In Chapter 8, details on how the guidelines were developed are provided to assist teachers and parents on steps to follow in identifying children with ADHD in the management and treatment of ADHD with comorbid internalising disorders. In Chapter 9, conclusions about the study are provided based on the findings and recommendations targeted to parties concerned are made. In this chapter, the gaps that emerged but which were not the focus of the present study are mentioned. These are the gaps that will serve as future research directions.

1.13 Conclusion

This chapter outlined an introduction on the background of the study, including, the aim of the study, problem statement, hypotheses, objectives of the study, significant of the study and definitions of concepts that are used in the current study. The study went further to investigate the comorbidity of internalising disorders on Attention Deficit Hyperactivity Disorder (ADHD). The existence of these comorbid disorders is about understanding that children with ADHD have internalising disorders and how they impair children's normal functioning.

CHAPTER TWO

THEORETICAL FRAMEWORK

2.1 Introduction

This chapter will focus on the theories or models of ADHD and internalising disorders, and in particular, anxiety and depression. The chapter looked at those theories that guided the current study. Theories of ADHD include: (1) The Dynamic Developmental Theory of Sagvolden et al. (2005), (2) Barkley's Theory of Executive Functions (1997), (3) Brown's Model (2006), (4) Seargeant's Cognitive-Energetic Model (2000) and (5) Sonuga-Barke's Dual-Pathway Model (2002). For this research study, the Dynamic Developmental Theory of ADHD was used as a guideline in understanding the heterogeneity of ADHD. Various viewpoints on the comorbidity of internalising disorders (anxiety and depression) will also be discussed.

2.2 Theories of ADHD

2.2.1 The Dynamic Development Theory

The Dynamic Developmental Theory (DDT) is based on the hypothesis that altered dopaminergic function that is, hypofunctioning mesolimbic, mesocortical and nigrostriatal dopamine branches, plays a vital role by failing to control dopaminergic signal transmission adequately (Sagvolden, Johansen, Aase & Russell, 2005). The theory also recognises neurobiological factors whereby dopamine dysfunction is seen as a core in influencing behaviour manifestation.

It is for this reason why the researcher chose this theory in particular because most behaviours seen in children with ADHD can be as a result of the hypofunctioning of dopamine in those branches. Therefore, behavioural functioning becomes a major challenge when they are compared to their normal counterparts. Behaviors often displayed by children with ADHD and comorbid disorders are impaired continuously and comparatively to children with pure ADHD.

Behaviour as explained by dopamine dysfunction system

Dopaminergic pathways are sets of projection neurons in the brain that synthesise and release dopamine. They have a crucial role in regulating behavioural functioning. Any disruption of dopamine flow within the branches is perceived to affect other neurotransmitter systems and behaviour that is dependent on these systems. A decline in the dopamine level activity in the thinking areas was strongly linked to cognitive problems (learning and memory deficit), poor concentration, difficulty initiating or completing tasks, inability to "lock onto" tasks and lack of motivation (Sagvolden et al., 2005). As indicated by Johansen and colleagues (2002), a dysfunctional dopamine system can be caused by factors such as genetic transmission, environmental pollutants and drug abuse by the mother when pregnant. The following are the three dopamine projection branches: mesolimbic, mesocortical and nigrostriatal systems as they appear on (Figure 2.1).

- *Mesolimbic dopamine pathway*

The pathway connects the ventral tegmental area, which is located in the midbrain, to the nucleus accumbens. The release of dopamine from the mesolimbic pathway into the nucleus accumbens regulates motivation and desire for rewarding stimuli and facilitates reinforcement. A hypofunctioning mesolimbic dopamine branch produces altered reinforcement of behaviour and deficient extinction of previous reinforced behaviour. This gives rise to delay aversion, development of hyperactivity in novel situations, impulsiveness, deficient sustained attention, increased behavioural variability and a failure to inhibit responses (Sagvolden et al., 2005).

- *Mesocortical dopamine system*

The mesocortical pathway connects the ventral tegmentum to the prefrontal cortex. It is essential to the normal cognitive function of the dorsolateral prefrontal cortex and is involved in cognitive control (Executive Functions), like behaviour planning, impulse control, working memory and cognitive flexibility. A hypofunctioning mesocortical dopamine branch will cause attention response deficiencies (deficient orienting responses, impaired saccadic eye movements, and poorer attention responses toward a target) and poor behavioural planning (deficient executive functions).

Therefore, children with ADHD may be cognitively impulsive with a lack of planning, organisation and unable to remember important issues. Due to their impulsiveness, these individuals are inclined to act without forethought. In this regard, a hypofunctioning mesocortical dopamine system may be associated with challenges such as insufficient attention and poor behavioural organisation (Sagvolden et al., 2005).

- *Nigrostriatal dopamine system*

The nigrostriatal dopamine branch usually originates in the substantia nigra and projects mostly to the basal ganglia and controls mostly movement. Therefore, a hypofunctioning dopamine system in children with ADHD is associated with multiple motor dysfunctions like poor motor control (clumsiness, longer reaction time, poor response timing, poor handwriting, and weak coordination) of various body parts (Gillberg, 2003; Meyer & Sagvolden, 2006), and deficient nondeclarative habit learning and memory (Johansen et al., 2002; Sagvolden et al., 2005).

These impairments may lead to marked developmental delay, clumsiness, neurological “soft signs”, and a failure to inhibit responses when fast responses are required. Sagvolden et al. (2005) suggest that the time available for associating behaviour with its consequences will be shorter in children with ADHD than in neurotypical comparisons, if dopamine systems are hypofunctioning. They also indicate that hypofunctioning dopamine systems lead to a deficient behavioural extinction process. This will cause excessive behaviour, usually known as hyperactivity, and increased behavioural variability, that can be interpreted as a failure to inhibit responses. This theory disentangles aspects of various deficient executive functions in ADHD into impulsiveness caused by inefficient reinforcement, deficient extinction of previous acquired behaviour, and impaired motor control. The concept of impulsiveness has both a motor and a cognitive component (Johansen et al., 2002; Sagvolden et al., 2005).

Figure 2.1 Model of the Neurobiological theory of Sagvolden et al. (Johansen et al., 2002)

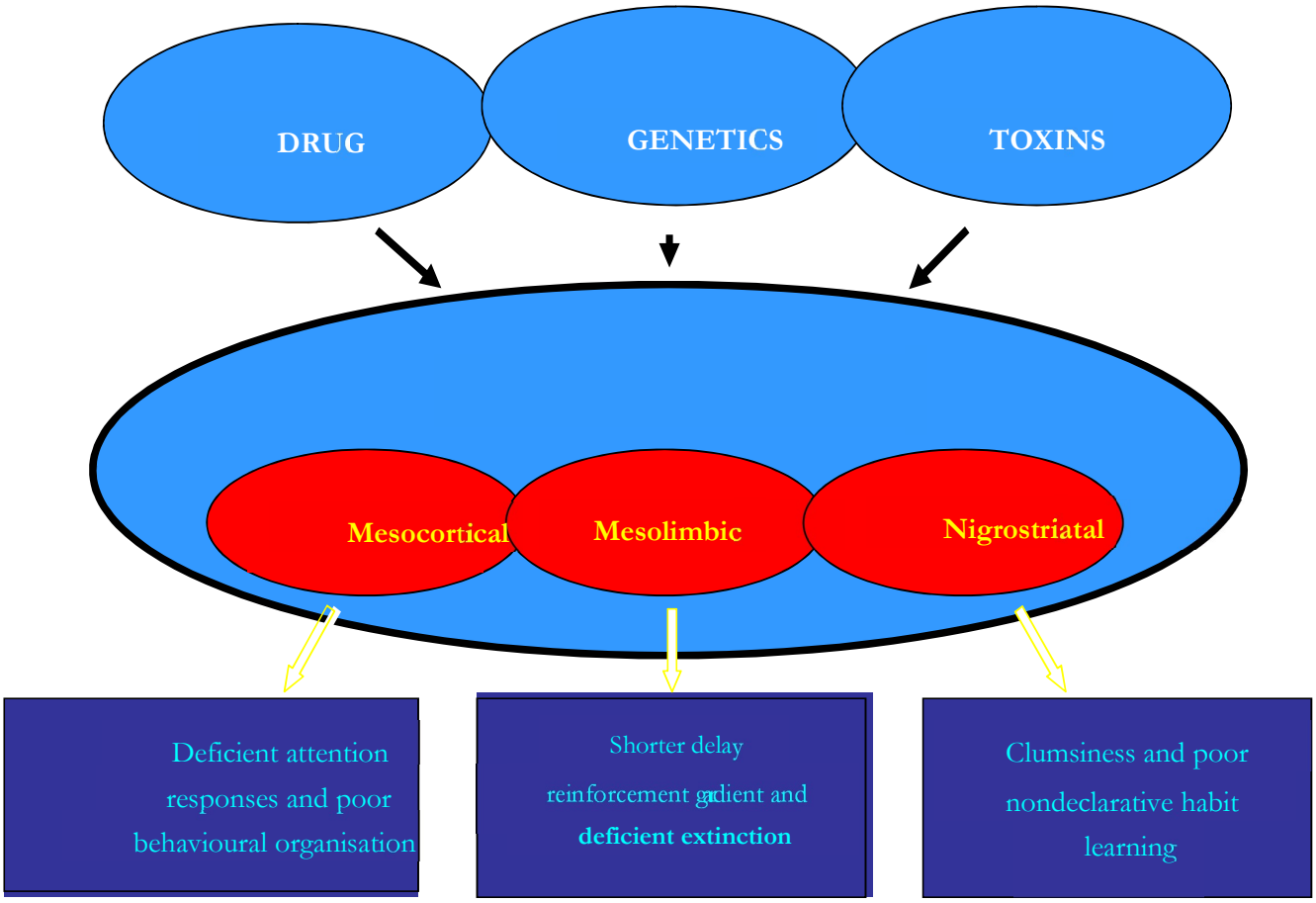


Figure 2.1 shows dysfunction of dopaminergic systems resulting from drug abuse, genetic transmission, or environmental pollutants may cause ADHD symptoms by interacting with frontostriatal circuits.

The exposure of an individual to external factors may also be viewed as influencing an individual in developing the symptoms and behaviours. Behavioural, emotional and cognitive properties can eventually become permanent behavioural patterns which are the result of individual predisposition interacting with the environment. (Sagvolden, Johansen, Aase & Russell, 2005). The developmental theory of ADHD continues to suggest that reduced dopaminergic transmission also bring changes in behavioural mechanisms (Sagvolden et al., 2005). This behavioural mechanism was said to arise as a result of deficient reinforcement of successful behaviour as well as deficient extinction (elimination) of unsuccessful behaviour, and all these are linked to a low

level of dopamine activity (Standford & Tannock, 2012).

- *Altered reinforcement processes*

Children with ÀDHD in most instanes show alterations in reinforcement sensitivity (Luman, Tripp & Scheres, 2010). Hence, more frequent reinforcers are needed to change behaviour (Johansen, Aase, Meyer & Sagvolden, 2002; Sagvolden et al., 1998). Clinical observations indicate that children with ADHD have a "motivation" problem, which means that they often prefer smaller options that are rewarding now and not be in favour in delaying larger rewards in the long term (Sagvolden, Johansen, Aase & Russell, 2005; Sonuga-Barke, 2002). As indicated by Kollins, Lane and Shapiro (1997), stronger and more salient reinforcement will be essential to control their behaviour. Dopamine, in particular, is essential for reinforcement of successful behaviour and reinforcement reduces impulsiveness in ADHD individuals, as these individuals are not always cognitively impulsive, and they are able to manage to plan ahead, organise themselves and remember important matters when the reinforcement schedule is suitable for them (Johansen, Killeen, Russell, Tripp, Wickens, Tannock, Williams & Sagvolden, 2009). Research body thus far has shown that the key symptoms of ADHD being sustained attention, overactivity, and impulsivity, can be seen as a result of altered reinforcement mechanisms and shorter delay of reinforcement gradient (Sagvolden et al., 2005; Sonuga-Barke, 2002).

Methylphenidate (Ritalin) is a dopamine agonist which prevents the re-uptake of dopamine and has a positive effect in improving both hyperactivity/impulsiveness and inattention. Cognitively, many deficient executive functions are identified in persons with ADHD and impulsiveness being caused by altered reinforcement and deficient extinction of behaviour as well as impaired motor control. There are also other neuromodulators affecting the frontal lobe activity, and thus imbalances in more aspects of this system will impact on problems with organising and controlling own behaviour (Rapoport, Van Voorhis, Tzelepis & Friedman, 2001). Motor impulsiveness is predominant in infants and young toddlers, while cognitive impulsiveness is more prevalent in older children and adolescents.

This implies that ADHD impulsiveness can be understood as a maturational lag (Sagvolden, Johansen, Aase & Russell, 2005). Attention problems associated with the ADHD inattentive subtypes are often seen as poor focus of attention and less accurate information processing (Johansen et al., 2002), such problems eventually lead to reading disorders, learning disability which may be related to a reduced IQ (Sagvolden et al., 2005).

2.2.2 Barkley's Model of Executive Functions and ADHD

According to Barkley (1997), deficits in behavioural inhibition is the primary cause of ADHD, and as a result, creates disturbances in five neuropsychological functions such as working memory; internalisation of speech; self-regulation of affect; behaviour analysis and synthesis; and motor control, called the Executive Functions (EF). Executive Function refers to brain functions that activate, organise, integrate and manage other functions. It enables individuals to account for short-term and long-term consequences of their actions and to plan ahead. It allows a person to make evaluations of their actions and make the necessary adjustments when those actions do not have the desired results. Barkley (1997) breaks EF down into the following four areas:

Nonverbal working memory: It is the temporary or working memory because it represents remembering to do. Children with ADHD were found to be less well controlled by internally represented information than normal children. They are more controlled by external stimuli and have difficulties with nonverbal working memory, planning and a sense of time (Barkley, 2001).

Internalization of speech (verbal working memory): Internalisation of speech is the process of turning speech on the self in the form of dialogic conversation with oneself that becomes increasingly more private, covert and internalised. This is the main contributor to the development of self-control (Barkley, 1997).

Self-regulation of affect/motivation/arousal: There are three essential functions provided by the prefrontal lobes, which create self-regulation. Firstly, there must be a system that provides for the inhibition of more automatic and dominant responses that

have as their function the maximization of immediate consequences. Secondly, the inhibitory system provides for the power to interrupt ongoing behavioral patterns should information from immediately past behaviours in the sequence indicate errors or ineffectiveness of the going pattern. Thirdly, this inhibitory system functions to control potential sources of interferences that could disrupt or destroy the activities taking place within the working memory (Barkley, 2001).

Behaviour Analysis and Synthesis: This is the ability of humans to generate novel, complex, hierarchically organised, and goal-directed behaviour, and planning. Goal-directed behavioral flexibility and creativity are believed to reflect the executive function of reconstitution (Barkley, 2001) .Barkley's model is based on the idea that inabilities to self-regulate lie at the root of many challenges faced by persons with ADHD. He explains that a person with ADHD may be unable to delay responses, therefore acting impulsively and without sufficient consideration of future consequences, either positive or negative (Barkley, 1997).

2.2.3 Brown's Model of ADHD

The model was developed by Brown in 2006 following a period of 25 years of clinical interview and research with children, adolescents and adults presenting with ADHD. The model fully paid attention and was able to explain executive functions and the cognitive management system of the human brain and in particular that of an ADHD individual. The model was based on the view that ADHD involves developmental impairments of executive functions. The model depicts the following six separate clusters which appear to function interchangeably to help one manage many tasks of daily life:

(a) Organising, prioritising and activating for tasks (Activation). This refers to one's ability to plan their duties in order, however in children with behavioural disorders it can be different since they are more disorganised and easily become overwhelemed by tasks.

(b) Focusing, sustaining, and shifting attention to task (Focus). This may suggest one's ability to remain and fully achieving a certain task before moving to another without being distructed, which can be complex task in the case of an individual with ADHD as they easily get distructed outside stimuli affecting more of their attention.

(c) Regulating alertness, sustaining effort, and processing speed (Effort). This reflects one's total ability to pay attention, utilising and ability to retrieve information learned. This is an area where ADHD individuals' struggles with shifting and retrieving previously learned information. Due to failure, individuals in most instances refraim from trying to attempt hence they easily give up.

(d) Managing frustration and modulating emotions (Emotion). It can be expected that one is able to regulate his/her feelings and emotions. Children with ADHD in most instances they easily outburst with emotions and they are irrational in their decision making as a result. In this regard they are not able to handle frustration hence they are impulsive as a result.

(e) Utilising working memory and accessing recall (Memory). This enables one's abilty to apply and utilise past learned information for the purpose of future goal. Children struggling in this area mostly struggles with manipulating concepts and as such becomes overwhelmed. Not being able to recall may impair their ability to learn.

(f) Monitoring and self-regulating action (Action)

Some of these impairments agree with views from other models like those of Barkley (1997), Sagvolden et al., (2005) and Sergeant (2000). Not being able to control own actions may result to impaired behaviour later in life. Most children with impaired behaviour result are likely to engage in multiple risk behaviours.

According to Brown, it is very typical of individuals with ADHD to experience difficulties in getting organised, initiate work-related tasks and in self-activating daily routines. They tend to be forgetful as they have difficulties in recalling learned tasks. Problems with sustained attention, maintenance of energy for work-related tasks, daytime drowsiness, slow processing of information, inadequate task completion are the results. Mood fluctuations, frequent frustrations and lack of motivation are some of the symptoms observed in persons with ADHD.

2.2.4 Cognitive-Energetic Model

The model suggests that there may be certain aspects of inhibition which may be deficient in ADHD children and that this depends on the energetic state of the child (Sergeant, 2000). This model focuses on the ADHD deficiency at three distinct levels of information processing: cognitive mechanisms such as response output, energetic mechanisms such as activation and effort and control systems of executive functioning.

The model suggests that disruptive disorders have common deficiencies in EF control systems and may possibly differentiated either at an energetic level or at specific cognitive stages. Thus, the cognitive-energetic model tries to incorporate both top-down and bottom-up processes (Sergeant, Geurts, Huijbrechts, Scheres & Oosterlaan, 2003). The cognitive-energetic model does not suggest a single EF deficit in ADHD. It supposes that a range of EF-functions can cause differences in symptomatology (Sergeant, 2000).

The model suggests that differences between ADHD and ODD/CD are the result in terms of reward mechanisms which influence inhibitory control (Smith & Jonides, 999). Anxiety disorders are predicted to be oversensitive to signals of punishment or reward. The model further emphasises that nonspecific anxiety and arousal in ADHD divert attention from the focused task, i.e. causing poor behavioural activation (Schatz & Rostain, 2007). In children with ADHD and comorbid anxiety, the level of information processing is rather slow and thus causes impairment on behaviour as seen in ADHD.

2.2.5 Dual Pathway Model of ADHD

This model has been developed by Sonuga-Barke (2002). It holds the view that ADHD can be seen as the result of two dissociable psycho-patho-physiological pathways, mediated by distinctly different psychological processes and which is imbedded in functionally segregated and yet related brain circuits. The model has developed from bringing together executive dysfunction and delay aversion. ADHD is represented as a disorder of dysregulation of cognition, action, and cognitive-energetic state. The model supposes that the brain-behaviour link in ADHD is interceded by neuropsychological deficits associated with dysregulation of behavioural and cognitive activity leading to dysfunctional patterns of action and engagement. Moreover, the link

between ADHD and EF deficits justifies portrayal of ADHD as an EF disorder as deficits in working memory, planning, and set-shifting have been identified (Sonuga-Barke, 2003).

There seems to be a relation between ADHD and hypersensitivity to delay and consequent difficulties in waiting for desired outcomes and working effectively over extended period. Impulsiveness also implicates that children who are delay-aversive cannot escape or avoid actual delay (under fixed time conditions). Therefore, behaviours which create non-temporal stimulation such as fidgeting, can be regarded as hyperactivity, while attention to non-temporal stimuli redirecting children from the tasks they have to complete, could indicate inattentiveness. This model emphasises that a biologically based shortened delay reward gradient leads to a tendency to discount future rewards and an associated preference for immediate rewards (Sonuga-Barke, 2003).

2.3 ADHD and comorbid disorders

Psychiatric comorbidity is a clinical important dimension of ADHD heterogeneity. According to Faraone, Asherson, Banaschewski, Biederman, Buitelaar, Ramos-Quiroga and Franke (2015), only a small proportion of individuals with ADHD is free of comorbidity while at the other end of the spectrum, some patients show a complex pattern of multiple problems, including communication disorders, intellectual disabilities, sleep disorders, substance use disorders, specific learning disabilities, externalising disorders (ODD and CD) and internalising disorders (anxiety and depression), which are the focus of the present study.

Little is known about how comorbidity affects ADHD symptomatology and related deficits like neuropsychological functioning. Of research on comorbidity, particular comorbid disorders have received more attention than others. For example, externalising disorders (ODD and CD) are present in approximately 50% of children with ADHD and much research has been done in this area (Newcorn, Halperin, Jensen, Abikoff, Arnold, Cantwell, Conners, Elliott, Epstein & Greenhill, 2001). Although comorbidity with these disorders is very high, it is also important to note that internalising disorders such as anxiety and depression are comorbid in approximately 25% of children with ADHD in the general population, and around 30% to 40% of

clinical referred children (Tannock, 2009).

2.3.1 Anxiety

It has been suggested that ADHD with comorbid anxiety disorder may be a distinct entity (Jensen et al., 2001). Better interpreting anxiety in ADHD will advance the treatment choices (Tsang, Kohn, Efron, Clarke, Clark, Lamb & Williams, 2015). There are indications that stimulant medications may be less effective and produce more severe side effects when ADHD is comorbid with anxiety disorder, even though these medications are standard treatment for ADHD (March, Swanson, Arnold, Hoza, Conners, Hinshaw & Abikof, 2000). Anxiety is comorbid with ADHD in 25%-30% of cases (Bedard & Tannock, 2008; Jensen, 2009; Larson, Russ, Kah & Halfon, 2011; Tsang et al., 2015). Children with anxiety disorder may suffer from general Anxiety Disorder (GAD), Social Anxiety Disorder (SAD) or even Obsessive-Compulsive Disorder (OCD) (American Psychiatric Association, 2013). Most studies find that GAD is the most common anxiety disorder that is concurrent with ADHD symptoms (Elia, Ambrosini & Berrettini, 2008).

The highest rate of anxiety disorder is found in the combined subtype/presentation of ADHD-Combined (ADHD-C) (Levy et al., 2005; Tsang et al., 2015). When the genders are compared, girls, with and without ADHD, show more anxiety symptoms than boys (Littman, 2012; Skogli, Teicher, Andersen, Hovik & Oie, 2013). Several studies found that children with ADHD and comorbid anxiety diagnoses have a greater severity of ADHD symptoms than children who do not have anxiety symptoms. This may have significant clinical and practical implications for intervention methods for ADHD (Faraone et al., 2015).

Neurobiology:

Anxiety traits may be a result of impairment in the Mesolimbic Dopamine (DA) system (Levy, 2004). The DA system serves as a domain where reward and delay of reinforcement are determined by tonic/ phasic DA relationships, which result in impulsive "fearless" response (Levy, 2004). Impairment in these dopaminergic systems will result in provoking anxiety traits which reflect ADHD symptoms like impulsivity (Levy, 2004). The theory is also in line with the DDT model which

emphasizes dopamine disruption in the dopaminergic branches (Sagvolden et al., 2005).

Comorbid anxiety in children with ADHD is then viewed as a result of an impaired synaptic process which has fear response and aggression (Levy, 2004). Therefore, the impairment to the pre-frontal cortex will continue to cause more negative inputs on the amygdala (which is an area of the brain responsible for decision making) and more anxiety related processes (Levy, 2004). Impairment in both dual pathways, that is, the phasic mesolimbic DA relationships and prefrontal cortex, as well as hippocampal inputs will have implications for comorbidity in ADHD (Levy, 2004). Increased amygdala activity corresponds to more emotional experience as persons with anxiety disorders show aggravated amygdala response (Etkin & Wagner, 2007).

Children with comorbid ADHD and anxiety function poorly at school and receive more mental health services than those with ADHD or anxiety only (Hammerness, Geller, Petty, Lam, Bristol & Biederman, 2010). The inability to function academically may be caused by increased fear of failure which interferes with performance (Vloet, Konrad, Herpetz-Dahlmann, Polier & Gunther, 2010).

2.3.2 Depression

Childhood depression is a common, serious and persistent mental health problem that has a negative impact on the academic, behavioural and social development of the child (American Psychiatric Association, 2013). Children with ADHD display higher rates of depression than children without ADHD. Studies show that the rate of depression in children with ADHD in community samples ranges from 12% to 50% (Angold et al., 1999; Daviss, 2008). Comorbid depression in clinically referred children occurs even at higher rates (Daviss, 2008). Symptoms of depression in children occur several years after the onset of ADHD symptomatology (Daviss, 2008).

Depression is more associated with the inattentive (ADHD-PI) and combined (ADHD-C) subtypes than with the hyperactive/impulsive subtype (ADHD-HI) (Power, Costigan, Eiraldi & Leff, 2004; Chronis-Tuscano, Molina, Pelham, Applegate, Dahlke, Overmyer & Overmyer, 2010; Ulloa, Sánchez, Sauceda & Ortiz, 2006; Volk, Neuman & Todd, 2005). The comorbidity of ADHD and depressive disorders increase the severity of

attention problems. This can be explained by the presence of inattention symptoms in clinical depression, which may increase the problems with sustained attention that is typical of children with ADHD (Di Trani, Roma, Elda, Daniela, Pasquale, Silvia & Renato, 2014). The presence of impulsiveness as in the ADHD-C subtype may predict a suicide risk (Tuscano et al., 2010). Most studies on ADHD and comorbid depression found significant gender differences with girls with ADHD having a higher incidence of depression symptoms than boys (Angold, Costello & Erkanli, 1999; Barkley, 2014; Biederman, 2008; Gershon, 2002). However, a study by Bauermeister, Shrout and Chávez (2007) could not find gender differences in the rates of depression among children with ADHD.

Theoretical explanations:

There are several theories that explain the link between ADHD and depression. Daviss (2008) suggests that children with ADHD are more prone to negative feedback at school, in social situations and in the family environment. This negative feedback results in low self-esteem, demoralisation and ultimately, depression. However, this hypothetical relationship is not supported by empirical evidence. Studies have shown that children with the most severe symptoms of ADHD would more likely receive more negative feedback, and not the most affected by depression (Posner, Siciliano, Wang, Liu, Sonuga-Barke & Greenhill, 2014). Another theory suggests that ADHD associated vulnerabilities to depression in some children due to genetically shared neurobiological anomalies. An interaction between neurobiological vulnerabilities and environmental effects, may have stronger effects on children with ADHD who carry vulnerabilities for depression (Posner et al., 2014). It has been suggested that the combination of ADHD and a depressive disorder can represent a subtype of ADHD, with shared and specific features related to aetiology, outcome and clinical presentation.

2.3.3 Self-esteem

Self-esteem is an individual's sense of his or her value or worth or the extent to which a person values, approves or appreciates, or likes himself or herself (Blascovich & Tomaka, 1991). It is generally considered as the evaluative component of the self-concept, a broader representation of the self that includes cognitive and behavioural aspects as well as evaluative or affective ones (Blascovich & Tomaka, 1991).

According to a previous study, a primary school child with ADHD frequently begins to be seen as being different as classmates start to develop the skills and maturity that enable them to learn successfully in school. Although a sensitive teacher may be able to adapt the classroom to allow an able child with ADHD to succeed, more frequently the child experiences academic failure, rejection by peers and as a result, low self-esteem (Landgraf, Abetz & Ware, 1999).

Research on the relationship of ADHD and the development of self-esteem is inconclusive, as some studies indicate that these individuals often have low self-esteem, and others report that children and adolescents with ADHD often overestimate their abilities, perceptions of self and self-concepts. Thus, as a protection mechanism, they enhance the appreciation of happiness that they feel with their lives which makes it difficult to see the impact of ADHD on the self-esteem of children (Glass, Flory, Martin & Hankin, 2011; Miranda-Casas, Presentacion-Herrero, Colomer-Diago & Rosello, 2011).

Several studies found high rates of peer rejection for children displaying ADHD-related behaviours (Hinshaw, Zupan, Simmel, Nigg & Melnick, 1997; Hodgens, Cole & Boldizar, 2000). This peer rejection of children with ADHD leads to children being isolated and low self-esteem. The rate of peer rejection is particularly high for children displaying both aggression and ADHD. Peer rejection status is stable over time, reflecting the chronic nature of these children's interactional difficulties (Parker & Asher, 1987). Children with this disorder perceive peers and classmates at school as less frequent social support than do their non-ADHD counterparts (Demaray & Elliot, 2001).

Sisto and de Cássia Martinelli (2004) found that experiencing failure in performance on academic tasks can generate feelings of insecurity and lack of confidence in children, as studies are the main activity during childhood and adolescence. As it is characteristic of most children with ADHD, there is an impact on school performance. It is likely to impact on the formation of school self-concept, which is related to the representations of one's accomplishments, school abilities and assessments that the person does about themselves (Sisto & de Cássia Martinelli, 2004). Children's self-esteem has been found to be sensitive to the perception of parents, teachers and peers (Wheeler & Carlsson, 1994). Findings regarding the nature of self-esteem in children

with ADHD are contradictory. Hoza, Gerdes, Hinshaw, Arnold, Pelham, Molina and Odbert (2004) reported that children with ADHD tend to overestimate their own competence, reporting an inflated estimation of self-worth called positive illusory bias. They overestimate their perceptions of themselves most strongly in areas where they have the greatest skill deficit (Hoza et al., 2004).

Barber, Grubbs and Cottrell (2005) reported lower self-esteem in children with ADHD when compared to controls. They reported to be lower on the subscales for behaviour, scholastic skills, physical capacity and global self-worth. Barber and colleagues (2005) also explained the influence of peers and stated that children of school age are extremely sensitive of peer approval, evaluating themselves and their competence. In terms of academic or athletic abilities, the authors recommended school-based support groups to improve self-esteem in children with ADHD (Barber et al., 2005). They noted that the cumulative effect of years of low self-esteem might have major consequences. Pisecco, Wristers, Swank, Silva and Baker (2001) found that children's early history of behavioural problems and low self-esteem were associated with the development of more serious social problems. They also found that social rejection causes emotional pain in the lives of ADHD children, which may result in low self-esteem (Pisecco et al., 2001).

According to a study by Bussing, Zima, and Perwien (2000), ADHD alone does not appear to be associated with lower levels of self-esteem in school age children, but rather the presence of co-occurring internalising problems, either alone or in combination with an externalising disorders like disruptive behaviour disorders, could result in a lower self-esteem.Negative responses by parents, teachers and peers to the affected child's impulsivity and hyperactivity may contribute to his or her feelings of low self-esteem (Barkley, 2006). The teachers constantly reminding the students to behave, sit quietly, and pay attention may create a negative self-image in these children, which can have a negative impact on the ability to make friends. The possible biological influences on impulsivity, hyperactivity and inattention combined with attempts to control these children may lead to rejection and poor self-esteem (Barkley, 2006).

2.3.4 Disruptive behaviour disorders (Externalising)

Externalising disorders are characterised by maladaptive behaviours directed towards an individual's environment, which causes impairment or interference in life functioning (Jensen, Hinshaw, Kraemer, Lenora, Newcorn, Abikoff & Conners, 2001; Kolko, Dorn, Bukstein & Burke, 2008). Oppositional behaviour, aggression, and antisocial behaviour are the psychiatric symptoms most frequently comorbid with ADHD. They are often the symptoms most troubling to parents and teachers (Pliszka, 2003). About half of children with ADHD will meet criteria for either Oppositional Defiant Disorder (ODD) or Conduct Disorder (CD). Similarly, a large percentage of children with ODD/CD will in turn meet criteria for ADHD (Pliszka, 1998). The prevalence of comorbid ODD and CD in clinical samples is 50% to 65% with co-occurring ODD and about 20% with co-occurring CD (Kadesjo, Hagglof, Kadesjo & Gillberg, 2003; Posner et al., 2007; Wilens, Biederman & Spencer, 2002). In community-based samples, the prevalence is lower, with about 20% of children having co-occurring ODD and 14% CD (Wichstrom, Berg-Nielsen, Angold, Egger, Solheim & Sveen, 2012). Evidence indicates that this comorbidity is more common in boys than in girls (Biederman, Mick, Faraone, Braaten, Doley, Spencer & Johnson, 2002).

Regarding the comorbidity between CD, ODD, and ADHD, existing literature seems to be sufficiently consistent to conclude that it may be primarily attributed to genetic influence (Dick, Viken, Kaprio, Pulkkinen & Rose, 2005; Martin, Levy, Pieka & Hay, 2006; Nadder, Rutter, Silberg, Maes & Eaves, 2002; Tuvblad, Zheng, Raine & Baker, 2009) and unique environment effects (Derks, Hudziak & Boomsma, 2007; Kerekes, Lundström, Chang, Tajnia, Jern, Lichtenstein & Anckarsäter, 2014; Tuvblad et al., 2009). There also seems to be sufficient literature to state that children with ADHD have a predisposition to manifest behaviours that are common to ODD and CD, including the antisocial behaviour that these children often display (Nadder et al., 2002).

There is a strong relationship between CD and ODD (Rowe, Maughan, Pickles, Costello & Angold, 2002), which seems to share genetic influences (Knopik, Bidwell, Flessner, Nugent, Swenson, Bucholz & Heath, 2014), specifically a unique genetic influence that increases the tendency for externalisation in both disorders (Dick et al.,

2005) and, most likely, in ADHD, although each disorder may also have its own genetic characteristics.

2.4 Conclusion

Various theoretical perspectives were presented on how ADHD is linked to the impaired neurotransmitters. Dopamine dysfunction transmitter remains the crucial role in the manifestation of all symptoms and behaviours related to ADHD and may play a role in comorbid disorders. Other neurotransmitters such as serotonin and noradrenaline are also implicated. The manifestation of behaviours and symptoms such as inattention, impulsivity and hyperactivity are highly regulated by the influx of the secretion of these transmitters, especially dopamine. Other theoretical frameworks emphasised executive functioning as playing a pivotal role in the psychopathology of ADHD.

Psychiatric comorbidity is another clinically important dimension of ADHD heterogeneity. At one extreme, a small proportion of clinic-referred individuals are free of co-morbidity; at the other end, some patients have a complex pattern of multiple problems, including mood disorders and anxiety (internalising) disorders, disruptive behaviour (ODD and CD, externalising) disorders, and low self-esteem, among many more. Consideration of a patient's comorbidity profile is important, as it will influence treatment planning (Faraone et al., 2015). Although the focus of the present study is on the comorbidity of ADHD with internalising disorders, the role of self-esteem and a comparison of internalising disorders with the occurrence of externalising comorbid disorders will also be addressed.

CHAPTER THREE

ATTENTION DEFICIT HYPERACTIVITY DISORDER

3.1 Introduction

The literature review for the current study was done through several engine search such as (PubMed, Google-Scholarand PsychInf) using the following terms: ADHD, anxiety, comorbidity, depression, DSM criteria, internalising disorders, and treatment. Journal articles published between 1999 and 2018 were used as a source of obtaining information. The researcher carefully assessed the abstracts of journals obtained from the engine searches and also acquired full-text of those journals.

This chapter provides an overview of attention deficit hyperactivity disorder among children with regard to its clinical description, prevalence, etiology or causes, other contributing factors, as well as various methods of treatment to the disorders. Attention Deficit Hyperactivity Disorder as expressed in the Diagnostic Statistical Manual of Mental Disorders (DSM-5), according to Sadock and Ruiz (2015), it is a neuropsychiatric condition affecting preschoolers, children, adolescents, and adults around the world and is characterised by a pattern of diminished sustained attention, and increased impulsivity or hyperactivity. The onset of the disorder is prevalent from as early as childhood i.e. from the early age of 3, but becomes clearly visible and diagnosed by the age of 7 going upwards. The presence of ADHD symptoms can only be confirmed with impaired inattention and or hyperactivity and impulsivity which can be observed in at least two settings i.e. school and home environment and must have been seen to be interfering with the child's normal functioning. It is during this period that children are quite expected to be able to pay attention and follow instructions especially in areas such as home and school environment. In her study, Anderson (2000) found that at the age of 7, executive functioning is fully developed. Executive functioning therefore enables one's ability to plan, organise, process information and thinking is fully developed, hence a lot can be expected from these children.

Historically, ADHD is predominantly understood as a childhood disorder. ADHD is a diagnostic disorder mainly for children with substantial difficulties with attention, impulsivity and overactivity. In the early twentieth century, ADHD was first recognized as a disorder in 1902 by a British doctor, Dr. Still (Lange, Reichl, Lange, Tucha & Tucha, 2010). He labeled the disorder as "Defect of Moral Control" because he strongly believed that the disorder was as a result of a medical problem more than anything else. Observation showed that children with "moral control" displayed some strange abnormal defects which were in relation to cognitive relation to environment (capacity for reasoning comparison), moral consciousness (intellectual capacity) and volition (Lange et al., 2010). They further expressed that other concepts referring to the disorder such as brain damage, minimal cerebral dysfunction and hyperactivity were seen as a sign to ADHD.

The disorder was later described and diagnosed as "post encephalitic behaviour disorder" around 1917-1918 due to the effect it had on the brain and behaviour which subsequently led to the disorder ADHD (Barkley, 2006; Lange et al, 2010). The impact caused by encephalitis was found to have caused extreme damaged children physically and mentally as indicated by Rafalovich (2001). Many of those children who survived the condition displayed remarkable abnormal behaviour which led to significant change in personality, emotional instability, cognitive deficits, learning difficulties, sleep reversals, depression and poor motor control (Conners, 2000) which are some character traits defining ADHD (Barkley, 2006).

Since then, the criteria for diagnosis have continually changed and in 1968, the first criteria appeared in the DSM II (Diagnostic and Statistical Manual). These were modified in 1980 in the DSM III, then again in 1987 (DSM III R). Significant changes were introduced to the next editions, DSM IV in 1994 and DSM IV TR in 2000. Later literature suggests the presence of the following distinctive subtypes of the disorder: • Inattentive Subtype (pure ADD) • Hyperactive – Impulsive Subtype (classic ADHD) • Combined Subtype • ADHD with Co-morbidities (Presented at the 2009 AAPS Annual Scientific Meeting, San Diego, June 23, 2009 Nagui Hanna, MD). ADHD in existence often comorbid with either one or two psychiatric disorders which can be regarded as internalising and externalising disorders. For the purpose of the current study the researcher will only limit herself on internalising disorders i.e. anxiety and depression.

According to In-Albon (2012), internalising disorders are the most common mental disorders in children and adolescents. Developmentally, children are often regarded to be immature to express their emotions hence through acting out behaviour or poor scholastic performance one could identify if the child is having a crisis. Often children's emotional state is ignored and not being attended to, and this may eventually become permanent and therefore pose a risk for the development of further mental disorders later in life (In-Albon, 2012). Because these children continue to suffer in silence for long without receiving adequate treatment, internalising disorders deserves more attention in relation to early recognition, prevention and better management (In-Albon, 2012).

Comorbid internalising disorders may be observed to have a negative impact on children with normal functioning if diagnosed with ADHD as compared to their normal counterparts. In their study, Carter, Wagmiller, Gray, McCarthy, Howrwitz and Briggs-Gowan (2010) expressed that 1 out of 5 children continue to meet the criteria for a mental disorder with impairment. In support to this perspective, In-Albon (2012) further emphasised that anxiety disorders in particular are the most common mental disorders in children, then followed by attention deficit and hyperactivity disorder (ADHD) and aggressive behavioural disorders. It can be understood that young children may suffer from mental illness although at the most they are not easily noticed.

Other behaviours in children become noticeable when they are unable to cope in a home and school situations, especially if the behaviour affects normal daily functioning and is continuing for a period of six months. Therefore, the comorbidity of other psychiatric disorders may complicate what is supposed to be known as pure ADHD. In a study conducted by Baldwin and Dadds (2008), children who were clinically diagnosed as having ADHD were found to be highly co-existing with a range of other psychiatric disorders. In support to this perspective, most of the children who are sufferers of these psychiatric disorders become rejected by their peers, have low self-esteem and emotional problems. As such, children with ADHD continue to have impairment in academic functioning, social and interpersonal situations quality of life become compromised and they continue to meet great challenges in social functioning (Sadock & Ruiz, 2015).

3.2 Prevalence of ADHD

Erskine, Ferrari, Nelson, Polanczyk, Flaxman, Vos, Whiteford and Scott (2013), found ADHD to have a worldwide prevalence of 5.29% in children aged 18 years and younger. The disorder is generally more prevalent in males than in females (American Psychiatric Association, 2013). The Global Burden of Disease Study 2010 for the first time has profiled ADHD and Conduct disorder (CD) as one of the burden of diseases affecting children (Erskine, Ferrari, Nelson, Polanczyk, Flaxman, Vos, Whiteford & Scott, 2013). Common symptoms among children manifest themselves thorough poor scholastic performance, rejection by their peers, learning problems and aggression (Biederman, 2005). In areas such as Africa where the population of children constitute up to 40%, the prevalence of ADHD among school children ranges from 5.4% to 8.7% respectively (Bakare, 2012). Although most studies on ADHD were conducted in European countries, few studies were also done locally around ADHD showing that the disorder is prevalent in other regions. Emerging research documents on ADHD were from South Africa, Nigeria and Democratic Republic of Congo.

Various studies conducted in African countries such as Democratic Republic of Congo on co-existing symptoms and risk factors among African school children with ADHD symptoms among school children was at 6% (Kashala, Lundervold, Sommerfelt, Tylleskar & Elgen, 2006). Another study conducted in Ethopia on the prevalence of mental and behavioural disorders in children showed the prevalence of disorder at 1.5% (Ashenafi, Kebede, Desta & Alem, 2001). The prevalence was quite small but yet informative. In a study conducted in Nigeria on ADHD among Nigerian primary school children, the prevalence was at 8.7% range. Another study conducted on problems and pro-social behaviour among Nigerian children with intellectual diasability also found the prevalence of 8.7% (Bakare, Ubochi, Ebigbo & Orovwigho, 2010). Several studies done in South Africa also showed the prevalence of ADHD to be at 5% (Meyer, Eilertsen, Sunde, Tshifularo & Sagvolden, 2004; Zeegers, Rabie, Swanevelder, Edson, Cotton & Van Toorn, 2009). Although the time prevalence of ADHD does not differ with ethnicity, a notable study was able to show that the prevalence of ADHD does not differ between countries in Europe, Asia, Africa, America and including Australia (Polanczyk, Willcut, Salum, Kieling & Rohde, 2014).

3.3 ADHD and gender differences

The manifestation of ADHD may be influenced by a number of factors, such as gender, age, and comorbid disorders. ADHD affects boys more than girls with a male to female ratio of 3:1 in a population based studies (Barkley, 2006) and between 5:1 to 9:1 in a clinical sample (Graetz, Sawyer, Hazell, Arney & Baghurst, 2001). As stated by Sciutto and Eisenberg (2007), girls continue to remain underidentified and underdiagnosed because of the expression of the disorder. Dirlikov, Rosch, Crocetti, Denckla, Mahone and Mostofsky (2015) found sexed-based difference in the cortical morphology of functions in the frontal lobe suggest the difference in the expression of the symptoms between boys and girls.

In comparison between females and males, findings revealed that females with ADHD had fewer hyperactive/impulsive symptoms and more inattentive symptoms when compared to males with ADHD (Biederman, Kwon, Aleardi, Chouinard, Marino, Cole, Mick & Faraone, 2005). Affected boys with ADHD were therefore observed to show problems in the way of hyperactivity/impulsiveness (Taylor et al, 1998). Gender differences in terms of presentation of ADHD symptoms showed that boys were mostly diagnosed with combined type (ADHD-C) or hyperactive/impulsive (ADHD-HI), while girls are mostly diagnosed with the predominantly inattentive presentation (ADHD-PI) (Pila-Nemutandani & Meyer, 2016). However, both genders showed some impairments in executive functions. Since ADHD symptoms at the most are characterised by externalising behaviour, it was observed that boys with ADHD had more externalising disorders comparatively to normal developing boys, while on the other hand females had the tendency to display more of internalising disorders when compared to normal girls (Quinn, 2008; Gershon, 2002). As indicated by Rucklidge (2010), less disruptive behavior in females with ADHD may lead to referral bias even though they display equal level of impairment (Sciutto, Nolfi & Bluhm, 2004).

Quinn (2008) found that the diagnosis of ADHD in females in particular can be overlooked due to the presence of co-existing symptoms which often cloud the diagnostic image. About 75% of children having ADHD probably have one other psychiatric disorder. According to Skogli, Teicher, Andersen, Hovik and Oie (2013),

co-existing psychiatric problems appear to be common in a clinical practice, in which females with ADHD were found to show more of internalising disorders while boys have externalising disorders. Epidemiological studies on the other hand are of the opinion that internalising disorders both in girls and boys occur with same frequency during childhood (Merikangas, He, Burstein, Swanson, Avenevoli, Cui, Benjet, Georgiades & Swendsen, 2010), unlike during adolescents wherein female adolescents display higher prevalence of internalising disorders than male adolescents.

Both males and females with ADHD were found to be more impaired and disturbed when compared to their gender matched non- ADHD counterparts (Gershon, 2002). However, females with ADHD were observed to be specifically impaired in inattentiveness than males with ADHD (Gershon, 2002). Gerson (2002) and Levy, Hay, Bennett and McStephen (2005) are of the opinion that girls with ADHD display a higher rate of internalising symptoms especially anxiety than boys. In support of this view, Rucklidge and Tannock (2001) argue that girls with internalising disorders continue to exhibit less aggressive, peer aggression, lower levels of inattention (Levy et al., 2005) and disruptive behaviour, but are more at a risk of substance abuse.

Females with ADHD are equally impaired as males with ADHD in terms of cognitive functioning, neurological disorders and academic problems than normal boys (Tannock, 2005). However, impairment in response inhibition, working memory, planning and time perception was found to be more prevalent in both genders (Toplak, Rucklidge, Hetherington, John &Tannock, 2003). These behaviours are not easily seen by parents or teachers compared to physical behaviors which girls tend to show less frequently.

3.4 Clinical symptoms of ADHD

The main clinical symptoms which characterised the disorder are inattention, hyperactivity and impulsivity. In most cases, these symptoms are pronounced and therefore having a negative impact on behaviour which is often observed and defined as inappropriate. The symptoms are mostly understood as a result of various factors such as faulty executive functions, deficient behavioural inhibition, and dopamine dysfunction in the brain. As a result, the manifestation of inappropriate behavioural

development by children becomes clearly observed a lot more.

3.4.1 Deficient sustained attention

Inattention as one of the key symptoms of ADHD, is characterised by short attention span, distractibility, perseveration, failure to finish tasks, inattention, poor concentration, an inability to control attention in response to extremely imposed demands, challenges in initiating tasks or difficulties in selectively attending to relevant stimuli while filtering out unnecessary noise (Sadock & Ruiz, 2015). Similar challenges can be experienced in children with comorbid disorders such as internalising disorders because of the shared symptoms with ADHD.

According to Baldwin and Dadds (2008), young children with inattention problems, often have difficulties completing their schoolwork which may consequently lead to negative outcomes. This problem frequently arises when one is given boring, tedious, protracted or repetitive activities which are less appealing to them. Children with ADHD often fail to show the same amount of persistence, motivation and will power of others their age when uninteresting, however, important task must be performed (Barkley & Murphy, 2006). Again, children with ADHD would report becoming bored with such tasks hence will shift from one uncompleted activity to another without completing these activities. It is therefore common for them to lose concentration while taking tedious boring tasks. Therefore, they become easily distracted during periods and as a results may have problems with completing routine assignments without direct supervision, being unable to function independently (Barkley & Murphy, 2006).

Functional mapping of the brain electrical activity indicates multilevel deficits in sensory processing in ADHD children (Pliszka, Liotti & Woldorff, 2000). It can then be understood that inattentive behaviour observed in ADHD could be as a result of a combination of cognitive and sensory-processing deficits. As indicated by Douglas (1999), attention problems of ADHD are described as trouble with “sustaining attention” commonly occurring in situations where stimuli are widely spaced in time. It could be that attention problems result from changed motivational processes, as they seem to be evident “only when the ability to concentrate is stressed by the task which is not interesting (Taylor, 1998).

3.4.2 Hyperactivity

Hyperactivity is characterised by excessive level of activity often observed in ADHD as restlessness, fidgeting and general increase in gross body movement (Sadock & Ruiz, 2015). Taylor (1998) elaborated that rating hyperactivity involves an element of overstepping clear social rules which are judged according to situational appropriateness. Hyperactivity is often spotted in some situations such as the classroom where certain appropriate behaviour is expected, but might not be observed in others such as play (Porrino, Rapoport, Sceery, Ismond & Bunney, 1983). At school, children with ADHD have the tendency to attack a test rapidly but only answer the first two questions, while at home they cannot be put off for even a minute (Sadock & Ruiz, 2015). In young children, the disorder often shows excessive running, climbing and other gross motor activity (Barkley & Murphy, 2006).

3.4.3 Impulsiveness

Impulsiveness is also regarded as a key defining character of ADHD and has always been explained as being due to faulty executive functions (EF). According to Sadock and Ruiz (2015), behaviours such as as action before thought, abrupt shifts in activity, lack of organization, jumping up in class, memory and thinking deficits, specific learning disabilities, blurting out answers before questions have been completed, having difficulty waiting one's turn when this is appropriate, and frequent interruption and intrusion upon activities of other people are all character traits of impulsivity (Sagvolden, Aase, Johansen & Russell, 2005). Generally, impulsiveness means acting without reflecting and failure to plan ahead. In response suppression, children with ADHD display difficulties waiting for delayed rewards, hence they prefer immediate rewards over delayed ones. According to Sadock and Ruiz (2015), impulsiveness and an inability to delay gratification are the character traits of ADHD.

Lezak, Hoieson and Loring (2004) refer to EF as psychological process which enables one's ability to plan, organize, execute and regulation of goal directed behavior. All this functions are regulated by the frontal lobe and more especially the prefrontal cortex (Lezak et al., 2004). Impulsive behaviour can therefore be understood as a result of executive dysfunction caused by behaviour disinhibition.

3.5 Diagnostic criteria and primary symptoms

3.5.1 Background of diagnostic criteria

The Diagnostic and Statistical Manual of Mental Disorders (DSM) is a manual used by psychiatrists and therapists as a yardstick which is specifically used for diagnosing ADHD in children. The new Fifth Edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013) has made several provision to the diagnostic criteria of ADHD in children, youth and in adults. In the previous Diagnostic and Statistical Manual of Mental Disorders Fourth Edidtion Text Revised, symptoms of ADHD had to be presented by the age seven years, whereas the new DSM-5 recognize that several inattentive or hyperactive-impulsive symptoms" must be presented prior to the age of twelve years (Sadock & Ruiz, 2015) rather than the age of seven years. Again, the DSM IV-TR only recognized two subtypes which are the inattentive type and hyperactive/impulsive type (APA, 2013). The DSM-5 criteria, have replaced subtypes by three specifiers which are: (1) combined presentation, predominantly inattentive presentation, and predominantly hyperactive/ impulsive presentation. The manual further had several alterations in the criteria for ADHD which had an impact on the prevalence both in children and adult population (Faraone, Asherson, Banaschewski, Biederman, Buitelaar, Ramos-Quiroga, Rohde, Sonuga-Barke, Tannock & Franke, 2015). Other important changes counted in the DSM-5 manual were increasing of the age mask.

Moreover, the DSM-5 has an inclusion for comorbid ADHD and autism spectrum diagnosis within the criteria unlike in the previous DSM- IV-TR criteria. Again, the DSM-5 is more elaborative on the symptoms and gives examples for more clarity and a better understanding of the developmental differences of ADHD in terms of diagnosing. The DSM-5 also has a diagnostic criterion for adolescents up to the age of seventeen years and older as well as adults, whereby only five symptoms instead of six symptoms of either inattention or hyperactivity and impulsivity are recognised (Sadock & Ruiz, 2015). However, numerous criticisms in relation to the new DSM-5 were put in place when it was compared to the previous DSM-IV-TR. The following were pointed out (a) it was criticized for utilizing many child psychiatric examples (Levy, 2014), (b) many

new disorders were introduced that might overlap with 'normal behaviour', and it has loosened requirements for many of the existing disorders, (c) has reduced a threshold for adult attention hyperactivity deficit disorder (ADHD).

There are two manuals which are in use for the diagnosis of the disorder ADHD and ICD-10 and were developed by the American Psychiatric Association (APA) and the World Health Organization (WHO) known as DSM-5 (American Psychiatric Association, 2013) and ICD-10 (World Health Organization, 1993). Both the DSM (Diagnostic and Statistical Manual of Mental Disorders) and ICD (International Classification of Disorders) have been highly successful in facilitating communication between clinicians, researchers and administrators in establishing diagnostic reliability. There has been a controversial issue on the ICD 11 with regard to the new classification of disorders. Interestingly, it was observed that, both previous manuals provided similar list of symptoms, but recommended different ways of establishing a diagnosis with a lower prevalence predicted by the ICD-10 requirements (Levy, 2014). Currently, the ICD-11 is said to be due for publication in 2017 but consensus from representatives of more than 200 countries is still needed before approval (Levy, 2014).

In order for a child to meet the diagnostic criteria and to confirm the diagnosis of ADHD, the disorder must have been seen to cause impairment in at least two settings or more (Levy, 2014), moreover, impairment from inattention and/or hyperactivity and impulsivity must have been observed in those settings (i.e. home and school) to a level that it interferes with developmentally appropriate social or academic functioning (Sadock & Ruiz, 2015). Below are the DSM-5 criteria for ADHD and will be followed by the ICD-10 criteria for Hyperkinetic disorder.

3.5.1.1 DSM-5 diagnostic criteria

The new DSM-5 (American Psychiatric Association, 2013) stipulates that in order for an individual to meet the diagnostic criteria for the disorder ADHD, the following criteria should be used as a guideline to determine the actual diagnosis.

A. A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and /or (2):

1. Inattention: Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

(a) Often fails to give close attention to details or make careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
(b) Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
(c) Often does not seem to listen when spoken to directly (e.g. mind seems elsewhere, even in the absence of any obvious distraction).
(d) Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
(e) Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belonging in order; messy, disorganized work; has poor time management; fails to meet deadlines.
(f) Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
(g) Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, and mobile telephones).
(h) Often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts.
(i) Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults returning calls, paying bills, keeping appointments)

2. Hyperactivity and impulsivity: Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: The symptoms are not soley a manifestation of oppositional behaviour, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

(a) Often fidgets with or taps hands or feet or squirms in seat.
(b) Often leaves seat in situations when remaining seated is expected (e.g. leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).
(c) Often runs about or climbs in situations where it is inappropriate. (Note: In adolescents or adults, may be limited to feeling restless).
(d) Often unable to play or engage in leisure activities quietly.
(e) Is often “on the go”, “acting as if driven by a motor” (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).

(f) Often talks excessively.

(g) Often blurts out an answer before a question has been completed (e.g., completes people” sentences; cannot wait for a turn in conversation).
(h) Often has difficulty waiting his or her turn (e.g., while waiting in line).
(i) Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people’s things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

B. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.

C. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).

D. There is clear evidence that the symptoms interfere with, or reduse the quality of, social, academic, or occupational functioning.

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

substance intoxication or withdrawal).

Three subtypes of ADHD:

In DSM-5 (American Psychiatric Association, 2013), three behavioral patterns which specify ADHD were identified. Individuals with ADHD have the tendency to show several signs of being constantly inattentive and not show other signs. Again, they may have a pattern of being hyperactive and impulsive far more as compared to those of their chronological age, or may show all three behavioral patterns. Similar subtypes were recognized in the previous DSM-IV-TR (American Psychiatric Association, 2000). The current DSM-5 recognizes three subtypes of ADHD which according to the criteria symptoms must have been predominant for the past six months. The subtypes are clearly outlined as follows:

Attention-Deficit/Hyperactivity Disorder, Combined Presentation (ADHD-C): this presentation should only be used when six or more symptoms of inattention and six or more symptoms of hyperactivity/impulsivity are predominant for a period of six months to a level that they cause impairment. Individuals who present with ADHD often display both criterion.

Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive presentation (ADHD-PI): this presentation should be used only if symptoms of inattention are predominant for a period of six months to a level that they cause impairment. Individuals who display this presentation must meet criterion for inattention and a criterion for hyperactivity/impulsivity. This presentation generally does not show major hyperactive-impulsive behavioural pattern and referred to as ADD.

Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive/Impulsive presentation (ADHD-HI): this presentation should be in use only if six or more symptoms of hyperactivity/impulsivity have been predominant for a period of six months to a level that it causes impairment. In this case, symptoms of inattention should not be predominant for the past six months. Following is the criteria as outlined according to the ICD-10 criteria for Hyperkinetic disorder.

3.1.5.2 ICD-10 diagnostic criteria for hyperkinetic disorders

According to ICD-10 version 2010, when making a diagnosis for Hyperkinetic disorders the following must be taken into consideration.

- All three of A, B and C, listed below.

- Onset of the disorder is no later than the age of 7 years.
- The criteria should be met for more than a single situation, for example, the combination of inattention and hyperactivity should be present both at home and at school, or at both school and another setting where children are observed, such as a clinic
- The symptoms in A and C must cause clinically significant distress or impairment in social, academic or occupational functioning.

A. At least six of the following symptoms of inattention have persisted for at least six months, to a degree that is maladaptive and inconsistent with the developmental level of the child.
Inattention:

- Often fails to give close attention to details, or makes careless errors in school work, work or other activities.
- Often fails to sustain attention in tasks or play activities.
- Often appears not to listen to what is being said to him or her.
- Often fails to follow through on instructions or to finish school work, chores or duties in the workplace (not because of oppositional behaviour or failure to understand instructions).
- Is often impaired in organizing tasks and activities.
- Often avoids or strongly dislikes tasks, such as homework, that requires sustained mental effort.
- Often loses things necessary for certain tasks and activities, such as school assignments, pencils, books, toys or tools.
- Is often easily distracted by external stimuli is often forgetful in the course of daily activities.

B. At least three symptoms of hyperactivity have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the developmental level of the child.

Hyperactivity:

- Often fidgets with hands or feet or squirms on 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, only feelings of restlessness may be present).
- Often unduly noisy in playing or has difficulty in engaging quietly in leisure activities.
- Often exhibits a persistent pattern of excessive motor activity that is not substantially modified by social context or demands.

C. At least one of the following symptoms of impulsivity has persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the developmental level of the child.

Impulsivity:

- Often blurts out answers before questions have been completed.
- Often fails to wait in lines or await turns in games or group situations.
- Often interrupts or intrudes on others (for example, butts into others' conversations or game).
- Often tasks excessively without appropriate response to social constraints.

3.6 ADHD and culture

A long standing debate by several researchers on whether ADHD might be a cultural thing has been on existence for sometimes (Timimi & Taylor, 2004) while others emphasized that geographical location to some extent may have some influence on the epidemiology of the disorder ADHD and ADHD symptoms (Adewuya & Famuyiwa, 2007; Polanczyk, de Lima, Horta, Biederman & Rhode, 2007). However, a conclusion reached was on the view that culture and geographical location have absolute no influence on the epidemiology of ADHD on the entire world (Adewuya & Famuyiwa,

2007; Plonczyk et al., 2007).

