

# **Cognitive Functioning of Child Protection Clients in Secure Care: A Neuropsychological Study**

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## Abstract

The aim of this research was to carry out a systematic prospective study of the cognitive functioning of young persons residing in a secure care facility. These adolescents have been identified as being at an immediate risk for harm and are placed in a secure facility to establish safety. Typically, these young persons have been in protective care for some years, and represent a cohort of maltreated children at the severe end of the spectrum. More recently, it has been recognized that as a group, these children are exposed to risk factors for neuropsychological deficit. The present study adopted a neuropsychological perspective to document the pattern and extent of their cognitive impairments.

Participants' cognitive functioning was assessed with a number of instruments from the following domains: learning and memory, processing speed, executive functioning and attention, language, visuo-perceptual function, as well as measures of depression, anxiety and posttraumatic stress. The Secure Welfare group included 49 adolescents recruited from the Victorian Department of Human Services Secure Welfare Service, aged between 12-16 years ( $M=14.5$ ,  $SD=1.2$ ). A comparable control group ( $n=52$ ) of participants aged between 12-16 years ( $M=14.5$ ,  $SD=1.2$ ) also matched on gender and SES were recruited from secondary schools in Melbourne, Australia.

The results of the study indicated that Secure Welfare participants performed significantly worse than controls in all cognitive domains, including working memory, executive functioning, learning and memory, visuo-perceptual function and processing speed. Overall cognitive functioning, as represented by the WISC IV FSIQ was almost one standard deviation below the population mean. The data suggests that most adolescents with histories of maltreatment experience a number of cognitive difficulties, and, these difficulties are not specific to those identified as intellectually disabled. The implications of such deficits are potentially profound, influencing academic performance, adaptive behaviour and social functioning. As these deficits are not consistent with a specific neuropsychological disorder, these adolescents remain misunderstood and unsupported in their activities across various aspects of functioning.

## **Declaration**

“I, Vidanka Ruvceska declare that the PhD thesis entitled Cognitive Functioning of Child Protection Clients in Secure Care: A Neuropsychological Study is no more than 100 000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole, or in part, for the award for any other degree or diploma. Except where otherwise indicated, this thesis is my own work”.

Signature\_\_\_\_\_

Date\_\_\_\_\_

## **Dedication**

*To my daughter Samantha, for arriving during this journey, and teaching me the most important lessons of all...*

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## **Chapter 1: Introduction**

### *1.1 Cognitive Development in Childhood and Adolescence*

The periods of childhood and adolescence are associated with considerable physical, psychological and cognitive development. Cognitive development in childhood, is quite rapid and extensive, with entry into formal education and acquisition of other skills such as sports and music being characteristic of this time (Korkman, Kemp, & Kirk, 2001).

Cognitive development during childhood includes the development of functions associated with reading and language, memory and learning, visuospatial skills and motor skills. By adolescence, most cognitive processes are established and the rate of cognitive development slows (Korkman et al., 2001). Ongoing maturation of existing cognitive processes and further development of specific brain regions continues to occur well into late childhood and adolescence (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Luna et al., 2001). This localised development during adolescence is related to behavioural changes, including, increased self awareness, identity formation and enhanced cognitive flexibility (Blakemore & Choudhury, 2006; Giedd, 2004). This development occurs as a result of normal maturational progression within the brain in line with environmental experiences influencing the plastic reorganization of the brain (Spear, 2004b; Spessot & Plessen, 2004).

Cognitive development during adolescence is characterized by a significant level of maturation occurring predominantly in the frontal and prefrontal lobes of the brain (Anderson et al., 2001; Thatcher, Walker, & Giudice, 1987). The prefrontal cortex is one of the last cortical regions to complete full myelination (Fuster, 1989). The frontal and prefrontal brain regions are typically associated with processes which facilitate higher order thinking and executive functioning (Fuster, 1989; Lehto, Petri, Kooistra, & Pulkkinen, 2003). It has been proposed that frontal lobe lesions are likely to interfere with cognitive skills of working memory, concept formation, inhibitory control, cognitive flexibility and problem solving (Fuster, 1989; H. S. Levin, Culhane, Hartmann, Evankovich, & Mattson, 1991; H. S. Levin et al., 2004). It has been

suggested that the development of executive functions is largely experience dependent, and adolescence is the sensitive period for the acquisition of these skills (Blakemore & Choudhury, 2006). Executive functions are the slowest to develop and have a trajectory that continues through late adolescence and early adulthood (Steinberg, 2004).

### *1.2 Brain Development during Childhood and Adolescence*

Development of the brain and nervous system is an intricate and complex process that begins soon after conception. The neural tube of the foetus in early gestation forms the brain and spinal cord in later development. During foetal development, the process of neurogenesis occurs, where neurons are formed and migrate to predetermined locations forming the layers of the neural tube (Kolb & Fantie, 1997). Once neurons embark on their journey of migration, they go through a period of differentiation, acquiring features that are typical to the brain region they will form (Noback, Strominger, Demarest, & Ruggiero, 2005; Perry, 2002). This process involves the development of axons and dendrites, during foetal development and is followed on by dendritic arborisation after birth (Zillmer, Spiers, & Culbertson, 2008). Axonal and dendrite formation coincide with the establishment of a small number of synapses during the foetal period, whilst synaptic density rapidly increases following birth (Sanes, Reh, & Harris, 2006). During the end stages of migration and well into adulthood, neuronal axons undergo myelination, myelin is a protective fatty sheath, that increases neural impulse conduction and forms the white matter of the brain (Noback et al., 2005). The efficiency of neuronal connections is further enhanced by processes of synaptic pruning. Neuronal connections with no or limited sensory output are removed, whilst those that are frequently stimulated become strengthened, this process also appears to continue many years after birth (Pfefferbaum et al., 1994; Schore, 2001c). This process appears to be a result of both genetic and environmental processes, where numbers of synaptic connections made by the neuron and level of activation received (provided by environmental stimuli) determine whether the neuron will remain (Perry, 2002).

Structural brain development typically occurs in parallel to cognitive development. A classic study by Yavkolev and Lecours (1967) suggested that myelination of various brain regions occurs throughout childhood and adolescence and continues well into the third decade of life. In support of this notion, magnetic resonance imaging (MRI) studies have indicated that there are age related increases in white matter density during childhood and adolescence, particularly in pathways supporting motor and speech functions (Paus, 2005; Paus et al., 1999). Sowell, Thompson, Leonard, Welcome, Kan and Toga (2004) demonstrated that local brain growth measured by increases in cortical thickness occurs at an approximate rate of 0.4-1.5 mm per year between the ages of five and eleven, particularly in the frontal and occipital regions. Significant thickening of cerebral matter was found in the regions surrounding Broca's and Wernicke's areas, the areas that are most commonly associated with the major aspects of speech and language. Extensive cortical thinning was indicated in the right frontal and bilateral parietal and occipital association cortices. As would be expected, significant increases in performances on tasks of verbal functioning measured using the vocabulary subtest of the Wechsler Intelligence Scale for Children- Revised (WISC-R) were associated with cortical thinning in the left frontal and parietal regions. In another MRI study it was observed that white matter density increased in a linear fashion during adolescence at similar rates within each of the four major brain regions (frontal, temporal, parietal and occipital) (Giedd, 2004) thus increasing the efficiency of cognitive processing during adolescence. Sowell et al (2002) also suggested that there was a significant increase in cerebral white matter between the ages of 7 and 16, coinciding with a slight decrease in grey matter during the same period. These changes were predominantly found in the fronto-parietal regions. Increases in myelination and white matter density have been shown to coincide with increases in brain size, brain weight and cognitive functioning (Sowell et al., 2002; Spreen, Risser, & Edgell, 1995).

Increases in cortical grey matter in specific regions of the brain have also been identified during the adolescent period. Just prior to the teenage years, it has been reported that the brain experiences another wave of grey matter overproduction, predominantly in the frontal, parietal and temporal areas (Giedd et al., 1999; Thompson et al., 2000). This period of massive overproduction of neurons has been

found to be quickly followed by a sharp decrease of grey matter at the temporo-parietal region due to synaptic pruning with the beginning of the adolescent period (Gogtay et al., 2004; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999; Thompson et al., 2000). This localized structural development has been associated with the enhancement in skills of language and visuospatial functioning (Thompson et al., 2000).

The frontal cortex continues to develop into young adulthood, Sowell et al (1999) suggested that a decrease in grey matter within the frontal lobes coincides with a progressive increase in myelination in the cerebral cortex between adolescence and adulthood. The frontal lobes demonstrate a maturational process which occurs in an anterior progression, with the pre frontal cortex being one of the last regions to experience grey matter reduction in late adolescence and early adulthood (Gogtay et al., 2004). Huttenlocher (1979) conducted a classic study of post-mortem brain samples of individuals ranging in ages from newborn to 90 years. It was found that the brain experienced a gradual decline in synaptic density between the ages of two and sixteen, coinciding with a small loss of neurons. Huttenlocher also demonstrated that synaptic density in the medial pre frontal cortex reached peak levels at ages three to four years, these levels remained relatively constant until mid to late adolescence when synaptic pruning in the region is thought to occur at a rapid rate. Magnetic Resonance Imaging (MRI) studies have supported these results, indicating that grey matter decreases are generally localised to the frontal and parietal regions during late childhood and adolescence (Jernigan, Trauner, Hesselink, & Tallal, 1991; Sowell et al., 1999).

The extent of synaptic pruning and myelination that occurs within the adolescent brain is largely experience dependent, that is, that those connections which are used frequently are retained and strengthened, whilst those that are used minimally are lost (Cheetham, Hammond, Edwards, & Finnerty, 2007; Cragg, 1975; Kolb, Gibb, & Gorny, 2003; Kolb, Gibb, & Robinson, 2003; Kolb & Whishaw, 1998). This suggests that the onset of adolescence coincides with a period of brain plasticity, where structural changes within the brain lead to the acquisition of new skills related to self regulatory behaviours known as executive functions. As a result, cognitive development within the period of adolescence is dependent on the availability of



stimulating experiences, like those required in infancy and childhood (C. A. Nelson & Carver, 1998). Therefore, opportunities to develop specific skills during adolescence need to be accessible in order to be established within the brains' circuitry (Blakemore & Choudhury, 2006).

Brain activation research using functional MRI (fMRI) has shown maturation of function across widely distributed brain regions (Luna et al., 2001) during childhood and adolescence. Development of specific regions such as the prefrontal cortex, during these periods has also been demonstrated using fMRI (K. M. Thomas et al., 1999). The authors suggest that refinement of synaptic pruning and increased axonal myelination sub serve functional brain development (Luna et al., 2001; K. M. Thomas et al., 1999). Singer (1995) suggests that the structural development of cortical connections are reliant on environmental experience, furthermore, synapses of associative connections are at risk of being removed due to poor levels of activity and only those synapses that are frequently coactivated become permanent within the brain's circuitry. Quantitative electroencephalographic (QEEG) measurement has also provided evidence of incremental maturation of cortical electrophysiological activation consistent with the cognitive stages denoted by Piagetian theory (Hudspeth & Pribram, 1990; Thatcher et al., 1987).

Gender differences in brain maturation during adolescence suggest that developmental processes occurring during this period differ for males and females. Quantitative MRI studies have consistently found that males have larger cerebral volumes than females (Giedd et al., 1996; Lenroot & Giedd, 2006; Pfefferbaum et al., 1994; Sowell et al., 2002), and these volumes relate to differential levels of neuronal density, where increased cortical grey matter accounts for larger brain size in males (Reiss, Abrams, Singer, Ross, & Denckla, 1996). When measuring size of specific brain structures, Giedd, Castellanos, Rajapakse, Vaituzis and Rapoport (1997) found that sex differences in the development of the basal ganglia, with the caudate being larger in females and the globus pallidus larger in males. Males also demonstrated marked increases in lateral ventricular size and the amygdala. Females also showed an increase in amygdala size, however not to the same degree as that for males, marked increases were however found in hippocampal size for females. Sowell et al (2002) also reported sex differences in brain structure volumes of individuals aged between

seven and sixteen years. Females demonstrated greater volumes of the caudate, thalamus and basomesial diencephalon, they also indicated higher grey matter density within the mesial and lateral regions of the temporal cortices.

Giedd (2004) found that cortical grey matter thickness reaches its peak between the ages of 12 and 16. Sex differences were also noted, indicating that peak density levels of grey matter were reached earlier in females, with the exception of the temporal lobes where males developed peak levels earlier. Cortical grey matter loss, in relation to synaptic pruning has been shown to occur earliest in the sensory-motor regions and latest in the prefrontal cortex (Giedd, 2004). This coincides with the major structural and functional changes of the prefrontal cortex during puberty and adolescence (Blakemore & Choudhury, 2006). The prefrontal cortex is a region associated with executive functioning, these functions become most prominent during the adolescent period. Giedd et al (1997) suggested that these differential patterns in neurodevelopment in males and females may provide important explanations for the observed sex differences in child neuropsychopathology such as Attention Deficit Hyperactivity Disorder (ADHD) and depression.

In summary, brain development during childhood and adolescence is profound, involving the maturation of both cognitive skills and physiological components. The impact of environmental and interpersonal factors on such a complex period of development represents an important field of inquiry. The remainder of this review aims to explore the impact of abuse during childhood and adolescence in relation to various aspects of cognitive development.

### *1.3 Neuropsychological Development in Childhood and Adolescence*

Structurally, the brain undergoes major development during the childhood and adolescent years. Although brain size has been shown to reach adult weight by five to ten years of age (Huttenlocher, 1979; Lemire, Loeser, Leech, & Alvord, 1975), dynamic changes occur in the proportions of white and grey matter in the brain throughout the childhood and adolescent periods (Gogtay et al., 2004; Reiss et al., 1996; Sowell et al., 2002). These important processes within the brain reportedly

coincide with the developmental progression of cognition and behaviour during childhood and adolescence.

### *1.3.1 Overall cognitive functioning*

Thompson, Cannon, Narr, van Erp, Poutanen, Huttunen, Lonnqvist, Standertskjold, Kaprio, Khaledy, Dail, Zoumalan & Toga (2001) completed a combination of MRI scans and cognitive tests for 20 normal adult monozygotic and dizygotic twin pairs. They demonstrated that density of frontal grey matter was associated with higher cognitive functioning, where cognitive performance results were manipulated to form 'Spearman's *g*' a numerical value which represents intellectual function similar to the full scale intelligence quotient (FSIQ). Similarly, in a study of 85 typically developing children and adolescents aged between five and seventeen years, it was found that larger total grey matter volume in the brain significantly predicted higher FSIQ scores accounting for approximately 15 percent of the variance (Reiss et al., 1996). More specifically, the prefrontal grey matter volume significantly predicted 20 percent of variance in FSIQ.

It has also been long debated whether overall cognitive function is attributable to genetic or environmental factors. It has been suggested that genetic factors account for up to 62 percent of the variance of overall cognitive functioning, and the remaining 38 percent was accounted for by environmental factors (Boomsma & van Ball, 1998). Whilst others have argued that environment, namely socioeconomic status (SES) accounts for approximately two thirds of the variance in IQ, particularly for those who come from a lower SES bracket (Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Years of completed formal education is also considered an important environmental factor that has a relationship with performance on FSIQ. An adult standardisation sample for the Wechsler Adult Intelligence Scale-Revised (WAIS-R), indicated a linear relationship between years in formal education and FSIQ (Matarazzo & Herman, 1984).

### *1.3.2 Memory and learning*

Memory is defined by the processes of encoding, storage and retrieval of information. These processes provide the brain with the ability to maintain a record or image of prior events and experiences (Squire & Kandel, 2000). Learning involves the

consolidation of information within memory following repeated presentation of the information over a period of time. The ability to retrieve this information from memory when required is evidence that learning has taken place. Essentially learning is the acquisition of information or behaviour as a result of experience (Purdy, Markham, Schwartz, & Gordon, 2001). Like many cognitive functions, memory can not be isolated to a single structure or system in the brain, rather it involves a number of associated structures and interplaying processes (Kail, 1984).

The brain structures responsible for learning and memory function have been associated with the limbic system, with particular emphasis on the hippocampus, however there are a number related brain structures which are involved in memory functioning (M. H. Johnson, 2005; Packard & McGaugh, 1992). The areas of the brain commonly associated with learning and memory function are the medial temporal lobes and the lateral prefrontal cortex (Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000; Fell et al., 2003; Kirchhoff, Wagner, Maril, & Stern, 2000; Otten, Henson, & Rugg, 2002a, 2002b; Squire & Zola-Morgan, 1991). The process of learning relates to altered neural activity that causes changes in the strength of synaptic connections within these brain areas as a result of experiences (S. J. Martin & Morris, 2002).

The capacity for memory and learning increases with development, with most notable changes evident during the childhood period (Schneider & Pressley, 1997). Early studies have suggested that the accelerated development of these processes during childhood is attributable to schooling related experiences (Sharp, Cole, & Lave, 1979). It has been suggested that schooling strongly influences the development of memory strategies in children, as they are required to remember aspects of the syllabus being taught (Schneider, Knopf, & Stefanek, 2002). Studies which provide participants with mnemonic strategies as a form of memory training have shown that children benefit most from this type of training and show higher level of performance than adults at follow up (Brehmer, Li, Muller, von Oertzen, & Lindenberger, 2007). It has also been suggested that children more readily add these strategies to their repertoire of cognitive skills, showing enhanced memory performance that persists over time (Brehmer et al., 2008).

Performances of young children on tasks of verbal and non verbal memory have been shown to increase as a function of age, prior to the commencement of formal schooling (Simcock & Hayne, 2003). These authors also found that children with superior language skills performed better on tasks of non verbal memory than those with poor language skills. It has been stated that phonological memory skills are closely associated with language acquisition (Gathercole, 1999). The evidence for this association has also been presented in terms of the speech processing impairments apparent in children with specific language disorder (Joanisse & Seidenberg, 1998). These impairments have been related to deficits in the phonological short term memory store which result in the poor level of language learning in these children (Baddeley, Gathercole, & Papagno, 1998).

Incremental changes in learning and memory capacity have also been demonstrated in school children aged between five and sixteen years for both verbal and visuospatial information (Anderson & Lajoie, 1996; Kramer, Delis, Kaplan, O'Donnell, & Prifitera, 1997). When compared to older children and adolescents, five to six year olds showed lower recall, a flatter learning curve and poorer recognition (Kramer et al., 1997). The older adolescents (15-16 yrs) in this study showed the best performance on recall and recognition tasks, they also demonstrated highest levels of memory strategy use. This suggests that development of strategy use coincides with increases in performance on tasks of learning and memory.

Kramer et al (1997) also indicated that there were gender differences in the development of learning and memory. Girls consistently outperformed boys on recall, recognition and delayed recall tasks. Girls also utilised a higher level of memory strategies than boys. The discrepancies between male and female performance were greatest in the 13 years and older age group. Possible explanations for these gender differences include; sex differences in brain morphology (Giedd et al., 1997; Gilmore et al., 2007) and the effects of gonadal hormones on cognitive function (Sisk & Foster, 2004). Gender differences on memory and learning performance as measured by the Rey Auditory Verbal Learning Test (RAVLT) and the Block Span task were found in the Anderson and Lajoie (1996) normative study, where girls performed better than boys. However, it was indicated that these differences were minimal, and

that it was unnecessary for normative data to be divided by gender for child populations.

### *1.3.3 Working memory*

Working memory function involves the cognitive skill of holding and manipulating information within short term memory simultaneously. Working memory capacity underlies higher order processes such as reasoning and complex problem solving (Baddeley, 1986). The original model of working memory was theorised by Baddeley and Hitch (1974) composed of the central executive which is the attentional controlling system and the two slave systems used for temporary storage of specific forms of information. The phonological loop provides a temporary space for rehearsal and storage of verbal information, whilst the visuospatial sketchpad as its name would suggest is the unit for manipulation and storage of visual and spatial information (Baddeley, 1992). More recently, a fourth component to the original model was added, termed the episodic buffer, which has been described as a limited capacity storage system that integrates a variety of sources of information (Baddeley, 2000).

At the structural level, the working memory system is thought to be largely mediated by structures in the frontal lobes of the brain (Owen et al., 1999; R. S. Scheibel & Levin, 2004; E. E. Smith & Jonides, 1999). Evidence of activation in the parietal regions of the brain during performance on tasks of working memory has also been reported (Collete & van der Linden, 2002; E. E. Smith & Jonides, 1998). Aspects of working memory skills also appear to operate according to the lateralisation of the brain, with visual-spatial working memory tasks activating the right side and verbal working memory tasks activating the left side of the brain (E. E. Smith & Jonides, 1997). The prefrontal cortex has been identified as a major centre for working memory function, where its role is to actively hold information for short periods of time which is required for subsequent motor and cognitive activities (Curtis & D'Esposito, 2003; Curtis, Zald, & Pardo, 2000; Owen et al., 1999). Possibly playing the role of the central executive component of the working memory model (D'Esposito et al., 1995).

Brain imaging studies have shown that increases in working memory function coincide with increases in brain activity in the frontal and parietal cortices of children and adolescents aged between 9 and 18 (Klingberg, Forssberg, & Westerberg, 2002). Kwon, Reiss and Menon (2002) reported similar results in a sample of 7-22 year olds, suggesting that underlying processes of working memory develop during childhood, adolescence and into young adulthood. Luciana and Nelson (2002) showed that working memory was not functionally mature by the age of 12 years, suggesting that development continued into the adolescent period. The activity of the dorsolateral prefrontal cortex (DLPFC) has been associated with age related changes in working memory (Klingberg et al., 2002; Kwon et al., 2002). Crone, Wendelken, Donohue, van Leijenhorst, and Bunge (2006) conducted an fMRI study looking at working memory related brain activation in 8-25 year olds. They suggested that both adults and adolescents demonstrated activation of the DLPFC during the manipulation component of the working memory task, whilst the children within the sample showed no activation in this region. A recent study showed similar results, suggesting that the DLPFC reaches adult level maturity during adolescence (Brahmbhatt, McAuley, & Barch, 2008), however others have suggested that the level of activation in the DLPFC may continue to increase into adulthood (Scherf, Sweeney, & Luna, 2006).

Differential development of subcomponent processes of working memory has been demonstrated in a sample of adolescents and young adults. Luciana, Conklin, Hooper and Yarger (2005) found simple working memory function was established between ages 11-12. Whilst complex working memory skills requiring more executive control showed incremental development up to the age of 16 and remained stable in the 18-20 year age groups. Other studies have suggested that basic level working memory was present at four years of age, at six years, the three-component model outlined by Baddeley and Hitch (1974) was demonstrated (Gathercole, Pickering, Ambridge, & Wearing, 2004). From six years onwards, working memory function continued to increase at a considerable rate throughout the childhood period and into mid adolescence. Working memory function and capacity has been shown to improve after the age of 12, and similar to previous studies, basic working memory skills peaked much earlier than complex skills which matured between the ages of 16 and 17 (Conklin, Luciana, Hooper, & Yarger, 2007), whilst some have suggested that

working memory doesn't reach adult levels until 19 years of age (Luna, Garver, Urban, Lazar, & Sweeney, 2004).

It has been shown that younger children are more efficient at using visual working memory as opposed to verbal working memory (Hitch, Halliday, Schaafstal, & Schraagen, 1988). As children get older, they develop the ability to convert visuospatial information into a phonological form, providing a greater space for maintaining information and thus enhancing working memory performance (Pickering, 2001). The executive control component of working memory becomes most prominent in the adolescent period, coinciding with performance that is closest to adult levels (Luciana et al., 2005).

Working memory function has been associated with performances on other cognitive tasks and measures of scholastic aptitude (Cowan et al., 2005). Children with poor performances on national curriculum assessments also demonstrated limited working memory skills (Gathercole & Pickering, 2000), indicating that these skills are necessary for performances on tasks of reading, comprehension and mathematics (Bull, Johnson, & Roy, 1999; Hansen & Bowey, 1994; McLean & Hitch, 1999; Swanson, 1994). Enhanced working memory function in children has been associated with higher scores on measures of general intelligence, mathematics, reading, spelling, general knowledge and receptive vocabulary (Swanson, 1996). These findings indicate that deficits in working memory can influence an individual's functioning over a number of different areas.

