ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND LOW BIRTH WEIGHT

by

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DECLARATION

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

Signature ..A Haycock.....

Date .....2005/09/15.....

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LIST OF ABBREVIATIONS

ADHD	Attention-deficit/hyperactivity disorder
AGA	Appropriate for gestational age
ANOVA	Analysis of variance
CD	Conduct disorder
CMV	Cytomegalovirus
CNS	Central nervous system
CPM	Coloured progressive matrices
DBD	Disruptive behaviour disorder
DCD	Developmental coordination disorder
DSM	Diagnostic and Statistical Manual of Mental Disorders
ELBW	Extremely low birth weight
FAS	Foetal alcohol syndrome
FBW	Full-term birth weight
HGV	Head-circumference growth velocity
HIV	Human immunodeficiency virus
IUGR	Intrauterine growth restriction
IQ	Intelligence quotient
LBW	Low birth weight
MRI	Magnetic resonance imaging
NBW	Normal birth weight
ODD	Oppositional defiant disorder
SES	Socio-economic status
SGA	Small for gestational age
ToL	Tower of London
VLBW	Very low birth weight
WCST	Wisconsin Card Sorting Test

ABSTRACT

The aim of this study was to investigate the number of attention-deficit/hyperactivity disorder (ADHD) symptoms on the disruptive behaviour disorder (DBD) rating scale among low-birth-weight (LBW) children in comparison with normal-birth-weight (NBW) children. A further aim was to establish whether LBW children have poorer performances on ADHD-sensitive tests (particularly the Tower of London Test, Maze Coordination Test and Wisconsin Card Sorting Test). The sample (110 children) was drawn from seven randomly selected urban, mainstream primary schools in the Polokwane magisterial district in South Africa. The 55 LBW children (weighing 2 000 g and below) were matched with 55 NBW children (above 3 000 g) according to age, sex, socio-economic status, language and racial group. The LBW boys and girls tended to be more vulnerable to ADHD symptoms on the DBD rating scale, particularly inattention, than their NBW counterparts. Similarly, problems with motor and executive problems were significant among the LBW boys and girls. The increased occurrence of ADHD symptoms among LBW children may be the consequence of neurological events due to being born with LBW. These children seem to have a biological predisposition to developmental difficulties, such as ADHD.

GLOSSARY

CONCEPTUAL CLARIFICATION OF TERMS

Abruptio placentae: Abruptio placentae refers to bleeding from a normally situated placenta causing its complete or partial detachment from the uterine wall after the 24th week of gestation. It is often associated with hypertension and pre-eclampsia.

Anaemia: Anaemia is a condition in which there is reduced delivery of oxygen to the tissue. Anaemia is not a disease but a symptom of any of a number of different disorders. It can be caused by factors such as poor diet, blood loss, congenital defects of haemoglobin, exposure to industrial poisons, diseases of the bone marrow, or any other disorder that upsets the balance between blood loss through bleeding or destruction of blood cells and production of blood cells.

Auto-immune disease: Auto-immune disease is one of a number of otherwise unrelated disorders caused by inflammation and destruction of tissue by the body's own antibodies (auto-antibodies).

Bicornate: Bicornate refers to having two processes or projections.

Cardiovascular: Cardiovascular relates to the heart and the blood vessels or the circulation.

Cerebral palsy: Cerebral palsy is a nonspecific term used to describe a persistent qualitative motor disorder caused by nonprogressive damage to the brain. Although manifested primarily in motor dysfunction, the disorder also may involve sensory deficits and impairment of the intellect. Most cases are diagnosed

before the age of three. However, prior to age eight or nine, function lost by damage to one part of the brain can be taken over by another part of the brain. Hence, many people consider that brain damage occurring at any time prior to this age can lead to cerebral palsy.

Erythroblastosis: Erythroblastosis is the presence in the blood of the nucleated precursors of the red blood cells (erythroblasts). This may occur when there is an increase in the rate of red cell production.

Gastrointestinal: Gastrointestinal is a term relating to the stomach and intestines.

Haematology: Haematology refers to the science dealing with the morphology of blood and blood-forming tissues, and with their physiology and pathology.

Hydrops: Hydrops is an abnormal accumulation of fluid in body tissue or cavities.

Iatrogenic: Iatrogenic is a term describing a condition that has resulted from treatment, as either an unforeseen or inevitable side-effect.

Inanition: Inanition is a condition of exhaustion caused by lack of nutrients in the blood. This may arise through starvation, malnutrition, or intestinal disease.

Intrauterine growth restriction (IUGR): IUGR is the condition resulting in the birth of a baby whose birth weight is abnormally low in relation to its gestational age (i.e. small for dates). Causes include maternal disease (e.g. infection, malnutrition, high blood pressure, smoking and alcoholism), poor socio-economic conditions, multiple pregnancies (e.g. twins) and foetal disease. It may be associated with preterm birth.

Intraventricular haemorrhage: Intraventricular haemorrhage is the extravasation of blood into the ventricular system of the brain.

In vitro fertilisation: In vitro fertilisation is the fertilisation of an ovum outside the body, the resultant zygote being incubated to the blastocyst stage and then implanted in the uterus.

Low birth weight: Babies born with low birth weight (less than 2 500 g) are born either pre-term (less than 37 completed weeks of gestation) or small for gestational age (SGA – less than 10th percentile for gestational age) or both.

Morbidity: Morbidity is the state of being diseased. Morbidity is also referred to as the ratio of sick to well in a community.

Mortality: The incidence of death in the population in a given period.

Obstetrics: Obstetrics refers to the branch of medical science concerned with the care of women during pregnancy, childbirth, and the period of about six weeks following the birth, when the reproductive organs are recovering.

Placenta: The placenta is an organ within the uterus by means of which the embryo is attached to the wall of the uterus. Its primary function is to provide the embryo with nourishment, eliminate its waste, and exchange respiratory gases. This is accomplished by the close proximity of the maternal and foetal blood systems in the placenta.

Placenta previa: Placenta previa is a placenta situated wholly or partially in the lower and noncontractile part of the uterus. When this becomes elongated and stretched during the last few weeks of pregnancy, and the cervix becomes stretched either before or during labour, placental separation and haemorrhage will occur.

Pre-eclampsia (pregnancy-induced hypertension): Pre-eclampsia is defined as high blood pressure (greater than 140/90 mmHg) developing during pregnancy in

a woman whose blood pressure was previously normal. It is often accompanied by excessive fluid retention and less often by the presence of protein in the urine.

Premature birth: Premature birth refers to the birth of a baby weighing less than 2 500 g. Usually this is indicative of preterm birth but it can also be caused by IUGR. Birth weights of less than 500 g are almost invariably incompatible with life.

Preterm birth: Preterm birth occurs when a baby is born before 37 weeks (259 days) of gestation (calculated from the first day of the mother's last menstrual period). Birth at less than 23 weeks is at present incompatible with life.

Renal: Renal relates to or affects the kidneys.

Seizure: Seizure is defined as the sudden attack or recurrence of a disease; a convulsion or attack of epilepsy. Convulsions are a series of involuntary contractions of the voluntary muscles. Convulsive seizures are symptomatic of some neurologic disorders; they are not in themselves a disease entity.

Sequelae: Sequelae are any disorders or pathological conditions that result from a preceding disease or accident.

Teratogen: Teratogen refers to any substance, agent or process that induces the formation of developmental abnormalities in a foetus. Known teratogens include drugs such as alcohol, infections such as German measles and also irradiation with X-rays and other ionising radiation.

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Chapter 1

GENERAL INTRODUCTION AND BACKGROUND

1.1 Introduction

Since 1940 hyperactive, inattentive and impulsive children have been labelled as having "minimal brain dysfunction", "brain-injured child syndrome", "hyperkinetic reaction of childhood", "hyperactive child syndrome", and, most recently, "attention-deficit disorder" (Barkley, 1998). Developmentally inappropriate levels of inattention, over-activity and impulsiveness are regarded as the main clinical symptoms of what is currently called attention-deficit/hyperactivity disorder or ADHD (Johansen et al., 2002).

ADHD causes clinically significant impairments in social, academic or occupational functioning (American Psychiatric Association, 2000). Therefore, ADHD is among the most common causes of behavioural disturbances in schools. It constitutes one of the largest groups of patients in the clientele of child psychiatrists and child neurologists (Lou, 1996).

Cantwell (1996) reports not only developmental delay in children with ADHD, but also the development of more serious psychopathology. The presence of ADHD should be considered as a marker for other psychiatric and developmental conditions, such as learning disabilities, oppositional defiant

disorder (ODD), antisocial behaviour, conduct disorder (CD), depression, anxiety, substance abuse, poor social skills and others (Accardo, 1999; Barkley et al., 2002). These functionally impairing symptoms of ADHD may continue into adolescence and adult life (Taylor et al., 1998). Cantwell (1996) notes that, if untreated, ADHD predisposes a child to psychiatric and social pathology in later life. ADHD therefore constitutes one of the strongest risk factors known for mental illness in early adult life. Increased understanding of the disorder will lead to improved and more focused approaches to intervention.

The direct and immediate causes of the difficulties experienced by children with ADHD are unknown (Barkley, 1998). According to Cantwell (1996) there is interplay of both psychosocial (such as parental care or environmental toxins) and biological factors (such as genetics or brain abnormalities) that may contribute to the syndrome of ADHD. Low birth weight (LBW) seems to be one of the various intertwined contributing factors in the development of ADHD (Astbury, Orgill & Bajuk, 1987).

LBW children are vulnerable to neurodevelopmental disturbances due to prenatal complications and neonatal risk factors (Breslau et al., 1996a; Lindahl & Michelsson, 1986).

Avery, Fletcher and MacDonald (1999) describe specific types of developmental morbidities in LBW cohorts, for example social problems, lower attention, cognitive delays (for example lower intelligence quotient [IQ]), remedial

assistance, speech and language disorders, persistent neuro-motor abnormalities and perceptual problems. Breslau, Johnson and Lucia (2001) confirm that deficits in academic areas occur through childhood and adolescence. Adults also show lower occupational success and income.

Elgen, Sommerfelt and Markestad (2002) document that 27% of the LBW children compared to 9% of normal-birth-weight (NBW) children were diagnosed with a psychiatric disorder. Harrison (2002) found that 22% of LBW children had at least one psychiatric disorder, the most common being ADHD (16%). The risk of either a diagnosis or symptoms of ADHD in premature children increases as birth weight decreases (Chapieski & Evankovich, 1997).

In general, the risk of neurodevelopmental and intellectual problems is higher as birth weight and gestational age decrease (Behrman, Kliegman & Jenson, 2000). Greater rates of behavioural problems are associated with decreasing birth weights (Harrison, 2002).

Survival of very-low-birth-weight (VLBW) infants is improving, but short-term outcomes (18 to 22 months) and longer-term outcomes (school performance) of the survivors are not improving (Jobe, 2001). As an increasing number of babies are born and continue to survive with birth weights less than 1 000 g, it is important to optimise their chances of a healthy, productive life (Campbell, 2001a; Subramanian, 2002).

1.2 Objective of the study

This research proposes to study the quantity of ADHD symptoms among LBW children in comparison with NBW children, as well as to determine whether LBW children have lower performance on ADHD-sensitive tests than NBW children.

The greater part of current literature reflects that there is a relationship between ADHD and LBW. Literature also indicates an association between greater rates of behavioural problems and decreasing birth weight. Existing data, however, do not clearly indicate the nature of the relationship between ADHD and LBW regarding inattention, over-activity and impulsiveness. Literature is also not clear about the relationship between ADHD, LBW and gender.

1.3 Purpose and significance of the study

National and international efforts are made to understand the nature and consequences of the relationship between ADHD and LBW. According to Botting et al. (1997) there is profound concern internationally about the long-term problems of very premature children who survive. Studies designed to work out ways to reduce these long-term effects are urgently needed.

Research findings obtained from this study will assist in understanding the nature and significance of ADHD symptoms in LBW children, as well as in the development of early effective intervention programmes. These programmes

should aim at continuous monitoring of development, including identification, diagnosis and treatment of problem areas. The focus of these programmes should be on preventing possible pathological symptoms from reaching a full diagnostic level, especially in the light of ADHD (and LBW) as a marker of other comorbid disorders. As mentioned in section 1.1, LBW children often have biologically based behavioural problems. Therefore, intervention programmes should be operated from a multi-professional team approach, including the child, parents, teacher, psychologist, psychiatrist, paediatrician and other significant role players. It is hoped that a reduction in long-term problems in LBW children will then be achieved.

Bregman (1998) emphasises that further research on LBW is essential in order to determine academic functioning and educational needs of children born with LBW, as well as determining other treatments (such as medical, behavioural and educational techniques). Further research is the only way to maximise the outcomes of LBW children.

1.4 Delineation of the study

Chapter two focuses on the definition of LBW in accordance with the different populations and various levels. The incidence of LBW will then be referred to. Correlation between risk factors relating to LBW and causes of LBW will further be elaborated on, with specific reference to intrauterine growth restriction (small for gestational age [SGA]) and prematurity. Reference to

outcomes of LBW in the neonatal stage will be summarised, for short-term outcomes will not be the focus of this study. The aim of the chapter is to familiarise the reader with the consequences of LBW and to refer to treatment of children born with LBW.

Chapter three provides a general discussion of ADHD. The aim of this chapter is to explore the following relevant aspects of ADHD in this study, namely defining ADHD, diagnostic criteria and assessment, incidence, gender differences, symptoms, comorbid disorders, aetiological factors, consequences of ADHD and treatment.

Chapter four reflects on available research on the relation between LBW and ADHD and attempts to explore the nature of this hypothesised relationship. The following areas are the focus of discussion: the incidence of ADHD in LBW children, biological structures involved in LBW children with ADHD, risk factors, gender, comorbidity and treatment.

Chapter five is directed at a discussion of problem formulation and hypotheses.

Chapter six focuses on the methodology of data collection.

Chapter seven reports on the results of the study.

In chapter eight the results, limitations of the study and suggestions for future research will be discussed.

Chapter 2

LOW BIRTH WEIGHT

2.1 Introduction

LBW will be defined in this chapter in accordance with the different populations and various levels. The incidence of LBW will then be referred to. Correlation between risk factors relating to LBW and causes of LBW will further be elaborated on with specific reference to intrauterine growth restriction (SGA) and prematurity. Reference to outcomes of LBW in the neonatal stage will be summarised, for short-term outcomes will not be the focus of this study. The main aim of the chapter is to familiarise the reader with the consequences of LBW. Treatment of children born with LBW will be addressed briefly.

2.2 Definition of LBW

LBW infants (Avery et al., 1999; Behrman et al., 2000) constitute the following populations:

- Appropriately grown premature infants, delivered prior to 37 weeks' completed gestation, who weigh 2 500 g or less.
- Full-term infants, born at or after 37 completed weeks' gestation who weigh 2 500 g or less. These infants are SGA, that is, they display intrauterine growth restriction (IUGR). Papageorgiou and Bardin (1999)

state that infants with IUGR are smaller than normal at any gestational age. According to Taeusch and Ballard (1998) SGA is usually defined as birth weight below the 10th percentile; at term this may vary from 2 500 g to 2 750 g. Sohl and Moore (1998) further state that at times the terms SGA and IUGR do not necessarily reflect identical clinical situations. Some perfectly normal, constitutionally small babies are below the 10th percentile for gestational age at birth, whereas some babies born above the 10th percentile exhibit growth restriction.

- Infants born both prematurely and SGA.

LBW infants form a heterogeneous group, perhaps needing separate types of medical treatment and intervention and facing different developmental outcomes (Bukatko & Daehler, 2001). According to Behrman et al. (2000) prematurity and IUGR are associated with increased neonatal morbidity and mortality.

Campbell (2001a), as well as Breslau et al. (2000), defines various levels of low birth weight as follows:

- LBW – birth weight between 1 500 g and 2 500 g
- VLBW – birth weight between 1 000 g and 1 500 g
- Extremely low birth weight (ELBW) – less than 1 000 g.

2.3 Incidence

Behrman et al. (2000) state that during 1997, 7.5% of live-born neonates in the United States weighed less than 2 500 g. Approximately 30% of LBW infants in the United States have IUGR and are born after 37 weeks. In developing countries, approximately 70% of LBW infants have IUGR. According to Sohl and Moore (1998) 4% to 8% of all infants born in developed countries have IUGR. March of Dimes (2003) reports that more than 60% of LBW babies are preterm.

2.4 Risk factors

There seems to be a correlation in the literature between risk factors and causes of LBW. Various causes of LBW will be elaborated on in section 2.5. Risk factors (Breslau et al., 1996a; Campbell, 2001a) related to LBW are many and complex, and include the following:

- Environmental (for example working conditions, violence towards women, prenatal care, poverty), housing (National Institute of Health, 1999) (for example lead paint, safety, crowding and pollution)
- Behavioural (for example smoking, nutrition, alcohol and substance use, physical activity)
- Social (for example stress, lack of social support)
- Biological (for example pregnancy under 20 or over 40 years)

- Previous premature birth
- Maternal illness, such as genital or urinary tract infections, hypertension and diabetes mellitus
- Multiple gestations: this has become more common because of treatment for infertility
- Low maternal weight gain and low pre-pregnancy weight (National Institute of Health, 1999).

2.5 Aetiology

It is difficult to separate factors associated with prematurity from those associated with IUGR. A positive correlation exists between both premature birth and IUGR and low socio-economic status (SES). Families of low SES have relatively high incidences of maternal undernutrition, anaemia, illness, inadequate prenatal care, drug addiction, obstetric complications and maternal histories of reproductive inefficiency (Behrman et al., 2000). The degree to which genetic versus environmental factors influence the variance in birth weight among various populations is difficult to determine (Behrman et al., 2000).

Aetiology of IUGR

According to Sohl and Moore (1998) and Papageorgiou and Bardin (1999) growth restriction can be classified into asymmetric or symmetric growth restriction.

Asymmetric growth restriction (usually IUGR occurring late in pregnancy) refers to a decrease in weight, but length and head size are not restricted.

In symmetric growth restriction all indices are decreased, brain and body.

This division is apparently an oversimplification. Mixed patterns of abnormal growth are often noted (Sohl & Moore, 1998).

Symmetric IUGR usually has an earlier onset and is associated with diseases that affect foetal cell numbers. These diseases refer to conditions with chromosomal, genetic, malformation, teratogenic, infectious or severe maternal hypertensive aetiologies. Asymmetric IUGR usually has a late onset and is associated with poor maternal nutrition or exacerbation of maternal vascular disease (such as pre-eclampsia, chronic hypertension) (Behrman et al., 2000). Thus, factors intrinsic to the foetus cause symmetric growth restriction, whereas external factors are associated with asymmetric growth (Papageorgiou & Bardin, 1999). The causes of IUGR (Sohl & Moore, 1998) can be grouped into intrinsic and extrinsic types, which are summarised in Table 2.1.

Table 2.1 Foetal growth restriction (Sohl & Moore, 1998 p. 92 adapted)

Intrinsic causes
Constitutional (for example parents of small stature)
Genetic (especially trisomy 18, 13 syndromes)
Toxic: alcohol, nicotine, virus, hydantion, coumarin
Infectious: TORCH (toxoplasmosis, other infections, rubella, cytomegalovirus, herpes simplex), syphilis, malaria
Teratogenic: radiation, drugs
Extrinsic causes
Decreased maternal nutrient delivery
Maternal cardiac disease
High altitude
Maternal anaemia (for example sickle cell)
Maternal starvation/inanition
Placental dysfunction
Hypertensive disease
Auto-immune disease (for example lupus)
Placental infection (for example syphilis)
Reduced placental area (multifoetal gestation, placental infarction)

- **Constitutional influences on growth:** The genetic contribution to birth weight is approximately 40%, whereas environmental factors contribute 60%. Small mothers are more likely to produce small babies. Maternal influences on foetal growth are greater than paternal ones. During childhood and young adulthood maternal and paternal influences are more even (Sohl & Moore, 1998).

- **Race:** According to Klaus and Fanaroff (2001) studies in the United States have demonstrated a significantly higher rate of LBW in African Americans when compared with their white contemporaries. Perinatal and neonatal mortality rates remain much greater in the black population. The origins of this problem remain unclear. Breslau et al. (1996a) report that social class and membership of a racial minority are strongly associated with biologic determinants of LBW. The National Institute of Health (1999) notes that these disparities remain despite the controlling of adverse economic and social conditions.

- **Genetic and developmental influences:** Chromosome influences are significant on foetal growth (Sohl & Moore, 1998). Males at term weigh 150 g to 200 g more than females. Infants born with trisomy 13 or 18 are often severely and symmetrically growth restricted. Syndromes that cause multiple genital abnormalities, massive congenital malformations and foetal cardiac anomalies are associated with IUGR (Sohl & Moore, 1998).

- **Congenital infection:** Infections occurring at a critical time in foetal development can cause enough disruption of foetal cells to result in IUGR. Intrauterine infections usually have more severe consequences when they occur early in gestation. Most of the agents associated with the TORCH syndrome (toxoplasmosis, other infections, rubella, cytomegalovirus [CMV] infection, herpes simplex) cause IUGR. It is not

entirely clear if a human immunodeficiency virus (HIV)-caused embryopathy involving IUGR exists. Bacterial infections, with the exception of syphilis, have not been associated with IUGR (Sohl & Moore, 1998). The National Institute of Health (1999) also refers to genital tract and oral microbial infections and inflammation as causes of LBW.

- **Smoking:** In developed countries (Klaus & Fanaroff, 2001), it is the single most important contributor to LBW. Rates of IUGR in smokers are 3 to 4.5 times higher than in non-smokers, with average birth weights decreasing by 70 to 400 g. These adverse effects are particularly pronounced in babies born to older women. According to the American Heart Association (2002) the effects of maternal smoking during pregnancy seem to be dose-related (American Heart Association, 2002). Paternal smoking was associated with a decline in infant birth weight of 112 g. Whitaker et al. (1997) note that postnatal maternal smoking might also play a role.

  Elimination of smoking would diminish SGA rates by 20% to 30% (Klaus & Fanaroff, 2001). However, the mean duration of gestation is not affected by maternal smoking (American Hearth Association, 2002).

  Whitaker et al. (1997) state that nicotine alters the receptors in the basal ganglia of the foetal brain. According to Ernst, Moolchan and Robinson

(2001) prenatal exposure to nicotine may lead to dysregulation in neurodevelopment. This can indicate a higher risk of psychiatric problems, including substance abuse.

A variety of other recreational drugs, including alcohol, marijuana, cocaine and amphetamines, have also been associated with adverse foetal effects. With the exception of foetal alcohol syndrome, the effect of these drugs is not as well established or as pervasive as that of tobacco. Certain prescription drugs, anticonvulsants in particular, can result in foetal growth restriction and specific malformation syndromes. Even consumption of coffee may pose a risk (Klaus & Fanaroff, 2001). According to Hulse et al. (1997) maternal cocaine use causes LBW. A greater effect is associated with heavier use. Evidence is inadequate that marijuana, in the amount typically consumed by pregnant woman, causes LBW (English et al., 1997).

- **Maternal disease states:** Maternal vascular disease, for example chronic hypertension, pregnancy-induced hypertension, severe diabetes with vasculophathy and chronic renal disease, has been associated with poor foetal growth. The common thread in all these diseases is vascular pathology that reduces uteroplacental perfusion (Sohl & Moore, 1998). Klaus and Fanaroff (2001) emphasise that pre-eclampsia is not only a contributor to foetal growth restriction, but is also the factor that carries

the most unfavourable prognosis in terms of severity of growth deficit. Maternal hypertension (Sohl & Moore, 1998) unaccompanied by underlying vascular or renal disease is unlikely to be associated with IUGR. Maternal medical complications that compromise delivery of oxygen to the foetus are associated with IUGR. Severe maternal anaemia, such as that associated with sickle cell disease, is also linked with IUGR. Up to 30% of infants born to women with sickle cell disease weigh less than 2 500 g at birth (Sohl & Moore, 1998).

- **Altitude:** A two- to three-fold greater rate of LBW is observed at altitudes higher than 2 000 m than at sea level (Klaus & Fanaroff, 2001).

- **Maternal nutritional states:** Some research evidence demonstrates the potential benefits of nutritional intervention in the mother who was poorly nourished before pregnancy, but not in women who were well-nourished before pregnancy (Klaus & Fanaroff, 2001). Sohl and Moore (1998) state that maternal malnutrition has been known for many years to be a cause of IUGR, although the magnitude of this effect is moderate. Larroque et al. (2001) state that undernutrition during vulnerable periods of brain development may have long-term effects on brain development that can affect learning and memory. According to Wynn and Wynn (1997), as well as Breslau et al. (2001), poor nutrition and infection are significant causes of LBW.

- **Prior obstetric and family history:** Women younger than 15 years of age or older than 45 years of age who have a history of miscarriages or unexplained stillbirths after 20 weeks' gestation, or have had prior preterm deliveries are at increased risk of delivering a growth-restricted baby (Klaus & Fanaroff, 2001). Those aged 35 and older are at a 20% to 40% increased risk of LBW and preterm delivery (Tough et al., 2002).

  Familial factors appear to play a role in the birth weight of babies. Mothers of LBW infants were frequently LBW infants themselves (Klaus & Fanaroff, 2001).

- **Obstetric factors:** Multiple pregnancies are associated with IUGR. Up to 25% of twins are born with IUGR. Placental crowding in some of the foetuses probably contributes to higher rates of IUGR in higher order gestations. At 38 weeks the average birth weight of a triplet is at the 10^{th} percentile for singletons (Klaus & Fanaroff, 2001; Sohl & Moore, 1998). Vohr et al. (2004) confirm that increased short-term and long-term morbidities are associated with higher order multiples. Tough et al. (2002) found that almost 45% of the observed increase in the delivery of infants weighing less than 1 250 g and 50% of the increase in delivery before 35 weeks (which is partly related to multiple births) were accounted for by in vitro fertilisation.

- **Placental contributions:** There are placental pathologic correlates of known causes of IUGR (intrauterine infections, chromosomal anomalies, hypertensive disorders) and gross placental and cord abnormalities (for example extensive infarction and abnormal cord insertions), which are likely to result in restricted foetal growth (Klaus & Fanaroff, 2001).

Sohl and Moore (1998) summarise the various influences on foetal birth weight in Table 2.2.

Table 2.2 Factors affecting foetal weight at birth (Sohl & Moore, 1998 p. 91)

Factor	Comment
Sex	Males weigh more than females
Race	White babies at term weigh more than black babies
Constitutional factors	As a rough general rule, small parents produce small babies and large parents produce large babies; maternal constitutional factors have a greater influence on foetal growth than paternal factors
Multiple gestation	Singletons are larger than twins, which are larger than triplets, and so forth
Maternal disease states and maternal placental infarction	Diseases include obesity, diabetes, hypertension, infections and substance abuse, among others

IUGR is commonly associated with conditions that are also related to preterm delivery. Therefore, IUGR is an increasingly common finding among infants born at earlier gestational ages (Papageorgiou & Bardin, 1999). Thus, IUGR often occurs with a variety of maternal conditions that are associated with preterm delivery, for example very young or advanced maternal age, low maternal prepregnancy weight, poor maternal weight gain, maternal illness, lower SES status, race, pre-eclampsia, diabetes, intrauterine infections and substance abuse (Papageorgiou & Bardin, 1999).