According to Meyer and Aase (2003) and Meyer et al. (2004), little is known about the disorder in the African continent. However, a research study done among a heterogeneous group in Limpopo indicated similarities in prevalence, sex ratios and subtypes in South African children when compared to US and European countries (Meyer et al., 2004), affecting 3% to 10% of primary school children. Meyer et al. (2004) further found that different patterns of disruptive behaviour disorders displayed by ADHD individuals were similar across cultures as assessed by the DSM-IV. In the past, the disorder ADHD was widely researched in developed countries like Europe and North America yet few studies are available from Africa. Emerging studies from African countries documented information on the epidemiology of ADHD, symptoms and associated comorbidities among African children (Bakare, Ubochi, Ebigbo & Orovwigho, 2010; Meyer, 1998; Meyer, Eilertsen, Sundet, Tshifularo & Sagvolden, 2004; Ofovwe, Ofovwe & Meyer, 2006; Wait, Santon & Schoeman, 2002).

In study by Adewuya and Famuyiwa (2007) on the prevalence and co-morbid conditions among Nigerian primary school children, aspects such as method of assessment, diagnostic criteria employed, source of information and inclusion of impairments in operational definition criteria may be looked at with caution as they may have an influence in the prevalence rate of the disorder. In support to similar findings, a study done by Zuddas, Ancilletta, Muglia and Cianchetti (2000) on a neuropsychiatric disorder with childhood onset found that, although clinical tools used to assess mental health were often not found to be valid for African children, yet continued to show factor structure similar to those in European studies. In Kinshasa, Congo a study conducted on co-existing symptoms and risk factors among African school children with ADHD yielded the co-existence of hyperactivity-inattention, emotional and behavioural problems in the same children as compared to those in developed countries (Kashala, Lundervold, Sommerfelt, Tylleskar & Elgen, 2006).

Studies conducted in developed countries in Europe and Mexico focused on psychopathologies associated to ADHD in school age children, revealed that school age children with ADHD who came to psychiatric attention services were highly comorbid with (anxious depressive symptoms, delinquent behaviour and internalising symptoms) (Ulloa, Sanchez, Saucede & Orbitz, 2006). This shows that, comorbid

internalising disorders among school age children were already been acknowledged and as such they were already receiving attention services within hospitals. Moreover, another study observed that a high number of children with ADHD refered for clinical evaluation were first observed by teachers (Sax & Kautz, 2003), showing that the role of effective intervention between teachers and clinicians at hospitals regarding ADHD and child mental health is highly acknowledged. Other studies focused mainly on neurolodevelopmental disorders in children and adolescents such as epilepsy, autism and ADHD being at the risk for multiple psychiatric diagnoses (Germann, Eppinger, Mostofsky, Dcicco-Bloom & Maria, 2015; Jones, Siddarth, Almane, Gurbani, Hermann & Caplan, 2016).

This still remains a huge gap in African countries in recognisinising and intervening on mental illness among ADHD children. In countries like Nigeria where mental health is considerably underresourced and a huge treatment gap still exists (Ibukum, Oluyemi, Abidemi, Suraj & Ola, 2015), the role of a clinical evaluation may be essential. This may be the same in Sout Africa, Limpopo province, where mental health is not well researched. Other factors that lead to limited information within African continent on ADHD and coexisting disorders include lack of data on mental health among children as well as the influence of socio demographic factors (Kashala et al., 2006). However, a study conducted in Kinshasa, Congo on co-existing symptoms and risk factors among African school children having ADHD, showed that co-existence of hyperactivity and emotional behaviour were prevalent. A study by Chinawa, Odetunde, Obu, Chinawa, Bakare and Ujunwa (2014), showed that ADHD is a neglected illness in a developing country. In fact, most studies conducted in Africa, for example, Kinshasa, Nigeria, and DRC focused largely on the prevalence and pattern of the disorder in school children, the burden of ADHD and associated factors of ADHD (Wamulugwa, Kakooza, Kitaka, Nalugya, Moore, Sajatovic & Katabira, 2017).

Research work done on ADHD and comorbid disorders in Africa is quite limited in particular South Africa, Limpopo province among Sepedi speaking children. One emerging study by Mokabane, Pillay and Meyer (2017) on the comorbidity of attention deficit hyperactive disorder (ADHD) and major depression in primary school children, was able to confirm the prevalence and existence of internalising disorder in African primary school. However, the study only looked at the existence of one internalising

disorder i.e depression particularly in the Capricorn district. The study went further to explain that certain behaviours need to be well explored since they may have a different meaning depending on different cultures. Other studies conducted appeared to show attention on ADHD and comorbid internalising disorders among adolsencents, giving a perspective that internalising disorders are prevalent during adolescent phase and not among younger children. Internalising disorders as part of psychiatric conditions still remain neglected among this age group.

There are insufficient studies done in South Africa on the comorbidity of internalising disorders on ADHD symptoms among children in Limpopo, Lepelle Nkumpi, hence the current study seek to investigate the burden of internalising disorders (anxiety and depression).

3.7 Aetiology of ADHD

The aetiology of ADHD seems to be a complex issue to many researchers since there seem to be no single entity leading to the cause of ADHD. However, a combination of factors plays an influential role in the development of ADHD. Most researchers after thorough investigation finally came to an understanding that children with ADHD have a challenge such that they are unable to inhibit their impulsive motor responses (Barkley & Murphy, 1998). There are various factors influencing the eatiology or the cause of ADHD, such as genetic factors, environmental factors and neurological factors (Bradley & Golden, 2001). This section will outline and discuss the following factors which contribute to the development of ADHD:

3.7.1 Brain development.

Gied, Blumenthal, Jeffries, Castellanos, Liu, Zijdenbo, Paus, Evans and Rapoports (1999) outlined in their study on brain development during childhood and adolescence, that scientific research came to a realization that a total brain of a young adult becomes fully attained by the age of 5, while total cerebral volume (TCV) reaches its maximum volume by early adolescence (Courchense, Chisum, Townsend, Cowles, Covington, Egaas & Hinds, 2000). Meanwhile, Sadock and Ruiz (2015) emphasized that the brain of a human normally goes through major developmental spouts at different age levels: 3 to 10 months, 2 to 4 years, 6 to 8 years, 10 to 12 years and 14 to 16 years. This

shows that as early childhood throughout to adolescence the brain is undergoing reconstruction.

Studies using MRI were able to show maturation in brain development especially in children. Findings revealed that children's brain especially the pre frontal cortex expands at approximately 1mm each year (Sowell, Thompson, Leonard, Welcome, Kan & Toga, 2004). This is the area where executive functioning such as planning complex cognitive behaviour, decision making, judgement, impulse control, attention and moderating social behaviour are mediated. The issue of gender related differences became an emerging factor in terms of brain development parttern due to some noticeable differences in both boys and girls. Findings revealed that certain parts of the brain especially the cerebrum and the cerebellum were somehow larger in boys more than girls by 7- 10% in boys (Sowell, Trauner, Gamst & Jernigan, 2002). This was found to be inconsistent with the growing size of the cortical gray matter, which was also found to be larger in boys more than in girls by 10% (Sowell et al., 2002). This may suggest why some behavioural traits and symptoms observed in ADHD individuals are more pronounced in boys in particular.

Other noticeable changes were in the subcortical regions in which the putamen and globus pallidus were also found to be larger in boys, after controlling for total cerebral volume (Gied, Snell, Lange, Rajapakse, Casey & Kozch, 1996). Contrary to this, part of the subcortical region which is the caudate was observed to be larger in girls rather than in boys after controlling the TCV (Sowell et al., 2002). Further explorations on the alteration of the brain were identified. Findings revealed many abnormalities in the global thining of the cortex (in the medial and superior prefrontal and precentral regions) (Shaw et al., 2006), reduction in the density of the dorsolateral prefrontal cortex (Sowell et al., 2003), reduction in surface area, and decreased cortical folding (Cortese, 2012). All these were associated with the impairment of ADHD. As explained by Shaw, Gillian, Liverpool, Weddle, Malek, Sharp, Greenstein, Evans, Rapoport and Giedd (2011), the degree of cortical thinning which is a process which happens normally in the development of the brain is usually associated with severe hyperactivity and impulsivity in ADHD children and those without.

3.7.1.1 White and gray matter development

The fundamental abnormalities in dopamine transmission which was previously known to cause ADHD was challenged and it was suggested that the actual cause of the disorder may lie within the structural difference in the grey matter in the brain (del Campo et al., 2013). Studies further revealed reduced gray matter volumes in fronto-striato-cerebellar and limbic networks which are known to subserve cognitive functions such as attention and executive functions. In support to this, patients with ADHD were found to show significant impairments in attentional performances. This gray matter was found to be localized in the right lentiform nucleus and extend to the caudate nucleus (Nakao, Radua, Rubia & Mataix-Cols, 2011).

The white and gray matter undergoes development through various phases as a person develops. What was observed was that the absence of total brain volume changes covers complex changes in gray and white matter more especially during late childhood and adolescence (Sowell et al., 2002). In young children, it was discovered that there was an increase in the neuron myelination in the white matter in the frontal cortex, parietal cortex and right internal capsule (Sowell, Thomson, Holmes, Bartth, Jerigan & Torga, 1999), which may suggest an increase in myelination of those brain areas (Courchesne et al., 2000; Sowell et al., 2002).

Although there seem to be an increasing growth pattern in the gray matter in the cerebrum, over a period of time, gray matter continues to decrease from early childhood to post-adolescence through which a process of selective pruning of neurons takes place (Courchesne et al., 2000). Following a thorough examination study on the gray-white matter segmentation in a population of ADHD individuals, emerging results showed a significant reduction in both gray and white matter for the right prefrontal cortex (PFC) (Overmeyer, Bullmore, Suckling, Simmons, Williams, Santosha, & Taylor, 2001), and left prefrontal cortex (PFC) (Kates et al., 2002). Similar findings were confirmed by Mostosky, Cooper, Kates, Denckla, Kaufmann (2002) who reported significant reduction in the grey matter restricted to the left PFC, with reduced gray matter in both hemispheres but more particularly on the right hemisphere. As indicated by Depue, Burgess, Bidwell, Willcutt and Banich (2010), poor outcomes on measures of processing speed, response inhibition and response variability are as a result of

reduction of gray matter in the right inferior frontal gyrus.

Again, children diagnosed with hyperkinetic disorder when compared to their non-counterpart were observed to have reduced gray matter more especially in right side in the posterior cingulate gyrus, superior frontal gyrus, and putamen, and bilaterally in the globus pallidus (Lemaitre, Goldman, Sambataro, Verchinski, Meyer-Lindenberg, Weinberger & Mattay, 2012). In this regard, more reduction in the white matter was mainly in the left hemisphere (Krain & Castellanos, 2006). Contrary to findings of prefrontal gray and white matter volumes, Sowell, Thompson, Leonard, Welcome, Henkenius, Toga and Peterson (2003) observed an increased density in gray matter by 15%-30% in the posterior temporal lobes and inferior parietal lobe bilaterally in ADHD sufferers. Other noticeable structures which were found to be significantly smaller in children with ADHD when compared to their non-counterpart were the corpus callosum, genu and splenium (Hill, Yeo, Campbell, Hart, Vigil & Brooks, 2003). The size of the brain was found to be playing a role in ADHD individuals when compared to non ADHD individuals.

3.7.1.2 Decreased brain volume

Studies using the magnetic resonance imaging (MRI) have significantly proven that children with ADHD have a smaller brain on average, when compared to children with healthy brains throughout childhood to adolescence (Castellanos, Lee, Sharp, Jeffries, Greenstein, Clasen, et al., 2002; Durston, Hulshoff Pol, Schnack, Buitelaar, Steenhuis, Minderaa, et al, 2004). Similarly, studies reported abnormal volume and cortical thickness in several brain regions (Valera, Faraone, Murray & Seidman, 2007), total brain volume (Castellanos, Gied, Marsh, Hamburger, Vaituzis et al., 1996), the corpus callosum (Giedd, Rumsey, Castellanos, Rajapakse, Kaysen, Vaituzis, Vauss, Hamburger & Rapoport, 1996), prefrontal regions (Hill, Yeo, Campbell, Hart, Vigil & Brooks, 2003), the basal ganglia (Filipek, Semrud-Clikeman, Steingrad, Kennedy et al., 1997), and the temporal and parietal cortices (Shaw, Eckstrand, Sharp, Blumenthal, Lerch, Green-stein, Clasen, Evans, Gied, Rapoport, 2007). In a longitudinal study, children with ADHD showed a delay in the peak of cortical thickness maturation between 3-5 years, with the greatest delays in frontal as well as temporal brain regions (Shaw et al., 2007), suggesting that myelination process is in progress.

Furthermore, an overall reduction in total brain volume was found to be higher in children with ADHD in comparison to age and sex when matched to children without ADHD (Castellanos & Acosta, 2004). In a study conducted by Durston, Tottenham, Thomas, Davidson, Eigsti, Yang and colleagues (2003), children with ADHD showed an overall cerebral volume that was 3,2% smaller than in normal children. In addition to that all major lobes of the brain which is the frontal, parietal, temporal, and occipital were found to be significantly smaller and thus affected.

Research studies revealed that children with ADHD were found to have abnormal brain activation in the frontal subcortical cerebellar circuits comparative to non- ADHD individuals (Durston et al., 2003). Disruption of circuits mostly in the frontal brain regions, basal ganglia, the cerebellar hemispheres and sub-region of the cerebellar vermis were seen to have an influence in some key symptoms of ADHD (Durston et al., 2003). In support to these findings, prefrontal lobes of the brain are said to be responsible for executive functioning such as behavioural inhibition, regulation of emotional impulses and motivation, planning behaviour, and using external feedback in organizing behaviour across time (Tripp, Ryan & Peace, 2002). Therefore, any form of damage to this area of the brain can result to disruption in behaviour regulation as indicted by Bradley and Golden (2001).

Findings of the white matter area deficits indicated that structural deficits in ADHD affect the structural interconnectivity between regions as well as the entire neural network (Konrad & Eickhoff, 2010). A diffusion tensor imaging study was able to outline several white matter located in several different bunches in the left fronto-temporal regions and right parietal-occipital regions (Silk, Vance, Rinehart, Bradshaw & Cunnington, 2009). Results showed an abnormal white matter development in ADHD in different cortical regions which were previously regarded as dysfunctional. Therefore, it can be concluded that truly there is an abnormal development taking place within fronto-parietal cortical networks that may strengthen the cognitive and attentional disturbances related with ADHD (Silk et al., 2009).

3.7.1.3 Frontal cortex

The role of frontal brain in relation to the disorder ADHD has been scientifically tested by many. In a study that was conducted among 12 years old boys with and without ADHD, findings indicated a decreased size of the frontal lobe which accounted 48% of the reduction in total cerebral volume (Mostofsky, Cooper, Kates, Denckla & Kaufmann, 2002). In ADHD children, the prefrontal cortex (PFC) was observed to be significantly smaller when compared to normal children and their unaffected siblings (Durston et al., 2004), suggesting that all functions coordinated in that area becomes implicated. Again in a sibling study conducted a decreased left occipital gray and white matter volume in both boys with ADHD and their brothers was also found.

Gender differences on the other hand was observed to play a significant role during puberty in which, Blackemore and Choudhury (2006) highlighted that the high secretion level of testosterone resulted to the increase of grey matter volume in the inferior frontal gyrus in boys. Moreover, boys were found to have a greater cortical grey matter volume as compared to girls. Brain asymmetry on the part of the prefrontal regions was identified with some differences in terms of size. The right asymmetry of the prefrontal cortex was seen to be greater that the left asymmetry of the prefrontal cortex (Watkins, Paus, Lerch, Zijdenbos, Collins & Neelin, 2001). Then, the significant reduction in size of the asymmetry in children with ADHD was as a result of a decrease in right prefrontal regions (Watkins et al., 2001).

Other interesting findings about the size of the PFC sub regions was that the right dorsolateral prefrontal volume was quite smaller in investigated children with non-comorbid ADHD (Yeo, Hill, Campbell, Vigil, Petropoulos, Zamora, & Brooks, 2003). Moreover, children with ADHD appeared to have a reduced brain surface by up to 4 mm bilaterally in the lateral anterior temporal cortices and inferior portion of dorsal prefrontal cortices well as in the right parietal cortex as indicated by Hesslinger, Tebartz van Elst, Thiel, Haegele, Hennig and Ebert (2002).

3.7.1.4 Cerebellum

The cerebellum is another part of the brain which has been acknowledged to have an influential role in ADHD. The cerebellum is generally known for its role in motor control, cognitive functions such as attention and language, regulating fear and pleasure responses. Again the cerebellum is also known to be involved in non-motor functions such as timing and attentional shifting through connections with frontal regions (de Zubicaray, Zelaya, Andrew, Williams & Bullmore, 2000). In children with ADHD, these functions can be a complicated task as a result of the disorder.

Studies using the MRI were able to measure the total volume of the cerebellum, as well as the volume and its components such as the cerebellar vermis and lobes. Studies have shown quite a smaller cerebellar hemispheric volume which was about 6% of the cerebellum in ADHD (Durston et al., 2004). It is further observed that cerebellar hemispheric volume remains stagnated throughout adolescence and even after controlling for TCV (Castellanos et al., 2002). Other discoveries were the smaller vermal volume found in ADHD children than controls (Hill et al., 2003). The decrease in size of the posterior inferior lobe of the cerebellum in children with ADHD was located specifically to the lobules VIII-X, while decrease in other parts of the cerebellar lobules could not be found (Hill et al., 2003).

3.7.1.5 Structural findings in girls with ADHD

ADHD is largely known to be prevalent in boys more than in girls. However structural abnormalities in girls with ADHD was examined by several researchers. Research findings found that total cerebral volume in girls with ADHD was significantly smaller when compared to their non-counterparts (Castellanos et al., 2001). More findings indicated that after controlling the TCV and vocabulary, girls with ADHD continued to show significantly smaller volumes in the posterior – inferior lobules of the cerebellar vermis (Castellanoset et al., 2002).

3.7.1.6 Association between brain structures and functioning

Many research studies were able to examine the structure and functioning of the brain on two aspects which is the regional brain volumes and measures of functioning such as behavioural rating scales and neurological tests. They found that smaller brain volumes were connected with greater ADHD symptom severity particularly in girls (Castellanos et al., 2001). Contrary to similar findings, in their study Castellanos and colleagues (2002) were able to establish that the frontal and temporal gray, caudate, and cerebellar volumes negatively correlated with global clinician ratings as well as parent ratings of child attention problems.

Studies by functional and structural neuroimaging managed to establish abnormalities of the brain which might be a possibility to the neuropathophysiology of ADHD (Bush, 2011). What was observed was the hypofunction of the brain regions in the cingulo-frontal-parietal and cognitive-attention network. These are regarded as the main components of neural systems that are related to ADHD, together with cognitive/attention networks, motor systems and reward/feedback-based processing system (Bush, 2011). This type of network is regarded as the executive control circuit since it underpins goal directed executive process and also provides flexibility to information processing in response to changing task demands (Liston, Matalon, Hare, Davidson & Casey, 2006).

Other investigations found that smaller left caudate head and white matter volumes were associated with higher Child Behaviour Checklist (CBCL) more especially on externalising scores (Semrud- Clikeman, Steinguard, Filipek, Biederman, Bekken & Renshaw, 2000). It can be understood that a decrease in volume of brain structure may have a huge influence on the behaviour as well as brain functioning of an individual. Sowell et al. (2003) stated that reduced total cerebral volume was strongly associated with more attention problems, while reduced posterior inferior vermal volumes was significantly associated with global functioning and CBCL anxiety-depression scores. It may be understood that these children generally may experience extreme challenges in multiple areas of functioning and therefore exhibit elements of depression or anxiety whenever they are assessed.

Other studies were able to examine the relationship between brain volumes and tests of neuropsychological functioning in ADHD children. In a study conducted on the neuropsychological test performance of children with ADHD relative to test norms and parent behaviour ratings, showed that children with ADHD performed poorly to test norms and measures sensitive to fronto-executive functioning (span of attention, sustained attention, response inhibition and working memory) and also on memory tests which required free recall/retrival, a skill reliant on intact frontal/subcortical functioning (Muir-Broaddus, Rosenstein, Medina & Soderberg, 2002). This task performance usually is known to be sensitive to the prefrontal cortex and as a result, children with ADHD often display executive function deficits especially in response inhibition (Krain & Castellanos, 2006).

A study conducted by Casey, Castellanos, Giedd, Marsh, Hamberger, Schubert, Vauss, Vaituzis, Dickstein, Safratti and Rapoport (1997) on relation between specific frontostriatal structures (prefrontal cortex and basal ganglia) and response inhibition deficits observed in ADHD, showed that performance of children with and without ADHD displayed a positive correlation with prefrontal cortex, caudate and globus pallidus volumes on ADHD task performance. Moreover, a strong correlation between sensory selection task performance and prefrontal and caudate volumes were said to be more localized to the right side while response selection and response execution tasks were found to be correlating with caudate symmetry as well as left globus pallidus size.

This was a contradiction to a study conducted by Hill, Yeo, Campell, Hart and Vigil (2003), on comparison of magnetic resonance imaging size differnces in many brain regions and neurocognitive function in ADHD children. Findings revealed that larger volumes in total superior prefrontal cortex and right superior cortex were both associated with worse performance particularly on a test measuring attention i.e. (Conners'continuos performance test, CPT). Similar findings further showed that performance on the Conners's CPT results significantly correlated with the dorsolateral volumes in ADHD children. Castellanos and colleagues (2002) further elicited that differences in decrease brain volumes seem to represent a continuous deficit probably arising from genetic and environmental factors. It appears that many key brain structures are affected in ADHD. Krain and Castellanos (2006) are of the view that

larger volumes predicts poor performance on the CPT, variability and reaction time, this implies that ADHD children are more likely to display some neurological impairments when they are assessed. It was further observed that the dysfunctionality of the right dorsolateral region in children with ADHD lead to greater disruption in which attention becomes affected (Krain & Castellanos, 2006).

3.7.2 Genetics

Research body so far have demonstrated the influential role of genetics on the etiology of ADHD. In understanding genetics fully, we also need to look at the probability and the impact of this gene manifests itself through families, adopted children and monozygotic (MZ) and dizygotic (DZ) twins. Studies were able to show that ADHD is familial and the familiarity is clearly due to genetic influences (Faraone, Biederman & Friedman, 2000). Furthermore, genes meaningfully contribute to the onset, persistence and remission of ADHD (Faraone, Asherson, Banaschewski, Biederman, Buitelaar, Ramos-Quiroga, Rohde, Sonuga-Barke, Tannock & Franke, 2015). Comparatively, the level of ADHD was found to be higher in children with biological relatives with ADHD more than families of children without ADHD (Faraone, Biedermtan & Friedman, 2000). In support to this similar findings, familial researches have shown that the increased risk of ADHD on parents and siblings is two and eight folds (Faraone et al., 2005). This implies that the relative risk of having ADHD was observed to be similar for relatives of both boys and girls with ADHD. Moreover a study conducted among Caucasians and African American signify that the risk of ADHD seems to cut across demographic groups (Faraone et al., 2000). About 30-35% of the full siblings of ADHD probands also meet the criteria for ADHD or rather display symptoms of the disorder unlike in a general ADHD population. It can then be understood that the familiality of ADHD may be slightly recognized as genetically influenced (Willcutt et al., 2012).

Several studies made an attempt to identify the specific genes involved in the aetiology ADHD and what they came across was that, ADHD children were likely to have several missing DNA segment or duplicated segments of DNA which was not observed when comparing them to their non-counterparts (Nordquist & Oreland, 2010). This missing DNA segments refers to chromosomal regions which contains genes that co-segregate with the disorder within families (Cortese, 2012). The chromosomal regions which were

identified to be having genes involved in the aetiology of ADHD were 5p13, 11q22-25 and 17p11 (Coghill & Banaschewski, 2009). These genes play a crucial role in the development of attention deficit hyperactivity disorder.

ADHD is a genetic disorder which can be observed at diferrent regions or bins within the chromosome. Therefore, each bin carries a gene which can be implicated to the disorder ADHD. Other studies further revealed a region on chromosome 16 at bin 16.4 to be implicated as well as other nine regions i.e. bins 5.3, 6.3, 6.4, 7.3, 8.1, 9.4, 15.1, 16.3 and 17.1 which may have genes of interest for ADHD (Zhou, Dempfle, Arcos-Burgos, Bakker, Banaschewski, Biederman et al., 2008). On the other hand, Williams, Zaharieva, Martins, Langley, Mantripragada, Fossdal, & Thapar (2010) argue that ADHD is not caused by a single genetic change, rather it is caused by a number of genetic changes like CNV's as they interact with the environment. The study as a result acknowledged that ADHD is a neurodevelopmental disorder rather than a behavioural disorder (Faraone & Mick, 2010).

Genetic molecular studies identified an elevated dopamine transporter gene of varying lengths, as a result of polymorphism (Madras, Miller & Fischman, 2002). Other researchers have shown a significant association with ADHD and the genes encoding the DA receptor subtypes D4 and D5 (Faraone & Mick, 2010). DA betahydroxylase, the synaptosomal- associated protein 25, the serotonin transporter and serotonin 1B receptor were all associated with ADHD. There seem to be no single gene playing a significant role in the development of ADHD. However, close to 27 different candidate genes were identified and also said to be influential in the neurotransmitter that may have a key role in the pathophysiology of ADHD (Comings, Chen, Blum, Mengucci, Blum, & Meshkin, 2005). The main genes implicated in ADHD are DAT, DARD 4, DRD 5, DBH, ADRA 2A, SNAP 25, 5HTTLPR, HTR 1B and FADS 2, all being said to be associated with the neurotransmitter dopamine (Coghill & Banaschewski, 2009). It is said that candidate genes such as alpha 2A adrenergic receptor (ADRA 2A), serotonin receptor (HTR 1B), and some other proteins are still yet to be explored.

Adoptive studies were able to find that biological relatives of children with ADHD are, more likely to have hyperactivity than adoptive relatives (Faraone et al., 2005). On the other hand, Willcutt (2008) holds the view that biological relatives of an individual who is adopted at birth are related genetically to the individual, even though they do not

experience the same environmental influences. Family environmental influences were then said to play a key role in the etiology especially in the elevated rate of the disorder among adoptive relatives (Willcutt, 2008).

ADHD remains to be the most heritable psychiatric disorder (Cortese, 2012). This was supported by several studies conducted on the concordance of ADHD rate, in which a higher rate of concordance among MZ pairs was estimated at 58% - 82% comparative to same-sex DZ pairs which was at 31%-38% (Levy, McStephen & Hay, 2001). In comparison, monozygotic (MZ) twins shared most of their genes while dizygotic (DZ) share half of their segregating genes on average, which suggest that ADHD is purely heritable. A direct estimation of the extent to which genes, shared environmental factors, and non-shared environmental factor may influence the disorder was also provided through twin analyses method (Polmin, DeFries, McClearn & McGuffin, 2001). As much as genetic bases may have a key role in the etiology of the disorder, it further shows that multiple environmental factors have an influence in the development of ADHD.

3.7.3 Environmental factors

Studies previously conducted have shown that environmental factors are direct causal of ADHD. Environmental factors in the development of ADHD may include pre-natal, peri-natal and post-natal in origin (Cortese, 2012). Certain environmental factors may have an effect on the child i.e. before, during and after pregnancy.

Prenatal factors

Factors such as maternal smoking, maternal anaemia, exposure to cocaine, lead or alcohol, iodine deficiency, low birth weight (>1, 5 kg) (Banerjee, Middleton & Faraone, 2007; Scassellati, Bonvicini, Fraone & Gennarelli, 2012) and small head circumference are associated with ADHD (Millichap, 2008). All of these during pre-natal are detrimental to the unborn child and this could result into a child developing ADHD related conditions. In one study conducted, children exposed to prenatal smoking and hozygous for the DAT1 10-repeat allele were said to be at the high risk of developing hyperactivity, impulsivity and oppositional symptoms (Kahn, Khoury, Nichols & Lanphear, 2003). Similarly, exposure to prenatal alcohol and DAT1 genetic was also

seen to be linked with the increased risk for ADHD (Brookes, Mill, Guindalini, Curran, Xu, Knight, Chen, Huang, Sethna, Taylor, Chen, Breen & Asherson, 2006).

Perinatal factors

During pregnancy, factors such as maternal smoking, increased maternal stress during pregnancy as well as pregnancy and delivery complications such as toxaemia, preeclampsia, and poor maternal health, advanced maternal age, long duration of labor, fetal distress and ante partum haemorrhage can have an implication in the development of ADHD (Boulet, Boyle & Schieve, 2009). Brain development during foetal phase can be changed by chemical agents such as alcohol, nicotine and drugs, by mother's behaviour and health as well as by environmental effects on the mother (Mulder, De Medina, Huizink, Van den Bergh, Buitelaar & Visser, 2002). Exposure of a foetus to a toxic or diseased prenatal environment such as opiates, methadone, marijuana and cigarette during pregnancy result to ADHD and conduct problems.

As indicated in by Castellanos et al. (2002) that male offspring of nutritionally deprived pregnant women especially in the first and second trimester may be a risk factor for developing antisocial personality disorders when they become adults. This may give clear perspective why ADHD is prevalent and external in males more than females.

Postnatal factors

Studies indicated that malnutrition at the age of three exposes children to neurocognitive defects which at a later stage may predispose them to persistent aggression and hyperactivity (Andersen, Thompson, Rustein, Hostetter & Teicher, 2000). Breastfeeding is a crucial factor in the early developmental life of a child. In support to this it was indicated that breastfeeding beyond 6 months is crucial for neurodevelopment and cognition that support self-regulation and self-control (Stadler, Musser, Holton, Shannon & Nigg, 2016).

Breastfeeding duration was further said to strengthen the development of brain white matter connectivity in infants and toddlers (Tawia, 2013) as well as increasing the white matter development in 8 years children, especially boys in fibre tracts such as superior longitudinal fasciculus, cingulum, body of corpus collosum and posterior thalamic radiations (Ou, Andres, Cleves, Pivik, Snow, Ding & Badger, 2014). These areas

according to Nagel, Bathula, Herting, Schmitt, Kroenke, Fair and Nigg (2011) are similar to those observed in ADHD. A comparison study among children with and without ADHD, findings revealed that shorter duration of breastfeeding was associated with child ADHD rather than initiation of breastfeeding. It may then be suggested that breastfeeding may serve as a buffer for future development of ADHD.Factors like childhood illness such as meningitis, viral infections, encephalitis, anemia, epilepsy and cardiac disease may be associated with ADHD (Millichap, 2008), leading to more complications to the disorder.

Massive evidence by researchers showed that malnutrition at age three was related to lower IQ at age three and eleven (Liu, Raine, Venables, Dalais & Mednick, 2003). Malnutrition was seen by others as a precursor to multiple neurocognitive deficits, which predicts the beginning of antisocial behaviour and continuous externalising behaviour problems throughout childhood and adolescents (Liu et al., 2003).

Incidences such as high levels of family problems and emotional stress during pregnancy (23%), early injurious accidents (7%), surgery within the first month of life (5%) and trauma during delivery (2%) may be responsible for ADHD (Bradley & Golden, 2001). In support to this, risk factors which can be in a form of cerebral trauma, and toxins and drugs as indicated by Millichap (2008) may also expose the child to develop typical symptoms of ADHD. In a study conducted in South Africa showed that risk factors such as birth complications, prenatal psychiatric disorder, maternal ADHD, nonmaternal child care, early traumatic life events, and short periods of breastfeeding were associated with ADHD in South Africa (Van Dyk, Springer, Kidd, Steyn, Solomons & Van Toorn, 2015).

Exposure to vehicular pollution and lead exposure were notably linked to ADHD (Siddique, Banerjee, Ray & Lahiri, 2011). Similar findings further indicated that exposure to this harmful chemicals during the first year of life was associated with (higher hyperactivity scores at seven years of age (Newman, Ryan, LeMasters, Levin, Bernstein, Hershey, Lockey et al, 2013). Moreover, it was further discovered that Blood Lead Level (BLL) which was less than 10 µg/dL in children were predictable to at least one type of ADHD and this association was found between BLL and combined subtypes of ADHD by many researchers (Daneshparvar,Mostafavi, Jeddi, Yunesia,

Mesdaghinia, et al, 2016). This may imply that less blood lead level may be linked to lower ADHD scores.

There is a developing trend of research these days which show that there is direct association between genes and environmental factors in contributing to ADHD. Alcohol consumption and psychosocial adversity, parental conflicts, parental psychiatric illness or parental aggression (Manjunath, Kishor, Kulk. arni, Shrinivasa & Sathyamurthy, 2016) are all environmental factors and if they occur during pregnancy that is likely to affect the genes which later contribute to the development of ADHD (Nigg, Nikolas & Burt, 2010).

More researchers paid attention on how environment and heritability influences each other in the development of ADHD. They observed that gene-environment (G × E) interaction may be the main mechanism by which environmental risk factors increase the possibility of ADHD (Faraone, Asherson, Banaschewski, Biederman, Buitelaar, Ramos-Quiroga, Rohde, Sonuga-Barke, Tannock & Franke, 2015). Furthermore, the genetic polymorphism of 5-HTTLPR which is located in the promoter of SLC6A4 is involved in ADHD key symptoms, hyperactivity and impulsivity and also moderate the effects of stress on ADHD (van der Meer, Hartman, Richards, Bralten, Franke,Oosterlaan, Heslenfeld, Faraone, Buitelaar & Hoekstra, 2014)This may suggest that environmental factors may have a great influence on genetic interaction.

3.7.4 Psychosocial factors

There are many signs of psychosocial difficulties, such as family adversity and low income which have been found to be associated with child mental health problems, including ADHD (Pheula, Rohde & Schmitz, 2011). Factors such as severe chronic abuse, maltreatment, and neglect were associated with behavioural symptoms that overlap with ADHD such as poor attention and poor impulse control (hyperactivity) (Sadack & Ruiz, 2015). Children who were institutionalized and suffered emotional deprivation often displayed ADHD key defining symptoms such as hyperactivity and also have poor attentional span (Sadock & Ruiz, 2015). However, these signs tend to disappear with time when a child is put in a conducive environment depending on the child's temperament, genetic familial factors (Sadock & Ruiz, 2015). Johnston and Mash (2001) indicated that most studies done on parenting and ADHD only focused

on families of children with ADHD yet overlooked the fact that many parents in those families also experience elevated ADHD symptoms.

Children who come from background where there is constant family conflict were seen to have a high incidence of ADHD symptoms comparative to their non-counterparts (Thapar, Cooper, Eyre & Langley, 2013). Lifford, Harold and Thapar (2009) were of the view that there is a relationship between parent child hostility and ADHD symptoms. They observed that it is the child's ADHD symptoms which impacts on mother-son hostility more than the hostility playing a role in ADHD. It can be understood that the degree at which the disorder present itself may put a lot of strain and even create a rough relationship between the individual and significant others.

3.7.5 Dopamine dysfunction

Several researchers postulated that ADHD is associated with certain changes in dopamine functioning which may interfere with one's ability to focus, sustain attention or with memory formation and retrieval (Johansen, Aase, Meyer & Sagvolden, 2002; Sagvolden, Johansen, Aase & Russell, 2005). Studies of the neurotransmitters also pointed abnormalities in dopamine (DA) and norepinephrine (NE) to be associated with ADHD (Bond, Hadjipavlou, Lam, McIntyre, Beaulieu, Schaffer & Weiss, 2012).

The chemical dopamine is mostly found in the areas of the brain known as orbital frontal regions, and its many connections through pathways of nerve fibers into a structure known as the caudate nucleus comprising the striatum, which itself connects further back into a deeper area of the brain called the limbic system. These brain areas are those responsible to inhibit behavior, sustain attention, and inhibit our responses (Castellanos, 1997). In a study by Johansen, Aase, Myer and Sagvolden (2002) showed that chronic intake of dopamine agonists such as cocaine, crack and amphetamine will cause a down- regulation of dopamine synthesis which may result to behavioural characteristics of ADHD.

3.8 Comorbid disorders

The diagnosis of Attention Deficit/Hyperactivity Disorder as indicated by Barkley and Murphy (1998) may suggest a significant risk for other coexisting psychiatric disorder. As indicated by Patel, Patel and Patel (2012) that children with severe ADHD symptoms often have higher chance of developing other psychiatric disorders. In support to this, a survey published on children's health, observed that psychiatric and physical comorbidities were indeed common in children with ADHD (Larson, Russ, Kkahn & Halfon, 2011), while on the other hand the presence of comorbid disorders made the diagnosis of ADHD even more difficult (Patel, Patel & Patel, 2012).

ADHD symptoms and core features of other disorders may overlap and make the diagnosis difficult to differentiate. ADHD often coexist with multiple disorders such as conduct disorder, oppositional defiant disorder, bipolar disorder, to mention a few. This section will exclude comorbid anxiety and depression since they will be clearly outlined in the next chapter of the current study. The following are the psychological problems associated with ADHD:

3.8.1 Conduct Disorder (CD)

Conduct Disorder remains the common disruptive behaviour disorder within children with Attention Deficit Hyperactivity Disorder (Jensen, Hinshaw, Kraemer, Lenora, Newcorn, Abikoff, March, Arnold, Cantwell, Conners & Elliott, 2001). Conduct Disorder as outlined in the DSM-5 criteria is expressed as a repetitive and persistent pattern of behaviour in which the basic rights of others or major age-appropriate societal norms or rules are violated (American Psychiatric Association, 2013). According to Patel et al. (2012), about 20-45% of children presenting with ADHD do meet the criteria for Conduct Disorder (CD). Baker (2012) further emphasized that the presence of CD is greater in individuals coming from low socioeconomic status. According to Sadock, Sadock Cancro, Sussman, and Ahmad (2005), ADHD is again associated with psychosocial factors such as low socio economic level, harsh, punitive parenting, family discord, and a lack of social competence.

ADHD with comorbid CD seems to contribute to the severity of the condition, thus resulting to poor prognosis which is followed by substance abuse and antisocial

personality disorder later in life (Barkley, 2004). In a correlational study done by Logan, Shachar and Tannock (1997) on ADHD and CD, it was hypothesized that both conditions seemed to share similar risk factors and moreover one disorder had a greatly influences on the other. Other studies are of the opinion that ADHD and CD are associated with deficits in the executive functioning (Banaschewski, Hollis, Oosterlaan, Roeyers, Rubia, Willcutt, et al., 2005).

3.8.2 Oppositional Defiant Disorder (ODD)

Oppositional Defiant Disorder is another comorbid disruptive behavioural disorder which is commonly associated with ADHD (Patel et al., 2012). Oppositional Defiant Disorder is mainly characterized by a pattern of angry/irritable mood, argumentative/defiant behaviour, or vindictiveness (American Psychiatric Association, 2013). According to Patel et al. (2012), symptoms of ODD have a habit to occur more often with people whom the child is close to or knows such as family or caregivers. Moreover, these children are said to form an intermediated group between those have ADHD alone and those with ADHD and conduct disorder. About 21% to 60 % of children with ADHD were said to have ODD symptoms (Cunningham & Boyle, 2002; Wilens, Biederman & Spncer, 2002). On the other hand, Masi and Gignac (2017) estimated that the incidence for the disorder at 5 -10%. In a clinical setting, they discovered that ODD was associated with ADHD in 25 – 75% of patients.

Verlinden, Jansen, Veenstra, Jaddoe, Hofman, Verhulst, Shaw & Tiemeier (2015) emphasized that the presence of ADHD and ODD behavioural problems at a young age may predispose children to bullying involvement in early elementary school. Children with both ODD and ADHD were observed to be more impaired, however using treatment helped to reduce the risk of complications like depression, CD, or substance abuse (Masi & Gignac, 2017). For treatment of behavioural disorders like ODD, the involvement of a psycho educator whom will assist in introducing rules which aims at re-establishing generational boundaries (Masi & Gignac, 2017). Baker-Ericzén, Jenkins, and Haine-Schlagel (2013), further elaborated that the use of behavioural treatments which were previously supported as well as combining medical approach with psychosocial approach will be essential in order to improve the pharmacotherapy of ADHD.

3.8.3 Bipolar Disorder

Distinguishing between ADHD and bipolar disorder in childhood can be rather difficult. Masi and Gignac (2017) further found that the periodicity aspect of bipolar disorder as opposed to the continuous symptoms in ADHD which is another crucial differentiating point. Bipolar disorder is characterized by symptoms of grandiose perception or psychosis, decreased need to sleep, tachypsychia, euphoria and hypersexuality. But in children, bipolar often seems to be a rather chronic mood dysregulation with a mixture of elation, depression, and irritability. It should be taken into account that most children with ADHD do not have bipolar disorder, however, it should be noted that during prepuberty, bipolar disorder could be manifested with less clearly defined cycles (Masi & Gignac, 2017). Moreover, there are symptoms that can be present both in ADHD and bipolar, such as logorrhea, pressure of speech, psychomotor instability and distractibility.

Van Meter, Moreina and Youngstrom (2011) in their study on the analysis of epidemiology of pediatric bipolar disorder, observed that in indiviuals from 7- 21 years-old, adolescents who participated had an incidence rate of 1.8% for bipolar disorder I and II. However, up to 20% of the cases of bipolar disorder co-occur with ADHD (Masi & Gignac, 2017). The comorbidity of ADHD and bipolar disorder put patients at a risk of developing other comorbid disorders, have more suicide attempts, less educational achievement, more legal problems and as a result have poor prognosis.

Treatment for both disorders is quite a pre-requisite although it can be complex. The use of substances on the other hand may have devastating side effects hence they must be taken through the guidance of a medical practitioner. Stimulant medication as a form of treatment for ADHD symptoms can increase the risk of manic episode in the case of bipolar (Masi & Gignac, 2017). In the management of both disorders, caution is made that, drugs such as stimulants be discontinued until such time when the mood is stabilized, and thus re-administration of stimulants may be done (Van Meter, Moreina & Youngstrom 2011; Perroud, Corderaa, Zimmermanna, Michalopoulos, Bancila, Prada & Aubry, 2014).

3.8.4 Disruptive Mood Dysregulation Disorder (DMDD)

DMMD is a new diagnostic that has been described in the new DSM-5 (APA, 2013). DMMD is often characterized by a chronic dysphoria associated with a minimum of three severe anger episodes per week over a period of a year (Masi & Gignac, 2017). In this regard, episodes of anger are associated with severe and persistent irritability.

Masi and Gignac (2017) further observed that among individuals ranging between 9-19 years old had an incidence rate for DMDD ranging at 3.3% and amongst those 1.8% were said to be severe. DMDD was further found to be highly comorbid with ADHD and in support to this up to 87% of DMDD children were found to be having ADHD (Leibenluft, 2011). Other similar findings showed that 93.8% of children with DMMD presented with symptoms of ADHD (Dickstein, Milham, Nugent, Drevets, Charney, Pine & Leibenluft 2005; Brotman, Schmajuk, Rich, Dickstein, Guyer, Costello, Egger, Angold, Pine, & Leibenuft, 2006). In managing the condition, both pharmacological and non-pharmacological treatment should be used at the same time.

3.8.5 Tics and Tourette's syndrome

Tics often has a phonic or movement aspect (blinking being the most common). Tourette's syndrome (TS) is a disorder with an onset from early childhood and often comorbid with ADHD. TS is characterized by multiple physical (motor) tics, for example blinking being the most common and at least one vocal (phonic) tic. People with Tourette have various nervous tics and repetitive mannerisms, such as eye blinks, or facial twitches or grimacing.

TS is also regarded as a neurodevelopmental disorder with a common comorbididty with ADHD. As indicated by Freeman (2007), the co-occurance of both ADHD and TS is 55%, which at a later stage may have major impacts on the child's outcome. The use medication such as stimulants on treating both disorders was found to be complex and therefore both disorders should be treated simultaneously with caution. However, stimulants were observed to be more effective on treating ADHD symptoms.

3.8.6 Autistic Spectrum Disorder (ASD)

The new DSM-5 has formally acknowledged the comorbidity of Autistic Spectrum Disorder (ASD) and ADHD under the classification of neurodevelopmental disorders. Both disorders seem to have similar genetic, clinical and neurological factors. In a clinical sample of 58% of children with autism, 85% of children with a continuum ASD were confirmed to have a clinical picture of ADHD (Masi & Gignac, 2017). A study conducted by Konst, Matson, Goldin and RieskeHow (2014) among children with ASD, revealed that ASD symptom severity were linked with inattention/impulsive symptoms, which are key symptoms of ADHD (Konst, Matson, Goldin & Williams, 2014). The management of both disorders may require psychopharmacology wherein psychoeducation, behavioral therapy and medication are used simultaneously. ASD patients with ADHD may respond adequately to ADHD medication, but it should be administered with caution in order to minimize adverse effects like dysphoria (Konst et al., 2014).

3.9. Assessment and diagnosis of ADHD

ADHD is a child psychiatric disorder which requires proper assessment by an appropriate and qualified mental health practitioner such as a psychiatrist or psychologist. Since the principal signs of inattention, impulsivity and hyperactivity may be stimulated on the basis of a detailed history of a child's early developmental pattern and where a child's behaviour can be clearly observed, (Sadock & Ruiz, 2015). In addition to ADHD symptoms, having a detailed history about the presence or absence of any existing co-morbid disorders, patient's medical and developmental history will assist in order to rule out any possible developmental and medical conditions or medications that may predispose, mimic or worsen ADHD (Shier, Reichenbacher, Ghuman & Ghuman, 2013).

Furthermore, the diagnosis of ADHD as stipulated in the diagnostic and statistical manual of mental disorders (DSM-5), requires that patients must show minimum of 6 of the 9 inattentive or hyperactive/impulsive symptoms for a period of at least 6 months (American Psychiatric Association, 2013). In diagnosing ADHD, it is paramount that

clinicians always document the persistence, impairing symptoms of either hyperactivity /impulsivity or inattention in at least two different settings i.e. in school and at home (Sadock & Ruiz, 2015).

Collateral information obtained from school teachers in relations to ADHD symptoms in class, behavioral problems at school, problems with peer-relations, and level of academic performance and achievements plays an essential role (Shier et al., 2013). Another reliable source of information can be through parents' report about their children since they best understand the child's functioning in a home environment (Swanson, Lerner, March & Gresham, 1999). Generally, information can be obtained from both parents and teachers through rating scales or questionnaires. Moreover, the use of psychological tests may be useful especially that they can provide added information and give support in discovering comorbid conditions from several sources and not just on the results of tests. The use of psychological tests can help clinicians to address the important questions underlying the evaluation of ADHD such as (1) is the diagnosis justified? (2) If the diagnosis is not justified, are there other explanations that better accounts for the symptoms? (3) If the diagnosis is justified, are there comorbid conditions that should be justified and be treated? When all these are considered, a diagnosis can be done.

There are various methods of assessment tools that may be used to assess and distinguish between children with ADHD and normal control groups. Those tests include cognitive tests, individual neuropsychological tests and general neuropsychological batteries. Testing materials such as Parent/Teacher Disruptive Behavioural Disorder Rating Scale (DBD), Barkley and Murphy (1998) should be administered to both parents and teachers prior to diagnosing the disorder. The scale is used to assess the presence and degree of ADHD symptoms of the participants. The Beck Youth Inventories (BYI) is a well-accepted self-report scale that measure stress that children experience in association with mental health problems (Beck, Beck, Jolly & Steer, 2005).

The inventory also has 2 sub inventories which are the Beck Depression Inventories for Youth (BDI-Y) assessing symptoms of depression as well as the Beck Anxiety Inventory for Youth (BANI-Y) which reflects on children's fears, worries about school performance and physiological symptoms associated with anxiety (Beck et al., 2005).

Both sub-scales are in line with the depression and anxiety criteria of the DSM IV TR. More detailed information on these tests/scales will be discussed in chapter 5 of the current study.

3.10 Treatment of ADHD

Treatment of ADHD requires a comprehensive behavioral, psychological, educational, and sometimes medical evaluation. Treatment for ADHD simply means implementing strategies and interventions which will help to manage the symptoms of ADHD more effectively. Managing ADHD in most instances is multidisciplinary such that it requires assistance of other disciplines like mental health, educational, and medical professions at various points in different times (Barkley & Murphy, 2006). Treatment involves the individual or their family members to be educated about the nature of the disorder as well as methods of management, positive and proactive behavioral interventions that provides structure, consistency, predictability and teach appropriate skills, parent training to teach and support effective parenting approaches for a child with ADHD and modifications, support and accommodation to increase success at school (Barkley & Murphy, 2006). It is important to treat ADHD so that we decrease symptoms, enhance functionality, and improve well-being for the individual and those closer to them (Miller, Johnston, Klassen, Fine & Papsdorf, 2005). As such treatment must be long-term to assist those with ADHD as an ongoing management process of their disorder. The following treatment modalities were found to be effective in the management of the disorder and will be fully outlined:

3.10.1 Psychopharmacology

The use of pharmacological treatment is regarded as one of the first clinical strategies in the treatment for ADHD (Sheir et al., 2013). Following a number of concerns by many researchers indicating that the treatment suppress growth in those using treatment (Harpin, 2005), sufficient evidence for the efficacy and safety of both stimulants and non-stimulants in the treatment of ADHD was brought forward (Sheir et al., 2013). Treating ADHD by using psychostimulants has been the most preferred method because of its effectiveness in normalizing the rate of cortical thinning observed in children as well as adolescents with ADHD (Shaw, Sharp, Morrison, Eckstrand, Greenstein, Clasen, Evans & Rapoport, 2009). Similar effects of the

stimulant were also observed in normalizing the larger right anterior cingulate volume especially in children who were under treatment when compared to normal controls not on medication (Sermrud-Clikeman, Pliszka, Bledsoe & Lancaster, 2012).

The effectiveness of stimulants was viewed by others for its purpose in strengthening the connectivity of front parietal regions on fMRI comparative to children with ADHD (Wong & Stevens, 2012). They further found that the effect of these stimulants as a matter of fact improves working memory performance task as well as core symptoms of ADHD i.e. inattention, hyperactivity and impulsivity. The use of psychostimulants such as Methylphenidate was found to increase activation in some brain regions such as the left ventro-lateral dorsomedial frontal, parietal cortices and fronto-striatal regions. These stimulants therefore seem to have a role in normalizing poor brain activation level in the frontocingulate during error processing (Rubia, Halari, Cubillo, Smith, Mohammad, Brammer & Taylor, 2011). It can be understood that poor brain activation may result to some dysfunctionality leading to ADHD symptoms.

It is a fact that treatment with stimulants does affect alpha, gamma and theta activity in the brain of those suffering from ADHD (Shier et al., 2013) as indicated by studies using magnetoencephalography (MEG). Others were on point to say alpha activity in the brain seem to lessen when attention was directed towards a stimulus (Fu, Foxe, Murray, Higgins, Javitt & Schroeder, 2001), while the activity of the gamma in this regarded is more critical especially in the coordination of information processing. Exposure to any form of a stimuli may be necessarily in order to capture the attention of those with ADHD so that information may be well processed.

Wienbruch, Paul, Bauer and Kivelitz (2005) revealed that methylphenidate has a great influence in the frontal regions or rather cortical processing of children whom were diagnosed with different subtypes of ADHD. They discovered that theta activity seemed higher in the left hemisphere following the administration of methylphenidate than before. Moreover, a positive correlation was also found between D2 test improvement in attention and methylphenidate induced power changes in the theta group over the left frontal region.

Other psychostimulants drugs such as methylphenidate (Ritalin, Ritalin-SR, Concerta, Metadate, CD, Metadate ER), dextroamphetamine (Dexedrine, Dexedrine spansules, Vyvanse), and detroamphetamine and amphetamine salt combinations (Addrella, Adrella XR) were said to show improvement in symptoms of ADHD (inattention, hyperactivity and impulsivity) (Sadock & Ruiz, 2015). They also found that the new methods of preparation of these drugs including, Methylin, a chewable form of methyphenidate; Daytrana, a methylphenidate patch; and dexmethylphenidate, the D-enantiomer (Focalin), have a longer acting form Focalin XR. The aim of these drugs as indicated by Sadock and Ruiz (2015) is to maximize the target effect and minimize the adverse effects in those with ADHD or whose dose was limited by side effects.

Nonstimulants were also acknowledged as rendering effective management in treating ADHD. According to Food and Drug Administration (FDA), few drugs such as atomoxetine (Strattera), norepinephrine uptake inhibitor showed to have improvement in ADHD symptoms (Sadock & Ruiz, 2015). It was further indicated that nonstimulants such as Strattera should be consumed with caution since they may lead to the risk of developing suicidal thoughts or behavioural symptoms similar to those children taking antidepressant (Sadock & Ruiz, 2015). However, Strattera as a nonstimulant is known for its involvement in the selective inhibition of presynaptic norepinephrine transporter.

Atomoxetine on the other hand is a highly selective presynaptic norepinephrine reuptake inhibitor and it is known for its effectiveness in treating inattention and impulsivity in children and adults with ADHD (Kohn, Tsang & Clarke, 2012; Shier et al., 2013). Studies further recommended that Atomoxtine should be the initial treatment in the case of comorbid ADHD/anxiety (Kunwar, Dewan & Faraone, 2007). However, the use of this treatment has been proven to have short-term and long-term efficacy (Donnelly, Bangs, Trzepacz, Jin, Zhang, Witte, Ball & Spencer, 2009) when administered in younger children with comorbid oppositional defiant disorder (ODD), comorbid anxiety disorders (Geller, Donnelly, Lopez, Rubin, Newcorn & Sutton, 2007), and in children with Personality Defiant Disorder (PDD).

Using atomoxetine together with stimulants was said to have significant improvement on ADHD symptom particularly on individuals who does not respond fully to atomoxetine (Wilens, Hammerness, Utzinger, Schillinger, Georgiopoulus, Doyle, Martelon & Brodziak, 2009). In situations where ADHD symptoms improve but with

excecebeting symptoms of anxiety, psychomtherapy should be employed in order to target those anxiety symptoms, whereas with severe anxiety symptoms specific treatment for anxiety with medication, psychotherapy or both should be used first then focus on ADHD later (Kunwar, Dewan & Faraone, 2007). Several side effects of the substance in most instances include, diminished appetite, abdominal discomfort, dizziness, vomiting, somnolence, fatigue and irritability.

3.10.2 Psychosocial Intervention

Sadock and Ruiz (2015) stated that treating children with ADHD requires interventions such as psychoeducation, academic organization skills remediation, parent training, behavior modification in the classroom and at home, cognitive behavioral therapy as well as social skills training. In support to this view, interventions such as behavioral parent skills training (BPT), skills training and cognitive executive functioning training were also proven to be effective in the management of pediatric (Johnston & Park, 2015). Since ADHD coexist with other disorders, evaluation and treatment of these other learning or psychiatric disorders must be given attention (Sadock & Ruiz, 2015). This may suggest that psychosocial intervention as a treatment modality calls for a multidisciplinary approach which aims at assisting those who are suffers of ADHD.

According to Jonston and Park (2015), behaviour parent training in the past was the preferred strategy due to its effectiveness and was said to be a well-established treatment for children with ADHD (Daley, Van der Oord, Ferrin, Danckaerts, Doepfner, Cortese & Sonuga-Barke, 2014). BPT strategy aimed at educating parents on systematic and effective ways of coping with their children's ADHD symptoms and related disruptive behaviour problems. Furthermore, the approach strengthens a positive parent-child relationship in which it encourages appropriate child behaviour with reward systems, while implementing consistent consequences for misconduct (Johnston & Park, 2015).

Classroom intervention approach also aimed at improving children's functioning within a home environment, with peers, school interventions targeting classroom behavior and academic performance and looking at the effect of both behaviour and academic outcomes (DuPaul, Eckert & Vilardo, 2012). Other treatment ingredients include establishing of clear rules and the use of visual reminders and the use of positive and

negative consequences. Structured environment was proven to fade anxieties in children with ADHD. Both parents and teachers should work hand in hand in developing solid expectations for the child and explore methods of rewards when expectations are met as agreed upon (Sadock & Ruiz, 2015).

Children with ADHD often get rejected by their peers in most circumstances. Hence, it is important to assist them by improving peer regard while reducing social impairments as part of classroom intervention (Johnston and Park, 2015). As such, multiple interventions which mainly targeted at improving the social lives of children with ADHD by addressing peers' negative impression were put in place (Mikami, Griggs, Lerner, Emeh, Reuland, Jack, & Anthony, 2013). By training teachers in various principles whereby they can adjust their own responses to a child with ADHD to be more tolerant and positive, to train the peer group to refrain from rejecting children with ADHD and to draw positive characteristics of children with ADHD in order to dismantle peers' negative impressions (Johnston & Park, 2015). This intervention was found to be efficient for children during the school year.

Other treatments in a form of skills training worked directly with the child by specifically targeting compensatory skills such as organizational skills to improve functioning in areas damaged by ADHD (Abikoff, Gallagher, Wells, Murray, Huang, Lu & Petkova, 2013). The main purpose of this intervention is to assist children and youth to develop necessary organizational skills and combine this type of training with rewards for utilizing these skills.

Since children with ADHD struggle with poor executive functioning skills. These skills include planning, organizing time and materials, decision making, shifting from one activity to another, controlling emotions and learning from past mistakes. Improving cognitive or executive functioning by using intervention approaches which aimed at improving what are known to be core executive functioning deficits in children with ADHD will be beneficial (Johnston & Park, 2015).

3.10.3 Cognitive Behavioural Therapy (CBT)

Cognitive behavioural therapy is regarded as one of the best psychosocial intervention or treatment for ADHD (Ramsay, 2010). The therapy has further gained empirical support for its efficiency as psychological treatment for other disorders such as depression and anxiety. The therapy emphasizes on the interaction of thoughts, behaviours, and emotions and their influence on the current functioning. In general, children with ADHD are easily rejected by their own peers and that will make them develop negative perception about everything. In most cases, children develop distorted thinking which ultimately affects behaviour. CBT therefore acknowledges that negative thinking leads to negative behaviour.

CBT plays a significant role by instilling and maintaining effective coping strategies while improving individuals' quality of life. The therapy therefore targets several aspects such as helping kids to think about their thoughts, feelings and behaviour, replace negative thoughts with ones that are more realistic and positive, moreover, helps kids build self-esteem, which is negatively affected by ADHD (Brown, 2010). Although effective management of ADHD requires both medication and behaviour therapy in order to prevent long term difficulties such as substance abuse or antisocial behaviour among children with ADHD (Evans, Owens & Bunford, 2014), it remains important for children to take part in behaviour therapy because ADHD medication stop working the moment you stop taking them, but behaviour therapy will continue to teach children skills which will benefit them as they mature (Brown, 2010).

3.10.4 Behaviour modification

The National Institute of Mental Health and Scientific Sources holds the view that behaviourally oriented psychosocial interventions such as behaviour modification was found to be efficient in the management of ADHD. Children with ADHD experience challenges which are way beyond their symptoms of inattentiveness, hyperactivity and impulsivity as well as poor academic performance and behaviour at school, poor realtionships with their peers and sibblings and poor relationships with their parents. Behaviour modification being the only non- medical treatment modality, will then provide skills to parents and teachers to help them deal with children with ADHD. Moreover, the approach will teach skills to children with ADHD that will help them

overcome their impairments (Subcommittee on Attenion-Deficit/ Hyperactive Disorder Steering Committee, 2011).

Behaviour modification further enables children with ADHD to use skills learned in their interaction with other children. Thus learning these skills will be useful to these children throughout their lives. To obtain best results, behaviour modification gives emphasis that parent, teacher and child intervention should be carried out simultaneously and the following components should be adhered to (Attention-Deficit, Subcommittee, 2011):

(a) Start with the goals that the child can achieve in small steps.

(b) Be consistent-across different times of the day, different settings, and different people.

(c) Provide consequences immediately following behaviour.

(d) Implement behavioural interventions over a long haul-not for a few months.

(e) Teaching and learning new skills takes time, and children's improvement will be gradual.

