

https://dspace.org/

The relationship between cognitive functioning and early childhood factors in children with speech and language impairment.

Milligan, Robyn

2012-02-28
http://hdl.handle.net/10539/11366

Downloaded from a DSpace repository. https://dspace.org/

The Relationship between Cognitive Functioning and Early Childhood Factors in Children with Speech and Language Impairment

Robyn Milligan
Department of Psychology
University of the Witwatersrand

Supervisor: Prof. Kate Cockcroft

A Research Report submitted to the Faculty of Humanities,
University of the Witwatersrand, Johannesburg, in partial fulfilment of the requirements for the degree of Master of Arts in Psychology by Coursework and Research Report.

Johannesburg, November 2010.

Declaration

I hereby declare that this research report is my own independent work, and has not been presented for any other degree at any other academic institution, or published in any form. It is submitted in partial fulfilment of the requirements for the degree of Masters of Arts in Psychology by Coursework and Research Report at the University of the Witwatersrand, Johannesburg.

_____ _____

Robyn Milligan November 2010

0604256g

Acknowledgments

This thesis would not have been possible without the help and encouragement of the following people:

The staff at inter-library loans. You really are this universities best-kept secret, and deserve esteemed recognition for your efficient and friendly service.

To the principals, staff and children at the pre-school where the research was conducted. Your welcoming hospitality and assistance in every step of this process is greatly appreciated.

To Jarrod Payne, for helping me find my way through SAS, and statistics in general.

To Nicky Israel, for all the extra, unnoticed assistance from random phone calls, and emails throughout the year. Thank you for letting me use your MA as a high benchmark for which to strive.

To my friends in MACC 2010, for their continual support, emails, mid-night sms's, good laughs, a few tears and unforgettable memories.

To Kate Cockcroft. I have so appreciated your detailed concern, guidance and support through this process, and the amount of time that you have invested in my growth. Not only have I learnt to value you as a mentor, but have learnt much from your style of research and practice. Thank you for never being too busy to explain yourself to me, and for giving me the freedom to pursue this project as I liked.

To Tracey le Roux, for lending me all your speech and language material, your brain at all hours of the night, and your open ears and warm heart through quite a busy year. I hope to one day return the favour.

To my mom, dad, nana and Lisa, for helping me whenever I needed you, be it printing, supper or a good laugh. Thank you for your enduring support and love throughout my studies.

To Craig, thank you for all you sacrificed this year in getting me through Masters, for bringing me innumerable cups of tea, editing my work, staying up with me when I was tired until all hours of the morning and being my biggest supporter in this whole process. As much as I have loved this experience, I hope that our lives will never again be as busy as this year. The kettle won't survive another anyway.

And to the Lord, the Author and Perfector of my faith, '*The heart of the discerning acquires knowledge, the ears of the wise seek it out (Wise men and women are always learning, always listening for fresh knowledge*)' Proverbs 18:15.

Abstract

It has long been the quest of developmental theorists to understand the mechanisms behind cognitive functioning and the acquisition of language. Studies have identified that there is an interdependent, dialectical relationship between cognitive development and language acquisition. The development of language, in typical populations, is therefore dependent on a baseline cognitive skill, which, once acquired, capacitates the further development of cognition. However, very little is known about this process within atypical populations, particularly those with speech and language impairment. This study examined the relationship between cognitive functioning and early predictive factors in an atypical population of pre-school children with speech and language impairment using three measures of cognitive functioning (as measured by the WPPSI (Wechsler Pre-School and Primary Intelligence Scale), the Griffiths and the JSAIS (Junior South African Individual Scale)) and a range of demographic, diagnostic and early developmental childhood factors. Results identify factors such as parental levels of education, family structure, gender and pregnancy and early childhood health as the main influences of cognitive performance. They also highlight the pervasive influence of speech and language impairment on non-verbal and processing speed abilities. The presence of genetic conditions as well as multiple diagnoses was frequently found to have significant associations with poor cognitive performance. The study also highlighted two things of unexpected interest. The first refers to the role of handedness (particularly undifferentiated handedness) in identifying cognitive difficulty which is related to hemispheric lateralisation, and its relationship to the various diagnostic groups represented within the sample. The second considers the high proportion of diagnostic co-morbidity and the common cognitive profiling patterns across diagnostic categories in abilities *outside* of the verbal range to highlight potential directions for future research. The implications of these overlaps are considered within existing research on brain laterality, hemispheric dominance and neurological immaturity.

Keywords: cognitive development; diagnosis; early development; speech and language impairment; pre-schooler; WPSSI, Griffiths, JSAIS.

Table of Contents

Chapter 1: Introduction....7
Chapter 2: Literature Review....9
Diagnostic associations with language and cognitive development....13
Autistic Spectrum Disorder (ASD).....13
Epilepsy.....14
Difficulties in the diagnosis of speech and language disorders with no apparent aetiology....14
Expressive Language Disorder Spectrum (ELD).....17
Mixed Receptive and Expressive Language Disorder Spectrum (MRELD).....17
Mental Retardation (MR).....18
Genetic Disorders.....19
Co-morbid Conditions.....22
Rationale and Aims....38
Research Questions....39
Chapter 3: Method....40
Design.....40
Participants and Sampling.....40
Materials and Instruments.....41
The Wechsler Pre-school and Primary Intelligence Scale (WPPSI).....41
The Junior South African Individual Scale (JSAIS).....44
Griffiths Mental Development Scales.....46
Independent Variables.....47
Procedure.....48
Threats to Validity.....51
Data Analysis....52
Chapter 4: Results....54
Normality of the Data.....56
The Wechsler Pre-primary and Primary Scale of Intelligence (WPPSI).....57
The Junior South African Intelligence Scale (JSAIS)....63
The Griffiths Scales of Mental Development.....68
The Influence of Diagnosis on Cognitive Functioning.....73
The Prediction of Age of Symptom Identification and Subsequent Intervention.....75
Chapter 5: Discussion....78
General Descriptive Differences.....78
Pregnancy and Early Childhood Health.....84
The Pervasiveness of Speech and Language Impairment.....86
Brain Laterality and Childhood Impairment.....88
Further implications of the study....96
Limitations of the current study.....97

Recommendations for Future Research. 100
Conclusion 100
References 102
Appendix A 117
Ethics Certificate 117
Appendix B 118
Description of the WPPSI-III Subtests 118
Appendix C 121
Description of the Subscales of the Griffiths Scales of Mental Development 123
Appendix E 125
Intake Forms for the Pre-Primary School 125
Appendix F 131
Kolmolgorov-Smirnoff Tests of Normality for the WPPSI, JSAIS & Griffiths 131

Chapter 1: Introduction

The study of cognitive functioning and development has been firmly established as one of the cornerstones of psychological assessment and interest in the last century. These investigations began formally in the early 19th century with the work of Simon and Binet and their assessment of children's intellectual functioning (Kamphaus, 1993). Theories of intelligence, and its subsequent assessment, have since blossomed into a sub-discipline of psychology that attempts to thoroughly investigate and accurately monitor the changing cognitive capacities as they occur within typically developing children. Published studies indicate that there are certain relationships that we can expect in a typically developing child population. For instance, research suggests that pregnancy complications and poor maternal and neo-nate health are negatively associated with cognitive development, and increase vulnerability to impairment (Kay, Ferguson, Molfesa, Peach, Lehman, Molfesa, 2007; Herrera, Salmeron & Hurtado, 1997). Other research has indicated that children from lower socio-economic brackets and that those whose parents are poorly educated, are at higher risk for developing certain cognitive impairments (Ginsborg, 2006).

Alongside these understandings of cognitive development, it has also been ascertained that cognition and language development have a highly interdependent relationship. It is believed that the two mature alongside one another in oscillating patterns. As one of them matures and expands, so it capacitates the other to develop and catch-up to the other. This pattern is repeated to increasing degrees (arguably into adulthood) as humans continue to enhance both their linguistic and intellectual bases (Baird, 2008; Owens, 2004). This dialectic relationship is very complex, with multiple variables influencing the technicalities of the process at various times along the linear time-periods that constitute both cognitive and linguistic acquisition. While some influences have been identified, the *precise* mechanisms behind this 'catch-up' process have not yet been identified, and it is unclear exactly which variables are essential for this process of optimal dual-development.

While researchers continue to investigate the mechanisms and predictors of linguistic and cognitive development within typically developing children, very little is known about this process within an atypical population – particularly children who have speech and language impairments (Hughes, Sapp & Kohler, 2006). There are two hypothetical situations that result from the presence of language impairment and its symbiotic relationship with cognitive development. This first is that, despite the presence of language impairment, cognitive functioning can be predicted in very similar ways to that of typical populations. Alternatively, the existence of language impairment could cause intellectual functioning to develop in a very different way. If this is so, the cognitive profiles of children with language impairment are likely to be somewhat different to that of typically developing children. Further, the variables that are associated with these differences can be identified and compared to those known to predict performance in typical populations.

An understanding of these mechanisms within an atypical population has the potential to offer much needed direction in the study of children whose development differs from expected norms. Speech

and language impairment is symptomatic of many childhood disorders and syndromes (Autistic Spectrum Disorder, Specific Language Impairment, Learning Disabilities), and can potentially inhibit development in other areas of functioning (motor control, social awareness and emotional expression) if not intervened with appropriately. The biggest obstacle to appropriate intervention across these impaired populations is perhaps the lack of knowledge about the mechanisms and influences of the various domains of their development. By understanding the ways in which these differ from typically developing populations, it becomes possible to direct appropriate intervention toward affected individuals, so as to maximise their functioning and potential. Further, understandings about the influences of these impairments also serve to profile at-risk families, groups and individuals for vulnerability to future impairment. Preventative and compensatory intervention is often the best chance at maintaining functional and optimally developing children. This study reports on the descriptive characteristics and early developmental predictors of cognitive functioning in a sample of 164 pre-school children with speech and language impairment.

Chapter 2: Literature Review

The following chapter seeks to comprehensively discuss the nature of language and cognitive development within pre-school children, and the various attributions highlighted by previous research that are believed to influence its development in different manners. These contributions are discussed with particular reference to the sample of this study, namely pre-school children with speech and language impairment.

The typically developing child shows remarkably rapid acquisition of the skills of extracting meaning and communicating using speech. The exact process whereby children acquire the use of language and speech is not yet fully understood, but it is believed that a range of linguistic and cognitive processes are required to smoothly integrate to form a complementary proficiency (Baird, 2008). Infants are innately motivated to attend to stress patterns, rhythms and spaces of speech. Later, between 6 and 9 months this ability is fine-tuned as auditory perception increases the child's familiarity with their own voice. After this, the child learns to read other people's mental states to infer a speaker's meaning. This skill, merged with joint attention, where the child can switch between a speaker and an object, allows for the conceptualisation of the changing meaning of words within sentences. Typically, by the age of 5, a child has not only mastered the fundamental structural aspects of language but has acquired a knowledge of pragmatics, and can use both verbal and non-verbal signals to understand and convey a variety of different meanings within context (Baird, 2008).

The age range by which children are expected to learn language and talk is wide, and highly dependent on the circumstances in which language is elicited. Studies indicate that 16 month toddlers can understand between 78 and 303 words, and produce between 0 and 154 words. Many children who are late to talk will catch up spontaneously with no intervention or explanation for their delay (about 60% recover by themselves between aged 2 and 3 years) (Baird, 2008). However, this makes it difficult to assess who will recover spontaneously and who requires early intervention to assist with this acquisition (Fenson, Dale, Rezsnick, Bates, Thal & Pethick, 1994). Additional factors such as child temperament, intelligence and family structure have all been associated with differing rates of language acquisition. However, further research is needed to identify the exact pathways by which these pathways interact (Law, 1992).

In light of the variation in rate of language acquisition, it is also important to highlight from the outset the distinction between language 'disorder' and 'delay'. While there might exist considerable overlap in the use of the two terms, they in fact refer to separate and distinct clinical populations. The former comprises a group for whom the pattern of language development has been disturbed and has a consequent poor prognosis. The latter comprises a group of children who develop language normally but at a slower rate than their peers, and have a better prognosis. However, the distinction is not always used precisely even in academic literature. Some argue that the distinction is easier to make in hindsight, after various treatments are evaluated for their degree of effectivity (Law, 1992).

Thought and language cannot be studied or fully understood in isolation of each other. The Piagetian term 'egocentric speech', implies that both thought and verbal expression provide meaning to each other through increased growth and interaction with the outside world (Vygotsky, 1962). To learn any type of language, children require the effective functioning of an array of cognitive processes, including the ability to perceive sequenced acoustic events of short duration; the ability to attend actively, to respond to and anticipate stimuli; the ability to use symbols; the ability to invent syntax from the language of their environment; and the capacity to do all these things simultaneously (Owens, 2004). Likewise, cognitive development is heavily reliant on the effective interpretation and use of language.

It is worth noting the contributions of socio-cognitive responsiveness and working memory to the development of cognition and language. Firstly, socio-cognitive responsiveness refers to the skills that underlie the communication relationships between people (joint attention, theory of mind and imitation). This skill was highlighted by Vygotsky, in his term, the Zone of Proximal Development (ZPD), which refers to an extended skill set that a child is capable of learning within an environment of full social interaction (i.e. being taught by an able instructor or parent). Hence, children develop both their cognition and language skills if they are given maximum use of their ZDP and develop good socio-cognitive responsiveness skills (Botting, 2006; Vygotsky, 1978). Working memory refers to the ability to actively hold information in mind and manipulate it to achieve complex tasks such as reasoning, comprehension and learning. These include executive attention, information integration, processing and retrieval (Baddeley, 1998).

Within a Vygotskian framework, cognitive development is considered an active process in which language plays an essential role. Language is considered to develop interdependently with cognition, and is fundamental to all knowledge, "both as an interpersonal communication system, and as a cognitive, representational system" that facilitates development (Garton, 1992, p. 3). The development of thought and that of language, do not, however, progress in parallel but as "two growth curves that cross and re-cross, straighten out and run side by side, even merge for a time, but they always diverge again" (Vygotsky, 1962, p.33). Studies by Botting (2005) also show that language and cognitive development have a reciprocal relationship. A sample of 82 children with Specific Language Impairment (SLI) and other linguistic impairments, and matched on base-line non-verbal ability, were assessed at ages 7, 8, 11 and 14 years with appropriate IQ tests. It was found that not only did their verbal scores remain relatively constant (as expected), but that their *non-verbal* performance scores actually declined upon consecutive follow-ups in later years, specifically between the ages of 8 and 11 years. On average, the sample experienced an average decline of 20 IQ points over the duration of the study. These researchers, and others, attribute this decline to the inter-relatedness of language and cognitive development in pre-school children, proposing that the poor verbal ability that is characteristic of children with SLI subsequently impaired non-verbal cognitive development (evidenced by lower performance scores) (Clegg, Hollis, Mawhood & Rutter, 2005; Krassowski and Plante, 1997; Paul & Cohen, 1984).

Considering this interdependent relationship, it can be assumed that both cognition and language are likely to develop differently in atypical populations. While the *precise* ways in which the cognitive abilities of children with SLI would differ in this regard is not well documented, yet there are studies indicating that the difficulty in researching the issue lies in the equivocal nature of the diagnostic category of SLI (which necessitates a non-verbal IQ of over 85 IQ points) and its overlap with other childhood disorders (Owens, 2004).

There is much debate about the levels of cognitive functioning in hearing and language-impaired populations. Some studies have found that hearing-impaired pre-schoolers fall within the lower average limits on IQ scales (85-90) (Savage, Evans & Savage, 1981), while other writers have found that 'in many aspects of cognitive skill, [hearing-impaired] respondents are of equal to and even superior to the hearing…. [and] it is reasonable to conclude that the cognitive abilities of these children are essentially normal' (Rodda & Grove, 1987, p. 183). Recent research however, supports the ideas of Vygotsky's dialectical interdependence between cognitive development and language acquisition, and suggests that cognitive functioning is compromised in populations who have co-morbid language impairments (Hansson, Sahlen & Maki-Torkko, 2007; Lyxell & Holmbergm 2000; Marton, Abramhoff & Rosenzweig, 2005; Miller & Gilbert, 2008).

Attention is now directed to the possible causes and associated conditions of speech and language impairment. Hearing loss can interfere with the development of many different skills that may or may not be related to cognitive ability (Sattler, 1988). Language impairment is usually one of the major consequences of hearing impairment, as it is difficult for children to develop adequate understandings of sound, phonics and the manipulation of their own vocal chords if they are unable to correctly perceive an externally available model. In the next section, the basic characteristics and causes in some of the childhood disorders that are characterised by language impairment are considered, namely Autistic Spectrum Disorder (ASD), SLI and its two major subtypes, Language Learning Disorder (LLD) and mental retardation (MR), various congenital and genetic conditions and associated disorders and impairments that are often considered secondary to the diagnosis of language impairment, since these are the main diagnostic categories of the children comprising the sample of this study.

Speech and language problems can be classified within four categories: receptive (understanding), expressive language, dysfluency (stuttering) and other. These categories are not mutually exclusive and because of their inter-relatedness with cognitive processes and language learning, it is quite common that speech and fluency (expressive processes) are related to receptive concerns. Speech and language difficulties can also be classified according to their underlying causes, normally to a genetic or congenital condition, or co-morbid hearing loss. While these are recognised as valid and researched aetiologies, demographic factors and syndromes that have a speech and language symptom profile are equally valid.

Some of these are noted in Table 1.1 below. This makes it very difficult to differentiate between primary, secondary or even consequent factors of influence. For instance, while most etiologies are secondary to occurrences like deafness, motor disorder, structural palatal problems, acquired brain disorder and so forth; they are sometimes antecedent to these very things themselves. The distinction between primary and secondary diagnoses therefore is a complex process that is often not resolved (Baird, 2008).

Table 1.1
Factors associated with increased risk of speech and language impairment in children (Baird, 2008)

Etiologies leading to secondary speech and language impairment
Hearing Impairment
Genetic Disorders
Pre-natal exposure to substances such as anti-epileptic drugs, alcohol and narcotics
Acquired epileptic aphasia (sometimes through lesions in Traumatic Brain Injury)
Acquired disorders resulting in neurological damage (e.g. strokes)
Oromotor structural defects (e.g. cleft palate)
Motor dysfunction of central origin (e.g. cerebral palsy) or of peripheral origin (e.g. neuro-muscular disorders)
Impoverished environment (socially or linguistically)
Syndromes in which speech or language impairments are associated
Autistic Spectrum Disorder (ASD)
General Learning Difficulties
Anxiety Disorders (usually associated with Elective Mutism)

Diagnoses of speech and language delays have a broad range of possibilities. When the child struggles to grasp language to a degree that is disproportionate to other aspects of development (non-verbal ability, motor function, cognition), professionals usually investigate possible Specific Language Impairment (SLI) (Baird, 2008).The diagnosis of *exclusive* speech and language problems is also difficult as nomenclature, terminology and strict criteria for diagnosis within the discipline of speech pathology differs between therapists. For example, a linguistic problem might be considered phonological in nature by some, while others will diagnose with a focus on motor aspects and use terms such as ‘verbal apraxia’, or ‘speech articulation problem’. This is further complicated by the need to distinguish children diagnosed with speech impairment because they suffer a *disorder* from those experiencing a *delay*. In practice, this is very complex and often not possible (Baird, 2008).

In an attempt to remedy the diagnostic dilemma that often faces professionals from across disciplines, disorders are considered as primary if there is a known and obvious underlying aetiology for the speech and language impairment (i.e. if a child has ASD, the syndrome, and not a speech and language diagnosis, will be considered to be primary as his symptoms are accounted for by the ASD). However, when there is no obvious or known aetiology for the impairment, then the deficits are diagnosed as a speech and language impairment (Baird, 2008). The procedure of specifying diagnostic priority (with regards to primary or secondary diagnosis) in this study was modelled as close as possible to the abovementioned best-practice strategies by current professionals. Where this was unclear, it was modelled on striations in the

manuals of cognitive functioning measures, and similar studies – the details of which are provided in the Procedures section.

With reference to this study, it is necessary to define speech and language disorders in light of their existence as both an autonomous diagnostic category of childhood impairment and a symptom of other disorders. Generally speaking, speech and language impairment refers to children's inadequate abilities to understand, interpret, perceive and produce both speech and language in a way that is expected within their developmental level (Law, 1992). In order to constrain the material in this study to that of the psychological, the impairments identified in the sample were defined by diagnosis in the Diagnostic and Statistical Manual for Mental Disorder (4th Edition, Text Revision) (DSM-IV-TR). Further, all children within the sample were included because of their placement at a pre-primary school for children with special speech and language needs. This placement is based on investigations to ensure that their impairments are not a consequence of 'normal' delay that can be rectified through weekly speech therapy.

Diagnostic associations with language and cognitive development.

There are several childhood disorders that have associated speech, language and cognitive impairments. All except the genetic conditions are identified as disorders usually identified in childhood by the DSM-IV-TR (2000). Those that were present in the sample of this study are described in some detail below.

Autistic Spectrum Disorder (ASD).

Autism is a lifelong developmental disability that occurs in varying degrees of severity. It is characterised by a wide range of impairments in social, interaction and communication skills, where sufferers may spend long periods of time repeating particular motor activities and focusing on irrelevant aspects instead of the larger environment (Mash & Wolfe, 2005; McCauley, 2001).

Children with autism experience profound difficulties in relating to people, have limited social expression and have deficits in facial recognition. Their behaviour is characterised by high anxiety and irritation, and often screaming and crying in a tantrum-like manner. They also participate in self-stimulatory behaviours such as rocking, spinning and hand-flapping and have a general pre-occupation with stereotyped and restricted patterns of behaviour (Owens, 2004; DSM-IV-TR, 2000). Their inability to understand events from another's perspective, namely the absence of Theory of Mind, is a highly specific, high level cognitive deficit. Children with autism also display deficits in executive functioning (planning and behaviour regulation). Despite also showing a variety of other perceptual abnormalities (weak central coherence), unusual reactions to stimuli and abnormal responses to low-level perceptual tasks (irrelevant detail), these cognitive shortfalls should not be mistaken for generalised dysfunctional cognition. In fact, a few autistic cases have been found to have an above average intelligence, and sometimes possess savant-like talent in a particular task (e.g. mathematics, music). Typically, however, the intelligence levels of

people with autism are below average – often attributed to incomplete development because of their verbal impairment (Hulme & Snowling, 2009).

Language Impairment (LI) is also one of the first indicators of autism, as between 25 and 60 per cent of the autistic population remain mute or non-speaking. Accounting for the great variation in language use and ability within this population, autistic children have difficulty in communicating what is situationally relevant, speaking in a context-appropriate register, and failing to comply with turn-taking in conversation (Owens, 2006).

Autism has been significantly associated with prenatal complications. It has a strong biological and neurological basis as 65 per cent of children with ASD have abnormal brain patterns. Because autism can often be traced in the family lineage, there is recent research that suggests that it might be genetically based (Owens, 2004).

Epilepsy.

One of the most common types of Epilepsy that is associated with speech and language impairment in childhood is the Landau-Kleffner type, often known as Landau-Kleffner Syndrome. This is a highly heritable subtype that localises brain dysfunction in the perisylvian region of the brain which has a devastating impact on language development. It most commonly presents in children between the ages of 4 and 7 years old, where parents usually notice a gradually diminishing use of language, accompanied by profound receptive language impairment. The receptive difficulties may be severe enough to include poor responses to external sounds. While overt seizures are not usually part of the initial presentation, they do occur. Sleep EEG's show a continuous spike wave activity, which in severe cases maybe occur up to 80% of the time (Robinson, Baird, Robinson & Simonoff, 2001)

Other epilepsy types also connected to linguistic impairment is that of Rolandic, and more mixed types that are difficult to identify. Usually clinicians find epileptiform EEG abnormalities in sleep in children with speech and language impairments. However, it is unclear whether this has etiological significance (Picard, Heraut, Bouskraoui, Lemoine, Lacert & Delattre, 1998). Within this research study, it was unclear whether children with Epilepsy experienced the highly genetic subtype of Landau-Kleffner, or if they had another subtype that could have been caused by pre-natal conditions, drug-use or particular brain lesions. For that reason, they were categorised in an autonomous category, although it is understood that they could have had multiple etiologies.

Difficulties in the diagnosis of speech and language disorders with no apparent aetiology.

Specific Language Impairment.

Probably one of the most common expressive language disorders, Specific Language Impairment (SLI) is usually diagnosed by eliminating the existence of other disorders. Children with SLI are distinct from those with more general language impairment because they are said to be of 'normal' intelligence, have no

etiologically related neurological symptoms, have no hearing loss or primary emotional disorder or neuromotor impairment and do not have a predominant environmental disadvantage. Thus, their language skills are markedly below their cognitive skills, without any obvious explanation for their difficulties (Law, 1992, McCauley, 2001).

There are also diagnostic issues around the criteria for the confirmation of SLI. While it is usually diagnosed according to the disproportional impairment of language learning within a normal nonverbal IQ, this should be carefully considered within an understanding of the interconnectedness of verbal and non-verbal reasoning. Further studies suggesting overlap with other disorders such as ASD and Williams Syndrome (WS) in particular make it difficult to understand as a disorder (Tager-Flusberg, 2004; Thomas, Grant & Barham, 2001). These difficulties in distinction also relate to broader issues within health practices which take for granted the underlying mechanisms behind current diagnostic nomenclature. Such practices assume the existence of an essentialised condition underlying (sometimes random) categorisations of disorders according to overt symptoms. Hence, it is sometimes unhelpful to create a 'catch-all' diagnostic category such as that of SLI for a variety of difficulties.

This very issue was demonstrated by research conducted by the Early Language Learning Project, a longitudinal study funded by the National Institute of Health (NIDCD) in the United States, which examines the linguistic processing abilities of toddlers with varying rates of language acquisition. In one section of this study, the language skills of late talkers were matched and compared to those of typically developing toddlers throughout the pre-school period. The late-talkers were also periodically assessed for their speech quality and characteristics. The study made use of a reasonably sized sample (53 children in each group) and had sound and well thought through methods and statistical analyses. The findings from the study suggest that there is a significant degree of overlap in the patterns and mechanisms of language development for typically developing talkers and late-talkers, as well as certain salient similarities between aspects of late-talkers' skills and those usually reported with children with SLI. This not only suggests that language development proceeds in a dynamic fashion where changes in skills and proficiency are triggered by skills that *subserve* language, but that this proficiency occurs along a *continuum* of severity and delay. Further the very low proportion of late-talkers (7%) that are classified as having SLI suggest that the latter might simply exist within the lower ranks of late-talkers. The existence of a language endowment spectrum relates to broader theoretical issues regarding the continuous and dichotomous characterisations of language based skills (Weismer, 2007). These debates are beyond the scope of this research, but they do point to an important consideration – that language disorders reveal more similarities than differences, which makes discrete distinctions between diagnoses a difficult and perilous thing to attempt.

In an attempt to contain the boundaries of this research within that of the psychological, attention will be turned to the DSM-IV to provide explanation of the diagnosis of communication disorders. The DSM-IV lists five types of communication disorders: Expressive Language Disorder, Mixed Receptive Expressive Language Disorder, Phonological Disorder and Communication Disorder Not Otherwise

Specified (NOS). A careful examination of the associated features and characteristics of these classifications highlight that there is a very faint distinction between these disorders. An example of this is that the Phonological Disorder is the most common associated feature of Expressive Language Disorder.

There were numerous reasons why the diagnostic categorisations used in this study were formulated as they were. The wide range of symptoms that often overlap with criteria offered by the DSM-IV make it very difficult to specify diagnostic equivalents within the domain of speech pathology. Further, because this is a psychological study, it was decided that diagnoses of the research sample given by speech therapists about specific speech and language deficits would, as far as possible, be translated to DSM-IV equivalents. This was done to not only compress the wide range of descriptive diagnoses offered into larger categories with less detail, but also to allow for an intra-discipline comparison across disorders within psychology. As mentioned, research suggests that speech disorders may exist on a continuum of severity and not in multiple discrete categories (Weismer, 2007), which allows for the existence of *spectrums* instead of discrete categorisations. Also, standardisation studies on special populations for the largest and most important dependent variable of the study, namely the WPPSI-III, were conducted with DSM-IV and not speech pathology diagnoses (Wechsler, 2002).

Within the sample of the current study, the identification and description of speech and language impairments were wide, which made it difficult to create uniformity across the sample. In an attempt to remedy this variation, children who suffered a speech and language impairment as their primary condition (i.e. that had no other known underlying cause) were categorised following the method used in a widely cited study by Hill (2001). The author separated the sample diagnosed with SLI according to the two major DSM-IV definitions, namely Expressive Language Disorder (those experiencing difficulties in expression and articulation), and Mixed Receptive-Expressive Language Disorder (those experiencing difficulties in language interpretation and perception, and hence struggling to achieve appropriate speech). Other DSM-IV diagnoses of Communication Disorders (Phonological Disorder, Stuttering and Not Otherwise Specified) were not explicitly identified within the sample and were therefore considered redundant. This division of participants with SLI according to the DSM-IV specification is an accepted practice amongst researchers (McCauley, 2001). Further, because of the wide variation in abilities, it was decided to consider the two categorisations of impairment as existing on a spectrum. In order to fully explain this, the characteristics of Expressive Language Disorder Spectrum and Mixed Receptive-Expressive Language Disorder Spectrum are discussed in some detail next.

Expressive Language Disorder Spectrum (ELD).

Expressive Language Disorder is diagnosed if the conditions outlined in Table 1.2 below are satisfied (DSM-IV-TR, 2000).

Table 1.2
Diagnostic Criteria for Expressive Language Disorder (315.31)

A.	The scores obtained from standardised individually administered measures of expressive language development are substantially below those obtained from standardised measures of both non-verbal intellectual capacity and receptive language development. The disturbance may be manifest clinically by symptoms that include having a markedly limited vocabulary, making errors in tense, or having difficulty recalling words or producing sentences with developmentally appropriate length or complexity.
B.	The difficulties with expressive language interfere with academic or occupational achievement or with social communication
C.	Criteria are not met for Mixed Receptive-Expressive Language Disorder or a Pervasive Developmental Disorder.
D.	If mental retardation, a speech-motor or sensory deficit or environmental deprivation is present, the language difficulties are in excess of those usually associated with these problems.

It is also characterised by limited speech and vocabulary, difficulty acquiring new words, word-finding errors, limited grammatical structure, very little variety in sentence types, unusual word order, omission of critical parts of sentences and a generally slow rate of language development. Non-linguistic functioning (as measured by non-verbal IQ) and language comprehension skills are in normal limits. These features refer to a type of verbal apraxia.

Mixed Receptive and Expressive Language Disorder Spectrum (MRELD).

Children diagnosed with this disorder have very similar difficulties to those presented in Expressive Language Disorder and have additional impairments in their receptive language development. They have difficulty understanding words, sentences or particular types of words or terms. In severe cases they have difficulties in their auditory processing (the discrimination of sounds, association of sounds and symbols, storage, recall and sequencing). Because the development of expressive language in childhood relies heavily on the reception of language and the skills that are involved in acquisition of language input, a pure Receptive-Language Disorder, or aphasia, is impossible in children. This is because aphasia is classified as the loss of *previously acquired language use*, and children are in the process of acquiring language and so cannot lose a skill that they never possessed. Cranberg, Filley, Hart & Alexander (1990) considered *acquired* aphasia in children, but all eight children in the study had left hemispheric lesions – making the disorder similar to that found in Wernicke's adult aphasia (Cranberg, Filley, Hart & Alexander, 1990). Comprehension deficits are the primary features that differentiate the disorder from others. It is also very difficult to diagnose, as children often give tangential responses or appear not to be paying attention, and so can be confused with AD/HD (American Psychiatric Association, 2000). Specific criteria for MRELD can be found in Table 1.3 below (DSM-IV-TR, 2000).

Table 1.3
Diagnostic Criteria for Mixed Receptive-Expressive Language Disorder (315.32)

A.	The scores obtained from a battery of standardised individually administered measures of both receptive and expressive language development are substantially below those obtained from standardised measures of non-verbal intellectual capacity. Symptoms include those for Expressive Language Disorders as well as difficulty understanding words, sentences or specific types of words such as spatial terms.
B.	The difficulties with receptive and expressive language interfere with academic or occupational achievement or with social communication
C.	Criteria are not met for a Pervasive Developmental Disorder.
D.	If mental retardation, a speech-motor or sensory deficit or environmental deprivation is present, the language difficulties are in excess of those usually associated with these problems.

Language Learning Disability (LLD) is considered to be a type of learning disorder (in a similar way to mathematics and reading learning disorders are identified). Yet, it has very similar characteristics to those identified as existing on the spectrum of impairment of Mixed Receptive-Expressive Learning Disorder. LLD was identified in four of children in the sample, but because of the high proportion of symptomatic overlap it was included in the category of MRELDS for statistical parsimony. Its characteristics and etiologies are briefly summarised below.

Language Learning Disability (LLD).

In general, LLD is a sub-category of a learning disability and is characteristic of 75 per cent of the learning disabled population who have difficulty learning language. As a result, these children have memory difficulties and have overall slow oral language development, despite having an average intelligence (Owens, 2004). There is usually great disparity between their verbal and non-verbal scores and they usually have accompanying social and behavioural problems. LLD is diagnosed in the early school going years, and can usually be identified with co-morbid perceptual deficits. Thus, children with LLD do not perceive in a normal fashion (as a result of peripheral nervous system (PNS) dysfunction) and struggle with listening, reading, comprehension. These children battle to find words in conversations and thus need a longer time to respond verbally (Owens, 2004). Such pre-schoolers tend to have poor attentional selectivity, and concentrate on inappropriate stimuli, which deficits in working memory (Owens, 2004). It also has a nine times greater incidence in boys than in girls (Hulme & Snowling, 2009).

Mental Retardation (MR).[1]

Children with mental retardation also show noticeable language delays and impairments, but are differentiated from those with LLD in that they have an accompanying poor generalised intelligence. They show a considerable range of impairments that involve general limitations to daily living – the most acute being difficulties in learning to communicate due to limited speech and language skills (Mash & Wolfe,

[1] The term 'mental retardation' and not the more politically correct substitute 'intellectual impairment' is used because of its diagnostic function in the DSM-IV.

2005). Theoretically, MR is diagnosed if the child's IQ is two standard deviations or more below the mean on a standardised IQ test, with significant limitations in the areas of communication, self-care, home-living, social skills and health and safety that manifest before the age of 18. Four degrees of severity are identified as reflecting the level of impairment (Mild MR [IQ = 50-70], Moderate [IQ = 35-50], Severe [IQ = 20-35], and Profound [IQ below 20]). In 25 per cent of the MR population, language comprehension and production is below the level of cognition (Owens, 2004).

Mental retardation was not included as a diagnostic category in the study for two reasons. The first is that while it *is* recognised that certain children in the sample would satisfy the criteria for mental retardation (i.e. IQ scores below 70 points), they were never identified as such in their clinical files. Most were given a primary diagnosis of a genetic condition, and were admitted to a pre-primary school that specialised in the development of their speech and language deficiencies. It was assumed that they would benefit from being in an environment that specialised in speech and language development in particular, and not a more generalised special needs school. Secondly, it is very difficult to identify if language impairment causes the intellectual impairment, or if the intellectual impairment precedes the language delay. In cases such as neonate hypoxia (often resulting in Cerebral Palsy) it is obvious that the language impairment is one of many deficits resulting from the brain damage. However, in cases of unknown aetiology, it is difficult to establish which precedes which. In these cases, children were identified as having a primary diagnosis of either ELDS or MRELDS (usually the latter). Technically this violates the diagnostic criteria for SLI (which stipulates a minimum non-verbal IQ of 85) which is the over-riding diagnostic category of these two DSM striations. As discussed earlier, there is much controversy surrounding the exclusionary and often contradictory criteria of SLI in any case (Bishop, 1997). Many of children suffered from conditions which had a genetic disorder which have accompanying language impairments. These are described below.

Genetic Disorders.

Genetic disorders refer to abnormalities within the chromosomal material of human cells. The chromosome, a single large package of tightly coiled DNA material in every somatic cell, contains genes and systemic regulator elements that are responsible for a wide range of our functioning. In some cases of reproduction, an extra chromosome is incorporated into the gamete so that there are three, and not two, copies. In others, whole chromosomes or parts thereof are deleted in the process of replication. These alterations manifest as a variety of disorders. There is extensive evidence to suggest that most chromosomal conditions which affect cognitive development also affect speech and language acquisition (Law, 1992). An increase in chromosomal abnormalities, particularly in the sex chromosomes, has been reported in children with Klinefelter's Syndrome (XXY, XXYY, XXXY), XYY Syndrome and Trisomy X (XXX) which are all characterised by delayed speech and language learning difficulties. In other instances there

are defects within particular genes within chromosomes and these disorders can be profiled according to particular patterns of strength and weakness within speech and language acquisition. For the sake of brevity, only those genetic disorders identified within the sample of the study are explained (Baird, 2008).

