A Comparison of Working Memory Profiles in HIV-Infected and HIV-Exposed Uninfected Children

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Abstract

Conventional psychometric measures, such as the IQ score, have significant limitations in addressing the assessment needs of linguistically and culturally diverse communities. In response, working memory assessment has been identified as a promising alternative to these constraints. It is a better predictor of scholastic success than IQ, and is essential in the acquisition of fundamental literacy and numeracy concepts in school beginners. While there is a lot of theoretical and empirical support for working memory performance in typically developing populations, less is known about its functioning in the context of atypical development; particularly in children who are infected with, or exposed to HIV in utero. This study compared the working memory (AWMA) and general neuropsychological functioning (NEPSY-II) of 273 South African school beginners (6-8 years). The sample consisted of both HIV-infected (n = 95), and HIV-exposed (n = 86) children, as well as an uninfected, unexposed typically developing control group (n = 92). Significant differences were found between the three groups on measures of working memory and general neurocognitive functioning, where the processing component of working memory appeared to be particularly impaired in the two HIV-affected atypical groups. A within-group analysis of the relative strengths and weaknesses of each of the three groups showed that both storage and processing skills in the verbal domain appeared to be general weaknesses, while visuospatial working memory was a relative strength. The former is believed to be influenced by issues of linguistic test bias in the multilingual sample, while the latter is posited to be a consequence of this very multilingualism, which affords these children an executive functioning advantage. The two HIV-affected samples also showed significant deviations in the structure of their working memory when compared to the typically developing control group. However, within-group structural comparisons of a number of working memory models showed that the four factor model comprising separate components of the verbal and visuospatial simple and processing components of working memory was still favoured, even in conditions of atypical development. The study contributes to the growing body of working memory research by presenting the working memory profiles of HIV-infected and HIV-exposed, uninfected children. It also assists in identifying HIV-exposed, uninfected children as a vulnerable and under-researched clinical group which could benefit from further intervention, as well as foregrounding working memory as a less biased alternative in the assessment of paediatric cognitive functioning.

Keywords: working memory, HIV-infected, HIV-exposed uninfected, neurodevelopment, children.

Declaration

I declare that this dissertation is my own, unaided work. It is being submitted for the degree of Doctor of Philosophy at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at any other university.

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Robyn Milligan (Ms.)

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Chapter 1: Introduction

It has long been the intention of researchers and clinicians to identify the predictors of academic success in younger generations. Since the advent of the twentieth century, the development of the IQ score afforded researchers this opportunity, and has since been considered a universal means of predicting a child's cognitive potential, and subsequent scholastic success. Increasingly, however, the growing cultural heterogeneity of communities around the world has highlighted the limitations and cultural irrelevance of the IQ test, and professionals have been forced to look to other means of assessing and developing cognitive functioning and academic potential (Jensen, 1980; Hilliard, 1984). Over the last two decades, the importance of executive functioning in the prediction of academic success has changed the focus of the measurement of cognitive functioning from that of verbal proficiency and the ability to remember large amounts of culturally located content, to the ability to attend to, control, inhibit and adapt cognition using efficient strategies to problem solve in the here and now (Blair & Peters-Razza, 2007; Hughes & Ensor, 2011). One of the salient cognitive constructs reliant on executive functioning is working memory which has evolved rapidly as a front runner to replacing IQ (Alloway & Capello, 2013).

The debate regarding the definition of working memory is vast, but can be understood simply as the brain's ability to consciously and temporarily store and manipulate information; a skill essential to people of all ages, in a variety of contexts. The popularity of working memory is increasing because of its importance in everyday functioning; for example remembering an email address, following a map, and learning to drive (Alloway, 2006). It has also been found to be an excellent predictor of academic success, and a better measure of this than the traditional IQ score (Alloway & Gregory, 2013; Alloway & Alloway, 2013). It has reduced cultural and socioeconomic bias when compared to conventional intelligence and scholastic measures (Alloway & Capello, 2013), has externally valid assessments in many language groups (Leseman, Mayo, & Scheele, 2010; Messer, Leseman, Boom, & Mayo, 2011), and is understood to be reliably measurable in children as young as four years old (Alloway, Gathercole, Willis, & Adams, 2004).

There is a growing, and well-established knowledge base on the working memory functioning of typically developing children, three aspects of which will be summarised briefly. Firstly, within typically developing children, working memory is fractionated which

means that it is not a unitary construct (similar to the initial conceptualisation of IQ as representing an underlying 'g'), and takes on a modular structure with a number of inter-related components which work together. This fractionation extends into adulthood, but its structure is not consistent during childhood, and the components have differential rates of maturity at different stages of development. The number of discrete components, and their relative contribution to the larger success of the working memory system differ as the construct matures according to neuroanatomical and functional changes in the developing brain (Alloway & Gathercole, 2006a; Alloway & Alloway, 2013; Jarvis & Gathercole, 2003). The second is that working memory capacity is limited, but remains stable over time. Regardless of the model used to explain the functioning of working memory, most would attest to some explanation of where the model fails to store and process increasing volumes of information. Unlike the conceptualisation of intelligence by an IQ score, where high performers are capable of holding an almost infinite amount of information in long-term memory, working memory is unconcerned with content retention and focuses instead on the ability to problem solve complex cognitive adaptations in the here and now. This capacity is limited by processes of decay and interference (Miyake & Shah, 1994). Despite these limits in volume, working memory capacity is believed to remain stable over time, and will increase incrementally with age until it reaches adult capacity around the age of 15 years (Gathercole, Pickering, Ambridge & Wearing, 2004). Thirdly, working memory is strongly associated with academic success, and remains a powerful cognitive skill related to measures of reading, writing, spelling, mental arithmetic, measurement and spatial abilities, and computational scores in both typical and atypical school-going children (Alloway & Copello, 2013; Berninger & Swanson, 1994, 1995; Caramazza, Miceli, Villa, & Romani, 1987; DeStefano & LeFevre, 2004; Margolin, 1984; Swanson & Sachse-Lee, 2001; Swanson, Saez, Gerber, & Laefsedt, 2004).

Working memory research has also provided evidence for the way in which this ability deviates in atypically developing populations. A large collection of studies have profiled working memory in children with Attention Deficit/Hyperactivity Disorder (AD/HD), specific language impairment (SLI), Autistic Spectrum Disorder (ASD), Downs Syndrome, Williams Syndrome, developmental coordination disorder, dyslexia, dyscalculia, and general intellectual disabilities (Alloway, 2006b; Alloway & Gathercole, 2006b; Gathercole & Baddeley, 1990; Henry, 2012; Lazar & Frank, 1998; Passolunghi & Siegel, 2001, 2004;

Rucklidge & Tannock, 2002; Steele, Minshew, Luna, & Sweeney, 2007; Swanson, 2006). This research has provided evidence of the fractionation of working memory in these populations, and the relative deficits present in a variety of working memory abilities; phonological storage and processing, broad spectrum executive functioning, and visuospatial storage. While this evidence does converge to identify particular strengths and weaknesses within these samples, very little of this research measures working memory comprehensively. Instead, components of the construct are assessed separately, with little replication in the psychometric measure employed, or control of the severity of impairment. Hence, an understanding of working memory functioning within atypical samples is often secondary to the investigation of another cognitive construct.

The investigation of working memory in atypical populations is further complicated by issues of equipotentiality. While there are a number of shared symptoms and areas of impairment common to different atypical populations, these deficits do not necessarily have the same underlying etiological brain structure, yet could respond to cognitive intervention in a similar way. Conventionally, the difficulties experienced by atypical populations are understood by reified diagnostic categories which could limit the ways in which practitioners attempt to remediate deficits. By way of example, the working memory profiles of children with developmental coordination disorder and those with ADHD both have common visuospatial span difficulties (Alloway & Gathercole, 2006b; Alloway, 2007). Yet, a common working memory intervention would not be a typical treatment response, as impairment in the one population is conceptualised as a motor disorder, while the other is understood as a disorder of attention. In response, the proposition of 'endophenotypes' in the conceptualisation of cognitive profiling in atypical samples is particularly helpful because it disputes the often conflating classifications related to etiology, and instead focuses on common understandings which assist in functional remediation (Bishop, 2006).

Empirical support for the abovementioned praxis is further limited by a lack of generalisability to children from regions in the world with the largest child populations; sub-Saharan Africa (Population Reference Bureau, 2014). The large majority of studies profiling the working memory functioning of atypically developing children have been done in Western contexts where children predominantly speak English as their first language, attend English-medium schools and have a familiarity with standardised testing and assessment. They also usually come from homogenous middle-class communities, and have access to

basic needs and appropriate, specialised medical care. In contrast, the application of findings from these studies to non-Western contexts is limited by compounding factors within often resource-deprived child populations, such as poverty, malnutrition, parental unemployment and the stark lack of access to resources which are known to negatively affect developmental outcomes (Feinstein, 2003; Paxson & Schady, 2015).

Debate regarding the contextual influence of a disadvantaged environment on cognitive development is particularly evident in the context of HIV within Sub-Saharan Africa. Research regarding HIV is commonly vexed by a series of conflating, extraneous variables endemic to HIV infection around the world, where it is predominantly found in contexts plagued by poverty, poor education, unemployment, and differential access to resources and health systems. This context is also oft associated with lower neurodevelopmental outcomes which often compounds the disabling effect of viral infection or exposure within the family and larger community (Salter-Goldie, DeMatteo, King, Wells, and the Multisite Co-investigators, 1997, as cited by Blanchette et al., 2002).

Even without consideration of these environmental influences, it is well established that the HI virus itself has a compromising and detrimental effect on neurodevelopment. HIV associated encephalopathy is common in HIV infection, with deficits apparent in motor, linguistic and cognitive domains (Whitehead, 2012). While there do exist a handful of studies which show no significant functional impairment within these samples (Bagenda et al., 2006; Blanchette, Smith, King, Fernandes-Penney, & Read, 2002), they appear to have small sample sizes, or are recognised as belonging to a rare profile of HIV progression characterised by long periods of latent asymptomaticity (Webster, 2009).

There is also a large, and increasingly specific body of research across scientific domains which considers the effect of antiretroviral therapy on neurocognitive functioning. To date, the findings remain equivocal, with outcomes from empirically sound studies around the world varying in their indication that antiretroviral drugs have particularly damaging side effects to neurodevelopmental outcomes (Jeremy, Kim, Nozyce, Nachman, McIntish, Pelton, Togev et al., 2005), to those which advocate that treatment has resulted in the delay of otherwise certain neuro-functional impairment (Koekkoek, de Sonneville , Wolfs, Licht, & Geelen, 2008). Research in this domain is plagued by a number of methodological limitations which challenge the trusted pillars of scientific empiricism. These

include an almost impossibly large number of regimen combination permutations investigated across studies which make comparable review nearly impossible, differences in the clade of HIV being investigated (which differs according to geographical region), contextual and socio-demographic influences which conflate treatment outcomes (poverty, gender relationships) and differing treatment approaches to drug administration around the world (driven largely by socio-political and economic beliefs regarding public health). Further, the empirical study of antiretroviral therapy (ART) effectiveness is also faced with ethical challenges, as clinical-researchers remain unable to isolate the effect on prevention of mother to child (PMTCT) prophylaxis on young children, where it is illegal and unconscionable to provide differentiable levels of care in the context of paediatric HIV.

The intersection of these two ideas, the effect of HIV infection, and the effect of ART exposure on neurodevelopment, is located in the growing interest in research to HIV exposure, particularly the HIV-EU (HIV-Exposed, Uninfected) child. Due to increases in the effectiveness of administration of ART prophylaxis, the HIV-EU child population are rapidly dwarfing the number of children born with HIV infection, and will soon be an under-researched population of interest within the fields of health and development (Filteau, 2009) The HIV-EU child is believed to have a unique neuroimmunological profile as they are exposed to the immunological side effects of HIV in utero (as a result of immune activation in the mother), as well as exposure to the prophylactic, often damaging effects of PMTCT regimens, yet remain uninfected themselves (Bunders, et al., 2006; Garay & McAllister, 2010; Nyoka, 2008). There has been limited research into the general functioning and health of this often overlooked population. Generally, there is strong evidence to support the idea that this group have an altered physiological profile, with significant differences identified in infant growth outcomes, haematological parameters, abnormalities in immunological cells and an increased incidence of metabolic dysfunction (Claudio et al., 2013; Kuhn, Meddows-Taylor, Gray, & Tiemessen, 2001; Le Chenadec, Mayaux, Guihenneuc-Jouyaux, & Blanche, 2003). Recent efforts to investigate the relative neurocognitive functioning in the context of HIV-exposure have produced equivocal findings. Some research has indicated that their neurodevelopment falls within similar ranges to that of typically developing children (Kandawasvika et al.,2015), while others have noticed significant degrees of impairment in areas of verbal performance, sequencing and the verbal, memory and quantitative indices of the McCarthy's Scales (Brackis-Kott et al., 2009; Kerr et al., 2014; Levenson et al., 1992). This

research however, is often done on very young children, using crude measures of cognitive functioning.

The neurodevelopmental profile of the HIV-EU child is therefore a valid area of potential investigation to the psychological research community. This study sought to investigate this, with a particular emphasis on working memory, as it is essential to the acquisition of literacy and numeracy skills in foundation phase education, as well as everyday functioning. It is also a far more valid measure of predictive school performance than IQ testing, and is less pervious to the effects of socioeconomic status (SES) and linguistic proficiency (Campbell, Dollaghan, Needlemen, &, Janosky, 1997; Engel, Dos Santos &, Gathercole, 2008; Laing & Kamhi, 2003). This is particularly important as psychometric assessment has inherent biases toward samples which fall outside of the Western, English speaking norm of standardised testing.

With regards to working memory, the study sought to investigate two axes of interest. The first was a between-group comparison of three groups of children to identify salient nodes of difference between the HIV-infected (HIV-I) and HIV-exposed uninfected (HIV-EU) groups in comparison to an unexposed control (HIV-UU) group. The second was an internal within-group profiling of the strengths and weaknesses within each group. The working memory structure of the two HIV-affected (HIV-I and HIV-EU) groups were also compared against theoretically expected structural models in an attempt to understand the degree to which the maturation of the construct was similar to those of typically developing children of the same age. The study also assessed general neurocognitive functioning in a smaller sub-sample to investigate the broader performance profile within the following cognitive domains: attention and executive functioning, language, memory and learning, sensorimotor abilities, social perception and visuospatial processing.

Theoretically, the study makes a contribution to the growing field of working memory as a competitive alternative to IQ testing in the assessment of paediatric cognitive functioning. Moreover, it does this with a child population who are distinct in two ways. Firstly, it considers the working memory profiles of two atypical paediatric populations; those of HIV infection, and HIV exposure. While these groups would conventionally not be considered under the same umbrella as that of neurodevelopmental disorders, there are a number of salient cognitive similarities between these two groups and other atypical

populations with cognitive difficulties. To that end, the conceptualisation of these together within the 'endophenotypic' paradigm proposed by Bishop (2006), positions this research as an integrated and complementary addition to the existing research on other neurodevelopmental disorders. Secondly, the research considers the working memory profiles of children who do not speak English as their first language, and who come from resource-deprived, culturally-heterogeneous communities which are vastly removed from the context wherein the majority of this field of research has been drawn from in the past. While there are limitations in the methods of psychometric testing within samples such as these, the findings have a far-reaching applicability to the very many children who face similar difficulties, come from similar communities in sub-Saharan Africa, but whose predicaments remain under-researched. Practically, the findings from this study are therefore well-positioned to inform the assessment and remedial intervention of working memory in linguistically diverse, non-Western contexts, particularly those affected by HIV. A chapter summary is presented below.

Chapter 2 outlines the theoretical conceptualisation of working memory as a construct which has only really found substantial empirical traction in the last decade. It begins with a broader consideration of the importance of executive functioning, and then presents working memory as a viable alternative to the limitations of the conventional use of intelligence testing. This introduction of the construct is located within a discussion of its relationship to academic functioning, and its relative robustness to the effects of SES and second language testing, although less so to the latter. The discussion then presents a historical development of the construct, and foregrounds Baddeley's Multicomponent Model as a robust and theoretically sound model to the available empirical evidence in support of working memory functioning. A number of competing models are then critically compared against this model which aim to provide an alternative account of working memory functioning. The chapter concludes with a discussion of the development of working memory through childhood and presents the theoretical expectations of what is predicted by way of working memory structure in typically developing school beginners.

Chapter 3 begins by presenting the working memory profiles of a number of atypical paediatric populations, where HI viral infection and exposure is later positioned within this larger group of paediatric impairment. A thorough discussion of the epidemiological background to HIV, along with a critical overview of the empirical research regarding its

neurocognitive impairment in both adult and paediatric populations follows. The chapter concludes with the presentation of the HIV-EU child, and presents the limited equivocal research regarding typical health and development outcomes regarding this sample, as well as hypotheses regarding why atypical working memory development is likely.

Chapter 4 is a presentation of the methods employed during this study. It begins by exploring the aims of the research in light of the available literature, and then describes the sampling methods and demographic details of the final sample. It then presents the practical and ethical procedures followed in carrying out the data collection, describes the psychometric instruments used and briefly presents the data analytic tools used for statistical analysis.

Chapter 5 documents the statistical outcomes of these analyses in the Results chapter. It begins by describing how the identification of covariates was done, and then how these were used to attempt to control for the effects of extraneous variables. The use of a number of multivariate analyses were then conducted on the larger sample who were only assessed on measures of working memory, and then also on a smaller subset who were also measured on more general neuropsychological outcomes. These analyses included both the identification of between-group differences, as well as a within-group profiling within each clinical group. The chapter then concludes with a final set of analyses which employed confirmatory factor analysis to compare model fit on five theoretically informed working memory structural models. This assisted in identifying the dominant fractionations present at the age, as well as the degree to which these were predictive of typical working memory structures.

Chapter 6 is a critical discussion of the salient findings of the study in relation to broader theoretical issues in the existing knowledge bases. These refer to issues of working memory componential structures and developmental fractionation within both typical and atypical samples. They also refer to the functioning and existence of strengths and weaknesses in the working memory performance of HIV-infected, and HIV-exposed children. The chapter concludes with a discussion of the strengths and weaknesses of the present study, as well as the theoretical and practical contributions which it makes to existing knowledge.

Chapter 2: Working Memory

Introduction

The primary focus of this chapter is the theoretical conceptualisation of working memory. It begins by briefly outlining the construct, and locating it within broader understandings of executive functioning, and the importance of both of these for academic success. The relationship between working memory and the environmental influences of socioeconomic status and language is then discussed. The focus of the chapter shifts to discuss the historical progression of working memory from a basic account of attention and short term storage, to the well-researched and empirically validated Multicomponent Model proposed by Baddeley (1974, 1986, 2000). While this model is foregrounded throughout the study, the discussion also presents three alternative theoretical models of working memory performance and the ways in which they aim to account for the short term storage and processing capacities of the brain. The focus of this theoretical understanding is later directed towards the construct's structure and emphasis in development, particularly the early and middle years of childhood. This chapter ends with a critique of the psychometric measurement of the construct and previous working memory research, as well as a presentation of the theoretically informed expectations of working memory structure in typically developing school beginners.

Working Memory

Working memory is the brain's ability to not only store, but manipulate information. It was initially conceptualised in the 1970's as an active component of short term memory which is capable of directing attention to a task despite interference or distraction (Baddeley & Hitch, 1974). It has largely been recognised as being independent of long term memory stores, yet retains a strong relationship with this essential element of memory. It has also been found to be highly related to other cognitive activities, particularly skills essential for success at school (Alloway & Alloway, 2010; Gathercole, Pickering, Knight &, Stegman, 2004). Because of these connections to long term memory and scholastic success, working memory is often confused as being another form of intelligence, or a collective term for executive functioning. These next two sections differentiate working memory from these two constructs.

Executive Functions.

Increasingly, research within both typical populations and developmental psychopathology has pointed to the importance of executive functioning in understanding the origins of

success and impairment in childhood learning (Bull & Scerif, 2001; Henry, 2012; Swanson, 1999). Despite the growing interest in these skills, there is little consensus about the exact definition of executive functioning, particularly within children. This is largely due to methodological weaknesses in the available empirical research, which is characterised by low construct validity between tasks, and the failure to base executive functioning research within developmentally appropriate frameworks. To this point, the majority of research to date has been guided by existing knowledge regarding frontal lobe functioning in adults and has failed to recognise developmental changes within executive functioning at different stages throughout childhood (Brocki & Bohlin, 2004). In an attempt to provide a compelling theory of executive function which concerns the specific cognitive processes which are controlled and coordinated during complex tasks, Miyake, Friedman and Emerson et al. (2000) propose executive functioning to be 'general purpose control mechanisms that modulate the operation of various cognitive sub-processes and thereby regulate the dynamics of human cognition' (p. 50). The most common executive skills which these authors refer to are described briefly: working memory (sometimes referred to as updating), switching or cognitive flexibility, planning, inhibition and fluency.

Working memory is an almost impossibly difficult construct to define briefly, but generally refers to the conscious and concurrent storage and processing of information. Switching or shifting-set involves moving backwards and forwards between multiple tasks, operations and sets, or moving fluently between different sets of instructions. In order to do this successfully, the process of updating or self-monitoring is used which requires the monitoring and encoding of incoming stimuli, and then appropriately revising items currently held in the working memory system by replacing no-longer relevant information with newer and more relevant information. Planning or problem-solving involves the organisation and planning of a sequence of actions, while inhibition refers to the ability to deliberately inhibit dominant and automatic responses. Lastly, fluency refers to the ability to generate items according to particular rules, and usually involves spontaneous, creative production of stimuli according to a categorisation (Henry, 2012).

One of the key issues to emerge out of executive research is that executive functioning is not a unitary construct, but represents a number of distinct yet related skills. A recent twin study has provided support for this belief. Friedman, Miyake, Young, DeFries, Corely, and Hewitt (2008) showed that executive functions collectively draw on a common,

highly heritable factor which explains why there is a high degree of inter-correlation between these functions. A number of studies have sought to identify discrete operational differences in executive functioning. Within adult samples, Miyake et al. (2000) identified the clearly separable functions of switching, working memory (updating) and inhibition using a confirmatory factor analysis on a group of 137 English speaking undergraduate students from the United States. This factor structure is supported by other studies considering adults (Anderson, 2002; Garon, Bryson, & Smith, 2008; Huizinga, Dolan, & van der Molen, 2006; Lehto, Juujarvi, Kooistra, & Lulkkinen, 2003; van der Sluis, de Jong, & van der Leij, 2007). The convergence of these findings, however, is not absolute, and some do not consider more than three factors in their research. Hence, the identification of these discrete executive functions might be reified (Henry, 2012).

Studies regarding the structure of executive functioning in children have found different results, suggesting that there is a developmental fractionation of the different types of executive functioning that develop at different maturational points. One such study identified only inhibition and updating as separable constructs in a sample of 11-12 year old children, but failed to identify a third distinct executive factor of shifting. Verbal and visuospatial complex working memory were found to have common associations with updating, but not with inhibition (St Clair-Thompson & Gathercole, 2006). While this study clearly showed the separable components of executive functions, it also highlighted the unitary nature of executive functioning within children as well. These findings establish that it is both the common associations between executive functions as well as their discrete differences which supports their complementary and synchronous management of complex cognition.

Another developmental study only identified three factors over three periods of maturation in a study assessing a range of executive functions in three groups of children within three age bands (6-12 years: 6-7.5; 7.6-9.5; 9.6-11.5 year cohorts) (Brocki & Bohlin, 2004). The authors identified inhibition, processing speed/arousal and working memory/fluency as these three discrete factors. The authors do concede that the requirements of the verbal fluency task required analysis, synthesis, strategy generation, and the need to maintain in mind which words have already been said. This latter task placed a strong demand on complex verbal working memory, and, as proposed by a similar study with converging findings (Barkley et al, 2001), could just be named working memory,

without the reference to fluency. Developmentally, this study also found striking advances in the inhibition factor which emerged between 7.6-9.5 years, and 9.6 to 11.5 years. The speed/arousal factor indicated a major gain in development between the first (6-7.5 years) and second age groups (7.6-9.5 years). Performance improvements in the working memory factor were most notable at the ages of 8 and then 12 years (Brocki & Bohlin, 2004). The aforementioned research concluded that executive functioning in both adult and child samples is made up of a number of complementary, yet distinct skills. Importantly, working memory is a consistent product of executive functioning fractionation and identified as only one of these discrete executive skills within the larger group of skills which have differential rates of maturity throughout development.

Working Memory, Academic Attainment and IQ.

These introductory paragraphs have contextualised working memory as an important executive function, but also differentiated it from other executive functions which serve similar functions. Despite executive functions being responsible for much of what we describe as intelligence, research evidence has provided support that executive functioning is not a substitute for IQ, nor is it uniquely related to measures thereof (Henry, 2012). A similar relationship exists between working memory and intelligence, where they are both related to academic learning but remain dissociable cognitive skills (Alloway & Alloway, 2013).

Working memory is believed to be an essential skill from the beginning of formal schooling (preschool) (Alloway et al., 2005) to tertiary level study (Alloway & Gregory, 2013). There are also strong links between working memory, learning and academic achievement, where working memory (measured by verbal and visuospatial complex span tasks) has been strongly associated with national curriculum test scores at 7, 11, and 14 years (Gathercole & Pickering, 2000a, 2000b; Gathercole et al., 2004; Jarvis & Gathercole, 2003). Within typically developing children, scores on working memory measures are predictive of reading achievement independent of phonological skills, and as a construct is believed to be essential in the acquisition of reading (Swanson, Saez, Gerber, & Laefsedt, 2004), writing (Berninger & Swanson, 1994, 1995), and spelling (Carramazza, Miceli, Villa, & Romani, 1987; Margolin, 1984). Children with reading difficulties have been found to have limited storage and processing capacities, and show significantly poor performance on measures of working memory (Alloway & Copello, 2013). Working memory capacity limitations are also

associated with mental arithmetic and mathematics performance, which draw on skills requiring working memory (DeStefano & LeFevre, 2004). These include counting, mental arithmetic, measurement and spatial abilities (Geary et al., 2004; Maybery & Do, 2003; Swanson, 2004), where low working memory scores have been found to be closely related to poor performance on arithmetic word problems (Swanson & Sachse-Lee, 2001) and poor computational skills (Bull & Scerif, 2001). Verbal and visuospatial complex working memory scores account for a small yet unique degree of variance in performance on school-based language test scores (tested in English), while visuospatial scores are usually linked to performance in mathematics and science. (Jarvis & Gathercole, 2003).

Working memory capacity is therefore an excellent predictor of academic attainment in children. Children with high working memory scores are typically excellent readers, with similarly excellent mathematical abilities. This direct relationship is believed to extend across the lifespan and is as significant in university students as it is in children in their first year of school (Alloway & Gregory, 2013; Alloway et al., 2005). In contrast to this, intelligence, as measured by IQ scores, has conventionally been referred to as the best predictive measure of scholastic success because of the alignment between IQ test content and scholastic curriculum. Some theorists have considered these two constructs so highly correlated that they could be considered as isomorphic properties (Colom, Rebollo, Palacios, Juan-Espinosa, & Kyllonen, 2004; Colom, Abad, Quiroga, Shih, & Flores-Medona, 2008; Jensen, 1998; Stauffer, Ree, & Caretta, 1996 as cited by Alloway & Alloway, 2010), and posit that it is IQ which is the common underlying factor between working memory and learning (Nation, Adams, Bowyer-Crane, & Snowling, 1999; Stothard & Hulme, 1992). However, there is increasing empirical evidence in dispute of this view, and instead supports IQ and working memory as being related yet dissociable constructs, where working memory shares unique links with learning after statistically accounting for IQ (Alloway, Gathercole, & Pickering et al., 2006; Alloway & Alloway, 2010, 2013; Swanson, 1999).

One such study supporting the distinction between working memory and intelligence measured a group of 98 children (4.3-5.7 years) at two points in time, six years apart, on standardised measures of working memory, IQ and learning. The findings conclude that not only are working memory and IQ dissociable constructs, but also that working memory has unique links to learning outcomes outside of the already established pathways between what IQ measures and learning. Further, the study found that measures of working memory

in school beginners was a more powerful predictor of subsequent academic success than IQ scores (Alloway & Alloway, 2010). These findings provide strong support that working memory is a stable, reliable construct (six years), and is confirmed by other research also showing that while working memory increases with age, its relative capacity remains consistent (Alloway, Gathercole, & Pickering et al., 2006; Swanson, 1999).

One reason for this relationship is that unlike IQ, working memory measures the potential to learn, while IQ tests generally measure content already learnt (Alloway & Copello, 2013). The use of IQ scores as a measure of intelligence, have oft been critiqued for their reification of cognitive functioning into a single numerical quantification (referred to as Full Scale IQ (FSIQ)). The striation of this score into sub-components representing different abilities (Verbal IQ (VIQ), Performance IQ (PIQ)) offers a mechanism for the overlap between working memory performance and intelligence, where the non-verbal or fluid intelligence components of an IQ tests are posited as accounting for the associations between IQ and working memory. Particular attention has been given to this distinction between fluid intelligence (g_f) and working memory (Engle, Tuholski, Laughlin, & Conway, 1999; Fukuda, Vogel, Mayr, & Awh, 2010; Unsworth, Brewer, & Spillers, 2009). Studies of empirical relationships (Chooi, 2012; Kane et al., 2005; ; Kyllonen & Christal, 1990; Oberauer et al., 2005), brain-based behaviour and imaging (Gray, Chabris, & Braver, 2003; Jung & Haier, 2007), as well as cognitive training and interventions have again yielded the two as related, yet unidentical abilities. The failure of some studies to induce transfer effects in g_f or general IQ to working memory performance provide further support for the two as distinguishable constructs (Bergman, Nutley et al., 2011; Buschkuehl et al., 2008; Holmes, Gathercole, & Dunning, 2009; Van der Molen et al., 2010). The existence of significant near transfer effects between working memory training and increased performance on attention and matrix reasoning skills is believed to be the results of a shared neural connection, particularly in periods of neurodevelopment when plasticity and synaptogenesis are prolific (Borella, Carretti, Riboldi, & De Beni, 2010; Klingberg, 2005; Schmiedek, Lovden, & Lindenberger, 2010). The relationship between the two therefore appears to be based on a shared fundamental neural network in the dorsolateral prefrontal cortex which relates to short term memory storage and processing speed – essential to both working memory and intelligence, but distinct in structure and function. To that end, working memory might be related to intelligence, but represents a distinct construct that is fractionated itself.

Working Memory, Language and Socioeconomic Status.

Another way in which working memory and intelligence have been linked is through their common association with long term memory. Both of these cognitive capacities are influenced by the interaction with the external environment, particularly the influence of socioeconomic status (SES), which has long been linked to school success (Alloway & Copello, 2013). Further, one of the difficulties in measuring the distinction between working memory and intelligence (as measured by conventional IQ tests) concerns the inherent bias in the assessment of cognitive function in non-Western, multilingual children. While this bias is often described as being 'Western' in nature, it frequently manifests on linguistic and cultural platforms where test-takers not familiar with English or Western cultural assumptions are systematically discriminated against. The global legacy of discrimination often dictates that these linguistic and cultural biases are predominantly experienced by people in lower socioeconomic brackets (Campbell, et al., 1997; Hoff, 2003; Hoff & Tian, 2005; Lezak, 1995). It is in response to these limitations regarding the validity of IQ testing that working memory has been investigated as a less-biased manner of assessing cognitive functioning.

While initially designed as aptitude tests (which predict the potential to learn), conventional IQ tests have predominantly fallen in the realm of achievement testing (the measurement of skills and knowledge which has already been learnt). This is because of their reliance on English proficiency, and that success requires familiarity with an inherent set of contextual assumptions that children from a diverse set of ethnic groups and socioeconomic classes may not have (i.e. snow, safe relationships with law enforcement, democratic governments). Conversely, the procedures and stimuli presented to measure working memory are designed to be equally unfamiliar to all test-takers, and thus have less vulnerability to the sociocultural and linguistic biases that traditional intelligence measures possess. While they still make use of language span and repetition tasks, their emphasis on spontaneous and immediate processing and storage are less bound to long-term knowledge which has particular contextual locations (Alloway, 2009; Colom et al., 2008).

There has been promising research in the area of paediatric language evaluation to promote the use of working memory assessment measures as less biased than conventional tests. Laing and Kamhi (2003) encourage the use of processing dependent linguistic measures to assess language abilities in linguistically diverse communities. Campbell,

Dollaghan, Needlemen, and Janosky (1997) administered a series of verbal working memory tasks (nonword repetition, competing language processing task, various verbal complex span tasks), as well as a knowledge-driven measure of oral vocabulary, listening comprehension and verbal analogies to 156 typically developing American school children between the ages of 11 and 14 years. Both African American and white children were represented in this sample. The results found that African American children (both typically developing and with an already identified language impairment) performed significantly poorer than the white group of typically developing children on the knowledge-based measures of language assessment. However, performance on the verbal working memory measures were similar for both groups of typically developing children, and they were able to identify those with language impairment without the assistance of the knowledge-based measure. These results suggest that processing-dependent measures, such as verbal working memory subtests, hold considerable promise for distinguishing between children with language disorders, whose poor language performance reflects fundamental psycholinguistic deficits, and children with language differences attributable to differing experiential and cultural backgrounds. This is not to suggest that working memory measures are impervious to the effects of language or culture; however they appear to be less biased when compared to conventional psychometric testing which is usually based on previous knowledge acquisition.

There is also equivocal research regarding the validity of working memory assessment in children from different socioeconomic brackets. Tine (2013) conducted a study in the United States on 186 sixth grade children (*M* = 11.3 years). She found that not only were both verbal and visuospatial measures of working memory significantly lower in low socioeconomic brackets, but that there were further significant differences between the urban and rural poor. The study found that while low-income urban children had low, yet symmetrical verbal and visuospatial working memory scores (40^{th} percentile), low-income rural children had far poorer visuospatial working memory scores (29^{th} percentile) than verbal working memory scores (45^{th} percentile). The authors suggest that the poorer working memory performance in the lower income bracket is a response to the mind's attempt to adjust to the events of a chronically stressful life (Evans & Schamberg, 2009). The longer a child lives in poverty, the higher the chronic stress and the greater the burden on the internal allostatic load – an index of cumulative physiological wear and tear. In critique

of these findings, the author does concede that the samples from high, low, urban and rural communities were not matched, and that the working memory differences between low and high income children were likely to be a reflection of language ability differences between the two groups that vary systematically with race and minority status. While the study shows validity in its distinction between high and low income earners, it makes no comparison of working memory with other cognitive measures, nor does it recognise that working memory might be less influenced by SES than other more conventional measures of cognitive capacity. Similar findings are evident in two other studies which also included older children in the sample. For example, Farah et al. (2006) found significant deficits in spatial memory and the n-back task in a group of African American pre-teenage children (10-13 years) (*n* = 30) coming from low socioeconomic brackets when compared to an age and gender matched control group from a higher socioeconomic bracket. Evans and Schamberg (2009) also report working memory deficits as a function of low SES in a longitudinal comparative study of 195 17-year olds, where approximately half grew up below the American poverty line, and the other half grew up at levels two to four times the poverty line. A regression analysis revealed that the greater the proportion of life growing up in poverty (birth to 13 years), the shorter the span of sequential information that the participants could accurately hold in visuospatial short term stores. None of these studies compared the validity of working memory assessment against more conventional cognitive tests in children from low income brackets. Conversely, working memory performance appears to be less impervious to the effects of SES in younger children. Evidence in support of this follows here.

Engel, Dos Santos, & Gathercole (2008) found evidence contrary to that of Tine (2013) in their assessment of a group of Brazilian children from both low and high socioeconomic classes who were matched on age, sex and non-verbal ability (*N* = 40, *M* = 6.11 years, SD = 3.75). Children from high and low income groups were compared using the Automated Working Memory Assessment (AWMA), and vocabulary measures of expressive and receptive vocabulary in Portuguese. The results found that while groups differed significantly on measures of receptive and expressive language, no significant distinctions were found between the subtests of the AWMA. This suggests that working memory is a less biased measure of cognitive potential than measures of expressive and receptive vocabulary (often assessed in IQ and aptitude tests) which rely on crystallised knowledge

and are associated with opportunity and previous learning. The results of Alloway et al. (2004) in their assessment of 4-5 year olds converge with this finding. Messer, Leseman, Mayo, and Boom (2010) also found that working memory performance was the same between a group of immigrant children (4 year olds) from a low income area, and a group of wealthier children who spoke the same language, when assessed in their native language. The Engel et al. (2008) and Messer et al. (2010) studies have indexed SES by parental education which has a number of limitations in heterogeneous groups (Higgs, 2002; Luo, Wilkins, Kramer, 2006; Education, Employment & Workplace Relations, 2009).

Alloway and Alloway (2008) investigated the relationships between intelligence (traditional IQ scores), working memory and learning in a diverse group of 98 children from low, middle and high poverty indices in the United Kingdom. Information regarding each participants' mother's educational level and the age at which the mother left school was also collected as they are considered relatively accurate measures of socioeconomic status and quality of the home environment in culturally homogenous Western contexts (Luo, Wilkins, & Kramer, 2006). Maternal education was significantly correlated with IQ and academic literacy skills, but was not significantly associated with working memory. Significant associations between working memory and learning remained even after statistically accounting for maternal education. These findings align well with previously presented research in this area, and suggest that working memory assessment is less affected by the consequences of environmental and economic deprivation than conventional cognitive measures. Research evidence to the contrary is plagued by conflating issues of language, and the contextual distinction between rural and urban poverty, which is vulnerable to extraneous effects (Tine, 2013). Despite equivocal evidence regarding the validity of working memory assessment as a less biased means of assessing cognitive potential in children from low socioeconomic brackets, there is nevertheless a substantial body of support for its use as a far purer measure of a child's learning potential than conventional psychometric assessment (Alloway et al., 2004, Alloway et al, 2005; Ellis Weismer et al., 2000).

These conclusions have generally been drawn from studies conducted in Western contexts where English speaking children were assessed in English. In studies where children who speak English as a second language were used (e.g. Engel et al., 2008), they were assessed in their native language. Despite, valid measures of working memory currently not

being available in local languages, a South African study also compared English measures of working memory with more traditional measures of English vocabulary and non-verbal IQ in a sample of Grade One learners from both high and low socioeconomic brackets using the AWMA. While the results found that working memory measures were consistently less affected by socioeconomic status than vocabulary and non-verbal IQ measures, language was found to be highly correlated with socioeconomic status which conflated the findings (Moolla, 2012). This is an inherent limitation of psychometric assessment within the South African context, but one that is not unique to measures of working memory. In fact, it would apply even more strongly in traditional assessment of cognitive functioning, and that all else being equal, working memory appears to be a less biased measure than IQ (Laing & Kamhi, 2003). The focus of this chapter now to turns to the definition of working memory. It begins with an account of the construct's chronological progression over the last forty years.

Historical Development of Working Memory

Working memory is widely agreed upon as playing an essential role in complex cognition, and has come to be known as the cognitive mechanism responsible for the maintenance of task-relevant information while performing a cognitive task (Baddeley & Hitch, 1974). However, despite the familiarity with the term, there is very little agreement about the definition of the construct. One of the most prominent of these distinctions is the difference between working memory and short term memory. There are also other contentions around the definition of the construct which consider whether it is a unitary or non-unitary construct, the various domains that it can carry information in, its neurobiological location and its functional capacity for everyday living.

This section seeks to critically discuss some of the proposed models defining the construct of working memory. The intention behind this approach was to assist in the answering of one of the aims of this study, which was to determine whether the working memory development of the two HIV affected (HIV-Infected (HIV-I), and HIV-Exposed, Uninfected (HIV-EU)) samples is similar to that expected of typical populations. Usually, this question would be answered by a simple comparison of the samples' psychometric scores with normed estimates. However, because of the complex and often biased nature of psychometric measurement within non-Western contexts, a model-based approach is believed to be a fairer means of answering the question. The discussion presents the models in chronological order, and begins with the model of Atkinson and Shriffin.

Atkinson & Shiffrin's Model of Working Memory (1968).

This model is a predecessor to that of Baddeley's (1974) and is actually a model of memory of which working memory is only a component. It asserts that memory has three components – a sensory registry where sensory perception occurs, a short-term store (what is currently referred to as working memory), and long-term memory (an indefinite store of short-term memory rehearsals). The authors propose that representational encoding is domain specific and that perception into these buffers is achieved by a series of multiple registers – one for each sense. This componential sensory register does not process the stimuli, but detects and holds the information in short-term memory. The subsequent funnelling and buffering of particular information is only done under controlled attention in order to prevent higher-order cognitive overload, and prevent loss due to interference and decay (Atkinson & Shiffrin, 1968; Miyake & Shah, 1999).

The domain specified representational buffers have been expanded upon within the visual and auditory systems, and have come to be known as iconic and echoic memory respectively. This buffer holds information for visual stimuli (shape, size, colour, but not meaning), and is seemingly limitless in the size of stimuli that it can hold. However, without rapid encoding into the short-term store, information rapidly decays after only half a second. Similarly, echoic memory holds a limitless perception of the superficial aspects of sound (pitch, tempo, rhythm) but has a longer duration (between one and five seconds) which can be as long as twenty seconds in the absence of interference (Shiffrin, 1975; Miyake & Shah, 1999).

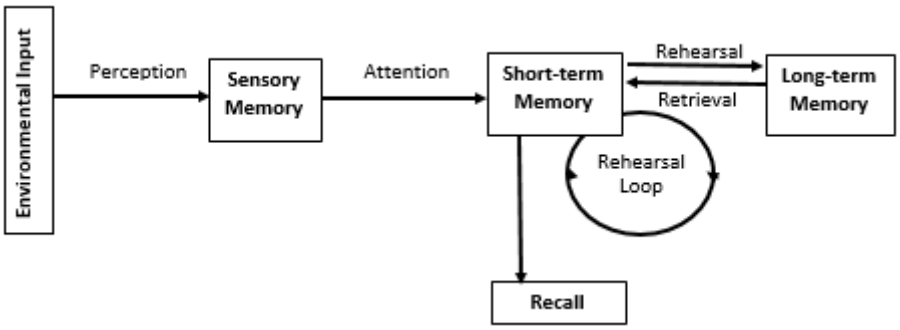

Figure 2.1. Atkinson & Shiffrin Memory Model. Adapted from "Multistore Memory of Model – Atkinson and Shiffrin (1968)" By S.A. McLeod Retrieved from www.simplypsychology.org/multi-store.html

Without rehearsal, information in the short-term store can be held for up to twenty seconds. Its capacity is limited by a vulnerability to decay and interference, and is found to be at seven (+/-2) chunks. The transmission of this information to long-term stores is cited as being a result of repetition (encoding), and retrieval is done through a series of cues by association, recency and context. This has been refuted as being incomplete in light of other evidence which suggests that rehearsal is not a key process in the transfer of information from the short-term store to long term memory. In contrast, semantic encoding has been found to have longer traces in memory.

This model is widely criticised for its simplicity and inability to explain the neurocognitive structures behind representational domain specificity, as well as the processing and control of attention to guide only specific information into the short-term store. Critics have argued that it is in fact the sensory registers who act as the executive of attention, and should not be a separate system. The model also assumes that the short term storage of information in that component would guarantee automatic transfer into long term stores. This is contrary to what is now known about the links between elaborate and encoded processing and better learning (Craik & Lockhart, 1972). The deficits of this model also became apparent in clinical cases of neuropsychological damage. Should the model's assumptions regarding the essential pathway between short and long term memory be correct, then deficits in the former should automatically create long term memory difficulties as well. In some cases, patients who had only a two digit recall span, had intact long term memories which supported evidence for its shortcomings. Further, if the short term memory was solely responsible for complex cognitive processes of rehearsal, processing and manipulation, then patients with short term memory deficits should have profound functional impairment in everyday living. This was not the case in acute cases of neuropsychological impairment, and encouraged further research into a 'working' component of the memory system (Baddeley, 2012).

In response to these short-falls, Baddeley and Hitch (1974) proposed a simple, three component model of working memory following a series of empirical studies on typically developing undergraduate students. They split attentional control from temporary storage into two capacity-limited, domain-specific loops which was responsible for either verbal or visuospatial information. A domain-general central controller was termed the central-executive, and remained generally under-investigated for almost a decade. This was because

of its assumed complexity, and the inability of existing theories of attention at the time to explain the essential role of attention within the central executive as the controller of action, and not necessarily in relation to perception. Baddeley and Hitch drew on the work of Normal and Shallice's attentional system to explain the function of the central executive.

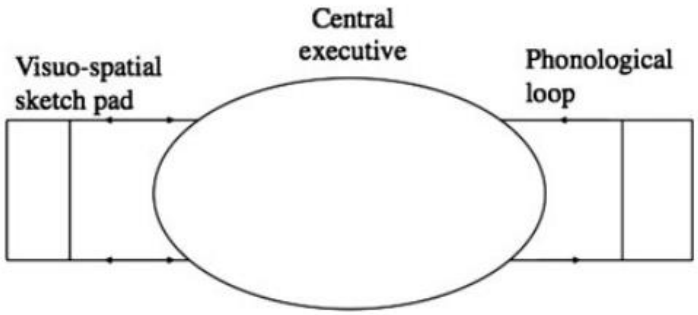

Figure 2.2. Baddeley & Hitch's (1974) initial working memory model. Adapted from "Working Memory: Theories, Models and Controversies" by A.D. Baddeley, 2012*, Annual Review of Psychology, 63,* p. 6.

Norman & Shallice's Model of Executive Attention (1980).

This model is not so much a model of working memory, but rather one of executive attention which is an essential feature of working memory functioning. The authors propose a model of executive functioning of attentional control that specifies how cognitive schemata are either activated or suppressed in novel or routine contexts. Particular environments (stimulus conditions) will activate a particular response set of schemata. The initiation of appropriate schema under routine or well-learned situations is controlled by a cognitive process called 'contention scheduling' which inhibits schemata for control of cognitive apparatus. Here, attention is automatic, does not require conscious control and is triggered in response to familiar environmental stimuli (driving a familiar route) (Friedenberg & Silverman, 2010).

Under novel circumstances, this attentional control is determined by the supervisory attentional system (SAS). The SAS has control over 'contention scheduling' as well, and serves to monitor conscious, deliberate planning of actions within novel situations that cannot be solved by previously existent schema, or when error prevention is essential. It is also responsible for novel problem solving where previous cognitive strategies have failed.

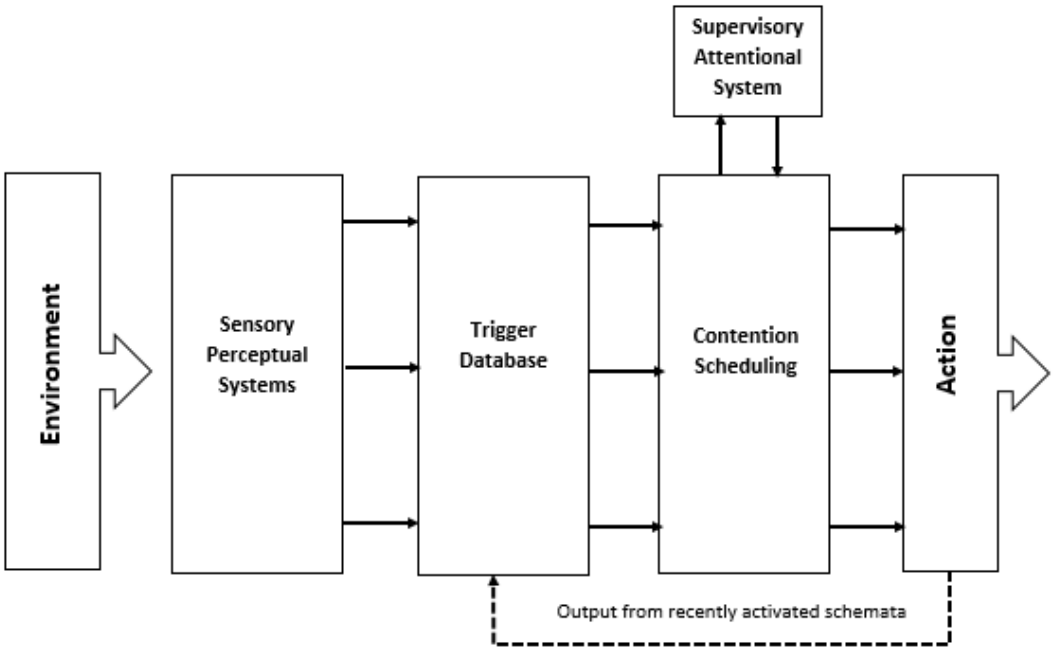

Figure 2.3. Norman & Shallice's Supervisory Attentional System (SAS). Adapted from "Attention to Action: Willed and Automatic Control of Behvaiour" by D.A. Norman & T. Shallice, 1980, *Centre for Human Information Processing, 99,* p. 9.

To that end, the SAS enables planning, decision making, problem solving, error management (inhibition and correction) and the initiation of actions. It also accounts for the priming of responses in anticipated tasks. The model makes no direct reference to memory stores, and presumably operates in a similar way to long-term working memory where it relies on a store of previously learnt procedures from which to direct attention and elicit a response. While this explanation does not holistically explain the central executive, it assists in explaining the executive homunculus controlling the two domain-specific temporary storage systems. The synchronous functioning of these three components are together regarded as the basis of what was later to become the model of working memory that we have today; the hypothetical capacity-limited system that provides the temporary storage and manipulation of information that is necessary for performing a wide range of cognitive activities. An explanation of the mature version of this model follows (Miyake & Shah, 1999; Shallice & Burgess, 1996).

Baddeley's Multicomponent Model (2000).

As described previously, in the original model of working memory created by Baddeley and Hitch (1974), the construct had three main parts: a central executive, a phonological loop

and a visuo-spatial sketch pad. More recently, a fourth component, the episodic buffer, was added to the model (Baddeley, 2000).

The most important component of the Multicomponent Model is the central executive which serves as a 'headquarters', and regulates information flow within the working memory system, retrieves information from other memory systems and processes and temporarily stores this information (Gathercole & Baddeley, 1993). This attentional control is however limited in capacity, and delegates responsibilities to the two slave systems of the phonological loop and the visuospatial sketch pad. The phonological loop is specialised for the short-term storage of verbal material, while the visuospatial sketch pad is responsible for the short term store of images, pictures and locations. A diagrammatic depiction of these components is found in Figure 2.4. A few features of this model are of particular relevance to a broader understanding of the model. Firstly, each of the three components is limited in capacity, and hence relies on each other to carry out multi-modal processes. Secondly, while there is a bi-directional transfer of information between the two slaves and the central executive, there is no direct corresponding path between the two slave systems themselves. Hence, they can only indirectly communicate via the central executive (Gathercole, Alloway, Willis &, Adams, 2006). Lastly, Baddeley (2000, 2007) also extended the model and added a fourth component, the episodic buffer. This is believed to hold representations that integrate phonological, visual and spatial information which is not covered by the aforementioned slave systems (i.e. semantic information, musical information), bind the storage of larger 'chunks' of information, and integrate these with existing schemas in long-term memory. A more thorough discussion of each component of working memory follows.

The Central Executive.

The central executive is the most important, but least understood, subsystem of the three-component model of working memory, and acts as a central management system between the two slave systems. Previously the central executive was oft considered a 'ragbag' of collective functions which did not exclusively belong to the phonological loop or sketch-pad. However, more recently it has become known as providing a supervisory, delegatory function between the two as a pivotal 'middle-man' which is responsible for the regulation of complex cognitive processes, a capacity for the temporary activation of long

term memory, the coordination of multiple tasks, shifting between tasks and retrieval strategies, and the capacity to inhibit and attend to information in a selective manner (St Clair-Thompson & Gathercole, 2006). As discussed earlier, Miyake, Friedman, Emerson et al. (2000) identified three key executive functions believed to be pivotal to the central executive: shifting, updating and inhibition.

These three discrete components of the central executive have been found to be empirically dissociable in adult populations, but only that of updating and inhibition are evident in paediatric samples of children under the age of 12 years of age. Updating is also commonly associated with verbal and visuospatial complex span tasks in children, and is believed to represent a domain-general ability crucial to working memory (St Clair-Thompson & Gathercole, 2006).

There are particular points of interest that are worth noting about the central executive. Firstly, in addition to regulating inputs and outputs from the two slave subsystems, the central executive (together with the episodic buffer) is also active in the retrieval of information from long-term memory (existing schema), and regulates functioning

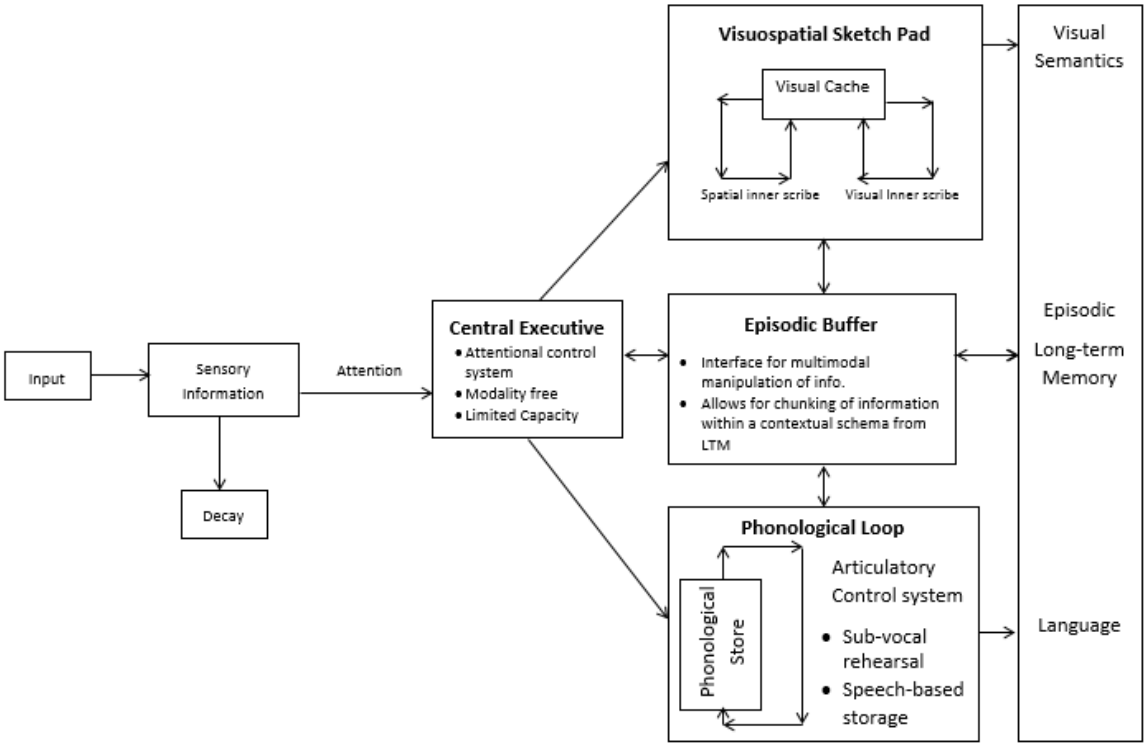

Figure 2.4. Baddeley's Multi Component Model. Adapted from "Working Memory: Theories, Models and Controversies" by A.D. Baddeley, 2012, *Annual Review of Psychology, 63*, p. 16.

according to this existing knowledge. Secondly, the central executive has a finite capacity for attention and is thus vulnerable to distraction and interference from task demand overloads when doing too many things at once (Baddelely, 2000). Hence, tasks that are already automated and exist in well-established schema's (e.g. driving) are far less taxing on the system than new tasks that require attentional manipulation from multiple inputs (e.g. learning a new computer game). The capacity and efficiency of the central executive is believed to form the basis of the general factor '*g*' of intelligence, and that it is comprised of and responsible for a series of relatively independent sub-processes, such as planning, task coordination, conscious awareness and the mind's ability to select and control action (Shallice & Burgess, 1991).

Experimental studies initially done by Baddeley and Logie (1999) provide clear evidence for the distinction between the central executive (responsible for processing) and the two slave systems (primarily responsible for domain specific storage). They did this by examining the extent to which increasing processing demands resulted in poorer performance on verbal and visuospatial storage capacities. Participants were given a processing task (to verify the accuracy of arithmetic sums in a fixed time period), a storage task (the immediate serial recall span for sequences on unrelated words), and a processing-and-storage task (the combination of the two tasks). The results revealed that the increased demand of the storage task had no effect on the processing task and visa a versa. Three subsequent experiments by the authors have confirmed similar results, suggesting that the central executive is not responsible for storage but for processing. Hitch, Halliday and Litter (1989) also conducted a series of studies with children which managed to determine that the developmental changes in the phonological loop and central executive are dissociable, and by so doing provided evidence for the fractionation of separately existing components, even in childhood (Gathercole & Baddeley,1993). Baddeley himself concedes that it is empirically insufficient to determine that the central executive is separate and differentiated in function from the phonological loop and visuospatial sketchpad, but to then classify it as an all-encompassing supervisory homunculus (Miyake & Shah, 1994). Hence, subsequent research has focused on identifying particular executive functions such as the focusing of attention, switching attention, and the activation of representations within LTM. These operations within the central executive remain the nexus of central executive research as science

continues to determine to what degree these are fractionated, whether these exist in a hierarchy, or whether they are all driven by a singular underlying capacity.

Neuroanatomical studies of brain-injured patients have provided substantive evidence for the specific functionality and existence of the central executive. Central executive functioning is primarily believed to be located in the frontal lobes, as patients with damage in these areas struggle to consciously control their actions (termed 'dysexecutive syndrome'). Data from patients with frontal lobe damage, and the consequent influence on executive functioning deficits, have done much to support this belief (Kane & Engle, 2002; Roberts, Robbins, & Weiskrantz, 1998; Stuss & Knight, 2002). Wilson and Baddeley (1988) also found that some amnesic patients were capable of demonstrating excellent immediate recall of a prose passage, but had almost complete absence of delayed recall. This indicates that in the absence of activation by the central executive (not capable of storing any information itself, and hence severing any connection to long term memory), performance significantly declines.

Current research regarding the central executive is focused around the degree to which it is fractionated, and how discrete the underlying dissociable abilities are. Baddeley foregrounds focused attention, the capacity to divide attention simultaneously, and switching as essential skills. Other theorists have posited anything between six (verbal storage-and-processing coordination, visuospatial storage-and-processing coordination, dual-task coordination, strategic retrieval, selective attention, and shifting (Fournier-Vicente, Larigauderie, & Gaonach, 2008), four (manipulating and updating of information, dual-task coordination, inhibition and shifting processes) (Collette & van der Linden, 2002) and no (Parkin, 1998) dissociable functions of the central executive.

It is this idea, that the central executive has no fractionable functions, which is the source of one of the biggest criticisms to Baddeley and Hitch's conceptualisation of the central executive. Parkin (1998) argued that the central executive doesn't exist, and that is a reified abstraction created to provide account for otherwise unaccountable phenomena. He argues that the central executive is a concept that emerges from a range of research evidence because there is a lack of more rigorous and theoretically sound constructs to explain the results. He proposes that the lack of a single identifiable and measurable brain region responsible for executive function, coupled with the varied executive tasks subsumed

by different neural networks as evidence for the non-existence of a central executive, and suggests instead that executive tasks are completed by a series of non-overlapping control processes.

Baddeley (1998) has rebutted these criticisms by outlining a series of incorrect assumptions about the theoretical conceptualisation of the homunculus-like nature of the central executive. He refutes the idea that a coherent functional concept (such as that of the central executive) must have a unitary anatomical location in the brain, and argues that its location as an essential part of a cohesive system (like that of working memory) does not prohibit it to be a fractionable entity in and of itself. To the contrary, Baddeley argues that the idea of a homunculus can be particularly helpful in the current stage of working memory research as it affords researchers the opportunity to analyse the separability and relatedness of each of the executive processes in an attempt to eventually render the homunculus redundant. He further defends his approach by arguing that he himself considers the theoretical conceptualisation of the working memory system to be a relatively loose framework that is valuable not only in its capacity to explain existing data, but on its productivity in generating good, answerable questions linked to empirical methods that can be widely applied.

There are numerous questions regarding the central executive which have arisen out of such an approach which are concerned with the construct's measurable capacity, the source of its limitations, and how it can facilitate changes in processing ability according to practice-effects or the passage of time. In response to these, there have been some studies indicating the presence of transfer effects following executive process training and intervention (Salminen, Strobach, & Schubert, 2012; Thorell, Lindqvist, Bergman Nutley, Bohlin, & Klingberg, 2009). While research evidence is encouraging, there is much yet to be learned about the central executive, and its relationship to and interaction with the two slave systems. Much research regarding these relationships has been done on the verbally based system, the phonological loop. For example, Zhenshu, Ming, and Xiaolin (2008) compared the process of updating (central executive) in both verbal and visuospatial working memory in a number of experiments. The results found that the central executive system alone played an important role in the updating function of verbal working memory, while the phonological loop was responsible for the processes of serial recall. In contrast, the visuospatial sketchpad and the central executive were found to work together in the

updating of visuospatial information. This suggests that updating verbal and visuospatial working memory are not two parallel processes, and that there is an asymmetricity in the interactions between the two slave systems and the central executive. Research evidence regarding the verbal slave system, the phonological loop, is now considered.

The Phonological Loop.

The phonological loop (PL) is responsible for the storage and processing of verbal material within the working memory system, and is made up of two parts: a phonological store and an articulatory rehearsal mechanism. In the phonological store, words are stored very briefly (usually for about 2 seconds) and then decay from the system if they are not rehearsed. This if referred to as 'trace decay'. The articulatory rehearsal mechanism is responsible for a subsequent process called sub-vocal rehearsal. This sub-vocal rehearsal ('looping' of the words on the articulatory control system) boosts the activation of the decaying material. It is necessary for this to be repeated numerous times in order for it to be remembered, or later stored in long term memory (LTM) (Baddeley, 2007; Gathercole & Baddeley, 1993).

There are four main pieces of empirical support for the existence of the phonological loop. The first is the effect of *phonological similarity.* Conrad (1964) found that when patients mis-recalled letters in a sequence of consonants, the error was phonologically similar to the letter missed (i.e. D as B not R). Other studies have demonstrated that similar sounding letters like V P B G, are recalled less well than dissimilar letters, X A Y W. Similarity of sound in words also has also been found to have a detrimental effect on recall, whereas semantic similarity (words that have a similar meaning) have comparatively little effect (Conrad & Hull, 1964). This provides evidence that the representation of verbal code is phonological (based on sound), where follow-up studies have proven this with both similar letter sequences and monosyllabic words (Baddeley, 1986; Conrad & Hull, 1964; Wickelgren, 1965).

The presence of the phonological loop is also supported by the concept of *articulatory suppression*. Here studies required participants to memorise letters while maintaining irrelevant speech, or having the information presented visually. Memory for this verbal information is impaired because the mental rehearsal is believed to block the articulatory rehearsal process, and allows memory traces in the phonological store to decay (Baddeley, Thompson, & Buchanan, 1975; Baddeley, 1986). Evidence of the phenomenon known as the *word length effect* also supports the presence of the phonological loop.

Studies have found that working memory performance for verbal information is directly influenced by the length of memory items. Monosyllabic words were much easier to remember than five syllable items which are rehearsed less on the articulatory control system due to their length and hence suffer quicker decay. Studies therefore conclude that there exists trace decay within the phonological loop as words that are longer to say are refreshed less frequently (Baddeley, 1986; Gathercole & Baddelely, 1993).

Lastly, research exploring the relative capacity *of transfer of information between representational codes* (verbal and visuospatial) supports the essential functioning of sub-vocal rehearsal of the phonological loop. Murray (1968) presented lists of letters varying in length and acoustic confusability in either visual or auditory form. He found that with visually presented items, encoding is switched from a visual to verbal code through a process of naming and sub-vocal rehearsal. The process of simultaneous articulatory suppression prevents this transfer, which emphasises the importance of the phonological loop to provide rehearsal necessary for the maintenance of verbal information.

Neuropsychological evidence in a patient population of aphasic patients with developmental verbal dyspraxia offers further support for the functions of the phonological store and the articulatory rehearsal processes of the phonological loop. Six patients who had difficulty in articulating sounds, syllables and words were tested on measures of verbal memory span and other tasks tapping articulatory rehearsal. Findings indicated significantly impaired memory performance, which did not appear to be influenced in a typical manner by the effects of word length and phonological similarity. The authors suggested that patients were unable to set up the speech motor codes necessary for articulation, which was caused by a deficiency in the articulatory rehearsal process. It is important to point out that the developmental apraxia was because of a neurological dysfunction in initiating movement of motor parts required for speech, and not because of muscle weakness or paralysis. These results are in contrast to those of other studies in which patients with dysarthria, where speech difficulties are a result of neurological injury to the motor component of the speech system, show a normal capacity for rehearsal, and indicate that sub-vocal rehearsal is essential to short term verbal storage (Waters & Rochon, 1992).

The storage capacities of the phonological loop have been widely criticised by Nairne (2002). He argues that the simplicity of our understanding of short term verbal stores

(coupled with their vulnerability to decay, reliance on active processes of rehearsal and their access to long term information) is unsupported by empirical evidence. He cites empirical studies which question whether the correlation between span and articulation are indeed a result of rehearsal, and instead show support for other factors which contribute to memory success (familiarity, frequency and phonological complexity). In the place of the traditional modular understanding of the phonological loop, Nairne proposes a cue-driven model of immediate retention, similar to that of long term memory, which he suggests is better able to account for the effects of decay and interference. There have been other critics which have called for the abandonment of the idea of a phonological store, and suggest instead its replacement with a perceptual system that is reliant on a gestural rehearsal system able to perform all that the former is unable to accomplish (Jones, Hughes, & Macken, 2007; Macken & Jones, 2003). Baddeley and Larsden (2007) concede on a number of similarities between their own work and that of Jones, Hughes and Macken (2007), and attribute differences to semantic attributions to different systems (i.e. the relabelling of the verbal rehearsal mechanisms to a 'gestural component' of a perceptual system, or of the episodic buffer as a 'object oriented episodic record'). However, their primary difference is accounted by J, H & M's insistence that perceptual buffers can account for short term storage, while Baddeley and Larsden suggest that their denial of the need for short term phonological storage is premature, as it has strong empirical support from a number of fields.