3.11 Prognosis

ADHD is a debilitating disorder with fewer symptoms diminishing over a period of time. Most children who were initially diagnosed with ADHD, combined type, exhibit fewer impulsive, hyperactive symptoms as they grow older, and by the time they reach adulthood, will meet criteria for ADHD, inattentive type (Sadock & Ruiz, 2015). ADHD symptoms have devastating outcomes to personal, academic and social life of an individual. Those children whose symptoms persist into adolescent are at the risk of developing externalising behaviours, while on the other hand those with ADHD are at the risk of developing comorbid internalising disorders. At social as well as academic level school drop out, rejection by peers and low self esteem are more common. The oucome of ADHD in childhood is associated to the amount of persistent comorbid psychopathology especially CD and social disability (Sadock, Sadock, Cocro, Sussman & Ahmed, 2005).

In children with ADHD, the presence of comorbid disorders predicts poor social adjustment later in life, while, the presence of and severity of inattention in children with ADHD predicts low qualityof life at a later stage. Early identification of the disorder is crucial for effective functioning among the clinically diagnosed and minimize the developmental risk conferred by ADHD (Kutcher, Aman, Brookks, Buitelaar, Daalen, Fegert, Findling, Fisman, Greenhill, Huss & Kusumakar, 2004). Treatment approaches have been successful in diminishing symptoms of ADHD and comorbid disorders. Several studies showed that ADHD with treatment interventions yield positive responses by showing increased attention span, decreased impulsiveness and improved mood (Sadock & Ruiz, 2015). Prognosis of a child with ADHD is good suppose early identification and intervention is initiated at an advance stage.

3.12 Conclusion

ADHD is a persistent disorder and often coexist with other psychiatric disorders. Children presenting with symptoms of ADHD requires carefull and thoughrough assessment by a medical professional. It then becomes important that proper diagnosis of ADHD is done through the use of stipulated diagnostic criteria in order to rule out other possible conditions. There are also contributing factors showing a great influence in the development of ADHD and further causing multiple impairments on the execuitve functioning, psychological functioning and behaviour of the individual. The management of ADHD may require collaborative intervention strategies in order to manage symptoms from continuing to cause more harm and compromise success. Swanberg, Passno and Larimore (2005) stated that ADHD suffers with proper instruction, skills and mentoring can lead a normal life.

CHAPTER FOUR

COMORBIDITY OF INTERNALISING DISORDERS AND ADHD

4.1 Introduction

ADHD is a neuropsychiatric disorder and often comorbid with multiple psychiatric disorders, such as learning disabilities, conduct disorder, depression and anxiety disrders (Schatz & Rostain, 2006). Apart from the key symptoms, children with ADHD are known to have deficits with executive functioning, emotional regulation and social skills (Rinsky & Hinshaw, 2003). The comorbidity of internalising disorders such as anxiety and depression may be quite pressing on the symptoms often observed in ADHD. The sad part is the existence of comorbid conditions is continuing to make the diagnosis of ADHD even more challenging (Patel & Patel, 2012). Moreover, having to struggle with two conditions, the symptoms of each disorder becomes more intense. Clinically, these children are more impaired than if they had either disorder at a time than having both disoders.

ADHD as characterized by symptoms of inattention, hyperactivity and impulsivity, coupled with comorbid disorders may cause a huge decline in an individual's social and academic functioning, academic achievement and low social competence (Vollebregt, Dongen-Boomsma, Slaats-Willemse & Buitelaar, 2014). A huge decline in terms of one's quality of life is increasing and therefore becomes risky for the onset of multiple malfunctioning. Children with ADHD and comorbid disorders suffer in silent as some of these comorbid are often less noticeable and thus not given adequate attention. Developmentally, children are not matured enough to verbalize their emotions and feelings rather they express through acting out behaviour. As indicated by (Raguram, Weiss, Channabasavanna & Devins, 1996) children in most cases use somatic complains as a way to verbalize or express distress. This was the same for children with emotional disorders such as depression and anxiety that they will frequently report somatic complaints including headache, abdominal pain, fatigue, back pain and musculoskeletal pain (Leirbakk, Clench-Aas & Raanaas, 2015). Generally, comorbid conditions are distinct diagnoses that exist simultaneously with

ADHD. They do not go away once the primary condition in this case, ADHD is treated. Comorbid conditions exist in parallel with ADHD and require their own specific treatment (Wigal & Wigal, 2007). Caution should be taken in this regard, especially in the manner which these disorders present themselves.

The symptoms of ADHD and those of other disorders tend to overlap which creates a confusion whether they are symptom clusters and not true comorbidities (Kunwar, Dewan & Faraone, 2007). It is important to screen for the presence of other comorbid disorders when a child is diagnosed with ADHD (Masi & Gignac, 2017). The impact of comorbid internalising disorders i.e. anxiety and depression on ADHD symptoms, inattention, hyperactivity/impulsivity as well as self-esteem in these children, which form part of this study will be outlined.

4.2 Prevalence and prognosis of comorbid disorder

ADHD is a common disorder for children and extremely comorbid with more than 2/3 of ADHD individuals having psychiatric disorders related (Masi & Gignac, 2017). Studies on children with ADHD confirmed that psychiatric and physical comorbidities were common in children with ADHD (Larson et al., 2011). About 67% of children diagnosed with ADHD, 33% had one comorbidity disorder, 16% had two, and 18% had three or more comorbid disorders (Patel & Patel, 2012). It can be acknowledged that the prevalence of comorbid disorders along with ADHD is quite high. Both ADHD and depression/anxiety are associated with significant impairments with an estimated prevalence of 2.2-6.5% under the age of 13 years (Bittner, Egger, Erkanli, Jane, Foley & Angold, 2007). Multiple impairments observed among children in a form of scholastic challenges, rejection by others and social mulfunctioning may be as a result of these disorders which may suggest further assessment. More still needs to be explored about the long term effect of these comorbid disorders especially among boys and girls.

As indicated by American Psychiatric Association (2000), ADHD is 2 to 3 times more prevalent in boys than in girls. ADHD in girls can remain unnoticed because of the clinical presentation of ADHD symptoms when they are compared to boys. About 5.6% of girls were said to be diagnosed with ADHD during childhood and the disorder further continuing into adolescent (Visser, Zablotsky, Holbrook, Danielson & Bitsko, 2015).

However, both genders with ADHD were seen to be equally impaired (Newcorn et al., 2001) in social, academic and familial domains (Tung, Li, Meza, Jezior, Jessica, Kianmahd, Hentschel, O'Neil & Lee, 2016). Research work in the past zoomed on ADHD and comorbid externalising disorders particularly in male population. Over a period of years, longitudinal studies were able to show that girls with ADHD often experience internalising problems (Biederman et al., 2010), while children with ADHD and comorbid depressive and anxiety disorders had worse outcomes than those with one disorder (Daviss, 2008).

Gender differences in observation of ADHD symptoms proposed that girls with ADHD may be more likely to have the inattentive type of ADHD and as such may suffer from internalising symptoms and inattention whereas boys had hyperactive and aggressive symptoms (Biederman & Faraone, 2004). In girls with ADHD, what was more noticeable was a strong relationship between ADHD symptoms in particular inattention and anxiety symptom (Baldwin & Dadds, 2008). These symptoms were further connected through temperament and behavioural problems. Generally, girls with ADHD tend to display lower rates of hyperactivity, greater intellectual impairments, and lower rates of comorbid oppositional defiant disorders, conduct disorder, higher rates of internalising problems and less aggression when compared to boys (Gershon, 2002). This may suggest that in comparison, girls have more internalising problems while boys have externalising problems. Since boys have a trend to display behaviours that are explicitly hyperactive and more disruptive, they easily get referred for treatment (Coles, Slavec, Bernstein & Baroni, 2012). In support to this, damages caused by ADHD were said to be even more impairing, in girls especially with their peers (Mikami & Lorenzi, 2011). This shows that girls with ADHD still remain unnoticed hence they are less referred, while the intensity of the damage caused by ADHD becomes worse.

Often children with comorbid disorders in particular anxiety tend to display a strange pattern of response to treatment recommended for modification, especially when anxiety is an issue (Costin, Vance, Barnett, O'Shea & Luk, 2002), suggesting that the presence of other disorders may have a great influence in terms of treatment option to a point that having treated one condition does not necessarily mean the other condition is under control or managed. ADHD is always associated with negative outcomes, which may be worsened by the presence of comorbid disorders. The prognosis and

the outcome of children with comorbid ADHD may appear to be worse than children with ADHD only (Marikangas et al., 2010). It can be overwhelming for an ADHD child to deal with multiple conditions which can be too much for a child to cope with. ADHD can be regarded as a predictor to multiple pathologies. Others emphasized that similar impairments can be observed in both boys and girls with regard to poor mental health, academic and social outcomes (Biederman et al., 2005).

4.3 Comorbidity of anxiety disorder with ADHD

ADHD and anxiety are psychiatric disorders and all causing major impairments in functioning should one present with both. Up to 50% of children with Attention Deficit Hyperactivity Disorder (ADHD) meet criteria for comorbid anxiety disorder (Sciberras, Lycett, Efron, Mensah, Gerner & Hiscock, 2014). There seem to be a strong link between anxiety and ADHD in a sense that anxiety may give the impression related to some of the behaviours that can be seen with Attention Deficit Hyperactivity Disorder (ADHD). Problems such as difficulties in concentration and inattention are always common in children with ADHD as well as those presenting with anxiety. It can be suggested that the impact of anxiety may seem to have devastating effect on the lives of children with ADHD, hence it is for this reason the study is exploring further on the impact of internalising disorders i.e. anxiety on ADHD symptoms.

Anxiety disorders are a group of mental disturbances characterized by anxiety as a central symptom and can also be expressed in a form of thinking, behaviour and physical reaction (Huberty, 2004). School going children after the age of 8 are intellectually more matured and therefore anxiety provoking events are more abstract. Often their anxiety is provoked by preoccupation about their school grades, rejection by their peers, coping with a new school, and having friends and these could hinder the child's ability. In support to the above, Brown (2000) found that anxiety in ADHD may reflect a concern about competency and performance. Furthermore, the preoccupation in thoughts which is fear of poor cognitive performance seen in ADHD may definitely end up continuing to hamper the cognitive performance which in the beginning was the source of anxiety. Previous research findings managed to demonstrate that as much as anxiety reduce the symptom of impulsivity (Schatz & Rostain, 2006), it continues to impair the cognitive functioning and the executive

functioning of these children. Studies further observed that children with ADHD are prone to the risk for anxiety than children without ADHD, moreover, they are not able to manage their emotions and easily get overwhelmed with emotions. Therefore, their anxiety provoked level as such may mimic those symptoms similar to ADHD causing them to become distracted and forgetful especially when they are supposed to handle complex issues as indicated by Tuckman (2009). It then becomes evident that these children in particular will have poor scholastic achievements as well as challenges in other areas of functioning, hence the comorbidity of anxiety may be seen as influential in all these impairments seen among these school children.

Most pathogenesis happening in ADHD were seen as a result of anxiety which was believed might have a connection behind all these pathogenesis (Levy, 2004). The role played by anxiety may have a significant influence in the disorder ADHD causing multiple dysfunctions. Both ADHD and comorbid anxiety may be viewed as a marker that distinguishes subtypes of ADHD (Schatz & Rostain, 2006). Moreover, individuals with ADHD and comorbid anxiety tend to display different presentation when compared to those with pure ADHD (Pliszka, Carlso & Swanson, 1999). Comorbid conditions in ADHD particularly anxiety was seen to have a way in inhibiting impulsivity while making attentional tasks worse (Brown, 2000). On the contrary, children with ADHD and anxiety were further said to be less likely to display off tasks and hyperactive behaviour and have longer reaction times than pure ADHD.

Generally, children with ADHD and anxiety may appear to have poor attention and thus come across challenges in those tasks which require more of their attention and concentration. In support to this view, March et al. (2000) also confirmed that the ADHD/anxiety group was more inattentive than impulsive. Moreover, their hyperactive behavior may remain subtle which makes them unnoticeable when they are compared with children with pure ADHD. Inattention as character marker of ADHD, may reflect similar symptoms in individuals with ADHD and comorbid anxiety.

As much as ADHD and anxiety may appear to have some negativity on sufferers, some positive elements that were noticeable about anxiety was that ADHD children performed better on stop signal paradigms, which is a test that measures response inhibition (Brown, 2000; Manassis, Tannock & Barbosa, 2000). They further showed that children with ADHD and anxiety are specifically challenged with activities which

require working memory tasks and serial addition tasks in most cases. Typically, ADHD /anxiety children made more errors when presented with digits at longer intervals comparatively to non-anxious children (Schachar & Tannock, 1995), which suggests huge impairment of working memory. As indicated by Schatz and Rostain (2006), the administration of methylphenidate only improved working memory in the non-anxious children and not the anxious one. It may only suggest that these are children who will experience extreme challenges in areas which require calculations, reasoning and planning at the most.

In support to the above, the interest on the effect of anxiety on ADHD became the focus area in which more focus was on how anxiety affects impulsivity and cognitive functioning (Schatz & Rostain, 2006). Children with ADHD at the most have cognitive impairment or problems. Cognitive problems refer to difficulties with attention, reward response, executive function and cognitive processes and there is no single cognitive dysfunction which characterizes all children with ADHD (Nigg, Nikolas & Burt, 2010). In a study conducted on the effect of reward and response cost on response inhibition in ADHD, disruptive, anxious and normal children, it was discovered that increased response inhibition in anxious children became clear in situations where there were extreme demands for response inhibition (Oosterlaan, Logan & Sergeant, 1998). Tannock (2009) found that anxiety at certain level may enhance vigilance and regulate impulses, however, may disrupt more cognitively processes like working memory. Anxiety may be viewed as having a dual role in the cognitive functioning. The presence of anxiety may have an effect both as a remedy on cognitive deficits seen in ADHD while on the other hand decreasing impulsivity (Beck & Schatz, 2006). Although there are some benefits that comes with anxiety traits, there are still some difficulties that are noticeable in children with anxiety.

According to Huberty (2004), other character traits often observed in children with anxiety disorders are difficulties in concentration, memory problems, difficulties with problem solving and academic performance, impulsiveness and sleep problems to mention a few. Both ADHD and anxiety disorders appear to share similar symptomatology in terms of cognition, behaviour and physical. Again, both disorders can be independent of each other and therefore a challenge to individuals in understanding how both disorders works. A study conducted by Sciberras, Lycett,

Efron, Mensah, Gerner and Hiscock (2014) on anxiety in children with ADHD, also confirmed that children with ADHD and comorbid anxiety have poor quality of life and more problematic behavior and daily functioning especially when less than two anxiety comorbidities are present than children without anxiety. They further outlined that multiple anxiety comorbidities were linked with poorer functioning for children with both ADHD-Inattentive and ADHD –Combined presentation.

Jensen, Hinshaw, Kraemer, Lenora, Newcorn, Abikoff, March, Arnold, Cantwell, Conners and Elliott (2001), viewed anxiety as a predictor and outcome and further associated with negative affectivity and disruptive social behaviour than with fearful/phobic behaviours. Other viewed the presence of anxiety as having an effect on conduct disorder. Anxiety may therefore be seen as a gateway to other external disorders. Inattention as a symptom appears to be a character marker in both ADHD and anxiety which seem to be a challenge in these individuals i.e. affecting their ability to concentrate and finish school work (Baldwin & Dadds, 2008). Inattention and anxiety since were said by others to be having a strong link and more common in girls than boys (Baldwin & Dadds, 2008). This behaviour often may be unnoticed since the symptoms are not external unlike CD and ODD, which may put them at the risk of being easily referred.

Other challenges experienced by these individuals are peer rejection, academic difficulties and this is likely to put these children at risk of developing negative outcomes (Keiley, Lofthouse, Bates, Dodge & Pettit, 2003). Often children with ADHD as a result of their behaviour they are misunderstood either by caregivers, teachers and their own peers, and therefore underachieve in their school work which perhaps makes them to be labelled as being intellectually not fitting. Therefore, they receive negative attitude from the rest which makes them even more vulnerable to anxiety, hence this anxiety level will continue to exacerbate ADHD symptoms perhaps they have the disorder. This was supported by Tsang, Kohn, Efron, Clarke, Clark, Lamb and Williams (2015) who found that ADHD with comorbid anxiety was associated with greater ADHD severity. As indicated by Skinner and Piek (2001), anxiety in children with ADHD may lead to withdrawal in most activities and therefore lead to fear of future participation. Several characteristics of children with ADHD and comorbid anxiety were associated with neurocognitive impairments. Children with ADHD and anxiety were

observed to have reduced auditory emotion recognition especially when using dichotic listening tasks (Manassis, Tannock & Barbosa, 2000).

In a comparison group with anxiety disorders, children with ADHD and anxiety were further found to have less auditory perception of anger (Manassis, Tannock, Young & Francis-John, 2007). These children in particular display some low level of emotion recognition which is observed as a result of inattention arising from of their anxiousness which ultimately impacts negatively to their ability to pay close attention. Generally, ADHD children with anxiety have a tendency to perform worse on more cognitively complex and mentally effortful tasks (Jarrett, Wolff, Davis, Cowart & Ollendick, 2016). Keiley and colleagues (2003) further elaborated that harsh parental discipline may result to both internalising and externalising behaviours mostly displayed by these children. This was supported in a study conducted emphasizing that the prevalence of ADHD, depression/anxiety in children is higher when parents have low socio-economic position.

4.4 Comorbidity of depression disorder with ADHD

ADHD is the most common diagnosis in child psychiatry which appears to be more challenging to diagnose and treat when there is a comorbid depressive disorder (Brunsvold, Oepen, Federmn & Akins, 2008). The prevalence of depression among individuals with ADHD ranges from 13% to 27% particularly in a community population whereas in a clinical sample it is estimated at 60% (Furman, 2005). ADHD has its onset from early childhood but comorbid disorders like depression does not have a specific onset, however, develops post pubertal phase, yet the age of risk for depression onset is not clear (Eyre, Langley, Stringaris, Leibenluft, Collishaw & Thapar, 2017).

Children presenting with ADHD and depression often are more impaired than those with ADHD or depression alone (Blackman, Ostrandr & Herman, 2005), with a high increase rate in girls more than boys (Brunsvold et al., 2008). Individuals with ADHD and depression occurring together display even greater levels of psychosocial impairment than individuals with either ADHD or depression (Biederman, Ball, Monuteaux, Mick, Spencer, Mc-Creary, Cote & Faraone, 2008; Daviss, 2008), suggesting that ADHD in comorbid with other disorders (depression) can be disastrous should one present with such a diagnoses. Therefore, identifying those with ADHD

who are at the risk of depression is rather important for the purpose of early intervention and prevention (Eyre et al., 2017).

Depressive disorders as stipulated in the DSM IV TR (American Psychiatric Association, 2000) are characterized by a pervasive and persistent low mood that is accompanied by low self-esteem and by a loss of interest or pleasure in normally enjoyable activities. In children, the episode lasts from 7 to 9 months. As outlined in the DSM criteria, symptoms of childhood depression often include academic decline, withdrawal from friends and family, thoughts or actions of self-harm, problems with sleep, feelings of being misunderstood, unexpected crying. These signs and symptoms can prevail without being noticed in most children or perhaps become noticed at a later stage following major life complications.

Studies have made several investigations and findings about the effect of comorbid depression on ADHD. Many impairments as a result were outlined and indeed children with ADHD and comorbid depression were found to be extremely impaired. It was observed that patients with ADHD also suffer from “demoralization”, or a low/sad mood and chronic unhappiness (Turgay & Ansari, 2006). Children with ADHD are largely at risk of receiving negative responses in a form of academic setbacks, social rejection and family tension (Postner, Siciliano, Wang, Liu, Sonuga-Barke & Greenhill, 2014) and eventually they end up feeling frustrated and demoralized as a result of their symptoms.

Part of their depression may arise over a fact that they lack control over what happens in their environment as well as negative interaction with school and at home. With continuous negative experience over a period of time they become discouraged and demoralized. In view of Herman, Lambert, Reinke and Lalongo (2008), an increasing number of impairments due to ADHD as well as the negative environmental circumstances may lead to those with ADHD to be in a depressive state and eventually develop depression. It can be suspected that the emotional state arising from their incompetence may be a risk factor to other impairments in these individuals, hence it is for this reason that the impact of depression on how they influence the symptoms of ADHD be well explored. Major depression in most cases exacerbates the symptoms and dysfunctions of ADHD and as such individuals with ADHD who later develop major depression are likely to present with severe dysfunctions than those who have major

depression alone (Turgay & Ansari, 2006). ADHD and comorbid depression in children is mostly associated with school difficulties, school refusal, withdrawal, negativism, aggression and antisocial behavior (Spencer, Biederman & Mick, 2007). All these may lead to a feeling of worthlessness when they are compared with children with ADHD and without depression.

The new DSM-5 (APA, 2013) has come up with a new category of Disruptive Mood Dysregulation Disorders (DMDD) and irritability which are said to be related to mood disorders. Depressed children often show more irritability and inattention than sadness, which in most instances leads to a misdiagnosis of ADHD. Irritability may be defined as a tendency to react with anger, grouchiness or tantrums disproportionate to the situation (Stringaris & Goodman, 2009). Children with ADHD often they are touchy and easily annoyed as one of the items outlined in the DBD scale. In their view, Vidal-Ribas, Brotman, Valdivieso and Leibenluft (2016) and Whelan, Stringaris, Maughan, Barker (2013), stated that irritable dimension has been associated elevated risk of emotional disorder and depression in the general population. It can be supported that the presence of depression may be a risk factor in exacerbating symptoms of ADHD.

An attempt in examining children with ADHD and depression revealed that children with ADHD and comorbid depression show more impairments in social and academic competence, have aggression and are suicidal (Blackman, Ostrander & Herman, 2005). This was also supported by the study conducted by Connor, Edwards, Fletcher, Baird, Barkley and Steingaurd (2003), which found that severe symptoms of ADHD and high rates of aggression particularly in children with ADHD and depression. Moreover, these children are likely to develop conduct problems associated with ADHD and depression which can be observed through impaired academic and social functioning (Blackman et al., 2005). Depression as an internalising disorder coexisting with ADHD may be viewed as a gate way to conduct disorder which is a more externalising disorder.

There seem to be a relationship between internalising symptoms and social functioning in children with ADHD and depression. Depression as it is regarded as an internalizing disorder may pose a risk factor leading to multiple impairments as well as negative outcomes. This supports the notion and the findings by several researchers that internalising disorders are more prevalent among girls than externalising disorders,

therefore proper diagnoses and treatment option are crucial (Subcommittee on Attenion-Deficit/ Hyperactive Disorder Steering Committee on Quality Improvement and Management, 2011).

It was observed by other researchers that female patients with both depression and comorbid ADHD have earlier ages of onset of depression, longer durations of depressive episodes and higher rates of suicide and hospitalization than those with depression alone (Biederman et al., 2008). Even after following a successful treatment for their depression, these individuals continued to have higher rates of depressive recurrence when compared to those with depression alone (Rohde, Clarke, Lewinsohn, Seeley & Kaufman, 2001). Solomon, Keller, Leon, Mueller, Lavori, Shea, Coryell, Warshaw, Turvey, Maser and Endicott (2000), further emphasized that about 60% of persons with depression are likely to experience major depression as a recurrent illness. They further emphasized that the risk of the recurrence disorder increases with every episode and therefore decreases as the road to recovery increases.

Others observed further that the use of medication to treat ADHD symptoms continue to cause side effects like insomnia, appetite, changes, sleepiness, tearfulness and moodiness which are similar to symptoms of depression (Diler, Uguz, Seydaoglu, Erol & Avci, 2007). It looks like the management of ADHD may result to extreme negative outcomes which may be seen as a predictor for depression. The use of treatment for the management of ADHD was viewed as the leading cause of negative side effects and suicidal thoughts being one of the effects (Emilsson, Gustafsson, Ohnstrom & Marteinsdottir, 2017). According to Spencer, Biederman and Mick (2007), the diagnosis of major depression may predict lower psychosocial functioning, higher rate of hospitalization and impairments in interpersonal and family functioning in children with ADHD with depression. The presence of depression looks like it directly worsens ADHD symptoms. Similar results were found in relation with comorbid mania in children with ADHD wherein additional psychopathology, psychiatric hospitalization, severe impaired psychosocial functioning and family history of mood disorder were expected (Spencer, Biederman & Mick 2007).

The relationship between ADHD and depression was believed to have shared neurobiological abnormalities which may connect the two disorders (Posner et al., 2014). Both disorders appear to have neurbiological vulneralities and environmental affects which through interaction, information which can send depressive signals from the environment may have a strong influence or effect in a hyperactive child who already has neurobiological vulnerabilities of depression (Posner et al., 2014). Studies using magnetic resonance imaging, identified hippocampal abnormalities with depression (MacQueen, Campbell, McEwen, Macdonald, Amano, Joffe, Nahmias & Young, 2003). Findings revealed that depressed individuals had reduced hippocampal volumes, impaired hippocampal functioning and hippocampal hyperperfusion (Videbech Ravnkilde, Fiirgaard, Clememensen, Egander, Rasmussen, Christensen, Sangill & Rosenbergy, 2001) as detected by Positron Emission Tomography (PET).

Other emerging studies on the analysis of hippocampal and amygdala volume in ADHD children further revealed enlarged hippocampal volumes bilaterally in ADHD (Plessen, Bansal, Zhu, Whiteman, Amat, Quackenbush, Martin, Durkin, Blair, Royal, Hugdahl & Peterson, 2006; Perlov, Philipsen, van Elst, Ebert, Henning, Maier, & Hesslinger, 2008). These abnormalities happening in the brain may be associated with the developing of depression in these children. According to Vakalopoules (2007), these children continue to display neuro-cognitive deficits i.e. memory loss, poor concentration and rumination which are said to be related to the hypofunctioning monoaminergic system.

According to Vermetten, Vythilingam, Soutwick, Charney and Bremner (2003), adhering to treatment with antidepressants partially normalize hippocampal volumes as well as neurogenesis within the hippocampus which are believed to play a crucial role in the responsiveness of an individual to therapies for depression (Sahay & Hen, 2007). Birmaher, Axelson, Monk, Kalas, Clark, Ehmann, Bridge, Heo and Brent (2003), also stated that the identification and treatment of pediatric MDD at an early stage may reduce devastating long term results in these individuals.

4.5 Comorbid disorders on self esteem

According to Dan and Raz (2015), ADHD affects many aspects of life such as poor peer relationships, aggression and learning problems, which in turn are associated with academic failure and increased risk for development of self-esteem. So far many studies have reported negative relationships between ADHD and self-esteem (Edbom, Granlund, Lichtenstein & Larsson, 2008). Self-esteem as defined by Palaniappan (2016), is a cognitive and emotional concept of an individuals has about himself. The concept is further said to be based on the relationship between perceived competence and the individual's aspirations in any one specific area of life.

ADHD from its early onset and diagnosis is a childhood disorder with symptoms often identifiable either at home or school environment. According to Jellinek (2010), a child with ADHD by the age of ten would have been exposed to non-directive responses or corrective comments from their teachers. This is too much criticism for a child and therefore crumbles their self-esteem. The link between ADHD and self-esteem was based on the multiple failures that individuals report when they are in different settings i.e. school and family, which leads them to experience a lower perception of their competency as compared to their peers (Mazzone, Postorino, Reale, Guarnera, Mannini, Armando, Fatta, Peppo & Vicari, 2013). Although, the use of medication to certain extend was found by many to reduce behaviours that elicit negative comments by 50% to 75%, the burden of criticism and the feeling that a lot of what he is about is not acceptable still remains with them (Jellinek, 2010).

Quite often children with ADHD are taking treatment for the management of their symptoms. However, that continues to expose them to challenges of self-esteem by family members queering the benefits of treatment and siblings criticizing them for taking a pill (Jellinek, 2010). On the contrary, these individuals may have learning disabilities which are common comorbidities in children with ADHD (Jellinek, 2010). Other challenges observed in children with ADHD is difficulty with reading social cues which may put them at the risk of rejection by others and not to be popular amongst others. As clearly said by Jellinek (2010), children with low self-esteem suffer from the pain of being themselves. In addition, these children always anticipate failure rather than success no matter how much effort they can put, suggesting that with continuous

negative corrective comments thrown at them will only make their situation become worse.

The intensity of ADHD puts those with the disorder at the risk of criticism and negative inputs by others, hence they experience crisis based on perceived competence and the importance to succeed in that particular area. In these individuals' acknowledgement by one self and those other important people is so significant that it puts them under extreme pressure and at risk for impairment in self-esteem (Palaniappan, 2016). This suggests that ADHD children are so much preoccupied in thoughts and conscious about being judged by the world, hence, they worry much and have low self-esteem. It can be understood that positive self-esteem is crucial for a healthy psychological development among those individuals.

Research studies in the past were able to show that children and adolescents suffering from ADHD have low levels of social skills and self-esteem when they are compared to their normal counterparts (Shaw-Zirt, Popali-Lenane & Chaplin, 2005**;** Bussing, Zima & Perwien, 2000). In the same way, Slomkowsk, Klein and Mannuzza (1995) stated that social function and self-esteem may play a role on future outcome in ADHD. On the other hand, Graetz, Sawyer and Baghurst (2005) found that children with ADHD have impaired self-concept and self-esteem. They further explained self-concept as the totality of the individual's cognitive image, definition of the self, ideas, beliefs and attitude about the self. One may argue that frustration in a form of interpersonal crisis may arise as a result of failure to pull through this journey of self-discovery.

Pisecco, Wristers, Swank, Silva and Baker (2001) indicated that among ADHD children, poor self-concept was related to academic competence, contributed to the development of disruptive antisocial behaviours in early adolescence. Nevertheless, the same group of children were again found to have more internalising behaviour and had lower self-esteem than the hyperactive type who exhibited externalising behaviour and had a higher self-esteem (Graetz, Sawye & Baghurst, 2005). Perhaps it can be suggested that there may be a link between internalising behaviour, low self-esteem and externalising behaviour and high self-esteem that can be observed in these groups.

A study conducted by Silverstone and Salsali (2003) was able to show that low self-esteem increases the exposure for development of psychiatric disorders (i.e. anxiety depression, eating disorder and substance disorder) and the presence of a psychiatric disorder, which in turn lowers self-esteem. On the contrary, increased anxiety was found to lower self-esteem in children with comorbid disorders (Taylor & Pilar, 1992). Low self-esteem may therefore be seen as a precursor to this multiple psychiatric disorders that one may have. In his study Palaniappan (2016), demonstrated that comorbidities do have a great influence on the level of self-esteem in children with ADHD. He further indicated lower self-esteem especially in areas of social domain in ADHD children presenting with comorbid internalising disorders. Others further observed that aggressive boys with ADHD displayed higher rates of depressive symptomatology and lower levels of self-esteem than their counterparts (Treuting & Hinsaw, 2001).

Comorbid disorders in a form of anxiety and depression may pose negative outcomes on the well-functioning of an individual. Therefore, Salsali and Silverstone (2003) affirmed that self-esteem is a vital factor of psychological health and often goes along with psychiatric disorders. Self-esteem thus becomes an etiological component for psychiatric disorders and moreover is a role player in quality of life in these individuals, (Ruggeri, Bisoffi, Fontecedro & Warner, 2001). Psychological wellbeing in this regard for a healthy development is essential. Research study conducted continues to elaborate that individuals with major depression do display low self-esteem (Harter & Jackson, 1993). Other researchers showed that the more anxious an individual becomes, the more the level of self-esteem decreases (Taylor & Pilar, 1992). Lowered self-esteem as observed by Erkolahti, Saarijarvi, Llonen and Hagman (2002) becomes a psychological trademark in most individuals with psychiatric conditions.

According to Biederman (2005), ADHD touches many aspect of life which also relate with academic failure and high risk for development of low self-esteem. A study by Dan and Raz (2015) showed a strong relationship among ADHD, self-esteem and test anxiety. In their study, they observed that most children develop some level of test anxiety and therefore display specific reaction towards examination situations. Their test anxiety level in particular affected many areas of functioning. Dan and Raz (2015) further outlined that most of these individuals experience some impairments in areas

such as emotional area: (wherein they experience mental stress, excitement and fear), cognitive area, (they experience some forgetfulness, problems in concentration and fear of failure) and behavioural area (where they often experience different lifestyles and improper ways of studying). All these challenges experienced by these individuals are similar to those of ADHD symptoms and internalising disorders (Whitaker, Sena, Lowe & Lee, 2007).

ADHD and comorbid disorders may be a setback towards achieving goals and succeeding in one's life. In order to provide effective intervention much focus should be on treating ADHD. Beside medication and behaviour modification, Jellinek (2010) emphasized on the new approach that will preserve and enhance self-esteem with few guidelines:

(a) Set reasonable expectations more especially for children with ADHD. When you set goals too high, they will ensure a sense of failure.

(b) Expect normal variations in a child's performance. With ADHD children some days performance may reach its peak and some days may be low.

(c) Consider other factors. When evaluating performance of children with ADHD, take into account: comorbidities, family problems and chronic illness.

(d) Focus on building strengths rather than remediating weaknesses whenever possible. If a child is good in certain activities capitalize on that because it will only make them feel good about themselves, rather than spending more hours trying to overcome his weakness.

(e) Think about summer as a time to take the pressure off and cultivate successes. Encourage families to consider energetic camp, one with activities suitable to the individual child, rather than endless hours of academic skills building and remediation.

(f) Encourage play! Always remind families that kids with ADHD crave moments of senseless fun with their parents.

Through attaining these guidelines, it will help in improving children with ADHD's psychological emotional wellbeing. In support of this, positive self- esteem may assist

the children with ADHD in order to cope with failures or difficulties in everyday life (Edbon, Granlund, Lichenstein & Larson, 2008).

The use of medication was investigated further to assess the effect of these drugs as a way of improving self-esteem in children with ADHD. A study conducted reported that individuals who were on methylphenidate showed significantly higher self-esteem comparative to un-medicated individuals (Frankel, Cantwell, Myatt & Feeinberg, 1999). In addition, another study conducted revealed improvement of self-esteem that in turn lead to an impact on emotional well-being and quality of in those individuals treated with atomoxetine (Sobel, Bansal, Mia, Sanchetz, Mazzone, Durkin, Liu, Hao, Ivanon, Miller, Greenhill & Peterson, 2010).

4.6 Comorbid disorders and ADHD subtypes

Attention Deficit Hyperactivity Disorder is a heterogeneous disorder characterized by different subtypes and several comorbidities (Biederman, 2005). DSM criteria only recognizes three subtypes which are the predominantly inattentive (ADHD-I), predominantly hyperactive (ADHD-H) and the combined type (ADHD-C) (American Psychiatric Association, 2013), with each subtype presenting itself differently in relation with the psychiatric disorder. Children with ADHD-Combined Subtype (ADHD-C) have more severe ADHD symptoms than children with ADHD-Inattentive subtype (ADHD-I) (Gross-Tsur, Goldzweig, Landau, Berger, Shmueli & Shalev, 2006). A study by Willcut, Nigg, Pennington, Solanto, Rohde, Tannock, Loo, Carlson, McBurnett and Lahey (2012), demonstrated that children with ADHD-C were 6.5 times more likely to meet criteria for comorbid Generalized Anxiety Disorder when compared to healthy individuals. They further indicated that during adolescence phase, particularly the ADHD-Inattentive subtype (ADHD-I) and ADHD-Hyperactive/Impulsive (ADHD-H/I) were likely to meet criteria for co-occurring generalised anxiety disorder.

Research studies using parents and teachers showed that children with ADHD-C displayed more symptoms of more internalising symptoms than children with ADHD-I (Gross-Tsur et al., 2006). Moreover, children in the clinical groups did not seem to differ in terms of internalising disorders. This was in line with the idea that these children depending on their clinical groups are likely to have a recurrence of psychiatric disorders (Willcut et al., 2012). Though the rates of comorbid anxiety and depression

did not differ between the two subtypes (Power, Costigan, Eiraldi & Leff, 2004), signifying that there might be a slight difference between the comorbid psychiatric disorders. A study by Carlson and Mann (2002) on sluggish cognitive tempo predicting different patterns of impairments, illustrated that children with ADHD particularly the inattentive subtype seems to display some impairments such as sluggish cognitive tempo and this subgroup in particular had more anxiety and depression problems and fewer externalising problems. In the same way, Mayes, Calhoun, Chase, Mink and Stagg (2008) observed that similar ADHD subtype had a slower processing speed than children with ADHD-C subtype and this could relate to co-occurring anxiety having a negative impact on ADHD symptoms.

Others observed a high incidence of internalising disorder especially depression among the ADHD-C subtype than in ADHD-I subtype (Ushijima, Usami, Saito, Kodaira & Ikeda, 2012). In contrary to this, the predominantly inattentive subtype commonly had greater comorbid depression (Hurtig, Ebeling, Taanila, Miettunen, Smalley, McGough, Loo, JÄrvelin & Moilanen, 2007). In their study, Cueli, Gonzalez-Castro, Alvarez, Nunez and Fernandez (2014) on anxiety and selective attention in ADHD subtypes, observed that ADHD Combined subtype displayed higher anxiety traits, while the inattentive subtype showed higher levels of state anxiety. The severity of internalising disorders seems to be dependable on the ADHD subtype. Having two or more ADHD subtypes might be associated with poorer functioning than a child having one or just pure ADHD as this may lead to poor quality of life, daily functioning and behaviour.

Castanga, Calamia and Davis (2017), reported that children with ADHD-Combined subtype (Inattentive and Hyperactive/impulsive) are sensitive to personal failure. Being sensitive makes them develop an attitude of reframing to participate in activities because they already perceived themselves as not being able. Self-esteem may be accompanied by anxiety and depression making ADHD symptoms even worse. Continuous use of negative and corrective words may be rather too much for them because they are constantly being judged wrong for their behaviour which they have no control over.

4.7. Treatment of comorbid disorders

The use of psychostimulants has been the first-line of treatment for individuals with attention-deficit/hyperactivity disorder (ADHD) (Faraone et al., 2005). Having ADHD and comorbid conditions complicates treatment and makes the diagnoses even more difficult (Bailey & Ofoemezie, 2013). Faraone, Ashherson, Banaschewski, Biederman, Buitelaar, Ramos-Quiroga, Rohde, Sonuga-Barke, Tannock and Franke (2015), maintained that, the management of ADHD in the presence of other disorders can be impossible hence the most serious disorders must be treated first. This was in line with what Katzna, Bilkey, Chokka, Fallu and Klassen (2017) who suggested that the most impairing condition should generally be treated first especially when ADHD coexist with other psychopathologies. Again the process of diagnosing now and then requires the medical professionals to determine whether if the comorbid is a separate condition or if it is a result of ADHD symptoms (Bailey & Ofoemetzie, 2013). In doing one will be able to arrive at the actual diagnosis of the disorder then the rightful treatment method since there is some similarities of symptoms in both disorders.

Amphetamines and methylphenidate have primary effects to increase the central dopamine and norepinephrine activity which plays a significant role in executive and attentional function. Since individuals with ADHD experience an imbalanced secretion of chemicals or neurotransmitters around many brain areas, the supply of stimulants thus mediates the situation in order to avoid behaviors that may arise as a result of these chemical imbalance. Research body so far is of the view that multiple neurotransmitters and brain structures play a vital role in ADHD. On the other hand, neurobiological factors mediating behaviours linked with ADHD shared similarities to a certain extend with those involved in comorbid disorders (Farb & Ratner, 2014; Schwartz & Kilduff, 2015). Much attention on comorbidities should be taken into account when considering pharmacotherapy in an individual with ADHD.

The outcomes of using stimulants may be complicated to a certain extent if there are comorbid disorders. Bleck, DeBate and Olivardia (2015) maintained that the disorder anxiety adds another element to ADHD, as you are both developing strategies for ADHD symptoms while working with the resulting anxiety at the same time. As a result, sufferers continue to remain stuck in their old behaviour for long because of the

paralyses caused by anxiety, furthermore this may even hider strategies that they may learn from to help them to be on top of ADHD (Tuckman, 2012). As suggested by others, some anxiety disorders may only be assisted once ADHD symptoms are treated and managed (Bailey & Ofoemezie, 2013). However, anxiety disorders in a long run are been overlooked because some side effects of stimulant medication are anxiety or nervousness (Baily & Ofoemezie, 2013).

Although stimulant medication may be regarded to be effective, observed side effects which were as a response to medication were increased heart beat and dry mouth. Therefore, due to lack of tolerance to stimulants, individuals may be put on non-stimulant along with a selective serotonin reuptake inhibitor, which has anxiety reducing effects (Kellogg, 2016). On the other hand, others suggested that one may opt to take medication for the other disorder and again undergo behavioural therapy for the other disorder especially if they are skeptical to take several medications at once (Tuckman, 2012). The use stimulants associated with serotonergic drive improves comorbid anxiety associated with ADHD (Heal, Smith, Gosden & Nutt, 2013).

The chemical dopamine is often implicated in depressive symptoms. Therefore, the use of psychostimulants may have an influence on improving depressive symptoms which is likely to differ from antidepressants (Malhi, Byrow, Basset, Boyce, Hopwood, Lyndon, Mulder, Porter, Singh & Murray, 2016). On the contrary, the use of methylphenidate and modafinil was observed by others as neither effective when it comes to reducing depressive symptoms (Faraone & Larsson, 2018). Faraone and collegues (2015) combined treatment mostly used for the management of ADHD in individuals with comorbidity.

Generally, psychostimulants were observed to be ineffective in treating undifferentiated symptoms of depression. However, few studies supported the efficacy of psychostimulants for bipolar depression (Dell'Osso & Ketter, 2013). Pursuing this further, others argued that lack of efficacy of psychostimulants are as a result of poorly defined psychopathology and as such psychostimulant effects may be more pronounced in selected symptoms domains (Hegerl & Hensch, 2017). It can be observed that the use of stimulants may result to side effects while providing treatment on the other hand. Finally, atomoxetine has been a success story in the management of ADHD with the comorbidity of anxiety and depression (Bangs, Emslie, Spencer,

Ramsey, Carlson, Bartky, Busner, Duesenberg, Harshawat, Kaplan, Quintana, Allen & Sumner, 2007; Kratochvi, Newcorn, Arnold, Duesenberg, Emslie, Quintana, Sarkis, Wagner, Gao, Michelson, & Biederman, 2005).

The treatment of ADHD with comorbid disorders requires to be handled with caution. Since ADHD is a childhood disorder with some biological defects implicated. It will be essential that to observe that comorbid internalising disorder comes along as a result of the severity of ADHD. Therefore, treating comorbid disorders at first is important so that effective strategies in treating ADHD may be employed for the well-functioning of those with the disorder.

4.8 Conclusion

The existence of comorbid disorders complicates and worsen the severity of ADHD disorder. It looks like having to deal with one clinical diagnoses is much better than having to cope with multiple diagnoses. It is during elementary phase where children experience challenges that may affect them emotionally and may also struggle to deal with esteem issues. Thus, it can be understood that children have a more burden of deaseases to cope with, which can be overleming foe them considering their developmental level. It becomes important that educators and parents are made aware of such turmoil and how severely can hamper with the child's future outcomes. Several studies thus far have identified impairments related to ADHD and cormorbid disorders. Therefore, more attention to this co-existing should be given so that eventually these children are referred and be given proper intervention.

CHAPTER FIVE

RESEARCH METHODOLOGY

5.1 Introduction

This section outlines various levels in which the methodology of the current study was conducted. I start with a discussion on qualitative research which is an approach that was used for the current study. The strength and weakness of this approach are emphasised. In the second part of the chapter, I discuss the research design that was chosen as appropriate for this study. Following that, I briefly give an outline of the quasi research design, including the experimental design that was used in the research exploration. The third section looks at the location for the study. The fourth section provides information on how sampling of the population was done. The section on data collection provides information on how data was collected. The data collection method that was chosen is explained including the data collection instruments. The data analysis method is described in detail while matters relating to confidentiality of participants are highlighted. The last part of this chapter focuses on the ethical considerations for the present study. The research methodology for the current research study will include the research approach, research design, location of the study, population, sample, research instruments, entry negation, pre-testing data collection, data analysis, dissemination of results and ethical considerations

5.2 Research approach.

Quantitative research is a research which is conducted using a range of methods which use measurements to record and investigate aspects of social reality (Bless, Hingson, Smith & Kagee, 2006). On the other hand, Cresswell and Poth (2016) defined quantitative research as a means of testing objective theories by examining the relationships among variables and these in turn can be measured, typically on an instrument, so that numbered data can be analyzed using statistical procedures. Similarly, Polit and Beck (2008) clearly indicated quantitative as a numeric information that results from some type of formal measurement and that is analyzed with statistical procedures. It is through this research method that data is collected or coded in

numerical forms and to which statistics may be applied to determine the significance of the findings (Blanche, Blanche, Durrheim & Painter, 2006). The research is further said to be conducted within the traditional scientific method which is a systematic and controlled process and that the findings will be based on empirical evidence.

The researcher employed quantitative research approach for this current study because it enabled her to obtain an in-depth information within a brief period of time. The quantitative study does not consume a lot of time and is characterized by objectivity. This was the preferred method since it enables the researcher to describe variables, examine relationships among variables. Again, the researcher was able to ask specific narrow research hypotheses to determine the cause and effect interaction between variables (Mertler & Charles, 2005; Burns & Groove, 2011). Through this approach, the researcher obtained more information from teacher's and parent's responses about the child's behaviour as it was observed in class or at home and both the teacher and parent were required to mark the appropriate column depending on the level of degree at which the behaviour is being expressed. Moreover, children themselves were requested to rate their own emotions and feelings at a certain level of degree. The approach was more effective and more reliable in terms of responses. Unlike with qualitative approach whereby children not honestly be expressive and comprehend the level of pathology regarding own behaviour.

This research approach came with the benefits that it enabled the researcher to expand her population for screening purposes in order to select the clinically diagnosed ADHD children. By having a large population increases the chances of identifying those children who may possibly have ADHD. Suggesting that you may not find an ADHD diagnosed child in every participant you may come across. It was during this approach whereby a large sample size was randomly selected from a larger population with the aim to generalize the results to this population. Mokobane, Pillay and Meyer (2017) on their study where they researched on the comorbidity of Attention Deficit Hyperactive Disorder and major depression in primary school children, also employed a quantitative approach and quasi experimental design similar to the current study. Another study done in Nigeria on primary schools on the prevalence and co-morbid conditions (Adewuya & Famuyiwa, 2007), and also a study by Meyer et al (2004) on heterogenous group in Limpopo, did use the quantitative approach in the same way as the current

study.

5.3 Research design

In the current study, a quasi-experimental research design was employed as the subjects could not be randomly assigned. According to Shadish, Cook and Campbell, (2002), quasi-experimental research design, like experimental design does test casual hypotheses and by definition the design lacks random assignment. Shadish and collegues (2002) further stated that a quasi- experimental design identifies a comparison group that is as similar as possible to the treatment group in terms of baseline characteristics. It is a comparative study where the performance of emotional disorders was compared between children with ADHD and a matched control group. Boys and girls were analysed separately. Children with ADHD and a neurotypical control group are compared on areas of comorbid internalising disorders (anxiety and depression) and self-esteem. The gender groups were compared.

This was a research design of choice for the study because it gave the researcher an opportunity to have both the experimental group and a control group which were subjected to comparison and intervention. For the purpose of the study comparison was only done between variables and no intervention was conducted since it was not the focus of the present study. The researcher further compared score of those who were clinically diagnosed and matched them to non ADHD group. The cases were compared with the control group and were matched according to age and gender. Since teachers were not supposed to diagnose ADHD, they were requested to screen all learners in all grades so that every child had a chance of equal opportunity to participate in the study. Kashala and colleugues (2006) in their study on co- existing symptoms and risk factors among African school children with ADHD and those in developing countries also employed a quasi-experimental research design. Although a study by Mayes and colleugues (2009) focused on differences in sleep problems in children with ADHD subtypes and comorbid anxiety, depression and Oppositional- Defiant Disorder, the same design with the current study was employed. A study done on internalising disorders as common mental disorders in children and adolescents by In- Albon (2012) also used similar design as the current study.

5.4 Location of the study and population

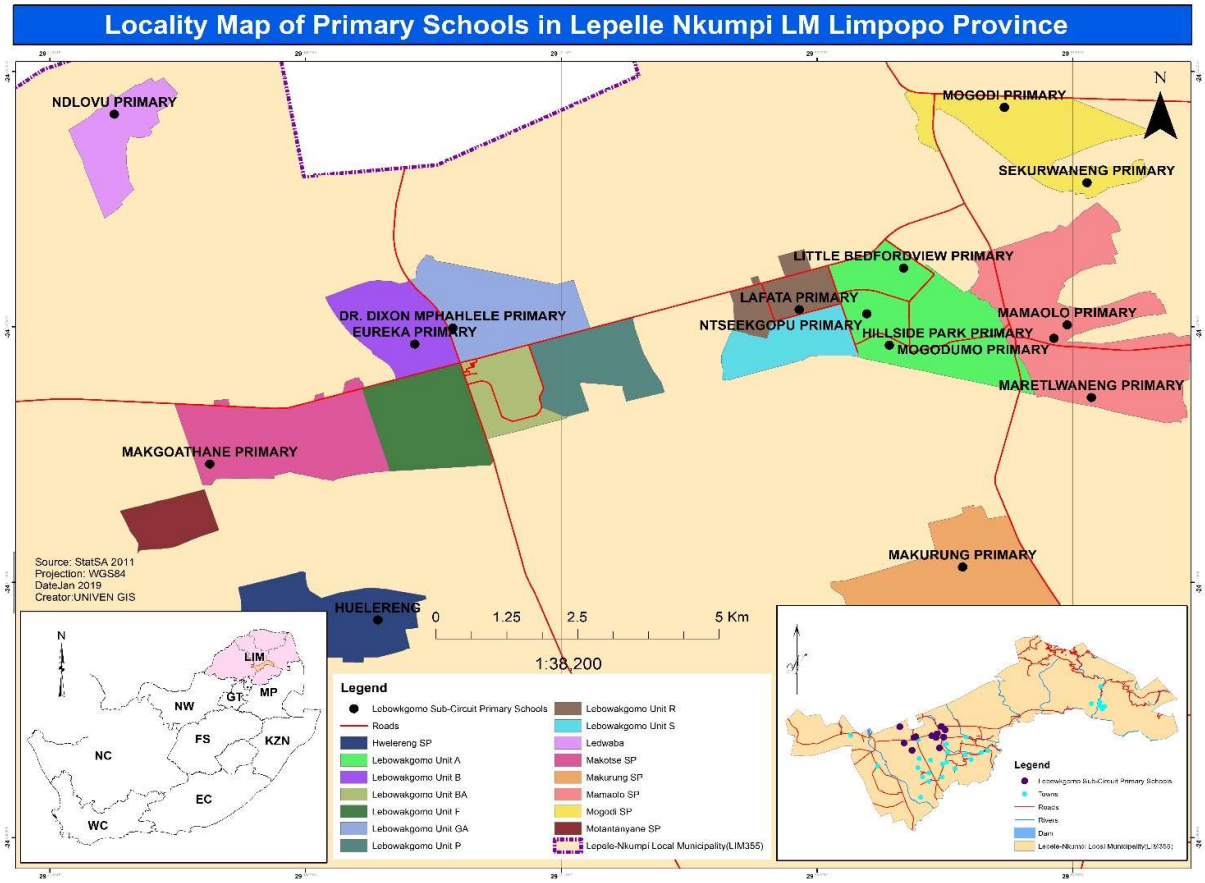


Figure 5.1. A map showing primary schools in Lepelle-Nkumpi Municipality under Lebowakgomo sub circuit (STATSSA, 2011)

Lepelle-Nkumpi Municipality is one of the local municipalities within the Capricorn District Municipality in Limpopo Province. The municipality is located 55km south of the Capricorn District Municipality and Polokwane city. The municipality is pre-dominantly rural with a population of 227 965 and covers 3,454.78km, which is 20.4% of the district's total land area. The municipality is divided into 29 wards, four of them being a township called Lebowakgomo, one of the Capricon district growth point. All sittings of the Provincial Legislature take place at Lebowakgomo Old Parliament and Lepelle-Nkumpi Municipality, Capricorn District in Limpopo Province.

Social facilities available within the municipality includes 108 primary schools, 90 secondary schools, 1 FET College, 21 primary health care clinics, 3 hospitals, 4 police stations and one magisterial court. For the purpose of this study, data was collected from children in the Lepelle-Nkumpi municipality under Capricorn District in Lebowakgomo sub-circiut. The study focused on primary schools in Lepelle Nkumpi municipality. The targeted primary schools in Lepelle Nkumpi Municipality were Ntseekgopu primary school, Mogodumo primary school, Dr Dixon Mphahlele primary school, Eureka primary school, Little Bedfordview primary school, Maretlwaneng primary school, Hwelereng primary school, Ndlovu primary school, Mamaolo primary school, Sekurwaneng primary school, Hillside primary school, Lafata primary school and Mogodi primary school (see Appendix I & J). Mahlasedi special school is one school within Lebowakgomo sub-circuit which was excluded since it was categorised as MMI (mild moderate intellectual) school for children with mild to moderate intelligence. The school offers a different curriculum from other main stream schools. Children who were accepted in the school specialise with skilled and semi-skilled work.

According to Brink, Van der Walt and Van Rensburg (2012), population is the entire group of persons or objects that is of interest to the researcher. School going children located in Lebowakgomo sub-circuit within Lepelle-Nkumpi Municipality were used for this study. Most parts of the municipality are still very rural even though there are fewer semi-urban areas. Many children who were observed as referrals by teachers for psychological intervention came from around this area indicated above. Lepelle-Nkumpi Municipality has many schools around and as such there should be many psychologists providing intervention to these individuals.

5.5 Sampling and sample size

A sample of 250 school children from Lebowakgomo sub circuit were purposively selected. Sampling refers to the process of selecting representative unites of a population for study in a research investigation (Akinsola, 2005). The schools were purposively selected and screening was done within semi urban schools in Lebowakgomo sub circuit. The researcher was interested in children from these schools because most referrals by teachers were children coming from the above mentioned sub circuit. The researcher therefore requested that teachers screen all

learners in all grades by completing a DBD scale for each learner and again parents of the same learners were also requested to complete the same scale completed by the teacher. Following that scores obtained from the teacher and the parent were compared for any discrepancies.

The researcher therefore identified those learners who were clinically diagnosed as having ADHD and then formed part of the sample that was used in the current study. The participants were further assessed on an emotional scale for further comparison. The control and experiment were conducted on the homogeneous group.

The sample consisted of two hundred and fifty children, boys and girls, from the Lepelle Nkumpi Municipality, recruited from the nine primary schools in Lebowakgomo sub-circuit, Limpopo Province, South Africa. The 250 children (94 boys and 156 girls, aged 6 – 12) were recruited following screening for ADHD of the general population of primary school children. The learners were screened for ADHD using the Disruptive Behavior Disorders Rating Scale (DBD) based on their teachers and parents ratings (Pelham, Gnagy, Greenslade, & Milich, 1992).

Participants with scores ≥39 on the Hyperactive/Impulsive (H/I) sub-scale of the DBD were classified Hyperactive/Impulsive (ADHD-HI) and those having a score ≥39 on the inattentive sub-scale (ADHD-PI) were classified as predominantly Inattentive (ADHD-PI). Participants who met the criteria on both scales were classified as the ADHD-Combined (ADHD-C) subtype. The cut-off point for the neurotypical control group (non-ADHD) was set as the 85th percentile or below in order to decrease the risk for false negatives in this group (Meyer, Eilertsen, Sundet, Tshifularo, & Sagvolden, 2004). Thus, children with scores on the H/I scale less than 35 and on the Inattention scale less than 35 were selected as controls. The control group was matched with the ADHD group for gender and age. The final sample, therefore, consisted of 250 children, 125 (47 males and 78 females) with ADHD and 125 (47 males and 78 females) as neurotypical controls (see Table 1).

5.6 Inclusion criteria

Polit and Beck (2008) indicate that inclusion criteria are the criteria which specify population characteristics. Burns and Grove (2011) describe inclusion criteria as the characteristics that must be present for an element to be included. The study only focused on learners from primary schools from Lepelle-Nkumpi municipality in Lebowakgomo sub circuit. Participants were both boys and girls ranging between the ages of 6 -13 years.

5.7 Exclusion criteria

Exclusion criteria are the population defined in terms of characteristics that people do not possess (Polit & Beck, 2008). In this research study, adolescents from the ages of 14- 19 years were excluded from the study because during this phase most of them are experimenting with substances and already engaging in risky behaviours. Children with an IQ <80 and children who were reported to have a history of neurological problems (e.g. head injuries, epilepsy, cerebral palsy, etc.) or severe psychiatric disorders (as indicated by the parents/guardians on the demographic questionnaire) were excluded from the study. None of the children who participated in the study was on psychostimulant medication at the time of testing. Also children who were on prescribed psychotropic medication for ADHD or related disorders within 6 months of the time of evaluation were excluded from participating in the study.

Children whose parents or guardians did not return consent forms were excluded from participating in the study. Only one primary school in the sub district was eliminated from the study because the school is for learners with a presentation of mild to moderate intellectual functioning and the curriculum of the school caters skills and semi-skilled work, which is entirely different from other schools.

5.8 Entry negotiation

The researcher initially obtained a letter of permission from the Provincial Department of Education to conduct a research in primary schools in Lebowakgomo sub-circuit within Lepelle-Nkumpi Municipality (See Apendix I). Following that, the Provincial Department of Education further instructed the researcher to obtain another letter of

permission from the circuit manager within Lebowakgomo sub-circuit which was issued out to school principals allowing teachers to assist the researcher to conduct her study. After obtaining permission from the Department of Education, a letter of request for permission was sent to the school principals so that learners will be made available through the assistance of teachers since data collection was done during school lessons. Also a letter of consent to parents was sent especially that learners were under age and cannot consent for themselves. Parents were consulted since they formed part of the study.

5.9 Measuring instruments

Measuring instruments refer to assessment tools or method that the researcher used in order to obtain information (Shea, Grimshaw, Wells, Boers, Andersson, Hamel, Poter, Tugwell, Moher, & Bouter, 2007). The researcher used Teacher/Parent Disruptive Behavioural Disorders Rating Scale (DBD), Beck Youth Inventories Second Edition for Children and adolescents (BYI-II). The DBD scale was the most preferred assessment tool because thus far it is the only standardised tool for Sepedi speaking people and it user friendly. While the Beck inventories scale is a simple scale written in a simple English langue which children from lower grade can easily understand. The use of both tools was relevant in assisting the researcher to obtain the purpose of the study as well as achieving the objectives and hypotheses of the study. It is for these reasons that the researcher decided to apply these screening tools towards achieving her goal. A full description of the DBD and Beck scale is discussed underneath.

5.9.1 Disruptive Behaviour Disorders Rating Scale

Description of the DBD Scale

The screening device that was used to assess participants was the Disruptive Behaviour Disorders rating scale (DBD) (Pelham, Jr, Gnaggy, Greenslade, & Milich, 1992) that was used for screening children who formed the two groups' i.e. ADHD group and non-ADHD group. The DBD assesses the presence and degree of ADHD-related symptoms (inattention and hyperactive/impulsive); Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) as formulated in the DSM IV TR.

There are 18 items on the scale that measure ADHD-related symptoms. Teachers and parents were asked to rate the behaviours of the children on a four-point scale comprising of the following options: not at all (0); just a little (1); pretty much (2); and very much (3). The total score was added up and compared to the cut-off point of the 95th percentile, which has previously been identified as clinically significant (Barkley, 1997; Barkley & Murphy, 2006). The children meeting the criteria for inclusion in the ADHD group were selected for further testing. They were matched for gender and age with children who did not meet the inclusion criteria (85th percentile and below), obtained from the screening process. They formed the neurotypical control group.