*Hydrocephalus.*The primary characteristic of this condition is the excessive accumulation of cerebrospinal fluid on the brain, caused by abnormal dilation of the entrances to the brain ventricles. It may be a congenital condition caused by genetic abnormalities, or by developmental disorders such as Spinal Bifida or Encephalocele, and sometimes as a result of traumatic brain injury, meningitis or subarachnoid haemorrhaging. Symptomatically, it is often noticed in infancy due to the unusually large head circumference, and can be accompanied by poor motor function, poor memory and generally poor cognitive functioning. The degree of severity can be broad (Aschoff, Kremer, Hashemi & Kunze, 1999).

Fragile X Syndrome. Fragile X Syndrome is a leading cause of intellectual impairment, and is the result of a mutation in a single gene (FMR1) located on the X chromosome. It results in the inability to produce protein FMRP which is involved in processes of neural myelination, the correct functioning of neural synapses and the appropriate organisation and activation of neural activation. As an X-linked disorder it affects the sexes differentially, and surprisingly affects double the male (1 in 4000) population as it does the female (1 in 8000). While there are many cognitive functions in this population that are impaired, it is especially language skills that are affected, including those involved in the processing of sequential information, auditory short term memory and the direction and sustaining of attention. People affected show relatively strong abilities to process simultaneous information, long-term memory and the ability to distinguish the self from the other. They also show high degree of co-morbidity with hyperarousal, hyperactivity and anxiety disorders and sometimes display autistic-like behaviours that are sufficient to warrant a diagnosis of ASD in 10-40% of individuals (Abbeduto & McDuffie, 2007).

Bronchio-OTO-Renial Syndrome. Bronchio-OTO-Renal (BOR) syndrome is a genetic disorder that mainly affects the areas of the inner ear and the kidneys, and is caused by a mutation on the EYA1 gene on chromosome 8. The symptom profile usually includes hearing loss, periauricular (ear) pits, branchial cysts and mild kidney dysfunction. The ear pits, tiny holes immediately in the front of the ear are usually accompanied by figural malformations of the ear. Branchial cysts are small openings which are located on the lower neck area and drain fluid occasionally. While not all features of the syndrome are expressed in all carriers, few carriers lack all of the features. The ear pits, branchial cysts and hearing loss are the most common symptoms. Of most interest in this study is the accompanying hearing loss which can vary in severity and cause – being sensorineural, conductive or a mix of the two (Fraser, Ling, Clogg, Nogrady & Gorlin, 2005; Fraser, Sproule, Halal & Optiz, 2005). Within the context of this study, it was assumed that

children would require intervention for their speech impairment that would result from the hearing loss characteristic of the ear pits in BOR Syndrome.

Williams Syndrome. This is characterised by a single gene defect where there is a deletion of genetic material from chromosome 7. The syndrome in general arouses a lot of interest because of claims that affected children have normal language skills despite their low IQ. However, within the context of early learning, these claims are highly unlikely. Although language acquisition in this population is usually delayed, they do make use of fluent and complex language. Closer inspection usually reveals that there exist major limitations in comprehension (Baird, 2008; Bates, 2004; Brock, 2007).

Ehlers Danlos Syndrome. This genetic disorder is actually quite far removed from those usually associated with speech and language impairment. It is caused by mutations on multiple genes that alter the structure, production and processing of collagen, or the proteins that interact with its formation. It is predominantly characterised by highly flexible fingers and toes, skin hyperelasticity, muscle weakness and easy bruising. There are no associated cognitive impairments or overt speech impairments, but the disorder does result in a high and narrow palate which can cause poor speech formation and acquisition. In utero, carriers often experience premature rupture of the placental membranes which can cause complications and foetal distress during pregnancy (Beighton, De Paepe, Steinmann, Tsipouras & Wenstrup, 1998; Gedalia, Press, Klein & Buskila, 1993; Lawrence, 2005).

Pierre Robin Syndrome. This disorder is strictly not a genetic condition, but rather a congenital disorder arising in neonatal conditions. It is believed to be a result of developmental malformations that occur in-utero. It is understood that during the formation of the bones, the jaw and collar bones become stuck, which prevents the jaw from developing normally. Symptomatically, it is characterised by an usually small jaw, posterior displacement or even retraction of the tongue and upper airways obstruction. Its most salient and predominant feature is an often U-shaped cleft palate. Most grow into normal and healthy adults. The implications of the disorder within the atypical population of interest is the presence of the cleft palate and breathing problems in infancy that prevent correct speech development and create a vulnerability to foetal distress and sickness (Jakobsen, Knudsen, Lepinasse et al., 2006; van den Elzen, Semmekrot, Bongers, Huygen & Marres, 2001).

*Prader-Willi Syndrome.*This is another rare genetic disorder due to deletions or partial expression of seven genes (q 11-13) on chromosome 15 (Curfs & Fryns, 1992). It is characterised by a myriad of differing symptoms that change through the lifecycle. In utero, these infants are frequently in abnormal positions

and have excessive amniotic fluid which often results in complicated and stressful birthing procedures. Infants are characterised by lethargy, hypotonia, and high rates of feeding difficulties which often result in a failure to thrive (Cassidy, 1997). Within early childhood, speech delay is one of its most common features, as well as excessive hunger (often leading to childhood obesity), behavioural problems, poor motor function and below average intellectual functioning. Only the top 5% of the clinical population are believed to reach their 'ceiling' IQ limit of 85 (Holm, Cassidy & Butler, 1993).

Co-morbid Conditions.

One of the most marked associated factors of language impairment is the presentation of co-morbid behaviour problems (between 48 and 53% of both a clinical and non-clinical population), as children without the ability to fully understand their environments and engage with it reciprocally are at high risk to develop social and behavioural problems (Crowley, 1992). The mechanisms behind this association are not clear. One hypothesis is that the frustration incurred from poor comprehension and expression is antecedent to social and emotional problems which manifest in alternative and often socially inappropriate behaviour. Studies supporting this view identify that it is often only language impaired children (and not speech impaired children) who display behavioural problems, indicating that it is perhaps specific to the frustration felt through poor comprehension. Other hypotheses are that both linguistic and behavioural difficulties are caused by a common factor, or that they both appear spontaneously from separate and unrelated aetiologies. Other theorists suggest that emotional and behavioural problems are associated with language disorders because of the inappropriate parental response as a result of poor coping skills with the developmental delay (Cantwell & Baker, 1987; Botting & Conri-Ramsden, 2000; Beitchman, Hood & Inglis, 1990).

Attention-Deficit/Hyperactivity Disorder (AD/HD).

AD/HD is the psychiatric disorder most commonly associated with both Expressive and Mixed Receptive-Expressive Language Disorders (American Psychiatric Association, 2000), and is more highly correlated with language impairment than emotional or social psychiatric problems (Bruce, Thernlund & Nettelbladt, 2006; Cohen, Vallance, Barmick, Im, & Menna, 2000). Studies suggest that AD/HD has stronger associations with receptive language impairments than those predominated by speech. A 15 year longitudinal study tracking children with co-morbid diagnoses of language impairment and AD/HD ($N_{3\ years} = 87, N_{15\ years} = 71$) with good resistance against attrition and a matched control against 49 of the sample showed that pre-school language impairment is *not* a risk factor in itself for poor mental health in later life. However, the risk is increased with increased severity of language impairment and low nonverbal IQ (Snowling, Bishop & Stothard et al., 2006). While the study did have a relatively homogenous sample (with regard to social class and parental education), it did not assess early infant information and so cannot account for these differences in predicting risk-factors like adversity and inadequate infant caregiving.

Other studies have examined the similarities between children with SLI and children with AD/HD and have found both to overlap in areas of poor working memory and cognitive processing. This suggests that there might exist a language deficit that is characteristic of AD/HD (Cohen et al., 2000). This is supported by the same study's findings of similarities between the psycholinguistic profiles of children with AD/HD and those of other children suffering mild to moderate sensorineural hearing loss (Redmond, 2005). This is not to suggest that AD/HD is at all similar to the deficits in comprehension experienced by SLI sufferers, but simply that they have underlying common features. Within the context of this atypical population, one wonders whether the co-morbid diagnosis of AD/HD is simply a misinterpretation of the frequent inattention characteristic of Receptive Language Impairment, or if the associated features of language impairment in some children with AD/HD have been incorrectly diagnosed as a secondary diagnosis rather than a primary (and more serious) symptom.

Anxiety Disorders.

Anxiety disorders (specifically separation anxiety and generalised anxiety disorder), often accompanied by enuresis, are also commonly associated with Expressive Language Disorder (American Psychiatric Association, 2000). Within this study, it was not included as an original diagnostic category, but was added during the process of data collection in response to the unexpectedly high proportion of children experiencing anxiety disorders.

Motor Integration Disorders.

Motor dysfunction, Developmental Co-ordination Disorder, and difficulty processing muscle movements smoothly are all associated characteristics of Expressive and Mixed Expressive-Receptive Language Disorders (American Psychiatric Association, 2000). While it is believed that both gross and fine motor control are significantly poorer in children with language impairment, their rhythm and timing of movement is within normal age-appropriate ranges (Zelaznik & Goffman, 2010). One study sought to investigate this link by looking at previous studies concluding the correlation. It was found that substantial co-morbidity exists in children with SLI and those with poor motor skill, suggesting that SLI is not a specific disorder of language but rather that it includes a broader range of difficulties. Current theoretical understandings of the disorder (possibly because it is determined by a process of exclusion) do not account for the wide-ranging difficulties. In order to remedy this, the author suggested that neuroimaging studies and detailed explanation of shared cognitive process be undertaken to understanding a potentially novel diagnostic category (Hill, 2001).

Other causes of language impairment can be due to traumatic brain injury, various genetic syndromes not mentioned here, developmental disorders and toxin-induced syndromes (e.g. Foetal Alcohol Syndrome). Hearing impairment will also have a major impact on a child's ability to acquire and use language as an aid to his cognitive development. Birth complication, chronic ear infections, and sometimes

the over- and ineffective use of antibiotics can cause hearing impairment and have long-term effects of language development, even if full hearing returns (Guy, 1999). Because of the high co-morbidity between diagnoses and proposed etiologies, multiple diagnoses were considered as a category on their own. This was done to prevent over-representation, and to allow for the cognitive profiles to indicate whether multiple impairments had a poorer association with cognitive performance than having a diagnosis of a single disorder.

In typically developing children, by definition, cognitive development and language acquisition occur within normal developmental periods. Atypical cognitive development in atypical populations, however, relies on more than simply biological symptoms that can be given a diagnosis. The interaction of neural pathways with external factors which exert an *external* influence are also important to consider in understanding the mechanisms which underlie these cognitive-linguistic processes within this population. Attention is now turned to these influences.

The external factors which influence the development of cognitive processing within the sample were gleaned from an intake questionnaire that was mainly concerned with the family's demographic information and child's early developmental history. This combination of factors is by no means exhaustive and is simply a reflection of the information available within the data of the study. Further, these items have been condensed to include more general factors that relate to available research. The discussion begins with a consideration of the age of identification of the difficulties and subsequent intervention. The age at which a child's difficulties are identified and intervened upon are often considered as paramount in order to lessen the effects of impairment on future development. Language is no different from other developmental categories in children, in that its optimum acquisition must happen within a specific developmental period (known as critical periods). Within language acquisition, this is optimally around 2.5 years as this is when children typically start to speak (Law, 1992). Therefore the early identification and intervention of difficulty in its acquisition or fluency is of paramount importance if the effects of the impairment and its consequent deficiencies and limitations are to be compensated for (Rossetti, 2001; Ruben, 1991). There are multiple studies which highlight the benefit of early intervention for children experiencing developmental difficulties early on in life, which has been found to not only improve rates of language and cognitive development (Resnick, Eyler, Nelson, Eitzman & Bucciarelli, 1987), but have secondary effects on better feeding practices, weight-gain, increased routine and behaviour (Als, Lawhon & Duffy 1994), better school readiness (Johnson & Walker, 1991), improved mother-child interaction and long-term developmental outcomes (Spiker, Ferguson & Brooks-Gunn, 1993). These interventions often occur through indirect processes such as education of staff, individualised care, multi-modal intervention strategies, home-based visitation, health care worker screening and parental training (Rossetti, 2001). These processes are often associated with maternal level of education, socio-economic status, and access to primary health care and education. Additionally, early intervention is also estimated

to be cost effective in reducing the capital output required later on in remedial support for children not afforded the benefit of best-practice intervention at a young age. Estimates vary, but are believed to be between a 7:1 and 3:1 ratio of cost output. Early intervention becomes considerably more favourable when economic benefits are deliberated (Barnett, 1993).

A second factor identified from the biographical questionnaire was h**e**aring loss, which is usually described according to the site of lesion. Any problem of transmission of sound through the outer or middle ear causes conductive hearing loss, while the term 'sensorineural hearing loss' refers to hearing loss due to damage to the peripheral auditory system (i.e. the cochlea or auditory nerve). In rare cases, children present with apparent difficulties in hearing, listening and deriving meaning from sound but upon inspection they have normal hearing. In these cases, the problem is highly localised within the central nervous system and its ability to interpret neural transmissions from the auditory nerve. This is known as central auditory dysfunction.

The causes of hearing loss vary according to its type and can include infection, congenital conditions, genetic malformations and traumatic brain injury. While the impact of hearing impairment on the acquisition of speech and language might be taken for granted, it is untrue that any degree of hearing loss will cause a proportionally equal delay in speech and language acquisition. In fact it seems that there exists a threshold past which hearing impairment legitimately causes speech delay. It is difficult to measure with quantitatively based audiology equipment and so a qualitative definition has had to suffice (''where the hearing loss reduces the intelligibility of a speech message to a degree inadequate for accurate interpretation and learning'') (Emanuel & Herman, 1992, p. 88). Complete deafness has an obvious impact on the development of speech and language acquisition as without the ability to hear and perceive sound, there is limited means by which it can be attuned to and replicated. The degree of hearing loss, provided it is beyond the highly subjective threshold, is usually proportional to the degree of speech and language delay or impairment. While some sensorineural hearing impairment is gradually progressive, most remain consistent over the childhood years. Deafness can also be a product of bacterial and pneumococcal meningitis in infancy (7% of all cases experience this) (Baird, 2008).

An obvious factor that emerged from the biographical questionnaire, and which has important influence on language and cognitive develop is that of pre- and peri-pregnancy maternal health, as exposure to various forms of toxins can cause significant damage to the development of children while in utero. Maternal prenatal exposure to alcohol and cocaine has been found to have highly significant deficits in the child, including cognitive impairment, learning disabilities, delays in language acquisition as well as receptive and expressive language deficits (Cone-Wesson, 2004). Similarly, peri-natal exposure to nicotine found in cigarette tobacco is found to be equally adverse to the development of the central nervous system (indicated by lower scores on verbal and performance IQ). These infants showed a marked delay in

speech sound discrimination and speech processing due to sub-optimal brain activity (Herrera, Salmeron & Hurtado, 1997; Kay, et al., 2007)

The length of labour, the mode of delivery, complications during pregnancy and premature delivery and birthweight are also related to the health of the mother-infant dyad, and are believed to hold possible influence for future cognitive and language development. In a review of three studies comparing mode of infant delivery with cognitive ability (measured at 24 months) and maternal health, it was found that unassisted vaginal birth and delivery by caesarean section were highly recommended as they had no significant impact on the child's later development (Berghella, Baxter & Chauhan, 2005; Litt, Armon, Seidman, Yafe & Gale, 1993). Another study considered the long-term impacts of forceps delivery on both maternal health and infant development (measured by appropriate milestones in sight, hearing and motor development). The study showed that the majority of the 313 women using unassisted-instrumental delivery had shorter labours, with less complications, only 1 had an Apgar score of less than 7, and none required a blood transfusion (compared to 15 in the forceps group). While this study found that there were no significant consequences to the child's health, many mothers reported temporary facial scarring on their infants from the instruments and major complications such as delayed developmental milestones (measured at 6 and 12 months) (Johanson, Wilkinson, Bastible, Ryan, Murphy, & O'Brein, 1993)

Parents are often very concerned that complications and difficulty during birth are the cause of their children's problems, and it is sometimes not far from the truth. However, a difficult pregnancy or birth may actually be a product of pre-existing foetal difficulties. It is obviously not possible to tease out the causes in this case, but birthing factors remain an important variable to consider when deliberating the influence of pregnancy on future health and growth of the child (Baird, 2008). Birthweight is possibly the best indicator of a child's health and post-natal development. One Dutch study found that 42 per cent of their cohort of infants born at 1500g had at least mild hearing loss. These children later reported significant educational and cognitive difficulties, with a language impairment prevalence (measured at 3 years) of 22% (Weisglas-Kuperus, Baerts, Graaf, Zanten & Sauer, 1993). Other studies reveal significantly high rates of neurosensory and cognitive impairment, together with more subtle neuropsychological and behavioural problems in low birth weight infants (Hack, Klein & Taylor, 1996; Boyle). Another found that low birthweight pre-schoolers had significantly lower scores in perceptual motor skills, defects in spatial attention and a higher incidence of perceptual motor disabilities. Thirty six of the 85 infants in the study were pre-33 weeks and showed severe motor, perceptual, cognitive and behavioural disorders due to perinatal brain damage (Torrioli, Frisone, Bonvini, Luciano, Pasca, Lepori et al., 2000). Together the studies indicate that premature birth is often associated with speech, language and cognitive delay (Law, 1992). A study comparing matched cases of pre-term births with a cohort of normal 38 week gestation infants, found that the pre-term children (measured at 5 years) had significantly lower IQ quotients than their

matched peers, as well. They also showed particular problems in non-verbal reasoning and simultaneous information processing, and were at increased risk for AD/HD (Johnson, 2007).

Breastfeeding is considered to be a related factor to infant health, and has also been researched as a positive influence in the development of language and cognition. Many studies have found significant and positive associations between breastfeeding and infant intelligence. However, in a meta-analytic review of this literature, the better quality research suggests that this is in fact actually untrue (Anjali, Concato & Leventhal, 2002). In a longitudinal study of pre-school intelligence in New Zealand of over 500 infants, it was concluded that breastfeeding made no significant difference to intelligence, despite a trend for higher intelligence to be associated with breastfeeding for a longer time. In the group of infants who fell within the lowest 10 percentiles and were subsequently classified to be of small gestational age (SGA), it was found that breastfeeding was significantly associated to IQ at age 3.5 years. Further, those that were breastfed for longer than 12 months had on average a 6 point higher IQ than those who were not (Slykerman, Thompson, Becroft, Robinson, Clark, Wild et al., 2005). Another study using nearly ten times the sample size had congruent conclusions to the New Zealand study, suggesting that while there may be many benefits to both mother and child through breastfeeding, intelligence was not one of them. The impact of the practice seems to most benefit infants who need additional nutrition (Der, Batty & Dreary, 2006). In the same study, maternal intelligence was significantly associated with breastfeeding, and was more strongly associated with the nutritional practice than her race, education, age, poverty status, smoking, home environment, child's birthweight or birth order. In this way, maternal intelligence is considered be a pathway to increased intelligence that is often hidden in the association with breastfeeding.

Culturally determined family dynamics play a big role in the interactions and expectations of children according to their birth order within the family hierarchy. Two studies both conclude that birth order significantly influences IQ, with first or second born children tending to have a higher IQ than siblings born later (Boomsma, van Beijsterveld, Beem, Hoekstra, Polderman, Bartels, 2008; Lewis & Jasker, 1983). These studies had sample sizes of approximately 200 and 626 respectively and made use of sound empirical methods. Abdel-Khalek and Lynn (2008) reject these findings and argue that neither family size nor birth order have any significant effect on IQ. This study had a large sample size (N = 4643) and made use of across age-group correlations. Despite these disparities, it was still considered a variable of interest in the current study.

Living with the responsibility of a child with a language, hearing or cognitive impairment is understandably stressful and demanding. Parents and family members have described their lives as being continually draining, living in a constant psychological fear of anticipated misfortune, feeling shame and personal guilt, worrying about what would happen to the child if they passed away and experiencing

community isolation and discrimination (Dhar, 2009). Parents of children with Autism also felt that their interpersonal relationships suffered as a result of their child's limitations. Further, the typical behaviours (mostly anxiety) of children with Autism significantly decreased when parents pro-actively sought to reduce inter-familial conflict (Kelly, Garnett, Attwood & Peterson, 2008). Thus, it is likely that a family which is closely-bonded, with limited conflict, will serve as a social support for both the parents, and child. Thus, the function of social support is considered to be a protective factor for children's functioning and development within the study.

Parental level of education is considered to an important influence in the levels of stimulation, time and resources that are available to small children as their cognitive and linguistic skills undergo critical periods of development. In support of studies that exclusively consider maternal education as a predictor of intelligence, Ginsborg (2006) in her meta-analytic review of this issue, found that intelligence could be correlated with paternal education. She also found that many families (especially those that were socially disadvantaged) are headed by single mothers. These studies concluded that the lower the level of maternal education, the lower the cognitive capacity of the child.

Some components of verbal IQ (such as the appropriate use of tense in language development) are acquired regardless of maternal education (Rice, Wexler & Hershberger, 1998), while others (such as a child's receptive and productive language use, and vocabulary size) were found to increase as the number of years of maternal education increased (Paradise, Dollaghan, Campbell, Feldman, Bernard, Colborn et al., 2000). A local study that measured cognitive ability using the Griffith's Mental Developmental Scales also found that better performance on the General Quotient and Locomotor Scales was correlated with professional mothers. However, years of maternal education did not discriminate infants in terms of their social, fine motor, language, hearing, processing speed or practical reasoning abilities. It was only gross motor ability which was distinguished according to maternal education (Cockcroft, Amod & Soellart, 2008). Other studies have also identified the significant role of maternal education in providing environments in which their pre-schoolers get sufficient sleep. This longitudinal study concluded that children's sleep was related to intellectual ability and later, academic achievement (Buckhalt, El-Sheikh, Keller & Kelly, 2009).

Considering these differentiations from an ecological approach, factors such as the mother's own personality and developmental history, parental knowledge about child development and the level of stimulation in the home are found to influence later child intelligence. Other mediators of maternal education and cognitive development include the nature of interaction between the mother and child, the quality and quantity of child-directed speech, and the language environment (neighbourhood deprivation) (Ginsborg, 2006). Hence, to a large extent, maternal education is heavily related to socioeconomic status (SES) and the relative social disadvantage that low-income families experience.

The SES of the current sample was not considered a predictor in the study as there was no way to accurately measure it from the available information. However, because of its strong overlap with parental

education and its influence on healthy development, it is mentioned here, with the understanding that parental education is as close a proxy variable for it as possible. Despite the high collinearity between maternal education and SES, family income has been found to be a better predictor of global (verbal and non-verbal) IQ than maternal education. More specifically, poverty, which often manifests itself in poor health and nutrition (particularly in the peri-natal period) as well as exposure to environmental pollutants, can set off long-term neurological deficits (Ginsborg, 2006).

It is also unfortunate that children born in circumstances of social disadvantage are almost always continually excluded from opportunities that could improve their development. Living in equally deprived neighbourhoods (with limited access to good early development centres, libraries and health care) they often slip into the intergenerational cycle of social deprivation – unable to break through barriers to inclusion (Bray, 2006; Doherty & Landell, 2006).

Conversely, the presence of sufficient nutrition, caregiver attention, age-appropriate stimulation, health care and security all promote healthy development – affecting arenas of speech and cognition within their respective critical periods (Brooks-Gunn, Klebanov & Duncan, 1996). While some studies suggest that lower socio-economic groups perform poorly on tests of language acquisition (due to the abovementioned factors), it is also more likely that their performance is a function of the structure and biases of the test and parental expectation rather than specific linguistic skill (Law, 1992).

The discussion lastly considers the impacts of early childhood illness, and the development of handedness and their relationships to language development and cognitive performance.

The existence of childhood illness can severely debilitate the typical paths of development in children. An explanation of all of these is impossible, but those from the demographic questionnaire were considered where they were believed to have had an impact on the cognitive performance of the children in the sample. The illnesses experienced by the children in the sample were divided into four main categories, namely; chronic ear infections, chronic upper respiratory infections (URTI's), dysfunction or infection with their brain, heart or lungs and nutritional disorders and problems that was classified as 'failure to thrive'. There is a high degree of overlap between the presence of chronic ear infections and URTI's as the infection usually affects the same system and will result in temporary hearing loss which is believed to be the cause of impaired speech and language development (Law, 1992). These are differentiated in the intake interview, and are categorised as separately, but believed to influence the hearing of the child and general vulnerability to illness. The categorisation of major organ dysfunction and infection also assumes homogeneity in a group that include Meningitis, Encephalitis, temporary heart failure, pleurisy, and collapsed lungs.

While human handedness might seem an unrelated factor to cognitive and language development, it is considered to an early childhood indicator of possibly neurological immaturity in the brain, and is

hence considered as a variable for consideration in this study (Baird, 2008). Human preference for hand usage is hemispherically based, and is believed to develop between the ages of 60 and 70 months as the human brain matures (Coryell, 1985). Within the context of childhood neurological disorder, recent neuroimaging research considers handedness as marker variable for potentially dysfunctional neurological processes in the brain (Dick, Richardson & Saccuman, 2008). Studies have found that, in the event of even minor brain-damage, left-handedness is preferred, suggesting that non-right handedness and cognitive dysfunctions are loosely associated (Bishop, 1990). Scientists have yet to determine the exact reasons behind this, as there are many speculated determinants of cerebral dominance and its subsequent dysfunction. However, there does seem to be sufficient evidence that cerebral abnormality, especially in language impaired populations, is significantly associated with non-right-handedness (Bishop, 1990). A study of 12 cases identified a clinical condition called Pathological Left-Handedness. This is characterised by a left-hemisphere brain lesion that occurs before the age of six, and significantly correlates left-handedness with critical speech deficits in the fronto-temporal/fronto-parietal cortex (Satz, Orsini, Saslow & Henry, 1985). This does not confirm that left-handedness, as such, is *always* associated with poor intellectual performance, as there are other studies which confirm that genetically induced right-hemispheric dominance (causing left-handedness) is frequently associated with *strong* visuo-spatial ability, mathematical strength and musical ability (Aggleton, Kentridge & Good, 1994; Nicholls, Shah & Shields, 2009; Bishop, 1990).

While there is equivocal research about the associations between left-handedness and poor intellectual potential, there do seem to be marked and significant associations between left-handedness and poor performance in children with *existing* diagnoses or impairments (neuromotor problems, learning disorders, AD/HD). Handedness is primarily considered to be a function of laterality, hemispheric dominance and neuro-anatomical functioning and maturity in the brain. Hence, in the case of left or undifferentiated handedness in children with particular impairments, practitioners have reason to believe that neurological functioning is in some way compromised. Increasingly, neuroimaging has highlighted the commonalities between imperfect brain asymmetry, and structural and functional abnormalities in ASD, AD/HD and disorders of language impairment (Bishop, 2008; Dick, Richardson & Saccuman, 2008; Hill, 2001; Rommelse, Atlink, Oosterlann, Buschgens, Buitelaar, Sonneville & Sergeant, 2007). Further, structural imperfections in children with SLI often occur in brain regions responsible for motor function (Dick, Richardson & Saccuman, 2008). While these connections are not always clear, there is a case for the investigation of handedness in a sample predominated by diagnoses of SLI, ASD, AD/HD and motor dysfunction.

In light of the biological and externally located factors that have been found to influence the development of language and cognitive processing abilities in *typical* populations, it is necessary to consider why the understanding of these factors in an *atypical* population is important.

Speech and language problems are some of the most common developmental concerns within the preschool years (Baird, 2008). Therefore, in light of the above predictors of less than optimal development and infant maturation, it is necessary to understand what implications these have for care-giving and future research. The abovementioned diagnostic, peri-natal and early-developmental influences of cognitive and language development give direction and guidance to optimal conditions of early development.

All of the diagnostic criteria mentioned (Autism, intellectual impairment, Language Learning Disability, Specific Language Impairment, Epilepsy, AD/HD) that have associated language and cognitive impairments indicate a strong biological and genetic basis. This implies that parents who themselves, or have family members, who suffer from these conditions should consciously screen for signs of the disorders and their related symptoms as early as possible. In this way, infants and young children can receive intervention and compensatory care and stimulation early, and by so doing reduce the impact of the impairment as much as possible. Researchers and practitioners (e.g. Ruben (1999) and Rosetti (2001)) stress the importance of early symptom identification and intervention in young children. By highlighting and acting on 'delays' and tangents in normal growth patterns as early on in the child's life as possible, developmental trajectories can be shifted so as to achieve the best possible adult outcome. As an example, if parents correctly identify hearing impairment in their infant prior to 8 months of age, cochlear implants can be fitted with such precision and accuracy that the child will manage mainstream schooling with little to no extra assistance (Archbold, Harris, O'Donoghue, Nikolopoulos, White & Richmond, 2008).

The effect of pre-, peri-, and post-natal care is also of great importance in light of the efficacy of early intervention. Because it is established that pregnancy complications, maternal and infant health (physical and mental) and obstetric conditions have a significant impact on the long-term outcome of child development, it is in the best interests of the child to ensure that their in-utero journeys are as healthy and well-managed as possible. Further, in light of the importance of early intervention, mothers whose infants have experienced complications should be aware that there is an increased risk of developmental impairment. Caregivers should be cognisant of this, and make every effort to monitor developmental progress and delay.

From a more ecological and preventative approach, the influence of externally located predictors of impairment should also be considered in light of the efficacy of early intervention. Parental education, lower SES, family structures and degrees of social/familial support have been found in varying degrees and multiple directions to predict aspects of cognitive and language impairment. Therefore, in contexts where parental education, SES and familial support is low, interventions can be directed toward ensuring that cognitive and language impairments are reduced and prevented as much as possible. Examples of such interventions include early screening of developmental delays that highlight signs of possible impairments which ensures early interventions. Programs that educate parents around optimal conditions for healthy

early childhood development (sufficient stimulation, regular health check-ups, and ranges for normal developmental milestones) could be used to ensure optimal maturation (Law, 1992; Rosetti, 2001).

Since the focus of this study is on how the abovementioned factors impact on a child's cognitive development, the nature and assessment of cognitive functioning warrants some attention. While the study and measurement of the often nebulous notion of intelligence has firmly established itself as a keystone of interest within the discipline of psychology, it is not without its share of controversy. Some suggest that there are as many definitions and constructions of intelligence as there are theorists who theorise around it. Herein lies one of its biggest disputes – its definition and subsequent measurement.

Debates regarding the reification of the term and its use in discriminatory practices in the past are beyond the scope of this study. However, it is important to consider that the assessment of cognitive functioning in children has a long history which is not without controversy. Childhood cognitive assessment was formalised with the work of Simon and Binet in 1905. They desired to establish a means by which children with educational difficulties could be identified and consequently receive additional assistance (Kamphaus, 1993). The intention between the measurement of the intellectual capacities of children is very similar within current practice, as they are used to track appropriate child development and identify particular areas of strength and difficulty within the child's range of functioning.

Two of the measures used within the study, the Wechsler Pre-Primary and Primary Scale of Intelligence (WPPSI) and the Junior South African Intelligence Scale (JSAIS) are based on Wechsler's model of intelligence. Wechsler viewed intelligence as a complex interaction of abilities that produce intelligent behaviour (persistence and motivation) that reflects a quality similar to Spearman's 'g'. He separated these tasks into a two factor model of verbal and performance based scales (Deary, 2000). His tests are based on the premise that intelligence is a global entity because it characterises the performance of the individual as a whole, and that it is specific because it is composed of elements or abilities that are distinct from each other. Hence, Wechsler developed subtests that highlighted particular aspects of cognitive functioning, namely abstract reasoning, perceptual organisation, verbal comprehension, quantitative reasoning, memory and processing speed. While there are advantages to measuring cognitive functioning within certain psychometric divisions, Wechsler himself noted that these dichotomies are not representative of neurological literalities or splits, but rather that cognitive functions are frequently interrelated and interact with each other, which makes it very difficult to measure a pure domain of cognitive functioning. Even a very 'simple' category like processing speed requires the individual to discriminate between visual stimuli, process the information and provide a motor response. Conversely, the ecological validity of a *general intelligence* is supported by its strong ability to predict job-performance and psychological well-being, for example, while more discrete domains of cognitive functioning do not show the same degree of predictive ability. Hence, the psychological measurement of pure factors may be useful diagnostically, but do not

always translate into real world knowledge that is useful (Wechsler, 2002; Gardner, Kornhaber & Wake, 1996).

The limitations of intelligence testing persist in their inability to adequately test all domains of cognitive functioning in a meaningful and practical way. Wechsler also recognised the influence of external features such as fatigue and motivation in creating invalid results. Further, related factors such as academic achievement, executive functioning and motor skills also influence performance on intelligence tests. Performance on intelligence tests, therefore, reflects only a portion of what comprises intelligence, which Wechsler defined as the "capacity of the individual to act purposefully, to think rationally and to deal effectively with his environment" (Wechsler, 1944, p.3). In this definition he avoided defining intelligence in purely cognitive terms because he believed that these factors only comprised a portion of 'intelligence'. The results of factor-analytic studies account for only a portion of the overall variance within intelligence, and another group of attributes is believed to contribute to the remaining variance. These could include planning, goal awareness, enthusiasm, field dependence and independence, impulsiveness, anxiety and persistence – all measures which are not directly tapped by standardised measures of intellectual ability. While the Wechsler tests rely on the results of factor analytic studies suggesting the presence of underlying factors, they also consider age appropriate developmental shifts that accompany various stages of development (Wechsler, 2002).

Cognitive assessments are also limited when used to assess children with various developmental concerns, for example, the testing of speech and language impaired children with tests that rely largely on verbal comprehension (Hughes, Sapp & Kohler, 2006). One of this study's primary debates concerning the theoretical assumptions of assessment is the relationship between language acquisition and cognitive ability within pre-schoolers. Intelligence assessments attempt to lay out certain tasks that test particular mental abilities that are similar to the functional, everyday demands of life. However, the tests, their instructions and their underlying assumptions are highly biased towards people who have a degree of verbal understanding and fluency. Therefore, poor performance by a language impaired child on an intelligence test is not a definite and absolute indicator that they have below average cognitive functioning. Assessors are therefore required to carry out 'intelligent' testing and ensure that as far as possible the verbally based nature of tests do not unfairly discriminate against children with verbal deficits.

There are two major retaliations to this argument. The first is that the mental tasks measured by the psychometric subtests, test ability that is expected or even demanded in everyday life. For that reason, some argue that IQ tests are not biased, but instead are an accurate reflection of what is likely to be the child's current functioning in everyday life. By ignoring this translation, the limitations of the child are ignored which sometimes result in unfair decisions around their educational placement and required intervention. The other is that IQ tests have a performance or non-verbal subscale which relies only on verbal instructions (and then the standardised rules of instruction are often adapted to accommodate the special needs of children with speech-language impairments), and most have additional composite scales

that tap into other abilities such as working memory, processing speed and numerical skill. In a study comparing the construct validity of non-verbal subscales of various tests of intelligence, the authors concluded that the construct measured by non-verbal tests of the Leiter International Performance Scale (a test often used within the discipline of speech pathology and audiology) is very similar to the non-verbal scale of the Wechsler Intelligence Scale for Children. Correlations between the two on a sample of 40 children with speech and language impairment was 0.71 which is moderately high (Mean (WISC) = 77.2; Mean (LIPS) = 73.7, SD = 17.2) (Mask & Bowen, 1984). Sattler (1988) also recommends the administration of the Wechsler scales to children with speech and language impairments to allow for a profile of their strengths and weaknesses, but suggests that only the non-verbal scales be considered as an accurate picture of their intellectual potential.

Further, when considering the place of cognitive functioning within the diagnosis of speech and language impairment, there is often an underlying relationship of reciprocity. Some speech and language disorders are purely otomotoric and the speech deficits are the only real hindrance to the child's functioning, while cognitive functioning remains intact. However, for some more severe speech and language impairments that have associated poor cognitive performance, it is often very difficult to tease out whether the poor intellectual functioning is a product of the limits that language deficits have placed on their ability to actively engage and learn from the world, or if they are an 'essentialised' co-morbid feature of the speech-impairment that would remain if the language deficits were somehow corrected. It is also very difficult to know when to give a primary diagnosis of intellectual impairment, and when it is a secondary or associated feature of a primary language deficiency. For example, some genetic disorders such as Angelman's Syndrome and congenital conditions like Cerebral Palsy affect language and speech, but also have very apparent and severe impacts on intellectual potential. While the two might be related in some way, it is likely that they are both individually characteristic of the biological base for the disorder.