2.5.1 Aetiology of prematurity

Behrman et al. (2000) state that premature birth of infants whose LBW is appropriate for their preterm gestational age is associated with medical conditions in which there is inability of the uterus to retain the foetus, interference with the course of the pregnancy, premature separation of the placenta or an undetermined stimulus to effective uterine contractions before term (Table 2.3).

Table 2.3 Identifiable causes of premature birth (Behrman et al., 2000 p.477)

Foetal
Foetal distress
Multiple gestation
Erythroblastosis
Non-immune hydrops
Placental
Placental dysfunction
Placenta previa
Abruptio placentae
Uterine
Bicornate uterus
Incompetent cervix (premature dilation)
Maternal
Pre-eclampsia
Chronic medical illness (e.g. cyanotic heart disease, renal disease)
Infection (e.g. group B streptococcus, urinary tract infection, bacterial vaginosis)
Drug abuse (e.g. cocaine)
Other
Premature rupture of membranes
Polyhydramnios
Iatrogenic

Overt or symptomatic bacterial infection of the amniotic fluid and membranes (such as group B streptococci) may initiate preterm labour. Bacterial products may induce premature uterine contractions or a local inflammatory response with focal membrane rupture (Behrman et al., 2000).

2.6 Expected consequences of LBW

In general, the risk of neurodevelopmental and intellectual problems is higher as birth weight and gestational age decrease (Behrman et al., 2000; Klaus & Fanaroff, 2001). However, Elgen, Sommerfelt and Ellertsen (2003) found no

differences in cognitive functions between those children weighing less than 1 500 g at birth and those weighing between 1 500 and 2 000 g. The risk of morbidity depends on the aetiology, but babies for whom no diagnosis is made still have a higher risk of future problems than babies within the normal weight range (Campbell, 2001b).

According to Larroque et al. (2001) previous research with younger children have not always detected significant differences in intelligence tests or school indicators. Research findings of Breslau et al. (2001) imply that most of the differences between LBW and NBW academic achievements at age 11 could be eliminated by eliminating differences in cognitive abilities at age six. Therefore, interventions to improve academic performance of LBW children should focus on the preschool years. Elgen et al. (2003) found that motor problems and low verbal IQ at five years of age in LBW children each doubled the risk of experiencing a school problem at 11 years of age. LBW children without behavioural problems at five years of age are not expected to develop behavioural problems later.

Short-term outcomes and two-year outcomes are predicted by various neonatal diseases, for example intraventricular haemorrhage and respiratory distress syndrome. The more diagnoses an infant has and the more severe those diagnoses, the more likely it is that the infant will have adverse long-term outcomes (Jobe, 2001). Low parental education, single-mother status, parental

SES and other indicators of the environment in which the preterm infant is raised are strong predictors of behavioural and school problems (Breslau et al., 2001; Greenbaum & Auerbach, 1992; Gross et al., 2001; Jobe, 2001). Gross et al. (2001) confirm that preterm children are particularly vulnerable to non-optimal environmental influences, which have a greater negative impact on school outcome (for example parental death, family composition, geographic moves and family instability).

Researchers found a disproportionate number of boys compared to girls who experienced school dysfunction and required remedial assistance (Papageorgiou & Bardin, 1999). Reuters (2004) reports in Beeld, a local newspaper, that premature boys have the tendency to have a smaller brain than premature girls, owing to reduced white and grey matter, especially in the areas controlling reading, language, emotion and behaviour. Boys consequently experience more scholastic problems, such as language, reading and speech difficulties. Girls may be protected by hormones or more X-chromosomes against the consequences of premature birth.

According to Bhutta et al. (2002), 50% of LBW or preterm children are more likely to be enrolled in special education classes. Although several of the LBW children with earlier problems were no longer demonstrating all of these, an almost equal number of previously unrecognised children had manifested academic and social problems, thus resulting in a relatively stable number of such

problems over time (Papageorgiou & Bardin, 1999). Elgen and Sommerfelt (2002) also reported that twice as many LBW children had school problems. York and DeVoe (2002) consequently found that fewer premature children graduated from high school. Whereas behaviour such as over-activity, temper tantrums and perseveration had greatly subsided, symptoms of neuropsychiatric disturbance, including distractibility, irritability, unhappiness, low frustration tolerance, fears, disobedience, poor motivation and sleep difficulties, persisted or increased (Papageorgiou & Bardin, 1999).

Specific types of developmental morbidities described in LBW cohorts include cognitive delays (i.e. lower IQ), speech and language disorders, persistent neuro-motor abnormalities, including difficulties with balance and coordination, and perceptual problems (Breslau et al., 2001; Papageorgiou & Bardin, 1999). According to McGrath et al. (2000) reading and mathematic achievements scores were the lowest for the preterm groups classified as neurologically suspect or abnormal. Thus, compromised neurological status adversely affects cognitive and school performances.

Chapieski and Evankovich (1997) report that preterm children are particularly vulnerable to psychiatric disturbances. Ten percent of a sample of premature children exhibited an emotional disturbance severe enough to interfere with their development and to make them difficult to manage. Elgen et al. (2002) document that 27% of LBW children compared to 9% of NBW children were

diagnosed with a psychiatric disorder. Chapieski and Evankovich (1997) found a higher level of internalising behaviour problems, including anxiety, among nine-year-old VLBW males. Breslau et al. (1996b) found no relation between LBW and other common childhood disorders, namely childhood anxiety disorder and ODD. According to Chapieski and Evankovich (1997) children born prematurely do not seem to be at increased risk of conduct problems, in the presence of controlled adverse environmental conditions. Research data are however inconsistent. Children with a birth weight of less than 1 500 g seem to show lower levels of social competence, especially in the presence of lower intellectual functioning, poor neurological status, family instability and low SES (Chapieski & Evankovich, 1997; Harrison, 2002).

2.6.1 Neonatal outcomes

Though neonatal outcomes will not be the focus of this study, relevant aspects will be addressed briefly before general longer-term outcomes are discussed.

All very premature babies are developmentally behind normal babies of the same age for the first six to nine months. Most have caught up by 12 to 18 months. Many premature babies show disturbances of reflexes, posture and muscle tone in the early months, which usually resolve by 12 to 18 months without specific treatment (Campbell, 2001b).

Behrman et al. (2000) list the following neonatal problems associated with premature infants:

- Respiratory, for example respiratory distress syndrome, chronic lung disease, congenital pneumonia and apnea
- Cardiovascular, for example bradycardia, hypotension and hypertension
- Hematologic, for example organ haemorrhage
- Gastrointestinal, for example poor gastrointestinal function
- Metabolic-endocrine, for example hypocalcemia
- Central nervous system (CNS), for example intraventricular haemorrhage, hypotonia, deafness, seizures and retinopathy of prematurity
- Renal, for example hyponatremia
- Other, for example infections

According to Chapieski and Evankovich (1997) some of these above-mentioned neonatal problems may have long-lasting effects on the CNS.

The frequency of cerebral palsy, seizure disorders and other neurological problems increases, especially in the case of VLBW or ELBW (Bhutta et al., 2002; Bukatko & Deahler., 2001; Stathis et al., 1999). According to Bregman

(1998) mental retardation and severe visual impairments comprise the highest percentage of severe outcomes in infants born before 27 weeks.

2.6.2 Full-term SGA infants

Sohl and Moore (1998) state that some researchers have found a lower mean IQ in school-age SGA children, and the lower IQ is generally associated with SES or gender (i.e. boys). Studies of populations of children with mental retardation demonstrate a disproportionately greater number of SGA children than expected, although the studies do not differentiate among causes of IUGR.

Sohl and Moore (1998) further note that speech and language problems, hyperactivity, attention deficit, learning disability and minor neuro-motor dysfunction are more frequent in full-term SGA children than in appropriate-for-gestational-age (AGA) children. Learning deficits or academic failure occurs in as many as 45% to 50% of full-term male SGA school-age children with normal intelligence. Even if there is no evidence of congenital infection or anomalies, the earlier the growth retardation (e.g. noted before 26 weeks' gestation) the higher the incidence of lower IQ, academic difficulties, behaviour problems and attention deficit. Deficits in academic areas (Breslau et al., 2001), such as vocabulary, spelling and mathematics, occur throughout childhood and adolescence. O'Keeffe et al. (2003) also confirm an increased prevalence of learning difficulties in SGA children.

2.6.3 Preterm SGA infants

The preterm SGA infant has the disadvantages of both prematurity and IUGR (Taeusch & Ballard, 1998). It is difficult to determine whether the prematurity or the IUGR makes the greatest contribution to adverse neurodevelopmental outcome.

Major disability occurs in 7% to 23% of preterm SGA children. Most preterm SGA children have normal intelligence, but the mean IQ is often lower than in full-term AGA children and sometimes even preterm AGA children. In preterm children without major disability, minor neuro-motor dysfunction, visual-perceptual abnormalities, academic difficulties (such as reading and mathematics), and behaviour problems are far more common than in the general population. Learning deficits occur in 36% to 50% of preterm SGA children at eight to 11 years. An increased incidence of hyperactivity was found in eight-year-old preterm SGA children when compared with preterm AGA and full-term control groups (Sohl & Moore, 1998).

2.6.4 Outcomes of VLBW infants

In the case of VLBW infants, approximately 50% to 60% will have normal outcomes and 40% to 50% will have some degree of impairment ranging from mild-moderate outcomes (20% to 30%) to severe ones (approximately 20%). These children remain at high risk of learning difficulties at school age (Astbury et al., 1987; Botting et al., 1997; Bregman, 1998; Ulvund, Smith & Lindemann,

2001). Breslau and Chilcoat (2000) and Breslau et al. (1996a) also confirm increased rates of behavioural and cognitive problems in VLBW children at school age. Bregman (1998) further states that deficits in early information-processing skills are consistent with findings of deficits in academic performance, often resulting in a specific learning problem. VLBW infants may not catch up, especially in the presence of chronic sequelae (for example mental retardation, seizures, sensory impairment), insufficient nutritional intake or an inadequate caretaking environment.

The following cognitive outcome proportions in VLBW children at eight years of age were reported: 4.6% had a very low IQ (<70) and 13.9% had a low IQ (70 to 84). Of those with an IQ higher than 84, 12.0% had language disability, 12.0% had performance disability, 21.4% had visual-motor disability and 36.1% were apparently normal (Papageorgiou & Bardin, 1999). Bregman (1998) confirms that these children were more likely to be classified as subaverage at age six with an IQ below 85. York and DeVoe (2002) and Botting et al. (1997) report that VLBW individuals had significantly lower mean IQ scores than controls, a higher frequency of subnormal IQ (below 70) and borderline IQ (70 to 84).

Papageorgiou and Bardin (1999) state that there was interaction of prematurity and social class on IQ, verbal tests, academic achievement and attention, with premature children of lower SES scoring lowest on these

measures. According to Bregman (1998) VLBW children are at increased risk of attention deficiencies and hyperactivity, which will be elaborated on in Chapter 3. This is probably a significant factor contributing to overall poorer school performance. It is also consistent with the finding in infancy of poor state modulation and organisation correlating with later learning deficits. Botting et al. (1997) confirm that VLBW children are at increased risk of psychiatric symptoms, especially ADHD.

Although VLBW girls did not differ significantly from full-term birth weight (FBW) girls, suggestive trends in the same direction as the boys may indicate that there is an increased risk of behavioural problems in both genders but that these sequelae become evident at an earlier age in boys than in girls (Papageorgiou & Bardin, 1999).

Sykes et al. (1997) indicate that both male and female VLBW children were rated by their teachers as expressing more behavioural problems than their controls. They were also less well adjusted to the school environment. It is speculated that the problem behaviour reflects a failure in self-regulatory functions. Botting et al. (1997) also reported poorer social skills in the VLBW group. Increased depression and anxiety, particularly generalised anxiety disorder, were found among VLBW children (Botting et al., 1997).

The survival of VLBW infants is improving but short-term (18 to 22 months) and longer-term outcomes (school performance) of the survivors are not

improving (Jobe, 2001). Bregman (1998) confirms a reduction in mortality but stable neurodevelopmental morbidity.

2.6.5 Outcomes of ELBW infants

ELBW infants are at increased risk of neurological abnormalities, developmental delays and functional delays at 18 to 22 months' corrected age (Vohr et al., 2000). As can be expected, most children with major neurosensory impairments have subaverage cognitive development. However, developmental outcomes are poor even for those without major neurosensory impairments (Greenbaum & Auerbach, 1992; Hack et al., 2000; Stathis et al., 1999).

The classroom behaviour of ELBW children was rated by teachers as poor, even for children who had not failed a grade (Papageorgiou & Bardin, 1999; Saigal et al., 2001). Arithmetic reasoning, mathematics, reading comprehension, balance, fine motor coordination and perceptual function were common weaknesses in these children (Papageorgiou & Bardin, 1999). Breslau et al. (2001) state that LBW has greater effects on mathematics than on reading. Mathematics is more strongly related to non-verbal processing, including visual-perceptual and motor functions. LBW children tend to show greater impairments in these areas. This finding is consistent with evidence that early neurological insults are more closely related to deficits in visual than in verbal abilities.

Whereas some children grow out of neurodevelopmental problems, infants with a birth weight of less than 1 000 g frequently experience behavioural problems, learning difficulties and school failure (Jobe, 2001)

Despite an overall mean IQ of 90 (range 50 to 141) at a mean age of 10 years, 64% of these children had been or currently were in special school educational programmes. Saigal et al. (2001) report that at school age, ELBW children make use of a disproportionate amount of resources for remedial education and psychological therapy for hyperactivity and emotional problems. This results in greater dependence on the family, health care system and government resources.

2.7 Treatment

Physical and occupational therapy, as well as early intervention developmental programmes, should be some of the options available for cognitive and neurodevelopmental problems. Primary health care professionals play a significant role in assisting in the follow-up and implementation of a management plan, as well as supporting and educating parents. Such programmes should be coordinated with the infant's paediatrician, as well as with the follow-up clinic (Campbell, 2001b; Chapieski & Evankovich, 1997; Subramanian, 2002). Social skills training, including enhancement of problem-solving and adaptive skills, is an essential dimension in the treatment of LBW children (Chapieski & Evankovich, 1997). Long-term follow-up to school age is

important for children with prematurity, IUGR or both (Chapieski & Evankovich, 1997; Sohl & Moore, 1998).

As an increasing number of babies are born and continue to survive with birth weights of less than 1 000 g, it is important to optimise their chances of a healthy, productive life (Campbell, 2001b; Subramanian, 2002).

2.8 Summary

LBW infants constitute three populations, named premature infants, SGA infants who display IUGR and infants with both. Levels of low birth weight are classified into LBW (1 500 g to 2 500 g), VLBW (1 000 g to 1 500 g) and ELBW (less than 1 000 g).

There is correlation between risk factors and causes of LBW with reference to the following: constitutional influences on growth (small mothers are more likely to produce small babies), race, genetic and developmental influences, congenital infection, smoking and substance abuse, maternal disease states, altitude, maternal nutritional states, family history, obstetric factors (multiple pregnancies) and placental contributions.

It is difficult to separate factors associated with prematurity from those associated with IUGR. IUGR is commonly associated with conditions that are also related to preterm delivery or maternal conditions that are associated with preterm delivery. Prematurity is associated with medical conditions in which

there is an inability of the uterus to retain the foetus, interference with the course of the pregnancy, premature separation of the placenta or an undetermined stimulus to effective uterine contractions before term.

The risk of neurodevelopmental and intellectual problems is generally higher as birth weight and gestational age decrease. Specific types of developmental morbidities described in LBW cohorts include cognitive delays (for example lower IQ), speech and language disorders, persistent neuro-motor abnormalities, difficulties with balance and coordination, visual-perceptual problems, hyperactivity, attention deficit, learning difficulties and behaviour problems. ELBW children make use of a disproportionate amount of resources for remedial education and psychological therapy. Survival of VLBW infants is improving but short-term and longer-term outcomes of the survivors are apparently not improving. Long-term follow-up to school age is important for LBW infants.

After mentioning difficulties with attention and hyperactivity as some of the consequences of LBW, ADHD will be discussed in the following chapter.

Chapter 3

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

3.1 Introduction

The discussion of LBW leads to the next variable, ADHD. The aim of this chapter is to explore the following relevant aspects of ADHD: defining ADHD, diagnostic criteria and assessment, incidence, gender differences, symptoms, comorbid disorders, aetiological factors, consequences of ADHD and treatment.

3.2 Definition

ADHD is defined as a disorder characterised mainly by developmentally inappropriate levels of inattentiveness, overactivity and impulsiveness, which cause severe impairments at home and school (Heptinstall & Taylor, 2002; Johansen et al., 2002; Plug et al., 1997). ADHD is regarded as a neurodevelopmental disorder, as it tends to be chronic and involve a significant disturbance in the acquisition of basis skills, due to neurobiological factors (Heptinstall & Taylor, 2002).

3.3 Diagnostic criteria and assessment

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) stipulates the following criteria for diagnosing ADHD (American Psychiatric Association, 2000):

A. Either (1) or (2)

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

(2) Six (or more) symptoms of hyperactivity/impulsiveness, which have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level.

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

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

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

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

Mercugliano (1999) states that subcategories of ADHD include ADHD – (primarily) inattentive type, ADHD – (primarily) hyperactive-impulsive type and ADHD – combined type, depending on the pattern of behaviour. Children with the predominantly hyperactive-impulsive type tend to have a more stable diagnosis over time than children with the predominantly inattentive type (Kaplan & Sadock, 1998).

A diagnostic assessment includes a parent and patient interview to identify symptoms of ADHD and other disorders, corroborative information from adults in other settings (usually teachers), a history and physical examination to identify medical disorders, as well as an assessment of academic functioning (Cantwell, 1996; Mercugliano, 1999; Robin, 2002; Stein, 2002). Collecting data from multiple sources is essential to address the possibility of biased reporting (Crystal et al., 2001). Stein (2002) also emphasises evaluation of the family environment and parenting style, which may have an important influence on a specific child. Table 3.1 provides guidelines for the diagnosis and evaluation of children with ADHD, using various resources.

Table 3.1 American Academy of Paediatrics Guidelines for diagnosis and evaluation for the child with ADHD (Leslie, 2002)

Recommendation 1: *In a child six to 12 years old who presents with inattention, hyperactivity, impulsiveness, academic underachievement, or behaviour problems, primary care clinicians should initiate an evaluation for ADHD.*
Recommendation 2: *The diagnosis of ADHD requires that a child meet DSM-IV criteria*
Recommendation 3: *The assessment of ADHD requires evidence directly obtained from parents or caregivers regarding the core symptoms of ADHD in various settings, the age of onset, duration of symptoms, and degree of functional impairment.*
Recommendation 4: *The assessment of ADHD requires evidence directly obtained from the classroom teacher (or other school professional) regarding the core symptoms of ADHD, the duration of the symptoms, the degree of functional impairment, and coexisting conditions. A physician should review any reports from a school-based multidisciplinary evaluation where they exist, which will include assessments from the teacher or other school-based professional.*
Recommendation 5: *Evaluation of the child with ADHD should include assessment for coexisting conditions.*

Accardo (1999) states that a comprehensive medical assessment assists in confirming the diagnosis of ADHD or identifies other conditions that mimic it (see Table 3.2). Various chronic diseases and neurological conditions produce a picture

of inattention, but with a range of other signs and symptoms (Accardo, 1999; Cantwell, 1996).

Table 3.2 Conditions that are associated with ADHD or mimic it (Accardo, 1999)

Medical disorder	**Developmental disorders**
Chronic diseases	Communication disorders
Hearing impairment	Learning disability
Sleep disorders	Mental retardation
Neurological disorders	**Psychiatric disorders**
Brain injury	Mood disorders
Tic disorder	Obsessive/compulsive disorder
Seizure disorder	Conduct disorders
Genetic/endocrine/metabolic disorders	
Syndromes:	
Fragile X	
Foetal alcohol syndrome	
Thyroid disease	

Edwards, Schulz and Long (1995) confirm that diagnosing ADHD is complicated by the possibility that some other primary problem is producing the ADHD symptoms. There is a variety of psychiatric, developmental, medical and environmental conditions that can either mimic ADHD symptoms or coexist with ADHD. Oppositional and conduct disorders frequently co-occur with ADHD

and it can be difficult to determine whether one or two conditions are present (Edwards et al., 1995).

Cantwell (1996) also emphasises appropriate cognitive assessment of ability and achievement, as well as appropriate adjunct assessments (such as speech and language assessment, and evaluation of fine and gross motor function in selected cases).

Crystal et al. (2001) mention that teacher information is more useful than parent information in discriminating among subtypes of children with ADHD. Researchers identified one way of addressing this challenge of biased reporting, by collecting data from multiple sources (Crystal et al., 2001; Edwards et al., 1995).

3.4 Prevalence

ADHD seems to be a heterogeneous group of disruptive behaviour disorders, affecting 3% to 6% of primary school children (Cantwell, 1996; Swanson et al., 1998). According to Swanson (2003) approximately 8% to 10% of school-aged children in the United States are affected with ADHD. Various studies estimate that between 2% and 9.5 % of all school-aged children worldwide have ADHD (Barkley, 1998). However, cultural influences may have an impact on the diagnosis of the disorder (Jensen et al., 1997). There might be slight geographical variations in the percentage of children diagnosed with ADHD (Alarcon et al., 1999; Meyer, 1998; Taylor, 1998). Little is known about

ADHD on the African continent. Research among the different ethnic groups of the Limpopo Province of South Africa indicates that ADHD is the most prevalent childhood disorder also in South Africa and that the prevalence rates for ADHD subtypes are similar to Western rates for both genders in all ethnic groups (Meyer et al., 2004).

According to Lou (1996) ADHD is among the most common causes of behavioural disturbance in schools. It constitutes one of the largest groups of patients in the clientele of child psychiatrists and child neurologists.

3.5 Gender differences

Recent evaluations suggest male-to-female ratios of 3:1 or 2:1 in community samples (Barkley, 1998; Stein, 2002; Swanson, 2003), which raise the possibility that affected females are different in some ways (Heptinstall & Taylor, 2002). Increasing interest developed in gender differences in ADHD, both in terms of the practical concerns regarding treatment and in terms of finding answers to the aetiology of the disorder which could account for the gender differences (Heptinstall & Taylor, 2002). Johansen et al. (2002), as well as Heptinstall and Taylor (2002) confirm that the disorder is more common in boys than in girls, but during adolescence and young adulthood relatively more females are affected. According to Heptinstall and Taylor (2002) the prevalence rate for ADHD among boys declines by nearly 20% per year between the ages of 10 and 20, while the prevalence among girls remains relatively constant.

Mercugliano (1999) states that the prominent decreases in the metabolism of female adolescents could suggest that either females have a more rapid rate of developing an "adult ADHD brain activity pattern" or that they have relatively more severe ADHD. Lower levels of inattention, internalising behaviour and peer aggression were more characteristic of ADHD girls from non-referred populations than ADHD boys (Heptinstall & Taylor, 2002).

Boys are generally more frequently afflicted with neurodevelopmental disorders than girls (Heptinstall & Taylor, 2002). One possibility is that girls appear to be more mature than boys at all developmental stages. Boys are one year behind girls in maturity at the time of starting school and this widens to two years at puberty. Prenatal and birth complications, infectious disease and neurodevelopmental abnormalities occur more frequently in boys. There is an agreement that affected girls develop a more severe form of the disorder. Because girls mature faster, they will acquire a relatively greater degree of divergence from their norm and will therefore be affected more severely than boys (Heptinstall & Taylor, 2002). Another possibility is that girls have a higher threshold, which implies that girls tend to be more seriously affected; they have a higher genetic loading and have more affected relatives (Heptinstall & Taylor, 2002).

Girls seem to have the tendency to be diagnosed with ADHD later, with more symptoms of the inattentive subtype and cognitive problems and fewer

symptoms of ODD, CD or aggressive/impulsive behaviour (Cantwell, 1996; Swanson, 2003). However, Heptinstall and Taylor (2002) report that girls of all ages had fewer attention problems and less hyperactivity than same-age boys on parent and teacher rating scales, but ADHD girls showed more inattentive behaviour during testing than ADHD boys. Accardo and Blondis (2001) are of the opinion that non-hyperactive girls with attentional disorders remain an under-diagnosed and untreated group.

According to Abikoff et al. (2002) girls with ADHD had relatively higher rates of verbal aggression to children, when compared to comparison girls. They further observed that boys with ADHD engaged in more rule-breaking and externalising behaviour than girls with ADHD. However, girls with ADHD should exhibit fewer externalising, observable forms of behaviour than do boys with ADHD, just as normal girls should also exhibit fewer than normal boys. Thus, according to Abikoff et al. (2002) objective observations should find that girls with ADHD show lower rates of rule-breaking and other externalising behaviour than boys with ADHD, but higher rates than non-impaired, normal girls. Crijnen, Achenbach and Verhulst (1997), as well as Heptinstall and Taylor (2002) confirm that across all cultures boys tend to have more externalising problems and girls more internalising problems. Therefore boys are more frequently referred for treatment (Heptinstall & Taylor, 2002).

Generalisation of research findings should however be done with caution. Hunt, Paguin and Payton (2001) state that although hyperactivity is more frequent in boys, distractibility and disorganisation can also be a major problem in girls.

3.6 Symptoms

The most common characteristics of children with ADHD are listed by Kaplan and Sadock (1998) in order of frequency: hyperactivity, perceptual motor impairment, emotional lability, general coordination deficit, attention deficit, impulsiveness, memory and thinking deficits, specific learning disabilities, speech and hearing deficits, and equivocal neurological signs.

The main clinical symptoms of ADHD (as mentioned in section 3.3) will be outlined as follow:

3.6.1 Attention deficit

According to the DSM-IV-TR six or more of the symptoms of inattention must be present, which have persisted for at least six months to a degree that is maladaptive and inconsistent with developmental level (American Psychiatric Association, 2000)

- Fails to give close attention to details
- Difficulty sustaining attention
- Does not seem to listen

- Does not follow through on instructions
- Difficulty organising tasks or activities
- Avoids tasks requiring sustained mental effort
- Loses things necessary for tasks
- Easily distracted
- Forgetful in daily activities.

Attention deficit seems to be characterised by slow retrieval and information processing, low levels of alertness and mild problems with memory/orientation (sluggishness, drowsiness, daydreaming) (McBurnett, Pfiffner & Frick, 2001).

According to Johansen et al. (2002) the attention problems associated with the ADHD inattentive subtype are general and non-specific. This is related to poorly focussed or selective attention (Beardsworth & Harding, 1996), less accurate information processing and possibly to stronger association with reduced IQ. Johansen et al. (2002) are of the opinion that ADHD inattentive subtype may have a different aetiology than the hyperactive/impulsive subtype.