#### *1.3.4 Executive functioning and attention*

Executive functioning refers to the range of higher order cognitive skills associated with self-regulatory and goal directed behaviours. Skills such as planning, organisation, perceptual set shifting, information updating, concept formation, perceptual reasoning and inhibitory control are just some abilities that fall under the umbrella of executive functioning (Baron, 2004; Lezak, Howieson, & Loring, 2004; Miyake et al., 2000; Strauss, Sherman, & Spreen, 2006; Zillmer et al., 2008). Some have suggested that there is a strong relationship between executive function capacity and general intelligence (J. Duncan, Burgess, & Emslie, 1995; J. Duncan, Emslie, Williams, Johnson, & Freer, 1996). Recent research of monozygotic twin pairs



reported that executive function capacity was largely influenced by genetic predisposition, possibly even more so than general intelligence (Friedman et al., 2008)

The executive functions are thought to originate in the frontal lobes (Stuss et al., 2002), however there is evidence to suggest that associations between the frontal lobes and posterior and subcortical brain regions also mediate executive functioning (Collete & van der Linden, 2002; R. Elliot, 2003). Studies which have examined patients with frontal lobe lesions, consistently report impairments in performances on tasks of executive function and their associated behavioural manifestations (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Draper & Ponsford, 2008; Lehtonen et al., 2005; Novack, Bush, Meythaler, & Canupp, 2001; Owen, Downes, Sahakian, Polkey, & Robbins, 1990; Tate, 1999). In child and adolescent populations, frontal lobe impairment and associated executive dysfunction is characteristic of developmental disorders such as Attention Deficit Hyperactivity Disorder (ADHD) (Amen & Carmichael, 1997; Barkley, Grodzinsky, & DuPaul, 1992; Castellanos et al., 2002) and Autism (Carper & Courchesne, 2000; Just, Cherkassky, Keller, Kana, & Minshew, 2007; Zilbovicius et al., 1995).

In normal populations of children and adolescents, evidence of executive functioning has been identified in the early stage of infancy (Diamond, 1985; Haith, Hazan, & Goodman, 1988). Development has been shown to continue through childhood (Isquith, Gioia, & Espy, 2004), however the most prominent period for the development of executive functioning is the adolescent stage of the lifespan (Kray, Eber, & Lindenberger, 2004; Luna & Sweeney, 2001). Klenberg, Korkman, and Lahti-Nuuttila (2001) observed the development of executive functioning in a sample of three to twelve year old Finnish children. The preschool children demonstrated basic level attention and inhibitory control of motor movements at three years of age. Maturity of these basic level functions was not reached until age six. At seven years of age, the executive skill of set shifting was established. Planning and focussed attention were shown to reach maturity between 8 and 10 years of age. The last skill to develop was verbal fluency, although full maturity of fluency was not evident in the sample, suggesting that it continues to develop beyond 12 years of age. Davidson, Amso, Anderson, and Diamond (2006) demonstrated similar findings suggesting that children of four years of age could complete tasks requiring basic level inhibitory

control and working memory. In contrast to Klenberg et al (2001), they suggested that perceptual set shifting demonstrated a longer period of development, not reaching adult levels at 13 years of age. In another study, children between the average ages of 7.6 years and 9.5 years showed considerable development of inhibitory control during this period, further improvement in these skills reportedly continued up to 11 years of age (Brocki & Bohlin, 2004). It was indicated that performance on tasks of inhibitory control appears to stabilise after this point, suggesting that these skills mature around 11 years. Others have shown that inhibitory functions reach adult levels at 15 years of age (Luna et al., 2004). Significant increases in verbal fluency were shown at the age of eight, with further increases appearing at the age of 12 years. Brocki and Bohlin (2004) suggested that the first peak in performance may be associated with children's ability to code information in its phonological form after the age of eight years. The later spurt in fluency functioning coincides with the findings of Davidson et al (2006) suggesting that fluency tasks demonstrate protracted development.

Development of executive functioning in late childhood and adolescence was examined by a group of Australian researchers (Anderson et al., 2001). They suggested that development of these functions slows during late childhood and adolescence, with only attentional control showing the most significant improvement at 15 years of age. Goal setting skills were shown to peak at the age of 12 years, whilst stable levels were apparent after this age. No significant improvements in cognitive flexibility were found, suggesting that these skills mature earlier in the childhood period.

Executive functions are utilised in a range of different cognitive and behavioural operations. In educational settings, it has been found that executive functions are significantly related to performances on tasks of English (Waber, Gerber, Turcios, Wagner, & Forbes, 2006) literacy (Blair & Razza, 2007), language learning (B. D. Singer & Bashir, 1999) and mathematics (Andrews-Espy et al., 2004; Blair & Razza, 2007; Bull & Scerif, 2001; Waber et al., 2006). Others have suggested that impaired executive functioning is related to poorer academic outcomes overall (Biederman et al., 2004). Executive function has also been associated with aspects of socio-emotional functioning. It has been suggested that poor inhibitory control is related to higher levels of aggression in children (Oosterlaan & Sergeant, 1996;

Raaijmakers et al., 2008). Poor performances on tasks of executive function have also been linked to a range of antisocial behaviours in children and adolescents (Kelly, Richardson, Hunter, & Knapp, 2002; Morgan & Lilienfeld, 2000).

### *1.3.5 Processing speed*

Processing speed is the measurement of how fast an individual is able to process information and complete tasks. It has been suggested that examining processing speed during the childhood period is particularly important as it is associated with structural changes in the brain and a wide range of cognitive abilities (Wechsler, 2003). Speed of processing plays a major role in intellectual function, due to its influence on a wide range of cognitive tasks, it is thought to be largely associated with general intelligence (DiLalla, 2000; Fry & Hale, 1996; Kail, 2000; Wechsler, 2003). Processing speed has also been associated with performances on tasks of working memory (Fry & Hale, 1996), arithmetic (Bull & Johnston, 1997) and reading (Kail & Hall, 1994).

Developmentally, processing speed has been shown to increase with age, stabilising during adulthood and then showing gradual decline in late adulthood when slowing of information processing occurs (Kail, 1991a, 1991b; Salthouse, 1996). Children perform much slower on tasks of processing speed than adults, one study compared performances between, four, five, and six year old children (Miller & Vernon, 1997). As expected, the four year olds demonstrated the slowest performance; gradual increases in speed were indicated in the subsequent age groups. Miller and Vernon also compared the children's performances to a group of young adults, significant decreases in the time to complete tasks was demonstrated in the adult group. Kail (1992) suggested that in comparison to adults, four-five year olds performed three times more slowly, whilst eight to 11 year olds performed twice as slowly. Brocki and Bohlin (2004) indicated that the greatest changes in development of processing speed occurred in the six year old- nine and a half year old age range. Similarly, large developmental gains in processing speed were shown in seven- nine year olds (Anderson et al., 2001). Anderson et al (2001) found relatively stable performances on tasks of processing speed between the ages of 11 and 15. These findings are in line with those of Luna et al (2004) suggesting that that processing speed reaches adult levels at approximately 15 years of age.

Sex differences have also been reported in relation to performances on tasks of processing speed. In children aged six-thirteen years, it was found that girls performed at a slower rate than boys (Brocki & Bohlin, 2004). Anderson et al (2001) reported similar findings showing poorer performances by girls in the earlier age groups, however when observing performances in the adolescent age groups, the reverse became apparent, with girls performing significantly faster than the boys.

Brain imaging studies have shown that white matter volume is significantly associated with processing speed (Posthuma et al., 2003). This suggests that increases in processing speed may coincide with greater nerve conduction in response to processes of myelination. Reduced processing speed has been found in elderly patients with cerebral white matter lesions, those with severe periventricular white matter lesions performed close to one standard deviation below the mean (de Groot et al., 2000). Children and adolescents with corpus callosal damage due to traumatic brain injury have also shown significant deficits in processing speed (Verger et al., 2001). Together, these findings provide further evidence that myelinated nerve pathways are associated with processing speed capacity. Some have suggested that deficits in processing speed are associated with impaired nerve pathways of the hypothalamus that connect with the amygdale and hippocampal formation (Frattali et al., 2001).

#### *1.3.6 Visuo-perceptual function*

Visuo-perceptual function refers to the ability to integrate visual and spatial information with fine motor movements. Classic measures of these skills require individuals to draw specified sequences of geometrical forms (Beery & Beery, 2004). Early research has suggested that visuospatial function was largely associated with the functioning of the right hemisphere and the motor area that lies on the same side of the non-dominant hand (Hartlage & Lucas, 1976; Mateer, 1983). Subsequent studies of patients with primarily left hemisphere damage have also demonstrated deficits in visuospatial skills, although the nature of these deficits were different from right hemisphere patients (Delis, Robertson, & Efron, 1986). Even though both hemispheres have been implicated in visuospatial function, it appears that the right hemisphere plays a greater role, as individuals with right hemisphere damage demonstrate more difficulties when completing drawing tasks (Damasio, 1985).

Children and adolescents with injuries that affect the interconnection between the two hemispheres, namely the corpus callosum have also demonstrated deficits in visuospatial function (Verger et al., 2001). Thus, pathways connecting the two hemispheres may also be responsible for visuospatial deficits. Animal studies (Mishkin, Ungerleider, & Macko, 1983; Ungerleider & Haxby, 1994) have suggested that there are two major pathways that mediate visual and spatial functioning, the occipitotemporal pathway for visual identification of objects and the occipitoparietal pathway responsible for integrating spatial relations between objects. Humans with visual and spatial deficits have demonstrated lesions in these pathways on post mortem analysis (Newcombe, Ratcliff, & Damasio, 1987). Visuo-spatial impairment has also been found in adults with cerebellar lesions (Schmahmann & Sherman, 1998; Wallesch & Horn, 1990). However, decreased cerebellar involvement was indicated in normally developing children performing motor tasks when compared to adults (Muller et al., 1998). Increased cerebral blood flow in the parietal regions (superior parietal cortex, intraparietal sulcus and inferior parietal lobe) was shown using positron emission tomography (PET) in human subjects who were making mentally planned movements of the body in external space (Bonda, Petrides, Frey, & Evans, 1995). Taken together, these findings indicate that visuospatial function is difficult to localise and is likely to be a consequence of communication between a number of different brain regions. Quintana and Fuster (1993) suggested that the visual, sensory and motor information, required of visuospatial function, is likely to be mediated by parietal and motor areas of the frontal lobes, whilst subcortical connections integrate this information (G. E. Alexander, DeLong, & Strick, 1986). Studies have found relationships between visuo-motor tasks and other cognitive abilities, including processing speed and executive function (Diamond, 2000; Roebers & Kauer, 2009). These findings suggest that the range of cognitive skills required to complete visuospatial tasks may activate various brain regions associated with different cognitive components required of the tasks. Structural changes of pathways mediating motor functions appear to develop through late childhood and adolescence (Paus et al., 1999), suggesting that physical changes in these skills will continue throughout these periods.

The limited research examining the development of visuospatial function has suggested that children are able to analyse spatial forms by the age of three (Tada & Stiles, 1996). These authors indicated that children change the way they manipulate and organise geometric forms with increasing age in a sample of three to five year olds. Similar findings were reported by Stiles and Stern (2001) suggesting that children between 24 and 48 months of age showed significant increases in their abilities to reproduce progressively more complex spatial forms with age. Giudice et al (2000) showed that simple visual scanning abilities were acquired at three years of age whilst more complex visual perceptual skills continued to develop through to ages eight and nine. Others have shown further improvements in visuospatial skills up to the age of 12 years (van Mier, 2006), although it has been suggested that visuospatial skills continue to develop through the adolescent period (Diamond, 2000; Rueckriegel et al., 2008).

### *1.3.7 Language*

Language goes through marked development during the childhood period. The rapid progression of language development during childhood is indicated by number of changes that occur in the organisation of words within a very short period of time. Early evidence of language development is shown in infant babbling by six months, followed on by the use of single words at approximately one year, formation of short sentences by age two, and complex organisation of language by about five years of age (Craik & Bialystok, 2008; Kuhl, 2004; K. Nelson, 1973). Research has indicated that there are sensitive periods for the development of language.

Hypothesised critical periods of language development have stemmed from studies that have examined children who have been deprived of linguistic stimulation during early childhood. A published report of an extreme case of deprivation is that of Genie, a 13 year old girl who had come to the attention of child welfare services after being isolated in a locked dark room since she was an infant by her parents (Fromkin, Krashen, Curtiss, Rigler, & Rigler, 1984). Genie was never spoken to, and was punished by her parents if she tried to communicate with them. Genie showed very little ability in spoken language after coming to attention of the authorities, however, with time her receptive vocabulary improved slightly, although her expressive speech did not develop beyond three word utterances. Her severe language deficits were

amongst a number of cognitive developmental impairments sustained as a result of her abuse history. As a result, it is difficult to ascertain whether her lack of language development was a result of inadequate stimulation during the 'sensitive period' or the effects of severe abuse on her cognitive development.

Other research examining the acquisition of a second language in non-English speaking immigrants lends greater support to the idea for a critical period in language development (due to the absence of a greater number of confounding factors). These studies consistently found that those that began learning English as a second language earlier on, within the childhood period, were more proficient in their English skills than those who learned the language after the adolescent period (Collier, 1987; J. Johnson & Newport, 1989; Oyama, 1976).

Language development occurs in the social context, thus various forms of verbal interaction between adults and children foster the development of complex language processes (Kuhl, 2004). It has been shown that infants show greater activity in left hemisphere activity when listening to speech (Molfese & Betz, 1988). The left side of the brain has been generally identified as the 'language centre', for the majority of right and left handed individuals (Pinel & Dehaene, 2010; Szaflarski et al., 2002; Szaflarski et al., 2006; Veroude, Norris, Shumskaya, Gullberg, & Indefrey, 2010). Post mortem studies have revealed that language development coincides with dendritic growth and synaptogenesis in the brain regions associated with language function (Huttenlocher & Dabholkar, 1997; A. B. Scheibel, 1990). Differential timing and location of dendritic growth was associated with the emergence of different aspects of language and verbal functioning. At nine months, right hemisphere areas related to the facial muscle movements of speech showed marked dendritic growth, and exceeded the development occurring in the left hemisphere (A. B. Scheibel, 1990). By age 15 months rapid increases in dendritic growth of the left Broca's area occurred, consistent with the onset of spoken language. Continued neural development in both left and right language centres occurred until six years of age indicative of lateralisation and adult level development of language structures (A. B. Scheibel, 1990). The auditory cortex undergoes synapse elimination until the age of 12, suggesting that refinement of verbal skills take place during this period (Huttenlocher & Dabholkar, 1997). Neurophysiological data demonstrates similar

results, suggesting that children show continual development of language areas up until 13 years of age, and possibly beyond this period (Hahne, Eckstein, & Friederici, 2004). Functional MRI studies have also demonstrated that the structures supporting language development in the right and left hemispheres in children become activated during the completion of language tasks, and increased activation in higher order areas of language function were demonstrated in older children (Szaflarski et al., 2006; Wood et al., 2004).

Children in the early periods of language development, show sharp increases in vocabulary acquisition with age. Fenson, Dale, Reznick, Bates, Thal, and Pethick (1994) estimated that children aged at 16 months had a vocabulary of 55 words, by 23 months this increased to 225 words and at 30 months the average number of words acquired was 569 words. Toddlers aged between 20 and 31 months demonstrated that they were able to acquire new words very quickly under laboratory conditions (Vincent-Smith, Bricker, & Bricker, 1974).

Anglin (1993) found that comprehension of words increased rapidly between the ages of six and ten. It was suggested that knowledge of complex words underwent a similar pattern of growth with increasing age. Anglin also reported estimated frequencies of words acquired by three age groups, the youngest children aged six had an average vocabulary of 10,398 words, whilst the oldest children aged ten had an average of 39,994 words. Some have argued that the ability to acquire new words was largely associated with the functioning of the phonological component of memory (Baddeley, Papagno, & Vallar, 1988; Bowey, 1996, 2001; Gathercole & Baddeley, 1990). This indicates that vocabulary acquisition may depend on the functioning of associated cognitive abilities.

Expression of language appears to occur at approximately 12 months of age, although some children show the ability to express a small number of words as early as eight months (Bates, Dale, & Thal, 1995). Between one and two, children show significant increases in comprehension of information and expressive language (Fernald, Perfors, & Marchman, 2006). Increases in comprehension are shown during the primary school years, with older children outperforming younger children on a comprehension task in a sample of 6-10 year olds (Best, Dockrell, & Braisby, 2006).



Age related increases in comprehension performance also continue throughout adolescence and young adulthood, suggesting that language experiences a protracted length of development (Duthie, Nippold, Billow, & Mansfield, 2008; Nippold & Haq, 1996; Nippold, Hegel, Uhden, & Bustamante, 1998; Nippold, Uhden, & Schwartz, 1997)

A number of other factors have been associated with the rate of language development in early childhood, some of these include: gender, with females outperforming males (K. Nelson, 1973), birth mothers employment status and level of education, with children of employed mothers developing language more rapidly (Dollaghan et al., 1999), time spent with other adults, limited or no time watching TV, and time spent outside the home (K. Nelson, 1973), maternal involvement and availability of play materials (Elardo, Bradley, & Caldwell, 1977; Murray, Kempton, Woolgar, & Hooper, 1993). Although a large number of environmental factors have been shown to affect the development of language, studies have also suggested that language functioning is also influenced by a strong genetic component (Hayiou-Thomas, 2008; Hoekstra, Bartels, & Boomsma, 2007).

### *1.3.8 Attachment and emotion*

It is well understood that emotional development relies heavily on the quality of early attachment relationships between caregiver and child. The behavioural manifestations of mother-infant attachment were first described by the work of Bowlby (1969) and Ainsworth (1969), and have been more recently extended with the understanding of the neurobiological processes that underlie this important relationship. According to Schore (1994; 2001c; 2005), the mother's emotional attunement with the child is the primary determinant of the child's capacity to self regulate emotion, cognitive function and behaviour. High concentrations of endorphins are released during these early mother-infant interactions, in response to activation of brain stem dopaminergic fibres (Schore, 1996). These endorphins are the biochemical markers of attachment, responsible for the pleasurable experiences related to social interaction (Bridges & Grimm, 1982; Hart, 2008; Herman & Panskepp, 1978; Kalin, Shelton, & Lynn, 1995).

It has been suggested that the maturation of the child's right hemisphere is dependent on the attachment experience with the mother (Schorer, 2000b). The right orbitofrontal cortex is one major cortical component of the limbic system. It has been identified as the region most strongly influenced by the attachment relationship (Schorer, 2001b, 2001c; Seigal, 1999). The orbitofrontal cortex is responsible for processing inter-relational signals that form the basis of social experiences (Schorer, 2001b). It is part of a large network that controls empathic and emotional relatedness, self awareness, personal identity and episodic memory (Schorer, 1994). This region also has a self-regulatory capacity, recognising and making sense of incoming emotional information (Balbernie, 2001). The autonomic nervous system is mediated by regions of the orbitofrontal cortex, thus inducing arousal in the presence of stress (Schorer, 1994). It is clear, from this evidence that the orbitofrontal cortex has a range of important functions. Given that its development is largely dependent on the early attachment interaction, it is foreseeable that there would be wide ranging consequences to the individual's emotional functioning in the absence of a quality attachment interaction during infancy (Schorer, 2005).

The attachment relationship during infancy serves as an internal working model that will form the basis of future social interactions and emotional regulation (Schorer, 2000a). A large proportion of children who have been deprived of a quality attachment relationship in infancy and early childhood due to child abuse and neglect demonstrate disorganised/disoriented insecure attachments (Carlson, Cicchetti, Barnett, & Braunwald, 1989; Schorer, 2001a). This form of attachment is associated with poor regulation of stressful stimuli, particularly that imposed by social interaction with others (Perry, 2001; van der Kolk & Fisler, 1994). It has been shown that adolescents with insecure attachment relationships show greater levels of stress in comparison to securely attached adolescents during conflictual interactions with their main attachment figure (Beijersbergen, Bakermans-Kranenburg, van IJzendoorn, & Juffer, 2008). These difficulties in coping with relational stress lead to the manifestation of dissociation and hyperarousal, which are characteristic of post traumatic stress disorder (Schorer, 2001a). Other consequences of disorganised/disoriented attachments include aggression, self destructive behaviour, eating disorders, substance abuse (van der Kolk & Fisler, 1994) impaired sense of self

and interpersonal difficulties (Briere & Elliot, 1994; Morton & Browne, 1998). The range of behaviours shown by these individuals lends support to the notion that those children who do not form a secure attachment to their caregivers during early life have not established the processes associated with right brain maturation resulting in deficient socio-emotional functioning.

#### *1.4 Gender differences in cognitive function*

Patterns of cognitive development appear to differ in boys and girls. Using MRI, it has been shown in a sample of 5-17 year olds, that boys have total cerebral volume that is approximately 10 percent larger in comparison to girls (Reiss et al., 1996). Furthermore, the difference in size was accounted for by a larger volume of grey matter in boys. Another study looking at a similar age range of children showed that male total cerebral volume was 12 percent larger than females (De Bellis, Keshavan et al., 2001). In this study, age by gender interactions indicated that males had more age related increases in grey matter reduction and white matter volume increases in comparison to females. Gender differences in brain anatomy have also been shown in adults, where males had greater total cerebral and white matter volumes than females, whilst females showed larger grey matter volumes (Gur et al., 1999). These gender differences in brain anatomy have been associated with differences in cognitive function between males and females. In adults, it has been shown that women are verbally superior, whilst males perform better on spatial tasks (Gur et al., 2000; Gur et al., 1999; Weiss, Kemmler, Deisenhammer, Fleischhacker, & Delazer, 2003). In contrast, Hyde and Linn (1988) conducted a meta-analysis of 165 studies of both children and adults that reported gender differences on verbal ability and found the differences were minimal and not significant. While the meta-analysis conducted by Voyer, Voyer, and Bryden (1995) showed that gender differences in spatial abilities, where males outperform females, were significant and of superior magnitude. Females have also shown better performance on tasks of comprehension (Hedges & Nowell, 1995), fine motor skills (J. A. Y. Hall & Kimura, 1995) and processing speed (Born, Bleichrodt, & van der Flier, 1987). Whilst males showed greater ability in tasks of visual working memory (Halpern & Wright, 1996) and fluid reasoning (Meehan, 1984).

### *1.5 Child abuse and neglect*

According to the World Health Organisation (2007) child abuse “refers to the physical and emotional mistreatment, sexual abuse, neglect and negligent treatment of children, as well as to their commercial or other exploitation” (p.7). Child abuse “relies on evidence of either ill-treatment of the child that has caused or is likely to cause significant harm to the child...” (cited in Glaser, 2000, p.98). A general definition of child abuse used within the state of Victoria, Australia is as follows, “Child abuse is an act or omission by an adult that endangers or impairs a child’s physical or emotional health and development. Child abuse is not usually a single incident, but takes place over time” (Responding to Child Abuse Victorian Government Publishing Service, 2002, p.5) According to Victorian legislation a person under the age of seventeen is classified as a child or young person (“Children, Youth and Families Act,” 2005).

#### *1.5.1 Types of abuse*

*Physical Abuse* can be defined as the “actual or likely physical injury to a child or failure to prevent physical injury (or suffering) to a child, including deliberate poisoning, suffocation, and Munchausen’s syndrome by proxy” (Veltman & Browne, 2001, p. 217). “Physical abuse refers to a situation in which a child suffers or is likely to suffer significant harm from an injury inflicted by a child’s parent or caregiver. The injury may be inflicted intentionally or may be the inadvertent consequence of physical punishment or physically aggressive treatment of a child” (Responding to Child Abuse Victorian Government Publishing Service, 2002, p.6).

*Sexual Abuse* “is the actual or likely exploitation of a child or adolescent” (Veltman & Browne, 2001, pp., p. 217) constituted by situations “in which a person uses power or authority over a child to involve the child in sexual activity and the child's parent or caregiver has not protected the child. It includes fondling of the child's genitals, masturbation, oral sex, vaginal or anal penetration by a penis, finger or other object, or exposure of the child to pornography” (Responding to Child Abuse Victorian Government Publishing Service, 2002, p.12).