While this research does not attempt to clarify these often blurry distinctions, it is important to hold these in mind while considering the identified predictors and associates of cognitive functioning within pre-schoolers.

Three measures of cognitive functioning were used to assess cognitive functioning of the pre-schoolers that formed the sample of the study, namely; the WPPSI, the JSAIS and the Griffiths Scales of Mental Development (GSMD). Research concerning the use of these tests with pre-school populations is considered below.

Psychologists have described the many changes that occur in a child's reasoning from ages five to seven as the five-to-seven-shift. This is relevant to test developers as one of the biggest challenges is to find tasks that are engaging enough to produce a valid assessment of cognitive functioning, yet limit the reliance on reading and writing tasks taught in formal schooling. The developmental period which the WPPSI, JSAIS and Griffiths Scales address encompasses some of the most profound challenges in children's

cognitive ability – most notably a series of qualitative shifts between distinct ways of reasoning. Some researchers reject the strict striation of the Piagetian stages, suggesting that they oversimplify the nature of pre-schooler reasoning, while others such as Siegler (1996) demonstrate that children *simultaneously* show strategies and concepts that are characteristically linked to a variety of developmental stages. In this way, development is understood to reflect a series of overlapping waves, in which one strategy or approach may predominate at a particular time, but not at the exclusion of others (Coalson & Zhu, 2002). Due to the many biological and environmental influences on cognitive development, it is necessary to have an objective and standardised means of its evaluation.

Within the study, most of the children were tested on either the Wechsler Pre-primary and Primary Scale of Intelligence-Revised (WPPSI-R) (*n* = 32) or the WPPSI-III (*n* = 59). For various reasons discussed in Chapter 3, these scores were considered to valid and equivalent representations of the same construct which increased the sample size and consequent statistical power of analyses (*n* = 91). Additionally, some children were tested on the Junior South African Individual Scale (JSAIS) (*n* = 18), and on the Griffiths Scales of Mental Development (*n* = 55). The use of three separate measures of cognitive functioning causes methodological limitations to the study, and a comprehensive discussion of these, as well as the characteristics of these measures is offered in Chapter 3

One of the most obvious limitations in using standardised measures of cognitive assessment with children who have speech and hearing impairments is the tests reliance on verbal responses and understanding. Sattler (1988), however, argues that the WPPSI-III *can* be used to test children with Autism or hearing-impairment, but the subtests that measure verbal and non-verbal language skills separately should be used. Additionally, increased use of visual instructions, non-timed tests, and performance-based tests should be used, as well as special score modifications and standardisations after testing depending on the degree of impairment. Despite many clinicians using the WPPSI-III with children with language and hearing impairment, Sattler (1988) concludes that the subtests that make up the Performance and Processing Speed Composites can be given to children with hearing impairment if they understand the instructions, but that that without a sufficient attempt at performance-based assessment, 'verbal…tests usually do not give an accurate picture of the hearing-impaired child's level of mental ability, but are more likely to measure the extent of language deficiency'(p. 101) (Lichtenberger & Kaufman, 2004; Sattler & Dumont, 2004). This in itself is not a reason *not* to test children with special needs, but simply highlights the importance of understanding the profiling capacity of tests to assess both strengths and weaknesses, rather than their capacity to attribute a numerical score and categorisation of a child as a whole.

Attention is now drawn to the capacity of the WPPSI particularly to assess the cognitive profiles of children with particular disorders which have characteristic speech and language impairment, and are thus relevant to this study.

Children with Autistic Spectrum Disorder (ASD). Coalson & Zhu (2002) expected that children with ASD would perform significantly worse than a matched control on the WPPSI-III. The WPPSI-III was administered to 21 children (aged 3:0 t0 6:11) with ASD. Participants were excluded from the study if they had existing cognitive functioning of more than 2 SD's below the mean (i.e. below 60). The sample was predominantly male (85.7%), which is appropriate given the higher prevalence of ASD in males than females. As predicted, the ASD group scored significantly lower than the matched control, with their PIQ being significantly higher than their VIQ mean. With the exception of Block Design and Object Assembly, all subtests showed significantly lower than average means. The fact that the WPSSI-III revealed poorer scores on nearly all subtests and scales does not necessarily make it an invalid instrument for children with ASD. The fact that it neatly profiles performance and ability within narrowly defined domains of cognitive functioning allows it to constructively measure particular strengths and weaknesses of the child. While it might be a measure of the degree of language deficiency, it also serves as a standardised means of comparing performance across the developmental span (Coalson & Zhu, 2002).

Children with Attention-Deficit/Hyperactivity Disorder (AD/HD). Previous research on the cognitive profiles of children with AD/HD have shown that IQ tests are actually a helpful diagnostic measure as children with the disorder usually achieve scores in the normative ranges for the VIQ, PIQ and FSIQ Indices, and perform poorly on measures of processing speed (Wechsler, 2002). Hence, researchers hypothesised that children with AD/HD would have lower mean scores than matched controls on the PSQ. The WPPSI-III was administered to 41 children aged 3:6-7:3 who had been diagnosed with AD/HD. As expected there were no significant differences between the two groups on the VIQ, PIQ and FSIQ Indices. Surprisingly, there was also no significant difference between their PSQ means either. This is believed to be because the research suggesting poorer PSQ scores within a group of children with AD/HD based their findings on a sample diagnosed with the predominantly inattentive subtype. The effects of this difference were possibly masked within the second study because of the inclusion of children with the hyperactive-impulsive or combined subtypes. While the relative similarity of results between the two groups suggests at a superficial level that the WPPSI-III is suitable and valid for use children with AD/HD, practitioners should bear in mind that almost half of the children in the sample were medicated at the time of testing and so the results could potentially have been skewed by this enhancement (Coalson & Zhu, 2002).

Children with Expressive Language Disorder (ELD). Expressive Language Disorder is believed to elicit substantially lower scores on measures of expressive language than on non-verbal intelligence. The WPPSI-

III was administered to 23 children (aged 4:0 to 6:11) who were identified as having ELD. Children who were totally mute were excluded from the study. The group mean Composite Scale differences between the group with ELD and the matched controls were greatest for the VIQ (0.72) and FSIQ (0.65), with the effect size of the VIQ being greater than that of FSIQ, and the significance of the VIQ being only marginal, and not significant. Within the subtests, Similarities, Word Reasoning and Comprehension were significantly lower than for the matched controls. This is consistent with research suggesting that the vocabularies of children with ELD may normalise by the time they enter school, yet their abilities in tasks requiring verbal reasoning, drawing conclusions and sequential reasoning remain impaired. In this way, the WPPSI-III has been approved as a useful tool in diagnosing language delay and its relative severity. Debates persist regarding whether any measure of intellectual functioning (verbal or non-verbal) is a sufficient and valid measure of cognitive functioning of children with language acquisition or expression delay (Coalson & Zhu, 2002)

Children with Mixed Receptive-Expressive Language Disorder (MRELD). The majority of research on children with MRELD suggests that they have significantly lower mean scores than matched controls on the VIQ and FSIQ. To test this, the WPPSI-III was administered to 27 children aged 4:0 to 7:3 who satisfied the MRELD criteria. Mute children were excluded from the study. As predicted, the VIQ, GLC and FSIQ scales were all significantly lower than those of the matched control. The PIQ and PSQ scores were also significantly lower within the MRELD group, as was performance on all of the subtests, but Block Design, Matrix Reasoning and Object Assembly. These results are consistent with research which confirms that children with language disorders tend to have global deficits in cognitive functioning, yet slightly better performance on their non-verbal scores in comparison to the verbal scale and generally slower reaction times and processing speed (Coalson & Zhu, 2002).

In light of the tests used with special populations, developers of the WPPSI-III acknowledge that while the measure produces results that are consistent with research and theory, its utility is somewhat limited to clinical diagnosis and intervention (Wechsler, 2002). To that end, while it may be a valid measure for testing the cognitive performance of the individual on the day, it is not necessarily a measure of their underlying capacity or potential for cognitive functioning. On the other hand, no measure of cognitive measure can claim to measure an underlying cognitive capacity or potential. The tasks offered are simply a measure or replica of other tasks necessary for daily functioning within the real world. Consequently, cognitive assessments within atypical populations are valid in that they accurately reflect an individual's capacity to accomplish necessary tasks within everyday life.

The Griffiths Scales of Mental Development have been thoroughly researched by a group of researchers in Port Elizabeth (Allan, 1988; Allan, 1992; Bhamjee, 1991; Knoesen, 2003; Luiz, 1997; Heimes,

1995; Sweeney, 1994). Their collective findings promote the use of the Griffiths as a valid and meaningful assessment of the development of young children. While they acknowledge that it has not been normed on a South African sample, it is believed to be a culture-fair test as it is minimally reliant on culturally specific pictures, words and concepts. Differences between British and South African samples are believed to be due to time delays in data collection, and the Flynn effect, and not culturally located advantages (Allan, 1988; Foxcroft, 1985). The Flynn effect is is described as a systematic and considerable rise in intelligence test scores over the world (Resing & Tunteler, 2007).It has been tested on White, Indian and Black South African children, and while it is considered to be a valid instrument, researchers caution against blind interpretation and comparison of the scores as South African infants are generally more advanced than their British counterparts (Allan, 1988; Bhamjee, 1991; Soellart, 2003). The Griffiths has also been evaluated in the South African context as useful tool in the evaluation of pre-schoolers with Borderline intellectual impairment who attended a specialised pre-primary school. It was found to be effective in identifying particular problems, designing as well as re-evaluating appropriate intervention strategies (Houston-McMillan, 1997).

The above discussion has presented research regarding the interdependence of language development and cognitive functioning, identified some of the factors believed to influence language and cognitive development, and briefly examined three measures of cognitive functioning that were used with the sample of the current study. The discussion provided a background to relevant literature and research, as well as a rationale for this study, which is summarised below.

Rationale and Aims

Cognitive development and language acquisition are complex processes, which seem to be related in an interdependent interaction within typically developing populations. While less is known about the actual processes of cognitive functioning and development within atypically developing populations, both logic and research suggest that cognitive functioning would be different in populations that have language impairments. Further, research also shows that cognitive functioning is influenced by more than simply interactive language acquisition, but by external, socio-environmental factors. These include inherent demographic variables (age, language, gender), the diagnosis of particular disorders (Autism, SLI, language delay due to hearing impairment, AD/HD, neuro-motor impairment) and early developmental factors (pre- and peri-natal health, parental education, family structure). Hence, it would be interesting to consider the potential relationships between the demographic, diagnostic and early developmental factors of hearing and language impaired children and their cognitive functioning (as measured by the WPPSI, the JSAIS and the Griffiths). This research therefore aimed to identify the possible predictors of cognitive development within a language-impaired population. It also aims to show that possible differences and similarities

identified in the cognitive functioning of this atypical population refer to broader areas of diagnostic categorisation of childhood impairments.

Research Questions

The abovementioned research aims were operationalized as the following research questions.

1. What are correlations that exist between the independent variables and the subtests and composite scales of the dependent variables (WPPSI, JSAIS and Griffiths)?
2. Which demographic, diagnostic and early developmental factors predict cognitive functioning in children with language-impairment?

3a. Are there differences in cognitive functioning between different diagnoses associated with language impairment (i.e. Autism, AD/HD, congenital conditions)?

3b. Are different diagnoses that are symptomatic of language impairment correlated with higher or lower scores on the measures of cognitive functioning (WPPSI, JSAIS and the Griffiths)?

4. Which factors predict the age of identification?
5. Which factors predict the age of intervention?

Chapter 3: Method

Design.

The study used a non-experimental, exploratory design to investigate the relationship between various factors influencing childhood development and subsequent cognitive functioning. As an archival, retrospective record review of 164 clinical case files of pre-schoolers from a specialised Nursery School, its analysis was quantitative in nature, and makes use of longitudinal and correlational methods of analysis within secondary data analysis. Because the study makes use of ex post-facto research, it does not allow for experimental causality through the conditions of temporal precedence, co-variation and non-spuriousness. Therefore, it is not possible to draw causal conclusions from the results (Rosenthal & Rosnow, 2008). While this is a typical weakness of a non-experimental design, the nature of this particular sample and their difficulties makes it unethical for even the soundest of studies to imply causality. Ex-post facto associations are often the closest research can come to an explanation the nature of disorder and impairment, especially within the context of early development where there are multiple, often unquantifiable, factors of influence.

Participants and Sampling.

The sample consisted of 164 pre-schoolers from a specialised pre-primary school who were administered the WPPSI-R, the WPPSI-III, the JSAIS or the Griffiths Scales of Mental Development as an exit evaluation for school readiness. While the majority of children were tested in their final year at the school (usually age 6 years, and in the oldest class) in order to test their potential for learning in the first year of formal schooling (either Grade 0 or Grade 1), some children were tested earlier at the point of exit from the school. This was sometimes because their needs were best met by specialised education elsewhere, or that they were granted school exclusion due to impairment. The mean age across the sample was 61.38 months (5 years, 1 month), with a standard deviation (*SD*) of 13.44 months, and ranging between 35 (2 years 11 months) and 101 (8 years, 5 months). Children were excluded if they were not assessed using one of the abovementioned measures, and if they left the school after 2008. This is because the structure of the intake interview (used to capture the independent variables) changed in 2009, and it was desirable to maintain uniformity across the sample. Parents of the children granted permission for their confidential information to be used for anonymous research. In this way, informed consent was granted by their admission to the pre-school. Further, the pre-school has been granted institutional permission by the University of the Witwatersrand to conduct archival research. The certificate of ethical approval can be found in Appendix A.

Materials and Instruments.

Due to the archival nature of the study, it was impossible to ensure that all the cases within the sample were assessed using the same measure of cognitive functioning, at the same age, by a group of professionals who shared standards of assessment and diagnosis. Therefore, it was decided that three measures, the WPPSI, the JSAIS and the Griffiths, would be acceptable measures of preschool cognitive functioning.

The Wechsler Pre-school and Primary Intelligence Scale (WPPSI).

The original Wechsler Preschool and Primary Scale of Intelligence (WPPSI) was developed in the 1960's and was aimed at measuring the intellectual functioning of children aged 4:0 to 6:6. The original subtests, listed below in Table 2.1, were retained in the first revision of the test to form what is known as the WPPSI-R.

Table 2.1
Structure of the WPPSI-R

Verbal Scale	Non-Verbal Scale
Information	Geometric Design
Comprehension	Block Design
Arithmetic	Mazes
Vocabulary	Picture Completion
Similarities	Animal House (Optional)
Sentences (optional)	

The subsequent revision to create the WPPSI-III expanded the age-range to test the cognitive functioning of children from ages 2 years, 6 months (2-6) to 7 years, 3 months (7-3). The age range is further divided into two groups, which are referred to as the battery for younger and older children respectively (2-6 to 3-11, and 4-0 to 7-3). Within *all* the versions of the test, factor analytic studies report a two factor model of a Verbal and Performance factor (Coalson & Zhu, 2002). It also has an additional General Language Component (GLC), and for the older group, a Processing Speed Quotient (PSQ). Like most other tests of intelligence, it has a mean score of 100 and a standard deviation of 15. A diagrammatic and tabular description of the subtests can be found in Appendix B (Lichtenberger & Kaufman, 2004). Descriptions of its composite subtests are available in Appendix B.

The revision from the WPPSI-R to the WPPSI-III was undertaken in order to improve the test's psychometric properties, enhance its clinical utility, and improve its developmental appropriateness. Age-banding was introduced to accurately adapt to the changing cognitive processes and abilities of pre-schoolers. By dividing the test into two age-groups (2:6-3:11 and 4:0 to 7:3), the test is better able to measure the complex cognitive processes that rapidly shift and develop across time. For example, children who are three years old will understand basic logical processes, but will be unable to understand exceptions or violations to these rules. Similarly, because of normal lag periods inherent in linguistic and cognitive development, some children are impeded by normal, yet lagging language acquisition. To that end, subtests within the battery for younger children are designed to tap age-appropriate levels of cognitive ability and require little to no verbal expression.

The battery for older children is specifically aimed to target children preparing to enter formal schooling, and is thus of particular interest and relevance to this study. The subtests within this range provide quite specific profiling information about the child's verbal concept formation, abstract and fluid reasoning ability and processing speed. Other more minor additions and alterations to the WPPSI-III from its initial revision, the WPPSI-R, include an increased number of special group studies to enhance clinical utility in atypically developing populations. The WPPSI-III also makes an improved attempt at reducing the amount of expressive language needed to perform by reducing the amount of verbal responses required within subtests of Receptive Vocabulary, Picture Concepts, Similarities and Picture Naming. Its developmental characteristics are specified below (Lichtenberger & Kaufman, 2004).

Standardisation. The WPPSI is a norm-reference test, and is based on a deviation IQ (although this term is not completely true in relation to its original meaning within the Binet Scale) with a mean of 100 and a standard deviation of 15. This means that it measures a child in relation to expected performance levels of a *group* of children the same age. One of the major weaknesses of the point-scale score is that it is difficult when profiling the intellectual abilities of individuals to measure the child's relative delay or advance over time. For that reason, the test also has tables where the raw score for each subtest can be converted into an approximation of the child's mental age. The WPSSI-III was normed on a sample of 1700 American children who were selected to represent the demographic characteristics of age, gender, race, geographic region and parental education of the population in the United states. The sample was categorised into 9 age groups (each spanning approximately 5 months) of 200 children each, and was split equally between boys and girls (Coalson & Zhu, 2002).

Reliability. The WPPSI-III has a very high overall reliability. Its internal consistency (measured using split-half reliability) for the various subtests across age ranges varies between $r = 0.83$ and $r = 0.95$. Most of the reliability coefficients were improved substantially from the WPPSI-R subtests to the WPSI-III. This applies most notably to that of Object Assembly (0.63 to 0.85) and Similarities (o.86 to 0.95). The reliability of the composite scales range from 0.89 to 0.96 and are generally higher than those of smaller subtests. This is because they represent and summarise a broader range of intellectual abilities than can be indicated by the more narrowly defined subtests. The reliability coefficients of the composite scales of the WPPSI-III are nearly identical to those of the VIQ, PIQ and FSIQ of the WPPSI-R.

The WPPSI-III's internal consistency reliability for special groups was obtained using the split-half method from a sample of 395 children with special needs, and includes the diagnostic categories represented in this study. The subtest reliability for most special groups is either higher or very similar to those reported in the typically developing sample, suggesting that the WPPSI-III is equally reliable for assessing individuals with clinical diagnoses which is valuable for the current study.

The WPPSI-III also shows strong test-retest stability. Stability was assessed after an interval of 14 to 50 days, with a mean of 26 days, and the stability coefficients for the composite indices are all good, and range from 0.86 to 0.92 (Coalson & Zhu, 2002).

Validity. The content-validity of the WPPSI-III was obtained by a comprehensive literature review and consulting with expert reviewers who carefully examined the content of the WPPSI-R and proposed new means to improve the content coverage and relevance of the test material.

The convergent and divergent validity of the WPSSI-III was achieved through multiple inter-correlation studies. The presence of moderate to high correlations between all subtests supports the assumed existence of an underlying 'g' factor. The poor correlation between subtests belonging to the Verbal and Processing Speed Indices are also proof of divergent validity (i.e. Information and Coding: r = 0.32).

The construct validity of the WPPSI-III is supported by both exploratory and confirmatory factor-analytic models. For the 2-6 to 3-11 age group, a two factor model, verbal and performance, emerges; while for the older age-group (4-0 to 7-3) the factors of verbal, performance and processing speed (where the GLC is calculated under V-IQ, and PSQ is calculated under P-IQ and both are later included in FS-IQ) are identified to form a three factor model. In addition to the factor analyses, the content-validity of the WPPSI-III is further supported by correlations (ranging from 0.80 to 0.89) with other instruments that measure cognitive functioning, namely the BSID-II (Bayley Scales of Infant Development-II); WPPSI-R; WISC-III and the DAS (Differential Abilities Scale) (Lichtenberger & Kaufman, 2004; Sattler & Dumont, 2004).

During the development of the WPPSI-III, both the WPPSI-R and the WPPSI-III were administered to 176 children aged 3:0 to 7:3, with a testing interval of 8-58 days, and a mean of 28 days. While the mean composite scores on the WPPSI-III were slightly below 100, the researchers believe that this is a function of the sample. A comparison of the mean composite scores shows that those of the WPPSI-R are consistently higher than the WPPSI-III, yet only that of the PIQ is significant (t = 3.54, p <0.001). These gains over time are believed to be due to the Flynn effect, particularly in increases in the performance tests of the Wechsler batteries (Resing & Tunteler, 2007). These gains have been attributed to the increasing complexity of urban society and the accompanying improved nutrition and social influences such as the emphasis on on-the-spot problem solving, the reduction of family size, changes in the interests of children, the importance of and attendance of schooling and increased interest in complex information technology (video games, 'lego', computer games) (Resing & Tunteler, 2007).

Corrected correlation coefficients between the two instruments are 0.86, 0.70 and 0.85 for VIQ, PIQ and FSIQ respectively. The magnitude of the correlations suggests that *the WPPSI-III measures similar constructs as does the WPPSI-R.* Even though practitioners and researchers agree that it is appropriate for cognitive functioning testing on children with hearing and language impairment, it has not been normed on a South African population (Lichternberger & Kaufman, 2004; Sattler & Dumont, 2004).

Consideration is now given to the JSAIS – a South African measure of cognitive functioning that is also based on Wechsler's model of intelligence.

The Junior South African Individual Scale (JSAIS).

The JSAIS Scale was constructed at the request of the Education Department in 1967 to measure the cognitive abilities of younger children. It was designed with the intention of diagnosing cognitive impairment, predicting future scholastic achievement, and providing didactic and therapeutic guidance regarding developmental delays within various domains of the intellect. One of its major functions today is to assess for school readiness, and to develop a profile of differing intellectual abilities for the individual child. Therefore because the scale is not only required to yield a single numerical IQ score, but has utility within diagnosis and prognosis, it contains a number of relatively independent tests which simultaneously tap into an underlying and generalised intelligence (Madge, 1981).

Similar to the WPPSI, the JSAIS Full Scale IQ and the other two composite scales, the VIQ and the PIQ, are based on a point scale or deviation IQ and not an age scale. Mental ages can be calculated by a table that converts a subtest raw score to an expected age of functioning. The test is appropriate for use with children aged 3 to 7 year olds. It has a hierarchical structure, with a main full scale IQ score, and then 2 composite scales of Verbal IQ and Performance IQ. It has two other smaller scales that assess the child's numerical reasoning (Numerical Scale) and his working memory aptitude (Memory Scale) (Madge, 1981). A full description of the tests structure can be found in Appendix C.

The most recent version of those norms is believed to have been done in 1985. Upon consideration of the racial and language demographics of the sample (Table 2.2), the overwhelming majority are white English-speaking people (92.35%), which allows for a degree of relevance to the current sample.

Table 2.2
Home Language of the Standardisation Sample of the JSAIS

		n	*%*	*N*	*Missing Data*
Language	English	145	92.36	157	7
	Other	12	7.64		
Race	White	137	87.82	156	8
	Black	6	3.85		
	Coloured	7	4.49		
	Indian	6	3.85		

Standardisation. The norming of the point scale was done by psychologists, and administered to children aged 3:10 to 8:1 years who were attending pre- and primary school. Every attempt was made to ensure that the sample was representative of the population distribution of the four provinces of South Africa at the time, in terms of urban-rural and socio-economic background. The sample comprised 2000 children, with 200 boys and girls each in the different age groups. The sample represented Afrikaans-speaking and English-speaking children at a ratio 65:35, and there was also a split in the rural-urban divide

(30:70), while sufficient effort went into ensuring population representation across socio-economic status (Madge, 1981).

Reliability. Reliability was tested using the Kuder-Richardson Formula 8 reliability coefficients (r_{tt}), as well as an establishment of the standard errors of measurement (SEm) for each individual test and composite subtest. In general the reliability coefficients range from good (rtt = 0.88) to excellent (rtt = 0.97). Intercorrelations of the subtests and the composite scales show less reliability than that of the WPPSI, in that the correlation of the verbal subtests with each other are only moderate (0.35-0.56), indicating the presence of an underlying convergent inter-reliability. The presence of moderate, yet weaker correlations between verbal subtests and those of performance subtests (0.20 to 0.48), indicates that while the subtests are probably related to one another in some way as they all rely on an underlying 'g', they tap into different domains of cognitive functioning which is indicative of the differences and sometimes moderate correlations between subtests (Madge, 1981).

Validity. The content-validity of the JSAIS was assessed on the basis of an extensive literature review of research pertaining to the topic at hand. They concluded that the tasks are representative of the content within the non-testing environment being measured, and that it has a satisfactory degree of relevance to the construct being measured.

Because it was not possible to determine predictive validity (in terms of academic achievement) as the children were yet to enter formal schooling, teachers were asked to participate in the formation of a concurrent validity measurement. In order to establish criterion related validity. All the correlations between scaled scores and teacher's ratings were significant at p <0.01, with the average varying from 0.55 within the GIQ scale, 0.51 within the VIQ and 0.42 within the PIQ, Memory and Numerical Scales.

Factor-analytic techniques (unrotated Principal Factor Analysis) revealed that there does seem to be an underlying 'g' that underpins performance on all the subtests, and that is possesses strong construct validity. The strong loadings on VIQ and PIQ suggested a two factor model as a next step. Further, the fact that all of the individual subtests showed a loading of not less than 0.3 suggests that a construct validity of each of the individual tests exists (Madge, 1981).

Limitations of the JSAIS.

Because of the high number of subtests within the full JSAIS, the tester is highly vulnerable to scoring and clerical errors. It is also unnecessarily long for older children and while it was designed as an appropriate measure of school readiness, the wide age-range possibly makes the test too easy for the upper age-groups. The limited 'ceiling' of the test prevents the effective evaluation of the older children with very superior intellectual abilities. The test material is also best suited to what Piaget deemed the pre-operational child, and does not sufficiently address the more complex problem-solving strategies that are

possible by a child who has moved into operational thought (usually at age 6 to 7 years), although there are some tests that tap into this ability.

There are also limitations in the interpretation of scores that fall beyond three standard deviations from the mean, and it therefore treads into the murky waters of extrapolation to deduct scores that lie below 50 or beyond 150 (Madge, 1981).

Lastly, the Griffiths Scales of Mental Development are considered as a means of measuring cognitive functioning in children in a way that relies less on crystallised knowledge, and verbal skill than the other two tests.

Griffiths Mental Development Scales.

The Griffith Scales of Mental Development (Griffiths, 1954; 1970; 1984) were developed by the late Dr. Ruth Griffiths, to assess the developmental level of children from birth to two years. These scales are described in Appendix D.

The Griffiths is an age-scaled tests, which allows testers to calculate the number of months delay or advance the child possesses relative to age appropriate norms. Further, a General Quotient (GQ) is calculated by the following formulae to give a general indicator of all-round functioning. An international survey (Luiz, 1997) found that the Griffiths Scales are frequently used to identify problems such as general developmental delay and specific child developmental problems such as delayed speech and language acquisition.

Standardisation. As with all measure of cognitive functioning, reviews of the Griffiths Scales from the 1990's have shown increases in average IQ over time. This phenomenon, known as the Flynn Effect, which is especially evident in non-verbal IQ is noted in the Griffiths as the steadily increasing upward trend of the General Quotient. The norms for the Griffiths were revised most recently in 1996 on a British sample, and represented 1036 children from age 3 to 8 years, who were proportionally representative of gender, SES and urban-rural dwelling (Griffiths, 1996).

Reliability. With the exception of Subscale E for children below the age of 48 months, the Cronbach's alpha coefficients all comfortably exceed the conventional minimum acceptable values of 0.70. The creators of the Griffiths scales warn against the blind interpretation of these scores, as the ways in which the test is administered violates the assumptions underlying the coefficient. This is because the children are presented with increasingly difficult tasks until six consecutive failures occur, so the items are not truly independent. Further, the scales have built in consistency (six items prior to failure must be scored as correct before the sixth is considered as a 'fail'). This inflates the Cronbach alpha, making the Scales appear slightly better than they are (Griffiths, 1996).

Validity. A facet analysis was conducted on each subscale, and the results indicate that the items in each of the six subscales are representative of their respective content domain and that each satisfactorily measures the mentioned construct.

Research into the clinical use of the Griffiths with South African children began primarily in the 1980's and has subsequently received inconsistent research interest from across the country – the most notable from what is now known as the Nelson Mandela Metropolitan University (Port Elizabeth). It has been shown to be an excellent and appropriate measure for the precise identification of developmental weaknesses within children who are believed to have borderline intellectual functioning. This measurement also assists health professions to tailor the programs of various specialised nursery school programs to target these delays (Houston-McMillan, 1997)

The division of the Griffiths Scales into six subscales of development which measure a broad spectrum of activities, allows for a clear picture of individual strengths and weaknesses to be obtained at any stage in the child's development. Clinicians and practitioners who are expert in this profiling also consider the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) to allow for similar inferences and developmental specificities to be drawn. They do however consider the Griffiths to be a superior instrument in that it allows the changes measured during successive evaluations to be easily represented by graphs (Houston-McMillan, 1997). The Griffiths is also a suitable measure to work within settings that relies on the services of multidisciplinary teams as it yields data that is therapeutically workable for them all. The Griffiths also allows for the measurement of mental age (*MA*) and a General Quotient (GQ). It is important to bear in mind that there are potentially many South African children who function below their intellectual potential because of socio-cultural deprivation and understimulation. Because intelligence unused eventually becomes intelligence lost, their early assessment and intervention is critical. One of the most valuable contributions of child assessment is to articulate not only the general or overall development and intellectual potential of the child but to also specify particular areas of strength and weakness at an early (optimally preschool) age (Luiz, 1997). Kyle (1980) further advocates for the use of profile analysis of the subtests within a cognitive test to adequately measure the intelligence of hearing-impaired children.

Independent Variables.

The independent variables were derived mainly from the intake interview and questionnaire with the sample's caregivers upon admission to the school. This was usually around the age of three years. These variables include general demographic characteristics, maternal and infant in-utero information, and early developmental qualities (see Appendix E for attached intake forms). Information that was unavailable from the intake questionnaire was sought within other sections of the file, such as previous medical reports and teacher ratings of development in termly school reports. The fact that a large proportion of the data used

for the study is sourced from maternal report serves to limit the validity of the data. The sometimes underestimation and dishonest reporting of caregivers was evident in one case where the mother denied any injury or neglect in the intake interview, yet the paediatrician reported the child survived a 2m fall out of a window as a baby. In order to compensate for this limitation, as far as possible, the researcher relied upon diagnostic and assessment reports from other therapists, professionals and the child's teachers to ensure congruence in the information covered. Further, clinicians experienced in dealing with maternal report in diagnosing speech and language delays acknowledge that while the exact details of delay are often slightly incorrect, they have accurate insight into other characteristics about the nature of their children's delay (Baird, 2008).

Procedure.

After receiving approval from the University's Ethics Committee (Protocol Number – H100514), the researcher sorted through the case files supplied by the specialised pre-primary school, and those that contained an appropriate assessment were separated and coded. The data obtained from the parental questionnaire, and scholastic and therapeutic reports were captured and coded according to the breakdown in Appendix E in order to obtain a set of independent variables. Due to the overwhelming English-speaking majority, it became obvious that only two language categories were necessary, namely English and other. The alternative category included 8 children who spoke Afrikaans, isiZulu, Hebrew, Portuguese or who were bilingual in these varying combinations.

In order to retain relatively high statistical power, it was necessary to limit the number of categories within each variable (Howell, 1997). Therefore, parental education was narrowed to those not having passed their matric (or year 12 equivalent), those with just a matric, and then those who had studied further.

While the initial birth order was captured as a fraction (i.e. 1 of 3 children), it was later reduced to a binary category of either being the first or second child, or not.

Family attitudes and support were combined to form an indicator out of 16 where a high score indicated good social support.

The types of therapies that the child had received were recorded, but later discarded as a potential predictor as it became impossible to quantify the frequency and quality of intervention. It was also recognised that all of the children in the sample received intensive daily therapeutic input to ameliorate their difficulties.

The allocation of a diagnostic category to a child's difficulty posed serious concern as it was important to retain validity of the data and portray the data in a truthful way. Initially, all the child's diagnoses listed in the file from paediatricians, OT's, psychologists and speech therapists were recorded. Many children's cases reported the presence of a congenital disorder (these included Hydrocephalus,

Fragile X Syndrome, BOR Syndrome, Williams Syndrome, Landau-Kleffner Syndrome, Ehlers Danlos Syndrome, Pierre Robin Syndrome and Prada disorder, Willi Syndrome). While intellectual or language impairment is usually shared by these genetically transferred disorders they were compressed into a single category. Further, most cases reported difficulty with expressive language, and/or, difficulty in receptive or learning language. In the cases where this was specified as being a Language Learning Disorder, this diagnosis was allocated as the primary diagnosis. However, with the other categories (such as the genetic condition, ASD), it became difficult to tease out whether the expressive and receptive language impairments were a primary diagnosis within themselves, or if they were purely symptomatic of an existing condition. In these cases, expressive and receptive language impairments were disregarded and the genetic condition or ASD was prioritised as the primary diagnosis. AD/HD, co-morbid motor and co-morbid anxiety disorders were identified as co-morbid conditions that could co-exist alongside a diagnosis of a genetic condition, ASD or a speech or language impairment. Some children were diagnosed with multiple conditions (often ASD and AD/HD, or a genetic disorder and AD/HD). In this event, they were classified within a category of 'multiple disorders' in order to allow for the presence of more than one condition.

Lastly, when only a speech deficit or a language reception issue was identified, these were classified as existing within a spectrum of Expressive Language Impairment, or with Mixed Receptive and Expressive Language Impairment. This was done for multiple reasons explained in Chapter 2. It is difficult to ascertain precision and certainty within this because neither the child, nor his clinicians were available for consultation.

Pre-pregnancy maternal health was calculated to form an indicator out of six where a high score indicated poor pre-pregnancy health.

Maternal peri-pregnancy health was also calculated as a score out of 23 by combining a series of conditions which are listed in Table E1 in Appendix E, where a high score indicates poor peri-pregnancy health. In an attempt to create uniformity with the pregnancy length item for the sample, cases were given additional points for every week that their pregnancy lay outside of 35 and 42 weeks. Further, they were given an additional point if the length of labour exceeded 20 hours for the first child and 14 hours for subsequent children. This is because children are considered vulnerable, at risk or in potential foetal distress if their timing lies outside of these stipulated criteria (Oakes, 1994). In order to reduce the sub-categorisations within variables, it was not possible to categorise complicated births into those of 'breech', 'cord around the neck', 'placenta previa', 'forceps' and 'suction'. However, cases were allocated a point within this indicator if they had an emergency Caesarian delivery because of foetal distress, or if the baby was in the breech position, had the cord around their neck, or were delivered using either forceps or suction. While there is no guarantee that the child was necessarily damaged by using those two auxiliary methods of delivery, recent studies do not support their use and so they are included because of their contribution to the child's vulnerability to additional stress (Johanson, Wilkinson, Bastible, Ryan, Murphy & O'Brein, 1993).

The infant's post-birth health was also calculated with an indicator out of 14, where a high score indicated poor neonate health. In order to create across-sample uniformity, the condition of 'child at birth', and that 'after birth' was calculated according to the child's Apgar scores.

Many of the cases reported a normal prevalence of childhood diseases such as colds', flu, chicken pox, etc. In the case where chronic otitis media or upper respiratory tract infections were reported they were considered as separate illnesses, although it is likely that these have a high inter-correlation, and could indeed stem from the same underlying cause. Their identified difference would be where the caregiver reported the source of the infection. Some parents reported nutritional problems such as 'failure to thrive' which would indicate a vulnerability to under-nutrition and poor immunity, while others reportedly suffered from severe dysfunctions within their major organs (in all cases, the brain, lungs and heart). Again, detail was lost to accommodate the binary categories, and all these dysfunctions were grouped under one label. They usually included meningitis or encephalitis under the age of three, heart transplants, heart murmurs, needing resuscitation at birth, or major head injuries. The assumed homogeneity of an obviously quite diverse predictor reduced the study's validity in extracting detail from a varied sample.