The phonological loop also has strong ties to the learning of phonological information and its location in long term memory, particularly that of expanding vocabulary. One of the best examples of the close associations between working memory and long-term memory is that of language acquisition. This next section foregrounds a discussion of the relationship between verbal working memory and language acquisition where research findings suggest that both native and foreign language acquisition appear to be achieved through the ability to represent serial-order information in working memory. The discussion considers the interaction of language and working memory, and presents research evidence which would suggests that language perception and production, particularly in the learning of additional languages, are less reliant on simple verbal working memory skills, but rely instead on complex processes of attentional control. The continuous use of these executive processes

appear to provide the multilingual brain with greater mental flexibility, and account for a dialectical relationship between language learning and working memory capacity.

The relationship between verbal working memory and language was first measured in a seminal study by Daneman and Carpenter (1980) who found significant associations between complex reading span tasks and reading comprehension and scholastic measures of verbal aptitude. Subsequent studies also found high correlations between working memory capacity and language comprehension (Daneman & Merikle, 1996). Baddeley, Gathercole, and Papagno (1998) published an influential paper on the relationship between verbal working memory and language, and proposed that verbal working memory represents the "processes and mechanisms by which the sound patterns of the words of the native language are learned by the child"(p.159). Their work reviewed a large evidence base and concluded that the function of verbal working memory is primarily to learn language. They cite numerous studies which show that verbal working memory capacity (as measured by nonword repetition) and native vocabulary knowledge in children of various ages were significantly correlated (Bowey, 2001; Gathercole & Adams, 1993,1994), or were found to be correlated with the ability to learn new, unfamiliar words (Gathercole & Baddeley, 1990).

The use of nonword repetition (commonly assessed in verbal working memory measures) is a particularly important measure of language learning and has been used extensively to investigate these relationships. Before expanding on this relationship, it is important to understand why nonword repetition (sometimes referred to as nonword recall) is significant to this discussion. The capacity of the phonological loop is conventionally assessed using serial recall tasks in which verbal items are presented immediate recall in their original order. Although the loop is considered to be a storage device for distinct phonological information, it does not operate in isolation from more permanent lexical knowledge systems – hence immediate recall performance is strongly influenced by features other than the phonological loops efficiency (real words instead of non-words (Hulme, Maughan, & Brown, 1991), those low in every day frequency (Hulme et al., 1997). The repetition of non-words therefore does not activate existing lexical representations, and success requires the storage of their unique phonological segments in short term stores. Thus, nonword repetition may provide a purer assessment of phonological storage quality than serial recall measures (Gathercole, 2006).

The use of nonword repetition as a measure of verbal working memory and its correlation with language learning are evident in both typical and atypical populations. The associations between nonword repetition (as a measure of verbal working memory) and language acquisition have also been found to be impaired in populations of children who struggle to acquire language. Specific language impairment (SLI) is diagnosed in children who fail to develop language normally despite normal cognitive functioning. Experimental studies of these children have found that they have a disproportionate difficulty in acquiring the phonological forms of new words, and in the repetition of multisyllabic nonwords (Baddeley, 1998; Gathercole, 2006). In children newly diagnosed as having SLI, deficits in non-word repetition performance are believed to be more severe than the expected language deficits common to this population. This difference is believed to be present from preschool right through to adolescence, and extends even in older children whose language impairment has apparently resolved (Gathercole, 2006). Poor nonword repetition performance is also apparent in children diagnosed with dyslexia (Henry, 2012).

In typically developing children, the ability to repeat a nonword accurately has been closely and specifically related to vocabulary acquisition. In samples of children aged four, five and six, nonword recall was identified as an essential component of language acquisition, even after the confounding effects of age and non-verbal ability had been accounted for (Gathercole, 2006). Nonword repetition, however, may not always be as good an indicator of vocabulary learning potential in older children. Gathercole, Willis, Emslie, and Baddeley (1992) found that phonological memory is an important component in monolingual children up to the age of about 5 years. By eight years, it appears that prior vocabulary knowledge already stored in long term memory plays a far more important role in language learning than phonological storage. These findings have been confirmed by other studies in preschool and school beginner children (Haughey, 2002). The link between vocabulary knowledge and nonword repetition is therefore typically strongest during the early stages of acquiring a particular language. These findings are supported by neuropsychological imaging and anatomical studies which have found that the learning of new words and immediate serial recall (particularly in phonological sequences of nonwords) rely on the same neuroanatomical working memory structures (Gupta, 2009; Page & Norris, 2009; Szmalec, Duyck, Vandierendonck, Mata, & Page, 2009).

The associations between nonword repetition and language learning are premised on the assumption that they both rely on phonological storage. This hypothesis is widely supported by research evidence in typically developing populations (Gathercole et al., 1994), as well as those with verbal short term deficits (Baddeley, Papagno, & Vallar, 1988; Butterworth, Campbell, & Howard, 1986; Trojano & Grossi, 1995). This evidence indicates that nonword repetition ability is significantly constrained by phonological storage capacity, and that this capacity plays a key role in supporting learning of the sound structure of new words during vocabulary acquisition (Baddeley, Gathercole, & Papagno, 1998).

The findings described in the previous section have important implications for the acquisition of additional languages, as one of the key requirements to learn a new language is to learn its vocabulary. These words are initially heard as nonwords – a sequence of unfamiliar sounds and letters. This is relevant to the current study as the sample comprises children who have English as their second or even third language.

Service (1992) examined this relationship in a longitudinal study of Finnish children learning English. Nonword spans were a significant predictor of second language (L2) proficiency. Confirmatory findings were evident in a study by Cheung (1996) who found inverse correlations between nonword span with the number of trials it took 7th graders to learn new English words. This relationship existed only with students at lower levels of English proficiency, where students more proficient at English were seemingly less dependent on phonological learning. This is most likely a mirroring of what Gathercole et al. (1992) observed in children older than eight years as well. These two studies show strong support for the role of verbal working memory in the acquisition of new words and less so for the processing of familiar words in the beginning stages of learning a new language. The same relationship appears to exist between nonword repetition and vocabulary knowledge even in more advanced language learners. In Masoura and Gathercole's (2005) study of Greek children who had been studying English as a second language for an average of three years, nonword repetition ability was significantly related to knowledge of English vocabulary (r = .48, p < .001). However, the learning of not-yet-known English words paired with their Greek equivalents showed no significant association with nonword repetition scores, but was closely related to the children's existing English vocabulary. A review of other studies confirming these relationships (Ellis & Sinclair, 1996; Kormos & Safar, 2008; Martin & Ellis, 2012; Weissheimer, 2011) as well as a review by Hummel and French (2010)

leaves little doubt that verbal working memory capacity is heavily involved in the acquisition of an additional language.

The aforementioned discussion has presented findings which have shown that verbal working memory is crucial for language acquisition, but the reverse association is of equal interest, namely whether the processing and storage functions of working memory are also influenced by language processing. One piece of research which concerns this relationship is the executive control advantages for bilinguals. The research evidence suggests that the continuous language control and the repeated practice of switching between languages provides bilinguals with efficient executive control mechanisms. These are believed to have generalised transfer effects beyond the domain of language processing within those specific languages, and even extend into non-lingual tasks (Bialystok, Craik, Klein, & Viswanthan, 2004; Bialystok, Craik, & Ryan, 2006; Bialystok, Craik, & Freedman, 2007; Emmorey, Luk, Pyers, & Bialystok, 2008; Szmalec, Brusbaert, & Duyck, 2013)

There are a number of studies which vouch for the cognitive benefits of bilingualism. Bialystok, Craik and Freedman (2007) found that the onset of dementia is on average four years later in bilinguals than monolinguals. These advantages are believed to originate from the continuous demands to control the activation of lexical information or representations from the non-target language in order that they not interfere with current or ongoing language processing (Green, 1998).

There is a growing consensus between studies of bilinguals that languages frequently interact with, and are in competition with each other. This is evidenced by studies showing that reading speeds of bilinguals in their native language is faster if words in the additional language are similar (i.e. apple (English) and appel (Dutch), and that bilinguals experience tip-of-the-tongue phenomena more than monolinguals (Szamalec, Brusbaert, & Duyck, 2013). Despite this interaction and evidence of competition, there is also little evidence that bilingualism results in executive control failures. A number of studies have examined the error rates and speed/accuracy cost in samples who have both balanced and asymmetrical linguistic proficiency in more than one language. Bilinguals and monolinguals have been compared in a wide variety of tasks tapping executive control, with findings indicating that that they show better performance on a variety of executive tasks beyond those reliant on language control, but generalise to other situations where a dominant response must be

inhibited (Bialystok, Craik, Klein, & Viswanthan, 2004; Bialystok, Craik, & Ryan, 2006; Emmorey, Luk, Pyers, & Bialystok, 2008). Through the continuous control of language and language switching, it appears that bilinguals develop efficient control mechanisms which have generalisable effects to other cognitive functions. This idea is supported by a range of neuroimaging studies (fMRI (Wang, Xue, Chen, Xue, & Dong, 2007)) and PET scans (Abutelebi, 2008; Crinion et al., 2006) which have found that the brain regions involved in language switching tasks overlap significantly with the neural circuits identified in domain general executive control research (Brass & von Cramon, 2004). These studies are critiqued on the basis that they test processes of executive control, which is only an element of what the central executive achieves and largely ignore the other functions of the working memory system.

The findings from a number studies considering the effects of multilingualism on working memory capacity specifically (as opposed to executive functioning), have yielded mixed conclusions. Studies comparing the performance of simple working memory tasks in monolingual and bilingual children have found little to no support for a bilingual memory span advantage (Bialystok & Feng, 2010; Bonifacci, Giombini, Bellocchi, & Contemo, 2011; Engel de Abreu, 2011). These findings are criticised for being isolated to simple working memory tasks, and are believed to be biased as they rely on largely verbal measures and require children to recall lists of words or digits. Young bilinguals, such as those used in these studies, generally experience more difficulty than monolinguals with verbal processing. In both the Bialystok and Feng (2010) and Engel de Abreu (2011) studies, the bilingual groups obtained lower scores than monolinguals on measures of receptive and expressive vocabulary, which may be masking a latent bilingual advantage.

In a local study, Cockcroft and Alloway (2014) compared the working memory performance using the AWMA between a group of children who either spoke English as a first (n = 42, M = 87, SD = 5.3) or second language (n = 37, M = 104, SD =7.8). The results showed that the monolingual group significantly out-performed the multilingual group on measures of verbal working memory, and visuospatial short term- and working memory, even after accounting for the effect of non-verbal intelligence. No significant differences were identified on the measure of verbal short term memory. The authors attribute these findings to the fact that first and second language working memory capacities share substantial amounts of variance, regardless of native or foreign language processing. In that

way, high span individuals will possess more attentional resources to draw on than low span individuals, regardless of language (Alptekin & Ercetin, 2010).

More favourable findings have been found in studies measuring complex working memory tasks that are reliant on executive processes. Two such studies compared the performance of monolingual and bilingual children on different levels of working memory. In one study, 29 monolingual and 27 bilingual five year olds (*M* = 5.5 years, *SD* = 5.4) on a Simon type task, as well as measures to control for English receptive vocabulary and non-verbal intellect. The results found that bilingual children performed faster than monolinguals, and were more advanced in their progress in carrying our complex working memory tasks (measured by the lack of speed-accuracy trade-offs). In a second study, a group of 125 children (63 monolingual children, 62 bilingual) (*M* = 6.11 years, *SD* = 2.76) were compared on a computerised variant of the Corsi blocks which measures visuospatial working memory, where a spatially positioned matrix frogs (in a 3x3 matrix) were presented simultaneously or in a serial order. No language differences were reported for span, but bilingual children obtained higher scores than monolinguals in both conditions, particularly in the more difficult serial order condition (Morales, Calvo, & Bialystok, 2003). Similar findings regarding the superior executive processing of bilinguals are evident on a range of inhibition tasks involving interference suppression (Bialystok, Craik, & Ryan, 2006; Bialystok, Craik, & Luk, 2008; Carlson & Meltzoff, 2008; Martin-Rhee & Bialystok, 2008) and task switching (Bialystok & Martin, 2004; Carlson & Meltzoff, 2008).

The relative advantage of bilingualism is believed to be due to generalised superior capacities in a range of executive functions. The joint activation of several language systems in the mind of the bilingual is believed to create lexical conflict between competing responses. It is proposed that mechanisms of cognitive control are needed to resolve this conflict by maintaining activation of the relevant language in addition to suppressing interference from the non-target language (Bialystok, 1999; Ursino, Cuppini, & Magosso, 2010). This process draws on the executive processes of inhibition, selection, and maintenance or representations – all functions of working memory.

Before the discussion progresses, a brief post-script is necessary regarding the inconsistent references to both bilingualism and multilingualism within current literature. Within the South African context, the ability to speak more than one language is usually

referred to as multilingualism as the local context offers a diverse situation where children can often speak more than one African language in the home environment, and then learn English (and sometimes Afrikaans as well) at school. To that end, they are predominantly multilingual, rather than bilingual. Secondly, there are a number of contextual idiosyncrasies within this context which are different from the contexts of a lot of research regarding bilingualism. English may be taught at school as an additional language, but is also one of the dominant cultural languages in many communities (TV, media).

As discussed previously, there is considerable empirical support and subsequent critique of the phonological loop. Indeed it is this diversity of research evidence that was one of the initial drivers for the conceptualisation of the phonological loop in the first place. During the 1970's, the theoretical development of verbal working memory was in reaction to the limitation of a number of pre-existing models, but also to account for a range of experimental findings. The development of the visuospatial sketchpad has been less researched, probably because it was not born from the need to account for research findings. Rather, it was conceptualised and then sought research support from experimental evidence. This is likely to explain why there is such asymmetry in the volume of research support for the visuospatial sketchpad.

The Visuospatial Sketch Pad.

This component is specialised for the processing and storage of visual and spatial information, and of verbal information that is encoded in visual form (imagery). It was initially conceptualised as the seat of temporary storage and manipulation of spatial and visual information in the working memory system, as well as the planning of spatial movements Logie (1995) was perhaps more successful than Baddeley in proposing a more detailed model of the Visuospatial Sketch Pad (VSSP). He differentiates between a passive store, the visual cache, and an active component, the inner scribe. He suggests that the visual cache stores information about form and colour, while the scribe underpins the spatial, as opposed to the visual component of the system, and is involved in the storing of sequential information. The scribe is likened to the articulatory rehearsal component of the phonological loop, and is responsible for actively maintaining information in the passive visual store cache. Recent research also provides evidence to suggest that there might exist separate independent manipulative processes for the visual and spatial systems, rather than

a central scribe that serves both, but which is limited in its capacity to do both simultaneously (Mohr & Linden, 2005).

A series of studies similarly prove the existence of the VSSP, and its representational distinction from verbal working memory. Initially, Baddeley and colleagues (1975) attempted to produce visual image interference effects in order to prove the existence of a visual slave in the working memory system. The results report a marked tendency for imaginable phrases to be disrupted more easily when simultaneous processing of visual digits was required. Subsequent studies also showed that performance on visual letter tasks (describing features of a block capital letter image) was more accurate when the task was spoken than when it involved pointing – implying that a visuospatial memory task was separate to its verbal components (Brooks, 1968, Logie, 1995). Other studies requiring participants to track and place numbers in a matrix also suggested that simultaneous tracking disrupts visuo-spatial memory, but has a reduced effect on verbal equivalents (Baddeley, 1986). The distinction between verbal and visuospatial domains is also confirmed through a number of factor analytic studies (Alloway, Gathercole, Willis et al., 2004; Alloway, Gathercole, & Pickering, 2006; Gathercole & Jarvis, 2003; Gathercole, Pickering, Ambridge et al., 2004).

Upon further investigation of the visual-spatial sketchpad, researchers have found that its visual component is responsible for the construction and manipulation of mental images (i.e. doing mental calculations on an imagined abacus) and the encoding of unrelated sequences using mnemonic peg-word visualisations. A separate component is believed to be responsible for spatial location (i.e. mental mapping of a city) (Baddeley, 2007). This is supported by a series of six confirmatory experiments which found that a visual short term memory task was more strongly disrupted by visual rather than spatial interference, and that a spatial memory task was simultaneously more strongly disrupted by spatial rather than visual interference. The double dissociation supported a fractionation of the visuospatial sketchpad into separate visual and spatial components (Klauer & Zhao, 2004).

Neuropsychological research further supports the fractionation of the visuospatial sketch pad and the verbal domain. In a comparison of children with Williams and Downs Syndrome, researchers found that the often superior language skills characteristic of children with William's syndrome was accompanied by significantly better performance on a

measure of verbal short term memory, while children with Downs' Syndrome had significantly better performance on a visuospatial short term memory task. The authors attribute the double dissociation to the unique patterns of brain morphology characteristic of each syndrome profile, and provide evidence for the neurogenetic distinction between the two working memory representations (Wang & Bellugi, 1994). There also exists further neuropsychological support for the fractionation of the visual and spatial components of the sketchpad. Brain-damaged patients also show selective impairment in either aspect of the visuospatial system, with some patients displaying specific difficulties with visual (but not spatial) imagery, while others show selective impairment in the spatial imagery (Pickering, 2001). Further, the empirical measurement of ERP (electro-related potentials) in EEG research also display distinct underlying patterns of brain activation when measured during the retention of object and spatial information (Ruchkin, Johnson, Grafman, Canoune, & Ritter, 1997). These findings have been confirmed in a study using PET scans (Smith & Jonides, 1997).

Evidence for the modular structure and operation of the visuospatial sketchpad has been gathered from a much wider range of experimental paradigms than that for the phonological loop. The one paradigm has been focused on the short-term storage of visual and spatial images, while the other has involved studies that have explicitly required participants to generate, manipulate and inspect conscious visual images using particular theoretical paradigms, such as imagery mnemonics. The convergence of conclusions regarding these two types of evidence have assumed that they both reflect the operation of the sketchpad. Pearson (2001) argues that this fusion of conscious visual imagery and visuospatial short term memory is inappropriate, and suggests instead that Kosslyn's (1974, 1980) computational model of visual imagery has better explanatory power of the active and passive visuospatial processes which the sketchpad tries to account for. He argues that the computational model is able to able to provide a more detailed account of the processes and representations which underlie visuospatial storage and processing. His model has also been successfully implemented in a computer simulation in the processing of images, but has lacked the ability to successfully memorise these elements comprehensively. Kosslyn concedes that his visual buffer is ill-suited to the range of cognitive tasks attributed to the visual store, which could reflect deficits in both explanations of theory.

The visuospatial sketchpad is further critiqued on the grounds that if it really does encompass both visual imagery and visuospatial short term storage, then the effects of brain injury should result in impairment to both functions. Neuropsychological literature dissociates these processes which suggests that there are more components which underlie the activities of the sketchpad than the inner scribe and the visual cache (Pearson, 2001).

One of the other primary limitations in the initial computational working memory model was its inability to integrate information held in passive storage with pre-existing knowledge. In response, Baddeley (2000) added a fourth component to compensate for the obvious gaps in the models explanation of theory, the episodic buffer.

The Episodic Buffer.

The episodic buffer was introduced as a fourth component of the working memory model because Baddeley's (1974, 1986) existing understanding of the structure of the central executive proved insufficient. His model, comprising a central executive that hosts a general processing capacity, is therefore able to explain any result, but is not empirically effective in explaining the mechanics of how it works. Upon further research, the central executive, which was previously believed to play a purely attentional role and was itself incapable of storage, was able to manage separate attentional control processes which each performed a different function. Hence, the episodic buffer was introduced as an additional passive storage system that served as an interface between the three existing subsystems and long term memory. It is believed to serve as a storage facility for bound features (although unable to bind itself) that integrates perceptual information, subsystem information and long term memory into a limited series of *episodes*, or chunks. It is referred to a *buffer* in that it is believed to provide an interface between a number of different codes (visual, verbal and perceptual) as well as long term memory, semantic memory and episodic memory (Baddeley, 2000; Baddeley, 2007).

There is both empirical and neuropsychological support for the existence of the episodic buffer. The first is that Baddeley's original model (1986) lacked explanatory power without it. It is unable to account for the memory of large amounts of similar information, as well as the relationship of this information to long term memory, if these does not exist a passive store linking real-time information manipulation with content stored in long term memory (Chincotta et al., 1999). One such ability indicating the presence of this passive store is that of prose. It is widely accepted that when asked to recall a series of unrelated

words in sequence, most adults can recall five or six accurately. However, if the words comprise a meaningful sentence, a span of 16 or more is possible. This refers to the strategy of 'chunking', which Baddeley proposes is stored in a third 'transitional' storage system between the phonological store and long term memory which he named the episodic buffer (Baddeley, 2000). Its presence became further evident in neuropsychological research by Baddeley & Wilson (2002), who investigated the functioning of the episodic buffer in patients with impaired long term memory. As predicted, patients with impaired long-term memory were found to maintain good immediate prose recall. Further, this recall was better if the amnesic patient had good executive functioning, compared to those with more severe executive deficits.

There have been limited studies aiming to empirically verify the existence of the episodic buffer. However, these attempts have been widely criticised for lacking methodological rigour because it is not always certain that the episodic buffer functioning is actually the construct being measured (de Pontes Nobre, de Carvalho Rodrigues, Burges Sbicigo, da Rosa Piccolo, Zortea, Duarte Junior, et al., 2013). Baddeley (2012) himself recognises that accurate measurement of the episodic buffer is an ongoing and unresolved problem, and is quick to acknowledge the theoretical shortcomings of the episodic buffer in our current understanding of it. Some of these include the maintenance of rehearsal mechanisms, the impact of emotion of the working memory system and its relationship to consciousness.

Baddeley posits that like any buffer, the episodic buffer is limited in capacity which is assumed capacity of about four chunks. Studies have also shown that the episodic buffer appears to be relatively impervious to the effects of high demand of the central executive, but that maintaining this process of binding against distraction is attention demanding. This provides support for the ways in which the episodic buffer and the central executive are related, yet separable. Further, this process of maintaining binding in the face of distraction was evident in both verbal and visuospatial tasks which suggests that the buffer may be fractionated into components which are verbal, visuospatial, temporary or more enduring in nature. In this manner, the episodic buffer has been morphed into an entity that no longer has singularly responsibility for the binding of information, but has a passive storage component where bindings performed elsewhere can be maintained for further manipulation (Baddeley, 2012).

One of the primary criticisms of the episodic buffer is that there exists no clear methodological agreement for its independent assessment – an issue which Baddeley (2012) concedes is an unresolved problem. It is not always clear which process is being evaluated, particularly when assessment relies on standardised testing where such sub-processes are rarely specified, and experimental measurement of its operation often lack the ability to assess distinct binding processes (de Pontes Nobre et al., 2013). It is, however, the connection of Baddeley's multicomponent model with long term memory which has been one of the primary sources of its critique, where theorists such as Cowan (2005) and Ruchkin et al. (2003) describe working memory as an activated form of long term memory. Three alternate theories of working memory are discussed in relation to the ways that they account for the activities of working memory in a way other than that of the multicomponent model.

Cowan's Embedded Process Model (1999).

In contrast to the componential model proposed by Baddeley, Cowan (1999) proposes a model defined by functionality where there is a limited-capacity attentional focus that operates across areas of activated long term memory. Figure 2.5 provides a visual explanation of this process. In this approach, working memory is referred to as 'cognitive processes that are maintained in an unusually accessible state' (Cowan, 1999, p. 62). Cowan highlights three primary memory components as contributing compositely to working memory – activation, the focus of attention and awareness, and long-term memory. The large square represents all information in long-term memory, the irregular shape is the subset of memory that is in a temporarily heightened state of activation, and the small circle represents the current focus of attention or conscious awareness. The working memory system would then function to provide singular attention to that information that was currently in the conscious awareness of the individual. This would exist in the context of the type of memory that had heightened activation, and would draw on an understanding of long-term memory relevant to that being attended to. For example, if working memory was dealing with the navigation of a street map, the focus of attention would be on the current street, the activated memory would be the streets and direction previously navigated from, and the long-term memory would be the individual's previous navigational experience or memory of that particular area.

Cowan (1999) suggests that information is encoded in multiple ways depending on its representation, and that consequently the basic principles of encoding, maintenance and retrieval would be similar regardless of the representation. He does concede though, that despite his model allowing for more than the bi-modal representations of Baddeley (auditory and visuospatial) to include non-verbal sounds or tactile sensations, that the greatest interference appears to come from additional similar stimuli in that modality, suggesting that code-specific encoding is apparent in the temporary activation of memory. In contrast to Baddeley's model which suggests that maintenance of information is done through a modal-specific rehearsal mechanism, Cowan's model supports the idea that maintenance is done by a process of continuous active searching which serves to reactivate information in the area of focused attention. Effective retrieval of this information is therefore achieved when the correct item is directed into the focus of attention. This retrieval must occur within the time-limited framework of active searching (maintenance). This process of encoding, maintenance and retrieval is guided by the central executive.

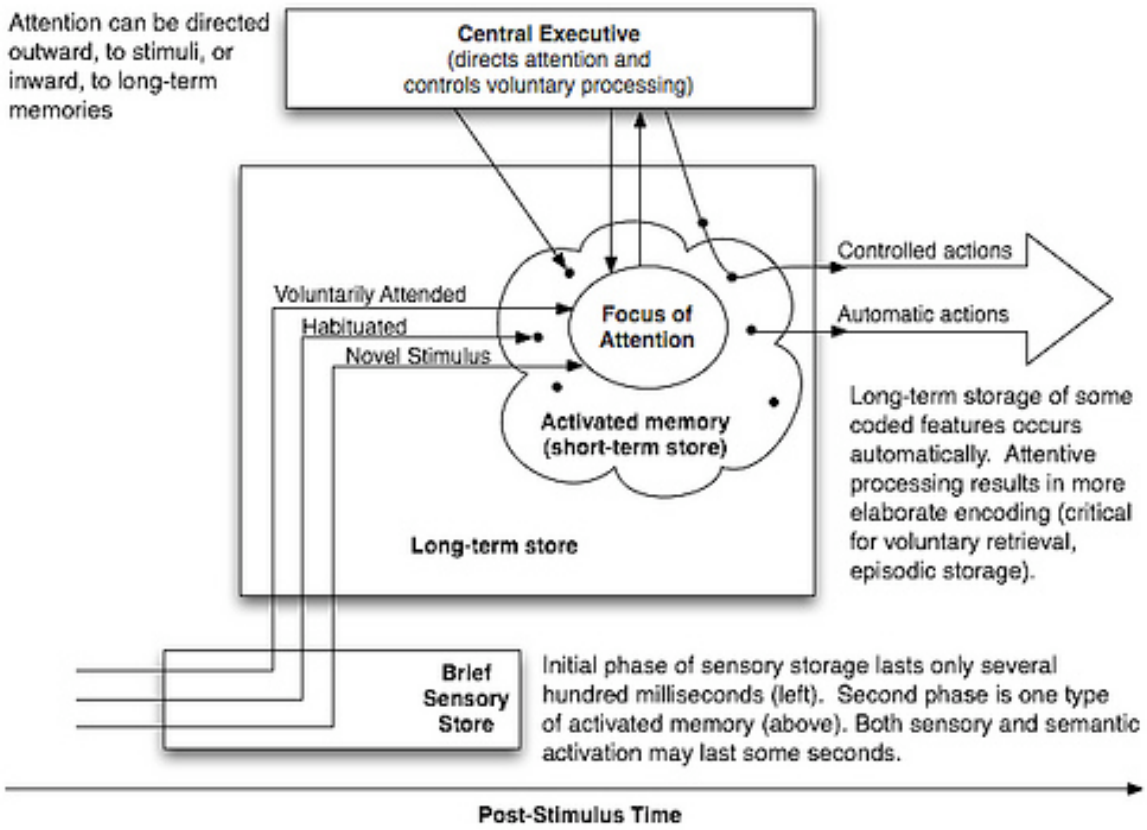

Figure 2.5. Cowan's Working Memory Model. Adapted from "Working Memory *Models of Working Memory: Mechanisms of Active Maintenance and Executive Control",* by A. Miyake & P. Shah, 1999, p.64. Cambridge: Cambridge University Press.

Again, in contrast to the multicomponent model, Cowan (1999) argues that the central executive acts as an attenuating filter that guides the processing by the activated memory and the focus of attention. While unable to fully filter out unattended to stimuli, the central executive serves to direct attention from the activated memory to the focus of attention to allow for the successful manipulation of cognitive information. The role of the central executive is not to filter out all unattended to stimuli, as all stimuli will activate some aspects of long term memory. Instead, this habituation to unattended stimuli allows for the attention to other stimuli, should it be sufficiently novel, or decided upon by the central executive.

Cowan's model lies in the middle of the spectrum of the unitary nature of working memory. While it does not distinguish between separate, modality-specific components like that of Baddeley's, it does differentiate between activation and attention by specifying differing levels of the latter. It also postulates a relationship to long-term memory, in that any information stored there that is associated to the task at hand is easily accessed as it is drawn into the activated memory (short term virtual memory).

Each aspect of this model has particular functional limitations. Evidence from various studies that have considered the effects of interference and decay across modalities have shown that there appears to be a time limit to the activation of memory, with memory fading within ten to twenty seconds unless it is reactivated through re-stimulation (Cowan et al., 1997). Conversely, the focus of attention appears to be limited by its capacity rather than time – limited to very few unrelated items (between three and five), while chunking or the use of an associative structure can raise this somewhat. One of the primary nodes of difference between that of Cowan and Baddeley is the limitation to the capacity of the attentional focus. Unlike Baddeley's proposition that mature, typical working memory storage capacity is seven units, he proposes that this is four (although he refers here to four chunks or episodes which may contain more than a single unit of information). The focus of Cowan's work is principally around the links between (in Baddeley's terms) the central executive and the episodic buffer. This is a particularly under-developed area within the multicomponent model, but their differences within this area of focus can best be understood in this way: Baddeley assumes that information from long term memory are downloaded onto the episodic buffer, while Cowan suggests that the 'links' or addresses to such information in long term memory are held in the active memory for ease of retrieval

(Baddeley, 2009). Cowan refers to the system or platform on which complex processing and storage is initiated and maintained as 'activated long term memory', but does not provide the same degree of explanation about what happens on this platform. Although he does offer some explanation of his model with verbal short term memory, it is very similar to that achieved by the phonological loop. To that end, while they differ in terminology and structural focus, there are many similarities between that of Cowan and Baddeley.

Engle, Kane and Tuholski's Inhibitory Control Theory (1999).

Engle, Kane and Tuholski (1999) developed their own model of working memory which deviated from Baddeley's model in its elaboration on the domain-specific codes of representation, as well as through its emphasis on the distinction between short-term and working memory. Engle et al. (1994) propose the idea that differences in controlled processing are central to an accurate understanding of the construct. They put forward a model which is similar in over-arching structure to the multicomponent model, but different in its unitary functionality. They suggest that the central executive acts as a supervisory attention system that is ultimately responsible for working memory capacity, and controlled and focused attention. It functions to activate working memory through a process of controlled information retrieval, to maintain this activation, and then block the interference through the inhibition of distractors.

The encoding, maintenance and retrieval of information is not specific to the auditory and visuospatial codes like that in the phonological loop and the visuospatial scratch pad, but instead operate as domain-specific codes that could be motoric, phonological, tactile, visual, spatial etc. in nature. Encoding and representations are as varied as the formats for perception, emotion and thought. The maintenance of information in the system is done through a process of controlled attention and activation (rehearsal being one of them). These take the form of a series of grouping skills, coding strategies and procedures necessary for maintaining activation by the central executive. Short-term memory is therefore a component of this system, embedded within long-term memory, that consists of those traces of information that are active above a particular attention threshold (easily lost due to decay or interference), or those that receive further activation by becoming the focus of attention. To that end, the many representational formats, controlled attention and the procedures and skills for maintaining this activation constitute a unitary system.

In order to support this view, the authors tested 133 subjects on a variety of tasks that they believed would test both working memory (reading span, operation span and counting span), and short-term memory (forward and backward word spans with dissimilar and rhyming words). They also administered the Ravens and the Cattell Culture Fair tests as a measure of fluid intelligence (g_f). A confirmatory factor analysis revealed that working memory and short term memory are highly related but separable constructs. Their analysis also showed that the model had a strong connection between working memory and fluid intelligence, which was not mirrored for short-term memory.

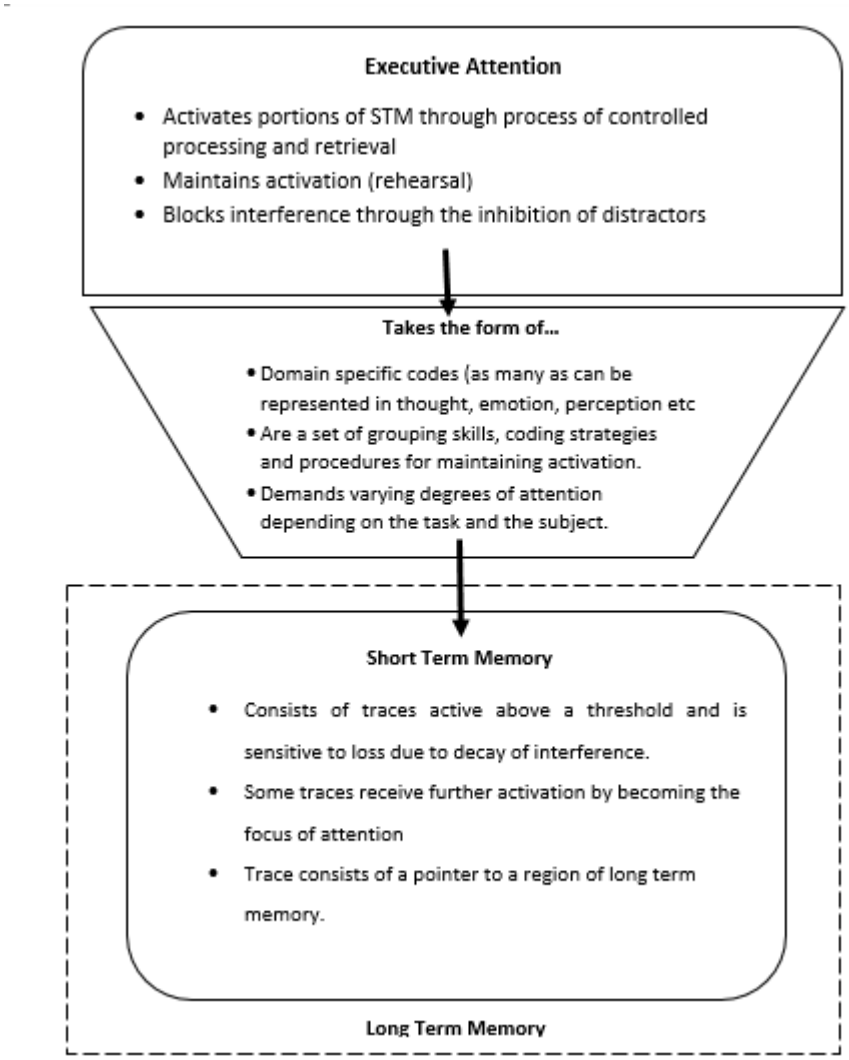

Figure 2.6. Engle, Kane & Tuholski's Inhibitory Control Theory of Working Memory. Adapted from "Models of Working Memory: Mechanisms of Active Maintenance and Executive Control", by A. Miyake & P. Shah, 1999, p. 102-133. Cambridge: Cambridge University Press.

Engle's model also makes significant contribution to the explanation of the capacity limits of the working memory system by nature of inhibitory processes, which are believed to be crucial to shielding memory content from interference and disruption. The central executive proposed by Baddeley is primarily a componential model made up of a number of executive functions. The singular reliance on a single, fractionated executive function – inhibition – for working memory capacity limitations is critiqued by Baddeley as being overly simplistic. However, the model is similar in that it relies on domain specific codes for encoding, representation and maintenance, makes a clear distinction between short term storage and active processing, relies on albeit a simplistic central executive to direct attention, and has the capacity to interface with trace recognition within long term memory.

Long Term Working Memory Model.

Ericsson and Delaney's (1999) model adopts a functionally defined approach which conceptualises working memory as the mind's ability to efficiently maintain selective access to information required to complete a given task from the vast resources (information and procedures) available in long term memory. This is performed by a wide range of different mechanisms, of which the traditional short-term working memory is only one. In order to do this, selective attention to both relevant procedures and presented, retrieved and generated information need to be maintained. Further this information has to be distinguished from the vast amount of other knowledge and procedures available in long term working memory.

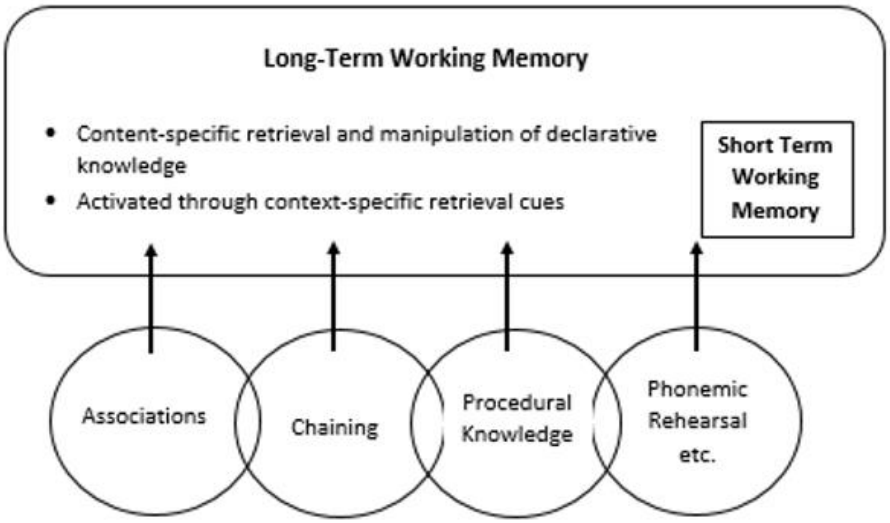

Figure 2.7. Ericsson & Delaney Long Term Working Memory Model. Adapted from "Models of Working Memory: Mechanisms of Active Maintenance and Executive Control", by A. Miyake & P. Shah, 1999, p. 257-297 Cambridge: Cambridge University Press.

Efficient long term working memory (LT-WM) then is the acquisition of a set of skills which allows people to anticipate the retrieval demands of information within a particular context, and then store only the relevant information in a way that allows optimal access to this information with appropriate cues. A myriad of cognitive strategies are employed here to assist with encoding and retrieval, such as temporal recency cues (recency effect), explicit retrieval structures (photographical memory, phonemic rehearsal), or association within complex cognitive structures assisting retrieval (mnemonics, chunking).

LT-WM is usually domain specific as the procedures and information elicited from long term memory are usually semantically linked to input material (stimuli), and that an association between these two exist in a particular context to allow for optimal functioning (i.e. the current challenge of teaching arithmetic in iPads in classrooms, but requiring tests and exams to be written in pen and paper format). However, some representations that require domain neutral skill (planning) cannot be semantically encoded because retrieval would require a replication of that encoding. Instead, LT-WM employs spatial and hierarchical cognitive strategies as an executive operator in order to allow adaptation to other activities.

Within this model, there is no universal capacity limit for how much information can be kept accessible during the performance of a specific task. Instead, as proficiency for a particular domain is developed, the cognitive mechanisms for LT-WM encoding and retrieval are acquired so that the retrieval demands of that domain are met. As the demand increases, so the mind deliberately refines representations and associations within the working memory system to monitor, plan and evaluate performance. Limitations (due to interference or decay) are considered a function of limitations in the various strategies in phonemic rehearsal, chunking, and mnemonic encoding.

Ericsson and Kintsch (1995) proposed this concept as a theoretical explanation of the performance of expert mnemonists, and suggest that the effective activation of long term memory is essential to the functioning of working memory. Baddeley (2012) contends that this does not warrant a novel conceptualisation of working memory, but rather provides substantive evidence for the ways in which working memory and long term memory interact successfully. He offers figure 2.9 in response to this model by way of explanation that working memory is a complex interactive system that is able to provide an interface

between cognition and action, and is capable of handling information in a range of modalities and stages of processing. Incoming information is therefore processed by systems that are influenced by long term memory – one of which is the working memory system.

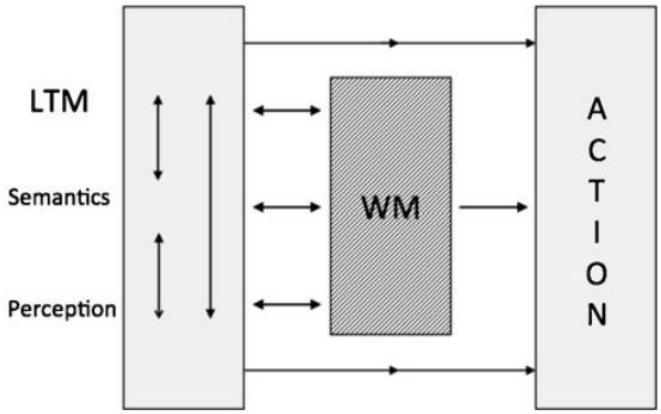

Figure 2.8. Baddeley's location of working memory within the environment and behaviour. Adapted from "Working Memory: Theories, Models and Controversies" by A.D. Baddeley, 2012, *Annual Review of Psychology, 63,* p.18.

The following section presents an integrative comparison and critique of these three alternative models to the multicomponent model proposed by Baddelely (2000). Issues of representation and model structure are central to any integrative discussion on the nature of working memory. More specifically, the processes of encoding, retrieval and in particular that of maintenance are essential processes which need accounting for if the model is to sustain major criticism. Cowan (1999) and Engle et al. (1999) posit that encoding consists of activating the appropriate features of long term memory, while Ericsson and Delany's model (1999) suggests that proficient LT-WM performance is actually due to the efficient creation of retrieval structures (encoded mnemonics) during initial perception which is done in a domain specific manner, often with semantic support. Within Baddeley's multicomponent model (2000), domain-specific encoding and retrieval are the responsibilities of the two modular salve systems; the phonological loop (primarily the phonological store) and the visuospatial sketchpad (the visual cache for visual information, and the inner scribe for spatial input) which encode, manipulate and process verbal and visuospatial input respectively (Miyake & Shah, 1999).

Maintenance of information is a fundamental component of any working memory model, and here most of the models agree that cognitive rehearsal (largely verbal) is

primarily the vehicle through which this is achieved. In cases where the rehearsal process is not specified, limitations to maintenance are usually accounted for by the nature of the control or executive attention mechanism. Retrieval is a difficult construct to discuss within this debate as its definition fluctuates rapidly throughout models. Within both Cowan and Engle's models, the retrieval of active and easily accessible information within long term memory is the very definition of working memory itself. Within Baddeley's model maintenance is delegated to the two structurally asymmetrical, yet functionally mirrored capacities of the articulatory rehearsal mechanism (for verbal information) and the inner scribe of the visuospatial sketchpad.

These three processes of encoding, maintenance and retrieval are inextricably linked to the representation of information within the model. Baddeley's (2000) model of domain specific slave systems is well-researched and shows sound empirical support. Their model postulates different codes for information housed in the phonological loop (auditory) and visuospatial sketchpad (visual), as well as specifying different codes within the latter – the visual cache for visual information and the inner scribe for the spatial component. Many other models support this view by also proposing domain specific codes or representations. Both Cowan's and Engle's models assume domain specific codes for working memory, but point out that all types are vulnerable to similar limitations (decay and interference). This is supported through empirical testing which relies on increased interference when similar codes interface with each other, and further by functional neuroanatomy where different regions of the perceptual and motor cortices have localised specificity for different types of encoded information (Miyake & Shah, 1999). Conversely, other models are less committed to domain specificity. Ericsson and Delaney (1999) do not specify the nature of codes in their model because they do not believe that skilled encoding within codes is essential for efficient working memory performance in everyday life.

Another pivotal issue in the issue of model consensus is that of the nature of what Baddeley termed the central executive, or the method of attentional control. The componential models (like that of Baddeley, Cowan and Engle) have no difficulty in identifying a central structure responsible for unitary control, attentional control and maintenance of other working memory functions (Miyake & Shah, 1999). In contrast, Ericsson's LT-WM model states that a central executive is not necessary as retrieval strategies are responsible for adequate recall. However, because there is very little

explanation as to exactly how this decision making is done if a central processing system is obsolete in relation to the system's functionality, the models explanatory power is subsequently diminished. To that end, a componential non unitary conceptualisation of working memory appears to have better theoretical and empirical support.

Subsequent to our understanding of the componential parts of working memory, is the constructs organisation as either a unitary or non-unitary entity. Within the models discussed here, there exists much debate about whether working memory is a single entity relying on inseparable pooled and interdependent resources, or if it a mechanistic model comprised of a series of complimentary sub-components which work together to form a greater, functioning whole. This distinction is of course not a polarised concept and there are models which lie on a spectrum somewhere between the two. One of the points of difference on this spectrum is the number of componential parts of which the model is made up. For instance, Engle, Kane, and Tuholski (1999) emphasise domain-general components of working memory, while Baddeley & Logie (1999) and Cowan (1999) propose explicit domain specific components. Many more models differ in the number of domain-specific representations exist, and at what level of the system they are sourced from (i.e. perception, encoding, maintenance or retrieval). The unitary nature of working memory does not have much in the way of empirical support. Experimental evidence across model types which tests procedural skills such as articulatory suppression or domain similar interference suggests that there does exist a degree of differentiation of the construct into operationally unique components. Further, the assistance of functional neuroanatomy and a study of deficit functioning in the context of legions across localised areas of the brain also suggests that the functional components of the model are separable. This would suggest that working memory is a non-unitary construct.

A componential non-unitary understanding of the model would therefore suggest that there is then no single, all-encompassing limiter of working memory capacity and functioning. Limitations to the system are created through limitations of the individual components, and are initiated as a result of simultaneous processing (i.e. articulatory suppression, excessive demand on executive resources). The more unitary models (LT-WM, architectural computerised models) are less specific about how the system's capacities are inadequate, while the componential (Baddeley, Cowan, Engle et al.) models can provide

clear evidence that limitations are enforced because of domain-similar interference, or decay due to capacity limitations of the central executive.

Lastly, the models also differ in their conceptualisation of the differences from, and relationships to, working memory, short-term memory and long-term memory. The multicomponent model is very clear in its distinction between the three, and its archetypal structure supports this by proposing that short-term storage does not allow for the subsequent manipulation and processing that is evident in working memory. Further, long-term memory is an essential and additional link to the model accessed through the episodic buffer, but whose contents remain a separate entity. Conversely, the model of Ericsson and Delaney (1999) support the idea that working memory is a form of long-term memory which is merely processed, attended to or lifted to consciousness in a way that allows for real-time cognitive processing.

The models discussed previously have a lot of similarity, and according to Baddeley (2012) appear to differ largely in their focus than represent a major theoretical distinction. The nodes of empirically verifiable commonality converge around the notion that working memory is non-unitary in nature, with specific, inter-related components which work together to create what we call working memory. Empirical support also provides evidence of an identifiable central executive or processor which is responsible for the activation of attention, inhibition, and maintenance of complex cognitive processes. There is also evidence in support of domain specific representation, which are usually differentiated between verbal and visuospatial stimuli, and include both short-term storage and information processing. Lastly, a theoretically robust model also appears to have strong links to, but remain separate from, long-term memory in the sense that they involve a degree of active processing therein.

The most empirically verified model aligned to these characteristics appears to be that of Baddeley (2000), which forms the theoretical understanding of working memory that this study is subsequently based on (Baddeley & Hitch, 1974, Baddeley & Logie, 1999; Baddeley, 2007). While there do exist other models of working memory, Baddeley's multicomponent model (2000) has been drawn upon in this research for multiple reasons. The first is that his model has considerable empirical research support that serve to verify the existence of its components (Pickering, 2001). Secondly, many of the other proposed

models are very similar to Baddeley's and appear as extensions or variations of the original model, with differences existing in their understanding of the association between working memory and long term memory (Cowan, 1995; Cowan, 2005; Kintsch, Walter, Patel, & Ericcson, 1999; Oberauer, 2002). Lastly, Baddeley's multicomponent model (2000) has sound theoretical alignment with a psychometrically sound measure of working memory, and the choice of instrument for this study. The Automated Working Memory Assessment (AWMA), developed by Alloway (2000) assumes the existence of a central executive and two functionally separate slave systems that are specialised for the synthesis of verbal and visual information.

The structural robustness of the multicomponent model (2000) is largely gained from its ability to provide a theoretical account of working memory functioning in the face of alternative and competing accounts of the functioning of working memory, as well as extensive experimental data. However, the majority of this empirical evidence has been gleaned from adult samples. It is therefore unclear whether the fractionation of working memory as evident in adult samples is present or identical in structure and dominance in younger typically developing children. The following section considers these issues within the context of development.

Developmental Structure of Working Memory

While the previous discussion has highlighted the salient features of a theoretically robust working memory model that is able to adequately account for empirical findings, it remains incomplete without its location within a developmental context. What follows is a discussion of the evidence for further developmental fractionation of working memory functioning at various points in childhood along two axes of difference: domain specificity, and the distinction between the simple (storage) and complex (processing) capacities of the working memory system. It culminates in the integration of the models of working memory and this section, and presents the typical expectations of working memory development for school beginners.

There are a number studies which have considered the structure of working memory and its development across the lifespan using confirmatory factor analysis (Alloway, Gathercole, Willis et al., 2004; Alloway, Gathercole, Pickering et al., 2006; Alloway & Alloway, 2013; Gathercole, Pickering, Ambridge et al., 2004). One such attempt investigated the structure of working memory and associative cognitive constructs in young children,

where researchers administered a series of measures assessing complex memory span, phonological short term memory, sentence repetition, phonological awareness and non-verbal ability to 633 children from broad demographic background in the United Kingdom (*M* = 59.5 months, *SD* = 3.7). This age band of child was tested as this is when children begin formal schooling in the UK. The results from a confirmatory factor analysis of the study support working memory as possessing a modular structure, with clear distinctions between the central executive, episodic buffer and phonological loop. Inter-model chi square difference tests reveal that a five factor model is the most appropriate for this age group, where loadings were significant when corresponding to the phonological loop (phonological short term memory measures), the central executive (complex memory span measures), the episodic buffer (sentence repetition measures), phonological awareness and non-verbal ability. The study shows clear evidence for the distinction between phonological short-term memory capacity (a component of working memory) and phonological awareness (a prerequisite for literacy), but makes little reference to the developmental importance of domain specificity within the early school context (Alloway, Gathercole, Willis et al., 2004).

Three subsequent studies have considered this very distinction within a developmental context. Alloway, Gathercole and Pickering et al., (2006) explored the verbal and visuospatial structure of working memory in children between 4-11 years of age using the twelve subtests of the AWMA. Sample sizes provided sufficient power (*N* = 708), and results were striated and compared across three age bands: 4-6 years, 7-8 years and 9-11 years. Similar to the method employed in the previous study, a series of confirmatory factor analyses were conducted using various theoretical models to identify which of those accounted for sufficient model fit. A series of chi square difference tests were also conducted across the age bands to identify the existence of developmental changes. The study made a number of salient findings. The first was that working memory steadily increased in capacity as children got older, but that verbal short term memory performance levelled off sooner than other tasks. Secondly, the results showed conclusive evidence for the existence of a three factor model, with related but separate constructs representing verbal and visuospatial storage, and a third factor representing the shared variance of verbal and visuospatial working memory tasks (a central executive responsible for the delegation of attention, inhibition and processing). Thirdly, the study identified a difference in the relationship between verbal and visuospatial working memory across development. While

the relationship between verbal short-term memory and the central executive remain constant across the age bands, it appears that younger children draw on more executive resources than older children when performing visuospatial short-term tasks. Lastly, the study found that the strength of association between verbal and visuospatial short term storage increases between the youngest (4-6 years) to oldest (7-11 years). This is believed to be because younger children rely more on visual codes initially, but begin to employ rehearsal strategies to recode visual material into verbal codes (Alloway, Gathercole & Pickering et al., 2006).

This hypothesis has found support from neuroimaging studies as well. The behavioural performance of a group of five and six year olds (*n* = 59), were compared to that of a group of eight and nine year olds (*n* = 92) on three tasks measuring the functioning of the lower prefrontal cortex (LPFC) – a visuospatial and auditory n-back working memory task, and a go/no-go response inhibition task. Multiple regression analyses showed significant correlations between the three components in the younger cohort, while three discrete and independent functions were apparent in the older cohort. The results suggest that in the LPFC, different modalities of working memory and response inhibition share a common neural system during early childhood, whereas different modalities recruit different neural systems in older children (Tsujimoto, Kuwajima, & Sawaguchi, 2007). Shah and Miyake (1996) also suggest domain specific fractionated systems for spatial and oral executive functioning in younger children (5-6 years).

The relative consistency of this three factor model of working memory, in place by the age of six years old, is supported by previous research by Gathercole, Pickering, Ambridge et al. (2004) who applied a very similar methodology using a manual measure of working memory performance (Working Memory Test Battery for Children). Their study measured the modular structure of working memory on 736 children between the ages of four and fifteen years from five different school across the UK. Their findings conclude that, in resemblance to the mature adult working memory model, there is a loose tripartite structure corresponding to the functions of the phonological loop, the central executive and the visuospatial sketchpad in place from as young as six years of age. Similar to Alloway, Gathercole and Pickering et al., (2006), the authors note a particularly significant association between central executive and visuospatial factors. This is in spite of the fact that the

visuospatial tasks were devised to minimize opportunities for verbal recoding and involved neither verbal inputs nor verbal recall, and provides support for claims that visuospatial memory is significantly dependent on support from domain-independent resources associated with the central executive. The authors also note that correlations between factors associated with the phonological loop and the central executive increase from 0.73 for the 6- to 7-year-old group to 0.90 and greater for the two older age groups (10- to 12-year-olds and 13- to 15-year-olds). The closer links between these two factors in older children is speculated to arise from developmental increases in complex processing efficiency. Hence, the demands placed on the working memory capacities in older children by the various complex span tasks (resequencing the numbers in the case of backward digit recall, dot counting in counting recall, and sentence processing in listening span) may diminish. This suggests that while the phonological loop and visuospatial sketchpad are clearly independent from an early age, the more complex processing capacities across both domains appear to develop later. Within the phonological loop, this is hypothesised to be due to a lag in the employment of rehearsal as a maintenance strategy.

In an attempt to understand developmental differences in verbal short-term memory capacities in children, a cross-sectional study considered the proven relationship between speech rate and memory span in a series of group experiments. Contrary to expectations, individual correlations between these two abilities (speech rate and memory span) were not significant when corrected for age. The results suggest that the speech rate of younger children (aged 5) did not predict memory span at all. Instead, speech rate was associated with holding the same amount of span for a longer period of time (5 seconds over the usual 2 second expectation). They also found little evidence of the articulatory suppression effect in children under 9, suggesting that the accurate use of rehearsal is less developed in young children, where they instead make errors or use it inconsistently (Henry, 1994). These immaturities in the verbal working memory system of pre-literate children are believed to be a function of developmental fractionation between the short term storage and processing components of the phonological loop. It appears that the rehearsal component necessary for efficient performance in complex verbal working memory tasks is limited in children under the age of nine years, and represents a developmental fractionation of the verbal domain. While both groups are capable of short term storage, complex verbal processing (as a consequence of adequate rehearsal) is only possible in older children.

There also appears to be a reduction in the reliance on the verbal short term component of the phonological loop in older children, as children over the age of eight years are believed to rely less on phonological storage to increase vocabulary, but to rely rather on semantic associations present in long term memory. However, while the established developmental association between nonword repetition and native vocabulary knowledge declines with increasing age beyond the middle childhood years, the link with the ability to learn novel words persists in older groups of children who have established working memory impairments (Gathercole, 2006) A longitudinal follow up of a group of eight-year-old children who were first recognised as having very poor nonword repetition abilities at five years of age still showed significant impairment in learning new phonological information under controlled laboratory conditions (Gathercole, Tiffany, Briscoe, Thorn, & The ALSPAC Team, 2005), despite having 'caught up' to normal native vocabulary knowledge (Gathercole et al., 2005). Hence, it appears that the delayed graduation from reliance on verbal short term- to working memory dominance within the verbal domain has lifelong consequences.

The development of short term visuospatial memory has been studied in two cohorts of children, aged five and seven years (Walker, Hitch, Doyle, & Porter, 1994). Children were required to remember both the location and colour of three coloured shapes that appeared in a random spatio-temporal order. The results found that children's memory, across cohorts, for shapes was impaired where the shapes were visually similar. This result is unsurprising and aligns well with previously well-substantiated claims that there are distinct abilities within the visuospatial memory system devoted to content (visuo) and location (spatial) which are sensitive to interference and decay. Secondly, the authors found that memory for location improved with age, while memory for colour remained the same when age was controlled for across the cohorts. They also found that older children were able to hold a visual representation of the conjunctions and features in shapes for a longer period of time (known as an object file) and make decisions based on it even when the object was no longer present (Walker, Hitch, Doyle, & Porter, 1994). These findings provide evidence that there are differential capacities for these skills to increase over time across developmental trajectories. This latter finding provides evidence for a similar, albeit not symmetrical, shift in the developmental dominance of visuospatial storage capacities to those capable of maintenance and processing. It would appear that younger children rely more heavily on

visuospatial storage, and that only older children are capable of manipulating the maintained material within the sketchpad.

Another study considering the developmental fractionation of visuospatial working memory in children observed different developmental trajectories in two spatial memory tasks (Logie & Pearson, 1997). Using both recall and recognition procedures, Logie and Pearson found that pattern memory developed more rapidly with age than memory for movement sequences. These results were, however, not consistent with the findings of a more recent study which suggests that the results in Logie and Pearson (1997) are a function of measurement scales, and not a developmental shift (Gathercole, Pickering, Ambridge et al., 2004; Pickering, 2001).

The aforementioned studies have provided clear evidence for a developmental distinction between passive short term and active processing capacities of the modular working memory structure, where the processing abilities in younger children are less developed. There was evidence for this in studies concerning both the verbal and visuospatial domain. The fractionation of working memory appears to be along the axis of domain specificity as well. Across a range of studies, findings converge to show that young children tend to code information in visual form, but that this soon switches to encode visual information through phonological mechanisms (Fenner, Heathcote, & Jerrams Smith, 2000; Hitch, Halliday, Schaafstal & Schraagen, 1988; Luciana & Nelson, 1998; Palmer, 2000; Pickering, 2001).

Gathercole and Jarvis (2003) found further evidence for domain specificity within the active processing component of working memory. Their study employed confirmatory factor analyses on the working memory measures of both 11 year old (n = 55) and 14 year old (n = 73 children. Their data did not provide strong evidence for the model of working memory incorporating a domain-general central executive and subsidiary domain specific storage systems. Instead there were significant dissociations between verbal and visuospatial working memory factors. Similar findings have been found in an older sample of undergraduate adults (Shah & Miyake, 1996). This evidence indicates that the limited processing capacity associated with the central executive appears to be domain specific. However, the extent to which this domain-specific fractionation of central executive resources extends back into the middle and early childhood years remains unknown at

present. It also stands in opposition to the domain general central executive originally proposed by Baddeley and Hitch (1974), but has limited empirical support. One explanation for this distinction could be its interaction with domain specific fractional lags between visuospatial and verbal material with young children.

These developmental studies offer support for a similar fractionation of working memory along the nexus of domain representation by providing evidence for an impartial reliance on one of the domain specific codes within the working memory system, and suggests that there may exist a 'switching over' from the reliance on visuospatial skill to verbal skill during the first two years of formal schooling. One study compared the phonemic and visual similarity effects in children between the ages of 5-10 years old (Longoni & Scalisi, 1994). Unsurprisingly, the researchers found evidence of phonemic similarity effect when information was presented in an auditory manner in both age groups. In other words, the recall of a set of phonologically similar words was much more difficult than the recall of a set of phonologically dissimilar words, regardless of age. However, when such similarity was presented in a series of similar or dissimilar pictures, the effect was only present in the older group. This suggests that younger children do not rely on a speech based code in memory like older children do, but are more modality-specific in their memory functioning. This refers to an immaturity in the cross-modal memory modes in younger children that develop as children learn to read. These findings were found to be independent of cultural differences or of the experimental material used (Longoni & Scalisi, 1994), and are consistent with the view that younger children rely more on visual codes initially, but recode verbally with increasing literacy (Hitch, Halliday, Schaafstal, & Schraagen, 1988). This domain dominance of visuospatial over verbal encoding in younger children is well-established in other literature as well (see: Alloway, Gathercole, & Pickering, 2006; Kemps, Rammalaere, & Desmet, 2000; Pickering, 2001).

Confirmatory evidence of this developmental domain preference was also found by Gathercole et al. (2004) who investigated developmental changes in the structure of working memory through a confirmatory factor analysis in children across a series of age groups (4-15 years). The authors found corresponding and increasing correlations between measures of the phonological loop and the central executive in older children. They posit that the processing demands of the complex verbal span tasks diminish in older childhood, which

could explain the preference for visuospatial processing to encode information into memory in pre-literate younger children under the age of seven. This assertion is supported by other researchers, but the mechanisms behind why this takes place remains unclear (see Hitch & Halliday, 1983; Hitch, Halliday, Schaafstal, & Schraagen, 1988, Pickering, Gathercole, Hall & Lloyd, 2001). Some suggest the shift is related to changes in the storage capacities of the visuospatial sketchpad (Logie & Pearson, 1997), while others propose that it relates to age related increases in the use of effective cognitive strategies, the accumulation of long-term knowledge in relation to the visuospatial sketchpad, or increased processing support by the central executive (Gathercole et al., 2004). Cowan, Nugent, and Elliot et al. (1999) propose that this distinction in the verbal short term stores in younger children relate to limits in the attentional capacity of the working memory system, which increase with age and consequently allow for the shift to preferential verbal encoding.

In summation of the developmental research, factor analytic studies confirm much of what is already known regarding working memory: the greatest gains in working memory capacity are found in childhood, with maximum capacity reached in the teenage years. The decline of functioning into old age appears to be a slow one, with visuospatial working memory performance declining more rapidly than verbal capacity (Alloway & Alloway, 2013). Working memory capacity appears to be a relatively stable construct which shows incremental growth relative to age across development. Additionally, there appear to be developmentally indicated fractionation of the construct within typical child populations. Firstly, the transition from a reliance on passive short term storage to active processing is apparent in younger children, with an increased capacity for complex manipulation only appearing at around the age of eight or nine. Further, there is also a domain-specific preference for visuospatial encoding in younger children, who only begin to switch to verbal encoding from around the age of six or seven years. Therefore, while the central executive in mature typical adult samples appears to be domain general, the synchronous functioning of executive functions responsible for complex processing remains domain specific in younger children because of the lag in utilising strategies such as rehearsal to recode visual information using verbal labels (Alloway, Gathercole, & Pickering, 2006; Hitch & Halliday, 1983; Hitch, Halliday, Schaafstal, & Schraagen, 1988).

One critique of the multicomponent model is its limited ability to adequately account for development throughout childhood. Very little explanation has been offered regarding the mechanisms behind increases in the capacities of any of the structures of the multicomponent model, but the increases in verbal span are believed to be because of an increase in articulatory speed within the sub-vocal rehearsal strategy (Kemps, Rammelaere, & Desmet, 2000). The argument is based on the logical suggestion that children have slower articulation rates than adults, so they rehearse information in the articulatory rehearsal mechanism more slowly. With age, increases in the reading or articulation rates allow for faster rehearsal rates, and hence, higher memory spans. While this simple explanation has received sound empirical support, it is not without critique (Henry, 2012). A number of correlational analyses have found significant associations between articulation in both adult and child samples, these effects disappear when individual data points are used instead of group means, and when age was controlled for in very young children (Ferguson, Bowey, & Tiley, 2002; Gathercole, Adams, & Hitch, 1994; Henry, 1994; Kail, 1992; Kail & Park, 1994). This is believed to be because the processing/rehearsal component of the verbal working memory system has not yet reached maturity in young children under the age of seven (Bebko & McKinnon, 1990; Flavell, Beach, & Chinsky, 1966). The use of correlational analyses in determining the relationship between memory span and articulation rate does not prohibit the possibility that a third variable is mediating the relationships as well. However, even in studies where the authors are able to imply a degree of causality where they attempted to train articulation rate and test for increases in memory span, the results show only small improvements in speech rate (Hulme & Muir, 1985). Subsequent studies aimed at matching children of different ages on articulation rate, and expecting subsequent matches in memory spans as well, have also only found partial support for the articulation rate hypothesis (Henry & Millar, 1991). Other studies attempting to provide empirical evidence for this articulation rate hypothesis have been criticised for using methods other than a single-syllable measures because of their confounding influence on children's memory (Ferguson et al., 2002). Within the visuospatial domain, the role of the inner scribe has not been found to mimic the rehearsal mechanisms present in the phonological loop, and so there is very little compelling evidence to explain why visuospatial span would increase as a function of chronological age (Smyth & Scholey, 1994).

The Pascuel-Leone model (1970) is a computational model of the development of attentional capacity that has been offered in response to this limitation. This model includes two levels of psychological constructs: schemes (adopted from Piagetian theory), and silent hardware operators. Schemes are believed to be the basic units of cognition; information-bearing, situation-specific rudimentary elements of thought, which differ in differ in content and modality. Content schemes represent cognitive states, while operator schemes allow for the transition from one state to another. Hardware operators are non-informational, content free processing resources which represent things like attention and learning, and are therefore applicable across situations. Cognitive functioning is determined by the interaction of these two levels. When an input is given (an internal or external stimuli), a number of schemes are activated. This activation represents the field of mental attention, which depends on the mechanistic activation of a number of operators. Pascuel-Leone made specific reference to the identified the M-operator and its role in the increasing capacity of the working memory system during maturation. He defined M-capacity as the maximum number of discrete chunks of information (independent schemes) that can be simultaneously activated by a single operator. The size of this M-operator is limited, and appear to increase in integer steps as children grow older. This is validated by empirical support which shows that M-capacity increases by one informational unit, every second year, from one unit at three years of age, to the adult capacity of seven at the age of 15 years (Kemps, Rammelaere, & Desmet, 2000).