3.6.2 Hyperactivity/impulsiveness

The DSM-IV-TR refers to six or more symptoms of hyperactivity/impulsiveness, which have persisted for at least six months to a

degree that is maladaptive and inconsistent with developmental level (American Psychiatric Association, 2000):

- Fidgets with hands or feet or squirms in seat
- Leaves seat in classroom inappropriately
- Runs about or climbs excessively
- Has difficulty playing quietly
- Is "on the go" or "driven by a motor"
- Talks excessively
- Blurts out answers before questions are completed
- Has difficulty awaiting turn
- Interrupts or intrudes on others.

3.6.2.1 Hyperactivity

According to Johansen et al. (2002) hyperactivity is characterised by a general increase in unnecessary gross body movements, which seem to be absent in novel situations.

3.6.2.2 Impulsiveness

Impulsiveness can be defined as a response that is executed with insufficient forethought, planning or control, and is therefore inaccurate or maladaptive (Solanto et al., 2001).

Johansen et al. (2002) argue that impulsiveness has both a motor and a cognitive component. "Motor impulsiveness" can be defined as bursts of

responses with short inter-response times. This behaviour seems to emerge in children with ADHD. "Cognitive impulsiveness" implies that private events (such as thoughts and plans) are dealt with for short sequences of time with rapid shifts, which result in problems with generating and following plans, difficulty with organising own behaviour, forgetfulness and inefficient use of time. Solanto et al. (2001) confirm that impulsiveness may be differentially expressed in motor, cognitive, social and emotional domains.

According to Johansen et al. (2002) children with ADHD inattentive subtype tend to be more socially withdrawn, experience greater academic problems and develop comorbid anxiety or other mood disorders (as discussed in section 3.7). However, early hyperactive-impulsive behaviour is associated more closely with externalising problems such as aggression, oppositional behaviour, adolescent delinquency and substance abuse (Johansen et al., 2002).

Tucha and Lange (2001) state that children with ADHD have poor motor coordination and perceptual motor skills. Kalff et al. (2002) identified impairments in working memory, visuo-motor ability, executive functions (especially inhibitory control), deficient motivation and difficulties with serial information processing.

3.6.3 Impaired executive functions

Barkley (1998) states that impaired self-control and behavioural inhibition are the central deficits in ADHD, particularly diminished responsivity to conditioned stimuli on which punishment is contingent (Olson, 2002). However, children with ADHD often manifest motivational problems that appear to be deficits in inhibitory control (Olson, 2002). Heptinstall and Taylor (2002) view impairments in attention and executive functions as the core impairments of ADHD. Mercugliano (1999) and Solanto et al. (2001) also found that both delay aversion (deficient timing and pacing of response) and inhibitory failure are implicated in ADHD. Delay can be reduced by engaging in activities or shifting attention.

Self-control can be defined as the ability to inhibit or delay one's initial motor (and perhaps emotional) responses to an event. Self-control is an essential foundation for the performance of any task. As children grow up, most obtain the ability to engage in mental activities, known as executive functions, that help them deflect distractions, recall goals and take the steps needed to reach them. For instance, in order to achieve a goal in work or play, people need to be able to remember their aim (use hindsight), prompt themselves about what they need to do to reach that goal (use foresight), keep their emotions reined in and motivate themselves. These functions can only be carried out successfully when a person is able to inhibit interfering thoughts and impulses (Barkley, 1998).

According to Blondis, Snow and Accardo (2000) executive processes include higher-level skills such as focused and divided attention, planning abilities, impulse control, mental flexibility and working memory. The executive functions can be grouped into four mental activities, as reflected in Table 3.3 (Barkley, 1998; Mercugliano, 1999).

Table 3.3 Executive functions (Barkley, 1998; Mercugliano, 1999)

1. Working memory, which refers to holding information in the mind while working on a task, even if the original stimulus that provided the information is gone. Such remembering is essential to timeliness and goal-directed behaviour. Working memory provides the means for hindsight, forethought, preparation and the ability to imitate the complex, novel behaviour of others, which are all impaired in individuals with ADHD.
2. Internalisation of self-directed speech: Most children, before the age of six, frequently speak out aloud to themselves, for instance by reminding themselves how to perform a particular task. Such private speech usually disappears by age 10 and evolves into inaudible muttering.
Internalised, self-directed speech enables one to reflect to oneself, to follow rules and instructions, to use self-questioning as a form of problem-solving and to construct "meta-rules", which form the basis for understanding the rules for using rules.
3. Control of emotions, motivation and state of arousal. Such control enables individuals to achieve goals by delaying or altering potentially distracting emotional reactions to a particular event and to generate private emotions and motivation.
4. Reconstitution, which consists of two separate processes: breaking down observed forms of behaviour and combining the parts into new actions not previously learned from experience. Reconstitution involves a great degree of fluency, flexibility and creativity. It enables individuals to propel themselves toward a goal without having to learn all the needed steps by rote. Children with ADHD seem to be less capable of reconstitution than other children.

According to Barkley (1998) a loss of behavioural inhibition and self-control leads to various disruptions in brain functioning, as indicated in Table 3.4. Table 3.4 indicates the consequences of specific impaired executive functions.

Table 3.4 A psychosocial model of ADHD (Barkley, 1998)

Impaired function	Consequence
Nonverbal working memory	Diminished sense of time. Inability to hold events in mind. Defective hindsight. Defective foresight.
Internalisation of self-directed speech (verbal working memory)	Deficient rule-governed behaviour. Poor self-guidance and self-questioning
Self-regulation of mood, motivation and level of arousal	Displays all emotions publicly; cannot censor them. Diminished self-regulation of drive and motivation.
Reconstitution (ability to break down observed forms of behaviour into component parts that can be recombined into new forms of behaviour in pursuit of a goal)	Limited ability to analyse forms of behaviour and synthesise new forms of behaviour. Inability to solve problems.

3.7 Comorbid disorders

Accardo (1999) and Fischer et al. (2002) state that the presence of ADHD should be considered as a marker for other psychiatric and developmental conditions, as indicated in Table 3.5. According to Leslie (2002) the symptoms associated with ADHD interfere with normal developmental milestones of

childhood and adolescence. Table 3.5 represents the prevalence of selected comorbid conditions in children with ADHD.

Table 3.5 Prevalence of selected coexisting conditions in children with ADHD (Stein, 2002)

Coexisting condition	Estimated prevalence (%)
Oppositional defiant disorder	35.2
Conduct disorder	25.7
Anxiety disorder	25.8
Depressive disorder	18.2

Comorbid conditions seem to be much rarer in younger children. They tend to become important during the transition to adolescence and especially with teenagers who have not been diagnosed or adequately treated previously (Accardo, 1999).

Children with ADHD who also have other comorbid conditions have more negative outcomes when compared with children who have ADHD only (Hechtman, 1999).

The following common comorbid disorders will be discussed:

3.7.1 Aggression

Aggression seems to be more characteristic of the ADHD-combined group than of the ADHD-inattention group (Crystal et al., 2001). According to Hechtman (1999) aggression in children with ADHD is highly predictive of

adolescent antisocial behaviour and adult violence. Physical fighting is an important predictor in the onset of CD.

3.7.2 Specific learning disability

Impaired performance with respect to motor planning and executive functioning may not be attributed completely to ADHD but may also partly be caused by learning disabilities. A recent study has demonstrated that children with ADHD and reading disability were impaired in various domains, including executive functioning. Thus, future research should also focus on learning disabilities in addition to ADHD in order to ascertain whether neuro-cognitive differences between groups are accounted for by ADHD or by learning disabilities (Kalff et al., 2002).

The group with comorbid hyperactivity and learning disability had significantly more oppositional or delinquent behaviour, lower self-esteem, more impulsiveness, fidgetiness, immaturity and inattentiveness. Their academic performance was worse than that of the control group but not worse than that of the other learning disabled group (Hechtman, 1999).

Taylor et al. (1998) report delayed language milestones, problems with expressive language and reading disabilities in hyperactive children. In one study two thirds of hyperactive children had learning problems to such an extent that individual or small-group remediation was needed (Hechtman, 1999).

According to Accardo (1999) day-to-day variations in academic performance are more suggestive of ADHD than of learning disabilities. Spencer, Biederman and Wilens (1999) note that children with ADHD have more grade repetitions, poorer marks, more placements in special classes and need more tutoring.

Children with either learning disability or ADHD often present with both disorders. Therefore, it is important to assess for both disorders when necessary (Fletcher, Shaywitz & Shaywitz, 1999).

Learning disabilities usually fall into the category of language-based disorders of learning, impaired mathematic performance, dysgraphia and pragmatic language disorders related to language use in social contexts. Formal psycho-educational testing for learning problems is required for those children whose developmental and school history suggests possible learning disabilities (Leslie, 2002).

Children with ADHD also show other difficulties in the area of executive functioning, such as inconsistent performance, delayed automatisation of skills and poor meta-cognitive abilities (for example organisation, time management and breaking tasks down into smaller components) that are not considered learning disabilities. Parents and teachers may inappropriately attribute these problems to laziness or lack of motivation (Leslie, 2002).

3.7.3 Oppositional defiant disorder

ODD is characterised by a pattern of negativistic, hostile and defiant behaviour (Spencer et al., 1999).

Approximately 40% to 60% of children with ADHD are comorbid for ODD. Hechtman (1999) found that the ADHD children who were also comorbid for ODD had more negative mother-child interaction, more home conflicts, and greater maternal psychological distress. Sixty percent of the ADHD group, but only 11% of the controls met criteria for comorbid ODD in adolescence.

Comorbid ODD is often a precursor of CD, which is a more serious disorder with significant long-term consequences (Hechtman, 1999).

Kalff et al. (2002) also report that children with comorbid ADHD and ODD or CD have significantly impaired performance on tasks measuring working memory and visuo-motor ability compared to children with pure ODD/CD.

3.7.4 Conduct disorder

CD is a severe disorder of habitual rule breaking defined by a pattern of aggression, destruction, lying, stealing or truancy (Spencer et al., 1999).

In an early study they found that the group with both hyperactivity and CD continued to have ADHD symptoms, aggressiveness, non-compliance, antisocial

behaviour and alcohol use at follow-up. Adolescents with ADHD who were comorbid for CD indulged in more cigarette and marijuana use (but not alcohol) and experienced more school expulsions and suspensions (Hechtman, 1999). Hyperactive individuals had significantly higher juvenile (46% versus 11%) and adult (21% versus 7%) arrest rates. Conduct problems in childhood and serious antisocial behaviour in adolescence are considered to be significant risk factors for becoming an adult offender. The authors concluded that children with both hyperactivity and CD problems are at increased risk of later criminality (Fischer et al., 2002; Hechtman, 1999; Olson, 2002; Spencer et al., 1999).

ADHD teenagers with more pronounced comorbid ODD and CD symptoms seem to be at highest risk for defiant driving skills or habits, and negative driving-related outcomes (Abikoff et al., 2002).

According to Accardo (1999) many of the behavioural symptoms interpreted as consistent with CD and ODD are frequently simply reflections of an undiagnosed and untreated ADHD. However, hyperactive behaviour in primary school children is considered to be a risk factor for the development of CD (Taylor et al., 1998).

3.7.5 Depression and anxiety

The rate of ADHD and comorbid depression and anxiety ranges from 20% to 40%, depending on the sample and diagnostic criteria (Accardo, 1999; Hechtman, 1999; Spencer et al., 1999). According to Cantwell (1996)

internalising problems, such as anxiety and mood disorders, may be underreported by parents or teachers, because externalising forms of behaviour are more observable.

According to Spencer et al. (1999) mania was detected in 11% of children with ADHD with a mean age of 11 years. Distractibility, impulsiveness, hyperactivity and emotional liability are characteristic of both ADHD and mania.

According to Spencer et al. (1999) comorbid association of approximately 25% between ADHD and anxiety disorders has been found in children with anxiety disorders and children with ADHD. Higher rates of ADHD have been noted in children of parents with anxiety and mood disorders than in children of comparison groups. Children with attention deficits without hyperactivity had higher rates of anxiety disorders than those with hyperactivity. Thus, ADHD with comorbid anxiety disorder may imply substantially worse outcomes in children with both disorders (Spencer et al., 1999).

Co-occurrence of ADHD and mood disorders has been found in 15% to 75% of cases in both epidemiologic and clinical samples of children and adolescents. Children with ADHD and their first-degree relatives seem to have a significantly higher rate of mood disorders than normal control children and their first-degree relatives (Spencer et al., 1999).

3.7.6 Substance abuse

Substance abuse was mostly linked to CD, later antisocial behaviour, arrest and continuation of ADHD symptomatology (Hechtman, 1999). Higher levels of smoking and drinking alcohol seem to characterise the high-ADHD group (Whalen, 2001). Fischer et al. (2002) state that antisocial personality disorder and substance use disorders were specifically overrepresented in early adulthood by ADHD children.

According to Kaplan and Sadock (1998) a childhood history of ADHD or CD or both increase the risk of an alcohol-related disorder as an adult. However, substance abuse disorders during adolescence appear to be related to the presence of CD rather than to ADHD alone (Kaplan & Sadock, 1998).

These above-mentioned findings are coherent with the results of Chilcoat and Breslau (1999), which indicate that children with a high level of externalising problems had a high risk of drug use (and vice versa), regardless of ADHD status. Thus, risk for early drug use in children with ADHD seems to depend on the level of externalising problems.

3.7.7 Personality disorders

Follow-up research by Fischer et al. (2002) found that significantly more hyperactive children had borderline personality disorder. Risk of this personality disorder is increased by the severity of childhood CD problems and the severity of teen CD. The prevalence of histrionic and passive-aggressive personality

disorders was significantly higher among the hyperactive group (Fischer et al., 2002).

3.7.8 Immature motor coordination

Weaknesses in right brain functions, such as mathematics and graphomotor skills, are common in children with ADHD. Children who can never write neatly may have a fine motor problem (or dysgraphia), but those who can sometimes write neatly do not have dysgraphia but rather an attentional or focussing problem (Accardo, 1999; Taylor et al., 1998).

According to Blondis (1999) clumsiness in children with ADHD has more to do with inattentiveness and impulsiveness (inability to stop, look and listen) than poor motor coordination. Children with ADHD usually outgrow clumsiness, while it is not the case if the ADHD child also has a comorbid developmental coordination disorder. Gillberg and Kadesjö (2000) state that clumsiness and poor motor coordination are considered to be clear markers of neurological dysfunction.

Gillberg and Kadesjö (2000) document that there is a strong relationship between ADHD and developmental coordination disorder (DCD), dyscoordination, or motor-perceptual dysfunction. About 50% of children with five or more ADHD symptoms, but less than 10% of those with four or fewer ADHD symptoms, also had DCD. Gillberg and Kadesjö (2000) further postulate that the overflow movements are considered as a sign of deficient

motor inhibition or motor control, which is seen as a crucial deficit in the syndrome of hyperactivity. This decreased ability to delay responding may be a core feature of ADHD.

According to Gillberg and Kadesjö (2000) ADHD children do not seem to have impaired input processing, but dysfunctional motor output. Timing, pacing and preparation to act are all deficient. Consequently, ADHD may primarily be a failure in intention, inhibition and capacity to delay responding. Therefore, attentional problems and motor control deficits sometimes need to be treated separately (Gillberg & Kadesjö, 2000).

3.7.9 Emotional disorders

Low self-esteem and insecurity often develop as a result of failures at school and interpersonal relationships (Leslie, 2002; Taylor et al., 1998). According to Hannah (2002) children with ADHD, especially those with aggression, are more likely to be disliked, ignored or rejected by others. Girls with ADHD were found to suffer more peer rejection while ADHD boys were more dominating and physically aggressive towards peers (Heptinstall & Taylor, 2002). Cantwell (1996) mentions that impulsiveness, hyperactivity and inattention often lead to difficulty in peer relationships, which first becomes evident in the elementary school age years. According to Greene and Ablon (2001) social functioning (of the child and significant others) is a significant predictor of long-term outcome for children with ADHD, even when the severity of ADHD

symptoms, conduct, problems, depression, aggression and other factors are controlled.

3.8 Aetiology

Various factors are in reciprocal interaction (Addiction Organisation, 2002; Rapport et al., 2001). According to Hunt et al. (2001) the CNS regulates attention, arousal, activity and affect by means of neurotransmitters, which are influenced by genetic factors. These genetic factors might be influenced by environmental factors (Edwards et al., 1995; Mercugliano, 1999).

Factors contributing to the development of ADHD are discussed below.

3.8.1 Genetic influences

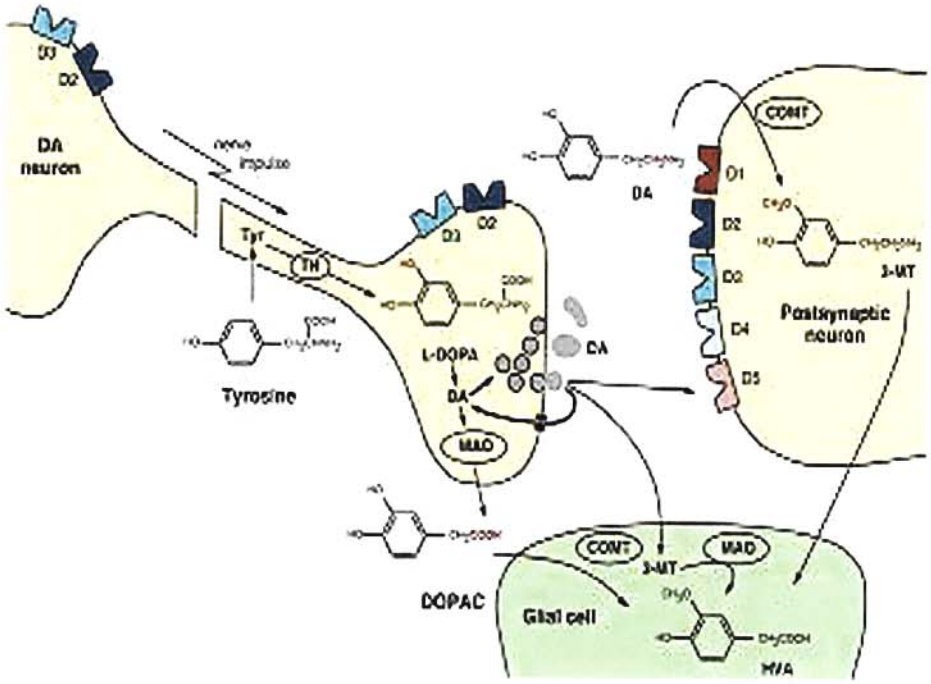
ADHD seems to run in families. Hunt et al. (2001) and Mercugliano (1999) state that studies of twins, siblings and adopted children reflect a significantly higher incidence of ADHD in biological relatives of patients compared with the general population. According to Barkley (1998) and Mercugliano (1999) the child of an adult with childhood-onset ADHD has a more than 50% chance of having ADHD. Barkley (1998) and Edwards et al. (1995) stipulate that heredity appears to play the largest role in the occurrence of ADHD. Parents of children with ADHD are more likely to have a history of numerous problems, such as antisocial behaviour, alcoholism and learning disabilities (Accardo, 1999; Edwards

et al., 1995), as well as depression and marital problems (Heptinstall & Taylor, 2002).

3.8.2 Neurochemistry of ADHD

Genetic studies focus on the chromosomes that regulate neurotransmitter production, release, reuptake and receptor sensitivity (Hunt et al., 2001). Alterations in dopaminergic and noradrenergic function appear to be central in the symptoms of ADHD, because the symptoms of ADHD are treated most effectively by medication (stimulants) that influences these neurotransmitters (Cantwell, 1996; Lou, 1996; Mercugliano, 1999; Swanson et al., 1998). According to Barkley (1998) dopamine is secreted by neurons, as indicated in Figure 3.1, in specific parts of the brain in order to inhibit or enhance the activity of other neurons, particularly those involved in emotion and behaviour.

Figure 3.1 A dopamine synapse (Waters, 1995)

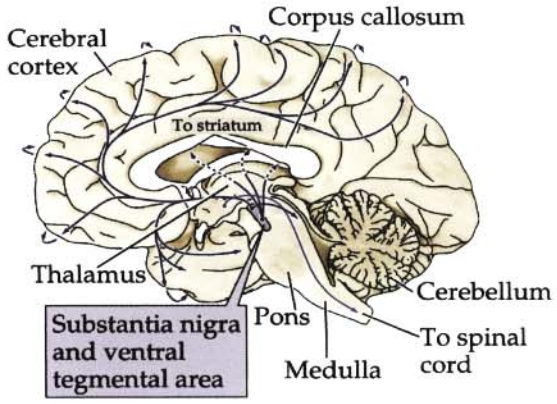


Johansen et al. (2002) confirm that ADHD symptoms may to a large extent be caused by a dysfunctioning dopamine system (see Figure 3.2).

A dysfunctioning meso-limbo-cortical dopamine branch (see Figure 3.3) will produce altered reinforcement and extinction processes that cause deficient sustained attention, hyperactivity, increased behavioural variability and impulsiveness (Hunt et al., 2001; Johansen et al., 2002). Solanto et al. (2001) confirm that children with ADHD have reduced sensitivity to reinforcement. As a result, more immediate, frequent or intense rewards are needed to maintain appropriate performance and behaviour. A dysfunctioning nigro-striatal

dopamine branch (see Figure 3.3) will contribute to poor motor control (Hunt et al., 2001; Johansen et al., 2002).

Figure. 3.2 Dopamine pathways (Purves et al., 2001)

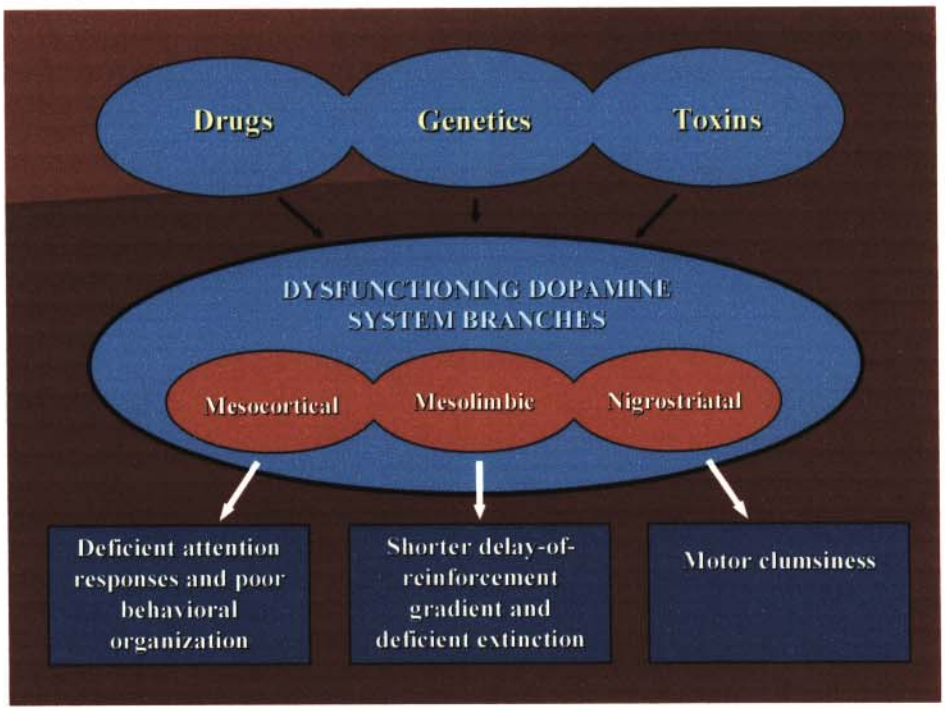


Dopamine dysfunctioning will probably be mainly genetically determined. However, non-genetic factors such as drugs of abuse and environmental toxins may also contribute to the regional differences found in the prevalence of ADHD (Sagvolden & Sergeant, 1998).

A deficit in dopamine is related to an increased binding capacity of the dopamine transporter. This implies that synaptic dopamine is cleared more rapidly in ADHD individuals in the striatum where reuptake via dopamine transporters is the most important means of removing dopamine from the synapse (relative to prefrontal cortex, where synaptic dopamine activity is

primarily decreased by diffusion and uptake by noradrenaline [norepinephrine] transporters). The question still remains whether increases in striatal dopamine transporter density represent the primary neurochemical deficit or a secondary overcompensation (Castellanos & Swanson, 2002). According to Lou (1996) the striatum is the cerebral structure with the richest endowment of dopaminergic synapses, which is coherent with the finding of striatal dysfunction as an anatomical basis of ADHD.

Figure. 3.3 A neurobiological model for ADHD (Johansen et al., 2002)



Different abnormalities might exist in two dopamine regions: under-activity in a cortical region (anterior cingulate) that results in cognitive deficits and over-activity in a sub-cortical region (caudate nucleus) that results in motor excesses.

Similarly different abnormalities may exist in two noradrenergic regions: under-activity in cortex (dorsolateral prefrontal) resulting in primary memory deficits and over-activity in a sub-cortical system causing over-arousal (Castellanos & Swanson, 2002).

The principal finding regarding ADHD is heightened activity of genes that regulate norepinephrine, concurrent with diminished activity of genes that regulate dopamine and serotonin. Thus, no one gene or neurobiological abnormality in isolation can account for ADHD disorders (Cantwell, 1996; Hunt et al., 2001; Johansen et al., 2002; Kaplan & Sadock, 1998). Whalen (2001) also emphasises that asymmetry and other structural brain differences vary among children with ADHD.

3.8.3 Anatomical differences in the brain

Alterations in frontal cortical and basal ganglia information processing may be central to the symptoms of ADHD (Mercugliano, 1999). Barkley (1998), Castellanos and Swanson (2002), and Swanson et al. (1998) confirm reduced size of the frontal lobes, basal ganglia, as well as of the cerebellum (specifically the posterior inferior cerebellar vermis), which is consistent with the notion that the relevant brain areas are hypofunctioning. According to Hunt et al. (2001) neurofunctional imaging studies have suggested inadequate activity in the prefrontal cortex of individuals with ADHD during attentional tasks. Research of Kalff et al. (2002) also suggest that the maturation of prefrontal structures in

children at risk of ADHD at a later age lags behind that of children of the same age without ADHD. Restricted blood flow and activity in frontal areas in ADHD children are reported, which increase after administration of medication (Grodzinsky & Diamond, 1992; Kaplan & Sadock, 1998). Lou (1996) states that a disturbance of the function of the striatum in ADHD and the cingulo-striato-thalamo-cortical loop subserving the fundamental faculty of awareness, results in impulsiveness, inattention and hyperactivity. Hypoperfusion (poor blood flow) has been found in the striatum. The Addiction Organisation (2002) views this lower level of activity in some brain areas as a possible cause of inattention. Accardo (1999) views ADHD as a neurological disorder itself.

3.8.4 Neurophysiological factors

According to Kaplan and Sadock (1998) major growth spurts normally occur in the human brain at several ages: three to ten months, two to four years, six to eight years, ten to 12 years, and 14 to 16 years. A maturational delay in the sequence sometimes manifests and results in ADHD symptoms, which appear to normalise by about age five.

3.8.5 Environmental factors

In the presence of a genetic predisposition to ADHD, environmental factors may enhance its expression (Jensen et al., 1997; Mercugliano, 1999; Taylor et al., 1998).