Developmental milestones were recorded from maternal report, and where possible from occupational therapists (OT) and paediatrician reports. It was very difficult to indicate a precise ceiling limit of these indicators, as they are wholly dependent on the individual child. In the cases where children had *yet* to reach a particular milestone, they were allocated a value beyond that in a normally developing child experiencing an appropriate delay. For example, some children did not babble or speak upon entering the pre-school (at earliest age of three years), and were often recorded as 'doesn't speak' by the caregiver. In this case, children were allocated the following values in order to indicate a delay, yet prevent their exclusion from the study (Owens, 2005), with the understanding that this could have underestimated the severity of their motoric delay:

Babbling: normally achieved at 4 months (ceiling value: 8 months)

Said first words: normally achieved at 12 months (ceiling value of 18 months)

Said first sentences: normally achieved at 18 months (ceiling values at 24 months)

Walking: usually achieved between 12 and 14 months (ceiling value at 18 months)

Toilet-training: varies between 12 and 30 months (mean usually 22 months) (ceiling value of 48 months) (Camurdan, Beyazova, Ilhan & Sahin, 2007; Sears, Maccoby & Levin, 1976).

A ceiling indicator was not given to children who never crawled, as it is developmentally appropriate for children to transition simply from sitting to walking – usually preferring a type of 'shuffle' or 'floor scurry' as a transitional alternative (Case-Smith, 2005).

Further, these values were then all converted to a 'mean' to form an indicator for motor milestone delay, speech milestone delay, and toilet training. While this value does not represent delay in months, it

became a comparative indicator relative to the entire sample, and dis not disadvantage or under-report the development of children who did not crawl.

The child's level of age-appropriate socialisation was calculated by adding the child's reported social abilities to form an indicator out of 22, where a high score displayed poor socialisation. Similarly, a behavioural indicator was calculated in much the same way to form a score out of 23 where a high score was an indicator of poor behaviour.

Handedness was captured from Nursery School reports from the class teacher where possible, or from maternal report in the intake questionnaire, where available.

Lastly, it needs to be re-iterated that, while the study did include the scores of the WPPSI, the JSAIS and the Griffiths, it did not consider them to be equivalent. The IQ deviation scores were considered as measures of the dependent variable for the WPPSI and the JSAIS, and the final quotient for each subscale (calculated as a percentage) was used for the Griffiths.

Threats to Validity.

There were a number of potential threats to validity, although it is difficult to determine their exact impact on the findings of the research.

The small sample sizes for all three of the measures of cognitive functioning affected the types of statistical analyses that could be applied to the data with statistical integrity. To that end, it was necessary to compact some of the categories in order to reduce the possible variability. Examples of this included the eradication of parental profession, or level of post-matric study. In this way people with a three-year diploma were considered to be the same as parents who were specialist physicians or advocates (and had an assumedly much higher earning power). This resulted in a loss of detail, and suggested that the statistical categories represented homogenous groups.

Further, the sample lacks external validity, and its generalizability to other contexts or populations from across South Africa is questionable. This is because the specialised pre-school is located in urban Johannesburg, is privately funded and has a low teacher to pupil ratio, making it probably one of the most expensive Nursery Schools in the country. This is only affordable to a particular demographic, and so the study is possibly only generalizable to urban, English speaking children with similar impairments who come from families in high income brackets.

Further, the fact that the study sought to profile children on not only *three different* measures of cognitive functioning, but that one of these measures is a combination of two revisions (the WPPSI-R and the WPPSI-III), reduces its ability to suggest that cognitive functioning is a containable and definable construct from which various extrapolations can be drawn.

Data Analysis.

Before carrying out the various statistical analyses, it was necessary to determine whether the collected data was suitable for parametric analysis. Random independent sampling was assumed, as was the possession of additive means. The three dependent variables all have an interval scale of measurement. Lastly, it was necessary to examine whether the data was normally distributed. This was done by calculating initial analyses of distribution which indicated normality by histograms, accompanying measures of central tendency and the Kolmogorov-Smirnoff tests for normality (Howell, 1997). Further, within each individual analysis, equality of variance was checked and acted upon accordingly.

Initially, descriptive statistics were calculated on the entire sample, and then again on the different samples categorised by the measure of cognitive function (the WPPSI, the JSAIS and the Griffiths) in order to investigate unique characteristics of the samples.

In order to assess the strength and direction of associations between different variables, both Pearson's and Spearman's correlation coefficients were calculated on all the sample striations.

Where non-normal data was found (the PIQ score of the WPPSI, the PSQ score of the WPPSI, the Personal-Social and General Quotient of the Griffiths), this was normalised using an appropriate transformation. In the case where data was left skewed, the square of the number transformed it to a normal curve, while the application of the square root converted right-skewed data to a normal curve.

It was also appropriate to establish whether significant differences existed between the various composite scales of the dependent variables. This was done in one of two ways. In the case of comparing two normally distributed variables, a two sample t-test was carried out. In the case where the data was not normal, the absolute value of the difference between the two scores was calculated. This was subjected to Wilcoxon's Signed Rank test. The non-normal data was also normalised using the abovementioned transformations and another two-sample t-test to re-test for significant differences. The two methods were employed to satisfy the differing beliefs of varying statisticians. While some believe that a non-parametric test (like that of the Signed Rank) allows for accurate data without changing the meaning of the number, others dispute the perception that the meaning of the transformed values change when converted to normal data.

It was not possible to test for differences between similar composite scales across measures. This was due to the fact that there were only 12 children in the entire sample who were tested on two of the three measures within a reasonably close enough time period and hence subject to cross-test validation. Further, a one-to-one relational comparison of cases was not possible with two-sample t-tests as this depends of the sequence of addition or matching. Alternatively, the testing of difference between means in this incidence is also not possible.

In order to satisfy the research question, a forward stepwise regression, at a confidence level of 95%, was calculated on every composite scale within the three measures. Stepwise regression is a popular

model-building technique by increasing the number of variables one at a time, and testing whether the additional variable contributes a significant degree of explainable variance. Variables are included in the order in which they possess the highest correlation with the dependent variable. After the initial inclusion, semi-partial correlations with the dependent variable are computed, and are added to the model accordingly (Tredoux & Durrheim, 2008). The confidence interval was lowered to 90% within the JSAIS sample in order to compensate for the reduced sample size. In the event that collinearity was high, variables with high degree of overlap (as indicated by the correlation coefficients) were removed in order to explain the most variance. An example of this is that the correlation between maternal and paternal level of education was very positively strong across the sample. In order to reduce this overlap in accounting variance, father's level of education was often removed. Studies promote the use of maternal education level has been identified as a good predictor of cognitive performance in childhood (Cockcroft, Amod & Soellart, 2008; Ginsborg, 2006).

Upon investigation, it also became clear that the age of symptom identification and the age at which intervention begins, continually predicted cognitive performance. However, they predicted it in an unexpected *positive* direction, whereby the later the age of identification ad intervention, the better the cognitive score. Reasons that account for this are discussed later in the report. However, it was decided that because of the strong influence that these two variables hold, that the predictors of these factors would be investigated. Further, because of the high collinearity (strong positive correlations) between them, it was also necessary to determine whether there existed a significant difference between the two. In order to ascertain whether or not the diagnosis given to a child makes a difference, parametric and non-parametric two sample t-tests were calculated, depending on the normality and variance equality present in the various dependent variables.

Because of the large number of independent variables Pallant's rule of thumb (which ensures at least 10 cases for every independent predictor variable) has been followed to retain statistical power for possible regression modelling (Pallant, 2007).

These analyses were used to answer the research questions offered by the study. The findings are presented in the following chapter.

Chapter 4: Results

The results of the study are based on a dataset comprising a variety of demographic and diagnostic factors, information about the child's early developmental history, and their performance on one of three measures of cognitive functioning. Because the study considered potential associations between a highly unique atypical population, it has limited external validity. However, a comprehensive understanding of the sample's characteristics could in itself be valuable. While the study does not have a matched comparison group with regards to developmental history, or measures of cognitive functioning, the body of knowledge concerning development within *typical* populations is broad and thorough, and allows for tentative descriptions and comparisons to be drawn from this study. Tables 3.1 and 3.2 describe the sample's demographics

Table 3.1
Descriptive Statistics for the Entire sample (Categorical Data)

	n	%	N	Missing data
Gender			164	0
Male	115	70.12		
Female	49	29.88		
Home Language			162	2
English	147	90.74		
Other	15	9.26		
Mother Education			146	16
No matric	8	5.48		
Matric	63	43.15		
Post-matric	75	51.37		
Father Education			146	16
No matric	9	6.16		
Matric	57	39.04		
Post-matric	80	54.79		
Family Type			162	2
Married	148	91.36		
Single	14	8.64		
Birth Order				
First or Second Born	148	91.36	162	2
Previous Family History	75	47.77	157	7
Diagnosis			158	6
ASD	22	13.41		
AD/HD	35	21.34		
Epilepsy	10	6.1		
Genetic disorders	14	8.54		
Multiple diagnoses	29	17.68		
Co-morbid motor problems	56	34.15		
Co-morbid anxiety disorders	29	17.68		
ELDS	28	17.72		
MRELDS	77	46.2		
Co-morbid hearing loss	30	22.56	133	31
On medication	36	21.95	18	0
Planned pregnancy	131	80.86	162	2
Breastfeeding	119	76.28	154	8
Childhood Illness				
Chronic ear infection	57	35.19	162	2
Chronic URTI	44	27.16	162	2
Major Organ Problems	25	15.43	162	2
Failure to Thrive	13	8.02	162	2
Handedness			163	1
Left handed	23	14.11		
Right handed	98	60.12		
Undifferentiated handedness	42	25.77		

Tables 3.1 and 3.2 show that the sample of children was largely English-speaking males from nuclear families where the parents were married. Parental education is believed to be closely associated with access to financial resources and social capital (Blumberg & Pfann, 2001), suggesting that the sample lies within a middle to high socio-economic bracket. This is also congruent with the cost of a private pre-primary school that provides an intensive therapeutic programme by trained professionals. Almost half of the sample reported a similar condition within another family member, suggesting that impairment is to some degree heritable. Of note is also the relatively high prevalence of chronic ear infection, and dysfunction of at least one major organ (brain, heart and lungs) prior to age three. This implies *potential* associations between poor childhood health and immunity and later impairment of some sort.

The mean birthweight of the children (usually indicative of poor infant health) seems to be within appropriate ranges. However, it has a very high standard deviation indicating high variability in the sample. Of note are the delayed speech milestones which would be expected within this particular population. Motor and toilet-training means all appear within the age-appropriate range.

Table 3.2
Descriptive Statistics for the Entire Sample (Continuous Data)

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Range*	*N*
Age of sample (months)	61.38	13.44	1.19	35	101	66	127
Age of symptom identification (months)	24.91	12.67	1.02	1	60	59	154
Age of intervention (months)	30.18	13.12	1.06	5	60	55	153
Social support final indicator	11.26	3.07	0.25	1	16	15	157
Pre-pregnancy indicator (6)	1.64	1.38	0.11	0	6	6	164
Peri-pregnancy indicator (23)	3.37	3.24	0.25	0	19	19	164
Neonate health indicator (14)	3.4	2.99	0.23	0	12	12	164
General pregnancy indicator (14)	2.8	1.98	0.15	0	10.33	10.33	164
Birthweight (grams)	3068	648.19	51.9	940	4650	3710	156
Breastfeeding length (months)	6.7	6.38	0.57	0.1	36	35.9	126
Babbled (months)	11.2	6.53	0.54	2	40	38	147
Said first words (months)	19.94	9.09	0.74	6	50	44	150
Said first sentences (months)	31.05	7.85	0.64	12	60	48	149
Final speech indicator	20.79	5.77	0.47	8	41	33	150
Age of sitting (months)	6.91	2.66	0.22	3	30	27	152
Age of walking (months)	14.84	4.58	0.37	9	48	39	154
Final motor indicator (months)	10.5	3.13	0.25	6.67	35.33	28.67	154
Toilet training (months)	35.09	8.18	0.67	16	60	44	151
Developmental delay indicator	22.01	4.18	0.33	12.33	36	23.67	156
Socialisation indicator (22)	6.53	2.99	0.23	0	14	14	164
Behaviour indicator (23)	7.24	3.31	0.27	0	18	18	156

The results for each research question will be reported under each of the three different measures of cognitive functioning, namely the WPPSI, the JSAIS and the Griffiths. This is because they are not strictly comparable means of measurement, as they are structured differently and require completion of different subtests. Despite these differences, there are studies that indicate that these tests *do* indeed measure the same construct.

Firstly, it is possible to theoretically align the constructs measured by the JSAIS and the WPPSI as they are both developed from the Wechsler model of intelligence, and thus have similar subtests, based on a two factor hierarchical model of verbal and non-verbal composites. It is more difficult to align the measured constructs of the JSAIS and WPPSI with the Griffiths as they are constructed on the assumptions of two different models of intelligence. Heimes (1983) investigated whether these disparities could be reconciled, and found that they share construct validity, despite the former being a norm-referenced tests and the other a criterion measured test. Using a sample of 18 girls and 14 boys from a local English speaking, middle class preschool, the researcher divided the sample into two groups and alternated administration of the JSAIS and Griffiths in order to remove experimenter effects. The findings suggest that the two tests indeed measure the same construct, as all but two of the between-test comparisons had a significantly strong, positive correlation ($r > 0.72$). The other two associations between the JSAIS PIQ and the Griffiths Performance scale ($r = 0.68$) and JSAIS Numerical Scale and the Griffiths Practical-Reasoning Subscale ($r = 0.43$) were not strongly related. On the whole, the study suggests that the two are comparable measures. However, despite evidence of validity across the measures, the numerical outputs generated by the scoring procedures are not equivalent and cannot be compared statistically. For this reason, they were separated into three smaller sets of results.

Normality of the Data.

For the purposes of utilising parametric techniques for the analysis, it was necessary to ensure that five assumptions were met. Random independent sampling and the possession of additive means were assumed, while the dependent variables all possessed an interval scale. The normality of the dependent variables was determined using graphical histograms, measures of central tendency and the Kolmogorov-Smirnoff test for normality. (See Table F1, Appendix F). Examination of the histograms and Kolmogorov-Smirnoff test suggest that at least five of the dependent variables were not normally distributed, namely the WPPSI PIQ, PSQ & GLC Indices, and the Performance and General Scales of the Griffiths. To remedy these, all five measures were transformed to normal distributions using appropriate measures,

For the additional research questions which sought to uncover which factors predict age of symptom identification and subsequent intervention, transformations of squaring, square rooting, log's and inversions and reflections did not allow for a normally distributed curve as indicated by Kolmogorov's test. Therefore, upon inspection of the histograms, it was decided to leave the two dependent variables as they were, since they displayed only slight kurtosis. (See Appendix F). Since Kolmogorov's test is extremely sensitive to discrepancies, both variables are considered *sufficiently* normally distributed to warrant parametric analysis. Non-parametric analyses were carried out as well to identify any discrepancies, or confirm significant responses.

Since the three measures of cognitive functioning had both subtle and overt differences, it was decided to present the results obtained on each separately, in the following order: the WPPSI, the JSAIS and The Griffiths.

The Wechsler Pre-primary and Primary Scale of Intelligence (WPPSI).

Tables 3.3 and 3.4 indicate the descriptive statistics of the sample assessed using the WPPSI. Identified descriptive differences within this sub-population could explain differences within later correlations and regressions.

Table 3.3
WPPSI Sample Descriptive Statistics (Categorical Data)

	N	%	Total N	Missing data
Gender			91	0
Male	68	25.27		
Female	23	74.73		
Home Language			90	1
English	88	97.78		
Other	2	2.22		
Mother Education			86	5
No matric	4	4.65		
Matric	35	40.7		
Post-matric	47	54.65		
Father Education			86	5
No matric	4	4.65		
Matric	27	31.4		
Post-matric	55	63.95		
Family Type			91	0
Married	85	93.41		
Single	6	6.59		
Birth Order			91	0
First or Second Born	48	52.75		
Previous Family History	41	47.13	87	4
Diagnosis			91	0
ASD	16	17.58		
AD/HD/ADD	22	24.18		
Epilepsy	4	4.4		
Genetic Disorders	8	8.79		
Multiple Diagnosis	28	30.77		
Co-morbid motor problems	31	34.07		
Co-morbid anxiety disorders	13	14.29		
MRELDS	16	17.98		
ELDS	42	46.67		
Co-morbid hearing loss	11	15.94	69	2
On Medication	22	24.18	91	0
Planned Pregnancy	79	86.81	91	0
Breastfeeding	66	74.16	89	2
Childhood Illnesses				
Chronic ear infection	28	31.11	90	1
Chronic URTI	22	24.44	90	1
Major Organ Problems	15	16.67	90	1
Failure to Thrive	7	7.78	90	1
Handedness			89	2
Left handed	11	12.35		
Right handed	55	61.79		
Undifferentiated handedness	23	25.84		

Table 3.4
WPPSI sample descriptive statistics (continuous data)

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Range*	*N*
Age of sample (months)	68.23	8.16	1.04	48	88	40	61
Age of symptom identification (months)	26.41	12.66	1.36	1	60	59	87
Age of intervention (months)	31.6	12.37	1.33	6	60	54	86
Social support final indicator	11.2	3.25	0.35	1	16	15	87
Pre-pregnancy indicator (6)	1.78	1.44	0.15	0	6	6	91
Peri-pregnancy indicator (23)	3.55	3.03	0.32	0	14	14	91
Neonate health indicator (14)	3.42	3.2	0.34	0	12	12	91
General pregnancy indicator (14)	2.92	1.94	0.2	0	8.33	8.33	91
Birthweight (grams)	3076.78	645.31	68.79	1080	4250	3170	88
Breastfeeding length (months)	6.59	6.4	0.75	0.1	36	35.9	72
Babbled (months)	10.81	5.87	0.63	3	36	33	86
Said first words (months)	19.75	8.27	0.89	6	39	33	87
Said first sentences (months)	31.59	8.46	0.91	12	52	40	86
Final speech indicator	20.76	5.73	0.61	8	41	33	87
Age of sitting (months)	6.72	1.66	0.18	3	12	9	88
Age of walking (months)	14.33	3.01	0.32	10	24	14	89
Final motor indicator (months)	10.17	2.04	0.22	6.67	15.67	9	89
Toilet training (months)	35.57	8.46	0.91	16	60	44	87
Developmental delay indicator	22	3.78	0.4	12.33	31.89	19.56	89
Socialisation indicator (22)	6.35	2.91	0.31	0	14	14	91
Behaviour indicator (23)	7.11	3.41	0.36	1	18	17	89

The WPPSI sample shows similar demographics to the entire group, with a slightly higher age of testing (86.23 months). They are also a largely male, English-speaking sample from educated, married parents. Of note is the high prevalence of ASD within this sample compared to those in the full sample. While the expected speech delays were apparent, generalised motor development was within age-appropriate ranges.

Table 3.5
WPPSI Assessment Descriptive Statistics

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Mode*	*Range*	*N*
Word Reasoning	8.76	2.14	0.47	3	12	8	9	21
Information	9.17	3.2	0.34	2	18	7	16	90
Vocabulary	9.47	3.08	0.32	3	18	8	15	90
Arithmetic	7.99	3.48	0.42	2	17	6	15	70
Sentences	7.41	3.54	0.58	3	19	5	16	37
Receptive Vocab.	7.14	2.32	0.62	3	12	6	9	14
Similarities	10.15	5.29	0.59	3	44	9	41	80
Comprehension	8.1	3.31	0.35	2	19	6	17	87
Picture Naming	8.6	2.35	0.61	5	12	7	7	15
Block Design	9.31	3.43	0.37	1	17	6	16	87
Matrix Reasoning	8.91	3.29	0.69	2	17	9	15	23
Picture Concepts	10.33	2.66	0.54	5	15	9	10	24
Geometric Design	7.58	2.9	0.35	1	13	8	12	67
Mazes	7.52	3.36	0.42	1	15	5	14	65
Symbol Search	7.43	2.17	0.45	4	13	7	9	23
Coding	7.58	2.98	0.61	4	14	6	10	24
Picture Completion	9.65	3.23	0.35	1	17	11	16	83
Animal Pegs	7.41	3.2	0.42	1	14	9	13	59
V-IQ	92.41	17.79	1.87	38	142	98	104	91
P-IQ	90.3	19.4	2.06	11	137	103	126	89
GLC	82.13	22.62	5.84	7	100	91	93	15
PSQ	80.4	24.2	4.84	9	119	75	110	25
FS-IQ	90.01	17.28	1.84	45	134	96	89	88

Note: VIQ = Verbal Scale Intelligence Quotient; PIQ = Performance Scale Intelligence Quotient; GLC = General Language Composite; PSQ = Processing Speed Quotient; FSIQ = Full Scale Intelligence Quotient.

Table 3.5 gives a breakdown of the subtest and composite scale responses of the WPPSI. Because the data was not normally distributed, Wilcoxon's Signed Rank Test was applied to the various composite scales to determine if there were significant differences between them. As can be seen in Table 3.6 significant differences were found to exist between all the composite scales and each other.

Table 3.6
Wilcoxon's Signed Rank Test of difference between composite scales of the WPPSI

	VIQ (*S*)	PIQ (*S*)	PSQ (*S*)	GLC (*S*)	FSIQ (*S*)
VIQ	-	1914***	264***	68***	1785***
PIQ	-	-	125.6***	68***	1785***
PSQ	-	-	-	52.5***	115.5***
GLC	-	-	-	-	60***

Note: *** $p < 0.0001$

A parametric matched pair's t-test was also run on the normalised transformations which confirmed the significance of these results.

Within these subtests of the WPPSI, results are generally within the low average range, with the highest variability existing on the Similarities subtest. The subtests of the Verbal Scale are within the average range, with some subtests comprising the Performance Scale and Processing Speed Index being considerably below those of the VIQ and in the low average range. These include Geometric Design (*M* = 7.58), Mazes (*M* = 7.52), Symbol Search (*M* = 7.43) and Coding (*M* = 7.58).

It is evident that the average performance of the group of children tested on the WPPSI is slightly below the expected means of 10 and 100 for scaled subtest scores and composite scales respectively, and the majority fall within the low average ranges. Of note is that while it is expected that the language based subtests and composite scales would be the poorest performing types given the sample's language impairment, it is actually the subtests relying on processing speed that have faired the poorest. The standard deviations of the various scales is also quite high (up to 24.20), indicating high variability in performance.

In order to explore the relationship between the early developmental factors and the measure of cognitive functioning, correlational analyses were carried out between the VIQ, PIQ, PSQ and GLC composite scales of the WPPSI. It was decided that the correlations with the FSIQ composite scale would be of little real meaning as the score would have been accounted for by its compositional sub-scales. Both the Pearson Product Moment Correlation Coefficients (r) and the Spearman Rank-Order Correlation Coefficient (r_s) (a non-parametric assessment of correlation between two variables) were carried out since the database included both normally and non-normally distributed data. These are shown within Table 3.7 which includes only significant correlations.

Table 3.7
WPPSI Correlation Coefficients

	VIQ (n = 91)	PIQ (n = 89)	GLC (n = 15)	PSQ (n = 25)
Maternal Education	-	-	0.79**	-
Paternal Education	-	-	0.71**	-
Symptom Identification	0.27**	0.36**	-	-
Age of Intervention	0.22*	0.29**	-	-
Epilepsy	-0.24**	-	-	-
Genetic Disorders	-0.25*	-0.21*	-	-0.42*
Multiple Diagnoses	-0.26*	-	-	-
Co-morbid Motor Diagnoses	-	-	0.64*	-
Breastfeeding Length	0.29*	-	-	-
Neonate Health	-0.26*	-0.24*	-	-
General pregnancy indicator	-0.25*	-0.22*	-	-
Motor Delay	-0.24*	-0.34*	-	-
Toilet Training	-0.32**	-0.28**	-	-
Developmental Delay	-0.37**	-0.27*	-	-
Socialisation Indicator	-	-0.22*	-	-
Behaviour Indicator	-	-	-	-
Right Handedness	0.23*	-	-	0.62**
Undifferentiated Handedness	-0.24*	-	-	-

Note: a indicates correlational analysis by Pearson's correlation (r) because these variables did not have a normal distribution; b indicates correlation by Spearman's correlation (r_s).
*p < 0.05. ** p < 0.01

Of interest in these tables are the particularly strong positive correlations between parental education and performance on the GLC ($r_{(s)}$ = 0.79, p <0.05; $r_{(s)}$ = 0.71, p <0.05). This is believed to overestimated due to the small sample size, since subtests comprising the GLC and PSQ are optional subtests and were not administered to the full sample. It is unlikely that professionals would choose to test these in young children who already have difficulties if they are not entirely necessary. Unexpected findings within these correlations are the moderate correlation between the presence of multiple disorders and performance on the GLC (r = 0.64, p < 0.05). This could be a statistical manipulation due to the small sample size. Further, the high number of variables associated with performance on the VIQ and PIQ subscales may have resulted in the weak correlations.

From these correlations, linear regression lines to estimate the role of these variables in predicting the measures were calculated using a forwards elimination model. In this way, factors were entered into the model on the basis of the strength of their correlation with the dependent variable. Subsequent additions were made on the basis of semi-partial correlations with next highest strength with the dependent variable. Predictors were kept in the model on that basis that they explained a significantly different amount of variance in the dependent variables within the context of other variables simultaneously accounting for a part of the variance. The model also allowed for the identification of the amount of variance that each variable is able to explain (indicated by ΔR^2), while the total amount of variance accounted for by the model is indicated by R^2. In order to limit the number of predictor variables within the bounds of the dependent variable's sample size, it was necessary to eliminate predictors that shared a large amount of variance with another. In these instances, predictors that shared variance were identified by their correlation coefficient, and then included or excluded on the basis of theoretical decisions. For example, there was generally a high correlation between maternal and paternal education. By removing paternal education, it allowed maternal education to explain a degree of unshared variance within the dependent variable. Paternal education was removed on the basis that previous research has indicated that maternal education is a stronger predictor of intellectual functioning than that of the father (Ginsborg, 2006). Similarly, there was frequently a high degree of overlap between the existence of a previous family history and the presence of a genetic condition. While it is obvious how these two are related, it was decided to include the genetic condition over the family history as the medical diagnosis provided for a more precise and comparable predictor than that based on maternal report. Space does not allow for a detailed explanation of the procedures with all of the many regressions, which were all carried out in a similar way.

Powerful statistical techniques have similar limitations and assumptions that are difficult to circumvent. The most important assumption of multiple regression is linearity where it is assumed that the

relationship between the predictor and dependent is linear. However, all except three (pre and peri-pregnancy health) of the predictor variables were binary, and hence did not have a directly scatterplot. Secondly, the technique assumes that the residuals (the difference between the actual data points differ from the calculated line) are normally distributed. The F-test which calculated significant explained additional variance is fairly robust in respect to minor violations of normality. Lastly, sample size and the presence of outliers can have profound impacts on the creation of regression lines and should be accounted for accordingly. Within this sample there were no major outliers, and Pallant's rule of thumb was followed with only minor deviations (Tredoux & Durrheim, 2002).

Tables 3.8 to 3.11 display the forward step-wise regression lines which seek to predict the measures of cognitive functioning. Only the variables that accounted for a significant amount of variance in the dependent variable are outlined in the tables and included in the model.

Table 3.8
Regression Tables for WPPSI VIQ (n = 91)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2	C(p)
Step 1							
Gender	16.25	5.07	2440.39	10.27	0 < 0.01	0.19	-2.46
Step 2							
Genetic Disorders	-25.87	10.62	1266.03	5.94	0.02	0.1	-5.23
Step 3							
ASD	-18.31	8.5	910.33	4.64	0.04	0.07	-6.66
Total R^2						0.36	

The significant predictors for the VIQ, namely gender and the presence of a genetic condition of the Autistic Spectrum, account for only 36.46% of the total variance in the measured verbal skills of the sample. This leaves the majority of variance unexplained, and is possibly accounted for by other factors and processes that are not identified within this study.

Table 3.9
Regression Tables for WPPSI PIQ (n = 91)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2	C(p)
Step 1							
Age of Symptom Identification (months)	106.62	37.08	76559602.00	8.27	0.01	0.16	-8.36
Step 2							
Gender	2074.03	967.82	39239891.00	4.59	0.04	0.08	-9.57
Total R^2						0.24	

The variance in the PIQ of the WPPSI is accounted for predominantly by the age of symptom identification and gender. This two-factor model accounts for even less of the variance – only 24.4%.

Table 3.10
Regression Tables for WPPSI PSQ (n = 25)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2	C(p)
Step 1							
Co-morbid Anxiety Disorders	5870.624	1272.73	203721874	21.28	<.0001	0.331	38.0597
Step 2							
ASD	4726.533	1736.623	61729272	7.41	0.0094	0.1003	28.2064
Step 3							
Right	2103.137	850.6465	45411381	6.11	0.0177	0.0738	-6.6628
Total R^2						0.5051	

The model predicting performance in the PSQ accounts for almost half of the variance, yet this is most likely because of the smaller sample size. While the presence of an anxiety disorder accounts for the most variance, the presence of ASD and right handedness account for the rest of the significantly attributable variance.

Table 3.11
Regression Tables for WPPSI GLC (n = 15)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2	C(p)
Step 1							
Maternal Education	20.33	6.24	372.1	10.63	0.01	0.57	38.06
Step 2							
Peri-Pregnancy Health	-1.37	0.45	160.34	9.38	0.02	0.25	28.21
Total R^2						0.82	

The notably smaller sample size within the GLC assessment allows for an increased explanation of variance (81.65%). Maternal Education and the health of the mother-infant dyad during pregnancy significantly account for the variance within these samples.

The Junior South African Intelligence Scale (JSAIS).

The analyses conducted on the WPPSI sample were repeated with the JSAIS sample. Descriptive tables of the categorical and continuous variables within the sample of children assessed using the JSAIS are shown below in Table 3.12 and 3.13.

Table 3.12
JSAIS Sample Descriptive Statistics (Categorical Data)

	n	%	Total N	Missing data
Gender			18	0
Male	9	50		
Female	9	50		
Home Language			18	0
English	15	83.33		
Other	3	16.67		
Mother Coding			15	3
No matric	1	6.67		
Matric	10	66.67		
Post-matric	4	26.67		
Father Coding			14	4
No matric	1	7.14		
Matric	7	50		
Post-matric	6	42.86		
Family Type			18	0
Married	16	88.89		
Not married	2	11.11		
First or Second Born	9	52.94	17	1
Previous Family History	9	52.94	17	1
Diagnosis				
ASD	1	5.56	18	0
AD/HD/ADD	4	22.22	18	0
Epilepsy	3	16.67	18	0
Genetic Disorders	1	5.56	18	0
Multiple Diagnosis	7	38.89	18	0
Co-morbid motor problems	7	38.89	18	0
Co-morbid anxiety disorders	3	16.67	18	0
MRELD	5	25	16	2
ELD	9	56.25	16	2
Co-morbid hearing loss	8	47.06	17	1
On Medication	3	16.67	18	0
Planned Pregnancy	13	76.47	17	1
Breastfeeding	12	75	16	2
Childhood Illnesses			18	0
Chronic ear infection	9	50		
Chronic URTI	4	22.22		
Major Organ Problems	3	16.67		
Failure to Thrive	2	11.11		
Handedness			16	2
Left handed	1	6.25		
Right handed	13	81.25		
Undifferentiated handedness	2	12.5		

Table 3.13
JSAIS Sample Descriptive Statistics (Continuous Data)

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Range*	*Sum*	*N*
Age of sample (months)	69.6	15.4171	3.98067	38	101	63	1044	15
Age of symptom identification (months)	26.88	12.58	3.05	6	48	42	457	17
Age of intervention (months)	31.82	14.93	3.62	6	57	51	541	17
Social support final indicator	10.76	2.75	0.67	5	16	11	183	17
Pre-pregnancy indicator (6)	1.11	1.02	0.24	0	3	3	20	18
Peri-pregnancy indicator (23)	2.28	2.87	0.68	0	11	11	41	18
Neonate health indicator (14)	2.78	2.41	0.57	0	7	7	50	18
General pregnancy indicator (14)	2.06	1.7	0.4	0	5.33	5.333	37	18
Birthweight (grams)	3013.75	539.62	134.9	1480	3700	2220	48220	16
Breastfeeding length (months)	4.49	3.95	1.14	0.5	13	12.5	53.85	12
Babbled (months)	10.4	4.95	1.28	2	24	22	156	15
Said first words (months)	22.4	9.3	2.4	7	36	29	336	15
Said first sentences (months)	30.53	4.81	1.24	18	42	24	458	15
Final speech indicator	21.11	5.53	1.43	10	34	24	316.6667	15
Age of sitting (months)	6.75	1.44	0.36	4	9	5	108	16
Age of walking (months)	14.31	3	0.75	10	20	10	229	16
Final motor indicator (months)	10.2	1.87	0.47	7.67	13.5	5.83	163.17	16
Toilet training (months)	32.88	7.66	1.91	22	48	26	526	16
Developmental delay indicator	21.3	2.73	0.68	16.67	24.44	7.78	340.83	16
Socialisation indicator (22)	5.78	3.24	0.76	0	10.67	10.67	104	18
Behaviour indicator (23)	7.07	3.31	0.85	1	13	12	106	15

Differences between the JSAIS sample and the WPPSI include the formers exactly equal distribution of gender, and lower levels of maternal education (only 26.6% had studied further than matric compared to 54.79% of the WPPSI). It also shows high proportion of children diagnosed as existing on the Receptive and Expressive Language Disorder Spectrum (25%) (17.72% of the WPPSI), and almost half having co-morbid hearing loss (47.06%) (22.56% of the WPPSI) and chronic ear infections (50%) (31.11% of the WPPSI). These shifts in characteristics are believed to be largely due to the reduced sample size of those children measured on the JSAIS. The profiling of the continuous variables reveals a similar picture to other samples where birthweight is appropriate with a high standard deviation, speech delay is apparent and motor co-ordination is within reasonable expectations.

Table 3.14 indicates the response set means on the JSAIS. This is necessary to see if there existed distinctive differences between composites to allow for the development of a profile. Multiple matched pairs t-tests were run on the normally distributed data to see if there were significant differences between the composite subscales. Significant differences were found between the PIQ and VIQ composite scales ($t = 4.64, p < 0.001$), and between Numerical and Memory Scales ($t = -2.40, p < 0.05$). It was not possible to calculate differences between the Memory and Numerical scales and other composites as the former are calculated with a mean of 10 and SD of 2.5, and not 100 and 15, as for the other composite scales. The subtest scores generally lie within the low average to average range, with Block Design and Missing Parts A showing relatively good performances. This is repeated in the average results in the PIQ Scale.

Table 3.14
JSAIS Assessment Descriptive Statistics

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Mode*	*Range*	*N*
Vocabulary	8.06	2.98	0.7	3	13	9	10	18
Ready Knowledge	8.44	2.85	0.67	3	13	8	10	18
Story Memory	7.94	3.04	0.72	3	14	5	11	18
Picture Riddles	8.06	3.26	0.77	4	15	4	11	18
Word Association	6.89	2.54	0.6	2	13	7	11	18
Form Board	11.61	3.65	0.86	7	20	9	13	18
Block Design	12	3.05	0.72	6	19	12	13	18
Missing Parts (A)	11.33	4.12	0.97	4	18	14	14	18
Absurd Situations (B)	9.5	4.05	0.95	4	16	9	12	18
Form Discrimination	10.44	3.31	0.78	2	15	11	13	18
Number & Quantity	8	3.65	0.86	2	14	8	12	18
Memory for Digits	8.75	3.53	0.88	3	18	7	15	16
VIQ	87.94	12.88	3.04	66	109	82	43	18
PIQ	105.67	14.9	3.51	78	129	98	51	18
Numerical Scale	8.13	3.1	0.77	3	16	8	13	16
Memory Scale	9.22	4.21	0.99	3	18	10	15	18
FSIQ	88.44	9.78	2.31	72	105	84	33	18

In order to investigate possible associations between early developmental factors and the measures of cognitive functioning on the JSAIS, Pearson's Product Moment Correlation Coefficients was utilised. There was no need to use non-parametric equivalents as all the dependent variables in the JSAIS were normally distributed. The significant results ($p < 0.05$) of this correlation analyses is shown in Table 3.15.

Table 3.15
Correlations between Developmental Factors and JSAIS Composite Scales

	VIQ (n = 18)	PIQ (n = 18)	Number Scale (n = 16)	Memory Scale (n = 18)
Family Type	0.51*	-	0.60*	-
Social Support	-	-0.53*	-	-
Chronic Ear Infection	-	-0.79**	-	-
Chronic URTI	-	-0.52*	-	-
Socialisation Indicator	-	-0.73**	-	-0.54*
Left Handedness	-	0.50*	0.69**	0.53*

Note. * p < 0.05. ** p < 0.01

Unexpected associations are highlighted in the moderate negative association between social support and performance on the PIQ scale (r = -0.54). Left-handedness was also found to have moderate positive associations with performance on the all but the VIQ subscales. These results, and especially those of the regressions are considered with reservation due to the small sample size explaining a high degree of variance.