While Baddeley's (2000) and Pascuel-Leone's (1970) models appear to be epistemologically independent of each other in their appraisal of the functioning of working memory, some authors have used the models in a mutually complimentary way to account for empirical data regarding the developing capacity of working memory during childhood (Ripaupierre & Bailleux, 1994; Kemps, Rammalaere, & Desmet, 2000). Pascual-Leone's model generally provides a coherent account of the development of the central capacity of the working memory system, while Baddeley's model explains the fractionation of the phonological and visuospatial components in early childhood.

Expectations of Typically Developing Working Memory in Children

In summary of the two previous sections, research evidence has converged to render a four factor model of working memory in typical adult populations; a central executive, the phonological loop, the visuospatial sketchpad, and the episodic buffer. As discussed, the

episodic buffer is a difficult construct to measure and is largely ignored in psychometric assessment, and most between-group research is satisfied with the identification of three discrete factors in typical adult samples.

This structure is, however, not evident in typical populations of children. There appears to be a two way fractionation within typical samples; younger children have a dominant reliance on short term storage in the context of their under-developed complex processing capacities, as well as an asymmetrical reliance on visuospatial codes as they are less able to employ rehearsal strategies to encode verbally. It is expected then that the working memory structure in typically developing children would be fractionated into four components; visuospatial short term memory, visuospatial working memory, verbal short term memory and verbal working memory. The delay in acquisition of complex processing strategies would also imply that the visuospatial domain would be dominant in younger children as the rehearsal strategies necessary to encode verbally are yet to fully mature. A ranked dominance between the four factors is also therefore expected to be as follows; visuospatial short term memory, followed by visuospatial working memory, verbal short term memory and then verbal working memory. The final section of this chapter offers a theoretical and methodological critique to the evidence presented in support of this structural expectation within typical samples.

Problems with Psychometric Measurement

The psychometric measurement of various aspects of children's development has long been the work of both theorists and clinicians. The standardised measurement of cognitive functioning has its roots in the work of Binet in the early 1900's. While current thinking on developmental assessment has since evolved into a widely accepted and thoroughly researched industry, it remains the focus of contentious debate from both within and outside the developmental fraternity. The third world context of this study offers even further complexity to this debate by including issues of multilingualism, ethnocentric bias and socioeconomic influence (Foxcroft et al., 2001).

The purpose of developmental assessment tools is to identify and monitor areas of delay or atypical functioning in children. This supports the creation of appropriate and timeous intervention strategies, and allows clinicians to monitor progress over a period of time (Whitehead, 2012). Various standardised psychometric measures have been designed for this purpose, which attempt to provide clinicians and researchers a standardised means

by which to make clinical judgements regarding performance that is relatively free of the subjective bias of individual case study. Coupled with these large scale benefits, psychometric assessment is also plagued by limitations in the applicability of norm-referenced scores to samples dissimilar to those used in the original test construction. Three of these limitations are highlighted as conceptual precautions applicable to this study and its method.

Firstly, the psychometric assessment of development makes particular assumptions about the universal and sequential course of human development, and leaves little room for individual variation. The application of age-related scores collude with these often incorrect suppositions, and threatens the introduction of Type 1 error, where children are incorrectly labelled as atypical, and the equifinality of developmental outcomes is disallowed. Secondly, psychometric measures of development have a strong Western leaning, and issues of linguistic bias further serve to undermine their utility within non-Western settings (Meiring, van de Vijver, Rothmann, & Barrick, 2005; van de Vijver, & Tanzer, 2004). Lastly, all psychometric assessment runs the risk of reifying the construct under inspection through attempts at its measurement (Gould, 1981; Rose, 1988). While this is particularly apparent in the psychometric measurement of intelligence, the assessment of working memory is less vulnerable to this threat as it measures novel abilities that are far more difficult to learn from a previous context. The threat remains nonetheless. The following discussion presents a critique of the psychometric measurement with specific reference to the measurement of working memory

As if the current contention around the definition of the construct and its fractionation across the lifespan was not enough, the measurement of working memory is also plagued by methodological difficulties. Both experimental and applied attempts to examine Baddeley's (2000) model have assumed reified understandings of its multiple components. These simplified assumptions are generally as follows: that the measurement of the phonological loop is done through the assessment of the temporary storage and maintenance of phonological information, and that the measurement of the visuospatial sketchpad is done through the assessment of non-verbal information. The domain-general central executive is correctly assumed to be an attentional controller which allocates resources to the two slave systems, coordinates cognitive processes and focuses, switches and divides attention, and the episodic buffer assumed to provide a passive link between

these codes and semantic links to long term stores. Assessment of the central executive therefore would collectively measure the *working* component of working memory, while the measurement of the episodic buffer would then need to assess the temporary storage of bound information (both visual and phonological) and provide a link between this information and long term memory.

There are some difficulties with this process. Firstly, it incorrectly assumes the existence of a domain general central executive in samples of children. While only one study has found domain specific fractionation of the central executive in an adult sample, dissociable verbal and visuospatial complex working memory factors have been identified more commonly in younger samples (Gathercole & Jarvis, 2003; Gathercole, Pickering, Ambridge, & Wearing, 2004). Executive processes of the central executive are usually measured in a general, domain general manner which disallow the confounding effects of the influence of domain specific representations. This is particularly important in atypical samples where these may be deficits in domain specific encoding (SLI, dyslexia) and that the associations between these deficits and executive control could be misinterpreted.

Secondly, verbal phonological stimuli are sometimes presented visually instead of in an auditory manner. There are two implications with this mono-dimensional method assessment. Firstly, within typical populations, visual stimuli are usually converted to a sub-vocal phonological process anyway, which could mean that the results from this method incorrectly infer functions of the phonological loop which are actually confounded by latent functioning of the episodic buffer (Alderson, Kasper, Patros, et al, 2014; Kasper, et al, 2012). Secondly, the switch from visual to verbal encoding via sub-vocal rehearsal is not always evident in atypical populations, which means that results could be misrepresenting confounding influences.

As intimated in the previous point, research regarding working memory performance generally ignores the presence of the episodic buffer. This is particularly evident in studies of children, and is largely because it difficult to quantify and then measure. This is perhaps a reflection of the position of current episodic buffer research which presents it in vague, indistinct terms which are difficult to operationalise experimentally. One area of difficulty in research regarding the episodic buffer is its close association with language. Rudner and Ro (2008) provide clear evidence for significant interaction of the episodic buffer with the

central executive as well as the phonological loop, which has a notable influence on language processing. Its functioning is therefore difficult to isolate because of the pervasive dominance of language in most experimental outputs. Nobre et al. (2013) present a meta-analytic review of the tasks used to assess the episodic buffer over the last thirteen years. Some tasks were found not to meet experimental criteria that were needed to evaluate the episodic buffer, while some of those using standardised tests could not provide sufficient theoretical argument or empirical support that they were indeed recruiting the episodic buffer and not another component. This creates large inconsistencies in the knowledge of what the episodic buffer does, and how it can be measured with integrity in subsequent studies.

Working memory research usually takes two approaches: Within- and between-group differences approach, and the less popular latent variable approach. The most common method examines within- and between-group differences on separate phonological and visuospatial tasks that vary with the amount of mental processing and manipulation required. Further, forward span tasks are reified as measures of short term storage or rehearsal processes, while backward span tasks are incorrectly considered pure measures of the functioning of the central executive. This methodology however, incorrectly assumes that forward span tasks do not rely on central executive input, or that backward span tasks do not require the assistance of temporary storage and rehearsal in either of the salve systems. For instance, in complex sentence and counting span tasks, storage and processing are performed concurrently. Within both typical and atypical populations it is possible that individual differences determine that some may have more difficulty with sentence span task because of the requirement to generate a word. Alternatively, impairment in the counting span task may be slower because of the need to visually scan material. Similarly, increasing the processing load could result in a diminished maintenance capacity. Thus it is possible that deficits in span do not actually reflect true working memory impairment. In response to this, researchers have recently started using a latent variable approach which covaries shared variability between phonological and visuospatial tasks, and consequently attempts to statistically estimate the unique contributions of each component to working memory functioning (Alderson, Kasper, Hudec, & Patros, 2013).

There has also been a lot of criticism of the methods supporting the theoretical explanation for developmental increases in phonological stores as children mature. The role

of long-term memory and existing knowledge in the development of phonological short term storage has also been largely ignored within the context of working memory research in children. Generally, while articulation rates may be implicated in explaining the development of phonological short term storage in typical children, they are certainly not the only relevant factor (Henry, 1994; Henry, 2012; Turner, Henry, & Smith, 2000).

Conclusion

This chapter has provided a comprehensive discussion of working memory, and its relationship to executive functioning, intelligence, socioeconomic status and language. The relationship between working memory and language was given particular attention in light of its relationship to multilingualism in culturally diverse contexts such as South Africa. The discussion of working memory has also located the theoretical development of the construct within a critical appraisal of competing and alternative models, as well as providing a clear account of how the fractionation and maturity of the construct is expected to develop differently in typical children. The following chapter considers working memory performance in atypical child populations, and provides an extensive review of its functioning in the context of HIV infection and exposure.

Chapter 3: Working Memory and the Human Immunodeficiency Virus

Introduction

The previous chapter dealt with the historical development of working memory, its theoretical definition and its development across childhood within typical populations. This chapter locates this theoretical debate within atypical development – particularly that of HIV infection, and exposure. It begins by profiling the working memory functioning characteristic of a range of neurodevelopmental disorders in an attempt to highlight common deficits and how these might be understood to relate to other disorders with different etiologies. It then presents a review of empirical evidence regarding the associated neurocognitive effects of HIV infection, and HIV exposure on both adult and paediatric samples, and closes with a comment on the confounding environmental effects of HIV-associated impairment.

Working Memory Profiles in Atypical Development

There is a wide body of research evidence profiling the working memory difficulties characteristic of particular types of neurodevelopmental disorders. Those of specific language impairment, dyslexia, dyscalculia, AD/HD, ASD and DCD will be discussed here. The relevance of these profiles to that of HIV infection and exposure exists within a tension between etiology and symptom presentation. HIV infection not only manifests with similar neuropsychological symptoms as those of common neurodevelopmental disorders, but there are also moderate levels of comorbidity between HIV-I (HIV-infected) status and other neurodevelopmental disorders (e.g. ADHD, 20%; (Kumar, Shekar, Pandiyan, Das, Nahar, Raveendra, & Hongally, 2014); SLI, 18.4%; (Rice, Buchanan, Siberry et al., 2013)). The primary distinction between the two is that HIV associated deficits are largely the consequence of viral infection, while neurodevelopmental disorders have a strong predisposition from genetics.

The identification of etiological factors within neurodevelopmental disorders is widely studied, and clinical meta-analyses usually identify a multifactorial explanation foregrounding a genetic predisposition which manifests in particular environmental stimulations. It has long been the desire of researchers to reduce multi-factorial explanations regarding the etiology of neurodevelopmental disorders to a parsimonious, single-factor theory that can be the sole focus of intervention. This is particularly apparent in SLI and ASD which are believed to primarily neurogenetic in origin (Bishop, 2002, 2006;

Chaste & Leboyer, 2012; Folstein & Rosen-Sheidley, 2001; Freitag, 2007; Le Couter, Bailey, Goode, Pickles, Robertson, Gottesman et al., 1996). While the identification of genetic components can be helpful, an understanding of isolated gene mutations is unlikely to make progress in the treatment of such disorders because of their complex etiologies and symptom presentations. Bishop (2006) suggests that neurodevelopmental disorders instead be considered in terms of common underlying discrete deficits ('endophenotypes'), rather than reified clinical diagnostic labels. In this way, our understanding of the ways in which discrete deficits respond to treatment can be applied broadly across neurodevelopmental profiles. It also makes it possible to identify where compensation of one discrete difficulty using alternative skills is possible, or where they produce an additive risk when existent in combination.

Hence, while the neuropsychological effects associated with HIV-infection are often not a consequence of a typical neurodevelopmental disorder (predominantly genetic in origin), an understanding of how the HIV working memory profile might align with that of other atypically developing populations remains useful. Firstly, it presents a method by which the profiling of strengths and weaknesses was done in other research. Many of the profiling studies, however, use between-group comparison as a means of significance testing, while the current study uses both between- and within-group differences to establish a characteristic working memory profile. Secondly, any similarities in the profiling of working memory performance within the two HIV-affected groups could add to the theoretical idea that diagnoses with complex etiologies be considered (and treated more effectively) by common discrete deficits instead of common genotypic understandings. The discussion begins with that of specific language impairment (SLI). This is a common neurodevelopmental disorder believed to have a strong genetic influence. However, its presentation as such is a challenge to clinicians as it is often defined as the catch-all diagnosis for language impairment in the absence of any other available cause. It is on this basis that the common discrete deficits in these disorders and that of HIV-infection and –exposure be considered.

Specific Language Impairment.

Specific language impairment (SLI) is a developmental disorder characterised by marked delays or dysfunction in the development of language, where language skills are not in line with other typically developing aspects of intellectual development. Language

development in this sample is typically late with a slow rate of development and weaknesses are generally in the areas of phonology, vocabulary and grammar (Bishop, 2006, Henry, 2012).

The findings of a series of robust studies have converged to indicate that children with SLI have marked difficulties with phonological short term memory, when using both nonword repetition or nonword span tasks (Dolloghan & Campbell, 1998; Edwards & Lahey, 1998; Gathercole & Baddeley, 1990; Laws & Bishop, 2003; Marton & Schwartz, 2003, Pickering & Gathercole, 2004), and word and digit span subtests (Henry, Messer, & Nash, 2012; Hick, Botting, & Conti-Ramsden, 2005). There is equivocal evidence for the degree of impairment in the visuospatial domains in children with SLI, where impairments are believed to be much smaller and more variable than the stark deficits apparent in the phonological loop (Archibald & Gathercole, 2006; Henry et al. (in press) provide evidence against deficits, while Hoffman & Gillam, 2004; and Hick et al., 2005 highlight deficits). There is strong emerging evidence that both the verbal (Archibald & Gathercole, 2006; Ellis Weismer et al., 1999; Henry, Masser, & Nash, 2012; Marton & Schwartz, 2003), and visuospatial domains (Henry et al., 2011; Im-Bolter et al., 2006; Marton, 2008) of the central executive are impaired in children with SLI when compared to their typically developing peers. Within the domain of broader executive function, there is also support for difficulties in inhibition, planning and fluency (Bishop & Norbury, 2005a, 2005b), but not in switching (Henry et al., 2011; Im-Bolter et al., 2006). There does not appear to be any work that explicitly examines the episodic buffer in children with SLI. However, Alt (2010) considered how fast these children could learn new words with fewer than three exposures. She found no evidence that the ability of children with SLI to use stored long term knowledge about which sounds are most commonly found together in the English language was impaired (Henry, 2012).

One of the limitations in the research regarding working memory and SLI is that many studies have inadvertently included children with dyslexia in their studies because of imprecision in the definition and distinction of the disorders. The overlap between the two are believed to be between 15-50% (Catts et al., 2005; McArthur et al., 2000), and have caused some researchers to argue that the disorders are caused by the same underlying problems (Kamhi & Catts, 1986; Tallal, 2003; Tallal, Allard, Miller, & Curtiss, 1997). Some researchers believe that SLI is an extended disorder of dyslexia, where children with dyslexia

experience only a single deficit in phonological skills and have consequent phonological short term memory difficulties, while children with SLI have weak phonological processing with additional difficulties with grammar, syntax and semantic skills (Bishop & Snowling, 2004). Others feel that the two are separate disorders with particular underlying cognitive deficits (Catts et al., 2005; Ramus, 2004; Ramus, Marshall, Rosen, & van der Lely, 2013, van der Lely, 2005). This they believe is evidenced by the fact that when phonological short term memory was assessed in carefully screened children with SLI and Dyslexia, the deficits within the SLI sample were relatively mild and performance significantly better than those of dyslexia (Catts, et al., 2005; Henry, 2012). This distinction represents a clear argument regarding the need for working memory profiling in children that have neurodevelopmental disorders, as it is likely that children with either dyslexia or SLI would have different working memory profiles, and would benefit from specific, concentrated intervention and remediation. The working memory profile of dyslexia is hence considered below.

Dyslexia.

Dyslexia is a neurodevelopmental disorder characterised by difficulties in the ability to decode or read words, and presents with poor reading accuracy and processing. There is an expectation that dyslexia is an isolated barrier to learning which presents only as a weakness in phonological processing, and development in other areas is usually typical (usually characterised by non-verbal IQ scores of greater than 80). This isolated diagnosis is uncommon as sufferers are often plagued by difficulties in reading comprehension as well (Henry, 2012).

Research evidence suggests that children with dyslexia have weak phonological short term memory for serially ordered verbal materials, regardless of whether this material is presented in a visual or auditory manner (Henry, 2012). These difficulties within phonological short term memory lie in both components of the phonological loop; the phonological store and the articulatory rehearsal mechanism. This is evidenced by phonological encoding errors (Johnston, 1982), and deficits non-word repetition (Roodenrys & Stokes, 2001), digits (Aaron, 2012) and verbal rehearsal (Macaruso et al., 1996; Spring & Capps, 1974). Conversely, children with dyslexia do not appear to have difficulties in visuospatial short term memory, which suggest that the visuospatial sketchpad is operationally intact. This is evidenced when the VSSP is assessed using serial picture span tasks, nonsense shapes and unfamiliar letters, (Liberman, Mann, Shankweiler, & Werfelman,

1982; Katz, Shankweiler, & Liberman, 1981; Swanson, 1978; Vellutino et al., 1973; Vellutino et al., 1975) as well as in spatial short term memory (Gould & Glenscross, 1990). With regards to the central executive, reading disabled children do appear to have difficulties in the concurrent processing and storage demands of working memory, which are apparent in both the verbal and visuospatial domains (Smith-Spark and Frisk, 2007; Swanson, 2003; Swanson, 2006). These difficulties are not better accounted for by the foregrounded difficulties in phonological short term memory, and are believed to account for a domain general executive dysfunction. There is little evidence to support the existence of difficulties in broader executive skills such as inhibition, planning and switching (Everatt, Weeks, & Brooks, 2008; Rieter et al., 2004 ; van der Sluis, de Jong, & van der Leij, 2004), but there are studies which report impairment, but have methodological limitations (Booth, Boyle, & Kelly, 2010; Swanson, 1993). While the research regarding the episodic buffer with children with dyslexia has been limited, the results of one study imply that there is no identifiable weaknesses in the binding of information from long term memory with verbal material stored in the phonological loop within this sample (Roodenrys and Stokes (2001). Attention is now turned to another isolated barrier to learning common in children when they begin formal schooling - dyscalculia.

Dyscalculia.

Dyscalculia is also referred to as an isolated mathematical learning disorder (MLD), and the two are used interchangeably here, despite some pieces of literature differentiating the two on the grounds that dyscalculia is a result of isolated brain injury, and MLD a neurodevelopmental disorder. Both dyslexia and dyscalculia are believed to have a similar neuro-genetic origin, have similar prevalence rates (4-7%) (Landerl, Fussenegger, Moll & Willburger, 2009), and comorbidity estimates from between 17-70% (Badian, 1983; Barbaresi et al., 2005; Dirks et al., 2008; Gross-Tsur et al., 1996; Landerl & Moll, 2010; Lewis et al., 1994; von Aster & Shalev, 2007). There are a number of shared cognitive factors, such as common phonological verbal deficits, to both disorders which some believe constitute a common underlying disorder which simply manifests differently (Geary, 1993; Robinson, Menchetti, & Torgesen, 2002; Vellutino, Fletcher, Snowling, & Scanlon, 2004; von Aster, 2000). Others profile the disorders based on domain specific cognitive deficits which and assume that the two have separable cognitive profiles (Landerl, Fussenegger, Moll & Willburger, 2009). The working memory profiles of dyscalculia support this second notion.

Children with dyscalculia are defined as those with a normal general intelligence (measure as >85 IQ score), and who have scores below the 25th percentile on a standardised maths test or standard mathematical achievement measure (American Psychiatric Association, 2000). This usually arises from deficits in the ability to represent or process mathematical information in its typical presentations (arithmetic, geometry and algebra). Comorbidity with ADHD are estimated at 26%, and with reading difficulties at 17%. The latter is believed to be under-inflated as some studies find as many as half of children with dyscalculia have comorbid reading and spelling difficulties (Badian, 1983).

Research findings suggest that children with dyscalculia appear to have typically functioning phonological short term memory ((Bull & Johnston, 1997, Mclean & Hitch, 1999, Passolumghi & Siegel, 2001, 2004; Passolumghi et al, 1999, Passolumghi, Marzocchi & Fiorillo, 2005), but that there are deficits in spatial working memory (Mclean and Hitch (1999; Passolunghi, 2006). Primary deficits within this population appear to be in the storage and manipulation demands governed by the central executive (Passolunghi & Siegel, 2001, 2004; Swanson & Sachse-Lee, 2001), particularly in tasks that require controlled processing of verbal or numerical information (listening or counting span tasks) (Hitch & McAuley, 1991; Siegel & Ryan, 1989). Within general executive functions, children in this population show difficulties in the process of inhibition, particularly an inability to control or ignore irrelevant information. The functioning of updating processes also appear to be atypical (Henry, 2012; Mclean & Hitch, 1999; Passolumghi & Pazzaglia, 2004). There have been no studies regarding the functioning of the episodic buffer within this sample to date, but the application of findings from other studies do suggest a degree of impairment (Passolunghi & Siegel, 2004).

Attention Deficit Hyperactivity Disorder.

Attention Deficit/Hyperactivity Disorder (AD/HD) is another common neurodevelopmental disorder characterised by deficits in executive functions which are believed to cause difficulties in maintaining attention, persistent hyperactivity and age-inappropriate impulsivity (Diagnostic and Statistical Manual IV, Text Revision, 2000). The difficulty with high-level cognitive processes responsible for goal directed behaviour is believed to be responsible for these children's difficulties with schooling and academic achievement (Henry, 2012).

Previous research regarding the working memory profiles of children with ADHD provides clear evidence that the efficient operation of the phonological loop appears to be intact (Goodyer & Sahakian, 2000; Lawrence, Houghton, Tannock, Douglas, & Whiting, 2002; Lazar & Frank, 1998; Williams, Stott, Felton, & Wood, 1989). There is limited evidence to suggest that the simple visual skills are similar to that of typical populations (Barnett et al., 2001; Kempton et al., 1999; McInnes et al., 2003; Tripp, Ryan, & Pearce, 2002), as these are limited by methodological issues regarding sample comorbidity. Equally, complex span deficits have repeatedly failed to achieve validity or significance after accounting for matching and IQ (Cornoldi et al., 2001; Kunsto, Oosterlaan, & Stevenson, 2001; Lazar & Frank, 1998; Siegel & Ryan, 1989; Siklos & Kerns, 2004; Rucklidge & Tannock, 2002; Willcutt et al., 2001). The evidence from these studies would suggest that the complex span difficulties within ADHD samples are not domain-specific in either the verbal or visuospatial representations. There is strong support for the impairment of broad executive functions (inhibition, planning, sustained attention) within the ADHD population. This is particularly evident in terms of working memory where the domain-general processing and storage domains are compromised under increased demand (Holmes, Gathercole, & Place et al., 2010; Sonuga-Barke, Dalen, Daley, & Remington, 2002). These processes do, however, appear to be fractionated and linear, as there are differential findings in different age groups (Henry, 2012). The functioning of the episodic buffer also appears to be impaired, although this appears to have only been assessed in one study (Alderson, Kasper, Patros et al, 2014).

This discussion now focuses on two less common childhood disorders, that of Autistic Spectrum Disorder (ASD) and Developmental Coordination Disorder (DCD). They are also believed to have a neurobiological origin and have relatively high rates of comorbidity with each other (Gillberg & Billstedt, 2000).

Autistic Spectrum Disorder.

Autistic spectrum disorders (ASD) refer to a range of heterogeneous, lifelong developmental disorders which are characterised by difficulties in three areas: reciprocal social interaction, communication and restricted or repetitive behaviours and interests. However, within this sample there is great variability in the degree and manifestation of impairment. It also has moderate co morbidities with other neurodevelopmental disorders;

ADHD (14-78% (Jang et al., 2013), tic disorders (22% (Canitano & Vivanti, 2007) and intellectual impairment (70%) (Tureck, Matson, Cervantes & Konst, 2014).

Research regarding the working memory profiles of children with this disorder is limited by the difficulty in definition of the degree of impairment as well as the heterogeneity with which it manifests. Despite the apparent lack of verbal expression and delay in language acquisition in some children with ASD, empirical evidence does not provide clear conclusions regarding the absence or dysfunction of inner speech in phonological short term memory tasks (Bennetto et al., 1996; Mottron, Morasse, & Belleville, 2001. See also Ameli, Courchesne, Lincoln, Kaufmann, & Grillon, 1988; Joseph, Steele, Meyer, & Tager-Flusberg, 2005; Williams, Goldstein, & Minshew, 2006; Whitehouse, Mayberry, & Durkin, 2006; Williams, Happé, & Jarrold, 2008). Conversely, studies concerning the visuospatial aspects of working memory are limited but do suggest that both visual and spatial components of the construct might be impaired (Minshew, Luna, & Sweeney, 1999; Joseph, McGrath, & Taher-Flusberg, 2005; Williams et al., 2005). Research evidence provides contradictory findings regarding the functioning of the central executive in this population. While some researchers suggest verbal processing deficits drive central executive impairment (Benetto et al., 1996; Joseph, Steele, et al., 2005), while others fail to confirm these findings (Ozonoff & Strayer, 2001), or have identified spatial deficits instead (Williams et al.,2005). There exists sound agreement that the broader executive functions of planning, switching and fluency are impaired (Geurts et al., 2004; Hughes, Russel, & Robbins, 1994; Ozonoff, Pennington, & Rogers, 1991; Rumsey & Hamburger, 1988, Turner, 1999), while that of inhibition has equivocal empirical support (Ozonoff & Jensen, 1999). While there has been little research into the functioning of the episodic buffer within this population, some studies do report that the recall of episodes is difficult for this population and imply that coherent binding of current information to long term stores could be impaired (Bruck, London, Landa, & Goodman, 2007; Williams et al., 2006). However, because of the close associations between the episodic buffer, language processing and phonological storage (Rudner & Ro, 2008), an accurate assessment of the functioning of the episodic buffer in an already language-compromised sample is challenging. This final section focuses on the working memory profiles of children diagnosed with Developmental Coordination Disorder (DCD).

Developmental Coordination Disorder.

Developmental Coordination Disorder (DCD) is recognised as a motor dyspraxia that affects movement as we all perception. It is characterised by general clumsiness and poor coordination of the body, poor posture, confusion of which hand to use, difficulties with reading, writing and holding a pen correctly. This has obvious implications for academic achievement and classroom learning (Alloway, 2006b; Archibald & Alloway, 2008).

All four components of the working memory profiles of children with DCD appear to be depressed when compared against typical samples, with particular impairment in the visuospatial domains (both simple storage and complex manipulation) (Alloway, 2006b). This suggests that the deficits are apparent in the memory and manipulation aspects of the visuospatial tasks rather than the motor component. The relative lack of impairment on the verbal tasks in relation to the visuospatial domain also suggests that functioning of the phonological loop (and its ability to store verbal information) remains intact. This is supported by the lack of interference effects in a similar spatial task assessing both spatial and verbal forms (Baddeley & Lieberman, 1980, as cited by Alloway, 2006b). These selective visuospatial deficits were confirmed in a larger, similar study with children with DCD (Archibald & Alloway, 2008). Other studies confirm that executive functioning also suffers deficits (Michel, Roethliberger, Neuenschwander, & Roebers, 2011; Roebers & Kauer, 2009). The measurement of depressed scores in the Alloway (2006b) and Archibald and Alloway (2008) studies employs a within-group design instead of a between-group comparison. This has limited validity when comparing across developmental disorders as was evidenced in many of the previous studies cited in other neurodevelopmental disorders as well as the current study.

Bishop (2006) suggested that neurodevelopmental disorders be understood by their discrete cognitive deficits and not by their etiological framework. Within this context, verbal short term memory deficits (and atypical functioning of the phonological loop) were evident in children with Dyslexia, DCD and SLI. Visuospatial sketchpad impairment was only identified in children with MLD, ASD and SLI, with only the spatial component believed to be impaired in ADHD and a marked difficulty apparent in DCD. Notably, all of the disorders have both verbal and visuospatial working memory deficits which emphasise both their importance in everyday functioning and academic success, but their vulnerability to compromise in atypical circumstances.

It is also important that when considering these studies through a methodological lens, none of the available research profiling working memory performance in atypical samples have considered the nature of the structure of the construct through a factor analytic paradigm. Instead, between-group differences are identified when compared against controls. Assessment is therefore usually focused on one aspect of working memory, and more general understandings of the construct's functioning as a whole is ignored.

The neurodevelopmental profiles characteristic of HIV infection and exposure are likely to be somewhat different to commonly understood neurodevelopmental disorders because symptomatology is primarily believed to be as a result of viral infection or exposure, and not due to the influence of gene-environment interactions. However, the profiling of working memory within all of these atypical populations remains useful for treatment protocols where there are common discrete deficits, particularly within the very large populations of HIV-I and HIV-EU children who often require practical rehabilitative intervention in order to cope in formal schooling. The profiling of working memory in atypically developing HIV subgroups is particularly valuable because this area of cognition is largely under-researched, and would add much to the theoretical understandings of the neurocognitive manifestations of the virus in various conditions (infection and exposure).

The relevance of the relationship between chronic disease and working memory to paediatric HIV is found primarily in this exact definition and distinction between viral infection as a neurodevelopmental disorder, or as a chronic disease. The former is concerned with manner in which the virus alters the typical development of the CNS, and the effect that these changes have on optimal cognitive performance. The latter, chronic disease, is concerned with the way in which a series of symptoms impacts on health in a manner which negatively impacts cognitive functioning. Typically, chronic disease is not associated with paediatric populations, as public health systems around the world tend to focus on adult populations suffering from the most common conditions: ischaemic heart disease, strokes, cancer, depression, Type 2 diabetes, arthritis, osteoporosis, chronic obstructive pulmonary disease (COPD) and chronic kidney disease. Chronic disease is defined as a prolonged course of illness that causes functional impairment or disability. It is etiologically complex with multiple risk factors, and usually has long latency periods (AIHW National Health Survey, 2005). While vertically transmitted paediatric HIV would not technically satisfy this definition, it is considered to be the most prevalent paediatric chronic

disease in the world because of its unique need for comprehensive, multidisciplinary and coordinated care which it shares with other paediatric chronic illnesses (Meyers & Weitzman, 1991; WHO, 2011).

The relationship between chronic disease and working memory deficits is well established, with deficits apparent even in conditions which do not directly affect the central nervous system. These include diabetes (Deary, Sommerfield, McAulay, Frier, 2003; Ryna, Freed, Rood, Cobitz, Waterhouse, & Strachan, 2006; Sommerfield, Deary, McAuley, & Frier, 2003), heart disease (Hayley, Sweet, Gunstad, Forman, Poppas, Paul, Tate & Cohen, 2007; Stetkiewicz-Lewandowicz & Borkowska, 2011*)*, kidney disease (Harrell, 2010), cancer (Hardy, Willard, Allen, & Bonner, 2012), and chronic pain (arthritis) (Dick & Rashiq, 2007). The mechanisms by which this happens vary greatly across conditions, and include the side effects to treatments (chemotherapy and radiation), neurochemical changes as a result of illness, or larger systemic interactions in the body where the specific relationship to neurocognitive impairment is unknown. While the relationship between HIV infection and exposure, and neurocognitive functioning within this study is considered through the lens of neurodevelopment, it is important to recognise that its effects are no doubt compounded by the experience of HIV as a chronic paediatric disease. This next section discusses the extensive literature base regarding the neuro-pathogenic effects of HIV infection. It begins with a brief contextualisation of the disease within sub-Saharan Africa.

Contextualisation of HIV

In the early 2000's, South Africa was faced with fastest growing and most severe HIV epidemic in the history of the world (van der Walt, Bowman, Frank, & Langa, 2007). There are multiple reasons behind the tardy response of both the formal and informal sectors to the lurking epidemic. To begin with, despite the international HIV health movement in the 1980's, the Apartheid government was heavy set on maintaining the status quo of their regime and paid little attention to the growing number of infections (Kauffman, 2004). Apartheid resistance movements were consumed with large scale political reform internally, which created an attention vacuum that rendered the growing pandemic invisible. Further, a series of structural and socio-political issues converged to create the 'perfect storm' for the making of a pandemic. These contextual enablers included the large-scale infrastructural degradation inflicted by Apartheid social engineering (migrant labour, same-sex hostels, systematic resource deprivation and the stripping of the traditional family structure), long-

standing poverty in post-Apartheid South Africa, issues of gender inequality, structural and ideological barriers to treatment, stigma towards the disease, and AIDS denialism. What ensued was a health problem of epic proportions. While recent governments have been successful in reducing the number of new vertical transmissions and HIV-associated deaths, the lived experience of HIV remains a salient issue in the national agenda (van der Walt, Bowman, Frank, & Langa, 2007). An epidemiological profile of HIV in South Africa to provides some context to this issue.

Epidemiology of the Human Immunodeficiency Virus.

An examination of the epidemiology of HIV over the last decade is useful for two reasons. Firstly, it assists in promoting HIV-EU children as an important and growing sub-section of the group of vulnerable populations in South Africa. Secondly it assists in identifying the socio-demographic associations of HIV infection in order to contextualise research findings.

The most recent release of national statistics identified the prevalence of HIV infection to be at 10.2% of the total population. The number of people living with HIV in South Africa increased from 4.09 million in 2002, to 5.51 million in 2014 (Statistics South Africa, 2014). These approximations are confirmed by estimations by UNAIDS (6.1 million, 12.2% prevalence rate) and the HSRC 2012 report (also 12.2%) (Shisana, et al., 2014) which identified a statistically significant increase in prevalence from 10.6% in 2008 to 12.2% in 2014. Approximately one-fifth of women of reproductive age are HIV positive. Prevalence estimates for vertical transmissions for children under 12 months are 1.3%, while for children under 5 years it is 1.7%. These estimates show no sex differences, but the declining rates of prevalence from 2008 to present suggest that PMTCT is becoming increasingly effective (Shisana, et al., 2014; UNAIDS, 2014).

An initial examination of prevalence estimates in the adult population would suggest that HIV intervention is largely ineffective as there seem to be more people living with the disease than ever before. These statistics are, however, unable to account for shifts due to people moving into different age brackets because they are still alive, largely because of effective, large scale roll-out of ART (Bor, Herbst, Newell, & Barnighausen, 2013). Consequently, the epidemiological curve has shifted with the peak prevalence for females moving from 25-29 year group (2008) to the 30-34 year age group (2012), and from 30-34

year age group (2008) to the 35-39 year age group (2012) for males, as dominantly infected age groups are receiving effective treatment, and therefore living longer.

Incidence statistics are more helpful in understanding the rate of national infection. Statistics SA (2014) reveal that HIV incidence has declined from 1.64 in 2002 to 1.11 in 2014. Other reports regarding declining infection rates are particularly encouraging. The UNAIDS report multiple times that infection rates in South Africa are declining, with estimations at 25% between 2001 and 2011 (UNAIDS, 2012) and 31% between 2004 and 2012 (UNAIDS, 2014). The HSRC study has similar results across demographic categories. There has also been significant progress in the reduction of vertical transmission from mothers to their children. In the UNAIDS (2012) report, South African estimates of declined rates of vertical infection were between 40-59%, and declined further in the 2013 estimates to 63%. Clinicians currently predict that only 5% of children born to HIV positive mothers will become infected with the disease themselves (Coovadia, 2012). This is statistically confirmed by the HSRC (2014) study which was particularly interested in the identification of sero-discordance between mother and child pairs. Within the 0-2 age group, results reveal that only 4.3% of mother-child pairs were both HIV positive, while a staggering 95.7% of dyads were discordant, with the mother being positive and the child negative. To that end, the HIV-EU child is increasingly the stereotypical product of effective HIV prevention, and represents a growing population which would benefit from clinical research and care.

Epidemiological studies of HIV affected populations provide useful information regarding the contextual location of the disease and its far-reaching effects. Table 3.1 highlights the 2012 HIV prevalence concentrations by gender, race, age and locality.

The survey confirms widely held beliefs regarding HIV prevalence, and identifies that Black Africans, particularly females, continue to be disproportionately affected by HIV, followed by people of mixed race. High prevalence of infection in the Black African group was associated with low levels of marriage, low socioeconomic status, and a number of unrelated social and behavioural factors (multiple partners, cohabitation, a lack of awareness, denial and stigma). HIV prevalence also appears to be higher in informal urban areas and low socioeconomic status (which are commonly associated in the wake of Apartheid). Orphanhood has also remained stable over the last decade between 16.8% (2002) and 16.9% (2012) of all HIV positive children 0-18 years. These statistics assist in

understanding HIV infection as a contextually nuanced experience of a disease that has multifactorial associations (Human Science Research Council, 2012).

Table 3.1
HIV prevalence among adults in the 15-49 age group by race, province and locality type, South Africa (2012)

		n	%
Sex			
	Male	6468	14.5
	Female	8252	23.2
Race			
	Black African	9363	22.7
	While	881	0.6
	Coloured	3013	4.6
	Indian/Asian	1418	1
Locality type			
	Urban formal	7882	14.7
	Urban informal	1518	29.9
	Rural formal	3408	22.6
	Rural Informal	1615	16.1
Province			
	Western Cape	1890	7.8
	Eastern Cape	1963	19.9
	Northern Cape	1207	11.9
	Free State	1071	20.4
	KwaZulu Natal	3536	27.9
	Nnorth West	994	20.3
	Gauteng	1673	17.8
	Mpumalanga	1125	21.8
	Limpopo	1261	13.9
	Total	**14720**	**18.8**

Empirical evidence regarding HIV.

The primary aim of this study was to investigate the working memory profiles of children who were exposed to, but not infected by the HI virus. In order to hypothesise why their working memory development and consequent functioning would be atypical, it is important to gain an understanding of what typical working memory performance within school beginners looks like (described in Chapter 2), and what previous research has found this effect to be in children who are infected by HIV. Critical review of the latter by way of previous studies makes this difficult for two reasons. Firstly, few assessments of paediatric neurocognitive functioning in the context of HIV infection have been conducted in resource-deprived, non-Western and linguistically diverse contexts such as the one this study is

located in, in South Africa. Secondly, none have considered working memory as their primary construct of assessment within these contexts. In response, this review considers the evidence from a broad range of empirical research investigating the effects of HI viral infection on working memory, but also on related neurocognitive constructs which might rely on similar skills (attention, inhibition, fluency, processing). It also considers research investigating the effect of HIV on language, and (briefly) motor skills. These sections have relevance to the present study in that they highlight the complexity of research within a linguistically diverse South African context, while the section on motor impairment serves to foreground other neurocognitive skills assessed using the NEPSY-II (one of the measures used in the current study).

The HI virus is a retrovirus, meaning that it contains a particular enzyme that allows the virus' genetic information to become part of the host cell's genetic material upon replication (Faulhaber & Aberg, 2009). It selectively attacks the CD4+ T lymphocytes which are responsible for orchestrating and coordinating the body's immune response to infection. HIV carries its genetic information in ribonucleic acid (RNA). With the assistance of its reverse transcriptase enzyme, it makes a mirror-image copy of itself which results in a double stranded DNA that carries instructions for replication. This is then inserted into the DNA of the original cell, which will eventually kill the CD4+ cell itself and release HIV copies into the bloodstream. The virus then makes another RNA encrypted version of itself and leaves the host cell to seek infection of other CD4+ cells (Trecarichi, Tumbarello, de Gaetano Donati et al., 2006).

HIV is also a neutrotrophic virus, and in the late stages of immune-compromise, leaves the nervous system vulnerable to an array of neurologic disorders (Faulhaber & Aberg, 2009). In 2007, the National Institute of Mental Health developed a classification with standardised diagnostic criteria for the neuro-cognitive effects of the virus on the nervous system. HIV Associated Neurocognitive Disorders (HAND) incorporates different degrees of impairment and was determined to include the features of cognitive impairment with motor dysfunction or behavioural/psychosocial symptoms that results from HIV infection itself. HIV Associated Dementia (HAD) is included within the HAND criteria and includes the impairment of attention and concentration, the slowing of mental speed and agility, the slowing of motor speed and apathetic behaviour (Antinori, Arendt, & Becker, 2007). Prior to the onset of overt AIDS, subsequent research has also identified particular declines in

executive functioning and higher order processing in HIV infected people (Bassel, Rourke, & Halman, 2002; Dawes, Suarez, Casey, Cherner, Marcotte, Lettendre et al., 2008). The mechanisms contributing to the development of neuropsychological impairment as a result of HIV infection remain incompletely understood. The next section documents what is known about these mechanisms which contribute to the neurotoxicity of HIV in the central nervous system before children are born.

The development of the foetal brain in utero relies heavily on the processes of migration, myelination and synaptogenesis. The migration of new neurons to the six layered neo-cortex in utero happens in a radial fashion, and in an 'inside-out' manner where newer cells pass between older cells to the surface. This process is regulated by interactions between other neuronal and glial cells, glycoproteins, GABA and glutamate (de Graaf-Peters & Hadders-Algra, 2006). HIV interferes with the appropriate functioning of glial cells and with the production of GABA (Wilfert at al., 1994), as well as reducing brain volume through processes of ischemic injury and lesioning in the perinatal brain (Barks, Sun, Malinak, & Silverstein, 1995).

Neural conduction (essential for the efficient 'firing' of neurons) is dependent on the successful myelination of axons, which begins at 12 weeks gestational age, and continues until middle age in adulthood (Anderson et al, 2011). This is the deposition of a fatty, insulator layer around the axons which assists in the rapid transmission of electrical impulse and ensures efficient conduction (de Graaf-Peters & Hadders-Algra, 2006). Disruption to this process usually results in decreased conduction speeds, increased conductive refractory periods and complete failure of axons to achieve action potential at all. Functionally, myelination deficits are believed to be responsible for general developmental delays. If disruption to this process occurs early on in gestation, it is believed to impair motoric abilities, whereas cognitive functioning is primarily affected in later disruption in childhood (McGrath at al., 2006). It is common for perinatal entry of the HI virus into the CNS to occur at periods critical for the process of myelination (Sanchez-Ramon, Bellon, & Resino, 2003), and are believed to alter typical white matter functioning which is associated with impaired cognitive and social functioning (Angelini, Triulzi, Guidici, Pinzani, & Plebani, 2000; Brouwers, van der Vlugt, Moss, Wolters, & Pizzo, 1995).

Additionally, in order for this neurological substrate to be appropriately organised, neural networks need to be created from this overproduction of neuronal connections. This takes place by natural processes of apoptosis (pre-programmed cell death) and synaptic pruning. This is when the synapse between two neurons ceases to exist, but the neurons do not die themselves. This elimination of connection is important for the processes of cognition as it supports learning and neural plasticity. These processes of synaptogenesis and synaptic pruning are mediated by environmental influences, the decreased presence of GABA, and neurotrophic competition. Should HIV be present in the brain during these processes, it is believed that its excitotoxic inflammatory by-products impair the efficient functioning of these opposing processes to impair the development of typical neuroanatomy. This is believed to manifest in the typical features of HIV associated neuronal injury: cortical atrophy (Belman at al., 1986; Brouwers at al., 1995; Decarli et al., 1993), neuronal loss in white matter and subcortical regions (Everall et al., 1991; Wiley et al., 1986), developmental delay (McGrath et al, 2006; Van Rie, 2007), abnormal reflexes and tonal abnormalities (Armstrong, Seidel, & Swales, 1993; Mitchell, 2001; Tardieu at al., 2000).

Pathophysiological studies investigating the presence of HIV antibodies and cytological pathways to infection prove that in many cases, HIV is present prior to birth. Evidence of inter-uterine HIV transmission identifies the viral component of the HI-virus in second trimester aborted foetuses and HIV antigens in the T4 cells of third trimester foetuses (Blanche, Rouzioux, & Moscato, 1989). Similar studies found HIV DNA in the chorionic villi of trophoblastic cells as young as 8 weeks gestational age. HIV DNA was also found in Hofbauer and endothelial cells of these aborted foetuses (Lewis, Reynolds-Kohler, Fox, & Nelson, 1990). Others suggest that HIV transmission occurs directly through the placenta and occurs across the duration of pregnancy from the first to third trimester (Maury, Potts, & Rabson, 1989). More specifically, doctors in Tanzania suggest that the HIV-1 genotype C is transmitted in utero in a significantly higher proportion than the distribution of HIV-1 genotype D, A or D and A combined. Clade C is the most prolific type of HIV in South Africa (Renjifo, Gilbert, Chaplin et al., 2004). Other research is more certain that the frequency of inter-uterine HIV transmission is believed to be low in the first trimester (<8%), and that transmission most likely occurs more in the later stages of pregnancy or through delivery (Brossard, Aubin, Mandelbrot et al., 1995).

The process of virally induced neuro-compromise would assumedly be different in these cases where vertical transmission occurs later on in gestation, as the brain remained protected from infection in those early and very vulnerable few months. Clinical findings in late stage infection appear to reveal a diffuse rather than a focal CNS disorder (Angelini, Zibordi, Triulzi et al., 2000), where HIV invades the brain soon after infection and can be found in the cerebrospinal fluid (CSF) of asymptomatic HIV positive patients. There appear to be two mechanisms - direct and indirect - of CNS invasion. The direct mechanism involves the infection of neural cells. Neurons themselves are not infected by HIV, and direct infection is done via supporting neuronal cells (macrophages and microglia). Microglial cells in the brain can be directly infected as they have viral receptors, and are commonly found in the basal ganglia and white matter of the CNS. Brain macrophages are the most frequently affected cells within the CNS. Activated brain macrophages secrete chemokines which attract more (often infected) leucocytes from the blood. The HIV virus therefore has the ability to infect and replicate itself within the brain, as seen on post mortem studies (Tardieu, 1998). Astrocytes and endothelial cells form part of the blood brain barrier. Their infection and resulting dysfunction allows further entry of toxic viral or cellular products, as well as HIV infected cells, into the CNS (Epstein & Gelbard 1999; Rausch & Stover 2001).

The indirect method of CNS infection is done via chemical poisoning. The HIV infected targets, usually microglia and macrophages, secrete a battery of inflammatory cytokines and neurotoxic factors, which are believed to be toxic to nearby neurons and cause injury to these surrounding cells. Cell damage in particular areas have been associated with high levels of these cytokines in surrounding CSF, which have also been significantly paired within limited cognitive skill (spatial memory) (Gonzalez-Scarano & Martin-Garcia, 2005; Koekkoek, de Sonneville , Wolfs, Licht, & Geelen, 2008; Rausch & Davis, 2001) . This is commonly referred to as HIV associated encephalopathy. The following discussion considers the large evidence base for the degree of neuro-compromise in the context of HIV infection. It begins broadly with the incidence and neuroanatomical effects of HIV-associated encephalopathy, and then how this manifests functionally in the delay of developmental milestones. The more general effects on cognitive development are foregrounded in the discussion, which then becomes increasingly specific as it focuses on motor and language development. The focus of this section culminates in a critical review of empirical evidence on the effect of HIV on working memory, and ends with a discussion on the biological basis of working memory

and the theories proposed which could explain the proposed compromised performance in working memory.

Encephalopathy.

HIV associated encephalopathy can be static (the attainment of milestones is apparent, but delayed in comparison to peers) or progressive (milestones previously attained are lost in either an acute or progressive manner) (van Rie, 2007). It is characterised by impaired brain growth, microcephaly, a loss of cognitive and motor function, abnormal tone and reflexes, decreased muscle strength, pyramidal tract symptoms, ataxia and seizures (Whitehead, 2012). Neurological complications in a sample of six children between the ages of six months and five years with AIDS were followed over fourteen months. The most frequent manifestations were encephalopathies, microcephaly and various electrophysiological abnormalities. There were also varying degrees of cortical atrophy with ventricular calcification. Two of the six children had CNS infections, neurological deterioration resulted in dementia in three of the children, while the other three had experienced cognitive impairment and developmental delay. Post-mortem examination of the three children who died showed cytomegalovirus encephalitis on one, and non-specific white matter changes and notable calcification of the basal ganglia in the others (Belman, Ultmann, Horoupian, Novick, Spiro, Rubinstein et al., 1985). The calcification of basal ganglia in infants who died of the virus without treatment were also noted in other studies of a similar nature (Belman, Lantos, Haroupian, Novick, Ultmann, Dickson, & Rubinstein, 1986). Angelini at al. (2000) report significant neurological manifestations of the virus in sixty-two vertically infected children (mean age seven years, three months), with identified encephalopathy, multifocal white matter alterations and cerebral calcification which was correlated with higher traces of viral load found in the CSF.

The incidence of HIV encephalopathies are highly variable. In fact prior to the introduction of cART, encephalopathy was reported in 35-50% of children with a diagnosis of HIV in the United States (Tardieu, Le Chenadec, Persoz, Meyer, Blanche, & Mayaux, 2000). This has since been reduced to less than 2% after the initiation and following of correct treatment regimens (Van Rie, 2007). Other studies report a range of encephalopathic indices, which begin as low as 22.5% (Foster, 2006), and begin to escalate to reports of 50% (Knight et al, 2000), 65.06% (De Carli, et al. 1993), between 60—90% (Msellati et al., 1993), and 83% (Brouwers et al., 1995). Comparison and control across studies is very difficult as

some included the influence of drug use during pregnancy, and occurred across various times where the sophistication and efficacy of treatments were highly variable. Regardless of prevalence, encephalopathy typically results in the delay of developmental milestones. This is usually pervasive across development and will be considered further in the following sections. A review of HIV associated impairment in general cognitive development follows.

Both local and international research suggests that HIV infection most often results in the delay or loss of developmental milestones, and has a particular and significant effect on the neurodevelopment of infants. In a study that investigated the neurodevelopmental functioning of 35 treatment naïve HIV infected children aged 18 -36 months using the Bayley Scales of Infant Development (hereafter the Bayley Scales (BSID-II)) and other screening measures, researchers found that the 60% had severe delays in cognitive functioning (van Rie, Mapuala, & Dow, 2008). Similarly, a local study of HIV-infected infants (0-2 years) shows significant delays when compared to an HIV exposed control group (Whitehead, 2012) on gross measures of infant motor development (BSID-II), but lacks the inclusion of an HIV-UU baseline and are focused on very young samples. Another longitudinal study compared the neurocognitive functioning of a treatment naïve population of 79 HIV infected (HIV-I), 241 HIV exposed, uninfected (HIV-EU) children and 116 uninfected, unexposed (HIV-UU) children (aged six to twenty-four months). The comparisons indicated that HIV infected children demonstrated greater deficits in motor development and neurologic status, and had more frequent and earlier onset of motor and neurologic abnormalities. The children were measured on the Bayley Scales and the Fagan Test of Infant Intelligence at six monthly intervals. Infant information processing was the only construct where there were no significant differences between the three groups (Drotar, Olness, Witznitzer, Guay, Marum, Svilar et al., 1997).

Cognitive Development.

Sherr, Meuller and Varrall (2009) found that eighty one percent of the included fifty four studies of HIV infected samples with control groups reported a detrimental effect on cognitive development. The authors concede that they have a strong Western bias (76% from United States and Europe), and that there is a lack of systematic developmental measurement, controlled trials and age- and gender-specific investigations which render the literature on the topic inadequate. Abubaker at al. (2008) completed a smaller review of six studies, and found that all measured domains of cognitive development were negatively

affected by viral infection. The finding had significant methodological limitations. Very few studies investigated the pathophysiological impact on language, were all done on very young children (<48 months) and appear to have been on a treatment naïve population that was studied prior to the roll-out of ARV treatment in Africa (Abubakar, van Baar, Van de Vijver, Holding, & Newton, 2008).The authors also assume that cognitive ability is a unitary construct and group all striated abilities into a singular function and report a single effect size. The idea that cognitive abilities might be underpinned by a singular and measurable 'g', as first proposed by Spearman (1927), might have theoretical validity, but is an outdated and reified psychometric concept. The second critique refers to the instruments used to measure cognitive ability in the studies included in this review. Those studies which measured children under two-years used the Bayley's Scales or the Fagan Test of Infant Intelligence. These instruments tend to be crude measures of cognitive functioning in young children which often lack validity as they are dependent on parental report which is notoriously unreliable. There are also low correlations between infant IQ scales and child IQ scales as the former attempt to measure quite abstract cognitive processes (higher order verbal skills) through a motor output observable in infancy. The Fagan Test of Infant Intelligence has also been heavily criticised for being a poor measure of visual recognition memory, as well as a poor predictive measure of intelligence in later childhood because of significant methodological weaknesses in its standardisation and poor reliability (Benasich & Bejar, 1992).

Therefore, while there is large scale agreement between studies that cognitive delay and below age-expected functioning is a consequence of viral infection, there is very little methodological replicability and control between these studies. Replicability is further complicated by issues of sample matching and ethical issues around treatment progression. Further, very few studies allow for the distinction between HIV infected, exposed and unexposed samples. A comparison of studies assessing treatment naïve and cART-initiated samples also lacks control. This is because wait-listing vulnerable, young samples on potentially life-saving medication has ethical implications; and the progression and diversity of treatment regimens across contexts makes the isolation of the preservatory and potentially masking effect of ART difficult, as different participants use different drug combinations (Armstrong, Seidel, & Swales, 1993; Foster at al., 2006; Levenson 1992;

Llorente, 2003; McGrath at al., 2006; Nozyce et al., 2006; Potterton et al., 2009; Van Rie, et al., 2007).

There are also a number of studies which do not agree about the detrimental effect of viral infection on cognitive development. For example, Bagenda et al., (2006) considered the health and neurodevelopmental progress of twenty-eight vertically infected, treatment naïve children between the ages of six and twelve years. While the HIV infected children showed evidence of acute malnutrition, they showed no significant impairment on subtests of the Kaufmann-ABC and WRAT-3 measures when compared to control groups of exposed and unexposed samples. Blanchette et al. (2002), conducted a study amongst poorer families in Canada, where he investigated the neurodevelopment of fourteen vertically infected children (with no maternal drug use) using subtests from the WISC-III and WRAT-R, and CT scans. Fifty percent of the infected children had abnormal CT scans, but there was no significant correlation between CD4+ counts and any area of neuropsychological performance. Excluding subtle fine motor deficits, neuropsychological performance was generally within the ranges expected of typically developing children. The study is however limited by very small sample sizes (N = 14), and the use of a sibling sourced control group which does not account for the effect of possible viral exposure. It also has limited external validity to the multilingual South African context as the participants had high levels of English proficiency. The majority of infected participants were also mildly or completely a-symptomatic. Bagenda et al., (2006) concedes the issues of asymptomaticity as a possible mediator of dysfunction, and suggests that the damaging effect of viral infection was buffered by the participants' delay in displaying symptoms (late start, asymptomatic, treatment naïve participants). The strong English language proficiency within this sample might also be reflective of the group being from a higher socioeconomic position, and therefore having personalised medical care which could better control their viral progression. These findings were confirmed in a local study of HAART-naïve, 'late progressors' in South Africa (Webster, 2009) who also found limited cognitive impairment in their sample of twelve children. Henry, et al., (1996) examined the effect of immune system dysfunction, HIV status and the psychosocial environment on cognitive development and found that the degree of immune system dysfunction was positively related to relative cognitive decline.

Therefore, while there are pockets of research evidence to support the relative imperviousness of cognitive functioning to the impact of HIV infection, it would appear that these studies are limited by methodological concerns. The conclusions of the large majority of research in this area suggest that HIV has a definite effect on cognitive functioning, but allow for external factors to mediate the degree of the neuropsychological dysfunction. Research supporting the existence of general cognitive dysfunction in children infected with HIV is, however, limited in its external validity as it fails to identify where these specific deficits lie, and is unable to provide cognitive profiles which can effectively inform intervention and treatment. This next section briefly documents previous empirical support for the effect of HIV on motor development. This has relevance to the present study as a motoric component was included in the neuropsychological battery of the NEPSY-II.

Motor Abilities.

The assessment of motor development is less vulnerable to the effects of bias within non-Western contexts as it is a non-verbal skill, and is independent of English proficiency to attain milestones. Research evidence regarding motor development would suggest that it is pervasively affected by HIV infection, and appears to be the most severely affected of neurodevelopmental domains. Motor dysfunction is often identified in the absence of any other identifiable markers of neurocognitive dysfunction, and is considered to be a reliable screening tool for HIV associated deterioration (Potterton, 2006; Whitehead, 2012).

There is a well-established body of South African research which converges to identify motor impairment as one of the primary areas of neurodevelopmental compromise in the context of HIV infection. Potterton and Eales (2001) employed a brief motor screening tool to identify motor dysfunction in an infected sample (n = 30, < 12 months old), and found a prevalence of 40%. Potterton, Stewart, Cooper et al. (2009) conducted a study on 122 HIV infected children (M = 18.5 months, SD = 8.1) in Soweto and found pervasive motor dysfunction across the sample. The Bayleys Scales identified 72% with severe motor delay, with motor development more severely affected than cognitive development. A similar study was conducted using the same instrument in Cape Town, South Africa, but employed a matched control group. The prevalence of significant motor delay in the HIV infected sample was 66.7%, compared to only 5.7% in the age matched control (Ferguson & Jelsma, 2009). Jelsma, Davids, and Furgeson (2011) found that, even when accounting for institutionalised care and the provision of an unexposed control group, significant differences were apparent

in the children's motor development (*M* = 52.8 months, *SD* = 10.9) when assessed using the Peabody Development Motor Scale – II.

In other developing contexts, a study of Rwandan children aged 6 months to 2 years observed that 40% of HIV-infected children had an abnormal neurodevelopmental examination at 18 months compared with only 5% of HIV-exposed uninfected children, where gross motor scores were found to be significantly lower at all studied time points in the HIV-infected children (Msellati, Lepage, Hitimana, Van Goetham, Van De Perre & Dabis, 1993). Generally, gross motor functioning appears to be more delayed than fine motor ability (Baillieu and Potterton, 2008; Jelsma at al., 2011; Msellati, 1993; Van Rie, 2007). Further afield, systematic reviews of studies considering the effect of HIV on motor development are equally as conclusive. In a review of studies from Sub-Saharan Africa, Abubakar et al. (2008) found that all studies investigating motor development report significant differences between infected children and controls. Differences are evident as early as the first six months of life, and increase with disease progression. Motoric impairment appears to remain evident in pre-school (Le Doare, et al., 2012; Van Rie et al., 2008) and school going populations (Boivin, et al., 1995; Phutanakit, et al., 2013). For a collection of evidence supporting the detrimental effect of viral exposure on motor development see: Angelini, Zibordi, Triulzi, et al., 2000; Barret, Tardieu, Rustin et al., 2003; Blanchette et al., 2002; Dawes, Suarez, Casey et al., 2008; Heaton, Marcotte, & Mind, 2004; Kerr et al., 2014; Llorente, 2003; McGrath, Fawzi, Bellinger et al., 2006; Mekmullica, Brouwers, & Charurat et al., 2009; Potterton, 2006; Reger, Welsh, Razani et al., 2002; Whitehead, Potterton, & Coovadia, 2014; Williams, Marino, & Malee, 2010; Williams, Seage, & van Dyke et al., 2012). It is interesting to note, that unlike cognitive development, working memory and language development, not a single study found in the literature searches for this thesis advocated for the imperviousness of motor development to HIV infection.

As with all extensively researched domains, the generalizability of results is conflated by methodological issues. These include: the great diversity in the demographic criteria and health of samples used, the differential inclusion of institutionalised children which could confuse results, the use of samples on different cART regimens, and the inclusion of studies which include cART naïve and initiated children, the use of different measures of assessment (while many relied in the BSID-II in infant studies), and the attempted comparison of

differential socioeconomic backgrounds. There is also the issue of including and comparing the results of studies from both the developed and developing world, where the former is affected by issues of perinatal drug exposure, but has excellent post-natal care, and where the opposite is found in the latter (Whitehead, 2012). Despite the various influences compromising the comparability of results, there is strong evidence that HIV affects motor development.

These next main section considers the effects of viral infection on language. However, before proceeding onto more specific neurocognitive functions, it is important to consider the effects of studies on general cognitive development within samples who have had exposure to ARV. While an inherent weakness of research is that it is not always clear or indicated whether samples have been exposed to ARV treatment or not, it would be amiss not to include the debate regarding the neuro-protective and neuro-toxic effects of ARV treatment on cognition in developmental samples. Because the focus of these studies is often on the effect or associations of the treatment regimen, the cognitive constructs are seldom assessed with any particular specificity or theoretical grounding, and are usually general, gross measures of overall cognitive performance. For this reason, these studies are discussed in relation to those reviewed in the aforementioned sections on the associations of HIV infection with general cognitive development. A brief discussion on ARV treatment precedes the review to contextualise the effects.

ARV Treatment.

Antiretroviral drugs are unable to remove the presence of the virus, but instead function to hinder its development further within the body. The five different types of ARV's currently available serve this function at different points of the virus' development in an attempt to interrupt viral replication at different points of its reproduction (Faulhaber & Aberg, 2009). These include: nucleoside analogue reverse transcriptase inhibitors (NRTI's), nucleotide reverse transcriptase inhibitors (NRTI's), and non-nucleoside transcriptase inhibitors; Protease inhibitors, entry inhibitors and integrase inhibitors. Conventionally, regimens of these types of antiretroviral prophylaxes have been used in varying permutations and combinations to render effective treatment, and were referred to as HAART (Highly Active Antiretroviral Treatment). Recently, scientists have moved away from this reference, and prefer to use cART (combination antiretroviral therapy) as a more accurate reflection of current HIV care. This is in response to a growing interest in single drug therapies where

patients are prescribed a single pill, or fixed combination dose, - preferred because of its cost effectiveness, its ease of adherence and the lower associated risks of long term treatment such as premature aging and metabolic dysfunction (Bangalore, Kamalakkannan, Parkar, & Messerli, 2007; Bangsberg, Kroetz, & Deeks, 2007). The terms HAART and cART are used interchangeably in this thesis as they straddle research done on both types of treatment.

The majority of children who become infected with HIV are infected by their mothers through vertical transmission of the virus, either in utero, during delivery or through breast milk (Kourtis, Bulterys Nesheim, & Lee, 2001). The risk of vertical transmission is exacerbated by high maternal viral load, placental infection, the presence of sexually transmitted infections and maternal malnutrition (John & Kreiss, 1996). Despite these risk factors, in developing countries where sound Prevention of Mother to Child Transmission (PMTCT) is practiced, the use of ARV's and sound feeding practices are believed to reduce the rate of vertical transmission to below 2% (Coovadia, 2011). In South Africa, the National Strategic Plan (2007-2011), has adjusted its PMTCT treatment guidelines to align with WHO recommendations, and currently estimate that its ARV coverage to HIV infected pregnant women is greater than 95% (South African National AIDS Council, 2010; South African National AIDS Council, 2011; World Health Organisation, 2011).

The use of the PMTCT drugs Azidothymidine ('Zidovudine') (AZT) and Nevirapine (NVP) has been so successful that its use for PMTCT is currently mandated in the National Strategic Plan 2012-2017 by the South African National AIDS Council. In-utero intravenous and oral delivery of cART to the neonate have been found to dramatically reduce mother-to-child transmission of the HI virus, in the absence of breastfeeding to <2% (Ioannidis, Abrams, Ammann et al., 2001). Its effective use in developed countries is currently believed to so significantly reduce the rate of MTCT that South Africa currently provides free PMTCT to all pregnant women in order to reduce the effect of the HIV epidemic and encourage its classification as an eradicable disease in the continent (Coovadia, 2011; Perinatal HIV Guidelines Working Group, 2001; South African National AIDS Council, 2012). However, despite these successes, the impact of ARV's in creating other unwanted and degenerating side-effects on the body cannot be underestimated.

With the introduction of effective PMTCT around the turn of the century, studies investigating the neurological and developmental profiles of both HIV-infected and HIV-exposed, uninfected children face a particular challenge in their inference of causality in their findings, as it is unclear whether salient deficits are due to HIV exposure in utero or the toxicity of the ART. This is complicated by the equivocal evidence regarding the safety of PMTCT. The following sections discuss related research findings with regards to neurodevelopmental effects from ARV treatment in childhood. A discussion on the effects of indirect exposure to ART through the placenta and at birth is discussed later in relation HIV-exposure.

Advances in the understanding of the neuropathogenesis of HIV has had a dramatic influence on the clinical course of HIV infection on the CNS. Both adult and paediatric populations from Western and developing contexts have shown substantial improvements in neurologic functioning, particularly the reduction in encephalopathies, when adherent on effective ARV regimens (Brouwers, et al. 1990; Gongvatana et al., 2009; Jeremy et al., 2005; Laughton, Cornell, Grove et al., 2012). However, while HAART has been very effective in decreasing mortality, it is less effective in protecting the CNS from the eroding effects of the virus, particularly in critical stages of neurodevelopment (Lindsey, Malee, & Brouwers et al, 2007; Smith & Wilkins, 2014).

Jeremy et al. (2005) conducted one of the first large scale studies of neurocognitive functioning in youth after the initiation of HAART, and found significant, pervasive and enduring impairment in all areas of neurodevelopment (cognition, short term memory, vocabulary and fine motor skills) up to the age of 17 years. While this study does well to assess a broad age range, and ensured that all children were following one of six well-controlled ART regimens, it is limited by the fact that there is not a single measure of cognitive performance suitable for all children in this sample. In these cases, the Bayley's Scaled of Infant Development-II was replaced with the WPPSI-R. Crude, motoric-based infant IQ scales such as the BSID-II have poor validity of cognitive functioning in older children and their measurement of IQ which is usually done through the verbal assessment of higher order functions. Further, the children had only been receiving ART for 16 weeks prior to assessment, which is unlikely to have had sufficient time alter neurocognitive outcomes.