The cut-off points were established based on the results from the prevalence study in which more than 6000 children in Limpopo province were assessed on the DBD (Meyer et al., 2004). In screening for ADHD, teachers and parents rated children for ADHD symptoms on the Disruptive Behaviour Disorders (DBD) rating scale (Pelham, Gnagy, Greenslade & Milich, 1992), which was standardised and normed for all the language groups and population groups in Limpopo Province, South Africa (Meyeret al., 2004).

5.9.2 The Beck Youth Inventories.

Descriptive of the inventory

The Beck Youth Inventories Scale for children and adolescents Second Edition (BYI-II) has 5 sub inventories. It is a simple multiple choice self-report inventory and each of the 5 inventories contains 20 statements about thoughts, feelings or behaviours associated with emotional and social impairment in children and adolescents. The scales were written at a second grade reading level. The Beck scale is suitable for children from the age of 6 to 12 years and participants took 5 to 10 minutes to complete each inventory (Beck, Beck, Jolly & Steer, 2005).

The Beck Youth Inventories (BYI) is a well-accepted self-report scale that measures stress that children experience in association with mental health problems (Beck et al., 2005). The BYI contains 5 scales that may be used separately or in combination to assess a child's experience of anxiety, depression, anger, disruptive behavior and self-concept. On screening internalising disorders, all the children who clinically met the criteria for ADHD and matched controls completed the Beck Youth Inventories- Second

Edition (BYI-II) (Beck et al., 2005). Children were assessed on all BYI- 5 sub scales with the assistance of trained Sepedi peaking research assistants. Children are therefore expected to describe how often the statement has been true for them during the past two weeks, including today.

5.10 Pre-testing

Banks and Olszewski (1997) stipulated that pretesting is used in identifying and correcting flaws in the instrument under development. Designing the perfect instrument can be an overwhelming task, however, researchers can create effective instruments. In order to determine the effectiveness of an instrument, it is essential to pre-test it before it is used (Goodwin and Finkelstein, 2005). This procedure helps researchers to determine the strengths and weaknesses of the instrument concerning question format, wording and order. Pretesting further enables the researcher to clarify the research study goals and objectives which guide the whole research initiative. Early identification and addressing of research problems will give the researcher enough time to improve the research instrument, ensuring quality data (Vadum & Rankins, 1998).

In pre-testing the scale, the researcher initially gave DBD scales to a group of teachers and parents who later did not form part of screening for the purpose of the study. This process was done on two occasions to confirm if the scale can be easily administered. During the actual screening of learners, this group of teachers and parents was excluded from screening process together with the learners who were screened by the same group. The main aim of this exercise was to measure if the tool is supposed to measure what it is supposed to for the purpose of the study. The researcher further evaluated the consistency of responses given by both teachers and parents. Pre-testing helped the research to give more clarity to some items which appeared to be more confusing to parents when responding to the questions on the scale i.e. DK (Don't Know) was changed to "write a question mark" to items for which you don't know (Appendix A). On the original DBD scale, item 10, 14 and 21were removed from the DSM-III-R nd are not included in the scoring for a DSM-IV diagnosis (see Appendix B). Therefore, the distributed scale entailed of 42 items instead of 45.

5.11 Reliability of the instrument

The consistency with which an instrument measures the attribute it is designed to measure is termed reliability, Golafshani (2003). Moreover, an instrument is only reliable if it measures accurately the true scores of the attribute under investigation. In this study, the reliability of the instrument was determined by the test-retest method. This determines the consistency when individual responses are compared on two separate occasions (Golafshani, 2003). This is meant to measure the correlation coefficient and the closer it is to one, the more excellent the result and modification will be done. The instrument in the present study is reliable to test what it is supposed to test.

At the initial phase, the scales were given to a sampled pre-testing participants (teachers, parents and children) to test the reliabity of the scales. The pre-testing was done three times to the same group to check if the instrument is userfriendly and, if participants understand the content of the scale. Through reliability testing, it came out that the participants were able to yield similar responses and as such no alteration or changes made to the instruments that were used to collect data. On the DBD scale, item number 17 (often blurts out answers before questions have been completed) both teachers and parents contributed similar information especially if the child does show symptoms of hyperactivity. Again on the BYI-II scale, children from different grades showed understanding and consistency in their responses.

The same results were also depicted with the Cronbach's alpha coefficients which depicted same internal consistency reliability in all five inventories ranging from .86 to .92 for ages between 7-14 years (Beck, yBeck, Jolly & Steer, 2005). The process of reliability also assited if there was no need for any questions to be refined. Moreover, the scales were administered in an interval of 3 weeks appart to assess if there was consistency. Therefore, the scales were eventually rolled out teachers, parents and children for screening purpose.

5.12 Validity of the instrument

The validity of the instrument refers to the extent to which it measures what it is supposed to measure (Golafshani, 2003). In this study, validity of the instruments was measured through reliability testing where the desired similar responses were yielded. The researcher in her study used construct validity to check if the scales (DBD and BYI-II) are measuring the concepts that are being discussed. Therefore, the instruments were validated to assess if they were designed to measure what they are supposed to measure. Since the instruments used were not newly constructed, their reliability and validity were already calculated. By comparing the content of the scales and the objectives and hypothesis of the study, the researcher found that there was great significant relationship between the two. On the BYI-II scale, it came out that children yielded similar expression of feelings which was consistent through the pilot study.

5.13 Data collection

Data collection is a term used to describe a process of preparing and collecting data (Creswell, 2016). For the purpose of the current study, the researcher collected data from a larger number of population in which she made use of the DBD rating scale as well as BYI-II to examine different variables, which were analyzed through the use of statistical programme which is a computer soft-ware programme used to analyze data. The researcher had a privilege to observe carefully while comparing how each variable influenced the other variable. Having a large number of population was effective and could not be doable through qualitative since it is based on the use of non-quantifiable data such as words, feelings, emotions (Bryman, 2016) since the data collected was quantified. The researcher was more interested in the quantitative study for broader or wider view which ultimately enabled the researcher to get results from many respondents. In addition, the research approach helped the researcher to obtain authentic and reliable information from both teachers and parents about children within Lepelle-Nkumpi Municipality. The use of scales was apprproaite as the right tools for assessing behaviour and internalising disorders in a larger population. The researcher preferred the quantitative data collection method because methods are based on purposive sampling and structured data collection instruments. As such findings are

easy to present, summarize, compare and generalize (Gonser, 2016).

5.14 Data collection procedure

Teachers were issued with copies of the DBD scales for completion for every learner in a particular grade. Parents were given letters outlining the purpose of the study and consent forms requesting their permission to allow their children to participate in the study. They were given a DBD scale to complete. Parents who were not conversant in English, were given a DBD scale which was translated into Northern Sotho (see Appendix B).

Following the completion of the questionnaire by both teachers and parents/guardians, only learners who were identified as having ADHD and their matched controls were further issued out with a BYI-II scale for completion. All children who took part in the research study were given a clear explanation of the reasons for their participation in the study. Participation was voluntary and assessments were completed during school hours. Participants took 20 minutes each to complete the Beck scale, while parents and teachers took 10 minutes to complete the DBD scale for each child. The researcher also observed that her research study did not interfere with school lessons.

5.15 Data analysis

Data analysis is the process that is conducted to reduce, organize and give meaning to the collected data (Burns, Grove & Gray 2011). They further explained that data analysis is the selection of appropriate statistical techniques to analyze the study data. The analysis techniques to be used were determined by the research objectives, research questions, hypothesis, research design and the level of measurement to be achieved by the instrument (Burns, Grove & Gray 2011). Inferential statistics provided the researcher with a chance to apply the information acquired from the sample to establish conclusions about the population and therefore draw up a conclusion about that population. Data analysis by means of statistical techniques helps researchers to investigate variables as well as their effect, relationship and patterns of involvement within our world (Welman, Kruger & Mitchell, 2006).

For the purpose of the study, the ADHD scores on DBD rating scales completed by both teachers and parents on a particular learner were correlated with scores obtained on the BYI-II, which is a test for assessing emotional disorders. Analysis of Variance (ANOVA) was used to investigate possible between-group differences in raw scores on the BYI-II, using the Statistica program, version 10 (Statsoft, 2011). The results were analysed with a 4 x 2 x 2 (ADHD–presentation x gender x age group) ANOVA for independent samples. *Post-hoc* analysis (Bonferroni correction was used to establish the difference within-groups).

5.16 Ethical considerations

For the purpose of this study, the researcher observed the following ethical considerations:

Permission to conduct the study

5.16.1. Institutional ethics

Before conducting the study, the researcher first presented the proposal in the Department of Psychology and again before the School of Health Science's Higher Degrees Committee for quality assurance. The proposal was then submitted to University Higher Degree Committee (UHDC) for approval. Ethical clearance to conduct the study was obtained from the University of Venda Ethics Committee (see Appendix L). A letter of permission from the Limpopo Provincial Department of Education permitting the researcher to conduct the research study was issued out with terms and conditions that at the end of the study, the researcher will provide the department with the findings of the research study (see Appendix H). A letter from the circuit manager in Lebowakgomo sub circuit requesting school principals to allow the researcher to conduct her study and further asked teachers to assist and avail learners for data collection was obtained (see Appendix I & J).

5.16.2 External ethics

(a) Informed consent

Blanche, Blanche, Durrheim and Painter (2006) define informed consent as the process of seeking explicit agreement from subjects to participate in a research project, based on their full understanding of the procedure involved and the likely effects. Before conducting this study, the researcher first informed parents of the learners about the purpose of the study before they can consent for their children since they are minors about what the purpose of the study, what she wanted to achieve, what she wanted to achieve with the study and who will benefit from the study. A letter of consent was then send out to parents and children whose parents did not give consent did not form part of the study (see Appendix D). Moreover, children who undertook the second scale were informed about the purpose of the study and what was expected from them. This assisted the researcher to get more relevant information because the respondents were free to participate in the study.

(b) Non-harm to participants

It was essential that the researcher ensures that participants were not exposed to any undue physical or psychological harm (Leedy & Ormrod, 2001). The researcher employed principles of honesty, respect and sympathy to all those who participated in the study. For those children who were diagnosed with having ADHD, the researcher ensured that they were referred to the hospital for further intervention in a form of Behaviour Modification and Psychotherapy, and where possible they were referred to the hospital for psychiatric evaluation and treatment.

(c) Confidentiality

It is always important that the researcher safeguard data collected in a secured condition or environment. Bless, Higson-Smith and Kagee (2006) define confidentiality as the protection of information as information provided by participants, particularly sensitive and personal information should be protected and made not available to anyone other than the researcher. The researcher applied the confidential ethics in the study because the obtained is personal and it will not be disclosed to other people. The researcher

reassured the respondents that any information they have provided on the scale will not be shared with other respondents. After data was collected, the researcher collected the scales and put them in a safe place.

(d) Anonymity

The data provided by the participants must not be connected immediately and obviously with the name or any other identifier. Generally, researchers assign a number to participant's data to ensure that the data remains anonymous (Bless, Higson-Smith and Kagee, 2006). The researcher requested teachers when completing scales to give each learner a code that they can be identified with in order to protect their identity. The researcher applied the ethic of anonymity by not asking the teachers, parents and respondents to state or write their names and address on the scales. It was agreed during the first meeting with parents and teachers that on the space for name of participants a seriel code i.e. P1 will be used as a psydo name. All scales had the same code for comparison, scoring and analysis purpose. Before participants started completing the scales the researcher also informed them that they will remain anonymous and their information that their information will not be distributed to others. Therefore, the names and details of the respondents remained anonymous throughout the study.

5.17 Conclusion

The methodology used in the current study was relevant and reliable for the purpose of the current study. The study applied a quantitative approach in which a quasi-experimental research design was employed which lead to the arrival of the actual population sample. The area where the sample was drawn from was clearly specified by an indication of a map and moreover, the study focused only on school going children from primary schoos. Two scientific and standardised assessment tools which are the DBD and BYI-II were used for screening disruptive behaviours and emotional challenges of participants. Permission to visit schools from different stakeholders i.e. Limpopo Department of Education and Lebowakgomo Circuit was obtained and letters of permission are attached as appendices in the current document. The protocol on how the researcher will observe ethical issues prior to the study were clearly presented in this chapter. Data analysis of information obtained from children in various schools was

analysed through Statisctical soft-wear in which analysis of varience (ANOVA) was employed.

CHAPTER 6

PRESENTATION OF RESULTS

6.1. Introduction

The aim of this research was to explore the comorbidity of internalising disorders i.e. anxiety and depression on Attention Deficit Hyperactivity Disorder symptoms (inattention, hyperactivity/impulsiveness). The following hypotheses and null hypotheses were developed:

Hypotheses

- Children with ADHD have more symptoms of anxiety than children without ADHD.
- Children with ADHD have more symptoms of depression than children without ADHD
- Children with ADHD have lower self-esteem than children without ADHD.
- Internalising comorbidities (anxiety and depression) are more prevalent in girl than in boys who have more Externalising comorbidities (Oppositional Defient Disorder (ODD), Conduct Disorder (CD), anger issues and disruptive behaviours.

Null hypotheses:

Research hypothesis 1:

Children with ADHD have more internalising disorders than children without ADHD

Null hypothesis 1(a)

Children with ADHD have no more anxiety disorders than children without ADHD

Alternative hypothesis 1(a)

Children with ADHD have more anxiety disorders than children without ADHD.

Null hypothesis 1(b)

Children with ADHD have no more depression symptoms than children without ADHD

Alternative hypothesis 1(b)

Children with ADHD have more depression symptoms than children without ADHD.

Research hypothesis 2

Children with ADHD, because of their comorbid internalising disorders, have a lower self-esteem than children without ADHD.

Null hypothesis 2

Children with ADHD have no lower self-esteem than children without ADHD.

Alternative hypothesis 2

Children with ADHD have a lower self-esteem than children without ADHD.

Research hypothesis 3

Internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who exhibit more externalising comorbidities (ODD), (CD), anger issues, and disruptive behaviours

Null hypothesis 3(a)

Girls do not have more anxiety symptoms than boys.

Alternative hypothesis 3(a)

Girls have more anxiety symptoms than boys.

Null hypothesis 3(b)

Girls do not have more depression symptoms than boys.

Alternative hypothesis 3(b)

Girls have more depression symptoms than boys.

Null hypothesis 3(c)

Boys have no more symptoms of ODD than girls.

Alternative hypothesis 3(c)

Boys have more symptoms of ODD than girls.

Null hypothesis 3(d)

Boys have no more symptoms of CD than girls.

Alternative hypothesis 3(d)

Boys have more symptoms of CD than girls.

Null hypothesis 3(e)

Boys express no more anger than girls.

Alternative hypothesis 3(e)

Boys express more anger than girls.

Null hypothesis 3(f)

Boys show no more disruptive behaviours than girls.

Alternative hypothesis 3(f)

Boys show more disruptive behaviours than girls.

This chapter will give a report of the results obtained when the collected data was analysed to accept or reject the proposed hypotheses.

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

(i) Demographic and ADHD characteristics of the sample.

(ii) Gender distribution of the sample

(iii) Descriptive statistics in table form.

(iv) Inferential statistics: ANOVA, to establish between-group differences (ADHD v. Control) and *post-hoc* analysis (Bonferroni correction) establish the within-group differences (subtype differences, males v. females).

(v) Graphical representation of the results.

6.2 Demographics

6.2.1 Demographic and ADHD characteristics

Table 6.1: Demographic and ADHD characteristics of the sample

	ADHD-HI n = 18 7.2%		ADHD-PI n = 44 16.6%		ADHD-C n = 63 25.2%		Control n = 125 50%		Test	*p*
	N (%)	M (SD)	N (%)	*M* (SD)	n (%)	*M* (SD)	n (%)	*M* (SD)		
Gender									x^2 = 12.21	0.07
Male	13 (5.2%)		11 (4.4%)		23 (9.2%)		47 (18.8%)			
Female	5 (2.0%)		33 (13.2%)		40 (16.0%)		78 (31.2%)			
Age (years)		9.44 (1.03)		10.02 (1.20)		9.39 (1.42)		9.72 (1.18)	*F* = 0.12	0.73
DBD Scores										
HI		40.66 (1.11)		27. 69 (7.33)		40.05 (1.34)		21.90 (1.18)	*F* = 144.80	< 0.001*
Inattention		28.69 (5.98)		41.41 (1.78)		39.98 (1.61)		19.91 (5.47)	*F* = 379.65	< 0.001*

Table 6.1 illustrates the demographic and ADHD characteristics of the sample.

Figure 6.1 illustrates the geder distribution of the sample.

Figure 6.1 Gender distribution of the sample (n= 250)

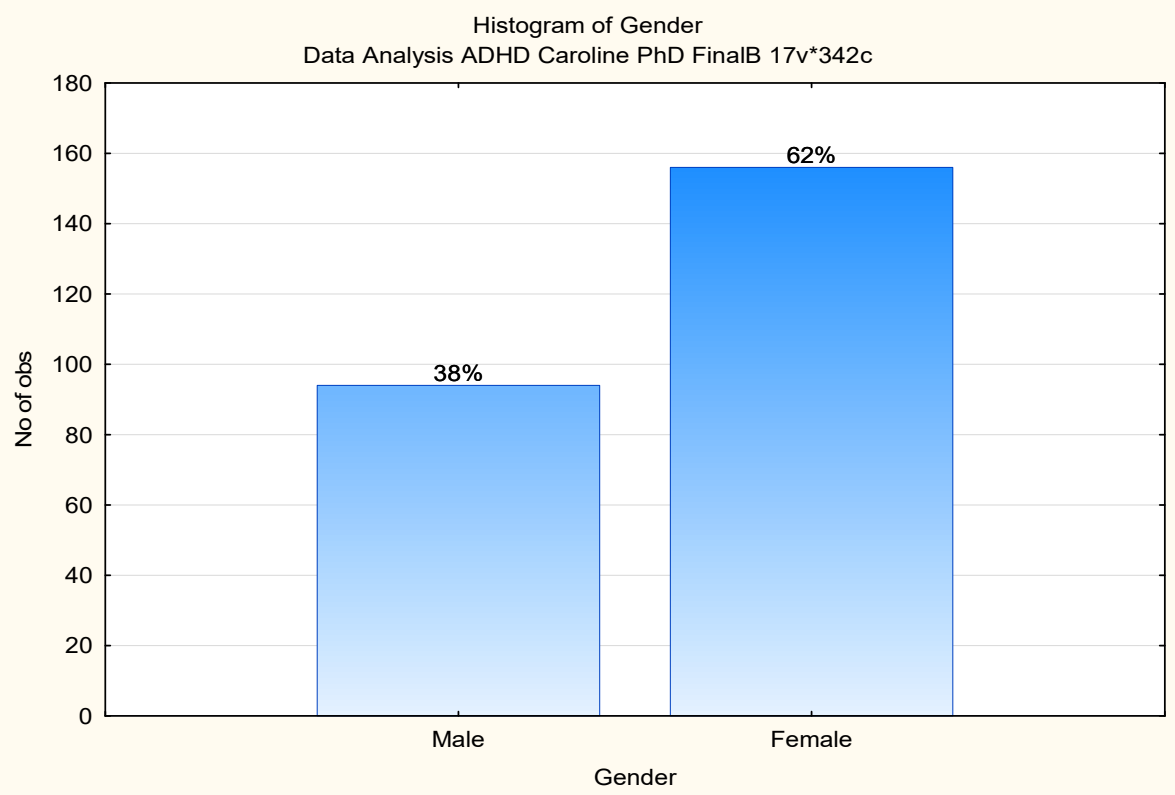


Figure 6.1 depicts that more females (62%) participated in the study than males (38%).

Figure 6.2 illustrates the age distribution of the sample.

Figure 6.2 Age distribution of the sample (n= 250)

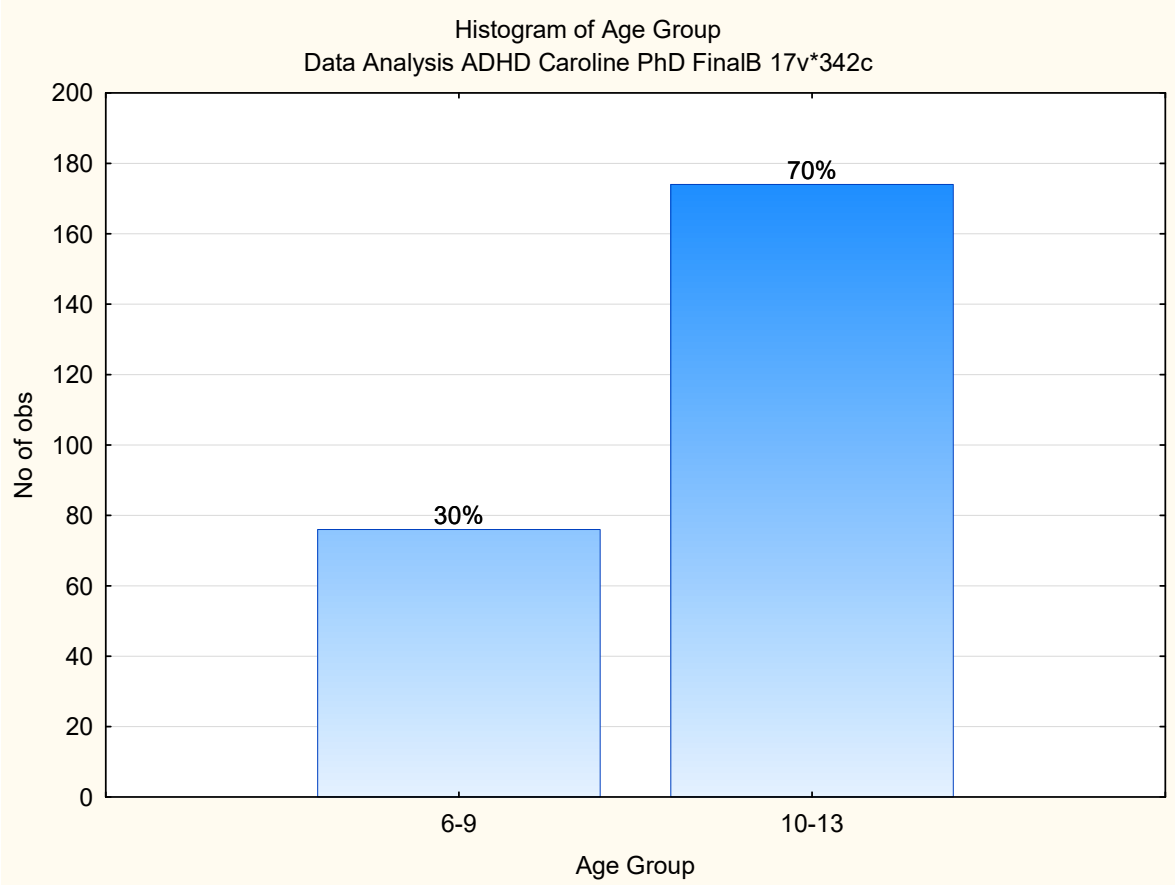


Figure 6.2 depicts that the age group between 10-13 years (70%) had more ADHD symptoms while age group 6-9 years (30%) also had symptoms of ADHD.

6.3 Anxiety

Table 6.2 illustrates the descriptive statistics for the results of anxiety scores of the ADHD subtypes.

Table 6.2 Descriptive Statistics for Anxiety scores

Subtype	Gender	N	Mean ± SD
ADHD-HI	*Male*	13	25.15 ± 5.40
	Female	5	21.80 ± 5.45
ADHD-PI	*Male*	11	30.09 ± 5.97
	Female	33	33.58 ± 4.61
ADHD-C	*Male*	23	29.87 ± 6.31
	Female	40	33.80 ± 5.74
Control	*Male*	47	19.98 ± 8.39
	Female	78	19.53 ± 6.02

Analysis of variance (ANOVA)

Table 6.2 depicts the results of the ANOVA on Anxiety and subtypes.

***Table 6.3* Analysis of Variance for Anxiety scores**

	DF	*F*	*P*	η_p^2
ADHD	3, 242	62.68	0.00*	0.44
Gender	1, 242	0.65	0.42	0.00
ADHD x Gender	3, 242	2.55	0.06	0.03

*$p < 0.001$

There was a statistically significant difference between the anxiety scores of the ADHD group and the neurotypical controls: F (3, 242) = 62.68, $p < 0.001$, $\eta_p^2 = 0.44$.

There was no effect of gender, neither main, nor interacting. Therefore, the gender groups were not analysed separately.

6.3.1 Post-hoc analysis (Bonferroni correction):

Post-hoc analysis revealed that there was a significant difference between the ADHD-

HI subtype when compared to control group (p = 0.03). The other groups (ADHD-PI and ADHD-C) also showed statistically significant difference (p < 0.001) when compared to control group. Another significant difference observed was among the ADHD-HI versus the ADHD-PI and ADHD-C subtypes (p < 0.001). Both the ADHD-PI and ADHD-Comb subtype had more anxiety symptoms than the ADHD-HI subtype.

Figure 6.3 Graphical representation of the results obtained on the Beck Anxiety Subscale.

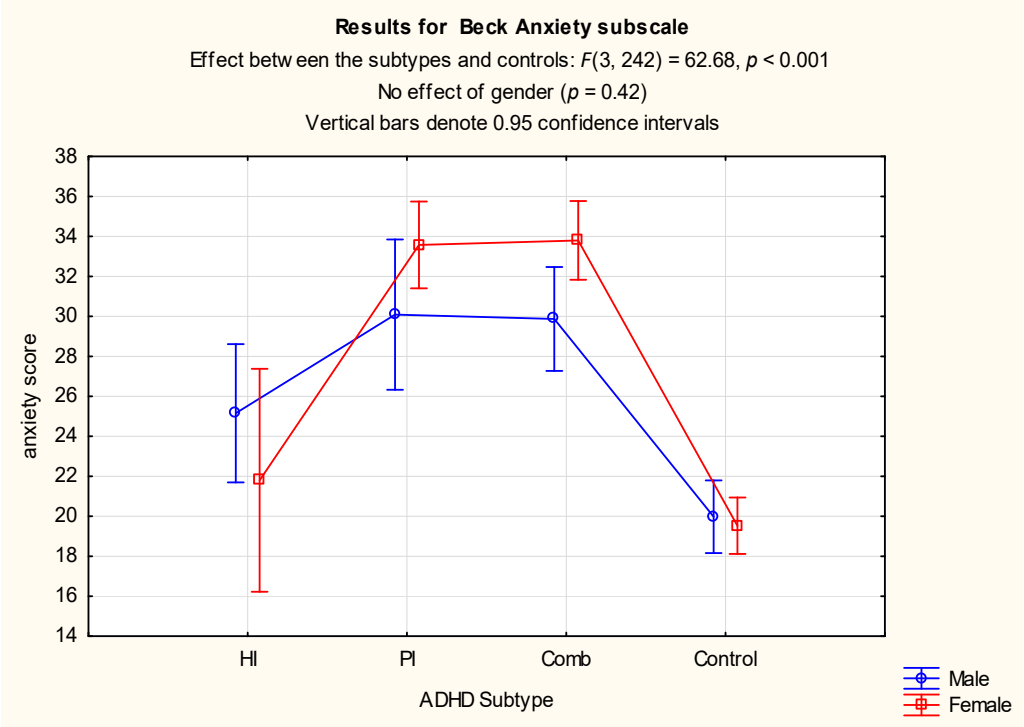


Figure 6.3 indicates that there was a significant difference between ADHD subtypes on anxiety scores. Al three ADHD subtypes had a significantly higher score on the scale than the Controls. The ADHD-PI and ADHD-C subtypes also showed a higher level of anxiety when compared to ADHD-HI subtype.

6.4 Depression

Table 6.4 gives the descriptive statistics for the results of depression scores on the Beck scale for the ADHD subtypes.

Table 6.4 Descriptive statistics for the depression scores

Subtype	Gender	N	Mean ± SD
ADHD-HI	*Male*	13	23.23 ± 4.32
	Female	5	21.20 ± 8.04
ADHD-PI	*Male*	11	29.73 ± 4.96
	Female	33	34.70 ± 4.81
ADHD-C	*Male*	23	28.43 ± 5.57
	Female	40	34.65 ± 5.88
Control	*Male*	47	17.11 ± 7.69
	Female	78	20.00 ± 5.58

Analysis of variance (ANOVA)

Table 6.5 depicts the results of the ANOVA on Depression and subtypes.

Table 6.5 Analysis of Variance for depression scores

	DF	F	*P*	η_p^2
ADHD	3, 242	84.86	0.000**	0.51
Gender	1, 242	8.14	0.004*	0.03
ADHD x Gender	3, 242	2.28	0.08	0.02

*$p < 0.05$, **$p < 0.001$

There was a statistically significant difference between the depression scores of the ADHD subtypes and the neurotypical control group: F (3, 242) = 84.86, $p < 0.001$, $\eta_p^2 = 0.51$.

There was a main effect of gender, F (1, 242) = 8.14, $p < 0.05$, $\eta_p^2 = 0.03$ but no interacting effect, therefore the gender groups were analysed separately.

6.4.1 Post-hoc analysis (Bonferroni correction)

Post-hoc analysis revealed that there was a significant difference between the Controls and all three the subtypes, with the ADHD group having significantly more symptoms of depression. In the ADHD-HI group, only the males differed significantly from the control group (p = 0.03), while there were no significant differences between the females of the ADHD-HI and the females of the control group. For the ADHD-PI and ADHD-C subtype, there were no gender differences and both subtypes differed significantly from the control group (p< 0.001).

Figure 6.4 Graphical representation of the results of the Beck Depression Subscale

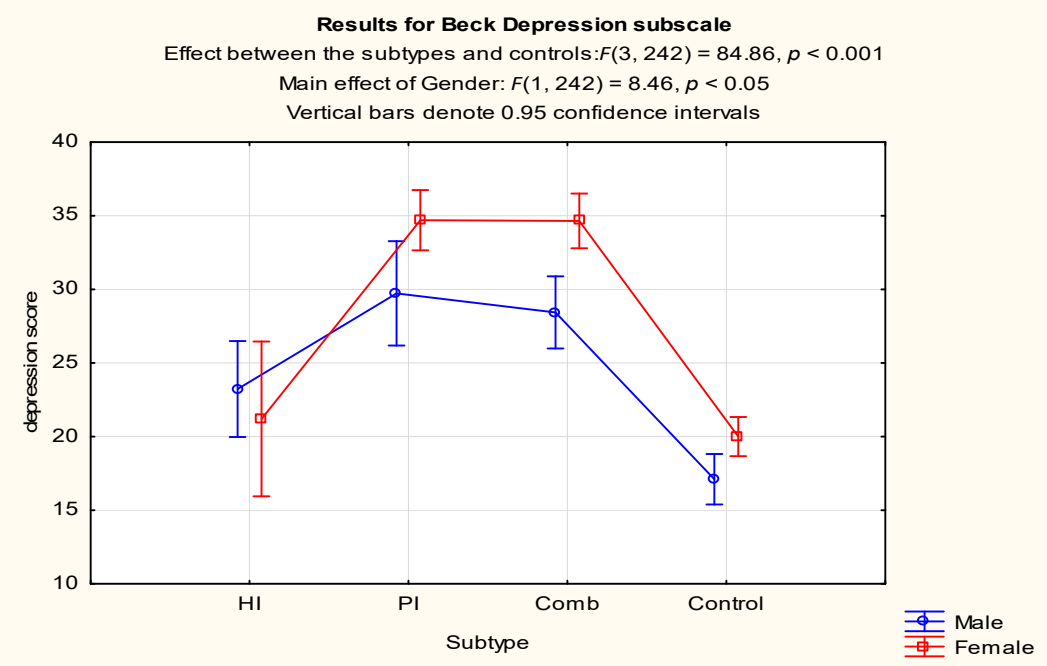


Figure 6.4 shows that there was a difference in scores on the depression scale between the ADHD subtypes and the neurotypical control group with the ADHD group having significantly more symptoms of depression. The exception was the ADHD-HI subtype, where the difference was only significant for the males. No significant difference in depression symptoms was found for the female ADHD-HI group when compared with the controls.

6.5 Self-esteem

Table 6.6 illustrates the descriptive statistics for the results of self-esteem scores of the ADHD subtype.

Table 6.6 **Descriptive statistics for self-esteem scores**

Subtypes	Gender	N	Mean ± SD
ADHD-HI	*Male*	13	26.15 ± 9.15
	Female	5	24.20 ± 0.45
ADHD-PI	*Male*	11	29.55 ± 8.95
	Female	33	24.18 ± 0.77
ADHD-C	*Male*	23	24.96 ± 6.18
	Female	40	24.25 ± 0.49
Control	*Male*	47	33.15 ± 9.56
	Female	78	28.14 ± 6.06

Analysis of variance (ANOVA)

Table 6.7 depicts the results of the ANOVA on self-esteem, subtypes and gender.

Table 6.7 **Analysis of Variance for self-esteem scores**

	DF	*F*	*P*	η_p^2
ADHD	3, 242	13.70	0.000**	0.15
Gender	1, 242	8.46	0.004*	0.03
ADHD x Gender	3, 242	1.78	0.15	0.02

*p <0.05, **p < 0.001

There was a statistically significant difference between the self-esteem scores of the ADHD subtypes and the neurotypical control group: F (3, 242) = 13.70, p<0.001, η_p^2 = 0.15.

There was a main effect of gender, F (1, 242) = 8.46, p < 0.05, η_p^2 = 0.03 but no interacting effect, therefore the gender groups were analysed separately.

6.5.1 Post-hoc (Bonferroni correction):

Post-hoc analysis revealed that there were differences on the self-esteem scale when the ADHD-C group was compared with the Controls. Both males ($p < 0.001$) and females ($p = 0.04$) had significantly more symptoms of low self-esteem. Only the males of the ADHD-HI subtype differed significantly from the Control males ($p = 0.02$), with more symptoms of low self-esteem. There were no significant differences between the female ADHD-HI group and the Control females. The ADHD-PI group did not differ significantly from the controls on the self-esteem scale; this was true for both males and females.

Figure 6.5 Graphical representation of the results on the Beck Self Esteem Subscale

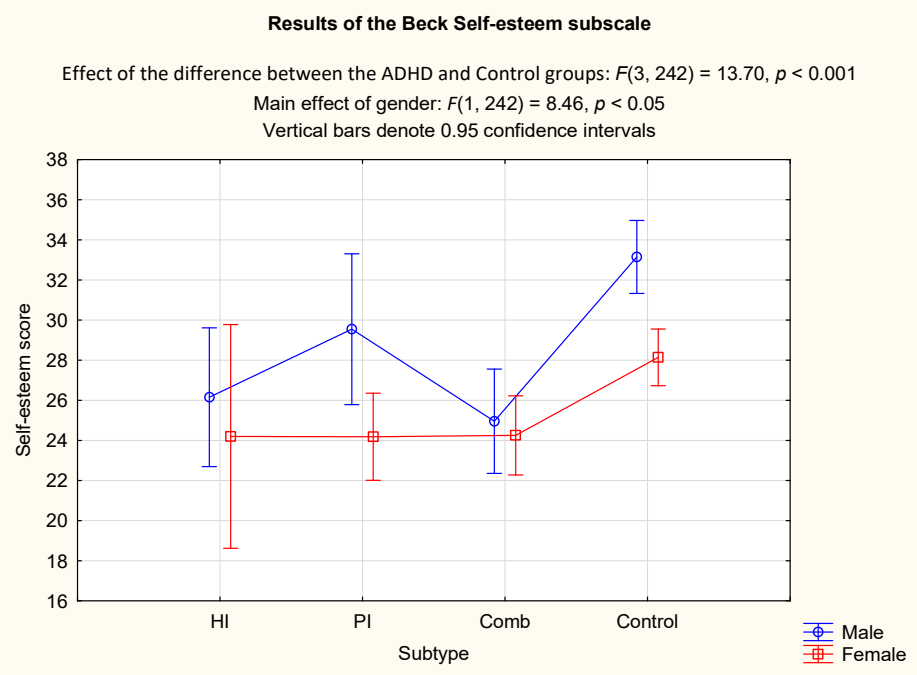


Figure 6.5 points out that there is a significant difference ($p < 0.001$) between the ADHD-C subtypes and controls. No differences in self-esteem were found for the ADHD-PI group. Only the males for the ADHD-HI subtype had lower self-esteem than the control males. There was no significant difference for the females.

6.6 Oppositional Defiant Disorder (ODD)

Table 6.8 illustrates the descriptive statistics for the results of ODD scores of the ADHD subtypes.

Table 6.8 **Descriptive statistics for ODD scores**

Subtype	Gender	N	Mean ± SD.
ADHD-HI	*Male*	13	31.77 ± 4.27
	Female	5	28.60 ± 6.23
ADHD-PI	*Male*	11	30.36 ± 8.42
	Female	33	26.36 ± 6.14
ADHD-C	*Male*	23	33.52 ± 3.44
	Female	40	26.05 ± 5.43
Control	*Male*	47	14.81 ± 6.71
	Female	78	14.10 ± 6.88

Analysis of Variance (ANOVA)

Table 6.9 depicts the results of the ANOVA for ODD as a function of subtypes and gender.

Table 6.9 **Analysis of Variance for ODD symptoms**

	DF	F	P	η_p^2
ADHD	3, 242	100.90	0.000**	0.56
Gender	1, 242	10.78	0.001*	0.04
ADHD x Gender	3, 242	5.02	0.002*	0.06

*p <0.05, **p < 0.001

There was a statistically significant difference between the ODD scores of the ADHD subtypes and the neurotypical control group: F (3, 242) = 100.9, p < 0.001, η_p^2 = 0.56.

There was a main effect of gender, F (1, 242) = 10.78, p < 0.001 η_p^2 = 0.04 and an interacting effect, F (3, 242) = 5.02, p = 0,002, η_p^2 = 0.06, therefore the gender groups were analysed separately.

6.6.1 Post-hoc analysis (Bonferroni correction)

Post-hoc analysis revealed that all three the subtypes differed significantly from the neurotypical control group at the $p < 0.001$ level. This was true for both males and females. All ADHD subtypes of both genders had substantially higher scores on the ODD scale than the controls. The observed gender differences were between the scores of the males and females. For all three subtypes, the males scored significantly higher than the females on the ODD scale. This, however, was not the case for the controls, where males and females did not differ significantly.

Figure 6.6 Graphical representation of the results of DBD- ODD subscale

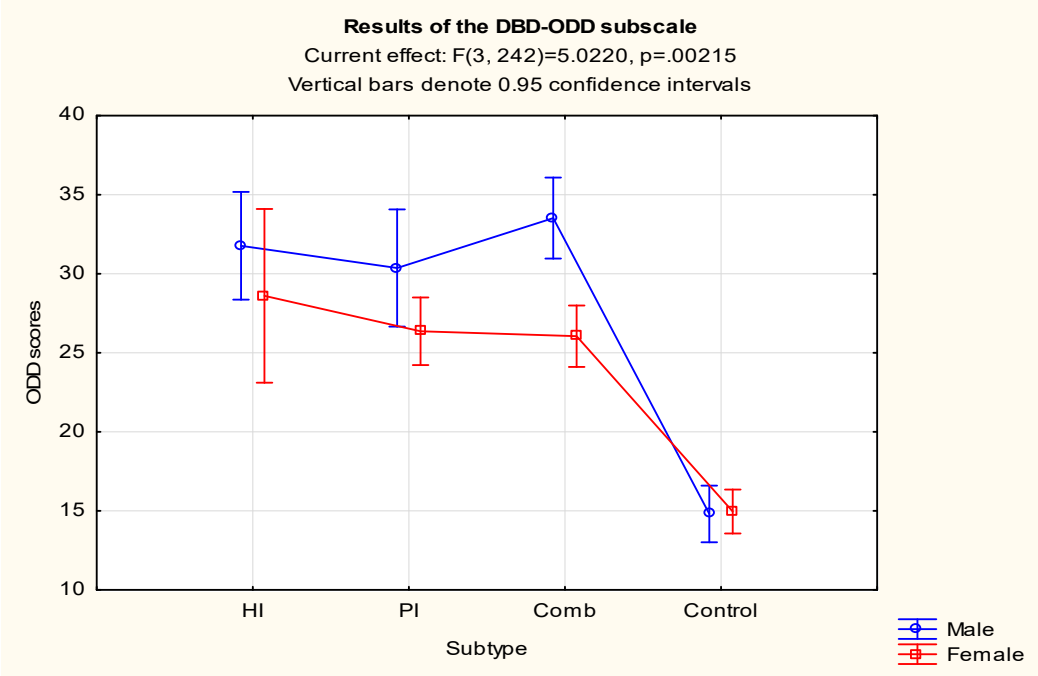


Figure 6.6 denotes a difference in scores on the ODD scale between the ADHD subtypes and the controls, with all three subtypes scoring significantly higher than the controls. There is a significant difference in the scores of the males and females with the males for all three subtypes having higher scores than the females. This was not true for the control group.

6.7 Conduct Disorder (CD)

Table 6.10 illustrates the descriptive statistics for the results of CD scores of the ADHD subtypes.

Table 6.10 **Descriptive statistics for CD scores**

Subtype	Gender	N	Mean ± SD
ADHD-HI	*Male*	13	33.46 ± 7.29
	Female	5	27.60 ± 7.02
ADHD-PI	*Male*	11	24.00 ± 0.25
	Female	33	22.36 ± 8.78
ADHD-C	*Male*	23	37.43 ± 5.13
	Female	40	26.5 5 ± 6.80
Control	*Male*	47	12.70 ± 8.19
	Female	78	13.19 ± 8.52

Analysis of Variance (ANOVA)

Table 6.11 depicts the results of the ANOVA

Table 6.11 Analysis of Variance for CD scores

	DF	*F*	*p*	η_p^2
ADHD	3, 242	84.30	0.000**	0.51
Gender	1, 242	10.05	0.002*	0.04
ADHD x Gender	3, 242	6.85	0.000**	0.08

*$p < 0.05$, **$p < 0.001$

There was a statistically significant difference between the CD scores of the ADHD subtypes and the neurotypical control group: F (3, 242) = 184.30, $p < 0.001$, $\eta_p^2 = 0.51$.

There was a main effect of gender, F (1, 242) = 10.05, $p < 0.002$ $\eta_p^2 = 0.04$ and an interacting effect, F (3, 242) = 6.85, p = < 0.001, η_p^2 = 0.08, therefore, the gender groups were analysed separately.

6.7.1 Post-hoc analysis (Bonferroni correction):

The results of the *post-hoc* analysis showed that all three subtypes scored significantly higher than the controls on the CD scale. The *p*-value for the ADHD-HI females was 0.003 when compared with the control females, while for the other groups, male as well as female it was < 0.001. The ADHD-PI males also scored significantly lower on CD symptoms than the ADHD-C males ($p < 0.001$). Gender differences were only observed for the ADHD-C group ($p < 0.001$).

Figure 6.7 Graphical representation of the results of DBD- CD subscale

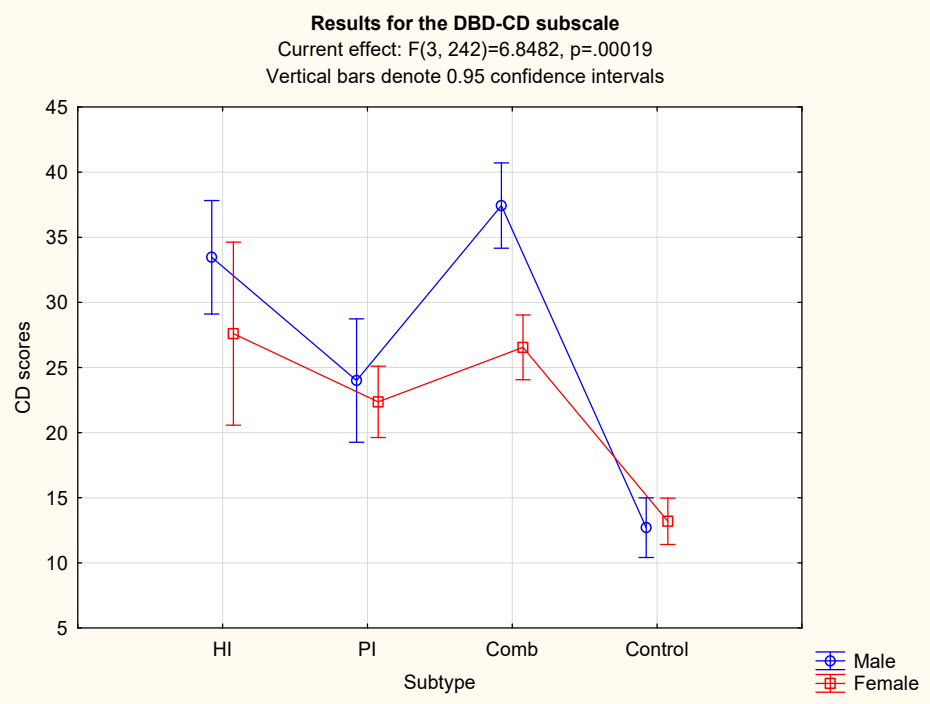


Figure 6.7 denotes that the ADHD subtypes scored significantly higher than the control group on symptoms of CD. Males, mainly, scored higher female, especially the ADHD-C subtype. The ADHD-PI males showed substantially fewer CD symptoms than ADHD-C males.

6.8 Anger

Table 6.12 illustrates the descriptive statistics for the results of the Anger scores of the Beck Subscale.

Table 6.12 Descriptive statistics for the Anger subscale

Subtype	Gender	N	Mean ± SD
ADHD-HI	*Male*	13	29.46 ± 7.99
	Female	5	27.80 ± 3.63
ADHD-PI	*Male*	11	20.82 ± 7.05
	Female	33	20.30 ± 6.73
ADHD-C	*Male*	23	27.00 ± 5.10
	Female	40	25.83 ± 6.28
Control	*Male*	47	15.85 ± 7.69
	Female	78	15.91 ± 8.30

Analysis of Variance (ANOVA)

Table 6.13 depicts the results of the ANOVA on Anger of the ADHD subtypes and gender.

Table 6.13 **Analysis of Variance for the Anger scores**

	DF	*F*	*p*	η_p^2
ADHD	3, 242	33.70	0.00*	0.30
Gender	1, 242	0.40	0.53	0.00
ADHD x Gender	3, 242	0.13	0.94	0.00

*$p < 0.001$

There was a statistically significant difference between the Anger scores of the ADHD subtypes and the neurotypical control group: F (3, 242) = 33. 698, $p < 0.001$, $\eta_p^2 = 0.30$. There was no effect of gender, neither main, nor interacting, F (1, 242) = 0.40, $p = 0.53$ $\eta_p^2 = 0.00$ Therefore, the gender groups were not analysed separately.

6.8.1 Post-hoc analysis (Bonferroni correction):

Post-hoc analysis revealed that there was a significant difference between the ADHD-HI and the Control group ($p < 0.001$). When the ADHD-PI subtype was compared with the Controls, there was also a significant difference ($p = 0.003$). Between the Combined subtype and Control group, there was also a significant difference ($p < 0.001$). In each case, the anger scores for the ADHD subtypes were significantly higher than those of the controls. There was no significant difference when the ADHD-HI subtype was compared to ADHD-C type, but a significant difference was observed between the ADHD-PI and ADHD-C subtypes ($p < 0.001$). The ADHD-PI subtype had fewer anger symptoms than the ADHD-C subtypes. Figure 6.6 illustrates the scores of the subtype and genders.

Figure 6.8 Graphical representation of the Anger results of the Beck subscale

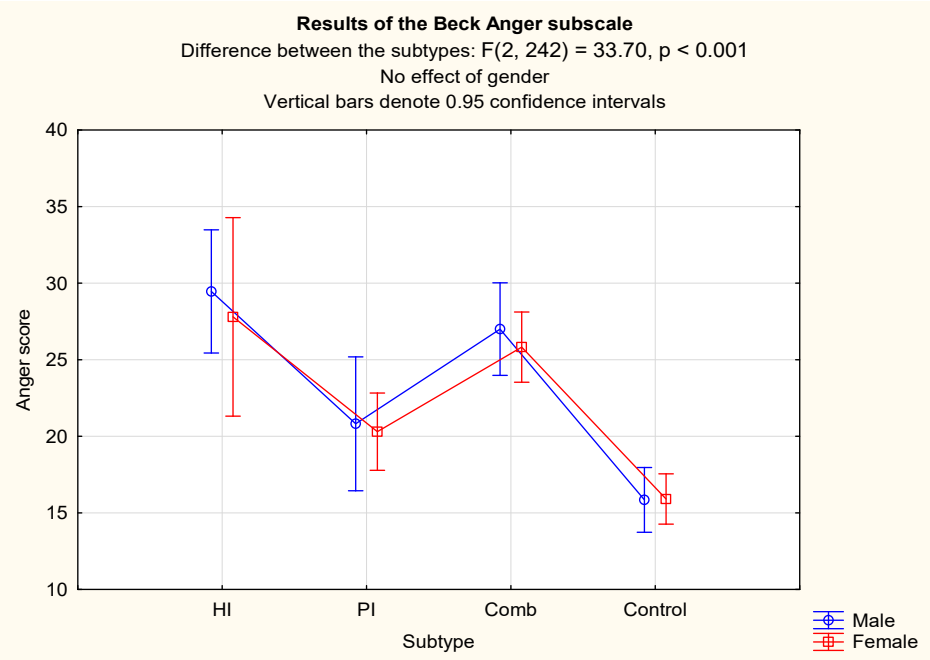


Figure 6.8 demonstrates that there is a difference between all three subtypes and the control group. The subtypes scored significantly higher on the anger scale than the controls. This was true for both males and females. The ADHD-HI and ADHD-C subtypes showed more anger than the ADHD-PI subtype.

6.9 Disruptive Behaviour

Table 6.14 illustrates the descriptive statistics for the results of Disruptive Behaviour scores of the Beck Subscale.

Table 6.14 **Descriptive statistics for the Disruptive Behavior subscale**

Subtype	Gender	N	Mean ± SD
ADHD-HI	*Male*	13	26.31 ± 5.42
	Female	5	22.20 ± 2.77
ADHD-PI	*Male*	11	18.91 ± 7.25
	Female	33	16.97 ± 7.92
ADHD-C	*Male*	23	23.57 ± 5.33
	Female	40	24.20 ± 5.52
Control	Male	47	12.36 ± 7.13
	Female	78	12.38 ± 7.41

Analysis of Variance (ANOVA):

Table 6.15 depicts the results of the ANOVA on Disruptive Behaviour of the ADHD subtypes and gender.

Table 6.15 **Analysis of Variance of the results of the Disruptive Behaviour Scale**

	DF	F	*p*	η_p^2
ADHD	3, 242	43.33	0.000*	0.35
Gender	1, 242	1.24	0.27	0.01
ADHD x Gender	3, 242	0.64	0.59	0.01

*$p < 0.001$

There was a statistically significant difference between the Anger scores of the ADHD subtypes and the neurotypical control group: F (3, 242) = 433, $p < 0.001$, $\eta_p^2 = 0.35$. There was no effect of gender, neither main, nor interacting, F (1, 242) = 1.24, $p = 0.27$ $\eta_p^2 = 0.01$ Therefore, the gender groups were not analysed separately.

6.9.1 Post-hoc analysis (Bonferroni correction):

Post-hoc analysis revealed that there was a significant difference ($p < 0.001$) between the ADHD-HI, ADHD-PI and ADHD-C subtypes when they were compared with the Control group, with all the ADHD subtypes having more symptoms of disruptive behaviour than the neurotypical controls. When the ADHD-HI subtype was compared to ADHD-PI subtype a significant difference ($p < 0.001$) was observed**.** Moreover, a significant difference between ADHD-PI and ADHD-C was depicted ($p<0.001$), with the ADHD-PI subtype showing fewer symptoms of disruptive behaviour. However, no significant difference was noticeable between ADHD-HI and ADHD-C.

Figure 6.9 Graphical representation of the results of the Beck Disruptive Behaviour subscale

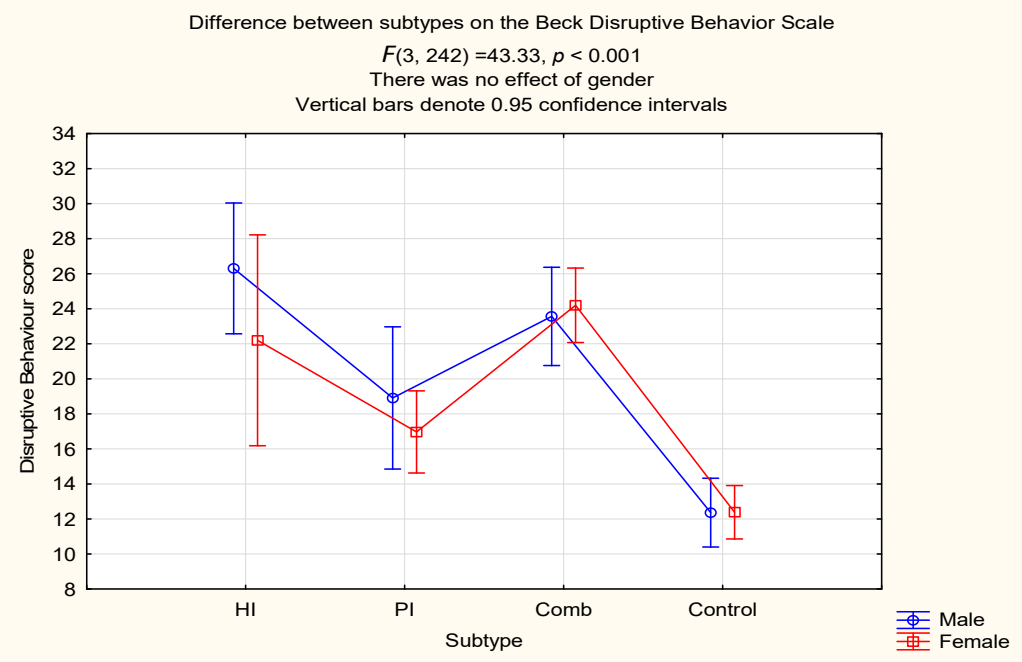


Figure 6.9 illustrates a significant difference in symptoms of disruptive behaviour between all three the ADHD subtypes and controls, with the ADHD groups having more symptoms of disruptive behaviour. The ADHD-HI and ADHD-C subtypes had significantly more symptoms of disruptive behaviour than the ADHD-PI subtype. There were no gender differences.

6.10 Hypotheses testing

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

Hypothesis 1: Children with ADHD have comorbid internalising disorders
1 (a) Children with ADHD have more symptoms of anxiety than children without ADHD. This sub-hypothesis can be accepted as the results show that children with ADHD had significantly higher scores on the Anxiety subscale of the Beck Inventory. This was true for all three subtypes. No gender differences were observed.

1 (b) Children with ADHD have more symptoms of depression than children without ADHD. This sub-hypothesis can also be accepted as the results show that children with ADHD had significantly higher scores on the depression subscale of the Beck Inventory. This was true for all three subtypes except for the females of the ADHD-HI subtype. This can be ascribed to the small sample size (n = 5).

Hypothesis 2: Children with ADHD have lower self-esteem than children without ADHD.
Hypothesis 1 which stated that children with ADHD have more internalising comorbid disorders than neurotypical comparisons, can, therefore, be accepted.

Hypothesis 2 can be partially accepted as only the children with ADHD-C and the boys with ADHD-HI had more symptoms of low self-esteem than the neurotypical controls. The ADHD-PI group, both males and females did not have significantly lower self-esteem than the controls. The girls with ADHD-HI also did not differ in this aspect from the controls, but this may be ascribed to the small size of the sample.

Hypothesis 3: internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who have more externalising comorbidities (Oppositional Defiant Disorder (ODD), Conduct Disorder (CD), anger and disruptive behaviour issues.
Hypothesis 3 must be largely rejected. The results did not show gender differences for the internalising comorbidities (anxiety and depression), except for depression results in the ADHD-HI girls, which result is probably unreliable because of their small

numbers. Although ADHD girls also had significantly more ODD symptoms than controls, the boys scored significantly higher than girls for symptoms of ODD. On the CD scale, only in the ADHD-C group showed major gender differences with the boys having considerably more symptoms of CD than girls. This was not observed in the ADHD-HI and ADHD-PI subtypes. No significant gender differences were found for disruptive behaviour and anger.

It can, therefore, be concluded that there are no differences between the genders for internalising disorders and that boys with ADHD suffer in the same way as girls from comorbid anxiety and depression. For externalising comorbidities (ODD and CD) however, more boys are affected, especially in the combined type of ADHD. Disruptive behaviours and anger seem not to discriminate between the genders. A discussion of the results will follow in the next chapter.

6.11 Conclusion

The findinigs of the current study were in line with literature conducted on the comorbidity of internalising disorders and ADHD. The presentation of the results confirmed the hypothesis of the current study that children with ADHD have comorbid internalising disorders (anxiety and depression) when they were compared to children without ADHD. The results also showed that children with ADHD do have low self-esteem. Moreover, the results went further to reveal that internalising disorders (anxiety and depression) are common in both genders and also externalising disorders (ODD, CD and anger issues) in boys and girls with ADHD. Interestingly, different ADHD subtypes (ADHD-I, ADHD-H and ADHD-C) showed different levels of symptoms of internalising, low self-esteem and externalising disorders. Both ADHD, anxiety and depression often comorbid in children and the costs of ADHD are more damaging (Leirbakk, Clench-Aas & Raanaas, 2015).

CHAPTER SEVEN

DISCUSSION OF RESULTS

7.1 Introduction

Research findings in the past have shown that ADHD is often comorbid with internalising disorders, i.e. anxiety and depression (Sagvolden et al., 2005). The main purpose of the study was to investigate whether children with ADHD have more comorbid internalising disorders (anxiety and depression) than a control group without ADHD symptoms and whether gender and subtype affect the expression of symptoms. Secondly, the study wanted to establish whether children with ADHD differed in self-esteem from their neurotypical counterparts and whether within-group differences could be found (gender and subtype). Lastly, a comparison was made of externalising disorders (ODD and CD) between the ADHD and control group as a function of gender and subtype, because according to the literature, males exhibit more externalising symptoms than females, while females show considerably more internalising symptoms (Biederman et al., 2002).

In investigating the prevalence of these psychiatric disorders, the researcher employed the DBD Scale (Meyer et al., 2004) for establishing symptoms of ADHD and externalising comorbidity as well as the BIY-II Scale (Beck, Beck & Jolly, 2005) to assess depression, anxiety and self-esteem. The results of the study indicated that children with ADHD have significantly more symptoms of both anxiety and depression than their neurotypical counterparts and they also showed significantly lower self-esteem. The results also confirmed that both boys and girls with ADHD showed more externalising comorbidity than the neurotypical controls, but that they were significantly more pronounced in boys.

7. 2 Demographic results

A sample of 125 children, clinically diagnosed with ADHD of both genders was used for the study. Both genders showed positive symptoms of inattention and hyperactive/impulsiveness on the DBD scores. The results showed thtat, there was no significant difference in age between the ADHD (M = 9.54 ± 1.48) and the Control (M = 9.61 ± 1.36) groups: $F(1, 228) = 0.12$, $p = 0.73$.

There was a significant difference in the scores on the Hyperactive/Impulsive scale of the DBD rating scale between the ADHD (M = 45,88 ± 5.70) and Control (M = 21.90 ± 1.18) groups: $F(1, 228) = 144.80$, $p < 0.001$.