In order to further investigate possible predictive models of cognitive performance on the JSAIS, a forward stepwise regression was carried out. It was very difficult to apply Pallant's rule of thumb in this case, but every effort was made to leave out variables with high collinearity. The manual exclusion of

certain variables within this regression does reduce its ability as a valid comparison against the WPPSI. The results are presented in Tables 3.16.

Table 3.16
Regression Tables for JSAIS Composite Scales (n = 18)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2	C(p)
Verbal Scale							
Step 1							
Co-morbid hearing loss	-20.07	8.78	755.01	5.22	0.06	0.47	27.35
Step 2							
Mean Indicator General Pregnancy	4.51	1.76	492.20	6.55	0.05	0.30	16.32
Step 3							
Co-morbid motor problems	12.53	4.81	236.23	6.78	0.06	0.15	8.90
Step 4							
Multiple Diagnosis	-7.86	2.83	100.39	7.71	0.07	0.06	
Total R^2						0.98	
Performance Scale							
Step 1							
Chronic ear infections	-18.11	4.56	1180.84	15.78	0.00	0.55	1.15
Total R^2						0.55	
Numerical Scale							
Step 1							
Social support final indicator	-1.02	0.50	59.73	4.18	0.08	0.34	16.33
Step 2							
Gender	-4.37	2.19	41.45	3.98	0.09	0.24	10.24
Step 3							
Paternal Education	3.00	1.24	36.06	5.87	0.05	0.21	5.1973
Total R^2						0.79	
Memory Scale							
Step 1							
Social support indicator	-1.02	0.50	59.73	4.18	0.08	0.34	16.68
Step 2							
Maternal Education	4.55	1.95	50.05	5.45	0.05	0.29	8.76
Total R^2						0.63	

There is not much similarity between the models predicting performance on the JSAIS and those predicting performance on the WPPSI. The smaller sample sizes of the JSAIS allowed for a greater proportion of total variance to be explained. Within the Verbal Quotient, co-morbid hearing loss (46.52%), general pregnancy health of infant and mother (30.33%), co-morbid motor problems (14.56%) and the existence of multiple diagnoses (6.18%) contribute to a total prediction of 97.59% of the variance in the verbal scores. This is a very good model that explains a lot of the difference in scores. However, this precision is not reflected in the PIQ, where only the presence of chronic ear infections account for a significant proportion of the variance (54.83%). Within the two subordinate Numerical and Memory scales, the predictors accounted for a total of 78.83% and 63.03% of the variance respectively. Within the Numerical Scale, social support (34.31%), gender (23.81%) and paternal education (20.71%) all account for significant degrees of unshared variance, while social support (34.31%), and maternal education (28.75%) are the predictors for the Memory Scale.

It is important to recognise that the large amounts of explained variance proposed by this model is exaggerated by the small sample size, and should be interpreted with caution.

Lastly, consideration is given to the Griffiths Mental Development Scales as the final measure of cognitive functioning.

The Griffiths Scales of Mental Development.

The same statistical procedures that were used with the WPPSI and JSAIS were carried out with the Griffiths. Descriptive statistics of the GSMD are displayed in Table 3.17

Table 3.17
Griffiths Sample Descriptive Statistics (Categorical Data)

	n	%	Total N	Missing data
Gender			55	0
Male	38	69.09		
Female	17	30.91		
Home Language			54	1
English	46	85.19		
Other	8	14.81		
Mother Coding			45	10
No matric	3	6.67		
Matric	18	40		
Post-matric	24	53.33		
Father Coding			46	9
No matric	4	8.7		
Matric	23	50		
Post-matric	19	41.3		
Family Type			53	2
Married	47	88.68		
Not married	6	11.32		
Birth Order				
First or Second Born	27	50	54	1
Previous Family History	25	47.17	53	2
Diagnosis				
ASD	5	9.09	55	0
AD/HD/ADD	9	16.36	55	0
Epilepsy	3	5.45	55	0
Genetic Disorders	5	9.09	55	0
Multiple Diagnosis	10	18.18	55	0
Co-morbid motor problems	18	32.73	55	0
Co-morbid anxiety	13	23.64	55	0
MRELD	10	15.69	51	4
ELD	26	50	52	3
Co-morbid hearing loss	11	23.4	47	8
On Medication	11	20	55	0
Planned Pregnancy	39	72.22	54	1
Breastfeeding	41	80.39	51	4
Childhood Illnesses				
Chronic ear infection	20	37.04	54	1
Chronic URTI	18	33.33	54	1
Major Organ Problems	7	12.96	54	1
Failure to Thrive	4	7.41	54	1
Handedness				
Left handed	8	15.69	51	4
Right handed	28	54.9	51	4
Undifferentiated	17	34.69	49	6

The sample that was assessed using the Griffiths had broadly similar demographic characteristics to those of the other samples, who were largely English-speaking males from highly educated, two parent families. The birthweight and speech delays are as expected within this atypical sample, and only the age of walking (15.96 months) is slightly delayed. Further, the Griffiths sample showed an unusually high incidence of co-morbid anxiety disorders, and has a much younger mean sample age (50.76 months) relative to the JSAIS and WPPSI samples. This is possibly because the Griffiths is more suitable for younger children or those with more severe impairments as its methods are less reliant on verbal fluency and previously learnt information. It is assumed that professionals would have chosen the Griffiths over other methods to establish a more accurate and therapeutically valid profile of younger children's abilities.

Table 3.18
Griffiths Sample Descriptive Statistics (Continuous Data)

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Range*	*N*
Age of sample (months)	50.76	10.8	1.51	35	81	46	51
Age of symptom identification (months)	21.62	12.33	1.74	1	60	59	50
Age of intervention (months)	27.18	13.51	1.91	5	60	55	50
Social support final indicator	11.53	2.9	0.4	5	16	11	53
Pre-pregnancy indicator (6)	1.58	1.36	0.18	0	5	5	55
Peri-pregnancy indicator (23)	3.44	3.65	0.49	0	19	19	55
Neonate health indicator (14)	3.56	2.81	0.38	0	10	10	55
General pregnancy indicator (14)	2.86	2.12	0.29	0	10.33	10.33	55
Birthweight (grams)	3069.4	692.7	96.06	940	4650	3710	52
Breastfeeding length (months)	7.52	6.83	1.05	0.25	24	23.75	42
Babbled (months)	12.17	8.01	1.18	2	40	38	46
Said first words (months)	19.52	10.42	1.5	7	50	43	48
Said first sentences (months)	30.25	7.53	1.09	18	60	42	48
Final speech indicator	20.74	6.02	0.87	12.67	39	26.33	48
Age of sitting (months)	7.31	4.09	0.59	4	30	26	48
Age of crawling (months)	10.01	4.68	0.69	6	36	30	46
Age of walking (months)	15.96	6.76	0.97	9	48	39	49
Final motor indicator (months)	11.19	4.67	0.67	7.17	35.33	28.17	49
Toilet training (months)	34.94	7.86	1.13	18	48	30	48
Developmental delay indicator	22.27	5.16	0.72	13.17	36	22.83	51
Socialisation indicator (22)	7.07	2.99	0.4	0	13	13	55
Behaviour indicator (23)	7.52	3.19	0.44	0	15	15	52

Table 3.18 highlights the relative response profiles on the actual measure. Both the mean mental age and the quotient of each subscale are offered. The mental age is meaningless, unless it is analysed in comparison to other subscales and the general mean age of testing for the sample. What are more comparable are the subscale quotients. Most lie within the low average to borderline range, while only Hearing and Speech and Practical Reasoning are below average. This deficit is expected within the Hearing and Speech skill-set, and the deficits in Practical reasoning are possibly related due to the child's poor communication skills that make it difficult to negotiate social situations and problem solving through a process of verbal reasoning. The standard deviations are within normal limits, indicating limited variation among the sample.

Table 3.19
Griffiths Assessment Descriptive Statistics

	Mean	*Std Dev*	*Std Error*	*Minimum*	*Maximum*	*Mode*	*Range*	*N*
Locomotor (*MA*)	45.5	14.23	1.95	22	84	50	62	53
Locomotor (*%*)	91.81	18.58	2.53	50	133	65	83	54
Personal-Social (*MA*)	43.44	13.14	1.79	20	76	48	56	54
Personal-Social (*%*)	87.69	16.08	2.17	55	140	100	85	55
Hearing & Speech (*MA*)	36.86	14.97	2.08	9	76	40	67	52
Hearing & Speech (*%*)	73.31	20.55	2.82	20	120	89	100	53
Eye-Hand Coordination (*MA*)	40.17	12.42	1.69	18	76	32	58	54
Eye-Hand Coordination (%)	80.54	14.25	1.92	45	107	88	62	55
Performance (*MA*)	42.67	12.48	1.7	10	78	40	68	54
Performance (*%*)	87.52	16.38	2.21	50	125	77	75	55
Practical Reasoning (*MA*)	40.55	15.16	2.12	20	104	26	84	51
Practical Reasoning (*%*)	78.48	16.64	2.31	44	119	68	75	52
General (*MA*)	44.19	17.72	2.43	20	98	31	78	53
General (*%*)	82.73	13.87	1.87	45	112.5	76	67.5	55

Note. MA is Mental Age, % is the scaled quotient.

Multiple matched pairs t-tests were conducted across the six Griffiths Scales to potentially develop a profile of cognitive performance characteristic of the sample, and are shown in Table 3.20.

Table 3.20
Significant differences between subscales

	Locomotor (*t*)	Personal-Social (*t*)	Hearing & Speech (*t*)	Hand-Eye Coordination (*t*)	Performance (*t*)	Practical-Reasoning (*t*)
	M = 91.81	*M* = 87.69	*M* = 73.31	*M* = 80.54	*M* = 87.52	*M* = 78.48
Locomotor	-	-	-6.61***	-5.23***	-	-5.16***
Personal-Social		-	5.27***	-3.71**	-	-3.93***
Hearing & Speech			-	-2.65*	4.36***	-2.38*
Hand-Eye Coordination				-	-3.83***	-
Performance					-	3.37***
Practical-Reasoning						-

Note. *** $p < 0.0001$

The Hearing and Speech subscale had consistently significantly poorer means than all the other subscales. The Hand-Eye Coordination Subscale, an indicator of fine motor control, is also significantly poorer than the Locomotor and Performance Subscales. This possibly indicates generalised fine motor difficulties in this sample. The Practical Reasoning Subscale, responsible for indicating abstract reasoning, working memory and problem solving was significantly poorer than all scales except the Hearing and Speech Scale. While the deficits in verbal skills are expected within this sample, the other weaknesses suggest that children in this sample experience difficulty in fine motor control and higher-order reasoning as well.

To investigate the associations between these subscales and the independent variables, Pearson's Product Moment Correlations Coefficient (r) and a non-parametric equivalent, Spearman's Rank Order

Correlation Coefficient (r_s) were used, since the data sets comprised both normally distributed and non-normal data. These are outlined in Table 3.21.

Table 3.21
Correlations Between the Subscales of the Griffiths

	Locomotor (n = 54)	Personal Social[a] (n = 55)	Eye-Hand (n = 55)	Performance (n = 55)	Practical Reasoning (n = 53)	Hearing & Speech (n = 55)	General[a] (n = 55)
Father Coding	-	-0.32*	-	-	-	-	-
Family Size	-	-	-	-	-	-	0.28*
Previous Family History	-	-	-	-	-	-	0.28*
Anxiety Disorders	0.34*	-	-	-	-	-	-
Multiple Diagnoses	-	-	-	-	-	0.33**	
ELDS	-	-	-0.29*	-0.30*	-	-	-
Motor Delay	-0.37*	-	-	-0.37**	-	-	-0.30*
Pre-pregnancy Indicator	-	-	-	-	0.34*	-	-
Chronic URTI	-	0.31*	-	0.32*	-	-	-
Speech Delay	-	-0.37*	-	-	-	-	-0.29*
Socialisation Indicator	-	-	-	-0.29*	-	-	-
Toilet Training	-	-0.32*	-	-	-	-	-0.36*
Developmental Delay	-	-	-0.35*	-	-	-	-
Undifferentiated Handedness	-	-0.34*	-	-	-	-	-
Left Handedness	-	-	-	0.53*	-	-	-

Note: Spearman's Rank Order Correlation coefficient (r_s) was used to calculate the correlation between the subscales indicated by [a] because were not normally distributed.
Also, * $p < 0.05$, ** $p < 0.01$.

Most of the correlations are of weak to moderate strength. The positive association between family size and General Scale Performance was expected, as were the negative associations between the presence of MRELD, motor delay, speech delay, and undifferentiated handedness and the various subscales. However, other results are somewhat unexpected, such as the presence of an anxiety disorder being strongly related to strong performance on the Locomotor subscale, and that previous family history of a disorder is weakly associated to general performance. Further, poor pre-pregnancy health has a slightly moderate correlation with performance on the Practical Reasoning Subscale ($r = 0.32$), and the presence of Chronic URTI's have a moderate correlation with performance on the Personal-Social subscale ($r_{(s)} = 0.31$). The association between left handedness and the Performance subscale supports the hemispheric localisation of non-verbal activity in the right hemisphere.

These correlations were used as a basis to construct regression models that are able to predict the performance on the Griffiths. (Shown in Table 3.22). Only variables that accounted for a significant degree of variance in the dependent variable were included.

Table 3.22
Regression Tables for Griffiths Subscales (n = 50)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2	C(p)
Personal Social Subscale							
Step 1							
ELDS	-0.79	0.27	4.66	8.46	0.01	0.23	27.3497
Step 2							
MRELD	0.91	0.31	3.77	8.73	0.01	0.19	16.32
Step 3							
Family Type	1.22	0.44	2.73	7.92	0.01	0.14	8.90
Total R^2						0.56	
Eye-hand Subscale							
Step 1							
ELD	-8.63	3.78	968.63	5.23	0.03	0.09	2.00
Total R^2						0.09	
Practical Reasoning Subscale							
Step 1							
Multiple Diagnosis	16.95	6.75	1663.63	6.30	0.02	0.19	-2.44
Step 2							
Age of Symptom Identification (months)	-0.61	0.27	1169.74	5.10	0.03	0.13	-4.14
Step 3							
Birth Order	3.12	1.37	1025.85	5.20	0.03	0.12	-5.39
Total R^2						0.44	
Performance Subscale							
Step 1							
Age of symptom identification (months)	-0.56	0.24	986.08	5.38	0.03	0.16	-5.33
Step 2							
Age of symptom identification (months)	-0.49	0.23	758.33	4.65	0.04	0.12	-6.26
Total R^2						0.28	
Step 1							
Multiple Diagnosis	2088.21	778.18	25582263.00	7.20	0.01	0.20	1.54
Step 2							
Apraxia	-2118.20	611.70	30591536.00	11.99	0.00	0.24	-4.93
Step 3							
Undifferentiated	-1519.88	545.64	15831338.00	7.76	0.01	0.13	-7.31
Total R^2						0.58	

While the Locomotor and Hearing and Speech Subscales included significant correlations, they did not allow for the construction of a regression model. Therefore, the prediction of performance on these scales is not possible. It is likely, that they are predicted by variables and interactions that are not included in this study. The correlation table displays some interesting results. While we would expect some of the more obvious directional associations, it is interesting that previously family history of a similar impairment is positively associated with General performance on the Griffiths .

In general, the models account for a moderate amount of total explained variance. The presence of impairment on the MRELDS and ELDS predicts performance on some of the subscales. The age of symptom identification and intervention are also repeated as predictors on two of the scales. Handedness and the

presence of multiple diagnosis are predictors on single Griffiths scales, but were identified in previous measures (7.38% on the WPPSI, and 6.18% on the JSAIS). Other demographic factors such as birth order and parental marital status are also singular predictors of the measure's subscales. It is interesting to note that not one of the independent predictors could account for a significant amount of variance within the Locomotor and Hearing-Speech subscales, despite there existing a range of 83 and 100 within the quotients respectively.

The Influence of Diagnosis on Cognitive Functioning.

In order to ascertain whether there exist differences in the cognitive functioning of children who have various paediatric diagnosis, multiple two sample-t-tests were calculated between all three measures. Where the dependent variable was not normally distributed, non-parametric t-tests were employed.In the latter cases, parametric tests were also applied to confirm the certainty of such a difference. The equality of variance was tested within every example to determine whether parametric or non-parametric tests were preferable. The results for those categories which revealed significant differences are presented in Table 3.23.

Table 3.23
Diagnostic Differences within Cognitive Functioning (t)

	WPPSI (n = 91)				Griffiths Scales of Mental Development (n = 54)						
	PIQ	VIQ	PSQ	GLC	Locomotor	Personal-Social	Eye-Hand	Performance	Practical-Reasoning	Hearing & Speech	General
Genetic Disorders											
t-value	-1.95* [a]	2.46*	-1.97* [a]	-	-	-1.91* [a]	-	-	-	-	-
Mean	46.57	93.78	14.09	-	-	29.31	-	-	-	-	-
SD	65.33	16.87	11.92	-	-	34.12	-	-	-	-	-
Multiple Diagnoses											
t-value	-	2.51*	-	-	-	-	-	-	-2.29*	-	-
Mean	-	95.44	-	-	-	-	-	-	89.1	-	-
SD	-	17.54	-	-	-	-	-	-	19.71	-	-
ASD											
t-value	-	-	-1.86* [a]	-1.77* [a]	-	-	-	-	-	-	-
Mean	-	-	14.91	9.27	-	-	-	-	-	-	-
SD	-	-	17.11	7.6	-	-	-	-	-	-	-
Co-morbid-motor Diagnoses											
t-value	-	-	-	2.33* [a]	-	-	-	-	-	-	-
Mean	-	-	-	10.92	-	-	-	-	-	-	-
SD	-	-	-	8.57	-	-	-	-	-	-	-
Apraxia											
t-value	-	-	-	-	-	-	-	-	-	-	-1.81* [a]
Mean	-	-	-	-	-	-	-	-	-	-	30.32
SD	-	-	-	-	-	-	-	-	-	-	54.61

Note: [a] indicates that when parametric equivalents were applied, the results were not significant.

Within the different predictors, the means were in favour of the absence of genetic disorder, the absence of multiple diagnoses, the absence of ASD, the presence of co-morbid motor diagnoses and the absence of impairment in the Expressive Language Disorders Spectrum.

* $p < 0.05$

There were no significant differences between diagnostic categories and performance on the JSAIS, which makes across measure comparison for these tests somewhat limited. The validity of these significant differences are also limited by the fact that the study has no comparison against which to measure impairment and cognitive functioning. In essence, differences in one diagnostic category are measured against another group that has different, yet still existent, types of impairment. Generally, these significant differences indicate that cognitive functioning is significantly better in children who do not suffer genetic conditions, and do not have ASD, multiple diagnoses or Expressive Language Disorders. The only counter-intuitive result is the significantly better performance on the GLC by children who *have* co-morbid motor impairment (M = 10.92, SD = 8.57). This might be a skewed result because of the sample size of 15 children in the GLC sample.

The Prediction of Age of Symptom Identification and Subsequent Intervention.

Within previous regressions seeking to predict the influences of cognitive functioning as measured by the WPPSI, JSAIS and Griffiths, the age of symptom identification and intervention continually emerged as giving a good account of the variance. However, within the correlations, the association between these variables and cognitive functioning was in an unexpected *positive* direction, implying that the later a child's symptoms are identified and intervened upon, the better their cognitive functioning. The implicit converse of this relationship is also puzzling and hopefully untrue. While possible explanations for this result are offered in Chapter 5, both correlations and multiple regression models were utilised in attempt to uncover the associates and predictors of the variables of age of identification and intervention.

It would be logical to assume that there is a high likelihood that symptom identification would be followed closely by age of intervention. To establish if a difference existed between these variables, Wilcoxon's non-parametric Signed rank test was used on the non-normal data, and revealed that there was a significant difference across the sample (S = 2093, $p < 0.0001$), and that the variables were separated by a mean of 5.35 months (SD = 6.58 months).

As discussed previously, it was decided that, while both of the dependent variables did not satisfy Kolmogorov-Smirnoff's criteria for normal distribution, an analysis of the histograms and measures of central tendency allowed the deduction that they were *sufficiently* normal to employ parametric regression techniques. A Spearman's Rank Order Correlation was utilised to identify those dependent variables which had significant associations with age of symptom identification or intervention, and the demographic and developmental factors in the study. The results of significant correlations are shown in Table 3.24.

Table 3.24
Age of Symptom Identification (ASID) and Intervention (ASIN) Correlation

	ASID (n = 164)	ASIN (n = 164)
ASID	-	0.84***
ASIN	0.84***	-
Maternal Education	-0.17*	-0.23**
MRELDS	0.19*	-
Genetic Disorders	-0.21*	-0.17*
ELDS	-	0.16*
Major Organ Problems	-0.17*	-0.19*
Co-morbid Hearing Loss	-0.30**	-0.32**
Toilet Training	-0.17*	-0.19*
Undifferentiated Handedness	-	-0.34*

Note. * $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$

There are expected high, positive associations between age of symptom identification and intervention. All of the significant associated variables are expected to exist within the direction that they do and will be discussed later in Chapter 5.

Further, forward step-wise regression modelling was employed to narrow down these relationships. These predictors were excluded in a similar manner to that followed in earlier regression analyses. Only three variables (paternal education, developmental delay, general health during pregnancy) were excluded due to overlapping variance within other variables. These are speech, motor and toilet-training delay, and pre and peri-pregnancy health. These models are outlined in Tables 3.25.

Table 3.25
Regression Tables for Age of Symptom Identification and Intervention (n = 164)

	Parameter Estimate	Standard Error	Type 11 SS	F-Value	Pr>F	ΔR^2
Age of Symptom Identification						
Step 1						
Major Organ Problems	-10.02	3.41	1069.82	8.62	0< 0.01**	0.08
Step 2						
Failure to Thrive	9.02	3.59	639.90	6.31	0.01*	0.05
Step 3						
MRELDS	15.00	6.44	623.90	5.42	0.02*	0.04
Step 4						
Maternal Education	-4.09	1.75	605.05	5.48	0.02*	0.04
Step 5						
Co-morbid hearing loss	-5.83	2.72	551.18	4.59	0.03*	0.04
Step 6						
AD/HD/ADD	4.73	2.25	470.99	4.41	0.04*	0.03
Total R^2						0.28
Age of Intervention						
Step 1						
Paternal Education	-4.82	1.95	861.28	6.12	0.02*	0.16
Step 2						
Co-morbid hearing loss	-7.12	3.02	818.42	5.54	0.02*	0.05
Step 3						
Previous Family History	5.67	2.24	814.33	6.40	0.01*	0.05
Step 4						
Major Organ Problems	-9.71	3.81	758.70	6.49	0.01*	0.05
Step 5						
Chronic Ear Infections	5.91	2.41	806.69	6.02	0.02*	0.00
Step 6						
Multiple Diagnoses	5.69	2.82	501.24	4.06	0.04*	0.03
Total R^2						0.29

Note: ** $p < 0.01$, * $p < 0.05$.

In general, the models do not explain much of the total variance within the dependent variables. The first (age of symptom identification) only accounts for 28.08% of the variance in the age of symptom identification, and the second (age of intervention) shows miniscule improvements in the age of intervention (28.69%). Age of symptom identification seems to be predicted by conditions present early in the child's life, and serve as warning signs for vulnerability to future impairment. These include major organ problems under 36 months (7.59%), failure to thrive (4.53%) and hearing loss (3.90%). These are possibly also affected by the child's expressive language ability (2.19%) and maternal education (2.11%). Age of intervention is predicted by similar variables, and includes previous family history of a similar disorder (5.13%) and the presence of multiple diagnoses (3.15%).

This chapter has presented the statistical analyses employed to answer the research questions. In summation, the results on the various composite scales of the different measures could potentially provide insight into the nature and process of cognitive functioning within this atypical sample. Further, the results from the correlation and regression analyses identify some of the influences of cognitive functioning within the sample. These include the age of symptom identification and intervention, the condition of pregnancy and general childhood health, gender and wider-reaching factors such as parental education and family structure. The high co-morbidity between seemingly different diagnoses suggests the pervasiveness of speech and language impairment in other areas of childhood growth and functioning. Lastly, the high proportion of children with left and undifferentiated handedness within the abovementioned diagnostic groups was interesting. The implications and tentative explanations of these results and their relationship to existing literature are discussed in Chapter 5.

Chapter 5: Discussion

This research investigated the characteristics of an atypical population of pre-schoolers with speech and language impairments, with the intention of understanding potential associations between early childhood factors and cognitive functioning. This chapter discusses and explains the results and locates them in future research and practice.

General Descriptive Differences.

Speech and language impairments have long been known to recur in families. While the heritability of some conditions is obvious through the stark influence of genetic disorders evident in the study, the biological basis of some disorders is less obvious and the presence of a similar condition in a family member can be a good marker that points to the need for early professional intervention. Within the sample, 47.77% of children were reported as having a previous family history of their condition. This is supported by a study similar to the current study (i.e. a sample from a preschool specialising in speech and language impairments), which found that over 40% had a relative with similar problems, with 28% having a parent of sibling with a similar condition (Robinson, 1991).

When considering the performance profiles on the three measures of cognitive functioning, it was initially unclear whether children from the atypical population with a speech or language impairment would show similar performances to typically developing children. In terms of the performances of the children who were tested on the WPPSI, the Picture Naming subtest showed average performance. This is surprising, as the subtest relies heavily on verbal retrieval and long-term memory. These results are supported by those from a study which suggested that picture naming within a language impaired population of children approached that of typically developing children. This is believed to be a result of right-hemispheric compensation on the part of children with language impairment compared to typically developing children (Saccuman et al. 2007 as cited by Dick, Richard & Saccuman, 2008). This capacity for neural plasticity suggests that there is every possibility that children who experience speech and language impairment can compensate for localised functional impairment elsewhere, and develop within appropriate ranges.

Within the Griffiths sample, as expected, children performed significantly poorer on the Hearing and Speech subscale. However, they also had consistently and significantly weaker performances on the Hand-Eye Coordination and the Practical Reasoning subscales. This suggests that these children experience pervasive deficits in abstract, non-verbal reasoning and fine-motor control, as well as the expected difficulties with verbal material. Upon initial inspection, one would assume that children with verbal deficits would rely more heavily on their working memory and short-term non-verbal encoding in order to compensate for their deficits with verbal reasoning. The relationship between working memory and short-term memory within language impaired children has actually attracted much recent research interest

because of its unexpected relationship. Findings suggest that not only do children with language impairment struggle to retain and manipulate verbal information (which is expected), but that their abilities to handle non-verbal information is sometimes poorer than both controls and their verbal skill (Archibald & Gathercole, 2006). One of the key issues is the relationship between language impairment in childhood, and working memory difficulties. Debate persists in establishing the direction of causality between the two, as scholars seek to confirm whether poor memory ability limits language learning or if language capacity is in some way quantitatively linked to the degree and quality of memory function (Gathercole & Baddeley, 1990).

Within this debate, children's manipulation of non-verbal information is considered to be the best indicator for causality. Deficits in memory functioning cannot be attributed wholly to language deficits as the memory limitations of children with language impairment make them less accurate in performing non-verbal visuo-spatial tasks than typically developing children (Bavin, Wilson, Mmaruff &Sleeman, 2005). The results of the current study using the WPPSI, JSAIS and Griffiths, partially agree with these deficits evident in previous research, however the children did not performed poorly on *all* tasks relying on non-verbal manipulation across the subtests. When considering these profiles more broadly, the results of both the WPPSI and the JSAIS samples revealed low-average performances on tasks relying on working memory and short-term storage. While working memory was within average limits, most subtest scores tapping this ability fell within the low average ranges, with standard deviations suggesting that several fell below this range. These are indicated as follows: WPPSI PSQ: *M*= 80.40, *SD* = 24.20, WPPSI Arithmetic: *M*= 7.99, *SD* = 3.48; JSAIS Memory for Digits: *M*= 8.75, *SD* = 3.53; JSAIS Story Memory: *M*= 7.94, *SD* = 3.04. Performances in the low average range are found on the WPPSI subtests of Geometric Design (*M* = 7.58, *SD* = 2.90) *M*azes (*M*= 7.52, *SD* = 3.36), Symbol Search (*M*= 7.43, *SD* = 2.17), Coding (*M*= 7.58, *SD* = 2.98) and Animal Pegs (*M*= 7.41, *SD* = 3.20). The other performance based scores were average or slightly above average. Despite these mixed results, it is evident that the sample experienced a relative weakness in their working memory and short-term retrieval.

The implications for identification and treatment are therefore important within this atypical population. Therapists encourage the promotion of strategies that promote working memory and higher order cognitive tasks. Suggestions include making conscious cognitive attempts to teach encoding in a way that lengthens the 'stay' of information in on the phonological loop, and to switch encoding from auditory encoding to strategic visualisation in order to promote short-term recall. Studies on these strategies show that the adoption of only one strategy (and not both simultaneously) works successfully, particularly with older children and has shown to improve their reading ability. This is possibly because the processing of two strategies overloads the child's already burdened processing capacity. With younger children, such strategies were found to be no more effective than traditional intervention, possibly because of the heavy demands that working memory places on the brain's processing capacity (Dixon, Joffe & Bench, 2001). Luiz (1997) reports that parents and therapists sometimes ignore areas of cognitive functioning (non-verbal, processing-speed skills) that are not overtly associated with speech and language skills in children who

have speech and language impairments. This is often done in an attempt to compensate for their current deficits, but actually serves to stagnate important and appropriately developing skills. In light of the abovementioned connections between working memory and language impairment, it is important for therapists and parents to make an effort not to ignore seemingly 'non-verbal' skill development and stimulation in their attempt to rectify more apparent deficits.

Within the WPPSI profile, performance on the PSQ was consistently poorer than the other three composites (M= 80.40, SD = 24.20 compared to VIQ (M = 92.41, SD = 17.79), PIQ (M = 90.30, SD = 19.40) and GLC (M = 82.13, SD = 22.62)), and was significantly different from the VIQ (S = 126.5, $p < 0.0001$) and PIQ Scales (S = 264, $p < 0.0001$). The PSQ is comprised of the Symbol Search and Coding subtests which rely on working memory, rapid mental manipulation and response to the material. This deficit could be accounted for by the abovementioned difficulties in non-verbal processing. Ottem (2003) conducted a maximum-likelihood confirmatory factor analysis on the WPPSI-III scores of 198 Norwegian language-impaired children (mean age 5.6 years), and concluded that, while the original two factor model proposed by Wechsler sufficiently describes the data, a four factor model which includes the factors of 'processing speed' and 'knowledge dependent' was the most parsimonious for explaining the ideal structure of the WPPSI. However, other studies have indicated that higher-order processing speed deficits are one of the common overlapping symptoms of children who experience SLI and Autism, which suggest that SLI and Autism, maybe overlapping heterogeneous disorders that exist along a continuum (Tager-Flusberg, 2004). In the current study, there was a significant difference between the PSQ and VIQ scores children with ASD (t = -1.87, $p < 0.05$) and MRELDS (t = 1.97, $p < 0.05$), as well as significant differences between the PSQ and PIQ scores of children with AD/HD (t = 10.42, $p < 0.0001$), ASD (t = 2.18, $p < 0.05$) and ELD (t = 7.41, $p < 0.0001$). All the other diagnostic categories, with the exception of the most severely impaired category (genetic conditions) did not have significant differences between their PSQ and other IQ composites. While the results of this study do not indicate sufficient symptom overlap to support the suggestions of Tager-Flusberg (2004), neither do they contradict these ideas. This issue, along with other findings from this research study, is discussed in the final section of this chapter.

A final comment on the cognitive profile specific to children who experienced impairment on the MRELD Spectrum is also necessary. These were children who had difficulty learning (perceiving and interpreting) language and expressing speech. Most of the children in the group were diagnosed as having SLI, but because of the great variation in impairment severity and the need to limit the number of analysed variables (phonological disorder, language delay as a result of deafness) these were condensed to a more generalised category. The diagnostic definition of SLI is very similar to those offered by the DSM-IV for ELD and MRELD, and stipulates the 'delayed acquisition of language skills, occurring in conjunction with normal functioning in intellectual, social-emotional and auditory domains' (McCauley, 2001, p. 114). Additionally, the child must not present with mental retardation, psychiatric problems or neuro-motor impairments,

and must have a non-verbal IQ of above 85. The profile of these children revealed a PIQ mean of 91.69 (*SD* = 10.56) which fits the definition. However, many of these children were diagnosed too early to identify whether they experienced co-morbid psychiatric difficulties, or neuro-motor impairments. Later discussions will also reveal the relatively high proportion of co-morbidity of motor impairment, AD/HD and anxiety disorders within the sample. The presence of these should *diagnostically* exclude the diagnosis of SLI. This is not to suggest that the sample was incorrectly diagnosed, but rather highlights broader concerns regarding the criteria for the diagnosis of SLI as an impairment confirmed by exclusion. This is highlighted amidst much debate regarding the value of the current diagnostic criteria and the controversial decisions which often lie in its wake (Bishop, 1997; Krassowski & Plante, 1997)

The descriptive differences in the age of symptom identification and intervention are now addressed. In the regression models for cognitive performance on the Griffiths and the WPPSI, the variables representing the age of symptom identification and subsequent intervention were identified as explaining a significant degree of variance, despite having weak to moderate correlations with various subscales. However, upon inspection of the correlations between the predictors and the measure of cognitive function, the direction of this association is in the opposite direction (positive) than was expected ($r_{(s)Identification} = 0.26$, $p < 0.01$; $r_{(s)intervention} = 0.23$, $p < 0.01$). Multiple studies promote the benefit and value of early identification through infant screening procedures and timely intervention which provide disadvantaged children with as much compensatory stimulation for their weaknesses as possible. Such interventions have been found to consistently and significantly improve a child's language, academic, and socio-emotional development against a matched comparison group up to 5 years post intervention (n = 40) (Calderon & Naidu, 1998; Gaines & Mussiuna, 2007). Within the current study, it seems that *later* identification and intervention predict better cognitive functioning, while *earlier* intervention seems to predict poorer intellectual functioning. Upon closer inspection, it seems that the two variables of identification and intervention are possibly mediated by the severity of impairment and diagnostic category. Unfortunately, impairment severity was not investigated or recorded as a variable of interest.

However, when looking at the mean ages of identification and intervention in months, it seems that genetic conditions are usually identified much earlier than less severe conditions that tend to manifest only within more structured forms of education (disorders like AD/HD and speech and language impairments). From the list of genetic conditions present in the sample, it is quite likely that other features of the condition (like poor muscle tone, deformed features, cleft palate and poor immunity), and not language impairment, which relies on later detection would lead to early identification and subsequent intervention. To establish whether severity was indeed a mechanism by which timeous identification and intervention occurred, the means of the age at which each occurred are grouped according to diagnosis in Table 4.1 below.

Table 4.1
Mean Age of Symptom Identification and Intervention (Sorted by Diagnosis) in months

	Age of Symptom Identification			Age of Intervention		
	n	*m*	*SD*	*N*	*m*	*SD*
AD/HD/ADD	27	28.11	12.74	27	31.41	11.74
ASD	12	22.83	8.99	12	28.92	7.08
MRELDS	6	19.8	7.82	5	21.4	8.17
ELDS	39	23.6	13.56	39	29.68	14.43
Epilepsy	3	20	12.49	3	28	24.25
Genetic Disorders	7	12.33	9.89	7	21.33	10.71
Multiple Diagnoses	27	25.39	11.61	26	33.93	13.91

An independent t-test indicated that there is a significant difference between both the age of symptom identification and subsequent intervention and the presence of a genetic condition ($t = 2.67$, $p < 0.01$; $t = 2.15$, $p < 0.05$ respectively). No other significant differences between timeous identification and intervention, and the other diagnostic categories were found. The rest of the means are indicative of age-appropriate detection, with ASD, ELDS and MRELDS all identified when children should be speaking (around 24 months), and AD/HD identified as the child enters basic schooling. For the diagnostic categories other than genetics, it was not possible to confirm that severity predicts early identification and intervention as it exists on a spectrum. Hence, this is considered as a tentative explanation for the unexpected results. Another possibility is that impact of early identification and intervention has been minimised within the sample, as all children in the sample attended a specialised pre-primary school from age three years. Thus, their average age of identification and intervention is young in comparison to children who would not have access to formal schooling, assessment and medical intervention so early. It is possible that the children in this sample have benefited as much as they can from early intervention, and the effects may be more evident in samples who only receive intervention slightly later.