Significant improvements have been found in a study with a similar age cohort in the Democratic Republic of the Congo after 6-12 months of ART (Van Rie, Mapuala, & Dow, 2008; Van Rie, Mapuala, & Stewart, 2009).Two South African cohort studies found high pre-ART levels of neurocognitive delay in a sample of thirty well-nourished and immunologically stable HIV-I pre-schoolers. The introduction of cART showed no significant improvement or deterioration in their performance on the Griffiths Mental Development Scales, even after 6 months of treatment (Lowick, Sawry, & Meyers, 2012; Smith, Adams, & Eley, 2008). In an evaluation of the cognitive functioning of a school age sample in the Netherlands, the correlational analyses resulted in only two significant outcomes, showing that higher CD4+ treatment initiation and longer treatment duration were associated with better working memory function and attentional control (Koekkoek, de Sonneville , Wolfs, Licht, & Geelen, 2008). The Children with HIV Early Antiretroviral Therapy (CHER) trial demonstrated that early initiation of ART in South African children resulted in favourable, short-term neurodevelopmental gains, decreased infant mortality and disease progression, in comparison to later initiation of ART (Laughton, Grove, Kibb, Springer, Dobbels, Janse van Rensburg et al., 2009). Therefore, their presence in the body does not appear to be initially damaging. As a result, the state has recommended that all HIV positive children under the age of five years be started on ART regardless of CD4+ count or WHO staging (SANAC, 2014).

Evaluative reviews of such studies have inherent threats to their validity. Griner, Williams, & Read et al., (2011) conducted a trend analysis on ARV use across a series of large cohort studies and confirmed that there is an increasing number of antiretrovirals used in utero for the purposes of PMTCT. While this regimen diversity increases treatment effectiveness, it complicates the validities of observational studies which consider the effect of ARV exposure. The large majority of evaluative studies regarding ARV toxicity (or lack thereof) are also written by foreign researchers, studying foreign populations. A South African doctor, Mark Cotton (Head of the Paediatric Infectious Diseases Unit, Stellenbosch University) wrote a paper concerning the toxicity of ART on infants (Cotton, 2002). He considers multiple reviews of large patient databases available at the time of going to print, and then compares the health profiles of patients overseas and those in South Africa. In most of those studies, about 71% of patients were either asymptomatic or mildly symptomatic, while 68% of mothers seen in South African hospitals are either in Stage B (moderate) or C (severe) by WHO standards of disease progression. Cotton concludes that

ARV toxicity to foetus' are of particular concern in South Africa as "drug reaction profiles might be different in South Africa due to the more severe disease profiles experienced" (p. 15). Further, "given the limited and relatively short-term experience with all antiretroviral agents in pregnancy, long-term follow up of infants exposed to these medications is important" (Anderson, 2001, p. 291). Overwhelmingly, it would appear that any possible risk second to ART exposure is less damaging than the almost certain long-term damage as a result of viral infection. The discussion now increases in the specificity of its direction, and attention is turned to two areas believed to be vulnerable to HIV infection, and of particular importance to the context of this study; language and working memory.

Language.

The effects of HIV infection on language development are difficult to isolate, and almost impossible to define without the influence of psychometric bias or the inclusion of a series of external, yet related factors such as socioeconomic status, school quality, exposure to Western curricula and English proficiency. The effects of the virus on language development are also difficult to identify with any degree of specificity because many studies consider the construct in infants (children under 24 months), and are therefore actually assessing a series of pre-lingual skills which are highly dependent on motor function. The subtle compromise apparent in higher order constructs might only be apparent in older children. Nevertheless, this section discusses the research findings of the effect of HIV infection on language development.

Wolters, Brouwers, Civitello, and Moss (1997) conducted a longitudinal follow-up study of the expressive and receptive language abilities of forty four HIV infected children (*M* = 5.4 years). Psychometric measurements using the CELF-R (Clinical Evaluation of Language – R), as well as CD4+ counts and CT scans, were taken prior to the commencement of ARV's, and every six months for two years. Results indicated that while cognitive functioning (as measured using the BSID-II, McCarthy's Scale of Children's Abilities or the WISC-III) remained stable over time, expressive language was significantly more impaired than receptive language and appears to be differentially affected by the disease. While no detrimental changes were noted in either expressive or receptive language in the first six months following the initiation of HAART, significant and gradual decline was apparent in both domains between the 6-month and 24- month marks. Prevalence studies support this notion, and identify both receptive and expressive language impairment in 35% of children

born to HIV positive mothers - regardless of whether they are HIV infected themselves. Linguistic measures reveal that perinatal HIV exposure is associated with performance below the 21st percentile (Rice, Buchanan, Siberry, et al., 2012). In a review of studies considering the general neurodevelopment of HIV infected children, Le Doare et al., (2012) found that there is evidence for deficits in higher order language development in older, school going children, with scores remaining below typical norms even after the initiation of ART. Subtle linguistic deficits are also noted in even clinically stable HIV infected paediatric samples (Boivin, Busman, Parikh et al., 2010). Van Rie et al. (2007) also assessed a broad span of language tasks using the Rosetti Infant Toddler Language Scale, and found that 85% of infected pre-schoolers in the sample had delays in language expression, and 77% had delays in comprehension. The most comprehensive study of viral infection on language development in school-going populations was done by Brackis-Kott, Kang, Dolezal et al., (2009) who compared language performance in 206 HIV infected children (9-16 years) with a control group using the Picture Peabody Vocabulary Test (PPVT-III) and the WRAT-3. HIV status was significantly associated with poor performance on both measures, even after adjusting for demographic influences. Many of these children were retained for an additional year at school, or attended special education classes. Other studies confirm similar findings in younger populations (See: Martin, Wolters, Toledo-Tamula at al., 2006; Sherr, Meuller & Varrall, 2009; Webster, 2009).

The development of language in children seems to be particularly affected by viral influence, as it is commonly understood that language remains relatively intact in adult viral infection, and only begins to deteriorate with acute disease progression (Dawes, Suarez, & Casey, 2008). This could be because of the sensitivity of the developing brain coupled with the consequence of vertical transmission, or because chronic otitis media is a common symptom of HIV infection in childhood, and affects hearing at a critical period in which language is developing (Layton & Scott, 2000; Potterton, 2006). Blanchette et al., (2002), however, find no sign of language impairment in relation to their control group when using subtests of WISC-III, and make the point that previous studies identifying 'HIV associated language impairment' could be measuring a second language issue as these studies usually make use of bilingual samples from low socioeconomic brackets.

While these findings suggest that language development is impaired in the context of HIV infection, it is important to point out that many of these studies actually measure a

degree of English proficiency and exposure to a particular set of Western linguistic standards, rather than a developmental capacity to learn language. Further, the generalisability of these findings are limited in much the same way as those in other cognitive domains are. However, working memory assessment is considered a processing-based measure of cognitive potential and has been found to be a sufficiently accurate measure of verbal working memory, even in bilingual children who were not tested in their dominant language (Cockcroft, 2014). Therefore, while not impervious to the effects of bias in language, working memory is a better attempt than conventional testing.

Working Memory.

This final section refers to one of the primary aims of this study and discusses the associations between working memory and HIV infection. The biological basis of working memory is presented and then discussed in relation to a review of empirical evidence on previous research regarding working memory and HIV. This study does not make use of any direct assessment or investigation of the neuro-functional components of the brain responsible for working memory. However, a brief synopsis of its neurobiological basis is provided here to provide a context in order that functional deficits might be understood according to their neurological location when interacting with the HI virus.

Understanding working memory as a cognitive construct through the lens of functional neuroanatomy affords the opportunity to hypothesise how it should be affected in the context of HIV exposure – a phenomenon which to date, has not yet been decided on. The neurobiological basis of working memory is particularly interesting. Its fractionated, modular structure implies that there is localised specificity of the multiple discrete functions that comprise the greater system. However, efficient working memory functioning relies on the existence of a dynamic construct, comprised of a series of interacting and interrelated parts across the cortex, which communicate through multiple neural networks. This componential, yet effusively connected structure positions the construct as simultaneously vulnerable and resilient to the diffuse effect of viral exposure through CSF. Its broad-based distribution across multiple regions across the cortex make it vulnerable to viral-associated damage. However, this very distribution also affords the construct the capacity to circumvent damaged regions and compensate for these through processes of neurodevelopmental plasticity (Diwadkar, 2011; Müller, 2006; Shepherd, 2014). This idea remains an area for future research in the context of developmental viral exposure.

Empirical evidence regarding the HIV-associated functioning of the modular components of the Multicomponent Model (2000) will now be considered.

Phonological Loop.

Neuroanatomically, the phonological loop is supported by structures in the left hemisphere of the brain (Alloway, 2007). When measuring the storage and rehearsal phases of verbal working memory tasks, activity is found in Broca's area (involved in speech production) in addition to supplementary and premotor areas (involved in movement) in frontal cortex. More specifically, the left supramarginal gyrus is the primary location of the phonological store, and Broca's region believed to be pivotal to the articulatory rehearsal process. In addition, different networks are involved in retrieval as compared with storage in the left lateralized frontal cortex (Paulesu et al., 1993; Salmon, van der Linden, Collette et al., 1996). The following sections discuss previous research of the functional effects of HIV on the phonological loop.

York, Franks, Henry and Hamilton (2001) conducted a thorough assessment of the functioning of the phonological loop in a comparison of 36 asymptomatic and symptomatic HIV-I adults. They assessed the phonological store of the phonological loop by assessing two phenomena which have previously been known to provide evidence for its existence. The phonological similarity effect was measured by asking participants to recall increasingly longer lists of both phonologically similar and dissimilar letter spans, while the irrelevant speech effect was measured by asking participants to recall and increasingly longer list of phonologically dissimilar letters while listening to irrelevant speech. The results found that the HIV-I symptomatic group had significantly diminished performance on both measures, while the HIV-I asymptomatic group showed no depressed performance on the irrelevant speech measure. The articulatory control and rehearsal function of the phonological loop was measured using the word length effect (the recall of increasingly longer lists of visually presented words) and the articulatory suppression effect (perform the previous task while repeating out-loud 1,2,1,2…). Both groups demonstrated intact articulatory rehearsal processes when measured on this task. These findings suggest that HIV-infected adults have a deficient ability to store verbal information in working memory, but that information that is successfully encoded and gains access to the phonological store can be successfully rehearsed and processed by the articulatory rehearsal process (York, Franks, Henry, & Hamilton, 2001). These findings were similar, but less thoroughly assessed in a study of 147

HIV-I male adults in measures of Reading and Digit Span tests (Stout, Salmon, Butters et al., 1995). Similarly, Klaas et al., (2002) also document typical performance by HIV- infected children on a measure of immediate verbal recall, but concede that performance in the infected group was worse in areas of recall rather than in recognition, indicating an association between viral infection and impairment in short term verbal storage. The following two sections draw on research similar to that of Klaas et al., (2002) which have a particular focus on children.

Blanchette et al. (2002) used the Digit Span test of the WISC-III on a between-group comparison of 11 HIV-I children (M = 9.4 years) and found no difference in performance on short term verbal storage in his comparison of HIV infected children and a sibling sourced control group. These findings are limited with reference to an HIV-EU group because of the sibling sourced control group, where four had an infected parent. This reduces the findings applicability within the growing population of HIV-EU children. The sample sizes were also small (n = 11). Martin, Wolters, Toledo-Tumala et al. (2006) also found that their sample 41 children with vertically acquired HIV infection (*M* = 11.2 years, *SD* = 2.5) performed in the average range of performance on the Digit Span (*M* = 8.1, *SD* = 2.6) subtest of the WISC-III. They did concede that poorer performance on both forward and backward digit span tasks was associated with greater neuroanatomical abnormality when measured by CT scan topography. However, again, these findings are on children from white, English speaking children whose caregivers had an average education duration of 13.6 years. These qualities can all buffer the effects of viral infection, and reduce the degree of associated impairment.

There are numerous limitations with the studies that have been included in this section. Firstly, there is equivocal support for the use of word and digit span tests as a reliable and valid measure of verbal short term storage, particularly within a population who speak English as a second language. Secondly, only one study has focused their investigation of verbal working memory using Baddeley's conceptualisation of the construct as a complex processing function. Further, there is a process of developmental fractionation of verbal short term stores where it can be expected that the skill is more developed in adult samples (as those presented here), than in children. Lastly, even the paediatric samples used here were not school beginners where there is a typical developmental transition from visuospatial encoding to that of verbal or auditory stimuli. This reduces the applicability of this profiling to the school beginners assessed in the present study. The next section

presents the neuroanatomical location of the visuospatial sketch pad in the brain, and then reviews empirical support for its functioning in relation to HIV infection.

Visuospatial Sketchpad.

The functioning of the visuospatial sketchpad has been found to activate regions in the superior occipital gyrus which appears to house the temporary storage of visual information. Simultaneous activation of the inferior parietal regions also occurs when visual mental imagery tasks are elicited, while spatial memory is believed to be more parietally based (Farah, 1988). Both rely principally on the right hemisphere of the brain (Baddeley, 2007). A review on previous research regarding the functional manifestations of HI viral infection on the visuospatial sketchpad follows.

Reger, Welsh, Razani et al. (2002) analysed the findings of 41 primary studies of HIV-infection in a meta-analysis of studies measuring both immediate and delayed visual memory. Significant deficits were only apparent in immediate visual recall tasks, and only when measured between an HIV-UU control and an adult group with mature disease progression (AIDS). They suggest that declines in visual memory occur only the final stages of HIV-infection. Visual memory deficits were also apparent in a study of 523 HIV infected adults who were assessed on the Rey Complex Figure-Immediate Recall. These measures have limitations in that they are not pure measures of an increasing visual span (Sacktor, McDermott, Marder et al., 1994). Within paediatric samples, a Dutch study investigating the neuropsychological profiles of twenty two HIV positive children (aged 6-17 years), explored within-group performances on measures of working memory, attention, information processing, executive functioning and visuospatial perception. These constructs were assessed using a general measure of intelligence (Snijders-Oomen Non-verbal Intelligence Test (SON-R), and a battery from the Amsterdam Neurospychological Tasks (ANT). In this battery a visuospatial memory span task was used to measure the functioning of the visuospatial sketchpad by assessing visuospatial temporal order in a serial matrix task. The authors found that while HIV infected children functioned in the average range of non-verbal intelligence, their performance on this task was significantly below average compared with normative data for their ages (Koekkoek, de Sonneville, Wolfs, Licht, & Geelen, 2008). The next sections considers the neuroanatomical location of the active processing components

of working memory (referred to as the central executive) and the supporting research regarding its functioning in relation to HIV infection.

The Central Executive and Broader Executive Functions.

Both neuropsychological and neuroimaging evidence supports a central executive organisation based on functional instead of modal specificity (Owen, 1997; Smith & Jonides, 1997; Tulving et al., 1994). The anterior cingulate cortex and bilateral regions of the dorsolateral prefrontal cortex have been identified as regions implicated in different tasks relying on selective attention, processing capacity, the activation of attention, and the preparation and activation of movement (Collette & Van der Linde, 2002; Salmon et al., 1996). Neuropsychological studies of brain damaged patients have produced contradictory findings in the isolated location of executive functioning within the frontal lobes. While the presence of frontal lobe lesions was associated with executive dysfunction in some studies, others show signs of dysexecutive syndrome following posterior brain damage. A series of neuroimaging studies have attempted to reconcile these distinctions by demonstrating that the different executive functions depend of the dual intervention of both prefrontal and posterior (mainly parietal) regions (Collette & Van der Linden, 2002). It is perhaps better to understand executive functioning as relying on a distributed cerebral network that is not restricted to anterior cerebral localisation. To that end, the activation of a number of prefrontal regions in a large number of executive tasks suggests that functioning be understood as a series of interacting networks rather than a one-to-one association between region and cognitive function (Collette & Van der Linden, 2002). The following sections present empirical research regarding these essential cognitive functions.

Central Executive.

There has been a larger volume of published literature regarding the complex processing task performance in HIV-I samples. In order to replicate the presentation of profiling in the discussion on other atypical samples at the outset of this chapter, findings have been separated into domain specific verbal and visuospatial working memory components. A further reason for this is because of the developmental fractionation of the construct expected at the school beginner level. In many of the adult studies presented here, no such distinction is made as impairment is attributed to the domain general functioning of

the central executive. The discussion begins with studies concerned with verbal working memory performance.

The verbal and spatial working memory performance of 50 HIV infected adults were compared to a control group (n = 23) on a series of n-back alphabetised, spatial stimuli tasks. The results suggest that significant performance deficits were equally apparent in both verbal and spatial domains. The study implicates central executive dysfunction as a likely substrate for these differences (Hinkin, Hardy, Mason, Castellon, Lam, Stefaniak, & Zolnikov, 2002). Another study of 553 HIV infected adults identified a consistent pattern of impairment in three subtest measures of verbal working memory (Letter-Number Sequencing, Paced Auditory Serial Addition Test-50, and COWAT (a measure of verbal fluency) (Dawes, Suarez, Casey et al., 2008). The authors mistakenly assume that verbal fluency is a true measure of storage and processing demands within the verbal domain which limits the validity of their findings in a theoretically pure sense. In a similar study comparing verbal working memory in seropositive and HIV-negative male drug users, impaired verbal working memory performance was significantly more common in HIV symptomatic adults, when compared to controls. This was assessed using a Listening Span Test and a Self-Ordered Pointing Task (Farinpour, Martin, Seidenberg, Pitrak, Persell, Mullane, et al., 2000). These findings are confirmed by significant deficits performance by HIV-I individuals in the Verbal Memory Span test which was used to determine verbal working memory performance in the York et al (2001) study which has been described previously. These differences were however no longer significant after level of education was included as a covariate in the model. Lastly, the auditory working memory performance (measured with the Letter-Number Span Task) was significantly lower in the HIV-I group in a between-group comparison of 41 HIV-I adults with 37 HIV-UU controls matched on age, education and estimated IQ. Heaton et al., (2004) also found depressed scores in a group of 276 HIV infected adults on a neuropsychological measure of working memory. However, this domain score was comprised of both Digit Span (forwards) and the Arithmetic subtests of the WAIS-R. These test different components of the working memory model, and reflect a failure to adequately operationalise and then profile the construct.

There has been very little direct assessment of working memory within paediatric samples. Boivin, Busman and Parikh et al. (2010) note that even in clinically stable infected children, HIV had significant deficits in visual-spatial analysis, attention, executive

functioning, planning and working memory when compared to HIV-EU children in a control group. These Ugandan children (*N* = 60, 6-16 years) were assessed on the Cogstate computerised neuropsychological battery which employs playing cards and a Groton maze task to measure visual motor tracking, attention and learning. None of the tasks are language dependent, and hence have sound cross-cultural validity. In contrast, Koekoek et al. (2008) included a measure of visuospatial manipulation, which they consider to be a sufficient proxy for visuospatial working memory, in the ANT (Amsterdam Neurospychological Tasks) (discussed previously) and found performance of the HIV-I to be within typical ranges in a sample of 22 children (*M* = 9.46 years, Range: 6-17 years) . Similarly, a between-group comparison of 26 HIV-I, yet asymptomatic adults with an HIV-UU control group on a dual task working memory task. While the findings suggest that central executive functioning was normal, the HIV-I group had slower response times in comparison to the control. The applicability of this study is limited by the relative health of the infected group, where asymptomatic disease progression is often correlated with better neurocognitive outcomes (Webster, 2009).

Converging evidence from a number of similar studies suggest that working memory deficits in HIV positive adult, and to a limited extent, child populations are evident across multiple informational domains, and involve multiple-component factors of the construct (Bassel, Rourke, Halman, & Smith, 2002; Heaton, Marcotte, Mindt et al., 2004; Morgan, Woods, Weber, Dawson, Carey, Moran, Grant et al., 2009; Stout, Salmon, Taylor et al., 1995; Wood, Hinkin, Castellon, & Yarema, 1998; Woods, Weber, Cameron, Dawson, Delano-Wood, Grant, et al.,2010).

The application of these findings to the local context have limitations, as the majority of these adult studies are conducted in Western contexts with samples who have high levels of English proficiency. Some sample sizes are low (N = 23-35 (Bassel, et al., 2002; Morgan, et al., 2009), and there are often issues of previous or comorbid drug abuse. There are also very few studies which assess the domain specific processing and storage demands of either verbal or visuospatial working memory in paediatric samples. This is a glaring deficit in the available literature as this distinction is developmentally significant. Further, findings regarding impairment on the domain general features of the central executive in adult samples can not necessarily be used to make inferences about the working memory

functioning of children due to developmental changes that occur in working memory at different life stages (Bayliss, Jarrold, Gunn, & Baddeley, 2003).

Broader Executive Functions.

This section has been included because of the conflation of central executive functioning and that of more general executive functions in previous research regarding samples infected with HIV, particularly within the context of developmental fractionation in children. Therefore, while there is very little direct of the central executive in HIV samples, there is some on the related construct of executive functions which have been included here to provide a comprehensive review. Broadly, this section assesses abilities such as fluency, inhibition, planning, problem solving, set-shifting, updating and self-monitoring within the context of HIV. Within adult HIV-I samples, these elements of executive functioning have been assessed in a piece-meal manner. Heaton et al. (2004) assessed executive functioning through the Category Test (a measure of problem solving) and the Trail making Test (B) (a measure of shifting set), and found evidence of functional deficits in 54% of the sample. Similarly, Lovejoy, and Suhr (2009) conducted a meta-analytic review of neuropsychological functioning and adherence to medication in HIV-I adults. They found 11 studies suitable for inclusion and concluded that all studies found impairment in executive functions. Executive functioning was assessed the construct using at least one of the following five tests: Trail making Test B (cognitive shifting/complex tracking), Stroop Colour Word Test 2 and Interference Trial (inhibition), the Short Category Test (cognitive flexibility) and the Odd Man Out (visual discrimination). Some measures also included the Digit Span subtest of the WAIS-III, and the COWAT which is a measure of verbal fluency. Because results are reported in according to an aggregate domain score, the identification of direct deficits within functioning is not possible. This is again a reflection on the failure of studies to accurately refine and distinguish the components of the working memory mode.

Koekkoek et al. (2008) also investigated executive functioning in their research of children. They assessed verbal fluency (timed word generation task), set shifting (responding to a moving object under specific parameters), and two simple pursuit and tracking tasks (planning a trajectory of objects) in HIV infected children. The authors found depressed performance in the HIV-I group on measures of shifting set, and that the measures of speed and accuracy were also significantly less accurate and slower on tasks that impose executive

functioning demands – the manipulation and monitoring of working memory content in a pattern recognition task. Interestingly, as more cognitive tasks were imposed (increased cognitive load), so more and more children were unable to perform the task and gave up – hence the available results are believed to actually underestimate the working memory deficits in the sample (Koekkoek, de Sonneville, Wolfs, Licht, & Geelen, 2008). The following section presents the neuroanatomical position of the episodic buffer, and presents what little empirical evidence regarding its HIV-associated functioning is available.

Episodic Buffer.

Baddeley (2007) acknowledged that it would be difficult to pinpoint localised regions responsible for the functioning of the episodic buffer because of its strong connection to the evasive location of the central executive. Prabhakaran et al. (2000) used fMRI and saw episodic buffer activation within multiple regions of the frontal lobes. The connection between the central executive and the episodic buffer is also believed to be represented by activation in the left parahippocampal region (Salmon et al., 1996)

There are no direct measures of the episodic buffer in HIV infected samples, but there is one which shows evidence that one of the two primary functions of the episodic buffer are at least existent in HIV infected samples. Woods, Weber, Cameron et al. (2010) investigated the use of spontaneous strategy use in the protection of visual memory deficits in older HIV-I adults. When questioned on their use of strategy in the Self-Ordered Pointing Test (SOPT) (an experimental measure of working memory which involves the generation, monitoring and maintenance of a response set to a series of complex visual stimuli), 30% replied that they grouped or 'chunked' the designs based on the visual characteristics of the stimuli. While the proportion of the sample able to engage in the binding of larger amounts of information for the purposes of short term storage might be low in comparison to that of typical populations, it does provide evidence that it is operational within both young (<40 years) (26.2%) and older (>50 years) HIV-I adults (32.6%).

Much of the research evidence within the area of working memory in HIV-I samples has a number of limitations. Firstly, the studies tend to make use of gross measures of working memory which often measure the construct by proxy of generalised intelligence testing or other neuropsychological measures, instead of measuring a clearly

operationalised construct that measures a theoretically supported model of working memory itself (Bassel et al., 2002; Puthanakit et al., 2013; Sherr, Mueller, & Varrall, 2009; van Rie et al., 2008). Secondly, some of the research incorrectly conflates short-term memory with working memory, and thus probably underestimates working memory deficits (Blanchette at al., 2002; Morgan et al., 2009; Le Doare, Bland, & Newell, 2012; Martin et al., 2006). Lastly, none of the research considers the effect of HIV infection on the two dominant domain-specific representations of working memory (verbal and visuospatial) separately and then compares them to identify if there is a difference between the two (Heaton at al., 2004; Martin et al., 2001).

The profiling of working memory performance in HIV-infected samples is further limited by of the lack of consistency in psychometric measures used to assess the construct, understandings regarding the definition and operationalisation of the components of the working memory model, and relative demographic variability of the samples used. The samples also had differential access to cART regimens which conflate the effects of the virus and its treatment. Further, not one of these studies examines working memory itself through a single psychometric measure, but assesses it through various subtests which are believed to collectively measure the components of working memory. The findings are further limited in their applicability to local populations because of the inherent English language proficiency and test-wiseness in these samples. The primary failing of this body of research is the lack of research findings in paediatric samples where a developmental fractionation of the components of the model are expected. Currently, findings from adult samples are extrapolated to that of children which violates theoretical understandings of what is expected of children at various ages.

Despite these limitations, research evidence would suggest that HIV infection is associated with impairment in both the phonological loop and visuospatial sketchpad. Impairment in verbal short term storage appears to be apparent in the earlier stages of infection, while visual memory impairment is likely to only become evident in the later stages of infection. Impairment in the functioning of the central executive is increasingly becoming a primary node of recognition for HIV-infection, although these is very little research in the developmental sphere to differentiate this performance on the domains. Broader executive functioning also appears to be compromised in areas of problem solving

and set-shifting, while the functioning of the episodic buffer appears to be intact and operational. The degree to which this functioning is optimal is yet to be determined.

It is important to link these empirical findings to an understanding of the biological basis of working memory. The aforementioned studies imply that all components of the model are compromised in the context of HIV infection, albeit with differential severity. Two complementary theories of neurobiology propose how this dysfunction occurs in the brain. The first foregrounds the localised responsibility (one to one) relationship of anatomical structure and function and proposes that direct neuronal damage to the cortical regions responsible for working memory abilities is what compromises functioning. The second proposes a homeostatic paradigm for considering demand excess on any one of the modular components.

The theory regarding localised damage proposes that neurological changes associated with HIV are a result of damage to the subcortical or fronto-striatal brains systems of HIV-I patients. Verbal and visuospatial working memory rely on efficient functioning of these circuits, and it stands to reason that functioning might be therefore compromised in the presence of the virus (Ernst, Chang, & Arnold, 2003). Neurologically, studies indicate that because HIV predominantly affects white matter and deep grey matter structures (basal ganglia), with cortical atrophy usually in the frontal and temporal regions, neurocognitive deficits are expected in domains related to these areas – particularly working memory because of its broad cortical localisation (Martin et al., 2006). Changes in basal ganglia, cerebral white matter and prefrontal cortical matter are believed to be responsible for an interruption in the fronto-striatial circuitry in HIV-I infection and is responsible for compromise (York et al., 2001).

The second theory, the executive functioning hypothesis, is supported by studies which bring together the associations between observable impairment in performance (empirical/experimental evidence) and the underlying neurobiological compromise. This theory does not implicate dysfunction to any one aspect or operation of working memory to a particular localised region, but suggests rather that deficits occur when activity demands place stress on more than one component of the model: short-term memory storage (verbal and visual), information maintenance across time delays, and memory manipulation. Hence, it is not the engagement of a particular domain or combination of working memory

components that indicates HIV dysfunction, but that the task-demand on a particular part needs to exceed a particular level for impairment to be observed (Martin, Sullivan, Reed, Fletcher, Pitrak, Weddington, & Harrow, 2001). Further, unlike the incremental effect of HIV disease progression on general cognitive functioning (Bagenda, 2012), working memory performance appears to be poorly affected by viral infection in both the early and late stages (York, Franks and Henry et al., 2001). Neurologically, this is unsurprising as the HIV infected CSF is distributed within the ventricles and around the meninges of the brain, suggesting that the symptoms of primary infection are diffuse rather than localised (Koekkoek, et al, 2008). One theory suggests that it is the injury to the neural substrate caused by the HIV infection that may necessitate greater attentional modulation of the neural circuits. This uses a greater part of the brain's reserve and the additional activation of the frontal lobes is necessary to carry out the tasks. Hence, HIV infection alone (and not ART exposure) appears to be sufficient to elicit working memory deficits.

These two theories outline the proposed mechanisms by which working memory impairment is believed to take place in the context of HIV infection. There is unfortunately no hypotheses as to how working memory deficits could be explained in the HIV-EU brain. There are two hypotheses as to why the CNS of such children could be functionally different, and these will be expanded upon in the following section. The one refers to the indirect chemical poisoning hypothesis regarding general CNS neuro-compromise in HIV-infection (described above), while the second refers to an in utero neuroimmunological phenomena responsible for typical neurodevelopment in the foetus which is compromised in conditions of maternal infection and foetal exposure. What follows is a similar modelling of the discussion on HIV infection to that of HIV-exposure. It begins with a definition of HIV-exposure in the HIV-EU child, and the estimated epidemiology of HIV-EU populations. It also includes a critical discussion of empirical support for an alternative neuropsychological profile as an explanation for why atypical working memory performance and structure would be anticipated in this growing population.

The HIV Exposed Uninfected Child

The HIV Exposed-Uninfected population is a growing and under-researched paediatric group, in need of clinical care and investigation. The term 'HIV exposed' refers to a child who is born to an HIV positive mother, and whose status is not yet determinable because of the window periods of the HI virus post-parturition. 'HIV Exposed, Uninfected (HIV-EU)' refers to

infants born to HIV positive mothers, but who remain uninfected themselves. They are considered to be HIV-EU because they have a degree of exposure to the virus in utero, and could have also been exposed to a range of antiretroviral drugs that have prevented transmission. This is usually only determined at age 18 months, after a rapid HIV test can accurately identify the absence of HIV antibodies (SANAC, 2010)

Estimations of the number of children who comply with this demographic categorisation are extremely difficult to come by (Kuona, et al., 2014). In 2009, conservative estimates were that only 18% of children born to HIV-infected mothers would remain exposed, but uninfected themselves (Filteau, 2009). However, because of increasing success of prevention of mother-to-child HIV transmission programmes, this number is fast increasing. In a recent UNAIDS (2013) report regarding HIV-exposure in South Africa, a 63% increase was noted in the number of HIV-EU infants declared not positive, with only 5% of children born to positive mothers remaining HIV-reactive.

The study of the neurodevelopmental consequences of vertical HIV exposure (and not by needle-stick injury as is common to health practitioners), is conflated by the inescapable pairing of the consequences of viral exposure (in utero), and that of antiretroviral treatment (of both mother while pregnant and immediately at birth). While there are cases of infants who are born to HIV infected mothers who are neither on treatment themselves, nor receive prophylactic treatment at birth who are not HIV infected themselves, these are rare. This is a consequence of large-scale access to antiretroviral treatment in South Africa following reformed legislation in 2004, and the subsequent prioritisation of pregnant women to receive such treatment in their antenatal care. In the context of viral exposure, researchers are therefore often unable to identify associations between deviations in typical development to that of viral exposure, or the introduction of ART.

Atypical development within the HIV-EU population could be a consequence of viral exposure, ART exposure or a combination of the two. For ethical reasons, and as a result of excellent access to PMTCT, the separation of these effects is not possible. For this reason, a critical discussion of the consequences of the latter is included in a subsequent discussion. This next section documents evidence on the development and functioning of the HIV-EU child. In a typical pregnancy with an HIV infected mother, the cells of the foetus are never

infected with the virus (Chantry, Cooper, Pelton, Zorilla, Hillyer, & Diaz, 1995). In some cases, vertical transmission does however take place through the placenta, indicating that the placental barrier is semi-permeable to maternal HIV. The blood of HIV-EU infants have also found to have circulating viral proteins, as well as HIV-1 specific T-cells which suggests that the immune systems of exposed children are functionally altered by the presence of HIV or its products in the maternal environment (Clerici, Sison, Berzofsky, et al., 1993). These 'side effects' of HIV infection to the developing foetus have lead the research community to believe that the HIV-EU has an atypical developing profile which are apparent in a number of systems throughout the body.

There is mixed evidence concerning the general health of the HIV-EU child, but the biological profile of this vulnerable population does appear to be compromised, and is likely to have an impact on cognitive development. Significantly higher rates of being small for gestational age and gender (SGAG) are apparent in exposed, uninfected infants (Sofeu, Warszawski, Ateba et al., 2014), as are underdeveloped growth profiles in samples from developing African nations (Uganda (Muhangi, Lule, Mpairwe, et al., 2013); South Africa (Bobat, Coovadia, Moodley et al., 2001); and Malawi (Landes, van Lettow & Chan et al., 2012). HIV-EU children also had significantly lower birth weights, less subcutaneous fat, and decreasing mid arm circumferences over time when compared to national United States standards (Neri et al., 2013) and British controls (Newell, Borja, & Peckham, 2003). HIV-EU children also appear to have altered and deficient haematological profiles when compared unexposed controls, with significant deficiencies in lymphocyte, neutrophil and platelet counts (Le Chenadec, Mayaux, Guihenneuc-Jouyaux, & Blanche, 2003; Pacheco et al., 2006), as well as haemoglobin levels (El Betuine & Duarte, 2006).

Significant immunological changes are unsurprisingly associated with HIV exposure in infancy (Bunders, et al., 2005a; Bunders, Pembrey, Kuijpers, & Newell, 2008; Clerici et al., 2000; Farquhar et al., 2011; Heidari et al., 2011; Hygino at al., 2008; Kuhn, Meddows-Taylor, Gray, & Tiemessen, 2001; Nyoka, 2008; Rowland-Jones et al., 1993; Velilla, 2008; Warning, Ziegler & Ffrench, 2008). It is difficult to demonstrate that these responses are a type of primed protective immunity for which exposed infants are primed, or if it is simply an epiphenomena of exposure. The only available study linking this altered immunological profile and a functional manifestation has been that of Slogrove, Cotton, & Esser (2010) who document serious infectious morbidity (pneumonia, cytomegalovirus, sepsis, haemorrhagic

varicella and meningitis) in eight HIV-EU infants following altered immunological functioning in response to maternal HIV infection. Additionally, HIV-exposure has been associated with a number of other physiological dysfunctions, including metabolic disorders (Claudio et al., 2013), and mitochondrial injury and dysfunction (Brogly et al., 2007; Duong van Huyen, 2003; Noguera et al. 2004).

There appears to be equivocal evidence regarding the neurodevelopmental associations with viral exposure. In a review of the neurodevelopmental outcomes of the HIV-EU child, Le Doare et al. (2012) conclude that studies of infants (0-2 years) do not demonstrate any global developmental delay after controlling for the influence of extraneous psychosocial elements (maternal substance use), but that there appears to be subtle deficits in cognition, motor function, expressive and receptive language and behaviour in older children, which first manifest during preschool (3-5 years). These findings are confirmed by the small body of local research in this area. Springer, Laughton, Tomlinson et al. (2012) compared the developmental outcome of 17 HIV-EU and 20 HIV-UU infants using the Griffiths Scales of Mental Development. There were no significant differences between the two groups, apart from a poorer performance of the HIV-EU group on the Personal/Social scale, which is believed to be function of cultural bias. The Griffiths also relies on parental self-report which can prove unreliable as well as crude measures of cognitive functioning. Van Rie, Mupuala, and Dow (2008) compared the neurodevelopment of HIV infected (*n* = 35), HIV-EU (*n* = 35) and HIV-UU (*n* = 90) children between the ages of 18-72 months using the Bayley's Scales of Mental Development II (BDMS-II), and three other psychometric measures of motor function (Peabody Developmental Motor Scales), language (Rosetti Infant Toddler Language Scale) and non-verbal intelligence (Snijders-Oomen Nonverbal Intelligence Test). They found that while the HIV infected group was most severely affected by the virus, HIV-EU had significantly more motor and language expressive delays than the HIV-UU children. The study lacked generalizability to school beginners as they only considered the neurodevelopment of very young children who are yet to fully acquire language, or engage in higher order cognitive thinking where the more subtle effect of HIV exposure could be apparent. The reliance on the Bayley's Scales as a valid predictive measure of childhood cognitive functioning is also weak, as it relies heavily on motor performance which is a crude measure of cognitive functioning, as well on parental self-report which is unreliable. The sample sizes are also relatively small, and the study therefore

makes no allowance for the inclusion of covariates to account for influences on development other than HI-viral exposure. The children in this study were cART naïve which excludes the potentially neuro-protective effects of ARV treatment.

The studies included in the aforementioned review have focused on the neurocognitive associations with HI viral exposure, and have ignored the possible correlation with ARV exposure. This next section is centred on research in the same population but focuses on the effect of ARV prophylaxis. This distinction in focus is an inseparable consequence of the logistical inability to study the two potential influences in isolation.

The extent to which HIV-EU children are affected by in utero exposure to ARVs remains unclear and impossible to separate from the effect of HI viral exposure as both are present at birth. There are mixed findings concerning the safety of peri- and post-natal ARV exposures which occur during critical periods of development of the CNS. Both animal and human studies warn about potential teratogenicity, carcinogenicity and mutagenicity of early ARV exposure (Anderson, 2001; Bishop, Witt, Tice & Wolfe, 2004; Poirier, Olivero, Walker & Walker, 2004; Chersich, Urban, Venter, Wessels, Krause, Gray, Luchters & Viljoen, 2006) which affect the effective functioning of bone, renal, cardiovascular, haematological, neurologic and reproductive systems (Smith & Wilkins, 2014; Sibiude, Mandelbrot, & Blanche et al., 2014). Despite these claims, there are also studies promoting the safety of PMTCT to both mothers and their infants, which are discussed below.

A study of 727 HIV-EU infants given Zidovidine at birth (M = 38.3 months) found no evidence of tumours in the children. However, due to the children's young age, the researchers stress the importance of long term follow up of carcinogenicity for these infants with in utero ARV exposure (Hanson, Antonelli, Sperling, Oleske, Cooper, Culnane et al., 1999; Jibril & Egunsola, 2013). Another study also investigating the neonatal health of HIV-EU infants exposed to Zidovidine, Lamivudine or a combination of both, found that there was no difference in the infants' gestational age, Apgar scores, platelets or lymphocyte count. The only area of concern was a reduction in haemoglobin levels at birth (Beitune & Duarte, 2006). Similarly, while some studies reported low birth weight amongst HAART-exposed infants (Heidari, Mofenson, & Cotton et al., 2011; Ekouevi, Coffee, Becquet et al., 2008; Townsend, Cortina-Borja, & Peckham et al., 2007;), a rebutting body of evidence offers support for the fact that HIV-EU infants exposed to HAART were not at risk for premature

delivery, low birth weight or low Apgar scores (Toumala, Shapiro, & Moffenson, 2002), and that in cases where this was evident, they were able to correct for any birth deficiencies in the first six months of life (Briand, Mandelbrot, & Le Chanadec et al., 2009; Cotter, Garcia, & Duthely at al., 2006; Powis, Smeaton, & Ogwu et al., 2011;). Table K1 in Appendix K provides a review on the safety and side-effects of ARV's on HIV-EU infants (Maron, Guar, & Flynn, 2010; Mofenson & Munderi, 2002).

In a study of 1037 HIV-EU infants enrolled in the Paediatric AIDS Clinical Trial Groups investigated previous claims that in utero exposure to nucleoside reverse transcriptase inhibitor (NRTI) (Lamividuine, 3TC or Zidovidine), causes mitochondrial dysfunction (Blanche, Tardieu, Rustin, Slama, Barret, Firtion, et al., 2003). These findings are supported by the work of Brogly, Ylitalo, Mofenson, Oleske, Van Dyke, Crain et al., (2007) and Noguera, Fortuny, Sanchez , Artuch, Vilaseca, Munoz-Almagro, et al., (2002). In contrast to this, a review of 1008 HIV-EU children in a European Collaborative Study showed no clinical manifestations of mitochondrial dysfunction, nor were the deaths in 30 HIV-EU infants accounted for by mitochondrial dysfunction (European Collaborative Study, 2003; The Perinatal Surveillance Review Working Group, 2005). The relative safety of ARV regimens are supported by studies considering mitochondrial toxicity who found negligible clinical effect in light of the benefit of effective HIV prophylaxis (Culnane et al, 1999; Hanson et al., 1999; Modenson & Munderi, 2002).

A similar study was conducted on 374 HIV-EU infants (9-15 months), and results confirm that only the language subtest of the BSID-III had a significantly lower score for infants exposed to Atazanavir ($p = 0.01$). While the authors support the safety of perinatal ARV exposure, they recommend continued monitoring for neurodevelopmental outcomes in older HIV-EU children (Sirois, Huo, & Williams e al., 2013). Similarly, Culnane et al., (1999) found no long-term effects of Zidovudine on a longitudinal cohort of 234 HIV-EU children who were followed for 36 months on measures of growth, immunological parameters, cognitive/developmental functions (assessed using either the Bayley's Scales or McCarthy Scales), and mortality data. Sirois et al. (2013) also found no developmental markers of dysfunction when using the Bayley's-III in an evaluation of the effect of cART on HIV-EU infants. Similarly, a large retrospective review of both European and US cohorts showed no evidence of neurodevelopmental dysfunction (Alimenti, Forbes, Oberlander, et al., 2006; European Collaborative Study (2003) and The Perinatal Safety Review Working Group (2000).

The study does, however, concede that the investigation of other points in time in the child's life, and the use of more precise measures of development, particularly for areas of attention and memory, may be necessary to assess the effects of HIV and ARV exposure to the developing child. The largest study of this population to date followed 1840 HIV-EU infants for 3 years (Williams, Marino, Malee, Brogly, Hughes, Mofenson et al., 2010). The study compared the profiles of 1694 ARV exposed infants with 146 HIV exposed infants who were not administered PMTCT. Their neurodevelopment was assessed using the Bayley Scales, and the results suggest that there is no significant cognitive or motor impairment differences associated with HIV or ARV exposure in utero. The study did not have an unexposed control group against which to compare. Despite these positive findings, the study does suggest that the further study of ARV-exposed, uninfected children is warranted as they develop into school-going children. The study makes purely observational correlations between exposure and neurocognitive functioning which the authors concede as a limitation, despite including many relevant confounding variables in their models as covariates.

There are currently only four published studies which consider the neurocognitive effect of HIV exposure on school going children, and only one on school beginners. Levenson et al. (1992) found cognitive deficits in both samples when measured using the Quantitative, Verbal and Memory indices of the McCarthy's Scales in their comparison of HIV-I and HIV-EU school going children from the United States (*M* = 5 years, *SD* = 20 months). These findings are relatively meaningless because the groups but had vastly unequal sample sizes (HIV-I: 41; HIV-EU: 8), and no normative comparisons or use of an HIV-UU control group on which to gauge performance. The inferences thereof should be interpreted with caution. The other two studies examined HIV exposure as secondary issues to cognitive deficits primarily apparent in HIV-I samples (Kandawasvika et al., 2015; . Brackis-Kott, 2009). Similar to the design of the current study, Kandawasvika et al. (2015) compared three groups of children (6-8 years) on the McCarthy Scales (HIV-I:32, HIV-EU: 121, HIV-UU:153). No between-group differences were measured, but the authors report similar percentages of typical cognitive functioning between exposed (18%) and unexposed (14%) groups in their study. Similar to the current study, participants were from resource-deprived backgrounds, but differed in that their mothers were all cART treatment naïve. This was also the only study which disclosed whether their samples had received cART. Brackis-Kott (2009) compared two

groups of older children (9-16 years) (HIV-I: 206, HIV-EU:134) on language measures, and reported poor verbal performance by HIV-EU children on the Peabody Picture Vocabulary Test. The performance on the HIV-EU group was, however, higher than the HIV-I group, but the study make no comparison to an HIV-UU group.

The most comprehensive study of the neurocognitive profiles of school-going HIV-EU children was done by Kerr, Puthanakit, Vibol et al., (2014) in Thailand and Cambodia. The study assessed the neurodevelopmental outcomes of HIV-EU (n = 160) and HIV unexposed, uninfected controls (n = 167) on a broad neurodevelopmental battery, comprising the Beery Visual Motor Integration Test (BVMI), Colour Trails, Perdue Pegboard, Child Behaviour Checklist (CBCL) and either the WPPSI-III or Stanford-Binet II. After adjusting for the psychosocial and environmental influence of caregiver education, income, age and ethnicity, the HIV-EU group had statistically lower scores on VIQ, FSIQ and Binet Bead Memory Test than HIV-UU controls. No significant differences were identified between the two on PIQ, other Binet memory tests, Colour Trail, Perdue Pegboard, Beery VMI or CBCL.

Research findings on the HIV-EU population has salient limitations. There are very few studies considering this under-investigated population, and the interpretation of results from those that exist is difficult because of the heterogeneity of populations with regards to sample size, socio-demographics, maternal substance use, and the lack of information about the maternal ARV regimen in pregnancy. Most studies also use normative data for comparison instead of an HIV-UU control group. These scales are often not normed to socioeconomically disadvantaged or linguistically diverse groups, and so the influence of HIV, ART and social disadvantage are often conflated. Many studies are quick to therefore concede to the need for further research which considers the more subtle influence of HIV exposure on older children, using psychometric measures which assess neurodevelopment in a more complex and specified manner. Despite these methodological concerns, it would appear that the research evidence on the HIV-EU child converges to highlight that in utero HIV exposure appears to alter the immunological and developmental profile of the HIV-EU child, and that there is a strong likelihood that this influences their cognitive development.

Previously, this discussion briefly identified the two hypothetical pathways which could explain working memory impairment in the context of HIV-EU samples. The implications of these hypotheses are now extrapolated to what is known about HIV-EU

samples in order to propose two mechanisms by which working memory impairment could also occur in this population. The first is related to the indirect, chemical poisoning hypothesis of CNS impairment in HIV infection. Here the CNS of HIV infected children is believed to be damaged as a result of the excitotoxic effects of inflammatory by-products to infection. While the cells of the HIV-EU foetus of the pregnant, HIV-reactive mother are not infected with the virus themselves, there is nothing to say that these excitotoxic by-products could not diffuse into the CSF of the foetus, via the placenta and initiate a similar inflammatory response to the developing HIV-UE brain (Gonzalez-Scarano & Martin-Garcia, 2005; Koekkoek, de Sonneville , Wolfs, Licht, & Geelen, 2008; Rausch & Davis, 2001). The only confirmation of this hypothesis to date is evident in the assessment of 1840 HIV-EU infants using the Bayley's Scales (Williams, Marino, Malee et al., 2010). The authors observed a trend suggestive of lower neurodevelopmental functioning associated with higher maternal load. This suggested the possibility that a maternal cytokine (a toxic by-product to cytological infection) response could be identified in the infant's neurodevelopmental performance when viral load was poorly controlled during pregnancy. What follows here is the second neurobiological proposal for working memory impairment in the HIV-EU population.

Research regarding HIV exposure within the experimental domains of cognitive psychology and neuro-virology appear to be converging to produce a related set of knowledge regarding the functional cause and manifestation of viral exposure. At a micro level, virology research is discovering the cellular effect of exposure, and presents HIV-EU infants as having an altered immunological profile, but to date has been unable to ascertain exactly how these cellular changes affect functioning later on in life. On the other end of the spectrum, neurocognitive research is discovering the effect of exposure on functioning, but is unable to chronicle exactly how or why this happens. Neuroimmunology is a relatively new field of research at the intersection of neuroscience and immunology, and seeks to explain how immune function can affect neurodevelopment.

Recent research regarding the relationship between in utero and early infancy immune activation, and neurodevelopmental changes offers a hypothesis at to the connection between these two, but has not yet been researched in the context of HIV exposure. In a review of recent neuroimmunological research, Garay and McAllister (2010) highlight the beneficial role of immune molecules in the developing CNS, and the efficient

functioning of neurogenesis, neuronal migration, axon guidance, synapse formation, activity dependent refinement of circuits and synaptic plasticity. However, the presence of chronic or severe maternal infection can significantly disrupt the balance or normal neural-immune cross-talk in the foetus, and result in permanent structural changes in the developing brain.

Following the body's identification of the presence of infection, the immune system rapidly responds to the invasion of pathogens by releasing a number of signalling molecules, which in turn induce an infection-fighting response by the innate immune system. These same signalling pathways are used by the vulnerable and developing brain to coordinate processes of cell proliferation, differentiation and migration. Within the context of maternal viral infection, there is the potential for a conflation of communication between these two systems, resulting in deficient neurological functioning (Smith & Patterson, 2009). For example the effect of chronic elevation of cytokines has been researched in the pathogenesis of autism and schizophrenia (Garay & McAllister, 2010). A series of in vivo animal models have been established in rats and mice to study this association, and research continues to identify the factors which can modulate the neurodevelopmental effect (timing and acuity of infection, gene-environment interactions, sex, age). This has been investigated for the influenza virus, bacterial endotoxins and with specific prof-inflammatory cytokines, but no studies have yet investigated this effect in HIV maternal infection and viral exposure on a foetus (Meyer, Feldon, & Fatemi, 2009).

The discussion thus far has been held across the tension between understanding or hypothesising the etiological mechanisms which underlie a particular neurocognitive profile, and the similarities and differences in the discrete deficits apparent in these and other atypical populations (like those of neurodevelopmental disorders). Bishop (2006) argues that we can often do very little about the complex etiologies behind the neurocognitive profiles in atypical populations, and that the focus should be on the identification of common 'endophenotypes' which can assist in directing treatment protocols. Consequently, this study seeks to profile the working memory capacities of HIV-I and HIV-EU samples in an attempt to do just that, and by so doing, draws on the large body of knowledge concerning assessment and intervention with other neurodevelopmental disorders. However, the etiological complexity behind these discrete deficits in the two HIV-affected populations (HIV-I and HIV-EU) is quite unlike the strongly genetic component found in disorders such as SLI. The consideration of HIV therefore, exists on the outskirts of what Bishop (2006)

proposes, because the etiology regarding what is likely to be responsible for HIV-associated deficits are more controllable than genetics (i.e. ARV toxicity, regimen adherence, maternal viral load). There are also a vast number of uncontrollable factors which no doubt also influence cognitive performance of all children, which will be briefly discussed here.

Confounding Environmental Effects: Socioeconomic Status

It has long been the goal of developmental theorists to identify the factors influencing neurodevelopment. This proves particularly difficult in research areas such as HIV where there is a large degree of variability in the neurocognitive functioning of infected children, and the contexts from which they come. It is therefore not possible to determine whether compromised neurodevelopment relates specifically to HIV status, or whether it is secondary to other factors. There is also a broad array of psychosocial conditions that often accompany both HIV infection and exposure in the developing world which make it difficult to accurately attribute cause to effect. Hence, one of the greatest challenges in conducting neurodevelopmental research within this population is the complicated interplay of time-varying contributions from biological risk (heritable), medical factors linked to HI viral infection, and the myriad of social and environmental factors linked to living with HIV.

Some of these factors are related to HIV infection in the family, and include frequent absences from school due to medical reasons, illness and death of parents, siblings and family members, cultural stigma and a multigenerational burden of poverty and care that comes from comes from HIV infection (Lewis, Kaiken, & Hoyt, 1994; Salter-Goldie, DeMatteo, King, Wells and the Multisite Coinvestigators, 1997, as cited by Blanchette et al., 2002). Other factors are less specific to HIV infection itself, but exist in the same sociocultural milieu in which HIV is prolific. These include indirect associations with exposure to violence, family instability, poor educational systems and marginalised social status. Their influence in development is important, but almost impossible to isolate from the biological effect of viral infection and treatment itself. Essentially then, there are two issues at hand. The first is that neurodevelopment is a multifaceted phenomenon influenced by intrinsic and external factors. Some of those external factors are found in the developmental environment, and include contextual influences such as socioeconomic status, home environment, levels of nutrition, parenting etc. which have an effect on functioning, even in the absence of HIV disease. The second is that the influence of HIV on neurodevelopment is not an isolated intrinsic influence, but is frequently associated with some of those

environmental influences as well. Nevertheless, while it is not possible to control for these factors, it is important to consider their influence.

It is well established that HIV is prolific in the developing world, and that prevalence estimates are higher among populations of low socioeconomic status (SES). Studies regarding this association are often limited in that there is a two way causality between HIV status and SES, and that there is a simultaneous effect of SES on HIV incidence (Barnighausen, Hosegood, Timaeus, & Newell, 2010; Hargreaves, Morison, Chege et al., 2002; Lurie, Fernandes, Hughes, & Arevalo, et al., 1995). There is also equivocal evidence regarding the influence of socioeconomic status and cognitive development. A common finding among research in both developed and developing countries has identified that higher levels of SES in childhood (as measured by income, wealth or parental education) are associated with better cognitive development (Paxson & Schady, 2007). The pathways between these associations are frequently debated, with better parenting skill, increased stimulation, lower incidence of maternal depression and parental stress often proposed. However, in the developing world, it is often malnutrition and subsequent child health that is considered the greater determinant of cognitive development. Hence, the corollary of the association is not always directly apparent because it is mediated by other factors. Despite these mediators, it is generally accepted that children from poorer backgrounds are at greater risk for poorer cognitive development (Feinstein, 2003; Johnston, Low, de Baessa, & MacVean, 1994). Socioeconomic status is a difficult psychometric measure to quantify, but frequently manifests in the quality of the home environment, and the quality interactions between child and caregivers.

The influence of the home environment and the quality of developmental stimulation is a difficult concept to quantify and measure empirically. There have been numerous attempts to operationalise the construct, with the most common being the HOME Inventory which is designed to assess the degree of stimulation and support available to the child in the home environment. Studies of this nature also lack validity because they lack generalisability across different socio economic brackets. Excluding the influence of child health (particularly HIV), it is generally agreed that the quality of the home environment (usually assessed by parental attitude, parental involvement, stimulation and accessibility to play materials) has moderate, positive correlations with cognitive development, particularly in higher resourced environments (Engle, Black, Behrman et al., 2007; Walker, Wachs,

Gardner et al., 2007). Within lower socioeconomic brackets, however, children appear to be 'doubly disadvantaged' (i.e. poor socioeconomic status, is often coupled with either poor health, nutrition and HOME scores *and* subsequent poor cognitive development) – an association which is believed to remain stable across the lifespan from as young as three years old (Bradley, Caldwell, Rock et al, 1989; Feinstein, 2003). These influences are far-reaching within the vulnerable environment of HIV. Boivin, Green and Davies et al., (1995) argue that the effect of HIV extends beyond the neurologic and functional impairment because it undermines the social and nutritional strength of the home environment. Coscia et al., (2001) observed that home environment (which included parental environment, play, availability of stimulation materials) was a significant mediator of cognitive functioning in HIV infected children, and Dobrova-Krol (2010) found that rearing environment had a more significant effect than HIV infection in a sample of sixty four Ukranian matched controls on measures of general cognitive functioning.

Lastly, numerous studies report an association between poor cognitive development and malnutrition, and cite iron and vitamin deficiencies, and anaemia as pathways to the correlation (Paxson & Schady, 2007). Experts in this field suggest that this relationship is mediated by poor child health, where malnourished children are more likely to have recurrent infections, and will be less likely to learn (Dickson, Awasthi, & Williamson 2000; Miguel & Kremer 2004). Again, these effects are exacerbated within developing settings where poverty, low levels of education and poor access to health care undermine efforts to control childhood nutrition.

> 'Subsequently, such second-order effects [malnutrition, home environment, socioeconomic status] may ultimately be as much at fault for the verbal, social, and global intellectual deficiencies of the HIV-infected child as the direct effects of the virus itself…Within the context of the overwhelming human and economic need associated with life for African children, the additional burden of [HIV] on family members can have devastating effects on the development of those children irrespective of their infection status' (Boivin, Green, Davies et al., 1995, p. 20).

This inseparability of HIV-infection and –exposure from the HIV-associated environmental influence represents an inherent limitation to the validity of observational social science research which attempts to understand the direct relationships between

behavioural manifestations of impairment and their causes. The links to possible sources of impairment soon become blurred when considered within this holistic lens. However, the inclusion of the environment as a powerful node of developmental influence on the health of vulnerable populations like those associated with HIV also represents an opportunity to intervene more effectively. While there is very little that can be done to adequately reverse HIV infection or exposure, much more can be done to support communities affected by the disease to be more resilient to its negative consequences.

Conclusion

The previous two chapters have documented that working memory is an essential skill to everyday functioning and academic skill, and is a far better predictor of scholastic success than conventional measures of intelligence, even in samples who do not speak English as a first language or who come from resource-deprived settings unfamiliar with knowledge driven curricula.

There are multiple explanations of working memory, but the Multicomponent Model developed by Baddeley (2000) appears to be the most theoretically robust in accounting for the vast amount of empirical data regarding working memory. Typical adult working memory functioning is best accounted for by a four factor modular structure comprised of the domain general central executive, two domain specific slave systems (phonological loop and visuospatial sketchpad), and the episodic buffer. This fractionation of working memory is not consistent through childhood in typical samples, where there appears to be a further domain specific fractionation of the executive processes of the central executive. To that end, the working memory structure in typical school beginners is best accounted for by a five factor model: verbal short term memory (phonological loop), visuospatial short term memory (visuospatial sketchpad), verbal working memory, visuospatial working memory (domain-specific fractionation of the central executive), and the episodic buffer. The episodic buffer is particularly difficult to measure in atypical child samples, but has been done in a handful of studies.

A large body of research evidence has also converged to profile the working memory abilities in a number of atypical populations of children whose neurodevelopment is not expected to follow the predicted rates and patterns of maturity to those in typical populations. While HIV infection, nor HIV-exposure is not technically a neurodevelopmental disorder because their etiology is largely related to effects of the virus, ART medications,

and a series of HIV-associated environmental influencers, it is helpful to consider HIV under the same umbrella term as there may exist common areas of impairment. To that end, a comprehensive understanding of the working memory profiles in HIV-infected (HIV-I) and HIV-exposed (HIV-EU) samples make a significant contribution to the existing knowledge of how working memory functions in atypical development.

There is a large body of evidence regarding the neurocognitive effects of both HIV-infection and ART in adult and paediatric samples. The findings generally agree that HIV infection is associated with a range of neuropsychological deficits, with working memory and motor impairment usually the first abilities to show impairment in the early stages of infection. However, this research does not offer a comprehensive investigation of how HIV impacts on the various components of working memory, particularly within paediatric samples where there is an age appropriate developmental fraction of the construct which has yet to be researched. There is equivocal support for the effects of ART, with most agreeing that the relative degree of ART-associated neurocognitive impairment is far smaller than the effects of not taking the drugs to prevent disease progression.

With respect to the HIV-EU population, there is limited research regarding their neurocognitive profiles after the initiation of formal schooling. There is equivocal support for an altered heath and developmental profile in younger HIV-EU children which suggest that working memory impairment in school beginners is not unlikely. However, this research is still in its infancy, and has a lot of inherent methodological limitations. More generally, research regarding the working memory performance in atypical samples is limited in its applicability to the large majority of children in the world who do not come from middle-class Western contexts, do not speak English as their first language, nor have a familiarity with conventional psychometric testing.

To that end, this cross-sectional study aimed to investigate the working memory profiles of the two HIV-affected samples (HIV-I, and HIV-EU). It did this by comparing working memory performance on three groups of children: HIV infected (HIV-I), HIV-Exposed, Uninfected (HIV-EU) and a control group of HIV uninfected, unexposed children (HIV-UU). It had two broad research aims which evolved from the limitations in the existing literature.

Firstly, it sought to determine whether there were significant differences between the three groups on two different psychometric measures of neurocognitive function: the Automated Working Memory Assessment (which was administered to all participants), as well as a selective battery of subtests from the Developmental Neuropsychological Assessment (NEPSY-II) (which was administered to a subgroup of 30 participants in each group). This was done in order to establish if there were between-group differences on either of the measures between the three groups. A within-group comparison of significant strengths and weaknesses was also undertaken within each of the groups to allow for a cross-group profiling pattern.

Secondly, it would also be helpful to compare the working memory performance of the three groups to the developmental expectations of typical children. In order to avoid relying on normative psychometric comparisons which would include a significant degree of measurement bias, the typicality of working memory structure was assessed using two factor analytic paradigms. The first, a multi-group confirmatory factor analysis identified whether the relative structure of working memory within each of the three groups was significantly different from one another. The second factor analysis was employed to determine whether the working memory profiles of the three groups developed according to theoretically expected phases and rates of development. It was hypothesised that the HIV-UU group would typical working memory development, and that the HIV-infected and HIV-EU groups would have atypical working memory development. This analysis of factor structures within each of the groups was believed to allow for a less biased conceptualisation of the construct within linguistically diverse, atypical samples. The following chapter documents the method employed to achieve this.

Chapter 4: Method

Aims

This cross-sectional study aimed to investigate the neurocognitive profiles of three groups of children: HIV infected (HIV-I), HIV-Exposed, Uninfected (HIV-EU) and a control group of HIV uninfected, unexposed children (HIV-UU). This method was based on the following two broad research aims:

1. To determine significant between-group differences, and the profile of within-group strengths and weaknesses of each the three groups on two different psychometric measures of neurocognitive function.
    a. Automated Working Memory Assessment (administered to all participants)
    b. Selective subtests of the Developmental Neuropsychological Assessment (NEPSY-II) (administered to a subgroup of 30 participants in each group)
2. To determine whether the working memory profiles of the three groups developed according to theoretically expected phases and rates of development. This was measured against the following two hypotheses:
    a. The HIV-UU group had typical working memory development.
    b. The HIV-infected and HIV-EU groups had atypical working memory development.

Design

The study fell within the positivist, quantitative, descriptive paradigm of knowledge production (Rosenthal & Rosnow, 2008). While it compared and correlated scores across three groups of children within the sample, there was no experimental manipulation of the independent variable (HIV status), and hence used a simple cross-sectional, ex post facto, non-experimental, correlational design.

A brief discussion regarding the distinction between a correlational and quasi-experimental design is of relevance here. A strictly correlational design is one with non-directional associations between two variables, where it is usually not possible to determine the variable responsible for the effect in the other. Within this study, a strictly correlational design would therefore imply that HIV status could affect working memory performance, and that working memory performance could affect HIV status. It is obvious that the latter implication is impossible, which suggests that the research is not correlational in a true sense.

The alternative to this would be a quasi-experimental design which attempts to investigate an inherent degree of causality. There are two ways in which this experimental causality could be achieved. The first would be the manipulation of an experimental condition which is impossible in this context. The second is through the random assignment of participants to the three levels of the independent variable. In the context of this study, the random assignment into groups was not possible because this was pre-determined at birth by factors largely uncontrollable by both the parent and the attending doctor. However, Rosenthal and Rosnow (2008) suggest that there are alternatives to that design, namely *non-equivalent group designs* where the researcher has no control over how subjects are assigned to their different groups, and a *catch-all-correlational design*, where the correlation in this case would refer to a retrospective co-variation of X, and then Y. This leads to conclusions being made on the basis of pseudo-causality, but which lacks the empirical rigour to make definite conclusions. Therefore, while the study is neither purely correlational, nor does it satisfy the requirements of the quasi-experimental approach, the strength of its internal validity is sourced from having elements of both of these proposed alternatives.

In addition to this, the operationalisation of the independent variable as 'HIV status' is simplistic. HIV cannot be referred to, or conceptualised simply as the presence or absence of exposure/infection to a virus. It would be short-sighted to assume that the effects and manifestations apparent in empirical studies are simply a consequence of viral infection, or exposure. The experience of HIV infection exists within a complex, highly diverse socio-cultural milieu where it is impossible to identify whether it is the virus, or the confounding and co-morbid issues of lack of employment, poor nutrition, and poor access to services that create the behavioural manifestations often linked to the virus. The interplay of these vectors with HIV status are nuanced and almost impossible to disentangle. These have been discussed already, but is important to keep in mind when considering the results of this study.

Participants

The initial intention was to include 80 children in each group to obtain moderate effect sizes (Soper, 2014), which would provide a total sample size of 240 (N = 80). After beginning the research, it became clear that it would be better to test to saturation within the HIV-I and

HIV-EU groups at the various sites to obtain a complete a data set as possible. To that end, data was collected as stipulated in Table 4.1.

Table 4.1
Total Number of Participants and Final Sample Numbers

	HIV-I	HIV-EU	HIV-UU	Total
Used	95	86	92	273
Unusable	18	6	4	28
Total Collected	113	92	96	301

Not all data collected was included in the final analyses (9.3%) and some had to be discarded due to experimenter error in collection (administering tests incorrectly), technological failures on computers (errors in saving results completely, power failures at the hospital), child fatigue or refusal to continue, early identification of child unsuitability to the assessment, and the failure of caregivers to return to the assessment procedure (same-day attrition). Caregivers were often very concerned about forfeiting their position in the queue for treatment to see their child's doctor, and preferred to get to the pharmacy before midday to avoid waiting for staff to finish their lunch break. These are the types of issues that further complicated the data collection process as it put pressure on both assessor and child to complete the assessment quickly. Despite the attrition, the final sample size yields excellent power ($P = 0.996$) when considering an average sample size of 90 across groups, and good effect sizes ($\Delta = 0.750, \alpha = 0.05$) (Friendly, 2014).

Selection criteria.

Children and their caregivers were invited to participate in the study if they satisfied the following criteria:

1. Were between the ages of 6-8 years old.

This was a contentious issue in the selection criteria during the phase of data collection. Initially, only children in Grade One were selected, with an average age of seven years old, while allowing for children who ranged between 6 and 8 years old at the time. However, it became clear that this would be limiting and biased on two fronts. Firstly, the insufficient number of children in Grade One meant that the power requirements for sufficiently large sample size number would not be satisfied. Further, only about two in every three Grade One's were appropriately placed in their grade for their age. Therefore, the age range was widened to include children who were between the ages of 6 and 8 years old, and use

scaled scores which were corrected statistically for age when comparing results. It was recognised that the difference in didactic stimulation and educational demand placed on two 8 year old children born on the same day, but who happen to be in either Grade One or Grade Three is very different, and exists as a limitation in the study. That being said, however, there is equally no guarantee that the relative quality of Grade 2 teaching at some schools is significantly better than that at another.