Environmental factors are also known to cause ADHD. These include the effects of foetal exposure to alcohol and benzodiazepines, as well as other adverse factors in pregnancy, such as pre-eclamptic toxaemia, maternal smoking, low foetal heart rate during labour and small head circumference at birth (Taylor et al., 1991; Taylor et al., 1998). Toxins, such as lead, alcohol, cigarette smoke or other drugs, appear to be capable of contributing to ADHD (Addiction Organisation, 2002; Mercugliano, 1999).

According to Mercugliano (1999) suboptimal parenting skills and particular parenting characteristics have not been shown to cause ADHD. However, specific kinds of parent-child interaction are believed to be helpful or harmful to symptom expression. In a study of the effects of medication on parent-child interaction, it was found that medication that was effective for disruptive behaviour led to more positive mother-child interaction. This indicates that maternal negative behaviour was the result rather than the cause of the child's difficult behaviour (Mercugliano, 1999).

However, Edwards et al. (1995) state that it is unclear whether observed family dysfunction is caused by having a child with ADHD, or whether the child's misbehaviour is caused by parental and/or family dysfunction.

The minority of children are detectably affected by food additives and allergenic whole foods. However, results of studies on iron deficiency and supplementation, deficiencies in essential fatty acids, zinc, and other minerals, and

the relationship of behaviour to the relative dietary content of protein and carbohydrate suggest that further research in this area is indicated (Cantwell, 1996; Mercugliano, 1999). The National Institute of Mental Health (1996) emphasises that ADHD is usually not caused by too much television, food allergies, excess sugar, poor home life or poor schools.

According to Swanson et al. (1998) foetal distress due to environmental factors may damage striatal neurons and affect the development of the frontal lobe basal ganglia neural networks.

Heavy alcohol use during pregnancy has been linked to foetal alcohol syndrome (FAS), a condition that can lead to LBW, intellectual impairment and certain physical defects (Addiction Organisation, 2002; Cantwell, 1996; Johansen et al., 2002). Many children born with FAS show much the same hyperactivity, inattention and impulsiveness as children with ADHD. Drugs such as cocaine (including the smokeable form known as crack) seem to affect the normal development of brain receptors. These brain cell parts are involved in the transmission of incoming signals from our skin, eyes and ears, and help to control our responses to the environment (Addiction Organisation, 2002).

3.8.6 Medical history

Brain injury caused by any prenatal, perinatal and postnatal problem can contribute to a risk of ADHD. The most common long-term sequelae to severe head trauma (with or without skull fracture) are the development of ADHD

(Accardo, 1999). Infants with birth weights of less than 1 500 g or with intraventricular haemorrhages seem to have an increased incidence of developmental disability and ADHD, which will be discussed in Chapter four (Accardo, 1999).

According to Grodzinsky and Diamond (1992) brain damage resulting from encephalitis, lead poisoning and head injury can lead to cognitive impairments and behavioural symptoms similar to ADHD. Accardo (1999) indicated that genetic syndromes that cause mental retardation or that have an impact on brain development are likely to contribute to ADHD.

3.8.7 Traumatic experiences

According to Accardo (1999) emotional stress can precipitate depressive symptoms, conduct disorders, oppositional and anxious behaviour along with attentional deficits, but never attentional deficits alone. Sudden onset of hyperactivity, attentional deficits and learning or other behavioural problems in a school-age child with no developmental risk factors might indicate possible child abuse or sexual abuse (Accardo, 1999).

Taylor (1994) mentions that institutionalisation and failures of attachment are associated with the aetiology of ADHD. Kreppner, O'Conner, Rutter and the English and Romanian Adoptees Study Team (2001) state that attentional problems and overactivity are associated with early disruptive care such as

institutional rearing and parental breakdown. According to Taylor et al. (1998) the quality of relationships within the family and at school are predictors of outcome.

3.8.8 Working memory

According to Rapport et al. (2001) failure of working memory not only leads to disorganised behaviour, but also motivates children to redirect their attention to other stimuli in the environment. This phenomenon is described as *stimulation seeking*. The inability to maintain working memory representations leads to behaviour that increases the rate at which input is delivered to working memory so as to compensate for the rapid rate at which representations fade. Consequently, the rate at which stimulation impinges on working memory increases. Redirection of attention can be alternatively conceptualised as a form of escape from monotonous or high task demand conditions and observed by others as hyperactivity and impulsiveness (Rapport et al., 2001).

3.9 Consequences

According to Taylor et al. (1998) ADHD often persists into adolescence and adulthood. Olson (2002) reports that 25% to 50% of hyperactive children are judged to be functioning normally in later life. According to Kaplan and Sadock (1998) ADHD symptoms persist into adulthood in 15% to 20% of cases, though the severity tends to diminish over time (Olson, 2002). Though physical hyperactivity may diminish, inattention, impulsiveness and accident proneness persist in adolescence (Kaplan & Sadock, 1998; Robin, 2002).

ADHD puts sufferers at risk of a range of abnormalities in personality development (Olson, 2002; Taylor et al., 1998). As already mentioned, the adverse outcomes include delinquency and other antisocial behaviour, as well as underachievement in school. Olson (2002) reports that boys whose symptoms of hyperactivity had not remitted were four times more likely to develop CD in adolescence than others. Moreover, two-thirds of those who developed CD progressed to drug and/or alcohol abuse. Cantwell (1996) states that, if untreated, ADHD predisposes a child to psychiatric and social pathology in later life.

High-risk behaviour is associated with ADHD in adolescents, such as poor driving habits, more accidents, sexual intercourse at an early age, more sexual partners, less use of birth control, more sexually transmitted diseases, a greater frequency of testing for HIV and more teen pregnancies than non-ADHD individuals (Robin, 2002).

Hechtman (1999) and Olson (2002) are of the opinion that the long-term outcome of ADHD is influenced by characteristics of the child, such as severity of symptoms, comorbidity and IQ, which interact with family parameters such as parental pathology, SES status and family adversity, as well as treatment (types and duration).

3.10 Treatment

A multimodal approach has been followed for many years in the treatment of children with ADHD, which includes medication, parent and child education, behaviour management and classroom accommodation (Kaplan & Sadock, 1998; Meyer & Aase, 2003; Silver, 1999; Stein, 2002). Cantwell (1996) and Swanson et al. (1998) emphasise the combination of two primary modalities of treatment for ADHD, including pharmacological (stimulant medication) and behavioural treatment (with psychosocial interventions), based on the argument that multiple areas of impairment require multiple modalities of treatment.

Frequent monitoring of a child with ADHD should be guided by obtaining information about target behaviour, educational output and medication side effects. Regular communication with parents, teachers and the child should be established at the initiation and implementation of a treatment plan. Normal developmental changes in behaviour, increasing educational expectations, as well as changes in the home and school environment should always be considered during monitoring (Kaplan & Sadock, 1998; Leslie, 2002; Meyer & Aase, 2003; Robin, 2002; Stein, 2002). According to Robin (2002) it is essential to deal with comorbidities and remaining problems. Whalen (2001) emphasises that treatment should match the unique aspects of the child's problem(s).

3.10.1 Pharmacological

Hunt et al. (2001) state that pharmacological treatment includes psychostimulants, noradrenergic agents, antidepressants, anticonvulsants (for anti-aggressive and mood-stabilising purposes) and neuroleptics.

ADHD has been treated mainly with stimulant medication (Bennett et al., 1999; Hunt et al., 2001; Spencer et al., 1999; Stein, 2002). Commonly used stimulants in children and adolescents include the following: dextroamphetamine (Dexedrine®), methylphenidate (Ritalin®), amphetamine/dextroamphetamine (Adderall®) and pemoline (Cylert®) (Bennett et al., 1999). Methylphenidate is usually the treatment of choice (Cantwell, 1996; Swanson, 2003; Swanson et al., 1998; Tucha & Lange, 2001). Silver (1999) confirms that methylphenidate and dextroamphetamine are the primary treatments of choice. These medicines are not only effective in treating the core ADHD symptoms (hyperactivity, impulsiveness and inattention), but often improve children's ability to follow rules and interpersonal relationships (Bennett et al., 1999; Hunt et al., 2001; Stein, 2002; Swanson, 2003). According to Tucha and Lange (2001) improvements were found following treatment with methylphenidate regarding aspects of handwriting, such as legibility, accuracy, spacing and uniformity. Hunt et al. (2001) state that psychostimulants enhance a sense of self and self-control.

Recent research supports the use of two other non-stimulant forms of medication for ADHD, namely tricyclic antidepressants (imipramine,

nortriptyline, desipramine and amitriptyline) and bupropion (also an antidepressant). However, there are significantly fewer studies of these drugs compared to stimulants (Silver, 1999; Spencer et al., 1999; Stein, 2002). Swanson et al. (2003) state that tricyclic antidepressants are second-line therapy for those who respond poorly to stimulants, who cannot tolerate stimulant treatment or who have comorbid conditions.

These stimulant medicines release and inhibit the reuptake of catecholamines, mainly dopamine, in the CNS (Bennett et al., 1999; Castellanos & Swanson, 2002; Swanson et al., 1998). According to Lou (1996) methylphenidate enhances the release of only dopamine, while dextroamphetamine releases both dopamine and norepinephrine. The effectiveness of these drugs peak at about one hour after each dose and dissipate in about four hours (Swanson et al., 1998). Thus, stimulants do not produce long-term changes in achievement or long-term prognosis. The benefits of stimulants stop as soon as the medication wears off (Pelham et al., 2000).

According to Jensen et al. (1997) medication may sometimes be the only available or most effective way to help a specific child. However, compliance with medication is a significant problem due to multiple doses during the day together with long duration of treatment (Swanson, 2003).

Adesman (2003) emphasises that there is no preferred medication or dosage that meets the needs of all children. Medication can be combined and

sculpted as needed to meet individual needs, with minimum side effects (Adesman, 2003; Leslie, 2002).

Clinical effects of stimulants

Research confirmed long-term adverse effects of stimulants in the area of growth restriction (height and weight), which require careful monitoring (Bennett et al., 1999). Bennett et al. (1999) also found that those who are treated appropriately with stimulant medication are not at higher risk of substance abuse than their peers who are not treated with stimulants.

The short-term effects of stimulant medication refer to mild levels of decreased appetite, insomnia, anxiety, irritability and emotional lability in more than 50% of the sample. The possible development of abnormal motor movements or tics is of considerable concern. Behavioural rebound or the deterioration of behaviour to a level worse than baseline as the medication wears off, are other frequent adverse effects. High rates of passive and submissive behaviour were found when children received stimulants medication (Bennett et al., 1999).

3.10.2 Behaviour therapy

Parent training in behaviour therapy and teacher training in classroom intervention appear to be effective (Hannah, 2002; Meyer & Aase 2003; Stein, 2002; Swanson, 2003; Swanson et al., 1998). Behavioural programmes attempt to enhance parents' understanding of a child's behaviour and to teach specific skills

of behaviour modification, such as techniques for giving commands, rewarding adaptive and positive social behaviour (positive reinforcement), as well as decreasing or eliminating inappropriate behaviour (for example punishment) (Hannah, 2002; Stein, 2002). Hannah (2002) and Swanson (2003) also emphasise social skills training and cognitive-behavioural treatment (such as self-reinforcement, self-monitoring and problem-solving strategies).

According to Kaplan and Sadock (1998) and Rapport et al. (2001) behavioural interventions focus on a wide range of behaviour such as academic performance, rule following, social skills or peer and parent interactions. These interventions are used in various settings (for example at home, school, extracurricular, leisure) and require involvement by others (such as mental health professionals, parents, teachers, peers and siblings). Barkley (1998) emphasises that a more structured environment can be a significant complement to medication.

Parent management training seems to be effective in reducing the child's disruptive behaviour, as well as increasing the parents' own self-confidence and decreasing family stress (Cantwell, 1996).

3.10.3 School placement

Some children with ADHD may need individual tutoring, some may need a self-contained special class (for academic reasons) and others with complex problems may need a special school (Cantwell, 1996; Stein, 2002). Silver (1999)

emphasises the importance of addressing learning disabilities, as discussed in section 3.7.2.

3.10.4 Child-focussed interventions

Individual psychotherapy is often indicated in the treatment of depression, low self-esteem, anxiety, poor impulse control and anger control, as well as improvement of social skills and problem-solving skills (Cantwell, 1996). Heptinstall and Taylor (2002) postulate that treatment to promote psychological adjustment in the longer term needs to be implement, based on the strengths and weaknesses of the child's development and environment. Education for individuals with ADHD is also an essential part in the management of ADHD (Bennett et al., 1999; Robin, 2002). Mental health professionals may play a significant role in cases of family-related stress, psychopathology in the family, domestic violence, substance abuse and other conditions (Leslie, 2002).

Medical treatment alone seems to be more effective for the core symptoms of ADHD compared to behavioural treatment alone (Greene & Ablon, 2001; Rapport et al., 2001; Stein, 2002; Swanson, 2003). However, according to Hannah (2002) a combination of behavioural intervention and medication is more effective than either treatment alone. According to Hechtman (1999) the impact of other factors (such as family functioning) may be more important in predicting outcome than medication itself.

3.11 Summary

ADHD is characterised by developmentally inappropriate levels of inattentiveness, overactivity and impulsiveness, which affect approximately 3% to 6% of primary school children. The disorder seems to be more common in boys than in girls. However, during adolescence and young adulthood relatively more females are affected. Females seem to have more inattention symptoms, while males have more hyperactive symptoms.

ADHD is considered to be a marker for other psychiatric and developmental conditions. Therefore, mental health professionals should be alert to comorbid disorders such as learning disabilities, ODD, CD, depression, anxiety, substance abuse and others.

Aetiological factors are intertwined. Biological factors (for example genetics) are influenced by environmental factors (such as prenatal influences, exposure to toxins, physical abuse) and vice versa. Alterations in dopaminergic and noradrenergic functions appear to be the mainly factors implicated in the symptoms of ADHD.

ADHD, especially inattention and impulsiveness, often persists into adolescence and adulthood. High-risk behaviour is associated with ADHD in adolescents.

A multimodal treatment approach is followed, including medication (mainly stimulants), education of the parents, child and teacher, as well as behavioural management (with psychosocial interventions) in various settings.

Chapter 4

THE RELATION BETWEEN ATTENTION-DEFICIT/ HYPERACTIVITY DISORDER AND LOW BIRTH WEIGHT

4.1 Introduction

Elaboration on both dependent and independent variables, that is, ADHD and LBW, calls for a clarification of the relationship between these two variables. The aim of this chapter is to reflect available research on the relation between LBW and ADHD and to explore the nature of this hypothesised relationship. The following areas will be the focus of discussion: the incidence of ADHD in LBW children, biological structures involved in LBW children with ADHD, risk factors, gender, comorbidity and treatment.

4.2 Incidence of ADHD in LBW children

Before discussing the incidence of ADHD in LBW children, it is important to note that increasing survival rates for the smallest infants consequently imply increasing rates of disability in some domain(s) (Lou, 1996; Winders & Burns, 2001).

Most research studies confirm that there is a significant relationship between LBW and ADHD (Bhutta et al., 2002; Breslau et al., 1996b; Breslau et al., 2000; Harrison, 1996; Mick et al., 2002; Saigal et al., 2001; Szatmari et al.,

1990; Ulvund et al., 2001; Winders & Burns, 2001). Botting et al. (1997) state that the prevalence of ADHD in the 12-year-old VLBW group was 22% in comparison to 6% in the control group. Whitaker et al. (1997) found that 22% of the six-year-old LBW sample had at least one psychiatric disorder, the most common being ADHD (15.6%). Ulvund et al. (2001) report a 27% prevalence of ADHD in eight- to nine-year-old VLBW children. According to Barlow and Lewandowski (2000) preterm children were four to six times more likely than the national estimates of 3% to 5% of the general population to be diagnosed with ADHD. Increased problems with inattention and hyperactivity have been reported for samples of VLBW children ranging in age from five years to adolescence (Chapieski & Evankovich, 1997).

Bhutta et al. (2002) state that 67% of 15 studies that assessed children for ADHD found a significantly higher prevalence of attention problems, 69% of 13 studies demonstrated a significantly higher prevalence of externalising symptoms, while 75% of 12 studies found a significantly higher prevalence of internalising symptoms in preterm children. In short, the meta-analysis performed by Bhutta et al. (2002) shows that children born preterm have a 2.64-fold risk of developing ADHD, as well as frequently manifesting externalising or internalising behaviour at school age.

Behavioural and attention problems were more common in infants weighing less than 750 g at birth (Avery et al., 1999). Szatmari et al. (1990) report

that 16% of children between 500 to 1 000 g birth weight had attention deficiencies with hyperactivity, compared with 6.9% of controls.

According to O'Keeffe et al. (2003) there are small but significant differences between SGA children and normal control children regarding attention problems, poor concentration and hyperactivity/distractibility.

York and DeVoe (2002) noted that the presentation of ADHD may differ among premature babies. Premature infants with ADHD have more significant attention problems, but less hyperactivity.

Chapieski and Evankovich (1997) report more inconsistent findings of studies, which have included children with birth weights above 1 500 g. However, Breslau and Chilcoat (2000) indicate that the increased prevalence of psychiatric problems also applies to LBW children with a birth weight of more than 1 500 g. Stathis et al. (1999) also report no difference in the prevalence of ADHD between controls and children with ELBW, small head circumference or those with changes in head-circumference growth velocity (HGV) during the first year. Sommerfelt et al. (1996) found that inattention and hyperactivity were not prominent among five-year-old LBW children. Elgen, Lundervold and Sommerfelt (2004) indicated no specific identifiable attentional dysfunctions in the LBW group, as well as continued uncertainty about the nature of the reported impaired attention among LBW children.

Harrison (1996) emphasises that some premature children have symptoms of ADHD that do not reach the full diagnosis. Ideally a behaviour continuum should be referred to rather than an absolute diagnosis.

4.3 Biological structures involved in LBW children with ADHD

According to Cherkes-Julkowski (1998) preterm children are at increased risk of developmental difficulties because the CNS is not fully organised at the time of birth for sustaining itself in the extra-uterine environment. Prematurity places a child at risk of failure to achieve the self-regulation required for autonomic function. Preterm children are also at risk of dysregulation of attention and arousal. Cherkes-Julkowski (1998) further postulates that attention regulation reflects the regulation of the CNS to a great extent. Children with a compromised CNS have difficulty regulating internal arousal well enough, in order to tolerate environmental stimulation. They tend to allocate arousal/attention resources to internal-state regulation rather than to the external stimulus field. These problems with attention have been found in almost all groups of premature children with disabilities at school age (Cherkes-Julkowski, 1998). According to Winders and Burns (2001) impairments in the development of the self-regulation of attention may explain deficits in the acquisition of cognitive skills as well as other deficits. Specific variables may directly, indirectly or in both ways influence mechanisms and processes underlying the development of attention in LBW children.

Harrison (1996) confirms that children with birth weights below 1 000 g have specific biologically based problems of attention. Michelsson (1986) documents that prenatal insults and neonatal risk factors have an additive effect on the origin of neurodevelopmental disturbances.

Whitaker et al. (1997) state that parenchymal lesions/ventricular enlargement increase the risk of normal intelligent children to contract any disorder, ADHD and separation anxiety, irrespective of sex or social advantage. Perinatal white matter lesions are correlated with ventricular enlargement and with ischemic/infarctive lesions of the basal ganglia, brainstem and cerebellum. Cortical development might be adversely affected in preterms by germinal matrix haemorrhage/intraventricular haemorrhage and ischemic white matter injury because of their effects on late migration, organisation and myelination. In spite of inconsistent results, white matter injury (parenchymal lesions/ventricular enlargement) seems to increase the risk of some psychiatric disorders at age six years in LBW children significantly, especially for ADHD. Thus, elevated rates of ADHD in LBW children do not seem to be caused by birth weight or gestational age independently, but by higher rates of parenchymal lesions/ventricular enlargements (Whitaker et al., 1997).

Premature infants are particularly prone to developing ADHD, owing to repeated hypoxic-ischemic events with modest systemic circulatory failure. The striatum is vulnerable in these repeated hypoxic-ischemic events (Lou, 1996).

Whitaker et al. (1997) confirm that maturation of the striatum, which plays an important role in the improvement of behavioural inhibition that normally occurs in middle childhood, is deficient in ADHD. Post-mortem studies demonstrated that ischemic/infarctive white matter lesions were associated with ischemic/infarctive lesions of the basal ganglia, as well as hypoperfusion (poor blood circulation). These findings are confirmed by structural and functional brain imaging studies of children with ADHD, which have found abnormalities in the basal ganglia, specifically the corpus striatum (caudate and putamen). The striatum receives dopaminergic input (the neurotransmitter mainly involved in ADHD) from the substantia nigra. Alterations of the dopaminergic system have been associated with ischemic brain injury in human neonates. The striatal dopaminergic system seems to be more vulnerable to ischemic injury than other striatal neurotransmitter systems. As these children undergo puberty, which is accompanied by cortical maturation and synaptic pruning, it is possible that parenchymal lesions/ventricular enlargement will increase the risk of disorders that typically have a later onset and in which abnormalities of cortical-basal ganglionic circuits have been implicated. However, this last statement needs further investigation (Whitaker et al., 1997).

Harrison (1996) notes that the metabolic activity and blood flow patterns of children with suboptimal gestational or perinatal conditions showed hypoperfusion in the frontal lobe and symmetrically throughout both

hemispheres of the brain. This finding is consistent with an aetiological role for early hypoxic-ischemic lesions (for example periventricular leucomalacia).

Krageloh-Mann et al. (1999) report that low oxygen delivery to the brain was found in 63% of preterm born children, in contrast to 12.5% in those with normal magnetic resonance imaging (MRI), which indicates neonatal hypoxia-ischemia as an significant factor. MRI abnormalities were mainly periventricular lesions, especially periventricular leucomalacia. Symptoms of ADHD were related to these mild MRI abnormalities.

According to Stathis et al. (1999) the first two years of life is a critical stage for normal head growth and brain development. Cortical glial cell multiplication, myelination and growth of the neocerebellum are problems often associated with reduced head size during this critical period. Stathis et al. (1999) further postulate that a small head circumference or a fall in the rate of head growth during the first two years of life, particularly the first four months after birth, has been associated with long-term disability. IUGR and a small head size at eight months in infants below 1 500 g at birth were predictors of cognitive dysfunction, poor academic achievement and behavioural problems at eight years. However, HGV seems to be a poor predictor of future learning problems after eight months of age. No association was found between head circumference or HGV and ADHD (Stathis et al., 1999).

Bhutta et al. (2002) document that eight-year-old children born preterm showed disproportionately smaller volumes of the sensorimotor cortex, other cortical areas, the corpus callosum, amygdala, hippocampus and basal ganglia. These smaller volumes were associated with significantly lower cognitive scores, an increased incidence of ADHD and other behavioural disorders. Bhutta et al. (2002) propose that the biological and environmental insults associated with preterm birth may promote some of these anatomical differences, since developing neurons are more vulnerable to cell death during the perinatal period.

Breslau et al. (1996b) state that brain injury as well as abnormal brain development during prenatal and perinatal stages have been suggested as the mechanisms in subtle visual-motor and visual-perceptual deficits, as well as hyperactivity in preterm children. Stathis et al. (1999) confirm that ADHD in ELBW children has also been associated with structural and functional changes in the developing brain. Bhutta et al. (2002) emphasise that physiological instability and exposure to early adverse experiences may have a persistent impact on brain development leading to negative cognitive and behavioural outcomes. According to Abernethy, Palaniappan and Cooke (2002) attention deficit in children born with VLBW does not correlate with conventional markers of perinatal brain injury, but may be related to global brain growth and the development of key structures (such as the caudate nuclei and hippocampal formations).

4.4 Risk factors

The risk of either a diagnosis or symptoms of ADHD in premature children increases as birth weight decreases (Botting et al., 1997; Chapieski & Evankovich, 1997). However, Elgen et al. (2002) found that behavioural problems and psychiatric disorders were as common in children born with a birth weight of less than 1 500 g compared to children born with a birth weight of between 1 500 and 2 000 g.

Chapieski and Evankovich (1997) also state that VLBW infants with temperamental abnormalities (such as being less adaptable, having irregularities in habits, being less persistent and more withdrawn) early in life may be most at risk of exhibiting symptoms of ADHD. Temperamental problems appear to be more common in children with a birth weight of less than 1 500 g (Chapieski & Evankovich, 1997). Temperament should be viewed with caution, as parents' perceptions about their children may be biased. Research on early temperament and later behaviour problems in premature infants is currently insufficient.

According to Chapieski and Evankovich (1997) and Harrison (1996) premature children who exhibit neurodevelopmental problems (such as ventricular enlargement and/or periventricular leucomalacia) and those who were sicker infants appear to be at increased risk of ADHD. Breslau et al. (1996a) also state that perinatal complications increase the risk of attention deficiencies in

children with comorbid disorders and with no family history of attention deficiencies.

The role of environmental factors in the expression of ADHD symptoms in premature children is unclear. However, available data indicate that ADHD symptomatology in premature children is associated with adverse social conditions such as lack of parental involvement, negative maternal attitude and lower SES (Chapieski & Evankovich, 1997). Breslau et al. (1996a) report interaction between social class and VLBW in connection with hyperactivity and motor-visual coordination. Bhutta et al. (2002) confirm inconsistencies regarding the effect of SES on neurodevelopmental outcomes of preterms. However, a recent study of preterms found that family factors were stronger predictors of school performance than were perinatal complications (Bhutta et al., 2002). Cherkes-Julkowski (1998) also confirms that caretakers who can help their at-risk infants to self-regulate attention and arousal often have infants who achieve adequate CNS organisation and consequent richer opportunities to learn.

Robson and Pederson (1997) found that attention problems were predicted by temperament, environment and the interaction between developmental status and quality of home environment.

Breslau et al. (1996b) emphasise that LBW infants with a familial vulnerability to ADHD might be at increased risk of ADHD. This aspect needs further research.

4.5 Gender

Chapieski and Evankovich (1997) report inconsistencies between studies investigating the relation between LBW, ADHD and gender. It appears that there is some indication that males may be more at risk, in spite of insufficient research on sex differences (Chapieski & Evankovich, 1997; Whitaker et al., 1997).

O'Keeffe et al. (2003) mention that girls with more severe growth restriction were at increased risk of attentional difficulties.

4.6 Comorbidity

According to Breslau et al. (1996a) ODD was the most common comorbid disorder in both LBW and NBW children with ADHD. Breslau et al. (1996a) further state that ADHD in LBW did not differ from ADHD in NBW children with respect to associated comorbidity. About 50% of LBW as well as NBW children with ADHD met criteria for at least one additional disorder.

Botting et al. (1997) state that little research has been done on the possible long-term impairments hyperactivity or attention deficit may cause VLBW children or about other psychiatric outcomes of VLBW. Botting et al. (1997) found that ELBW children with hyperactivity were not developing antisocial behaviour or CD as frequently as matched peers who had also been recognised as hyperactive. This might imply a different aetiology or development of attention deficit in ELBW infants to that of NBW babies. The lower incidence of

comorbid antisocial behaviour may also indicate that VLBW children may present with a "purer" form of ADHD than that found in the general population, for whom social factors may be more important. A significant increase in anxiety was reported in VLBW children (Botting et al., 1997; Breslau et al., 1996a; Chapieski & Evankovich, 1997).