Lastly, the fact that genetic or congenital conditions, which are usually more severe, predict poor cognitive functioning may appear deterministic and fatalist, as it seems that regardless of early identification they will generally be behind their peers developmentally. However, the unexpected positive association does not necessarily negate the benefits that early identification can have on children with severe congenital disorders, and might actually promote sound management and normal and functional lifestyles for children and their families who would otherwise have to live with severe disability.

After discussing possible reasons for the unexpected association, attention is turned to the possible predictors of identification and intervention. Knowing the factors that usually precede these two variables could help practitioners and community social policy to better identify and help at-risk families. The regression models for the prediction of the age of symptom identification and intervention yielded results that are explainable within the context of the study. The presence of a previous family history of the disorder, co-morbid hearing loss and major childhood illnesses in infancy (heart, brain or lung dysfunction, chronic infection or nutritional failure) all predicted earlier identification and intervention. This was expected since children with these conditions are expected to have a higher vulnerability to developmental

delay and impairment later on (Lieu, Tye-Murray, Karzon & Piccirillo, 2010). Further, higher levels of parental education were also predictive of earlier intervention. This could be because of two reasons. Research suggests that while the *exact* mechanisms behind associations between maternal intelligence and child development are unknown, there is credibility to suggestions that better educated mothers are better able to recognise and intervene in developmental problems appropriately because they are more connected to public health services (Brooks-Gunn et al., 1994). One study supporting this view found that early enrolment into auxiliary support programs for at-risk toddlers was negatively associated higher with maternal education (Clements, Barfield, Kotelchuck & Wilber, 2008). The other possible reason is that maternal education serves as a proxy indicator for social economic status. It is not possible to equate parental occupation with socio-economic status within the South African context because of the broad-based participation of many parents in the informal sector, but it can be assumed that people who are better educated have increased capacity for higher-income employment, and are therefore more able to provide their children with access to services that best suit their developmental needs.

There were two unexpected results in the correlational associations between age of identification and intervention. These include the weak, yet *positive*, association between the presence of an impairment classified under the Expressive Language Disorder Spectrum (ELD) and the age of intervention. This could be because the developmental milestone for speaking is at about 24 months, and then an age-appropriate lag of a few months is tolerated as representing a 'normal' developmental delay. The deferred intervention could be due to this graduated expectation. Undifferentiated handedness was also *negatively* associated with early identification. This is an unexpected result, since handedness differentiation generally occurs between 60 and 72 months (Coryell, 1985). It is possible that the high associations between undifferentiated handedness and poor performance on measures of cognitive functioning indicates the presence of an underlying impairment. The importance of handedness as an indicator of functional delay and neurological immaturity is discussed again within the context of hemispheric structure and brain laterality.

Attention is now drawn to a discussion of the differences between cognitive functioning and the different diagnostic categories of childhood disorders that existed in the sample. Investigations of potential significant differences between the different diagnostic groups and cognitive performance are congruent with the results discussed up to this point. The sample of children with genetic or congenital conditions showed significant differences between various measures of cognitive ability. From the symptom profile and relative impairments present in these conditions, it is apparent that they have the most severe and generalised impairment, and would hence show the starkest differences in cognitive performance. Unfortunately, the data did not allow for the differentiation of impairment severity as this would have concluded this assumption statistically, and not only theoretically.

The presence of significant differences between children with multiple diagnoses and their relatively poorer cognitive functioning also aligns with theoretical assumptions about the links between

the severity of impairment and the child's relative cognitive potential (McCauley, 2001). The fact that children with multiple diagnoses, or particularly severe conditions like ASD *and* an Expressive Language Disorder, had significantly poorer cognitive performance was expected. The only incongruent result within this set of analyses was the significantly *better* intellectual performance of children with co-morbid motor diagnoses (usually Developmental Coordination Disorder, or a sensory integration difficulty) on the GLC of the WPPSI. This is believed to be a product of the limited methodology of the study, and is not an indicator of an underlying linguistic strength in children with co-morbid motor function impairments. Because of the characteristics of the sample, very few children were administered the GLC (*n* = 18), and so the difference is possibly exacerbated by the quotient's small sample size.

The presence of particular diagnoses was also included within the correlational and regression analyses which sought to identify predictors of cognitive performance. The resulting predictions are relatively congruent with expectations based on the literature review. For example, the presence of ASD is a significant predictor of poor verbal performance on the VIQ of the WPPSI. This is an appropriate relationship as it is well known that children with ASD have particularly poor verbal skills (McCauley, 2001). Further, the pervasiveness of speech and language impairments are again recognised in the significant prediction of cognitive ability by the Personal-Social and Eye-hand Coordination Quotients of the Griffiths within impairments on the Expressive, or Mixed Receptive-Expressive Language Disorder Spectrums. While we would expect these diagnoses to predict poor performance on those tasks very reliant on *verbal* understanding and reasoning, it is less obvious that they would have such a wide-ranging effect on tasks heavily dependent on motor ability. This also points to bigger issues underpinning the neurological basis of speech and language-based diagnosis and motor co-morbidities which will be discussed later.

Pregnancy and Early Childhood Health.

The presence of complications at birth, foetal distress, maternal ill-health and neonate well-being did not predict as much of the variance in cognitive ability as originally expected. These variables only showed significant prediction for the VIQ scale of the JSAIS, and the GLC of the WPPSI. Within the correlation matrices, general health of child and mother during pregnancy was significantly associated with VIQ in a moderate, negative direction. However, general health during pregnancy was also moderately associated with Practical Reasoning Performance on the Griffiths in a *positive* direction. This is an unexpected finding that has no alignment with existing research but makes logical sense. Breastfeeding was significantly associated with strong performance on the VIQ scale, which aligns with research indicating that breastfeeding promotes a good nutritional basis for further learning (Noble & Emmett, 2006; Slykerman et al., 2005). The prediction of cognitive ability by both the GLC of the WPPSI (*n* = 15) and the verbal composite of the JSAIS (*n* = 18) is supported by research suggesting that in-utero conditions, parturition and neonate health are vitally important for the future development of infants (Torrioli, et al., 2000; Weisglas-Kuperus, et al., 1993). Further, these conditions have the potential to have a profound impact upon the verbal ability of children later on, which is confirmed through these two significant predictions.

However, the fact that this result presented in the JSAIS and the GLC, the smallest cohorts within the sample, suggests two things. Either it is not a strong predictor in general within this atypical population and hence only manifests as significant within sample sizes that allow for greater explanation of variance. Alternatively, and possibly linked to the first sample, is that the way in which the indicators of pregnancy well-being were constructed within the database limited their explanatory power relative to other variables. While it should not have mattered that the indicators of health during pregnancy were measured using interval measures and were therefore relative to each other, it is likely that that they had high collinearity with other variables that were better predictors of cognitive functioning, and so were disregarded as unimportant.

The presence of childhood illnesses also possessed moderate explanatory power in relation to other variables in the prediction of intellectual ability. Chronic ear infection was a likely predictor of verbal performance (as in the JSAIS PIQ) as it has been found to be associated with poor language development in children (Law, 1992). While previous research on the relationship between Otitis Media with Effusion (OME) has been equivocal, there have been some studies which suggest a possible link between the two. A study that followed 10 Dutch children matched on their degree of language impairment up to 9 years of age, found that the presence of OME resulted in poorer sensitivity to voicing cues, phonetic identification and auditory perception (Groenen, Crul, Maassen, & van Bon, 1996). Other practitioners have interpreted these and similar findings to suggest that, while OME does not strictly *cause* language and speech impairment, both the ear infection and language impairment are usually confounded by environmental factors. These environmental factors (i.e. low socioeconomic status, poor home stimulation, poor parental availability) might predispose a child to develop OME which then exacerbates their already increased risk for language impairment (Bishop, 2008; Paradise et al., 2000). While it is believed that children in the current sample belonged to high SES brackets, there are a number of as yet unidentified predisposing factors that could have led to their vulnerability (Roberts, Rosenfel & Zeisel, 2004).

Within the regression models for the JSAIS and the WPPSI, male gender significantly predicted good performance on the JSAIS Numerical Scale and both the PIQ and VIQ scales of the WPPSI. These results are confirmed by special population validity studies done by the WPPSI-III developers and other researchers, who found that, while the majority of children with speech and hearing impairments are male, they show less cognitive impairment than their female counterparts (Baird, 2008; Tomblin, Records, Buckwalter, Zhang, Smith & O'Brein, 1997; Wechsler, 2002). The over-representation of males within the regression analyses could also be due to the relatively small sample sizes, especially within the JSAIS cohort.

Parental Education was found to predict strong performance on the GLC of the WPPSI, the Numerical and Memory Scales of the JSAIS, and the Personal-Social subscale of the Griffiths. Further, the presence of a nuclear family with two parents was found to predict high achievement on the VIQ of the JSAIS and the Personal-Social subscale of the Griffiths, and was significantly associated with performance

on the JSAIS Numerical Scale (r = 0.60). The older the child relative to other siblings (a function of birth order), was also found to predict strong performance on the Practical-Reasoning subscale of the Griffiths. Parental level of education and the existence of a nuclear family are considered as the best possible proxy variables for SES, such as a degree of financial support and resources, parental attention and social capital available to the child (Doherty & Landell, 2006; Fish & Pinkerman, 2003). These are believed to be more broad-spectrum determinants of cognitive function that were not possible to measure in the study. It was assumed that a two-parent family would have the greater potential for a higher income as opposed to a single parent family. The increased availability of financial resources often translates into more frequent, and better quality therapeutic intervention and opportunity for stimulation. This increased financial support could also allow parents to offer their children increased contact time and attention with their children, which is an indicator of better stimulation and opportunity for learning. This is contrasted with single parent families who potentially have less time with their children, and by circumstance may not be able to offer them the same degree of quality care and stimulation. This is supported by research suggesting that nuclear families with two parents are better able to provide a stimulating and educationally enhancing environment for young children within periods of particular vulnerability (Volonté, 2010; Wise, 2003). However, there is research that disputes the importance of 'whole' families in improving pre-school IQ, but, this research self-admittedly concluded that spousal self-report measures of the quality of their relationships were actually contradictory to what psychometric evaluations thereof found, and that there was serious limitation to degree of variance available from spousal report (Poresky & Whitsitt, 2001). Other studies are congruent with the current research, and state that maternal education level consistently predicts verbal performance in pre-schoolers because of the assumed increased knowledge of the requirements for childhood stimulation and nutrition and their increased socio-economic status which often increases access to resources generally (Poresky & Whitsitt, 2001; Wachs & McCabe, 2001). It is important to recognise that the precise mechanisms by which maternal education impacts on the child's intelligence are not fully understood. In fact, current cross-cultural research supports the possibility that *different* mechanisms exist in different contexts, and that it is difficult to generalise even the strongest studies with strong external validity to other populations (Der, Batty & Dreary, 2006).

The Pervasiveness of Speech and Language Impairment.

Poor performance on the various cognitive subtests was significantly associated with delays in motor milestones, toilet training, poor socialisation and generally poor behaviour relative to age-appropriate expectations. While it is well established, and perhaps expected, that children with speech and language impairments will experience problems in terms of their capacity to make use of age-appropriate socialisation and exhibit generally poor behaviour (often an indicator of an etiological diagnosis like ASD that explains the speech and language impairment), the co-morbid motor and toilet-training delays are possibly less well known (Noterdaeme & Amorosa, 1999; Snowling et al., 2006). Reasons for these relations come from structural neuroscience which, through studies of adult aphasia, suggests that speech and

language skills are highly localised within particular regions of the brain. Due to the high co-morbidity of poor motor control and development in children with speech and language impairments, recent research has sought to understand the possible links between the two (Archibald & Alloway, 2008). Contrary to widely held beliefs, co-morbid motor deficits may *not* be a product of poor stimulation and limited educational opportunity as a result of a speech/language learning disorder. This hypothesis has been generated on the grounds that in previous studies, co-morbid motor problems were only identified much later in the children's lives once they were in an academic environment, and were required to undertake increasingly more independent tasks of self-care. It was assumed that because of their later identification, the motor deficits were a *consequence* and not a simultaneous symptom of poor quality educational opportunities and parent engagement as a result of an existing speech impairment. Instead, they suggest that there may exist common cerebellar deficits, inter-hemispheric deficits, and patterns of atypical brain development that affect both speech and language development and motor function simultaneously (Estil, Whiting, Sigmundsson & Vnglaldsen, 2003; Gaines & Missiuna, 2006; Kaplan, Wilson, Dewey & Crawford, 1998). These shared etiologies and proposed overlaps leads to consideration of the role of handedness and diagnostic profiling within the sample's results.

Handedness is believed to be an indicator of cerebral dominance, and is developmentally expected to differentiate between the age of 60 and 72 months (Coryell, 1985). Most children have established at least partial dominance between the ages of 3 and 4 years, but it is not uncommon to find a degree of ambidexterity within older children. Within the sample of this study (mean age is 61.38 months), right-handedness dominated (60.12%), followed by differentiated left-handedness (14.11%) and undifferentiated handedness (25.77%). Most of the global population are right-handed, yet it is surprising to see such a large proportion of undifferentiated handedness. Handedness was repeatedly identified as a significant predictor or associate of cognitive functioning within the sample. Within the cohort assessed by the WPPSI, right handedness was a significant predictor of performance within the PSQ composite scale, and was significantly associated with good performance on the VIQ and PSQ scales. Undifferentiated handedness was also significantly associated with poor performance on the VIQ scale, and on the Personal-Social Subscale of the Griffiths. Left handedness, on the other hand, was significantly associated with *strong* performance on the PIQ, Numerical and Memory Subscales of the JSAIS and on the Performance subscale of the Griffiths. The latter results are congruent with research that suggests that a genetically induced right-hemispheric dominance (which causes left-handedness) is frequently associated with strong visuo-spatial ability, mathematical strength and musical giftedness (Aggleton, Kentridge & Good, 1994; Nicholls, Shah & Shields, 2009)

The abovementioned results are surrounded by quite equivocal research and have interesting implications in terms of neurodevelopmental disorders that become apparent in childhood. Recently, Levy's theory (1972) which proposed that left-handedness predicted spatial inferiority, as well as the work

of Palmer and Clark (1963) which indicated that mental instability was associated with left-handedness, has been widely criticised, and has been disproven. Studies comparing the spatial abilities, visual motor performance, reading ability, visuo-spatial lateralisation and verbal comprehension between left and right handedness, all indicate that there exist no significant differences between differentiated left- or right-handedness and cognitive performance (Faurie, Vianey-Laud & Raymond, 2006; Rommelse, et al, 2007; Wellman, 1983; Wellman, 1985). However, children who fall within the lower intellectual ranges, or have existing diagnoses of motor impairments, learning disorders, AD/HD or language impairment, appear to show significant differences between their cognitive performance and the presence of left-handedness (Ferrari, 2007; Natsopoulos, Kiosseoglou, Aphrodite, 2004; Rommelse et al., 2007). Further, these studies show robust evidence that left-handed (and mixed-handed) children performed significantly worse on nearly all measures of development than right-handed children, and cannot explain these differentials in terms of different socioeconomic characteristics of the household, parental attitudes, or investments in learning resources (Johnston, Nicholls, Shah & Shields, 2009).

Moreover, the poor performance predicted by undifferentiated handedness within the current study is a topic that has recently proven useful in understanding neurological processes, and the subsequent pathologies that arise from immature neurological development or dysfunction. While the human brain is both structurally and functionally asymmetrical, there is some connection between imperfect brain asymmetry, language development and handedness. This is since differences in brain lateralisation are reliably identified through right-, left- and mixed-handedness – although the specifics of prediction have not yet been definitely ascertained. Further, atypical laterality and undifferentiated handedness have been associated broadly with language impairment in children and various psychopathologies in adulthood (Rodriguez & Waldenstrom, 2008). Left, and undifferentiated handedness is therefore considered to be a variable marker for underlying neurological dysfunction (Ferrari, 2007). The relationship between handedness and neurological immaturity is now considered within broader profiling patterns found within the descriptive statistics in the current study.

The following and final section reflects on results that were not overtly or intentionally sought from the study. However, upon inspection of the varying co-morbidities between diagnostic groups and their common cognitive profiles, it was decided to investigate these within existing research. The reader is advised to consider the results of this study and their links to neurological research with caution, as the results only tentatively support existing and growing knowledge on the subject.

Brain Laterality and Childhood Impairment.

As the understanding of pathology transcends into the realm of neuroscience, it is important to consider the role of brain abnormality and neurological dysfunction as an aetiology or transitory explanation for speech and language impairment, especially in light of the connections between handedness, brain

laterality and cognitive performance that were made within the current sample. Few studies have managed to include multiple groups (children with SLI, AD/HD, Autism and motor impairments) in their research, due to the difficulties in finding relatively homogenous groups and the limited sample sizes that are usually too small to conduct statistically sound analyses. Conclusions about such groups and their common features and etiologies are usually drawn across different studies, and researchers later attempt to highlight similarities between them.

The current research study allowed for a descriptive examination of the cognitive profiles of children whose diagnoses span these categories, and hence allowed for tentative conclusions to be drawn. There are two findings from the study that are aligned to recent neuroscience research and may account for the frequently overlapping symptoms displayed in children with SLI (referred to in this study under the category of MRELD and ELD Spectrum), ASD, AD/HD and motor impairment. Firstly, an analysis of the cognitive profiles of children meeting these diagnostic criteria suggests that children across these groups performed poorly in the *same* subtests. Moreover, the poor performance was concentrated in subtests that surprisingly *do not* rely on verbal reasoning, word recognition or semantic understanding, but occurred within subtests that rely on processing capacity, working memory (both verbal and visuo-spatial), spatial orientation, auditory processing and perceptual organisation. Because the sample had no comparison group it was not possible to identify whether these non-verbal weaknesses were significantly different from typically developing children. Therefore a face-value comparison is one of the best possible means to get a general idea of the profiling. Table 4.2 represents these values.

Table 4.2
Mean Comparison of Subtests According to Diagnosis

	ASD		AD/HD		Motor Impairment		MRELDS		ELDS	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Word Reasoning	9.38	1.60	8.75	2.50	9.56	1.74	9.00	1.41	9.38	1.60
Information	9.43	3.31	8.23	3.22	9.23	2.68	8.44	1.67	9.43	3.31
Vocabulary	9.31	2.80	9.05	2.82	9.29	2.71	8.88	3.16	9.31	2.80
Arithmetic	7.44	3.33	6.47	2.50	7.08	2.76	8.08	2.75	7.44	3.33
Sentences	7.00	3.19	6.80	3.79	8.08	3.92	7.40	2.51	7.00	3.19
Receptive Vocabulary	7.17	1.17	7.00	4.58	8.71	1.98	7.50	0.71	7.17	1.17
Picture Naming	9.83	1.72	7.00	2.00	9.00	2.38	10.00	1.65	9.10	3.38
Similarities	10.65	6.85	8.55	3.24	10.85	7.50	9.67	3.06	8.29	3.94
Comprehension	8.29	3.94	7.55	3.61	8.03	3.12	8.00	2.39	8.85	2.60
Block Design	9.10	3.38	8.64	3.26	8.90	2.90	9.56	2.73	10.00	2.36
Matrix Reasoning	10.00	2.36	10.00	5.39	8.56	2.13	9.20	1.64	11.90	2.28
Picture Concepts	11.90	2.28	9.60	2.97	10.89	2.67	11.80	3.11	7.22	3.23
Geometric Design	7.22	3.23	6.06	2.51	6.17	2.74	7.08	2.23	7.50	3.95
Mazes	7.50	3.95	6.76	2.70	7.23	3.21	6.82	2.32	7.80	1.81
Symbol Search	7.80	1.81	6.75	1.89	7.88	1.81	7.60	1.82	7.91	2.59
Coding	7.91	2.59	6.83	2.32	7.40	2.88	8.80	2.68	10.65	6.85
Object Assembly	8.85	2.60	8.15	2.30	8.87	2.56	9.44	2.79	9.82	3.06
Picture Completion	9.82	3.06	9.55	3.20	9.18	2.98	9.38	2.83	7.21	3.12
Animal Pegs	7.21	3.12	6.57	2.50	6.48	3.33	8.00	3.02	9.83	1.72
V-IQ	91.76	19.92	88.00	14.68	91.90	13.59	90.81	11.66	91.76	19.92
P-IQ	91.14	18.92	87.59	18.87	88.38	11.07	91.69	10.56	91.14	18.92
PSQ	88.90	10.34	85.25	9.85	87.74	16.27	89.40	11.41	88.90	10.34
GLC	91.50	7.06	83.00	9.39	93.29	4.54	92.50	2.12	91.50	7.06
FS-IQ	90.38	18.55	84.86	14.89	88.19	13.70	89.75	9.77	90.38	18.55

Paired t-tests revealed significant differences between some of these subtests. Table 4.3 highlights that while some of the subtests relying on processing speed, spatial orientation, working memory and auditory perception (indicated in the top row) are significantly poorer than subtests relying on verbal reasoning, and word recognition, these subtests are significantly poorer than non-verbal subtests that also rely on a degree of spatial organisation. Therefore, if *only* the non-verbal subtests relying on processing speed, spatial orientation, working memory and auditory perception had poorer performances than verbal subtests relying on verbal reasoning, the results would indicate that children who experience impairments on the range of difficulties specified (motor impairment, ASD, AD/HD and speech and language difficulties) experienced similar, distinct weaknesses in similar domains of cognitive functioning. However, because these subtests were also significantly weaker than other non-verbal subtests which also rely on spatial organisation (i.e. Block Design) and hence share an overlap in that cognitive domain, this in not conclusive. The presence of shared and significantly weaker performance tentatively suggests that the hypotheses of Hill (2001) could be confirmed through further investigation of the current sample.

Table 4.3
Paired t tests between subtests of the WPPSI

Subtest (*M*)	Arithmetic (7.99)	Geometric Design (*7.58*)	Mazes (*7.52*)	Symbol Search (*7.43*)	Coding (*7.58*)	Animal Pegs (*7.41*)
Information (*9.17*)	3.18**	4.05***	3.7***	2.69*		4.23***
Vocabulary (*9.47*)	4.83***	5.66***	4.48***	2.71*		4.22***
Comprehension (*8.10*)		4.00**	4.36***			3.73**
Similarities (*10.15*)	3.50**	2.27*	2.02*	2.89*		2.05*
Block Design (*9.31*)	4.25***	5.42***	5.05***	2.70*		5.43***
Picture Concepts (*10.33*)				6.62***	5.01***	
Object Assembly (*9.23*)	6.43***	7.24***	7.77***			4.62*
Picture Completion (*7.41*)	4.11***	5.81***	5.75**			5.58*

Secondly, it was rare for a child to present with a singular diagnostic label. Many of the children presented with a primary diagnosis as ASD, Epilepsy, Genetic Conditions or an Expressive or Mixed Receptive-Expressive Language Disorder. They would often have a secondary diagnosis or co-morbidity of AD/HD (*n* = 35 (21.34%)), motor impairment (*n* = 56 (34.15%)) an anxiety disorder (*n* = 29 (17.96%) and a multiple diagnosis (*n* = 29 (17.86%)).

The relatively high co-morbidity of other diagnoses with language impairment and ASD has two possibly explanations. The first is that the presence of ASD and language impairment predisposed children to develop attentional and motor difficulties that are later diagnosed as co-morbid diagnoses of AD/HD, anxiety disorders or motor impairment. Alternatively, both the primary and secondary diagnoses are caused by the same underlying aetiology, but are simply mediated by different psychological mechanisms. There is a body of recent research which supports the latter explanation (Bishop, 2008; Weismer, 2007). It suggests that, while these disorders might present differently symptomatically, there is a strong possibility that they indeed represent a similar underlying neurological aetiology which is simply differentiated by differences in neurological maturation (Hill, 2001).

Within child populations with SLI, neuroimaging studies suggest abnormalities in the proportions and symmetry of different cortical regions (Herbert et al., 2006; Leonard, Eckert, Given, Virginia and Eden, 2006). This lack of normal brain asymmetry may indicate dysfunction in the lateralised specialisation of language. Further, the recent growth in the use of magnetic resonance imaging (MRI) is increasingly used to conduct studies of typically and atypically developing children. Within a sample of children with language impairment, imaging has identified regions of asymmetry, particularly in the planum temporale, an area that is known for its involvement in the processing of speech and acoustic information. In some cases, this asymmetry is inconsistent with images showing reversed, exaggerated or left-ward asymmetry. Additional anatomical deviations from typical child brains include significantly smaller and atypically symmetrical triangular parts of the inferior frontal gyrus, and pre-frontal abnormalities, particularly in areas responsible for motor control (Dick, Richard & Saccuman, 2008). Other imaging that focused on the functioning (not necessarily structural) abnormalities of the brain showed that in a sample of children with SLI there existed a smaller and weaker pattern of activation in the left hemispheric language-regions than

those of controls. This is believed to be related to the difficulties individuals with SLI have in decoding the phonological structure of words and pseudo words (Friederici, 2006; Hugdahl et al., 2004).

Laterality studies have also been done with child populations with ASD in an attempt to shed light on the same processes of brain maturation and asymmetry that were considered within the context of SLI above. In one study, 47 autistic children with a history of early language impairment, 22 autistic individuals with normal language acquisition and 112 non-disordered individuals were compared on a standardised measure of lateral preference. The first group showed significantly more atypical cerebral dominance than both healthy participants and autistic individuals with normal language acquisition. The data indicates maturational disturbances in establishing lateral preference rather than the presence of left handedness. The authors suggested that atypical establishment of cerebral dominance may be one cause of impaired language development in Autism (Escalante-Mead, Minshew & Sweeney, 2003). Moreover, similar to findings about the unexpected lack of asymmetry in the planum temporale (PT) within a sample of children diagnosed as having SLI, neuroimaging studies on 12 adults with Autism matched to controls indicated that there is a lack of expected asymmetry in the Autistic group and not in the controls (as indicated by significant differences between the volume of the PT in both left and right hemispheres (Rojas, Camou, Reite & Rogers, 2005). These studies, as well as those considering the neurological structure and function of brains in children with SLI suggest that there may be a common underlying dysfunction across both diagnostic groups.

There are also studies that consider the lateralisation of brain functioning and hemispheric structures within populations who have AD/HD. Many studies have sought to investigate the relationships between AD/HD, language impairment, motor dysfunction and the frequent perceptual impairment that often accompanies these disorders. Hill (2001) found that children who suffer from SLI and those who have a diagnosis of AD/HD often have co-morbid motor impairments (usually given a diagnosis commonly used in Occupational Therapy, Developmental Coordination Disorder), and perceptual difficulties. Further, the most commonly associated psychiatric diagnosis given to children with language impairment is AD/HD (estimated at 63.6% of 7-14 year olds with language impairment in Canada) (Cohen, et al., 2000).

One study attempted to identify neurological processes explaining the poor motor control that is often associated with AD/HD. In a sample of 350 children with AD/HD, 195 non-affected siblings and 271 non-related participants without AD/HD, the researchers found significant differences in terms of poorer motor functioning of children with AD/HD relative to the two comparison groups. Motor control deficits were most pronounced in the left hand, which are believed to be related to right hemispheric brain pathology in children with AD/HD (Rommelse, et al., 2007). These findings are supported by another study ($n_{AD/HD} = 56$, $n_{comparison} = 52$) which confirmed that the abnormal spatial asymmetry evident in the motor control of children with AD/HD was related to dysfunction in right-hemispheric neural networks (Chan, Mattingley, Huang-Pollock, Ebglish, Hester, Vance & Bellgrove, 2009). Another mixed-method analysis of

other research and a case study indicated that the motor deficits in children with AD/HD are most likely related to neurological immaturity in the corpus callosum, which has more generalised effects on brain development, lateralisation and functioning and accounts for the common underlying neurological associations with Developmental Co-ordination Disorder and Dyslexia (Roessner, Banaschewski, Uebel, Becker & Rothenberger, 2004).

Hill (2001), has written widely on the potential underlying etiologies of the common symptoms within AD/HD, SLI and DCD. In one study, she found that children with SLI show weakness in auditory processing, non-verbal reasoning, picture naming, word recognition and on non-linguistic tasks such as reaction time, mental manipulation of figures and other timed motor tasks. Similar results were found within the current study, as the children performed poorly on tasks relying on *non-verbal* reasoning and working memory, particularly in the group experiencing impairment on the MRELD Spectrum. These results are still within average ranges, but are within the lower end of the average spectrum, with standard deviations indicating that several cases fell below average limits.

Hill (2001), convinced that the co-morbidity between motor impairment and SLI represented a related underlying neurological dysfunction, undertook a meta-analytic review of over 5000 studies that studied the potential overlap between symptoms of children diagnosed with Developmental Coordination Disorder and SLI. While it revealed that the two disorders shared considerable overlap in terms of perceptual skills and fine and gross motor coordination and are possibly sourced from the same aetiology, they are mediated by *different* psychological mechanisms. This suggests that SLI cannot be explained as a discrete neurological dysfunction, but refers to a greater and more generalised processing deficit. Previously, these were explained by Tallal's (1973) temporal processing theory and Kail's (1994) Generalised Slowing Hypothesis (GSH) (Hill, 2001).

The former, Tallal's temporal processing theory, suggests that the processing difficulties experienced by children with SLI arise from difficulties in integrating sensory information that converges in rapid succession to the central nervous system. This convergence deeply affects the phonological system, which compounds language impairment. Kail's GSH postulates that the processing speed difficulties in language impaired children are associated with some general versus specific aspect of cognitive development. According to this theory, children with SLI execute each component of a series of tasks much slower than a typical child would, increasing the absolute time of processing. Another suggestion is that children with SLI have reduced information processing capacity in comparison to their normally developing peers, which has the potential to influence performance on non-linguistic tasks as well. The reduced processing capacity of children within the sample was evident by their weakest performance on the WPPSI Processing Speed Quotient which fell in the below average range (M = 80.40, SD = 24.20).

An understanding of the overlapping symptomatology of language impairment with ASD, AD/HD and motor impairment can help practitioners better comprehend the atypically developing child within the

bigger picture of neurodevelopmental disorders. It leads to tentative suggestions that SLI is not simply a specific disorder of language but that it represents a broader range of difficulties, of which language impairment is just one. For example, in a selection of studies investigating the behavioural and neurological phenotypes and genetic aetiology of Autism and SLI, authors suggest that SLI and Autism, as well as Dyslexia, are indeed the same thing, but are differentiated only by subtypes which are mediated by varying environments and neurological processes (Bishop, 2000; Tager-Flusberg, 2004).

An explanation of these overlaps and hypotheses for the existence of a language-impairment-ASD-motor function continuum is now discussed. Childhood neurodevelopmental disorders are typically classified into distinct categories – one of them being language-related. However, language impairment has *also* been classified symptomatically under attention-based neurodevelopmental disorders like AD/HD and ASD. This overlap of symptoms across diagnostic categories suggests that the distinctions made between discrete neurodevelopmental disorders are somewhat artificial. This is possibly because the focus of previous research has been to fractionalise disorders and emphasise definitions based on specific and recognisable symptoms which assume a collective homogeneity of impairment, and mutually exclusive symptom profiles. The high co-morbidity between attention, language and motor dysfunction suggests that many children experience these as *simultaneous deficits* and that those who have highly specific deficits are the exception rather than the rule.

These shared symptom profiles between quite different diagnostic categories lead to two inter-related explanations that both rely on recent advances in the neuro-imaging of children's brains. The first are imaging studies of children with SLI. While there is no evidence of visually obvious lesions, there is evidence of atypical hemispheric asymmetry, particular atypical perisylvian asymmetries in such children. Ojemann (1984) found that sequential motor movement and language share a common brain mechanism that appears to be located within the lateral perisylvian cortex of the dominant hemisphere. Further, studies linking the co-morbidity of motor and language impairment have not yet found lesions or dysfunction in the cerebellum, but have found that the cerebellum (and particularly the neo-cerebellum and dorsolateral prefrontal cortex) functionally link the learning of motor tasks, cognitive and language skills and selective attention (Akshoomoff & Courchesne, 1992; Leiner et al., 1991).

The second, and far more likely option, suggests that deficits indicate an underlying neurological immaturity. This would explain why other developmental disorders also show co-morbid motoric deficits. Locke (1997) suggests that the neurological immaturity common in many childhood disorders does not negate the separateness of the processes of language, motor function and attention, but instead that they are all regulated by a common process of neurological development. This model also assumes the existence of critical periods that underlie neurological development. Supporting this is the work of Haynes and Naidoo (1991) which showed that only 44% of 156 British children with severe developmental language delays were walking by 15 months, and 34% of them were not walking until 18 months or later. Within the current sample, the mean age of walking was 14.84 months (*SD* = 4.58) months, which is also

somewhat delayed. As time passes, the neurological immaturity is unable to "catch up" and so other cognitive delays are seen – including prominent language delay within the critical period in which the linguistic mechanisms are required to develop. Conclusively, if neuro-maturational development is slow, then delayed development of linguistic, motoric and attentional-perceptual skills will occur. If these are not addressed in time, they forego critical periods of particular vulnerability to their development and persist in their domino effect on other aspects of cognitive development. In this way, the motoric or linguistic delays reflect biological developmental changes that are a function of the maturational processes of the nervous system. Thus, co-morbid cross-ability delays may be caused by widespread neurological immaturity (often signified by abnormal brain anatomical asymmetry, and undifferentiated handedness), and are not a consequence of each other.

In critique of neuro-imaging studies of children, it is important to recognise that the structural and functional differences between individuals are subtle, and that identification of these subtleties is done through a quantitative computerised process rather than being evident to the naked eye. Further, despite the technological sophistication of imaging, it does not indicate the direction of causal pathways by which language impairment is structured. Thus, it is unknown whether the structural brain differences observed in children with language impairment are the underlying cause, or whether they are at least partly due to differences in the child's language use, and his effect on the language environment. These issues are difficult to answer because of the relatively small sample sizes of the cited studies and the highly heterogeneous character of the samples (diagnosis, age, gender, upbringing) used. It is also important to remember that at times, the neurological dysfunction thought to cause speech impairment is not always a genetic malformation but a result of environmental effects such as meningitis, head injuries and intercerebral problems, such as strokes which affect general brain functioning or localised areas of speech and language processing (Baird, 2008). This makes it difficult to ascertain whether differences are truly linked, or if they are co-incidental, based on shared environmental influences.

Despite the research indicating potentially pioneering developments within the neurological explanations of childhood disorder, neuroscience is still a long way away from uncovering the possible mechanisms through which these occur. The variation in severity of the co-morbidities suggests the existence of some sub-types which could possibly explain the differing psychological mechanisms through which the differences manifest, as postulated by Hill (2001) and Powell and Bishop (1992). While these differences are not confirmed or disproved in the current study, the unexpected profiling of lowered performance in non-verbal tasks that was common to language impairment, AD/HD, ASD and motor impairment, as well as the high degree of overlap between discrete diagnoses suggests that there is some validity to the hypotheses postulated by the authors who promote the existence of impairments on a continuum.

Further implications of the study.

The original intention of the study was to identify factors that could accurately and significantly predict the cognitive performance of this particular atypical population, so as to identify potential risk and protective factors to their intellectual ability. The identification of these factors could inform educational and caregiving practices of other children with similar impairments, so as to minimise the impact of their speech and language impairments. This proved to be difficult to accomplish, as the three measures of cognitive functioning did not allow for a uniform profiling procedure, and results were sometimes skewed due to small sample sizes. However, across the three measures, certain variables were identified as either promoting or limiting cognitive performance.

The strong influence of high levels of parental education and the benefit of belonging to a two parent family, and to a lesser degree social support from extended family and employees, could be seen as protective factors against further vulnerability and impairment. Practitioners should therefore be aware of the converse of this situation and learn to act quickly in cases of high vulnerability to further impairment. Single parent families with poor socio-economic status who have a child with difficulties should be identified and given additional support to as to prevent decline.

General pregnancy health, the presence of chronic ear infections and major organ dysfunction of the heart, brain or lungs under the age of three years were moderately predictive of poorer intellectual performance across the sample. Breastfeeding was also moderately associated with better performance. These early childhood factors have obvious implications for infant childcare. As with demographic structures of economic and social capital, so vulnerable families for poor childhood health, poor infant nutrition and poor in-utero care could be identified and provided with additional support to prevent impairment. Likewise, in the event that a child experienced these difficulties, parents and practitioners should be careful to monitor their development and implement appropriate strategies to compensate for possible difficulties as early as possible.