Further, there are practical reasons why grade choice is an insufficient indicator of inclusion into the study. The first of these refers to the recent overhaul of the public school admission system in 2010. According to the Department of Basic Education, children are now to start school in Grade 1 if they are 5, turning 6 by the 30 June of that year. Previously, national school admission policy had admitted children to Grade 1 in the year that they were turning 7. The large numbers of children incorrectly placed in their grade could be a function of poor administration and communication to the public about these changes (Department of Basic Education, 2014; South African Schools Act, 1996).

The second reason for not relying absolutely on grade as an inclusion criterion is based on the findings that grade allocation was not a clear indication of South African learners' literacy and numeracy abilities, and children were often found to be at least one to two grades behind their expected outcomes. The researchers argue that grade completion is not an indication that the required numeracy and literacy competencies have been attained, and that learner achievement is 'strikingly, abysmally low' (Spaull & Taylor, 2012, p.5). To that end age, instead of grade, was used as the marker for participant inclusion.

The particular age group of 6-8 years was chosen for three additional reasons. The first is that the most recent research on the effects of HIV Exposure have only assessed much younger children and measured less refined neurological functions (see Culnane, Fowler, Lee et al., 1999; Van Rie, Mupuala, & Dow, 2008; Whitehead, Potterton, & Coovadia, 2014; Williams, Marino, Malee, Brogly, Hughes, Mofenson et al., 2010). Further, research indicates that children's working memory functioning is used profusely at this age due to the rapidly growing acquisition of literacy and numeracy capabilities, and it is therefore a prime age for this to be assessed (Alloway, 2006; Henry, 2012; Palmer, 2000). Lastly, within the South African context, major PMTCT roll-out only happened in 2004, with effective horizontal dissemination to pregnant women happening at an even slower rate

(Ingle, May, Uebel, Timmerman et al., 2010; Johnson, 2012; Kagee, 2008; Mills, et al., 2006). Therefore, it is unlikely that there will be many HIV-EU children over the age of 8 years.

2. Children had to attend a mainstream school. The exclusion of children at schools catering for learners with specialised educational needs (LSEN) was necessary to guarantee a degree of control regarding a baseline level of typical cognitive functioning and learning potential within children of the specified age limit.
3. Children were included in the HIV-I (HIV-infected) group if they were HIV positive and were currently on ART. They also needed to have been virally suppressed for 6 months. This meant that they were adherent and responding well to their anti-retroviral medication, suffered no debilitating side effects from the medication, and had stable CD4 counts and viral loads. This was assessed by the Voluntary Counselling and Testing (VCT) counsellors who screened participants on a daily basis.
4. Children were included in the HIV-EU (HIV-Exposed, Uninfected) group if they were currently HIV negative, but their biological mother was HIV positive prior to giving birth to them.
5. Children were included in HIV-UU (HIV-Unexposed, Uninfected) if both they and their biological mother were HIV negative at the time of giving birth.

Exclusion criteria.

Studies of the neurological functioning of children in institutions suggests that their neurodevelopment may be different from typical children who have consistent caregivers because of the impact of attachment on the development of the brain (Schore, 2001; Schore, 1994). For this and other ethical reasons, children living in institutions were excluded from the study. However, many children in the study were not cared for by their parents but by grandparents, aunts and extended family, sometimes in the absence of being a single or double orphan. This speaks to a more general point about the social circumstances of HIV affected children, and the relative non-existence of the nuclear family structure within large sections of the South African population.

Similarly, children with any form of neurological compromise such as Epilepsy, Traumatic Brain Injury, or any recent contamination of Meningitis or Encephalitis were also excluded from the study in order to reduce the confounding effects of other neurological impairments on test performance. Children whose cognitive functioning was in the

borderline ranges of performance were also excluded from the study for similar reasons. If children attended schools for Learners with Special Educational Needs (LSEN), they were also excluded as this indicated a lower than average level of cognitive capacity. Further, children who had recently suffered a debilitating illness in the last six months which was believed to negatively affect their development and learning because of long term school absence or general lethargy for learning, were also excluded from participation in the study. Only two cases of recent Meningitis were identified and then excluded. All other children who met the inclusion criteria were found to be sufficiently healthy to participate.

A diagnosis of Attention Deficit/Hyperactivity Disorder (AD/HD) was not an identifying feature of exclusion in the study, but in hindsight should have been, as co-morbid attentional difficulties were often identified within the sample. While these cases were identified and referred to specialised psychiatric child services, the study has little guarantee that this screening was consistent across assessors as the research assistants were not all professionally qualified psychologists. Further, there could exist the possibility that the identified co-morbid attentional difficulties were a function of HIV infection or exposure itself, and were therefore inseparable from the HIV status (Misdrahi et al., 2004; Scaharko, 2006)

Sample demographic details.

This section describes the basic demographic details of the three groups of participants that were administered the primary working memory measure, the AWMA. A smaller subset of this sample was also administered to a selected battery of subtests from the Developmental Neuropsychological Assessment (NEPSY-II) to assess more general neuropsychological performance.

Descriptive demographic statistics of the AWMA sample.

Table 4.2 represents the nominal comparisons across the three groups, and Table 4.3 compares the continuous data of age, socioeconomic status, preschool attendance and class size. A between-group comparison of the salient demographic information suggests that there is a relatively equal distribution of gender across the three groups, with the HIV-UU group having slightly more female participants than male participants. There also appears to be particular differences between the groups in their home and school language preferences. None of the participants in the HIV-EU group are first language English speakers, while only a third of them attend English schools. Within HIV-I, just under a tenth

of the participants are first language English speakers, while a fifth are identified as such in HIV-UU. Similarly, about two thirds of participants from the HIV-I and HIV-UU groups attend English schools. The majority of children across all three groups were in Grade's One or Two, while only two thirds of the sample were appropriately placed in their grade for their age.

There are also salient between-group differences in the socio-economic demographic markers of the sample. The measurement of socioeconomic status (SES) is a contentious issue, and has compounded complexities within developing contexts characterised by widespread income and educational inequality such as South Africa. Within some contexts, parental education level is considered a moderately accurate measure of SES (Finkelstein, Kubzansky, Capitman, & Goodman, 2007). However, this marker becomes inaccurate in environments where there are high unemployment levels of school leavers (or people with that qualification) as a result of economic factors. There are also a series of demographic variables which are believed to covertly indicate the socioeconomic standing of a family relative to their ability to send their children to preschool, their access to social grants, the type of dwelling they live in, and whether they are obliged to look after a family member with a disability. Further, families were often not supported by a single breadwinner, but received proportions of support from multiple sources (social grants, pensions of older family members, extended family members, part-time work etc.), which made the accuracy of determining this construct challenging. In South Africa, the use of a nationally bench-marked quantitative score representing SES is becoming increasingly popular because of its relevance to the majority of the population. The LSM (Living Standard Measure) is the industry standard when considering consumer patterns in South Africa, and has been developed by the South African Advertising Research Forum (SAARF, 2012). The study employed all of these measures to gain a comprehensive understanding of SES, however only the LSM was used for analysis.

The large majority of children in the HIV-I and HIV-EU groups attended pre-school, while only two thirds in HIV-UU did so. The prevalence of a family member with special physical or mental needs was approximately one in ten in the HIV-I and HIV-UU groups, but doubled in the HIV-EU group. Special needs excluded common lifestyle chronic illnesses such as HIV, hypertension and diabetes. Similarly, only two thirds of participants in HIV-I and HIV-UU rely on the child support grant, while eighty percent of participants in HIV-EU are benefactors thereof. The majority of participants lived in houses, but far more participants

from the HIV-I and HIV-EU groups lived in shacks, than do from the HIV-UU group. Lastly, approximately one in ten participants from all groups did not have information regarding their mothers. In stark contrast, half of the children in the HIV-I and HIV-EU groups did not know anything about their fathers, while only a third in HIV-UU lacked this information. Further, the majority of mothers in the HIV-I and HIV-EU groups did not finish high school, while the majority in the HIV-UU group did. About a fifth of mothers from the HIV-I and HIV-UU groups also went on to complete a tertiary level qualification. In contrast, the majority of fathers from the HIV-I and HIV-UU groups did finish high school, and a fifth went on to complete tertiary study. The majority of fathers from HIV-EU did not finish high school, and none completed a tertiary education.

These results suggest that are two nodes of primary difference between the three groups – language and socioeconomic status. The HIV-EU group appears to have the least English language fluency in comparison to the other two groups, as noted by their school and home language indications. The HIV-UU group appears to have a better English language proficiency than the HIV-I group. Similar to this ordering, the HIV-EU group also appears to be particularly disadvantaged with the worst parental educational levels, and the highest levels of dependence on the child support grant. They also appear to have the highest burden of familial care (taking responsibility for a disabled family member), suggesting that the presence of a family member with special needs is more likely to increase dependence on alternate state-funded assistance like the child support grant. These socioeconomic differences are confirmed by the significant differences between Living Standards Measures ($p < 0.0001$, $\alpha = 0.05$), with significant differences between the HIV-I and HIV-EU group, and the HIV-EU group and the HIV-UU group.

It would also be amiss not to recognise that while issues of language and socioeconomic status might appear to be unrelated constructs, there is a large body of local literature to support a relationship between the two within the South African context owing to the historical legacy of Apartheid (Alexander, 2005; Taylor, 2014). The discrimination against politically black people under the previous regime has had long-standing economic consequences in post-Apartheid South Africa. Socioeconomic distinctions persist even twenty years into democracy, with the majority Coloured (mixed race) and black South Africans who bear the brunt of Apartheid's economic legacy and remain some of the poorest communities in the country. In contrast the white population are generally far

wealthier, and have access to stable incomes affording them a lifestyle similar to many in the first world. Within the white population, there is also a distinction between the one-third of English origin and the two-thirds of Afrikaner origin. Despite Apartheid policies explicitly designed to improve the lot of Afrikaners at the expense of other race groups, Afrikaners do not share income equality with English-speaking South-African-born Whites; although nearly equally well-educated, they tend to have lower incomes and are less likely to work at non-manual jobs and, specifically at jobs which earn a salary opposed to a wage (Treiman, 2005). The large majority of the Coloured community is also Afrikaans speaking, which makes research distinctions based on race, language and income inequality in the post-Apartheid context a challenge.

Descriptive demographic statistics of the NEPSY-II sample.

The following section describes the demographic statistics of the subsample of 30 matched trios to whom the NEPSY-II was administered. Generally, the sample is similar to that of the complete sample described above. Salient differences between them are evident in the skewed distribution of gender in the NEPSY-II sample which has a predominantly female subsample when compared to the more equal gender distribution in the larger set of participants. Despite there being no participants in the NEPSY-II sample who speak English as a first language, there are more of them who attend an English speaking school than the AWMA sample. This has implications for the ease of test administration and a degree of test-wiseness that participants possess. Lastly, it appears that both the HIV-I and HIV-EU groups from the AWMA response set have an increased burden of care (as indicated by their living standards, the presence of a disabled family member) which is not as salient in the smaller NEPSY-II subset.

Table 4.4. represents the nominal comparisons across the three groups, and Table 4.5 compares the continuous data of age and socioeconomic status. An eye-ball comparison of the three groups who were administered the NEPSY-II suggests that they are generally very similar. While all between-group trios were matched on age, gender and English as first or additional language, there remain stark differences between the three groups on other demographic variables. All participants spoke English as an additional language.

Table 4.2.
Frequency Tables for Nominal Variables of HIV-I, HIV-EU and HIV-UU (AWMA Sample) (N = 275)

	HIV-I (n = 95)			HIV-EU (n = 88)			HIV-UU (n = 92)		
	Frequency	Valid Percent	Missing Data	Frequency	Valid Percent	Missing Data	Frequency	Valid Percent	Missing Data
Gender			0 (0)			0 (0)			0 (0)
Male	46	48.4		41	46.59		37	40.22	
Female	49	51.6		47	53.41		55	59.78	
Home Language			0 (*0*)			2 (2.3)			3 (3.3)
English First Language	7	7.37		0	0		18	20.22	
English Additional Language	88	92.63		88	100		71	73.78	
Home Language			0 (0)			2 (2.3)			3 (3.3)
Afrikaans	5	5.3		2	2.33		15	16.85	
Arabic	0	0		2	2.33		6	6.74	
Bemba	0	0		0	0		1	1.12	
English	7	7.4		0	0		18	20.22	
French	1	1.1		2	2.33		1	1.12	
Ndebele	2	2.1		0	0		0	0	
Pedi	6	6.3		3	3.49		4	4.49	
Shona	1	1.1		2	2.33		0	0	
Sotho	7	7.4		12	13.95		8	8.99	
Tsonga	3	3.2		5	5.81		0	0	
Tswana	18	18.9		19	22.09		25	28.09	
Venda	2	2.1		2	2.33		2	2.25	
Xhosa	7	7.4		13	15.12		1	1.12	
Zulu	36	37.9		24	27.91		8	8.99	
School Language			0 (*0*)			3 (3.4)			3 (3.3)
English First Language	64	67.37		29	34.12		60	67.42	
English Additional Language	31	32.63		56	65.88		29	32.58	
School Language			0 (0)			3 (3.4)			3 (3.3)
Afrikaans	1	1.1		0	0		0	0	
English	64	67.4		29	34.12		4	4.49	
Pedi	4	4.2		5	5.88		60	67.42	
Shona	0	0		2	2.35		3	3.37	
Sotho	1	1.1		7	8.24		5	5.62	
Tsonga	0	0		4	4.71		0	0	
Tswana	11	11.6		11	12.94		13	14.61	
Xhosa	1	1.1		8	9.41		0	0	

Zulu	13	13.7		19	22.35		4	4.49	
Grade			0 (*0*)			2 (2.3)			4 (4.3)
0	1	1.1		8	9.3		7	7.95	
1	35	36.8		34	39.5		41	46.59	
2	40	42.1		43	50		34	38.64	
3	19	20		1	1.2		6	6.82	
Attended Pre-school			1 (1.1)			5 (5.7)			3 (3.3)
Yes	90	4.3		80	96.39		66	74.16	
No	4	95.7		3	3.61		23	25.84	
Special Needs Family Member			1 (1.1)			6 (6.8)			3 (3.3)
Yes	10	10.5		18	21.95		10	11.24	
No	84	88.4		64	78.05		79	88.76	
Type of Dwelling			0 (0)			2 (2.3)			3 (3.3)
Flat	15	15.8		6	6.98		15	16.85	
House	61	64.2		58	67.44		67	75.28	
Room	1	1.1		1	1.16		0	0	
Shack	17	17.9		20	23.26		7	7.87	
Shelter	1	1.1		1	1.16		0	0	
Receive Child Support Grant			0 (0)			2 (2.3)			5 (5.4)
Yes	64	67.4		79	91.9		66	75.86	
No	31	32.6		7	8.1		21	24.14	
Age Appropriate for Grade			0 (0)			2 (2.3)			3 (3.3)
Yes	67	70.5		49	57		51	57.3	
No	28	29.5		37	43		38	42.7	
Maternal Education			15 (15.8)			10 (11.4)			12 (13)
No schooling	0	0		0	0		0	0	
Primary School not completed	5	6.3		5	6.4		0	0	
Only Primary School Completed	8	10		3	3.8		1	1.25	
Secondary school not completed	31	38.8		52	66.7		23	28.75	
Secondary school completed	21	26.3		17	21.8		37	46.25	
Tertiary Education completed	15	18.8		1	1.3		19	23.75	
Paternal Education			48 (50.5)			47 (53.4)			33 (35.9)
No schooling	1	2.1		1	2.4		0	0	
Primary School not completed	0	0		2	4.9		0	0	
Only Primary School Completed	4	8.5		3	7.3		3	5.08	
Secondary school not completed	13	27.7		20	48.8		15	25.42	
Secondary school completed	16	34		15	36.6		24	40.68	
Tertiary Education completed	13	27.7		0	0		17	28.81	

Note. Sample size for the HIV-EU group was calculated at n = 88 for demographic purposes. This was reduced to n = 86 when data affecting between- and within-group analyses (covariates) were used, due to unsuitable and missing test scores

Table 4.3.

Descriptive Demographic Statistics for the AWMA Sample

	HIV-I (n = 95)			HIV-EU (n = 86)			HIV-UU (n = 92)		
	Mean	Std. Dev.	Range	Mean	Std. Dev.	Range	Mean	Std. Dev.	Range
Age in months	88.98	10.15	71-107	88.28	10.51	67-106	84.54	10.35	60-106
No. years in preschool	2.37	1.48	0-6	2.51	1.62	0-7	2.15	1.52	0-6
Class size	31.96	9.58	11-50	35.42	10.78	5-81	33.08	9.99	15-76
LSM	6.55	1.77	10-Feb	5.88	1.13	2-10	6.95	1.65	2-10

The HIV-UU group shows the only noticeable language deviation, with 30% speaking Afrikaans at home. The HIV-EU appears to have the least attendance at English schools (37%), while HIV-I group (47%) and HIV-UU group (73%) have increased proportional attendance. Only half of the participants in the HIV-I and HIV-EU groups appear to be appropriately placed in their grade for their age, while 86% of HIV-UU are appropriately placed.

With regards to socio-economic markers, the HIV-UU group also appears to be the least dependent on the child support grant. The majority of mothers from the HIV-UU group had finished school, while only one in five mothers from the other two groups had done so. The majority of fathers from the HIV-UU group had also finished school, while only 13% and 16% from the HIV-I and HIV-EU groups respectively had done so. There was also a noticeable difference in the availability of fathers between the groups, with 73.3% of participants in the HIV-I group having no contact with their fathers. Generally, this data suggests that the HIV-UU group appears to be economically advantaged when compared to the other three groups. These differences are however not confirmed by an analysis of variance of the Living Standards Measure ($p = 0.065$, $\alpha = 0.05$).

Table 4.5

Comparative Descriptive Demographic Statistics (NEPSY-II Sub-sample)

	HIV-I			HIV-EU			HIV-UU		
	Mean (*SD*)	Min	Max	Mean (*SD*)	Min	Max	Mean (*SD*)	Min	Max
LSM_{raw}	9.52 (5.04)	2	20	11.20 (4.45)	4	21	12.48 (5.04)	4	22
Age at Assessment	87.03 (10.64)	72	103	87.62 (10.41)	72	104	87.48 (10.29)	71	102

Table 4.4
Demographic Comparison of NEPSY-II Sample

	HIV-I (N = 30)			HIV-EU (N = 30)			HIV-UU (N = 30)		
	Frequency	Valid %	Missing (%)	Frequency	Valid %	Missing (%)	Frequency	Valid %	Missing (%)
Gender			0 (0)			0 (0)			0 (0)
Male	10	33.3		10	33.3		10	33.3	
Female	20	66.7		20	66.7		20	66.7	
Home Language			0 (0)			0 (0)			0 (0)
Afrikaans	0	0.00		0	0.00		9	30.00	
Arabic	0	0.00		3	10.00		5	16.67	
Bemba	0	0.00		0	0.00		1	3.33	
French	1	3.33		1	3.33		0	0.00	
Ndebele	0	0.00		0	0.00		0	0.00	
Pedi	4	13.33		1	3.33		2	6.67	
Shona	0	0.00		0	0.00		0	0.00	
Sotho	3	10.00		5	16.67		5	16.67	
Tsonga	0	0.00		2	6.67		0	0.00	
Tswana	7	23.33		0	0.00		7	23.33	
Venda	0	0.00		1	3.33		0	0.00	
Xhosa	6	20.00		4	13.33		0	0.00	
Zulu	9	30.00		11	36.67		1	3.33	
School Language			0 (0)			0 (0)			0 (0)
English	14	46.67		11	36.67		22	73.33	
Additional Language	16	53.33		19	63.33		8	26.67	
School Language			0 (0)			0 (0)			0 (0)
Afrikaans	0	0.00		0	0.00		2	6.67	
Arabic	0	0.00		0	0.00		0	0.00	
English	14	46.67		11	36.67		22	73.33	
French	0	0.00		0	0.00		0	0.00	
Ndebele	0	0.00		0	0.00		0	0.00	
Pedi	4	13.33		2	6.67		1	3.33	
Shona	0	0.00		0	0.00		0	0.00	
Sotho	3	10.00		3	10.00		2	6.67	
Tsonga	0	0.00		2	6.67		0	0.00	
Tswana	5	16.67		0	0.00		3	10.00	
Venda	0	0.00		1	3.33		0	0.00	
Xhosa	1	3.33		4	13.33		0	0.00	
Zulu	3	10.00		7	23.33		0	0.00	
Grade			0 (0)			0 (0)			0 (0)

0	1	3.33		2	6.67		2	6.67
1	11	36.67		14	46.67		14	46.67
2	16	53.33		12	40.00		12	40.00
3	2	6.67		2	6.67		2	6.67
Attended Pre-school			0 (0)			0 (0)		0 (0)
Yes	27	90.00		28	93.33		27	90.00
No	3	10.00		2	6.67		3	10.00
Special Needs Family Member			0 (0)			0 (0)		0 (0)
Yes	1	3.33		3	10.00		5	16.67
No	29	96.67		27	90.00		25	83.33
Type of Dwelling			0 (0)			0 (0)		0 ()
Flat	2	6.67		2	6.67		3	10.00
House	22	73.33		20	66.67		23	76.67
Room	1	3.33		1	3.33		0	0.00
Shack	4	13.33		7	23.33		4	13.33
Shelter	1	3.33		0	0.00		0	0.00
Receive Child Support Grant			0 (0)			0 (0)		0 (0)
Yes	22	73.33		26	86.67		20	66.67
No	8	26.67		4	13.33		10	33.33
Age Appropriate for Grade			0 (0)			0 (0)		0 (0)
Yes	17	56.67		16	53.33		26	86.67
No	13	43.33		14	46.67		4	13.33
Maternal Education			7 (23.33)			1 (3.33)		2 (6.67)
No Schooling	0	0.00		0	0.00		0	0.00
Primary School Completed	0	0.00		2	6.67		0	0.00
Only Primary School Completed	3	10.00		2	6.67		0	0.00
Secondary School Not Completed	13	43.33		17	56.67		9	30.00
Only Secondary School Completed	6	20.00		8	26.67		17	56.67
Tertiary Education Completed	1	3.33		0	0.00		2	6.67
Paternal Education			22 (73.3)			11 (36.67)		8 (26.67)
No Schooling	0	0.00		0	0.00		0	0.00
Primary School Completed	0	0.00		1	3.33		0	0.00
Only Primary School Completed	2	6.67		1	3.33		0	0.00
Secondary School Not Completed	1	3.33		10	33.33		5	16.67
Only Secondary School Completed	4	13.33		5	16.67		15	50.00
Tertiary Education Completed	1	3.33		0	0.00		2	6.67

Sampling Procedure

The study employed a non-probability haphazard/convenience sampling strategy and invited interested parties to voluntarily participate in the study if they wished to do so (Rosenthal & Rosnow, 2008). The identification of participants happened primarily at a paediatric ARV clinic in a Johannesburg government hospital, but also included an adult public tertiary hospital and a number of primary health care facilities in the surrounding areas. These institutions are all free public service hospitals and primary health clinics which serve poorer communities. The three sub-samples were taken from across this catchment region. Historically, the patients of these institutions tend to be poor people with limited access to resources.

The sampling procedure also differed by necessity across the groups, and implicitly introduced particular biases within the group allocations which could have executed an effect on the results. The HIV-I group were the initial group to be identified and invited to participate, as they were easiest to gain access to. Voluntary Counselling and Testing (VCT) counsellors at the paediatric ARV clinic were briefed and educated on the inclusion and exclusion criteria and screened for patients in their daily work. Gaining staff cooperation was a challenge to the study, and it took a while for this process to gain sufficient momentum. Three staff members collected information of just under 150 potential participants in HIV-I over a period of about 18 months. If the child was already at the clinic for their three-monthly check-up with the doctors, they were invited to participate, and were assessed prior to their appointment while their parent or caregiver waited in the queue. In this case, and on the instruction of the Head of Paediatrics at the hospital, participants were *not* provided with the R150 stipend to compensate them for their travel expenses in order to prevent ethical issues for staff and patients in future visits. If children were called to see the doctor during the assessment, it was paused and was re-commenced when their appointment was over. If caregivers and their children were willing to participate, but unable to be tested on the day of their clinic appointment, they were booked in for an appointment on a suitable day and were then compensated with R150 each. All children (including accompanying siblings) were given a pack of refreshments (crisps and a juice) following their assessment. Parents were also offered verbal feedback describing their child's working memory profile and advised as to how it could be improved. An explanatory brochure was provided as well (See Appendix A).

The identification of the HIV-EU participants took longer, and was also done by trained VCT counsellors and nursing staff in their adherence sessions with patients prior to them having their vitals checked, and then waiting to see their doctor. At the paediatric unit, staff approached parents of children who already had infected children attending the clinic, and asked whether they had other children who were exposed, but not infected. The reliability of their blood results was confirmed by these women in participants' files and they were invited to participate. This was a very successful approach and accounted for more than half of the eventual 92 HIV-EU participants. A similar procedure was put in place by nursing staff at an adult ARV clinic, who were also able to screen for HIV-EU participants. This also worked very well, as the majority of these parents had given birth at the aforementioned paediatric hospital, were familiar with the ARV unit there, and were happy to come to the hospital for their appointment. There were a few (less than 5) cases of HIV-EU participants identified by nursing staff in various departments across the hospital who were familiar with the study.

Participants in the HIV-UU were initially identified and screened by nursing staff in the Paediatric Out Patient Department. This was later discontinued due to staff resource limitations, and participants were then identified by VCT counsellors doing testing on the wards, and by members of the psychology and psychiatry department who could see by patient's files that they were eligible for invitation to the study.

Administration Procedure

The head nursing sister of the paediatric ARV unit identified three VCT counsellors and nursing staff and allocated them to the study. These three women were trained on the screening procedure and given material with which to recruit possible participants An initial pilot study was conducted with eight HIV-I participants to determine the suitability of the testing procedure to the sample. Their data was not included in the final results and there were two salient lessons that were included in subsequent administration. Firstly, even after translation, participants struggled to understand the instructions to the Backwards Recall, Mr X and Spatial Recall subtests of the AWMA. To assist this, children were offered a piece of paper that showed the numbers used in the trial/teaching items so that the assessor could point to numbers as they were taught forwards and then backwards. Small laminated pictures of the teaching items in the other two subtests were also provided to show

participants the rotations necessary to conceptualise the items (Appendix B). While this proved helpful for many children in the HIV-I group, and some in the HIV-EU group, it was evident that very few participants in the HIV-UU group needed this teaching aid. This could have been due to a greater degree of English language proficiency in the control group, and/or an increased familiarity with new scholastic material, but could also reflect a cognitive advantage where they simply learnt new material with greater ease. This should have been accounted for in the initial pilot study children from all three groups should have been part of the pilot, and not assumed that administration would be the same across the striated sample.

Over the two year period of data collection, four research assistants worked periodically to assist with data collection. All of these women were post-graduate Psychology students who had been extensively trained in psychometric test administration. They were all trained on the material, and then supervised by the author to ensure reliability and uniformity across assessors. There was also a standardised testing procedure that we all followed to ensure consistency (refer to Appendix C).

As far as possible, the instructions to the subtests were translated into the home language of the child being tested. During the pilot study, this was done by the child's accompanying caregiver, or an available VCT counsellor or nurse. However, there were two difficulties were raised during this process. The first was that it became apparent from listening to their vernacular translations that caregivers did not always fully understand the instructions themselves, and were therefore not adequately explaining what needed to be done. When these parents were given translated written instructions to the subtests, they were unable to read them. In order to avoid embarrassment in front of their children, this strategy was subsequently abandoned. Alternatively, in a few instances, caregivers would prime or prompt their children to give the correct answers, or hit or shame them when they got items incorrect. Consequently, the study employed a translator who could speak many of the Nguni and Bantu languages common to South Africa. She was a trained VCT counsellor herself, worked in the North Western regions of Soweto as a home based carer, and ran maternal HIV support groups from the clinic on a weekly basis.

A subgroup of 30 HIV-EU participants were recruited and invited to participate in subsequent testing on the NEPSY-II. These children were then matched to children from the

HIV-I and HIV-UU databases and a further 60 children from these two groups were also tested. Matching was done to within 1.5 months of the ages of the participants HIV-EU group, gender and English as first or second language. There were in fact no trio's who spoke English as a first language, and one of the trio's data became unusable after scoring because of an incompatibility with the age thresholds within the scoring manual.

In order to make comparisons between a reasonably homogenous set of children, there were 30 groups of matched trios across the sample, who were matched on age (born within three months of each other), gender and whether they spoke English as an additional language or not. A table comparing this information can be found in Appendix D.

Ethical Considerations

Ethical clearance was obtained by the Medical Research Ethics Committee (Human Subjects) of the University of the Witwatersrand (protocol number: M120902). Approval for the study was obtained through the relevant public hospitals (see Appendix E). In addition, informed consent was obtained from caregivers prior to commencing assessment, and written assent was sought from the child (Appendix F). In the event that the caregiver could not read or understand English, a translator was used. In the absence of a translator, an available nursing sister or VCT counsellor assisted in ensuring consent was informed and voluntary. Each participant was given an information sheet detailing the intention of the study. Confidentiality of results was assured, but due to the individual nature of psychometric testing, anonymity was not possible. Parents were offered a brief verbal summary of their child's profile of strengths and weaknesses after the assessment, and were also provided with a pamphlet as to how their child's working memory skills could be improved and practiced at home (Appendix A). Children and their caregivers were also referred to auxiliary (usually allied) services when this was deemed appropriate.

There were two ethical issues that permeated the data collection process. The first was the disclosure of the mother's (and sometimes child's) status to the child. The assessors were always very careful to ensure that this was not done, and children were asked to leave the room when this was spoken about. It was evident that very few children knew their own status or that of their parents, and many parents said that they would address this with their children in adolescence. Professionals at the hospital suggest that disclosure is done by the parent and attending medical professional to the child when they are about 8 years old.

The second ethically contentious issue was the travel stipend of R150 stipulated by the Medical Ethics Board. The reimbursement of travel expenses for research participation is a consistent problem when conducting research within low SES communities. There needs to be a balance between remunerating participants for their time and cost of travel, and the influence that this stipend has on participation.

Instruments

Automated Working Memory Assessment.

This study's methodological approach was formulated around Baddeley and Hitch's (1974) theoretical model of working memory. Hence, the Automated Working Memory Assessment (AWMA) (Alloway, 2007) was chosen as an appropriate assessment tool. It is an individual, computer based assessment of working memory that provides a practical and convenient screen for significant working memory problems from early childhood (4 years) to adulthood (22 years). The long form of the test consists of 12 subtests that combine to form four measures of the different parts of working memory. The test has a pre-determined sequence, is automatically scored by its internal software, and a brief summative report of the individual child's performance is generated to highlight the relative strengths and weaknesses. The test was felt to be appropriate because it is relatively quick to administer, is a standardised test battery which means that the scores follow the same pattern as that of current IQ testing *(M* = 100; *SD* = 15), and is highly effective at identifying students at risk (Alloway, 2011). It also appears to be relatively impervious to environmental influence such as the quality of social and intellectual stimulation in the home, the number of years spent in preschool (Alloway, 2011) and financial background (Engel, Dos Santos, & Gathercole, 2008).

The AWMA is divided into four indices of performance, with three subtests in each. The child is given three practice items to learn what is required before beginning. Each level offers six attempts, four of which have to be correct to proceed to the next level. Each level increases in difficulty with an added length to the item. A brief summary of what is required of the child follows.

Verbal Short Term Memory.

Digit Recall: The child hears a sequence of numbers and has to recall each sequence in the correct order.

Word Recall: The child hears a series of words and has to recall the words in the correct order.

Non-Word Recall: The child hears a series of non-words and has to recall the sequence of non-words in order.

Visuospatial Short Term Memory.

Dot Matrix: The child is presented with a series of 4 x 4 matrices in which a red dot moves around. The child is required to tap the screen to indicate where the dot has been in the correct order. Each dot is held on the screen for 2 seconds.

Mazes: The child is shown a maze with a red path shown on it for 3 seconds. The child has to trace on the screen where they remember the red path being.

Block Recall: The child is shown a video of a series of blocks being tapped. The child has to reproduce the sequence of tapping the blocks in the correct order after the video has been stopped.

Verbal Working Memory.

Listening Recall: The child is presented with a series of spoken sentences. The child has to verify the veracity of the sentence by stating if it is 'true' or 'false', and then recall the last word in that sentence. The number of sentences within each item increases, and words are recalled after all veracity decisions are made. The sentences were translated into the child's home language. Children used this to understand the meaning of the sentence, but still preferred to report the last word of each sentence in English.

Counting Recall: The child is presented with a number of randomly placed red circles and blue triangles. They are required to count the number of red circles, and then tally up the total.

Backwards Recall: The child is required to recall a sequence of spoken digits in the reverse order.

Visuospatial Working Memory.

Odd One Out: This task is made up of two parts. In the first, the child is presented with a series of three shapes, and has to identify which one of the three is different to the rest (i.e. a circle and two triangles). In the second part, they have to remember the serial order of

where the odd shape was located on the screen. Memory for location is only allowed after all odd shapes have been identified, and the sequence of order is important.

Mr X: This task is also made up of two parts. In the first part, a child is shown a picture of two Mr X's (a fictitious, but likable cartoon man). The child is required to identify whether Mr X with the blue hat is holding the red ball with the same hand as the Mr X with the yellow hat. The Mr X with the blue hat is frequently rotated around four axes. At the end of each trial, the child is required to recall the location of the red ball in sequence by pointing to a picture with eight static compass points.

Spatial Recall: In this task, the child initially views a picture of two arbitrary shapes where the shape on the right has a red dot on it. The child has to identify whether the shape on the right is facing the same direction as the shape in the left. The shape on the right is also frequently rotated along three axes (similar to that in the Mr X task). At the end of each trial, the child has to recall the location of each red dot, in sequence, by pointing to a picture with three compass points.

The AWMA has not been standardised on a South African population, and the scoring procedure draws on British norms to produce scaled scores. An attempt to compensate for this has been done by including age as a covariate in the study. It was not possible to rely on raw scores, as sound across-subject comparison requires that all participants answer the same number of items within each subtest. The AWMA is designed to discontinue a subtest after three consecutive incorrect responses, which would differ across participants. However, this was not seen as a major limitation since comparison with the UK sample was made redundant as the analyses only made between-group comparisons with no reference to normative data. The AWMA also relies on computer-based testing, and the English instructions are spoken by a British female avatar. This was unlikely to influence performance as the instructions were translated into the child's vernacular language, but familiarity with mode of administration, test format and the understanding of instructions can affect test performance in cross-cultural contexts (Foxcroft, 2004).

The test reliability of the AWMA is reported in Alloway et al., (2006), and test validity in Alloway et al. (2008). Test re-test reliability was assessed on a sample of 128 British individuals (M = 10.4 years, SD = 5 years), and the correlational coefficients ranged between 0.69 and 0.89 suggesting sound accuracy and consistency between the four week period

across the sample. The validity of the working measures of the AWMA (Backwards Digit Recall and Listening Recall) was measured against performance on the Working Memory Index of the Wechsler Intelligence Scale for Children Fourth UK Edition (WISC-IV). Three quarters of children identified as having poor working memory by the AWMA also obtained standard scores of 85 or less in the WISC Working Memory Index (Alloway, 2007).

Non Verbal Intelligence: Raven's Coloured Progressive Matrices.

The Ravens Coloured Progressive Matrices (RCPM) (Raven, Raven, & Court, 1998) was used to obtain a standard measure of general cognitive functioning, and to control for potential cognitive differences between the groups. This is used as it is assumed to be a relatively culture fair, non-verbal means of assessing an inherent '*g*'. The Raven's CPM measures logical problem solving ability and is designed for young children ages 5:0-11:0 years and older adults. The test consists of 36 items in 3 sets (A, Ab, B), with 12 items per set. The Raven's CPM produces a single raw score that can be converted to a percentile based on normative data collected from various groups (Raven, Raven, & Court, 1998).

The retest reliability of the RCPM when normed on children from the United States has been found to be 0.90 over the whole range of development (Raven et al., 1990). A split-half reliability estimate of 0.90 was found with no gender or ethnicity differences reaching significance (Jensen, 1974 as cited by Raven et al. 1990). In a subsequent study by Carlson and Jensen (1981), the split -half reliability estimate of 0.85 was established; with the estimates at ages 6, 7 and 8 generating estimates of 0.65, 0.86 and 0.85 respectively. There has however, been some evidence that when the test is administered to very young children, the CPM generates lower reliability estimates (Sattler, 1982). Valencia (1984) explored the test's reliability for Anglo and Mexican-American schoolchildren in grade 3 and found it to be acceptably high and equal for both cultural groups.

Validity studies between the RCPM and the WISC reveal correlations of 0.91, 0.84 and 0.83 between the Full Scale, Verbal and Performance IQ's respectively (Martin & Wiechers, 1954). Further, highest subtest correlations were found with the Block Design (a processing or fluid measure) subtest (0.74) and lowest with the Information subtest of the WISC (a crystallised or experience-based measure) (0.47), suggesting that the test is a culturally reduced measure of non-verbalised abstract thinking (Martin & Wiechers, 1954).

Sentence Repetition Test.

The Sentence Repetition Test (Redmond, 2005) is a very brief measure of English language proficiency included in order to control for differences between the groups. It includes sixteen 10-word sentences that are each between 10 and 14 syllables long, with an even number of active and passive sentences. These particular parameters of stimuli length and sentence types were chosen to ensure that sufficient errors in a typically developing group would be produced in order to permit group comparison. Inter-rater reliability was calculated by independent comparisons of marked responses (number of agreements/number of agreements + number of disagreements), and a value of 95% (sentence recall probe) and 98% (past tense elicitation probe) were found. Children are required to recall and repeat the sentences exactly and are given a percentage score based on their performance (either a 0, 1 or 2) (Redmond, 2005). While the SRT was originally developed as a cross-linguistic screen for children with Specific Language Impairment, subsequent use and research of the test has been found to be particularly sensitive to English proficiency in children who are not first language English speakers (Komeili, Marshall, 2013; Komeili, Marshal,l & Chait, 2013).

Recall probes likes that of the Sentence Repetition Test have also been used to measure the functioning of the episodic buffer component of working memory (Alloway, et al., 2004). This is because it integrates information from short term memory with existing long-term language processing systems. Typically, the test is believed to be able to differentiate between short term memory deficits (related to working memory), and limited language knowledge (as a proxy for English proficiency in this study). The former is identified by the presence of word order errors where the meaning remains intact, while limited language proficiency is implicated if the meaning of the sentence response is altered, or if the participants produces a grammatically or semantically incorrect sentence in response (Vance, 2008).

In the current study, the score from the SRT was used as a covariate to attempt to control for language proficiency differences between the groups. While sentence recall probe tasks such as the SRT have also been used to measure the functioning of the episodic buffer in previous working memory research with child samples (Alloway et al., 2004; Jordaan, 2012), it was not employed in this manner in the current study as it had already

been used as a measure for English proficiency within a multilingual sample. However, it's exclusive use as a covariate does not preclude it from also measuring abilities of the episodic buffer and phonological loop. To that end, the SRT's reliance on verbal short term memory could have conflated the purity of its assessment of language proficiency in the two clinical sub-samples in this study because of the effect of HIV status on memory. This remains an unavoidable limitation in using this type of measure, particularly in multilingual samples.

Demographic Questionnaire.

A demographic questionnaire was administered to all participants, and assisted in the identification of confounding variables that could affect the data. These include home language, language of educational instruction, access to pre-school education, parental education level and occupation, as well as quantitative assessment of the quality of living amenities – all of which contribute to a child's cognitive development (See Appendix F) (Tinajero & Loizillon, 2010). It was not possible to control for all of these factors across the three samples, but the quantifiable ones were included in an assessment of covariance to identify and then attempt to compensate for their extraneous influence.

The Developmental Neuropsychological Assessment-II (NEPSY-II).

The NEPSY-II (Korkman, Kirk, & Kemp, 2007b) was administered to a smaller sub-set of participants in each sample in an attempt to identify between-group differences in a broader spectrum of neurocognitive functions. The NEPSY batteries are based on the theoretical foundations of Luria where he assumes that designated brain functions correspond with selected assessment tasks (Korkman, Kirk, & Kemp, 2007b). The subtests are divided into six domains of performance that cover a broad range of functioning; namely Attention and Executive Functioning, Language, Memory and Learning, Sensorimotor Abilities, Social Perception and Visuospatial Processing. The creators of the measure stress that these domains are not empirically derived, or even statistically independent. Their intent is to focus the choice of the administrator to select appropriate tests for whatever they suspect needs to be assessed. This has implications for this study, as the NEPSY-II does not provide total domain scores. For the purposes of research, the corresponding subtests were collapsed within each domain to create a score. This was following previous research done in other areas of atypical development or psychopathology by Calderoni, Muratori,

Leggero, et al., 2013; Lind, Haataja, Rautava et al., 2010; Warren Foss-Feig, Malesa et al., 2012. The formulae and explanation are available in Appendix G. The creation of domain scores inherently reify cognitive constructs, and reduce the detail with which psychometric assessment can pinpoint strength and weakness within clinical settings. However, within research settings, this if often offset against their provision of the means to assess large groups of samples on a set of constellated skills.

While every effort was made to administer all the subtests of the NEPSY-II, not all subtests were administered. The test was piloted on five children in the unit, and the following were excluded:

Table 4.6
Subtests Excluded from NEPSY-II Administration

Subtest	Reason for Exclusion after Piloting
Animal Sorting	Relied heavily on language to explain their answers; struggled to translate the instructions adequately for the children to understand what to do. Many could not grasp the instruction.
Clocks	When asked to tell the time, children would often ask to show me on their caregivers' cell-phone. They are very unaware of analogue time, and it was felt that assessment of this was unfair as they might not have been taught it.
List Memory & Delayed	Heavy reliance on English understanding; could not guarantee the same word frequency in languages after translation.
Memory for Designs & Delayed	Performance was consistently very poor. This subtest was actually administered, but because performance on this was so poor, it was discontinued as it was believed that a cultural unfamiliarity with these types of tasks was behind the poor visual memory across the sample.
Narrative Memory	Heavy reliance on English understanding; could not guarantee the same word frequency in languages after translation.
Phonological Processing	Heavy reliance on English understanding and phonetic building. Phonemes seem an unfair was to test language in children who do not speak it as a first language.
Theory of Mind	Affect recognition component was adequate, but the questions assessing idioms and metaphors had a strong linguistic and cultural bias which would be unfair to administer.
Word List Interference	Strong reliance on language, and even after translation, it would be unclear whether the subtest was assessing a degree of English proficiency or actual verbal memory.

The NEPSY-II battery was standardised on 1200 American children from 3 to 16 years of age, with only 50 children in each age band. The test shows good reliability, where most subtests achieved moderate to high internal consistency estimates. There was also good inter-rater reliability results for Clocks, Design Copying, Memory for Names, Theory of Mind, Word Generation, Visual Memory Delayed and Visual Motor precision, ranging from 0.93 to 0.99. Test-retest reliability estimates across seven of the striated age groups showed little change in scores (*M = 21* days). Notably, the reliability of subtests was relatively stable for both typical and atypical samples which has particular relevance for the clinical samples that this study considers.

The development of the subtests of the NEPSY-II are based on strong theoretical and evidence based foundations. However, the lack of domain scores makes it difficult to assess the NEPSY-II's construct validity. There does appear to be sound domain specific inter-correlation between the subtests, which supports the test multitrait-multimethod model in both normative and clinical samples. A series of confirmatory factor analyses for ages 5-12 were not supportive of the tests' six-factor domain structure, and preferred a 'loosely fitting' four-factor model, which included Sensorimotor Abilities, Visuomotor Abilities, Language, and Memory and Learning (excluded the Attention and Executive Functioning and Social Perception domains). With regards to its criterion validity, the creators rely heavily on concurrent validity data. The NEPSY-II's correlations with other intellectual batteries (WISC-IV, DAS) are moderate to strong, as are it's correlations with achievement batteries (WIAT-II). The correlations between the NEPSY-II and more specific neurocognitive batteries are also moderate to strong (DKEFS, CMS and BBCS). The NEPSY-II creators also employed ten special atypical group studies which revealed moderate relationships with other diagnostic tests (i.e. the Devereux Scales of Mental Disorder show specific relationships with Autism (Comprehension of Instructions) and Conduct Disorder (Affect Recognition). There was however, no subgroup for children with HIV.

Overview of Data Analysis

All analyses were conducted using SPSS 22.0. Data was initially cleaned and descriptive statistics, frequencies and an exploration of normality were conducted. In the event of non-normal data, a series of transformations were conducted in an attempt to normalise data. An explanation of the relative success of these calculations and their practical utility within

the analysis follows in Chapter 5. Missing data was found in less than 3% of the 4692 observed scores due to equipment malfunction, experimenter error or participant withdrawal from the study. In these cases, scores were manually imputed to represent the mean of that particular index in order to satisfy the missing data requirements of SPSS.

Between-group difference on the Automated Working Memory Assessment (AWMA).

A primary aim was to determine whether there were significant differences between the groups on the four indices measures of the AWMA. In order to identify possible covariates for the analysis, a series of parametric analyses of variance (ANOVAs) were conducted on the proxy variables for English proficiency, non-verbal intelligence and socio-economic status, as well as age at assessment. Next, a multivariate analysis of covariance (MANCOVA) was conducted with age, SES (measured by LSM_{raw}), English proficiency (measured by SRT) and non-verbal intelligence (measured by RCPM) as covariates. Post-hoc ANOVA's and then t-tests identified where these specific differences lay. A within-group profile of working memory performance within each of the three groups was also created using a series of repeated measures ANOVA. Descriptive statistics and correlations was also used to examine more detailed patterns within each of the three sub-samples.

Neurocognitive profiles on the Developmental Neuropsychological Assessment-II (NEPSY-II).

The NEPSY-II was administered to a subgroup of each of the three larger research samples (29 trios). Covariates were identified in the same way as for the AWMA database, and identified as English proficiency (measured by SRT_{BoxCox}) and non-verbal intelligence (measured by the RCPM). Next a multivariate analysis of covariance was conducted on the six created domain scores. Further post-hoc analysis by way of ANOVA's and t-tests were later conducted to identify the pairwise location of these differences. A within-group profile of general neuropsychological performance within each of the three groups was also created using a series of repeated measures ANOVA. Descriptive statistics and correlations was also used to examine more detailed patterns within each of the three sub-samples.

Confirmatory factor analysis.

Factor analysis is a statistical tool which employs the matrix of correlations or covariances among measured observed variables (i.e., items) to identify a set of more general unobserved latent constructs (i.e., factors) that explain the covariances among the

measured variables. These latent constructs are created by the responses on the items of a measure or scale. When these items are measuring the same latent construct, their shared variance is manifested by their common factor (e.g., the latent modular structure of the multicomponent model in adulthood) (Varni, Limbers, & Newman, 2009).

The use of confirmatory factor analysis was used to determine whether working memory development within the three sub-samples was progressing within expected age ranges. This is an unusual tool to employ for this purpose. Ordinarily, an analysis of variance of the deviations of scaled scores from standardised normed population estimates would suffice. However, the psychometric use of scaled scores for this purpose within a linguistically diverse, and non-Western sample is coloured by issues of cultural and linguistic bias. Instead, the CFA method in this study draws on previous research regarding the structure of working memory, and hypothesises that because typical working memory functioning of school beginners is usually supported by a four factor model (of a domain specific fractionation of central executive, and two domain-specific slave systems for visuospatial and verbal representation), typical development of the construct within these samples should follow suit. Conversely, if this working memory model does not fit within the samples, it implies a degree of atypical development.

Confirmatory factor analysis (CFA) was employed to determine the degree of typical working memory development in two ways. Initially a multi-group CFA was used to compare model fit between the groups according to the theoretically informed four factor structure believed to be the expected structure in typically developing school beginners (verbal short term memory, verbal working memory, visuospatial short term memory and visuospatial working memory). Following this, a series of CFA's were then used within each of the three groups to assess model fit of AWMA performance according to five theoretically proposed models. A series of chi squared difference tests were also employed to identify significant differences between these models within each group.

Conclusion

This chapter outlined the method employed for this study. The initial section provides a detailed description of the procedures employed to gain access to the three sample groups, some of whom are difficult to locate because of ethical and practical constraints. It is structured around the two broad aims of the research. The first is the comparison of the

performance of the three groups on two measures of neurocognitive functioning: working memory (as measured by the AWMA), and six domains of general neuropsychological functioning (as measured by the NEPSY-II). The second employs a comparative confirmatory factor analysis of five theoretically informed models of working memory structure within all three groups to determine whether working memory structure is following a similar progression to that of typical populations. The next chapter presents the results of the statistical analyses employed to achieve these two aims.

Chapter 5: Results

Introduction

This chapter is organised around the two main research aims of the study. The first part considers the between-group comparisons, and within-group profiling on two psychometric measures, namely the Automated Working Memory Assessment (AWMA), and the Developmental Neuropsychological Assessment-II (NEPSY-II). The second component investigates whether the working memory profiles of the three groups of children (HIV infected (HIV-I), HIV-Exposed, Uninfected (HIV-EU), and HIV-Uninfected, Unexposed (HIV-UU)) are developing in a manner expected of typical children within the same age range. This was done through two applications of a confirmatory factor analysis. These two sections are preceded by a thorough examination of the descriptive data of the three groups.

Descriptive Data

This section documents the descriptive results of the three groups' performance on the AWMA, the NEPSY-II, and a number of extraneous variables which were analysed for possible inclusion as covariates in subsequent inferential analyses. Table 5.1 reports the mean scores and the standard deviations within each subtest, and cumulative index of the AWMA. The table also presents a description of the performance of a smaller subset of children who completed a selection of subtests from the NEPSY-II. In order to assess for significant differences in the various aspects of neurocognitive functioning, the subtest scaled scores of the NEPSY-II were collapsed into six subscales, namely, Attention and Executive Functioning, Language, Social Perception, Visuospatial Processing, Memory and Learning and Sensorimotor Abilities. A technical explanation of how this was done is offered in Appendix G.

An analysis of the skewness and kurtosis statistics was conducted for each of these, as well as the standard error of each, which were used to calculate individual z-scores. Together, these give a numerical indication of the normality of the distribution. Tables presenting the z-score data are available in Appendix H. The skewness and kurtosis statistics met the criteria for normality in all variables of the AWMA, except for Verbal Working Memory which had a slight left skewing. A logarithm (log base 10) transformation was applied to this variable which created a more normal distribution. All six of the domain scores of the NEPSY are believed to be sufficiently normal to warrant parametric analysis.

Kline (1998, 2005) argues that for research within the social sciences, skewness values higher than three, and kurtosis values higher than 10 are problematic.

A between-group comparison of the AWMA and NEPSY-II descriptive statistics appear to be as theoretically expected. HIV-I appears to have the weakest profile of the sample, while HIV-UU generally has the strongest, with HIV-EU performing between somewhere between these two groups. An account of significant differences are provided later in this chapter.

Table 5.1

Descriptive Test Statistics for AWMA and NEPSY-II Performance Across Three Groups (N = 273)

	Mean (SD)	Range	Skewness	Kurtosis	Mean (SD)	Range	Skewness	Kurtosis	Mean (SD)	Range	Skewness	Kurtosis
AWMA (n = 273)	HIV-I (n = 95)				HIV-EU (n = 86)				HIV-UU (n = 92)			
Digit Recall	86.21 (14.11)	64-120	0.40	-0.33	90.47 (17.00)	60-125	0.16	-0.88	100.21 (17.05)	11-126	-1.66	6.91
Word Recall	77.54 (13.32)	30-127	0.57	2.53	76.15 (14.2)	63-120	1.50	1.78	94.43 (18.46)	63-129	-0.07	-1.19
Non-Word Recall	100.97 (17.87)	59-145	-0.16	0.27	95.18 (17.15)	59-137	0.14	-0.32	110.62 (18.03)	69-137	-0.31	-0.68
VSTM	85.6 (14.62)	59-131	0.33	0.09	84.44 (16.41)	59-129	0.77	0.33	102.41 (16.97)	69-129	-0.22	-0.82
Listening Recall	73.13 (15.31)	2-109	-0.17	4.36	84.56 (17.58)	62-139	1.15	1.27	95.57 (16.89)	63-131	0.01	-0.69
Listening Recall Processing	77.88 (9.18)	66-114	1.68	3.19	83.13 (13.25)	66-134	1.83	4.03	90.13 (12.77)	73-135	0.81	0.77
Counting Recall	87.85 (13.65)	55-129	0.19	0.32	93.44 (13.74)	70-130	0.55	0.27	103.37 (20.36)	14-141	-0.77	3.16
Counting Recall Processing	89.05 (11.17)	71-128	1.13	2.26	93.94 (12.57)	75-131	1.10	1.35	104.22 (16.32)	74-133	0.41	-0.93
Backwards Digit Recall	80.57 (13.3)	58-119	0.29	-0.29	84.56 (14.76)	58-136	0.48	0.57	99.42 (16.34)	64-143	0.24	0.09
VWM	77.18 (11.13)	61-107	0.69	-0.46	85.13 (14.13)	61-121	0.68	-0.17	99.7 (16.26)	66-131	0.35	-0.62
Dot Matrix	86.61 (16.72)	61-148	0.59	0.56	93.22 (14.71)	64-132	0.16	-0.23	99.3 (17.17)	65-148	0.68	0.18
Mazes Memory	83.51 (15.81)	48-133	-0.22	0.68	86.94 (17.91)	48-129	0.27	-0.65	97.09 (16.44)	59-133	0.10	-0.46
Block Recall	85.94 (13.57)	47-120	-0.19	-0.04	90.87 (13.02)	61-120	-0.06	-0.48	97.46 (16.08)	70-131	0.46	-0.56
VSSTM	82.76 (15.85)	2-131	-0.30	0.54	88.74 (15.21)	63-126	0.47	-0.55	97.29 (17.6)	63-139	0.50	-0.45
Odd One Out	88.73 (17.53)	59-130	0.44	-0.82	98.9 (15.93)	62-133	0.01	-0.56	108 (17.36)	71-133	-0.50	-0.70
Odd One Out Processing	90.15 (15.36)	71-130	0.70	-0.71	97.86 (14.55)	71-144	0.57	0.37	107.55 (16.6)	75-144	-0.04	-0.54
Mister X	92.33 (15.92)	62-144	0.56	0.37	99 (13.62)	71-133	0.13	-0.27	107.11 (19.42)	71-155	0.29	-0.56
Mister X Processing	88.02 (9.8)	71-125	1.40	3.49	92.53 (13.2)	11-121	-2.34	16.56	103.09 (19.58)	30-139	-0.07	0.72
Spatial Recall	87.65 (15.43)	60-126	0.17	-0.64	95.56 (14.1)	64-135	0.55	0.57	102.32 (15.01)	70-135	0.08	-0.46
Spatial Recall Processing	87.94 (11.27)	70-142	1.73	5.43	94.79 (13.05)	75-142	1.69	3.21	101.8 (15.04)	80-148	0.68	-0.17
VSWM	87.21 (16.38)	61-132	0.42	0.49	97.23 (14.14)	62-132	0.06	-0.01	107.25 (17.98)	71-139	0.09	-0.99
NEPSY-II (N = 87)	HIV-I (n = 29)				HIV-EU (n = 29)				HIV-UU (n = 29)			
Att. & Exec. Functioning	49.87 (7.91)	36-65	-0.16	-0.85	52.36 (12.52)	27-95	1.16	4.29	66.4 (9.98)	45-83	-0.36	-0.38
Language	31.52 (11.74)	7-54	-0.22	-0.49	36.04 (14.21)	12-60	-0.12	-1.02	58.86 (10.51)	36-78	-0.10	-0.71
Memory & Learning	31.17 (1.84)	13-53	0.51	0.04	53.79 (11.85)	12-58	-0.10	-0.75	57.09 (8.89)	44-85	1.18	2.46
Sensorimotor Abilities	42.89 (10.61)	18-64	-0.34	0.05	50.22 (7.7)	38-67	0.61	0.04	59.23 (8.02)	46-75	0.18	-0.95
Social Perception	47.89 -(7.00)	33-63	-0.47	0.42	48.39 (11.48)	23-66	-0.68	0.00	61.73 (8.77)	30-75	-1.61	4.77
Visuomotor Processing	35.84 (9.82)	20-59	0.42	0.05	37.7 (10.09)	21-61	0.49	-0.19	58.14 (9.94)	38-86	0.31	1.08

In order to present a detailed descriptive analysis of the data, it was necessary to investigate whether there were significant associations between the various tests administered. A series of Pearson correlations were therefore examined between all subtests and indices of the neurocognitive and working memory measures. Table 5.2 presents the parametric correlations between the psychometric and demographic variables of the AWMA sample in each of the three groups. Table 5.3 presents similar data regarding the smaller NEPSY-II subsample.

Table 5.3
Parametric Correlations of NEPSY-II Subsample (N = 90)

		RCPM	Attention & Executive Functioning	Language	Memory & Learning	Sensorimotor	Social Perception	Visuospatial Processing
SRT	r	0.482**	0.376**	0.749**	0.693**	0.386**	0.519**	0.547**
RCPM	r	-	0.496**	0.679**	0.604**	0.434**	0.415**	0.674**
Att. & Exec. Functioning	r		-	0.577**	0.520**	0.549**	0.323**	0.671**
Language	r			-	0.839**	0.611**	0.582**	0.757**
Memory and Learning	r				-	0.574**	0.532**	0.748**
Sensorimotor	r					-	0.383**	0.712**
Social Perception	r						-	0.593**

** $p < 0.01$. (two-tailed)

This final descriptive section presents the data commencement of inferential analysis in order to determine which variables they were to be included as covariates in further analyses.

Of the additional variables gathered from the larger AWMA sample, the Sentence Repetition Test (SRT) (a proxy for English proficiency) and the Living Standards Measure (LSM) (a measure of socioeconomic status (SES)) were found to be non-normal. The former had a strong negative skewness, while the latter had a steep central kurtosis. Within the smaller NEPSY-II sub-sample, SRT was the only variable found to be non-normal. An explanation of subsequent transformations to these non-normal variables follows in subsequent sections. Table 5.4 presents their descriptive markers, and skewness and kurtosis statistics. Table I1 in Appendix I presents further checks for normality and histograms.