According to O'Callaghan and Harvey (1997) ELBW children experiencing symptoms of ADHD are likely to suffer social and learning impairments, which should be the focus of clinical attention, rather than perinatal risk factors.

Cherkes-Julkowski (1998) notes that in the context of a number of identified disorders, the possibility should be kept in mind that no one disorder is primary, but all may be manifestations of the same underlying condition.

According to Botting et al. (1997) it is possible that the combination of ADHD, motor impairments and poor mathematical ability seen in some VLBW children may indicate a deficit in mechanisms that may link these abilities, such as visuospatial or working memory impairment.

According to Harrison (1996) new research data indicate that behaviour problems in premature children may worsen with age, though data seem to be inconsistent.

The relationship between LBW and ADHD seems to vary by level of IQ. LBW children with an IQ below 80 showed the highest rate of ADHD. Those

with an IQ between 80 and 99 exhibit an intermediate rate. LBW children with an IQ greater or equal to 100 were not more likely than NBW children to be diagnosed with ADHD (Breslau et al., 1996b).

4.7 Treatment

Treatment of LBW children with ADHD seems to be similar to NBW children with ADHD, as discussed in Chapter two. Treatments thus include interventions such as psychopharmacology, parental counselling, classroom interventions, direct therapy with the child to improve organisational, social and adaptive skills, as well as psycho-education of the child, parents and school system (Chapieski and Evankovich, 1997).

4.8 Summary

Most research studies demonstrate that ADHD is the most common psychiatric disorder among LBW children. However, various studies found no significant difference in the prevalence of ADHD between NBW and LBW children.

It appears that brain injury, as well as abnormal brain development during prenatal and perinatal stages, puts LBW children at increased risk of developing symptoms of ADHD. These findings support the hypothesis that LBW children, especially those born with birth weights of less than 1 000 g, have specific biologically based problems of attention.

Lower birth weight, possible temperamental abnormalities, neurodevelopmental problems, poor medical status, environmental factors and a family history of ADHD increase the risk of LBW children to develop ADHD symptomatology. Lower IQ in LBW is associated with higher rates of ADHD.

Treatment of ADHD in LBW children seems to be similar to ADHD treatment in NBW children.

Chapter 5

PROBLEM STATEMENT

5.1 Introduction

As stated in section 4.2, increasing survival rates for the smallest infants imply increasing rates of disability (Lou, 1996; Winders & Burns, 2001). Jobe (2001) confirms that short-term and longer-term outcomes are not improving, though survival rates are increasing.

Avery et al. (1999) describe specific types of developmental morbidities in LBW cohorts, for example social problems, lower attention, cognitive delays (for example lower IQ), remedial assistance, speech and language disorders, persistent neuro-motor abnormalities and perceptual problems. Elgen et al. (2002) document that 27% of LBW children compared to 9% of NBW children were diagnosed with a psychiatric disorder. Chapieski and Evankovich (1997) report that 10% of a sample of premature children exhibited an emotional disturbance severe enough to interfere with their development and to make them difficult to manage.

LBW seems to be one of the various intertwined contributing factors in the development of ADHD (Astbury et al., 1987). Harrison (2002) also found that 22% of LBW children had at least one psychiatric disorder, the most common

being ADHD (16%). ADHD is characterised by developmentally inappropriate levels of inattention, over-activity and impulsiveness (Johansen et al., 2002), which cause clinically significant impairments in social, academic or occupational functioning (American Psychiatric Association, 2000). Cantwell (1996) notes that, if untreated, ADHD predisposes a child to psychiatric and social pathology in later life. ADHD therefore constitutes one of the strongest risk factors known for mental illness in early adult life.

The focus of this chapter is to highlight the purpose of the study as a whole, by addressing the problem statement, aim of the study and the hypotheses.

5.2 The problem: The relation between LBW and ADHD

5.2.1 Problem statement

The main focus of this study is to investigate the prevalence of ADHD symptoms among current primary school children, born with LBW, in the Limpopo Province.

LBW children seem to be vulnerable to neurodevelopmental disturbances due to prenatal complications and neonatal risk factors (Breslau et al., 1996a; Lindahl & Michelsson, 1986).

Most available research reflects a significant relationship between LBW and ADHD, as indicated in section 4.2 (Bhutta et al., 2002; Breslau & Chilcoat, 2000; Breslau et al., 1996a; Harrison, 1996; Mick et al., 2002; Saigal et al., 2001; Szatmari

et al., 1990; Ulvund et al., 2001; Winders & Burns, 2001). However, various studies reported inconsistent findings (Chapieski & Evankovich, 1997) or no significant prevalence of ADHD symptoms among children born with LBW (Elgen et al., 2003; Sommerfelt et al., 1996; Stathis et al., 1999).

Research findings also appear to be inconsistent regarding the specific nature of present inattentive and/or hyperactive-impulsive symptoms. Attention problems are significant among premature babies, but they exhibit less hyperactivity (Papageorgiou & Bardin, 1999; York & DeVoe, 2002). Elgen et al. (2003) could find no identifiable pattern of inattention among LBW children.

Accardo (1999) and Barkley et al. (2002) state that the presence of ADHD should be considered as a marker for other psychiatric and developmental conditions, such as learning disabilities, ODD, antisocial behaviour, CD, depression, anxiety, substance abuse, poor social skills and others. These functionally impairing symptoms of ADHD may continue into adolescence and adult life (Taylor et al., 1998). Cantwell (1996) notes that, if untreated, ADHD predisposes a child to psychiatric and social pathology in later life. ADHD therefore constitutes one of the strongest risk factors known for mental illness in early adult life.

It is essential to determine if there is a relationship between LBW and ADHD, as well as to clarify the nature, aetiology and consequences of such a possible relationship. Determination of functioning and needs in specific areas

(behavioural and neuropsychological) will enable professionals and significant people to develop relevant treatment strategies. It is hoped that research data will enable all parties involved to optimise the outcomes of LBW children, where necessary.

5.2.2 Aim of the study

The main aim of this research study was two-fold:

1. to study the number of ADHD symptoms on the disruptive behaviour disorder (DBD) rating scale among LBW children in comparison with NBW children
2. to establish whether the performance of LBW children is lower on ADHD-sensitive tests than that of NBW children.

5.2.3 Hypotheses

Research hypothesis 1:

Children with LBW will have a higher incidence of ADHD, ODD and CD symptoms than children with NBW. There will be differences in scores between the genders.

Null hypothesis 1:

Children with LBW will not have a higher incidence of ADHD, ODD and CD symptoms than children with NBW. There will be no differences in scores between the genders.

Specific null hypotheses derived from Null hypotheses 1:

1.1 Children with LBW will not have more inattentive symptoms on the DBD rating scale than children with NBW. There will be no differences between the genders.

1.2 Children with LBW will not have more hyperactive/impulsive symptoms on the DBD rating scale than children with NBW. There will be no differences between the genders.

1.3 Children with LBW will not have more ODD symptoms on the DBD rating scale than children with NBW. There will be no differences between the genders.

1.4 Children with LBW will not have more CD symptoms on the DBD rating scale than children with NBW. There will be no differences between the genders.

Research hypothesis 2:

Children born with LBW will perform less well in tests that measure the cortical areas supplied by the mesocortical dopamine branch, in other words, executive functions, than children with NBW. There will be differences in scores between the genders.

Null hypothesis 2:

Children born with LBW will not perform less well in tests that measure the cortical areas supplied by the mesocortical dopamine branch (executive functions), than children with NBW. There will be no differences in scores between the genders.

Specific null hypotheses derived from Null hypotheses 2:

2.1 Children with LBW will not have lower scores on the Tower of London (ToL) Test than children with NBW. There will not be differences in performance between the genders.

2.2 Children with LBW will not have higher perseverative error scores on the Wisconsin Card Sorting Test (WCST) than children with NBW. There will not be differences in performance between the genders.

2.3 Children with LBW will not make more non-perseverative errors in the WCST than children with NBW. There will not be differences in performance between the genders.

2.4 Children with LBW will not achieve fewer categories on the WCST than children with NBW. There will not be differences in performance between the genders.

Research hypothesis 3:

Children born with LBW will perform less well in the test that measures the cortical areas supplied by the nigro-striatal dopamine branch, in other words, motor functions, than children with NBW. There will be differences in scores between the genders.

Null hypothesis 3:

Children born with LBW will not perform less well in the test that measures the areas supplied by the nigro-striatal dopamine branch (motor functions), than children with NBW. There will be no differences in scores between the genders.

Specific null hypothesis derived from Null hypothesis 3:

3.1 Children with LBW will not have higher scores on the Maze Coordination Test with the dominant hand than children with NBW. There will be no differences in performance between the genders.

3.2 Children with LBW will not have higher scores on the Maze Coordination Test with the non-dominant hand than children with NBW. There will be no differences in performance between the genders.

The next chapter will elaborate on the statistical tests employed to accept or reject these previously formulated hypotheses.

Chapter 6

METHODOLOGY

6.1 Introduction

This chapter will be reporting on the research design applied, sampling and demographic characteristics of the sample. Descriptive statistics for responses on the biographical data questionnaire (Appendix F) will be summarised in Tables 6.7 to 6.10. The measurement instruments, procedures and methods of analysis will also be referred to.

6.2 Research design

This research investigation will focus on the prevalence of ADHD symptoms in LBW children by means of participant observation in the form of neuropsychological testing, as well as a rating scale. Scores on neuropsychological tests and the rating scale should be able to verify the hypotheses.

A control group of children with NBW will be used to determine differences in the prevalence of ADHD symptoms in LBW children and NBW children. A control group is characteristic of an experiment, which refers to quantitative research.

This is a quantitative research study, with a quasi-experimental research design, as the subjects cannot be randomly assigned to the conditions of the independent variable, because they already exhibit the variable. Methods used in this study, such as direct observation, a questionnaire and a rating scale, are also characteristic of quantitative research (Brynard & Hanekom, 1997).

6.3 Sample

The sample was drawn from seven randomly selected urban, mainstream, primary schools in the Polokwane magisterial district. These seven schools were representative of all cultures. All primary school children with a birth weight of 2 000 g and lower were included in the sample. The control group (consisting of children with a birth weight above 3 000 g) was matched with the experimental group for age, sex, SES, language and racial group.

The demographic characteristics regarding age and birth weight are reflected in Table 6.1

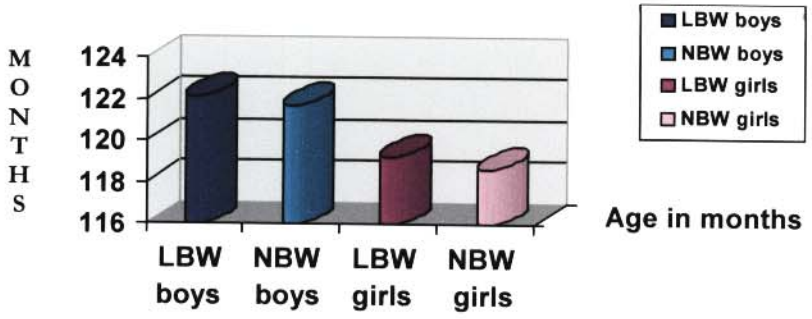
Table 6.1 Demographic characteristics (age and birth weight groups)

Group	N	Age in months Means	Birth weight Means
LBW boys	21	122.14 ± 23.95	1620.00 ± 340.24
LBW girls	34	119.26 ± 20.90	1620.32 ± 280.09
NBW boys	21	121.71 ± 23.96	3797.86 ± 547.02
NBW girls	34	118.68 ± 21.47	3585.44 ± 308.59
All groups	110	120.10 ± 22.01	2643.37 ± 1091.14

The differences in age between the normal and LBW groups were statistically insignificant (p=0.92) while the differences in birth weight between the normal and LBW group were statistically significant (p=0.00).

Figure 6.1 illustrates the mean age in months for gender groups.

Figure 6.1 Mean age (in months) for the gender and weight groups



The mean birth weight in grams for gender groups is illustrated in Figure 6.2.

Figure 6.2 Mean birth weight for the genders

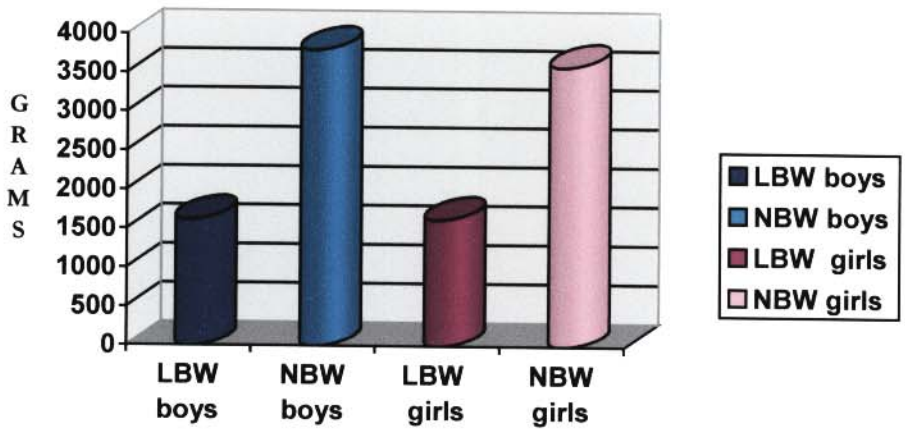


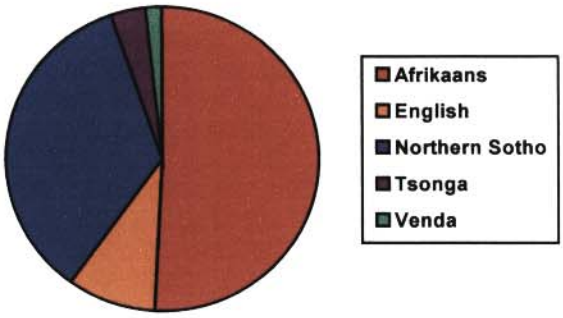
Table 6.2 reflects the language distribution of the sample.

Table 6.2 Language distribution of the sample

	Frequency	Percentage
Afrikaans	56	50.9
English	10	9.1
Northern Sotho	38	34.5
Tsonga	4	3.6
Venda	2	1.8
Total	110	100.0

Figure 6.3 illustrates the language distribution of the sample.

Figure 6.3 Language distribution of the sample



The gender distribution of the sample is indicated in Table 6.3

Table 6.3 Gender distribution

	Frequency	Percentage
Male	42	38.2
Female	68	61.8
Total	110	100.0

The gender distribution of the sample is illustrated in Figure 6.4.

Figure 6.4 Gender distribution of the sample

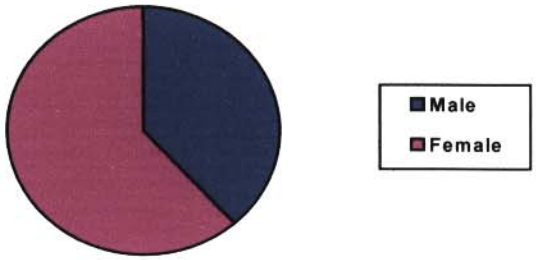


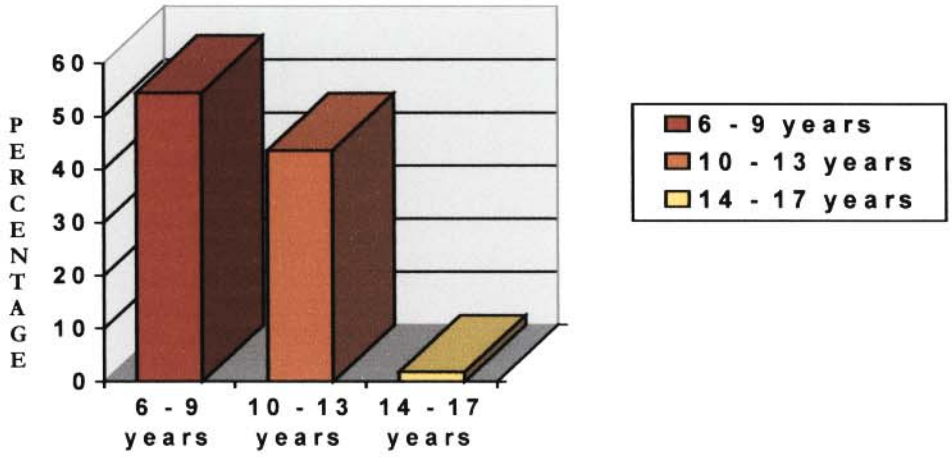
Table 6.4 gives an overview of the differentiation of the respective age groups.

Table 6.4 Differentiation of age groups

	Frequency	Percentage
6-9	60	54.5
10-13	48	43.6
14-17	2	1.8
Total	110	100.0

Figure 6.5 illustrates the differentiation of age groups.

Figure 6.5 Distribution of age groups



The grade distribution of the sample is represented in Table 6.5

Table 6.5 Grade distribution

Grade	Frequency	Percentage
1	18	16.4
2	14	12.7
3	29	26.4
4	16	14.5
5	11	10.0
6	18	16.4
7	4	3.6
Total	110	100.0

The grade distribution is illstrated in Figure 6.6.

Figure 6.6 Grade distribution of the sample

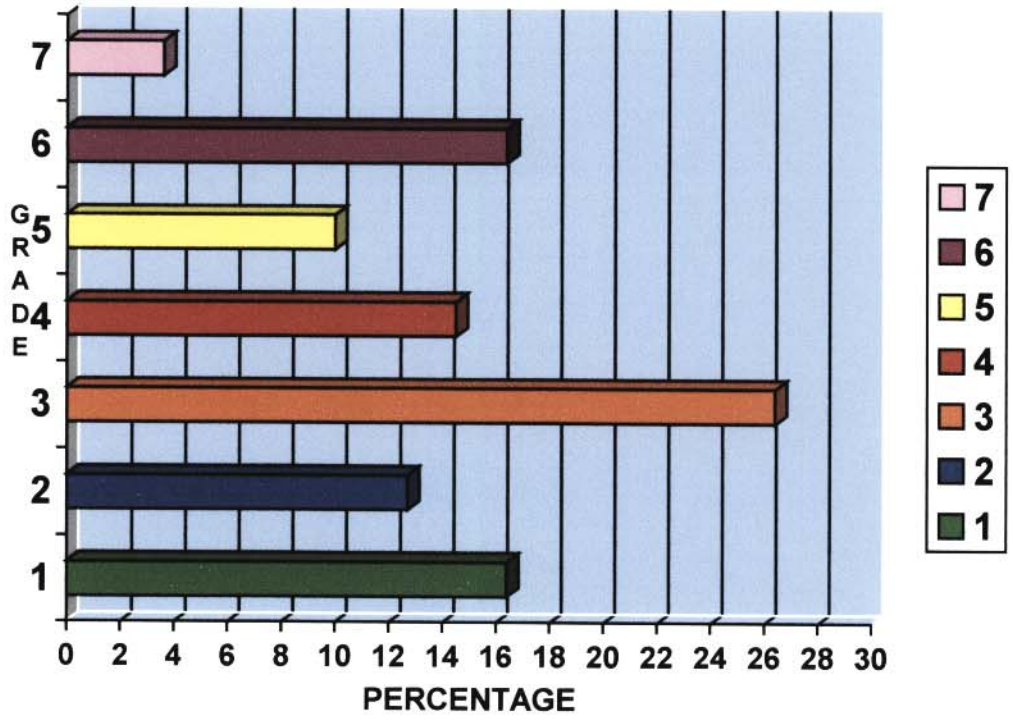


Table 6.6 represents the length of pregnancy in weeks.

Table 6.6 Length of pregnancy in weeks

Weeks	Frequency	Percentage
24	1	0.9
28	13	11.8
30	4	3.6
32	9	8.2
35	2	1.8
36	3	2.7
37	1	0.9
38	9	8.2
40	37	33.6
41	1	0.9
Total	80	72.7
Unknown	30	27.3
Total	110	100.0

Thus, 29% of the respondents were born prematurely, excluding the missing responses.

Figure 6.7 illustrates the length of pregnancy in weeks.

Figure 6.7 Duration of pregnancy

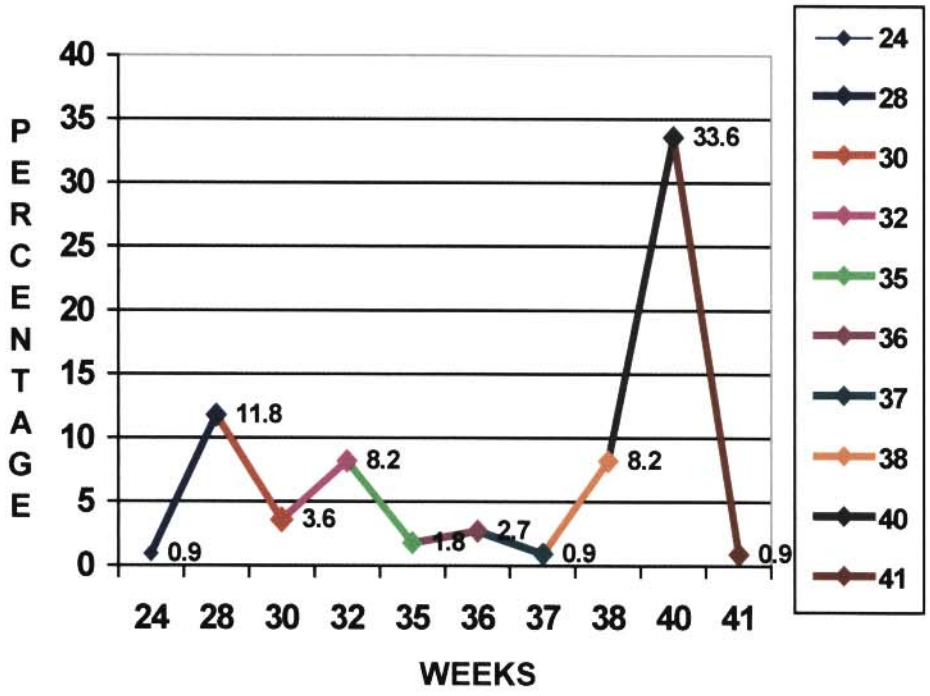


Table 6.7 gives an overview of the occurrence of specific conditions during pregnancy or delivery (see Appendix F). The results of Table 6.7 to Table 6.10 reflect the total responses of the sample group, including those who did not respond at all.

Table 6.7 Occurrence of conditions during pregnancy/delivery – Descriptive statistics for birth weight groups

	LBW				NBW			
	Boys		Girls		Boys		Girls	
During pregnancy/delivery the following occurred:	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Received proper medical care	16	76	22	65	15	71	24	71
Followed a well-balanced diet	17	81	21	62	15	71	24	71
Bleeding	6	29	11	32	4	19	3	9
Excessive weight gain (more than 20 kg)	4	19	6	18	5	24	4	12
Low weight gain by mother	4	19	6	18	1	5	4	12
Toxaemia / pre-eclampsia	2	10	3	9	2	10	1	3
Rh factor incompatibility	1	5	0	0	1	5	3	9
Frequent nausea or vomiting	2	10	4	12	2	10	4	12
Maternal anaemia	1	5	2	6	0	0	0	0
Serious illness (e.g. infections, hypertension or diabetes mellitus) or injury	2	10	4	12	1	5	1	3
Took prescription medication	2	10	5	15	3	14	1	3
Took illegal drugs	0	0	0	0	0	0	1	3
Used alcoholic beverage	2	10	0	0	0	0	2	6
Smoked cigarettes	4	19	3	9	1	5	2	6
Was given medication to ease labour pains	5	24	2	6	3	14	5	15
Delivery was induced	3	14	2	6	9	43	6	18
Forceps were used during delivery	0	0	3	9	3	14	3	9
Had a breech delivery	1	5	0	0	0	0	0	0
Had a caesarean section delivery	7	33	13	38	7	33	10	29

Table 6.8 indicates the occurrence of specific conditions, which affected the child during delivery or in the first few days after birth (see Appendix F).

Table 6.8 Conditions affecting the child during delivery or in the first few days after birth – Descriptive statistics for birth weight groups

	LBW				NBW			
	Boys		Girls		Boys		Girls	
During delivery or in the first few days after birth the following occurred:	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Injured during delivery	0	0	0	0	1	5	1	3
Cardiopulmonary (heart) distress during delivery	2	10	1	3	0	0	0	0
Delivered with cord around neck	0	0	0	0	0	0	2	6
Had trouble breathing following delivery	8	38	7	21	0	0	0	0
Needed oxygen	10	48	16	47	0	0	1	3
Turned blue	4	19	5	15	0	0	2	6
Was jaundiced, turned yellow	4	19	6	18	1	5	3	9
Had an infection	2	10	2	6	0	0	0	0
Had seizures	0	0	0	0	0	0	0	0
Was given medication	8	38	10	29	0	0	0	0
Born with a congenital defect	1	5	0	0	0	0	0	0
Was in hospital for more than seven days	11	52	23	68	1	5	1	3

Table 6.9 reflects the health and temperamental characteristics during the first 12 months of the child (see Appendix F).

Table 6.9 Health and temperamental characteristics during the first 12 months of the child – Descriptive statistics for birth weight groups

	LBW				NBW			
	Boys		Girls		Boys		Girls	
During the first 12 months the child had the following health and temperamental characteristics:	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Difficult to feed	5	24	11	32	0	0	1	3
Difficult to get to sleep	2	10	7	21	0	0	5	15
Colicky	1	5	6	18	1	5	3	9
Difficult to put on a schedule	1	5	7	21	0	0	3	9
Alert	12	57	17	50	11	52	15	44
Cheerful	14	67	20	59	16	76	22	65
Affectionate	9	43	17	50	15	71	19	56
Sociable	13	62	19	56	16	76	22	65
Easy to comfort	14	67	20	59	16	76	19	56
Difficult to keep busy	3	14	3	9	2	10	3	9
Overactive, in constant motion	5	24	7	21	2	10	6	18
Very stubborn, challenging	0	0	7	21	2	10	8	24

Table 6.10 shows the occurrence of health problems at any time of the child's life (see Appendix F).