*Emotional Abuse* is the “actual or likely severe adverse effect on the emotional and behavioural development of a child caused by persistent or severe emotional abuse or rejection (Veltman & Browne, 2001, p. 217). The definition in the state of Victoria characterises emotional abuse as “a situation in which a child’s parent or caregiver repeatedly rejects the child or uses threats to frighten the child. This may involve name calling, put-downs or continual coldness from the parent or care giver, to the extent that it significantly damages the child’s physical, social, intellectual or emotional development” (Responding to Child Abuse Victorian Government Publishing Service, 2002, p.12). As it has been extensively noted, emotional abuse is usually present in all forms of abuse, children are only classified as emotionally abused if it is the sole documented abuse type (Glaser, 2000; Veltman & Browne, 2001).

*Neglect* has been defined as “failure to provide the proper or necessary support, education required by law, medical, surgical or any other care necessary for the child’s well being” (Culp et al., 1991, p.380). It occurs when “...a child’s parent or caregiver fails to provide a child with the basic necessities of life, such as food, clothing, medical attention, or supervision, to the extent that the child’s health and development is, or is likely to be, significantly harmed” (Responding to Child Abuse Victorian Government Publishing Service, 2002). Some authors have also categorised emotional abuse as part of neglect, arguing that lack of appropriate parental interaction which is characteristic of emotional abuse can also be interpreted as a form of neglect (Dubowitz, Papas, Black, & Starr, 2002).

Much of the research literature within this area to date has categorised children on the types of maltreatment experienced described above, however this poses significant problems. Difficulties arise when attempting to classify these children according to maltreatment type as many experience more than one type of abuse. For example, a child experiencing physical abuse may also be exposed to simultaneous violent verbal attacks characteristic of emotional abuse whilst residing in an impoverished environment pertaining to neglect. A social welfare perspective would

classify such a child as physically abused, however when considering the actual experience of the child, a definitive maltreatment type is often not reliably ascertained and may be best described as mixed. Milburn, Lynch and Jackson (2008) indicated that the most current child abuse statistics according to the Department of Human Services, Victoria are underestimates of the prevalence of abuse, the main reason being that workers frequently arbitrarily decide on a single abuse type regardless of other abuse being experienced by the child. According to a report conducted by Frederico, Jackson and Black (2005) it was observed that a single specific abuse type was unlikely to occur. Most children and adolescents experienced more than one abuse type and it was suggested that emotional abuse commonly co-occurred with each category of abuse. It has also been found that those who experience sexual abuse up to three times more likely to experience other abuse types (Dong, Anda, Dube, Giles, & Felitti, 2003).

### *1.6 Take Two Berry Street Program*

Berry Street is the largest independent child and family welfare organisation in the state of Victoria. The organisation plays a major role in providing various services to at-risk children and their families. Services provided are focussed on fostering the wellbeing of youth and their families at various levels including, housing, education, employment, foster care, outreach, abuse prevention programs and family counselling.

The Take Two program is a state wide intensive child and adolescent mental health service funded by the Department of Human Services, Victoria. The program is provided by a partnership between Berry Street Victoria, Austin Child and Adolescent Mental Health Services and Latrobe University School of Social work and Social Policy and Mindful- Centre for Training and Research in Developmental Health. Take Two is primarily involved in the treatment of clients of the Department of Human Services child protection branch. Take Two was developed in response to the Stargate Early Intervention Programme for Children which was a mental health service targeted at treating children and adolescents who had been placed in out-of home care for the first time due to substantiations of child maltreatment (Milburn et al., 2008). The clinicians involved in this program identified the high needs of these young

persons and the necessary involvement of a systems perspective in treatment; including the young person/s, biological parents, legal carers and guardians and child protection professionals.

Take Two's role within Secure Welfare is to provide therapeutic assessment, crisis intervention, brief interventions and recommendations to families and DHS case managers working with each individual child that is referred. Take Two's evaluation of their role within Secure Welfare has shown that high number of young persons utilizing the service demonstrate a number of different of maltreatment types within their histories with sexual abuse being most prevalent among cases (Frederico et al., 2005). Young persons also exhibit particularly challenging behaviours and various mental health issues associated with trauma during childhood (Frederico et al., 2005).

### *1.7 The Secure Welfare Service*

Most young people involved with child protection due to maltreatment history live in various forms of accommodation within the community. Some stay with their families under specific provisions made by child protection, some reside in foster care placements, whilst others are housed in shared residential units under the supervision of social and youth workers. A small number of these young people may be placed in secure accommodation due to significant risk of being harmed, harming themselves or harming others (Falshaw & Browne, 1997). In the United Kingdom, young persons presenting to these facilities showed high rates of aggression, substance misuse, self harm, and, social, family and educational problems (Kroll et al., 2002). The aim of secure accommodation is to provide the young person with a place of safety and containment, as well as access to therapeutic services to deal with trauma related issues.

The Department of Human Services (DHS) Child Protection Secure Welfare Service is a temporary accommodation unit that is comprised of two services (each comprising a 10 bed capacity), one for young males and the other for young females aged 10-17. It is a locked facility that is set up with highly structured routines to help contain young persons following a significant crisis for a maximum period of 21 days, in exceptional situations this period may be extended for a further 21 days ("Children,

Youth and Families Act," 2005; Department of Human Services, 2007). To be eligible to access the service, young persons need to be current DHS Child Protection clients "...who are at immediate and substantial risk of harm and for whom no alternative safe options exist, and subject to a Child Protection Order or Interim Order" (Department of Human Services, 2007, p. 2). A long history of maltreatment is highly prevalent among the young persons who utilize Secure Welfare (Frederico et al., 2005). Admission to the service is likely to be initiated by a significant crisis within the young person's life putting themselves at risk of serious harm such as further maltreatment, domestic violence, drug abuse, suicidality, criminal offending, absconding from placement, challenging behaviour, engagement with known paedophiles and prostitution (Department of Human Services, 2009). The Secure Welfare Service provides young persons with access to a medical practitioner, drug and alcohol nurse and mental health professionals. It also has an on site school managed by the Victorian Department of Education. The service aims to form a management plan to reintegrate the young person into the community in liaison with protective workers, case managers and mental health professionals. Where appropriate, referrals to external agencies may also take place for purposes of child engagement with therapeutic services.

According to Johnson (2006) within the 2005-2006 period, a total of 500 admissions were made to Secure Welfare, with females outnumbering males at almost a two to one ratio. Young women account for 66 percent of all admissions and 60 percent of number of days in placement. The average length of stay within the service for both males and females was eight and a half days. The statistics indicate a trend demonstrating that admissions to Secure Welfare are increasing incrementally, and for young women in particular, the rise in admissions is increasing more rapidly.

### *1.8 Custodial Treatment Strategies for Child Protection Clients*

The Department of Human Services Secure Welfare Service Victoria is the only service of its type in Australia. Literature on other similar services at an international level is quite sparse and that which is available is usually focussed on adolescents put in secure accommodation as part of their involvement with the legal



justice system following a crime. O'Neill (2001) conducted a study on similar secure accommodation in England, where there are 31 secure units operating, offering 500 beds to young people. Scotland has six secure units, with a total of 112 places available (The Scottish Government, 2007). The English and Scottish secure welfare services differ from the Victorian service in that young people may be admitted for one of two reasons:

1. As a form of remand in relation to a committed offence via the criminal justice route, or
2. As a form of protection via the welfare route, where a child is at risk of harm (the primary reason for admission in the Victorian service).

The English and Scottish services also have the capacity to provide young persons with accommodation in the unit for one year, and in some circumstances longer (The Scottish Government, 2007). As stated in a previous chapter, the Victorian service is limited to providing accommodation for a brief period of three weeks, and in exceptional circumstances 6 weeks ("Children, Youth and Families Act," 2005).

All the units described here provide young persons with educational facilities within the service to avoid disruption to schooling. In the Victorian service, the curriculum is focussed on meeting the needs of the individuals admitted to the service rather than following the format of mainstream education. Issues of drugs and alcohol, mental health and sexual health are just some of the main topics covered.

Psychotherapeutic and medical care are available in all of these services, however it has been suggested that psychotherapy is limited in the English secure care service where containment and control appear to be more of a focus than management of psychological issues (T. O'Neill, 2001). Whilst in the Scottish system, 14 percent of young persons received mental health treatment, and 39 percent received assistance in maintaining mental health and well being (The Scottish Government, 2007). The United States also have residential treatment programs that are locked facilities, however these are non-federal government funded and rely on limited state government budgets, fundraising and health insurance claims to provide their services (Frederico, Jackson, & Black, 2006).

### *1.9 Characteristics of Children in Protective Care*

Children and adolescents in protective care share common experiences of various types of maltreatment, including physical, emotional, sexual and neglect during the developmentally sensitive period of the childhood years (Veltman & Browne, 2001). The consequences of such maltreatment on cognitive, affective and social functioning and development remain unclear, however the limited research available suggests that these children and adolescents experience long term interpersonal, educational, cognitive and behavioural dysfunction (Carrey, Butter, Persinger, & Bialik, 1995; Holland & Gorey, 2004; Veltman & Browne, 2001).

Maltreated children and adolescents have been shown to display more challenging behaviours in the school environment and are more likely to be truant and miss large periods of school (Nugent, Labram, & McLoughlin, 1998). This may relate to specific factors associated with the abuse experience such as neglect where parents are unavailable to take their children to school or in older children or adolescents it may be a consequence of absconding from the maltreating environment leading to homelessness. Research has also suggested that maltreated children expressed high levels of both reactive and verbal aggression, it has also been found that maltreated children demonstrate lower verbal IQ scores (Connor, Doerfler, Volungis, Steingard, & Melloni, 2003; Connor, Steingard, Anderson, Cunningham, & Melloni, 2004; Scerbo & Kolko, 1995). A study of children from mothers deemed at risk for performing acts of maltreatment before giving birth showed evidence of self-regulation and higher levels of behaviour problems when assessed at five years of age (Schatz, Smith, Borkowski, Whitman, & Keogh, 2008). These factors affect the individual's ability to function in social situations, leading to significant behavioural disturbances and poor social competence (Feldman, Salzinger, Rosario, & Alvarado, 1995). These difficulties tend to carry on through to the adolescent period, Arata, Langhinrichsen-Roling, Bowers and O'Brien (2007) found that adolescents with maltreatment histories were more aggressive and were more likely to engage in substance abuse, delinquent and promiscuous behaviours. It has also suggested that adolescents and young adults with histories of maltreatment were more likely to engage in risky sexual behaviours, relating to more incidences of sexual transmitted diseases and putting them at greater risk of acquiring HIV (Allers, Benjack, White, &

Rousey, 1993; Greenberg et al., 1999). Others have found that adolescents presenting before a court for criminal offending were most likely to have histories of maltreatment (Falshaw & Browne, 1997; Swahn et al., 2006). Abused females in particular, were more likely to engage in delinquent behaviours that later developed into criminal offending (Baldry, 2007; Makarios, 2007).

Concomitant to their abuse, a large proportion of these children also suffer from varying types of psychopathology, namely depression, anxiety (McCloskey, Figueredo, & Koss, 1995; McLeer et al., 1998; Silverman, Reinherz, & Giacona, 1996) and post traumatic stress (Barrett, Green, Morris, Giles, & Croft, 1996; Beers & De Bellis, 2002; Cahill, Kaminer, & Johnson, 1999; De Bellis & Putnam, 1994; Glaser, 2000; McCloskey et al., 1995; Silverman et al., 1996; van der Kolk, 2003). Symptoms consistent with Borderline Personality Disorder, including dependency, suicidality, violence, impulsivity and substance use were also found more commonly amongst adolescents with histories of maltreatment (Grilo, Sanislow, Fehon, Martino, & McGlashan, 1999). Wonderlich et al (2000) suggested that adolescents with histories of maltreatment, particularly females demonstrated more eating disordered behaviours, and, were at higher risk for developing an eating disorder (Corstorphine, Waller, Lawson, & Ganis, 2007; Hodson, Newcomb, Locke, & Goodyear, 2006; Kendler et al., 2000). Maltreatment also puts adolescents at higher risk self injurious behaviours (Husain, 1990), hopelessness, depression and suicide (Arata et al., 2007; Husain, 1990).

Adolescents in secure care commonly share issues of substance abuse. Wall and Kohl (2007) reported that a high proportion of adolescents with histories of maltreatment had substance abuse issues, or were at higher risk of developing substance dependence (Kendler et al., 2000). It has been proposed that victims of child maltreatment are more likely to develop substance addiction in order to cope with stress. The relative ease of access to alcohol also puts this population at greater risk for alcohol abuse and dependence (D. F. Becker & Grilo, 2006; Hyman, Paliwal, & Sinha, 2007; Widom, White, Czaja, & Marmorstein, 2007; Zlotnick et al., 2007).

Lisak & Bezterczey (2007) suggested that the experience of maltreatment leads to substance abuse and criminal offending. Furthermore, victims of

maltreatment experience severe developmental problems indicating disturbances in academic, occupational and relational experiences, making the transition to adulthood a major difficulty for this population.

#### *1.10 Impact of Child Maltreatment on Overall Cognitive Function*

More recently, it has been recognized that as a group, maltreated children are exposed to risk factors for neuropsychological deficit (Beers & De Bellis, 2002; Carrey et al., 1995; Glaser, 2000; Mezzacappa, Kindlon, & Earls, 2001; Pears & Fisher, 2005; Porter, Lawson, & Bigler, 2005; Tapert & Brown, 1999; M.H Teicher, 2002; Veltman & Browne, 2001). Strathearn, Gray, O'Callaghan and Wood (2001) observed that the general cognitive functioning of neglected infants was reduced and continued to show a progressive decline at four year follow up. A similar study found that neglected infants showed persistent cognitive dysfunction despite efforts to reverse the impact of the neglectful environment (L. Singer, 1986).

Physically abused infants displayed similar cognitive outcomes, Appelbaum (1977) found that general developmental retardation in addition to specific deficits in language and gross motor skills were characteristic of a physically abused group of infants. The proportion of children in both abused and neglected groups with IQs below 70 falling in the intellectually disabled range (also known as mental retardation in some countries and literature) was almost ten times of that of the control group in another study (Sandgrund, Gaines, & Green, 1974). Consistent with these findings a maltreated sample of seven to 13 year olds performed significantly more poorly on FSIQ and verbal IQ (VIQ) in comparison to a comparable control group, though measures of other cognitive domains were not included (1995). Dubowitz, Papas, Black and Starr (2002) found that the cognitive development of an entire sample of neglected preschoolers was impaired at follow up (age five years). More specifically, the children displayed: limited impulse control, poor academic performance, poor language comprehension, lower IQ scores and restricted creativity (Dubowitz et al., 2002). The combination of failure to thrive in early infancy and parental neglect were examined in relation to their impact on cognitive functioning, it was found that infants with both of these risk factors fell 15 points lower on IQ falling within the low

average range in comparison to controls (Mackner, Starr, & Black, 1997). The authors also found that infants with only one risk factor (i.e. either failure to thrive or neglect) did not differ significantly from controls on IQ, falling within the average range.

Hoffman-Plotkin and Twentyman (1984) explored the cognitive capacities of both neglected and physically abused preschoolers and found that both groups scored lower on all measures of cognitive functioning including IQ and language ability when compared to controls. Observationally, the abused and neglected children were least ready to learn, immature and poorly equipped for the demands of social interactions with peers (Hoffman-Plotkin & Twentyman, 1984). These problems also continue into school age years, children with histories of neglect and sexual abuse have been found to show major difficulties in engaging within the school environment, limiting their ability to achieve academic success (Daignault & Hebert, 2009; Dodge-Reyome, 1994). More specifically, it has been suggested that students with maltreatment histories are inattentive, have difficulties regulating their behaviour, are more dependent on teacher's help and are more withdrawn from the classroom (Daignault & Hebert, 2009; Erickson, Egeland, & Pianta, 1989). However when compared to children of similar socioeconomic status without histories of maltreatment, it was found that both groups performed similarly within the classroom, indicating that it may be opportunities for academic stimulation at home are more relevant to classroom behaviour rather than experience of maltreatment (Dodge-Reyome, 1994).

### *1.11 Specific Cognitive Deficits Associated with Child Maltreatment*

Deficiencies in language comprehension and expression seem to be commonly experienced by abused and neglected children. The impoverished communicative functioning of maltreated infants as measured by standardized tests and observations has been attributed to patterns of maternal ignoring and lack of verbal stimulation (Allen & Wasserman, 1985; Coster, Gersten, Beeghly, & Cicchetti, 1989; Koluchova, 1972). It has been found that children who were abused, neglected and both abused and neglected experienced delay in general language skills and cognitive functioning (Culp et al., 1991). The research suggests that neglected children experience the most

severe deficits in measures of both expressive and receptive language when compared to physically abused children and controls (Culp et al., 1991; Fox, Long, & Langlois, 1988). In contrast, Alessandri (1991) did not find any differences between maltreated children and controls on standardized measures of language functioning, however observational data suggested problems of social communication and peer interaction. Similarly, adolescent victims of childhood maltreatment did not differ significantly from their non maltreated counterparts on measures of comprehension and expressive vocabulary, however differences were observed in their functional communication abilities (McFayden & Kitson, 1996). Oates, Gray, Schweitzer, Kempe and Harmon (1995) found that abused children presented minimal developmental gains in language functioning following intervention suggesting a persisting language problem.

More recently, studies into childhood abuse have utilized a neuropsychological perspective to explore specific domains of cognitive functioning affected by history of maltreatment. However, the availability of such research is limited and expresses diverse findings. Experience of childhood abuse and neglect has been associated with poor functioning on measures of visuospatial processing and language (Pears & Fisher, 2005). Beers and De Bellis (2002) administered a comprehensive neuropsychological battery to a group of children with maltreatment related posttraumatic stress disorder. Statistically significant differences on measures of attention, executive functioning and learning were found, other deficits on measures of language, memory and learning, visuospatial skills and psychomotor performance were no longer significant after Bonferroni corrections were applied. Primary school aged children with experiences of childhood neglect have been found to show a number of impairments in a range of cognitive domains (De Bellis, Hooper, Spratt, & Woolley, 2009). When compared to controls, the neglected children in this study showed significantly lower performances on measures of overall cognitive function, language, visuospatial skills, learning and memory, attention and executive function and academic achievement that remained after Bonferroni corrections were applied. This study also compared groups of neglected children with PTSD and those without. Although PTSD was negatively correlated with a number of cognitive variables, statistically significant differences between the neglect only and neglect with PTSD groups were only found on one measure of delayed recall.

Porter et al (2005) examined the impact of childhood sexual abuse on cognitive functioning. It was found that sexually abused children performed poorer than controls on the Test of Memory and Learning (TOMAL) subtests of attention and concentration, however after controlling for socioeconomic status and IQ no significant differences were found. In this important study, the authors themselves noted that only a limited range of measures were used, increasing the likelihood that deficits which would've been otherwise detected were missed. Measures of executive functioning, working memory and visuospatial functioning were not administered and their inclusion would have tested for deficits in these higher order cognitive functions. The authors proposed that the measures used in the study may have been limited in the range of cognitive functions that could be assessed. The TOMAL was also used in a study by Palmer et al (1999), and consistent with Porter et al, no significant differences between the sexually abused and control groups were found on this test. Additional deficits of FSIQ and Verbal IQ performance were reported for the sexually abused group, providing further evidence for language impairment in abused populations.

Mezzacappa, Kindlon and Earls (2001) specifically tested executive functioning in maltreated boys, the findings demonstrated that maltreated boys show limited improvement on measures of impulse control and inhibition with age. The authors suggest that child maltreatment may impede the expected developmental progression of competence in executive functioning. Similarly, young adult female survivors of child sexual abuse were found to have significantly poorer functioning in attention, inhibitory capacity, memory functions and scholastic aptitude, providing further evidence for the suggestion that childhood sexual abuse may be associated with neuropsychological deficit (2004). One other study in this area has reported no significant effects of maltreatment on cognitive abilities (Samet, 1997).

### *1.12 Childhood Traumatic Brain Injury and Cognitive Function*

Physical abuse can have direct effects on brain structure and functioning if it involves direct blows to the head, strong forces of pushing and shoving, or repeated shaking. Experiences of this nature can result in childhood traumatic brain injury,

which has been shown to result in a range of detrimental outcomes, including death (Karandikar, Coles, Jayawant, & Kemp, 2004; Makaroff & Putnam, 2003).

#### *1.12.1 Traumatic brain injury defined*

According to the Guidelines for Surveillance of Central Nervous System Injury (Thurman, Sniezek, Johnson, Greenspan, & Smith, 1995), Traumatic Brain Injury (TBI) “can be summarised as an occurrence of injury to the head (arising from blunt or penetrating trauma or from acceleration-deceleration forces) that is associated with symptoms or signs attributable to the injury: decreased level of consciousness, amnesia, other neurological or neuropsychological abnormalities, skull fracture, diagnosed intracranial lesions- or death” (cited in Thurman, Alverson, Dunn, Guerrero, & Sniezek, 1999, p. 603). Blunt force and acceleration-deceleration injuries are commonly termed closed head injuries as the skull remains intact, whilst penetrating injuries are known as open head injuries as the skull and dura are pierced by an external object (Lezak et al., 2004).

#### *1.12.2 Neuropathophysiology of traumatic brain injury*

A number of processes occur in the brain during a head injury. During the event of trauma, the brain may be placed under significant stress, causing deformation, stretching and compression of brain tissue (H. S. Levin, Benton, & Grossman, 1982). These forces, if strong enough, can cause contusion of the brain and axonal tearing (diffuse axonal injury), often leading to neuronal death (Zillmer et al., 2008). These primary injuries may be followed on by, cerebral oedema, intracranial haemorrhage, increased intracranial pressure, lowered blood pressure and ischaemia, resulting in further brain damage (H. S. Levin et al., 1982).

Injury can occur in the brain tissue underlying the point of impact (coup injury) or tissue closest to the region that is opposite to the point of impact, known as a countercoup injury (Drew & Drew, 2004). Countercoup injuries occur as the brain is detached from the skull at the point of impact and hits the opposite side. Strong acceleration and deceleration forces can lead to profound diffuse axonal injury, causing extensive damage to axons and blood vessels (Silver, McAllister, & Yudofsky, 2004).



### *1.12.3 Child maltreatment related traumatic brain injury*

Research has shown that children who present with head injury often show evidence of other types of physical abuse, such as fractures when being examined in the medical setting (Leventhal, Thomas, Rosenfield, & Markowitz, 1993; J. A. O'Neill, Meacham, Griffin, & Sawyers, 1973). In a sample of children with strong clinical evidence of physical abuse aged three weeks to 16 years, skull fractures were the second most common type of injury shown on radiologic examination (Merten, Radkowski, & Leonidas, 1983). Similarly, Merten, Osborne and Leonidas (1984) reported that 70 percent of children presenting with intracranial injury also showed evidence of skeletal injuries. This suggests that child victims of physical abuse are at great risk for incurring traumatic brain injuries. Most concerning is that most of these injuries remain undetected, particularly in infants. Evidence of this was shown in a study of 173 abused infants, where approximately 30 percent of cases with abuse related head injuries were not identified at their initial presentation to medical treatment (Jenny, Hymel, Ritzen, Reinert, & Hay, 1999). Furthermore, brain imaging studies found signs of previously undetected head trauma, in 45 percent of abused children aged one month to six years, presenting for treatment of further head injuries (Ewing-Cobbs, Kramer et al., 1998).

Children with maltreatment related TBI, also show different patterns of brain pathology when compared to children with non-inflicted TBI. Sub-dural haematomas were more common amongst children with abuse related TBI, whilst those with non-inflicted TBI commonly showed epidural haematomas and shear injuries (Ewing-Cobbs, Kramer et al., 1998). Computed Tomography (CT) analyses of 712 physically abused children indicated similar findings, where sub-dural haemorrhage and increased extra-cerebral fluid volumes were the most commonly identified lesions in the sample (Merten et al., 1984). This study also reported that a high proportion of children (45%) presented with skull fractures, suggesting that the craniocerebral trauma was consistent with impact head injuries. This is an important consideration, as in many instances, victims of child abuse may also have TBI related cognitive deficits.