Table 5.2a
Correlations of Tests for HIV-I Group (N = 95)

		LSM_{raw}	SRT	RCPM	Digit Recall	Word Recall	Nonword Recall	**Verbal Short Term Memory**	Listening Recall	Counting Recall	Backwards Digit Recall	**Verbal Working Memory**	Dot Matrix	Mazes Memory	Block Recall	**Visuospatial Short Term Memory**	Odd One Out Recall	Mr X Recall	Spatial Recall	**Visuospatial Working Memory**
Age (months)	r	-.207*	.208*	-.114	-.133	-.299**	-.035	**-.155**	-.029	.075	-.010	**-.041**	-.093	-.048	-.067	**-.134**	.065	-.085	-.106	**-.071**
	p	.044	.043	.272	.197	.003	.733	**.132**	.784	.467	.921	**.690**	.371	.643	.517	**.196**	.530	.411	.305	**.495**
LSMraw	r		-.080	.157	.079	.276**	.196	**.213***	.028	.029	-.018	**.071**	.384**	.297**	.189	**.362****	.026	.169	.146	**.136**
	p		.442	.129	.446	.007	.056	**.038**	.790	.779	.863	**.492**	.000	.003	.067	**.000**	.800	.102	.159	**.190**
SRT	r			.073	.306**	.100	.255*	**.301****	.336**	.267**	.240*	**.364****	.160	.197	.101	**.155**	.329**	.254*	.158	**.303****
	p			.485	.003	.335	.013	**.003**	.001	.009	.019	**.000**	.120	.056	.329	**.134**	.001	.013	.126	**.003**
RCPM	r				.007	-.059	-.082	**-.035**	.168	.211*	.138	**.261***	.217*	.159	.151	**.241***	.207*	.218*	.246*	**.279****
	p				.946	.571	.427	**.736**	.103	.040	.183	**.011**	.034	.124	.143	**.018**	.044	.034	.016	**.006**
Digit Recall	r					.484**	.530**	**.806****	.308**	.354**	.511**	**.500****	.456**	.544**	.427**	**.394****	.382**	.404**	.361**	**.444****
	p					.000	.000	**.000**	.002	.000	.000	**.000**	.000	.000	.000	**.000**	.000	.000	.000	**.000**
Word Recall	r						.583**	**.749****	.121	.265**	.258*	**.303****	.384**	.436**	.268**	**.343****	.296**	.379**	.403**	**.415****
	p						.000	**.000**	.241	.009	.012	**.003**	.000	.000	.009	**.001**	.004	.000	.000	**.000**
Nonword Recall	r							**.865****	.379**	.261*	.351**	**.449****	.302**	.419**	.204*	**.184**	.237*	.342**	.298**	**.323****
	p							**.000**	.000	.011	.000	**.000**	.003	.000	.047	**.075**	.021	.001	.003	**.001**
Verbal Short Term	**r**								**.378****	**.376****	**.446****	**.535****	**.417****	**.568****	**.342****	**.345****	**.327****	**.449****	**.440****	**.466****
Memory	**p**								**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.001**	**.001**	**.001**	**.000**	**.000**	**.000**
Listening Recall	r									.297**	.415**	**.735****	.241*	.238*	.179	**.239***	.231*	.248*	.229*	**.268****
	p									.003	.000	**.000**	.018	.020	.082	**.020**	.025	.015	.025	**.009**
Counting Recall	r										.296**	**.664****	.443**	.545**	.392**	**.501****	.455**	.557**	.526**	**.604****
	p										.004	**.000**	.000	.000	.000	**.000**	.000	.000	.000	**.000**

Table 5.2a (continued)
Correlations of Tests for HIV-I Group (N = 95)

		LSM_{raw}	SRT	RCPM	Digit Recall	Word Recall	Nonword Recall	**Verbal Short Term Memory**	Listening Recall	Counting Recall	Backwards Digit Recall	**Verbal Working Memory**	Dot Matrix	Mazes Memory	Block Recall	**Visuospatial Short Term Memory**	Odd One Out Recall	Mr X Recall	Spatial Recall	**Visuospatial Working Memory**
Backwards Digit Recall	r											**.722****	.352**	.390**	.233*	**.269****	.420**	.376**	.282**	**.433****
	p											**.000**	.000	.000	.023	**.009**	.000	.000	.006	**.000**
Verbal Working Memory	**r**												**.489****	**.533****	**.352****	**.473****	**.477****	**.510****	**.426****	**.555****
	p												**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**
Dot Matrix	r													.689**	.605**	**.835****	.571**	.509**	.477**	**.612****
	p													.000	.000	**.000**	.000	.000	.000	**.000**
Mazes Memory	r														.526**	**.709****	.481**	.542**	.510**	**.590****
	p														.000	**.000**	.000	.000	.000	**.000**
Block Recall	r															**.679****	.465**	.416**	.527**	**.546****
	p															**.000**	.000	.000	.000	**.000**
Visuospatial Short Term Memory	**r**																**.548****	**.528****	**.546****	**.636****
	p																**.000**	**.000**	**.000**	**.000**
Odd One Out	r																	.482**	.542**	**.817****
	p																	.000	.000	**.000**
Mr X Recall	r																		.656**	**.823****
	p																		.000	**.000**
Spatial Recall	r																			**.859****
	p																			**.000**

*p<0.05, **p<0.01
Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Short Term Memory, VSWM: Visuospatial Working Memory

Table 5.2b

Correlations of Tests for HIV-EU Group (N = 88)

		LSMraw	SRT	RCPM	Digit Recall	Word Recall	Nonword Recall	**Verbal Short Term Memory**	Listening Recall	Counting Recall	Backwards Digit Recall	**Verbal Working Memory**	Dot Matrix	Mazes Memory	Block Recall	**Visuospatial Short Term Memory**	Odd One Out Recall	Mr X Recall	Spatial Recall	**Visuospatial Working Memory**
Age (months)	r	.097	.414**	-.081	-.172	-.168	-.065	**-.176**	-.103	-.136	-.014	**-.109**	-.355**	-.427**	-.228*	**-.371****	-.118	-.183	-.168	**-.180**
	p	.370	.000	.451	.112	.122	.555	**.106**	.346	.213	.896	**.320**	.001	.000	.035	**.000**	.278	.092	.123	**.097**
LSMraw	r		.293**	-.110	.090	.123	.153	**.150**	-.044	-.002	-.010	**-.039**	-.048	.193	-.070	**.046**	.205	-.028	.046	**.103**
	p		.006	.305	.408	.258	.161	**.168**	.686	.982	.929	**.718**	.662	.075	.521	**.673**	.058	.801	.675	**.344**
SRT	r			.128	.135	.251*	.223*	**.237***	.019	.089	.075	**.051**	-.103	-.027	.028	**-.011**	.214*	-.023	.157	**.153**
	p			.235	.216	.020	.039	**.028**	.862	.413	.491	**.643**	.343	.804	.797	**.923**	.048	.833	.148	**.161**
RCPM	r				-.043	.037	-.038	**-.032**	.042	.151	.361**	**.202**	-.006	-.028	.095	**.012**	.085	.185	.193	**.181**
	p				.697	.737	.726	**.772**	.699	.165	.001	**.062**	.955	.796	.382	**.913**	.437	.088	.075	**.095**
Digit Recall	r					.501**	.515**	**.812****	.209	.462**	.309**	**.428****	.451**	.430**	.480**	**.521****	.467**	.253*	.302**	**.417****
	p					.000	.000	**.000**	.053	.000	.004	**.000**	.000	.000	.000	**.000**	.000	.019	.005	**.000**
Word Recall	r						.595**	**.824****	.338**	.321**	.128	**.368****	.261*	.330**	.362**	**.365****	.321**	.292**	.348**	**.378****
	p						.000	**.000**	.001	.003	.239	**.000**	.015	.002	.001	**.001**	.003	.006	.001	**.000**
Nonword Recall	r							**.852****	.320**	.411**	.195	**.378****	.450**	.388**	.447**	**.453****	.465**	.305**	.337**	**.464****
	p							**.000**	.003	.000	.071	**.000**	.000	.000	.000	**.000**	.000	.004	.002	**.000**
VSTM	**r**								**.340****	**.487****	**.253***	**.472****	**.484****	**.476****	**.536****	**.554****	**.510****	**.334****	**.398****	**.506****
	p								**.001**	**.000**	**.019**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.002**	**.000**	**.000**
Listening Recall	r									.364**	.193	**.718****	.163	.139	.159	**.154**	.168	.365**	.286**	**.342****
	p									.001	.076	**.000**	.133	.202	.143	**.157**	.121	.001	.008	**.001**

Table 5.2b (continued)

Correlations of Tests for HIV-EU Group (N = 88)

		LSMraw	SRT	RCPM	Digit Recall	Word Recall	Nonword Recall	**Verbal STM**	Listening Recall	Counting Recall	Backwards Digit Recall	**Verbal Working Memory**	Dot Matrix	Mazes Memory	Block Recall	**Visuospatial STM**	Odd One Out Recall	Mr X Recall	Spatial Recall	**Visuospatial WM**
Counting Recall	r										.527**	**.786****	.475**	.532**	.572**	**.594****	.452**	.403**	.575**	**.596****
	p										.000	**.000**	.000	.000	.000	**.000**	.000	.000	.000	**.000**
Backwards Digit Recall	r											**.695****	.353**	.272*	.355**	**.352****	.439**	.191	.322**	**.404****
	p											**.000**	.001	.011	.001	**.001**	.000	.078	.002	**.000**
VWM	**r**												**.422****	**.401****	**.456****	**.468****	**.435****	**.418****	**.463****	**.551****
	p												**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**
Dot Matrix	r													.564**	.701**	**.840****	.561**	.288**	.502**	**.571****
	p													.000	.000	**.000**	.000	.007	.000	**.000**
Mazes Memory	r														.629**	**.865****	.519**	.434**	.551**	**.632****
	p														.000	**.000**	.000	.000	.000	**.000**
Block Recall	r															**.854****	.508**	.393**	.491**	**.592****
	p															**.000**	.000	.000	.000	**.000**
VSSTM	**r**																**.585****	**.401****	**.580****	**.661****
	p																**.000**	**.000**	**.000**	**.000**
Odd One Out	r																	.333**	.455**	**.784****
	p																	.002	.000	**.000**
Mr X Recall	r																		.542**	**.753****
	p																		.000	**.000**
Spatial Recall	r																			**.821****
	p																			**.000**

*p<0.05, **p<0.01

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Short Term Memory, VSWM: Visuospatial Working Memory

Table 5.2c

Correlations of Tests for HIV-UU Group (N = 92)

		LSMraw	SRT	RCPM	Digit Recall	Word Recall	Nonword Recall	**Verbal Short Term Memory**	Listening Recall	Counting Recall	Backwards Digit Recall	**Verbal Working Memory**	Dot Matrix	Mazes Memory	Block Recall	**Visuospatial STM**	Odd One Out Recall	Mr X Recall	Spatial Recall	**Visuospatial Working Memory**
Age	r	.157	.315**	-.139	.138	.038	.134	**.105**	-.036	.078	.016	**.015**	-.101	-.096	-.077	**-.100**	.068	-.174	-.096	**-.084**
(months)	p	.134	.002	.187	.191	.716	.201	**.319**	.732	.458	.876	**.888**	.336	.363	.466	**.343**	.520	.097	.362	**.428**
LSMraw	r		.219*	.011	.064	.148	.106	**.078**	.114	-.032	-.098	**-.025**	-.131	-.004	-.229*	**-.135**	-.129	-.159	-.203	**-.191**
	p		.036	.921	.541	.158	.316	**.460**	.278	.759	.353	**.810**	.214	.968	.028	**.201**	.220	.130	.052	**.069**
SRT	r			.290**	.446**	.537**	.530**	**.593****	.370**	.328**	.205*	**.397****	.147	.313**	.099	**.217***	.206*	.131	.139	**.187**
	p			.005	.000	.000	.000	**.000**	.000	.001	.050	**.000**	.162	.002	.349	**.038**	.049	.213	.185	**.074**
RCPM	r				.183	.327**	.093	**.236***	.156	.141	.320**	**.274****	.235*	.356**	.281**	**.334****	.221*	.378**	.239*	**.339****
	p				.082	.001	.377	**.023**	.138	.181	.002	**.008**	.024	.000	.007	**.001**	.035	.000	.022	**.001**
Digit Recall	r					.374**	.297**	**.550****	.342**	.289**	.268**	**.449****	.279**	.287**	.335**	**.351****	.361**	.074	.191	**.238***
	p					.000	.004	**.000**	.001	.005	.010	**.000**	.007	.006	.001	**.001**	.000	.486	.068	**.022**
Word Recall	r						.696**	**.895****	.449**	.414**	.342**	**.559****	.464**	.584**	.375**	**.551****	.388**	.422**	.488**	**.508****
	p						.000	**.000**	.000	.000	.001	**.000**	.000	.000	.000	**.000**	.000	.000	.000	**.000**
Nonword	r							**.860****	.210*	.218*	.060	**.267***	.263*	.329**	.223*	**.315****	.282**	.232*	.311**	**.321****
Recall	p							**.000**	.045	.037	.573	**.010**	.011	.001	.032	**.002**	.006	.026	.003	**.002**
VSTM	**r**								**.401****	**.373****	**.276****	**.507****	**.415****	**.516****	**.395****	**.508****	**.431****	**.323****	**.435****	**.462****
	p								**.000**	**.000**	**.008**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.002**	**.000**	**.000**
Listening	r									.447**	.404**	**.816****	.362**	.499**	.430**	**.500****	.352**	.345**	.329**	**.408****
Recall	p									.000	.000	**.000**	.000	.000	.000	**.000**	.001	.001	.001	**.000**

Table 5.2c (continued)

Correlations of Tests for HIV-UU Group (N = 92)

		LSMraw	SRT	RCPM	Digit Recall	Word Recall	Nonword Recall	**Verbal Short Term Memory**	Listening Recall	Counting Recall	Backwards Digit Recall	**Verbal Working Memory**	Dot Matrix	Mazes Memory	Block Recall	**Visuospatial STM**	Odd One Out Recall	Mr X Recall	Spatial Recall	**Visuospatial Working Memory**
Counting	r										.308**	**.632****	.507**	.490**	.485**	**.565****	.363**	.432**	.459**	**.492****
Recall	p										.003	**.000**	.000	.000	.000	**.000**	.000	.000	.000	**.000**
Backwards	r											**.730****	.366**	.313**	.418**	**.420****	.426**	.340**	.382**	**.457****
Digit Recall	p											**.000**	.000	.002	.000	**.000**	.000	.001	.000	**.000**
VWM	**r**												**.576****	**.622****	**.650****	**.715****	**.525****	**.525****	**.524****	**.625****
	p												**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**	**.000**
Dot Matrix	r													.560**	.715**	**.879****	.508**	.612**	.518**	**.653****
	p													.000	.000	**.000**	.000	.000	.000	**.000**
Mazes	r														.596**	**.825****	.459**	.665**	.579**	**.679****
Memory	p														.000	**.000**	.000	.000	.000	**.000**
Block Recall	r															**.888****	.503**	.578**	.569**	**.654****
	p															**.000**	.000	.000	.000	**.000**
VSSTM	**r**																**.558****	**.718****	**.639****	**.763****
	p																**.000**	**.000**	**.000**	**.000**
Odd One	r																	.420**	.505**	**.767****
Out	p																	.000	.000	**.000**
Mr X Recall	r																		.729**	**.865****
	p																		.000	**.000**
Spatial	r																			**.875****
Recall	p																			**.000**

*p<0.05, **p<0.01

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Short Term Memory, VSWM: Visuospatial Working Memory

Table 5.4

Descriptive Demographic Statistics for Complete Sample and NEPSY-II Subsample Performance Across Three Groups

	Mean (SD)	Range	Skewness	Kurtosis	Mean (SD)	Range	Skewness	Kurtosis	Mean (SD)	Range	Skewness	Kurtosis
Complete Sample		HIV-I (n = 95)				HIV-EU (n = 88)				HIV-UU (n = 92)		
Age at Assessment	88.98 (10.15)	71-107	-0.068	-1.207	88.28 (10.51)	67-106	-0.12	-1.16	84.54 (10.35)	60-106	-0.07	-0.79
LSM_{Scaled}	6.55 (1.77)	2-10	-0.146	0.181	5.88 (1.13)	2-10	-0.48	3.07	6.95 (1.65)	2-10	0.17	0.13
LSM_{Raw}	11.15 (5.8)	0-24	0.191	-0.645	10.36 (4.04)	2-24	0.59	0.39	12.46 (4.47)	0-25	0.09	0.53
SRT	7.91 (6.95)	0-30	0.77	0.129	10.69 (7.64)	0-30	0.51	-0.35	17.15 (9.83)	0-32	-0.08	-1.06
RCPM	18.16 (21.72)	5-90	1.872	2.765	29.03 (28.15)	5-95	0.84	-0.76	54.67 (32.47)	5-95	-0.31	-1.39
NEPSY-II Subsample		HIV-I (n = 29)				HIV-EU (n = 29)				HIV-UU (n = 29)		
Age at Assessment	87.03 (10.64)	72-103	-0.05	-1.52	87.62 (10.41)	72-104	-0.19	-1.34	87.48 (10.29)	71-102	-0.12	-1.44
LSM_{Scaled}	5.61 (1.57)	2-9	-0.44	0.62	6.02 (1.41)	3-9	0.12	-0.03	7.12 (1.77)	3-10	-0.06	-0.28
LSM_{raw}	9.52 (5.04)	2-20	0.14	-1.17	11.20 (4.45)	4-21	0.33	-0.49	12.48 (5.04)	4-22	0.19	-0.56
SRT	6.36 (7.58)	0-30	1.56	2.30	10.83 (9.22)	0-30	0.15	-0.97	23.03 (8.71)	8-32	-0.40	-1.39
SRT_{BoxCox}	2.58 (*2.93*)	-0.58-9.65	0.66	-0.34	4.31 (*3.07*)	-0.58-9.66	0.75	-0.57	7.99 (*2.07*)	3.97-10.05	-0.25	-1.61
RCPM	15.07 (5.59)	6-26	1.28	0.38	13.82 (5.11)	4-24	1.38	0.73	22.69 (5.27)	10-32	-1.18	0.46

Inferential Statistics

This section documents a series of analyses which investigated between-group differences in the working memory performance of the three groups as measured by the AWMA. It also documents an analysis of between-group differences of more generalised cognitive functioning, as measured by the NEPSY-II, on a smaller subset of the larger sample. It concludes with the presentation of within-group working memory and NEPSY-II profiles of each of the samples three groups.

The identification of covariates.

In order to analyse significant between-group differences, it was necessary to identify possible covariates that could conflate the results, and reduce the true error variance of other cognitive influences other than working memory. The following variables were considered as quantifiable measures of possible extraneous influence: Living Standards Measure Scaled Score (LSM) and Living Standards Measure Raw Score (LSM_{raw}) used as an indication of socioeconomic status, Sentence Repetition Test (SRT) used as a proxy for English proficiency, Age at Assessment (measured in months), and the Ravens Coloured Progressive Matrices (RCPM) which was the proxy for non-verbal intelligence. Both LSM_{raw} and LSM scaled scores were included in order to determine which had better normality. However, LSM_{raw} was chosen as it offered more variance.

There are limitations to the process of co-variation. Firstly, the inclusion of covariates is often the focus of theoretical contention, as it is based on the assumption that the influence of these covariates is, equal across the levels of the independent variable, and that the differences are a function of some extraneous, unwanted influence (Mayers, 2013). In this study, differences in constructs such as non-verbal intelligence could be influenced by the very same feature (HIV exposure) under investigation, and its use as a covariate could cancel out the effect of this influence. Conversely, the inclusion of a covariate allows for an isolated, 'purer' investigation of only the theoretical construct of interest – namely working memory. Secondly, the use of the Sentence Repetition Test as a proxy for English proficiency was not a direct assessment of what it intended to measure, since it relies on a degree of verbal short-term memory in an additional language. While the ability to remember a sentence in an additional language might be an accurate reflection of linguistic proficiency, despite its demand on memory, the tests reliance on the cognitive skills under investigation cannot be discounted. Further, the decision to use age instead of grade as a possible covariate was made for a number of reasons, which have been described in Chapter 4.

In order to identify covariates, an analysis of variance of potential extraneous influences was necessary. The assumptions of parametric testing on these aforementioned variables within each sample were assessed before further analysis could take place.

Table 5.5
Homogeneity of Variance for Possible Covariates (AWMA Sample)

	Levene's Statistic	df1	df2	P
Age at Assessment	0.095	2	272	0.909
LSM Scaled	12.716	2	272	<0.001**
LSM Raw Score	7.990	2	272	<0.001**
SRT	9.043	2	272	<0.001**
RCPM	1.420	2	272	0.244

*** p< 0.001 (two tailed).

Within the larger AWMA sample, Levene's test of homoscedasity (Table 5.5) shows that the LSM scaled and raw scores, as well as SRT do not have equal variance across the groups (measured by significant p values). This is because none of these three are normally distributed, with the LSM_{raw} scaled scores showing a steeped kurtosis, and the SRT being negatively skewed. Various transformations were applied to these three variables, but they made little difference to the overall performance. It was therefore decided to leave the variables unchanged for the following reasons. Firstly, the sample makes use of a linguistically diverse sample with variability in English proficiency. Further, a child's working memory performance – particularly the language components thereof – are strongly correlated with their verbal fluency (Miyake & Friedman, 1998; Payne & Whitney, 2002). Hence, this difference in language proficiency could be because of the very construct of interest in this study and the associated neurocognitive and socio-cultural influences that are inseparable from the different levels of the independent variables. Thirdly, the analyses use a large sample size with good statistical power, and makes use of tests which are statistically robust in the face of deviations from normality. Lastly, because of the negative skewness, a series of transformations were applied to the data in an attempt to improve normality. While these transformations made marginal difference to the data's skewness, they disturbed the kurtosis and distribution of variance within other levels of the independent variable. For that reason, the original SRT and LSM_{raw} scores were used in the identification of the covariates.

An analysis of variance between the groups (Table 5.6) indicate that Age at Assessment, LSM_{raw}, SRT and RCPM were all significantly different across the three groups, suggesting that they be included as covariates in an attempt to counteract their influence on the final analysis.

Table 5.6

ANOVA Identification of Covariates (Complete Sample N = 273)

	Sum of Squares	df	Mean Square	F	p
Age at Assessment					
Between Groups	1051.864	2	525.932	4.925	0.008**
Within Groups	29048.682	272	106.797		
Total	30100.545	274			
LSM Scaled					
Between Groups	53.014	2	26.507	11.029	0.001**
Within Groups	653.692	272	2.403		
Total	706.705	274			
LSM Raw Score					
Between Groups	202.095	2	101.048	4.294	0.015**
Within Groups	6401.127	272	23.534		
Total	6603.222	274			
SRT					
Between Groups	4181.994	2	2090.997	31.290	0.001**
Within Groups	18176.733	272	66.826		
Total	22358.727	274			
RCPM					
Between Groups	1570.789	2	785.395	24.764	0.001**
Within Groups	8626.687	272	31.716		
Total	10197.476	274			

**p<0.01 (two-tailed)

These covariates were not condensed into one component. Their correlation and components matrices did not show sufficient inter-correlation between the covariates (Table 5.8), and a multivariate analysis of covariance is a robust test that can handle up to ten covariates, although this reduces the degrees of freedom.

Table 5.8

Correlation and Covariance Matrix of Covariates (Complete Sample N = 273)

	Inter-Item Correlation Matrix				Inter-Item Covariance			
	Total Age in months	LSM Raw Score	SRT	RCPM	Total Age in months	LSM Raw Score	SRT	RCPM
Age at Assessment	1	-0.04	0.19	0.13	109.86	-1.92	18.43	8.56
LSM Raw Score	-0.04	1	0.17	0.1	-1.92	24.1	7.51	2.99
SRT	0.19	0.17	1	0.44	18.43	7.51	81.6	24.48
RCPM	0.13	0.1	0.44	1	8.56	2.99	24.48	37.22

Post-hoc analyses are presented in Table 5.7, where significant differences are indicated by an asterisk.

Table 5.7
Post-Hoc Pairwise Analyses for Identified Covariates (Complete Sample N = 273)

						95% CI	
			Mean Difference	Std. Error	p	Lower Bound	Upper Bound
LSM calculation	HIV-I	HIV-EU	0.67	0.23	<0.001**	0.22	1.12
		HIV-UU	-0.40	0.23	0.08	-0.85	0.04
	HIV-EU	HIV-I	-0.67	0.23	<0.001**	-1.12	-0.22
		HIV-UU	-1.07	0.23	<0.001**	-1.53	-0.62
	HIV-UU	HIV-I	0.40	0.23	0.08	-0.04	0.85
		HIV-EU	1.08*	0.23	<0.001**	0.62	1.53
LSM Raw Score	HIV-I	HIV-EU	0.78	0.72	0.28	-0.63	2.20
		HIV-UU	-1.31	0.71	0.07	-2.71	0.09
	HIV-EU	HIV-I	-0.78	0.72	0.28	-2.20	0.63
		HIV-UU	-2.09	0.72	<0.001**	-3.52	-0.67
	HIV-UU	HIV-I	1.31	0.71	0.07	-0.09	2.71
		HIV-EU	2.09	0.72	<0.001**	0.67	3.52
Age at Assessment	HIV-I	HIV-EU	0.69	1.53	0.65	-2.32	3.70
		HIV-UU	4.4	1.51	<0.001**	1.46	7.41
	HIV-EU	HIV-I	-0.69	1.53	0.65	-3.70	2.32
		HIV-UU	3.74	1.54	0.02*	0.71	6.77
	HIV-UU	HIV-I	-4.44	1.51	<0.001*	-7.41	-1.46
		HIV-EU	-3.74	1.54	0.02*	-6.77	-0.71
SRT	HIV-I	HIV-EU	-2.79	1.21	0.02*	-5.17	-0.41
		HIV-UU	-9.25	1.20	<0.001**	-11.60	-6.89
	HIV-EU	HIV-I	2.79	1.21	0.02*	0.41	5.17
		HIV-UU	-6.46	1.22	<0.001**	-8.86	-4.06
	HIV-UU	HIV-I	9.25	1.20	<0.001**	6.89	11.60
		HIV-EU	6.46	1.22	<0.001**	4.06	8.86
RCPM	HIV-I A	HIV-EU	-1.93	0.83	0.02*	-3.57	-0.29
		HIV-UU	-5.71	0.82	<0.001**	-7.33	-4.09
	HIV-EU	HIV-I	1.93	0.83	0.02*	0.29	3.57
		HIV-UU	-3.78	0.84	<0.001**	-5.44	-2.13
	HIV-UU	HIV-I	5.71	0.82	<0.001**	4.09	7.33
		HIV-EU	3.78	0.84	<0.001**	2.13	5.44

*p<0.05 (two-tailed), **p<0.001 (two-tailed)

Similarly, variables representing a quantifiable extraneous influence on the findings of the smaller NEPSY-II sub-sample were also assessed for normality. In order to satisfy the criteria for parametricity, SRT was transformed by a Box-Cox transformation to compensate for negative skewness and improve homoscedasity across the groups. This analysis employed

software developed by Wessa (2015) which has been used successfully in four of 88 cited sources (Butler, Barrett, Nowbath, & Upchurch, 2009; Kleifeld, Doucet, Prudova, & auf dem Keller, 2011; Mallat, Craig & Yoder, 2010; Zhu & Lee, 2008).

An assessment of homoscedasity of these possible extraneous influences indicate that they have sufficiently normal distribution to warrant parametric analyses (Refer to Table 5.9).

Table 5.9
Homogeneity of Variance for Possible Covariates (NEPSY-II Subsample)

	Levene's Statistic	df1	df2	P
Age at Assessment	0.056	2	86	0.945
LSM Raw	1.458	2	86	0.238
SRT Box Cox	2.017	2	86	0.139
RCPM	0.647	2	86	0.526

*p< 0.05

Following a parametric analysis of variance, only RCPM and SRT_{BoxCox} were identified as possible covariates within the smaller NEPYS-II sub-sample, and were therefore included in a multivariate analysis of covariance (Table 5.10) later on.

Table 5.10
ANOVA Analyses Identifying Covariates for NEPSY-II Analysis

		Sum of Squares	df	Mean Square	F	p
Total in months	Between Groups	5.727	2	2.864	0.026	0.974
	Within Groups	9393.037	86	109.221		
	Total	9398.764	88			
LSM Raw Score	Between Groups	133.157	2	66.578	2.824	0.065
	Within Groups	2027.742	86	23.578		
	Total	2160.899	88			
SRT Box Cox	Between Groups	453.064	2	226.532	30.303	<0.001**
	Within Groups	642.906	86	7.476		
	Total	1095.971	88			
RCPM	Between Groups	1344.885	2	672.443	23.660	<0.001**
	Within Groups	2444.216	86	28.421		
	Total	3789.101	88			

**p<0.01

Table 5.11 indicates the post-hoc analyses using the least squared differences criteria. SRT_{BoxCox} and RCPM had a sufficiently moderate correlation (r = 0.581) to justify not collapsing them into a single covariate. The effect on the loss of degrees of freedom was believed to be negligible.

Table 5.11
Post Hoc Pairwise Analyses for Covariate Identification for NEPSY-II Subsample Analyses

			Mean Difference	Std. Error	p	95% CI	
						Lower Bound	Upper Bound
SRT Box Cox	HIV-I	HIV-EU	-1.73	0.71	0.02*	-3.14	-0.33
		HIV-UU	-5.41	0.71	<0.001**	-6.81	-4.00
	HIV-EU	HIV-I	1.73*	0.71	0.02*	0.33	3.14
		HIV-UU	3.67*	0.72	<0.001**	-5.10	-2.24
	HIV-UU	HIV-I	5.41*	0.71	<0.001**	4.00	6.81
		HIV-EU	3.67*	0.72	<0.001**	2.24	5.10
RCPM	HIV-I	HIV-EU	4.58	6.97	0.513	-9.28	18.44
		HIV-UU	-42.32*	6.97	<0.001**	-56.18	28.46
	HIV-EU	HIV-I	-4.58	6.97	0.513	-18.44	9.28
		HIV-UU	-46.90*	7.09	<0.001**	-60.99	32.81
	HIV-UU	HIV-I	42.32*	6.97	<0.001**	28.46	56.18
		HIV-EU	46.90*	7.09	<0.001**	32.81	60.99

*p< 0.05 (two-tailed), **p<0.01 (two-tailed)

The identified covariates were included in subsequent multivariate analyses. The following section documents the multivariate analyses used to identify significant between-group differences on both the AWMA and NEPSY-II.

Multivariate analyses: Between-group differences.

This section documents the between-group differences on the larger AWMA sample, as well as the NEPYS-II sub-sample. The data from the larger sample are presented first as they investigated the primary research question concerning working memory profile differences in the three groups, followed by the identified between-group differences on smaller sub-samples of more generalised neuropsychological functioning using the NEPSY-II.

Between-group differences on the Automated Working Memory Assessment.

A multivariate analysis of covariance was used to examine the differences between HIV status (independent variables) and performance on each of the four indices of the AWMA, with LSM_{raw}, Age at Assessment, SRT and RCPM as covariates.

In order to employ a valid use of the MANCOVA, certain assumptions needed to be met. By definition, there need to be at least two dependent variables (this study employs four simultaneously at the least), which are parametric, and have data which is in interval form and has a reasonable normal distribution. Covariates also need to be interval data and distributed

normally across independent groups, however the multivariate analysis of covariance is also quite robust to these violations as long as the sample size exceeds 20). There also needs to be reasonable correlation between the dependent variables (anywhere between r = 0.3 and 0.9), and this correlation should not be significantly different across independent groups. This is measured through an examination of the variance-covariance matrices which are presented in Box's M test (where highly significant outcomes of p<0.0001 should be avoided.). There also needs to be a reasonable correlation between the dependent variables and the covariates, and there should not exist large between-group differences in these correlations in the three subsamples. This is referred to as 'homogeneity of regression slopes'. Lastly, there should also be homogeneity of between-group variance for each of the dependent variables, which is assessed using Levene's test (Mayers, 2013).

The majority of these assumptions were met by the data. In cases where there were violations, these were considered within a framework of the larger analysis. MANCOVA is a very robust analysis, which can produce valid results on even ordinal data that has previously satisfied the criteria for analysis sufficiently strong for publication (Mayers, 2013). In order to satisfy the normality assumptions for a MANCOVA, LogVWM and LSM_{raw} were used instead of the original scaled scores, for reasons discussed previously.

The result of the MANCOVA was significant for group, (Wilk's λ = 0.795, $F(8,526)$ = 7.982, $p < 0.0001$, *partial* η^2 = 0.108, *power* = 1, Hotelling's Trace = 0.244, $F(8,524)$ = 7.847, $p <$ 0.0001, *partial* η^2= 0.109, *power* = 1), indicating a significant difference in performance on one or more of the working memory indices between the three groups. Subsequent univariate analyses indicate that significant between-group differences were identified in all indices except Visuospatial Short Term Memory (Table 5.12). The Bonferroni correction method was used to protect against an inflated familywise error rate, due to the multiple comparisons that were done, and was consequently set α=0.0125.

Table 5.12
The Result of a Between-Group MANCOVA on the AWMA

		Type III Sum of Squares	df	Mean Square	F	P	Partial Ɲ2	Observed Power
Group	VSTM	4483.650	2	2241.825	11.283	0.000*	0.078	0.970
	LogVWM	0.144	2	0.072	18.158	0.000*	0.121	0.999
	VSSTM	1142.212	2	571.106	2.421	0.091	0.018	0.282
	VSWM	3453.853	2	1726.926	7.356	0.001*	0.053	0.840

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory
*p< 0.0125

A pairwise comparison highlights the differences between groups (Table 5.13). Within the Verbal Short Term Memory Index (VSTM), significant differences were only found between the HIV-EU and HIV-UU group, with the HIV-EU group's performance being poorer. Within the Verbal Working Memory Index (VWM), significant differences were found between all groups, except for the HIV-I and HIV-EU groups indicating that the latter were statistically indistinguishable from each other. There are no significant differences between groups in the Visuospatial Short Term Memory Index (VSSTM), and significant differences were only found between the HIV-I and HIV-UU groups in the Visuospatial Working Memory Index (VSWM). The performance of the HIV-I and HIV-EU groups remains statistically indistinguishable from each other in Visuospatial Working Memory Index. Figure 5.1 presents these findings graphically.

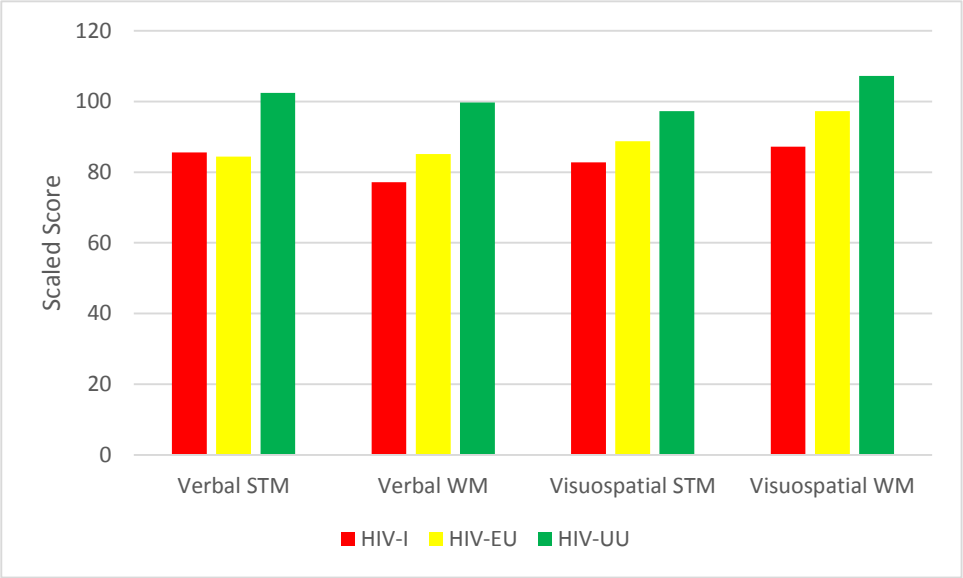

Figure 5.1. Comparative bar graph representing mean AWMA index performance of the three groups

The between-group working memory comparisons of the three groups indicated that verbal and visuospatial short-term memory performances remain less compromised under conditions of HIV infection and exposure than that of working memory performance. Conversely, both the verbal and visuospatial working memory performances of the HIV affected groups were significantly poorer than the unexposed control. The following section investigates the working memory profiles within each group.

Table 5.13
Pairwise Comparisons Between Groups on the Four Working Memory Indices

Dependent Variable			Mean Difference	Std. Error	p	98.75% CI Lower Bound	98.75% CI Upper Bound
Verbal Short Term Memory	HIV-I	HIV-EU	4.356	2.165	0.136	-1.901	10.613
		HIV-UU	-6.874	2.541	0.022	-14.219	0.471
	HIV-EU	HIV-I	-4.356	2.165	0.136	-10.613	1.901
		HIV-UU	-11.230	2.368	<0.001*	-18.073	-4.387
	HIV-UU	HIV-I	6.874	2.541	0.022	-0.471	14.219
		HIV-EU	11.230	2.368	<0.001*	4.387	18.073
Verbal Working Memory	HIV-I	HIV-EU	-0.027	0.010	0.016	-0.055	0.001
		HIV-UU	-0.068	0.011	<0.001*	-0.101	-0.036
	HIV-EU	HIV-I	0.027	0.010	0.016	-0.001	0.055
		HIV-UU	-0.041	0.011	<0.001*	-0.072	-0.011
	HIV-UU	HIV-I	0.068	0.011	<0.001*	0.036	0.101
		HIV-EU	0.041	0.011	<0.001*	0.011	0.072
Visuospatial Short Term Memory	HIV-I	HIV-EU	-3.353	2.358	0.469	-10.170	3.463
		HIV-UU	-6.000	2.769	0.093	-14.002	2.003
	HIV-EU	HIV-I	3.353	2.358	0.469	-3.463	10.170
		HIV-UU	-2.647	2.579	0.917	-10.102	4.809
	HIV-UU	HIV-I	6.000	2.769	0.093	-2.003	14.002
		HIV-EU	2.647	2.579	0.917	-4.809	10.102
Visuospatial Working Memory	HIV-I	HIV-EU	-6.592	2.353	0.016	-13.393	0.209
		HIV-UU	-10.148	2.762	0.001*	-18.132	-2.164
	HIV-EU	HIV-I	6.592	2.353	0.016	-0.209	13.393
		HIV-UU	-3.556	2.573	0.505	-10.994	3.883
	HIV-UU	HIV-I	10.148	2.762	0.001*	2.164	18.132
		HIV-EU	3.556	2.573	0.505	-3.883	10.994

*p< 0.0125.

Within-group profiles of working memory.

While an account of significant between-group differences allows a comparison of the novel HIV-EU group in relation to both viral exposure (HIV-I) and an unexposed, yet demographically similar control (HIV-UU), a within-group analysis of each group allows for the investigation of a working memory profile within each group. Consequently, a repeated measures ANCOVA was used to identify significant differences between the four working memory indices with each of the three groups. The variables representing LSM_{raw}, Age at Assessment, SRT and RCPM were used as covariates in all three sub-samples. Within the analysis of the HIV-I group, Mauchly's Test of Sphericity was violated ($W = 0.856$, $\chi^2 = 13.829$, $df = 5$, $p = 0.017$), and indicated that the variances of the differences between all combinations of the groups were not equal and increases the likelihood of a Type I error. However, three corrections were applied to these (Greenhouse-Geisser = 0.907, Huynh-Feldt = 0.979 and the Lower Bound = 0.333) violations

which automatically adjust the degrees of freedom. The ε (epsilon) test statistic is a measure of these three estimates, and indicates the degree of departure from sphericity, where a number close to 1 represents a small departure. Two of the three correctional statistics were very close to one, and the test was interpreted as being sufficiently valid. Bonferroni post hoc tests were also used to identify where these differences lay, with a corrected significance threshold of 0.0125. Table 5.14 provides a post-hoc summary of where these differences lay for the HIV-I group.

Table 5.14
Within-Group Repeated Measures ANCOVA Profile of Working Memory Performance (HIV-I: n = 95)

		Mean Difference	p	98.75% CI	
				Lower Bound	Upper Bound
Verbal Short Term Memory	Verbal WM	8.421*	<0.001*	4.501	12.341
	Visuospatial STM	2.839	0.639	-2.681	8.359
	Visuospatial WM	-1.611	1	-6.629	3.408
Verbal Working Memory	Verbal STM	-8.421*	<0.001*	-12.341	-4.501
	Visuospatial STM	-5.582*	0.001*	-10.066	-1.098
	Visuospatial WM	-10.032*	<0.001*	-14.545	-5.518
Visuospatial Short Term Memory	Verbal STM	-2.839	0.639	-8.359	2.681
	Verbal WM	5.582*	0.001*	1.098	10.066
	Visuospatial WM	-4.449*	0.01*	-8.789	-0.11
Visuospatial Working Memory	Verbal STM	1.611	1	-3.408	6.629
	Verbal WM	10.032*	<0.001*	5.518	14.545
	Visuospatial STM	4.449*	0.01*	0.11	8.789

*p<0.0125.

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory

Examining within-group differences among the AWMA index scores revealed significant differences in all three groups. Post hoc analysis of the significant differences in the HIV-I group (F(3,89) = 17.715, p < 0.001) showed that the Verbal Working Memory (VWM) Index was significantly lower than the other three indices (Verbal Short Term Memory (p<0.001; *Cohen's d* = -0.65), Visuospatial Short Term Memory (p<0.001; *Cohen's d* = -0.41), Visuospatial Working Memory (p<0.001; *Cohen's d* = -0.72). No other significant differences between indices were identified other than the Visuospatial Working Memory Index was significantly better than that of the Visuospatial Short Term Memory (p = 0.01; *Cohen's d* = -0.28).

Visuospatial working memory appears to be strongest index within this sample, followed by verbal short term memory and then visuospatial short term memory. Verbal

working memory appears to be a weakness in comparison to the other three. Since each subtest reflects slightly different aspects of working memory abilities within each index, further comparisons were run between the twelve subtests of the AWMA. Figure 5.2 represents these within-group index comparisons graphically. Table 5.15 represents the between-index post hoc differences of the twelve subtests of the AWMA in the HIV-I group.

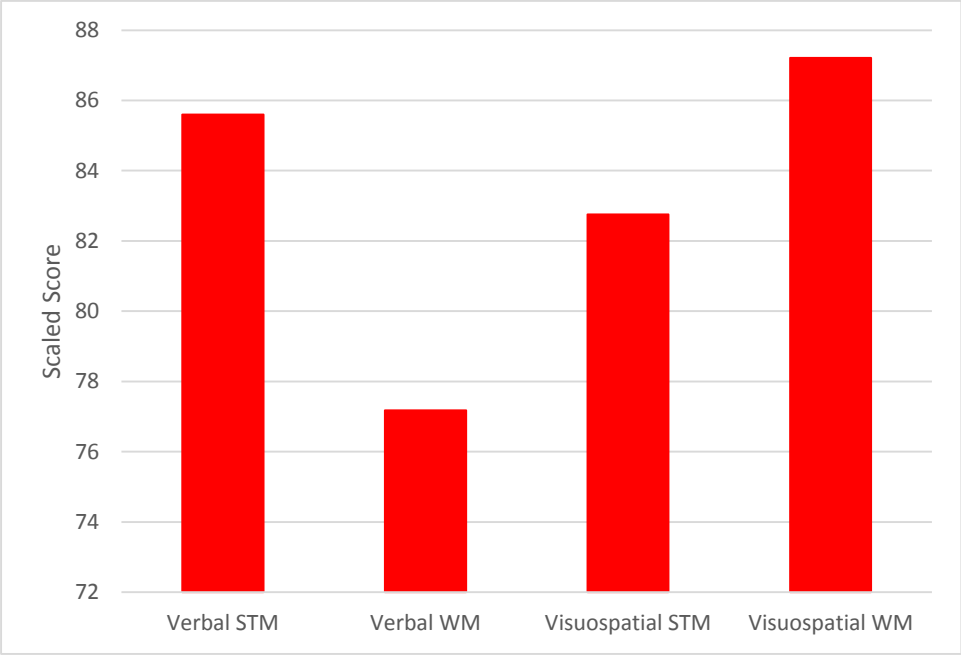

Figure 5.2. Bar graph representing mean AWMA index performance of the HIV-I group

Within the HIV-I group, it appeared that the poorest subtest scores constellate in verbal tasks, with working memory showing significantly more impairment than short term stores, with the exception of non-word recall which was in the average ranges. The reliance of the AWMA on English language was also apparent in the marked distinction between the Listening Recall subtest (reliant on words) and Counting Recall (reliant on numbers). This is, however, unsupported by correlations between the SRT and Listening Recall (r = 0.336) subtests being higher than between the SRT and Counting Recall (r = 0.267). While general performance on the visuospatial short term tasks significantly generally better than verbal tasks, it is the group's relative and significantly strength in Visuospatial Working Memory tasks that were most notable.

Table 5.15
Between Subtest Post Hoc Pairwise Differences Summary for the HIV-I group (n = 95)

Index	Subtest		Mean Difference	p	99.1% CI	
					Lower Bound	Upper Bound
VSTM	Digit Recall	Word Recall	8.671	<0.001*	2.82	14.521
		Nonword Recall	-14.758	<0.001*	-21.537	-7.978
	Word Recall	Nonword Recall	-23.428	<0.001*	-29.644	-17.213
VWM	Listening Recall	Listening Processing	-4.751	0.001*	-8.918	-0.583
		Counting Recall	-14.720	<0.001*	-22.202	-7.238
		Counting Processing	-15.921	<0.001*	-22.834	-9.008
		Backwards Digit Recall	-7.445	0.002*	-14.208	-0.682
	Listening Processing	Counting Recall	-9.969	<0.001*	-15.818	-4.121
		Counting Processing	-11.171	<0.001*	-16.275	-6.066
		Backwards Digit Recall	-2.695	1	-8.406	3.016
	Counting Recall	Counting Processing	-1.201	1	-3.083	0.681
		Backwards Digit Recall	7.275	0.005*	0.303	14.247
	Counting Processing	Backwards Digit Recall	8.476	<0.001*	1.898	15.054
VSSTM	Dot Matrix	Mazes Memory	3.095	1	-2.482	8.672
		Block Recall	0.671	1	-5.066	6.407
	Mazes Memory	Block Recall	-2.424	1	-8.631	3.783
VWM	Odd One Out Recall	Odd One Out Proc.	-1.418	0.166	-3.195	0.359
		Mr X Recall	-3.597	1	-10.946	3.752
		Mr X Processing	0.709	1	-5.758	7.177
		Spatial Recall	1.083	1	-5.65	7.817
		Spatial Processing	0.785	1	-5.253	6.824
	Odd One Out Proc.	Mr X Recall	-2.179	1	-9.329	4.971
		Mr X Processing	2.127	1	-3.835	8.09
		Spatial Recall	2.501	1	-3.857	8.859
		Spatial Processing	2.203	1	-3.266	7.672
	Mr X Recall	Mr X Processing	4.306	0.004*	0.235	8.377
		Spatial Recall	4.68	0.112	-0.978	10.338
		Spatial Processing	4.382	0.093	-0.83	9.594
	Mr X Processing	Spatial Recall	0.374	1	-5.024	5.771
		Spatial Processing	0.076	1	-3.445	3.596
	Spatial Recall	Spatial Processing	-0.298	1	-3.84	3.244

*p< 0.009

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory

The repeated measures ANOVA for the HIV-EU group did not violate Mauchly's Test of Sphericity. Within the HIV-EU group there were also significant within-group differences ($F(3,79) = 32.037$, $p < 0.001$, $\eta^2 = 0.549$). Table 5.16 presents post hoc analyses showing where these differences lay.

Table 5.16
Within-Group Repeated Measures ANCOVA Profile of Working Memory Performance (HIV-EU: n = 86)

Index		Mean Difference	p	98.75% CI	
				Lower Bound	Upper Bound
Verbal Short Term Memory	Verbal WM	-0.686	1	-5.787	4.415
	Visuospatial STM	-4.302	0.048	-9.339	0.735
	Visuospatial WM	-12.791	<0.001*	-17.974	-7.607
Verbal Working Memory	Verbal STM	0.686	1	-4.415	5.787
	Visuospatial STM	-3.616	0.142	-8.602	1.369
	Visuospatial WM	-12.105	<0.001*	-16.704	-7.505
Visuospatial Short Term Memory	Verbal STM	4.302	0.048	-0.735	9.339
	Verbal WM	3.616	0.142	-1.369	8.602
	Visuospatial WM	-8.488	<0.001*	-12.497	-4.479
Visuospatial Working Memory	Verbal STM	12.791	<0.001*	7.607	17.974
	Verbal WM	12.105	<0.001*	7.505	16.704
	Visuospatial STM	8.488	<0.001*	4.479	12.497

*p< 0.0125

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory

Post hoc analyses showed that the Visuospatial Working Memory Index was significantly higher than the other three indices (Verbal Short Term Memory (p<0.001; Cohen's d = 0.83), Verbal Working Memory (p<0.001; Cohen's d = 0.86), Visuospatial Short Term Memory (p<0.001; Cohen's d = 0.58). No other significant differences were identified. Visuospatial working memory appears to be strongest index within the HIV-EU group, followed by visuospatial short term memory. The two verbal indices appear to be weaknesses as they have relatively lower scores, with no significant difference between the working memory and short term memory components. Detailed analyses of where these differences were derived from were undertaken in a post-hoc analysis of the twelve subtests of the AWMA. Figure 5.3. represents this within-group index comparison graphically. Table 5.17 represents the between-index post hoc differences of the twelve subtests of the AWMA in the HIV-I group.

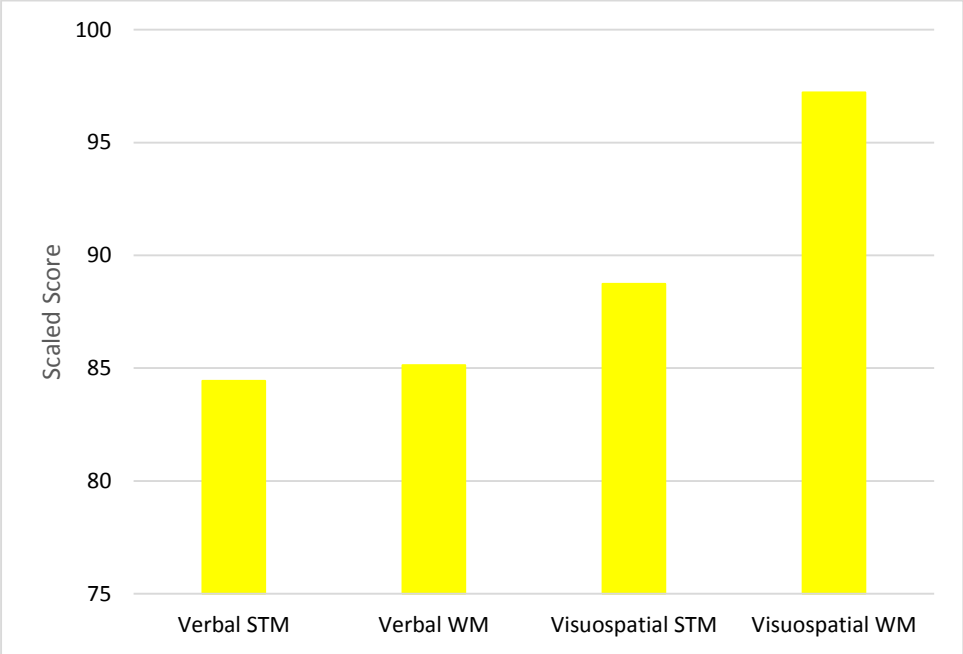

Figure 5.3. Bar graph representing mean AWMA index performance of the HIV-EU group

Within the subtest profile of the HIV-EU group, verbal skills appeared to be generally poorer than visuospatial skills. This is similar to the pattern identified in the HIV-I group. However, unlike the HIV-I group, there was no real distinction between Verbal Short Term and Verbal Working Memory performances. This is likely to be because in this sample, there was no pervasively elevated mean on the Non-Word Recall subtest like in the HIV-I group. However, in keeping with the profiling pattern evident in the HIV-I group within the verbal working memory subtests, HIV-EU participants were generally better at tasks relying on numbers rather than words.

Table 5.17
Between Subtest Post Hoc Pairwise Differences Summary for the HIV-EU group (n = 86)

					99.1% CI	
	Subtest		Mean Difference	p	Lower Bound	Upper Bound
VSTM	Digit Recall	Word Recall	14.327	<0.001*	7.037	21.616
		Nonword Recall	-4.706	1.000	-12.459	3.047
	Word Recall	Nonword Recall	-19.033	<0.001*	-25.650	-12.415
VWM	Listening Recall	Listening Processing	1.431	1.000	-2.527	5.390
		Counting Recall	-8.879	0.003*	-17.183	-.575
		Counting Processing	-9.377	0.001*	-17.633	-1.121
		Backwards Digit Recall	0.001	1.000	-9.300	9.302
	Listening Processing	Counting Recall	-10.310	<0.001*	-17.516	-3.105
		Counting Processing	-10.808	<0.001*	-17.804	-3.813
		Backwards Digit Recall	-1.430	1.000	-9.145	6.285
	Counting Recall	Counting Processing	-0.498	1.000	-2.024	1.029
		Backwards Digit Recall	8.880	<0.001*	2.710	15.051
	Counting Processing	Backwards Digit Recall	9.378	<0.001*	3.252	15.504
VSSTM	Dot Matrix	Mazes Memory	6.281	0.028	-.511	13.074
		Block Recall	2.348	1.000	-2.511	7.206
	Mazes Memory	Block Recall	-3.934	0.633	-9.620	1.753
VSWM	Odd One Out Recall	Odd One Out Proc.	1.041	1.000	-.795	2.876
		Mr X Recall	-0.102	1.000	-7.755	7.550
		Mr X Processing	6.369	0.287	-2.068	14.805
		Spatial Recall	3.343	1.000	-3.856	10.542
		Spatial Processing	4.112	1.000	-2.923	11.146
	Odd One Out Proc.	Mr X Recall	-1.143	1.000	-8.458	6.172
		Mr X Processing	5.328	0.857	-2.665	13.321
		Spatial Recall	2.302	1.000	-4.285	8.890
		Spatial Processing	3.071	1.000	-3.247	9.389
	Mr X Recall	Mr X Processing	6.471	0.004*	.305	12.637
		Spatial Recall	3.445	1.000	-2.579	9.470
		Spatial Processing	4.214	0.608	-1.849	10.277
	Mr X Processing	Spatial Recall	-3.026	1.000	-9.875	3.824
		Spatial Processing	-2.257	1.000	-8.354	3.840
	Spatial Recall	Spatial Processing	0.769	1.000	-2.318	3.855

*p< 0.009

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory

For the within-group profile of the HIV-UU group, Mauchly's Test of Sphericity was violated (W = 0.817, χ^2 = 17.357, df = 5, p = 0.004). However, the ε statistic produces correction very close to 1 (Greenhouse-Geisser = 0.874; Huynh-Feldt = 0.945; Lower Bound = 0.333), and still reveal significant within=group differences eve after correction, thereby not invalidating the results of the repeated measures ANOVA. Within the HIV-UU group, there were also

significant differences between the four working memory indices of the AWMA ($F(3,85) = 25.331$, $p < 0.001$, $\eta^2 = 0.226$). Table 5.18 presents the post hoc analyses of these differences.

Table 5.18
Within-Group Repeated Measures ANCOVA Profile of Working Memory Performance (HIV-UU: n = 92)

				98.75% CI	
Index		Mean Difference	p	Lower Bound	Upper Bound
Verbal Short Term Memory	Verbal WM	2.717	0.691	-2.700	8.135
	Visuospatial STM	5.120	0.014	-.074	10.313
	Visuospatial WM	-4.837	0.035	-10.262	.588
Verbal Working Memory	Verbal STM	-2.717	0.691	-8.135	2.700
	Visuospatial STM	2.402	0.420	-1.754	6.558
	Visuospatial WM	-7.554	<0.001*	-12.349	-2.760
Visuospatial Short Term Memory	Verbal STM	-5.120	0.014	-10.313	.074
	Verbal WM	-2.402	0.420	-6.558	1.754
	Visuospatial WM	-9.957	<0.001*	-14.080	-5.833
Visuospatial Working Memory	Verbal STM	4.837	0.035	-.588	10.262
	Verbal WM	7.554	<0.001*	2.760	12.349
	Visuospatial STM	9.957	<0.001*	5.833	14.080

*p<0.0125

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory

Post hoc analyses of the significant differences in the HIV-UU group showed that the Visuospatial Working Memory Index was significantly higher than both Verbal Working Memory (p<0.001; *Cohen's d* =-0.44) and Visuospatial Short Term Memory (p<0.001; *Cohen's d* = 0.56). There are no other significant differences between the indices. Visuospatial working memory appears to be strongest index within this sample, followed by verbal short term memory, verbal working memory and visuospatial short term memory respectively. A further detailed analysis of the working memory subtests was done using Bonferroni's post hoc method. Figure 5.4 represents this within-group comparison graphically. Table 5.19 represents the between-index post hoc differences of the twelve subtests of the AWMA in the HIV-I group.

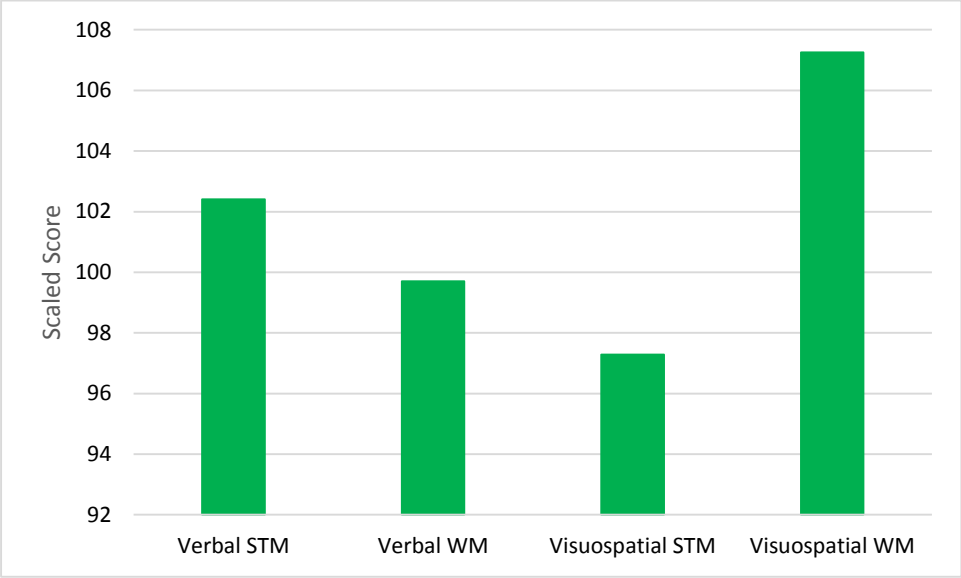

Figure 5.4. Bar graph representing mean AWMA index performance of the HIV-UU group

Within the HIV-UU group, the results provided evidence for a distinction between the two verbal score sets, where subtests reliant on short term verbal skill were marginally better than the working memory component, but showed no significant difference. There was evidence of significantly elevated performance on the Non-Word Recall scores, as well as the pattern of reliance on numbers being better than that on words (Counting Recall subtests). Again, similar to the HIV-I and HIV-EU groups, visuospatial working memory appears to be a relative strength of this sample.

An analysis of trends across the three within-group working memory profiles revealed a particular pattern of strengths and weaknesses. Verbal working memory performance was consistently the weakest index in the two HIV affected groups, but was not significantly weaker in the unexposed control group. The weaknesses in the Verbal Working Memory Index appeared to be derived from the groups' weaknesses in subtests reliant on words (Listening Recall), instead of digits (Counting and Backward Recall). In all three groups, verbal short term memory performance appeared to be stronger than the working memory component due to a general relative strength in the Non-Word Recall subtest. Visuospatial working memory was a persistent relative strength across the three groups.

Table 5.19
Between Subtest Post Hoc Pairwise Differences Summary for the HIV-UU group (n = 92)

	Subtest		Mean Difference	p	99.1% CI Lower Bound	99.1% CI Upper Bound
VSTM	Digit Recall	Word Recall	5.777	0.901	-2.929	14.483
		Nonword Recall	-10.409*	0.001	-19.611	-1.207
	Word Recall	Nonword Recall	-16.186*	0.000	-22.197	-10.174
VWM	Listening Recall	Listening Processing	5.441*	0.002	0.481	10.401
		Counting Recall	-7.799	0.042	-16.493	0.895
		Counting Processing	-8.646*	<0.001	-15.560	-1.731
		Backwards Digit Recall	-3.854	1.000	-11.471	3.762
	Listening Processing	Counting Recall	-13.240*	<0.001	-21.981	-4.499
		Counting Processing	-14.087*	<0.001	-20.881	-7.293
		Backwards Digit Recall	-9.296*	<0.001	-15.902	-2.689
	Counting Recall	Counting Processing	-0.847	1.000	-6.725	5.031
		Backwards Digit Recall	3.945	1.000	-5.571	13.460
	Counting Processing	Backwards Digit Recall	4.791	1.000	-2.639	12.222
VSSTM	Dot Matrix	Mazes Memory	2.216	1.000	-4.675	9.107
		Block Recall	1.849	1.000	-3.706	7.404
	Mazes Memory	Block Recall	-0.368	1.000	-6.516	5.781
VSWM	Odd One Out Recall	Odd One Out Proc.	0.446	1.000	-1.745	2.636
		Mr X Recall	0.891	1.000	-7.659	9.442
		Mr X Processing	4.914	1.000	-3.376	13.204
		Spatial Recall	5.676	0.176	-1.478	12.830
		Spatial Processing	6.196	0.030	-0.543	12.934
	Odd One Out Proc.	Mr X Recall	0.446	1.000	-7.927	8.818
		Mr X Processing	4.468	1.000	-3.639	12.575
		Spatial Recall	5.230	0.343	-1.817	12.278
		Spatial Processing	5.750	0.054	-0.794	12.294
	Mr X Recall	Mr X Processing	4.023	0.027	-0.316	8.361
		Spatial Recall	4.785	0.090	-0.891	10.461
		Spatial Processing	5.304	0.048	-0.677	11.286
	Mr X Processing	Spatial Recall	0.762	1.000	-5.121	6.645
		Spatial Processing	1.282	1.000	-4.228	6.791
	Spatial Recall	Spatial Processing	0.520	1.000	-3.231	4.271

*$p < 0.009$

Note. VSTM: Verbal Short Term Memory, VWM: Verbal Working Memory, VSSTM: Visuospatial Working Memory, VSWM: Visuospatial Working Memory

Between-group comparison on the NEPSY-II.

The second part of the first research question sought to identify whether there were significant differences between the six domains of neuropsychological performance of the NEPSY-II. Analyses of the relationship between HIV status and performance on the six collapsed NEPSY-II scales were therefore conducted using a multivariate analysis of covariance (MANCOVA), with

independent variable (HIV status), dependent variables (six collapsed indices of the NEPSY-II) and covariates (SRT_{BoxCox} and RCPM).

The MANCOVA results showed significant main effect differences on group (HIV status) (Wilk's λ = 0.570, $F(12,158) = 4.333$, $p<0.0001$, *partial* $\eta^2 = 0.25$, *power* = 0.997; Hotelling's Trace = 0.702, $F(12,156) = 4.565$, $p<0.0001$, *partial* $\eta^2 = 0.260$, power = 0.998). Subsequent post hoc univariate results indicated that there were significant differences found in all indices of the NEPSY-II, except Social Perception. These are reflected in Table 5.20. The between-group comparisons are represented graphically in Figure 5.5.

Table 5.20
Test of Between Subject Effects Identifying Significant Between Group Differences (NEPSY-II)

	Type III Sum of Squares	df	Mean Square	F	p	Partial Eta Squared	Observed Power
Attention & Executive Functioning	1710.574	2	855.287	8.622	<0.001*	0.170	0.857
Language	1170.316	2	585.158	6.162	0.003*	0.128	0.677
Memory & Learning	2086.726	2	1043.363	11.669	<0.001*	0.217	0.957
Sensorimotor Skills	1945.749	2	972.874	12.241	<0.001*	0.226	0.966
Social Perception	663.582	2	331.791	4.237	0.018	0.092	0.457
Visuospatial Processing	2311.847	2	1155.923	13.398	<0.001*	0.242	0.980

*p<0.008

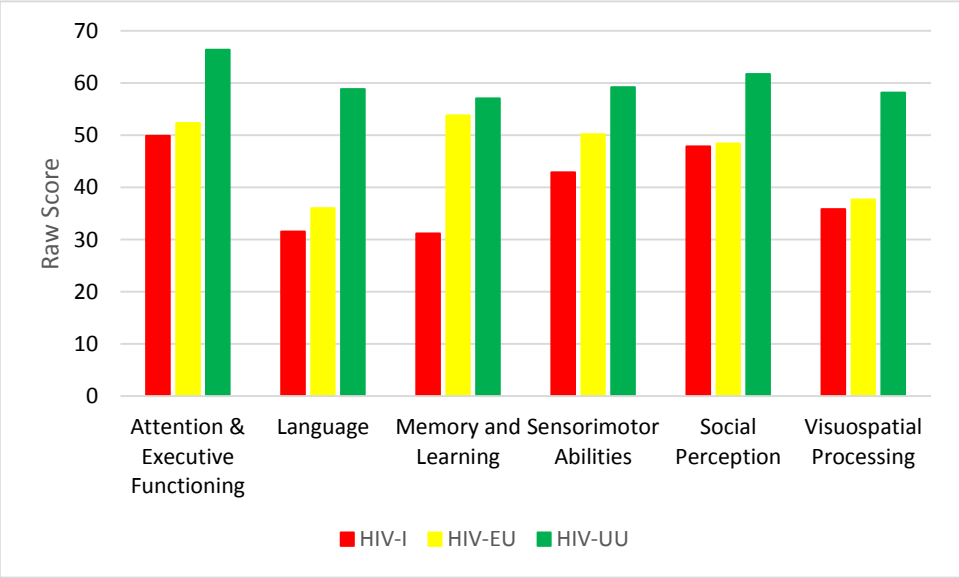

Figure 5.5. Comparative bar graph representing mean NEPSY-II domain performance of the three groups

Bonferroni post-hoc testing (shown in Table 5.21) revealed where these differences lay. Again, the Bonferroni correction method was used to protect against an inflated family-wise error rate and was consequently set to α=0.008.

Table 5.21
Pairwise Comparisons Between Groups on NEPSY-II Subtests

						99% CI	
Dependent Variable			Mean Difference	Std. Error	p	Lower Bound	Upper Bound
Attention & Executive Functioning	HIV-I	HIV-EU	-3.866	2.715	0.474	-12.067	4.336
		HIV-UU	-14.169	3.436	<0.001*	-24.551	-3.787
	HIV-EU	HIV-I	3.866	2.715	0.474	-4.336	12.067
		HIV-UU	-10.303*	3.280	0.007*	-20.213	-.394
	HIV-UU	HIV-I	14.169*	3.436	<0.001*	3.787	24.551
		HIV-EU	10.303*	3.280	0.007*	.394	20.213
Language	HIV-I	HIV-EU	-2.288	2.656	1.000	-10.313	5.737
		HIV-UU	-11.517*	3.362	0.003*	-21.675	-1.359
	HIV-EU	HIV-I	2.288	2.656	1.000	-5.737	10.313
		HIV-UU	-9.229	3.209	0.015	-18.925	.467
	HIV-UU	HIV-I	11.517*	3.362	0.003*	1.359	21.675
		HIV-EU	9.229	3.209	0.015	-.467	18.925
Memory & Learning	HIV-I	HIV-EU	-2.991	2.577	0.747	-10.778	4.796
		HIV-UU	-15.360*	3.262	<0.001*	-25.217	-5.503
	HIV-EU	HIV-I	2.991	2.577	0.747	-4.796	10.778
		HIV-UU	-12.369*	3.114	<0.001*	-21.777	-2.961
	HIV-UU	HIV-I	15.360*	3.262	<0.001*	5.503	25.217
		HIV-EU	12.369*	3.114	<0.001*	2.961	21.777
Sensorimotor Abilities	HIV-I	HIV-EU	-7.819*	2.430	0.006*	-15.160	-.478
		HIV-UU	-14.782*	3.076	<0.001*	-24.075	-5.490
	HIV-EU	HIV-I	7.819*	2.430	0.006*	.478	15.160
		HIV-UU	-6.963	2.936	0.060	-15.833	1.906
	HIV-UU	HIV-I	14.782*	3.076	<0.001*	5.490	24.075
		HIV-EU	6.963	2.936	0.060	-1.906	15.833
Social Perception	HIV-I	HIV-EU	0.361	2.412	1.000	-6.926	7.649
		HIV-UU	-7.714	3.053	0.040	-16.938	1.511
	HIV-EU	HIV-I	-.361	2.412	1.000	-7.649	6.926
		HIV-UU	-8.075	2.914	0.021	-16.880	.730
	HIV-UU	HIV-I	7.714	3.053	0.040	-1.511	16.938
		HIV-EU	8.075	2.914	0.021	-.730	16.880
Visuospatial Processing	HIV-I	HIV-EU	-2.015	2.542	1.000	-9.695	5.665
		HIV-UU	-15.801*	3.218	<0.001*	-25.522	-6.079
	HIV-EU	HIV-I	2.015	2.542	1.000	-5.665	9.695
		HIV-UU	-13.786*	3.071	<0.001*	-23.065	-4.506
	HIV-UU	HIV-I	15.801*	3.218	<0.001*	6.079	25.522
		HIV-EU	13.786*	3.071	<0.001*	4.506	23.065

*p< 0.008

Post-hoc analyses of the multivariate results indicated that there were no significant differences between any of the groups in the Social Perception Index. However, within the remaining five indices, the results showed that in all indices except Sensorimotor Abilities the performance of the HIV-I and the HIV-EU groups were statistically indistinguishable from each other. In all of these indices there were significant differences between the HIV-I and HIV-UU groups, and between the HIV-EU and HIV-UU groups, but notably not between the HIV-I and the HIV-EU groups. Conversely, within the Sensorimotor Index, the HIV-EU group was statistically indistinguishable from the HIV-UU group, and there were instead significant differences between the HIV-I group and the HIV-EU group, and between the HIV-EU group and HIV-UU group.

Within-group profiles on the NEPSY-II.

Attention is now drawn to the profiling of strengths and weaknesses of each of the three groups on the NEPSY-II. A repeated measures ANCOVA was used to identify significant differences between the six general domains of the NEPSY-II in each of the three groups. The variables representing, SRT_{BoxCox} and RCPM were used as covariates in all three sub-samples. The data from the HIV-I group showed that Mauchly's Test of Sphericity was not violated (W = 0.432, χ^2 = 21.056, df = 14, p = 0.102), and the results identified significant within-group differences $F(5,23) = 9.109$, $p < 0.001$, $\eta^2 = 0.664$, $power = 0.986$). Bonferroni post hoc tests were also used to identify where these differences lay, with a corrected significance threshold of 0.009. Table 5.22 provides a post-hoc summary of where these differences lay, while Figure 5.6. presents these within-group difference graphically.

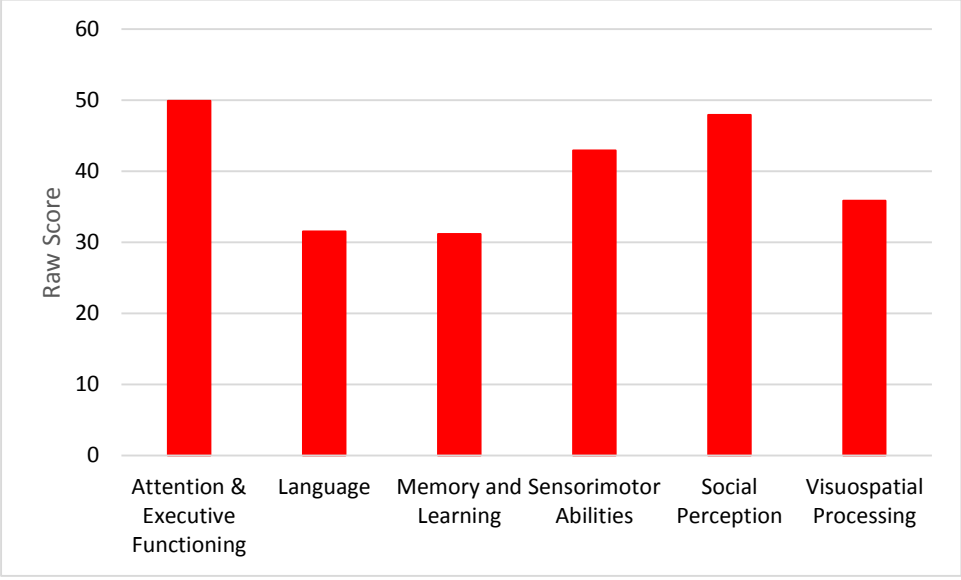

Figure 5.6. Bar graph representing mean domain NEPSY-II performance of the HIV-I group

Table 5.22
Within-Group Profile Post Hoc Pairwise Bonferroni Results (NEPSY-II) (HIV-I (n = 31))

					99.1% CI	
		Mean Difference	Std. Error	p	Lower Bound	Upper Bound
Attention & Executive Functioning	Language	18.346	1.64	<0.001*	11.92	24.77
	Memory & Learning	18.701	1.69	<0.001*	12.09	25.32
	Sensorimotor	6.98	2.02	0.03	-0.88	14.85
	Social Perception	1.99	1.79	1.00	-5.02	8.99
	Visuospatial Processing	14.039	1.68	<0.001*	7.49	20.59
Language	Memory & Learning	0.36	1.51	1.00	-5.55	6.26
	Sensorimotor	-11.362	1.95	<0.001*	-18.97	-3.75
	Social Perception	-16.361	2.15	<0.001*	-24.74	-7.98
	Visuospatial Processing	-4.31	1.93	0.51	-11.83	3.21
Memory & Learning	Sensorimotor	-11.718	2.17	<0.001*	-20.20	-3.24
	Social Perception	-16.716	2.07	<0.001*	-24.79	-8.65
	Visuospatial Processing	-4.66	1.47	0.06	-10.42	1.09
Sensorimotor	Social Perception	-5.00	2.21	0.49	-13.63	3.63
	Visuospatial Processing	7.055	1.63	<0.001*	0.67	13.44
Social Perception	Visuospatial Processing	12.054	1.80	<0.001*	5.01	19.10

*p< 0.009

Within the HIV-I group, Attention and Executive Functioning appears to be a relative strength, while Language, Memory and Learning and Visuospatial Processing are relative weaknesses.