Table 6.10 Health problems – Descriptive statistics for birth weight groups

	LBW				NBW			
	Boys		Girls		Boys		Girls	
The child experienced the following health problems at some time:	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Asthma	5	24	7	21	1	5	3	9
Allergies	10	48	11	32	5	24	6	18
Diabetes, arthritis or other chronic illness	1	5	1	3	0	0	1	3
Epilepsy or seizures	0	0	25	74	0	0	1	3
Febrile seizures	0	0	1	3	0	0	1	3
Heart or blood pressure problems	1	5	2	6	1	5	0	0
High fever (over 38°C)	4	19	7	21	1	5	3	9
Head injury with loss of consciousness	1	5	0	0	0	0	0	0
Lead poisoning	0	0	0	0	0	0	0	0
Lengthy hospitalisation	6	29	5	15	1	5	3	9
Speech or language problems	5	24	3	9	1	5	1	3
Chronic ear infections	3	14	8	24	1	5	4	12
Hearing difficulties	1	5	3	9	1	5	3	9
Eye or vision problems	2	10	2	6	0	0	1	3
Fine motor/handwriting problems	5	24	2	6	1	5	2	6
Gross motor difficulties, clumsiness	2	10	2	6	1	5	1	3
Appetite problems (overeating/under-eating)	2	10	7	21	4	19	6	18
Sleep problems (falling asleep, staying asleep)	0	0	3	9	3	14	2	6
Soiling problems	3	14	4	12	0	0	1	3
Wetting problems	1	5	4	12	4	19	3	9

6.4 Measurement instruments

The following measurement instruments were used in this study:

6.4.1 DBD rating scale

The DBD rating scale (see Appendix G) was used for assessment of the presence and degree of ADHD-related symptoms (inattention and hyperactivity/impulsivity); ODD and CD, as formulated in the DSM-IV (Pelham, Jr. et al., 1992; Pillow et al., 1998). This scale was translated into the official languages of the Limpopo Province (Afrikaans, Pedi, Venda, Tsonga and Setswana) and norms were established for those populations groups (Meyer et al., 2004). There are 18 items on the scale that measure ADHD-related symptoms. Teachers and parents were asked to rate the behaviour on a four-point scale consisting of the following options: not at all (0); just a little (1); pretty much (2); and very much (3).

The total score is added up for ADHD-related symptoms, ODD and CD, and compared to the cut-off point of the 93rd percentile, which has previously been identified as clinically significant (Barkley, 1997a; Barkley & Murphy, 1998).

6.4.2 ToL Test

The ToL Test was used for the measurement of a dysfunctioning meso-cortical dopamine branch that will produce frontal dysfunction resulting in cognitive impulsiveness (Lezak, Howieson & Loring, 2004). Barkley (1997b), as well as Shallice et al. (2002), referred to the ToL Test as a "frontal lobe test".

Shallice initially developed the ToL Test in order to assess frontal lobe damage in adult patients (Culbertson & Zillmer, 1998; Shallice et al., 2002).

The ToL Test has been used in normal and neuropsychological populations for the assessment of planning ability (Lezak et al., 2004; Phillips et al., 2001). This task requires working memory, especially non-verbal working memory (Barkley, 1998). The ToL Test is also considered to be a measure of executive function that taps planning and problem-solving skills, as well as impulsive response styles (Blondis et al., 2000).

The test entails the construction of a presented design using coloured beads mounted on three vertical pegs of different lengths, using the least number of moves possible (see Appendix 0) (Culbertson & Zillmer, 1998). Forethought and planning are essential in testing out various ways of removing and replacing the beads on the set of pegs to match the design presented by the tester. This task requires substantial mental planning that must occur before and while undertaking actual motor execution or arrangement (Lezak et al., 2004).

6.4.3 WCST

The WCST was developed by Berg (1948) and modified by Heaton (Heaton & Pendleton, 1981). It seems to be one of the most commonly administered psychometric measures of frontal lobe dysfunction (Hodges, 1994; Lezak et al., 2004; Quinlan, 2000; Skilbeck, 1996). In this study the WCST was used for measurement of the areas supplied by the meso-cortical dopamine

branch. Hypofunctioning in these areas will produce frontal dysfunction resulting in cognitive impulsiveness (Heaton & Pendleton, 1981). Monchi et al. (2001) confirm that the WCST has been used to assess prefrontal cortex and basal ganglia dysfunctioning. However, recent research questions the WCST as a measure of frontal dysfunction (Gregory, 2000; Lezak et al., 2004; Quinlan, 2000; Russell, 1994). Lezak et al. (2004) emphasise that though the WCST is sensitive to frontal damage, it does not localise lesions and is not a reliable brain-damage screen.

It is a neuropsychological test that assesses the ability to form abstract concepts, to sustain attention and to shift cognitive set flexibly in response to changing rules (also referred to as mental flexibility), while inhibiting inappropriate responses. It measures organisational capacity, attention-shifting and sustained attention (Gregory, 2000; Schmitz et al., 2002). This instrument also measures problem-solving abilities (Blondis et al., 2000; Russell, 1994). The WCST is thus sensitive to general executive functions (Blondis et al., 2000; Gregory, 2000; Monchi et al., 2001; Russell, 1994).

The computerised version (Ormond Software Enterprises, 1998) of the WCST (see Appendix M) was used for testing and scoring, in order to avoid errors in the complex administration and scoring (Quinlan, 2000). The subject has to match 128 test cards to reference cards according to the colour, shape and number in response to stimuli that are depicted. The subject receives accuracy

feedback after every sorting that he/she must use to determine what the correct sorting rule is. The sorting rule (according to colour, shape or number) changes after every 10 consecutive responses without notice, and the subject must shift to a new mode of classification (Lezak et al., 2004; Monchi et al., 2001; Quinlan, 2000). The test begins with colour as the basis for sorting, shifts to form, then to number, returns again to colour, and continues in this order.

Scores are derived for number of criteria met, number of errors, number of perseverative answers, learning to learn (shifting efficiently after initial concepts), and loss of set (deviation from the correct criterion after three correct trials). Perseverative errors seem to be prominent in the performance of individuals with frontal lobe damage (Quinlan, 2000). Hodges (1994) and Lezak et al. (2004) state that the number of categories achieved and number of perseverative errors are the most widely used scores. It is however essential to note that even normal children find traditional card-sorting tasks (such as the WCST) difficult until they are 10 to 12 years of age (Beardsworth & Harding, 1996).

In this study the testees' performances were evaluated according to the following criteria:

a) Set Shifting

Categories shifted: The sorting rule shifts after every 10 correct consecutive responses. The rule changes through colour, form and number. The number

changes are denoted as categories achieved. Categories achieved refer to the number of correct runs of ten sorts (Lezak et al., 2004).

b) Perseveration

Perseverative errors: A perseverative error occurs when the subject continues to sort according to a previously successful principle or (in the first series) when the subject persists in sorting on the basis of an initial erroneous guess (Hodges, 1994; Lezak et al., 2004). According to Russell (1994) perseveration is the opposite of mental flexiblity. Lezak et al. (2004) state that the perseverative error score is an indication of problems in forming concepts, profiting from correction, and conceptual flexibility.

Non-perseverative errors: A non-perseverative error is an answer that is incorrect but not perseverative. According to Lezak et al. (2004) it is the difference between the total number of errors and perseverative errors. These other errors may include guessing, losing track of the current sorting principle and an effort to devise a complex scheme, which usually indicates that a verbally clever person has failed to keep track of the pattern of the examiner's responses or to accept the simplicity of the pattern (Lezak et al., 2004).

6.4.4 Maze Coordination Test

Gregory (2000) states that most neuropsychological test batteries include motor measures of manipulative speed and accuracy. The Maze Coordination Test was used in this study for the measurement of a dysfunctioning nigro-striatal dopamine branch that on a behavioural level, will cause poor motor control (Lezak et al., 2004). The subject is requested to follow a trail as fast as possible using a stylus with the dominant and non-dominant hand respectively, from the starting point to the finishing point of the maze, without touching the sides (Matthews & Kløve, 1964). There are two trials, for every trial the tester notes the touches and time taken. In the first trial the child uses the dominant hand and in the second trial the non-dominant hand (see Appendix N).

Based on the assumptions of other motor tests (such as the Finger-tapping Test), it is also assumed in this study that the subject will have improved performance with his/her dominant hand. Significant deviations in performance might indicate some kind of impairment in the hemisphere opposite that of the slowed hand (Gregory, 2000).

6.4.5 Raven's Coloured Progressive Matrices

The Raven's Coloured Progressive Matrices (CPM) was used as an intellectual screening device (Gregory, 2000) in order to identify subjects with an IQ below 75.

According to Hodges (1994) the CPM was developed as a 'culture fair' test of general intellectual inability. The subject is provided with a large design, part of which is missing. Below are six different small pattern-samples, one of which the subject chooses to complete the larger design above. The CPM consists of 36 items, with norms for children and adults over 65. This test appears to be sensitive to brain damage in widely distributed areas, since normal performance depends upon visuo-perceptual, attentional and problem-solving skills. The CPM is a reasonable test of frontal lobe function in the absence of visuo-perceptual deficits (Hodges, 1994).

6.5 Procedure

Written permission was obtained from the Department of Education, Limpopo Province, as well as the principals of selected schools. Principals of the seven selected schools were provided with information (see Appendix A) during a personal visit. The Ethics Committee of the University of Limpopo approved the study.

Letters (see Appendix B) were sent out to the parents of all pupils at the selected schools. These letters contained general information on the research project, as well as a return slip to fill in the child's birth weight and identifying particulars. The clinical group was compiled based on feedback from parents. All children with a birth weight of 2 000 g and below were included. Some

parents of pupils with NBW also returned the slips provided, which were used to identify control group participants.

Teachers were involved in the identification of possible control group subjects for every LBW child. The parents of possible control group subjects were telephonically contacted in order to inform them about the research project and to determine their birth weight. A subject with a birth weight above 3 000 g that matched a specific child in the clinical group in terms of sex, language, age, SES and race was included.

Two of the seven schools had *ed lab* files available, which sometimes provided a clinic card with birth weight information. Some of the clinical and control subjects were identified by making use of these files.

Parents of the clinical and control groups were given a letter describing the study in more detail and were asked to complete the following documentation (see Appendix C for the control group and Appendix D for the clinical group): a consent form (see Appendix E); biographical data questionnaire (see Appendix F); and DBD rating scale (see Appendix G). Teachers were also asked to complete a DBD rating scale for every child included in the sample. Parents were repeatedly encouraged to provide the researcher with the necessary documentation, by means of telephone calls and/or letters (see Appendix J).

Children on ADHD medication were requested not to take any for at least 24 hours prior to assessment. Children with an IQ below 75, a history of relevant physical impairment (such as auditory or visual impairment), neurological trauma, psychosis or other severe psychiatric disorder, were not included in this study.

Teachers were requested to identify subjects' level of intellectual functioning, based on their observations. The intellectual functioning of those with possible borderline (IQ between 71 and 84) or mild mental retardation (IQ between 55 and 70) was screened with the CPM Test. As already mentioned, clinical or control group subjects with an IQ below 75 were excluded from the study and replaced (in the case of control group subjects or excluded from the study in the case of LBW).

The complete test battery (Maze Coordination Test, ToL Test and WCST) was administered to the experimental and control groups at six schools. A research data collection sheet (see Appendix H) was used to document each child's performance. The children were tested during school hours. Before the actual neuropsychological testing commenced, the children were introduced to the researcher and informed about the procedure described below.

The researcher and one assistant conducted the testing. The researcher is registered with the Health Professions Council of South Africa as an intern clinical psychologist and has experience in the administration of psychometric tests. The assistant was involved with the administration of the other tests on

various other study groups of the same project, before commencing with this process of administration and was also well-versed in the assessment instruments.

Feedback was given to parents (see Appendix L) and school principals (see Appendix K) in the form of a letter, after statistical analysis.

6.6 Method of analysis

The computer programs SPSS 11 (SPSS, 1999) and STATISTICA 6 (StatSoft, 2003) were employed.

Analysis of variance (ANOVA) models were also used to investigate possible between-group differences in raw scores.

Chapter 7

RESULTS

7.1 Introduction

The aim of this study was to investigate the number of ADHD symptoms among LBW children in comparison with NBW children, as well as to determine whether LBW children performed more poorly on ADHD-sensitive tests than NBW children.

This chapter will report on data collected for testing the respective hypotheses.

7.2 Realisation of sample

As indicated in Chapter 6 (section 6.3), the sample consisted of 55 children with LBW in the clinical group and 55 children with NBW in the control group, who were all tested using the ToL Test, WCST and Maze Coordination Test.

A total of 110 teacher DBD rating scales were distributed to the specific teachers of the various schools, and a total of 110 parent DBD rating scales, as well as 110 biographical data questionnaires, were distributed via the children to the parents.

A total of 110 teachers returned their DBD rating scales. Eighty-two parents (74.5%) returned their DBD rating scales and biographical data questionnaires.

7.3 Results of the study

In order to test the respective hypotheses, results of the above-mentioned tests and rating scale are presented in the following format:

The results for the rating scale and each test are presented separately and include: descriptive statistics (in table and graph form); ANOVA results investigating possible between-group differences (groups were divided according to gender and birth weight), the post-hoc (Scheffé) test to indicate where the differences occur. These results are presented for both gender groups and birth weight groups for each test or rating scale with reference to the specific hypothesis.

7.3.1 ADHD symptomatology

The first phase of this investigation is to determine whether children with LBW will have a higher incidence of ADHD symptoms on both the hyperactive/impulsive and inattention scales of the DBD rating scale, than children with NBW, as well as whether any gender differences exist. The results are illustrated below in section 7.3.1.1.

7.3.1.1 DBD rating scale

Only teacher DBD rating scales were used, in view of a 100% return, in comparison to a 74.5% return of parent DBD scales. Moreover, teacher information seems to be more useful than parent information, particularly in discriminating among subtypes of children with ADHD (Crystal et al., 2001).

Table 7.1 represents the descriptive statistics for the DBD rating scale for hyperactive/impulsive and inattentive scores, for the LBW and controls.

Table 7.1 DBD ratings – Hyperactivity/impulsivity and inattention – Descriptive statistics for LBW and control groups

Group	N	H/I Means	Inatt Means
LBW boys	21	7.14 ± 6.47	9.43 ± 7.77
LBW girls	34	4.82 ± 5.78	5.15 ± 5.67
NBW boys	21	4.62 ± 5.43	5.29 ± 6.36
NBW girls	34	3.15 ± 3.99	2.74 ± 3.48
All groups	110	4.71 ± 5.47	5.25 ± 6.10

Figure 7.1 illustrates the plot of means for hyperactivity/impulsivity and inattention scores on the DBD rating scale, for gender groups.

Figure 7.1 Hyperactivity/Impulsiveness and Inattention scores on DBD

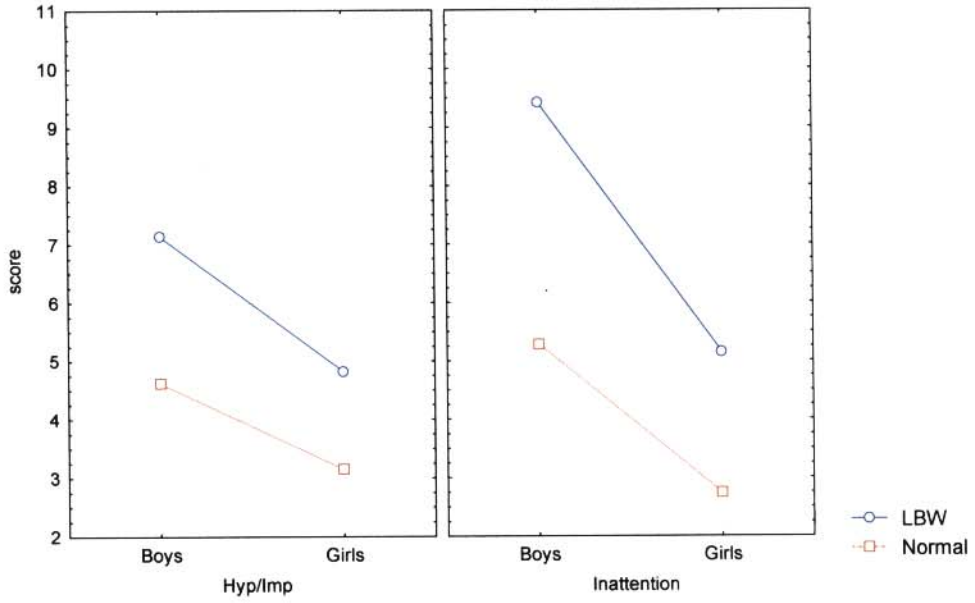


Table 7.2 reflects the results of the ANOVA for hyperactivity/impulsivity and inattention scores on the DBD rating scale, for the LBW and control groups.

Table 7.2 DBD ratings – Hyperactivity/impulsivity and inattention – ANOVA results for LBW and control groups

	df	**F**	***P***
H/I	3, 106	2.41	0.07
Inatt	3, 106	5.91	0.00*

* $p \leq 0.05$

The ANOVA shows statistically significant differences for only the inattention scale; however there was a tendency towards significance (p=0,07) for the hyperactive/impulsive scale.

The result of the post-hoc (Scheffé) test performed on the inattention scale of the DBD rating scale for gender groups, is demonstrated in Table 7.3.

Table 7.3 DBD ratings – Inattention – Post-hoc (Scheffé) results for the gender groups

	Inattention
Boys	0.02*
Girls	0.09

* $p \leq 0.05$

The ANOVA showed differences between the birth weight groups on the inattention scale. The post-hoc test indicated these significant differences for the LBW and NBW boys, but not for girls. However, there was a tendency towards significance for the girls (p=0.09).

Table 7.4 reflects the descriptive statistics for ODD and CD scores on the DBD rating scale, for the gender and weight groups.

Table 7.4 DBD ratings – ODD and CD – Descriptive statistics for the LBW and control groups

Group	N	ODD Means	CD Means
LBW boys	21	4.48 ± 4.45	1.52 ± 2.44
LBW girls	34	2.82 ± 4.28	1.56 ± 3.79
NBW boys	21	2.57 ± 3.36	0.67 ± 1.43
NBW girls	34	1.85 ± 2.57	0.41 ± 0,74
All groups	110	2.79 ± 3.75	1.03 ± 2.50

Figure 7.2 illustrates the plot of means for ODD and CD scores on the DBD rating scales, for the LBW and control groups

Figure 7.2 ODD and CD scores on DBD

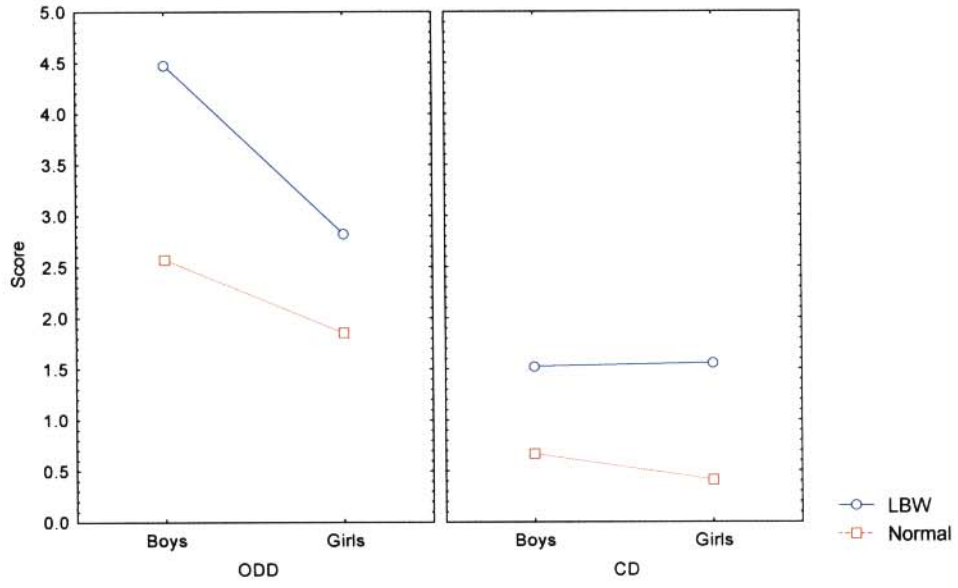


Table 7.5 represents the results of the ANOVA for ODD and CD scores on the DBD rating scale, for the gender groups.

Table 7.5 DBD ratings – ODD and CD – ANOVA results for gender groups

	df	F	*P*
ODD	3, 106	2.22	0.09
CD	3, 106	1.65	0.18

The ANOVA showed no significant differences between the birth weight groups on the ODD and CD scales, although there was a tendency towards significance for ODD (p=0.09).

Table 7.6 shows the correlations (Pearson's product-moment) between birth weight and all the DBD scores.

Table 7.6 Correlations (Pearson-r) between birth weight and DBD scales

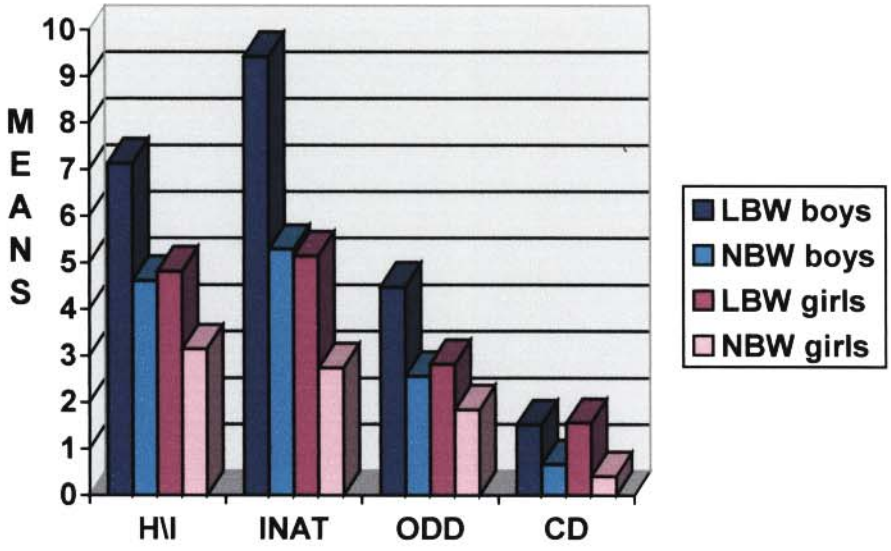
Variable	Correlation	Discrimination value
H/I	-.21*	Weak
Inattention	-.25*	Weak
Total ADHD	-.25*	Weak
ODD	-.18	None
CD	-.19	None

* $p \leq 0.05$

Thus, according to Table 7.6 there are weak significant correlations between birth weight and scores on the hyperactive/impulsive, inattentive and total ADHD scales, but no significant correlations for ODD and CD.

Figure 7.3 summarises the mean scores on all the DBD rating scales.

Figure 7.3 Mean DBD scores of the birth weight and gender groups



The descriptive statistics for full diagnoses for the LBW and control groups are indicated in Table 7.7

Table 7.7 Diagnoses – According to DBD scores

Diagnosis	LBW N	NBW N
ADHD	1	1
ODD	5	3
CD	6	0
ADHD and ODD	3	1
ADHD and CD	4	0

Table 7.8 depicts the numbers for each ADHD subtype for the LBW and NBW groups.

Table 7.8 ADHD subtypes diagnosed

ADHD Subtype	LBW N	NBW N
H/I	4	0
Inattention	1	0
Combined	3	1

7.3.2 Frontal areas dysfunction

The second phase of this investigation was to analyse whether children born with LBW performed less well on tests that measure the areas supplied by the meso-cortical dopamine branch (frontal areas: executive functions or behavioural planning), than children with NBW, as well as to determine differences in scores between the genders, with reference to the ToL and WCS Tests.

The results of the tests are reflected in sections 7.3.2.1 and 7.3.2.2.

7.3.2.1 ToL Test

Table 7.9 illustrates the descriptive statistics for the results of the ToL Test according to the gender and birth weight groups.

Table 7.9 ToL Test – Descriptive statistics for LBW and control groups

Groups	N	ToL Means
LBW boys	21	23.33 ± 3.28
LBW girls	34	23.00 ± 3.99
NBW boys	21	24.90 ± 3.85
NBW girls	34	23.88 ± 3.89
All groups	110	23.70 ± 3.82

Figure 7.4 illustrates the plot of means for gender groups, for scores on the ToL Test.

Figure 7.4 Tower of London (ToL)

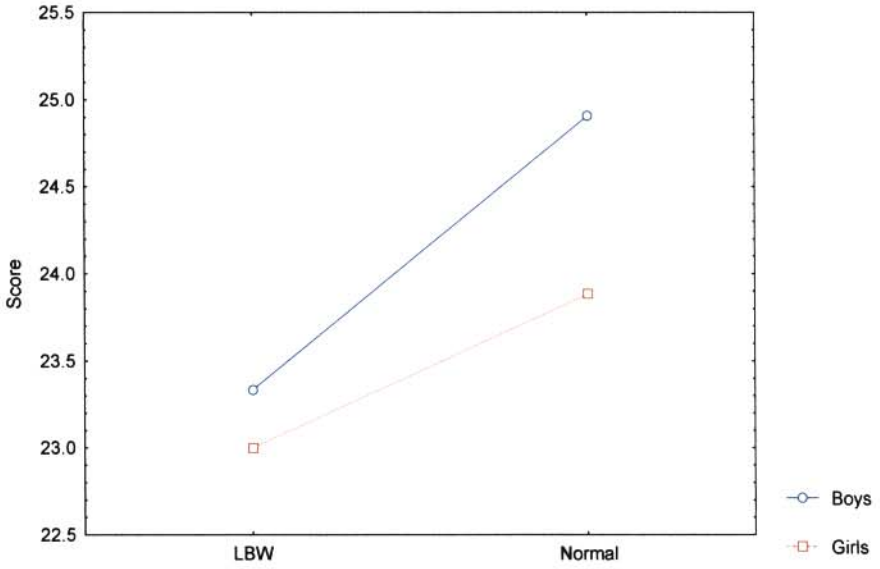


Table 7.10 illustrates the results of the ANOVA for the ToL Test for the birth weight and gender groups

Table 7.10 ToL Test – ANOVA results for LBW and control groups

Df	F	*P*
3, 106	1.18	0.32

The ANOVA indicated no significant differences between the LBW and control groups on the ToL Test.

7.3.2.2 WCST

Table 7.11 reflects the results on the WCST, for the LBW and control groups.

Table 7.11 Results of the WCST – LBW and control groups

Group	N	Categories achieved Means	Perseverative errors Means	Non-pers. errors Means
LBW boys	21	2.29 ± 1.85	30.29 ± 30.34	37.95 ± 24.40
LBW girls	34	2.53 ± 1.78	25.59 ± 24.33	41.29 ± 21.05
NBW boys	21	3.00 ± 2.14	18.48 ± 21.38	41.29 ± 21.98
NBW girls	34	3.68 ± 1.92	16.79 ± 12.58	37.12 ± 13.27
All groups	110	2.93 ± 1.96	22.41 ± 22.53	39.36 ± 19.73

Figure 7.5 illustrates the plot of means for categories shifted on the WCST for the LBW and control groups.