#### *1.12.4 Developmental and neuropsychological outcomes of children with traumatic brain injury*

As there is evidence to suggest that childhood physical abuse may lead to traumatic brain injuries, it is important to have an understanding of the neuropsychological outcomes associated with such injuries. Frontal lobe impairment as demonstrated by poor executive functioning was shown in a sample of children with TBI (Hanten, Bartha, & Levin, 2000). Slomine et al (2002) found that childhood TBI was associated with more perseverative errors on the Wisconsin Card Sorting Test (WCST) and poor letter fluency, indicating deficits in executive function. Adolescents and adults with childhood history of TBI were examined for executive function deficits seven to 10 years post injury. The results showed that those who had experienced severe TBI during childhood displayed more serious indications of executive dysfunction, particularly in areas of cognitive flexibility, abstract reasoning and goal setting (Muscara, Catroppa, & Anderson, 2008). Similar reports of paediatric TBI patients found that, social functioning, particularly those requiring executive related skills were greatly affected at long term follow up (Landry, Swank, Stuebing, Prasad, & Ewing-Cobbs, 2004).

TBI patients have also been shown to demonstrate continued difficulties on in a wide range of cognitive tasks at long term follow up, including attention, processing speed, memory (Ewing-Cobbs, Prasad et al., 1998; van Heugten et al., 2006), working memory, inhibitory control (Ewing-Cobbs, Prasad et al., 1998; Ewing-Cobbs, Prasad, Landry, Kramer, & DeLeon, 2004) and verbal learning (Roman et al., 1998). Children with severe TBI also demonstrated more behaviour problems, poor adaptive skills and limited performance on measures of academic achievement at approximately 4 years post injury (Taylor et al., 2002). Limited gains in academic achievement were also noted by Ewing-Cobbs, Barnes et al (2004) in individuals aged 10-20 years at five years post injury. Those identified with severe TBI, showed the most deficits at follow up, whilst the mild to moderate group demonstrated significant improvements over the follow up period. Other studies have also shown that children with mild traumatic brain injury have better outcomes following injury, demonstrating average cognitive abilities, although verbal fluency appeared to remain impaired (Mathias & Coats, 1999).

Landry, Swank, Stuebing, Prasad and Ewing-Cobbs (2004) examined maltreatment related TBI in a sample of infants aged 3 to 23 months. Social competence was measured in this group of children in comparison to controls, the findings indicated that the TBI group had difficulty initiating social behaviours and responding to social interaction. They also showed less positive affect and did not comply with simple requests. The authors of this study suggested that a maltreating environment poses additional pressures on developmental processes that would otherwise occur to improve functioning following TBI. Similar findings have been reported, where long term outcomes of children with paediatric TBI, particularly those who had severe injuries, were greatly affected by the family environment and socioeconomic status (Schwartz et al., 2003; Taylor et al., 2002).

#### *1.12.5 Shaken Baby Syndrome*

Shaken Baby Syndrome (SBS), also known as Abusive Head Trauma in other parts of the world, is a severe form of maltreatment related trauma resulting in a distinctive pattern of intracranial, retinal and cervical spinal cord injuries (Billmire & Myers, 1985). These significant injuries are the consequence of an infant being shaken violently by a caregiver, with strong acceleration, deceleration and rotational forces causing widespread brain damage (King, MacKay, & Sirnick, 2003; Kleinman, 1990).

In a large post-mortem study of 53 infants who had died due to SBS related injuries the most common injuries found were skull fractures, sub-dural haemorrhage and retinal bleeding (Geddes, Hackshaw, Vowles, Nickols, & Whitwell, 2001). In 82 percent of cases, cause of death was a result of increased intracranial pressure due to brain swelling. Widespread hypoxic brain damage was present in 77 percent of cases, and approximately 20 percent of cases showed evidence of axonal injury at the craniocervical junction or cervical cord. This study also showed differential brain pathology and injury dependent on age; those children below one year were more likely to show axonal injury, skull fractures and low level sub-dural bleeding. Older children showed, larger sub-dural haemorrhages and severe internal injuries, particularly in the abdomen.

Given the forces of SBS are thought to be similar to those that result in diffuse axonal injury (DAI), researchers have been interested in whether there is microscopic evidence in victims of SBS to support this notion. In order to answer this question, Geddes, Vowles et al (2001) used immunohistochemistry to examine the presence of axonal damage in the brains of 37 SBS infants below nine months of age post mortem. The findings showed vascular related axonal damage in approximately 35 percent of cases, although only 2 cases showed evidence of DAI. Consistent with previous findings, widespread hypoxic damage and axonal injury at the craniocervical junction were evident. The authors suggested that the lack of DAI may be attributed to one of two reasons; firstly, unmyelinated axons may have greater capacity to resist traumatic injury, or secondly, that the forces produced as a result of shaking are not strong enough to result in DAI.

Similar findings have been reported in case studies of SBS infants where CT scanning and MRIs were used for diagnostic purposes, on hospital admission. The case studies of 2 month old infants, one male (Parizel et al., 2003) and one female (Y. Chan, Chu, Wong, & Yeung, 2003) found evidence of a number of sub-dural haematomas, bilateral retinal haemorrhages, epileptic seizures and white matter lesions that were due to hypoxic ischemic brain injuries rather than DAI.

The SBS cases reported in the literature are those that were severe enough to present to hospital. The number of individuals with SBS injuries who have not come to medical attention is unknown. It could be argued that a large number of children and adolescents with abuse histories fall in this category. Thus, the cognitive impairment shown in these individuals may be a consequence of SBS related injuries that have not been documented at the time of injury.

#### *1.12.6 Developmental and neuropsychological outcomes of children with Shaken Baby Syndrome*

The severe and widespread injury related to SBS is related to equally profound developmental outcomes. A retrospective review of case records of children diagnosed with SBS under the age of five was undertaken to examine the characteristics of the children presenting with SBS and their developmental outcomes (King et al., 2003). Of the 364 cases examined, 295 survived the SBS related injuries.

Persistent neurological deficits remained in 55 percent of cases, whilst 65 percent had visual impairments. Only seven percent were recorded as having average intellectual functioning, whilst 48 percent had a moderate to severe degree of disability and 12 percent were in a coma or vegetative state. Other studies following up the outcomes of SBS children have consistently shown that SBS children; performed significantly poorer on measures of overall cognitive function, had long standing neurological deficits, severe motor impairments, visual impairments and epileptic seizures (Karandikar et al., 2004; Talvik et al., 2007). Ewing-Cobbs et al (1998) followed up children 1.3 months after injury. Cognitive testing indicated 45 percent of children fell within the intellectually disabled range and 25 percent had severe motor deficits. In a complementary study, Ewing-Cobbs, Prasad, Kramer and Landry (1999) conducted follow up at approximately 4.6 months after injury, SBS children indicated some minor improvements where performances on both overall cognitive function measures and motor function measures fell within the low average ranges. Longer term follow up (up to 90 months) of SBS children was conducted to examine neurologic and neuropsychological outcomes (Barlow, Thomson, Johnson, & Minns, 2005). Over one third of the sample demonstrated severe neurological deficits, requiring substantial aid and support. Severe impairments in speech and language skills were found in 64 percent of cases. Behavioural impairments, including, self harming, hyperactivity, impulsivity, tantrums, and rage reactions were shown in 52 percent of the sample. Almost half of the participants demonstrated difficulties in social interactions and adaptive behaviours.

SBS children were followed up during their early school age years and were assessed using a neuropsychological battery (Stipanivic, Nolin, Fortin, & Gobeil, 2008). Children with severe neurological deficits in relation to SBS were excluded from the study. The results indicated that SBS children performed significantly poorer than controls on FSIQ, performing almost one standard deviation below the population mean. Specific skills related to working memory, attention, executive functioning and learning and memory were also significantly impaired in comparison to controls. Taken together, these findings suggest that children who fall victim of SBS develop difficulties among several cognitive domains, consistent with the widespread damage that occurs as a result of their injuries.

### *1.13 Impact of Stress on Cognitive Development and Function*

The stress response, mediated by the hypothalamic-pituitary-adrenal axis (HPA axis), is an adaptive function which is activated in order to assist an individual during adverse or challenging circumstances. Prolonged stress, which may be a consequence of sustained child maltreatment can have various pathologic effects (De Bellis, 2004; Sapolsky, 1996). It has been postulated that a child's repeated internal responses to trauma become established in the neuronal circuitry, resulting in the pattern of symptomatology, namely dissociation and hyperarousal, that develop following prolonged trauma such as child maltreatment (Perry, Pollard, Blakely, Baker, & Vigilante, 1995).

During periods of stress, the HPA axis stimulates the secretion of glucocorticoids which have numerous target receptors around the body including the brain (De Bellis, Baum et al., 1999). The hippocampal region of the brain is the most rich in glucocorticoid receptors and functions to facilitate memory, attention and learning processes (Sapolsky, 1996). Excessive stress related secretion of glucocorticoids put hippocampal neurons at risk of deficient functional capacity or in extreme cases neuronal death (De Bellis, Keshavan et al., 1999; Sapolsky, 1996).

Animal studies have consistently shown that manipulating glucocorticoid levels to mimic those that would occur under extreme stress result in cognitive deficits as well as deterioration of brain structures. Arbel, Kadar, Silbermann and Levy (1994) found that increasing plasma corticosterone (a glucocorticoid) concentrations in rats was related to impaired cognitive functioning and neuronal loss within the hippocampus. It has been suggested that glucocorticoids inhibit glucose uptake within hippocampal neurons, reducing neuronal energy stores (Armanini, Hutchins, Stein, & Sapolsky, 1990). This lowered energy within neurons is then a trigger for a cascade of various processes leading to toxic levels of substances within the brain which cause degeneration of brain structures (Armanini et al., 1990). The hippocampus has been shown to be most at risk of neurological damage due to high plasma glucocorticoid levels (Packan & Sapolsky, 1990), particularly if these levels are maintained over a prolonged period of time (Sapolsky, Krey, & McEwen, 1985; Sapolsky, Uno, Rebert, & Finch, 1990; Uno, Tarara, Else, Suleman, & Sapolsky, 1989). In humans, the impact of glucocorticoids on the hippocampus has been investigated in patients with

Cushing's Syndrome. Results indicated that these patients had significantly smaller hippocampal volumes and these findings correlated with scores on tasks of learning and memory (Starkman, Gebarski, Berent, & Schteingart, 1992).

Neurotransmitter release during the stress response has also been implicated in the structural deterioration of the prefrontal cortex. Arnsten and Shansky (2004) suggested that the release of dopamine during stress impaired prefrontal functioning in adolescents, and that this may be related to the limited judgment adolescents display during emotional and stressful situations.

Studies have also suggested that maternal responsiveness during the early days of postnatal life may have an influence on the programming of the HPA axis (Ladd, Owens, & Nemeroff, 1996; Stanton, Gutierrez, & Levine, 1988). Liu and Meaney (1997) found that the adult offspring of maternal rats that received maternal care (such as licking and grooming) in the first 10 days of life demonstrated reduced plasma adrenocorticotrophic hormone and corticosterone responses to acute stress. Rats that were not handled during these early days secreted more glucocorticoids in response to stress, and at later ages, exhibited elevated basal glucocorticoid levels (Meaney, Aitken, van Berkel, Bhatnagar, & Sapolsky, 1988). This indicates that early maternal neglect may contribute to a hyperresponsive HPA axis, leading to high concentrations of glucocorticoids in the bloodstream following an episode of stress.

From a theoretical standpoint it could be postulated that stress induced by repeated incidence of child maltreatment could have an impact on brain structures and associated functioning such as memory, attention and learning. Many studies have reported that children and adolescents who have experienced trauma, interparental conflict and violence showed evidence of an altered stress response, indicated in both physiological and neuroendocrinological measures (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Perry, 2001; Perry & Azad, 1999; Perry & Pollard, 1998). Kaufman et al (1997) found that depressed maltreated children presented with significantly increased levels of adrenocorticotrophic hormone, the hormone responsible for the stimulation of glucocorticoid secretion. Women who had experienced sexual assault demonstrated acute high concentrations of cortisol in their blood streams, however those that had a history of previous assaults showed lower

levels of cortisol following examination (Resnick, Yehuda, Pitman, & Foy, 1995). The authors suggested that the acute cortisol response to trauma may be reduced in individuals who have experienced trauma in their pasts. In contrast, women with a history of childhood sexual abuse and post traumatic stress disorder diagnoses have demonstrated chronic high levels of cortisol (Lemieux & Coe, 1995). Baum, Cohen, and Hall (1993) offered the explanation that victims may reexperience their trauma in the form of memories throughout the course of their lives leading to reactivation of the HPA axis on a regular basis. De Bellis, Chrousos et al (1994) also found significant HPA axis dysregulation in sexually abused girls, suggesting that the severe stress resultant of sexual assault may have contributed to permanent adaptive changes in the physiological response to stress. Recent work has shown that children exposed to interparental conflict also have high cortisol reactivity; those children who were involved in the conflictual interactions exhibited particularly high levels of cortisol (Davies et al., 2008).

Increased HPA axis stimulation and thus increased activity of glucocorticoids in the brain may affect processes of neuronal migration, differentiation and synaptic proliferation, contributing to altered brain development (De Bellis, Baum et al., 1999). Stress hormones have also been shown to affect the process of myelination. It was found that injection of hydrocortisone (a glucocorticoid) suppressed the proliferation of cells that form the myelin sheath in rats (Bohn, 1980). Magnetic Resonance Imaging (MRI) scans of maltreated children with posttraumatic stress disorder (PTSD) have revealed that these children have significantly smaller intracranial and cerebral volumes, particularly in the region of the corpus callosum (De Bellis, Keshavan et al., 1999). Low IQ scores were found consistently in the maltreated group of children and coincided with the decrease in cerebral volume, the authors indicated that stress may have impaired brain development resulting in poor cognitive and behavioural functioning (De Bellis, Keshavan et al., 1999). A review by Bremner, Krystal, Charney and Southwick (1996) indicated that extreme stress instigated by exposure to child maltreatment has long term effects on memory. Other studies regarding adolescents without maltreatment history have suggested that stress during the adolescent stage alters prefrontal cortical function contributing to problems of inattention, poor inhibition, problem solving and working memory (Arnsten, 1999;



Gunnar, 1998). The lack of these skills relates to the difficulties adolescents have in situations where the capacity to process information quickly and at a higher order level is required. Yang & Clum (2000) have suggested that these factors put adolescents with maltreatment histories at greater risk of suicide.

The impact of stress on cognitive functioning has been explored in adult combat war veterans with post traumatic stress disorder (PTSD), results indicated that they had deficits in the ability to learn new material, memory (Yehuda et al., 1995b), concept formation, problem solving, decision making, verbal memory and visuospatial functioning (Barrett et al., 1996). This has been related to reduced right hippocampal volume in war veterans with PTSD (Bremner, Randall, Scott, Bronen et al., 1995; Gurvits et al., 1996). Similar findings have been reported in adult survivors of child abuse, with deficits in verbal short term memory (Bremner, Randall, Scott, Capelli et al., 1995). These deficits in memory have been related to decreases in hippocampal volume. Bremner et al (1997) found a twelve percent reduction in hippocampal volume of adult survivors of child abuse, the authors associate the reduction in volume to increased secretion of glucocorticoids and subsequent degeneration of hippocampal neurons. In support of these findings, Zhang, Xing, Levine, Post, & Smith (1997) found evidence of neuronal death in the hippocampus associated with the stress of maternal deprivation (prolonged separation from the mother) in rats.

The release of glucocorticoids in response to maternal stress during pregnancy may also be a factor that affects brain development in the foetus. Women who experience abuse, such as domestic violence are more likely to be in a situation where their children become victims of maltreatment themselves (Fantuzzo, Boruch, Beriama, Atkins, & Marcus, 1997). It could be hypothesised that in many cases, maltreated children are born to mothers who themselves are victims of abuse. One study reported that child abuse occurs alongside intimate partner violence in as high as 97 percent of cases (Kolbo, 1996). In instances of intimate partner violence, mothers may experience stress during pregnancy having elevated glucocorticoids within their bloodstreams that are also being circulated through the foetus. These elevations may have an affect on the developing brain structures of the foetus. In animal studies, it has been found that repeated doses of glucocorticoids during

pregnancy results in a reduction of myelination within the central nervous system of the foetus (Dunlop, Archer, Quinlivan, Beazley, & Newnham, 1997), reduced brain weight at term (Huang et al., 1999) and neuronal degeneration within the hippocampus (Uno et al., 1990). Therefore, children at risk of maltreatment may have brain impairments that are present before they have even been abused.

Taken together, these findings indicate that the physiological response to stress may be the underlying mechanism contributing to the cognitive deficits in victims of child maltreatment. As the stress occurs during a critical period of brain development, it is important to explore its impact on all domains of cognitive functioning, which in the literature to date is quite limited.

#### *1.14 Cognitive Functioning and Substance Abuse*

It has been shown that substance abuse rates peak during the adolescent period in normal populations (Melchior, Chastang, Goldberg, & Fombonne, 2008). As noted previously, engaging in substance abuse is a common feature of adolescents with histories of maltreatment and in particular, those that come to the attention of Secure Welfare services. The literature indicates that child maltreatment is a major risk factor for the development of substance abuse disorders/dependence during adolescence and adulthood (De Bellis, 2002; Malinosky-Rummell & Hansen, 1993). One study reported that abused and neglected individuals were one and a half times more likely to be involved in illicit substance abuse in middle adulthood when compared to controls (Widom, Marmorstein, & White, 2006). In a sample of 1,179 adolescents with histories of maltreatment 20 percent reported low levels of substance use whilst nine percent reported moderate to high levels (Wall & Kohl, 2007). Harrison, Fulkerson and Beebe (1997) found a strong relationship between child maltreatment and polysubstance abuse in an adolescent population. In comparison to their age matched peers, maltreated adolescents were more likely to; initiate substance abuse at an earlier age, experiment with a larger selection of substances and use the substances more frequently as a means of coping with painful emotions.

It is also important to consider the potential effects of prenatal exposure to substance abuse in maltreated populations. Clinical reports would indicate that it be

highly likely that many of these children and adolescents have been exposed to drugs during gestation. Reports have shown that over 30 percent of infants exposed to drugs prenatally were identified as abused or neglected during childhood (Jaudes, Ekwo, & van Voorhis, 1995). A lot of drug abusing women may not even realise they are pregnant until after the first trimester is close to complete. Exposure to teratogens during the first three months of pregnancy can lead to birth defects, developmental abnormalities and spontaneous abortion, as this is the most significant period for neurogenesis, migration and differentiation (Peterson, 1996).

As substance abuse is very strongly associated with maltreatment in childhood, it is important to examine its influence on cognitive development and functioning. The mechanisms underlying substance abuse in this population have been related to elevations of catecholamines and central CRH (which have been found to be typical in abused children) during development (De Bellis, 2002). De Bellis (2002) also suggested that dysregulation of a young person's HPA axis may lead to symptoms of negative effect, which increases the risk of the use of alcohol and other drugs for their 'self-medicating' properties. High concentrations of catecholamines have also been implicated in altered brain development, influencing neurogenesis and brain morphology (De Bellis, Keshavan et al., 1999; Sapolsky, 1996; Sapolsky et al., 1985; Sapolsky et al., 1990). As a consequence, substance abuse behaviours may be a product of the limited development of brain areas such as the prefrontal cortex which is responsible for executive functions and self-regulatory behaviours.

#### *1.14.1 Cannabis use and cognitive function*

The endogenous cannabinoid system extends throughout various regions of the central nervous system including the frontal and hippocampal regions (Iverson, 2003). As a result, cannabis abuse has the potential to affect various areas of the brain. Animal brain morphology studies have shown that prolonged cannabis use is associated with neuronal changes in the frontal, hippocampal and cerebellar regions of the brain (G. C. Chan, Hinds, Impey, & Storm, 1998; Gurvits et al., 1996; J. W. Harper, Heath, & Myers, 1977; Heath, Fitzjarrell, & Fontana, 1980; Romero, Garcia, Fernandez-Ruiz, Cebeira, & Ramos, 1995). Delta-9-tetrahydrocannabinol (THC), which is the main psychoactive component of cannabis has been used in experimental studies with animals to test whether it has any toxic effects on the neurons of the

central nervous system. As the hippocampus has large amount of cannabinoid receptors, Chan et al (1998) tested rat pup hippocampal neurons and found that THC is neurotoxic, causing cell death even at very low concentrations. They also suggested that these levels are comparable to the THC concentration in human plasma after consumption of cannabis. The destruction of hippocampal neurons by THC may be the mechanism underlying memory deficits associated with cannabis abuse (G. C. Chan et al., 1998). On the contrary, early imaging studies investigating the affects of cannabis use on brain morphology of humans found no evidence of cerebral changes in long term heavy cannabis smokers (Co, Goodwin, Gado, Mikhael, & Hill, 1977; Hannerz & Hindmarsh, 1983; Kuehnle, Mendelson, Davis, & New, 1977). Similar findings were reported in a later MRI study of 18 young adults who had been frequent users of cannabis for a period of two years or more (Block et al., 1999). The MRIs showed no evidence of total cerebral atrophy or regional cerebral atrophy in comparison to controls. Contrary to expectation, changes in grey and white matter densities were also absent. Block and colleagues did caution that their findings may be limited by the imaging techniques employed, which were not able to detect changes at the microscopic level. Furthermore, the participant's duration of marijuana use may have been too shorter a period to contribute to identifiable anatomical changes in the brain.

Wilson, Mathew, Turkington, Hawk, Coleman and Provenziale (2000) examined the relationship between age of cannabis use onset (before or after the age of 17 years) and its effect on brain architecture. Changes in brain volume were non-significant; however proportions of cerebral white and grey matter were significantly different for those who began use in early adolescence when compared to those who began use after the age of 17 years. A comparable control group of cannabis abstinent individuals was not included in this study. Those who began taking cannabis before the age of 17 demonstrated a significant reduction in grey matter, particularly in the frontal lobes and a significant increase in white matter. The authors interpret these findings using a developmental framework whereby ingestion of cannabis may interact with gonadal and pituitary hormones which influence the normal course of brain development during the adolescent period. Disparities in grey and white matter densities within specific brain regions were investigated by Matochik, Eldreth, Cadet,

and Bolla (2005) in a sample of heavy cannabis users using voxel based morphometry. Heavy cannabis users had lower grey matter density in the right parahippocampal gyrus and greater density in the bilateral region close to the precentral gyrus and right thalamus in comparison to a non-using control group. Lower white matter density was indicated in the left parietal lobe, whilst higher density was concentrated in the left parahippocampal and fusiform gyri. Longer term heavy marijuana users also showed increased white matter density in the left precentral gyrus. In the presence of cannabis, cannabinoid receptors in the neurons of the hippocampus demonstrate reduced neurotransmitter release to a level below that required to depolarise the postsynaptic membrane (Misner & Sullivan, 1999). This suggests that cannabis may also alter neuronal functionality in specific regions of the brain.

More recently, a study conducted with 16 adolescent cannabis users, found a decreased volume of white matter density and increased depressive symptomatology in these cannabis users (Medina, Nagel, Park, McQueeny, & Tapert, 2007). These results may be attributed to the identification that the white matter tracts of the animal and human brains are rich in cannabinoid receptors making them vulnerable to the toxic effects of THC (Iverson, 2003; Romero et al., 1995; Romero, Hillard, Calero, & Rábano, 2002). Volumetric changes in the brain regions and associated cognitive deficits of heavy long term adult cannabis users have been identified (Yucel et al., 2008). They found significantly reduced bilateral hippocampal and amygdale volumes in the cannabis users relative to controls. Alteration to hippocampal function was also demonstrated as cannabis users performed significantly poorer on the Rey Auditory Verbal Learning Test (RAVLT), a measure of verbal learning. Cannabis users showed deficits in total number of words recalled over five learning trials and number of words recalled after a 20 minute delay.