The within-group performance of the HIV-EU group on the NEPSY-II was also conducted. Within the HIV-EU group, Mauchly's Test of Sphericity was also not violated (W = 0.433, χ^2 = 19.337, df = 14, p = 0.154), and the results identified significant within-group differences $F(5,21) = 8.180$, $p < 0.001$, $\eta^2 = 0.661$, power = 0.967) after appropriate Bonferroni corrections (α = 0.009). Table 5.23 provides a post-hoc summary of where these differences lay, while Figure 5.7 represents these within-group differences graphically.

Table 5.23
Within-Group Profile Post Hoc Pairwise Bonferroni Results (NEPSY-II) (HIV-EU (n = 29))

					CI (99.1%)	
		Mean Difference	Std. Error	p	Lower Bound	Upper Bound
Att. & Exec. Functioning	Language	16.312	2.683	<0.001*	5.72	26.91
	Memory & Learning	16.560	3.032	<0.001*	4.59	28.53
	Sensorimotor	2.140	2.316	1.000	-7.00	11.29
	Social Perception	3.962	2.971	1.000	-7.77	15.69
	Visuospatial Processing	14.654	2.364	<0.001*	5.32	23.99
Language	Memory & Learning	0.248	1.890	1.000	-7.21	7.71
	Sensorimotor	-14.172	1.898	<0.001*	-21.67	-6.68
	Social Perception	-12.350	2.377	<0.001*	-21.74	-2.97
	Visuospatial Processing	-1.658	2.144	1.000	-10.12	6.81
Memory & Learning	Sensorimotor	-14.420	2.217	<0.001*	-23.17	-5.67
	Social Perception	-12.598	2.364	<0.001*	-21.93	-3.27
	Visuospatial Processing	-1.906	2.203	1.000	-10.61	6.79
Sensorimotor	Social Perception	1.822	2.179	1.000	-6.78	10.43
	Visuospatial Processing	12.514	1.472	<0.001*	6.70	18.33
Social Perception	Visuospatial Processing	10.692	1.880	<0.001*	3.27	18.12

*p<0.009

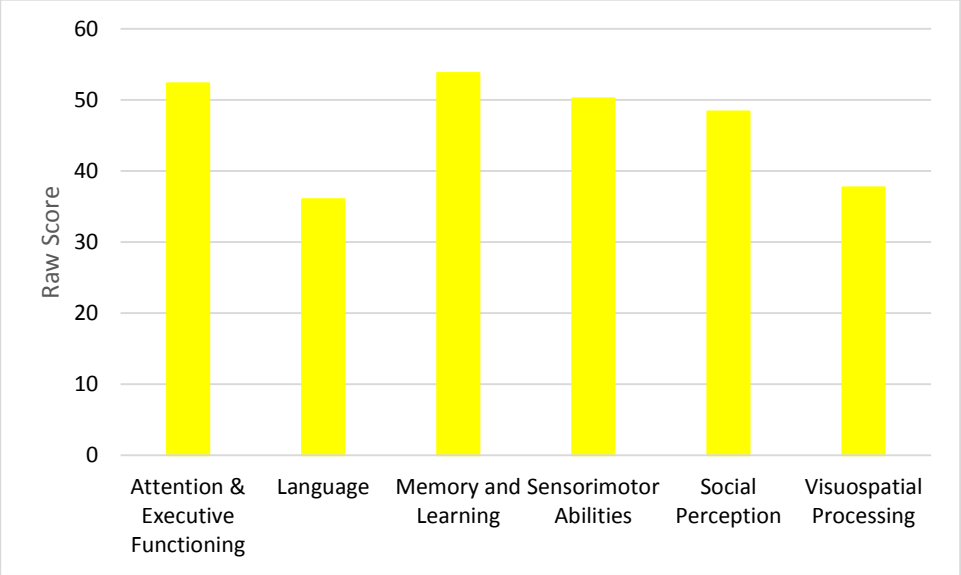

Figure 5.7. Bar graph representing mean domain NEPSY-II performance of the HIV-EU group

In a pattern very similar to that of the HIV-I group, Attention and Executive Function is identified as a relative strength, while Language, Memory and Learning, and Visuospatial Perception are identified as relative weaknesses.

Similarly, an examination of the HIV-UU group's performance on the NEPSY-II was also conducted. In the HIV-UU sample, Mauchly's Test of Sphericity was also not violated (W = 0.382, χ^2 = 21.246, df = 14, p = 0.097), and the results identified significant within-group differences $F(5,20) = 1.377$, $p < 0.001$, $\eta^2 = 0.256$, $power = 0.149$) after appropriate Bonferroni corrections ($\alpha = 0.009$). Table 5.24 provides a post-hoc summary of where these differences lay, while Figure 5.8. represents these within-group differences graphically.

Table 5.24
Within-Group Profile Post Hoc Pairwise Bonferroni Results (NEPSY-II) (HIV-UU (n = 29))

					CI (99.1%)	
		Mean Difference	Std. Error	p	Lower Bound	Upper Bound
Att. & Exec Functioning	Language	7.540	2.064	0.019	-0.61	15.69
	Memory & Learning	9.304	1.874	<0.001*	1.91	16.70
	Sensorimotor	7.163	1.730	0.006*	0.33	13.99
	Social Perception	4.669	2.474	1.000	-5.10	14.44
	Visuospatial Processing	8.247	1.752	0.001*	1.33	15.16
Language	Memory & Learning	1.764	1.306	1.000	-3.39	6.92
	Sensorimotor	-0.377	1.881	1.000	-7.80	7.05
	Social Perception	-2.871	1.601	1.000	-9.19	3.45
	Visuospatial Processing	0.707	1.523	1.000	-5.31	6.72
Memory & Learning	Sensorimotor	-2.141	1.883	1.000	-9.58	5.30
	Social Perception	-4.635	2.226	0.722	-13.42	4.15
	Visuospatial Processing	-1.057	1.666	1.000	-7.63	5.52
Sensorimotor	Social Perception	-2.494	2.101	1.000	-10.79	5.80
	Visuospatial Processing	1.084	1.605	1.000	-5.26	7.42
Social Perception	Visuospatial Processing	3.578	1.798	0.873	-3.52	10.68

*p< 0.009

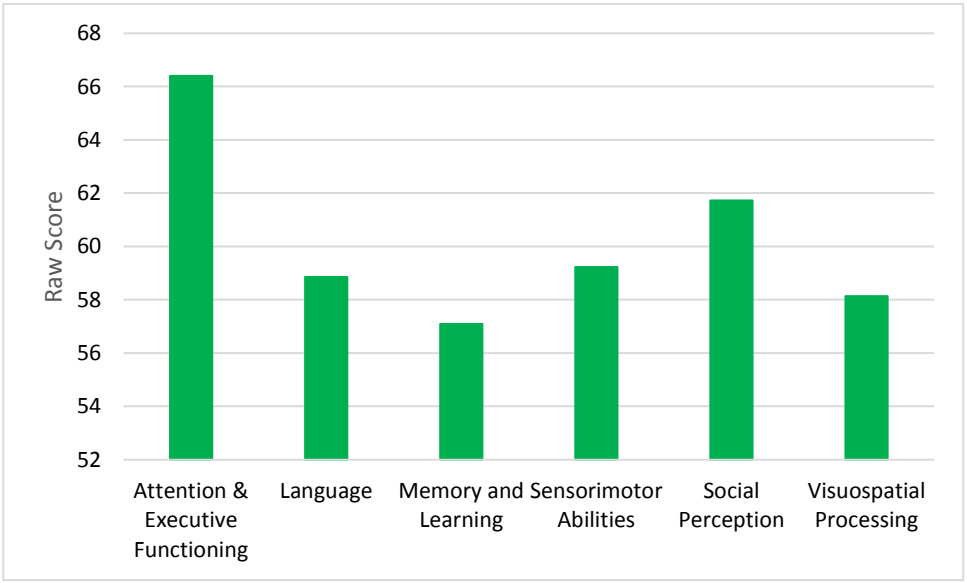

Figure 5.8. Bar graph representing mean domain NEPSY-II performance of the HIV-UU group

While the identification of these differences accounts for a limited variance (η^2 = 0.044), with poor power (*P* = 0.168), the Attention and Executive Functioning domain is again identified as a relative strength. There are no relative weaknesses, as the scores are relatively homogenous.

An analysis of trends across the three within-group neuropsychological profiles revealed a particular pattern of strengths and weaknesses. Within all three groups, the Attention and Executive Functioning domain was identified as a significant relative strength. Within the two HIV-affected groups, the domains of Language, Memory and Learning, and Visuospatial Processing were all identified as significant, relative weaknesses. However, this pattern of weakness was not apparent in the HIV-UU control group which had no identifiable relative weaknesses.

Investigation of Developmental Trajectories

This section reports on the results of confirmatory factor analyses (CFA) to assist in model comparison in two ways, a between-group, and a within-group comparison. The first, a multi-group CFA was used to compare the underlying latent factor structure of working memory between the three groups. The model used a four factor derivative of Baddeley's multicomponent model, which is aligned to the developmental fractionation and maturity of working memory within typically developing school beginners (see Figure. 5.9). The second use of CFA was to compare model fit within each of the three groups, to determine which theoretical account of working memory structure provides the best account of the data within each of the three groups.

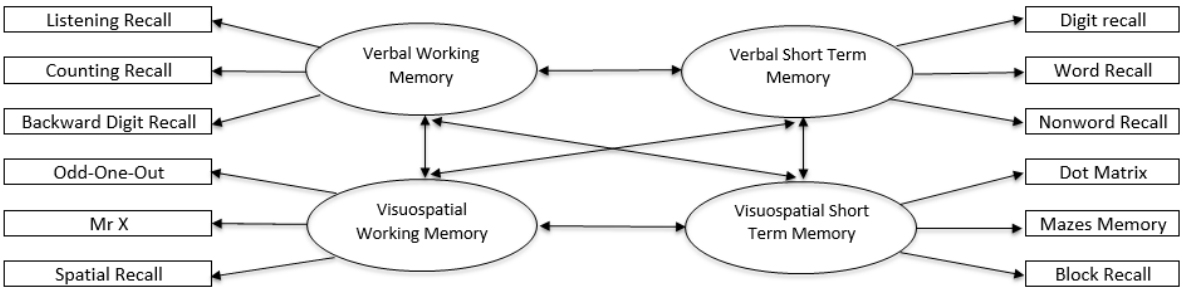

Figure 5.9. Developmental Fractionation of Working Memory in School Beginners

The choice of statistic used to indicate model-fit within CFA is contentious. Conventionally, the null hypothesis significance of the chi square test (χ^2) is the ultimate marker of model fit, where a non-significant result would mean that the observed sample co-variances do not differ significantly from the proposed (model-implied) population co-variances and the

model is accepted as showing good fit. This is considered an exact test fit. However, this test's sensitivity to large sample sizes and multivariate non-normal data sets makes it vulnerable to type 1 error. Given this, the research community (particularly in the social sciences) have come to developed and adopted 'approximate fit tests' (AFI's), which are a series of adjusted indices of the χ^2 statistic, which account for deviations in sample size, number of variables, number of degrees of freedom etc. (Barrett, 2007). This departure from binary null hypothesis significance testing is strongly rejected by purists in the scientific community who reject the approximate fit indices as a betrayal of scientific validity (McIntosh, 2007). There are a number of researchers who oppose this position, and vouch for the support of such indices under particular conditions. Hu and Bentler (1999) have released a series of 'golden rules' on the various cut off criteria for AFI's, but, supported by others (Markland, 2007) dissuade users from regarding them as absolute cut-off criteria for model fit. Commonly used indicators of model adequacy which are more sensitive to model fit than sample size include: the Bollen fit index (IFI), the goodness of fit index (GFI), the adjusted goodness of fit index (AGFI), and the root mean square error of approximation (RMSEA) which are known as absolute fit indices (AFI's). These determine how well the a priori model fits or reproduces the data according to expectations. The comparative fit index (CFI), the normed fit index (NFI), and the Ticker-Lewis index (TLI), are called relative fit indices. They provide a further measure of fit by comparing the hypothesised model against a null model in which the relationships between the latent variables are not specified and consequently set at 0. Fit indices greater than or equal to 0.90 are representative of a good fit. The root mean square error of approximation (RMSEA) also provides sound assessment of the degree to which the true model is reflected in the specified model. An RMSEA of 0.08 or lower is acceptable, and a value less than 0.05 is considered a good fit. (Alloway, Gathercole, & Pickering, 2006).

A multi-group confirmatory factor analysis (M-CFA) was used to examine the latent factor structure of working memory across the three groups, by testing for measurement invariance (also termed factorial invariance) simultaneously across three groups. An M-CFA with the 12 working memory tests of the AWMA was performed, using three tests to measure each of the four WM components according to the four-factor model of working memory expected of school beginners.

Measurement of factorial invariance assesses the degree to which a measure demonstrates similar factor structures with respect to the latent construct across groups.

Therefore, the presence of factorial invariance means that members of different groups respond to items in a similar manner, such that the operationalisations of the psychological constructs are similar across groups. Within the development of psychometric tests, the establishing of factorial invariance of a measure is reflective of the validity and reliability of the measure as it determines whether the conceptualisation of the latent constructs are different across groups. In such cases, the interpretation of any cross-group differences may be biased or incorrect since it is unknown whether differences are the result of true differences between groups in the latent constructs, or a result of differences in how members of different groups interpret and respond to test items (Varni, Limbers & Newman, 2009). In studies like the present study, however, the verification of factorial invariance across groups holds a different meaning, as there are clear distinctions by way of the independent variable (HIV status) as to why the groups are different, and as such it would not be unusual for there to be violations of factorial invariance.

Horn and McArdle (1992 as cited by Varni, Limbers, & Newman, 2009) defined hierarchical levels of factorial invariance, outlining the various ways in which groups may differ in their response to measures. This is typically assessed through several additive steps: (i) testing for configural invariance by defining the basic structure equation model to illustrate the data in all groups, in which all parameters were left free to co-vary in the three groups; (ii) testing for metric invariance, in which the factor loading was constrained to be equal in the three groups and was recognized as metric invariance; (iii) testing for scalar invariance through where the intercept of the factor loadings were constrained to be equal in the three groups, (iv) testing for mean differences in the latent factors, as the factor intercepts and residual variances were constrained to be equal, which is known as strict invariance (Byrne, 2010, Soliman, 2014).

An extension of Confirmatory Factor Analysis (CFA), Multi-group CFA was used in the current study to assess the invariance of measurement parameters across the three groups by using a series of nested models as described above. Table 5.25 summarises the results of the comparisons.

Table 5.25

Comparison of Multi-group CFA Measurement Invariance for AWMA Four Factor Model

	χ2	Df	χ2/df	p	NFI	IFI	TLI	CFI	RMSEA (CI 90%)
Configural	230.289	155	1.49	<0.001*	0.849	0.945	0.912	0.942	0.042 (0.03-0.054)
Metric	269.015	171	1.57	<0.001*	0.824	0.928	0.896	0.924	0.046 (0.035--0.056)
Scalar	443.987	195	2.28	<0.001*	0.709	0.813	0.769	0.808	0.069 (0.06-0.077)
Strict	506.123	219	2.31	<0.001*	0.669	0.781	0.763	0.778	0.07 (0.062-0.078)
	Δχ2	Δdf	Δχ2/df	Δp	ΔNFI	ΔIFI	ΔTLI	ΔCFI	ΔRMSEA (CI 90%)
Configural vs. Metric	38.726	16	0.09	<0.001*	0.025	0.017	0.016	0.018	0.004
Metric vs. Scalar	174.972	24	0.70	<0.001*	0.115	0.115	0.127	0.116	0.023
Scalar vs. Strict	62.136	24	0.03	<0.001*	0.04	0.032	0.006	0.03	0.001

*p< 0.05

In the M-CFA, all parameters remained free to co-vary to test for configural invariance across the three groups. The analyses revealed that the data were significantly different from the model (χ^2 =230.29, *df* = 155, *p*<0.001) which suggests an unacceptable model fit between the three groups. Additionally, while the fit indices are not vastly below acceptable cut off criteria, only the IFI and RMSEA are acceptable.

The factor loadings were constrained to be equal across both groups to examine the metric invariance. The results indicated an unacceptable model fit, (χ^2 =269.015, *df* = 171, *p*<0.001). The difference between the configural and metric invariance χ^2 values was significant (χ^2 = 38.726, *df* = 16, *p* = 0.001), and the differences in all of the fit index values were ≤.0.016. The values of the fit indices are shown in Table 5.25. The fit index values indicate that the regression slopes connecting the latent factors to the observed variables were not equal across the groups.

The intercepts and residuals were constrained to be equal across the two groups to examine the scalar invariance. The model that represented the scalar invariance yielded unsatisfactory values for both the χ^2 and fit indices (χ^2 =449.98, *df* = 195, *p*<0.001). There are significant χ^2 difference values between this and the metric model (χ^2 = 174.927, *df* = 24, *p*<0.001), with AFI differences all greater than 0.115.

The final test of factorial invariance is that of strict invariance. When all sources of variance in the model, as well as regression weightings (factor loadings) and intercepts are held

equal, the model again yields unsatisfactory values for both the χ^2 and AFI's (χ^2 =506.123, *df* = 219, *p*<0.001). The difference between the scalar and strict invariance χ^2 values was significant (χ^2 = 62.136, *df* = 24, *p*<0.001), and the differences in all of the fit index values were ≤.0.032.

These findings are unsurprising in light of the previous section documenting between-group multivariate analyses, and confirm the significant differences in the working memory structures between the three groups. They provide evidence that some of the groups do not possess the typical latent variable structures of working memory expected of school beginners. However, two of the three samples used in this study are considered atypical in their development because of their HIV status, and between-group differences were therefore to be expected. The next question to ask was whether the working memory structures expected of typical school beginners was evident or not within each subsample. In order to determine this, a within-group confirmatory factor analysis was conducted within each of the three groups on five possible models. In the series of models tested, paths between latent variables were left free to co-vary (see bi-directional arrows), as there were no theoretically informed assumptions about the direction or cause of influence (Alloway & Alloway, 2013). Within each case, the level of significance of the path weights between each observed variable and each latent factor was set to α = 0.05. Diagrams of the five theoretically tested models are provided below.

Model 1 (Figure 5.10) is a four factor model with separate domain specific working memory and short term memory constructs (Friedman & Miyake, 2000, Miyake et al., 2001, Alloway, 2007).

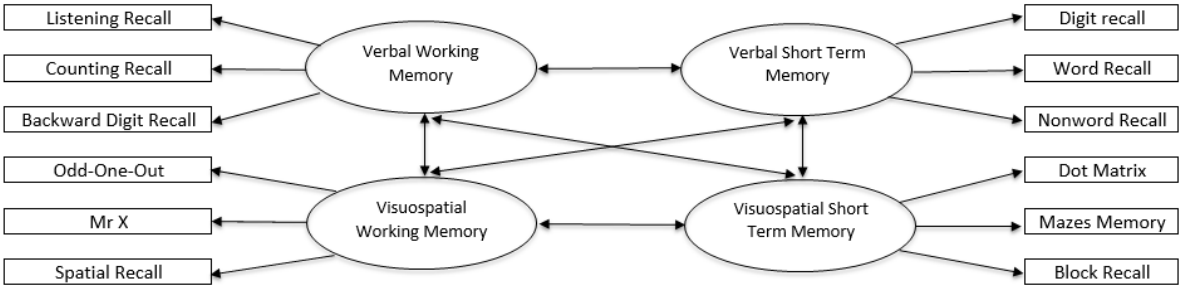

Figure 5.10. Four Factor Model (Adapted from(Alloway & Gathercole, 2006)

Model 2 (Figure 5.11) is typically referred to as Baddeley's Model, and is a three factor model with a single domain general working memory processor, and two separate storage components for verbal and visuospatial short term storage (Baddeley & Hitch, 1974; Baddeley, 2012).

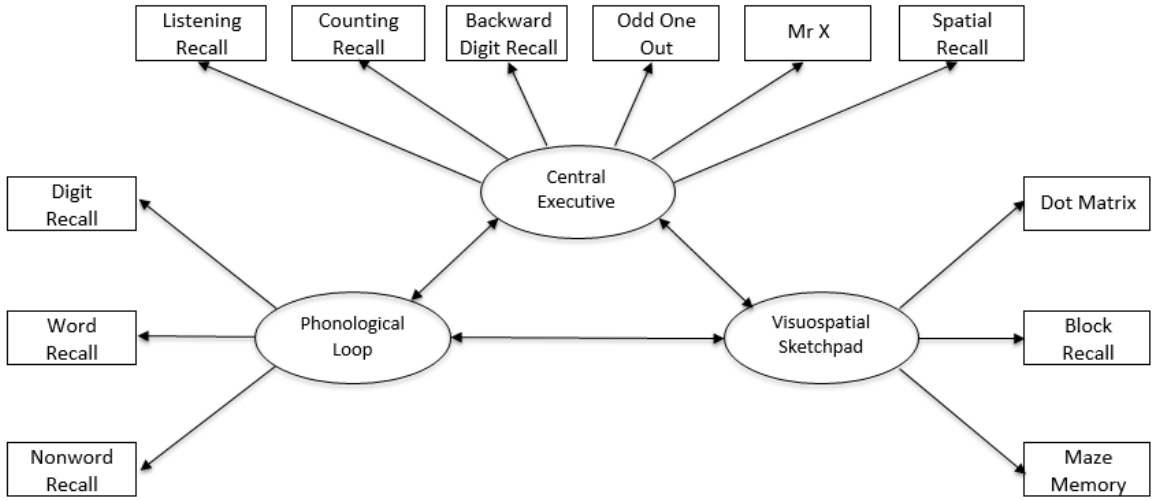

Figure 5.11. Three Factor Model (Adapted from Baddeley, 2012)

Model 3 (Figure 5.12) is a two factor domain specific model with latent constructs for verbal and visuospatial memory tasks. It seeks to determine whether working memory development within this age group is primarily fractionated along the axis of domain representation (Alloway, Gathercole & Pickering, 2006).

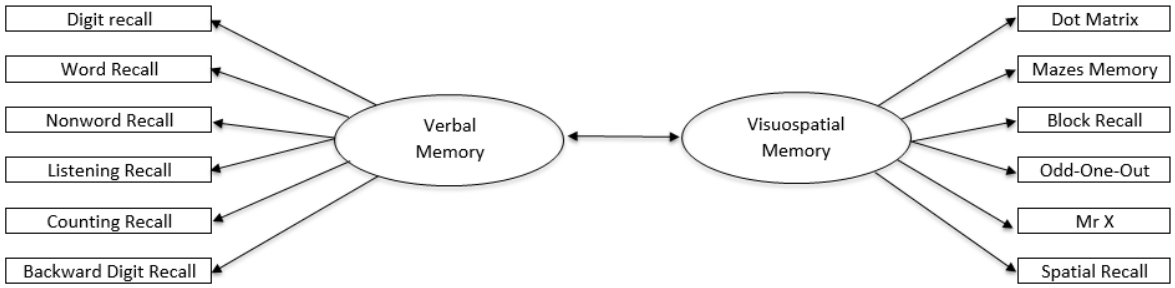

Figure 5.12. Domain Specific Two Factor Model (Adapted from Alloway, Gathercole & Pickering, 2006)

Model 4 (Figure 5.13) is a representationally neutral model, which also has a two factor structure which corresponds to short term and working memory constructs. Its domain generality seeks to identify whether the fractionation of working memory within school beginners is predominantly differentiated by a distinction between the processing short-term storage components of the model (Alloway, Gathercole & Pickering, 2006).

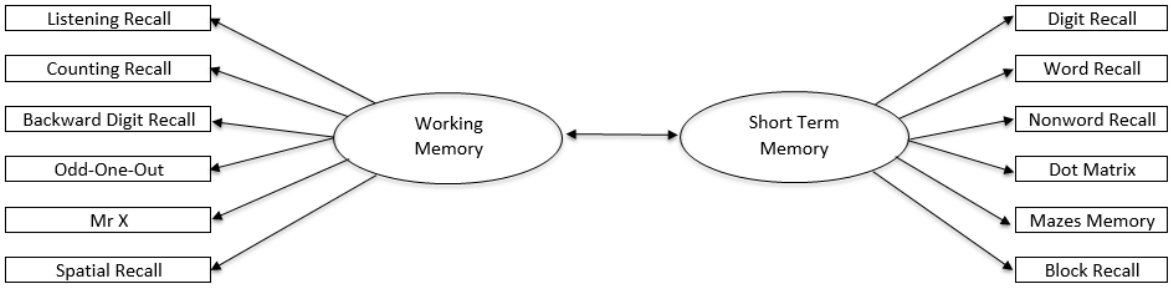

Figure 5.13. Storage vs. Processing Two Factor Model (Adapted from Alloway, Gathercole & Pickering, 2006)

Model 5 (Figure 5.14) is a general model which assumes that working memory is a unitary construct representative of 'g'. It is employed to differentiate working memory from general intellectual functioning (Alloway. Gathercole, Willis et al., 2004)

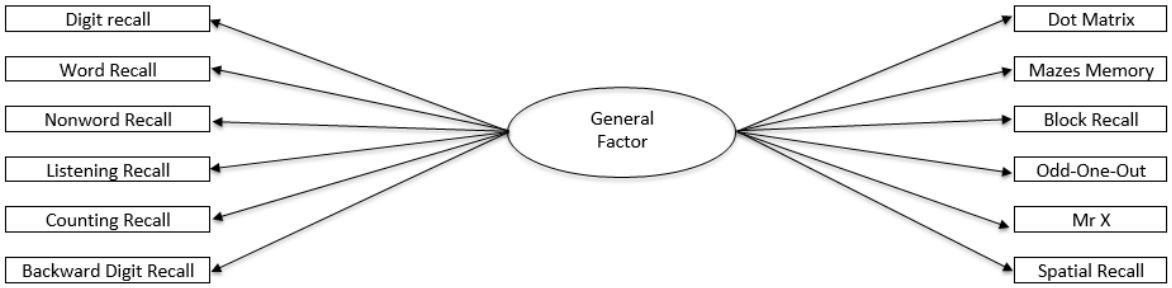

Figure 5.14. General Single Factor Model

Model 6 (Figure 5.15) is a four factor model representing mature working memory functioning in adults. It is fractionated according to the four dominant components of Baddeley's Multicomponent Model: phonological loop, visuospatial sketchpad, central executive and episodic buffer.

The presence of the episodic buffer in the working memory development of very young children has been established in a large sample of typically developing children (n = 633) as young as four years old, who speak English as their first language (Alloway, Gathercole, Willis et al., 2004). The distinct component of the episodic buffer was identified by a process of comparative model fit in a confirmatory factor analysis when measured using two sentence repetition tasks. This paradigm was chosen because the repetition of sentences involves the integration of temporary memory stores (the verbatim recall of content and order) with the

linguistic processing mechanisms of long term memory. The episodic buffer is believed to be integrate these two representations (Alloway & Ledwon, 2014; Rudner & Ro, 2008).

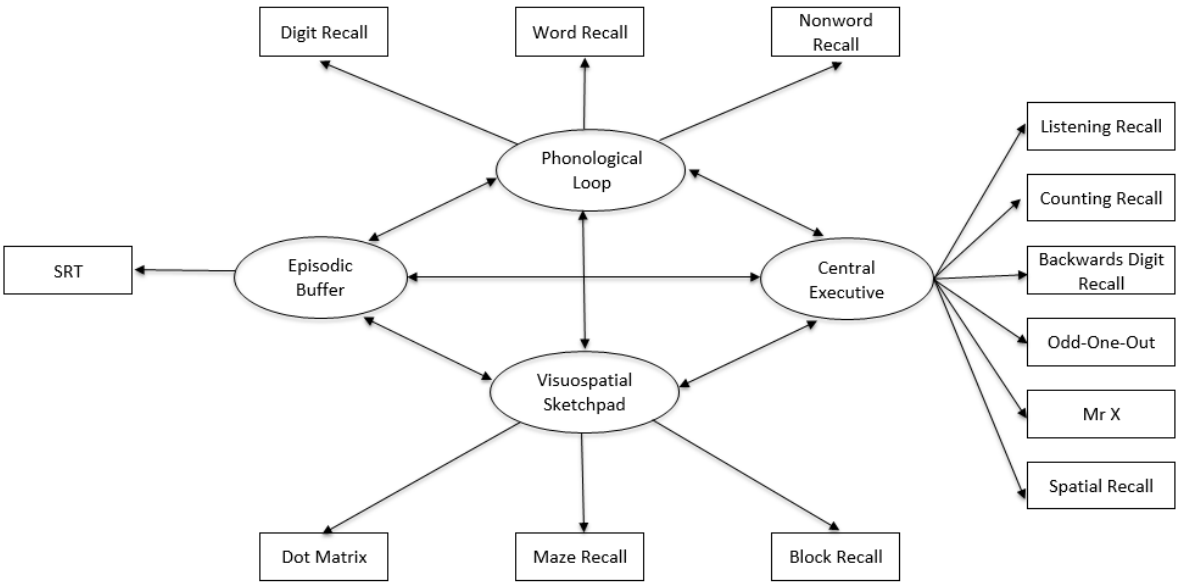

Figure 5.15. Four Factor Model of Mature Working Memory Performance (Adapted from Baddeley, 2012)

The authors chose two sentence repetition tasks, each consisting of ten sentences with age appropriate vocabulary. The first test contained simple active grammatical structures, while the second had both active and passive voices, and modified the embedding of either the subject or the object. Each sentence was spoken aloud, and the child was required to recall the sentence immediately. Scoring was indicated as either correct or incorrect, giving a maximum of 10 for each set.

This method was very similar to the Sentence Repetition Task (Redmond, 2005) employed in this study. It also had a combination of both active and passive voice with sentences of differing length. However, this model was not included in the analyses because of its assessment of the episodic buffer. None of the models in the three subsamples had an admissible solution using the SRT as a measure of the episodic buffer. This could be because it only relied on one observed variable, or because the correlations between it and other verbal measures were too high. However, this is unlikely as the highest correlation was between SRT and verbal short term memory (r = 0.458, p<0.001). Exploratory factor analysis, which used principal components analysis and Varimax rotation with Kaiser normalisations, shows that the SRT loads on the same factors as those of verbal short term memory, and the non-numerical

component of verbal working memory (Table 5.26). While such associations between executive functioning and language processing are theoretically accounted for (Rudner & Ro, 2008), the use of sentence repetition to measure the functioning of the episodic buffer is compromised in multilingual contexts such as this one. Jordaan (2012) has attempted to measure the functioning of the episodic buffer using Redmond's (2005) Sentence Repetition Test. In her study, three groups of children with differential English language proficiency were assessed; a group where both the children and their teachers only spoke English as an additional language (EAL context), a group who spoke English as an additional language (EAL) but were taught by teachers who spoke English as a first language (integrated context), and a group where both the children (L1) and their teachers only spoke English as their first language (integrated context). Significant differences were found between all three of these groups, with the group where neither the children nor teachers spoke English as a first language performed significantly lower than the EAL children in an integrated context (t = 4.31, p<0.001), and the L1 children in the integrated context (t = 7.89, p<0.001). There were also significant differences between the L1 children, and EAL children who were taught in the integrated context (t = 2.33, p = 0.0222, α < 0.05), with the former attaining higher results. Differences in performance between these three groups from different linguistic contexts would suggest that the SRT was influenced by variable levels of English proficiency, and the author conceded that the assessment of working memory functioning in English is compromised in contexts of multilingualism.

Table 5.26
Rotated Component Matrix for Varimax Factor Loading

	Component		
	1	2	3
Sentence Repetition Test	-0.022	**0.603**	**0.560**
Digit Recall	0.358	**0.641**	0.172
Word Recall	0.342	**0.734**	0.229
Nonword Recall	0.224	**0.847**	-0.006
Listening Recall	0.300	0.368	0**.529**
Counting Recall	**0.609**	0.286	0.331
Backwards Digit Recall	0.399	0.235	**0.609**
Dot Matrix	**0.818**	0.212	0.081
Mazes Memory	**0.750**	0.342	0.117
Block Recall	**0.811**	0.173	0.128
Odd One Out Recall	**0.628**	0.281	0.332
Mr X Recall	**0.717**	0.115	0.327
Spatial Recall	**0.743**	0.177	0.285

Note. Principal Component Analysis was employed as the extraction method, with Varimax Rotation Method with Kaiser Normalization. The rotation converged after 6 iterations. Factor loadings > 0.40 are in boldface.

Within this study, the SRT was used primarily as a measure of English proficiency within a sample who all spoke more than one language. To that end, phonological content in the temporary storage systems would not have the same semantic associations in long term memory because of reduced understanding in the child's second (or sometimes third or fourth) language. While there is local evidence that the processes of verbal working memory can be adequately measured in bilingual children using an additional language (English) (Cockcroft, 2014), it is unclear how this could be translated to the assessment of the episodic buffer in young, linguistically diverse children. This is because the children in that study (Cockcroft, 2014) were found to have a sound degree of English proficiency. The one available study which has attempted to assess the episodic buffer in bilingual children did so through the triangulation of three long term verbal measures: category fluency (the generation of as many items as possible in the categories of food and animals in a 60 second period), paired recall (the learned connection of pairs of words across six learning-and-recall trials), and memory for stories (free recall from three age appropriate stories). Within the study's bilingual group, the tests were administered in the child's dominant language. Replication of this method within the present study was deemed impossible, and the assessment of the episodic buffer in the present study was therefore not undertaken.

The following hypotheses were used to assess the degree of model fit within each group.

1. The HIV-UU group should have typical working memory development. This is characterised by a non-significant chi-square test result, and sufficiently satisfactory AFI's on Model 1.
2. The HIV-infected and HIV-EU groups should display signs of atypical working memory development. This is characterised by significant chi square results on a chi-square test and weak AFI's on Model 1.

Table 5.27 shows the comparisons between the chi-square and approximate fit indices (AFI's) of all three groups on the five models. The CFA within the HIV infected group (HIV-I) indicated that all models had a significant chi square result ($p = 0.05$), suggesting that none of them had sufficient model fit. Model 1, the four-factor model, appeared to have marginally better AFI's, with strong goodness of fit indices (NFI = 0.870, CFI = 0.953), and a low RMSEA (0.07).

Table 5.27

Comparison of CFA Results for Three Groups

			Chi Square			Approximate Fit Indices						
			χ^2	df	p	NFI	GFI	AGFI	CFI	IFI	NFI	RMSEA
	1	Four Factor	70.19	48	0.02*	0.870	0.889	0.819	0.953	0.955	0.936	0.07
	2	Three factor	78.768	51	0.008*	0.854	0.875	0.809	0.941	0.943	0.924	0.076
HIV-I	3	Verbal vs. Visuospatial	105.572	53	<0.0001*	0.804	0.842	0.768	0.889	0.892	0.862	0.103
	4	STM vs. WM	105.852	53	<0.0001*	0.804	0.833	0.754	0.888	0.891	0.861	0.103
	5	General	122.026	54	<0.0001*	0.774	0.815	0.733	0.856	0.86	0.824	0.116
	1	Four Factor	79.035	48	0.003*	0.828	0.869	0.786	0.921	0.924	0.891	0.087
	2	Three Factor	82.242	51	0.004*	0.821	0.868	0.797	0.920	0.923	0.897	0.085
HIV-EU	3	Verbal vs. Visuospatial	103.607	53	<0.0001*	0.774	0.823	0.739	0.871	0.875	0.839	0.106
	4	STM vs. WM	107.774	53	<0.0001*	0.765	0.820	0.736	0.860	0.865	0.826	0.11
	5	General	117.035	54	<0.0001*	0.745	0.806	0.720	0.839	0.844	0.804	0.117
	1	Four Factor	53.262	48	0.279	0.604	0.912	0.857	0.923	0.939	0.894	0.035
	2	Three Factor	62.726	51	0.126	0.482	0.894	0.838	0.788	0.833	0.725	0.05
HIV-UU	3	Verbal vs. Visuospatial	65.183	53	0.122	0.462	0.897	0.848	0.779	0.821	0.725	0.05
	4	STM vs. WM	64.397	53	0.136	0.469	0.893	0.842	0.793	0.833	0.743	0.049
	5	General	70.237	54	0.068	0.420	0.889	0.839	0.706	0.758	0.64	0.057

*$\chi 2 < 0.05$

Similarly, none of the models within the HIV-EU group attained a non-significant chi-square result which would have indicated sufficient model fit. The AFI's were again strongest in Model 1 (NFI = 0.828, CFI = 0.921, RMSEA = 0.087) and Model 2 (Baddeley) (NFI = 0.821, CFI = 0.920, RMSEA = 0.085). Conversely, all of the models in the HIV-UU group contained a non-significant chi square result, indicating sufficient model fit. The AFI's would again suggest that Model 1 has the best fit.

These within-group comparisons were confirmed by a series of chi square difference tests (Table 5.28). This statistic conducted a nested-model comparison between two competing models. It assumed that because solutions with fewer factors will always fit worse, a significant p value is indicative that the solution with more factors is a better model fit. It was not possible to compare the two two-factor models against each other as they each possess the same number of degrees of freedom which renders the calculation undefined. The results indicated that within all three groups, Model 1 (four factor) provided a significantly better account of the data than each of the other models. Table 5.29 presented the ranked order of how well each model fitted within each group. Path diagrams of the standardised estimates of each of these models can be found in Appendix J.

Table 5.28
Ranked Comparisons of Chi Square Difference Tests

Ranking	HIV-EI		HIV-EU		HIV-UU	
1	Model 1	AWMA	Model 2	AWMA	Model 1	AWMA
2	Model 2	Baddeley	Model 1	Baddeley	Model 4	STM vs.WM
3	Model 4	STM vs.WM	Model 4	STM vs.WM	Model 3	Verbal vs. V-spatial
4	Model 3	Verbal vs. V-spatial	Model 3	Verbal vs. V-spatial	Model 2	Baddeley
5	Model 5	General	Model 5	General	Model 5	General

It appears that the four factor model in all three groups provided the best relative model fit, and is thus the one used for between group comparisons. When relying on the chi-square results using Model 1 (four factor), both hypotheses were found to be true, with the HIV-EI and HIV-EU groups yielding a significant result, and the HIV-UU yielding a non-significant result. This suggests that the working memory development in the two clinical HIV-affected samples is atypical and does not follow the componential structure usually apparent in typical samples.

Table 5.29

Chi Square Difference Tests for Within Model Fit Comparisons

			HIV-EI				HIV-EU				HIV-UU			
			Model 2	Model 1	Model 4	Model 3	Model 2	Model 1	Model 4	Model 3	Model 2	Model 1	Model 4	Model 3
	$\Delta\chi^2$		43.258	51.836	16.174	16.545	34.793	38	9.261	13.248	7.511	16.975	5.84	5.054
Model 5	Δdf		3	6	1	1	3	6	1	1	3	6	1	1
	Δp		<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.002	<0.0001	0.005	0.009	0.015	0.024
Better Fit			Model 2	Model 1	Model 4	Model 3	Model 2	Model 1	Model 4	Model 3	Model 2	Model 1	Model 4	Model 3
	$\Delta\chi^2$			8.578	27.084	26.084		3.207	25.532	21.365		9.464	1.671	2.457
Model 2	Δdf	HIV-EI		3	2	2	HIV-EU	3	2	2	HIV-UU	3	5	5
	Δp			0.03	<0.0001	<0.0001		0.361	<0.0001	<0.0001		0.02	0.433	0.29
Better Fit				Model 1	Model 2	Model 2		Model 1	Model 2	Model 2		Model 1	Model 4	Model 3
	$\Delta\chi^2$				35.662	35.382			28.739	24.572			11.135	11.921
Model 1	Δdf				5	5			5	5			5	5
	Δp				<0.0001	<0.0001			<0.0001	<0.0001			0.04	0.035
Better Fit					Model 1	Model 1			Model 1	Model 1			Model 1	Model 1

*$\chi 2 < 0.05$

Conclusion

This chapter has presented the statistical analyses linked to the two broad research aims of this study. The first main finding is that significant differences exist between the three groups, primarily on measures of verbal and visuospatial working memory. With respect to the second aim, confirmatory factor analyses of five theoretically informed models indicated that, while the two HIV-affected groups show significant deviations in their working memory model structure when compared to the typically developing group, the developmentally expected four-factor structure is sufficiently robust in accounting for atypical development and remains the most appropriate model structure in the two atypical groups. The following chapter discusses these results with regard to their deviations from typical functioning in both the HIV-I an HIV-EU samples, and locates these findings within the literature reviewed at the outset of this thesis.

Chapter 6: Discussion

Introduction

This chapter critically discusses the results obtained from the analyses conducted in Chapter 5. The body of this discussion begins with a descriptive overview of the findings within each of the three subsamples. Five salient theoretical points of interest are then discussed cohesively. The chapter ends with a summative discussion of the theoretical and practical implications of these findings to the relevant groups, and their contribution to future research. The findings pertaining to the HIV-I group are presented first, followed by the HIV-EU group, and then HIV-UU group.

The rationale for this research centred on the investigation of working memory performance within two atypical paediatric populations – HIV infected, and HIV-exposed children. Research considering the relationship between working memory and HIV is presently welcomed by both the research and clinical fraternities, as it is able to inform clinical intervention, as well as contribute to the well-established foundation of previous working memory research. This is owed largely to a 'perfect storm' regarding the ontological location of recent working memory research within the current contexts of cognition, neurobiology, public health and education. The idea of an active processor within the memory system is not a new one, and the construct has been afforded the benefit of a strong empirical foundation with almost forty years of experimental and neuropsychological data providing theoretical support for its functioning and structure. Agreement on how exactly on how the structure and function of working memory operate together is, however, still contentious, and fuels much debate and, importantly, further research within the research community.

The robustness of the multicomponent model (Baddeley, 2000) is often disregarded by critics following the publication of unexpected results which do not align to theoretically informed expectations. In response to these criticisms, Baddeley (2012) retorts that negative results should instead be praised for their capacity to create further opportunities for investigation and validation. This is the mark of good science. Baddeley (2012) posits that it is the simple contribution of a simple theory (the multicomponent model), coupled with a few sound methods of investigation (such as dual task processing) which creates a theoretical working memory 'map' by which a construct is discovered (Toulmin, 1953, as cited by Baddeley, 2012). Here, the success of the theoretical development of a construct is judged by its productiveness, rather than its predictive accuracy, and future research is welcomed as filling empty spaces on this theoretical map.

Some of the well-established locations on this theoretical map of working memory refer to empirically sound ideas regarding its structure and function, particularly in typically developing children. Working memory has long been considered as having a fractionated, modular structure with different components responsible for the active, processing skills, while others are responsible for domain specific storage. The relative maturation of this fractionation has been identified as being different for children when compared to adults.

Working memory is also essential for scholastic success, and is pivotal in the acquisition of basic literacy and numeracy in school beginners as it plays a large role in learning to read, write, spell, as well as success in arithmetic and general problem solving. Working memory also appears to be relatively robust to the effects of socioeconomic status, maternal education, and to a lesser degree, second language psychometric assessment, in comparison to conventional methods of cognitive assessment. In fact, it has been found to be particularly efficient in bilingual samples - suggestive of a possible working memory bilingual executive functioning advantage, although this is strongly contested by equivocal research evidence. There is also growing evidence of the functional profiles of working memory in atypical samples, particularly those with neurodevelopmental disorders (ADHD, DCD, ASD, SLI, dyslexia and dyscalculia (Alloway, 2006b; Alloway & Gathercole, 2006b; Alloway & Gathercole, 2006c; Gathercole & Baddeley, 1990; Henry, 2012; Lazar & Frank, 1998; Passolunghi & Siegel, 2001, 2004; Rucklidge & Tannock, 2002; Steele, Minshew, Luna, & Sweeney, 2007; Swanson, 2006).

Baddeley's (2012) requirement for any good theory located in good scientific epistemology, ensures that there are a number of empty spaces on this theoretical map as well. One of these empty spaces refers to the working memory performance in atypical paediatric samples which is not caused by the interaction of neurogenetic elements common to many neurodevelopmental disorders (ADHD, SLI), like that of HIV infection. It is also unclear what the working memory functioning looks like in an HIV-EU paediatric population; a growing yet under-researched population which is quickly overtaking the number of children born HIV positive in Sub-Saharan Africa (Filteau, 2009). Previous research regarding working memory profiles of atypical populations has also been done on relatively homogenous, Western, English-speaking samples who have a familiarity with psychometric testing. However, the majority of children in the world, particularly those affected by HIV, are not from this context. Coupled with this, increasing levels of international migration have resulted in the influx of diverse, linguistically-mixed communities of children who require assistance from formal school systems for their academic success (Hashin,

2005; Rossi, 2008; Sward & Rao, 2008). The conventional methods of assessment of cognitive function have exacerbated limitations within these contexts, and clinicians, researchers and teachers are fast having to consider alternative ways of assessing and optimising cognitive potential. Working memory provides a promising alternative to current limitations in conventional psychometric assessment in its potential to contribute to these theoretical and practical empty spaces. The present study's focus on working memory within the vulnerable populations of HIV-affected children offers a promising contribution to the 'map'. The following three sub-sections summarise the main findings within each of the three groups assessed.

Working Memory Profiles of the HIV-I, HIV-EU and HIV-UU Groups

The working memory profiles of the HIV-Infected (HIV-I) sample.

The results of this study support and confirm the large body of knowledge regarding the- impaired and often atypical development and functioning of working memory in the context of HIV infection (Ernst, Chang & Arnold, 2003; See also: Farinpour, Martin, Seidenberg, Pitrak, Persell, Mullane, et al., 2000; Hinkin, Hardy, Mason, Castellon, Lam, Stefaniak, & Zolnikov, 2002; Koekkoek, et al, 2008). The negative effect of HIV infection was apparent in both the between-group comparisons of the HIV-I group, and the within-group profiling of strengths and weaknesses within this subsample.

The between-group analyses indicated that the performance of the HIV-I group was significantly worse than the matched HIV-UU control group in the verbal and visuospatial working memory domains. However, the HIV-I group's performance on the verbal and visuospatial short term storage tasks was not significantly distinguishable from either the HIV-UU control group, or the HIV-EU group.

The within-group profile of the HIV-I group revealed that verbal working memory was a relative weakness within this group, with significantly lower scores than all other indices. The mean score of this index was also below average ranges of performance. Conversely, visuospatial working memory was a relative strength and was significantly better than all but one other index (verbal short term memory). Despite its relative strength, the mean score of this index fell, however, within the low average range of performance (M = 87.21, SD = 16.38).

A more detailed analysis of subtest contributions to these differences revealed that within the verbal short term storage, Word Recall was particularly poor (M = 77.54, SD = 14.11, $p<0.001$*), while Nonword Recall was a particular strength (M = 100.96, SD = 17.87, $p<0.001$*).

Relatively elevated, or above average performance on the Nonword Recall is apparent in all three groups of the sample. Performance on the Nonword Recall subtest is correlated strongly with learning additional languages, which could explain some of the associations in this study, as all the participants could speak at least two languages (Gathercole, 2006; Haughey, 2002). A detailed discussion of the relationship between language learning and working memory performance will follow later in this chapter. Digit Recall appeared to be a more robust measure of short term verbal recall possible because of its reliance on numbers and not English words for representation. Similarly, within the verbal working memory index, the HIV-I group faired poorer on the two subtests reliant on linguistic representation and rehearsal within the working memory system (Listening Recall and Listening Processing), while measures of the same construct which employed numbers (Counting Recall, Counting Processing and Backwards Recall) were more robust to the effects of decay. This preference for tasks employing numbers had perhaps become a more automated skill in EAL children who may have more exposure to English numbers than English words and sentences. There were no particularly notable strengths or weaknesses within either the visuospatial short term or working memory subtests, besides the processing component of Mr X being significantly better than the recall component.

When more detailed cognitive functioning of the HIV-I group was explored using the NESPY-II, the group's performance also served to support the large body of evidence that general neuropsychological performance is impaired in the context of HIV infection (Abubakar, van Baar, Van de Vijver, Holding, & Newton, 2008; ; Armstrong, Seidel, & Swales, 1993; Foster at al., 2006; Levenson 1992; Llorente, 2003; McGrath at al., 2006; Nozyce et al., 2006; Potterton at al., 2009; Sherr, Meuller & Varrall. 2009; Van Rie, et al., 2007; Pollack et al). Here, the performance of the HIV-I group was significantly poorer than the HIV-UU group on all indices of the NEPSY-II, except that of Social Perception. The Social Perception index was measured by a single subtest (Affect Recognition) which may have reduced the variance in this domain, and subsequent ability for the statistical test to determine significant differences between the groups. While the result may reflect that affect recognition is preserved in the developing HIV-infected brain, this is not supported by research in adult samples which suggest that facial affect recognition is atypical in people with HIV (Clark, Cohen, Westbrook, Devlin, & Tashima, 2010; Heilman, Harden, Weber, Cohen, & Porges, 2013). A within-group analysis of the HIV-I groups performance on the NEPSY-II revealed that Attention and Executive Functioning was a strength relative to other domains. However, despite the domain's relative strength within this group, all subtest scaled scores were

in the below average range of performance, bar the three scores representing 'time' in the Inhibition subtest which were within average limits. This suggests that this group was able to complete the task within an age appropriate duration, but not without committing a significant number of errors. The Language, Memory and Learning, and Visuospatial Processing indices were all identified as relative weaknesses, with all scaled subtest scores falling well below average limits except for the global score of the Design Copying subtest, Fingertip Tapping (dominant and non-dominant), Route Finding, and Visuomotor Precision. These four scores were within average limits. These relatively elevated scores all rely on a visuospatial abilities and are less reliant on language, memory or complex processing. This constellation could reflect both the test bias of the NEPSY-II in multilingual, non-Western samples, as well as the differential associations of HIV with different cognitive capacities in children. The following section presents a similar set of findings from the HIV-EU group.

The working memory profiles of the HIV-Exposed (HIV-EU) sample.

The performance of the HIV-EU group aligned partially to hypothetical expectations, which proposed that while it would be unsurprising for this group to show a degree of impairment relative to unexposed control, they were unlikely to be as poor as the HIV-I group. A between-group analysis identified significant differences in the working memory performance of the HIV-EU group to the other two groups, but this followed a different pattern to that of the HIV-I group. The performance of the HIV-EU group on the verbal short term- and working memory indices was significantly lower than that of the HIV-UU control group, but no different to that of the HIV-I group. Findings in the visuospatial domain identified no significant differences between any of the three groups in the visuospatial short term index. However, in contrast to the pattern of difference with the verbal domain, the HIV-EU group were significantly better than the HIV-I group, and were not distinguishable from the HIV-UU control group in the visuospatial working memory index. The diminution of verbal performance in the HIV-EU appear to be as equally affected as the HIV-I group. These factors could include HIV-associated elements such as HI-viral exposure, ART toxicity, or the social circumstances surrounding living with HIV, where the HIV-EU had significantly poorer LSM_{raw} scores when compared to the HIV-UU group (t = 0.72, p<0.001**). It also represents the influence of English proficiency on psychometric assessment. The two HIV-affected groups also had comparatively poorer levels of English proficiency when compared to the HIV-UU group ($t_{HIV\text{-}I}$ = 1.20, p<0.001**; $t_{HIV\text{-}EU}$ = 1.22, p<0.001**).

A within-group profiling of the strengths and weaknesses of the HIV-EU group indicate that the visuospatial working memory index was a relative strength and significantly better than the other three indices. The mean performance score of the group fell within average ranges (*M* = 97.23, *SD* = 14.14). While there were no significant differences between the other indices, the verbal short term (*M* = 84.44, *SD* = 16.41) and working memory indices (*M* = 85.13, *SD* = 14.13) are both deflated relative to the visuospatial domain, and both lie on the cusp of the below average range of performance. A subtest analysis of within-group differences indicate that, again, the Nonword Recall (M = 95.18, SD = 17.15) subtest had the highest verbal score, and was significantly better than Word Recall (*M* = 76.15, *SD* = 14.2; $p<0.001^{**}$). Within the verbal working memory domain, the reliance on digits in the Counting Recall and Processing subtests appeared to be more robust, while the Listening Recall and Processing scores appeared to be more vulnerable to the effects of decay and English proficiency. There were no significant subtest strengths or weaknesses in the either of the visuospatial indices.

These results confirm the pattern emerging from the HIV-I group which suggest that visuospatial working memory appears to not only be more robust to the detrimental effects of HIV-exposure than other indices, but may represent an area of strength in this sample. This is possibly a consequence of the bilingual executive functioning advantage in the multilingual sample. The relative weakness of the verbal indices, in concordance with the trend in the HIV-I group, is likely to be a combination of the effects of HIV (viral exposure, ART toxicity and the socially disadvantaged environment thereof), as well as test bias in samples with lower levels of English proficiency. Similar to the HIV-I group, the HIV-EU group's subtest performance also suggests that the Nonword Recall subtest could be elevated due to the samples multilingualism and capacity to learn more than one language. The relative robustness of verbal tasks relying on digits (Counting Recall) and not words (Listening Recall) could reflect practice and exposure effects of English digits rather than words and sentences. There are relatively fewer digits to learn (possible the digits one to twenty), and are practiced with more frequency in comparison to a growing English vocabulary in the first two years of formal schooling.

When more detailed cognitive functioning of the HIV-EU group was explored using the NESPY-II, the group's performance was significantly worse on measures of Attention and Executive Functioning, Memory and Learning and Visuospatial Processing than the HIV-UU control group, but not significantly different from the HIV-I group. There were no significant differences on measures of Language or Social Perception between the HIV-EU group and the other two groups,

but they were significantly better than the HIV-I on the Sensorimotor index. These results suggest that the HIV-EU group was equally affected by the factors responsible for the poor performance of the HIV-I group on measures of Attention and Executive Functioning, Memory and Learning and Visuospatial Processing, but show differential associations on measure of Language and Social Perception. The deviations in the latter two domains could be the consequence of a statistical analysis relying on reduced variance (explained previously), as only a single subtest (Affect Recognition) was used to measure Social Perception. In the case of the Language subtest, the HIV-EU group had significantly better English proficiency than the HIV-I group (p = 0.02*), which could account for the more robust performance. The HIV-EU group's significantly better performance on the Sensorimotor index than the HIV-I group is consistent with existing literature which suggests that motor functioning is one of the first neurological skills to show impairment in conditions of HIV-infection (Blanchette, 2002; Drotar, 1997; Nozyce et al., 1994; Tardieu, 1998).

A within-group analysis reveals that while the Attention and Executive Functioning was a relative strength of the HIV-EU group's performance, all scaled subtest scores constituting this domain fell within the below average range except for that of the timed component of the Inhibition subtest. This suggests that children in this sample completed the task in age appropriate time limits, but their error profile was too large to maintain average performance. The Language, Memory and Learning, and Visuospatial Processing indices were identified as relative weaknesses, which is a pattern similar to that of the HIV-I sample. All of the scaled subtest scores of these indices fell within the below average range of performance, except for the scaled score components of the global score for the Design Copying, Fingertip Tapping (dominant and non-dominant hand), Imitating Hand Positions, and Visuospatial Precision subtests which were all within average limits. These subtests draw on non-lingual, motor, and visuospatial skills which appear to be less vulnerable to the effects of HIV exposure, and test bias in non-English samples. The following subsection documents the working memory profile of the HIV-UU group.

The working memory profiles of the HIV-Unexposed (HIV-UU) sample.

The performance of the HIV-UU group aligned well to hypothetical expectations, which proposed that they would be the best performing group of the three. The HIV-UU group was significantly better than both the HIV-I and HIV-EU groups on the Verbal Working Memory Index. They were also significantly better than the HIV-I group on the Visuospatial Working Memory Index, and significantly better than the HIV-EU group on the Verbal Short Term Memory Index. While statistically indistinguishable from the other two groups on the other working memory indices,

their performance was consistently above that of the other two groups, and importantly, always within average limits. This is in comparison to British normative data for this age group, and would suggest that the HIV-UU group does not deviate from normative expectations. Despite the group's multilingualism and issues of English proficiency and possible test bias, this would suggest that the AWMA is a valid measure of working memory in typically developing South African school beginners.

A within-group profile of strengths and weaknesses of the HIV-UU group revealed that, similar to the pattern in the previous two groups, verbal working memory was a relative weakness, and that visuospatial working memory was a relative strength. Both of these however were well within average limits of performance, and are believed to possibly reflect issues of test bias with regards to English proficiency, and the bilingual executive functioning advantage respectively. There was evidence of an age appropriate fractionation of the two verbal indices where short term stores were marginally better than the working memory counterpart. There results also show elevated performance on the Nonword Recall subtest (*M* = 110.62; *SD* = 18.03), as well the three subtests in the verbal working memory index which were reliant on digits, and not words (Counting Recall (*M* = 103.37; *SD* = 20.36), Counting Processing (*M* = 104.22; *SD* = 16.32) and Backwards Recall (*M* = 99.42, *SD* = 16.34). These results converge to continue a pattern from the trends observed in the other two group, and have been discussed previously. The relative strength of Nonword Recall performance is strongly correlated with language acquisition – something that the participants in this multilingual sample are familiar with. The general diminution of the visuospatial short term index within this sample is not believed to be reflective of a legitimate weakness, as this index still fell within average limits (*M* = 97.29; *SD* = 17.6), and could not be attributed to the poor performance in any one particular subtest. Rather, its statistical significance is only highlighted in comparison to the relative strength of the Visuospatial Working Memory index (*M* = 107.25, *SD* = 17.98). However, according to theoretical developmental trajectories, visuospatial short term performance in this aged cohort of children should have been stronger than the verbal short term component because of the developmental fractionation hypothesis. Hence, this relative diminution may have been the consequence of a misunderstanding of the test instructions or administration.

When more detailed cognitive functioning of the HIV-UU group was explored using the NESPY-II, the group's performance was more homogenous between domains, where the Attention and Executive Functioning index was identified as a relative strength only in relation to Memory

and Learning, Sensorimotor abilities and Visuospatial Processing. While this pattern is similar to that of the HIV-I and HIV-EU samples, within the HIV-UU group, this distinction had weak power (P = 0.168) and poor explained variance (η^2 = 0.044). This is not to suggest that the effect of the bilingual executive functioning hypothesis is less apparent in samples with greater English proficiency as is in the case with the HIV-UU group, but rather that the within-group differences are less discrete and identifiable in this group because their relatively sound levels of English proficiency reduce the effects of test bias in the other domains. In contrast to the subtest analysis of the performance of the other two groups, all scaled subtest scores of the NEPSY-II in the HIV-UU group were in the average range of performance, except that of the motor components of the Design Copying subtest (M = 6.66). This relative diminution could be the result of an error in the scoring of the detailed, fine motor abilities measured in this subtest.

One final point regarding the performance of the typically developing HIV-UU control group on the AWMA is necessary before the discussion moves to expand on the five major theoretical findings of the study. The HIV-UU control group came from a relatively low socioeconomic bracket, yet their performance on all subtests of the AWMA fell within the average range. This was in relation to British norms. The lowest subtest performance was on the Listening Recall Processing subtest (M = 90.13, SD = 12.77), while the highest was the Odd One Out Processing subtest (M = 107.55, SD = 16.6). The group's weaker performances were constellated in the verbal subtests reliant on words (instead of digits), while their strengths were in short term digit recall tasks and those relying on visuospatial working memory. This suggests that while the verbal subtests of the AWMA may hold some degree of cultural and linguistic bias, the test appears to be an appropriate measure of working memory performance in non-Western, linguistically diverse, typically developing South African children.

There are five salient discussion points which arise from these findings, and refer primarily to the two HIV-affected groups. The first is that there appears to be significant working memory impairment in both HIV-affected groups in comparison to the HIV-UU control group. The second is that in contrast to these deficits, short term memory appears to be relatively robust in the context of both HIV-infection and HIV-exposure. The third and fourth findings refer to the relative strength and weakness profiles where verbal skills are general weaknesses in all three samples, while visuospatial working memory appears to be a relative strength in all three groups. The fifth is the relationship between these findings and the performance of the three groups on the NEPSY-II. These will each be discussed below.

General Findings

Significant working memory impairment in atypical samples.

The data from the HIV-I group identified significant impairment in the two complex working memory indices, while that from the HIV-EU group was only significantly impaired on the verbal working memory measure when compared to the HIV-UU group. The visuospatial working memory of the HIV-EU group was however deflated in comparison although it did not reach significance. Within the context of HIV infection, these findings are well supported by other studies in adult and paediatric samples who have found impairment in both the verbal and visuospatial complex working memory capacities (Boivin, Busman, & Parikh et al., 2010; Dawes, Suarez, Casey et al., 2008; Koekoek et al.,2008; Farinpour, Martin, Seidenberg, Pitrak, Persell, Mullane, et al., 2000; Heaton et al., 2004; Hinkin, Hardy, Mason, Castellon, Lam, Stefaniak, Zolnikov, 2002;York, Franks, Henry, & Hamilton, 2001).

There are two theoretical hypotheses for HIV-associated working memory impairment. The first is through the neuroanatomical damage to prefrontal cortical matter in localised regions responsible for componential working memory functions (Martin et al., 2006; York et al., 2001). The other is known as the executive functioning hypothesis, and is where HIV-associated injury to the neural substrate necessitates greater attentional modulation of the neural circuits. When activity demands on these already stressed fronto-striatial circuits exceeds a particular threshold, and when more than one component is employed simultaneously, deficits begin to occur. This is because a greater part of the brain's reserve and the additional activation of the frontal lobes is necessary to carry out the tasks (Martin, Sullivan, Reed, Fletcher, Pitrak, Weddington, & Harrow, 2001). The explanation of working memory impairment as an initial disruption in the functional capacity of central executive networks is believed to provide support for the belief that executive functioning is one of the first neuropsychological functions to be affected in otherwise asymptomatic HIV infected adult patients (Reger et al., 2002). In this regard, Hinkin et al. (2002) remain adamant that working memory deficits across domains are primarily a result of executive functioning impairment secondary to viral infection, and not a result of localised damage of the isolated cortical regions responsible for verbal or spatial stores. The executive functioning hypothesis in the context of HIV infection is further supported by a large body of research in adult patients, while little exists regarding the mechanisms underlying working memory impairment in pre-schoolers or children (Heaton, Marcotte, Rivera et al., 2004; Heaton, Franklin, Ellis, & McCutchan, 2011; Marcotte, Wolfson, Rosenthal, & Heaton, 2004; Melrose, Tinaz, Castelo, & Courtney, 2008; Reger, Welsh & Razani, 2002). Hence, according to this hypothesis, it appears that

it is not the system's reliance on the specific encoding, maintenance or retrieval neuroanatomical structures within either domain (verbal or visuospatial) that appear to be affected by HIV infection, but rather that fallout occurs in the context of cognitive overload when demand exceeds capacity for mental manipulation (Alamargot, Terrier, & Cellier, 2007).

The executive functioning hypothesis gives further support to the belief that HIV-associated neuro-compromise is diffuse in nature, and relies on opportunistic infection of the CNS instead of localised damage. Neuro-imaging and neuropathology studies which have explored the cerebral substrates of the central executive provide sound support for the fact that executive functions not only make use of various frontal regions, but also rely on posterior (mainly parietal) regions. Such research confirms that executive functions rely on a distributed cerebral network across the cortex, and that its functioning is best understood in totality as the interaction of many networked regions rather than an isolated unitary localisation within the prefrontal cortex (Colette, van der Linden, & Salmon, 2001; Collette & van der Linden, 2002; Duncan & Owen, 2000; Heaton, Franklin, Ellis, & McCutchan, 2011; Parkin, 1998). Hence, it stands to reason that even in otherwise asymptomatic, immunologically stable HIV-I patients who show no signs of overt immuno-compromise, executive function is likely to be affected by the cytotoxicity of HIV infection of neuro-cells. Consequently, the subsequent effect on working memory would not be a latent by-product of acute infection, but could be considered one of the initial markers of neuropsychological degeneration in both adults and children. Heaton (2004) and Bassel et al. (2002) have thus both identified working memory as the primary driver between subjective reports of cognitive decline and its measurable effect on everyday functioning. In HIV-I individuals. Within the context of neurodevelopment, the decline of working memory could be considered a latent marker for the initial stages of neuro-compromise and an important area for early intervention within paediatric populations.

In relation to the HIV-EU group, the identification of working memory deficits in the current study are partially consistent with the very limited knowledge within this research field. Kerr et al. (2014) conducted an investigation into the general neurodevelopment of an exposed and unexposed sample in Thailand ($n_{HIV\text{-}EU}$ = 160; $n_{HIV\text{-}UU}$ = 167) and Cambodia (n = 202), using the Child Behaviour Checklist, the Beery Visual Motor Integration (VMI), the Stanford Binet-II and the WPPSI-III for a range of children between two to fifteen years. While no direct measure of working memory was used, the researchers did find that a significantly greater proportion of the exposed sample had salient attentional difficulties. However, the analyses also revealed no significant

differences in executive function ability as measured by the Colour Trails Test 1 and 2. The current study did not employ the Colour Trails Test 1 and 2, but did find significantly poorer scores in the Attention and Executive Functioning domain when compared to a HIV-UU control group (ΔM = -10.30, p = 0.007**). This domain made use of subtests measuring auditory attention, inhibition, planning, cognitive flexibility and fluency in the generation of patterns (Suchy, Kraybill, & Larson, 2010)

There are two hypothesised explanations for general neurocognitive compromise in the context of HIV-exposure which have been discussed previously. Due to the relatively under-researched nature of such samples, there is no available evidence regarding why the working memory capacities specifically would be compromised. The executive functioning hypothesis is unlikely to apply to HIV-EU children as this debilitating mechanism is only activated in the presence of excitotoxic by-products of HIV associated inflammation present in the CNS. It is more likely that working memory impairment is a consequence of ART toxicity, or of atypical development of neural networks responsible for efficient working memory performance, than a latent in-utero chemical response from gestation (Meyer, Feldon, & Fatemi, 2009). The latter explanation is a neuroimmunological hypothesis which suggests that the presence of maternal chronic viral infection disrupts mechanisms of typical neurogenesis in the foetus, and results in permanent structural changes in the developing brain (Garay & McAllister, 2010).