Fig. 7.5 Wisconsin Card Sorting Test

Categories shifted

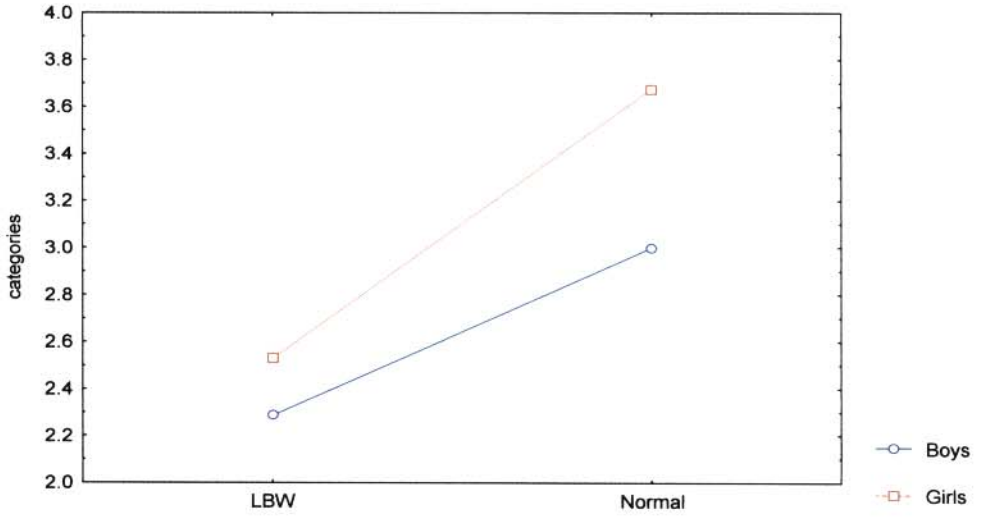


Figure 7.6 illustrates the plot of means for the number of perseverative and non-perseverative errors on the WCST, for the LBW and control groups.

Figure 7.6 Wisconsin Card Sorting Test

Perseverative and non-perseverative errors

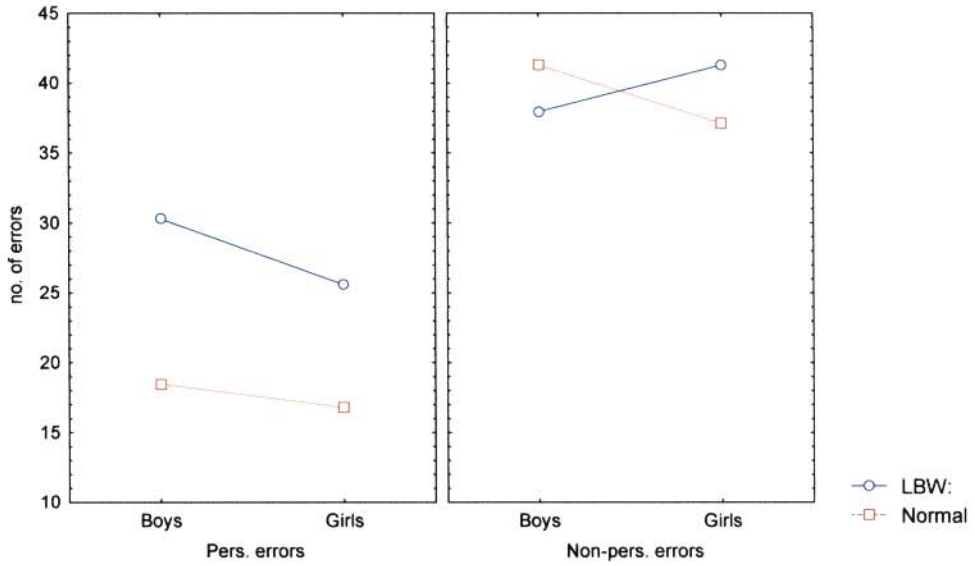


Table 7.12 gives an overview of the results of the ANOVA for the WCST, for LBW and control groups

Table 7.12 WCST – ANOVA results for LBW and control groups

	df	**F**	***P***
Categories	3, 106	3.04	0.03*
Pers. errors	3, 106	2.06	0.11
Non-pers. errors	3, 106	0.35	0.79

* $p \leq 0.05$

Table 7.13 indicates the results of the post-hoc (Scheffé) test performed on the categories achieved of the WCST, the LBW and control groups.

Table 7.13 WCST – Post-hoc (Scheffé) results for the number of categories achieved for the LBW and control groups

	WCST: Categories achieved
Boys	0.23
Girls	0.01*

* $p \leq 0.05$

The ANOVA showed significant differences between the LBW and NBW groups on categories achieved of the WCST, but not for perseverative and non-perseverative errors. The post-hoc (Scheffé) indicated differences in categories achieved for girls, but not for boys.

7.3.3 Motor dysfunctioning

The third phase of this investigation was to analyse whether children born with LBW performed more poorly on tests that measure the areas supplied by nigro-striatal dopamine branch (motor functions) than children with NBW, as well as to determine differences in scores between the genders, with reference to the following test:

7.3.3.1 Maze Coordination Test

Table 7.14 gives an overview of the descriptive statistics for the number of touches with the dominant hand and non-dominant hand on the Maze Coordination Test, for gender groups.

Table 7.14 Results of the Maze Coordination Test

Group	N	Dominant hand Means	Non-dom. hand Means
LBW boys	21	31.86 ± 11.88	44.57 ± 16.58
LBW girls	34	33.41 ± 13.55	51.47 ± 16.09
NBW boys	21	21.86 ± 9.96	30.71 ± 12.01
NBW girls	34	18.62 ± 8.53	35.68 ± 11.76
All groups	110	26.34 ± 12.87	41.31 ± 16.22

Figure 7.7 illustrates the plot of means for the number of touches on the Maze Coordination Test, for LBW and control groups.

Figure 7.7 Maze Coordination Test

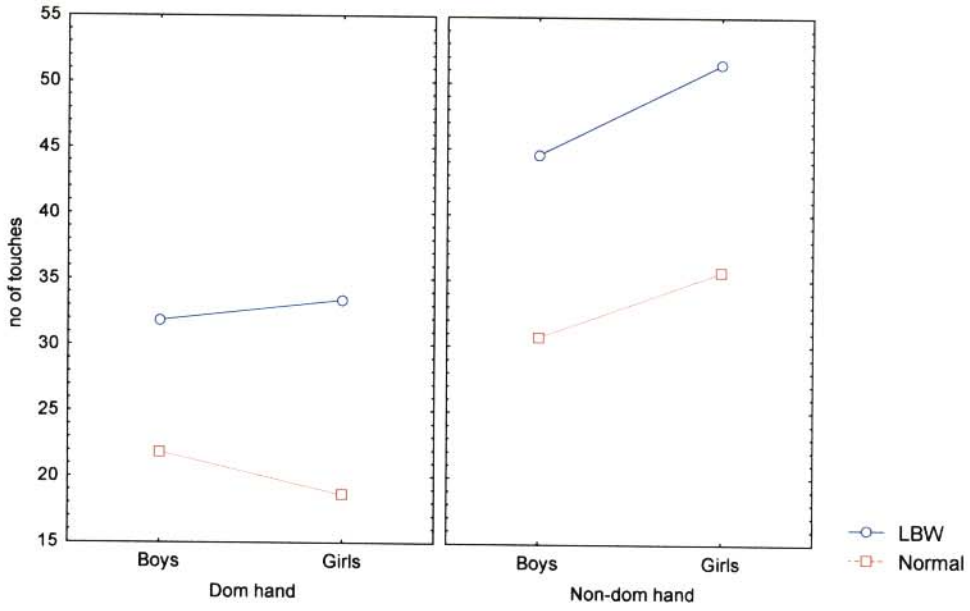


Table 7.15 illustrates the results of the ANOVA for the number of touches on the Maze Coordination Test, for the LBW and control groups.

Table 7.15 Maze Coordination Test – ANOVA results

	df	**F**	***P***
Dom. hand	3, 106	12.75	0.00*
Non-dom. hand	3, 106	11.79	0.00*

* $p \leq 0.05$

The results of the post-hoc (Scheffé) test, performed on the Maze Coordination Test, is illustrated in Table 7.16

Table 7.16 Maze Coordination Test – Post-hoc (Scheffé) results for touches for LBW and control groups

	Dominant hand	Non-dom. hand
Boys	0.04*	0.02*
Girls	0.00*	0.00*

* $p \leq 0.05$

The ANOVA showed significant differences between the birth weight groups on both dominant hand and non-dominant hand results of the Maze Coordination Test. Post-hoc (Scheffé) results indicated significant differences on both hands for boys and girls.

Table 7.17 provides the result of the correlations (Pearson-r) between birth weight and the specific test performances.

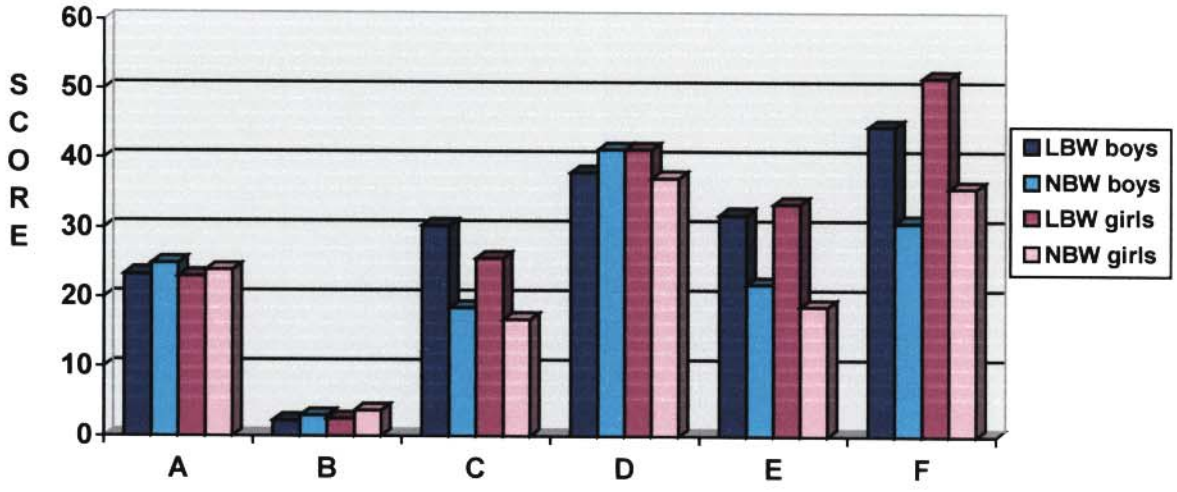
Table 7.17 Test Performances – Correlations between the test scores and birth weight (Pearson-r)

Test	Correlation	Discrimination value
ToL	0.13	None
WCST: Categories	0.22*	Weak
WCST: Pers. errors	-0.25*	Weak
WCST: Non-pers. errors	-0.2	None
Maze: dom. hand	-0.48*	Moderate
Maze: non-dom. hand	-0.50*	Moderate

* $p \leq 0.05$

Figure 7.8 summarises the mean scores on tests for the LBW and control groups.

Figure 7.8 Mean scores for the neuropsychological tests



A: ToL Test
B: WCST: Categories
C: WCST: Pers. errors
D: WCST: Non-pers. errors
E: Maze: Dom. hand
F: Maze: Non-dom. hand

7.4 Hypotheses testing

Based on the presented research results, the following can be concluded regarding the research hypotheses:

Research hypothesis 1 can be partially accepted. Children with LBW had significantly more inattentive symptoms on the DBD rating scale, but the hyperactive/impulsive scale did not discriminate clearly. There were differences in the performances of the gender groups on the inattentive subscale, while the hyperactive/impulsive subscale again did not discriminate clearly.

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

1.1 Rejected, as there were significant differences between LBW children and a control group with NBW on the inattentive symptoms of the DBD rating scale ($p=0.00$). There were significant differences between the boys ($p=0.02$) and those between the girls tended towards significance ($p=0.09$).

1.2 Accepted, as there were no significant differences between LBW children and a control group with NBW on the hyperactive/impulsive symptoms of the DBD rating scale. There was, however, a strong tendency towards significance ($p=0.07$). No significant gender differences were observed.

1.3 Accepted, as there were no significant differences between LBW children and a control group with NBW on the ODD symptoms of the DBD rating scale. There was, however, a tendency towards significance ($p=0.09$). No significant gender differences were observed.

1.4 Accepted, as there were no significant differences between LBW children and a control group with NBW on the CD symptoms of the DBD rating scale ($p=0.18$). No significant gender differences were observed.

Research hypothesis 2 is partially accepted, as there were significant differences between the scores of the LBW and the NBW group on one of the two tests measuring the areas supplied by the meso-cortical dopamine branch (prefrontal areas).

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

2.1 Accepted, as there were no significant differences between children with LBW and the NBW control group on the ToL Test ($p=0.32$). There were no significant differences in performance between the genders.

2.2 Accepted, as there were no significant differences between children with LBW and the NBW control group on the perseverative error scores of the WCST ($p=0.11$). There were no significant differences in performance between the genders.

2.3 Accepted, as there were no significant differences between children with LBW and the NBW control group on the non-perseverative error scores of the WCST ($p=0.79$). There were no significant differences in performance between the genders.

2.4 Rejected, as there were significant differences between children with LBW and the NBW control group and fewer categories achieved on the WCST ($p=0.03$). There were significant differences in performance between the girls of the two groups ($p=0.01$), but not between the boys ($p=0.23$).

Research hypothesis 3 is fully accepted, as there were significant differences between the scores of the LBW and the NBW group on the test that measures the area supplied by the nigro-striatal dopamine branch (motor functions).

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

3.1 Rejected, as there were significant differences between children with LBW and the NBW control group and more touches with their dominant hand on the Maze Coordination Test ($p=0.00$).

3.2 Rejected, as there were significant differences between children with LBW and the NBW control group and more touches with their non-dominant hand on the Maze Coordination Test ($p=0.00$).

Chapter 8

DISCUSSION OF RESULTS

8.1 Introduction

This study proposed to determine the number of ADHD symptoms among LBW children in comparison with NBW children. A further aim was to establish whether LBW children perform more poorly on ADHD-sensitive tests than NBW children.

In accordance with expectations, the LBW group performed more poorly than the NBW group on all measures, though significant differences were not always observed (Castellanos, 1997; Reeve & Schandler, 2001). The only exception to this finding was the non-perseverative category of the WCST, where the LBW boys performed insignificantly better than the NBW boys (see Table 8.1).

Table 8.1 Summary of results

Measure	ANOVA	Post-hoc	
		boys	girls
DBD			
Inattentive	0.00*	0.02*	0.09
Hyperactive/impulsive	0.07		
ODD	0.09		
CD	0.18		
Frontal tests			
ToL	0.32		
WCST: Categories	0.03*	0.23	0.01*
Perseverative errors	0.11		
Non-perseverative errors	0.79		
Motor test			
Maze Coordination Test: dom hand	0.00*	0.04*	0.00*
n-dom. hand	0.00*	0.02*	0.00*

*$p \leq 0.05$

The significant data results can be summarised briefly as follows:

1. The LBW group had significantly ($p=0.00$) more inattentive symptoms on the DBD rating scale, where the differences can be ascribed to LBW boys who had a higher score ($p=0.02$) than their NBW counterparts;
2. The LBW children achieved significantly ($p=0.03$) fewer categories on the WCST than the NBW children, where the LBW girls achieved fewer categories than the NBW girls ($p=0.01$); and
3. The Maze Coordination Test differentiated significantly on the number of touches with the dominant hand ($p=0.00$) and non-

dominant hand ($p=0.00$). The LBW boys performed significantly worse than the NBW boys with the dominant hand ($p=0.04$) and non-dominant hand ($p=0.02$). LBW girls had significantly more touches than their NBW counterparts with the dominant hand ($p=0.00$) and non-dominant hand ($p=0.00$).

Insignificant data results were observed in the following tests:

1. The ToL Test, where the differences in performance between the birth weight and gender groups were statistically insignificant ($p=0.32$);
2. Perseverative and non-perseverative errors on the WCST, where the difference between the birth weight and gender groups were insignificant ($p=0.11$ and $p=0.79$ for perseverative and non-perseverative errors respectively);
3. Hyperactive/impulsive subscale on the DBD rating scale, where differences between birth weight and gender groups were statistically insignificant ($p=0.07$); and
4. ODD and CD subscales on the DBD rating scale, where there were no significant differences ($p=0.09$ and $p=0.18$ for ODD and CD respectively). However, there was a tendency towards significance for the ODD scale.

8.2 Discussion of results

This study demonstrated a trend among LBW boys and girls towards poorer performances than their NBW counterparts on all ADHD-sensitive instruments, referring to the DBD rating scale, ToL Test, WCST and Maze Coordination Test.

The LBW children in this study tended to be more vulnerable to hyperactive/impulsive and inattentive symptoms, as reflected in increased scores on the hyperactive/impulsive and inattentive subscales of the DBD rating scale. The LBW boys generally had a greater tendency towards increased symptoms on all DBD rating scales, except for CD. In spite of the increased prevalence of ADHD symptoms, insignificantly more LBW children could be diagnosed with ADHD. This finding supports the studies of Sommerfelt et al. (2003) and Stathis et al. (1999), who found no difference in the prevalence of ADHD between the LBW and the NBW groups. Various other studies (Bhutta et al., 2002; Breslau & Chilcoat, 2000; Breslau et al., 1996a; Harrison, 1996; Mick et al., 2002; Saigal et al., 2001; Szatmari et al., 1990; Ulvund et al., 2001; Winders & Burns, 2001) did however identify an increased prevalence of ADHD among LBW children. Though ADHD symptoms among LBW children were not always severe enough to reach a full diagnosis of ADHD, the increased incidence of hyperactive/impulsive and inattentive symptoms can still be severe enough to debilitate their functioning, development and management. This finding is in

keeping with the suggestion of Harrison (1996), namely to refer to a behaviour continuum instead of an absolute diagnosis.

Significant differences on the inattentive subscale were demonstrated in this study, which support the findings of Bregman (1998), Breslau and Chilcoat (2000), as well as Sohl and Moore (1998). These differences were ascribed to meaningful differences between the LBW and the NBW boys. This is inconsistent with various other studies, which found that girls have the tendency to have more symptoms of the inattentive subtype (Cantwell, 1996; Swanson, 2003) and internalising problems (Abikoff et al., 2002), while boys tend to have more hyperactive symptoms (Hunt el al., 2002) and externalising behaviour (Abikoff et al., 2002; Crijnen et al., 1997). The finding of increased inattention among boys is however consistent with studies of Heptinstall and Taylor (2002), which state that girls of all ages have fewer attention problems and less hyperactivity than same-age boys on parent and teacher rating scales. Chapieski and Evankovich (1997) also found a higher level of internalising behaviour problems among nine-year-old VLBW males.

More inattentive symptoms among boys could be explained by the possibility that ADHD symptoms among boys remit later in life. As already stated, girls seem to be more mature than boys at all developmental stages. It is known that boys develop more slowly cognitively and physically than girls

(Heptinstall & Taylor, 2002), which relates to the theoretical perspective of ADHD as a neurodevelopmental disorder (Heptinstall & Taylor, 2002).

In this study tests were used that measure frontal lobe functioning. The frontal lobes are not fully developed until adulthood, which could be another explanation of more inattention among boys. The cited inconsistent finding, namely inattention among LBW boys, may possibly relate to the finding that attention deficit in LBW infants may have a different aetiology or development from that of NBW babies (Botting et al., 1997).

This finding of an increased prevalence of inattention among boys is in contrast to that of Elgen et al. (2003), Sommerfelt et al. (1996) and Stathis et al. (1999), who report no specific identifiable attentional dyfunctions in the LBW group. Chapieski and Evankovich (1997) also report inconsistencies between studies investigating the relation between LBW, ADHD and gender. Inattention among LBW gender groups, specifically boys, is an area that seems to need further investigation.

In this study the LBW boys and girls tended to have higher scores on the hyperactive/impulsive subscale of the DBD rating scale. These differences were however insignificant, although they tended towards significance, unlike other studies, which found an increased incidence of hyperactivity among LBW children (Bregman, 1998; Sohl & Moore, 1998). This finding correlates with

studies of York and DeVoe (2002), namely that attention problems are more significant in premature infants with ADHD and hyperactivity is less significant.

There was a trend toward increased scores in the LBW group on the ODD subscale of the DBD rating scale, which did not reach statistical significance. This does not support the findings of Breslau et al. (1996a), who state that ODD is the most common comorbid disorder in both LBW and NBW children. Even more insignificant findings regarding CD were observed. The findings for both ODD and CD correlate with studies of Botting et al. (1997), namely that LBW children with hyperactivity do not develop antisocial behaviour or CD as frequently as matched peers.

There was a statistically significant trend toward lower performance for the LBW group on the WCST and Maze Coordination Test, which appear to be useful measures of ADHD-related symptoms. The ToL Test did not differentiate clearly between the groups, which might imply that it is not such an effective measure of ADHD symptoms as the previously mentioned tests.

Significantly and insignificantly poorer performance on the WCST and ToL Test respectively indicate problems with executive functions in the LBW cohort (Blondis et al., 2000; Gregory, 2000; Monchi et al., 2001; Russell, 1994). These significant differences in the WCST and the tendency to perform more poorly on the ToL Test, may indicate the presence of a dysfunctioning meso-cortical dopamine branch, that produces possible frontal lobe dysfunctioning in LBW

children, which supports the findings of other authors (Heaton & Pendleton, 1981; Lezak et al., 2004).

The LBW girls performed significantly more poorly in categories of the WCST, which is difficult to explain.

This study found that LBW children of both genders were significantly more likely to experience motor control problems in comparison to their NBW counterparts, as indicated in the Maze Coordination Test results. Studies by Papageorgiou and Bardin (1999) and Breslau et al. (2001), as well as those of Sohl and Moore (1998) also demonstrated significant deficits in motor control for those born with LBW. Impaired motor control among the LBW cohort may indicate a dysfunctioning nigro-striatal dopamine branch (Johansen et al., 2002; Lezak et al., 2004).

The consistent pattern of differences in scores between the LBW and NBW groups indicate that children with LBW tend to have more ADHD-related symptoms than their NBW counterparts. It may be possible that developmental delays in LBW children do not completely resolve, although Campbell (2001b) indicates that they catch up by 12 to 18 months.

The increased occurrence of ADHD-related symptoms in the LBW cohort may be a consequence of neurological events that are related to being born with LBW, which is also postulated by Botting et al. (1997). Other studies have also

commented on the possible neurological aetiology of ADHD (Botting et al., 1997; Hunt et al., 2001; Johansen et al., 2002; Mercugliano, 1999). The previously mentioned possible dysfunctioning meso-cortical dopamine branch (as tested by the WCST and ToL Test) and nigro-striatal dopamine branch (as tested by the Maze Coordination Test) affirms the finding that LBW children have the biological predisposition to developmental difficulties, such as ADHD symptoms (Chapieski & Evankovich, 1997; Harrison, 1996; Whitaker et al., 1997).

8.3 Clinical implications of the study

The mentioned findings of increased ADHD symptoms and poorer performance on ADHD-sensitive tests among LBW children indicate that they are at increased risk of long-term impairments such as inattention, poor executive functions and problems with motor abilities. Intensive follow-up and management programmes have to be implemented on a continuous basis in order to identify, diagnose, treat and monitor such developmental delays. The focus of these programmes should be on preventing possible pathological symptoms from developing fully, especially in the light of ADHD symptoms. Awareness and treatment of psychiatric and neurological outcomes in LBW children among professionals, parents and the children themselves are of increased importance in combating long-term impairments (Botting et al., 1997). Follow-up interventions and guidance are crucial owing to the increasing number of LBW children, even those with VLBW and ELBW, and its financial implications for society.

8.4 Limitations of the study

No refined method was used to determine SES. Children were categorised into three groups, namely low, middle and high SES. The categorisation was only an estimation of SES on the grounds of basic information.

The assessment in this study did not include any medical examination to rule out unknown medical conditions that can mimic ADHD symptoms. The biographical data questionnaire did however explore aspects of medical history.

ODD and CD were the only comorbid disorders that were taken into account. This study did not focus on other psychiatric, developmental and environmental conditions that can either mimic ADHD symptoms or coexist with ADHD (Edwards et al., 1995).

8.5 Possibilities for further research

The inconsistent findings regarding increased inattentive symptoms among boys seem to be an area for further investigation, as well as the tendency of LBW boys to have increased symptoms on the DBD rating scale. Chapieski and Evankovich (1997) also report inconsistencies between studies investigating the relation between LBW, ADHD and gender. It is hoped that investigation of these inconsistencies will clarify the aetiology and treatment of ADHD symptoms among LBW gender groups.

Bhutta et al. (2002) found that physiological instability and exposure to early adverse experiences may have a persistent impact on brain development leading to cognitive and behavioural outcomes. Thus, the increased prevalence of ADHD symptoms in this study among LBW children may be a direct or indirect consequence of other variables. It is possible that variables such as pre-, peri- or postnatal complications or environmental factors could have enhanced ADHD symptoms in the children included in this study.

Breslau et al. (1996b) state that brain injury as well as abnormal brain development during prenatal and perinatal stages have been suggested as the mechanisms in subtle visual-motor and visual-perceptual deficits, as well as hyperactivity in preterm children. However, Abernethy et al. (2002) postulate that attention deficit in children born with VLBW does not correlate with conventional markers of perinatal brain injury, but may be related to global brain growth and the development of key structures (such as the caudate nuclei and hippocampal formations). Thus, the relation between perinatal problems and ADHD symptoms in LBW children is still uncertain.

Various questions on pre-, peri- and postnatal complications were included in the biographical data questionnaire, but were not used for statistical analysis in this study. It is hoped that the relation between the prevalence of ADHD symptoms and pre-, peri- and postnatal complications will prove whether ADHD

symptoms are caused by LBW as such or rather by pre-, peri- and/or postnatal problems, or by an interplay of both.

Research on the role of environmental factors in the expression of ADHD symptoms among LBW children seems to be inconsistent. A recent study of premature children found that family factors were stronger predictors of school performance than were perinatal complications (Bhutta et al., 2002). The impact of environmental influences was not investigated in this study, though various aspects were included in the biographical data questionnaire. It would be enlightening to explore to what extent environmental influences (such as low parental education, single mother status, parental SES, family composition, foetal exposure to toxins) predict behavioural and school problems (Addiction Organisation, 2002; Breslau et al., 2001; Greenbaum & Auerbach, 1992; Gross et al., 2001; Jobe, 2001; Mercugliano, 1999).

Impaired performance of the LBW group with respect to motor abilities and executive functioning may not be attributed completely to ADHD, but may also partly be caused by learning disabilities. A recent study has demonstrated that children with ADHD and reading disability were impaired in various domains, including executive functioning. Thus, future research should also focus on learning disabilities in addition to ADHD, in order to ascertain whether neuro-cognitive differences between groups are accounted for by ADHD, learning disabilities or both (Kalff et al., 2002).

It would be interesting to follow the children included in this cohort into adolescence or late adolescence. This may reveal whether ADHD symptoms during primary school years are precursors for more severe symptoms of disorders or only transient features in the development of LBW children, similar to those found in studies performed by Elgen et al. (2002) and Botting et al. (1997).

8.6 Concluding remarks

This study shows that LBW is associated with increased ADHD symptoms, as well as poorer performance on ADHD-sensitive tests, in comparison to the NBW counterparts of LBW children. This was particularly significant in the domains of inattention, executive functions and particularly motor abilities. These impairments most probably affect school functioning and other significant areas of life.

The identification of inconsistent findings in this study, as well as uncertainty regarding the nature of impaired attention among LBW children, stimulates the search to resolve questions on the nature and aetiology of ADHD symptoms, as well as gender differences.