As much of the literature focuses on adult populations it is difficult to report on the cognitive affects in adolescent users, even though it is usually within the adolescent period that substance abuse begins. Some have suggested that it may be difficult to detect cognitive impairment in adolescents on the premise that the length of time engaging in substance abuse has not been long enough to demonstrate deficits. Understanding the affects of cannabis on adolescents is of great importance as the

brain remains in a process of development during this stage, and it is unknown whether cannabis intervenes with these normal processes (Sundram, 2006). In a study of 17-20 year olds it was found that current marijuana users who smoked more than five joints per day showed a 4.1 point drop in full scale IQ (FSIQ) score in comparison to their preadolescent FSIQ measured from nine to 12 years of age (P. Fried, Watkinson, James, & Gray, 2002). This study also indicated that those who had been heavy users previously but were no longer using the substance showed no significant differences in their FSIQ scores at the two time intervals. Fried et al (2002) suggested that marijuana does not have a long term impact on overall cognitive functioning, however they questioned whether examining specific cognitive domains would demonstrate impairments. Taking this into consideration, Harvey, Sellman, Porter and Frampton (2007) found impairments on measures of attention, executive functioning, working memory and learning in a sample of adolescent regular cannabis users aged between 13 and 18 years. Impairments have also been demonstrated on tasks of executive function and prospective memory in a university sample (McHale & Hunt, 2008). In contrast, Teichner, Donohue, Crum, Azrin and Golden (2000) examined a global measure of cognitive functioning and demonstrated that cannabis and other substance abuse was not related to deficits in neuropsychological functioning in an adolescent sample, however they speculated that continued heavy use would result in pronounced cognitive impairment later on in life. They also suggested that adolescents may exhibit cognitive impairment during this stage as a result of secondary effects of substances such as head injuries from vehicle accidents, falls and physical assaults.

A study looking at the impact of short term and long term cannabis use on cognitive functioning in adults showed a number of neuropsychological impairments (Solowij et al., 2002). Specifically, learning and memory were found to be most impaired in long term users, indicating deficits in encoding, retention and retrieval. An inverse relationship was also found between duration of substance abuse (in years) and learning performance (Solowij et al., 2002). Pope, Gruber, Hudson, Cohane, Huestis and Yurgelun-Todd (2003) examined the cognitive functioning of early onset (before age 17) and late onset (after age 17) cannabis users, initial significant differences were found on verbal IQ (VIQ), verbal fluency and verbal memory

measures, with early onset users performing more poorly than late onset users and controls. However, after the results were statistically controlled for VIQ, no significant differences were found between the two cannabis using groups and controls. In a sample of adolescent female cannabis users, Medina, Hanson, Schweinsburg, Cohen-Zion, Nagel & Tapert (2007) showed that frequent episodes of use was associated with poorer cognitive functioning. Neuropsychologically, the adolescents demonstrated slower processing speed, poorer complex attention, story memory, planning and sequencing ability when compared to controls. In a 21 year longitudinal study of a birth cohort of 1265 children, cannabis use was investigated during the period between ages 14 and 21 (Fergusson, Horwood, & Swain-Campbell, 2002). Those that were regular users of the drug experienced a range of difficulties in adolescence and young adulthood including; polysubstance abuse, crime, depression and suicidality. These issues were more pronounced in the early onset users (age 14-15) than those who began taking cannabis in young adulthood. These outcomes may be related to the underlying structural and cognitive changes that occur within the brain in relation to cannabis use.

#### *1.14.2 Alcohol use and cognitive function*

Alcohol is a drug that appeals to the adolescent age group as it is readily available and relatively easy to obtain. Alcohol use commonly begins in the adolescent period, often before the legal drinking ages of 18 in Australia and 21 in the United States (Bauman & Phongsavan, 1999). Alcohol use and abuse is highly prevalent in adolescent communities, with insignificant differences across various socioeconomic groups (O'Malley, Johnston, & Bachman, 1998). It has been suggested that experience of stress may be one of the reasons why adolescence have a high propensity to alcohol use (Pereplechikova, Krystal, & Kaufman, 2008). Individuals may use alcohol as a coping mechanism that provides instantaneous effects, temporarily reducing the emotional dysregulation associated with a variety of sources of stress (Labouvie, 1986; Wagner, Myers, & McIninch, 1999; Wills, Sandy, & Yaeger, 2001; Wills, Sandy, Yaeger, Cleary, & Shinar, 2001)

A high proportion of adolescents below the age of 17 have reported regular episodes of binge drinking, which has been defined as having five drinks or more in a single session (Bauman & Phongsavan, 1999). Research has shown that those who

begin drinking at age 13 or less are more likely to develop lifetime alcohol dependence than any other age group (DeWit, Adlaf, Offord, & Ogborne, 2000; Grant, 1998). Genetic, psychological and life experiences are all issues that have been identified as risk factors for the development of adolescent alcohol abuse (Swadi, 1999). Individuals who suffer from varying psychopathologies including depression, anxiety and posttraumatic stress are at higher risk for becoming heavy drinkers (Clark & Bukstein, 1998; Clark, Pollock et al., 1997; Wu, Hoven, Okezie, Fuller, & Cohen, 2007). History of child maltreatment has also been identified as a major risk factor for the development of substance abuse issues, in these populations, rates of substance abuse and dependence are much higher than that found in the general population (Aarons et al., 2008; Clark, 2004; Swadi, 1999). Experiences of comorbid alcohol abuse and psychopathology are commonly found in maltreated populations (Clark, De Bellis, Lynch, Cornelius, & Martin, 2003; Perepletchikova et al., 2008). Clark, Lesnick, and Hegedus (1997) demonstrated that adolescents with alcohol use disorder were 6- 12 times more likely to have a physical abuse history and 18-21 times more likely to have a history of sexual abuse than controls. Evidence of excessive alcohol use in young adulthood has also been reported in women with histories of child abuse (Widom et al., 2007).

As there is evidence to suggest that individuals with a history of childhood maltreatment are more likely than the general population to engage in practices of chronic alcohol use and abuse it is important to understand the consequences of such behaviours on the functioning of the brain. Ethanol, the derivative of alcohol interacts with a number of different neurochemical systems in the brain, resulting in a number of functional and behavioural changes (Eckardt et al., 1998). Neuropsychologically, the acute effects of alcohol intoxication demonstrate impairments in various aspects of functioning including; planning and spatial recognition (Weissenborn & Duka, 2003), memory encoding and retrieval (Duka & Weissenborn, 2000; Duka, Weissenborn, & Dienes, 2001), working memory and response inhibition (Finn, Justus, Mazas, & Steinmetz, 1999), psychomotor function and memory (Heishman, Arasteh, & Stitzer, 1997; Hindmarch, Kerr, & Sherwood, 1991), response selection and organisation (Tharp, Rundell, Lester, & Williams, 1974) and reaction time (Huntley, 1974). It has although, been shown that these effects tend to resolve once ethanol has been



eliminated from the blood stream (Heishman et al., 1997). More concerning are the long term effects that occur as a result of frequent episodes of alcohol use over an extended period of time.

Studies examining adult alcohol patients have indicated varying degrees of atrophy in different regions of the brain. Torvik, Lindboe, and Rodge (1982) completed post mortem examinations of 545 alcoholic patients and 586 controls aged between 55 and 67 indicating that cerebral atrophy was found in 26.8 percent of all examined alcoholics. Further investigations indicated that alcoholic patients had a significant reduction in brain weight with a mean weight difference of 31 grams. Post mortem investigations of 22 brains of documented male alcoholics in comparison to controls indicated that alcoholic patients had significant neuronal loss in the superior frontal cortex, with an associated increase in glial cells in the same region (C. Harper & Kril, 1989). There were no significant losses of neurons in the motor cortex; however the size of neurons in this region were found to be significantly reduced.

An MRI study of individuals aged between 23 and 70 showed significantly reduced cortical grey and white matter and increased cerebrospinal fluid (CSF) levels in chronic alcoholic patients (Pfefferbaum et al., 1992). These changes were significantly greater than those expected in the normal ageing population. The authors also suggested that increased age was associated with a greater vulnerability to alcohol toxicity in the brain. Pfefferbaum, Sullivan, Rosenbloom, Mathalon, and Lim (1998) assessed the structural brain changes in alcoholic men over a five year period using MRI. A reduction of brain tissue volume was indicated in the prefrontal cortex. Accelerated reduction in grey matter was found in the anterior superior temporal lobe specific to alcohol dependent patients. The amount of alcohol ingested over the five year period significantly predicted the rate of cortical grey matter loss. It can be seen in the literature of adult alcohol dependent patients that protracted use of high doses of alcohol are associated with structural changes in the brain.

The adolescent population appears to be particularly vulnerable to the effects of alcohol abuse due to the immaturity of particular brain systems that serve to regulate alcohol intake (Spear, 2004a). White and Swartzwelder (2004) suggested that adolescent rat hippocampi inhabit a high proportion of NMDA receptors which are

extraordinarily sensitive to ethanol inhibition. This greater sensitivity to the inhibitory effects of ethanol relate to greater ethanol-induced inhibition of long-term potentiation which underlie processes of learning and memory (Blitzer, Gil, & Landau, 1990; Pyapali, Turner, Wilson, & Swartzwelder, 1999). The inhibitory effects of ethanol have also been extended to posterior cingulate cortex of adolescent rats, a region associated with visuospatial function, memory and cognitive shifting (Q. Li, Wilson, & Swartzwelder, 2002). Resistance to the sedative effects of alcohol has been demonstrated in adolescent rats (Little, Kuhn, Wilson, & Swartzwelder, 1996). This resistance has been associated with high numbers of undeveloped GABA<sub>A</sub> receptors in adolescent brains (Silveri & Spear, 2002). Studies have shown that adolescent rats lack sensitivity to ethanol induced motor impairments as opposed to adults (White, Bae et al., 2002; White, Truesdale et al., 2002). These factors may put adolescents at higher risk for heavy alcohol consumption as it takes more alcohol for them to experience the incapacitating sedative and motor effects associated with heavy drinking (Spear, 2004a; White & Swartzwelder, 2004).

De Bellis, Clark et al (2000) examined various brain structures of adolescents and young adults aged between 13 and 21 years using MRI. They found reductions in hippocampal volume of those individuals who had adolescent onset alcohol use disorder. Cortical grey and white matter densities, amygdala and corpus callosum sizes appeared unaffected when compared to controls. Earlier age of alcohol use onset was associated with smaller hippocampal volume. Medina, Schweinsburg, Cohen-Zion, Nagel, and Tapert (2007) found that adolescent alcohol users had smaller left hippocampal volumes and greater right>left asymmetry than concurrent alcohol/cannabis users and controls. These authors suggested that the combined use of marijuana and alcohol may relate to significant microstructural alterations (such as glial proliferation) which could be demonstrated as normal hippocampal volume, even though the functional capacity has been reduced. Investigations of changes in white matter microstructure have shown that adolescents with alcohol use disorder have subtle abnormalities in the splenium of the corpus callosum (Tapert, Theilmann, Schweinsburg, Yafai, & Frank, 2003). These subtle changes coincide with literature that has reported white matter microstructural impairments in corpus callosal splenium of alcoholic adults (Pfefferbaum et al., 2000).

Adolescents and young adults are more likely to engage in binge drinking behaviours (Courtney & Polich, 2009). Structural brain changes in relation to binge drinking have been compared in adolescent and adult rats (2000). Significant brain damage was shown in both adolescents and adults, however, differential regions were affected. Both groups showed equal damage in the olfactory bulbs, however the associated frontal cortical olfactory regions were only damaged in the adolescent rats. The piriform and perirhinal cortices were also impaired, however the anterior aspects were damaged only in the adolescent rats and the posterior aspects were damaged only in the adult rats. This study suggests that alcohol, and specifically binge drinking impacts brain structures in adolescents and adults differently. An fMRI Blood-Oxygen –Level- Dependent (BOLD) study of 18-25 year old alcohol dependent women showed that they had significantly less blood oxygen levels than controls in specific regions of the parietal and frontal lobes associated with spatial working memory function (Tapert et al., 2001). Those who had experienced the most episodes of withdrawal showed most impairment, suggesting that frequent binge and withdrawal episodes are most detrimental to brain function.

Deficits in cognition have also been identified in individuals who report frequent drinking episodes. Impairments in frontal lobe executive functioning are commonly reported in studies of alcoholic patients. Deficits in functions of planning, sustained attention and episodic memory were indicated in a study of young adult binge drinkers (Hartley, Elsabagh, & File, 2004). Difficulties in inhibiting impulsive responses were experienced in a sample of young adult female binge drinkers (Townshend & Duka, 2005). Poor inhibitory control has also been identified in adolescent (Soloff, Lynch, & Moss, 2000) young adult (Giancola, Zeichner, Yarnell, & Dickson, 1996; Noel, Bechara, Dan, Hanak, & Verbanck, 2007) and older adult (Hildebrandt, Eling, Brokate, & Lanz, 2004) long term heavy drinkers. Townshend and Duka (2005) showed that female binge drinking young adults performed poorly on tasks of spatial working memory. Working memory impairments have also been demonstrated in alcohol dependent adults (Ambrose, Bowden, & Whelan, 2001; Noel et al., 2007; Sullivan, Fama, Rosenbloom, & Pfefferbaum, 2002). Adolescent alcohol abuse has been associated with greater attentional deficits than controls (Tapert & Brown, 2000).

Memory and learning impairments are possibly the most reported cognitive effects of alcohol use (Birnbaum, Parker, Hartley, & Noble, 1978; Lister, Gorenstein, Risher-Flowers, Weingartner, & Eckardt, 1991). Retrieval deficits in immediate and delayed conditions were noted in a sample of adolescents with long term heavy alcohol use histories, visuospatial function was also deficient however this was associated with history of withdrawals (Brown, Tapert, Granholm, & Delis, 2000). Poor visuospatial function was however related to alcohol use in a sample of adult alcoholic women (Sullivan et al., 2002). Although the literature of adolescents with issues of alcohol abuse is quite limited, it can be seen it what is available that they are at great risk for structural and functional impairments of the brain.

#### *1.14.3 Methamphetamine use and cognitive function*

In Australia, and other parts of the world methamphetamine has become an increasingly popular drug since the beginning of its manufacture in the 1990's (Australian Institute of Health and Welfare, 2007; Brecht, O'Brien, von Mayrhauser, & Anglin, 2004). Methamphetamine use has been identified in various populations, with some using the substance recreationally whilst others have developed a more chronic abuse of the drug (National Drug Law Enforcement Research Fund, 2004). It was reported that methamphetamine was the second most common used drug type after cannabis in Australia (National Drug Law Enforcement Research Fund, 2004). Methamphetamine comes in three forms including powder (Speed), methamphetamine base (Base) and crystalline methamphetamine (Crystal or Ice) (Australian Institute of Health and Welfare, 2007). Use is most common in the young adult (20-29 year) age group, however up to eight to ten percent of adolescents have tried the drug between 16-17 years of age (National Drug Law Enforcement Research Fund, 2004). Experiences of violence and childhood maltreatment have been identified in methamphetamine users, in one study, 57.6 percent of female and 15.7 percent of male methamphetamine users reported a history of sexual abuse and domestic violence (J. B. Cohen et al., 2003).

Methamphetamine has more pronounced effects on the central nervous system than its predecessor amphetamine (National Institute on Drug Abuse, 2006). At the pharmacological level, methamphetamine blocks the transportation of dopamine across synapses (Volkow, Chan et al., 2001). The basal ganglia have the highest

proportion of dopaminergic neurons putting them at greatest risk for damage in the presence of methamphetamine (Chang, Ernst, Speck, & Grob, 2005). The neurotoxic effects of methamphetamine have been attributed to the significant loss of dopamine transporters in specific areas of the brain such as the caudate, putamen (McCann et al., 1998; Volkow, Chang et al., 2001; J. M. Wilson et al., 1996), nucleus accumbens and prefrontal cortex (Sekine et al., 2001; Sekine et al., 2003; J. M. Wilson et al., 1996). Significant reductions in serotonin transporter density have also been indicated in various brain regions of methamphetamine users including midbrain, thalamus, caudate, putamen, amygdala, anterior cingulate, frontal cortex and cerebellum (Sekine et al., 2006).

Methamphetamine users also exhibit cerebral glucose metabolism abnormalities in various regions of the brain, including the orbitofrontal cortex, cingulate, amygdala, striatum and cerebellum (Kim et al., 2005; London et al., 2004). Similarly, perfusion MRI has shown decreased regional cerebral blood flow in the basal ganglia and the right parietal lobes of methamphetamine abstinent users, however they also demonstrated increased cerebral blood flow in the temporoparietal and occipital regions in comparison to controls (Chang et al., 2002). The brain metabolite *N*-acetyl-aspartate, which is a marker of neuronal viability was significantly reduced in the cingula of recently abstinent methamphetamine users, indicative of neuronal damage within this region (Nordahl et al., 2002).

Thompson et al (2004) conducted a MRI study of 22 young adult methamphetamine users. The results showed extensive grey matter reduction in the cingulate, limbic and para- limbic cortices and significant increases in white matter. Methamphetamine users also had 7.8 percent smaller hippocampal volumes than controls and this reduction was associated with poor performance on memory tasks. The authors suggested that methamphetamine may alter neuronal activity leading to cell death, furthermore this neuronal damage may modify patterns of myelination and gliosis, resulting in the white matter hypertrophy indicated in methamphetamine users.

Cognitive deficits associated with affected brain regions of methamphetamine users have been reported. As the neurotoxic actions of methamphetamine have been

localised to the fronto-temporal regions, associated deficits in learning (Woods et al., 2005), verbal memory and motor functions have been identified (Volkow, Chan et al., 2001; Volkow, Chang et al., 2001). Impaired frontally based functions such as working memory, speed of processing (Chang et al., 2002), inhibitory control (Semple, Zians, Grant, & Patterson, 2005) abstract reasoning and cognitive shifting and flexibility (Kim et al., 2005) have also been demonstrated in methamphetamine users. Providing further evidence of frontal dysfunction, Paulus et al (2002) conducted a fMRI study which showed limited activation of prefrontal areas whilst completing a decision making task in methamphetamine dependent individuals. A study investigating the effects of methamphetamine use on a range of cognitive domains in young adult methamphetamine users showed deficits in memory, processing speed, inhibitory control and cognitive shifting and flexibility (Simon et al., 2000). Similar impairments were reported by Kalechstein, Newton, & Green (2003), indicating poor performances on tasks of attention, processing speed, learning and memory and executive verbal fluency tasks in methamphetamine dependent participants. These findings indicate that the neurotoxicity of methamphetamine results in altered brain structure and impaired functionality which are represented by the experiences of cognitive difficulties manifested by methamphetamine users.

#### *1.14.4 Polysubstance abuse and cognitive function*

Polysubstance abuse is defined as the use of two or more drugs simultaneously over an extended period of time. Studies have shown that engaging in alcohol and cannabis use puts individuals at greater risk for experimentation with harder drugs and polysubstance abuse (Brecht et al., 2004; Fergusson & Horwood, 2000; Fergusson et al., 2002; W. Hall & Solowij, 1998; Hawkins, Catalano, & Miller, 1992; Kandel, 2003; Yen, Hsu, & Cheng, 2007). Martin, Clifford, Maisto, Earleywine, Kirisci, & Longabaugh (1996) suggested that drug users commonly take a number of substances simultaneously. In Australia, it has been shown that adolescents aged 12-17 years frequently engage in multiple substance abuse (Australian Institute of Health and Welfare, 2007). Merril, Kleber, Schwartz, Liu and Lewis (1999) investigated the precursors to polydrug use in an adolescent sample. It was found that early alcohol and cigarette use was significantly associated with later cannabis use, and cannabis use was associated with abuse of other illicit drugs. Martin, Kaczynski, Maisto and

Tarter (1996) suggested that during adolescence, the frequency and extent of poly drug use tended to increase with age and was also associated with poor behavioural regulation and increased negative affect. Other studies have also shown that a high proportion of adolescent polysubstance abusers exhibit comorbid disruptive behavioural disorders and/or psychological disorders including depression, anxiety, PTSD and bipolar disorder (Schettini-Evans, Spirito, Celio, Dyl, & Hunt, 2007; Wilens, Biederman, Abrantes, & Spencer, 1997). This indicates that adolescents with impaired self regulatory behaviours and experiences of traumatic events have a greater susceptibility to polysubstance abuse. These adolescents may use various substances as a means to cope with emotional and behavioural problems (Labouvie, 1986; Rosselli & Ardila, 1998). Victims of childhood sexual abuse who demonstrated PTSD symptoms were shown to engage in polysubstance abuse, providing further evidence that a range of substances may be used for regulating emotional disturbances (Ullman, Townsend, Starzynski, & Long, 2006).

Research into the brain changes associated with polysubstance abuse is limited, and even more so on adolescent populations. Changes in white matter have been reported in individuals who use multiple substances; one study found that concurrent alcohol and cocaine abusers had less prefrontal white matter, particularly in the anterior cingulate (J. O'Neill, Cardenas, & Meyerhoff, 2001). Phospholipid metabolites of the white matter were significantly reduced in a sample of polysubstance abusers, indicating that using multiple substances alters phospholipid metabolism in white matter tracts (MacKay, Meyerhoff, Dillon, Weiner, & Fein, 1993). Similar metabolic alterations were shown in a sample of nine adult polysubstance users, furthermore decreased cerebral perfusion was also indicated in a high proportion of these individuals (Christensen et al., 1996). An MRI study examining the brain effects of polysubstance abuse found that users had significantly smaller pre frontal volumes than the controls (X. Liu, Matochik, Cadet, & London, 1998). However, these changes were attributed to smaller volumes of grey but not white matter as suggested by other studies. Electrophysiological changes have also been identified in polysubstance abusers, many have demonstrated abnormal electroencephalograph (EEG) patterns relative to controls (Roemer, Cornwell, Dewart, Jackson, & Ercegovac, 1995). This study also conducted quantitative

electroencephalograph (QEEG) analyses, indicating that polysubstance users have overall reduced interhemispheric coherence and greater right > left asymmetry of power in the frontal region of the brain. Medina et al (2007) examined the effects of polysubstance abuse on the brains of adolescents. They indicated that comorbid cannabis and alcohol use was not associated with gross structural changes in the brains of adolescents, however it was suggested that opposing effects of cannabis and alcohol may contribute to microstructural changes in the brain that are not identifiable using MRI.

It could be hypothesised that the impact of polysubstance abuse on cognitive functioning would be far reaching, given that different substances are affecting different aspects of the brain (Rogers & Robbins, 2001). Some have also suggested that polysubstance abusers demonstrate greater severity of impairments (Selby & Azrin, 1998) that are long-lasting and do not appear to show evidence of recovery even after one year of abstinence (Medina, Shear, Schafer, Armstrong, & Dyer, 2004).

The frontal lobes have been identified as particularly sensitive to the effects of substance abuse (Lyvers, 2000; Rogers & Robbins, 2001). Deficits in inhibitory control and other executive functions mediated by the prefrontal cortex were demonstrated in a sample of 35 substance abusing adults (Verdejo-García, Bechara, Recknor, & Perez-Garcia, 2006). Selby and Azrin (1998) found that polysubstance abusing men showed the greatest impairments in all aspects of neuropsychological functioning examined, including memory, visuo-motor functioning and executive function when compared to controls, cocaine-only abusers and alcohol-only abusers. Another study of polysubstance abusing men showed that impairments in recall memory were more severe than those experienced by alcohol dependent men (Bondi, Drake, & Grant, 1998).

A study examining memory functioning in a sample of polysubstance dependent women showed significantly poorer verbal learning ability, further analysis showed frequency of alcohol and cocaine use was also associated with impaired delayed recall and recognition ability (Medina, Shear, & Schafer, 2006). Reaction time, visuo-perceptual abilities (Nixon, Paul, & Phillips, 1998), memory, abstract reasoning (Rosselli & Ardila, 1998; Schrimsher, Parker, & Burke, 2007) and learning



(Gonzalez et al., 2004) were most deficient in adult multiple substance users when compared to individuals who abused single substances and controls. A study examining the differences in cognitive functioning between alcoholics and polysubstance abusers found that both groups performed at the same level on measures of visuospatial function, construction, learning and memory (Beatty, Blanco, Hames, & Nixon, 1997). Functioning was impaired on both groups, however the expected additive effects of abusing other substances were not apparent. Similar results were reported in relation to cannabis and Methylenedioxymethamphetamine (MDMA) abuse, where it was found that the affects of cannabis use alone showed impairments in learning and memory, however evidence of additional effects of MDMA in those that used both substances were not identified (Dafters, Hoshi, & Talbot, 2004). On the contrary, Croft, Mackay, Mills and Gruzelier (2001) showed that concurrent MDMA and cannabis users performed more poorly on measures of memory, learning, verbal fluency, processing speed and motor co-ordination than controls and cannabis only users.