While working memory impairment was evident in both HIV-affected samples, short term memory, particularly in the visuospatial domain, appeared to be less affected by associations with HIV. The following section discusses these findings in relation to existing literature.

The robustness of short term memory.

Notably, this study found no statistically significant differences between the HIV-I group and either of the other two groups in both the verbal and visuospatial components of short term memory. The HIV-EU group was significantly impaired when compared to the HIV-UU group in verbal short term memory, but showed no such significant difference from the other two groups in visuospatial short term storage.

Within the context of HIV infection and exposure, these findings are partially supported by similar research done with HIV-infected children in Thailand and Cambodia where researchers found no difference in the mean performance on similar short-term visual and verbal memory subtests (Bead Memory Test, Sentence and Digit Memory) between HIV-infected and HIV-

unexposed children (Kerr et al., 2014). Similarly, Bagenda et al. (2006) also conducted comparative assessments between HIV-infected and HIV-unexposed children, and found that there was no significant difference between the groups on the sequential and simultaneous processing of the K-ABC subtests of Number Recall, Word Recall, Spatial Memory, Photo Series and Face Recognition subtests which all measures short-term memory. However, these studies did not differentiate between short term- visuospatial and -verbal memory, in the way that the present study does. Despite this, the relative preservation of verbal short term storage is also supported by other studies of HIV-I children when compared to HIV-UU controls (Blanchette, et al., 2002; Klaas, et al., 2002; Martin, Wolters, Toledo-Tumala et al., 2006). These studies, however, are not completely comparable to the context of the present study as the samples were assessed in English which was believed to be their first language, had relatively small sample sizes (*N* = 14-41), and considered an older cohort of children (*M* = 9.8-10.5 years).

Within the visuospatial short term memory index, the findings from the current study do support previous work with HIV-I paediatric samples. For example, Koekkoek et al., (2008) found significantly below normative scores in measures of visuospatial short term memory (measured using visual memory of the location and serial order of an animated marker in a 3x3 matrix). The HIV-I group's performance in the current study was also below British AWMA normative ranges. However, comparison to these norms was believed to be biased, and instead comparisons with a demographically similar HIV-UU group were made. In this analysis, the performance of the HIV-I group was not significantly poorer than that of the HIV-UU group on the measure of visuospatial short term memory. Research in adult populations supports this finding by indicating that short term visuospatial impairment is usually characteristic of late stage infection, and that, like the children in the current sample who were in general good health and adherent to effective treatment regimens at the time of assessment, visuospatial short term memory is unlikely to show significant compromise during periods of good health (Reger, Welsh, Raxani et al., 2002).

The relative preservation of the visuospatial short term stores in the HIV-EU sample in the current study is supported by evidence which found that comparative performance on the Sentence Memory, Digit Memory and Object Memory subtests of the Stanford Binet-II did not differ significantly between the HIV-UU and HIV-EU groups (Kerr et al, 2014). This suggests that the more complex working memory components, which rely on greater executive attention in the context of increased cognitive load, appear to be more vulnerable to impairment than simple short term stores in the context of HIV exposure.

There are two primary hypotheses behind this possible preservation of short-term stores in the context of HIV infection. The first relates to the development of working memory throughout childhood, where there are debates about the developmental trajectories for the visual and verbal domains of the working memory system for children relative to adults. Evidence from a collection of studies converge to identify two clear axes of differentiation within the componential structure of the working memory model (Alloway, Gathercole, Willis et al., 2004; Alloway, Gathercole, Pickering et al., 2006; Alloway & Alloway, 2013; Gathercole, Pickering, Ambridge et al., 2004; Gathercole & Jarvis, 2003). The first refers to the distinction between the passive and active processing capacities of working memory, where there is a transition from a reliance on passive short term storage to active processing apparent in younger children, and an increased capacity for complex manipulation only appearing after the age of approximately seven years. The second refers to the domain specific distinction between the verbal and visuospatial representations within in each of the active and passive components. In this line of evidence, there is a domain-specific preference for visuospatial encoding in younger children, which begins to switch to verbal encoding from around the age of six or seven years (Alloway, Gathercole & Pickering, 2006; Hitch & Halliday, 1983; Hitch, Halliday, Schaafstal, & Schraagen, 1988). The current study's findings that the short term indices in the two HIV-affected group were no poorer than that of the control could reflect this relative proficiency in both verbal and visuospatial short term tasks, as the three sub-samples are all school beginners who were all negotiating this transition.

The second hypothesis about why short term memory stores may be preserved in the context of HIV infection refers to the process of HIV disease progression. Here, it is believed that a level of protection from acute infection is sustained by effective cART, which prevents damage to the short-term stores. Participants in the HIV-infected sample were required to be immunologically stable, with no hospitalisations due to illness in the last six months. While all were on cART, they had to be virally suppressed, compliant on the regimens, and with no significantly adverse side effects to cART. Therefore, while not clinically categorised as being HIV-asymptomatic, these children were relatively healthy. Hence, the verbal and visuospatial short term stores, which have a more localised neuroanatomical position in the cortex, could have been spared by the virus. On the other hand, the integrated and diffuse networks responsible for executive functioning across the anterior and posterior regions of the cortex may be more vulnerable to fall out in the presence of cyto-toxins. This line of reasoning is supported by the collective findings from 41 other adult studies, but is limited in application to this research because of the age of participants (Reger,

Welsh, Raxani et al. 2002). The supposition of these two aforementioned hypotheses is not mutually exclusive, and the results of the current study may reflect a combination of both.

The lack of variance in the Visuospatial Short Term Memory across the three groups could also be attributable to the construct's relative independence of language and HIV status, particularly in the visuospatial domain. However, there is much argument regarding the idea that even visuospatial information is coded verbally, and thus not free of linguistic influence (Pickering, 2001; Fenner, Heathcote, & Jerrams Smith, 2000; Hitch, Halliday, Schaafstal & Schraagen, 1988; Luciana & Nelson, 1998; Palmer, 2000). While visuospatial working memory does enjoy some research support as a construct less vulnerable to the effects of language proficiency (Campbell et al., 1997; Cockcroft, 2014; Laing & Kamhi, 2003), it is not without criticism. Other research has found strong support for the effect of language proficiency (particularly English) as a mediator to working memory success (Jordaan, 2012; Mailmela-Arnold, & Evans, 2005). The associations between working memory and language are evident in the within-group profiling of strengths and weaknesses within the samples of the current study.

Verbal working memory is a relative weakness for the HIV-affected groups.

The within-group analyses identified that both verbal short term and working memory scores were generally deflated in all three samples. However, only verbal working memory was identified as a relative weakness across the groups, while the verbal short term memory index maintained typical performance within HIV-UU group only. While no other known studies have profiled working memory in HIV-I and HIV-EU samples using within-group differences, the general impairment of verbal working memory in the context of HIV infection is evidenced by a number of between-group adult studies discussed previously (Klaas, et al., 2002; Stout, Salmon, Butters et al., 1995; York, Franks, Henry, & Hamilton, 2001,).

The performance of the HIV-EU group on measures of verbal short term memory demands special explanation prior to a discussion of the verbal working memory difficulties experienced by the larger sample more widely. The performance of the HIV-EU group on the verbal short term index was the only one which was significantly worse than that of the HIV-I group. This is contrary to the expected pattern of performance, as the two HIV-affected groups were statistically indistinguishable on all other indices. The very poor performance of the HIV-EU sample on this index is believed to be a function of sampling, as well as the wider context in which HIV-EU children develop. From the socio-demographic analyses of the HIV-EU sample, it is apparent that this group of children were particularly disadvantaged in comparison to the other three groups.

Their socioeconomic status was the poorest of the three groups, and significantly lower than the HIV-UU group (LSM: $F(2, 272) = 4.29$, $p = 0.015$). The HIV-EU group also had the highest familial burden of care (looking after an immediate family member with special needs) (21.95%), the highest proportion of receiving the child support grant (91.9%), the lowest levels of maternal education (only 21.8% finishing high school), and the highest levels of paternal absenteeism (53.4%). In contrast, HIV infected children are frequently followed up on by specialised ARV clinics which offer them support and access to social and allied therapeutic services, while their exposed siblings seldom come into contact with auxiliary services. This lack of institutional care and support is a finding supported by international demographic profiling of HIV-EU children (Kerr, 2014). Further, despite the HIV-EU group's lack of HIV infection, they remain strongly affected by the daily consequences of families living with HIV. Single or double orphanhood, disability, poverty, unemployment and malnutrition are strongly correlated with HIV infection and have extremely negative consequences on child development (Barnighausen, Hosegood, Timaeus, & Newell, 2010; Hargreaves, Morison, Chege et al, 2002; Lewis, Kaiken & Hoyt, 1994; Lurie, Fernandes, Hughes, & Arevalo, et al., 1995; Salter-Goldie, DeMatteo, King, Wells and the Multisite Co-investigators, 1997, as cited by Blanchette et al., 2002).

The significantly poorer performance of the HIV-EU group relative to HIV-I group on the measure of verbal short term memory is also believed to be a reflection of the strong relationships between linguistic proficiency and general working memory performance (Jordaan, 2012). The HIV-EU sample within this study had the lowest numbers of children who speak English as a first language (0%) and attend English schooling (31.12%) of the three groups which could contribute to an account of their impairment in relation to an already impaired subsample (HIV-I group).

The focus of the discussion now returns to the generally deflated performance on the verbal working memory measures in all three samples relative to the Eurocentric norms of the AWMA. While this discussion refers to matters not associated with HIV status, it is important to recognise that as discussed in the previous point, the verbal working memory performance of the two HIV-affected groups was significantly lower than the control group. To that end, verbal working memory does appear to be compromised in the context of both infection and exposure, while also being further influenced by the possible explanations offered below.

There are three possible explanations for the relative diminution of verbal working memory in all three groups in relation to other components of the working memory model. The first is offered by the developmental fractionation hypothesis of the progressive trajectory of

working memory within children described above. There is sound empirical support for the notion that young children's working memory is primarily visuospatial in nature with a reduced proficiency in processing tasks until the age of about of seven or eight. Only as they begin to acquire literacy at around the age of six years, does verbal rehearsal begin to gain proficiency, and the complex processing capacities, particularly in the verbal domain, begin to reach maturity (Alloway, Gathercole, Willis, & Adams, 2004; Alloway, Gathercole, & Pickering, 2007; Gathercole, Pickering, Ambridge, & Wearing, 2004). The sample in the current study was tested at this node of transition, and so it is very likely that many participants were still gaining verbal working memory proficiency.

The second explanation suggests that the difference is a result of the order of test material presentation, as the verbal material is presented earlier than the visuospatial working memory measures allowing for a practice effect to emerge in the visuospatial task (Hinkin et al., 2002). The order of presentation of test materials in the AWMA is mixed, and presents a non-sequential succession of both verbal and visuospatial tasks (Alloway, 2007). While this explanation is unlikely to account for significant impairment, it may have had an influence.

The final explanation is that verbal working memory performance is intrinsically linked to linguistic processing and that performance suffers as a result of depressed English language proficiency. Jordaan (2012) assessed two groups of linguistically diverse school beginners in South Africa on measures of working memory (AWMA), sentence repetition and language processing. Differential working memory performances were found between children who spoke English as a first language, and those who came from linguistically diverse contexts who spoke English as an additional language. This suggests that working memory measures are not unbiased with respect to linguistically diverse children. Jordaan argues that the measures employed to measure verbal working memory, and the way in which this is assessed psychometrically, are heavily reliant on language knowledge and processing. She found that the effects of language limitations were not only evident in the verbal working memory index, but extended to those subtests that are assumed to rely less on language knowledge such as Digit Repetition, Dot Matrix and Spatial Recall and Processing, suggesting that these visuospatial tasks may draw on some elements of verbal ability. Jordaan (2012) recognises that deficits in these non-verbal subtests could be due to their reliance on adequate understanding of instructions, and not related to the measured construct itself.

Such research provides support for the idea that verbal working memory and long-term linguistic knowledge are unlikely to be distinct constructs, but rather that working memory is a reflection of an activated set of linguistic representations in long term memory (Jordaan, 2012; Mailmela-Arnold & Evans, 2005). This is similar to Cowan's model (Cowan, 1999). To that end, limited working memory capacity is a reflection of weak linguistic representations because access to these representations is dependent on linguistic efficiency. Consequently, working memory measures in linguistically diverse contexts may actually assess language processing; where the relationship between working memory and language processing depends on the difficulty of the language task and the level of language proficiency (usually English) (Jordaan, 2012).

The present study attempted to compensate for the effects of English proficiency by using the measure of Sentence Repetition as a covariate in the analyses, allowing the translation of instructions into the child's home language, encouraging a brief practice trial, as well as introducing additional material to explain the instructions in complex tasks. However, this remains one of the limitations of the study. There were differential levels of English proficiency between the groups (($F(2, 272) = 31.290$, $p = 0.001$) with significant differences exist between the two HIV-affected groups and the control only). The correlation between measures of verbal working memory and English language proficiency also differ across the groups (HIV-I: $r = 0.370$, $p<0.001$; HIV-EU: $r = 0.041$, $p = 0.705$; HIV-UU: $r = 0.435$, $p<0.001$).

Jordaan's (2012) research found significant differences in the working memory performance of South African children who had differential levels of English proficiency, and provides evidence that the standard psychometric assessment of the construct using the AWMA is thus biased. Her findings are, however, critiqued for being but one piece of evidence with a sample size of only 55 children, and had a focus on language processing, not general cognitive functioning. In contrast, other studies found no bilingual disadvantage on measures of verbal working memory in a comparison between 120 mono- and bilingual school beginners (e.g. Cockcroft, 2014). However, this has limited comparable validity to the current study as of the children in this study were proficient in English, and were being educated in English. Nevertheless, this may suggest that tests of verbal working memory may be able to give an indication of the efficient functioning of articulatory rehearsal and storage of the phonological loop, even when tested in a second language. It is necessary to point out that a strong rebuttal to this argument as well, which has proposed that working memory assessment is a more accurate measure of cognitive functioning that is less reliant on language learning than traditional assessments, and in

fact may offer a particular advantage to children who speak more than one language (Bialystok, Craik, Klein, & Viswanthan, 2004; Bialystok, Craik, & Ryan, 2006; Emmorey, Luk, Pyers, & Bialystok, 2008). This is discussed later, in support of the fourth finding from this study.

Within the verbal domain, a more detailed within-group analysis revealed three subtests identified as relative strengths in all three subsamples: Nonword Recall and Counting Recall and Counting Processing. The use of Nonword Recall has been widely used as a measure to determine language learning capacity in both typical and atypical samples of children, as it is a recognised means of establishing a child's capacity to learn unfamiliar vocabulary without an established lexical map within long term memory (Baddeley, 1998; Gathercole, 2006). Findings of performance in this subtest are therefore particularly pertinent to studies of multilingual children who have dual language processes competing for position within the phonological store.

There is equivocal support for the bilingual disadvantage effect on Nonword Recall performance within bilingual children. In two studies (Engel de Abreu, 2011; 2012), bilingual performance on measures of Nonword Recall was significantly poorer relative to their monolingual peers. In the former study, the effect disappeared once vocabulary had been controlled for, and the poorer performance was cited as being constrained by a smaller lexicon in the bilingual group. However, the effect persisted in the latter study after vocabulary was accounted for, and was found to be greater when tested in their second language when compared to their native language. This finding has relevance for the children in the present study who were also tested in an additional language to their home language. However, no significant differences on Nonword Recall performance were found in a South African comparison of mono- and bilingual school beginners (Cockcroft, 2014). The sample size in this study were small ($n_{monolingual}$ = 67, $n_{bilingual}$ = 53), and all bilingual participants were proficient in English and educated in English medium schools. While the present study did not investigate the extent of multilingualism, and correlate working memory differences with mono- and multilingualism, all participants in this study spoke more than one language, and 9.15% identified themselves as first language English speakers. Further, Nonword Recall scores within all three groups were significantly higher than the mean performance (HIV-I:*M* = 100.96, *SD* = 13.32 HIV-EU: *M* = 95.18, *SD* = 17.15 and HIV-UU: *M* = 110.62, *SD* = 18.03), and show differential yet significant correlations with SRT (a proxy for English proficiency) (HIV-I: $r = 0.219$, $p = 0.03$; HIV-EU: $r = 0.246$, $p = 0.022$; HIV-UU: $r = 0.514$, $p<0.001$). These findings support moderate, positive associations between English proficiency and performance on Nonword Recall.

The significantly better performance on Nonword Recall within this multilingual sample is important for two reasons. Firstly, its relevance to the multilingual sample provides further evidence that children who can speak more than one language could have an increased capacity to learn vocabulary, and subsequent additional languages in similar orthographies. It also provides positive support for the bilingual advantage in this multilingual sample which is discussed in greater detail later on. The proposition that working memory ability supports the learning of additional languages is considered here.

Results from an important study by Papagno and Vallar (1995) indicate that this association between Nonword Recall and language learning extends to both high and low language learning capacities. They compared the nonword repetition and novel word learning abilities of young adults classified as either polyglots (who were proficient at a minimum of three languages, or non-polyglots). One of their key findings was that polyglots had superior nonword repetition scores to the non-polyglots, where nonword repetition is understood to represent the capacity to learn additional vocabulary. Other studies have also found associations between established dual linguistic proficiency in bilinguals, and the capacity to learn additional languages (Abu-Rabia & Sanitsky, 2010; Weiqiang, 2011). While it is hypothesised that an established strength in vocabulary learning is one of the reasons for this lingual advantage, the links are only associational, and there is no empirical support for a causal effect yet. However, some authors do vouch for a working memory capacity interaction hypothesis with multilingualism (van den Noort, Bosch, & Hugdahl, 2006). Bialystok (1987) argues that bilingual children have an increased metalinguistic awareness, and this increases bilinguals' control of linguistic processes, such as having a greater ability to detect grammatical or syntactical errors, and recognize words in continuous speech in additional languages. Within the present study, this might represent a unique advantage to the participant sample who, because of an established metalinguistic awareness, are able to learn more than two language more easily than monolinguals.

The relationship between additional language learning and Nonword Recall is not new, and was initially put forward by two seminal studies by Service (1992) and Cheung (1996). However, the relationship between these two skills in children who speak English as a second language (ESL) remains uncertain. The children in these two studies were learning English as a Foreign Language, and were not children who spoke English as a second language. Foreign language English learners learn English as an academic subject in their own country, while English as Second language learners learn English in a country where it is the primary language of communication. This

differentiates the learning context of these two groups. It is likely that a combination of varying sociocultural factors could affect the significance of phonological working memory in children who speak English as a second language. These factors may not be present in the learning environment of monolingual and 'English as a Foreign Language' learners. Therefore, given the different circumstances in which children who speak English as a second language learn English, it cannot be assumed that they learn in the same manner as young monolingual children. For example, their first language may be suffering from attrition, which may impact English language learning experiences (Baddeley, 2002; Haughey, 2002; Kayser, 1995). One example of this interaction is evident in the findings from Cockcroft and Alloway (2012) who found that a group of ESL South African children had similar levels of phonological awareness to that of monolingual English speakers from the UK, except on a task detecting a middle sound oddity. They proposed that the influence of the ESL group's Sotho and Nguni home languages, which emphasises the initial and final phoneme of a word and offers middles phonemes less stress, interacted with their processing of English phonemes in an atypical way. To that end, specific linguistic knowledge from the ESL child's first language may affect language development in an additional language, and that different languages may support specific processing skills. Within the South African context, this is further complicated by the fact that English is esteemed as a language associated with good education and wealth, and that it is not always taught correctly in schools (Broom, 2004; De Klerk, 2006). Further, English, in some cases, is not a child's second language but their third or fourth language which could change the associations with the language learning as well.

The second important implication of the relative strength of Nonword Recall within all three samples refers to its use as an assessment measure of verbal working memory capacity. Within a subtest analysis across the three groups of this study, Nonword Recall and the two subtests, Counting Recall and Counting Recall Processing, were identified as the only consistent relative strengths across the three subsamples. In Cockcroft's (2014) comparison of 120 mono- and bilingual South African school beginners, lexical knowledge (vocabulary) predicted significant variance within these two AWMA subtests (Nonword Recall, and Counting Recall) in the monolingual group, but notably not in the bilingual group. She proposed that these two subtests may be less dependent on the bilingual child's knowledge of English proficiency and may hold the least bias for the assessment of working memory when using the AWMA. She also suggests that Nonword Recall could be a relatively pure measure of phonological storage in school beginners

who are assessed in English as their second language. The findings from the current study support this evidence.

Visuospatial working memory as a relative strength in all three groups.

The fourth finding is related to the issue of accurate working memory measurement within linguistically diverse groups of children, and concerns the fact that visuospatial working memory was identified as a relative strength in all three samples. The visuospatial dominance over verbal skill within this aged sample is unsurprising as there is strong theoretical support for a visuospatial supremacy over verbal skill within this age group (Gathercole et al., 2004; Hitch & Halliday, 1983; Hitch, Halliday, Schaafstal, & Schraagen, 1988, Pickering, Gathercole, Hall, & Lloyd, 2001). However, this finding was unexpected in light of the hypothesised ranking of working memory indices within the developmental fractionation hypothesis, where it was assumed that visuospatial short term memory would outperform its working memory counterpart.

The visuospatial working memory dominance may have been a consequence of practise effects, as these subtests are presented in the latter half of test administration. However, this was unlikely to have been solely responsible for the pervasive strength of this index across all three groups, as all three groups have moderate effect sizes ($Mean_{Cohen's\ d}$ *= 0.59; Range:* 0.28 to 0.86). Rather, within school beginners, the subtests employed to measure visuospatial working memory rely heavily on executive processes to manipulate and then recall a number of spatial locations and shapes. There is also sound evidence for the bi-directional effect of multilingualism on executive processes within working memory which could support the dominance of visuospatial working memory in this multilingual sample over visual short term storage. One of the two directions are referred to as the bilingual advantage where bilingual samples show better performance on a variety of executive tasks not reliant on language (inhibition, switching, executive control) (Bialystok & Martin, 2004; Bialystok, Craik, & Ryan, 2006; Bialystok, Craik, & Luk, 2008; Carlson & Meltzoff, 2008; Martin-Rhee & Bialystok, 2008;). While some studies have found no evidence for a bilingual advantage in working memory performance specifically, (Bialystok & Feng, 2009; Bonifacci, Giombini, Bellocchi, & Contemo, 2011; Cockcroft & Alloway, 2012; Engel de Abreu, 2011), there is little evidence for a bilingual disadvantage either. Some of these studies are also criticised for being isolated to simple working memory tasks, and are believed to be biased as they rely on largely verbal measures, and require children to recall lists of words or digits which present an inherent disadvantage to children who do not have strong English proficiency (Soliman, 2014).

The identification of a visuospatial strength in the present study is supported by the work of two other studies of similar aged children where bilingual children performed faster than monolinguals, and were more advanced in their progress in carrying out complex working memory tasks, and obtained higher scores than monolinguals in both conditions, particularly in the more difficult serial order condition (Morales, Calvo, & Bialystok, 2003). However, while these two studies were conducted with children who spoke English as their second language, they differed from the South African sample in that they all came from the same homogenous middle class community, all attended preschool, and all of their parents had a university level education, which could all have been mitigating factors. Further, they came from a predominantly English community, whereas this is not a homogenous assumption in South Africa.

Nevertheless, the identification of visuospatial working memory as a relative strength in all three samples proposes two positive consequences. The first is that while multilingual children in South usually come from disadvantaged circumstances, their linguistic skill might offer them a cognitive advantage in the field of working memory over their monolingual peers. The second is that visuospatial working memory may be a less culturally and linguistically biased means of measuring cognitive potential in groups of linguistically diverse children. This very distinction of the bias of more conventional measures of cognitive functioning is evident in the subsequent discussion of the groups' performance on the NEPSY-II which considered more generalised neuropsychological functioning.

The associations of HIV status with differential performance on the NEPSY-II.

As summarised earlier, a between-group analysis found significant impairment between the HIV infected and HIV-UU samples on all indices of the NEPSY-II except that of Social Perception. The performance of the HIV-EU group was indistinguishable from that of the HIV-I group, and also significantly worse than that of the HIV-UU control on all indices except that of Language and Social Perception. The HIV-EU group however, performed significantly better than the HIV-I group on the Sensorimotor abilities index.

The generally poor performance of the HIV-I group across five of the six domains of the NEPSY-II is unsurprising, and confirms widely help understandings that HIV infection has a pervasive and deteriorating effect on generalised neurocognitive functioning on school going children receiving cART. This study contributes to an already very large body of research which agree about the detrimental effect of the virus on cognitive development (Le Doare et al, 2012; Lowick, Sawry, & Meyers, 2012; Puthanakit et al., 2010; Sherr, 2009). While it is well established

that HI viral infection itself has a detrimental effect on the brain (Blanche, Tardieu, Duliege, et al., 1990; Enguland, Baker, Raskino, et al., 1996; Kandawasvika et.al, 2015; Sherr, 2014; Smith & Wilkins, 2014), it is difficult to establish its effect on separate aspects of cognition with certainty. This is because many studies are conducted with participants already on ART which have been found to lessen or prevent further neurocognitive damage (for example, Lowick, Sawry, & Meyers, 2012; Raskino, Pearson, Baker, et al., 1999; Smith, Adams, & Eley, 2008; Van Rie, Mapuala, & Dow, 2008; van Rie, Mapuala, & Stewart, 2009) and therefore report equivocal findings (for example, Brackis-Cott, Kang, Dolezal, Abrams, et al., 2009; Koekkoek, Sonneville , Wolfs, Licht, & Geelen, 2008; Jeremy, Kim, Nozyce, et al., 2005; Laughton, Grove, Kibb, Springer, Dobbels, Janse van Rensburg, et al., 2009; Smith, Malee, Leighty, et al, 2006; Thomaidis, Bertou, Critselis, Spoulou, Kafetzis, & Theodoridou, 2010)

The participating sample in this study was subject to the effect of both viral infection and ART, and it is therefore not possible to attribute associations directly to either of these elements, or to the contribution of an HIV-associated socioeconomic environment on development. However, from a review of the available literature, it would appear that while symptomatic HI infection has a significant impact on cognitive functioning, this is usually mediated by cART where less debilitating neurocognitive fall out is identified in areas of executive functioning (Jeremy, Kim, Nozyce, et al., 2005, Koekkoek, Sonneville , Wolfs, Licht, & Geelen, 2008), verbal skill (Brackis-Cott, Kang, Dolezal, & Abrams, 2009; Levenson, Mellins, Zawadzki, Kairam, & Stein, 1992; Koekkoek, Sonneville , Wolfs, Licht, & Geelen, 2008), behaviour (Thomaidis, Bertou, Critselis, Spoulou, Kafetzis, & Theodoridou, 2010) and memory (Jeremy, Kim, Nozyce, et al., 2005, Koekkoek, Sonneville , Wolfs, Licht, & Geelen, 2008). Unfortunately, most of these studies assessed children in a language other than their home language or made use of an interpreter, and only some accounted for this effect. The present study is also vulnerable to these threats to validity for similar reasons.

In the current study, no significant differences were found between any of the groups on the Social Perception Index, with between group mean differences being relatively minor. This suggests that social perception may be unassociated with HIV status. However, these results were based on participant performance in only one subtest (which has a total scaled score, and an error profiling component of six sub-scores) which limits its validity. Contradictory support for this finding comes from Springer and Laughton (2012) who conducted a local study to investigate the neurodevelopmental status of HIV-UE toddlers, and found deficits in the Personal/Social subscale

of the Griffiths Scales of Mental Development. However, there is very poor concurrent validity between the Griffiths and the NEPSY-II, and age differences in the sample may account for the differences in findings. Further, the Personal/Social subscale of the Griffiths is reliant on parental report which is notoriously unreliable (Jacklin & Cockcroft, 2013).

Performance of the HIV-I group on the Sensorimotor Index was significantly poorer than the HIV-EU and HIV-UU groups. Other studies investigating the effect of HIV-infection on motor abilities also attest to this finding, and conclude that motor functioning in HIV infected people is generally one of the first neurological skills to show significant impairment (Blanchette, 2002; Chase et al, 1995; Drotar, 1997; Nozyce et al., 1994; Pearson et al., 2000; Potterton & Eales, 2001; Tardieu, 1998).

In the present study the HIV-EU group performed significantly poorer than the HIV-UU controls on all measures except that of Language and Sensorimotor functioning. The measures which were comparably deficient correlate with specific impairment in executive functioning, attention, inhibition, initiation, cognitive flexibility, planning, short-term visual and verbal memory, and visuo-motor function when compared to a typical, matched sample. The findings are moderately consistent with those of two other studies which investigated the general cognitive functioning of older school-going HIV-EU samples. Thirty four percent of the exposed sample in the Child and Adolescent Self Awareness Health Study scored below the 10^{th} percentile in the Peabody Picture Vocabulary Test (Brackis-Cott et al., 2009). This measures receptive language abilities and has a good construct validity with the Weschler VIQ and FSIQ indices (Brackish-Cott, et al., 2009; D'Amato, Grey, & Dean, 1988). Similarly, Kerr et al. (2014) conducted a study of young school going children (5-11 years) using a selected battery of subtests from the WPPSI-III and Stanford Binet-II batteries, and found consistent and significantly lower scores on the VIQ, FSIQ and Stanford Binet Bead Memory subtest in HIV-EU samples when compared to HIV-UU controls. No significant differences were found in the PIQ of the WPPSI-III, or between the Sentence, Digit or Object Memory subtests of the Stanford Binet-II.

An attempt to identify convergence between the findings from the present study, and the very limited research on similar samples elsewhere, is complex. The other available studies do not all make use of between-group comparisons with HIV infected and HIV-UU groups, and instead use normative percentile scores which are standardised on predominantly Western, English speaking samples similar to the HIV samples of interest. Further, in the present study, the subtests relying on culturally- and linguistically-bound skills similar to that assessed by the Peabody and VIQ of the

WPPSI-III were removed (Kerr, et al., 2014), which makes comparison difficult. Comparison between the findings of the current study and other research is also limited by incongruences in the ages of samples. The CASAH study was conducted in children older than this cohort (9-16 years) (Brackis-Cott et al., 2009), and the WPPSI-III has a ceiling of 7 years, 2 months (Kerr, et al., 2014). Because of this, there appears to be very little overlap in the areas of deficit between that of the two previous studies and this study, largely because of the difference in psychometric measures employed. Consequently, the differences between these the present study are likely to methodological in nature.

The aforementioned difficulties in identifying meaningful degrees of overlap between the findings of the present study and the limited available literature on HIV-EU school going populations do not only refer to a lack of overlap in psychometric measures, but more broadly to issues of test construction, appropriate test use and test bias as well. It is therefore apt to discuss between group differences in the Language Index. Significant differences on the Language Index were only identified between the HIV-infected and HIV-UU group, while the HIV-EU group was not significantly different from either group, and mean scores resided in between those of the two other groups. Verbal impairment in the context of HIV infection is well-established in studies in many languages, and is supported by the findings of the current study (Becker, Lopez, Dew, & Aizenstein, 2004; Hardy & Vance, 2009; Reger, Welsh, & Razani, 2002). Compromised verbal performance in HIV-EU samples has been identified in cross-cultural contexts by (Brackiss-Cott et al., 2009; Kerr et al., 2014), but as discussed above, can only be partially supported by a descriptive analyses of language performance within this study. Issues of test bias, however, remain relevant to the assessment of this clinical population, and there are salient issues of bias within this test which undermine its construct validity in a sample that does not speak English as a first language. While every effort was made to only include subtests which were less affected by linguistic biases (subtests which could be interpreted and answered in a vernacular language with no effect on its correctness or not), test equivalence in this manner is never possible.

A within-group analysis of both the HIV-I and HIV-EU groups revealed that Attention and Executive Functioning were relative strengths, while Language, Memory and Learning and Visuospatial Perception were relative weaknesses. These weaknesses were not apparent in the HIV-UU group, where only the Attention and Executive Functioning index was identified as a relative strength in the HIV-UU sample, but had weak power ($P = 0.168$) and explained variance ($\eta^2 = 0.044$). The general relative strength of the Attention and Executive Functioning domain is a

complementary finding to the relative strength of visuospatial working memory in all three groups of the sample, and underscores support for the bilingual advantage in executive processes in children. The Attention and Executive Functioning domain of the NEPSY-II measures the self-regulatory constructs of inhibition, initiation, cognitive flexibility, planning, and sustained and selective attention. While there is an overlap between all of these skills and those employed in the three subtests of the visuospatial working memory index of the AWMA, transfer effects between executive and working memory processing skills are common and could also count for the common bilingual advantage apparent in all three groups (Brehmer, Westerberg, & Backman, 2012; Salminen, Strobach, & Schubert, 2012; Thorell, Lindqvist, Nutley, Bohlin, & Klingberg,, 2009).

Language, Memory and Learning, and Visuospatial Processing were relative weaknesses in both the HIV-I and HIV-EU groups, but not the HIV-UU group. The HIV-UU group also differs from the two HIV-affected groups on two nodes of distinction: HIV status and English proficiency. The associated differences with HIV status have been discussed previously, and could be the result of differential effects of viral infection, viral exposure or a particular socioeconomic environment characteristic of people living with HIV. The other difference refers to the HIV-UU group having the highest levels of English proficiency within the sample compared to the other two groups, and reinforces the earlier criticism of the use of conventional psychometric tests which rely heavily on language and cultural expectations, within linguistically diverse, non-Western samples. Although subtests heavily reliant on language processing were removed from the battery in the present study, the remaining subtests were still reliant on a particular cultural worldview. For example, the Memory and Learning index relied on the memory of a series of faces and names which have a particular cultural location (i.e. only one black face, and names such as Jacob, Mimi, Carl, Jenny, Joe, Maria and Sam which sound foreign, and would not have an existing lexical location in long term memory). Further, while subtests from the Visuospatial Perception index would be superficially free of influence of language and cultural bias, there are elements which could disadvantage non-Western children. For example, the use of a road map to find a house is a suburban concept, whereas many township houses and shacks have a simple reference to a number within a geographical area. There is also evidence that Block Design subtests (as used in this index) have a particular cultural bias, and discriminated against those with deprived educational backgrounds (Rosselli & Ardila, 2003; Shuttleworth-Edwards et al., 2013)

Descriptive analyses of the three samples in this study show that a total of fourteen languages was spoken by this study's participants, and that all participants were at the least

bilingual. This has implications for testing in South Africa and in particular this study, as both local and international research have highlighted the difficulties and limitations of psychometric testing in second (or in this case, third or fourth) languages (see Bedell et al., 1999; Claasen, 1997; Foxcroft, 2004; Van de Vijver & Rothmann, 2004; Van Eeden & Mantsha, 2007). Further, the reference to 'language' as a variable of influence in studies such as these, which include it as an extraneous influence, can be over-simplified and misleading. In linguistically diverse contexts, language is a complex construct that refers to far more than simple proficiency, but also represents a discursive cultural accessibility which intersects with notions of class, power and educational status (In the current study, parametric correlations indicated that children who did not speak English as their first language, were associated with poorer socioeconomic status, (r = -0.169, p = 0.005). By way of example, in many cases where participants' caregivers in the current study identified themselves as first language English speakers on the questionnaire, it was obvious through testing that this was in fact not the child's dominant language. While many in this small group who identified themselves as English speakers may have been proficient in English, their primary linguistic exposure at home was clearly not English. This further conflates the distinction between participants into the two categories (English First Language and English Additional Language), and represents the vested interest that caregivers had in portraying their children as 'English speaking'. It is common for parents to promote English in their children as it is perceived as the language of economic empowerment and academic achievement (Broom, 2004; De Klerk, 2006). Caregivers also frequently identified their children as attending English-medium schools. This identification should not be thought of as an environment where children are taught exclusively in English. Due to the large numbers of children attending these schools who are not first language English speakers, teachers are regularly required to speak in a mix of other languages to promote classroom understanding (Jordaan, 2012; Webb, 2010).

While every effort was made to account for the influence of English proficiency by including Sentence Repetition as a measure of proficiency, linguistic aptitude remains a salient influence on cognitive functioning. This is apparent in the existence of moderate correlations between the Sentence Repetition Test and the verbal indices of the AWMA (verbal short term memory, r = 0.510, p<0.001; verbal working memory, r = 0.458, p<0.001) and four of the NEPYS-II indices (Language (r = 0.736, p<0.001), Memory and Learning (r = 0.683, p<0.001), Social Perception (r = 0.526, p<0.001) and Visuospatial Processing *(*r = 0.555, p<0.001)). Both the verbal and visuospatial working memory indices of the AWMA and the Attention and Executive

Functioning and Sensorimotor Indices of the NEPSY-II had low but significant correlations with the SRT (VSSTM: $r = 0.266$, $p < 0.001$; VSWM: $r = 0.346$, $p<0.001$; Attention and Executive Functioning: $r = 0.374$, $p<0.001$; Sensorimotor: $r = 0.405$, $p<0.001$), suggesting that while these constructs may be less vulnerable to the influence of English proficiency, they are still mediated by language These issues regarding the influence of language in this study highlight the difficulty of conducting applied research in linguistically diverse environments where language is not the variable of interest. It also highlights the need for psychometrically valid assessments in vernacular languages, and represents broader issues regarding the intersection of language with socioeconomic status, schooling, culture and power.

Working Memory Structure in the Context of HIV-Infection and –Exposure

The final section of this study concerns the degree to which the working memory development of the three groups followed the structural expectations of typical samples of children of the same age. This investigation was employed in an attempt to avoid comparing working memory performance of the three groups to established British norms on the AWMA, which would undoubtedly be biased because of issues of culture, language and test-wiseness.

Initially a Multigroup CFA (M-CFA) was employed to identify between-group differences in the underlying latent variable structure of working memory in the three groups. While the use of M-CFA for invariance testing has grown substantially in recent years, there has been a relative lack of studies that have examined the factorial invariance of cognitive constructs in paediatric samples. The majority of studies to date have been concerned with establishing configural and metric invariance, ignoring higher levels of factorial invariance such as scalar invariance, such as that employed here (Varni, Limbers, & Newman, 2009). The use of this technique within the knowledge domains of working memory development, and atypical paediatric samples is therefore relatively novel.

Measurement invariance across the three groups was examined through four additive steps. First, when all of the parameters were left free to co-vary in the three groups, the results showed an unacceptable configural invariance, which indicated that working memory was not conceptualised in a similar way across the three groups, as they had significantly different latent factor structures. When factor loadings were forced to be equal in both groups to examine the metric invariance, there were significant changes in the model fit. This implied unsuccessful metric measurement equivalence in both groups, which reflected the fact that the regression trajectories connecting the latent factors to the observed variables were not equal for the three groups. This

indicates that the relative contribution of the working memory measures to the factors (components) is unequal across the three groups. The measurement of scalar invariance was achieved by constraining both the intercepts and residuals to be equal in both groups. However, the analysis suggested an unacceptable scalar equivalence. Similarly, the results of the test for strict equivalence did not show an acceptable strict invariance across both groups, which indicated that there were significant latent factor structure differences between the groups.

These findings are unsurprising in relation to the significant between-group differences identified in the multivariate analyses. They suggest that there are significant differences in the latent variable structures of working memory in the three groups. While the method does not account for where these deviations lie, two of the three groups within the sample have atypical neurodevelopment. Within the HIV-I group, this has been observed in the convergence of a large body of research evidence supporting atypical cognitive functioning. However, there is less research in HIV-EU samples, the findings from this study suggest that working memory development in this group may be atypical. It would therefore stand to reason that the fractionation and maturation of the latter group's working memory structures would deviate from that expected of typically developing children of the same age. However, while this group's working memory factor structure deviated significantly from the typical expectations of the four-factor model, a within-group investigation of factor structure revealed that the four factor model provided in all three of groups. Hence, the four-factor structure remains robust to the effects of HIV status in comparison to other factor structures.

The findings from the within-group confirmatory factor analysis within the HIV-I and HIV-EU samples confirm the proposed hypotheses and showed that none of the proposed models achieved sufficient model fit when assessed for the presence of a non-significant chi-square test. This is in comparison to the HIV-UU group which achieved a non-significant chi square model fit for all proposed models, and would suggest that the working memory development of both the HIV-I and HIV-EU groups is *less* typical than that of the HIV-unexposed control group. However, when the two HIV-affected groups were analysed in isolation using AFI estimates and a chi-squared difference test, the findings suggest that while working memory development within these two groups is atypical with regard to the HIV-UU control group, their structures appeared to be aligned to developmentally expected fractionations. This is evidenced by the ranked support for a four factor model as the primary model providing best fit within both groups, where there was an age-expected differentiation of the verbal and visuospatial domains in the short term- and

working memory components. The HIV-I group achieved slightly better model fit than the HIV-EU for the four factor model, but these differences were small and are considered negligible (ΔM = 0.033).

The HIV-UU group's results confirmed the proposed hypothesis and yielded non-significant chi square results for all models. However, the AFI's and ranked test revealed that while the four factor model obtained the best model fit, the two two-factor models (structured on STM and WM, and verbal and visuospatial models) were preferred over the three factor model proposed by Baddeley (2000) which indicate mature adult working memory functioning. One explanation of this finding is that the age of the sample influenced this result. Their assessment at the node of transition within working memory fractionation, where there is a shift in emphasis from visuospatial to verbal encoding at about the age of seven years, may have exacerbated the effect of the two nodes of fractionated distinction (short term vs. processing component, and verbal vs. visuospatial). The modular functioning of a proficient domain-general central executive was not yet foregrounded, as the development of this samples' working memory is yet to fully mature. Alternatively, the two-factor models could have been dominant because of the particular relative strength of general visuospatial and visuospatial working memory skills in this sample.

The pervasive and consistent preference of the four factor model across the three groups offers sound support for the previous work of working memory experts who have proposed that working memory development is fractionated, and develops asymmetrically at various points in childhood (Alloway, Gathercole, Willis et al., 2004; Alloway, Gathercole, Pickering et al., 2006; Alloway & Alloway, 2013; Gathercole, Pickering, Ambridge et al., 2004; Gathercole & Jarvis, 2003). However, while these results confirm that working memory performance in childhood is fractionated, and shows differential rates of maturity at different points, they also show that these expectations are vulnerable to the influence of contextual factors, such as multilingualism, environmental practice effects and disease. This is evidenced by two main findings. Firstly, deviations in the HIV-UU model-fit rankings, and strengths and weakness profile from the expected pattern of typical development, indicate that the multilingualism of this subsample could have reversed the expected dominance of visuospatial short term storage, and instead provided the visuospatial working memory component with an increased proficiency possibly because of the effect of the bilingual executive advantage.

The second is that, while the working memory development in both the HIV- and HIV-EU samples favoured developmentally-expected fractionations of verbal and visuospatial short term-

and working memory, their model fit was significantly distinct from the typically developing control group, and suggests that there is a difference in the way in which working memory develops in the context of both HIV infection and/or exposure. Neurologically, typical developmental fractionation is a response to considerable maturation of the lateral prefrontal cortex at around the age of seven years of age, and is characterised by a dramatic reduction in neuronal density, a marked expansion in the dendritic trees of pyramidal cells and an increase in the volume of grey and white matter (Tsujimoto, Kuwajima, & Sawaguchi, 2007). HIV infection of the CNS is known to reduce the volume of grey brain matter (Barks, Sun, Malinak, & Silverstein, 1995), and interfere with the process of synaptic expansion (Sanchez-Ramon, Bellon, & Resino, 2003) which could adequately explain why the process of fractionation within HIV-I children is atypical. Explanations for atypical rates of fractionated maturity within HIV-EU samples hold less certainty because the exact mechanisms of neuronal impairment in HIV-EU are poorly understood. However, neuroimmunological animal studies concerning the interaction of the nervous and immune systems under conditions of in utero viral exposure have proposed that the blueprint for neurodevelopment is disrupted in conditions of maternal viral infection (foetal exposure), and which could result in atypical neural structures in the infant (Meyer, Feldon, & Fatemi, 2009; Garay & McAllister, 2010).

A model fit analysis also provides one final point regarding the distinction of working memory from general cognitive functioning. The ranked model fit comparisons of the three groups showed that the one-factor general model structure was the least preferred model and provided the poorest model fit across samples. This provides confirmatory support for the fractionated modular structure of working memory, and also differentiates it from general intellectual functioning. If working memory and IQ were indistinct constructs, working memory performance should also be underpinned by a reified '*g*' which determines success in the various indices. The distinct fractionation of working memory's modular structure contradicts this idea.

This section employed both a between-, and within-group approach to the investigation of working memory model structures within three different conditions of HIV status. The two salient findings from this section are seemingly contradictory. The between-group comparison of factor structures between the three groups suggests that the four factor structure usually reflective of working memory fractionation in typically developing school beginners, is compromised under conditions associated with HIV-status. On the other hand, the within-group analyses found that the four factor structure remained relatively robust to the effects of HIV association when compared to alternate working memory structures. This is supported by the findings of the

multivariate analyses in the current study, and indicates that working memory developmental functioning may be atypical in the contexts of HIV-infection and –exposure. However, despite these deviations in typical development, the four-factor model proposed by other paediatric working memory research remains the most robust to these influences within each group.

Summative Implications of Findings for the HIV-I, HIV-EU and HIV-UU Groups

This section summarises the findings and explanations for the three groups. There is of course always the possibility that these differences are a chance finding, and are uninfluenced by associations with the HI virus, its treatment, and the environment. However, the study had excellent power with a triangulated three-group design that allowed for the comparative observation of each group against two other baseline conditions. This increased the internal validity of the study, despite being correlational in nature.

The HIV-I group showed significant impairment in both their verbal and visuospatial working memory abilities. This was hypothesised to be a result of the executive functioning hypothesis which attributes executive control deficiencies to a limited capacity of the fronto-striatial cortical circuits to mediate the increasing attentional demand as a result of viral damage to both the structure and functional mechanisms responsible for this process. The verbal domain was a relevant weakness of this group, and is attributable to the consequence of HIV-associated neuronal damage to those cortical structures responsible for verbal processing, but also a function of test. Conversely, visuospatial working memory was a relevant strength in this group. This may represent a bilingual executive functioning advantage applicable to the entire sample who all spoke more than one language. This finding, together with the robust performance on the Nonword Recall and Counting Recall subtests also present working memory assessment as a potentially less biased means of assessing cognitive potential in linguistically diverse samples.

The HIV-EU group was also found to have impaired working memory performance relative to unexposed controls, and was statistically indistinguishable from the HIV-I group on both working memory indices of the AWMA. Similar to the HIV-I group, verbal scores were deflated in comparison to visuospatial performance, while visuospatial working memory was identified as a relative strength. The Nonword Recall and Counting Recall and Processing subtests were also relatively robust to the effects of bias, which provide further support for the use of these aspects of working memory assessment as a less biased measure of cognitive potential than IQ.

Between group comparisons of performance of the two clinical groups on the NEPSY-II indicate widespread impairment across domains, except that of Social Perception which appears to be relatively robust to the associations with HIV status. Measures of Language, Memory and Learning and Visuospatial Processing were relative weaknesses in both clinical groups and are believed to be a function of both HIV-associated impairment, as well as test bias. The Attention and Executive Functioning domain was identified as a relative strength in all three groups, and this provides support for the bilingual executive processing advantage hypothesised to explain the relative strength of visuospatial working memory in the multilingual sample.

The analysis of the developmental models converge across the three groups to provide evidence of developmental fractionation of the modular structure of working memory in both typical and atypical samples. However, differences in the degree to which this fractionation complied with expectations of typically developing children suggest that HIV status is associated with subtle differences in the construct.

An explanation of these differences is increasingly the mandate of researchers interested in the neurodevelopmental associations of HIV. There are four possible hypotheses that could explain these differences:

1. Neurodevelopmental effects are unrelated to viral or antiretroviral influences, and are instead the result of the familial and socioeconomic influence of HIV.
2. Neurodevelopmental effects are due to immunological changes in the infant as a result of maternal HIV infection.
3. Neurodevelopmental effects are due to the influence of PMTCT antiretrovirals.
4. Neurodevelopmental effects are a function of an interaction of all three previous hypotheses.

The first hypothesis locates the potential differences in neurodevelopment in both infected and exposed participants within the wider socioeconomic circumstances of HIV infection. A large body of research converges to present a series of correlations between HIV infection, socioeconomic markers and neurocognitive development which reveals the HI virus as more than a biomedical status, but as a set of associated lifestyle vulnerabilities that affects whole communities (Barnighausen, Hosegood, Timaeus, & Newell, 2010; Hargreaves, Morison, Chege et al., 2002; Lewis, Kaiken & Hoyt, 1994; Lurie, Fernandes, Hughes, & Arevalo, et al., 1995; Salter-Goldie, DeMatteo, King, Wells and the Multisite Coinvestigators, 1997, as cited by Blanchette et

al., 2002). HIV is notoriously associated with poor socioeconomic status, malnutrition, poor mental health, poor levels of education, single or double-orphanhood and unemployment. Coupled with this, HIV infection is predominantly found in poor communities, who, due to the legacy of Apartheid in South Africa, have the additional burden of resource limitations such as food security, housing threats, access to clean sanitation, access to quality schooling, changes in traditional family and community structures and the necessity of migrant labour (van der Walt, Bowman, Frank & Langa, 2007). While none of these factors can be individually responsible for poor cognitive functioning, they work together to create an environment where typical learning is challenging. HIV-EU children would have had an HIV-infected mother, and would thus be exposed to a very similar social environment to that of an HIV-infected child. Hence, in the same way that is not possible to isolate the effect of viral infection on neurodevelopment because of the environmental influence, it is impossible to remove the potentially detrimental effect of difficult living circumstances from understanding the influence of HI exposure.

The second hypothesis attributes the atypical development associated with HIV infection to both a direct and indirect mechanism of viral attack. The direct method is often termed the 'Trojan horse mechanism' and involves the infection of macrophages and microglia (Epstein & Gendelman, 1993; Pardridge, 2005). The supportive function of these cells means that they are frequently infected, and are commonly found in the basal ganglia and white matter of the CNS. Macrophages also secrete chemokines which attract more (often infected) leucocytes from the blood, providing the HI virus the ability to infect and replicate itself within the brain. The indirect method of CNS infection is done via chemical poisoning, where microglia and macrophages secrete a battery of inflammatory cytokines and neurotoxic factors, which are believed to be toxic to nearby neurons and cause injury to these surrounding cells (Gonzalez-Scarano & Martin-Garcia, 2005; Koekkoek, de Sonneville , Wolfs, Licht, & Geelen, 2008; Rausch & Davis, 2001). The explanatory mechanisms behind the associated impairment in HIV-EU are less developed. The first draws on the indirect mechanism of HIV infection explained previously, and proposes that the altered immunological profile in the foetus is a result of exposure to the inflammatory by-products of infection of the cells of the mother. The second refers to neuroimmunological interactions where alterations in the immunological profile of the developing foetus (as a result of in utero viral exposure) affect the typical development of essential neural structures of the CNS before birth (Garay & McAllister, 2010; Meyer, Feldon, & Fatemi, 2009).

The third hypothesis attributes exposure to ART as a mechanism to alter the immunological picture of both HIV-I and HIV-EU children. There is equivocal research to support both of these hypotheses, but a lot of their validity is limited in methodology (Cotton, 2002; Griner et al., 2011; Jeremy et al., 2005; Van Rie, Mapuala, & Dow, 2008; Van Rie, Mapuala, & Stewart, 2009). This is because it is almost impossible to isolate the effect of the maternal HI virus, maternal ART, and PMTCT regimens given to the infant at birth. In order to do this, a sample of three groups of children would be needed for comparison: the uninfected offspring of treatment naïve, infected mothers (where the likelihood of uninfected infants would be reduced); the offspring of mothers on cART, but whose infants do not receive PMTCT regimens at birth (unethical), and a group where both mother and infant receive all prophylaxis available. To date, no studies have made this comparison, and many use samples already on treatment (for example, Toumala, Shapiro, & Moffenson, 2002; Williams et al., 2010).

The fourth, and probably most likely, hypothesis allows for an interaction of all three of these hypotheses. Here, the effect on neurocognitive functioning is not simply a summative combination of the nett effects of the environment, maternal viral infection, and ART exposure, but an interaction of them in an additive manner. For example, the degree of maternal infection (as measured by viral load) has an impact on subsequent ART efficacy, and optimal PMTCT regimens. Maternal viral load has also been found to have a differential effect on the degree of foetal immunological alteration in exposed infants, as well as viral load in infected infants (Williams, Marino, Malee et al., 2010). In a study examining the interactive effect of the home environment and HIV infection on cognitive functioning in a group of 42 child-caregiver dyads, level of WHO clinical staging (a measure of HIV infection severity) was a mediator of the influence of the home environment on IQ (Coscia et al., 2001). To that end, it is highly likely that viral exposure in utero, ART exposure (both in utero and shortly after birth), as well as the socioeconomic environment interact to impede neurodevelopment in HIV-infected and HIV-exposed infants. The final sections that follow document the strengths and limitations of the study, as well as a summative outline of the theoretical and practical contributions of the study to current praxis.

Limitations of the Study

The study was not without its limitations. Firstly, its methodology did not allow for absolute between-group equivalence in terms of English proficiency, parental education, social capital, home stimulations etc. While socioeconomic status was statistically accounted for through the use

of the Living Standard Measure, some general factor would have been useful to account for the extraneous influence of socio-cultural advantage (e.g. available parenting, employed parents, parental education, access to preschool, assumed quality of preschool, and possible quality of schooling now). This is believed to be related to, but not the same as, socioeconomic status. While difficult, or near impossible to quantify, the use of a quantitative representation of a child's access to socio-cultural benefits would have been helpful. In hindsight, increased numbers in the pilot study should have highlighted these between-group demographic differences and allowed for the creation of a measurement for these things which could have been statistically co-varied across the three subsamples.

The demographic questionnaire and screening criteria also did not account for the various cART and PMTCT regimens used by the mother-child dyads. The validity of the study could have been improved had there been some knowledge about the various medications used, as well as more information about maternal health status. This was initially considered in the conceptualisation of this project, but it was disregarded for two reasons. Firstly, the aim of this study was to investigate the neurocognitive profile of the HIV-exposed child, and not to provide accurate causal inference about the reasons behind possible differences. Secondly, the number of permutations of drug combinations across the sample would have rendered the study invalid. ART clinics do not follow strict clinical guidelines, and drug regimens differ according to personal preferences by doctors, availability of medications at public health pharmacies, and individual patient immune response and adherence.

In an attempt to formalise and then compare the living conditions and possible socioeconomic status of the three groups, the demographic data includes a reference to 'Dwelling' and allowed for a forced choice between, 'house', 'flat', 'shack', 'shelter' and 'other'. These choices were often confusing to participants who frequently explained that while they did live in a house or a flat, the family actually resided in *one room* within that type of dwelling. Where 'room' was specified under the 'other' category, this was recorded. This striation is consequently misleading and should have been thought through prior to data collection in accordance with current data about South Africans (Statistics South Africa, 2013a, 2013b).

The study's method and use of psychometric measures did not include a means of measuring the episodic buffer which is an established component of the multicomponent model in children as young as between four and six years old (Alloway, Gathercole, Willis et al., 2004). This was largely due to the difficulty in assessing it accurately. Within samples who do not speak

English as a first language, the quantification of the functioning of the episodic buffer by linking it with phonological short term storage is conflated because there are inconsistencies in the semantic associations that such content would have in long term memory. The processing demand of Sentence Repetition in an additional language is also expected to be higher, and so the increased activation of executive functioning could mask or misinterpret the presence of the episodic buffer.

Lastly, a more thorough investigation of the sample's levels of multilingualism would have been helpful. All of the children in the sample spoke more than language, where English was frequently an additional language acquired only in formal schooling. Further, for many of the EAL participants, English was not their second language, but their third or fourth language. An investigation of the degree of multilingualism using a tool such as the LEAP-Q would have provided more useful information regarding proficiency, age of acquisition, environmental exposure, and how these interact with both HIV status, and working memory performance (Blumenfeld & Kaushanskaya, 2007; Soliman, 2014).

Strengths of the Study

The study's method and analyses possess a number of research strengths. Firstly, the comparison of three groups allowed for a clearer isolation of the under-investigated HIV-EU neurocognitive profile relative to both HIV infection and an unexposed control, which provided the opportunity to identify salient differences thereof. The specific age, gender and language matching of the NEPSY-II sample also allowed for good group equivalence on those nodes of influence. Secondly, previous investigation into the two clinical groups have conventionally used general intelligence batteries, or selected only a single component of the construct to be assessed. The present study utilised the full AWMA battery (verbal and visuospatial short term and working memory), and a substantial battery of the NEPSY-II. This allowed for a comprehensive picture of the neurocognitive profiles assessed by these instruments in all three groups.

The study investigated the working memory performance of a non-Western, linguistically diverse sample of school beginners and its associations with HIV status. The inclusion of this sample created greater generalisability to those children elsewhere in the world who are affected by HIV, and who are typically not the white and middle class samples often characteristic of the standardisation norms of Western psychometric measures. The study also made an effort to quantify and then control for the extraneous influences of socioeconomic status and English proficiency which are inherent in diverse samples within the South African context. The analysis

used of a confirmatory factor analysis to determine the degree of typical working memory development instead of using established normative comparisons. This was done to avoid between-group comparison of the three subsamples with Western norms, and by so doing, to reduce the discriminatory effects of test bias.

The establishment of between-group differences, as well as the use of confirmatory factor analysis also provided a promising direction for future research within the two HIV-affected samples. The presence or absence of group difference is often used as a basis for conclusions as to whether development might be delayed (proceeding in line with mental age, but delayed according to chronological age) or different (not proceeding in line with mental age). Even if this can be concluded, as broadly identified in the two HIV affected samples in this study, it does not identify why this has occurred. Thomas, Annaz, Ansari, Jarrold and Karmiloff-Smith (2009) argue for an alternative approach to understanding developmental disorders, by advocating for the use of 'developmental trajectories'. Here, the development of working memory would be examined across a wide age or mental age range. For example, memory span might be assessed in HIV-EU children between the ages of 3 and 16 years. Memory span is then plotted against chronological or mental age for every child to see if there are relationships between the two variables. A line of best fit would indicate how memory span changes with increases in chronological or mental age (Henry, 2012). In order to establish whether this development is atypical, the same procedure is used for a typical group comparison. Direct group matching is not necessary, but the typical group must have children of the same mental and/or chronological age. Comparison between the two lines of best fit allows for the identification of delayed onset or rate of development, and the distinction of typical and atypical development without the risks of attrition and conflating effects of viral exposure in longitudinal studies.

Theoretical Contributions

This thesis has made a number of theoretical contributions to the working memory 'map'. Firstly, it profiled the working memory performance of a sample of HIV-I children. The two primary findings of this HIV-I working memory profile are at first glance an apparent contradiction, but represent a distinction between the within- and between-group results of the group. The between-group analysis identified a general (verbal and visuospatial) working memory impairment relative to uninfected controls, while short term visuospatial performance within this sample was not significantly impaired compared to other groups. This suggests that working memory abilities may be more vulnerable to the effects of HIV and the associated factors thereof (ART, the

environment), while short term stores may be more robust to these influences. Within the HIV-I group, verbal short term storage and verbal working memory abilities appeared to be relative weaknesses, while visuospatial working memory appeared to be a relative strength. This would suggest that HIV associated impairment is primarily associated with impaired functioning of the phonological loop and the components of the central executive relevant to verbal functioning, while the visuospatial sketchpad appears to be relatively intact. However, this asymmetrical distinction which indicates a differential impact on the two slave systems could be a misrepresentation and may be a function of test bias within a sample who did not speak English as a first language.

The study also contributes to the working memory 'map' by providing a profile of the working memory performance of the relatively under-researched HIV-EU population of children. Similar to the profile of the HIV-I children, between-group analyses of the HIV-EU group show that viral exposure appeared to primarily impair both verbal and visuospatial complex working memory when compared to unexposed controls, while short term stores remained more robust. The within-group analysis of the HIV-EU group in isolation, showed that the visuospatial sketchpad appeared to remain relatively intact in comparison to the phonological loop and the central executive components related to verbal processing. This would suggest that the associations of HIV-exposure with the components of the working memory system are not impartial, and imply that there is selective impairment to the phonological loop and the verbally mediated aspects of the central executive. Again, this could however be a function of test bias within a linguistically diverse group of children who did not speak English as a first language. The performance of this group of children on the NEPSY-II also offered an insight into their general neurocognitive performance. While the NEPSY-II offers more detailed analyses than the outputs of conventional IQ tests, it is limited by its reliance on linguistic and culturally located subtests. Nevertheless, between-group performance indicated significant impairment on measures of Attention and Executive Functioning, Memory and Learning and Visuospatial Processing when compared to an unexposed control group, and were statistically indistinguishable from the performance of the HIV-I samples on all NEPSY-II domains, except that of Sensorimotor abilities. This would suggest that, even after the effects of language and test bias have been removed, HIV-EU school beginners show atypical cognitive performance that is similar to, but not as impaired as, HIV-I samples.

The third theoretical contribution that this study makes to existing working memory research concerns the structure of working memory development within non-Western, non-

English speaking, and atypical samples. The four factor structure, which differentiates performance along the axes of storage and processing, and domain representation (verbal and visuospatial), was apparent in the typical HIV-UU sample. However, this sample was not Western, nor did the participants speak English as a first language, and so this finding provides an indication of the robustness of the four-factor structure of working memory to the effects of culture and language within typically developing South African children. The within-group confirmatory factor analyses provided further evidence that this four factor structure was robust to the associations of HIV status as well. While the working memory structures of the two HIV-affected groups deviated from typical expectations, the four-factor structure obtained the highest levels of model fit within each of the groups, despite the effects of HIV infection and exposure. This supports the conceptualisation of the working memory structure of school beginners as comprising the phonological loop, the visuospatial sketchpad, and a central executive which has separate, domain-specific functions for verbal and visuospatial processing.

While this study has highlighted many areas of impairment within the two clinical samples, there are also a number of contextual factors characteristic of these two vulnerable populations which may provide them with a number of cognitive advantages. The salient one is the relative strength of visuospatial working memory, and performance on the Attention and Executive Functioning subtest of the NEPSY-II. This may to be a result of the multilingualism of the sample and the fact that speaking more than one language provides a bilingual executive functioning advantage.

The final theoretical contribution is that working memory assessment may provide a less biased means of cognitive assessment for linguistically diverse children who come from non-Western settings. Here the identification of Nonword Recall and the Counting Recall subtests of the AWMA as relative strengths in all three groups suggests that their relative independence from verbal instructions makes them promising tests of cognitive potential. Further, the homogeneity of performance on factors related to of visuospatial short term and working memory in the typical sample is also support for the use of these type of tests in a future where conventional psychometric testing is increasingly losing validity.

Practical Contributions and Directions for Future Research

The findings of this study, as well the reasons attributed to the neurodevelopmental differences, have implications for both future research and clinical practice. The differences identified with both clinical samples are not without consequence. Working memory, executive attention,

memory and visuospatial processing are critical to subsequent academic development and scholastic success. Souza, Santos, Valentini, Silva, and Falbo (2010) examined the long-term outcomes of 49 perinatally HIV infected adolescents in Brazil. Seventy percent had no sign of HIV infection, eighty one percent had normal CD4 counts, and fifty three percent had an undetectable viral load. However, despite their apparent health, quality of life and maintenance of viral suppression, half were failing their grade, and one in five had dropped out of school. The authors posited that poor language skill, as well the failure to detect HIV-associated neurocognitive impairments early enough were reasons for these results. A similar trend of high school drop-out rates, and poor school performance is apparent in South African schools (Beyers & Hay, 2011; Buchel, 2009; Dlamini, 2007). Therefore, the mandatory response to paediatric HIV in South Africa is no longer to save lives, but to save brains, and the practical response should be focused around the areas documented below:

Firstly, working memory deterioration should be considered as a primary marker of the effect of HIV infection in otherwise asymptomatic children, and be used as an indication to either initiate treatment or begin cognitive rehabilitation to prevent further impairment. While recent policy changes have made it mandatory for all HIV-I children under the age of five years to begin cART (Department of Health, 2014), there are many older children who do not yet qualify for treatment (CD4 not yet below 500cells/µl), and who suffer more subtle effects of neurocognitive impairment. These effects appear to be irreversible and occur during critical periods of neurodevelopmental regrouping that are essential for basic academic success (Laughton, 2013; Vimpani, Patton, & Hayes, 2004).

Secondly, research into the long-term effects of HIV exposure in school going children is essential if we are to understand where the deficits occur and intervene appropriately. To that end, longitudinal investigations concerning the cognitive profile of HIV-EU children should proceed with this population as they age in order to document their strengths and weaknesses. Coupled with this, the HIV-exposed child needs to be re-introduced as a clinical population in need of clinical care and multidisciplinary support. Further, both the exposed and infected child would benefit from early cognitive intervention to protect against neurocognitive weaknesses apparent later on in their formal schooling.

Thirdly, the use of working memory assessment offers a promising alternative to conventional IQ assessment (Engel, Dos Santos, & Gathercole, 2008), and needs further research and

psychometric credibility as a less biased means of assessing cognitive functioning within linguistically diverse, disadvantaged samples.

Conclusions

Baddeley's (2012) comparison of the development of any theory, particularly that of working memory, to that of a 'theoretical map' consisting of both well-established terrain and more unfamiliar areas, complements the findings of this thesis. His notion that a 'good' theory and the underlying 'good' science behind it, should not be judged by its degree of predictive accuracy, but should instead be judged by its productivity – both in the answering of knowledge gaps (what he refers to as 'empty spaces'), as well as the asking of good questions that direct future research (the subsequent creation of 'empty spaces'). This thesis has provided both of these. While it contributes to existing working memory theory through the profiling of two atypical HIV-affected samples, it also highlights the importance of working memory assessment as a promising alternative in the less biased assessment of atypical and diverse populations.

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