There was a significant difference in the scores on the Inattentive scale of the DBD rating scale between the ADHD (M = 36,69 ± 3.12) and Control (M = 19.91 ± 5.47) groups: $F(1, 228) = 379.65$, $p < 0.001$.

7. 3 Anxiety

The results of the current study showed that children with ADHD have more symptoms of anxiety than children without ADHD (figure 6.2). This was true for the boys as well as for the girls. Most studies show that children presenting with ADHD often meet the criteria for comorbid anxiety disorder (Lee, Falk & Aguirre, 2012; Mikami, Ransone & Calhoun, 2011; Sciberras et al., 2014). Both ADHD and anxiety are psychiatric disorders causing significant impairments in individual functioning. Findings of the current study supports the idea that up to 50% of children with ADHDH meet the criteria for comorbid anxiety (Sciberras et al., 2014). Having both ADHD and anxiety may cause severe malfunctioning.

ADHD children generally struggle with impulse control as they are too cautious about their behaviour while on the other hand, they are unable to manage negative thoughts which precipitate their anxiety level. Theoretically, such impairments are associated with hypofunctioning mesolimbic dopamine branch causing altered reinforcement of behaviour and deficient extinction of previous reinforced behaviourinction of previous reinforced behaviour that is causing anxiety traits (Levy, 2004). Imparments in these dopamine system will only result in provoking anxiety which will be seen as ADHD

symptoms such as impulsivity. Children with ADHD are sensitive to personal failure (Castanga, Calamia & Davis, 2017); hence constant negativity by others will only make them even more anxious. Self-report on anxiety symptoms by children was found to be vital since it is strongly associated with cognitive impairments (Bloemsma, Boer, Arnold, Banaschewski, Faraone, Buitelaar, Sergeant, Rommelse & Oosterlaan, 2013). Self-reporting by children about their emotions becomes a window through which the child expresses his/her emotional state as he or she experiences them; hence that self-report should be part of the assessment in a clinical and research setting (Bloemsma et al., 2013). Based on this notion, anxiety should be part of the assessment when screening and treating ADHD.

According to Mayes, Cahloun and Crowell (2000), symptoms of anxiety are often observed during the mid-school years, and already children in their early phase of life are prone to anxiety. Another study such as Mychailyszyn, Beidas, Benjamin, Edmunds and Podeli (2011) found that the school environment is anxiety provoking and children often become anxious during their school years. The results of the current study were obtained from children in a school environment, hence more symptoms of anxiety were yielded from the ADHD group. The current study supports the notion by Marikangas et al. (2010) who showed that when anxiety is disruptive, it is often linked with a crowd of cognitive, behavioural and emotional difficulties. It could be based on these findings that anxiety is a function of multiple impairments. Although a diagnosis of ADHD is made, specific symptoms of anxiety can be overlooked and the impairments as a result of anxiety symptoms are more severe (Masi & Gignac, 2017). It was found that most of the children with ADHD who met the criteria for General Anxiety Disorder by their own self report, were not described as anxious by their parents (Pliszka,1998), which suggests that parents may often be unaware of their child's internalising symptoms.

A study by Brown (2000) showed that anxiety underpins cognitive performance especially in children with ADHD in which attention is mostly affected. On the other hand, Halldorsdottir and Ollendick (2014), also found anxiety to be reducing symptoms of impulsivity in children with ADHD. On the contrary, positive aspects of anxiety in tasks which measure response inhibition such as the Gostop test were observed (Brown, 2000) and children with both ADHD and anxiety performed well. On the other

hand, Shatz and Rostain (2006) view anxiety as a remedy to some of the cognitive impairments seen in ADHD. Anxiety appears to have both some positive and negative influence on ADHD. According to Levy (2004), Dopamine system functions as a domain where reward and delay of reinforcement are determined by tonic/phasic Dopamine relationships, resulting in impulsive “fearless”response. Hence, children with anxiety will require contant reinforcement from time to time.

7.3.1 Subtypes:

The results further allude that the occurrence of anxiety was present in all three ADHD subtypes when compared to the control group. However, the level of anxiety was found higher in children with the ADHD-PI and ADHD-C subtypes than in the ADHD-HI subtype. Although the current study observed less symptoms of anxiety in the ADHD-HI subtype, Sciberras and colleugues (2014), also found that having anxiety comorbidities was linked with poor quality of life and behavior in children with ADHD-HI and ADHD-C. In the same way, others noted a possible differential between ADHD subtypes with comorbid anxiety which is lower in the ADHD-HI subtypes (Jensen, Martin & Cantwell, 1997), than in the ADHD-PI and ADHD-C subtypes suggesting that anxiety is linked to the inattentive component of ADHD and therefore may worsen ADHD key symptoms in the subtypes that are inattentive. Tannock (2009) suggested that the effect of anxiety in ADHD is to decrease impulsiveness, but to increase difficulties with working memory and effortful processing. Thus children with ADHD/anxiety are more likely to appear less overtly hyperactive and disruptive, but they are also more likely to be slowed down, or inefficient, compared to ADHD-only children. This is a possible explanation for the lesser incidence of anxiety in the hyperactive/impulsive subtype and the higher occurrence in the inattentive and combined subtypes.

Woo and Rey (2005) theorised that hyperactive-impulsive as a subtype might not be a true ADHD subtype since it does not include attention deficit, which is the primary deficit in ADHD. Most studies may not have been able to directly observe symptoms of hyperactive/impulsiveness because anxiety in ADHD inhibits impulsiveness. Therefore, children with ADHD and comorbid anxiety may show less impulsive behaviour but show more symptoms of inattention (Halldorsdottir & Ollendick, 2014).

Based on this findings, it could be for this reason that symptoms of impulsiveness are less noticeable in children in order to identify the disorder and it appears that anxiety, in particular, plays a dual role as an inhibitor while complicating other functions. Therefore, having comorbid anxiety may intensify symptoms of ADHD while symptoms of hyperactive/ impulsiveness may be suppressed.

The current study further found that children of the ADHD-C subtype have most symptoms of anxiety. This is in line with findings by Willcutt et al. (2012) who also found that specific subtype had a higher likelihood to meet the criteria for comorbid generalised anxiety disorder (GAD), suggesting that symptoms of anxiety plays a significant role in intensifying ADHD symptoms, that is, inattention and impulsiveness, causing even more cognitive impairments and some impairments in components of executive functions such as planning, working memory, organisation and inhibition (Manassis, Tannock, Young & Francis-John, 2007). Executive Functionings (EF) refer to a variety of behaviours and abilities linked to planning and strategy as well as maintaining behaviour in pursuit of some goal (Miyaki & Friedman, 2012; Riccio, Wolfe, Romine¸ Davis & Sullivan¸ 2004). EF is a system of interconnected behaviours or process to facilitate a goal-oriented behaviour and the ability to form mental representation of a task (Barkley, 1997). It can be based on this notion that symptoms of anxiety can cause possible impairments in those functioning domains. Clinically, inattention is related to some cognitive impairments and children with ADHD and comorbid anxiety struggle with poor concentration, reasoning, forgetfulness and learning (Manassis et al., 2007).

The results of the current study revealed that symptoms of anxiety were found in all ADHD subtypes, therefore, causing even more impairment on individual ADHD symptoms. This finding was confirmed by others who reported that anxiety intensifies symptoms of ADHD (Manassis, 2008). Therefore, both ADHD and anxiety constitute multiple impairments in these individuals, which are seen when tasks requiring EF must be executed. This may suggest that children with ADHD-C experience more challenges as a result of anxiety causing impairment in both ADHD symptoms (impulsiveness/hyperactivity).

7.3.2 Gender differences:

No significant gender differences were observed in the levels of anxiety in the children with ADHD. Both boys and girls showed more symptoms of anxiety than their neurotypical group, but the genders did not differ significantly from each other. Most studies, however, have shown gender differences in ADHD with comorbid anxiety in which a positive association of ADHD and anxiety were especially noticeable for girls (Baldwin & Dadds, 2008; Mikami & Lorenzi, 2011). Biederman, Kwon, Aleardi, Chouinard, Marino, Cole, Mick and Faraone (2005) reported fewer symptoms of hyperactive/impulsive symptoms and more inattentive symptoms in females more than boys, suggesting more impairment in females than in males. This differed from the current findings in which boys and girls scored generally higher on symptoms of anxiety than the neurotypical controls, but did not differ significantly from each other. Others studies, however also found that boys and girls with ADHD are impaired the same way (Newcorn et al., 2001) which confirm the findings of the current study, suggesting that anxiety symptoms co-occurring with ADHD have the same effect on both genders. Other studies confirmed the same findings with the current study in which they observed that internalising disorders in girls and boys occur with the same frequency during childhood (Merikangas et al., 2010). From the results of the current study, it can be concluded that anxiety symptoms are expressed equally in boys and girls.

Based on these findings, it can be suggested that both ADHD and anxiety overwhelm the child from being eager to engage in activities especially requiring mental efforts because they are not able to execute tasks efficiently. Symptoms of anxiety were found to impair working memory and children with both ADHD and anxiety were seen to perform worse on such tasks comparative to children with ADHD alone (Jarrett, Wolf, Thompson, Davis, Cwart & Ollendic, 2016).

Children with both ADHD and anxiety are faced with devastating outcomes as anxiety in some way inhibits impulsivity and response inhibition deficits, while it makes working memory deficits worse (Shatz & Rostain, 2006). Children with ADHD and anxiety are often more impaired when compared to children with one condition. Moreover, impairments such as attentional problems, school fears, and mood disorders and lower levels of social competence were strongly associated with ADHD and comorbid anxiety

(thus compromising the quality of life of these children). Early identification and intervention that may eliminate the complexity that may arise as a result of these disorders should be a priority (Bowen, Chavira, Bailey, Stein & Stein, 2008).

7.4 Depression

The results of the current study revealed that children with ADHD have more symptoms of depression than the neurotypical control group (figure 6.3). This was true for boys as well as for girls. Both ADHD and comorbid depression, according to Reinhardt and Reinhardt (2013), are associated with risk factors relevant in clinical practice and should be treated as an emergency situation. The identification of those with ADHD who are at the risk of depression is essential for early intervention and prevention (Eyre et al., 2017; Leslie, Weckerly, Plemmons, Landsverk & Eastman, 2004). Therefore, proper assessment is crucial for clinicians and psychiatrists and should treat ADHD and depression as emergency circumstances in order to avoid worse prognosis which may arise as a result of comorbidities (Lopez, Masana, Marti, Acosta & Gaviria, 2012).

Studies in the past on depression, regarded depression as a predominantly adult disorder, while children were considered as developmentally immature to experience depressive disorders and the low adolescent mood was then seen as part of 'normal' teenage mood swings (Maughan, Collishaw & Stringaris, 2013). But many studies showed that major depressive disorder is not only present in children but in 25% of cases co-occurs with ADHD (Daviss, 2008; Pliszka, 1998; Tannock, 2009). The results of the current study also showed that symptoms of depression were also observed among children clinically diagnosed with ADHD. Most ADHD groups showed significantly more symptoms of depression than their neurotypical counterparts. The only exception was the ADHD-HI female group who did not differ significantly in depression symptoms from the female control group. These findings were consistent with other previous studies on higher rates of depressive symptoms in children with ADHD than in matched controls (Bakare, 2012; Barkley, 2006; Biederman et al., 2002; Biederman, Monuteax, Mick, Spencer, Wilens, Klein. Price& Faraone, 2006; Daviss, 2008; Littman, 2012; Pliszka et al., 1999; Rucklidge, 2010; Skogli et al., 2013), the present results showed that children with ADHD had significantly more symptoms of depression than the same age neurotypical group.

Depression generally appears many years after the ADHD symptoms (Biederman et al., 2008; Daviss, 2008; Kovacs, Akiskal, Gatsonis & Parrone, 1994), therefore, it is often not observed in younger children. It can be suggested that children are not born with ADHD, but the severity of the disorder may cause cognitive and behavioural impairments leading to the malfunctioning of these children and as a result leading to the development of depression. Theories linking ADHD and depression suggested that, such children are prone to negative feedback at school, social situations and in family environment (Daviss, 2008). All these negativity thus results in low self estee, demoralisatio and severe depressioni. On the other hand, genetic neurobiological anomalities and environmental effects may aloso have a great influence especially on children who are born from a parent with a genetic vulnerability for depression (Posner et al., 2014). Moreover, depression in younger children often manifests as symptoms of irritability which may often been mistaken for symptoms of ODD (Bennett, Ambrosini, Bianchi, Barnett, Metz, & Rabinovich, 1997; Eyre et al., 2017). The cumulative effects of the impairments caused by ADHD and negative environmental circumstances may lead to de development of depression in children at a later stage (Blackman et al., 2005; Daviss, 2008). Others were of the opinion that young children with symptoms of ADHD may not exhibit with clinical depression (Cadman, Findon, Eklund, Hayward, Howley, Ceng & Asherson, 2016).

Children with ADHD and depression occurring together display greater levels of impairment than children with either ADHD or depression alone (Blackman et al., 2005; Daviss et al., 2006; Rohde et al., 2001). Depressive disorders occurring in children with ADHD are observed frequently, they are impairing and pose special challenges with regard to proper assessment and treatment. ADHD is a neurodevelopmental disorder characterised by symptoms of hyperactivity/impulsiveness which often place children at the risk of receiving negative responses (Posner, Siciliano, Wang, Liu, Sonuga-Barke & Greenhill, 2014) which triggers symptoms of depression. On the other hand, Barkley (2010) found that children with ADHD tend to become demoralised especially that they cannot control their behaviour.

7.4.1 Subtypes:

The results further revealed that there were significantly more symptoms of depression present in all ADHD subtypes than in the neurotypical group. A noticeable difference were the ADHD-HI females, who did not show a significantly difference in depressive symptoms when compared with the neurotypical females. However, this was not the case for the ADHD-HI males, who displayed significantly more symptoms of depression than the male controls.

The study further showed that there were more symptoms of depression among children within the ADHD-PI and ADHD-C subtypes. This was in line with findings by Ushijima, Usami, Saito, Kadaira and Ikeda (2012) who also found a high incidence of the internalising disorder, in particular depression, among the same ADHD subtypes. Other studies, on the other hand, found that ADHD children, especially the predominantly inattentive subtype, were said to have more withdrawn depressed behaviour and fewere problems with sustained attention when compared to the rest of ADHD sample (Capdevila-Brophy, Artigas-Pallarés, Blas Navarro-Pastor, García-Nonell, Rigau-Ratera & Obiols, 2014). This concurs partially with the present study where symptoms of depression were not found in the hyperactive/impulsive girls. It can be based on these findings that inattentive symptoms of ADHD may aggravate individual emotional functioning.

In their study on ADHD subtypes and depression, Chronis-Tuscano, Molina, Pelham, Applegate, Dahlke, Overmyer and Lahey (2010), observed that ADHD subtypes in children strongly predict adolescent depression and suicide attempt later in life, suggesting that each ADHD subtype having different impairments on behaviour and functioning of the child is a call for intense intervention.

Although studies found high levels of aggression and irritability in children with both ADHD and depression, they were not able to locate symptoms of depression among the respective inattention and combined ADHD subtypes (Eyre et al., 2017; Mick, Spencer, Wozniak & Biederman, 2005; Power, Costigan, Eiraldi & Left, 2004; Crystal, Ostrander, Chen & August, 2001). As was also to a large extend found in the present study, symptoms of depression were significantly more prevalent in children with the ADHD-PI and ADHD-C subtypes. The use of the BYDI-II Depression Scale for the

current study to some extent does not incorporate items relating to irritability which is often symptoms of ADHD which are associated with major depressive disorder in children (Eyre et al., 2017; Mick, Spencer, Wozniak & Biederman, 2005; Power, Costigan, Eiraldi & Left, 2004). Based on these findings, it could be suggested that other forms of behaviours which can be associated with depression are likely to be overlooked therefore other behaviours need to be well explored.

7.4.2 Gender differences:

The results of the current study allude that both females and males in the ADHD subtypes showed symptoms of depression compared to the neurotypical group, but there was a statistically difference between the males and the females, with the females having significantly higher levels of depression than the males. The only exception was the ADHD-HI where no differences in the level of symptoms of depression could be found. Even the girls of the control group showed significantly more symptoms of depression than their male counterparts. This finding was contradictory with findings by Mokobane et al. (2017) who did not find gender difference on the co-occurrence of ADHD symptoms with depressive symptoms. Generally, females showed more symptoms of depression than males in the current study. However, symptoms of depression in females with ADHD are more damaging and place them at a higher risk for more impairments than males.

A noticeable difference was also observed within the ADHD-HI group where no significant difference could be found between the genders, but this could be due to the small size of this group. The findings of the present study confirm studies which also reported that girls are at higher risk for depression symptoms (Biederman et al., 2002) while boys are at higher risk for ADHD symptoms (American Psychiatric Association, 2013). Girls with and without ADHD seem to show more depression than their male counterparts (Littman, 2012; Rucklidge, 2010; Skogli et al., 2013) since females tend to engage in rumination and self-focus in response to stressful life-events whereas males tend to cope with stress by distraction (Lewinsohn, Gotlib, Lewinsohn, Seeley & Allen, 1998). Not only do girls show higher rates of depression than boys, they also show a different course of depression symptoms over time. From early adolescence the gender ratio for depression increases greatly, when girls' rates begin to exceed

those for boys (Burke, Hipwell & Loeber, 2010). Some studies proposed that boys and girls with ADHD are equally impaired, but most found that ADHD is more impairing for girls even though it is less prevalent (Mikami & Lorenzi, 2011). Symptoms of depression among girls are still underestimated with adverse outcomes. It can be based on these findings that depression in both females and males should be identified soon after a child is clinically diagnosed as having ADHD.

The findings of the current study also confirm the findings by Hassan, Eissa, Hwedi, Hegazy and Essam (2013), who found more symptoms of depression among females and especially among ADHD-C and ADHD-PI subtypes. These findings are also attributed to the fact that the sample of the current study had more females participating than males. These findings suggest that depression exacerbates key symptoms of ADHD and causing extreme impairments, particularly in girls. Symptoms of depression are more impairing especially when a child has ADHD besides, suggesting that the child has double impairments. Literature has indicated that comorbid internalising disorders are common yet still underrated because of the manner in which they present themselves. The diagnosis of depression in an ADHD child is alarming because of the nature of the symptoms causing severe impairments (Reinhardt & Reinhardt, 2013), hence, early intervention and treatment are vital to eliminate the suffering and function.

7.5 Self-esteem

The findings of the current study found that children with ADHD have lower self-esteem when they were compared to their neurotypical group. However, this was only true for the ADHD-HI and ADHD-C subtypes when they were compared with the neurotypical comparisons.

The result is consistent with findings of past research that shows that ADHD has a negative impact on the self-esteem of children. Some of the studies that support the hypothesis of this study include Barber et al. (2005) which reported lower self-esteem in children with ADHD when compared to controls. Barkley, 2006; Edbom et al. (2008); Klimkeit, Graham, Lee, Morling, Russo and Tonge (2006); Pisecco et al. (2001); Slomkowski et al. (1995) and Taanila, Hurtig, Miettunen, Ebeling and Moilanen (2009), also showed that ADHD has a negative impact on the self-esteem of children. These findings are similar to findings by Foley-Nicpon, Rickels, Assouline and Richards (2012)

who also found lower scores on measures of self-esteem in children with ADHD than children without a diagnosis of ADHD. On the contrary, Newark, Elsaaer and Stieglitz (2016) found similar results but in adults, suggesting that identifying low self-esteem during childhood is essential since lower self-esteem is persistent and can manifest into psychopathological impairments during adulthood which individuals could have been assisted through psychotherapeutic intervention while they were still children (Cook, Knight, Hume & Quereshi, 2014).

ADHD affects many aspects of life such as poor peer relationships, aggression and learning problems, which in turn are associated with academic failure and increased risk for development of self-esteem (Dan & Raz, 2015). This negative relationship between ADHD and self-esteem was also reported by many researchers (Edbom, Granlund, Lichtenstein & Larsson, 2008).

Quite often children with ADHD are not well understood and therefore regarded as being disrespectful by most. Children are likely to become overwhelmed from a situation of not being understood while they cannot take control of their biological disorder, which places them to develop either anxiety or depression easily and later develop low self-esteem. It is for this reason that it becomes essential to manage symptoms of ADHD through intervention strategies in order to minimise the vulnerability of developing other disorders which may compromise the self-esteem of these children. Positive self-esteem is fundamental and is an influential predictor for specific outcomes such as academic, relationships and health (Robin, 2014).

There are also studies that do not support the hypothesis of the present investigation, namely, that children with ADHD have a lower self-esteem than typically developing children, like the study by Hoza et al. (2004) which reported that children with ADHD tend to overestimate their own competence, reporting an inflated estimation of self-worth called positive illusory bias. They overestimate their perceptions of themselves most strongly in areas where they have the greatest skill deficit. The outcome of the present study seems to be in agreement with this conclusion.

Most studies showed that both children and adolescents suffering from ADHD have low levels of social skills and self-esteem as compared to the general population (Shaw-Zirt, Popali-Lehane, Chaplin & Bergman, 2005; Bussing et al., 2000) and these

findings are consistent with other studies indicating that also adults with ADHD continue to have problems in many areas of functioning (Barkley, Murphy & Kwasnik, 1996; Murphy & Barkley, 1996) thus suggesting that social functioning and self-esteem may play a role on future outcomes in ADHD (Slomkowski et al., 1995).

Children with ADHD often have other key difficulties, but this varies widely from one child to the next. For example, some children have ADHD alone; others have externalising problems such as oppositional defiant disorder (ODD) or conduct disorder (CD) in addition to ADHD, while others have mood or anxiety problems (internalising disorders). In addition, there is an unfortunate subset of children with ADHD who have both co-occurring internalising and externalising problems. Clearly, self-esteem in children with these different combinations of difficulties would be expected to vary.

7.5.1 Subtypes:

The results of the current study further revealed that only children from the ADHD-HI, and ADHD-C subtypes showed lower levels of self-esteem compared to the neurotypical group. This was true for both males and females. Impulsiveness, which is a symptom of the disorder and a component of both ADHD-HI and ADHD-C, could affect the self-esteem negatively. When children have trouble inhibiting their behaviours, comments or responses, they may act before thinking or react without considering consequences. This could lead to their parents or teachers reacting harshly to them and could sometimes make them feel bad about what they have done thereby leading to a low self-esteem which shows a negative impact of the disorder (Santrock, 2002). Children presenting with both hyperactivity and inattention continuously find themselves in an overwhelming situation, in which individuals experience a distorted sense of self and disruption of the normal development of the self (Krueger & Kendall, 2001).

A study by Slomkowski, Klein and Mannuzz (1995) found that hyperactivity in children was associated with low self-esteem as adolescents. Similar findings with the current study were also observed wherein children in the ADHD-HI subtype also showed lower levels of self-esteem. Hyperactivity is an identifying symptom of ADHD and thus put children at the risk of been easily rejected and being referred. Based on this notion it

could be suggested that hyperactivity is a predisposing factor for multiple impairments that can be observed through various developmental phases of life from childhood, adolescent and through adulthood.

7.5.2 Gender differences:

There was a statistically significant result between the genders for the non-ADHD group where the boys had higher self-esteem than the girls. However, this result was not relevant to the present study. Gender differences have only been infrequently assessed in studies on self-esteem in ADHD. However, Ek, Westerlund, Holmberg and Fernell (2008) found significant gender differences with girls reporting lower self-esteem and poorer relationships with parents and peers, which was also found by Quinn and Wigal (2004) who noted that girls with ADHD reported more problems with parents and friends than boys with ADHD.

7.6 Internalising vs Externalising Disorders

ODD, CD as measured on the DBD rating scale and Anger and Disruptive Behaviour as rated on the Beck Inventory can all be regarded as externalising behaviours which are often found being comorbid with ADHD, especially among males (Barkley, 2004; Faraone et al., 2015). The results of the present study found that all four measured externalising disorders were significantly higher in the ADHD group than in their neurotypical comparisons. This was true for all three subtypes. However, for ODD and CD, there was an effect of gender, with the ADHD-C girls scoring significantly lower on the ODD and CD scales than the boys, however, still significantly higher than the control females. No gender differences were observed on the Anger and Disruptive Behaviour scales of the Beck Inventory.

The results of the current study further revealed that children with a clinical diagnosis of ADHD generally had more externalising and more internalising disorders when they were compared to the neurotypical group. The result was similar for both males and females. Children who were clinically diagnosed as having ADHD were observed to have anxiety and/or depression but also symptoms of externalising disorders. Both anxiety and depression seem to have significant impairments which complicate the presentation of ADHD. When a child has ADHD, having internalising disorders is more

alarming as it aggravates many of the ADHD symptoms. It should be fundamental that when ADHD coexist with other psychopathologies the one that is more impairing should be treated first (Katzma et al., 2017) and have ADHD treated as a separate disorder. Siroisi and colleugeus in Masi and Gignac (2017) stated that conduct disorder is a situation that contributes to the severity of the condition. Studies showed that children presenting with both conditions have a poor prognosis even in their adulthood (Masi & Gignac, 2017). It is for these reasons that early identification of ADHD comorbidities is important in order to prevent impairments that may arise as a result of these comorbidities.

7.6.1 Gender differences:

The prevalence of anxiety and internalising disorder, did not differ significantly between the genders, both the males and the females had higher levels of anxiety than the control group, but the level of anxiety did not differ between the genders. However, there was an effect of gender in the other internalising disorder under investigation, namely depression. Although, except for the female ADHD-HI group, all ADHD groups and even the female control group had higher levels of depression than their comparisons, suggesting that girls are more prone to symptoms of depression than boys. Gender differences were also found in ODD and CD symptoms, where the males of the ADHD-C group in both instances differed significantly from the female ADHD-C group in that the boys had significantly higher levels of both ODD and CD symptoms than the girls. No differences in gender were observed in symptoms of anger.

The results of the study partially confirmed most studies who observed internalising disorders (in the present study only depression) to be more in females whereas externalising disorders (ODD and CD) were more observed in males (in the present study only in the ADHD-C group) (Skogli, Teicher, Andersen, Hovik & Oie, 2013). Boys therefore, are at risk of being more easily referred because of their acting out behaviour than girls because who exhibit a more reserved nature. Previous studies showed that the frequency of ADHD is higher in boys than in girls while the comorbid anxiety and depression are more noticeable in girls (Quinn & Madhoo, 2014). These findings concurred partially with the findings of the current study which found comorbid anxiety equally in both genders, but depression more prevalent in girls. On the other hand,

ODD and CD is more frequently found in boys, at least in the combined subtype of ADHD as the present study indicates.

Based on the current findings, studies showed that developmentally girls mature faster than boys, which is the reason why girls with ADHD can develop better-coping strategies which enables them to be able to mask their symptoms way better than boys (Quinn & Madhoo, 2014), in other words, internalise their symptoms. Internalising disorders place girls with ADHD at the risk of having future adverse outcomes the same way as boys with ADHD. Both ADHD and internalising disorders will impact negatively on the child's scholastic performance.

In a study conducted in girls with ADHD, it was found that when they have increased ODD symptoms and aggression it predicted risky sexual impairments, involvement in abusive relationships and criminal behaviour (Biederman et al., 2010; Monuteaux, Faraone, Gross & Biederman, 2007). The results of the current study did not show any gender difference among children with ADHD and symptoms of disruptive behaviour since risk taking behaviour was not focus area of the study. Although studies indicated minimal agreement about the causes of gender differences in aggression, disruptive behaviour and delinquency (Loeber, Capaldi & Costello, 2013), this was in line with the current findings, suggesting that both ADHD and disruptive behaviour are not gender based.

7.6.2 Subtypes:

The results of the current study further revealed that only girls in the ADHD-HI showed significantly lower symptoms of depression than the controls. The clinical presentation of ADHD in girls is may often be underrated because symptoms of depression which are internalised are less noticeable than those of hyperactivity/impulsivity (Quinn & Madhoo, 2014), making it difficult to be identified and referred. The findings showed that symptoms of depression were associated more with inattentive symptoms, than hyperactivity/impulsiveness, as mostly the subtypes with the ADHD component of inattention showed symptoms of depression, suggesting that there is a strong link between inattention and internalising disorders. For externalising disorders, no differences between the subtypes were noted. For ODD, CD, Anger and Disruptive Behaviors all the ADHD groups had a higher prevalence of symptoms than the

neurotypical control group. As hyperactivity is a component common to all three subtypes, it can be assumed that external behaviours are linked to that component of ADHD.

In a study conducted by Jackson and King (2004) on gender differences in the effects of oppositional behaviour, more symptoms of ODD among boys and girls were found in the ADHD-C subtype. Similar findings were also observed in the current study although symptoms of ODD were also noticeable within the ADHD-HI, ADHD-PI subtypes and ADHD-C subtypes as well as in both genders, but significantly less in girls. The results also confirm that symptoms of hyperactivity/impulsivity predict possible development of conduct disorder. High incidence rates of CD were found among children with ADHD (Connor & Dorfler, 2009). The findings of the current study also revealed that males in the ADHD-PI reflected substantially fewer symptoms of CD when compared to males in the ADHD-C subtype, indicating that a combination of hyperactive/impulsive and inattentive symptoms may aggravate the condition.

The results of the current study further denote that children with all three ADHD subtypes showed more anger when they were compared to the neurotypical group. This was true for both males and females. A noticeable significant difference was that the ADHD-HI and ADHD-C subtype had significantly more anger symptoms than the ADHD-PI group, suggesting that hyperactivity/impulsiveness is an aggravating factor in expressing anger symptoms. Individuals with ADHD have low frustration tolerance and recurrent temper tantrums (Connor, Chartier, Preen & Kaplan, 2010). According to Carre, McCornick and Hariri (2011), impulsive aggression is characterised by behavioural disinhibition, with lack of planning and concern about consequences. Symptoms of impulsiveness which are seen as a lack of forethought can be associated with anger. This concurs with the current findings since it also found more symptoms of anger among the ADHD-HI group. Concurs

The results of the current study also showed that children with all three ADHD subtypes showed more symptoms of disruptive behaviour than the neurotypical group. The study further observed more symptoms of disruptive behaviour in children with the ADHD-HI and ADHD-C subtype. Children within the ADHD-HI and ADHD-C subtype were found to display externalising and social problems when they were compared to ADHD-PI children (Gaub & Carlson, 1997), suggesting that ADHD symptoms like

hyperactivity/impulsiveness are associated with complex behaviours and more difficulties in an individual's ability to succeed in life.

ADHD, as viewed by the clinical professional, is a disorder with deficits in behavioural inhibition and sustained attention (Barkley, Cook, Dulcan, Campbell, Prior, Atkins, Gillberg, Solanto-Gardner, Halperin, Bauermeister & Pliszka, 2002). The significant symptoms of inattention, hyperactivity and impulsivity can hinder many characteristics of an individual's life producing significant behavioural and cognitive difficulties (Cooper, 1999). Therefore, children within this category (ADHD-C) suffer deficits of both ADHD components which suggest significant behavioral impairments. Studies conducted alluded that ADHD-C subtype was associated with risk factors and conduct disorder symptoms (Counts, Nigg, Stawick, Rappley & Eye, 2005).

7.7 Concluding remarks

The results of the current study confirm the hypothesis that children with ADHD have comorbid internalising disorders (anxiety and depression) and children with ADHD have lower self-esteem than their neurotypical peers. The results obtained in this study were drawn from a community-based population. This explains that having ADHD symptoms and other symptoms related to the disorder may be not being noticed by parents and teachers and therefore proper assessment will not be implemented so that intervention can be initiated in order to prevent pathological behaviours that may come as a result of the disorder.

Moreover, children with ADHD and comorbid internalising disorders are double impaired hence intensive intervention should be a priority. Studies have indicated that ADHD behaviour is caused by impaired dopamine dysfunction, and as a result neurobiological processes are affected and impairment is caused in the executive functions domains (Johansen et al., 2002), which lead to mood and behavioural disorders. This to some extent increases the likelihood of developing anxiety and depression. Based on these findings, it can be well understood that children with ADHD suffer double impairment as a result of this comorbid disorders. Moreover, ADHD will have undesirable later consequences in the lives of these children. Based on this findings, it can be suggested that ADHD comorbidities are a gateway to risk-taking

behaviours. Therefore, establishing other extreme behaviours that can be linked to CD in girls needs to be well explored.

CHAPTER EIGHT

GUIDELINES ON THE ASSESSMENT AND MANAGEMENT OF ADHD

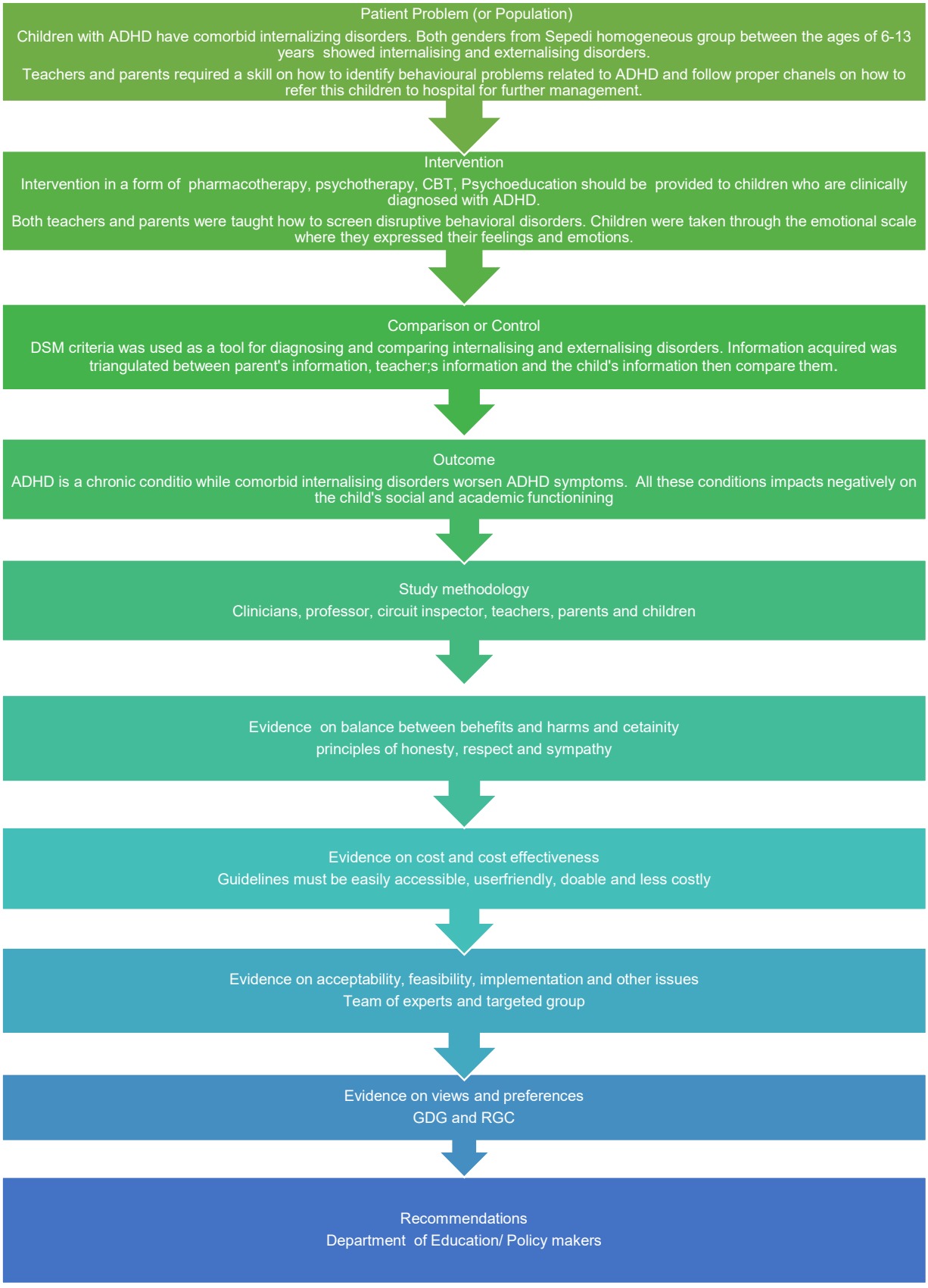
8.1. Introduction

ADHD is the most common neurobehavioral disorder of childhood and can profoundly affect the academic achievement, well-being and social interactions of children (Subcommittee on Attention Deficit Hyperactivity Disorder, 2011). The disorder is again associated with a broad range of functional impairments and negative outcomes resulting in a financial burden on families and society as a whole (Danckaerts, Sonuga-Barke, Banaschewski, Buitelaar, Döpfner, Hollis, Santosh, Rothenberger, Sergeant, Steinhausen & Taylor, 2010).

The assessment of ADHD by teachers and parents may often be limited towards assisting learners. Often an assessment of these nature requires a team effort i.e. both the teacher and the parent must be involved so that at the end, the child is not only viewed from one side i.e. from home or school. This has become a call for clinicians who make another assessment that they should assess for the possibility of other psychiatric disorders in order to make the actual diagnosis and also not misdiagnose. Terzian, Hamilton and Ericson (2011) stated that internalising disorders such as depressive or anxious mood, negative self-perception and emotional distress, if remain untreated can undermine the one's ability to succeed in school, live a healthy lifestyle, form and maintain close relationship with others. Clinical guidelines have become a valuable instrument for evidence-based medical practice, better quality of care, resource allocation and cost-efficient service provision (Melnyk, Fineout-Overholt, Gallagher-Ford & Stillwell, 2011). Guidelines development of the study are based on the current results which state that children with ADHD have internalising comorbidities, low self-esteem and that girls display more symptoms of internalising disorders than boys with externalising disorders. Therefore, guidelines were developed to assess and manage symptoms of ADHD.

Guidelines development in the current study were guided by the PICO (Population, Intervention, Comparator and Outcomes) as well as the GRADE (Grading of Recommendations, Assessment, Development and Evaluation) Workgroup, for quality assessment. All these were clinical medical tools which focused on clinical based evidence approach. The PICO was the mostly preferred tool for this study because it entails elements required in a question leading a systematic search of the evidence (World Health Organization,). Again, the PICO is a model relevant to define clinical questions in a research study. Below is a framework that outlined both the PICO and GRADE steps which were followed to define the clinical questions for the current study.

Figure 8.1 Framework for PICO and GRADE guidelines



8.2 Description of PICO and GRADE framework development

8.2.1 Population for the problem

The target population was school going children between the age of 6- 13 years of age. This was a homogenous group of boys and girls from primary schools in Lepelle-Nkumpi Municipality. The main problem was that children with ADHD had comorbid internalising disorders. ADHD poses a challenge to both teachers and parents hence there was a need to train them on how to screen for disruptive behavioural problems through the use of DBD scale. The guidelines were developed to assist teachers and parents to be able to identify possible symptoms and behaviour problems that are suspicious to be ADHD. This will assist by having those children suspicious of having ADHD to be referred early to the hospital for further management by professionals.

8.2.2 Intervention or exposure

Children who were screened for disruptive behavioural problems were referred and received proper diagnosis by the clinician. Clinician assessed for other comorbid disorders other than ADHD. Children were taken through an emotional scale where they expressed their innermost feelings without intimidation. It was through this process that clinician could identify the most impairing disorder and then assisted the child to overcome those disorder so that ADHD was treated as a separate condition. Intervention in a form of pharmacology, therapy, psychoeducation, and CBT and behaviour modification formed part of treatment plan. ADHD has devastating outcomes, however, early identification of these disorders before pathology emerge resulted to effective functioning eventually.

8.2.3 Comparisons or comparative intervention

The guidelines were compared with the DSM-5 criteria in identifying psychiatric disorders. Information obtained was triangulated between teacher's information, parent's information as well as children. The three scales were then compared to develop variables (anxiety, depression, self-esteem, anger, disruptive behaviour, ODD and CD). Both internalising and externalising disorders comorbid disorders were associated with ADHD symptoms. The ADHD group was compared to the neurotypical

group according to gender and ADHD subtypes.

8.2.4 Outcomes

ADHD if left untreated has devastating results. ADHD has negative results on scholastic performance and compromises quality of life and leads to school dropout. Having comorbid internalising disorders (anxiety and depression) impacts negatively on their cognitive functioning and worsen some ADHD symptoms. The newly developed guidelines will serve as an eye opener to policy makers, parents and teachers on how to provide the necessarynsupport to those learners experiencing challeges. This will be done with the notion that lenears can be assisted in order to perform to the best of their ability irrespective of their clinical diagnosis. The developed guidelines will further give reccomendations to Education department to strengthen the availability of services by psychologists at district level to conscientise psychologists to administer both scales (DBD and BYI-II) simultenously to arrive at the actual diagnosis prior to treatment intervention and to give recommendations to policy makers to amend policies which will accommodate children with ADHD. Moreover, children will benefit from learning in an environment that is non discriminative, while receiving an inclusive support system without being stigmatised.

8.2.5 Study methodology

This was a quantitative study because the reasearcher preferred to obtain more information in a numerical form. More information was obtained from teachers, parents and children themselves. Moreover, a quasi-experimental design was used because all primary school children were given an equal opportunity to participate in the research study. Study methodology used comprised of two groups i.e. the clinically diagnosed ADHD group and the non-ADHD group which were subjected to comparison and intervention.

8.2.6 Evidence on balance between benefits and harms and certainty

Participants in the study benefited from principles of honesty, respect and sympathy. Moreover, clinically diagnosed children were referred to the hospital for further intervention in a form of psychopharmacology, behaviour modification and

psychotherapy. The guidelines ensured that participants were protected from being harm by keeping ADHD children's identity anonymous at all times to be protected from the issue of stigmatization and being labelled by their peers. This was done through the permission of the parent or guardian of the child. Treatment adherence and constant support were ensured towards supporting the child. With the implementation of the guidelines, it will ensure that parents and teachers are enlightened about screening and identifying disruptive behaviours at an early phase. Children as a results will benefit from early intervention to curb the negative outcomes that may arise as a result of the disorders.

8.2.7 Evidence on cost and cost effectiveness

These guidelines will be run to all the stakeholders and it was concluded based on their responses regarding applicability and usability that these guidelines are user friendly. Developed guidelines were ensured that they are doable and less costly to those who can benefit from it. It was made certain that these guidelines were easily accessible and user-friendly to all members. Copies of the guidelines were put on notice boards of each class room and school reception which were made in a form of a flyer to remind teachers and parents on how to screen and manage ADHD in a situation where they are in contact with such a child.

8.2.8 Evidence on acceptability, feasibility, implementation and other issues

The guideline development group (GDG) were drawn from Lepelle-Nkumpi Municipality. This municipality was nominated because of the influx number of learners referred for scholastic assessment. The GDG comprised of 2 clinical psychologists from the hospital, one professor who specialised in the field of ADHD, one circuit manager, five teachers, five parents (School Governing Body-members). The two clinical psychologists and one professor acted as methodological experts. The guidelines were set for a target of twelve months in which the GDG had several meetings to refine the recommendations. The GDC ensured that whatever recommendation they come up with, was taken to the public to assess if their recommendations are acceptable, feasible and can be implemented. They took comments and suggestions made by the public and discussed in their meeting for improvements and kept record of amendments made.

8.2.9 Evidence on views and preference

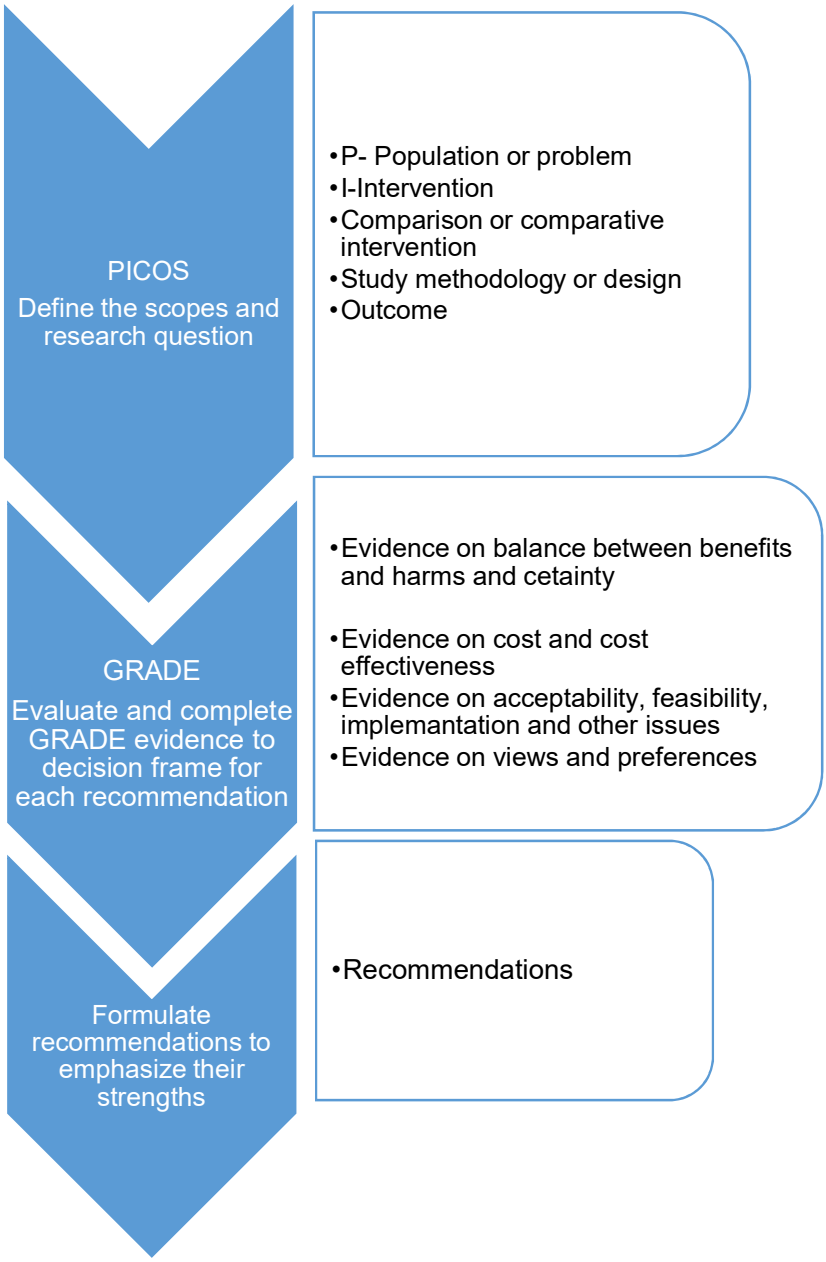
The GDG took comments made by others such as the people mentioned above, then assessed their views and preference with regard to recommendation made before they can be publicised. The GDG had the responsibility to check what is more suitable or not suitable by the members. Suggestions brought forward were then aligned with the recommendations to suit relevant members.

8.2.10 Recommendations

The GDG finally formulated recommendations and then assessed their strengths if they are supportive to teachers, parents and children. The GDG agreed that other relevant stakeholders should benefit from these recommendations. Recommendations made were: Limpopo Department of Education should provide educators with workshops on ADHD to ensure that they are more skilled in assisting those children, Information about ADHD and comorbid disorders should form part of the new curriculum during teachers training so that they know how to screen, manage and teach children with ADHD so that they achieve well and become successful. Policy makers to make policies which do not discriminate children having ADHD from acquiring knowledge and from other learners and they should be treated fairly. Upon agreement on the specified recommendations, the guideline panel then agreed on recommendations which were based on evidence, values and preference. The panel took into account that recommendations were valid for a period of twelve months and were subjected to change.

Figure 8.2 illustrates the method of integrating values and preferences in developing guidelines. The guideline development group framed the recommendations based on evidence, values and preferences.

Figure 8.2 Intergration of values and prefernces for guidelines



8.3 Guidelines development

8.3.1 Setting objectives

The main objectives of the study were to determine whether children with ADHD have comorbid internalising disorders (anxiety and depression), furthermore to determine if children with ADHD have low self-esteem and whether internalising comorbidities are common in girls than externalising disorders which are common in boys. Many studies thus far have confirmed that psychiatric and physical comorbidities were common in children with ADHD. Multiple impairments observed among children in a form of scholastic challenges, rejection by others and social malfunctioning may be as a results of these disorders.

8.3.2 Targeting your audience

Guidelines targeted at teachers and parents in order to be able to screen children presenting with disruptive behavioural disorders either at home or school environment. Early identification of children with behavioural problems prevented children from engaging in risk taking behaviours, school dropout and developing pathological behaviours which could have been curbed at an early phase. Planning for guideline development assisted teachers and parents towards helping ADHD children was proposed.

8.3.3 Timelines

Guidelines were developed over a period of twelve (12) months to allow the guideline panel to critique the content and if the guidelines covered evidence on all the necessary questions. Below is a time line for guidelines development:

Table 8.1 Time line framework for guidelines development

Activity	Responsible Team	Target
Guidelines Proposal	GDG	Jan-Feb 2018
Submission of 1st draft	GDG	March- Apr 2018
Corrections and submission	GDG	May 2018
Approval	GRC	June 2018
Developing Guidelines	GDG	Jul- Aug 2018
Distribution of guidelines	Team of Experts	Sept 2018
Distribution of guidelines	Teachers, parents, children	Oct 2018
Evaluation	Internal and External partners	Nov 2018
Translation	GRC	Dec 2018
Dissemination	Teachers, parents, children	Jan 2019

8.3.4 Funding

An amount of R175 000.00 awarded from Research and Publication Committee assisted in organising meetings with the guideline panel to synthesize the guidelines for relevancy. Consultations with a panel of experts (clinicians, professor, circuit inspector, teachers and parents) about the significance and editing of the guidelines also consumed a lot of funding.

8.3.5 Existing guidance and resources

There were existing guidelines which were similar to the current study. Although they only give an outlay of ADHD description, symptoms and treatment, the current guidelines included the comorbidity of internalising disorders which brought in awareness on other psychiatric disorders which often co-exist with ADHD while making

the disorder to appear to be in its worse form. Hence the current developed guidelines would assist both teachers and parents not to only pay attention to behaviour but also emotional functioning of the child as they come in contact with them so that they act immediately on the child's situation.

8.3.6 The evidence base

The use of other scales such as the BYI-II served as a screening tool which assessed the children's emotional functioning further confirmed that psychiatric disorders were prevalent in ADHD children. Also information obtained from the teachers' and parents' DBD scale confirmed that children with ADHD have symptoms of anxiety and depression, and low self-esteem and other externalising disorders (ODD, CD). Therefore, guidelines development was influenced by the evidence from the results of the current study.

8.3.7 Who was involved?

The Guideline Review Committee (GRC) was established to give expert knowledge and directives in the development of guidelines. The committee consisted of the secretariat, who coordinated and gave support on guidelines development. The GDG consisted of 2 clinicians, 1 professor specializing in the field of ADHD, circuit inspector, 5 school teachers, 5 parents and 5 children who fully participated in the development of guidelines.

Another group of experts was established and the team was comprised of clinicians and professor with an expert knowledge in the field. These teams were selected because they have provided primary care and have advocated that ADHD is a chronic condition. Through their expert knowledge, they gave constructive inputs on the guidelines development, reviewed and approved the completed draft of the document on guidelines. The same group was responsible to implement the recommendations in the guidelines.

8.3.8 Type of publication

Following the approval and implementation of guidelines, information was distributed in a form of flyers which were made accessible to parents and teachers and children at the school reception. Guidelines were put on notice boards in each and every class as a reference for teachers to remind themselves about the steps to follow each time they are in contact with a child with behavioural and emotional problems.

8.3.9 Translation

For the purpose of values and preference, guidelines were translated into Northern Sotho which is the language mostly spoken in Lepelle-Nkumpi Municipality. Most parents and children who were not conversant in English found the guidelines user-friendly. The budget allocated from RPC was able to pay for translation and editing of guidelines.

Figure 8.3 Planning for guidelines

Figure 8.3 illustrates the guidelines process

8.4 Scoping the guidelines

The developed scope for guidelines was for learners from mainstream primary schools in Lepelle-Nkumpi municipality in Lebowakgomo subcircuit. Both boys and girls ranging between the ages of 6 -13 years formed part of the scope of guidelines. Those children with an IQ <80 and a history of neurological problems (e.g. head injuries, epilepsy, cerebral palsy, etc.) or severe psychiatric disorders (as reported by the parents/guardians) were excluded in the guidelines.

The guidelines further excluded children on psychostimulant medication and prescribed psychotropic medication for ADHD or related disorders within 6 months of the time of evaluation. One primary school in the sub district was excluded in the scope of guidelines because the school is for learners with a presentation of mild to moderate intellectual functioning and the curriculum of the school caters skills and semi-skilled work.

The developed guidelines scope of practice was applicable to school teachers, parents, children, clinicians. Moreover, the scope of guidelines also applied to other stakeholders such as Limpopo Department of Education and policy makers with a hope they can make effective changes.

Children who were identified as having ADHD and comorbid disorders were referred to the hospital for pharmacological and therapy intervention. Of those children who were referred they showed improvement in their scholastic and social functioning and also there was a huge improvement on managing unwanted behaviour through behaviour modification and reinforcement for a well achieved behavior. Those who were prescribed medication (Ritalin) some experienced the negative side effects of the drug such as difficulty falling asleep and symptoms of hyperactivity especially after school hours especially that Ritalin must be taken in the morning so that it improves their inattention. Most parents complained about children gaining weight and their concern whether they should give their children treatment during recess.

The table below indicates the entire scoping process:

Table 8.2: Scope, purpose and stakeholders

Title of the guidelines:	**Towards developing guidelines that will improve screening of ADHD and internalising disorders among primary school children in Lepelle-Nkumpi, Limpopo Province.**
Principal authors	PhD Candidate: Mrs Takalani MC Promoter: Prof Mulaudzi MT Co-Promoter: Prof Sodi T
Guideline development group (GDG)	**Managers** 1. Acting Head of Department of Education, Provincial Government 2. Circuit Manager Lebowakgomo Sub circuit 3. Statistician: Expert in the field of ADHD 4. 2 Clinical Psychologists from Lebowakgomo hospital **School Personnel** 1. School principals from 5 different schools within the sub circuit 2. 5 SGB members who are representatives of parents
Duration	12 months, from December 2017 to December 2018
	The main purpose is to provide an evidence-based clinical practice guideline (CPG) that is consistent with the stipulated DSM criteria Guidelines and model of assessment and to encourage interventions that would capitalize on benefits and minimize harm to children with ADHD and comorbid disorders in primary schools in Limpopo Province This CPG is independent of and not intended to replace the standards of the DSM criteria
Clinical problem	The impact of comorbid internalising disorders on ADHD symptoms among primary school children
Care providers	Policy makers, teachers and parents of children in primary schools in Lebowakgomo sub circuit
Consumers	Children who meet the clinical diagnosis of ADHD and comorbid internalising disorders
Review date	Three (3) years or when there is new study recommendations or new DSM in place by the American Psychiatric Associassion (APA)
Setting for guideline implementation	Primary schools within the Lebowakgomo sub circuit in Lepelle-Nkumpi Municipality, Limpopo province

8.5 Guidelines and recommendations

Table 8.3: Recommendation 1

Provision of workshops/training on ADHD for early identification	
Guideline 1	
1.1	Provide teachers with adequate training on behavioural and psychological problems affecting learners in primary schools
1.2	Ensure that teachers are well informed about the impact of ADHD and comorbid disorders on scholastic performance, social functioning, self esteem and behaviour outcomes in the foundation phase are well trained.
1.3	Equip teachers with intense knowledge as part of their training curriculum to improve awareness and how to act promptly when confronted with such children
1.4	Improve skills, that provides more teachers with the ability to do screening of any suspicious behaviour related to ADHD
1.5	Have proper and good practice to screen and refer for further management
1.6	Encourage collaboration between teacher, parent and children for positive outcomes
1.7	Protect diagnosed children from victimisation and rejection to prevent unnecessary school drop-outs
1.8	Create a conducieve environment for understanding between the teacher and the parent

Table 8.4: Recommendation 2

Supply of DBD Scales to be used for screening and assessment	
Guideline 2	
2.1	Provision of DBD scales in all primary schools e.g with the secretary for accessibity
2.2	Copies of DBD scales should be available at the admin desk for accesibilty by teachers and parents
2.3	The admin office to ensure that scales are available for staff and parents should there be a need to screen a learner

2.4	Avail scales both English and N Sotho for those parents who cannot be conversant in English
2.5	Ensure training for teachers and parents to avoid misunderstanding
2.6	Provide support and monitor the process

Table 8.5: Recommendation 3

Establishing school outreach services for assessment of children in primary schools with psychologists	
Guideline 3	
3.1	Establish and maintain outreach services in schools for assessing children for disruptive behaviours
3.2	State clearly the process of assessment and ensure confidentiality
3.3	Provide collaboration between teachers and parents for effective outcomes
3.4	Emphasise on the role of the teacher and the parent as key informants about the child's behaviour
3.5	Explain the consequences of disruptive behaviours and comorbid disorders on the child's functionality and possible late outcomes
3.6	Provide personal consultation and coaching
3.7	Provide school support programmes for teachers and children
3.8	Establish monitoring and evaluation

Table 8.6: Recommendation 4

Establish and maintain in-service education plan to update teachers on new developments about ADHD and comorbid disorders	
Guideline 4	
4.1	Provide teachers with the latest information on ADHD and other possibilities that impacts on the child data base for researched or evidence based care on all high risk maternal and child health cases
4.2	Document all areas that need development and follow strictly these areas to develop teachers
4.3	Include workshop trainings for teachers which are in line with research

4.4	Provide support to teachers experiencing difficulties and frustration working with ADHD children
4.5	Allow time for consultation and debriefing

Table 8.7: Recommendation 5

Provide parents with psychoeducation on the behavioural symptoms of ADHD	
Guideline 5	
5.1	Target parents during parents meetings and teacher/parent consultation to be vigilent
5.2	Create awareness to parents on ADHD symptoms and its impact on performance and behaviour
5.3	Train parent on how to screen disruptive behaviour.
5.4	Encourage collaborative interaction between parents and teachers towards assisiting the child for effective functioning
5.5	Emphasise on the role of parents involvement as key informants about their children's behaviour
5.6	Create a platform for consultation and support
	Provide consistent monitoring

Table 8.8: Recommendation 6

Request for timeous training for teachers on ADHD related matters	
Guideline 6	
6.1	Train teachers on ADHD related matters such as behavioural and psychological symptoms
6.2	Send teachers for training inorder to be well informed about the new trends of ADHD and other co-existing behavioural problems
6.3	Provide sufficient funding for training skills in schools
6.4	Promote life time learning that include interest in speciality training
6.5	Allow continuous in service post teachers training for curbing pathological behavioural problems at primary level

Table 8.9: Recommendation 7

Provision of information to policy makers with anticipation to improve on current policies	
Guideline 7	
7.1	Establish, observe and maintain principles that accommodates children with differences
7.2	Establish policies which provides flexibility for specialized teaching
7.3	Stipulate policies which protects children with ADHD
7.4	Ensure that children with ADHD receive education from main stream school like any normal children
7.5	Embrace differences and refraim from discrimination of children
7.6	Advocate for children in case of illtreatments
7.7	Revisit the established policies and refine them if there is a need
7.8	Allow constructive and meaningful feedback by teachers and parents
7.9	Provide continuous followup on established policies for effectiveness

Table 8.10: Recommendation 8

Establish more posts for psychological services in schools for assessments	
Guideline 8	
8.1	Provide staff establishment within the department by creating more pobs for Educational psychologists
8.2	Equipe schools with proffessionals to provide psychological services
8.3	Educational psychologists to adopt schools at district level for these services
8.4	Establish school outreach programs
8.5	Encourage collaboration between teachers and children for effective outcomes
8.6	Maximise full support for children who are clinically diagnosed
8.7	Provide support for children undertaking treatment

8.8	Ensure monitoring and evaluation of the process
8.9	Suggest for inclusive education system

Table 8.11: Recommendation 9

Provision of therapeutic intervention which is anticipated to improve care provided by psychologists	
Guideline 9	
9.1	Ensure that clinicians adhere to thourough assessment i.e. assessing for other psychiatric disorders (anxiety and depression) when diagnosing ADHD
9.2	Administer children with both DBD and BYI-II for inclusive assessment
9.3	The most imparing disorder must be attend to first and manage ADHD as a separate disorder
9.4	Assess for the possibility of treatment intake for the child and monitor side effects of the drugs
9.5	Provide tailored therapy intervention depending on the individual needs
9.6	Maintain collaborative interaction between clinician, parent, teacher and child
9.7	Provide personal consultations and support to all members for distress
9.8	Give tasks/activities to the child and followup on goal directed behaviour
9.9	Encourage collaboration between clinicians and educators in problem solving
9.10	Provide contneuous monitoring

Table 8.12: Recommendation 10

Provision of a setting which is expected to improve functionality of the child provided by teachers and parents	
Guideline 10	
10.1	Create a conducive environment which enables learning dispite the clinical diagnosis i.e. at home and school
10.2	Provide educational support for children who are clinically diagnosed and on treatment
10.3	Provide remedial leassons for children with short attention span and allow the child to execute activities themselves
10.4	Provide reinforcement strategies for improvement and good behaviour to nature self esteem (use start charts and use positive words)
10.5	Help chidren to master emotional skills,, build confidence, self regulation, problemsolving and setting boundries
10.6	Labelling and corporal punishment should be prohibited as they may worsen the child's behaviour and diminish their esteem
10.7	Provide academic support and consultation to children
10.8	Give tasks/activities to the child and followup on goal directed behaviour
10.9	Teachers and parents to work hand in hand towards assisting the child for effective functioning
10.10	Provide children with responsibilities that will boost their confidence

8.6 Concluding remarks

In conclusion, the management of comorbid internalising disorders and ADHD require a careful assessment because of the similarities of symptoms. Studies showed that the prognosis and the consequences of children with comorbid ADHD are worse than children with ADHD only (Merikangas et al., 2010). Guidelines will bring awareness to teachers that school children irrespective of their age, do go through emotional turmoil and each time they manifest strange behaviours it can be a warning to act promptly. Therefore,

early identification and intervention of symptoms co-existing with the disorder ADHD are crucial because they will minimise the severity of ADHD.