A multiple regression analysis of a sample of polysubstance abusers investigated which substances predicted poorer performances on executive related processes of working memory, inhibitory control, cognitive flexibility and abstract reasoning (Verdejo-García, López-Torrecillas, Aguilar de Arcos, & Pérez-García, 2005). It was found that different substances contributed to performances on different components of executive functioning where MDMA contributed to poor performance on tasks of working memory and abstract reasoning, cocaine contributed to inhibitory control performance, whilst cannabis influenced performance on the cognitive flexibility component of executive functioning. A similar study using fMRI to assess brain activation during cognition of MDMA users and the contributions of other drugs to performances on tasks of working memory, attention and associative memory were examined (Jager et al., 2008). It was suggested that polysubstance use was related to poor associative memory performance, however no effects on working memory and attention were indicated by MDMA users. Multiple regression analysis showed that amphetamine had a greater influence on associative memory performance than MDMA. Both drugs did impact upon associative memory related brain functioning, however deficits were identified in different aspects of the brain associated with this

function. The isolated effects of individual drugs on different mechanisms and pathways within the brain may be one explanation for these results.

Schweinsburg, Schweinsburg, Cheung, Brown, Brown, and Tapert (2005) conducted a fMRI study of adolescents with issues of polysubstance abuse (cannabis and alcohol) and alcohol alone abuse. It was found that polysubstance abusers showed different brain response abnormalities than alcohol alone abusers, presenting with less activation of the inferior frontal and temporal regions, and more activation in other prefrontal areas in response to a spatial working memory task. Neuropsychologically, adolescents with heavy polysubstance abuse practices showed deficits in tasks of attention that persisted at four year follow up (Tapert & Brown, 1999). This study also identified impairments in visuospatial function, however this was attributed to severity of withdrawal experiences rather than the actions of the drugs on their own. Similarly, Tapert, Granholm, Leedy, and Brown (2002) followed up the neuropsychological functioning of polysubstance abusing adolescents after eight years and found learning, retention and attentional difficulties that were associated to the effects of heavy substance abuse. In agreement with the previous study by Tapert and Brown (1999) experiences of withdrawal were related to poorer functioning on tasks of visuospatial functioning.

These findings suggest that engaging in polysubstance abuse relates to brain and cognitive impairments within a number of different areas. Combinations of different substances relate to variable effects making this group of substance users most vulnerable to a range of deficiencies. The minimal adolescent literature has also identified impairments associated with polysubstance abuse similar to those in adults, which appear to persist over time.

#### *1.14.5 Prenatal drug exposure and cognitive function*

There is a large body of literature to suggest that children exposed to alcohol and illicit drugs whilst in-utero show evidence of impaired cognitive functioning. Given that a large proportion of children who are maltreated have also been subject to prenatal drug exposure, it is important to examine how these experiences may possibly contribute to the cognitive functioning of abused children and adolescents.

Research has implicated the role of a dysregulated stress response system in children prenatally exposed to cocaine and other drugs. The finding that infants exposed to cocaine prenatally showed elevated cortisol reactivity lends support to this theory (Eiden, Veira, & Granger, 2009). It has been suggested that prenatal exposure to cocaine, and commonly other substances, alters arousal and regulatory systems that directly relate to prefrontal information processing systems (Mayes, 2002). Similarly, children with Foetal Alcohol Spectrum disorders indicated prefrontal impairments as demonstrated by poor performances on measures of verbal executive functioning (Rasmussen & Bisanz, 2009). However, it was also indicated that a number of other factors influence this system, exacerbating potential effects on information processing capacity. Many of these factors were environmental, such as postnatal home conditions and parental functioning and maternal factors such as age, education and pregnancy history (Mayes, 2002).

Studies have consistently reported that children exposed to substances prior to birth show lower scores on measures of overall cognitive functioning (Alessandri, Bendersky, & Lewis, 1998; Behnke, Eyler, Garvan, & Hou, 2002; Huizink & Mulder, 2006; Jacobson, Jacobson, Sokol, Chiodo, & Corobana, 2004; Mattson & Riley, 1998; L. Singer et al., 1997; L. T. Singer et al., 2002; L. T. Singer et al., 2004). These children have also been shown to exhibit motor deficits and visuospatial impairment across a range of studies (Behnke et al., 2002; Chang et al., 2005; Frank, Augustyn, Knight, Pell, & Zuckerman, 2001; Mattson & Riley, 1998; L. Singer et al., 1997). Impaired attention and executive function have been indicated in children prenatally exposed to substances, where specific deficits in selective and sustained attention, problem solving and poor inhibitory control were commonly reported (Azuma & Chasnoff, 1993; Chang et al., 2004; Frank et al., 2001; P. A. Fried & Smith, 2001; Mattson & Riley, 1998; Noland et al., 2005). Bandstra et al (2002) found that children exposed to cocaine before birth showed persisting language deficits from ages three to seven years. Memory and learning impairments have also been reported in this population (Chang et al., 2002; Mattson & Riley, 1998). Others have shown that children showed limited information processing capacity and speed at seven and a half years of age (Burden, Jacobson, & Jacobson, 2005; Jacobson, Jacobson, Sokol, Martier, & Ager, 1993). At the brain structural level, brain imaging of three to 16 year

old children indicated that prenatal exposure to substances related to smaller subcortical volumes, particularly in structures of the basal ganglia and limbic system (Chang et al., 2004).

These cognitive and brain impairments have also been shown to coincide with behavioural problems such as externalising behaviours, high distractibility, poor emotional regulation, poor academic functioning and a greater susceptibility to developmental disorders such as ADHD (Frank et al., 2001; Huizink & Mulder, 2006; Jacobson et al., 2004; L. T. Singer et al., 2004).

Collectively, these studies show that children and adolescents that have been prenatally exposed to abused substances are at great risk of developing a range of neuropsychological impairments. However, it has been strongly suggested that the postnatal environment bears great influence on developmental outcomes in these children (Jacobson et al., 2004; L. T. Singer et al., 2004). Therefore, it could be argued that children and adolescents subject to maltreatment, who also have a history of prenatal drug exposure, are a cohort of children who have compounding factors likely to result in severe cognitive impairments.

### *1.15 Psychopathology and Cognitive Function*

Experience of maltreatment puts individuals at great risk of developing psychological and psychiatric disorders. Most commonly reported psychopathologies related to maltreatment include, depression, anxiety (De Bellis, Broussard et al., 2001; McCloskey et al., 1995; Silverman et al., 1996), post traumatic stress disorder (Barrett et al., 1996; Beers & De Bellis, 2002; Cahill et al., 1999; De Bellis & Putnam, 1994; Glaser, 2000; McCloskey et al., 1995; Silverman et al., 1996; van der Kolk, 2003) and borderline personality disorder (Grilo et al., 1999; Milburn et al., 2008). It has been identified within the literature that particular psychopathologies are related to cognitive deficits.

#### *1.15.1 Depression and cognitive function*

According to the DSM-IV-TR (American Psychiatric Association, 2000) some of the key symptoms of depression include low mood and lack of motivation to

engage in activities previously performed, including those that were found pleasurable. With these symptoms in mind, it may be considered a condition relevant only to emotional functioning, however the literature would suggest that diagnosed individuals also experience a number of cognitive deficits (R. L. Levin, Heller, Mohanty, Herrington, & Miller, 2007). Like other neurological disorders, depression has been defined in terms of a distinctive profile of cognitive deficits alongside its emotional symptomatology (Austin, Mitchell, & Goodwin, 2001).

The hippocampus has been implicated in the pathogenesis of depression and has also been thought to be responsible for the pattern of cognitive impairments associated with the disorder (S. Becker & Wojtowicz, 2006). Depression commonly co-occurs with anxiety, a condition of chronic stress (Hankin, Abramson, Miller, & Haefl, 2004). As the experience of stress has been proposed as a possible contributing factor to the development of depressive illnesses, similar to literature previously presented, the high levels of glucocorticoids resultant of stress may be responsible for altered hippocampal morphology in these patients (S. Becker & Wojtowicz, 2006). Recent animal studies have shown that neurogenesis does not end at childhood, but continues on into adulthood in specific regions of the brain including the dentate gyrus of the hippocampus (Yoshida, Hashizume, & Tanaka, 2004). It has been suggested that this ongoing neurogenesis within the dentate gyrus is related to the neurobiological processes underlying learning and memory (Warner-Schmidt, Madsen, & Duman, 2008).

Becker & Wojtowicz (2006) proposed that there was a lack of neurogenesis in the hippocampi of depressed individuals. This was evidenced by the reduction of hippocampal size in depressed patients and its relationship to duration of depressive illness, functionally, these individuals also performed poorly on tests of recollection memory (MacQueen et al., 2003) and spatial memory (N. F. Gould et al., 2007). A recent review by Levin et al (2007) indicated that cognitive skills mediated by the prefrontal cortex, including memory, attention and executive functioning were also deficient in clinically depressed individuals. After completing an extensive cognitive battery a group of unmedicated individuals with major depressive disorder showed impairments in memory, executive functioning and decision making (Taylor- Tavares

et al., 2007). Specific difficulties were indicated in skills of spatial working memory, set shifting, inhibitory control and decision making.

#### *1.15.2 Posttraumatic stress disorder and cognitive function*

Multiple experiences of violence and physical and/or sexual abuse in childhood are major risk factors for the development of posttraumatic stress disorder (PTSD) (Briggs & Joyce, 1997; R. D. Duncan, Saunders, Kilpatrick, Hanson, & Resnick, 1996; MacMillan et al., 2001; Triffleman, Marmar, Delucchi, & Ronfeldt, 1995). Childhood sexual abuse appears to be strongly associated with the experience of PTSD in adulthood, and the severity of the abusive experiences are related to the severity of symptoms (Briggs & Joyce, 1997). As its name would suggest, the onset of PTSD is usually preceded by a specific traumatic event (such as a car accident), or accumulation of events (such as repeated episodes of maltreatment over time). The symptomatology of PTSD is organized under three symptom domains in the DSM-IV-TR. A person is clinically diagnosed with PTSD, if they experience one or more symptoms as classified in three specific symptom domains, three further domains have also been described, although an individual does not need to experience symptoms that fall within those categories to be diagnosed with PTSD. To be diagnosed with PTSD, individuals must experience at least one symptom that falls under the intrusive recollection domain, three symptoms in the avoidant/numbing domain and two symptoms in the hyper- arousal domain (American Psychiatric Association, 2000). The three other associated criteria relate to the nature of the stressor itself, the duration of the symptoms and the impact of those experiences on the daily life of the individual (American Psychiatric Association, 2000).

The severe episodes of anxiety and stress associated with PTSD have been associated with biological changes in the hypothalamic-pituitary-adrenal (HPA) axis which is responsible for mobilizing an individual when facing a stressful event (Sapolsky, 1996; Sapolsky et al., 1990). Research has shown that individuals diagnosed with PTSD have higher cortisol levels associated with maladaptive functioning of the HPA axis (Lindauer, Olff, van Meijel, Carlier, & Gersons, 2006). Altered cortisol levels have also been demonstrated in children with experiences of trauma and childhood abuse (Bevans, Cerbone, & Overstreet, 2008; Bruce, Fisher, Pears, & Levine, 2009) As presented previously, the hippocampus is a region with

high concentrations of glucocorticoid receptors , (Packan & Sapolsky, 1990; Sapolsky et al., 1985; Sapolsky et al., 1990; Starkman et al., 1992; Uno et al., 1989). Enhanced stress responses associated with PTSD symptoms may correspond with excessive release of glucocorticoids (Sapolsky et al., 1990), glutamate (Moghaddam, 2002), corticotrophin releasing hormone (Brunsen, Eghbal-Ahmadi, Bender, Chen, & Baram, 2001), decreased neurogenesis (E. Gould, McEwen, Tanapat, Galea, & Fuchs, 1997), impaired long term potentiation (C. Li, Maier, Cross, Doherty, & Christian, 2005) and inhibition of brain derived neurotrophic factor (Duric & McCarson, 2005). This suggests that the hippocampus of PTSD patients may be at risk of damage due to these neurobiological changes.

In one study police officers with PTSD, demonstrated smaller hippocampi and higher morning salivary cortisol levels (Lindauer et al., 2006). Magnetic Resonance Imaging research of Vietnam combat war veterans with PTSD has consistently reported reduced hippocampal size in these individuals (Bremner, Randall, Scott, Bronen et al., 1995; Gilbertson et al., 2002; Gurvits et al., 1996). Similar deficiencies in hippocampal size have also been reported in adults with histories of child abuse and female victims of intimate partner violence (Bremner et al., 1997; Bremner et al., 2003; Notestine, Stein, Kennedy, Archibald, & Jernigan, 2002). Contrastingly, children with abuse related PTSD showed no changes in hippocampal size (De Bellis, Hall, Boring, Frustaci, & Moritz, 2001), however it could be suggested that the pathophysiology of PTSD may correspond with structural changes in the brain over a longer period of time. Furthermore, De Bellis, Keshavan, Spencer, and Hall (2000) also indicated that children with PTSD showed no changes in hippocampal size, however decreased of N-acetyl-aspartate was found in the medial frontal cortex, suggestive of reduced neuronal functionality within this region. Some studies of adults with PTSD related to child abuse history and other trauma also showed no changes in hippocampal size (Jatzko et al., 2006; Pederson et al., 2004), however these findings put into question the influence of age at time of trauma, severity of trauma, use of psychotropic medication and frequency of trauma episodes.

Smaller hippocampal volumes (Bremner et al., 1993; Johnsen & Asbjornsen, 2009; Johnsen, Kanagaratnam, & Asbjornsen, 2008; Lindauer et al., 2006; Tischler et al., 2006; Yehuda et al., 1995b) and altered hippocampal function (Werner et al.,

2009) in PTSD patients has been associated with performances on tasks of verbal learning and memory suggesting that PTSD puts individuals at risk of developing neuropsychological deficits.

United Nations war veterans with PTSD demonstrated similar deficits, indicating poor performances on tasks of figural and logical memory, and immediate and delayed recall of verbal information (Geuze, Vermetten, de Kloet, Hijman, & Westenberg, 2009). These deficits were also shown to be associated with poor social and occupational functioning. Jelinek et al (2006) found verbal and visual memory deficits in a mixed sample of PTSD patients who had experienced a range of different types of trauma. Holocaust survivors with PTSD have also demonstrated deficits in verbal learning, increased age was associated with more severe deficits suggesting that PTSD may accelerate cognitive degeneration (Yehuda, Golier, Halligan, & Harvey, 2004). A meta-analysis of 28 studies demonstrated that verbal memory deficits were consistently found in patients with PTSD (Jenkins, Langlais, Delis, & Cohen, 1998; Johnsen & Asbjornsen, 2008). These deficits have also been described in PTSD patients with histories of abuse (Bremner, Randall, Scott, Capelli et al., 1995; Bremner, Vermetten, Afzal, & Vythilingam, 2004). Whilst others found no evidence of deficits in a similar sample of women (Stein, Hanna, Vaerum, & Koverola, 1999).

Other studies have found deficits that extend into other domains of cognitive function. Gilbertson, Gurvits, Lasko, Orr, and Pitman,(2001) found that combat veterans with PTSD experienced deficits across three major domains of cognitive function including, memory and learning, attention and executive function. Specifically, immediate and delayed memory, attention span, set-shifting, and cognitive flexibility were impaired. Others have also found multiple neuropsychological deficits in PTSD patients including; learning, memory, attention and working memory (Vasterling, Brailey, Constans, & Sutker, 1998; Veltmeyer et al., 2005), verbal memory, attention and processing speed (Samuelson et al., 2006; Stein, Kennedy, & Twamley, 2002), attention, memory, learning, set shifting and cognitive flexibility (Koenen et al., 2001; Koso & Hansen, 2006).



There is strong evidence to support that PTSD patients experience cognitive dysfunction, particularly in the learning and memory domain, however some have suggested that low premorbid functioning may put individuals at greater risk for developing PTSD (Buckley, Blanchard, & Neill, 2000; Parslow & Jorm, 2007).

### *1.16 Research Design and Methodological Issues*

Examining groups of children with histories of maltreatment raises a number of significant methodological issues that need to be considered in study design. The degree of variability in maltreatment experiences is reflected in the difficulties of quantifying such experiences for the purposes of research. It has been described previously in this thesis that the identification of a single abuse type is problematic as many of these children experience more than one type of abuse, and emotional abuse in particular has been said to occur alongside all other abuse types (Claussen & Crittenden, 1991; Frederico et al., 2005; Milburn et al., 2008).

#### *1.16.1 Severity of maltreatment*

Severity of maltreatment is another area of ambiguity in the literature, as there is not a generally accepted standard for rating severity (Kinard, 1994; Porter et al., 2005). Some have even suggested that severity has been defined in a somewhat “ad hoc fashion” and is often based on arbitrary groupings of experiences (Chaffin, Wherry, Newlin, Crutchfield, & Dykman, 1997, p. 570). As a result, the differential impact of abuse severity on cognitive function amongst various research samples of this kind has been difficult to ascertain.

A small number of studies that have included a measure of severity have shown significant relationships between maltreatment severity and cognitive impairment (Carrey et al., 1995; Palmer et al., 1999), whilst others have not (Porter et al., 2005). Carrey et al (1995) examined severity in terms of 2 categories, where greater severity was associated with greater degrees of harm. For example, those who had experienced penetration during episodes of sexual abuse were assigned to the greater severity category, whilst those who reported experiences of fondling with no penetration were assigned to the lower severity category. The same method was used

as a measure of severity for the all four abuse types. Others have used a standardised measure, known as the Wolfe's History of Victimization Form (HVF) to report abuse related variables, where legal guardians, and/or the young person's therapist were asked to estimate the number of abuse episodes, abuse duration, perpetrator-child relationship and the type of force used (Palmer et al., 1999; Porter et al., 2005). Another international standardised measure of maltreatment type and severity has been developed by Barnett and Cicchetti (1993). This measure, known as the Maltreatment Classification and Rating System (Barnett & Cicchetti, 1993) accounts for both maltreatment type and severity. Ratings for each maltreatment type (e.g. Physical abuse) are devised using a six-point Likert scale, scores start from zero, representing no evidence of abuse type, to five, representing severe level of abuse type. Under each maltreatment type, descriptions of abuse experiences representing a particular severity score are provided.

For the purposes of the current study a similar Australian measure of maltreatment type and severity was used. At the local level, academics and workers in child protection have attempted to tackle the issue of measuring maltreatment severity. As a result, the Take Two Harm Consequences Assessment Referral Tool (T2 HCA) was devised (S. Thomas et al., 2004). All Victorian Department of Human Services child protection clients have a T2 HCA prior to being referred to the Take Two program. When compared to the Maltreatment Classification Rating System (Barnett & Cicchetti, 1993), the T2 HCA provides more detailed descriptions of maltreatment experiences considered under each of the severity rankings (Extreme, Serious and Concerning). However, similar to other severity measures, the T2 HCA is limited by its reliance on the child protective workers knowledge of the client.

Victims of child abuse with experiences of multiple out-of-home placements have been identified as a group at risk for greater neuropsychological deficit (Carrey et al., 1995; Goodman, 1996; Pears & Fisher, 2005). It has been suggested that being placed in out-of-home care and number of out-of-home placements may be an indicator of greater severity of abuse when comparing studies with victims of abuse without placement experiences (Porter et al., 2005). Studies that haven't indicated severity factors have shown mixed results, with some finding significant effects of

abuse severity on cognitive measures (Beers & De Bellis, 2002), whilst others have indicated no such effects (Samet, 1997).

#### *1.16.2 Age of maltreatment onset and duration of maltreatment*

Age of maltreatment onset, maltreatment duration and frequency of maltreatment experiences are also factors that may have considerable influence on how an individual functions as a consequence of altered development (Briere, 1992; Romano & De Luca, 2001). Contrary to expectation, in the Porter et al (2005) study variables of abuse duration and frequency as measured by the HVF, were not related to cognitive performance. Abuse onset was recorded as starting below age five or starting after age five, no significant differences were found in terms of cognitive functioning in relation to age of abuse onset in this study.

Duration or age of maltreatment onset also did not appear to be related to the cognitive variables in Carrey et al's (1995) research, where onset was recorded as either before or after seven years of age and duration was indicated as less than or more than one year. Contrastingly, others have shown that maltreatment duration was associated with lower FSIQ in a sample of participants with maltreatment related PTSD (De Bellis, Keshavan et al., 1999). Similarly, children exposed to frequent episodes of interpersonal violence and other types of maltreatment were shown to have significantly poorer capacities of overall cognitive function and language skills (Saltzman, Weems, & Carrion, 2006). Information regarding, abuse onset, frequency and duration could not be ascertained for a large proportion of participants in the Mezzacappa et al (2001) report, as a result, the authors of this study could not examine the influence of these variables in the final analysis.

#### *1.16.3 Determining developmental and medical history*

Information regarding developmental and abuse history is often difficult to ascertain, as children with these experiences are often subject to disorganised and transient lifestyles (Dunlap, Golub, Johnson, & Wesley, 2002). As a consequence records of medical history and notifications of maltreatment are incomplete or absent. These young persons are frequently subject to different caregivers, particularly if they have been placed in out of the home (James, Landsverk, & Slymen, 2004; Pears & Fisher, 2005). Young persons with maltreatment histories commonly have parents

who are not contactable, and those who are, have incomplete accounts of their child's developmental and medical history. Recall of such events is difficult as these parents are more likely to experience intermittent episodes of domestic violence, substance abuse and emotional disturbance (Dunlap et al., 2002; Widom, 1989; Wolfe, 1985). Records kept by child protection are often variable in these aspects (Munro, 1998). This may be a consequence of the legal priorities associated with child protection. The high rate of staff turnover in child protection agencies is also an issue, as certain information gets lost or isn't transferred when handover to a new case manager or child welfare agency occurs (National Council on Crime and Delinquency, 2006; Sloper, 2004).

These issues are also relevant to obtaining information regarding substance use. Current substance abuse and substance abuse history are important to consider as these issues have been associated with maltreated populations, and it is well known that substance abuse affects brain structure and cognitive function. Key studies examining the influence of maltreatment history on cognitive function in children and adolescents, failed to report measures of substance abuse in their methodologies (Mezzacappa et al., 2001; Porter et al., 2005). Although, Porter et al did exclude those with histories of prenatal drug exposure from their study, information regarding current or previous drug abuse in the participants themselves was not indicated. Experiences of current or previous substance abuse issues were part of the exclusionary criteria in other studies of this type (2002; Navalta, Polcari, Webster, Boghossian, & Teicher, 2006).

Research consisting of adolescent samples requiring details regarding substance use has typically used self report questionnaires to gauge this information (Newcomb, Maddahian, & Bentler, 1986). It has been suggested that interview data regarding this type of information has limited validity, particularly if details required are highly sensitive and relate to issues of legality (Turner et al., 1998). Whilst others have recommended conducting face to face interviews with the adolescent and the caregiver to obtain detail regarding such issues (Weissman et al., 1987). A sample of adolescent and young adult participants, in a recent study, reportedly provided limited information regarding experiences of drug use on interview, as they had indefinite recollections of these experiences (Baliz, 2008). Although the impracticalities

associated with toxicological avenues to obtain drug related information were considered, it was recommended by Baliz that attempting such procedures would provide an objective assessment of current drug use. However, toxicological data also has its limitations, as it cannot provide indications of previously abused substances.

#### *1.16.4 Full Scale IQ- Matching variable or dependent variable?*

In neuropsychological research it is conventional practice to covary for demographic variables that significantly differ between groups. Covarying for full scale IQ is also common in neuropsychological studies where FSIQ is significantly different between groups. However, it is questionable whether such practices are appropriate, particularly when the groups differ considerably in relation to the independent variable. Examples of research in this area that have covaried for IQ have indicated varying results.

In the Porter et al (2005) study, significant differences between the abused and non abused groups were found on measures of learning and memory, whilst controlling for socioeconomic status. However, given that the two groups also differed on FSIQ, further analysis was undertaken, adding FSIQ as second covariate. The result of this was that the differences between the two groups on the cognitive measures were no longer significant. By contrast, Mezzacapa et al (2001) reported significant differences between abused and non abused groups on measures of executive function remained significant after covarying for FSIQ. Others that have found significant differences in FSIQ between maltreated and non maltreated groups, have treated FSIQ as a dependent variable suggesting that maltreatment history has a significant effect on FSIQ (De Bellis et al., 2009; Pears & Fisher, 2005; Perez & Widom, 1994). Porter et al (2005) also made comment that the differences in FSIQ between abused and non abused samples may be attributable to the effects of adverse life events such as trauma. Dennis et al (2009) pointed out that it was imperative that researchers considered environmental variables that were likely to be related with pre-existing group differences. Therefore, it could be argued that experience of trauma is a likely explanation for group differences in FSIQ in situations where groups are matched on other demographic variables such as age, gender and SES.