It is hoped that continuous research on the nature of the relation between LBW and developmental difficulties, such as ADHD, will optimise the treatment

and outcomes of these children, given the context that the number of LBW children is increasing.

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Appendix A
Request to school to participate in research

UNIVERSITY OF THE NORTH
SCHOOL OF SOCIAL SCIENCES
PSYCHOLOGY

Private Bag X1106
Sovenga
0727

e-mail: meyera@unorth.ac.za

15 August 2003

Dear Sir or Madam,

RESEARCH PROJECT: ATTENTION-DEFICIT/HYPERACTIVITY IN THE LIMPOPO PROVINCE

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

The child is usually disorganised, has problems with planning his/her activities and may be very forgetful. These are severe problems with sustained attention, especially in the classroom situation. The child also has problems with sitting still, is overactive and fidgets. Problems with gross and fine motor co-ordination occur frequently.

The cause of ADHD is not known yet, but research suggests a genetic origin. Pollutants and poor nutrition may also play a role. It is not caused by failure to discipline or control the child. Research has shown that children with low birth weight are at risk of developing the disorder, as the disorder is four times more prevalent in these children.

ADHD children not diagnosed and treated at an early age are at risk of future delinquent behaviour, psychiatric problems and substance abuse. The financial cost for society will be considerable. The families of these children experience undue stress and ADHD has a severe impact on academic activities at schools.

In order to identify children at risk of ADHD at an early age, it was decided to do research on children with low birth weight whether they are at risk of developing ADHD or not.

The Department of Physiology, University of Oslo, Norway, has therefore developed a culture-free non-verbal test sensitive to impulsiveness, inattention and motor activity, the three major symptoms of ADHD. Together with tests for planning deficiencies and gross and fine motor co-ordination, we are hoping to have established a test battery, which measures the functions of the brain areas that have been implicated to play a role in the disorder. At the same time we will try to establish the role of birth weight in the disorder. This is an approved departmental research project of the University of the North in collaboration with the University of Oslo, Norway.

Method:

If you should agree to participate in this project, the research team will visit your school and screen the children for low birth weight. A control group matched for age and gender will also be selected. The following questionnaires/tests will be administered:

Biographical data questionnaire
Disruptive behaviour disorder rating scale
Tests for fine motor coordination
Tests for planning abilities
Tests for over-activity, impulsiveness and impaired sustained attention

The data will be used for statistical analysis only and in no circumstances will the identity of the child and the school be revealed. The children selected will not necessarily be suffering from ADHD.

Thank you for your co-operation.

.....
Anelia Haycock BAHons
Researcher

.....
Prof. Terje Sagvolden PhD
Research collaboration UiO

.....
Prof. Anneke Meyer PhD
Project leader UNIN

Appendix B
Letter to parents: Identification of LBW children

PO Box 11022
BENDOR
0699

2003-08-26

Dear parent / guardian

RESEARCH PROJECT: RELATIONSHIP BETWEEN LOW BIRTH WEIGHT AND PSYCHOLOGICAL FUNCTIONING

The Discipline of Psychology of the University of the North is investigating the relationship between low birth weight and possible psychological problems.

Current primary school children, born with a birth weight below 2.5 kg, are needed for this research project. Confidentiality is guaranteed.

Free psychological services will be provided in relevant cases.

We would appreciate it if you could complete the slip and return it to the class teacher before 12 September 2003.

Thank you for your collaboration.

Anelia Haycock
Researcher
Cell no: 082 489 9498

Name of child: .....

Grade: .....

Birth weight: .....

Name of school: .....

Name of parent: .....

Telephone number: .....

Appendix D

Letter to parents of LBW children: Request for documentation

PO Box 71022
BENDOR
0950

2003-09-17

Dear parent / guardian

RESEARCH PROJECT: RELATIONSHIP BETWEEN LOW BIRTH WEIGHT AND PSYCHOLOGICAL FUNCTIONING

Thank you for responding to the previous letter. As previously noted, the Discipline of Psychology of the University of the North is investigating the relationship between low birth weight and possible psychological problems.

Current primary school children, born with a birth weight below 2,5 kg, are needed for this research project.

If you allow your child to be part of this research we would appreciate it if you could complete the following documents and return these to the class teacher on the 1st of October 2003:

- Consent form
- Biographic information
- DBD questionnaire
- Please send a copy of your child's clinic sheet to confirm his/her birth weight, if possible.

Four different tests will be administered in respect of each selected child at his/her school. All information will be treated confidentially.

Free psychological services will be provided in relevant cases.

Thank you for your collaboration. Your involvement in this research project will contribute to more effective psychological treatment for children.

.....

Amelia Haycock
Researcher
Cell no: 082 189 0498

UNIVERSITY OF THE NORTH
ETHICS COMMITTEE

PROJECT TITLE: Attention-Deficit/Hyperactivity Disorder and Low Birth Weight

PROJECT LEADER: Prof. Anneke Meyer

CONSENT FORM

I, _____ hereby voluntarily consent to my child participating in the following project: **Attention-Deficit/Hyperactivity Disorder and Low Birth Weight.**

I realise that:

1. The study deals with the relationship between attention-deficit/hyperactivity and low birth weight;

2. The procedure or treatment envisaged may hold some risk for me that cannot be foreseen at this stage;

3. The Ethics Committee has approved that individuals may be approached to participate in the study;

4. The experimental protocol, i.e., the extent, aim and methods of the research, has been explained to me;

5. The protocol sets out the risks that can be reasonably expected as well as possible discomfort for persons participating in the research, an explanation of the anticipated advantages for myself or others that are reasonably expected from the research and alternative procedures that may be to my advantage;

6. I will be informed of any new information that may become available during the research that may influence my willingness to continue my participation;

7. Access to the records that pertain to my participation in the study will be restricted to persons directly involved in the research;

8. Any questions that I may have regarding the research, or related matters, will be answered by the researchers;

9. If I have any questions about, or problems with the study, or experience any undesirable effects, I may contact a member of the research team;

10. Participation in this research is voluntary and I can withdraw my participation at any stage;

11. If any medical or psychological problem is identified at any stage during the research, or when my child is vetted for participation, such condition will be discussed with me in confidence by a qualified person and/or my child will be referred to a doctor or psychologist;

12. I indemnify the University of the North and all persons involved with the above project from any liability that may arise from my participation in the above project or that may be related to it, for whatever reasons, including negligence on the part of the mentioned persons.

.....	.....
SIGNATURE OF RESEARCHED PERSON	SIGNATURE OF WITNESS
.....	.....
SIGNATURE OF PERSON THAT INFORMED THE RESEARCHED PERSON	SIGNATURE OF PARENT/GUARDIAN

SIGNED AT _____ this _____ day of _____ 2003

EARLY DEVELOPMENTAL MILESTONES

A. At what age did your child first accomplish the following:

1. Sitting without help _____
2. Crawling _____
3. Walking along, without assistance _____
4. Using single words (e.g. "mama", "dada", "ball", etc.) _____
5. Putting two or more words together (e.g. "mama up") _____
6. Bowel training, day and night _____
7. Bladder training, day and night _____

HEALTH HISTORY

A. Date of child's last physical examination: _____

B. Has your child had the following at any time:

1. Asthma	Never	Past	Present
2. Allergies	Never	Past	Present
3. Diabetes, arthritis, or other chronic illness	Never	Past	Present
4. Epilepsy or seizures	Never	Past	Present
5. Febrile seizures	Never	Past	Present
6. Heart or blood pressure problems	Never	Past	Present
7. High fever (over 38°C)	Never	Past	Present
8. Head injury with loss of consciousness	Never	Past	Present
9. Lead poisoning	Never	Past	Present
10. Lengthy hospitalization	Never	Past	Present
11. Speech or language problems	Never	Past	Present
12. Chronic ear infections	Never	Past	Present
13. Hearing difficulties	Never	Past	Present
14. Eye or vision problems	Never	Past	Present
15. Poor motor/handwriting problems	Never	Past	Present
16. Cross motor difficulties, clumsiness	Never	Past	Present
17. Appetite problems (overeating/under-eating)	Never	Past	Present
18. Sleep problems (falling asleep, staying asleep)	Never	Past	Present
19. Soiling problems	Never	Past	Present
20. Wetting problems	Never	Past	Present
21. Other health difficulties – please describe _____			

20. Other problems – please describe _____

1. Did any of the following conditions affect your child during delivery or within the first few days after birth?

1. Injured during delivery	No	Yes
2. Cardiopulmonary (heart) distress during delivery	No	Yes
3. Delivered with cord around neck	No	Yes
4. Had trouble breathing following delivery	No	Yes
5. Needed oxygen	No	Yes
6. Turned blue	No	Yes
7. Was jaundiced, turned yellow	No	Yes
8. Had an infection	No	Yes
9. Had seizures	No	Yes
10. Was given medication	No	Yes
11. Born with a congenital defect	No	Yes
12. Was in hospital for more than seven days	No	Yes

INFANT REACTIVITY AND TEMPERAMENT

A. During the first 12 months, was your child:

1. Difficult to feed	No	Yes
2. Difficult to get to sleep	No	Yes
3. Colicky	No	Yes
4. Difficult to put on a schedule	No	Yes
5. Alert	No	Yes
6. Cheerful	No	Yes
7. Affectionate	No	Yes
8. Sociable	No	Yes
9. Easy to comfort	No	Yes
10. Difficult to keep busy	No	Yes
11. Overactive, in constant motion	No	Yes
12. Very stubborn, challenging	No	Yes

Appendix F

Biographical data questionnaire

BIOGRAPHICAL DATA INFORMATION

Child's No: .....

CHILD AND FAMILY INFORMATION

Child's name and surname: .....

Date of birth: ..... Age: .....

Sex: M / F Language: .....

Height: ..... Weight: .....

School: ..... Grade: .....

Is child in special education? Y / N If so, what type? .....

Computer experience: Y / N

Dominant hand: Left / Right Dominant foot: Left / Right

Medication (if any): .....

Father's name: ..... Age: .....

Years of education: ..... Occupation: .....

Contact number: .....

Mother's name: ..... Age: .....

Years of education: ..... Occupation: .....

Contact number: .....

Address: .....

Home phone: .....

Please list all the other children in the family:

Name:	Age:	Grade:
_____	_____	_____
_____	_____	_____
_____	_____	_____

	YES	NO
Does the family have a TV?		
A car?		
Electricity in the house?		
Water in the house?		
Is the child adopted?		
Are parents married?		
Separated?		
Divorced?		

TEACHER / PARENT DBD RATING SCALE

ID: _____

Child's name: _____ **Form completed by:** _____

Sex: M / F **Age:** _____ **School:** _____

Grade: _____ **Date Completed:** _____

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

TICK THE COLUMN THAT BEST DESCRIBES THIS CHILD. PLEASE PUT A QUESTION MARK NEXT TO ANY ITEM 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 favours to avoid obligations (i.e. "cons" others)				
5. Often initiates physical fights				
6. Has been physically cruel to people				
7. Often talks excessively				
8. Has stolen items of nontrivial value without confronting a victim (e.g. shoplifting, but without breaking and entering; forgery)				
9. Is often easily distracted by extraneous stimuli				
10. Often truant from school, beginning before age 13 years				
11. Often fidgets with hands or feet or squirms in seat				
12. Is often spiteful or vindictive				
13. Often blames others for his or her mistakes or misbehaviour				
14. Has deliberately destroyed others' property (other than by fire setting)				
15. Often actively defies or refuses to comply with adults' request or rules				
16. Often does not seem to listen when spoken to directly				
17. Often blurts out answers before questions have been completed				
PLEASE ALSO COMPLETE REVERSE SIDE				

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

RESEARCH: DATA COLLECTION

Child No:_____ **Child's name:** _____

Language: _____ **Sex: M / F** **School:** _____

Tested by: _____ **Date:** _____

Dominant hand: L / R

1. Maze Coordination Test

Dominant hand

	Touches (counter)	**Time (timer)**
1st trial		
2nd trial		
Total		

Non-dominant hand

	Touches (counter)	**Time (timer)**
1st trial		
2nd trial		
Total		

2. Tower of London

Task no.	No. of moves	Child's solution	Score (0-3)
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			

Sum (max 36) _____

3. VI-VI

4. Wisconsin Card Sorting Test

CPM

Child no: _____

Child's name: _____

Name of School: _____

Date: _____ Sex: M / F

	A	AB	B
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
TOTAL			

TOTAL: _____

IQ: _____

Appendix I
Letter to principal: Notification of testing dates

PO Box 11022
BENDOR
0699

2003-10-09

..... Primary School
POLOKWANE
0699

Attention: The Principal

RESEARCH (RELATION BETWEEN LOW BIRTH WEIGHT AND PSYCHOLOGICAL PROBLEMS): DATA COLLECTION

Psychological testing of all identified children (112) from various schools will take place between 14 October and 4 November 2003. Unfortunately no specific dates can be given, owing to difficulty with estimating children's work pace.

I appreciate your assistance and support.

Yours truly,

.....

Anelia Haycock
Researcher
Cell no: 0824899498

Appendix J
Letter to parents: Final request for documentation

Dear parent/guardian

RESEARCH PROJECT: RELATIONSHIP BETWEEN LOW BIRTH WEIGHT AND PSYCHOLOGICAL FUNCTIONING

The following documentation regarding your child, ..., is still outstanding:

- Consent form
- Biographic information
- DBD questionnaire
- Please send a copy of your child's clinic chart to confirm his/her birth weight, if possible.

Please inform me if you need another copy of the above-mentioned documentation.

Thank you for your collaboration. Your involvement in this research project will contribute to more effective psychological treatment for children.

.....
Anelia Haycock
Researcher
Cell no: 082 489 9498

Appendix K
Feedback to school principals

P.O Box 1473
GROENKLOOF
0027

1 July 2004

Dear school principal

Research 2003: Relation between low birth weight and possible psychological problems

I would like to thank you for your cooperation. A reliable and valid study would not be possible without your support.

Specific aspects were measured in this study, namely:

- Fine motor skills;
- Executive functions, such as cognitive flexibility, attention shifting, sustained attention, problem-solving, planning, forming of abstract concepts, and inhibition of inappropriate responses;
- Attention;
- Hyperactivity and impulsivity;
- Conduct behaviour: Violation of the basic rights of others or important societal norms/rules (e.g. serious violations of rules, aggression to people or animals, destruction of property, deceitfulness or theft); and
- Oppositional defiant behaviour: Negativistic, hostile and defiant behaviour (e.g. often loses temper, often argues with adults, blames others for his/her misbehaviour, touchy or easily annoyed by others, spiteful or vindictive).

The study found that the low birth weight group generally performed more poorly on all the above-mentioned measures than the normal birth weight group. Generalisations

should however be made with caution. It should be noted that there were exceptions where LBW children performed above average.

Parents are to be individually informed of the performance of their child. They are referred to the following professional people at the Polokwane Provincial Hospital, should their child need therapy:

- Clinical psychologists
- Occupational therapists, who address problems such as motor skills and executive functions.

Parents are advised to consult with their child's 2003 and 2004 class teachers regarding the necessity of therapy, should they question the test results.

It was an honour to work with you in the process of enhancing the well-being of the future of our children. I will appreciate it if you can thank all your personnel on my behalf.

Please do not hesitate to contact me if you require more information.

Regards

.....

Anelia Haycock
Researcher
Cellphone number: 082 489 9498

Appendix L
Feedback to parents

P.O Box 1473
GROENKLOOF
0027

1 July 2004

Dear parent / guardian

Research 2003: Relation between low birth weight and possible psychological problems

I would like to thank you for your collaboration. A reliable and valid study would not be possible without your support.

Specific aspects were measured in this study, namely:

- Fine motor skills;
- Executive functions, such as cognitive flexibility, attention shifting, sustained attention, problem-solving, planning, forming of abstract concepts, and inhibition of inappropriate responses;
- Attention;
- Hyperactivity and impulsivity;
- Conduct behaviour: Violation of the basic rights of others of others or important societal norms/rules (e.g. serious violations of rules, aggression to people or animals, destruction of property, deceitfulness or theft); and
- Oppositional defiant behaviour: Negativistic, hostile and defiant behaviour (e.g. often loses temper, often argues with adults, blames others for his/her misbehaviour, touchy or easily annoyed by others, spiteful or vindictive).

The study found that the low birth weight group generally performed more poorly on all measures than the normal birth weight group. Generalisations should however be made

with caution. It should be noted that there were exceptions where LBW children performed above average.

If your child performed below the average of his/her group regarding the various skills, it will be indicated in the table below. If not, you can assume that your child performed in the average or above average range.

According to available tests results and the rating scale, it seems that your child experiences difficulty in the following area(s), if any:

Fine motor skills	
Executive functions	
Attention	
Hyperactivity/impulsivity	
Conduct behaviour	
Oppositional defiant behaviour	

The following professional people can be contacted at the Polokwane Provincial Hospital, should your child need therapy:

- Clinical psychologists
- Occupational therapists, who address problems with motor skills and executive functions.

The 2003 and 2004 class teachers can be consulted regarding the necessity of therapy, should you question the above-mentioned results.

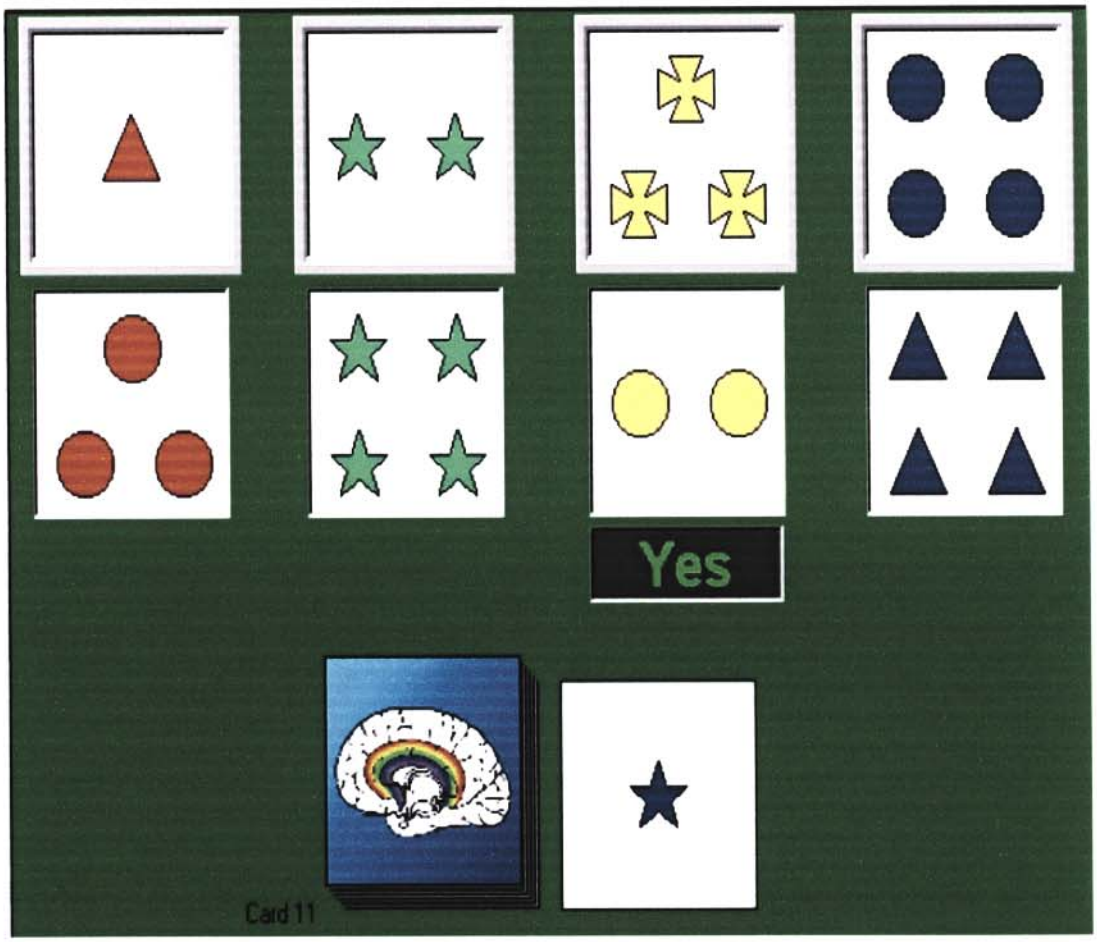
Please do not hesitate to contact me if you require more information.

Regards

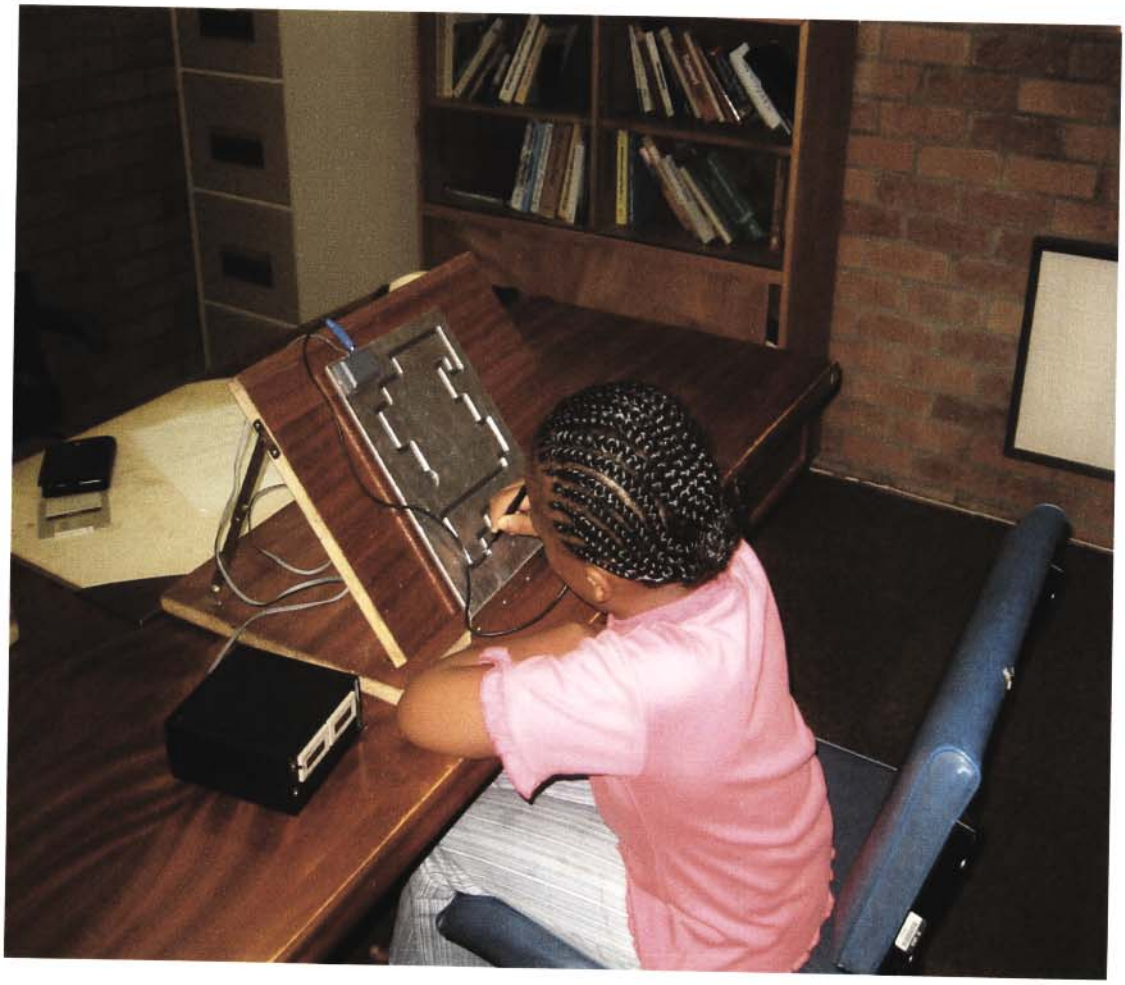
.....

Anelia Haycock
Researcher
Cellphone number: 082 489 9498

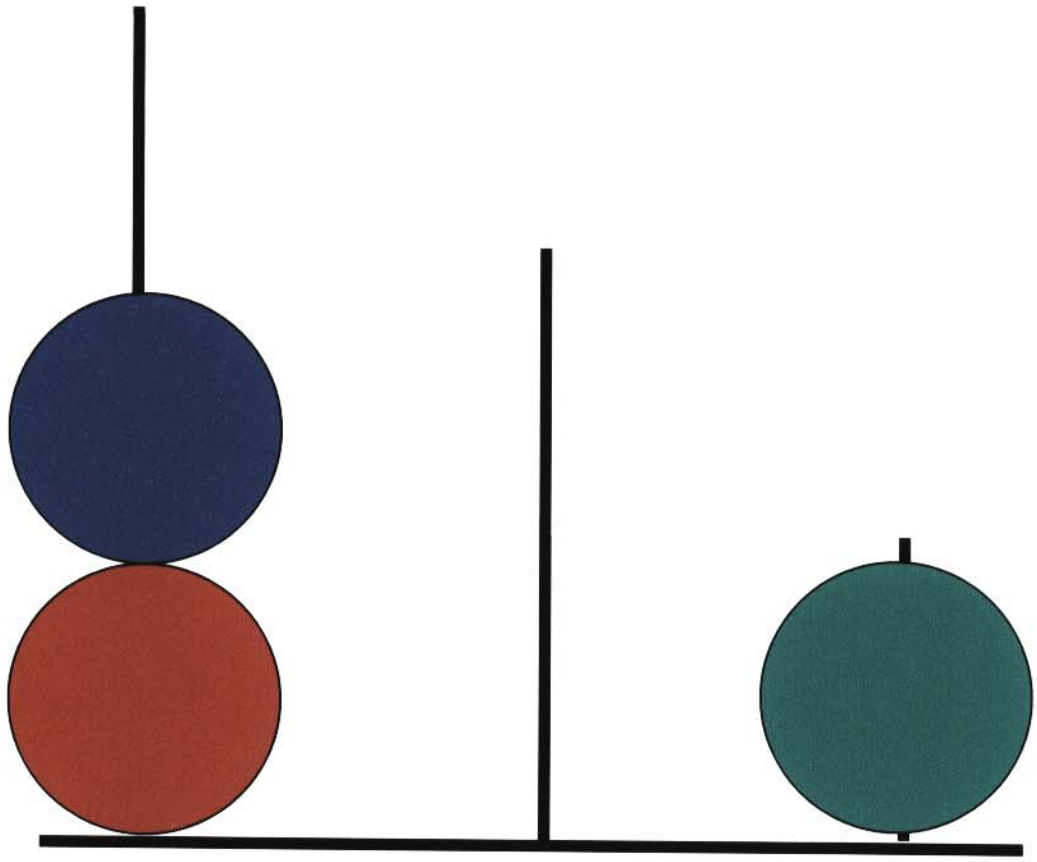
Appendix M
Wisconsin Card Sorting Test



Appendix N
Administration of the Maze Coordination Test



Appendix O
Tower of London Test



Task 7: 4 moves