The management of ADHD and comorbid internalising disorders is therefore a reciprocal process which requires the intervention of a teacher, parent and a clinician towards assisting these children so that they have a meaningfull and successful future. With these guidelines, they will serve as a yardstick to assist teachers and parents through the process of screening children with disruptive behavioural problems. Perhaps teachers will reframe from the misperception that these children are mentally disabled and not fit to be in mainstream school.

Based on these guidelines, several recommendations were identified to bridge the gap. The first recommendation was for the education department to create posts for Educational psychologist at provincial and district level to facilitate assessment and intervention process at primary schools. Secondly, a recommendation was made for policy makers to amend the current policies to accommodate children with neurobehavioural/psychiatric disorders and also to ensure that those policies do not victimise and discriminate against those wiothout ADHD.

Thirdly, a recommendation to psychologists that, caution should be applied when administering DBD as a screening tool when assessing behavioural disorders. The use of a DBD scale by the psychologists should not at all times warrant the diagnosis of ADHD since there may be other mimics of the disorder. It can be concluded that DBD scale should always and at all times be used alongside with the BYI-II scale for the purpose of inclusive assessment. It can be concluded that the assessment and management of ADHD and comorbid disorders require the involment of differnet stakeholder with each having a key role play towards assiting the child to achieve.

CHAPTER 9

SUMMARY, LIMITATIONS, RECOMMENDATIONS AND CONCLUSION

9.1 Introduction

In this final chapter, the focus is on the limitations, recommendations and implications for future research. Conclusion will thereafter be made based on what the study found.

9.2 Summary of the results

The result of the present study can be summarized as follows:

Findings of the present study confirm that children with ADHD have comorbid internalising disorders (anxiety and depression). There is a strong evidence that children with ADHD have more symptoms of anxiety than children without ADHD. Children who participated in the study were clinically diagnosed with ADHD obtained high scores on measures of anxiety when assessed on the BYI-II emotional scale. Comorbid internalising disorder i.e. anxiety was noted within ADHD subtypes particularly within ADHD-PI and ADHD-C, moreover, ADHD symptom inattention was significantly linked to anxiety.

There is also a strong evidence that children with ADHD have more symptoms of depression than children without ADHD. Children who were clinically diagnosed with ADHD, were also found to be having depression when assessed on the BYI-II emotional scale. Comorbid internalising disorder i.e. depression was mostly observed among the ADHD-PI and ADHD-C with females showing more symptoms of depression than males. There is a significant difference that children with ADHD have lower- self-esteem than children without ADHD. All children diagnosed with ADHD had lower levels of self-esteem in particular females more than males. The non ADHD male group showed a higher level of self-esteem.

The study further establishes that internalising comorbidities (anxiety and depression) are more prevalent in girls than in boys who have more externalising comorbidities (Oppositional Defiant Disorder (ODD), Conduct Disorder (CD) and anger issues. The result of the study showed that internalising disorders i.e. anxiety and depression were prevalent in both genders, whereas externalising disorders such as ODD and CD were prevalent in both genders but more pronounced in boys who participated in the study. Externalising behaviour in a form of disruptive behaviour can be observed as acting out behaviours. Studies showed that expression of psychopathology in children can be associated with immature development of cognition and emotion (Patel, Patel & Patel, 2012), suggesting that children are not matured enough to express their emotions and feelings like adults do. Hence, it can be understood that disruptive behaviours in children with ADHD can be alarming hence these children may need further referral in order to be helped.

Results of the current study have observed that ADHD is never in isolation but rather comorbid with other psychiatric disorders. It can then be suggested that a child presenting with multiple disorders have double impairment regardless of their gender difference. Children with ADHD and comorbid disorder continue to suffer in silence and continue to become worse without any form of intervention. In general, the findings of the current study make a strong case that both ADHD, depression and anxiety have overlapping symptoms and are associated with major impairments (Leirbakk, Clench-Aas & Raanaas, 2015). Therefore, in managing ADHD, several comorbid must first be addressed and be managed so that ADHD is managed separately.

9.3 Limitations of the study

- The sample of the current study was too homogeneous. The sample consisted of Sepedi speaking children within the same municipality. This may not represent the populations of South Africa and therefore generalization of the results may be affected.

- No psychometric tests were used to measure the cognitive processing and executive functioning of participants following their diagnosis of ADHD and comorbid internalising disorders. The use of psychometric testing could have assisted in identifying impairments resulting from tasks which measure response inhibition associated with anxiety and specific internalising disorders.

- The sample consisted of more female participants than males. Perhaps a balanced ratio in terms of gender could have yielded different results towards the findings. A larger sample in the future is required for purpose of more information in order to do a comparison. The bigger the sample to yield better results.

- The attitude of parents and learners in responding to questionnaires might have affected the results. Most parents did not give a true reflection of their children's disruptive behaviours due to the fact that their children might be recommended to be placed in a special school.

9.4 Clinical implications

The comorbidity of internalising disorders on ADHD symptoms may lead to devastating outcomes and can complicate the child's functioning and ability to succeed. Many children in primary schools are suspected to have ADHD and comorbid internalising disorders yet remain unidentified with no further intervention provided. The severity of ADHD may cause emotional disorders which further lead to pathological behaviours. Having this comorbid disorders compromise on the child's quality of life and later may resort to risk taking behaviours.

Intensive training to educators about ADHD must be a priority since they observe and interact with these children every day. Parents need to be made aware of the high prevalence of neurodevelopmental comorbidities among school-aged children with ADHD and the impact it will do on their children's functioning. Availing of DBD scales and BYI-II scales should be considered in all primary schools so that if a child is seen to be having problems, a quick screening is done and the child is properly referred.

The present study showed clearly that children with ADHD have high rates of comorbidity, which warrant timely intervention and appropriate treatment methods. It is therefore important that co-occurring disorders are seen as predictors and moderators of treatment outcomes and which treatments work best for whom.

Teacher's attitude towards these children may have an influential role in triggering symptoms of depression. However, ADHD like other disorders, affects educational performance, and therefore teachers should be informed on the impact of ADHD and comorbid disorders occur with ADHD. Furthermore, more knowledge by teachers will

be essential for early identification of these children at a primary phase. Based on the current findings, it can be suggested that emotions are signals and when a child is seen to be depressed immediate referral and intervention should be offered because such symptoms may have a negative impact on the academic ability and success of that child.

Proper screening by professionals by identifying the possibility of other comorbid disorders will be essential to crafting tailored therapeutic intervention suitable for the child. The presentation of ADHD then becomes a gateway to psychiatric disorders which must be addressed earlier to prevent pathologies that may arise as a result of comorbid disorders. Hence, more awareness, and proper training of teachers in identifying and managing children with ADHD should be a priority so that they can provide effective intervention and proper referrals.

Moreover, depression in children is often displayed by irritability and inattention which further leads to the misdiagnosis when in co-occurs with ADHD (Alston, 2011). Depression aggravates the cognitive impairments present in these children. It becomes imperative that assessing ADHD and other suspicious behaviours associated with the disorder be done at an early age to prevent future adverse outcomes that may arise as a result of this disorder.

Disruptive behaviour, noticeable behaviour which is more out of control or wild is commonly expressed. Therefore, teachers should recognise the child's dangerous behavior as alarming and be able to refer for diagnosis and treatment. Having disruptive behaviour can be an additional burden to what these children are already battling with. Disruptive behaviour particularly in classrooms has become a considerable challenge for learning and again a risk factor for students' academic achievement (Narhi, Kiiski, Peitso & Savolainen, 2015; Nash, Schlosser & Scarr, 2016). Besides, a disruptive classroom behavioural climate hampers the learning process and lowers the school performance of the entire class (Blank & Shavit, 2016). Identifying ADHD and disruptive behaviours are crucial as both disorders must be attended separately for the success of these children.

9.5 Clincal implications for theory

Criteria as described in DSM-5 (American Psychiatric Association, 2013) for different psychiatric disorders may produce comorbidity caused by symptom overlap or vagueness in defining a symptom. There are many examples of symptom overlap in DSM-5, for example restlessness is symptomatic both of ADHD and generalised anxiety disorder, and similarly poor concentration is present in depressive disorders as well as in ADHD American Psychiatric Association (2013). Increased motor activity and distractibility are listed in the DSM criteria for both mania as pointed out by Kaiser, Schoemaker, Albaret and Geuze (2015).

Therefore, clinicians may interpret the disorders differently and may be in disagreement on whether a symptom is present or absent and will find it problematic to which diagnostic category to assign the symptoms to. For instance, if a child is irritable, it is due to a major depressive episode, a manic episode, or ODD? (Eyre et al., 2017). Such conflicts will lead to marked differences about the degree to which various disorders are comorbid with each other (Wilens et al., 2009).

The question also arises which disorder is the primary disorder or the comorbidity. If a child has ADHD and anxiety or depression, does the one disorder cause a risk factor for the other? For instance, do children with ADHD develop depression because of demoralisation of dealing with negative feedback from parents, teachers, and peers? In this view, comorbidity results from the primary disorder being an aetiological case for the other (Daviss, 2008).

Comorbidity can be caused by differences in psychosocial as well as biological and genetic factors. The ADHD + comorbidity group may have a differential exposure to major societal stressors such as poverty, crime or exposure to crime or violence. The child with ADHD + comorbidity may have been exposed to certain family experiences not shared by the child with ADHD alone. For example, children with comorbid anxiety may have been exposed to domestic violence or divorce. Biological factors may include differences in genetic markers, brain anatomy, neurochemistry or physiology. Recent studies indicate that this may play a role (Faraone et al., 2015).

A number of studies have looked at whether children with ADHD and comorbidities are different from those with ADHD alone in terms of stimulant responsiveness of the inattention and hyperactivity symptoms. Pliszka (2003) suggested that children with comorbid ADHD and internalising disorders might show a less robust response to stimulant medication than would children with ADHD without internalising disorders. Graham, Banaschewski, Buitelaar, Coghill, Danckaerts, Dittmann, Döpfner, Hamilton, Hollis, Holtmann, & Hulpke-Wette (2011), indicated that only 30% of children with symptoms of depression or anxiety showed improvement on the medication. Further research and guidelines for the use of stimulants for the treatment of ADHD and comorbid internalising disorders are therefore imperative.

Advances in the understanding of the neurobiology of ADHD will provide us with new ways to intervene with children with ADHD and comorbidity which can be seen as difficult patients to treat. When the multifactorial causes of ADHD are fully understood, it will be possible to accurately subtype ADHD on a genetic and/or neurological level. More advanced research in the prevalence and aetiology of of ADHD and its comorbidities may be able to determine if the two (or more) conditions are independent, or genetically linked. Neuroimaging studies and advances in genetics are paving the way to fully understand this complex disorder. The ultimate goal of research in this area is to improve treatment. Children with ADHD and a comorbid psychiatric disorder are among the most challenging patients to treat. There is still gross misunderstanding among the family, medical insurance companies, mental health systems and schools. Thus, the mental health professional feels limited in what he or she can do. Well-informed knowledge about both pharmacological and psychosocial intervention is of the utmost importance.

9.6 Clinical implications for policy makers

The results of the current study showed that children with ADHD and comorbid internalising disorders have difficulties with inatention and impulsivity which in turn impacts negatively in areas of learning. Lack of dopamine has been found to be linked to many behavioural problems seen in these children. All these challenges are attributed to impairments in the doparminegic systems which keep on provoking internalising disorderd (anxiety and depression) which as a results manifest ADHD

symptomatologies (Levy et al., 2004).

Children with ADHD lack reinforcement gradients, hence constant reinforcement by educators is required in order to boost their attention and esteem. This is essential to children with such difficulties because it assists in regulating their behaviour by diminishing unwanted behaviour rather than punishing them, while strenghthening good behaviour (Johansen et al., 2002)

Policies on ADHD and comorbid internalising disorders are silent. The finding of the present study may assist policicy makers in amending the existing policies which will accommodate learners with comorbid disorders from being discriminated and acquire quality education. In addition, the findings may assist policy makers in the development of curriculum which provides training to teachers on behavioural problems affecting children at school. Policy makers will then strengthen that skilled teachers are procduced so than they can be able to educate those children in class who require special attention within the same environment. At the end, there will be a policy which accommodates inclusive education which caters for those with behavioural and emotional problems without any form of discrimination may be developed. Support system through collaboration with other departments such as Department of Health, where community outreach services are extended to schools to assist these children is crucial. Early intervention where these comorbid disorders can be curbed before these children manifest complex behaviours prior their adolescent phase will help.

9.7 Recommendations

- The same study could be done again using a more heterogeneous and diverse sample.
- The use of Teacher/Parent Disruptive Behaviour Disorders Rating Scale should never be used in isolation but together with the Beck Youth Inventory for Children and Adolescents Scale so that an inclusive diagnosis of the actual disorder of the child could be made. This could also assist both clinicians and medical experts make a proper diagnosis.
- Educators should be trained to complete a DBD scale if there is a suspicious behavioural problem about a particular learner and again that learner should complete BYI-II scale. Moreover, both scales should be taken to the hospital through the consent

of a parent/guardian whereby the clinical psychologist and the psychiatrist will intervene further.

- More research in the field of psychology is needed, as the comorbidity of internalising disorders on ADHD is under-researched in South Africa particularly in Limpopo province.

- Screening for the presence of other comorbid disorders when a child is diagnosed with ADHD is important because both ADHD, anxiety and depression may be associated with severe impairments. Early identification of ADHD from early ages is important in order to curb impairments that may arise later due to ADHD

- The Department of Education and the Minister of Education should provide a workshop on ADHD and comorbid disorders for educators.

9.8 Conclusion

In conclusion, the findings of the study show that children with ADHD have comorbid internalising disorders. It is important to understand that ADHD in most cases co-exist with other disorders which in presentation may have similar presentation in terms of symptoms. These symptoms may cause confusion in assessment, diagnosis and even treatment of the actual disorder. Therefore, causing the complexity of the disorder in terms of clinical presentation. This suggests that a child presenting with multiple disorders is at risk of multiple impairments which compromise his/her ability to succeed in life.

Early identification of ADHD at a primary phase is important so that other existing disorders may be dealt with sooner and then focus on pure ADHD as a presenting problem. In this regard, children having ADHD symptoms should be given out most attention and intervention before they can engage in risky behaviours. Research studies have indicated that the comorbidity of internalising disorders may complicate the severity form of the disorder (Rommelse, Fliers, Martin, Buschgens, Hartman, Buitelaar, Faraone, Sergeant & Oosterlaan, 2009; Gillberg, Gillberg, Rasmussen, Kadesjo, Soderstrom, Rastam, Johnson, Rothenberger, & Niklasson, 2004), which is observed by inattention and not being able to pay attention and focus on task that requires their attention.

REFERENCES

Aase, H., & Sagvolden, T. (2005). Moment-to-moment dynamics of ADHD behaviour. *Behavioral and Brain Functions, 1*(1), 12.

Abikoff, H., Gallagher, R., Wells, K. C., Murray, D. W., Huang, L., Lu, F., & Petkova, E. (2013). Remediating organizational functioning in children with ADHD: Immediate and long-term effects from a randomized controlled trial. *Journal of Consulting and Clinical Psychology, 81*(1), 113 - 128.

Achenbach, T. M., Howell, C. T., Quay, H. C., Conners, C. K., & Bates, J. E. (1991). National survey of problems and competencies among four-to sixteen-year-olds: Parents' reports for normative and clinical samples. *Monographs of the Society for Research in Child Development*,56 (3), 1-130.

Adelman, H. S., & Taylor, L. (2010). *Mental health in schools: Engaging learners, preventing problems, and improving schools*. Thousand Oaks, CA, US: Corwin Press.

Adewuya, A. O., & Famuyiwa, O. O. (2007). Attention deficit hyperactivity disorder among Nigerian primary school children: prevalence and co-morbid conditions. *European Child and Adolescent Psychiatry, 16*(1), 10-15.

Airaksinen, E., Larsson, M., & Forsell, Y. (2005). Neuropsychological functions in anxiety disorders in population-based samples: evidence of episodic memory dysfunction. *Journal of Psychiatric Research, 39*(2), 207-214.

Akinsola, H. (2005). Research methods in medical and nursing practice. *Ibadan: Collage Press*

Aldridge, J. W., & Berridge, K. C. (1998). Coding of serial order by neostriatal neurons: a “natural action” approach to movement sequence. *Journal of Neuroscience*, *18*(7), 2777-2787.

Alston, C. S. (2011). *An examination of the relationship between reactive aggression and AD/HD symptomatology among female inmates* (Doctoral dissertation, Capella University).

Andersen, S. L., Thompson, A. T., Rutstein, M., Hostetter, J. C., & Teicher, M. H. (2000). Dopamine receptor pruning in prefrontal cortex during the periadolescent period in rats. *Synapse, 37*(2), 167-169.

Anderson, P. (2002). Assessment and development of executive function (EF) during childhood. *Child Neuropsychology, 8*(2), 71-82.

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 40*(1), 57-87.

Ashenafi, Y., Kebede, D., Desta, M., & Alem, A. (2001). Prevalence of mental and behavioral disorders in children in Ethiopia. *East African Medical Journal, 78*(6), 308-311.

American Psychiatric Association, (2000). Diagnostic and statistic manual of mental disorders IV TR. *Washington, DC: American Psychiatric Association*.

American Psychiatric Association, (2013). *Diagnostic and statistical manual of mental disorders (DSM-5®)*: American Psychiatric Publication.

Averbeck, B. B., Chafee, M. V., Crowe, D. A., & Georgopoulos, A. P. (2002). Parallel processing of serial movements in prefrontal cortex. *Proceedings of the National Academy of Sciences*, *99*(20), 13172-13177.

Bailey, R. K., & Ofoemezie, E. K. (2013). The Impact of Attention Deficit/Hyperactivity Disorder in African-Americans; Current Challenges Associated with Diagnosis and Treatment. In *Attention Deficit Hyperactivity Disorder in Children and Adolescents*: InTech.

Bakare, M. O. (2012). Attention deficit hyperactivity symptoms and disorder (ADHD) among African children: a review of epidemiology and co-morbidities. *African Journal of Psychiatry, 15*(5), 358-361.

Bakare, M. O., Ubochi, V. N., Ebigbo, P. O., & Orovwigho, A. O. (2010). Problem and pro-social behavior among Nigerian children with intellectual disability: the implication for developing policy for school based mental health programs. *Italian Journal of Pediatrics, 36(1)*, 37.

Baker-Ericzén, M. J., Jenkins, M. M., & Haine-Schlagel, R. (2013). Therapist, parent, and youth perspectives of treatment barriers to family-focused community

outpatient mental health services. *Journal of Child and Family Studies*, *22*(6), 854-868.

Baldwin, J. S., & Dadds, M. R. (2008). Examining alternative explanations of the covariation of ADHD and anxiety symptoms in children: a community study. *Journal of Abnormal Child Psychology, 36*(1), 67-79.

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

Banerjee, T. D., Middleton, F., & Faraone, S. V. (2007). Environmental risk factors for attention-deficit hyperactivity disorder. *Acta Paediatrica, 96*(9), 1269-1274.

Bangs, M. E., Emslie, G. J., Spencer, T. J., Ramsey, J. L., Carlson, C., Bartky, E. J., Busner, J., Duesenberg, D. A., Harshawat, P., Kaplan, S. L., Quintana, H., Allen, A. J., & Sumner, C. R. (2007). Efficacy and safety of atomoxetine in adolescents with attention-deficit/hyperactivity disorder and major depression. *Journal of Child and Adolescent Psychopharmacology, 17*(4), 407-420.

Banks, D., & Olszewski, R. (1997). *Estimating local dimensionality.* Paper presented at the Proceedings of the Statistical Computing Section of the American Statistical Society, ASA.

Barber, S., Grubbs, L., & Cottrell, B. (2005). Self-perception in children with attention deficit/hyperactivity disorder. *Journal of Pediatric Nursing, 20*(4), 235-245.

Barbosa, J., Tannock, R., & Manassis, K. (2002). Measuring anxiety: Parent-child reporting differences in clinical samples. *Depression and Anxiety*, *15*(2), 61-65.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Barkley, R. A. (2001). The executive functions and self-regulation: an evolutionary neuropsychological perspective. *Neuropsychology Review, 11*(1), 1-29.

Barkley, R. A. (2004). Adolescents with attention-deficit/hyperactivity disorder: an overview of empirically based treatments. *Journal of Psychiatric Practice, 10*(1), 39-56.

Barkley, R. A. (2006). The relevance of the still lectures to attention-deficit/hyperactivity disorder: a commentary. *Journal of Attention Disorders, 10*(2), 137-140.

Barkley, R. A. (2010). Why emotional impulsiveness should be a central feature of ADHD. *The ADHD Report, 18*(4), 1-5.

Barkley, R. A. (2014). Sluggish cognitive tempo (concentration deficit disorder?): current status, future directions, and a plea to change the name. *Journal of Abnormal Child Psychology, 42*(1), 117-125.

Barkley, R. A., Cook, E. H., Dulcan, M., Campbell, S., Prior, M., Atkins, M., Gillberg, C., Solanto-Gardner, M., Halperin, J., & Bauermeister, J. J. (2002). Consensus statement on ADHD. *European Child and Adolescent Psychiatry*, *11*(2), 96-98.

Barkley, R. A., & Murphy, K. R. (1998). *Attention-Deficit Hyperactivity Disorder: A Clinical Workbook*. New York: Guilford Press.

Barkley, R. A., & Murphy, K. R. (2006). *Attention-deficit Hyperactivity Disorder: A Clinical Workbook*: Guilford Publications.

Barkley, R. A., Murphy, K. R., & Kwasnik, D. (1996). Motor vehicle driving competencies and risks in teens and young adults with attention deficit hyperactivity disorder. *Pediatrics, 98*(6), 1089-1095.

Barkley Russell, A. (2006). Attention-Deficit Hyperactivity Disorder. A Hand book for Diagnosis and Treatment. New York: The Guilford Press.

Bauermeister, J. J., Shrout, P. E., Ramirez, R., Bravo, M., Alegria, M., Martinez-Taboas, A., Chavez, L., Rubio-Stipec, M., Garcia, P., Ribera, J. C., & Canino, G. (2007). ADHD correlates, comorbidity, and impairment in community and treated samples of children and adolescents. *Journal of Abnormal Child Psychology, 35*(6), 883-898.

Beck, J. S., Beck, A. T., Jolly, J. B., & Steer, R. A. (2005). *Beck Youth Inventories Manual: For Children and Adolescents*: Harcourt Assessment, Incorporated.

Beck Schatz, D., & Rostain, A. (2006). ADHD with Comorbid Anxiety: *A Review of the Current Literature.* 10, 141-149.

Bedard, A.-C., & Tannock, R. (2008). Anxiety, methylphenidate response, and working memory in children with ADHD. *Journal of Attention Disorders, 11*(5), 546-557.

Bell, E., Bryman, A., & Harley, B. (2018). *Business research methods*. Oxford university press.

Bennett, D. S., Ambrosini, P. J., Bianchi, M., Barnett, D., Metz, C., & Rabinovich, H. (1997). Relationship of Beck Depression Inventory factors to depression among adolescents. *Journal of Affective Disorders, 45*(3), 127-134.

Biederman, J. (2005). Attention-deficit/hyperactivity disorder: a selective overview. *Biological Psychiatry, 57*(11), 1215-1220.

Biederman, J., Ball, S. W., Monuteaux, M. C., Mick, E., Spencer, T. J., McCreary, M., Cote, M., & Faraone, S. V. (2008). New insights into the comorbidity between ADHD and major depression in adolescent and young adult females. *Journal of the American Academy of Child and Adolescent Psychiatry, 47*(4), 426-434.

Biederman, J., & Faraone, S. V. (2004). The Massachusetts General Hospital studies of gender influences on attention-deficit/hyperactivity disorder in youth and relatives. *The Psychiatric Clinics of North America, 27*(2), 225-232.

Biederman, J., Kwon, A., Aleardi, M., Chouinard, V. A., Marino, T., Cole, H., Mick, E., & Faraone, S. V. (2005). Absence of gender effects on attention deficit hyperactivity disorder: findings in nonreferred subjects. *American Journal of Psychiatry, 162*(6), 1083-1089. doi:10.1176/appi.ajp.

Biederman, J., Mick, E., Faraone, S. V., Braaten, E., Doyle, A., Spencer, T., Wilens, T. E., Frazier, E., & Johnson, M. A. (2002). Influence of gender on attention deficit hyperactivity disorder in children referred to a psychiatric clinic. *American Journal of Psychiatry, 159*(1), 36-42.

Biederman, J., Monuteaux, M. C., Mick, E., Spencer, T., Wilens, T. E., Klein, K. L., Price, J. E., & Faraone, S. V. (2006). Psychopathology in females with attention-deficit/hyperactivity disorder: a controlled, five-year prospective study. *Biological Psychiatry, 60*(10), 1098-1105.

Biederman, J., Petty, C. R., Monuteaux, M. C., Fried, R., Byrne, D., Mirto, T., Spencer, T., Wilens, T. E., & Faraone, S. V. (2010). Adult psychiatric outcomes of girls with attention deficit hyperactivity disorder: 11-year follow-up in a longitudinal case-control study. *American Journal of Psychiatry, 167*(4), 409-417.

Birmaher, B., Axelson, D. A., Monk, K., Kalas, C., Clark, D. B., Ehmann, M., Bridge, J., Heo, J., & Brent, D. A. (2003). Fluoxetine for the treatment of childhood anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(4), 415-423.

Bittner, A., Egger, H. L., Erkanli, A., Jane Costello, E., Foley, D. L., & Angold, A. (2007). What do childhood anxiety disorders predict? *Journal of Child Psychology and Psychiatry, 48*(12), 1174-1183.

Blackman, G. L., Ostrander, R., & Herman, K. C. (2005). Children with ADHD and depression: a multisource, multimethod assessment of clinical, social, and academic functioning. *Journal of Attention Disorders, 8*(4), 195-207.

Blakemore, S. J., & Choudhury, S. (2006). Development of the adolescent brain: implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry, 47*(3-4), 296-312.

Blanche, M. T., Blanche, M. J. T., Durrheim, K., & Painter, D. (Eds.). (2006). *Research in practice: Applied Methods for the SocialSsciences*. Cape Town, Juta and Company Ltd.

Blank, C., & Shavit, Y. (2016). The association between student reports of classmates' disruptive behavior and student achievement. *AERA Open, 2*(3), 2332-8584.

Blascovich, J., & Tomaka, J. (1991). Measures of self-esteem. *Measures of Personality and Social Psychological Attitudes, 1*, 115-160.

Bleck, J. R., DeBate, R. D., & Olivardia, R. (2015). The comorbidity of ADHD and eating disorders in a nationally representative sample. *The Journal of Behavioral Health Services & Research, 42*(4), 437-451.

Bless, C., Higson-Smith, C., & Kagee, A. (2006). *Fundamentals of social research methods : an African perspective*. Cape Town: Juta and Company Ltd

Bloemsma, J. M., Boer, F., Arnold, R., Banaschewski, T., Faraone, S. V., Buitelaar, J. K., Sergeant, J. A., Rommelse, N., & Oosterlaan, J. (2013). Comorbid anxiety and neurocognitive dysfunctions in children with ADHD. *European Child and Adolescent Psychiatry, 22*(4), 225-234.

Bond, D. J., Hadjipavlou, G., Lam, R. W., McIntyre, R. S., Beaulieu, S., Schaffer, A., & Weiss, M. (2012). The Canadian Network for Mood and Anxiety Treatments

(CANMAT) task force recommendations for the management of patients with mood disorders and comorbid attention-deficit/hyperactivity disorder. *Annals of Clinical Psychiatry, 24*(1), 23-37.

Boulet, S. L., Boyle, C. A., & Schieve, L. A. (2009). Health care use and health and functional impact of developmental disabilities among US children, 1997-2005. *Archieves of Pediatrics and Adolescent Medicine, 163*(1), 19-26.

Bowen, R., Chavira, D. A., Bailey, K., Stein, M. T., & Stein, M. B. (2008). Nature of anxiety comorbid with attention deficit hyperactivity disorder in children from a pediatric primary care setting. *Psychiatry Research, 157*(1-3), 201-209.

Bradley, J. D., & Golden, C. J. (2001). Biological contributions to the presentation and understanding of attention-deficit/hyperactivity disorder: a review. *Clinical Psychology Review, 21*(6), 907-929.

Brink, H., Van der Walt, C., & Van Rensburg, G. H. (2012). *Fundamentals of Research Methodology for Healthcare Professionals*: Cape Town, Juta and Company Ltd.

Bron, T. I., Bijlenga, D., Verduijn, J., Penninx, B. W., Beekman, A. T., & Kooij, J. S. (2016). Prevalence of ADHD symptoms across clinical stages of major depressive disorder. *Journal of Affective Disorders, 197*, 29-35.

Brookes, K.-J., Mill, J., Guindalini, C., Curran, S., Xu, X., Knight, J., Chen, C.-K., Huang, Y.-S., Sethna, V., & Taylor, E. (2006). A common haplotype of the dopamine transporter gene associated with attention-deficit/hyperactivity disorder and interacting with maternal use of alcohol during pregnancy. *Archives of General Psychiatry, 63*(1), 74-81.

Brooks Holliday, S., Ewing, B. A., Storholm, E. D., Parast, L., & D'Amico, E. J. (2017). Gender differences in the association between conduct disorder and risky sexual behavior. *Journal of Adolescence, 56*, 75-83.

Brotman, M. A., Schmajuk, M., Rich, B. A., Dickstein, D. P., Guyer, A. E., Costello, E. J., Egger, H. L., Angold, A., Pine, D. S., & Leibenluft, E. (2006). Prevalence, clinical correlates, and longitudinal course of severe mood dysregulation in children. *Biological Psychiatry, 60*(9), 991-997.

Brown, J. (2010). *The world café: Shaping our futures through conversations that matter*: Retrieved from ReadHowYouWant. com.

Brown, T. E. (2000). Attention-deficit disorders and comorbidities in children, adolescents, and adults: American Psychiatric Publishing Press Inc.

Brown, T. E. (2006). Executive functions and attention deficit hyperactivity disorder: Implications of two conflicting views. *International Journal of Disability, Development and Education, 53*(1), 35-46.

Brunsvold, G. L., Oepen, G., Federman, E. J., & Akins, R. (2008). *Comorbid depression and ADHD in children and adolescents: Consensus and controversy* 25, 13-17.

Bryman, A. (2016). *Social Research Methods*: Oxford University Press.

Buitelaar, J. K. (2002). Epidemiological aspects: what have we learned over the last decade. *Hyperactivity and Attention Disorders of Childhood, 2*, 30-63.

Burke, A., & Vorster, A. (2016). Prevalence rates of comorbid disorders in attention deficit hyperactivity disorder adults. *South African Journal of Psychology, 46*(2), 244-253.

Burke, J. D., Hipwell, A. E., & Loeber, R. (2010). Dimensions of oppositional defiant disorder as predictors of depression and conduct disorder in preadolescent girls. *Journal of American Academy of Child and Adolescent Psychiatry, 49*(5), 484-492.

Burke, J. D., Rowe, R., & Boylan, K. (2014). Functional outcomes of child and adolescent oppositional defiant disorder symptoms in young adult men. *Journal of Child Psychology and Psychiatry, 55*(3), 264-272.

Burns, N., Grove, S. K., & Gray, J. (2011). *Understanding Nursing Research. Building an Evidence-Based Practice.* Maryland Heights. *MO:* Elsevier Saunders.

Bush, G. (2011). Cingulate, Frontal and Parietal Cortical Dysfunction in Attention-eficit/Hyperactivity Disorder. *Biological psychiatry, 69*(12), 1160-1167.

Bussing, R., Zima, B. T., & Perwien, A. R. (2000). Self-esteem in special education children with ADHD: relationship to disorder characteristics and medication use. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(10), 1260-1269.

Cadman, T., Findon, J., Eklund, H., Hayward, H., Howley, D., Cheung, C., Kuntsi, J., Glaser, K., Murphy, D., & Asherson, P. (2016). Six-year follow-up study of

combined type ADHD from childhood to young adulthood: Predictors of functional impairment and comorbid symptoms. *European Psychiatry, 35*, 47-54.

Capdevila-Brophy, C., Artigas-Pallarés, J., Blas Navarro-Pastor, J., García-Nonell, K., Rigau-Ratera, E., & Obiols, J. E. (2014). ADHD Predominantly Inattentive Subtype With High Sluggish Cognitive Tempo: A New Clinical Entity? *Journal of Attention Disorders*, *18*(7), 607–616.

Carlson, C. L., & Mann, M. (2002). Sluggish cognitive tempo predicts a different pattern of impairment in the attention deficit hyperactivity disorder, predominantly inattentive type. *Journal of Clinical Child and Adolescent Psychology, 31*(1), 123-129.

Carré, J. M., McCormick, C. M., & Hariri, A. R. (2011). The social neuroendocrinology of human aggression. *Psychoneuroendocrinology, 36*(7), 935-944.

Carter, A. S., Wagmiller, R. J., Gray, S. A., McCarthy, K. J., Horwitz, S. M., & Briggs-Gowan, M. J. (2010). Prevalence of DSM-IV disorder in a representative, healthy birth cohort at school entry: sociodemographic risks and social adaptation. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(7), 686-698.

Casey, B. J., Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Schubert, A. B., Vauss, Y. C., Vaituzis, A. C., Dickstein, D. P., Sarfatti, S. E., & Rapoport, J. L. (1997). Implication of right frontostriatal circuitry in response inhibition and attention-deficit/hyperactivity disorder. *Journal of American Academy of Child and Adolescent Psychiatry, 36*(3), 374-383.

Castagna, P. J., Calamia, M., & Davis III, T. E. (2017). Childhood ADHD and Negative Self-Statements: Important Differences Associated With Subtype and Anxiety Symptoms. *Behavior Therapy, 48*(6), 793-807.

Castellanos, F. X. (1997). Toward a pathophysiology of attention-deficit/hyperactivity disorder. *Clinical Pediatrics (Phila), 36*(7), 381-393.

Castellanos, F. X., & Acosta, M. T. (2004). [The neuroanatomy of attention deficit/hyperactivity disorder]. *Revista de Neurologic, 38 Suppl 1*, 131-136.

Castellanos, F. X., Giedd, J. N., Berquin, P. C., Walter, J. M., Sharp, W., Tran, T., Vaituzis, A. C., Blumenthal, J. D., Nelson, J., Bastain, T. M., Zijdenbos, A., Evans, A. C., & Rapoport, J. L. (2001). Quantitative brain magnetic resonance imaging in girls with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 58*(3), 289-295.

Castellanos, F. X., Giedd, J. N., Marsh, W. L., Hamburger, S. D., Vaituzis, A. C., Dickstein, D. P., Sarfatti, S. E., Vauss, Y. C., Snell, J. W., & Lange, N. (1996). Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. *Archives of General Psychiatry, 53*(7), 607-616.

Castellanos, F. X., Lee, P. P., Sharp, W., Jeffries, N. O., Greenstein, D. K., Clasen, L. S., Blumenthal, J. D., James, R. S., Ebens, C. L., Walter, J. M., Zijdenbos, A., Evans, A. C., Giedd, J. N., & Rapoport, J. L. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *Journal of Amerrican Medical Association, 288*(14), 1740-1748.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry*, *60*(8), 837-844.

Castellanos, F. X., Sonuga-Barke, E. J., Scheres, A., Di Martino, A., Hyde, C., & Walters, J. R. (2005). Varieties of attention-deficit/hyperactivity disorder-related intra-individual variability. *Biological Psychiatry, 57*, 146-1423.

Catania, A. C. Learning. (1999). Learning 4 edition Prentice Hall *NJ, Englewoods Cliffs:*

Chinawa, J.M., Odetunde, O.I., Obu, H.A., Chinawa, A.T., Bakare, M.O., & Ujunwa, F.A (2014). Attention Deficit Hyperactivity Disorder: a neglected issue in the developing world. *Behaioural Neurology*, 2014. 1-6.

Chronis-Tuscano, A., Molina, B. S., Pelham, W. E., Applegate, B., Dahlke, A., Overmyer, M., & Lahey, B. B. (2010). Very early predictors of adolescent depression and suicide attempts in children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 67*(10), 1044-1051.

Coghill, D., & Banaschewski, T. (2009). The genetics of attention-deficit/hyperactivity disorder. *Expert Review of Neurotherapeutics, 9*(10), 1547-1565.

Coghill, D., Nigg, J., Rothenberger, A., Sonuga-Barke, E., & Tannock, R. (2005). Whither causal models in the neuroscience of ADHD? *Developmental science*, *8*(2), 105-114.

Coles, E. K., Slavec, J., Bernstein, M., & Baroni, E. (2012). Exploring the gender gap in referrals for children with ADHD and other disruptive behavior disorders. *Journal of Attention Disorders, 16*(2), 101-108.

Comings, D. E., Chen, T. J. H., Blum, K., Mengucci, J. F., Blum, S. H., & Meshkin, B. (2005). Neurogenetic interactions and aberrant behavioral co-morbidity of attention deficit hyperactivity disorder (ADHD): dispelling myths. *Theoretical Biology and Medical Modelling, 2(1)*, 50.

Conners, C. K. (2000). Attention-deficit/hyperactivity disorder—historical development and overview. *Journal of Attention Disorders*, 3(4), 173-191.

Connor, D. F., Chartier, K. G., Preen, E. C., & Kaplan, R. F. (2010). Impulsive aggression in attention-deficit/hyperactivity disorder: symptom severity, co-morbidity, and attention-deficit/hyperactivity disorder subtype. *Journal of Child and Adolescent Psychopharmacology, 20*(2), 119-126.

Connor, D. F., & Doerfler, L. A. (2009). Attention-deficit/hyperactivity disorder and comorbid oppositional defiant disorder or conduct disorder. *Current Attention Disorders Reports, 1*(1), 5-11.

Connor, D. F., Edwards, G., Fletcher, K. E., Baird, J., Barkley, R. A., & Steingard, R. J. (2003). Correlates of comorbid psychopathology in children with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(2), 193-200.

Connor, D. F., Glatt, S. J., Lopez, I. D., Jackson, D., & Melloni, R. H., Jr. (2002). Psychopharmacology and aggression. I: A meta-analysis of stimulant effects on overt/covert aggression-related behaviors in ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 41*(3), 253-261.

Cook, J., Knight, E., Hume, I., & Qureshi, A. (2014). The self-esteem of adults diagnosed with attention-deficit/hyperactivity disorder (ADHD): a systematic review of the literature. *ADHD Attention Deficit and Hyperactivity Disorders, 6*(4), 249-268.

Copeland, W. E., Angold, A., Costello, E. J., & Egger, H. (2013). Prevalence, comorbidity, and correlates of DSM-5 proposed disruptive mood dysregulation disorder. *American Journal of Psychiatry, 170*(2), 173-179.

Cortese, S. (2012). The neurobiology and genetics of attention-deficit/hyperactivity disorder (ADHD): what every clinician should know. *European Journal of Paediatric Neurology, 16*(5), 422-433.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60*(8), 837-844.

Costin, J., Vance, A., Barnett, R., O'shea, M., & Luk, E. S. (2002). Attention deficit hyperactivity disorder and comorbid anxiety: Practitioner problems in treatment planning. *Child and Adolescent Mental Health, 7*(1), 16-24.

Counts, C. A., Nigg, J. T., Stawicki, J. A., Rappley, M. D., & Von Eye, A. (2005). Family adversity in DSM-IV ADHD combined and inattentive subtypes and associated disruptive behavior problems. *Journal of the American Academy of Child & Adolescent Psychiatry, 44*(7), 690-698.

Courchesne, E., Chisum, H. J., Townsend, J., Cowles, A., Covington, J., Egaas, B., Harwood, M., Hinds, S., & Press, G. A. (2000). Normal brain development and aging: quantitative analysis at in vivo MR imaging in healthy volunteers. *Radiology, 216*(3), 672-682.

Creswell, J. W., & Poth, C. N. (2016). *Qualitative Inquiry and Research Design: Choosing Among Five Approaches*: SAGE

Crystal, D. S., Ostrander, R., San Chen, R., & August, G. J. (2001). Multimethod assessment of psychopathology among DSM-IV subtypes of children with attention-deficit/hyperactivity disorder: Self-, parent, and teacher reports. *Journal of Abnormal Child Psychology, 29*(3), 189-205.

Cueli, M., González-Castro, P., Álvarez, L., Núñez, J. C., & Fernández, E. (2014). EPA-0384 – Anxiety and selective attention in adhd subtypes. *European Psychiatry, 29*, 1.

Cunningham, C. E., & Boyle, M. H. (2002). Preschoolers at risk for attention-deficit hyperactivity disorder and oppositional defiant disorder: family, parenting, and behavioral correlates. *Journal of Abnormal Child Psychology, 30*(6), 555-569.

Daley, D., van der Oord, S., Ferrin, M., Danckaerts, M., Doepfner, M., Cortese, S., & Sonuga-Barke, E. J. (2014). Behavioral interventions in attention-deficit/hyperactivity disorder: a meta-analysis of randomized controlled trials across multiple outcome domains. *Journal of American Academy of Child and Adolescent Psychiatry, 53*(8), 835-847.

Demaray, M. K., & Elliot, S. N. (2001). Perceived social support by children with characteristics of attention-deficit/hyperactivity disorder. *School Psychology Quarterly*, 16, 68 – 90.

Dan, O., & Raz, S. (2015). The relationships among ADHD, self-esteem, and test anxiety in young adults. *Journal of Attention Disorders, 19*(3), 231-239.

Danckaerts, M., Sonuga-Barke, E. J., Banaschewski, T., Buitelaar, J., Döpfner, M., Hollis, C., Santosh, P., Rothenberger, A., Sergeant, J., & Steinhausen, H.-C. (2010). The quality of life of children with attention deficit/hyperactivity disorder: a systematic review. *European Child and Adolescent Psychiatry, 19*(2), 83-105.

Daneshparvar, M., Mostafavi, S. A., Zare Jeddi, M., Yunesian, M., Mesdaghinia, A., Mahvi, A. H., & Akhondzadeh, S. (2016). The Role of Lead Exposure on Attention-Deficit/ Hyperactivity Disorder in Children: A Systematic Review. *Iranian Journal of Psychiatry, 11*(1), 1-14.

Daviss, W. B. (2008). A review of co-morbid depression in pediatric ADHD: etiologies, phenomenology, and treatment. *Journal of Child and Adolescent Psychopharmacology, 18*(6), 565-571.

de Zubicaray, G. I., Zelaya, F. O., Andrew, C., Williams, S. C., & Bullmore, E. T. (2000). Cerebral regions associated with verbal response initiation, suppression and strategy use. *Neuropsychologia, 38*(9), 1292-1304.

del Campo, N., Fryer, T. D., Hong, Y. T., Smith, R., Brichard, L., Acosta-Cabronero, J., Chamberlain, S. R., Tait, R., Izquierdo, D., Regenthal, R., Dowson, J., Suckling, J., Baron, J. C., Aigbirhio, F. I., Robbins, T. W., Sahakian, B. J., & Muller, U. (2013). A positron emission tomography study of nigro-striatal dopaminergic

mechanisms underlying attention: implications for ADHD and its treatment. *Brain, 136* (11), 3252-3270.

Dell'Osso, B., & Ketter, T. A. (2013). Assessing efficacy/effectiveness and safety/tolerability profiles of adjunctive pramipexole in bipolar depression: acute versus long-term data. *International Clinical Psychopharmacology, 28*(6), 297-304.

Demaray, M. K., & Elliott, S. N. (2001). Perceived social support by children with characteristics of Attention-Deficit/Hyperactivity Disorder. *School Psychology Quarterly, 16*(1), 68.

Depue, B. E., Burgess, G. C., Bidwell, L. C., Willcutt, E. G., & Banich, M. T. (2010). Behavioral performance predicts grey matter reductions in the right inferior frontal gyrus in young adults with combined type ADHD. *Psychiatry Research: Neuroimaging, 182*(3), 231-237.

Depue, B. E., Burgess, G. C., Willcutt, E. G., Bidwell, L. C., Ruzic, L., & Banich, M. T. (2010). Symptom-correlated brain regions in young adults with combined-type ADHD: Their organization, variability, and relation to behavioral performance. *Psychiatry Research, 182*(2), 96-102.

Derks, E. M., Hudziak, J. J., & Boomsma, D. I. (2007). Why more boys than girls with ADHD receive treatment: a study of Dutch twins. *Twin Research and Human Genetics, 10*(5), 765-770.

Di Trani, M., Di Roma, F., Elda, A., Daniela, L., Pasquale, P., Silvia, M., & Renato, D. (2014). Comorbid depressive disorders in ADHD: the role of ADHD severity, subtypes and familial psychiatric disorders. *Psychiatry Investigation, 11*(2), 137-142.

Dick, D. M., Viken, R. J., Kaprio, J., Pulkkinen, L., & Rose, R. J. (2005). Understanding the covariation among childhood externalizing symptoms: genetic and environmental influences on conduct disorder, attention deficit hyperactivity disorder, and oppositional defiant disorder symptoms. *Journal of Abnormal Child Psychology, 33*(2), 219-229.

Dickstein, D. P., Milham, M. P., Nugent, A. C., Drevets, W.C., Charney, D.S., Pine, D.S. and Leibenluft, E. (2005). Frontotemporal alterations in pediatric bipolar disorder: Results of a voxel-based morphometry study. *Archives of General Psychiatry, 62*(7), 734-741.

Diler, R. S., Uguz, S., Seydaoglu, G., Erol, N., & Avci, A. (2007). Differentiating bipolar disorder in Turkish prepubertal children with attention-deficit hyperactivity disorder. *Bipolar Disorders, 9*(3), 243-251.

Dirlikov, B., Rosch, S.K., Crocetti, D., Denckla, M. B., Mahone, E. M., & Mostofsky, S. H. (2015). Distinct frontal lobe morphology in girls and boys with ADHD. *NeuroImage : Clinical, 7*, 222-229.

Donnelly, C., Bangs, M., Trzepacz, P., Jin, L., Zhang, S., Witte, M. M., Ball, S. G., & Spencer, T. J. (2009). Safety and tolerability of atomoxetine over 3 to 4 years in children and adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 48*(2), 176-185.

Douglas, V. I. (1999). Cognitive control processes in attention deficit/hyperactivity disorder. In *Handbook of disruptive behavior disorders* (pp. 105-138). Springer, Boston, MA.

DuPaul, G. J., Eckert, T. L., & Vilardo, B. (2012). The effects of school-based interventions for attention deficit hyperactivity disorder: A meta-analysis 1996-2010. *School Psychology Review, 41*(4), 387.

Durston, S., Hulshoff Pol, H. E., Schnack, H. G., Buitelaar, J. K., Steenhuis, M. P., Minderaa, R. B., Kahn, R. S., & van Engeland, H. (2004). Magnetic resonance imaging of boys with attention-deficit/hyperactivity disorder and their unaffected siblings. *Journal of the American Academy of Child and Adolescent Psychiatry, 43*(3), 332-340.

Durston, S., Tottenham, N. T., Thomas, K. M., Davidson, M. C., Eigsti, I. M., Yang, Y., Ulug, A. M., & Casey, B. J. (2003). Differential patterns of striatal activation in young children with and without ADHD. *Biological Psychiatry, 53*(10), 871-878.

Dwivedi, K. N., & Banhatti, R. G. (2005). Attention deficit/hyperactivity disorder and ethnicity. *Archives of Disease in Childhood, 90 (1)*, 10-12.

Edbom, T., Granlund, M., Lichtenstein, P., & Larsson, J. O. (2008). ADHD Symptoms Related to Profiles of Self-Esteem in a Longitudinal Study of Twins A person-oriented approach. *Journal of Child and Adolescent Psychiatric Nursing, 21*(4), 228-237.

Ek, U., Westerlund, J., Holmberg, K., & Fernell, E. (2008). Self-esteem in children with attention and/or learning deficits: the importance of gender. *Acta Paediatrica, 97*(8), 1125-1130.

Elia, J., Ambrosini, P., & Berrettini, W. (2008). ADHD characteristics: I. Concurrent co-morbidity patterns in children & adolescents. *Child and Adolescent Psychiatry and Mental Health, 2*(1), 15.

Emilsson, M., Gustafsson, P. A., Öhnström, G., & Marteinsdottir, I. (2017). Beliefs regarding medication and side effects influence treatment adherence in adolescents with attention deficit hyperactivity disorder. *European Child and Adolescent Psychiatry, 26*(5), 559-571.

Erkolahti, R. K., Saarijärvi, S., Ilonen, T., & Hagman, H. (2002). Self-image of anorexic and bulimic female adolescents. *Nordic Journal of Psychiatry, 56*(6), 447-450.

Erşan, E. E., Doğan, O., Doğan, S., & Sümer, H. (2004). The distribution of symptoms of attention-deficit/hyperactivity disorder and oppositional defiant disorder in school age children in Turkey. *European Child and Adolescent Psychiatry, 13*(6), 354-361.

Eriskine, H.E., Ferrari, A.J., Nelson, P., Polanczyk, G.V, Flaxman, A.D., Vos, T., Harvey, A.W., Scott, J.G. (2013). Research Review: Epidemiological modelling of Attention-Deficit/Hyperactivity Disorder and Conduct Disorder for the Global Burden of Disease Study 2010. Journal of Child Psychology and Psychiatry, 54(12), 1263-1274.

Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry, 164*(10), 1476-1488.

Evans, S. W., Owens, J. S., & Bunford, N. (2014). Evidence-based psychosocial treatments for children and adolescents with attention-deficit/hyperactivity disorder. *Journal of Clinical Child & Adolescent Psychology*, *43*(4), 527-551.

Eyre, O., Langley, K., Stringaris, A., Leibenluft, E., Collishaw, S., & Thapar, A. (2017). Irritability in ADHD: Associations with depression liability. *Journal of Affective Disorders, 215*, 281-287.

Faraone, S. V., Asherson, P., Banaschewski, T., Biederman, J., Buitelaar, J. K., Ramos-Quiroga, J. A., Rohde, L. A., Sonuga-Barke, E. J., Tannock, R., & Franke, B. (2015). Attention-deficit/hyperactivity disorder. *Nature Reviews Disease Primers, 1*,Article number 15020.

Faraone, S. V., Biederman, J., & Friedman, D. (2000). Validity of DSM-IV subtypes of attention-deficit/hyperactivity disorder: a family study perspective. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*(3), 300-307.

Faraone, S. V., Biederman, J., Monuteaux, M., & Spencer, T. (2005). Long-term effects of extended-release mixed amphetamine salts treatment of attention-deficit/hyperactivity disorder on growth. *Journal of Child & Adolescent Psychopharmacology, 15*(2), 191-202.

Faraone, S. V., & Larsson, H. (2019). Genetics of attention deficit hyperactivity disorder. *Molecular Psychiatry*, 24(4), 562-575.

Faraone, S. V., & Mick, E. (2010). Molecular genetics of attention deficit hyperactivity disorder. *Psychiatric Clinics of North America, 33*(1), 159-180.

Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., & Sklar, P. (2005). Molecular genetics of attention-deficit/hyperactivity disorder. *Biological Psychiatry, 57*(11), 1313-1323.

Farb, D. H., & Ratner, M. H. (2014). Targeting the modulation of neural circuitry for the treatment of anxiety disorders. *Pharmacological Reviews*, *66*(4), 1002-1032.

Filipek, P. A., Semrud-Clikeman, M., Steingard, R. J., Renshaw, P. F., Kennedy, D. N., & Biederman, J. (1997). Volumetric MRI analysis comparing subjects having attention-deficit hyperactivity disorder with normal controls. *Neurology, 48*(3), 589-601.

Finkelstein, J., & Goodwin, S. (2005). *The Sociological bent: Inside Metro Culture*. Southbank: Thomson.

Fischer, A. G., Bau, C. H., Grevet, E. H., Salgado, C. A., Victor, M. M., Kalil, K. L., Sousa, N. O., Garcia, C. R., & Belmonte-de-Abreu, P. (2007). The role of

comorbid major depressive disorder in the clinical presentation of adult ADHD. *Journal of Psychiatric Research, 41*(12), 991-996.

Foley-Nicpon, M., Rickels, H., Assouline, S. G., & Richards, A. (2012). Self-esteem and self-concept examination among gifted students with ADHD. *Journal for the Education of the Gifted, 35*(3), 220-240.

Frankel, F., Cantwell, D. P., Myatt, R., & Feinberg, D. T. (1999). Do stimulants improve self-esteem in children with ADHD and peer problems? *Journal of Child and Adolescent Psychopharmacology, 9*(3), 185-194.

Freeman, R. D. (2007). Tic disorders and ADHD: answers from a world-wide clinical dataset on Tourette syndrome. *European Child and Adolescent Psychiatry,* 16 Suppl 1, 15-23.

Fu, K. M., Foxe, J. J., Murray, M. M., Higgins, B. A., Javitt, D. C., & Schroeder, C. E. (2001). Attention-dependent suppression of distracter visual input can be cross-modally cued as indexed by anticipatory parieto-occipital alpha-band oscillations. *Brain Research Cognitive Brain Research, 12*(1), 145-152.

Furman, L. (2005). What is attention-deficit hyperactivity disorder (ADHD)? *Journal of Child Neurology, 20*(12), 994-1002.

Gabrielle, L. B., Rick, O., & Keith, C. H. (2005). Children with ADHD and Depression: A Multisource, Multimethod Assessment of Clinical, Social, and Academic Functioning. *Journal of Attention Disorders, 8*(4), 195-207.

Garralda, M. E. (1996). Somatisation in children. *Journal of Child Psychology and Psychiatry, 37*(1), 13-33.

Gaub, M., & Carlson, C. L. (1997). Behavioral characteristics of DSM-IV ADHD subtypes in a school-based population. *Journal of Abnormal Child Psychology, 25*(2), 103-111.

Geller, D., Donnelly, C., Lopez, F., Rubin, R., Newcorn, J., Sutton, V., Bakken, R., Paczkowski, M., Kelsey, D., & Sumner, C. (2007). Atomoxetine treatment for pediatric patients with attention-deficit/hyperactivity disorder with comorbid anxiety disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 46*(9), 1119-1127.

Germania, B., Eppinger, M.A., Mostofsky, S.H., DiCicco-Bloom, E., & MaRIA, B.L. (2015). Recent advances in understanding and managing autism spectrum disorders. *Journal of Child Neurology*, 30(14), 1887-1920.

Gershon, J. (2002). A meta-analytic review of gender differences in ADHD. *Journal of Attention Disorders, 5*(3), 143-154.

Giedd, J., Blumenthal, J., O. Jeffries, N., Castellanos, F., Liu, H., Zijdenbos, A., Paus, T., Evans, A., & Rapoport, J. (1999). *Brain Development during Childhood and Adolescence: A Longitudinal MRI Study,* 2, 861-863.

Giedd, J. N., Rumsey, J. M., Castellanos, F. X., Rajapakse, J. C., Kaysen, D., Vaituzis, A. C., Vauss, Y. C., Hamburger, S. D., & Rapoport, J. L. (1996). A quantitative MRI study of the corpus callosum in children and adolescents. *Developmental Brain Research, 91*(2), 274-280.

Giedd, J. N., Snell, J. W., Lange, N., Rajapakse, J. C., Casey, B. J., Kozuch, P. L., Vaituzis, A. C., Vauss, Y. C., Hamburger, S. D., Kaysen, D., & Rapoport, J. L. (1996). Quantitative magnetic resonance imaging of human brain development: ages 4-18. *Cerebral Cortex, 6*(4), 551-560.

Gillberg, C. (2003). Deficits in attention, motor control, and perception: a brief review. *Archives of Disease in Childhood, 88*(10), 904-910.

Gillberg, C., Gillberg, I. C., Rasmussen, P., Kadesjo, B., Soderstrom, H., Rastam, M., Johnson, M., Rothenberger, A., & Niklasson, L. (2004). Co-existing disorders in ADHD -- implications for diagnosis and intervention. *European Child and Adolescent Psychiatry, 13(1)*, I80-92.

Glass, K., Flory, K., Martin, A., & Hankin, B. L. (2011). ADHD and comorbid conduct problems among adolescents: associations with self-esteem and substance use. *ADHD Attention Deficit and Hyperactivity Disorders, 3*(1), 29-39.

Golafshani, N. (2003). Understanding reliability and validity in qualitative research. *The Qualitative Report*, *8*(4), 597-606.

Goodwin, S., & Finkelstein, J. (2005). *The sociological bent*. Amazon, Cengage Learning Australia.

Gonser, T. H. (2016). *System and method for document tagging templates. United.States. Patent No. 9,514,117*. Washington, DC: U.S. Patent and Trademark Office.

Graetz, B. W., Sawyer, M. G., & Baghurst, P. (2005). Gender differences among children with DSM-IV ADHD in Australia. *Journal of the American Academy of Child and Adolescent Psychiatry, 44*(2), 159-168.

Graetz, B. W., Sawyer, M. G., Hazell, P. L., Arney, F., & Baghurst, P. (2001). Validity of DSM-IV ADHD subtypes in a nationally representative sample of Australian children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(12), 1410-1417.

Graham, J., Banaschewski, T., Buitelaar, J., Coghill, D., Danckaerts, M., Dittmann, R. W, Döpfner, M., Hamilton, R., Hollis, C., Holtmann, & Hulpke-Wette, M. (2011). European guidelines on managing adverse effects of medication for ADHD. *European Child & Adolescent Psychiatry*, *20*(1), 17-37.