Some have suggested that practices of covarying for variables is problematic when there are significant differences between groups on these variables (Adams, Brown, & Grant, 1985, 1992), particularly when this is an attempt to produce statistical equivalence of groups that are significantly different from each other (Briere, 1992). In a very recent review of this issue, examples of research into childhood neurodevelopmental disorders were used to highlight the problem of covarying for FSIQ (Dennis et al., 2009). It was argued that differences on FSIQ between children with neurodevelopmental disorders and controls, were not related to sampling issues (where adjustments made by covariates would be appropriate) but pre-existing differences between the groups, not under experimenter control. It does not make theoretical sense to treat FSIQ as a covariate in research designs examining outcomes on other cognitive measures. The skills underlying performance on FSIQ reflect those of the cognitive measures serving as dependent variables, therefore controlling for IQ removes variability on the cognitive measures that are explained by the covariate itself (Dennis et al., 2009). It would seem illogical to suggest that a child's difficulty to take in and process information would not somehow be associated with performance on specific cognitive tasks which are actually dependent on these skills.

It has been stated that “there is simply no logical statistical procedure that can be counted on to make proper allowances for uncontrolled pre-existing differences between groups” (Lord, 1967, p. 305). Adjusting for covariates can be suitable in specific research designs, however unsuitable applications of this analysis can lead to highly conservative and invalid interpretations (Briere, 1988).

### *1.17 Study Rationale*

This review of the literature strongly suggests that maltreated children are vulnerable to developing a broad range of abnormalities in brain structure, neural function and cognition. The implications of such deficits are potentially profound, influencing academic performance, adaptive behaviour and social functioning (Barnett, Vondra, & Shonk, 1996; Daignault & Hebert, 2009; Dodge-Reyome, 1993; Eckenrode, Laird, & Doris, 1993; Gregory & Beveridge, 1984; Kendall-Tackett &

Eckenrode, 1996; Kurtz, Gaudin, Wodarski, & Howing, 1993). Early detection of cognitive deficits in these children may inform the introduction of interventions that can minimize the impact on aspects of daily functioning, producing more favourable outcomes.

Given that the majority of research in this area makes use of global cognitive measures, it is difficult to ascertain specific areas of deficit that can be targeted for intervention. An overall score of IQ provides limited information regarding specific deficits in cognitive functioning and runs the risk of missing extremely important strengths and weaknesses. For example, a child achieving a score of 125 on Verbal IQ (VIQ) and a Performance IQ (PIQ) score of 75 on the Wechsler Intelligence Scale for Children, will still achieve a full scale IQ (FSIQ) score of around 100, interpreted as 'average' general cognitive functioning, even though functioning on performance measures falls on the borderline intellectually disabled range. The discrepancy in component scores is potentially highly salient for the individual child. It is also important to be able to statistically monitor when participants in such studies have significant abnormalities in affective functioning (such as depression and anxiety) so that the co-occurring deficits in affective and cognitive functioning can be documented.

Research specifically examining cognitive deficits in maltreated children and adolescents has been based on quite small sample sizes. Most have relied on maltreated samples of fewer than 25 participants (Beers & De Bellis, 2002; Carrey et al., 1995; Mezzacappa et al., 2001; Palmer et al., 1999; Porter et al., 2005). One study had a maltreated sample of 99 children, although they were a much younger sample aged three to six years (Pears & Fisher, 2005). De Bellis et al (2009) included a sample of 61 maltreated participants, however these were separated into two groups, where those with a history of maltreatment and diagnosed with PTSD ( $n=22$ ) were separated from those maltreated participants who did not have PTSD ( $n=39$ ). Specific types of abuse were investigated in these studies with some looking at sexual and physical abuse only (Carrey et al., 1995; Mezzacappa et al., 2001), others also included witnessing of domestic violence (Beers & De Bellis, 2002) whilst some only examined sexual abuse (Palmer et al., 1999; Porter et al., 2005) or cases of neglect (De Bellis et al., 2009). Pears and Fisher (2005) considered all four abuse types and

made reference to those who experienced multiple abuse types. Indicators of maltreatment severity and duration were variable across all the studies, and given the difficulty in obtaining accurate information regarding developmental history, many used simplistic measures of these factors (e.g. duration of abuse noted down as less than or more than one year). On this basis, the current study will attempt to utilise a larger cohort of maltreated children who fall at the most severe end of the spectrum and have experienced multiple abuse types.

These key studies showed evidence of impaired cognitive function in maltreated samples, though the range of deficits shown may be related to the issues surrounding determination of abuse history and other factors related to sampling method and research methodology. Carrey et al (1995) showed that the maltreated sample performed significantly more poorly on FSIQ and VIQ in comparison to a comparable control group, though measures of other cognitive domains were not included. Similar results of impaired FSIQ and VIQ performance in a sexually abused sample when compared to a control group have been shown, although additional measures of memory and learning did not show a significant difference (Palmer et al., 1999). Differences in FSIQ were reported by Pears and Fisher (2005), specific skills in visuospatial function and language were also shown to be deficient in the abused group in comparison to controls.

Another study looking at the relationship between abuse-related PTSD and cognitive function included a comparable control group on the basis of demographic variables as well as FSIQ. The results indicated that the abused-PTSD group performed significantly worse on measures of executive function, attention and verbal memory (Beers & De Bellis, 2002). A more recent study of neglected school children has shown that neglected participants, regardless of PTSD diagnosis performed significantly more poorly on measures of learning and memory, attention and executive function, visuospatial skills, language and academic achievement (De Bellis et al., 2009). In Mezzacappa et al's (2001) study there appeared to be a trend towards differences in FSIQ between the maltreated and control groups, even though these were not significant, the authors undertook measures to control for FSIQ differences between groups. However as noted in the previous section (1.16.4), it has been cogently argued that it is inappropriate to take such statistical measures. They reported



significant differences between the abused and control groups on measures of executive functioning, specifically those skills related to impulse control. The study was limited in that it only involved male participants, therefore it could not be ascertained whether females with histories of abuse showed similar deficits in impulse control. Measures to control for demographic variables including FSIQ and SES were also utilised in the Porter et al (2005) study, as a consequence, differences in measures of memory performance between the abused and control groups were no longer significant. When FSIQ and SES were not included as covariates, the analysis showed that the abused group performed significantly poorer than the control group on tasks of learning and memory, attention and concentration and language (VIQ).

A systematic neuropsychological study will allow for the investigation of the impact of childhood maltreatment on a number of cognitive domains, enabling both the pattern and extent of deficits represented by this group of adolescents to be examined. It has been indicated that the deficits of maltreated children are not global, that is that most children showed deficits in specific areas rather than poor overall functioning (Gray, Nielsen, Wood, Andresen, & Dolce, 2000). This highlights the need for neuropsychological assessment, as it will provide an understanding of which cognitive areas are most problematic for maltreated adolescents. This in turn will allow for the more efficient allocation of resources to manage these specific areas within intervention programs. It has been indicated that “although maltreated children frequently manifest socioemotional behaviour problems that interfere with school performance, many maltreated children also manifest very fundamental deficits in behaviour that are fundamental to learning and school achievement but may be overlooked in purely clinical evaluations of adjustment” (Dodge-Reyome, 1994, p. 260).

### *1.18 Aims*

The aim of the research is to carry out a prospective systematic study of the cognitive profiles of children in a particular kind of protective care. That is, for those children at immediate risk of harm who have been placed in a secure facility to establish safety. These children have been in various forms of protective care for

many years, and represent a cohort of maltreated children at the severe end of the spectrum. The study aims to utilize a neuropsychological perspective to document the pattern and extent of cognitive impairments in these children and adolescents. The study also aims to examine the relationships between any observed cognitive deficits and characteristics of abuse history and aspects of affective functioning.

### *1.19 Hypotheses*

- (i) It is hypothesized that maltreated adolescents, in comparison to controls, will show significantly lower performances on overall cognitive function as represented by FSIQ score.
- (ii) It is hypothesized that maltreated adolescents will perform significantly more poorly than controls on measures of memory and learning.
- (iii) It is hypothesized that there will be significant differences between maltreated adolescents and controls on measures of executive functioning and attention.
- (iv) It is hypothesised that maltreated adolescents will show evidence of impaired spoken language skills in comparison to controls.
- (v) It is hypothesised that maltreated adolescents will show impaired performance on tasks of visuo-perceptual function of the visual analysis type in comparison to controls.
- (vi) It is hypothesized that greater extent or severity of cognitive deficits will be associated with greater abnormalities in affective functioning including, depression, anxiety and posttraumatic stress.

## **Chapter 2: Methodology**

### *2.1 Participants*

#### *Secure Welfare Group*

The sample consisted of a group of 56 (18 male and 38 female) adolescents aged 12-16 residing in a secure care facility located in Melbourne, Victoria. Seven of these participants (five male and two female) were excluded from the overall analysis as they obtained a FSIQ score below 70, falling in the intellectually disabled range. Participants were recruited via referrals from the Berry Street Take Two Program, who have a major role in the provision of clinical support services to children in protective care and secure welfare from local, metropolitan and rural communities in Victoria. Details of each young person's maltreatment history, family of origin and placements were taken from the Department of Human Services (DHS) Client Profile Document and the Take Two Harm Consequences Assessment. These documents were completed by the young person's DHS protective worker and forwarded to the Take Two Program. Specific information related to developmental history that was not present on the documents was followed up by contacting the young person's DHS protective worker, however in many instances, further details could not be obtained.

#### *Control Group*

A control group ( $n=59$ ), matching children by age, gender and SES were recruited from four government secondary schools in the western region of Melbourne, Victoria. Seven of these participants (four male and three female) were excluded from the main analysis as they obtained a FSIQ above 120, falling in the intellectually superior range. Participants were recruited via leading teachers (co-ordinators), after permission had been granted by the school principals and school council.

Participants from both groups were screened using a demographic questionnaire (see below) for major language and reading deficits, major visual and auditory deficits and major organ/systemic disease affecting the central nervous

system using semi structured interviews. Adolescents (both in the control and Secure Welfare group) who reported a history of identified psychiatric disorder (e.g. Schizophrenia) or major behavioural problems for example ADHD during the demographic interview were to be excluded from the study. In the case of the Secure Welfare group, those who had documented clinical reports by psychiatrists, psychologists and social workers in the DHS Client Profile document and/or the Take Two Harm Consequences Assessment were also to be excluded from the study. However, no participants involved in the current study were excluded on this basis as none of them reported clinical diagnoses of this nature during the demographic interview or had such issues documented in their histories.

## *2.2 Measures*

### *2.2.1 Demographic questionnaire*

A semi-structured interview schedule outlining demographic information including gender, age, education, medical history and substance use was conducted with each young person (see appendix 1 for Control group demographic questionnaire and appendix 2 for Secure Care group demographic questionnaire). For the Secure Care group, some relevant demographic information was obtained from direct interview with the participant, reading case records and speaking with protective/case workers. Parental occupation was recorded according to which person was regarded as the primary care-giver. The primary caregiver was defined as the individual that lived with the participant the longest period. For the control group, direct interview with the participant was the primary method of obtaining information about demographic information. A follow up interview with the parent/guardian of the young person was conducted if necessary to obtain further information in relation to the demographic questionnaire.

### *2.2.2 Socioeconomic status*

A measure of SES was obtained using an Australian normed instrument known as the Australian Socioeconomic Index 2006 scale (AUSEI 06, McMillan, Beavis, & Jones, 2009). Occupations are ranked from 0.0- 100.0. Occupations of low SES are scored toward zero, whilst occupations of high SES yield a score closer to 100. For example, factory process workers receive a score of 13.0, whilst medical

practitioners attract the highest score of 100.0. For the secure care group parental occupation was assigned to the individual that lived with the participant for the longest period. In circumstances where a participant had lived the longest in an adolescent residential unit under the care of residential youth workers (which is relevant to approximately 5% of the sample SW sample), parental occupation for these participants was deemed according to the qualifications of the individuals responsible for their care in the unit. The majority of care workers in residential units hold either youth or social work qualifications. For the control group, direct interview with the participant was the primary method of obtaining information about parental occupation.

### *2.2.3 Overall cognitive functioning*

#### **1. Wechsler Intelligence Scale for Children IV (WISC IV) (Wechsler, 2003)**

The WISC IV is a general measure of cognitive functioning. It allows for the calculation of a full scale intelligence quotient (FSIQ) as well as four separate indices of cognitive functioning including: verbal comprehension, processing speed, working memory and perceptual reasoning (Wechsler, 2003). Performance on these indices represents levels of functioning in the underlying cognitive systems, including; language, memory, perceptual and spatial processing, motor functions, attention and executive functions and social cognition. The separate indices of the WISC IV were also utilized as measures of specific cognitive domains as described below. The WISC IV was validated using a sample of 2200 children aged six years to 16 years and 11 months (Wechsler, 2003). It's internal and test-retest reliabilities are considered to be excellent for the majority of subtests with coefficients of .80 magnitude and above (Wechsler, 2003). The validity of the WISC IV is also good with significant correlations between subtests (e.g. the subtests of the Verbal Comprehension Index show moderate to strong relationships) indicating construct validity (Wechsler, 2003). It also appears to correlate well with other intelligence and developmental tests. Validity is further supported by the WISC IVs ability to discriminate clinical groups (i.e. mental retardation, ADHD, autism and giftedness) from controls (Wechsler, 2003). Further details of the reliability and validity measures of the WISC IV are provided in the interpretive manual (Wechsler, 2003).

#### *2.2.4 Memory and learning*

##### **1. Rey Auditory-Verbal Learning Test (RAVLT) (refer to appendix 3)**

The original format of the RAVLT (Rey, 1941) was utilized, however stimulus material, procedure for administration and scoring described by Strauss, Sherman, & Spreen (2006) was followed. The RAVLT consists of fifteen nouns which were verbally presented to the participant at the rate of one word per second. There were five consecutive trials in total. Each trial was followed by a free recall test, where the participant was required to recall, in any order, as many words as possible from the list presented. Upon completion of the fifth trial, an interference list of fifteen nouns (different to the original list) was presented verbally, followed by a free recall test of that list. After a 20-minute delay period, without further presentation of those words, the participant was asked to recall the nouns from the first list presented. Finally, a recognition task, where participants were required to identify the nouns from the first list within a larger list of words was completed. Trial totals of recalled nouns from A1 to A5 were summed to obtain a single total learning score for each participant. The score obtained on the free recall trial following the 20-minute delay was also noted as an indicator of retrieval capacity.

The psychometric properties of the RAVLT are quite good, the coefficient of alpha for the total score is .90 (Van den Burg & Kingma, 1999). Measures of test-retest reliability have produced moderate *r* values between .60 and .70 (Anderson & Lajoie, 1996; Mitrushina & Satz, 1991; Van den Burg & Kingma, 1999). The delayed recall scores correlate strongly with the total scores (Anderson & Lajoie, 1996; Mitrushina & Satz, 1991; Van den Burg & Kingma, 1999) providing some evidence of validity. Factor analyses have also shown that specific trials relate to acquisition, retention, storage and retrieval (Vakil & Blachstein, 1993). Studies have also shown that performance of the RAVLT correlates well with other measures of learning and memory, such as the Wechsler Memory Scales- Revised (WMS-R) (Johnstone, Vieth, J.C., & Shaw, 2000) and the California Verbal Learning Test (CVLT) (Crossen & Weins, 1994). The RAVLT is considered to be clinically useful in identifying those with memory deficits due to a range of neurological impairments (see Strauss et al., 2006 for review).

### 2.2.5 Working memory

#### 1. WISC IV Working Memory Index (WMI) (Wechsler, 2003)

The WISC IV Working Memory Index was designed to measure attention, concentration and working memory for verbal information (Wechsler, 2003). Where working memory is defined as the ability to maintain and manipulate information within memory. The WMI consists of two subtests, these are Digit Span and Letter-Number Sequencing. The standard method of administration as described by Wechsler (2003) was followed.

The digit span subtest includes two conditions, Digits Forwards and Digits Backwards. Both digits forwards and backwards tests consist of eight sets of digit strings, each string is one digit longer than the previous set. Digit strings were spoken (approximately one digit per second), by the researcher. In the digits forward test, the participant was expected to say each string of digits in the order presented. In the digits backwards test, the participant was advised to say the digit string in the reverse order of its presentation. A total digit span score was calculated according to the WISC IV manual (Wechsler, 2003), where each correct response obtained a score of one and an incorrect response obtained a score of zero. The overall total number of correct sequences was then recorded.

The letter-number sequencing subtest consists of 10 sets of letter-number sequences, where each sequence is one item longer than the previous set. The letter-number sequences were spoken (approximately one item per second), by the researcher. The participant was then required to manipulate the letter-number sequence, verbally expressing the numbers within the sequence in ascending numerical order and the letters within the sequence in alphabetical order. A total score was calculated according to the WISC IV manual, where each correct response obtained a score of one and an incorrect response obtained a score of zero. Total raw scores from each subtest were then calculated according to the WISC IV manual to produce an overall index score for working memory.

## 2. Swanson Sentence Span Task (Swanson, 1992)

The Swanson Sentence Span Task (SST) (refer to appendix 4) used in this study was a modified version of Daneman & Carpenter's (1980) Sentence Span Task for adults. The standard method of administration as described by Swanson (1992) was followed. The SST consists of five levels. Each level includes two sets of unrelated sentences and two comprehension questions in relation to the sentences (one for each set). The position of the answer to the question changes between sentences, with the restriction of never appearing within the last sentence of a set. The first level is comprised of 2 sentences within each set, then, with each level change, the number of sentences within each set increase by one sentence. The participant was instructed that they had to complete a number of steps within this task. First, they had to listen to the set of sentences, second, they had to answer a question in relation to one of the sentences, and third they would be required to recall the last word of each sentence within the order of which they were read. In order to conduct the task, the researcher read the set of sentences aloud to the participant (with a two second pause between each sentence). After the researcher had read the last sentence (with a two second pause following) the researcher read out the comprehension question in relation to one of the sentences to the participant. The participant was asked to answer the question (for example, "*Where did we wait?*" after listening to the sentence, "*We waited in line for an hour.*") The researcher then asked the participant to recall the last word of each sentence in the order of their presentation (for example, "*hour, freedom and excuse.*").

The reliability of the SST is excellent, where it has indicated coefficients of .80 magnitude and above (Swanson, 1992). Construct validity was also demonstrated by the significant correlations with other measures that tap into working memory capacity (Swanson, 1992).

### 2.2.6 Executive functioning and attention

1. Controlled Animal Fluency Test (CAFT) (formerly known as the Animal Fluency Test) (Tucker, Ewing, & Ross, 1996) (refer to appendix 5).



The Controlled Animal Fluency Test was used as a measure of verbal fluency. The participant was asked to name as many animals as possible in 60 seconds according to specified rules. The following three conditions are involved in the test:

- i. **Animals Automatic:** Participants were required to name as many different animals as possible in 60 seconds. Naming the same animal more than once was not allowed. No scores were given after the first time the animal appeared on the list if the same animal was repeated more than once, or altered slightly but was still the same animal (e.g. cat, kitten or little cat).
- ii. **Animals by Size:** Participants were required to order as many animals as possible from smallest to largest in 60 seconds, with each animal being slightly larger than the one before it.
- iii. **Animals by Alphabet:** Participants were required to name a single animal for each letter in order of the alphabet in 60 seconds. If the participant was silent for more than 15 seconds they were advised to go onto the next letter.

A relative difficulty –size score was calculated for each participant, this score represents the level of difficulty the participant experiences in the ‘animals by size’ condition, as it is mostly related to the executive functions. The relative difficulty-size score is computed using the following formula:

$$\frac{\text{Animals Auto score} - \text{Animals by Size score}}{\text{Animals Auto score}} \times 100$$

A relative difficulty-size score close to 100 indicates high difficulty, whilst a score closer to zero indicates low difficulty.

## 2. Controlled Oral Word Association Test (COWAT) (refer to appendix 6)

Stimulus material, procedure for administration and scoring of the COWAT followed the standard method as described in Strauss et al.(2006). This task is also a measure of verbal fluency, assessing the participant's ability to spontaneously generate words according to specified rules. Participants were required to produce as many words as possible within 60 seconds for each letter presented. The letters included in the task were F, A and S. Participants were instructed that they were not allowed to produce the names of places, people, products or the same word with different endings (for example, "stop" and "stopping"). A total score was calculated by adding the scores of each letter trial.

An Australian normative sample including 422 children aged seven to 15 years has been published by Anderson, Lajoie and Bell (1997). The internal consistency of each COWAT trial was high with an alpha coefficient of .83 (Tombaugh, Kozak, & Rees, 1999). Test- retest associations are strong with *r* values above .70 (Basso, Bornstein, & Lang, 1999; Tombaugh et al., 1999). Validity has been shown by moderate to strong correlations between variations in the letter combinations of the COWAT (e.g. FAS, CFL & PRW) (M. J. Cohen & Stanczak, 2000) and category fluency tasks (Kave, 2005). It has also been shown to correlate with of verbal intelligence, attention and processing speed (Anderson et al., 2001).

## 3. Trail Making Test Part B (TMTB) (refer to appendix 7)

The trail making test B is a measure of attention, speed, mental shifting and cognitive flexibility. The original version was adapted by Reitan (1955), however procedures for administration and scoring were followed according to Strauss et al. (2006). The stimulus is a single A4 sheet of paper covered with 25 randomly placed encircled numbers and letters. The participant was required to connect, alternating numbers and letters in numerical and alphabetical orders by making pencil lines, until reaching the end point number 13 (for example, 1 – A – 2 – B – 3 – C and so on). The participant was asked to complete the task as quickly as possible as they were being timed. The score was recorded as time in seconds required for completion. A score for number of errors produced was also noted. Procedure for administration and scoring of the TMTB followed the standard

method as described in Strauss et al (2006). The reliability of the TMTB is considered moderate with a coefficient of .67 (R. A. Cohen et al., 2001). The TMTB appears to be related to the executive function skills of perceptual shifting, sustained attention and inhibitory control. It has also demonstrated age related increases in performance in children aged between seven and 13 years (Kelly, 2000).

#### 4. Stroop Colour and Word Test (Golden, Freshwater, & Golden, 2003)

The Stroop Colour and Word Test is a measure of cognitive flexibility, selective attention and inhibitory control. Stimulus material, procedure for administration described by Golden et al. (2003) was used. The stimulus booklet includes three conditions. Each condition is presented in the same format, where 80 items are randomly presented in four lists, each list composed of 20 items. In the first condition (word), the participant was presented with a single page presenting the words red, green and blue written in black ink. The task of the participant was to read in order, as many words as possible within 45 seconds. In the second condition (colour), the participant was presented with a list of randomly coloured (red, green and blue) cross symbols. The task of the participant was asked to say out loud in order, the colours of the crosses as quickly as possible until the 45 second limit was reached. In the third condition (colour-word), the participant was given a list of words (red, green and blue), however they were written in an ink colour that was incongruent with the actual word (for example, the word red was written green ink). The participant was instructed to say out loud the colours of the ink that the words were written in, rather than the actual words. In all three conditions the participant was instructed to make corrections as they went along if they made errors and to begin from the start of the list again if they had completed reading all the words on the page within the time limit. The colour-word score was utilized as a measure of cognitive flexibility and inhibitory control. In order to obtain a colour-word score, the words read within the time limit were totalled for the third condition.

According to the Stroop Colour and Word Test manual (Golden et al., 2003) the reliability of the test has been shown over a number of studies, reporting

coefficients of .70 and above. It has been associated with the executive function skills of cognitive flexibility and perceptual set shifting (Lezak et al., 2004) and is sensitive to frontal lesions (Baron, 2004). The Stroop is considered a valid and reliable test, with coefficients above .83 on measures of test-retest reliability for each of the trials (Strauss et al., 2006).