As a final point with regards to the strongest influences of cognitive performance in children with speech and language impairment, it is important for practitioners to consider that no *one* factor has the absolute capacity to exclusively hinder or promote performance in a particular way. Impairment and development are highly idiosyncratic processes that differ according to the child and their unique environment. Profiling serves only as a framework upon which to base individual assessment, education and intervention. Bishop (2008) considered the various factors that are believed to cause speech and hearing impairment within a model similar to that in Figure 1.1. Each factor serves only as a risk or protective factor, and it is the particular and unique combination of an equally unique set of risk or protective factors which determine a child's level of development. Figure 1.1. illustrates that in many cases the causes or predispositions of impairment are actually unknown.

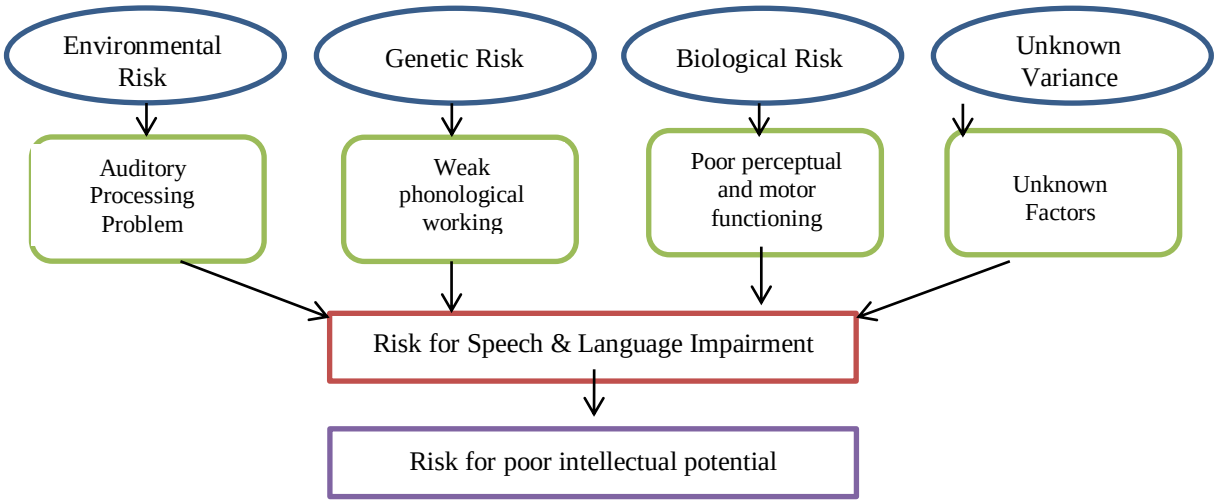


Figure 1.1. Diagram indicating possible pathways that predispose children to develop speech and language impairment and subsequent cognitive limitations. Adapted from Bishop, D. (2008). Specific Language Impairment, dyslexia and Autism: using genetics to unravel their relationship. In. C. F. Norbury, J. B. Tomblin & D. V. M. Bishop (Eds.). *Understanding Developmental language Disorders: From Theory to Practice* (pp.67-78). New York: Psychology Press.

The fact that age of identification and intervention were not predicted in the expected fashion does not dismiss them as unimportant variables when considering best-practice procedures in dealing with atypical and vulnerable groups. The vast majority of the sample were identified early in their lives and received intensive input weekly for several years. The fact that at least half entered mainstream schooling serves as testimony to the valued work of the health professionals at this specialised pre-primary school and the benefit of screening and early intervention.

The strong influence of genetic conditions and the severity of impairment that they yield is also something that can be prevented. Congenital deafness is an autosomal recessive genetic disorder on the GJB2 or GJB6 gene, and is particularly prevalent in the orthodox Jewish community (Rosner, Rosner & Orr-Urtreger, 2009). At risk groups for genetic or congenital conditions should be identified and advised to go for genetic counselling prior to starting a family so that all necessary precautions are made.

Lastly, the possibility that ASD, speech and language impairment, motor impairment and AD/HD represent a shared neurological immaturity in the brain has implications for intervention within this population. Some practitioners might assume that symptoms cluster as discretely as their diagnostic categories. Assessment and intervention of these groups should be comprehensive, multi-modal and frequent, since motor impairment and poor attentional capacity are usually identified quite late because of the increasing demands of formal schooling.

Limitations of the current study.

The current study's method and assumptions had certain flaws which limit its validity and power to contribute to the existing body of knowledge within this area.

To begin with, the study was archival in nature and relied upon a pre-existing body of knowledge that was translated from intake forms, medical, scholastic and psychological reports into electronic form. The

research had to be directed by the information that was available to the researcher, and does therefore not contain all the information necessary to answer some of the more interesting questions about this atypical population. There is therefore a large amount that is unknown, and could not be asked or sourced in a manner that would have been more useful to the objectives of the study, and to the statistical analyses being applied. From the information available, groups of data were converted into numerical indicators in order to make them statistically usable. The construction of these indicator scales (especially in the case of pregnancy information) from a wide variety of single questions, raises methodological concerns. Because the indicators were drawn up by the researcher, the final weighting of each question could have under- or overestimated certain information. It is believed that the influence of pregnancy and neonate health should have had a much higher impact than is reflected in this study. The underestimation could have also been a function of this particular population, or the pregnancy complications could have been overshadowed by collinearity within other variables. For example, maternal education and social support have been found to mediate pre-natal stress and nutrition (Talge, Neal, Glover, 2007).

The design of the study did not include a comparison group of typically developing children. While there is a large body of research informing the developmental processes and predictors of language and cognitive development in typically developing children, the study was not afforded the opportunity to statistically differentiate between these groups. Comparisons and distinctions between typically and atypically developing children were therefore made at face value.

Within the design of the study, there was incongruence between the measures of cognitive functioning. While all of the test scores can be converted to a mental age, and two of the tests operate on a deviation IQ score and are rooted within a Wechsler model of intelligence, they are in essence *not* the same construct. To remedy this, and maintain reliability across the study, the sample was segmented into three different smaller samples who were measured on the different tests (WPPSI, JSAIS and the Griffiths). This reduces the sample size, and limited the possible number of variables that could be considered for the prediction of cognitive functioning. Further, this reduced sample size also reduced the general statistical power of the analyses. The design is ex-post-facto in nature, cannot imply causality or even quasi-experimentality.

Within these measures of cognitive functioning, it was decided to equate the VIQ, PIQ and FSIQ scores sourced from the WPPSI-R and WPPSI-III, even though the two measures are not strictly comparable. The relative validity of the WPPSI-R to the WPPSI-III is a limitation when comparing them, but in order to increase statistical power through an increased sample size, their similar composite scales were equated, since the WPPSI-R is considered to be a valid convergent measure of the WPPSI-III (Wechsler, 2002).

With regards to the sample, the study made use of atypically developing children who were not all diagnosed and treated by the same health professionals. While there was a large degree of practitioner cross-over (which makes sense in light of the type and specialised needs of this population), the

procedures and criteria for diagnosis and case management might have varied across professionals, and could therefore limit the reliability of the study. The sample also attended a specialised pre-primary school that is serviced by highly-trained health professionals, a luxury that is only afforded by the very wealthy. While the school might offer subsidised funding to families who cannot afford the fees, the external validity and generalizability of the findings of the research are limited to children from similar circumstances.

The initial data collection for the study was collected several years ago and was beyond the researcher's control. This raises certain methodological weaknesses that limit the reliability of the information. The developmental history of the child was based on maternal report, which has been found to be both trustworthy and at times blatantly untrue. As far as possible, collateral was received from reports by doctors, paediatricians, and the motor milestones, handedness were derived from a standardised pre-primary nursery school report.

The study's method and analysis was also unavoidably based on faulty assumptions. Because of the strictly quantitative nature of the research, the sample and their characteristics were broken down into discrete groups that assume statistical homogeneity. However, highly unlikely, considering that most of the disorders exist on a spectrum and operate in very different ways. A mixed methods design would have allowed for a more fine-grained understanding of this differentiation.

The research study also makes particular theoretical assumptions that are controversial within developmental practice. The use and comparison of children to developmental norms are often considered problematic. These norms are age-appropriate abilities and expectations of *typically* developing children. Comparing other children, especially atypically developing children, to these standards is sometimes unfair because growth is therefore relative to a sample that usually has a large degree of unexplained variance in accounting for individual difference. In this way, researchers are caught in a bind as they cannot possibly create norms for multiple subgroups, as they will lose their generalizability to a greater population. Yet, the larger population becomes increasingly distant and more heterogeneous (increasing variability in individual difference) as it grows. The application of these norms to *atypical* populations becomes increasingly controversial as it places them at a disadvantage to a group about which little explainable variance is known.

Further, not only is the norming process of developmental milestone acquisition problematic, but the concept of discrete developmental stages within which various domains of development and growth should occur is similarly limiting. While there are of course thresholds beyond which delay is an acceptable recognition, sometimes lags are normal, and domains often play 'catch-up' to one another in a wave-like manner. For that reason, the use of normed comparison between larger populations and 'developmentally delayed' individuals in diagnosing delays as *disorders* is sometimes problematic, especially within countries for which Western norms may not apply.

Recommendations for Future Research.

A more methodologically sound way of understanding the factors that influence the cognitive potential of children with speech and language impairment would be to undertake a longitudinal study of children both with and without speech and language impairment throughout their childhood. In this way, a comprehensive intake interview could be conducted with parents at the commencement of the study, and the children's cognitive performance could be traced and compared throughout their development. In this way, intellectual assessment could be made uniform across the sample, and complimentary assessments of motor functioning and attention capacity could also be done periodically. Further, in order to ascertain a more accurate understanding of the influence of early identification and intervention, such a study could compare the results to an existing sample of children who have not had access to early intervention, or the benefit of a specialised pre-primary school. Methodologically, it would then be difficult to identify whether it is indeed the delayed or absent intervention, or a poorly resourced environment that associated with poorer cognitive performance. Despite these concerns, ethical integrity does not allow discrimination in any other way.

Perhaps the most salient question that has arisen from this research is the unexpected cognitive weaknesses in non-verbal reasoning and processing speed across the sample's various diagnostic categories, as well as the high degree of diagnostic co-morbidity between the participants. These results, together with the high proportion of undifferentiated handedness (25.77%) within the sample, suggest that recent research within the field of neuroscience is in the right direction. Neuroimaging studies use these external indicators of hemispheric dominance, symmetry and lateralisation within the brain across diagnoses that are believed to be discrete categories, and suggest that they have a common underlying neurological immaturity which are simply mediated by different processes which elicit their symptomological differences. There is no easy way to investigate these unknowns, and the answers possibly lie within the realm of neuroimaging and medical neuroscience. One suggestion is to not only compare the phonotypical overlaps in behaviour and performance (as many studies have done), but to consider underlying genetic etiologies for all the diagnoses. A group of studies has already identified common abnormalities on the FOXP2 gene on chromosome 7q31 in both children with SLI and Autism (Ashley-Koch, O' Brein Wolpert, Menold, Naeem, Basu, Donnelly et al., 1999; O' Brein, Zhang, & Nishimura et al., 2003; Warburton et al., 2000). Alternatively, advances in neuroimaging could reveal scientific evidence for the speculated commonalities.

Conclusion

This exploratory study identified a set of predictors of cognitive performance in an atypical population of pre-school children with speech and language impairment. One of the original intentions was to determine whether the development of language and cognition in an atypical population was any different to that of typically developing children. After analysis and interpretation, the identified predictors of cognitive

performance do not seem to differ significantly from those known to influence the construct within typically developing populations. However, the study has offered insight into the identification of broader patterns in the sample's characteristics and cognitive profiles that have highlighted particular commonalities across diagnostic characteristics. These findings have particular use in identifying and intervening with children at risk for future speech and language impairment and poor cognitive capacity. They also provide tentative direction for future research in neuroscience and the possible etiologies of childhood disorders that have previously been thought to be discrete and unrelated diagnostic categories.

References

Abbeduto, L. & McDuffie, A. (2007). Language learning and use as embedded social activities: Evidence from Autism and Fragile X Syndrome. In R. Paul (Ed.) *Language disorders from a developmental perspective* (pp.195-214). Mahwah: Lawrence Erlbaum Associates.

Abdel-Khalek, A., M., & Lynn, R. (2008) Intelligence, family size and birth order: Some data from Kuwait. *Personality and Individual Differences, 44,* 1032-1038.

Aggleton, J.P., R.W. Kentridge, & J.M.M. Good. (1994). Handedness and Musical Ability: A Study of Professional Orchestral Players, Composers, and Choir Members. *Psychology of Music, 22,* 148–56.

Akshoomoff, N. A. & Courchesne, E. (1992). A new role for the cerebellum in cognitive operation. *Behavioural Neuroscience, 106,* 731-738.

Allan, M. M. (1988). *A comparison of the performance of normal preschool South African children and British children on the Griffiths Scales of Mental Development.* (Unpublished Masters Thesis). University of Port Elizabeth, Port Elizabeth.

Allan, M. M. (1992). *The performance of South African normal preschool children on the Griffiths Scales of Mental Development: A Comparative Study.* (Unpublished Doctoral Thesis). University of Port Elizabeth, Port Elizabeth.

Als, H., Lawhon, G. & Duffy, F. (1994). Individualised developmental care for the very low birthweight preterm infant: Medical and neurofunctional effects. *Journal of the American Medical Association, 272, 835*-858.

Anjali, J., Concato, J. & Leventhal, J. M. (2002). How good is the literature linking breastfeeding and intelligence? *Paediatrics, 109,* 1044-1049.

Archbold, S., Harris, M., O'Donoghue, G., Nikolopoulos, T, White, A., & Richmond, H. L. (2008). Reading abilities after cochlear implantation: The effect of age of implantation on outcomes at 5 and 7 years after implantation. *International Journal of Paediatric Otorhinolaryngol, 72,* 1471-1478.

Archibald, L. M. D. & Alloway, T. P. (2008). Comparing language profiles: children with Specific Language Impairment and Developmental Coordination Disorder. *International Journal of Language and Communication Disorders, 43,* 165-180.

Archibald, L. M. D. & Gathercole, S. E. (2006). Short-term memory and working memory in specific language impairment. *International Journal of Language and Communication, 41, 675-693*

Ashley-Koch, A., Wolpert, C., Menold, M., Naeem, L., Basu, S., Donnelly, S. et al. (1999). Genetic studies of autistic disorder and chromosome 7. *Genomics, 61,* 227-236.

Aschoff, A., Kremer, P., Hashemi, B. & Kunze, B. (1999). The scientific history of hydrocephalus and ints treatment. *Neuropsychological Review, 22,* 67-93.

Baddeley, A. (1998). *Human memory: Theory and practice (Revised edition).* Boston: Allyn & Bacon.

Baird, G. (2008). Assessment and investigation of children with developmental language disorder. In C. F. Norbury, J. B. Tomlin & D. V. M. Bishop (Eds*.) Understanding developmental language disorders: From theory to practice* (pp. 1-22). Hove: Psychology Press.

Barnett, S. (1993). Benefit cost-analysis of preschool education: Findings from a 25 year follow-up. *American Journal of Orthopsychiatry, 63,* 500-508.

Bates, E. A. (2004). Commentary. Explaining and interpreting deficits in language development across clinical groups: Where do we go from here? *Brain and Language, 88,* 248-253.

Bavin, E. L., Wilson, P. H., Mmaruff, p. & Sleeman, F. (2005). Spatio-visual memory of children with specific language impairment: Evidence for generalised processing problems. *International Journal of Language and Communication, 40,* 319-332.

Beighton, P., De Paepe, A., Steinmann, B., Tsipouras, P., & Wenstrup, R. J. (1998). Ehlers- Danlos syndromes: Revised nosology, Villefranche, 1997. *American Journal of Medical Genetics, 77,* 31-37

Beitchman, J. H., Hood, J. & Inglis-Alison. (1990). Psychiatric risk in children with speech and language disorders. *Journal of Abnormal Child Psychology, 18,* 283-296, DOI: 10.1007/BF00916566

Berghella, V., Baxter, J. K., & Chauhan, S. P. (2005). Evidence-based surgery for caesarean delivery. *American Journal of Obstetrics and Gynaecology, 193,* 1607-1617.

Bhamjee, R. A. (1991). A comparison of the performance of normal British and South African Indian children on the Griffiths Scales of Mental Development. (Unpublished Doctoral Thesis). University of Port Elizabeth, Port Elizabeth.

Bishop, D. V. M. (1990). *Handedness and developmental disorder*. London: Mac Keith Press.

Bishop, D. V. M. (1997). *Uncommon understanding: Development and disorders of language comprehension.* East Susses: Psychology Press.

Bishop, D. V. M. (2008). Specific language impairment, dyslexia and Autism: Using genetics to unravel their relationship. In Norbury, C. F., Tomblin, J. B. & Bishop (Eds.) *Understanding Developmental Language Disorders: From Theory to Practice* (pp.67-78). Hove: Psychology Press.

Blumberg, B. F. & Pfann, G. A. (2001). *Social capital and the uncertainty reduction of self-employment. Discussion paper 303. The Institute for the Study of Labour (IZA).* University of Maastricht. Germany.

Boomsma, D. I., van Beijsterveld, T., Beem, A. L., Hoekstra, R. A., Polderman, T. J. C., & Bartels, M. (2008). Intelligence and birth order in boys and girls. *Intelligence, 36,* 630-634.

Botting, N. (2005). Non-verbal cognitive development and language impairment. *Journal of Child Psychology and Psychiatry, 46,* 317-326.

Botting, N. (2006). The interplay between language and cognition in typical and atypical development. In J. Clegg, & J. Ginsborg (Eds.), *Language and social disadvantage: Theory into practice* (pp. 28-43). Chichester: John Wiley & Sons Ltd.

Bray, M. (2006). Language and communication in young people with learning difficulties. In J. Clegg, & J. Ginsborg (Eds.), *Language and social disadvantage: Theory into practice* (pp. 106-121). Chichester: John Wiley & Sons Ltd.

Brock, J. (2007). Language abilities in Williams' Syndrome: A critical review. *Development and Psychopathology, 19,* 97-127.

Brooks-Gunn, J., Klebanov, P. K. & Duncan, G. J. (1996). Ethnic differences in children's intelligence test scores: Role of economic deprivation, home environment and maternal characteristics. *Child Development, 67,* 396-408.

Brooks-Gunn, J., Maiie C., McConuick, M. D., Shapiro, S., April, B. S., Benasich, A., & Black, G. W. (1994). The Effects of Early Education Intervention on Maternal Employment, Public Assistance, and Health Insurance: The Infant Health and Development Program. *American Journal of Public Health, 86,* 924-930.

Bruce, B. Thernlund, G. & Nettelbladt, U. (2006). AD/HD and language impairment: A study of the parent questionnaire FTF (Five to Fifteen). *European Child and Adolescent Psychiatry, 15,* 52-60.

Buckhalt, J. A., El-Sheikh, M., Keller, P. S., & Kelly, R. J. (2009). Concurrent and longitudinal relations between children's sleep and cognitive functioning: The moderating role of parent education. *Child Development, 80,* 875-892.

Calderon, R & Naidu, S. (1998). Further support for the benefits of early identification and intervention for children with hearing loss. *Volta Review, 5,* 53-84.

Cantwell, D. P. & Baker, L. (1987). Prevalence and type of psychiatric disorder and developmental disorder in three speech and language groups. *Journal of Communication Disorders, 20,* 151-160.

Camurdan, A. D., Koc, I., Beyazova, U., Ilhan, M. N., & Sahin, F. (2008). Toilet training in Turkey: The factors that affect timing and duration in different sociocultural groups. *Child: Care, Health & Development, 34,* 475-481.

Case- Smith, J. (2005). *Occupational Therapy for Children. 6th edition.* St Louis: Mosby.

Cassidy, S. B. (1997). Prader-Willi syndrome. *Journal of Medical Genetics, 34,* 917–23

Chan, E. Mattingley, J. B., Huang-Pollock, C., English, T., Hester, R., Vance, A. & Bellgrove, M. A. (2009). Abnormal spatial asymmetry of selective attention in ADHD. *Journal of Child Psychology and Psychiatry, 50,* 1064-1072.

Clegg, J., Hollis, C., Mawhood, L. & Rutter, M. (2005). Developmental language disorders: A follow up in later adult life – cognition, language and psychosocial outcomes. *Journal of Child Psychology and Psychiatry, 46,* 128-149.

Clements, K. M., Wanda, E., Barfield, D., Kotelchuck, M. & Wilber, N. (2008). Maternal Socio-Economic and Race/Ethnic Characteristics Associated with Early Intervention Participation. *Maternal and Child Health Journal, 12,* 708-171.

Coalson, D. & Zhu, J. (2002). WPPSI-III technical and interpretive manual. Wechsler Preschool and Primary Scale of Intelligence.

Cockcroft, K., Amod, Z., & Soellaart, B. (2008). Level of maternal education and performance of Black, South African infants on the 1996 Griffiths Mental Development Scales. *African Journal of Psychiatry, 11,* 44-50.

Cohen, N., Vallance, D. D., Barwick, M., Im, N, Menna, R. et al. (2000). The interface between AD/HD and language impairment: An examination of language, achievement, and cognitive processing. *Journal of Child Psychology and Psychiatry, 41,* 353-362.

Coryell, J. (1985). Infant rightward asymmetries predict right-handedness in childhood. *Neuropsychologia, 23,* 269-271.

Craig, H. B., & Gordon, H. W. (1988). Specialised cognitive function and reading achievement in hearing-impaired adolescents. *Journal of Speech and Hearing Disorders, 53,* 30-41.

Cranberg, L. D., Filley, C. M., Hart, E. J., Alexander, M. P. (1990). *Acquired Aphasia in Children: Clinical and CT investigations.* Proceedings of the Advanced Research Workshop, Portugal. NATO Science Series.

Crowley, M. (1992). Behavioural difficulties and their relationship to language impairment. In J. Law (Ed.) *Early identification of language impairment in children: Therapy in practice* (pp.63 -83). London: Chapman & Hall.

Curfs, L. M., Fryns, J. P. (1992). Prader-Willi syndrome: a review with special attention to the cognitive and behavioural profile. *Birth Defects Original Article Series, 28,* 99–104

Deary, I. J. (2000). *Looking down on human intelligence: From psychometrics to the brain*. Oxford; Oxford University Press.

Der, G., Batty, G. D. & Deary, I. D. (2006). Effect of breast feeding on intelligence in children: prospective study, sibling pair's analysis, and meta-analysis. *British Medical Journal*, 333, 945.

Dhar, R. L. (2009). Living with a developmentally disabled child: Attitudes of family members in India. *The Social Science Journal, 46,* 738-755.

Dick, F., Richardson, F., & Saccuman, M. C. (2008). Using magnetic resonance imaging to investigate the developmental langue disorders. In C. F. Norbury, J. B. Tomblin & Bishop, D. V. M. (Eds.) *Understanding Developmental Language Disorders: From Theory to Practice* (pp. 53-66). Howe: Psychological Press.

Dixon, G., Joffee, B., & Bench, R. J. (2001). The efficacy of visualising and verbalising: Are we asking too much? *Child Language Teaching and Therapy, 17,* 127-141.

Doherty, I., & Landells, J. (2006). Literacy and Numeracy. In J. Clegg, & J. Ginsborg (Eds.), *Language and social disadvantage: Theory into practice* (pp. 44-58). Chichester: John Wiley & Sons Ltd.

Emanuel, R., & Herman, R. (1992). The early identification of hearing loss and the effects of impaired hearing on language development. In J. Law (Ed). *Early identification of hearing impairment in children* (pp. 84-108). London: Chapman & Hall.

Escalante-Mead, P. R., Minshew, N., & Sweeney, J. A. (2003). Abnormal brain lateralization in high functioning Autism. *Journal of Autism and Developmental Disorders, 33,* 539-543.

Estil, L. B., Whiting, H. T. A., Sigmundsson, H., & Ingvaldsen, R. P. (2003) Why might language and motor impairments occur together? *Infant Child Development, 12,* 253–265.

Faurie, C., Vianey-Liaud, N., & Michel, R. (2006). Do left handed children have advantages regarding school performance and leadership skills? *Laterality, 11,* 57-70.

Fenson, L., Dale, P. S., Reznick, J. S., Bates, E., Thal, D. J., & Pethick, S. J. (1994). Variability in early communicative development. *Monographs of the Society of Research in Child Development, 59.*

Ferrari, M. (2007). Cognitive performance and left-handedness: Comparative analyses in adults with seizures, physical, psychological and learning disorders in a rehabilitation setting. *Journal of Rehabilitation, 73.* Downloaded on 12 October 2010 from http://findarticles.com/p/articles/mi_m0825/is_1_73/ai_n19094557/pg_6/

Fish, M., & Pinkerman, B. (2002). Language skills in low-SES rural Appalachian children: normative development and individual differences, infancy to preschool. *Applied Developmental Psychology, 23,* 539-565.

Foxcroft, C. D. (1985). The use of the Reitan-Indiana neuropsychological test battery in South Africa: cross-ethnic comparison of normal pre-school children. (Unpublished Doctoral dissertation). University of Port Elizabeth, Port Elizabeth.

Fraser, F. C., Ling, D., Clogg, D., Nogrady, B., & Gorlin, R. J. (1978). Genetic aspects of the BOR syndrome—branchial fistulas, ear pits, hearing loss, and renal anomalies. *American Journal of Medical Genetics, 2,* 241-252.

Fraser, F. C., Sproule, J. R., Halal, F., & Optiz, J. M. (2005). Frequency of the branchio-oto-renal (BOR) syndrome in children with profound hearing loss. *American Journal of Medical Genetics, 7,* 341-349.

Friederici, A. D. (2006). The neural basis of language development and its impairment. *Neuron, 52,* 941-952.

Gaines, R. & Missiuna, C. (2006). Early identification: are speech/language-impaired toddlers at increased risk for Developmental Coordination Disorder? *Child: Care, health and development, 33,* 325-332.

Gardner, H., Kornhaber, M. L., & Wake, W. K. (1996). *Intelligence: Multiple perspectives.* Orlando: Holt, Rinehart & Whinston Inc.

Garton, A. F. (1992). *Social interaction and the development of language and cognition.* Hove: Lawrence Erlbaum Associates Ltd.

Gathercole, S. E. & Baddeley, A. D. (1990). Phonological memory deficits in language disordered children: Is there a causal connection? *Journal of Memory and Language, 29,* 336-360.

Gedalia, A., Press, J., Klein, M., & Buskila, D. (1993). Joint hypermobility and fibromyalgia in school children. *Annals of the Rheumatic Diseases, 52*, 494-498.

Ginsborg, J. (2006). The effects of socio-economic status on children's language acquisition and use. In J. Clegg, & J. Ginsborg (Eds.), *Language and social disadvantage: Theory into practice* (pp. 9-27). Chichester: John Wiley & Sons Ltd.

Groenen, P., Crul, T., Maassen, B., van Bon, W. (1996). Perception of voicing cues by children with early otitis media with and without language impairment. *Journal of Speech & Hearing Research, 39,*

Guy, C. (1999). Sound advice: Protecting your child against the effects of hearing loss. *Today's Parent,* 16, 29-32.

Hack, M., Klein, N., & Taylor, H. G. (1995). School-age outcomes of children of extremely low birthweight and gestational age. *Seminars in Neonatology, 1,* 277-288.

Hansson, K., Sahlen, B., & Maki-Torkko. (2007). Can a 'single hit' cause limitations in language development? A comparative study of Swedish children with hearing impairment and children with specific language impairment. *International Journal of Language Communication Disorders, 42*, 307-323.

Haynes, C. & Naidoo, S. (1991). *Children with Specific Speech and Language Impairment.* London: MacKeith.

Heimes, L. (1983). The comparison of the JSAIS and the Griffiths Developmental Scale scores of 3-5 year old boys and girls. (Unpublished Masters Thesis). University of Port Elizabeth, Port Elizabeth.

Herbert, M. R., Ziegler, D. A., Deutsch, C. K., O'Brein, L. M., Kennedy, D. N., Filipek, P. A. et al. (2005). Brain asymmetries in Autism and developmental language disorders: A nested whole-brain analysis. *Brain, 128,* 213-226.

Herrera, J. A., Salmeron, B., & Hurtado, H. (1997). Prenatal biopsychosocial risk assessment and low birthweight. *Social Science and Medicine, 8,* 1107-1114.

Hill, E. L. (2001). Non-specific nature of specific language impairment: a review of the literature with regard to concomitant motor impairments. *International Journal of Language and Communication Disorders, 36,* 149-171.

Holm, V. A., Cassidy, S. B., & Butler, M. G. (1993). Prader-Willi syndrome: Consensus diagnostic criteria. *Paediatrics, 91,* 398–402.

Houston-McMillan, J. E. (1997). Borderline mentally handicapped pre-schoolers: Identification and treatment evaluation using the Griffiths Scales of Mental Development. In D. M. Luiz (Ed.) *Griffiths Scales of Mental Development: South African studies* (pp. 21-32). Research Papers C25 Publication Series: University of Port Elizabeth.

Howell, D. C. (1997). *Fundamental Statistics for the Behavioural Sciences (5th Edition).* Belmont: Thomson Higher Education.

Hugdahl, K., Gundersen, H., Thomsen, T., Rimol, L. M., Ersland, L., & Niemi, J. (2004). fMRI brain activation in a Finnish family with specific language impairment compared with a normal control group. *Journal of Speech, Language and Hearing Research, 47,* 162-172.

Hughes, D., Sapp, G. L., & Kohler, M. P. (2006). Issues in the intellectual assessment of hearing impaired children. Unpublished Research: University of Birmingham. Downloaded from http://eric.ed.gov/ERICWebPortal/search/detailmini.jsp?_nfpb=true&_&ERICExtSearch_SearchValue_0=ED493819&ERICExtSearch_SearchType_0=no&accno=ED493819 on the 16 October 2010.

Hulme, C., & Snowling, M. J. (2009). *Developmental disorders of language, learning and cognition.* Chichester: Wiley-Blackwell.

Jakobsen, L. P., Knudsen, M. A., Lespinasse, J. (2006). The genetic basis of the Pierre Robin Sequence. *Cleft Palate Craniofacial Journal, 43,* 155–159.

Johanson, R., Wilkinson, P., Bastible, A., Ryan, S., Murphy, H., & O'Brein, S. (1993). Health after childbirth: A comparison of normal and assisted vaginal delivery. *Midwifery, 9,* 161-168.

Johnson, S. (2007). Cognitive and behavioural outcomes following very preterm birth. *Seminars in Foetal and Neonatal Medicine, 12,* 363-373.

Johnson, D. & Walker, T. (1991). A follow-up evaluation of the Houston Parent-Child Development Centre: School Performance. *Journal of Early Intervention, 15,* 226-236.

Johnston, D. W., Nicholls, M. E. R., Shah, M., & Shields, M. A. (2009). Nature's experiment? Handedness and early childhood development. *Demography, 46,* 281-301.

Kamphaus, R. W. (1993). *Clinical assessment of children's intelligence assessment.* Massachusetts: Allyn & Bacon.

Kaplan, B. J., Wilson, B. N., Dewey, D. M., & Crawford, S. G. (1998) DCD may not be a discrete disorder. *Human Movement Science*, *17,* 471–490.

Kay, A. P. F., Ferguson, M., Molfesa, D. L., Peach, K., Lehman, C., & Molfesa, V. J. (2007). Smoking during pregnancy affects speech-processing ability in newborn infants. *Environmental Health Perspectives, 115,* 623-629.

Kelly, A. B., Garnett, M. S., Attwood, T., & Peterson, C. (2008). Autism spectrum symptomatology in children: The impact of family and peer relationships. *Journal of Abnormal Child Psychology, 36,* 1069-1081.

Knoesen, N. (2003). *An exploration of the relationship between the revised Griffiths Scales and Grade One Scholastic Development.* (Unpublished Masters Thesis). University of Port Elizabeth, Port Elizabeth.

Krassowski, E., & Plante, E. (1997). IQ variability in children with SLI: Implications for the use of cognitive referencing in determining SLI. *Journal of Communication Disorders, 30,* 1-9.

Kyle, J. G. (1980). Measuring the intelligence of deaf children. *Bulletin of the British Psychological Society, 33,* 54-57.

Law, J. (1992). *The early identification of language impairment in children: Therapy in practice.* London: Chapman & Hall.

Lawrence, E. J. (2005). The clinical presentation of Ehlers-Danlos syndrome. *Advanced Neonatal Care, 5,* 301–314.

Leiner, H. C., Leiner, A. L., & Dow, R. S. (1991). The human cerebro-cerebellar system: its computing, cognitive and language skills. *Behavioural Brain Research, 44,* 113-128.

Leonard, C., Eckert, M. Given, B., Virginia, B., & Eden, G. (2006). Individual differences in anatomy predict reading and oral language impairments in children. *Brain, 129,* 3329-3342.

Lewis, B. A., Freebairn, L. A., & Taylor, H. G. (2000). Academic outcomes in children with histories of speech sound disorders. *Journal of Communication Disorders, 33,* 11-30.

Lewis, M. & Jasker, J. (1983) Infant intelligence and its relation to birth order and birth spacing. *Infant behaviour and intelligence, 6,* 117-120.

Lichtenberger, E. O., & Kaufman, A. S. (2004) *Essentials of WPPSI-III Assessment.* Hoboken: John Wiley & Sons, Inc.

Lieu, J. E. C., Tye-Murray, N,, Karzon, R. K., & Piccirillo, J. F. (2010).Unilateral Hearing Loss Is Associated With Worse Speech-Language Scores in Children. *Paediatrics, 125,* 1348-1355.

Litt, R., Armon, Y., Seidman, D.S., Yafe, H., & Gale, R. (1993). The effect of mode of delivery on long-term outcome of low birthweight infants. *European Journal of Obstetrics & Gynaecology and Reproductive Biology, 52*, 5-10.

Locke, J. L. (1997). A theory of neurological development. *Brain and Language, 58,* 265-326.

Luiz, D. M. (1997). A child with hearing loss: A longitudinal study. In D. M. Luiz (Ed.) *Griffiths Scales of Mental Development: South African* studies (pp. 44-51). Research Papers C25 Publication Series: University of Port Elizabeth.

Lyxell, B. & Holmberg, I. (2000). Visual speech reading and cognitive performance in hearing-impaired and normal hearing children. *British Journal of Educational Psychology, 70,* 505-518.

Madge, E. M. (1981). *Manual for the Junior South African Individual Scales.* Pretoria: Health Sciences Research Council.

Marton, K., Abramoff, B., & Rosenzweig, S. (2005). Social cognition and language in children with specific language impairment (SLI). *Journal of Communication Disorders, 38,* 143-162.

Mash, E. J., & Wolfe, D. A. (2005*). Abnormal child psychology (3rd Ed.)*. Belmont: Thomson Wadsworth.

Mask, N. & Bowen, C. E. (1984). Comparison of the WISC-R and the Leiter International Performance Scale with average and above average students. *Journal of Clinical Psychology, 40,* 303-305.

McCauley, R. J. (2001). *Assessment of language disorders in children*. Mahwah: Lawrence Erlbaum Associates, Publishers.

Miller, C. A., & Gilbert, E. (2008). Comparison of performance on two nonverbal intelligence tests by adolescents with and without language impairment. *Journal of Communication Disorders, 41,* 358-371.

Natsopoulos, D., Kiosseoglou, G., & Xeromeritou, A. (2004).Handedness and spatial ability in children: Further support for Geschwind's hypothesis of 'pathology of superiority' and for Annett's theory of intelligence. *Genetic, Social, and General Psychology Monographs, 118,*

Nichols, J. D., Shah, M., & Shields, M. (2009). Nature's Experiment? Handedness and early childhood development. *Demography, 46,* 281-301.

Noble, S. & Emmett, P. (2006). Differences in weaning practices, food and nutrient intake between breast and formula-fed 4 month old infants in England. *Journal of Human Nutrition & Dietetics, 19,* 303-313.

Norbury, C. F., Tomblin, J. B., Bishop, D. V. M. (Eds.). *Understanding Developmental language Disorders: From Theory to Practice* (pp.67-78). New York: Psychology Press.

Noterdaeme, M. & Amorosa, H. (1999). Evaluation of emotional and behavioural problems in children using the Childhood Behaviour Checklist. *European Child and Adolescent Psychiatry, 8,* 71-77.

Oakes, D. (1994). *Your medical questions answered*. Cape Town: Readers Digest South Africa.

O'Brein, E., Zhang, X., Nishimura, C., Tomblin, J. B., & Murray, J. (2003). Associations of specific language impairment (SLI) to the region of 7q31. *American Journal of Human Genetics, 72,* 1536-1543.

Ojemann, G. A. (1984). Common cortical and thalamic mechanisms for language and motor function. *American Journal of Physiology, 246* (Special Issue; Regulatory Integrative and Comparative Physiology), R901-903.