Gross-Tsur, V., Goldzweig, G., Landau, Y. E., Berger, I., Shmueli, D., & Shalev, R. S. (2006). The impact of sex and subtypes on cognitive and psychosocial aspects of ADHD. *Developmental Medicine and Child Neurology, 48*(11), 901-905.

Hahlweg, K., Heinrichs, N., Kuschel, A., Bertram, H., & Naumann, S. (2010). Long-term outcome of a randomized controlled universal prevention trial through a positive parenting program: is it worth the effort?. *Child and Adolescent Psychiatry and Mental Health*, *4*(1), 14.

Halldorsdottir, T., & Ollendick, T. H. (2014). Comorbid ADHD: Implications for the treatment of anxiety disorders in children and adolescents. *Cognitive and Behavioral Practice, 21*(3), 310-322.

Hammerness, P., Geller, D., Petty, C., Lamb, A., Bristol, E., & Biederman, J. (2010). Does ADHD moderate the manifestation of anxiety disorders in children? *European Child and Adolescent Psychiatry, 19*(2), 107-112.

Harpin, V., Mazzone, L., Raynaud, J. P., Kahle, J., & Hodgkins, P. (2016). Long-Term Outcomes of ADHD: A Systematic Review of Self-Esteem and Social Function. *Journal of Attention Disorders, 20*(4), 295-305.

Harpin, V. A. (2005). The effect of ADHD on the life of an individual, their family, and community from preschool to adult life. *Archives of Disease in Childhood, 90(1)*, 2-7.

Harris, A. N., Stoppelbein, L., Greening, L., Becker, S. P., Luebbe, A., & Fite, P. (2014). Child routines and parental adjustment as correlates of internalizing and externalizing symptoms in children diagnosed with ADHD. *Child Psychiatry & Human Development*, *45*(2), 243-253.

Harter, S., & Jackson, B. K. (1993). Young Adolescents' Perceptions of the Link between Low Self-Worth and Depressed Affect. *The Journal of Early Adolescence, 13*(4), 383-407.

Hassan, G. A., Eissa, A. M., Hwedi, D., Hegazy, D., & Essam, A. (2013). Children with attention deficit-hyperactivity disorder and comorbid depression: a descriptive study. *Middle East Current Psychiatry, 20*(3), 140-145.

Hay, D., McStephen, M., & Levy, F. (2001). *The diagnostic genetics of ADHD symptoms and subtypes. Attention genes and ADHD.* Hove: Brunner-Routledge.

Heal, D. J., Smith, S. L., Gosden, J., & Nutt, D. J. (2013). Amphetamine, past and present–a pharmacological and clinical perspective. *Journal of Psychopharmacology, 27*(6), 479-496.

Hegerl, U., & Hensch, T. (2017). Why do stimulants not work in typical depression? *Australian & New Zealand Journal of Psychiatry, 51*(1), 20-22.

Herman, K. C., Lambert, S. F., Reinke, W. M., & Ialongo, N. S. (2008). Low academic competence in first grade as a risk factor for depressive cognitions and symptoms in middle school. *Journal of Counseling Psychology, 55*(3), 400-410.

Hesslinger, B., Tebartz van Elst, L., Thiel, T., Haegele, K., Hennig, J., & Ebert, D. (2002). Frontoorbital volume reductions in adult patients with attention deficit hyperactivity disorder. *Neuroscience Letter, 328*(3), 319-321.

Hill, D. E., Yeo, R. A., Campbell, R. A., Hart, B., Vigil, J., & Brooks, W. (2003). Magnetic resonance imaging correlates of attention-deficit/hyperactivity disorder in children. *Neuropsychology, 17*(3), 496-506.

Hinshaw, S. P., Owens, E. B., Sami, N., & Fargeon, S. (2006). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into adolescence: Evidence

for continuing cross-domain impairment. *Journal of Consulting and Clinical Psychology, 74*(3), 489-499.

Hinshaw, S. P., Zupan, B. A., Simmel, C., Nigg, J. T., & Melnick, S. (1997). Peer status in boys with and without attention-deficit hyperactivity disorder: Predictions from overt and covert antisocial behavior, social isolation, and authoritative parenting beliefs. *Child Development, 68*(5), 880-896.

Hodgens, J. B., Cole, J., & Boldizar, J. (2000). Peer-based differences among boys with ADHD. *Journal of Clinical Child Psychology, 29*(3), 443-452.

Hoza, B., Gerdes, A. C., Hinshaw, S. P., Arnold, L. E., Pelham Jr, W. E., Molina, B. S., Abikoff, H. B., Epstein, J. N., Greenhill, L. L., & Hechtman, L. (2004). Self-perceptions of competence in children with ADHD and comparison children. *Journal of Consulting and Clinical Psychology, 72*(3), 382.

Hoza, B., Pelham, W. E., Milich, R., Pillow, D., & McBride, K. (1993). The self-perceptions and attributions of attention deficit hyperactivity disordered and nonreferred boys. *Journal of Abnormal Child Psychology*, *21*(3), 271-286.

Huberty, T. J. (2004). Anxiety and anxiety disorders in children: Information for parents. *National Association of School Psychologist. Indiana University, 301*, S51-S54.

Humphreys, K. L., Katz, S. J., Lee, S. S., Hammen, C., Brennan, P. A., & Najman, J. M. (2013). The association of ADHD and depression: Mediation by peer problems and parent–child difficulties in two complementary samples. *Journal of Abnormal Psychology*, *122*(3), 854.

Hurtig, T., Ebeling, H., Taanila, A., Miettunen, J., Smalley, S. L., McGOUGH, J. J., Loo, S. K., JÄrvelin, M.-r., & Moilanen, I. K. (2007). ADHD symptoms and subtypes: relationship between childhood and adolescent symptoms. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*(12), 1605-1613.

Ibukun, A.I., Oluyemi, O., Abidemi, B., Suraj, A., & Ola, F. (2015). Literacy about Attention-Deficit/Hyperactivity Disoarder among primary school teachers in Lagos, Nigeria. *British Journal of Medicine and Medical Research*, 8(8), 684-691.

In-Albon, T. (2012). State of Research on Internalizing Disorders in Children and Adolescents: Is It Still in Its Infancy? *Verhaltenstherapie,* 22, 246-257.

Jackson, D. A., & King, A. R. (2004). Gender differences in the effects of oppositional behavior on teacher ratings of ADHD symptoms. *Journal of Abnormal Child Psychology, 32*(2), 215-224.

Janet, C., Alasdair, V., Rebbeca, B., Melissa, O. S., & L., L. E. S. (2002). Attention Deficit Hyperactivity Disorder and Comorbid Anxiety: Practitioner Problems in Treatment Planning. *Child and Adolescent Mental Health, 7*(1), 16-24.

Jarrett, M. A., Wolff, J. C., Thompson E. Davis, I., Cowart, M. J., & Ollendick, T. H. (2016). Characteristics of Children With ADHD and Comorbid Anxiety. *Journal of Attention Disorders, 20*(7), 636-644.

Jellinek, M. S. (2010). Don't Let ADHD Crush Children's Self-Esteem. *Clinical Psychiatry News, 38*(5), 12-12.

Jensen, P. S. (2009). Clinical considerations for the diagnosis and treatment of ADHD in the managed care setting. *American Journal of Managed Care, 15*(5 Suppl), S129-140.

Jensen, P. S., Hinshaw, S. P., Kraemer, H. C., Lenora, N., Newcorn, J. H., Abikoff, H. B., March, J. S., Arnold, L. E., Cantwell, D. P., & Conners, C. K. (2001). ADHD comorbidity findings from the MTA study: comparing comorbid subgroups. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(2), 147-158.

Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: implications for research, practice, and DSM-V. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(8), 1065-1079.

Johansen, E. B., Aase, H., Meyer, A., & Sagvolden, T. (2002). Attention-deficit/hyperactivity disorder (ADHD) behaviour explained by dysfunctioning reinforcement and extinction processes. *Behavioural Brain Research, 130*(1-2), 37-45.

Johansen, E. B., Killeen, P. R., Russell, V. A., Tripp, G., Wickens, J. R., Tannock, R., Williams, J., & Sagvolden, T. (2009). Origins of altered reinforcement effects in ADHD. *Behavioral and Brain Functions, 5(1)*, 7.

Johansen, E. B., Sagvolden, T., Aase, H., & Russell, V. A. (2005). The dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD): Present status and future perspectives. *Behavioural Brain Science,* 28(3), 397-418.

Johnston, C., & Mash, E. J. (2001). Families of children with attention-deficit/hyperactivity disorder: review and recommendations for future research. *Clinical Child and Family Psychology Review, 4*(3), 183-207.

Johnston, C., & Park, J. L. (2015). Interventions for Attention-Deficit Hyperactivity Disorder: A Year in Review. *Current Developmental Disorders Reports, 2*(1), 38-45.

Jones, J.E., Siddartrh, P., Almane, D., Gurbani, S., Hermann, B.P., & Caplan, R. (2016). Identification of risk for severe psychiatric comorbidity in pediatric epilepsy. *Epilepsia,* 57(11), 1817-1825.

Kadesjö, C., Hägglöf, B., Kadesjö, B., & Gillberg, C. (2003). Attention-deficit–hyperactivity disorder with and without oppositional defiant disorder in 3-to 7-year-old children. *Developmental Medicine and Child Neurology, 45*(10), 693-699.

Kahn, R. S., Khoury, J., Nichols, W. C., & Lanphear, B. P. (2003). Role of dopamine transporter genotype and maternal prenatal smoking in childhood hyperactive-impulsive, inattentive, and oppositional behaviors. *Journal of Pediatrics, 143*(1), 104-110.

Kaiser, M. L., Schoemaker, M. M., Albaret, J. M., & Geuze, R. H. (2015). What is the evidence of impaired motor skills and motor control among children with attention deficit hyperactivity disorder (ADHD)? Systematic review of the literature. *Research in Developmental Disabilities*, *36*, 338-357.

Kalff, A. C., de Sonneville, L. M., Hurks, P. P., Hendriksen, J. G., Kroes, M., & Feron, F. J. (2003). Low- and high-level controlled processing in executive motor control tasks in 5-6-year-old children at risk of ADHD. *Journal of Child Psychology and Psychiatry, 44*(7), 1049-1057.

Kann, R. T., & Hanna, F. J. (2000). Disruptive behavior disorders in children and adolescents: How do girls differ from boys? *Journal of Counseling & Development, 78*(3), 267-274.

Kaplan, B. J. (2016). Kaplan and Sadock's Synopsis of Psychiatry. Behavioral Sciences/Clinical Psychiatry. *Tijdschrift voor Psychiatrie, 58*(1), 78-79.

Karustis, J. L., Power, T. J., Rescorla, L. A., Eiraldi, R. B., & Gallagher, P. R. (2000). Anxiety and depression in children with ADHD: Unique associations with academic and social functioning [Press release], *Journal of Attention Disorders*, 4(3), 133-149

Kashala, E., Lundervold, A., Sommerfelt, K., Tylleskar, T., & Elgen, I. (2006). Co-existing symptoms and risk factors among African school children with hyperactivity-inattention symptoms in Kinshasa, Congo. *European Child and Adolescent Psychiatry, 15*(5), 292-299.

Kates, W. R., Frederikse, M., Mostofsky, S. H., Folley, B. S., Cooper, K., Mazur-Hopkins, P., Kofman, O., Singer, H. S., Denckla, M. B., Pearlson, G. D., & Kaufmann, W. E. (2002). MRI parcellation of the frontal lobe in boys with attention deficit hyperactivity disorder or Tourette syndrome. *Psychiatry Research, 116*(1-2), 63-81.

Katzman, M. A., Bilkey, T. S., Chokka, P. R., Fallu, A., & Klassen, L. J. (2017). Adult ADHD and comorbid disorders: clinical implications of a dimensional approach. *Biomed Central of Psychiatry,* 17(1), 302.

Kawulich, B. B. (2005). Participant observation as a data collection method. In *Forum Qualitative Sozialforschung/Forum: Qualitative Social Research,* 6(2), 43.

Keiley, M. K., Lofthouse, N., Bates, J. E., Dodge, K. A., & Pettit, G. S. (2003). Differential Risks of Covarying and Pure Components in Mother and Teacher Reports of Externalizing and Internalizing Behavior Across Ages 5 to 14. *Journal of Abnormal Child Psychology, 31*(3), 267-283.

Kellogg, C. (2016). *Back to Recovery: A Hermeneutic Phenomenological Inquiry into Childhood Psychotropic Medication Management*: Pacifica Graduate Institute.

Kendall, T., Taylor, E., Perez, A., & Taylor, C. (2008). Diagnosis and management of attention-deficit/hyperactivity disorder in children, young people, and adults: summary of National Institute for Clinical Excellence guidance. *Bio Medical Journal*, *337*, a1239.

Kerekes, N., Lundström, S., Chang, Z., Tajnia, A., Jern, P., Lichtenstein, P., Nilsson, T., & Anckarsäter, H. (2014). Oppositional defiant-and conduct disorder-like problems: neurodevelopmental predictors and genetic background in boys and girls, in a nationwide twin study. *PeerJournal, 2*, page 359.

Kessler, R. C., Adler, L., Barkley, R., Biederman, J., Conners, C. K., Demler, O., Faraone, S. V., Greenhill, L. L., Howes, M. J., Secnik, K., Spencer, T., Ustun, T. B., Walters, E. E., & Zaslavsky, A. M. (2006). The prevalence and correlates of adult ADHD in the United States: Results from the National Comorbidity Survey Replication. *The American Journal of Psychiatry, 163*(4), 716-723.

Killeen, P. (2005). Gradus ad Parnassum. Ascending strength gradients or descending memory traces? *Behavioral and Brain Sciences, 28*, 432-434.

Kim, M. H., Shimomaeda, L., Giuliano, R. J., & Skowron, E. A. (2017). Intergenerational associations in executive function between mothers and children in the context of risk. *Journal of Experimental Child Psychology*, *164*, 1-15.

Kirmayer, L. J. (2001). Cultural variations in the clinical presentation of depression and anxiety: implications for diagnosis and treatment. *Journal of Clinical Psychiatry,* 62, 22-30.

Klimkeit, E., Graham, C., Lee, P., Morling, M., Russo, D., & Tonge, B. (2006). Children should be seen and heard: self-report of feelings and behaviors in primary-school-age children with ADHD. *Journal of Attention Disorders, 10*(2), 181-191.

Klingberg, T., Fernell, E., Olesen, P. J., Johnson, M., Gustafsson, P., Dahlström, K., ... & Westerberg, H. (2005). Computerized training of working memory in children with ADHD-a randomized, controlled trial. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*(2), 177-186.

Knopik, V. S., Bidwell, L. C., Flessner, C., Nugent, N., Swenson, L., Bucholz, K. K., Madden, P. A., & Heath, A. C. (2014). DSM-IV defined conduct disorder and oppositional defiant disorder: an investigation of shared liability in female twins. *Psychological Medicine, 44*(5), 1053-1064.

Kohn, M. R., Tsang, T. W., & Clarke, S. D. (2012). Efficacy and Safety of Atomoxetine in the Treatment of Children and Adolescents with Attention Deficit Hyperactivity Disorder. *Clinical Medicine Insights. Pediatrics, 6*, 95-162.

Kolko, D. J., Dorn, L. D., Bukstein, O., & Burke, J. D. (2008). Clinically referred ODD children with or without CD and healthy controls: Comparisons across contextual domains. *Journal of Child and Family Studies, 17*(5), 714-734.

Kollins, S. H., Lane, S. D., & Shapiro, S. K. (1997). Experimental analysis of childhood psychopathology: A laboratory matching analysis of the behavior of children diagnosed with Attention-Deficit Hyperactivity Disorder (ADHD). *The Psychological Record, 47*(1), 25-44.

Konrad, K., & Eickhoff, S. B. (2010). Is the ADHD brain wired differently? A review on structural and functional connectivity in attention deficit hyperactivity disorder. *Human Brain Mapping, 31*(6), 904-916.

Konst, M. J., Matson, J. L., Goldin, R. L., & Williams, L. W. (2014). Socialization and nonverbal communication in atypically developing infants and toddlers. *Research in Developmental Disabilities, 35*(12), 3416-3422.

Kovacs, M., Akiskal, H. S., Gatsonis, C., & Parrone, P. L. (1994). Childhood-onset dysthymic disorder: clinical features and prospective naturalistic outcome. *Archives of General Psychiatry, 51*(5), 365-374.

Krain, A. L., & Castellanos, F. X. (2006). Brain development and ADHD. *Clinical Psychology Review, 26*(4), 433-444.

Kratochvil, C. J., Newcorn, J. H., Arnold, L. E., Duesenberg, D., Emslie, G. J., Quintana, H., Sarkis, E. H., Wagner, K. D., Gao, H., Michelson, D., & Biederman, J. (2005). Atomoxetine alone or combined with fluoxetine for treating ADHD with comorbid depressive or anxiety symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry,* 44(9), 915-924.

Krueger, M., & Kendall, J. (2001). Abstract. *Journal of Child and Adolescent Psychiatric Nursing, 14*(2), 61-72.

Kunwar, A., Dewan, M., & Faraone, S. V. (2007). Treating common psychiatric disorders associated with attention-deficit/hyperactivity disorder. *Expert Opininion on Pharmacotherapy, 8*(5), 555-562.

Kutcher, S., Aman, M., Brooks, S. J., Buitelaar, J., Van Daalen, E., Fegert, J., Findling, R. L., Fisman, S., Greenhill, L. L., & Huss, M. (2004). International consensus statement on attention-deficit/hyperactivity disorder (ADHD) and disruptive

behaviour disorders (DBDs): clinical implications and treatment practice suggestions. *European Neuropsychopharmacology, 14*(1), 11-28.

Landgraf, J., Abetz, L., & Ware, J. (1999). *Child Health Questionnaire (CHQ): A User's Manual.* Boston, MA: Health Act.

Lange, K. W., Reichl, S., Lange, K. M., Tucha, L., & Tucha, O. (2010). The history of attention deficit hyperactivity disorder. *ADHD Attention Deficit and Hyperactivity Disorders, 2*(4), 241-255.

Larson, K., Russ, S. A., Kahn, R. S., & Halfon, N. (2011). Patterns of Comorbidity, Functioning, and Service Use for US Children With ADHD, 2007. *Pediatrics, 127*(3), 462-470.

Lashley, K. S. (1951). The problem of serial order in behavior. In L. A. Jeffress (Ed.), *Cerebral Mechanisms in Behavior*. New York: Wiley.

Lee, S. S., Falk, A. E., & Aguirre, V. P. (2012). Association of comorbid anxiety with social functioning in school-age children with and without attention-deficit/hyperactivity disorder (ADHD). *Psychiatry Research*, *197*(1-2), 90-96.

Leedy, P. D., & Ormrod, J. E. (2001). Practical Research Design: Planning and Design: Upper Saddle River. *New Jersy: Merryll Prentice Hall*.

Leibenluft, E. (2011). Severe Mood Dysregulation, Irritability, and the Diagnostic Boundaries of Bipolar Disorder in Youths. *The American Journal of Psychiatry, 168*(2), 129-142.

Leirbakk, M. J., Clench-Aas, J., & Raanaas, R. K. (2015). ADHD with co-occurring depression/anxiety in children: The Relationship with Somatic Complaints and Parental Socio-Economic Position. *Journal of Psychological Abnormalities in Children*, 4: 137

Lemaitre, H., Goldman, A. L., Sambataro, F., Verchinski, B. A., Meyer-Lindenberg, A., Weinberger, D. R., & Mattay, V. S. (2012). Normal age-related brain morphometric changes: Nonuniformity across cortical thickness, surface area and grey matter volume? *Neurobiology of Aging, 33*(3), 617.e611-617.e619.

Leslie, L. K., Weckerly, J., Plemmons, D., Landsverk, J., & Eastman, S. (2004). Implementing the American Academy of Pediatrics attention-deficit/hyperactivity

disorder diagnostic guidelines in primary care settings. *Pediatrics, 114*(1), 129-140.

Levy, F. (2004). Synaptic gating and ADHD: a biological theory of comorbidity of ADHD and anxiety. *Neuropsychopharmacology, 29*(9), 1589-1596.

Levy, F. (2014). DSM-5, ICD-11, RDoC and ADHD diagnosis. *Australian & New Zealand Journal of Psychiatry, 48*(12), 1163-1169.

Levy, F., Hay, D., Waldman, I., & McStephen, M. (2002). Common Family Environment and The Comorbidity Among the Disruptive Behavior Disorders. *The ADHD Report*, *10*(1), 9-14.

Levy, F., Hay, D. A., Bennett, K. S., & Mcstephen, M. (2005). Gender differences in ADHD subtype comorbidity. *Journal of the American Academy of Child & Adolescent Psychiatry, 44*(4), 368-376.

Lewinsohn, P. M., Gotlib, I. H., Lewinsohn, M., Seeley, J. R., & Allen, N. B. (1998). Gender differences in anxiety disorders and anxiety symptoms in adolescents. *Journal of Abnormal Psychology, 107*(1), 109.

Lezak, M. D., Howieson, D. B., Loring, D. W., & Fischer, J. S. (2004). *Neuropsychological assessment*: Oxford University Press, USA.

Lifford, K. J., Harold, G. T., & Thapar, A. (2009). Parent-child hostility and child ADHD symptoms: a genetically sensitive and longitudinal analysis. *Journal of Child Psychology and Psychiatry, 50*(12), 1468-1476.

Liston, C., Matalon, S., Hare, T. A., Davidson, M. C., & Casey, B. (2006). Anterior cingulate and posterior parietal cortices are sensitive to dissociable forms of conflict in a task-switching paradigm. *Neuron, 50*(4), 643-653.

Littman, E. (2012). The secret lives of girls with ADHD. *Attention, 19*(6), 18-20.

Liu, J., Raine, A., Venables, P. H., Dalais, C., & Mednick, S. A. (2003). Malnutrition at age 3 years and lower cognitive ability at age 11 years: independence from psychosocial adversity. *Archives of Pediatrics and Adolescent Medicine, 157*(6), 593-600.

Loe, I. M., & Feldman, H. M. (2007). Academic and educational outcomes of children with ADHD. *Journal of Pediatric Psychology, 32*(6), 643-654.

Loeber, R., Capaldi, D. M., & Costello, E. (2013). Gender and the development of aggression, disruptive behavior, and delinquency from childhood to early adulthood. In *Disruptive Behavior Disorders* (pp. 137-160): Springer, New York.

Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and Inhibitory Control. *Psychological Science, 8*(1), 60-64.

Lonigan, C. J., & Phillips, B. M. (2001). Temperamental influences on the development of anxiety disorders. *The Developmental Psychopathology of Anxiety*, Oxford University Press, New York, pp. 60-91.

López, F. S., Masana, A. M., Martí, S. S., Acosta, S. G., & Gaviria, A. G. (2012). *The course of attention deficit/hyperactivity disorder in an outpatient sample.* Paper presented at the Anales de pediatria (Barcelona, Spain: 2003), 76(5), 250-255.

Luman, M., Tripp, G., & Scheres, A. (2010). Identifying the neurobiology of altered reinforcement sensitivity in ADHD: a review and research agenda. *Neuroscience and Biobehavioral Review, 34*(5), 744-754.

MacQueen, G. M., Campbell, S., McEwen, B. S., Macdonald, K., Amano, S., Joffe, R. T., Nahmias, C., & Young, L. T. (2003). Course of illness, hippocampal function, and hippocampal volume in major depression. *Proceedings of the National Academy of Sciences, 100*(3), 1387-1392.

Madras, B. K., Miller, G. M., & Fischman, A. J. (2002). The dopamine transporter: relevance to attention deficit hyperactivity disorder (ADHD). *Behavioral Brain Research, 130*(1-2), 57-63.

Mako, K. (2002). The prevalence of Attention Deficit/Hyperactivity Disorder (ADHD) among clinically referred and community based children: A comparative study. *University of the North. parental support, and coping strategies. Journal of Consulting and Clinical Psychology, 69*, 992-1006.

Malhi, G. S., Byrow, Y., Bassett, D., Boyce, P., Hopwood, M., Lyndon, W., Mulder, R., Porter, R., Singh, A., & Murray, G. (2016). Stimulants for depression: On the up and up? *Australian & New Zealand Journal of Psychiatry, 50*(3), 203-207.

Malhi, P., & Singhi, P. (2001). Diagnosis and management of children with attention deficit hyperactivity disorder. *The Indian Journal of Pediatrics, 68*(6), 547-555.

Manassis, K. (2008). *Keys to parenting your anxious child*: Barron's Educational Series.

Manassis, K., Tannock, R., & Barbosa, J. (2000). Dichotic listening and response inhibition in children with comorbid anxiety disorders and ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(9), 1152-1159.

Manassis, K., Tannock, R., Young, A., & Francis-John, S. (2007). Cognition in anxious children with attention deficit hyperactivity disorder: a comparison with clinical and normal children. *Behavioral and Brain Functions*, *3*(1), 4.

Manjunath, R., Kishor, M., Kulkarni, P., Shrinivasa, B., & Sathyamurthy, S. (2016). Magnitude of Attention Deficit Hyper Kinetic Disorder among School Children of Mysore City. *International Neuropsychiatric Disease Journal, 6(1)*, 1-7.

March, J. S., Swanson, J. M., Arnold, L. E., Hoza, B., Conners, C. K., Hinshaw, S. P., Hechtman, L., Kraemer, H. C., Greenhill, L. L., & Abikoff, H. B. (2000). Anxiety as a predictor and outcome variable in the multimodal treatment study of children with ADHD (MTA). *Journal of Abnormal Child Psychology, 28*(6), 527-541.

Martin, N. C., Levy, F., Pieka, J., & Hay, D. A. (2006). A genetic study of attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder and reading disability: aetiological overlaps and implications. *International Journal of Disability, Development and Education, 53*(1), 21-34.

Masi, L., & Gignac, M. (2017, June). ADHD and comorbid disorders in pedopsychiatry: Psychiatric problems, medical problems, learning disorders and developmental coordination disorder. In *ANNALES MEDICO-PSYCHOLOGIQUES,* 175(5), 422-429.

Matsumoto, N., Hanakawa, T., Maki, S., Graybiel, A. M., & Kimura, M. (1999). Role of [corrected] nigrostriatal dopamine system in learning to perform sequential motor tasks in a predictive manner. *Journal of Neurophysiology, 8(2), 978-998*.

Maughan, B., Collishaw, S., & Stringaris, A. (2013). Depression in childhood and adolescence. *Journal of the Canadian Academy of Child and Adolescent Psychiatry, 22*(1), 35.

Maughan, B., Rowe, R., Messer, J., Goodman, R., & Meltzer, H. (2004). Conduct disorder and oppositional defiant disorder in a national sample: developmental epidemiology. *Journal of Child Psychology and Psychiatry, 45*(3), 609-621.

Mayes, S. D., Calhoun, S. L., Bixler, E. O., Vgontzas, A. N., Mahr, F., Hillwig-Garcia, J., Elamir, B., Edhere-Ekezie, L., & Parvin, M. (2009). ADHD Subtypes and Comorbid Anxiety, Depression, and Oppositional-Defiant Disorder: Differences in Sleep Problems. *Journal of Pediatric Psychology, 34*(3), 328-337.

Mayes, S. D., Calhoun, S. L., Chase, G. A., Mink, D. M., & Stagg, R. E. (2008). ADHD Subtypes and Co-Occurring Anxiety, Depression, and Oppositional-Defiant Disorder: Differences in Gordon Diagnostic System and Wechsler Working Memory and Processing Speed Index Scores. *Journal of Attention Disorders, 12*(6), 540-550.

Mazzone, L., Postorino, V., Reale, L., Guarnera, M., Mannino, V., Armando, M., Fatta, L., De Peppo, L., & Vicari, S. (2013). Self-esteem evaluation in children and adolescents suffering from ADHD. *Clinical Practice and Epidemiology in Mental Health. 9, 96-102.*

Melnyk, B. M., Fineout-Overholt, E., Gallagher-Ford, L., & Stillwell, S. B. (2011). Evidence-based practice, step by step: sustaining evidence-based practice through organizational policies and an innovative model. *The American Journal of Nursing, 111*(9), 57-60.

Menon, V. (2011). Large-scale brain networks and psychopathology: a unifying triple network model. *Trends in Cognitive Sciences, 15*(10), 483-506.

Merikangas, K. R., He, J.-p., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., Benjet, C., Georgiades, K., & Swendsen, J. (2010). Lifetime Prevalence of Mental Disorders in US Adolescents: Results from the National Comorbidity Study-Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolesc Psychiatry, 49*(10), 980-989.

Mertler, C. A., & CharleDs, C. M. (2005). *Introduction to Educational Research (3rd edition)*: Boston, Pearson.

Meyer, A. (1997). Attention-Deficit/Hyperactivity Disorder among North Sotho speaking primary school children in South Africa, Prevalence and Sex Ratios. Journal of Psychology in Africa, 2(1), 186-195.

Meyer, A., & Aase, H. (2003). *Assessment and intervention in childhood disruptive behaviour disorders*. In N.S. Madu (Ed), Contributions to psychotherapy in Africa. (pp. 164-178) Polokwane (Pietersburg): UNIN Press.

Meyer, A., Eilertsen, D.-E., Sundet, J. M., Tshifularo, J., & Sagvolden, T. (2004). Cross-cultural Similarities in ADHD-Like Behaviour Amongst South African Primary School Children. *South African Journal of Psychology, 34*(1), 122-138.

Meyer, A., & Sagvolden, T. (2006). Fine motor skills in South African children with symptoms of ADHD: influence of subtype, gender, age, and hand dominance. *Behavioral and Brain Functions, 2*, 33-33.

Mick, E., Spencer, T., Wozniak, J., & Biederman, J. (2005). Heterogeneity of irritability in attention-deficit/hyperactivity disorder subjects with and without mood disorders. *Biological Psychiatry, 58*(7), 576-582.

Mikami, A. Y., Griggs, M. S., Lerner, M. D., Emeh, C. C., Reuland, M. M., Jack, A., & Anthony, M. R. (2013a). A Randomized Trial of a Classroom Intervention to Increase Peers' Social Inclusion of Children with Attention-Deficit/Hyperactivity Disorder. *Journal of Consulting and Clinical Psychology, 81*(1), 100-112.

Mikami, A. Y., & Lorenzi, J. (2011). Gender and conduct problems predict peer functioning among children with attention-deficit/hyperactivity disorder. *Journal of Clinical Child & Adolescent Psychology, 40*(5), 777-786.

Mikami, A. Y., Ransone, M. L., & Calhoun, C. D. (2011). Influence of anxiety on the social functioning of children with and without ADHD. *Journal of Attention Disorders*, *15*(6), 473-484.

Miller, A. R., Johnston, C., Klassen, A. F., Fine, S., & Papsdorf, M. (2005). Family physicians' involvement and self-reported comfort and skill in care of children with behavioral and emotional problems: a population-based survey. *Biomed Central Family Practice, 6*(1), 12.

Millichap, J. G. (2008). Etiologic classification of attention-deficit/hyperactivity disorder. *Pediatrics, 121*(2), e358-e365.

Miranda-Casas, A., Presentacion-Herrero, M., Colomer-Diago, C., & Rosello, B. (2011). Satisfaction with life of children with attention deficit hyperactivity

disorder: a study of possible protection and risk factors. *Revista de Neurologia, 52*, S119-126.

Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions: Four general conclusions. *Current Directions in Psychological Science, 21*(1), 8-14.

Mokobane, M., Pillay, B., & Meyer, A. (2017). Comorbidity of Attention Deficit Hyperactive Disorder (ADHD) and major depression in primary school children. *Journal of Psychology in Africa*, *27*(6), 541-544.

Monuteaux, M. C., Faraone, S. V., Gross, L. M., & Biederman, J. (2007). Predictors, clinical characteristics, and outcome of conduct disorder in girls with attention-deficit/hyperactivity disorder: a longitudinal study. *Psychological Medicine, 37*(12), 1731-1741.

Mostofsky, S. H., Cooper, K. L., Kates, W. R., Denckla, M. B., & Kaufmann, W. E. (2002). Smaller prefrontal and premotor volumes in boys with attention-deficit/hyperactivity disorder. *Biological Psychiatry, 52*(8), 785-794.

Muir-Broaddus, J. E., Rosenstein, L. D., Medina, D. E., & Soderberg, C. (2002). Neuropsychological test performance of children with ADHD relative to test norms and parent behavioral ratings. *Archives of Clinical Neuropsychology*, *17*(7), 671-689.

Mulder, E. J., De Medina, P. R., Huizink, A. C., Van den Bergh, B. R., Buitelaar, J. K., & et Visser, G. H. (2002). Prenatal maternal stress: effects on pregnancy and the (unborn) child. *Early Human Development, 70*(1-2), 3-14.

Müller, U. C., Asherson, P., Banaschewski, T., Buitelaar, J. K., Ebstein, R. P., Eisenberg, J., Gill, M., Manor, I., Miranda, A., & Oades, R. D. (2011). The impact of study design and diagnostic approach in a large multi-centre ADHD study. Part 1: ADHD symptom patterns. *Biomed Central Psychiatry, 11*(1), 54.

Municipality, T. L. (2009). *Local economic development strategy*. Available on http://epress. lib. uts. edu. au/journals/index. php/cjlg/article/view/2413.[Accessed online: 15 May 2014].

Murphy, K., & Barkley, R. A. (1996). Attention deficit hyperactivity disorder adults: comorbidities and adaptive impairments. *Comprehensive Psychiatry, 37*(6), 393-401.

Mychailyszyn, M. P., Beidas, R. S., Benjamin, C. L., Edmunds, J. M., Podell, J. L., Cohen, J. S., & Kendall, P. C. (2011). Assessing and treating child anxiety in schools. *Psychology in the Schools, 48*(3), 223-232.

Nadder, T. S., Rutter, M., Silberg, J., Maes, H., & Eaves, L. (2002). Genetic effects on the variation and covariation of attention deficit-hyperactivity disorder (ADHD) and oppositional-defiant disorder/conduct disorder (ODD/CD) symptomatologies across informant and occasion of measurement. *Psychological Medicine, 32*(1), 39-53.

Nagel, B. J., Bathula, D., Herting, M., Schmitt, C., Kroenke, C. D., Fair, D., & Nigg, J. T. (2011). Altered white matter microstructure in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*(3), 283-292.

Nakao, T., Radua, J., Rubia, K., & Mataix-Cols, D. (2011). Gray matter volume abnormalities in ADHD: voxel-based meta-analysis exploring the effects of age and stimulant medication. *American Journal of Psychiatry, 168*(11), 1154-1163.

Närhi, V., Kiiski, T., Peitso, S., & Savolainen, H. (2015). Reducing disruptive behaviours and improving learning climates with class-wide positive behaviour support in middle schools. *European Journal of Special Needs Education, 30*(2), 274-285.

Nash, P., Schlösser, A., & Scarr, T. (2016). Teachers' perceptions of disruptive behaviour in schools: a psychological perspective. *Emotional and Behavioural Difficulties, 21*(2), 167-180.

Nations, U. (2011). Human development report.

Newark, P. E., Elsässer, M., & Stieglitz, R.-D. (2016). Self-esteem, self-efficacy, and resources in adults with ADHD. *Journal of Attention Disorders, 20*(3), 279-290.

Newcorn, J. H., Halperin, J. M., Jensen, P. S., Abikoff, H. B., Arnold, L. E., Cantwell, D. P., Conners, C. K., Elliott, G. R., Epstein, J. N., & Greenhill, L. L. (2001). Symptom profiles in children with ADHD: effects of comorbidity and gender.

Journal of the American Academy of Child & Adolescent Psychiatry, 40(2), 137-146.

Newman, J. P., Wallace, J. F., Strauman, T. J., Skolaski, R. L., Oreland, K. M., Mattek, P. W., Elder, K. A., & McNeely, J. (1993). Effects of motivationally significant stimuli on the regulation of dominant responses. *Journal of Personality and Social Psychology, 65*(1), 165-175.

Newman, N. C., Ryan, P., Lemasters, G., Levin, L., Bernstein, D., Hershey, G. K., Lockey, J. E., Villareal, M., Reponen, T., Grinshpun, S., Sucharew, H., & Dietrich, K. N. (2013). Traffic-related air pollution exposure in the first year of life and behavioral scores at 7 years of age. *Environmental Health Perspective, 121*(6), 731-736.

Nigg, J., Nikolas, M., & Burt, S. A. (2010). Measured gene-by-environment interaction in relation to attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 49*(9), 863-873.

Nordquist, N., & Oreland, L. (2010). Serotonin, genetic variability, behaviour, and psychiatric disorders-a review. *Upsala Journal of Medical Sciences, 115*(1), 2-10.

Norén Selinus, E., Molero, Y., Lichtenstein, P., Anckarsäter, H., Lundström, S., Bottai, M., & Hellner Gumpert, C. (2016). Subthreshold and threshold attention deficit hyperactivity disorder symptoms in childhood: psychosocial outcomes in adolescence in boys and girls. *Acta Psychiatrica Scandinavica, 134*(6), 533-545

Ofovwe, C. E., Ofovwe, G. E., & Meyer, A. (2006). The prevalence of attention-deficit/hyperactivity disorder among school-aged children in Benin City, Nigeria. *Journal of Child and Adolescent Mental Health, 18*(1), 1-5.

Ohbayashi, M., Ohki, K., & Miyashita, Y. (2003). Conversion of working memory to motor sequence in the monkey premotor cortex. *Science*, *301*(5630), 233-236.

Ohrt, J., Webster, L., & De La Garza, M. (2014). The effects of a success skills group on adolescents' self-regulation, self-esteem, and perceived learning competence. *Professional School Counseling, 18*(1), 169-178.

Oosterlaan, J., Logan, G. D., & Sergeant, J. A. (1998). Response inhibition in AD/HD, CD, comorbid AD/HD + CD, anxious, and control children: a meta-analysis of

studies with the stop task. *Journal of Child Psychology and Psychiatry, 39*(3), 411-425.

Organization, W. H. (1993). ICD-10: The ICD-10 Classification of Mental and Behavioural Disorders: diagnostic criteria for research. In *ICD-10: the ICD-10 classification of mental and behavioural disorders: diagnostic criteria for research*.

Organization, W. H. (2012). *Guideline: Sodium intake for adults and children*: World Health Organization.

Ou, X., Andres, A., Cleves, M. A., Pivik, R. T., Snow, J. H., Ding, Z., & Badger, T. M. (2014). Sex-specific association between infant diet and white matter integrity in 8-y-old children. *Pediatric Research, 76*(6), 535-543.

Overmeyer, S., Bullmore, E. T., Suckling, J., Simmons, A., Williams, S. C., Santosh, P. J., & Taylor, E. (2001). Distributed grey and white matter deficits in hyperkinetic disorder: MRI evidence for anatomical abnormality in an attentional network. *Psychological Medicine, 31*(8), 1425-1435.

Palaniappan, P. (2016). Impact of Comorbidities on Self-Esteem of Children with Attention Deficit Hyperactivity Disorder. *The International Journal of Indian Psychology, Volume 3, Issue 3, No. 1*, 88.

Parker, J. G., & Asher, S. R. (1987). Peer relations and later personal adjustment: Are low-accepted children at risk? *Psychological Bulletin, 102*(3), 357.

Patel, N., Patel, H., & Patel, M. (2012). *ADHD and Comorbid Conditions*: Current Directions in ADHD and its Treatment, *INTECH Open Access, USA.*

Pelham, W. E., Jr., Gnagy, E. M., Greenslade, K. E., & Milich, R. (1992). Teacher ratings of DSM-III-R symptoms for the disruptive behavior disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 31*(2), 210-218.

Perlov, E., Philipsen, A., van Elst, L. T., Ebert, D., Henning, J., Maier, S., ... & Hesslinger, B. (2008). Hippocampus and amygdala morphology in adults with attention-deficit hyperactivity disorder. *Journal of psychiatry & neuroscience: JPN*, *33*(6), 509.

Perroud, N., Cordera, P., Zimmermann, J., Michalopoulos, G., Bancila, V., Prada, P., Dayer, A., & Aubry, J. M. (2014). Comorbidity between attention deficit

hyperactivity disorder (ADHD) and bipolar disorder in a specialized mood disorders outpatient clinic. *J ournal of Affective Disorders, 168*, 161-166.

Pheula, G. F., Rohde, L. A., & Schmitz, M. (2011). Are family variables associated with ADHD, inattentive type? A case-control study in schools. *European Child and Adolescent Psychiatry, 20*(3), 137-145.

Pila-Nemutandani, R. G., & Meyer, A. (2016). Behaviour planning and problem solving deficiencies in children with symptoms of attention deficit hyperactivity disorder from the Balobedu culture, Limpopo province, South Africa. *Journal of Child & Adolescent Mental Health, 28*(2), 109-121.

Pisecco, S., Wristers, K., Swank, P., Silva, P. A., & Baker, D. B. (2001). The effect of academic self-concept on ADHD and antisocial behaviors in early adolescence. *Journal of Learning Disabilities, 34*(5), 450-461.

Plessen, K. J., Bansal, R., Zhu, H., Whiteman, R., Amat, J., Quackenbush, G. A., Martin, L., Durkin, K., Blair, C., Royal, J., Hugdahl, K., & Peterson, B. S. (2006). Hippocampus and amygdala morphology in attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 63*(7), 795-807.

Pliszka, S. R. (1998). Comorbidity of attention-deficit/hyperactivity disorder with psychiatric disorder: an overview. *Journal of Clinical Psychiatry, 59*, 50-58.

Pliszka, S. R. (2003). Psychiatric comorbidities in children with attention deficit hyperactivity disorder. *Pediatric Drugs, 5*(11), 741-750.

Pliszka, S. R., Carlson, C. L., & Swanson, J. M. (1999). *ADHD with comorbid disorders: Clinical Assessment and Management*: New York, Guilford Press.

Pliszka, S. R., Liotti, M., & Woldorff, M. G. (2000). Inhibitory control in children with attention-deficit/hyperactivity disorder: event-related potentials identify the processing component and timing of an impaired right-frontal response-inhibition mechanism. *Biological Psychiatry, 48*(3), 238-246.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. (2003). *Behavioral Genetics*. American Psychological Association. Washinhton, DC.

Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *Amerian Journal of Psychiatry, 164*(6), 942-948.

Polanczyk, G. V., Willcutt, E. G., Salum, G. A., Kieling, C., & Rohde, L. A. (2014). ADHD prevalence estimates across three decades: an updated systematic review and meta-regression analysis. *Internatioinal Journal of Epidemiology, 43*(2), 434-442.

Polit, D. F., & Beck, C. T. (2008). *Nursing Research: Generating and Assessing Evidence for Nursing Practice*: Wolters Kluwer Health/lippincott Williams & Wilkins.

Porrino, L. J., Rapoport, J. L., Behar, D., Sceery, W., Ismond, D. R., & Bunney, W. E., Jr. (1983). A naturalistic assessment of the motor activity of hyperactive boys. I. Comparison with normal controls. *Archives of General Psychiatry, 40*(6), 681-687.

Posner, J., Siciliano, F., Wang, Z., Liu, J., Sonuga-Barke, E., & Greenhill, L. (2014). A multimodal MRI study of the hippocampus in medication-naive children with ADHD: what connects ADHD and depression? *Psychiatry Research: Neuroimaging, 224*(2), 112-118.

Poul, V., Barbara, R., Bente, F., Karin, C., Annette, E., Anton, R. N., Thorkil, C., Ryan, S., & Raben, R. (2001). Structural brain abnormalities in unselected in-patients with major depression. *Acta Psychiatrica Scandinavica, 103*(4), 282-286.

Power, T. J., Costigan, T. E., Eiraldi, R. B., & Leff, S. S. (2004). Variations in Anxiety and Depression as a Function of ADHD Subtypes Defined by DSM-IV: Do Subtype Differences Exist or Not? *Journal of Abnormal Child Psychology, 32*(1), 27-37.

Quinn, P., & Wigal, S. (2004). Perceptions of girls and ADHD: results from a national survey. *Medscape General Medicine, 6*(2), 2.

Quinn, P. O. (2008). Attention-deficit/hyperactivity disorder and its comorbidities in women and girls: an evolving picture. *Current Psychiatry Reports, 10*(5), 419-423.

Quinn, P. O., & Madhoo, M. (2014). A review of Attention-Deficit/Hy

Rafalovich, A. (2001).Tthe conceptual history of Attention Deficit Hyperactivity Disorder: idiocy, imbecility, encephalitis and the child deviant. *Deviant Behavior, 22*(2), 93-115.

Raguram, R., Weiss, M. G., Channabasavanna, S., & Devins, G. M. (1996). Stigma, depression, and somatization in South India. *American Journal of Psychiatry, 153*(8), 1043-1049.

Rajasekar, S., Philominathan, P., & Chinnathambi, V. (2006). Research methodology. *1-53*

Ramsay, J. R. (2010). *Nonmedication treatments for adult ADHD: Evaluating impact on daily functioning and well-being*: Washington, DC, US: American Psychological Association.

Rapport, L. J., Van Voorhis, A., Tzelepis, A., & Friedman, S. R. (2001). Executive functioning in adult attention-deficit hyperactivity disorder. *Clinical Neuropsychology, 15*(4), 479-491.

Reinhardt, M. C., & Reinhardt, C. A. (2013). Attention deficit-hyperactivity disorder, comorbidities, and risk situations. *Journal de Pediatria, 89*(2), 124-130.

Reitman, D., O'Callaghan, P. M., & Mitchell, P. (2005). Parent as coach: Enhancing sports participation and social behavior for ADHD-diagnosed children. *Child & Family Behavior Therapy, 27*(2), 57-68.

Rhodes, B. J., Bullock, D., Verwey, W. B., Averbeck, B. B., & Page, M. P. A.(2004). Learning and production of movement sequences: behavioral, neurophysiological, and modeling perspectives. *Human Movement Science*, *23(5)*, 699-746.

Riccio, C. A., Wolfe, M. E., Romine, C., Davis, B., & Sullivan, J. R. (2004). The Tower of London and neuropsychological assessment of ADHD in adults. *Archives of Clinical Neuropsychology, 19*(5), 661-671.

Rinsky, J. R., & Hinshaw, S. P. (2011). Linkages between childhood executive functioning and adolescent social functioning and psychopathology in girls with ADHD. *Child Neuropsychology, 17*(4), 368-390.

Robin, A. L. (2014). Family therapy for adolescents with ADHD. *Child and Adolescent Psychiatric Clinics, 23*(4), 747-756.

Rohde, P., Clarke, G. N., Lewinsohn, P. M., Seeley, J. R., & Kaufman, N. K. (2001). Impact of comorbidity on a cognitive-behavioral group treatment for adolescent

depression. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*(7), 795-802.

Rommelse, N. N., Altink, M. E., Fliers, E. A., Martin, N. C., Buschgens, C. J., Hartman, C. A., Buitelaar, J. K., Faraone, S. V., Sergeant, J. A., & Oosterlaan, J. (2009). Comorbid problems in ADHD: degree of association, shared endophenotypes, and formation of distinct subtypes. Implications for a future DSM. *Journal of Abnormal Child Psychology, 37*(6), 793-804.

Rowe, R., Maughan, B., Pickles, A., Costello, E. J., & Angold, A. (2002). The relationship between DSM-IV oppositional defiant disorder and conduct disorder: Findings from the Great Smoky Mountains Study. *Journal of Child Psychology and Psychiatry, 43*(3), 365-373.

Rubia, K., Halari, R., Cubillo, A., Smith, A. B., Mohammad, A. M., Brammer, M., & Taylor, E. (2011). Methylphenidate normalizes fronto-striatal underactivation during interference inhibition in medication-naive boys with attention-deficit hyperactivity disorder. *Neuropsychopharmacology, 36*(8), 1575-1586.

Rucklidge, J. J. (2010). Gender differences in attention-deficit/hyperactivity disorder. *Psychiatric Clinics of North America, 33*(2), 357-373.

Rucklidge, J. J., & Tannock, R. (2001). Psychiatric, psychosocial, and cognitive functioning of female adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, 40*(5), 530-540.

Ruggeri, M., Bisoffi, G., Fontecedro, L., & Warner, R. (2001). Subjective and objective dimensions of quality of life in psychiatric patients: a factor analytical approach: The South Verona Outcome Project 4. *British Journal of Psychiatry, 178*, 268-275.

Ruggeri, M., Gater, R., Bisoffi, G., Barbui, C., & Tansella, M. (2002). Determinants of subjective quality of life in patients attending community-based mental health services. The South-Verona Outcome Project 5. *Acta Psychiatrica Scandinavica, 105*(2), 131-140.

Sadock, B., & Ruiz, P. (2015). *Kaplan & Sadock's synopsis of psychiatry: Behavioral Sciences*: Espan, SA, Wolters Kluwer.

Sadock, B. J., Sadock, V. A., Cancro, R., Sussman, N., & Ahmad, S. (2005). *Kaplan & Sadock's Pocket Handbook of Clinical Psychiatry*: Philadelphia: Lippincott Williams & Wilkins.

Sagvolden, T. (1999). Attention deficit/hyperactivity disorder. *European Psychologist, 4*(2), 109-114.

Sagvolden, T., Aase, H., Zeiner, P., & Berger, D. (1998). Altered reinforcement mechanisms in attention-deficit/hyperactivity disorder. *Behavioural Brain Reseach, 94*(1), 61-71.

Sagvolden, T., Johansen, E. B., Aase, H., & Russell, V. A. (2005b). A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behavioral and Brain Science, 28*(3), 397-419.

Sagvolden, T., & Sergeant, J. A. (1998). Attention deficit/hyperactivity disorder--from brain dysfunctions to behaviour. *Behavioral Brain Research, 94*(1), 1-10.

Sahay, A., & Hen, R. (2007). Adult hippocampal neurogenesis in depression. *Nature Neuroscience, 10*(9), 1110-1115.

Salsali, M., & Silverstone, P. H. (2003). Low self-esteem and psychiatric patients: Part II – The relationship between self-esteem and demographic factors and psychosocial stressors in psychiatric patients. *Annals of General Hospital Psychiatry, 2*(1), 2.

Santrock, J. W. (2002). *Adolescence*.Ed.ke-6. New York; McGraw-Hill Companies.

Sax, L., & Kautz, K.J (2003). Whofirst suggests the diagnosis of Attention Deficit/Hyperactivity Disorder?. *The Aannals of Family Medicine*, 1(3), 171-174.

Sayal, K., Daley, D., James, M., Yang, M., Batty, M. J., Taylor, J. A., Taylor, J.A., Pass, S., Sampson, C.J., Sellman, E., Valentine, A., & Hollis, C. (2012). Protocol evaluating the effectiveness of a school-based group programme for parents of children at risk of ADHD: the 'PArents, Teachers and CHildren WORKing Together (PATCHWORK)'cluster RCT protocol. *Bio Medical Journal Open*, *2*(5), e001783.

Scassellati, C., Bonvicini, C., Faraone, S. V., & Gennarelli, M. (2012). Biomarkers and attention-deficit/hyperactivity disorder: a systematic review and meta-analyses.

Journal of the American Academy of Child and Adolescent Psychiatry, 51(10), 1003-1019.

Schachar, R., & Tannock, R. (1995). Test of four hypotheses for the comorbidity of attention-deficit hyperactivity disorder and conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*(5), 639-648.

Schatz, D. B., & Rostain, A. L. (2006). ADHD with comorbid anxiety: a review of the current literature. *Journal of Attention Disorders, 10*(2), 141-149.

Schellack, N., & Meyer, J. C. (2016). The management of attention deficit/hyperactivity disorder in children: updated 2016. *South African Pharmaceutical Journal*, *83*(4), 21-29.

Schmitzer-Torbert, N., & Redish, A. D. (2004). Neuronal activity in the rodent dorsal striatum in sequential navigation: separation of spatial and reward responses on the multiple T task. *Journal of neurophysiology*, *91*(5), 2259-2272.

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*(5306), 1593-1599.

Schwartz, M. D., & Kilduff, T. S. (2015). The neurobiology of sleep and wakefulness. *Psychiatric Clinics, 38*(4), 615-644.

Sciberras, E., Lycett, K., Efron, D., Mensah, F., Gerner, B., & Hiscock, H. (2014). Anxiety in children with attention-deficit/hyperactivity disorder. *Pediatrics, 133*(5), 801-808.

Sciberras, E., Ohan, J., & Anderson, V. (2012). Bullying and peer victimisation in adolescent girls with attention-deficit/hyperactivity disorder. *Child Psychiatry & Human Development, 43*(2), 254-270.

Sciutto, M. J., & Eisenberg, M. (2007). Evaluating the evidence for and against the overdiagnosis of ADHD. *Journal of Attention Disorders, 11*(2), 106-113.

Sciutto, M. J., Nolfi, C. J., & Bluhm, C. (2004). Effects of Child Gender and Symptom Type on Referrals for ADHD by Elementary School Teachers. *Journal of Emotional and Behavioral Disorders, 12*(4), 247-253.

Secnik, K., Swensen, A., & Lage, M. J. (2005). Comorbidities and costs of adult patients diagnosed with attention-deficit hyperactivity disorder. *Pharmacoeconomics, 23*(1), 93-102.

Seidman, L. J., Biederman, J., Faraone, S. V., Weber, W., & Ouellette, C. (1997). Toward defining a neuropsychology of ADHD: Performance of children and adolescents from a large clinically referred sample. *Journal of Consulting Clinical Psychology*, *65*(1), 150-60.

Selected writings of John Hughlings Jackson. (1932). London: Hodder and Stoughton.

Semrud-Clikeman, M., Pliszka, S. R., Bledsoe, J., & Lancaster, J. (2012). Volumetric MRI Differences in Treatment Naïve and Chronically Treated Adolescents With ADHD-Combined Type. *Journal of Attention Disorders, 18*(6), 511-520.

Semrud-Clikeman, M., Steingard, R. J., Filipek, P., Biederman, J., Bekken, K., & Renshaw, P. F. (2000). Using MRI to examine brain–behavior relationships in males with attention deficit disorder with hyperactivity. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(4), 477-484.

Sergeant, J. (2000). The cognitive-energetic model: an empirical approach to attention-deficit hyperactivity disorder. *Neuroscience and Biobehavioral Reviews, 24*(1), 7-12.

Sergeant, J. A., Geurts, H., Huijbregts, S., Scheres, A., & Oosterlaan, J. (2003). The top and the bottom of ADHD: a neuropsychological perspective. *Neuroscience and Biobehavioral Reviews, 27*(7), 583-592.

Seymour, K. E., Chronis-Tuscano, A., Iwamoto, D. K., Kurdziel, G., & MacPherson, L. (2014). Emotion regulation mediates the association between ADHD and depressive symptoms in a community sample of youth. *Journal of Abnormal Child Psychology*, *42*(4), 611-621.

Shadish, W. R., Cook, T. D., & Campbell, D. T. (2002). *Experimental and quasi-experimental designs for generalized causal inference*. Boston: Houghton Mifflin,.

Shaw-Zirt, B., Popali-Lehane, L., Chaplin, W., & Bergman, A. (2005). Adjustment, Social Skills, and Self-Esteem in College Students With Symptoms of ADHD. *Journal of Attention Disorders, 8*(3), 109-120.

Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., Clasen, L., Evans, A., Giedd, J., & Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences, 104*(49), 19649-19654.

Shaw, P., Gilliam, M., Liverpool, M., Weddle, C., Malek, M., Sharp, W., Greenstein, D., Evans, A., Rapoport, J., & Giedd, J. (2011). Cortical development in typically developing children with symptoms of hyperactivity and impulsivity: support for a dimensional view of attention deficit hyperactivity disorder. *American Journal of Psychiatry, 168*(2), 143-151.

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., Giedd, J., Castellanos, F. X., & Rapoport, J. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry, 63*(5), 540-549.

Shaw, P., Sharp, W., Morrison, M., Eckstrand, K., Greenstein, D., Clasen, L., Evans, A., & Rapoport, J. L. (2009). Psychostimulant treatment and the developing cortex in Attention-Deficit/Hyperactivity Disorder. *The American Journal of Psychiatry, 166*(1), 58-63.

Shea, B. J., Grimshaw, J. M., Wells, G. A., Boers, M., Andersson, N., Hamel, C., Porter, A. C., Tugwell, P., Moher, D., & Bouter, L. M. (2007). Development of AMSTAR: a measurement tool to assess the methodological quality of systematic reviews. *Bio Med Central Medical Research Methodology, 7*(1), 10.

Sheline, Y. I., Gado, M. H., & Kraemer, H. C. (2003). Untreated depression and hippocampal volume loss. *American Journal of Psychiatry, 160*(8), 1516-1518.

Sheppard, D. M., Bradshaw, J. L., Georgiou, N., Bradshaw, J. A., & Lee, P. (2000). Movement sequencing in children with Tourette's syndrome and attention deficit hyperactivity disorder. *Movement Disorders: Official journal of the Movement Disorder Society*, *15*(6), 1184-1193.

Shier, A. C., Reichenbacher, T., Ghuman, H. S., & Ghuman, J. K. (2013). Pharmacological Treatment of Attention Deficit Hyperactivity Disorder in Children and Adolescents: Clinical Strategies. *Journal of Central Nervous System Disease, 5*, 1-17.

Siddique, S., Banerjee, M., Ray, M. R., & Lahiri, T. (2011). Attention-deficit hyperactivity disorder in children chronically exposed to high level of vehicular pollution. *European Journal of Pediatrics, 170*(7), 923-929.

Sidlauskaite, J., González-Madruga, K., Smaragdi, A., Riccelli, R., Puzzo, I., Batchelor, M., & Fairchild, G. (2018). Sex differences in risk-based decision making in adolescents with conduct disorder. *European child & adolescent psychiatry*, *27*(9), 1133-1142.

Siklos, S., & Kerns, K. A. (2004). Assessing multitasking in children with ADHD using a modified Six Elements Test. *Archives of Clinical Neuropsychology, 19*(3), 347-361.

Silk, T. J., Vance, A., Rinehart, N., Bradshaw, J. L., & Cunnington, R. (2009). White-matter abnormalities in attention deficit hyperactivity disorder: a diffusion tensor imaging study. *Human Brain Mapping, 30*(9), 2757-2765.

Silverstone, P. H., & Salsali, M. (2003). Low self-esteem and psychiatric patients: Part I–The relationship between low self-esteem and psychiatric diagnosis. *Annals of General Hospital Psychiatry, 2*(1), 2.

Sisto, F. F., & de Cássia Martinelli, S. (2004). Estudo preliminar para a construção da escala de autoconceito infanto-juvenil (EAC-IJ). *Interação em Psicologia, 8*(2).

Skinner, R. A., & Piek, J. P. (2001). Psychosocial implications of poor motor coordination in children and adolescents. *Human Movement Science, 20*(1-2), 73-94.

Skogli, E. W., Teicher, M. H., Andersen, P. N., Hovik, K. T., & Oie, M. (2013). ADHD in girls and boys--gender differences in co-existing symptoms and executive function measures. *Biomed Central of Psychiatry, 13(1)*, 298.

Slomkowski, C., Klein, R. G., & Mannuzza, S. (1995). Is self-esteem an important outcome in hyperactive children? *Journal of Abnormal Child Psychology, 23*(3), 303-315.

Smith, E. E., & Jonides, J. (1999). Storage and executive processes in the frontal lobes. *Science, 283*(5408), 1657-1661.