Ottem, E. (2003). Confirmatory factor analysis of the WPPSI for language impaired children. *Scandinavian Journal of Psychology, 44,* 433-439.

Owens, R. E. (2005). *Language development: An introduction (6th Ed.).* New York: Pearson Education.

Pallant, J. (2007). *SPSS survival manual: A step-by-step guide to data analysis using SPSS for Windows (3rd. Ed.).* Maidenhead: Open University Press.

Palmer, R.D. & Clark, U. (1963). Hand differentiation and psychological functioning. *Journal of Personality, 31,* 445-461.

Paradise, J. L., Dollaghan, C. A., Campbell, T. F., Feldman, H. M., Bernard, B. S., Colborn, K. et al. (2000). Language, speech sound production, and cognition in three-year-old children in relation to Otitis Media in their first three years of life. *Paediatrics, 105,* 1119-1130.

Paul, R. & Cohen, D. (1984). Outcome of severe disorders of language acquisition. *Journal of Autism and Developmental Disorders, 14,* 405-442.

Picard, A., Heraut, F. C., Bouskraoui, M, Lemoine, M., Lacert, P., & Delattre, J. (1998). Sleep EEG and developmental dysphasia. *Developmental Medicine and Child Neurology, 40,* 595-599.

Poresky, R. H. & Whitsitt, T. M. (2001). Young girls' intelligence and motivation: Links with maternal employment and education but not systems theory. *The Journal of Psychology, 119,* 475-480.

Powell, R. P., & Bishop, D. V. M. (1992). Clumsiness and perceptual problems in children with specific language impairment. *Developmental Medicine and Child Neurology, 34,* 755-765.

Redmond, S. (2005). Differentiating SLI from AD/HD using children's sentence recall and production of past tense morphology. *Clinical Linguistics & Phonetics, 19,* 109–127.

Resing, W. C. M. & Tunteler, E. (2007). Children becoming more intelligent: can the Flynn effect be generalised to other child intelligence tests? *International Journal of Testing, 7,* 191-208.

Resnick, M., Eyler, F., Nelson, R., Eitzman, D., & Bucciarelli, R. (1987). Developmental intervention for low birth weight infants: Improved early developmental outcome. Pediatrics, 80, 68-74.

Rice, M. L., Wexler, K., & Hershberger, S. (1998). Tense over time: The longitudinal course of tense acquisition in children with specific language impairment. *Journal of Speech, Language and Hearing Research, 41,* 1412-1431.

Roberts, L. E., Rosenfeld, R. M., & Zeisel, S. A. (2004) Otitis Media and speech and language: A meta-analysis of prospective studies. *Paediatrics, 113,* 238-248.

Robinson, R. J. (1991). Causes and association of severe and persistent specific speech and language disorders in children. *Developmental Medicine and Child Neurology, 33,* 943-962.

Robinson, R. O., Baird, G., Robinson, G. & Simonoff, E. (2001). Landau-Kleffner syndrome: Course and correlates with outcome. *Developmental Medicine and Child Neurology, 43,* 243-247.

Rodda, M., & Grove, C. (1987). *Language, cognition and deafness*. Hillsdale: Lawrence Erlbaum Associates, Publishers.

Rodriguez, A. & Waldenstrom, U. (2008). Fetal origins of child non-right handedness and mental health. *Journal of Child Psychology and Psychiatry, 49,* 967-976.

Roessner, V. Banachewski, T., Uebel, H., Becker, A., & Rothenberger, A. (2004). Neuronal network models of ADHD – lateralisation with respect to inter-hemispheric connectivity reconsidered. *European Child and Adolescent Psychiatry (Supplement), 1,* 171-179.

Rojas, D. C., Camou, S. L., Reite, M. L., & Rogers, S. J. (2005). Planum temporale volume in children and adolescents with Autism. *Journal of Autism And Developmental Disorders, 35,* 479-486.

Rommelse, N. N. J., Altink, M. E., Oosterlaan, J., Buschgens, C. J. M., Buitelaar, J., De Sonneville, L. M. J., & Sergeant, J. A. (2007). Motor Control in Children with AD/HD and Non-Affected Siblings: Deficits Most Pronounced Using the Left Hand. *Journal of Child Psychology and Psychiatry, 48,* 1071-1079.

Rosenthal, R. & Rosnow, R. L. (2008). *Essentials of behavioural research: Methods and data analysis (3rd Ed.).* New York: McGraw Hill Publishers.

Rossetti, L. M. (2001) *Communication intervention: Birth to three (2nd Ed.).* New York: Thomson Learning.

Ruben, R. (1991). Effectiveness and efficacy of early detection of hearing impairment. *Acta Otolaryngol (Supplement), 482,* 127-131.

Sattler, J. M. & Dumont, R. (2004*). Assessment of children: WISC-IV and WPPSI-IV supplement*. San Diego: Jerome M. Sattler.

Sattler, J. M. (1988). *Assessment of Children (3rd Ed.).* San Diego: Jerome M. Sattler.

Satz, P., Orsini, D. L., Saslow, E., & Henry, R. (1985). The pathological left-handedness syndrome. *Brain and Cognition, 4,* 27-46.

Savage, R. D., Evans, L., & Savage, J. F. (1981). Psychology and communication in deaf children. Sydney: Grune & Statton.

Sears, R. R., Maccoby, E. E., & Levin, H. (1976). Patterns of child-rearing. Stanford: Stanford University Press.

Siegler, R. (1996). *Emerging minds: The process of change in children's thinking*. New York: Oxford University Press.

Slykerman, R. F., Thompson, J. M. D., Becroft, D. M., O., Robinson, E., Prior, J. E., Clark, P. M., Wild, C. J., & Mitchell, M. A. (2005). Breastfeeding and intelligence of preschool children. *Acta Pædiatrica, 94*, 832–837

Snowling, M. J., Bishop, D. V. M., Stothard, S. E., Chipchase, B. & Kaplan, C. (2007). Psychosocial outcomes at 15 years of children with a pre-school history of speech and language impairment. *Journal of Child Psychology and Psychiatry, 47,* 759 – 765.

Soellart, B. M. (2003). A comparison of the performance of normal black South African and British infants on the Griffiths Scales of Mental Development. (Unpublished Masters thesis). University of the Witwatersrand, Johannesburg.

Spiker, D., Ferguson, J., & Brooks-Gunn, J. (1993). Enhancing maternal interactive behaviour and child social competence on low birthweight, premature infants. *Child Development, 64,* 754-768.

Sweeney, K. (1994). *Cluster analysis of the Griffiths profiles of a white South African clinical population. (*Unpublished Masters Thesis), University of Port Elizabeth, Port Elizabeth.

Talge, N. M., Neal, C., & Glover, V. (2007). Antenatal maternal stress and long term effects on child neurodevelopment: how and why? *Journal of Child Psychology and Psychiatry, 48,* 245-261.

Taylor-Flusburg, H. (2004). Do Autism and Specific Language Impairment represent overlapping language disorders? In M. L. Rice & S. F. Warren (Eds.) *Developmental language disorders: From phenotypes to aetiologies* (pp. 31-52). Mahwah: Lawrence Erlbaum Associates Inc.

Thomas, S. C., Grant, J., Barham, Z., Gsodl, M., Laing, E., Lakusta, L., Tyler, L. K., Grice, S., Paterson, S., & Karmiloff-Smith, A. (2001). Past tense formation in Williams Syndrome. *Language and Cognitive Processes, 16,* 143-176.

Tomblin, J. B., Records, N., Buckwalter, P., Zhang, X., Smith, E., & O'Brein, M. (1997). Prevalence of Specific Language Impairment in Kindergarten Children. *Journal of Speech, Language, and Hearing Research*, 40, 1245-1260

Torrioli, M. G., Frisone, M. F., Bonvini, L., Luciano, R., Pasca, M. G., Lepori, R., Tortorolo, G., & Guzzetta, F. (2000). Perceptual-motor, visual and cognitive ability in very low birthweight preschool children without neonatal sound abnormalities. *Brain & Development, 22,* 163-168.

Tredoux, C. & Durrheim, K. (Eds.) (2002*). Numbers, hypotheses & conclusions: course in statistics for the social sciences.* Lansdowne: UCT Press.

van den Elzen, A. P., Semmekrot, B. A., Bongers, E. M., Huygen, P. L., & Marres, H. A. (2001). Diagnosis and treatment of the Pierre Robin sequence: results of a retrospective clinical study and review of the literature. *European Journal of Paediatrics, 160,* 47–53

Volonté, L. (2010). *Investing in family cohesion as a development factor in times of crisis (Doc 12103).* Report: Social, health and family affairs committee: Parliamentary Assembly of the European Union. Downloaded from http://assembly.coe.int/Main.asp?link=/Documents/WorkingDocs/Doc10/EDOC12103.htm on the 11 October 2010.

Vygotsky, L. S. (1978). Mind in society: The development of higher psychological processes. Boston: Harvard University Press.

Wachs, T. D. & McCabe, G. (2001). Relation of maternal intelligence and schooling to offspring nutritional intake. *International Journal of Behavioural Development, 25,* 444-449.

Warburton, P., Baird, G., Chen, W., Morris, K., Jacobs, W. B., Hodgson, S., & Docherty, Z. (2000). Support for linkage in Autism and specific language impairment to 7q3 from two chromosome

rearrangements involving bad 7q31. *American Journal of Medical Genetics (Neuropsychoatric Genetics), 96,* 228-234.

Wechsler, D. *The Wechsler Preschool and Primary Scale of Intelligence (WPPSI™-III).*

Weisglas-Kuperus, N., Baerts, W., de Graaf, M. A., van Zanten, G. A., & Sauer, P. J. J. (1993). Hearing and language in preschool very low birthweight children. *International Journal of Paediatric Otorhinolaryngology, 26,* 129-140

Weismer, S. E. (1997). Typical talkers, late-talkers and children with Specific Language Impairment: A language endowment spectrum? In R. Paul (Ed.) *Language disorders from a developmental perspective: Essays in honour of Robin S. Chapman* (pp. 83-102). Mahwah: Lawrence Erlbaum Associates.

Wellman, M. M. (1983). Variations in hand position, cerebral lateralization, and reading ability among right-handed children. *Brain and Language, 18,* 277-292.

Wellman, M. M. (1985). Information-processing abilities among left- and right-handed children. *Developmental Neuropsychology, 1,* 53-65.

Wise, S. (2003). Family structure, child outcomes and environmental mediators: an overview of the Development in Diverse Families Study. Research paper no. 30. Australian Institute of Family Studies.

Zelaznik, H. N. & Goffman, L. (2010). Generalized Motor Abilities and Timing Behaviour in Children With Specific Language Impairment. *Journal of Speech, Language, and Hearing Research, 53,* 383–393.

Appendix A

Ethics Certificate

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (NON MEDICAL)
R14/49 Cockcroft

CLEARANCE CERTIFICATE	**PROTOCOL NUMBER H100 514**
PROJECT	Centre for language and hearing impaired children data management and research system
INVESTIGATORS	Prof K Cockcroft
DEPARTMENT	Psychology
DATE CONSIDERED	14.05.2010
DECISION OF THE COMMITTEE*	Approved Unconditionally
NOTE:	

Unless otherwise specified this ethical clearance is valid for 2 years and may be renewed upon application

DATE 01.06.2010 **CHAIRPERSON** .....
(Professor R Thornton)

cc: Supervisor :

.....

.....

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10005, 10th Floor, Senate House, University.

I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

_____
Signature

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

Appendix B

Description of the WPPSI-III Subtests

Information	This verbal test is designed to assess the child's ability to acquire, retain and retrieve general factual knowledge. It draws on crystallised intelligence, long-term-memory and environmental retrieval. Auditory perception, comprehension and verbal expressive ability are also drawn on.
Vocabulary:	This test assesses a child's word knowledge and verbal concept formation, their knowledge 'bank', learning ability, long-term memory and degree of language development. Other abilities also utilised include auditory perception and comprehension, verbal conceptualisation, abstract thinking and verbal expression.
Word Reasoning	This new verbal subtest of the WPPSI-III is related to tasks measuring verbal reasoning, verbal comprehension, analogic and general reasoning ability, the ability to synthesise and integrate different types of information, verbal abstraction, domain knowledge, and the ability to generate alternative concepts.
Comprehension	This is a supplemental verbal subtest for the older age-band that assesses verbal reasoning and conceptualisation, the ability to evaluate and utilise past experiences, verbal comprehension and expression and the ability to demonstrate practical information. It also requires a degree of knowledge about conventional standards of behaviour, social judgment, maturity and common sense.
Similarities	This is also a supplemental verbal subtest for the older age-group that measure verbal reasoning and concept formation. It relies on auditory comprehension, memory, distinguishing between essential and non-essential features and verbal expression.
Receptive Vocabulary	This is a core subtest for the younger group and an optional verbal test for the older one. It assesses the child's ability to comprehend verbal directives, auditory and visual discrimination, auditory memory, auditory processing and the integration of visual perception and auditory input. Responses may also be influenced by phonological memory and working memory.
Picture Naming	This is a supplemental test for the younger group and an optional test for the older ones. It assesses expressive language ability, world retrieval from long-term memory, and association of visual stimuli with language.

Block Design	This is one of the cores Performance tests and is designed to measure the ability to analyse and synthesise abstract visual stimuli. It also draws on non-verbal concept formation, visual perception and organisation, simultaneous processing, visual-motor coordination, learning and the ability to separate figure and ground in visual stimuli.
Matrix Reasoning	This is also a core Performance test for the older group and taps into the fluid intelligence, and a subsequent reliable estimate of general intelligence, of the child in a way that is similar to Raven's Progressive Matrices. These matrix reasoning questions are believed to be largely culture-fair and language fee and require no hand-motor manipulation. They require the child to establish and conceptualise generalised rules of logic and succession within continuous and discrete pattern completion, classification, analogical reasoning and serial reasoning.
Picture Concepts	This core Performance test for the older group is a new subtest that is designed to measure abstract, categorical reasoning ability. The answers are attained through mental manipulation and reasoning behind concrete or abstract representations.
Picture Completion	This is a supplemental subtest for the older group and measures visual perception and organisation, concentration and visual recognition of essential details of objects.
Object Assembly	This is a supplemental subtest of the older group and a core test within the younger ages. It assesses visual-perceptual organisation, integration and synthesis of part-whole relationships, non-verbal reasoning and trail-and-error learning. It also draws on the child's spatial ability, visual-motor co-ordination, cognitive flexibility and persistence.
Symbol Search	This test makes up the Composite scale of the PSQ, and requires the child to make use of their short-term visual memory, visual motor co-ordination, cognitive flexibility, visual discrimination and concentration. It also taps auditory comprehension, perceptual organisation, and planning and learning abilities.
Coding	This test also forms a core subtest of the Processing Speed Composite for the older bracket. It seeks to access short-term memory, learning ability, visual perception, visual motor coordination, visual scanning ability, cognitive flexibility and motivation. It can also draw on visual and sequential processing ability which is an advanced skill to the lower ages of the targeted age-group.

Adapted from Lichtenberger, E. O., & Kaufman, A. S. (2004) *Essentials of WPPSI-III Assessment.* Hoboken: John Wiley & Sons, Inc.

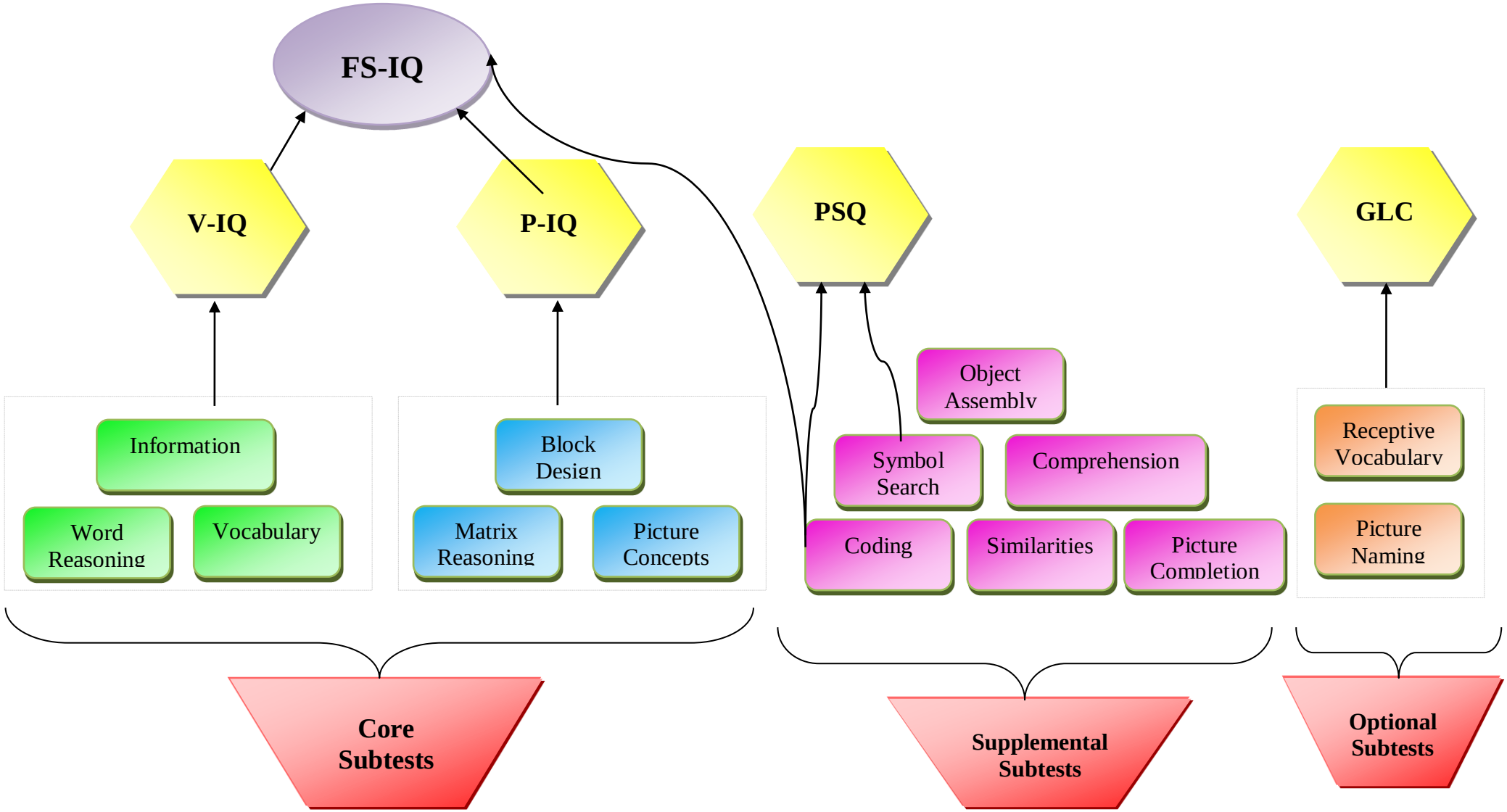


Figure 1.WPPSI-III Building Blocks for ages 4-0 to 7-3.
Note. FS-IQ = Full Scale IQ; V-IQ = Verbal IQ; P-IQ = Performance IQ; PSQ = Performance Speed Quotient; GLC = General Language Quotient.

Taken from Lichtenberger, E. O., & Kaufman, A. S. (2004) *Essentials of WPPSI-III Assessment.* Hoboken: John Wiley & Sons, Inc.

Appendix C

Description of the JSAIS Subtests

Verbal Subtest	
Vocabulary	This test assesses the child's recognition, comprehension, and interpretation of verbal symbols, the retrieval of associations from memory and the differentiation of different or ambiguous meanings.
Ready Knowledge	This test measures a child's general knowledge, facts needed for effective functioning within their everyday environments. Further, it requires the child to retrieve associations from long-term memory, productive language ability, reality orientation and indicative reasoning ability.
Story Memory	The aim of this test is to measure short-term memory for meaningful verbal material.
Number and Quantity Concepts	This subtest requires the child to manipulate quantitative material in a concrete way. It requires the child to count, apply basic mathematical processes, the evaluation of quantity, the comprehension of relational terms and spatial ordering.
Picture Riddles	This test measures concrete-practical reasoning, the comprehension of language stimuli, the construction of hypotheses and the re-interpretation of ideas and symbols to satisfy particular criteria.
Social Reasoning	This test assesses the child's capacity to understand their environment and its social norms and age-appropriate judgments accordingly. It relies on a degree of verbal understanding and fluency and is believed to be biased towards culture.
Word Association	This test requires the child to make relational associations in terms of verbal stimuli, and draw on conceptual thinking and

verbal fluency.

Performance Subtests	
Absurdities A: Missing Parts	This test assesses the child's ability to judge the correctedness of units of figural information, and draws on skills like visual memory for objects and environmental details, and visual discrimination.
Absurdities B: Absurd Situations	This test draws on the child's ability to notice absurdities, evaluate figural systems and discriminate visual material.
Form Discrimination	This test draws on similar skills drawn on in other subtests and relies on the child's ability to discriminate visual differences and similarities. It also relies on processes of form perception, spatial orientation, perceptual constancy, perceptual organisation and visual reasoning ability.
Block Design	This subtest measures the child's visual spatial reasoning, similarity identification, pattern analysis and re-coding, abstract conceptualisation, the ability to generalise, visual motor co-ordination, visual-motor speed and perception of spatial relationships.
Form Board	This test assesses a child's ability to perceive figural systems and transformations with insight. Successful performance requires the recognition and manipulation of 3D stimuli, part-whole relationship, form discrimination, visual organisational ability and psychomotor dexterity.
Memory for Digits	This subtest assesses auditory sequential working memory and mental reorganisation of stimuli.

Adapted from Madge, E. M. (1981). *Manual for the Junior South African Individual Scales.* Pretoria: Health Sciences Research Council.

Appendix D

Description of the Subscales of the Griffiths Scales of Mental Development

Scale A: Locomotor	This subscale allows the examiner to assess the child's gross motor skills including the ability to balance and coordinate and control movements. The items administered include age-appropriate activities such as walking up and down stairs, kicking a ball, riding a bike, jumping and skipping.
Scale B: Personal Social	The subscale assesses the child's proficiency in the activities of daily living, his level of independence and his ability to interact with other children. The items administered include age-appropriate activities such as dressing and undressing, competency using cutlery and knowledge of information such as date of birth and address.
Scale C: Hearing and Language	This subscale allows the examiner to assess the child's receptive and expressive language. The items administered include age-appropriate items such as naming objects and colours, repeating sentences, describing a picture and answering a series of questions about comprehension/similarities/differences.
Scale D: Eye and Hand Co-ordination	This subscale assesses the child's fine motor skills, manual dexterity and visual monitoring skills. The items administered include age-appropriate items such as threading beads, cutting with scissors, copying shapes and writing letters and numbers.
Scale E: Performance	This subscale allows the examiner to assess the child's visual-spatial skills, including the speed of working and precision. The items administered include age-appropriate activities such as building bridges and stairs, completion of form boards and pattern-making.

Subscale F: Practical-Reasoning	This subscale assesses the child's ability to solve practical problems, his understanding of basic mathematical concepts and questions about moral ad sequential issues. The items administered include age-appropriate activities such as counting and comparison of size, length and height. This subscale also assesses the child's knowledge of the days of the week, his visual sequential skills and his understanding of right and wrong.

Appendix E

Intake Forms for the Pre-Primary School

Table E1

Procedure for scoring independent variables

Demographic Information:						
Child Age		Years		months		continuous
Gender	male	female				categorical
Race	coloured	Black	Indian	white		categorical
Home Language	English	Afrikaans	isiZulu	Other		categorical
SES	Unemployed	Manual Labour	Clerical	Artisan	Professional	categorical
Parental Occupation						categorical
Family Structure:						
Categorisation of single or nuclear parent families				single	nuclear	categorical
A fraction measuring birth-order/no. of children in family			e.g. 1/3			continuous
Family Attitudes & Support:						
(1 = completely unsupportive. 5 = very supportive)						
(Allocation of 1 if yes and 0 if no and then added together to get an overall indicator)						
Family feelings on child's difficulties				1-2-3-4-5		Rating scale
Good family relationships				1-2-3-4-5		Rating scale
Relationship and support from grandparents				1-2-3-4-5		Rating scale
The presence of additional carers				yes	no	categorical
Previous family history of similar illnesses				yes	no	categorical
						Final Indicator
Age of symptom identification						continuous
Age of Intervention						continuous
Types of therapies received						categorical
Diagnosis						categorical
Medications						categorical

Maternal Health Pre-Pregnancy

(Allocation of 1 if yes and 0 if no and then added together to get an overall indicator)

Planned pregnancy or not?	yes	no	categorical	
Previous miscarriages and stillbirths?	yes	no		
Mother over the age of 35? (allocate 0.5 point if over 30)	yes	no		
Health of mother (on a 5 point scale)	1-2-3-4-5			
			Final Indicator	

Maternal Health Peri-Pregnancy

(Allocation of 1 if yes and 0 if no and then added together to get an overall indicator)

Condition of pregnancy	1-2-3-4-5			
Complications	yes	no		
Length of pregnancy to nearest week		weeks		
(add one point for every week below 38)				
Any surgical/medical treatment prescribed	yes	no		
Mother smoke during pregnancy	yes	no		
Mother consume alcohol during pregnancy	yes	no		
Mother drug use during pregnancy	yes	no		
Blood incompatibility	yes	no		
Length of labour		hours		
(add a point if abnormally long)				
Type of delivery			categorical	
(add a point if forceps or suction, placenta previ)				
Presentation			categorical	
			Final Indicator	

Birth-weight – continuous		kg	continuous	

Neonatal Health of Infant:

(Allocation of 1 if yes and 0 if no and then added together to get an overall indicator)

First Cry immediate	yes	no	categorical	Negative Scoring	
Stiffness/floppiness	yes	no	categorical		
Notable birth injuries	yes	no	categorical		
Jaundice	yes	no	categorical		
Normal Breathing	yes	no	categorical	Negative Scoring	
Need for incubator/oxygen/lights	yes	no	categorical		
Blood irregularities	yes	no	categorical		
Condition of child at birth	1-2-3-4-5		Rating scale		
Condition of child after birth	1-2-3-4-5		Rating scale		
Treatments	yes	no	categorical	Negative Scoring	
				Final Indicator	

Developmental Milestones

(There will be 1 point added for every unit delay beyond normal milestones)

Speech & Language:

Indicate awareness of maternal voice		months	categorical		
Babbled		months	categorical		
Said first words		months	categorical		
Said first sentences		months	categorical		
				Final Indicator	

Motor Milestones:

Age at which child sat:		months	categorical		
Crawled		months	categorical		
Walked		months	categorical		
				Final Indicator	

Toilet-Training:

Toilet-trained		months	categorical		
Bladder control		months	categorical		
Bowel control		months	categorical		
				Final Indicator	

Sleep:

Age at which child slept through		Months	continuous		

Socialisation & Play:

(Negative scoring will apply here)

Use of creative materials	yes	no	categorical	
Use of toys	yes	no	categorical	
Invention of imaginary scenes	yes	no	categorical	
Danger to themselves	yes	no	categorical	
Puts things away	yes	no	categorical	
Dependent on adult for attention during play	yes	no	categorical	
Socialisation with peers	1-2-3-4-5		Rating scale	
Socialisation with siblings	1-2-3-4-5		Rating scale	
Socialisation with teachers	1-2-3-4-5		Rating scale	
Identify with either parent, role-players	yes	no	categorical	
			Final Indicator	

Behaviour:

(Allocation of a 1 if positive, 0 is negative)

Aggression		yes	no	categorical	
Destructive behavior		yes	no	categorical	
Wetting		yes	no	categorical	
Soiling		yes	no	categorical	
Crying		yes	no	categorical	
Fear of	doctors/nurses	yes	no	categorical	
	objects	yes	no	categorical	
	dark	yes	no	categorical	
	loud noises	yes	no	categorical	
	storms	yes	no	categorical	
	nightmares	yes	no	categorical	
Shyness		yes	no	categorical	
Sleeplessness		yes	no	categorical	
Stealing		yes	no	categorical	
Temper-tantrums		yes	no	categorical	
Thumb/Object sucking		yes	no	categorical	
Whining		yes	no	categorical	
Jealousy		yes	no	categorical	
Masturbation		yes	no	categorical	

Hair Plucking	yes	no	categorical		
Head Banging	yes	no	categorical		
Frustration Behaviour	yes	no	categorical		
Cross-Dressing	yes	no	categorical		
				Final Indicator	
Handedness:					
Right/Left/Undecided			categorical		

Appendix F

Table F1

Kolmolgorov-Smirnoff Tests of Normality for the WPPSI, JSAIS & Griffiths

		Statistic (D)	p value
WPPSI	VIQ	0.0625	> 0.15
	PIQ	0.0784	> 0.15
	FSIQ	0.0446	> 0.15
JSAIS	VIQ	0.1155	> 0.15
	PIQ	0.1044	> 0.15
	FSIQ	0.1375	> 0.15
Griffiths	Locomotor	0.0938	> 0.15
	Personal-Social		
	Speech & Hearing		
	Eye-hand Coordination	0.0998	> 0.15
	Performance	0.0661	> 0.15
	Practical Reasoning	0.1201	> 0.15

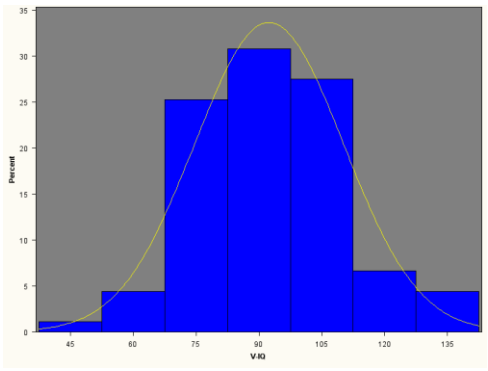


Figure F1. Histogram of WPPSI VIQ

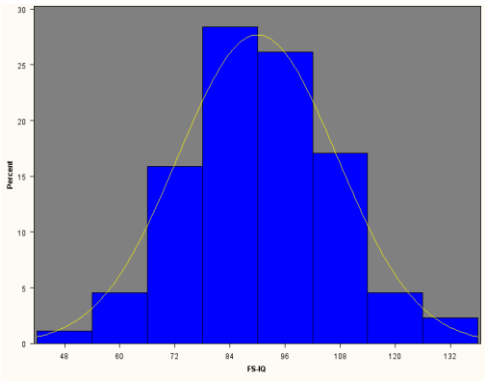


Figure F2. Histogram of the WPPSI FSIQ

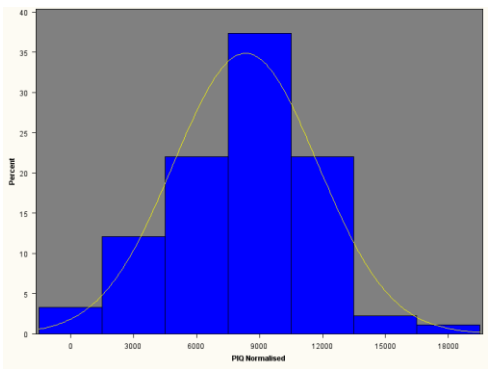


Figure F3. Histogram of WPPSI PIQ

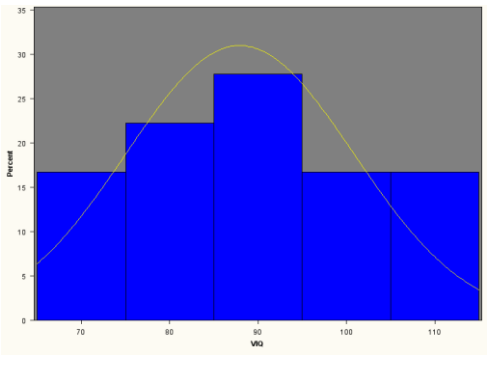


Figure F4. Histogram of JSAIS VIQ

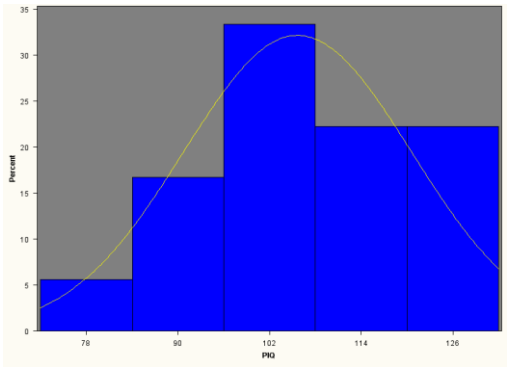


Figure F5. Histogram of JSAIS PIQ

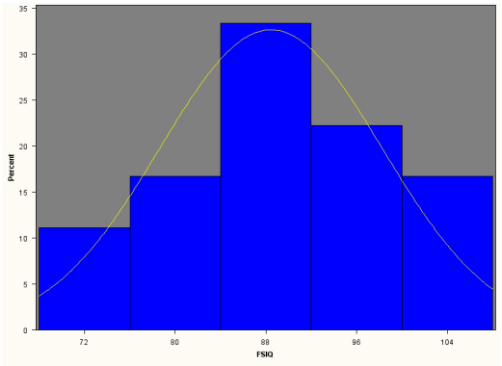


Figure F6. Histogram of JSAIS FSIQ

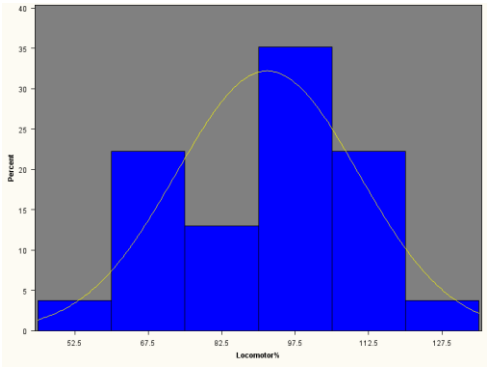


Figure F7. Histogram of Locomotor Subscale

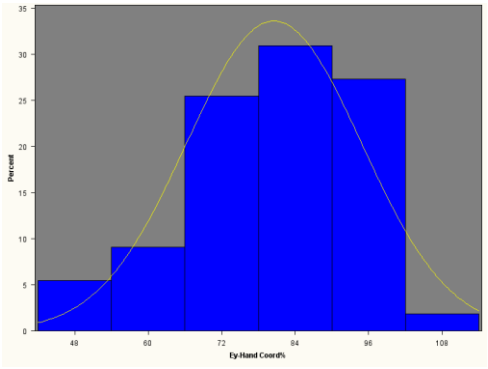


Figure F8. Histogram Eye-Hand Subscale

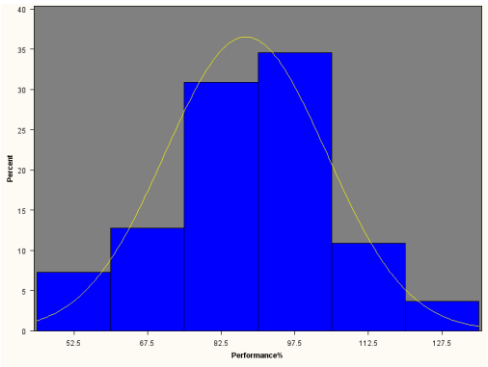


*Figure F9.*Histogram of Performance Subscale

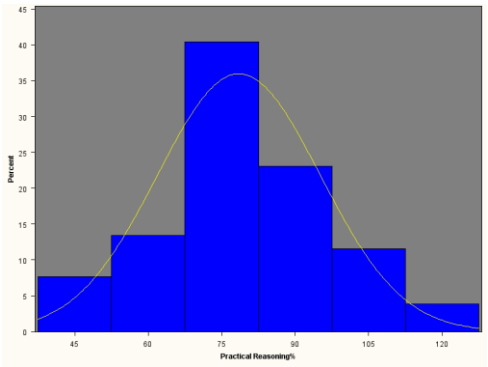
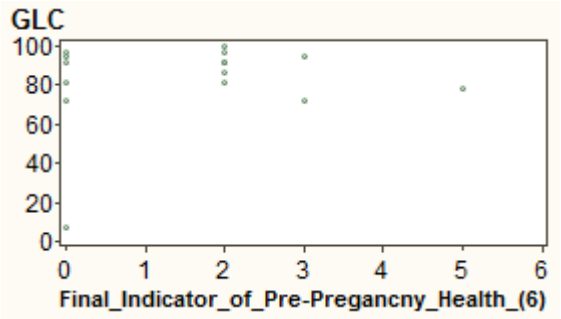


Figure F10. Histogram of Practical Reasoning Subscale


GLC
100
80
60
40
20
0
0
1
2
3
4
5
6
Final_Indicator_of_Pre-Pregancny_Health_(6)