Sobel, L. J., Bansal, R., Maia, T. V., Sanchez, J., Mazzone, L., Durkin, K., Liu, J., Hao, X., Ivanov, I., Miller, A., Greenhill, L. L., & Peterson, B. S. (2010). Basal ganglia

surface morphology and the effects of stimulant medications in youth with attention deficit hyperactivity disorder. *American Journal of Psychiatry, 167*(8), 977-986.

Solanto, M. V. (1998). Neuropsychopharmacological mechanisms of stimulant drug action in attention-deficit hyperactivity disorder: a review and integration. *Behavioral Brain Research, 94*(1), 127-152.

Solomon, D. A., Keller, M. B., Leon, A. C., Mueller, T. I., Lavori, P. W., Shea, M. T., Coryell, W., Warshaw, M., Turvey, C., Maser, J. D., & Endicott, J. (2000). Multiple recurrences of major depressive disorder. *American Journal of Psychiatry, 157*(2), 229-233.

Somer, D. R., Sukru, U., Gulsah, S., Nese, E., & Ayse, A. (2007). Differentiating bipolar disorder in Turkish prepubertal children with attention-deficit hyperactivity disorder. *Bipolar Disorders, 9*(3), 243-251.

Sonuga-Barke, E. J. (2002). Psychological heterogeneity in AD/HD--a dual pathway model of behaviour and cognition. *Behavioral Brain Research, 130*(1-2), 29-36.

Sonuga-Barke, E. J. (2003). The dual pathway model of AD/HD: an elaboration of neuro-developmental characteristics. *Neuroscience and Biobehavioral Reviews, 27*(7), 593-604.

Sowell, E. R., Thompson, P. M., Holmes, C. J., Batth, R., Jernigan, T. L., & Toga, A. W. (1999). Localizing age-related changes in brain structure between childhood and adolescence using statistical parametric mapping. *Neuroimage, 9*(6), 587-597.

Sowell, E. R., Thompson, P. M., Leonard, C. M., Welcome, S. E., Kan, E., & Toga, A. W. (2004). Longitudinal mapping of cortical thickness and brain growth in normal children. *Journal of Neuroscience, 24*(38), 8223-8231.

Sowell, E. R., Thompson, P. M., Welcome, S. E., Henkenius, A. L., Toga, A. W., & Peterson, B. S. (2003). Cortical abnormalities in children and adolescents with attention-deficit hyperactivity disorder. *Lancet, 362*(9397), 1699-1707.

Sowell, E. R., Trauner, D. A., Gamst, A., & Jernigan, T. L. (2002). Development of cortical and subcortical brain structures in childhood and adolescence: a structural MRI study. *Developmental Medicine and Child Neurology, 44*(1), 4-16.

Spencer, T., Wilens, T., Biederman, J., Wozniak, J., & Harding-Crawford, M. (2000). Attention-deficit/hyperactivity disorder with mood disorders. *Attention-deficit disorders and comorbidities in children, adolescents, and adults*. pp 79-124), Arlington, United States: American Psychiatric Publishing.

Stadler, D. D., Musser, E. D., Holton, K. F., Shannon, J., & Nigg, J. T. (2016). Recalled Initiation and Duration of Maternal Breastfeeding Among Children with and Without ADHD in a Well Characterized Case-Control Sample. *Journal of Abnormal Child Psychology, 44*(2), 347-355.

Stanford, C., & Tannock, R. (Eds.). (2012). *Behavioral neuroscience of attention deficit hyperactivity disorder and its treatment* (Vol. 9). Springer Science & Business Media.

StatSoft (2011). Statistica for Windows (Version 10) [Computer software]. Tulsa: OK: StatSoft. Inc.

Stats, S.A. "Statistics South Africa". *Formal census*.

Stockmeier, C. A., Mahajan, G. J., Konick, L. C., Overholser, J. C., Jurjus, G. J., Meltzer, H. Y., Uylings, H. B., Friedman, L., & Rajkowska, G. (2004). Cellular changes in the postmortem hippocampus in major depression. *Biological Psychiatry, 56*(9), 640-650.

Stringaris, A., & Goodman, R. (2009). Longitudinal outcome of youth oppositionality: irritable, headstrong, and hurtful behaviors have distinctive predictions. *Journal of the American Academy of Child & Adolescent Psychiatry, 48*(4), 404-412.

Subcommittee on Attenion-Deficit/ Hyperactive Disorder Steering Committee on Quality Improvement and Management (2011). ADHD: clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. American Journal of *Pediatrics*, 128(5), 2654.

Swanberg, D., Passno, D., & Larimore, W.N. (2005). *Why ADHD does not not mean disaster*. Caro Stream IL: Tyndale House Publishers.

Swanson, J., Lerner, M., March, J., & Gresham, F. M. (1999). Assessment and intervention for attention-deficit/hyperactivity disorder in the schools. Lessons from the MTA study. *Pediatrics Clininics of North America, 46*(5), 993-1009.

Taanila, A. M., Hurtig, T. M., Miettunen, J., Ebeling, H. E., & Moilanen, I. K. (2009). Association between ADHD symptoms and adolescents' psychosocial well-being: a study of the Northern Finland Birth Cohort 1986. *International Journal of Circumpolar Health, 68*(2), 133-144.

Tannock, R. (2005). Hypodopaminergic function influences learning and memory as well as delay gradients. *Behavioral and Brain Sciences*, *28*(3), 444.

Tannock, R. (2009). ADHD with anxiety disorders. In *ADHD comorbidities: Handbook for ADHD complications in children and adults.* (pp. 131-155). Arlington, VA, US: American Psychiatric Publishing, Inc.

Tawia, S. (2013). *Breastfeeding, brain structure and function, cognitive development and educational attainment*. *Breastfeeding Review*, *21*(3), 15.

Taylor, D. N., & Del Pilar, J. (1992). Self-Esteem, Anxiety, and Drug Use. *Psychological Reports, 71*(3), 896-898.

Taylor, E. (1998). Clinical foundations of hyperactivity research. *Behavioral Brain Research, 94*(1), 11-24.

Taylor, E., Sergeant, J., Doepfner, M., Gunning, B., Overmeyer, S., Mobius, H. J., & Eisert, H. G. (1998). Clinical guidelines for hyperkinetic disorder. European Society for Child and Adolescent Psychiatry. *European Child and Adolescent Psychiatry, 7*(4), 184-200.

Terzian, M., Hamilton, K., & Ericson, S. (2011). What Works to Prevent or Reduce Internalizing Problems or Socio-Emotional Difficulties in Adolescents: Lessons from Experimental Evaluations of Social Interventions. Fact Sheet. Publication# 2011-34. *Child Trends*.

Thapar, A., Cooper, M., Eyre, O., & Langley, K. (2013). What have we learnt about the causes of ADHD? *Journal of Child Psychology and Psychiatry, 54*(1), 3-16.

Timimi, S., & Taylor, E. (2004). ADHD is best understood as a cultural construct. *British Journal of Psychiatry, 184(1)*, 8-9.

Todd, R. D., Rasmussen, E. R., Wood, C., Levy, F., & Hay, D. A. (2004). Should sluggish cognitive tempo symptoms be included in the diagnosis of attention-

deficit/hyperactivity disorder? *Journal of the American Academy of Child and Adolescent Psychiatry, 43*(5), 588-597.

Toplak, M. E., Rucklidge, J. J., Hetherington, R., John, S. C., & Tannock, R. (2003). Time perception deficits in attention-deficit/ hyperactivity disorder and comorbid reading difficulties in child and adolescent samples. *Journal of Child Psychology and Psychiatry, 44*(6), 888-903.

Treuting, J. J., & Hinshaw, S. P. (2001). Depression and self-esteem in boys with attention-deficit/hyperactivity disorder: associations with comorbid aggression and explanatory attributional mechanisms. *Journal of Abnormal Child Psychology, 29*(1), 23-39.

Tripp, G., Ryan, J., & Peace, K. (2002). Neuropsychological functioning in children with DSM-IV combined type Attention Deficit Hyperactivity Disorder. *Australian and New Zealand Journal of Psychiatry, 36*(6), 771-779.

Tsang, T. W., Kohn, M. R., Efron, D., Clarke, S. D., Clark, C. R., Lamb, C., & Williams, L. M. (2015). Anxiety in young people with ADHD: Clinical and self-report outcomes. *Journal of Attention Disorders, 19*(1), 18-26.

Tuckman, A. (2009). *More attention, less deficit: Success strategies for adults with ADHD*: Florida USA, Specialty Press Plantation.

Tuckman, A. (2012). *Understand your brain, get more done*: Florida, USA: Speciality Press Inc

Tung, I., Li, J. J., Meza, J. I., Jezior, K. L., Kianmahd, J. S., Hentschel, P. G., O'Neil, P. M., & Lee, S. S. (2016). Patterns of Comorbidity Among Girls With ADHD: A Meta-analysis. *Pediatrics, 138*(4), 1-13.

Turgay, A. (2007). A Multidimensional Approach to Medication Selection in the Treatment of Children and Adolescents with ADHD. *Psychiatry (Edgmont), 4*(8), 47-57.

Turgay, A., & Ansari, R. (2006). Major depression with ADHD: In children and adolescents. *Psychiatry (Edgmont), 3*(4), 20.

Tuvblad, C., Zheng, M., Raine, A., & Baker, L. A. (2009). A common genetic factor explains the covariation among ADHD ODD and CD symptoms in 9–10 year old boys and girls. *Journal of Abnormal Child Psychology, 37*(2), 153-167.

Tye, C., Asherson, P., Ashwood, K. L., Azadi, B., Bolton, P., & McLoughlin, G. (2014). Attention and inhibition in children with ASD, ADHD and co-morbid ASD + ADHD: an event-related potential study. *Psychological Medicine, 44*(5), 1101-1116.

Uchida, R. R., Del-Ben, C. M., Santos, A. C., Araujo, D., Crippa, J. A., Guimaraes, F. S., & Graeff, F. G. (2003). Decreased left temporal lobe volume of panic patients measured by magnetic resonance imaging. *Brazilian Journal of Medical and Biological Research, 36*(7), 925-929.

Ulloa, L., & Messmer, D. (2006). High-mobility group box 1 (HMGB1) protein: friend and foe. *Cytokine and Growth Factor Reviews, 17*(3), 189-201.

Ulloa, R., Sánchez, S., Sauceda, J., & Ortiz, S. (2006). Psychopathology associated to attention deficit hyperactivity disorder in school age children. *Actas Espanolas de Psiquiatria, 34*(5), 330-335.

Ushijima, H., Usami, M., Saito, K., Kodaira, M., & Ikeda, M. (2012). Time course of the development of depressive mood and oppositional defiant behavior among boys with attention deficit hyperactivity disorder: differences between subtypes. *Psychiatry and Clinical Neuroscience, 66*(4), 285-291.

Vadum, A. C., & Rankin, N. O. (1998). *Psychological research: Methods for discovery and validation*. New York: McGraw-Hill.

Vakalopoulos, C. (2007a). Neurocognitive deficits in major depression and a new theory of ADHD: a model of impaired antagonism of cholinergic-mediated prepotent behaviours in monoamine depleted individuals. *Medical Hypotheses, 68*(1), 210-221.

Valera, E. M., Faraone, S. V., Murray, K. E., & Seidman, L. J. (2007). Meta-analysis of structural imaging findings in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 61*(12), 1361-1369.

van der Meer, D., Hartman, C. A., Richards, J., Bralten, J. B., Franke, B., Oosterlaan, J., Heslenfeld, D. J., Faraone, S. V., Buitelaar, J. K., & Hoekstra, P. J. (2014). The serotonin transporter gene polymorphism 5-HTTLPR moderates the effects of stress on attention-deficit/hyperactivity disorder. *Journal of Child Psychology and Psychiatry, 55*(12), 1363-1371.

van der Oord, S., Prins, P. J., Oosterlaan, J., & Emmelkamp, P. M. (2006). The association between parenting stress, depressed mood and informant agreement in ADHD and ODD. *Behaviour Research and Therapy, 44*(11), 1585-1595.

van Dyk, L., Springer, P., Kidd, M., Steyn, N., Solomons, R., & van Toorn, R. (2015). Familial-Environmental Risk Factors in South African Children With Attention-Deficit Hyperactivity Disorder (ADHD): A Case-Control Study. *Journal of Child Neurology, 30*(10), 1327-1332.

van Hulst, B. M., de Zeeuw, P., Bos, D. J., Rijks, Y., Neggers, S. F., & Durston, S. (2017). Children with ADHD symptoms show decreased activity in ventral striatum during the anticipation of reward, irrespective of ADHD diagnosis. *Journal of Child Psychology and Psychiatry, 58*(2), 206-214.

van Lang, N. D., Ferdinand, R. F., Ormel, J., & Verhulst, F. C. (2006). Latent class analysis of anxiety and depressive symptoms of the Youth Self-Report in a general population sample of young adolescents. *Behaviour Research and Therapy, 44*(6), 849-860.

Van Meter, A. R., Moreira, A. L., & Youngstrom, E. A. (2011). Meta-analysis of epidemiologic studies of pediatric bipolar disorder. *Journal of Clinical Psychiatry, 72*(9), 1250-1256.

Vella, G., Aragona, M., & Alliani, D. (2000). The complexity of psychiatric comorbidity: a conceptual and methodological discussion. *Psychopathology, 33*(1), 25-30.

Verlinden, M., Jansen, P. W., Veenstra, R., Jaddoe, V. W., Hofman, A., Verhulst, F. C., Shaw, P., & Tiemeier, H. (2015). Preschool Attention-Deficit/Hyperactivity and Oppositional Defiant Problems as Antecedents of School Bullying. *Journal of the American Academy of Child and Adolescent Psychiatry, 54*(7), 571-579.

Vermetten, E., Vythilingam, M., Southwick, S. M., Charney, D. S., & Bremner, J. D. (2003). Long-term treatment with paroxetine increases verbal declarative memory and hippocampal volume in posttraumatic stress disorder. *Biological Psychiatry, 54*(7), 693-702.

Vidal-Ribas, P., Brotman, M. A., Valdivieso, I., Leibenluft, E., & Stringaris, A. (2016). The status of irritability in psychiatry: a conceptual and quantitative review.

Journal of the American Academy of Child & Adolescent Psychiatry, 55(7), 556-570.

Videbech, P., Ravnkilde, B., Fiirgaard, B., Clemmensen, K., Egander, A., Rasmussen, N. A., Christensen, T., Sangill, R., & Rosenberg, R. (2001). Structural brain abnormalities in unselected in-patients with major depression. *Acta Psychiatrica Scandinavica, 103*(4), 282-286.

Visser, S. N., Zablotsky, B., Holbrook, J., Danielson, M., & Bitsko, R. (2015). Diagnostic experiences of children with attention-deficit/hyperactivity disorder. *National Health Statistics Reports,* 81, 1-7.

Vloet, T. D., Konrad, K., Herpertz-Dahlmann, B., Polier, G. G., & Günther, T. (2010). Impact of anxiety disorders on attentional functions in children with ADHD. *Journal of Affective Disorders, 124*(3), 283-290.

Volk, H. E., Neuman, R. J., & Todd, R. D. (2005). A systematic evaluation of ADHD and comorbid psychopathology in a population-based twin sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 44*(8), 768-775.

Vollebregt, M. A., van Dongen-Boomsma, M., Slaats-Willemse, D., & Buitelaar, J. K. (2014). What future research should bring to help resolving the debate about the efficacy of EEG-neurofeedback in children with ADHD. *Frontiers in Human Neuroscience, 8*, 321, 1-6.

Wait, J. W. V., Stanton, L., & Schoeman, J. F. (2002). Tuberculosis Meningitis and Attention Deficit Hyperactivity Disorder in Children. *Journal of Tropical Pediatrics, 48*(5), 294-299.

Wamulugwa, J., Kakooza, A., Kitaka, S.B., Nalugya, J., Kaddumukasa, M., Moore, S., Sajatovic, M., & Katabira, E. (2017). Prevalence and associated factors of attention deficit hyperactivity disorder (ADHD) among Ugandan children; a cross-sectional study. *Child and Adolescent Psychiatry and Mental Health*, 11(1), 18.

Watkins, K. E., Paus, T., Lerch, J. P., Zijdenbos, A., Collins, D. L., Neelin, P., Taylor, J., Worsley, K. J., & Evans, A. C. (2001). Structural asymmetries in the human brain: a voxel-based statistical analysis of 142 MRI scans. *Cerebral Cortex, 11*(9), 868-877.

Wechsler, D. (1974). In *Wechsler Intelligence Scale for Children – Revised*. Norwegian Psychology Association: Trondheim.

Welman, C., Kruger, S. and Mitchell, B. (2006). *Research methodology*, Cape Town: Oxford University Press SA

West, W., & Byrne, J. (2009). Some ethical concerns about counselling research. *Counselling Psychology Quarterly, 22*(3), 309-318.

Wheeler, J., & Carlson, C. L. (1994). The social functioning of children with ADD with hyperactivity and ADD without hyperactivity: A comparison of their peer relations and social deficits. *Journal of Emotional and Behavioral Disorders, 2*(1), 2-12.

Whelan, Y. M., Stringaris, A., Maughan, B., & Barker, E. D. (2013). Developmental continuity of oppositional defiant disorder subdimensions at ages 8, 10, and 13 years and their distinct psychiatric outcomes at age 16 years. *Journal of the American Academy of Child & Adolescent Psychiatry, 52*(9), 961-969.

Whitaker Sena, J. D., Lowe, P. A., & Lee, S. W. (2007). Significant Predictors of Test Anxiety Among Students With and Without Learning Disabilities. *Journal of Learning Disabilities, 40*(4), 360-376.

World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research* (Vol. 2). World Health Organization.

World Health Organization. (2016). WHO handbook for guideline development. 2012. *Geneva: World Health Organization*.

Wichstrøm, L., Berg-Nielsen, T. S., Angold, A., Egger, H. L., Solheim, E., & Sveen, T. H. (2012). Prevalence of psychiatric disorders in preschoolers. *Journal of Child Psychology and Psychiatry, 53*(6), 695-705.

Wienbruch, C., Paul, I., Bauer, S., & Kivelitz, H. (2005). The influence of methylphenidate on the power spectrum of ADHD children - an MEG study. *Biomed Central of Psychiatry, 5 (1)*, 29

Wigal, S. B., & Wigal, T. L. (2007). Special considerations in diagnosing and treating attention-deficit/hyperactivity disorder. *Central Nervous System spectrums, 12*(9), 1-16.

Wilens, T. E., Biederman, J., & Spencer, T. J. (2002). Attention deficit/hyperactivity disorder across the lifespan. *Annual Review of Medicine, 53*, 11 (1) 3-131.

Wilens, T. E., Hammerness, P., Utzinger, L., Schillinger, M., Georgiopoulous, A., Doyle, R. L., Martelon, M., & Brodziak, K. (2009). An Open Study of Adjunct OROS-Methylphenidate in Children and Adolescents Who Are Atomoxetine Partial Responders: I. Effectiveness. *Journal of Child and Adolescent Psychopharmacology, 19*(5), 485-492.

Willcutt, E. G. (2008). The Etiology of ADHD: Behavioral and Molecular Approaches. *Handbook of Cognitive and Affective Neuroscience of Psychopathology*.

Willcutt, E. G., Betjemann, R. S., McGrath, L. M., Chhabildas, N. A., Olson, R. K., DeFries, J. C., & Pennington, B. F. (2010). Etiology and neuropsychology of comorbidity between RD and ADHD: the case for multiple-deficit models. *Cortex, 46*(10), 1345-1361.

Willcutt, E. G., Nigg, J. T., Pennington, B. F., Solanto, M. V., Rohde, L. A., Tannock, R., Loo, S. K., Carlson, C. L., McBurnett, K., & Lahey, B. B. (2012). Validity of DSM-IV attention deficit/hyperactivity disorder symptom dimensions and subtypes. *Journal of Abnormal Psychology, 121*(4), 991.

Williams, N. M., Zaharieva, I., Martin, A., Langley, K., Mantripragada, K., Fossdal, R., Stefansson, H., Stefansson, K., Magnusson, P., Gudmundsson, O. O., Gustafsson, O., Holmans, P., Owen, M. J., O'Donovan, M., & Thapar, A. (2010). Rare chromosomal deletions and duplications in attention-deficit hyperactivity disorder: a genome-wide analysis. *Lancet, 376*(9750), 1401-1408.

Wong, C. G., & Stevens, M. C. (2012). The effects of stimulant medication on working memory functional connectivity in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 71*(5), 458-466.

Woo, B. S., & Rey, J. M. (2005). The validity of the DSM-IV subtypes of attention-deficit/hyperactivity disorder. *Australian & New Zealand Journal of Psychiatry, 39*(5), 344-353.

Yan, J. H., & Thomas, J. R. (2002). Arm movement control: differences between children with and without attention deficit hyperactivity disorder. *Research Quarterly for Exercise and Sport*, *73*(1), 10-18.

Yan, J. H., Stelmach, G. E., Thomas, K. T., & Thomas, J. R. (2003). Developmental differences in children's ballistic aiming movements of the arm. *Perceptual and Motor Skills*, *96*(2), 589-598.

Yeo, R. A., Hill, D. E., Campbell, R. A., Vigil, J., Petropoulos, H., Hart, B., Zamora, L., & Brooks, W. M. (2003). Proton magnetic resonance spectroscopy investigation of the right frontal lobe in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(3), 303-310.

Yin, H. H., Knowlton, B. J., & Balleine, B. W. (2004). Lesions of dorsolateral striatum preserve outcome expectancy but disrupt habit formation in instrumental learning. *European Journal of Neuroscience*, *19*(1), 181-189.

Yorbik, O., & Birmaher, B. (2003). Pharmacological treatment of anxiety disorders in children and adolescents. *Bulletin of Clinical Psychopharmacology*, *13*, 133-141.

Youngstrom, E., Loeber, R., & Stouthamer-Loeber, M. (2000). Patterns and correlates of agreement between parent, teacher, and male adolescent ratings of externalizing and internalizing problems. *Journal of Consultant and Clininical Psychology, 68*(6), 1038-1050.

Zeegers, I., Rabie, H., Swanevelder, S., Edson, C., Cotton, M., & Van Toorn, R. (2009). Attention deficit hyperactivity and oppositional defiance disorder in HIV-infected South African children. *Journal of Tropical Pediatrics, 56*(2), 97-102.

Zhou, K., Dempfle, A., Arcos-Burgos, M., Bakker, S. C., Banaschewski, T., Biederman, J., Buitelaar, J., Castellanos, F. X., Doyle, A., & Ebstein, R. P. (2008). Meta-analysis of genome-wide linkage scans of attention deficit hyperactivity disorder. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 147*(8), 1392-1398.

Zuddas, A., Ancilletta, B., Muglia, P., & Cianchetti, C. (2000). Attention-deficit/hyperactivity disorder: a neuropsychiatric disorder with childhood onset. *European Journal of Paediatric Neurology, 4*(2), 53-62.

APPENDICES

Appendix A: Teacher/ Parent DBD Rating Scale (English)

Child's Code: _____ Form completed by: _____

Sex: M/F: _____ Age: _____ School: _____

Grade: _____ Date Completed: _____

Home language: English /Afrikaans/ N-Sotho/ Xitsonga/ Tshivenda/ Setswana/Sesotho isiZulu/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 (0)	Just a Little (1)	Pretty Much (2)	Very Much (3)	
1. often interrupts or intrudes on others (e.g, butts into conversations or games)					H/I
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)					CD
3. often argues with adults					ODD
4. often lies to obtain goods or favours to avoid obligations (i.e., "cons others)					CD
5. often initiates physical fights with other members of his or her household					CD
6. has been physically cruel to people					CD
7. often talks excessively					H/I
8. has stolen items of nontrivial value without confronting a victim (e.g. shoplifting, but without breaking and entering; forgery)					CD
9. is often easily distracted by extraneous stimuli					Inatt
10. often truant from school, beginning before age 13 years					CD
11. often fidgets with hands or feet or squirms in seat					H/I
12. is often spiteful or vindictive					ODD
13. often blames others for his or her mistakes or misbehaviour					ODD
14. has deliberately destroyed others' property (other than by fire setting)					CD
15. often actively defies or refuses to comply with adults' request or rules					ODD
16. often does not seem to listen when spoken to directly					Inatt
17. often blurts out answers before questions have been completed					H/I
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)					CD

19. often has difficulty playing or engaging in leisure activities quietly					H/I
	Not at all (0)	**Just a little (1)**	**Pretty much (2)**	**Very much (3)**	
20. often fails to give close attention to details or makes careless mistakes in schoolwork, work or other activities)					Inatt
21. is often angry and resentful					ODD
22. often leaves seat in classroom or in other situations in which remaining seated is expected					H/I
23. is often touchy or easily annoyed by others					ODD
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)					Inatt
25. often looses temper					ODD
26. often has difficulty sustaining attention in tasks or play activities					Inatt
27. often has difficulty awaiting turn					H/I
28. has forced someone into sexual activity					CD
29. often bullies, threatens, or intimidate others					CD
30. is often “on the go” or often acts as “if driven by a motor”					H/I
31. often loses things necessary for tasks or activities (e.g toys, school assignments, pencils, books, or tools)					Inatt
32. often runs about or climbs excessively in situations in which it is inappropriate					H/I
33. has been physically cruel to animals					CD
34. often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)					Inatt
35. often stays out at night despite parental prohibitions, beginning before age 13 years					CD
36. often deliberately annoys people					ODD
37. has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)					CD
38. has deliberately engaged in fire setting with the intention of causing serious damage					CD
39. often has difficulty organising tasks and activities					Inatt
40. has broken into someone else’s house, building, or car					CD
41. is often forgetful in daily activities					Inatt
42. has used a weapon that can cause serious physical harm to others (e.g. a bat, brick, broken bottle, knife, gun.					CD

Total score
H/I _____
Inatt _____
ODD _____
CD _____

Appendix B: Teacher/ Parent DBD Rating Scale (Original Scale)

Child's Name: _____Form Completed by: _____

Grade: _____Date of Birth: _____Sex:_____DateCompleted_____

Check the column that best describes your/this child. Please write DK next to any items for which you don't 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 favors or 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 engages in physically dangerous activities without considering possible consequences (not for the purpose of thrill-seeking), e.g., runs into street without looking				
11. often truant from school, beginning before age 13 years				
12. often fidgets with hands or feet or squirms in seat				
13. is often spiteful or vindictive				
14. often swears or uses obscene language				
15. often blames others for his or her mistakes or misbehavior				
16. has deliberately destroyed others' property (other than by fire setting)				
17. often actively defies or refuses to comply with adults' requests or rules				
18. often does not seem to listen when spoken to directly				
19. often blurts out answers before questions have been completed				
20. often initiates physical fights with others who do not live in his or her household (e.g., peers at school or in the neighborhood)				

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

Appendx C: Teacher/ Parent DBD Rating Scale (Sepedi)

Khoutu ya ngwana…….....Bong…….E tladitswe ke……

Letsatsi leo e tladitswego ka lona……Mengwaga……

Polelo ya ka gae ……..

Mphato……

Lebedisisa kholomo yeo e hlalosago ngwana wa gago gabotsebotse. Ngwala GT mo o bonago o se na karabo ya maleba.

Ga ke tsebe-GT

	Le gatee	Gannyane	kudu	Kudukudu (Go feta tekanyo
1. O fela a tsenatsena babangwe ganong				
2. O kile a lala malalatle gabedi gomme a boa morago ga lebaka le letelele nakong ya ge a le ka fase ga tlhokomela ya bafepi (mohlokomedi yo mongwe)				
3. O rata go ngangisana le ba bagolo				
4. Gatsi o bolela maaka go rela lerato, go hwetsa seo a se nyakago goba go efoga maikarabelo				
5. Gantsi o fetla ntwa ka lapeng				
6. O kile a sorofatsa ba bangwe				
7. O bolela go feta tekanyo				
8 O kile a utswetsa yo mongwe thoto ye bohlokwa ntle le go ikopanya le motho yoo (mohlala, go utswa ka lebenkeleng ka ntlele go pshatla goba go tsena , go utswa ka bofora				
9. Gantshi sedi ya gagwe e tsewa ke tseo di sa mo amego				
10. Gantshi o ikamanya le ditiro tse kotsi ntle le go naganisisa ka ditlamorago tsa ditiro tseo (e se k age a katana le go ithabisa), mohlala , o kgona go tsena				
11.O na le moya wa botefeletso goba go gobatsa ba bangwe				
12.Gantsi o rogana goba o dirisa polelo ya go befa				
13.Gantsi o sola ba bangwe ka diphoso tsa gagwe				
14.O sentse dithoto tsa ba bangwe ka boomo				
15.Gantsi o tshela ditaelo goba a latola dikgopelo tsa ba bagolo				
16.Gantsi ga a bontshe a theeleditse ge babangwe ba bolela le yena				
17. O katana le go fetola motho pele				

18. Gantsi o kwana le go rumula bao bas a tswego thoko ya gabo, kudu sekolong le mo motseng				
19. Gantsi ga a kgone go bapala ka setu				
20.Gantsi o sitwa go fa mosomo sedi goba go dira diphoso tsa bosilo mosomong wa gagwe wa sekolo,ka gae le mabaleng a mangwe				
21.O dula a befetswe a ithumutse				
22.Gantsi o tlogela setulo sa gagwe le ge go nyakega gore a dule fase				
23.Ka mehla o befedisa ba bangwe				
24.Gantsi ga a kgone go latela ditaelo le go fetsa mosomo wa gagwe ka tshwanelo, e k aba ka gae, sekolong goba mosomong (e se k age a sa kwisise)				
25.Ga a kgone go itshwara, o befelwa ka pela				
26.Ga a kgone go fa mosomo sedi				
27.Ga a kgone go emela nako ya papadi yeo e mo lebanego				
28.O kile a gapeletsa go robalana le yo mongwe				
29.Gantsi o hlakisa ba bangwe, goba a ba tshosetsa				
30.O dula ale lebelong, goba o bonala a potlakile nke o a rakediswa				
31.Gantsi o timeditse dilo tse bohlokwa tseo di swanetsego go somiswa go phetha mosomo goba dikarolo (mohlala, dibapadisane tsa bana(diralokiswa), mesomo ya sekolo, diphentshele, dipuku didiriswa tse dingwe				
32. Gantsi o a kitima goba o namela le mo go sa nyakegego.(go merojana gob aba bagolo, se se ka hwetsago mabakeng a ge bas a iketla moyeng)				
33. O kile a sorofatsa diphoofolo				
34.Gantsi ga a nyake mosomo wo o mo gapeletsago go nagana bjalo ka mosomo wa sekolo				
35.Gantsi o thomile pele a eba le mengwaga e 13 go hlokomologa dikeletso tse batswadi bam o fago mabapi le go sepela boshego				
36.Gantsi o kgopisa batho ka boomo				
37. O kile a utswa ka go ikopanya le mothoyoo a mo utswetsago e ka ba go phamola dikanapa, go tseela motho				
38. O kile a tshuma hlaga ka maikemisetso a bosenyi				
39.Gantshi ga a kgone go beakanya mosomo wa gagwe ka tshwanelo				
40. O kile a pshatla le go tsena ntlong, moagong goba koloing ya o mongwe				
41. Gantsi o na le go lebala mesomo ya gagwe yeo a swanetsego go e dira letsatsi ka letsatsi				
42. O kile a dirisa sebetsa se se ka gobatsago ba bangwe bjalo ka mpheng, sethunya goba mphaka				

Appendix D: Beck Youth Inventories 2nd Edition for Children and Adolescents

COMBINATION BOOKLET

Please read instructions at the top of each inside page.

Background Information
Code:_____ Date of Birth_____
Today's date:_____ Location:_____
Sex: ☐ Female ☐ Male Grade:_____
ID:_____
Parent/Guardian
Name:_____

Notes

Here is a list of things that happen to people and that people think or fee. Read each sentence carefully, and circle the one word (Never, Sometimes, Often, or Always) that tells about you best.

THERE ARE NO RIGHT OR WRONG ANSWERS.

	0	1	2	3
1. I work hard.	**Never**	**Sometimes**	**Often**	**Always**
2. I feel strong.	**Never**	**Sometimes**	**Often**	**Always**
3. I like myself.	**Never**	**Sometimes**	**Often**	**Always**
4. People want to be with me.	**Never**	**Sometimes**	**Often**	**Always**
5. I am just as good as the other kids.	**Never**	**Sometimes**	**Often**	**Always**
6. I feel normal.	**Never**	**Sometimes**	**Often**	**Always**
7. I am a good person.	**Never**	**Sometimes**	**Often**	**Always**
8. I do things well.	**Never**	**Sometimes**	**Often**	**Always**
9. I can do things without help.	**Never**	**Sometimes**	**Often**	**Always**
10. I feel smart.	**Never**	**Sometimes**	**Often**	**Always**
11. People think I am good at things.	**Never**	**Sometimes**	**Often**	**Always**
12. I am kind to others.	**Never**	**Sometimes**	**Often**	**Always**
13. I feel like a nice person.	**Never**	**Sometimes**	**Often**	**Always**
14. I am good at telling jokes.	**Never**	**Sometimes**	**Often**	**Always**
15. I am at remembering things.	**Never**	**Sometimes**	**Often**	**Always**
16. I tell the truth.	**Never**	**Sometimes**	**Often**	**Always**
17. I feel proud of the things I do.	**Never**	**Sometimes**	**Often**	**Always**
18. I am a god thinker.	**Never**	**Sometimes**	**Often**	**Always**
19. I like my body.	**Never**	**Sometimes**	**Often**	**Always**
20. I am happy to be me.	**Never**	**Sometimes**	**Often**	**Always**

BSCI-Y

Total RS

Here is a list of things that happen to people and that people think or fee. Read each sentence carefully, and circle the one word (Never, Sometimes, Often, or Always) that tells about you best.

THERE ARE NO RIGHT OR WRONG ANSWERS.

	0	1	2	3
21. I worry someone might hurt me.	**Never**	**Sometimes**	**Often**	**Always**
22. My drams scare me.	**Never**	**Sometimes**	**Often**	**Always**
23. I worry when I am at school.	**Never**	**Sometimes**	**Often**	**Always**
24. I think about scary things.	**Never**	**Sometimes**	**Often**	**Always**
25. I worry people might tease me.	**Never**	**Sometimes**	**Often**	**Always**
26. I am afraid that I will make mistakes.	**Never**	**Sometimes**	**Often**	**Always**
27. I get nervous.	**Never**	**Sometimes**	**Often**	**Always**
28. I am afraid I might get hurt.	**Never**	**Sometimes**	**Often**	**Always**
29. I worry I might get bad grades.	**Never**	**Sometimes**	**Often**	**Always**
30. I worry about the future.	**Never**	**Sometimes**	**Often**	**Always**
31. My hands shake.	**Never**	**Sometimes**	**Often**	**Always**
32. I worry I might go crazy.	**Never**	**Sometimes**	**Often**	**Always**
33. I worry people might get mad at me.	**Never**	**Sometimes**	**Often**	**Always**
34. I worry I might lose control.	**Never**	**Sometimes**	**Often**	**Always**
35. I worry.	**Never**	**Sometimes**	**Often**	**Always**
36. I have problems sleeping.	**Never**	**Sometimes**	**Often**	**Always**
37. My heart ponds.	**Never**	**Sometimes**	**Often**	**Always**
38. I get shaky.	**Never**	**Sometimes**	**Often**	**Always**
39. I am afraid that something bad might happen to me	**Never**	**Sometimes**	**Often**	**Always**
40. I am afraid that I might get sick.	**Never**	**Sometimes**	**Often**	**Always**

BAI-Y Total RS

Here is a list of things that happen to people and that people think or fee. Read each sentence carefully, and circle the one word (Never, Sometimes, Often, or Always) that tells about you best.

THERE ARE NO RIGHT OR WRONG ANSWERS.

	0	1	2	3
41. I think that my life is bad.	**Never**	**Sometimes**	**Often**	**Always**
42. I have trouble doing things.	**Never**	**Sometimes**	**Often**	**Always**
43. I feel that I am a bad person.	**Never**	**Sometimes**	**Often**	**Always**
44. I wish I were dead.	**Never**	**Sometimes**	**Often**	**Always**
45. I have trouble sleeping.	**Never**	**Sometimes**	**Often**	**Always**
46. I feel no one loves me.	**Never**	**Sometimes**	**Often**	**Always**
47. I think bad things happens because of me.	**Never**	**Sometimes**	**Often**	**Always**
48. I feel lonely.	**Never**	**Sometimes**	**Often**	**Always**
49. My stomach hurts.	**Never**	**Sometimes**	**Often**	**Always**
50. I feel like bad things happen to me.	**Never**	**Sometimes**	**Often**	**Always**
51. I feel like am stupid.	**Never**	**Sometimes**	**Often**	**Always**
52. I feel sorry for myself.	**Never**	**Sometimes**	**Often**	**Always**
53. I think I do things badly.	**Never**	**Sometimes**	**Often**	**Always**
54. I feel bad about what to do.	**Never**	**Sometimes**	**Often**	**Always**
55. I hate myself.	**Never**	**Sometimes**	**Often**	**Always**
56. I want to be alone.	**Never**	**Sometimes**	**Often**	**Always**
57. I feel like crying.	**Never**	**Sometimes**	**Often**	**Always**
58. I feel sad.	**Never**	**Sometimes**	**Often**	**Always**
59. I feel empty inside.	**Never**	**Sometimes**	**Often**	**Always**
60. I think my life will be bad.	**Never**	**Sometimes**	**Often**	**Always**

BDI-Y Total RS

Here is a list of things that happen to people and that people think or fee. Read each sentence carefully, and circle the one word (Never, Sometimes, Often, or Always) that tells about you best.

THERE ARE NO RIGHT OR WRONG ANSWERS

	0	1	2	3
61. I think that my life is bad.	**Never**	**Sometimes**	**Often**	**Always**
62. I feel like screaming.	**Never**	**Sometimes**	**Often**	**Always**
63. I think people are unfair to me.	**Never**	**Sometimes**	**Often**	**Always**
64. I think people try to hurt me.	**Never**	**Sometimes**	**Often**	**Always**
65. I think my life is unfair.	**Never**	**Sometimes**	**Often**	**Always**
66. People bully me.	**Never**	**Sometimes**	**Often**	**Always**
67. People make me mad.	**Never**	**Sometimes**	**Often**	**Always**
68. I think people bother me.	**Never**	**Sometimes**	**Often**	**Always**
69. I get mad. I stay mad.	**Never**	**Sometimes**	**Often**	**Always**
70. When I get mad, I stay mad.	**Never**	**Sometimes**	**Often**	**Always**
71. When I get mad, I have trouble getting over it.	**Never**	**Sometimes**	**Often**	**Always**
72. I think people try to put me down.	**Never**	**Sometimes**	**Often**	**Always**
73. I feel people try to put me down.	**Never**	**Sometimes**	**Often**	**Always**
74. I feel mean.	**Never**	**Sometimes**	**Often**	**Always**
75. I feel like exploding.	**Never**	**Sometimes**	**Often**	**Always**
76. I think people are against me.	**Never**	**Sometimes**	**Often**	**Always**
77. I get angry.	**Never**	**Sometimes**	**Often**	**Always**
78. When I get mad, I feel mad inside my body	**Never**	**Sometimes**	**Often**	**Always**
79. I hate people.	**Never**	**Sometimes**	**Often**	**Always**
80. I get mad.	**Never**	**Sometimes**	**Often**	**Always**

BANI-Y
Total RS

Here is a list of things that happen to people and that people think or fee. Read each sentence carefully, and circle the one word (Never, Sometimes, Often, or Always) that tells about you best.

THERE ARE NO RIGHT OR WRONG ANSWERS.

	0	1	2	3
81. I steal.	**Never**	**Sometimes**	**Often**	**Always**
82. Other people get me into trouble.	**Never**	**Sometimes**	**Often**	**Always**
83. I think about running away from home.	**Never**	**Sometimes**	**Often**	**Always**
84. I do mean things.	**Never**	**Sometimes**	**Often**	**Always**
85. I break into cars, houses, or other places.	**Never**	**Sometimes**	**Often**	**Always**
86. I fight with others.	**Never**	**Sometimes**	**Often**	**Always**
87. I like getting people mad.	**Never**	**Sometimes**	**Often**	**Always**
88. I skip school.	**Never**	**Sometimes**	**Often**	**Always**
89. I hate listening to people.	**Never**	**Sometimes**	**Often**	**Always**
90. I argue with adults.	**Never**	**Sometimes**	**Often**	**Always**
91. I hurt people.	**Never**	**Sometimes**	**Often**	**Always**
92. I like being mean to others.	**Never**	**Sometimes**	**Often**	**Always**
93. I break the rules.	**Never**	**Sometimes**	**Often**	**Always**
94. I like it when people are scared of me.	**Never**	**Sometimes**	**Often**	**Always**
95. I like to hurt animals.	**Never**	**Sometimes**	**Often**	**Always**
96. I like to bully others.	**Never**	**Sometimes**	**Often**	**Always**
97. I tell lies.	**Never**	**Sometimes**	**Often**	**Always**
98. I like to trick people.	**Never**	**Sometimes**	**Often**	**Always**
99. I break things when I am mad.	**Never**	**Sometimes**	**Often**	**Always**
100. I swear at adults.	**Never**	**Sometimes**	**Often**	**Always**

BDBI-Y
Total RS

Appendix E: Consent letter to parents/ gardian

P.O.BOX 1555
LEBOWAKGOMO
0737

E-mail: Morongwa.takalani@univen.ac.za
Cell: 083 716 2921

Dear Parents/Guardian

Attention Deficit Hyperactivity Disorders (ADHD) is one of the common diagnosed neuro-cognitive behavioral developmental disorder among school going–age children. The child often has difficulties in paying attention, controlling his or her activity and is impulsive. Often children have problems in coping with their schoolwork and may not be getting along with other children. They are also unable to finish assignment without constant supervision and cause disruption in the family. ADHD therefore continues to pose a complex problem to sufferers, parents, caregivers and teachers.

Children diagnosed with ADHD were observed to have one or more psychiatric disorders. ADHD is found to be in co- existence with internalising disorders i.e. Anxiety and Depression which can be seen as having an impact on behaviour and academic functioning.

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. All these problems may be as a results of overlapping symptoms between ADHD and comorbid internalizing disorders (anxiety and depression).

The share symptoms in both ADHD and internalizing disorders in return becomes a challenge in assessing and treating these individuals. These children as a result are often mistakenly regarded as having low intellectual functioning hence being referred for further assessment or scholastic placement by the teachers.

Please contact me on the number above should you be agree in letting your child participate in this project. The research will be done in a form of completing a questionnaire.

The information will be used for statistical analysis only and in no circumstances will the identity of the child and the school be disclosed. The final report will be made available to all interested. Should you wish to consult us on any problems your child is experiencing, further referral to the Clinical Psychologist at hospital will be done where a child will be assisted.

You and your child's participation in this study is very important and will add value in gaining information on ADHD and comorbid internalizing disorders.

You're Sincerely

_____ _____

Signature **Date**

Appendix F: Consent form to parents/ guardian

P.O.BOX 1555
LEBOWAKGOMO
0703
Cell083716 2921

Research project on Attention Deficit Hyperactivity Disorders in Semi-urban school in Lepelle Nkumbi municipality.

I ……parent of …… give consent that my child

participate in the research study.

I have read the information on the proposed study and was provide the opportunity to ask questions about the purpose of the study. I accept and understood the content of the research study and that feedback will be given back to us as it was explained by the researcher.

I understand that participation in this research is completely voluntary and I am free to withdraw my child from participating at any given time.

I understand that responses given will remain confidential and the assessment forms will not entail names of the child for the purpose of confidentiality.

Yours Faithfully

_____	_____	_____
Signature of parent/ guardian	**Place**	**Date**

Appendix G: Foromo ya tumelelo le batswadi/ motswadi (Sepedi)

P.O.BOX 1555
LEBOWAKGOMO
0703
Cell: 083716 2921

Projeke ya dinyakišišo ka ga Attention Deficit Hyperactivity Disorder (ADHD) dikolong tša masepala wa Lepelle Nkumpi

Ke le (maina)……motswadi wa (maina a ngwana)…….. ke dumelela ngwana w aka go tšea karolo dinyakišišong tša porojeke ye.

Ke badile e bile ke sedimušitšwe ka ga di nyakišišo tša porojeke gape ka fiwa sebaka sa go botšiša diputšišo ka ga maikemišetšo a porokeke ye. Ke a amogela gape ke kwišiša maikemišetšo a projeke le gore re tla tsebišwa ka ga dipoelo bjalo k age monyakišiši a file hlathollo.

Ke a kwišiša gore bjale motswadi, ke ga ke gapeletšwe go tšea karolo, gape ke a lokologa go kgaola ngwana wa ka go tšeeng karolo nako efe goba efe.

Ke a kwišiša gore dikarabo tše ke fanago ka tšona e tlaba lekunutu gape difomo di ka se šupetše maina a ngwana.

Wa lena ka botshephegi

…… ……. …….

Motswadi **Lefelo** **letšatši**

Appendix H: Letter of request to the Department of Education

P.O.BOX 1555
LEBOWAKGOMO
0737
Cell: 083 716 2921

HEAD OF DEPARTMENT RESEARCH UNIT

Department of Education
PIETERSBURG
0700

Dear Sir/Madam

Research project on Attention Deficit Hyperactivity Disorders in Semi-urban school in Lepelle Nkumbi municipality.

Attention Deficit Hyperactivity Disorders (ADHD) is one of the common diagnosed neuro-cognitive behavioral developmental disorder among school going–age children. The disorder is mainly characterised by problems in allocating attention, regulating motor activity and controlling behavioral impulses. Attention Deficit Disorder is a disorder of the brain which as a result affects behaviour mostly displayed by these children and therefore having a serious impact on academic achievement and low social competence. ADHD therefore continues to pose a complex problem to sufferers, parents, caregivers and teachers.

Children diagnosed with ADHD were observed to reflect one or more psychiatric disorders. ADHD is found to be in co- existence with internalising disorders i.e. Anxiety and Depression which can be seen as having an impact on behaviour.

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 share symptoms in both ADHD and internalising disorders in return becomes a challenge in assessing and treating these individuals. These children as a result are often mistakenly regarded as having low intellectual functioning hence being referred for further assessment or scholastic placement by the teachers.

Method:

The research team will visit the participating schools and participated in the study. The participants will be assessed on a battery consisting of the DBD (Parent/Teacher Disruptive Behavioral Disorders Rating Scale), BDI-Y (Beck Depression Inventory for Youth) and BAI-Y (Beck Anxiety Inventory for Youth). The clinically diagnosed ADHD scores on DBD will be correlated with scores obtained on the BDI-Y and BAI-Y. The results will be analysed using the SPSS to show the comorbidity of internalising disorders i.e. depression and anxiety on ADHD symptoms.

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

Your approval of this very important study will contribute to the understanding of the impact of these internalising disorders thereof, which will enable professionals to identify and better management of children with coexisting internalised disorders.

You're Sincerely

Signature	**Date**

Appendix I: Letter of request for permission to the principal

P.O.BOX 1555
LEBOWAKGOMO
0737
Cell: 083 716 2921

The Principal

……. Primary School

Dear Sir/Madam

Research project on Attention Deficit Hyperactivity Disorders in Semi-urban school in Lepelle Nkumpi municipality.

Attention Deficit Hyperactivity Disorders (ADHD) is one of the common diagnosed neuro-cognitive behavioral developmental disorder among school going–age children. The disorder is mainly characterised by problems in allocating attention, regulating motor activity and controlling behavioural impulses. Attention Deficit Disorder is a disorder of the brain which as a result affects behaviour mostly displayed by these children and therefore having a serious impact on academic achievement and low social competence. ADHD therefore continues to pose a complex problem to sufferers, parents, caregivers and teachers.

Children diagnosed with ADHD were observed to reflect one or more psychiatric disorders. ADHD is found to be in co- existence with internalising disorders i.e. Anxiety and Depression which can be seen as having an impact on behavior.

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 share symptoms in both ADHD and internalising disorders in return becomes a challenge in assessing and treating these individuals. These children as a result are often

mistakenly regarded as having low intellectual functioning hence being referred for further assessment or scholastic placement by the teachers.

Method:

The research team will visit the participating schools and participated in the study. The participants will be assessed on a battery consisting of the DBD (Parent/Teacher Disruptive Behavioral Disorders Rating Scale), BDI-Y (Beck Depression Inventory for Youth) and BAI-Y (Beck Anxiety Inventory for Youth). The clinically diagnosed ADHD scores on DBD will be correlated with scores obtained on the BDI-Y and BAI-Y. The results will be analysed using the SPSS to show the comorbidity of internalising disorders i.e. depression and anxiety on ADHD symptoms.

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

Your approval of this very important study will contribute to the understanding of the impact of these internalising disorders thereof, which will enable professionals to identify and better management of children with coexisting internalised disorders.

You're Sincerely

_____ _____

Signature **Date**

Appendix J: Letter of permission from the Department of Education

LIMPOPO
PROVINCIAL GOVERNMENT
REPUBLIC OF SOUTH AFRICA

DEPARTMENT OF
EDUCATION

Ref: 2/2/2 Enq: MC Makola PhD Tel No: 015 290 9448 E-mail:MakolaMC@edu.limpopo.gov.za

Takalani MC
P O Box 1555
LEBOWAKGOMO
0737

RE: REQUEST FOR PERMISSION TO CONDUCT RESEARCH

1. The above bears reference.
2. The Department wishes to inform you that your request to conduct research has been approved. Topic of the research proposal: **"THE COMORBIDITY OF INTERNALIZING DISORDERS AND ATTENTION DEFICIT HYPERACTIVITY DISORDERS".**
3. The following conditions should be considered:

3.1 The research should not have any financial implications for Limpopo Department of Education.

3.2 Arrangements should be made with the Circuit Office and the schools concerned.

3.3 The conduct of research should not anyhow disrupt the academic programs at the schools.

3.4 The research should not be conducted during the time of Examinations especially the fourth term.

3.5 During the study, applicable research ethics should be adhered to; in particular the principle of voluntary participation (the people involved should be respected).

3.6 Upon completion of research study, the researcher shall share the final product of the research with the Department.

REQUEST FOR PERMISSION TO CONDUCT RESEARCH: TAKALANI MC

CONFIDENTIAL

Cnr. 113 Biccard & 24 Excelsior Street, POLOKWANE, 0700, Private Bag X9489, POLOKWANE, 0700
Tel: 015 290 7600, Fax: 015 297 6920/4220/4494

The heartland of southern Africa - development is about people!

4 Furthermore, you are expected to produce this letter at Schools/ Offices where you intend conducting your research as an evidence that you are permitted to conduct the research.

5 The department appreciates the contribution that you wish to make and wishes you success in your investigation.

Best wishes.

26/10/16

Ms NB Mutheiwana (Acting)
Head of Department

Date

REQUEST FOR PERMISSION TO CONDUCT RESEARCH: TAKALANI MC

CONFIDENTIAL

Appendix K: Letter of permission from Lebowakgomo circuit office

27. Jan. 2000 5:16 MALOPE IT No. 5480 P. 1/2

LIMPOPO
PROVINCIAL GOVERNMENT
REPUBLIC OF SOUTH AFRICA

DEPARTMENT OF EDUCATION
Lebowakgomo Circuit Office

CONFIDENTIAL

Ref.: 17/P Enquiries: Dibetso R F Tel.: 079 511 8732 Date: 29 November 2016

To: Principals of Identified Schools
Lebowakgomo Circuit
LEBOWAKGOMO

Sir/Madam

SUBJECT: REQUEST FOR PERMISSION TO CONDUCT RESEARCH: TAKALANI MC

1. The abovementioned matter refers.
2. Mrs. Takalani MC obtained permission from the Superintendent-General of Education to conduct a research in fulfillment of her studies to the following selected schools in Lebowakgomo Circuit:
   2.1. Dr. Dixon Mphahlele Primary School
   2.2. Eureka Full Service School
   2.3. Hillside Park Primary School
   2.4. Lafata Primary School
   2.5. Little Bedfordview Primary School
   2.6. Mogodumo Primary School
   2.7. Ntseekgopu Primary School
3. Your school is therefore requested to assist Mrs. Takalani with information and data she may require in order to fulfill her desired results.
4. We hope that you will cooperate with her and also thank you in advance.

Phome N D
Circuit Manager
pp

29/11/2016
Date

2016 -11- 29

DEPARTMENT OF EDUCATION,
Lebowakgomo Circuit Office
Tel 015 633 5059 Fax No. n/a

The heartland of Southern Africa -- development is about people

Appendix L: Letter of permission from Lebowakgomo circuit office

LIMPOPO
PROVINCIAL GOVERNMENT
REPUBLIC OF SOUTH AFRICA

DEPARTMENT OF EDUCATION
Lebowakgomo Circuit Office

CONFIDENTIAL

Ref.: 17/P Enquiries: Dibetso R F Tel.: 079 511 8732 Date: 01 August 2017

To: The Principals of identified Schools
Lebowakgomo Circuit
SUBJECT: REQUEST FOR PERMISSION TO CONDUCT RESEARCH: TAKALANI MC

1. The abovementioned matter refers.
2. Mrs Takalani MC obtained permission from the Suprintendent-General of Education to conduct a research in fulfillment of her studies to the following sampled schools in the circuit:
   2.1.Mogodi Primary School
   2.2.Mamaolo Primary School
   2.3.Maretlwaneng Primary School
   2.4.Huelereng Primary School
   2.5.Makgoathane Primary School
   2.6.Ndlovu Primary School
   2.7.Sekurwaneng Primary School
   2.8.Mahlasedi Special School

Phaladi V N
Circuit Manager
pp

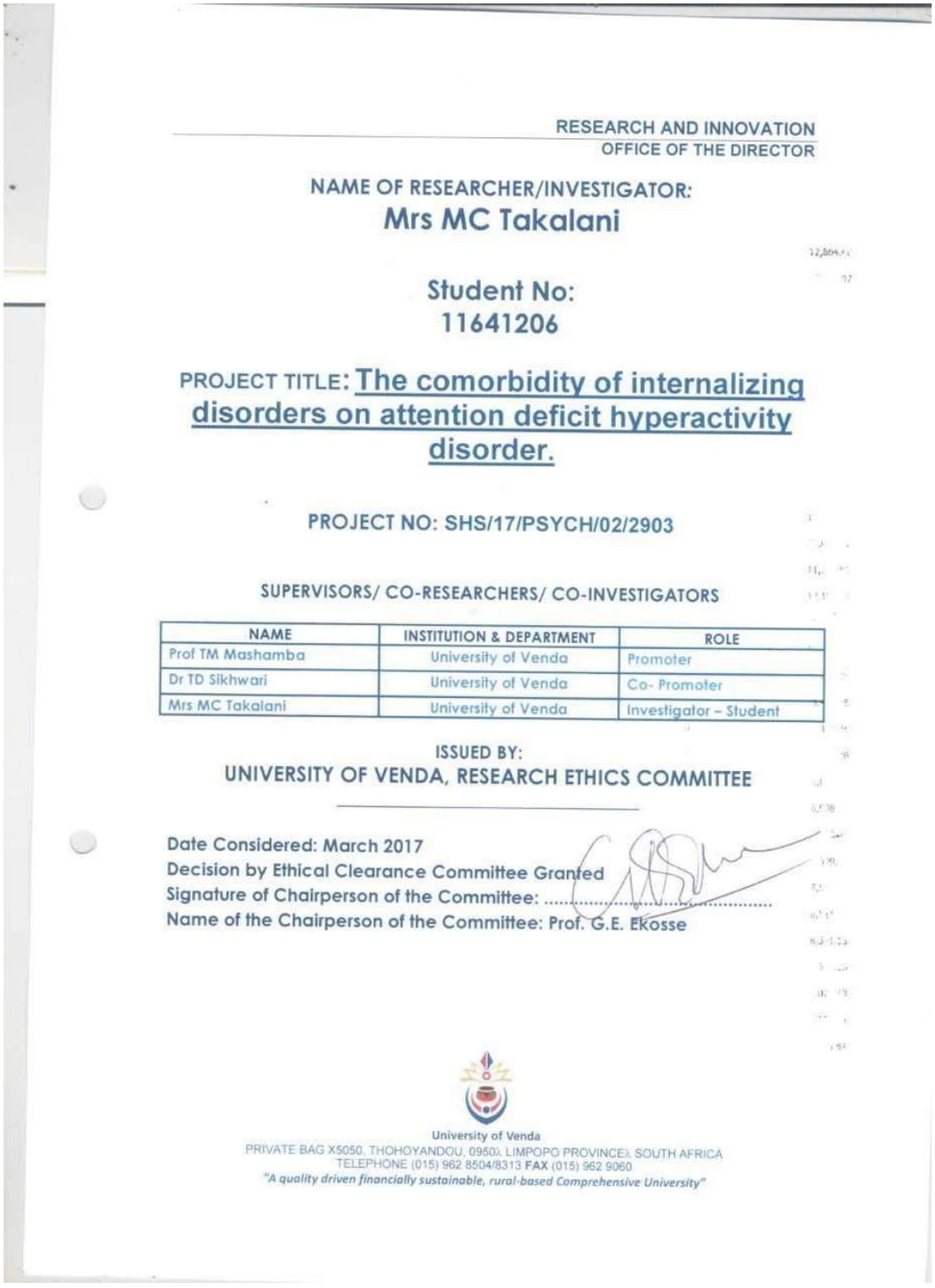
01/08/2017
Date

LIMPOPO PROVINCE
DEPARTMENT OF EDUCATION
LEBOWAKGOMO DISTRICT OFFICE
LEBOWAKGOMO CIRCUIT OFFICE
2017 -08- 01
PRIVATE BAG X25
CHUENESPOORT, 0745
KGORO YA TIUTO LIMPOPO PROVINCE

DEPARTMENT OF EDUCATION,
Lebowakgomo Circuit Office
Tel 015 633 5059 Fax No. n/a

The heartland of Southern Africa – development is about people

Appendix M: Ethical clearance certificate



RESEARCH AND INNOVATION
OFFICE OF THE DIRECTOR

NAME OF RESEARCHER/INVESTIGATOR:
Mrs MC Takalani

Student No:
11641206

PROJECT TITLE: **The comorbidity of internalizing disorders on attention deficit hyperactivity disorder.**

PROJECT NO: SHS/17/PSYCH/02/2903

SUPERVISORS/ CO-RESEARCHERS/ CO-INVESTIGATORS

NAME	INSTITUTION & DEPARTMENT	ROLE
Prof TM Mashamba	University of Venda	Promoter
Dr TD Sikhwari	University of Venda	Co- Promoter
Mrs MC Takalani	University of Venda	Investigator – Student

ISSUED BY:
UNIVERSITY OF VENDA, RESEARCH ETHICS COMMITTEE

Date Considered: March 2017
Decision by Ethical Clearance Committee Granted
Signature of Chairperson of the Committee: .....
Name of the Chairperson of the Committee: Prof. G.E. Ekosse

University of Venda
PRIVATE BAG X5050, THOHOYANDOU, 0950, LIMPOPO PROVINCE, SOUTH AFRICA
TELEPHONE (015) 962 8504/8313 FAX (015) 962 9060
"A quality driven financially sustainable, rural-based Comprehensive University"

Appendix N: Letter from language editor

P.O BOX 663

THOLONGWE

0734

02 December 2019

Dear Sir/Madam

This serves to confirm that I proof-read and edited dissertation entitled “The Comorbidity of Internalizing Disorders on Attention Deficit Hyperactivity Disorder” by Takalani M.C, student number: 11641206.

I have also suggested few amendments, provided the changes I recommended are effected to the text, the language is of an acceptable standard.

Please don’t hesitate to contact me for any enquiry.

Regards

Dr. Hlaviso Motlhaka (PhD, WITS, MA, IUP: USA)

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