### *2.2.7 Processing speed*

#### **1. WISC IV Processing Speed Index (PSI)**

The WISC IV Processing Speed Index consists of two conditions designed to measure a young person's speed of cognitive and visuospatial operations. In the coding B subtest of the PSI, the participant was shown a list of numbers from one to nine. Each number is assigned with a specific symbol. Below these items, numbers from one to nine are listed in non-numerical order. The participant was required to draw the symbol specific to each number in the box below it after being instructed to do so by the researcher. A two minute time limit is set for this subtest. The total number of correct digit-symbol pairs was calculated. There are 119 digit symbol pairs in this subtest, if the participant completed all items within the two minute time limit correctly, a time bonus was added to the total as outlined in the WISC IV manual (Wechsler, 2003).

Symbol search B is the second condition of the PSI. The participant was provided with 60 items, each item consists of two target symbols and five search group symbols. The participant was required to scan the search group for the identified target symbols. If either of the target symbols appeared in the search group, then the participant was required to tick yes in the boxes assigned to each item. If neither of the target symbols appeared in the search group, then the participant was required to tick no. A two minute time limit is set for this subtest. The correct number of responses was totalled to form a raw score.

Total raw scores from each subtest were then calculated according to the WISC IV manual (Wechsler, 2003) to produce an overall index score for processing speed.

### *2.2.8 Visuo-perceptual reasoning*

#### **1. WISC IV Perceptual Reasoning Index (PRI)**

The WISC IV Perceptual Reasoning Index is a measure of visuo-perceptual organization and reasoning. The index is formed by three subtests including block design, picture concepts and matrix reasoning.

Block design consists of 14 items. In each item, the participant was shown a picture of the design from the WISC IV stimulus book. The first items are comprised of 4 block designs whilst later items are composed of 9 blocks. Each block has two full red sides, two full white sides, and two half red-white sides. The participant was given instructions to manipulate the blocks as required to form the designs in the stimulus book. The stimulus book was shown to the participant, after which the participant was required to complete the design with the required number of blocks. Each item has a specified time limit, ranging from 45 seconds for earlier items to 120 seconds for the last few items. Time bonuses were allocated from items 9-14 which were included to form the total raw score.

Picture concepts is a measure of abstract reasoning consisting of 28 items. The participant was shown pages of two and three rows of pictures from the WISC IV stimulus book. The participant was required to choose one picture from each row to produce a set of pictures that follow a common theme or idea. The participant received a score of one if all of the pictures are chosen correctly and a score of zero if one or more incorrect pictures are chosen. All correct responses were then collated to form a total raw score.

Matrix reasoning is composed of 35 items. The participant was shown an incomplete design matrix from the WISC IV stimulus book, they were required to choose the missing section of the design out of five possible alternatives. A score of one was obtained for each correct response, and a score of zero for each incorrect response. A total of all correct responses formed the total raw score for this item.

Total raw scores from each subtest were then calculated according to the WISC IV manual (Wechsler, 2003) to produce an overall index score for perceptual reasoning.

#### **2. Beery-Buktenica Visual-Motor Integration Test (VMI) (Beery & Beery, 2004).**

The Visual- Motor Integration test is a measure of eye-hand co-ordination, visuoperceptual skills and visuospatial skills (Beery & Beery, 2004). The full 30-item form was used in this study. Participants were required to copy a sequence of geometric forms in the blank space below each geometrical form. Forms were scored according to the Beery VMI manual (Beery & Beery, 2004), with each correct form obtaining a score of one and an incorrect form obtaining a score of zero. The reliability of the Beery VMI is high, indicating a split-half correlation of .88 and an overall coefficient of alpha of .82 (Beery & Beery, 2004). According to the manual (Beery & Beery, 2004) the validity of the measure was examined by comparing results of tests measuring similar constructs. Moderate to strong associations between the Beery VMI and various tests were presented in the manual (e.g. Developmental test of Visual Perception & Wide Range Assessment of Visual Motor Abilities). Performances on the Beery VMI have also been shown to increase with chronological age, relate to non-verbal intelligence measures and academic achievement (see Beery & Beery, 2004 for review).

#### 2.2.9 Language

##### 1. WISC IV Vocabulary Index (VCI)

The WISC IV Verbal Comprehension Index was designed to measure verbal knowledge, verbal concept formation and verbal reasoning (Wechsler, 2003). The VCI consists of 3 subtests including, Similarities, Vocabulary and Comprehension. The Similarities subtest consists of 23 word pairs, matched by a common concept or idea. The task of the participant was to identify and describe the commonality between each word pair (for example: Q. *“How are pencils and pens alike?”* A. *“They’re both instruments used for writing”*).

The Vocabulary subtest consists of 36 words. The participant was required to define each word as descriptively as possible (for example: Q. *“What is a bicycle?”* A. *“A mode of transport with two wheels, a seat, handlebars and pedals”*).

The Comprehension subtest consists of 21 questions in relation to general principles and social awareness. The participant was asked to explain as descriptively as possible; the reasoning behind a specific process or idea (for example: Q. *“Why do police wear uniforms?”* A. *“So they can be easily identified in times of an*

*emergency*”), and how they would deal with specified situations (for example: (Q. “What would you do if you found someone’s wallet or purse in a store?” A. “Hand it in to the store manager”).

Each subtest of the VCI was scored according to the WISC IV manual (Wechsler, 2003), where each correct response was given a score of two and a partially correct response was given a score of one. Total raw scores from each subtest were then calculated according to the WISC IV manual (Wechsler, 2003) to produce an overall index score for verbal comprehension.

## 2. Peabody Picture Vocabulary Test III (PPVT) (Dunn & Dunn, 1997a).

The Peabody Picture Vocabulary Test is a measure of receptive vocabulary and level of vocabulary acquisition (Dunn & Dunn, 1997a). The standard method of administration as described by Dunn and Dunn (1997a) was followed. The PPVT test for form version IIIA was utilized within this study. Form IIIA consists of 17 sets of words, start points depend on the individuals age group, younger age groups begin at earlier sets whilst older age groups begin at later sets. As the specified age range for the participants within this study was 12-16 years, each participant began at set 10 of the form. Each set consists of 12 words, the testing was discontinued once the participant reached their ceiling set (obtaining eight or more errors in a set). In order to conduct this test, the participant was presented with the PPVT IIIA stimulus book. Each page of the stimulus book consists of four illustrations, numbered one to four. The researcher read a word from the test form and the participant was required to indicate which illustration corresponded to the particular word. The researcher then noted down whether the response was correct or incorrect. Scoring was conducted according to the PPVT manual (Dunn & Dunn, 1997a) and the PPVT norms booklet (Dunn & Dunn, 1997b).

Coefficients of internal consistency ranging between .92-.98, split half reliability of .81 and test-retest reliability of .91 and above, support the reliability of the PPVT (Dunn & Dunn, 1997a). In terms of validity, the PPVT correlates well with the Wechsler vocabulary subtests and other measures of intelligence and presents age related increases in performance (see Dunn & Dunn, 1997a for review).

### 2.3.0 Depression, anxiety and posttraumatic stress

#### 1. Trauma Symptom Checklist for Children (TSCC) (Briere, 1996)

The Trauma Symptom Checklist is a standardized measure of child and adolescent responses to unspecified traumatic events in a number of different symptom domains. The TSCC was first validated using clinical and child abuse centre samples (Briere, 1996). A sample of children who were outpatients at the Mayo clinic, or, who were the relatives of patients attending the clinic formed the non-clinical normative sample ( $n=3008$ ) (Freidrich, 1995). Construct validity of the TSCC was supported by Smith, Swenson, Hanson, and Saunders (1994) where types of specific symptoms of trauma correlated well with the symptom domains of the TSCC. A range of studies also indicating that each of the scales differentiated well between abused and non abused samples (D. M. Elliot & Briere, 1995; D. M. Elliot, McNeil, Cox, & Bauman, 1995), and reductions in symptom scale scores were observed following therapeutic intervention (Lanktree & Briere, 1995). In non-clinical/abused samples, the participants' experiences of stressful life events (e.g., parent divorce, death of a friend) were predicted by the TSSC-A which is an alternate form of the TSCC that does not include items related to the Sexual Concerns domains (Evans, Briere, Boggiano, & Barrett, 1994).

The TSCC consists of 54 items that are answered on a 4 point Likert scale from zero to three. The participants were required to rank how often they experienced each item, where 0=never, 1=sometimes, 2=lots of times, 3= almost all of the time. As directed in the manual, raw scores were totalled and converted to *T*-scores for each of the symptom domains outlined below; each domain had a separate *T*-score.

The domains measured include;

*Anxiety (ANX)*: Symptoms of generalized anxiety, hyperarousal, worry, specific fears (for example: of men, of women, of the dark) and unspecific fears such as fears of impending danger.

*Depression (DEP)*: Feelings of sadness, unhappiness and isolation. Thoughts related to self loathing, shame and guilt. Behaviours related to self harm and suicidality.



*Anger (ANG)*: Angry thoughts, feelings and behaviours. Feelings of hatred, feeling mean, being mad. Becoming argumentative, fighting and wanting to hurt others. Difficulties extinguishing angry behaviours.

*Posttraumatic stress (PTS)*: Intrusive memories of past traumatic events, presenting as thoughts, sensations and bad dreams. Cognitive avoidance of feelings associate with past trauma.

*Dissociation (DIS)*: The dissociation domain is structured including two subscales. The *Overt Dissociation subscale (DIS-O)*, refers to symptoms of derealisation, going blank, memory problems and emotional numbing. The *Dissociation Fantasy (DIS-F) subscale*, refers to behaviours such as daydreaming, pretending to be someone or somewhere else.

*Sexual concerns (SC)*: The sexual concerns domain is also structured including two subscales. The *Sexual Preoccupation (SC-P)* subscale refers to sexual thoughts or feelings that occur earlier than expected for the child's age. The *Sexual Distress (SC-D)* subscale refers to sexual conflicts, negative reactions to sexually related stimuli and fear of being sexually exploited.

Depression, anxiety and post traumatic stress were the only domains utilized for the purposes of this study. It has been identified within the literature that these forms of psychopathology may have an impact on cognitive functioning (Barrett et al., 1996; De Bellis, Keshavan et al., 1999; De Bellis & Putnam, 1994; Fertuck et al., 2006; Samuelson et al., 2006; Sapolsky, 1996; Silverman et al., 1996; M. H Teicher, Andersen, Polcari, Anderson, & Navalta, 2002).

### *2.3.1 Maltreatment history*

History of maltreatment for Secure Welfare participants was obtained from their individual DHS Client Profile Documents (CPD) and the Take Two Harm Consequences Assessment Referral Tools (T2 HCA) (see appendix 8). These documents were completed by the young person's DHS protective worker following referral to the Take Two program. These documents were then forwarded to the Senior Clinician in Take Two Secure Welfare, and were available to the researcher for review. The DHS CPD provided information about abuse type (i.e. sexual, physical,

emotional and neglect), duration of abuse, family networks and drug and alcohol issues. The first notification of abuse to DHS reported on the DHS CPD was used to determine duration of abuse. Time in years and months was calculated from this date until the period of assessment.

The Take Two HCA provided further information related to abuse type and severity of abuse. Its development (see appendix 9 for further information) was lead by Professor Shane Thomas from the School of Health, Latrobe University, Victoria, in collaboration with a number of individuals with considerable years of practice experience in child protection from organisations including Latrobe University Social Work Department, Department of Human Services and Take Two (S. Thomas et al., 2004). Indicators of abuse and predictive factors relating to behavioural and emotional disturbance and attachment difficulties were informed by reviewing the literature and thoroughly scrutinising published classifications of mental disorders and, trauma and childhood maltreatment. The development of the T2 HCA was largely informed by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), International Classification of Diseases and Related Health Problems (ICD-10), Victorian Risk Framework (VRF) and the Royal Children's Hospital Mental Health Service Stargate Program's Trauma and Attachment Screen.

The Take 2 HCA was used as a measure of maltreatment severity in this study. Severity of abuse/neglect in the Take 2 HCA was indicated on an ordinal scale with three levels, indicating concerning, serious and extreme levels of abuse, these were termed mild, moderate and severe respectively, for the purposes of this study. In the Take 2 HCA, the protective worker is required to provide information regarding five domains of abuse and neglect including; abandonment, physical harm and injury, sexual abuse, emotional and psychological harm, developmental and medical harm. There is also a second component where they are asked to indicate what impact these abuse/neglect experiences have on the young persons functioning. Each domain of maltreatment is associated with three categories, including extreme, serious and concerning. Listed under each of these categories are experiences of abuse and neglect deemed to fall under the specified level of severity. For example, 'dangerous self harm' is an option that falls in the extreme category of the emotional and psychological harm domain. In this study, the most frequently occurring category

across all maltreatment domains was deemed as the level of severity for each of the Secure Welfare participants. Therefore, if a participant had most experiences falling within the extreme category across a number of maltreatment domains, then their experiences would be classified as severe.

### *2.3.2 Substance use*

During the demographic interview, participants in both groups were asked to list types of substances used for a period of longer than three months. Further information regarding substance use for the Secure Welfare group was obtained from the DHS CPD.

## *2.4 Procedure*

### *i. Secure Care Group*

Following referral from the Take Two program and obtaining informed consent from parents/ guardians of the participants, individual appointments were made with the participants to complete the assessment. All assessments took place at the Department of Human Service Young Men's and Young Women's Secure Welfare Services. Prior to the commencement of the assessment, each participant had the assessment procedure explained and was given the opportunity to ask questions in relation to the assessment process and the research study. The participants were also informed that they were able to stop the assessment at anytime for a break and that they could withdraw at anytime if they did not want to continue with the assessment. After this information was explained, the participant was also given a copy of an informed consent form to sign. As a large majority of young people placed in Secure Welfare arrive in states of substance intoxication, it was ensured that those in the acute states of intoxication or withdrawal were not assessed. Reports of individuals in states of substance intoxication or withdrawal were made by Secure Welfare staff to the researcher. The researcher also observed participants in relation to substance effects both before and during the assessment to ensure that they weren't completing the assessment while under the influence of drugs. Those who were in this condition were not assessed until at least a week following their admission, and in some

circumstances, those that continued to show considerable withdrawal symptoms after this period were not assessed. In order to avoid fatigue, participants generally completed the assessment over two sessions each lasting approximately one hour. In some circumstances assessments were completed over a single session (lasting approximately two to three hours) upon the participant's request.

Participants were initially required to complete the short semi-structured interview regarding demographic information in order to screen for conditions specified within the exclusion criteria. The cognitive measures were then administered in the following order; Complete WISC IV, Beery VMI, PPVT IIIA, Stroop Colour-Word Test, RAVLT, COWAT, CAFT, Swanson SST, TMTB, RAVLT Recall and Recognition (20 Minute Delay) and TSCC, known as order A. A counterbalanced order (order B), the exact reverse of order A, was implemented to avoid testing effects. Every second participant completed the measures in order, ensuring that the 20 minute delayed recall and recognition trial required of the RAVLT was achieved.

Following the assessment the researcher provided feedback in the form of a neuropsychological report for each participant, outlining their performance on all the measures included in the research protocol. This report was then forwarded on to each participant's protective worker to be distributed to the participant and their parent/guardians. Protective workers (and if applicable, the participant's care network) were also invited to participate in a face to face feedback session, providing further information in relation to the neuropsychological report. Not all protective workers took up the opportunity for face to face feedback due to circumstances and events beyond the researcher's control.

## ii. Control Group

Principals of four secondary schools in the western region of Melbourne, Victoria were invited to participate in the study. Once approval from the principals was obtained, informed consent forms were distributed to the students and their parent/guardians via leading teachers. Those parent/guardians who agreed to their child's participation completed and signed the consent form and returned it to the researcher. An appointment time was arranged with the participant and the interviews

and assessments were completed during school hours in an appropriate space. Participants were inducted to the research and completed the assessment process in the same manner as the Secure Care participants. After they had signed the informed consent form, the demographic interview was conducted, followed by the cognitive and affective measures listed previously. The Control participants also received a neuropsychological report outlining their performance on the measures included in the research protocol. Further verbal feedback was reported on request of the participant and/or their parent/guardian.

Following testing, the performance of the Secure Care group and the Control group on the measures of cognitive and affective functioning were investigated. In order to explore the range and extent of cognitive deficits, performance on measures of cognitive capacity were compared between the Secure Care Group and the Control group.

### *2.5 Research Ethics Approval*

Ethical approval of the research project was obtained from the following research ethics committees; Victoria University Human Research Ethics Committee (appendix 10), Department of Human Services Human Research Ethics Committee (Victoria) (appendix 11), Berry Street Victoria Policy and Practice Committee (appendix 12) and the Victorian Department of Education Human Research Ethics Committee (appendix 13).

The project was also subject to approval by the various secondary school principals approached for involvement in the study. The principals were given formal invitation (appendix 14), outlining the details and purposes of the project. After approval was obtained, class room teachers were given informed consent forms inviting students and their parents/guardians for participation in the study.

The informed consent forms were published following the comprehensive format as required by the Department of Human Services Human Research Ethics Committee. Separate types of forms were made for the range of participants within the study. For the Secure group, different forms were given to the parent/guardian of

the child dependent on their custodial status. A version of the information and consent form was given to participants under the care of a parent/ guardian (appendix 15). There was a separate form (appendix 16) for those participants under a guardianship order, where DHS was named as their legal guardian. An individual informed consent form was also given to the Secure Care participant (appendix 17). The control group had two separate forms, one for the parent/guardian (appendix 18) and one for the participant (appendix 19).

## Chapter 3: Results

### 3.1 Demographic Variables for each Subject Group

All statistical tests were conducted utilizing the Statistical Package for the Social Sciences Version 17.0 (SPSS). Demographic characteristics of the two groups were examined using descriptive statistics, independent groups t-tests, correlations and chi squared. Demographic data for each of the groups is shown in Table 1 below. Data for these variables are shown as group means (*M*) and standard deviations (*SD*). Gender frequencies for each of the groups are reported in Table 2.

Table 1

Demographic Variables of the Control and Secure Welfare Groups

|                     | <b>CO group</b><br>( <i>n</i> =52) | <b>SW group</b><br>( <i>n</i> =49) | <i>p</i> -value |
|---------------------|------------------------------------|------------------------------------|-----------------|
| <b>Demographics</b> |                                    |                                    |                 |
| Age (years)         | 14.47(1.22)                        | 14.51(1.19)                        | 0.840           |
| Education (years)   | 9.84 (1.07)                        | 7.82(1.35)                         | 0.001***        |
| SES                 | 27.39(21.56)                       | 25.38(24.33)                       | 0.660           |

\*\*\*  $p < 0.001$  SES= AUSEI 06 socioeconomic status scale score (McMillan et al., 2009)

Table 2

Gender Distribution for the Control and Secure Welfare Groups

| <b>Gender</b> | <b>CO group (<i>n</i>=52)</b> | <b>SW group (<i>n</i>=49)</b> |
|---------------|-------------------------------|-------------------------------|
| Male          | 17                            | 13                            |
| Female        | 35                            | 36                            |

Group differences according to age, education and SES were examined using Independent t-tests. There were no significant differences between the groups for age,  $t(99) = -0.20$ ,  $p = 0.840$  and SES  $t(99) = 0.44$ ,  $p = 0.660$ . However, the SW group had

significantly less years of formal education than the control group  $t(91.14) = 8.29$ ,  $p = 0.001$ .

Pearson's Chi-square analysis was conducted to assess group differences based on gender. Table 2 shows the number of males and females in each group. There were no significant differences between the groups regarding gender  $\chi^2(1) = 0.46$ ,  $p = 0.498$ , suggesting that the numbers of males and females in each group was relatively even.

### *3.1.1 Relationship between education and cognitive performance for the Secure Welfare Group*

Pearson's Bivariate correlations were used to examine whether there were relationships between cognitive performance and years of education for the Secure Welfare Group. Table 3 presents all the cognitive measures and their associated correlation coefficients ( $r$ ).



Table 3

Bivariate correlations between education and the cognitive measures for the Secure Welfare group (n=49)

| Cognitive Measures       | Education | <i>p</i> - value |
|--------------------------|-----------|------------------|
|                          |           |                  |
| WISC IV-FSIQ             | .38       | 0.006**          |
| WISC IV-WMI              | .40       | 0.004**          |
| SSST                     | .20       | 0.166            |
| RAVLT A6- Retention      | .31       | 0.030*           |
| RAVLT A7- Delayed Recall | .33       | 0.021*           |
| RAVLT- Total             | .37       | 0.010**          |
| CAFT -RDS                | .16       | 0.270            |
| CAFT- Size               | .19       | 0.202            |
| COWAT                    | .36       | 0.010*           |
| TMTB                     | -.15      | 0.311            |
| TMTB- Errors             | -.07      | 0.638            |
| Stroop- C/W              | .29       | 0.041*           |
| WISC IV-PRI              | .31       | 0.030*           |
| VMI                      | .01       | 0.959            |
| WISC IV-PSI              | .39       | 0.005**          |
| PPVT                     | .17       | 0.242            |
| WISC IV-VCI              | .11       | 0.449            |

\*  $p < 0.05$  \*\*  $p < 0.01$

Pearson's Bivariate correlations showed that there were statistically significant but low strength associations between years of education and performances on measures of FSIQ, working memory, learning and memory, executive functioning, visuo-perceptual functioning and processing speed. Given the large number of correlation coefficients, Bonferroni corrections were applied to avoid inflating Type I error. The use of Bonferroni corrections for tests of significance resulted in the more

conservative alpha level of 0.003. When applying this alpha level, none of the correlation coefficients between the cognitive measures and years of education were significant for the Secure Welfare group.

### 3.2 Substance Abuse

The data related to type of substance abuse reported by participants is demonstrated in Table 4 in the form of frequencies and percentages.

Table 4

Frequencies of participants engaging in substance abuse by type

| Substance Type               | CO (n=52) |         | SW (n=49) |         |
|------------------------------|-----------|---------|-----------|---------|
|                              | Frequency | Percent | Frequency | Percent |
| Alcohol Only                 | 2         | 3.8     | 1         | 2.0     |
| Crystal Methamphetamine Only | -         | -       | 2         | 4.1     |
| Inhalants Only               | -         | -       | 2         | 4.1     |
| Polysubstance Abuse          | -         | -       | 44        | 89.8    |
| None                         | 50        | 96.2    | -         | -       |

Table 4 shows that almost 90% of participants in the Secure Welfare group reported that they engaged in the abuse of a range of substances. Approximately ten percent of the Secure Welfare participants reported engaging in only one type of substance use. The majority (96.1%) of participants in the control group reported no substance abuse, however two participants reported alcohol abuse.

### 3.4 Maltreatment type, Severity of Maltreatment and Duration of Maltreatment for Secure Welfare group

The data related to variables of abuse type and severities are reported in Table 5 in the forms of frequencies and percentages.

Table 5

Maltreatment type and severity for the Secure Welfare Participants (n=49)

|                                 | Frequency | Percent |
|---------------------------------|-----------|---------|
| <b>Maltreatment Type</b>        |           |         |
| Neglect Only                    | 2         | 4.1     |
| Mixed                           | 47        | 95.9    |
| <b>Severity of Maltreatment</b> |           |         |
| Mild                            | 1         | 2.0     |
| Moderate                        | 2         | 4.1     |
| Severe                          | 46        | 93.9    |

Of the 49 participants in the Secure Welfare group, only two were documented as having a single maltreatment type, the remainder of participants (95.9 %) were reported as having experienced multiple maltreatment types. Table 5 also shows that a large proportion (93.9 %) of the participants were documented as having maltreatment experiences that fell at the severe end of the spectrum, whilst only three participants fell in the mild to moderate ranges of severity. Duration of abuse ranged from four months to 15 years, with a median of seven years.

#### 3.4.1 Relationship between maltreatment duration and the cognitive variables

Pearson's Bivariate correlations were used to examine whether there were relationships between cognitive performance and duration of maltreatment in years for the Secure Welfare Group. Table 6 presents these in relationships with correlation coefficients ( $r$ ).

Table 6

Correlations of cognitive variables and maltreatment duration for the Secure Welfare group (n=49)

| Cognitive Measures       | Maltreatment Duration | <i>p</i> -value |
|--------------------------|-----------------------|-----------------|
| WISC IV-FSIQ             | -.06                  | 0.694           |
| WISC IV-WMI              | -.17                  | 0.228           |
| SSST                     | -.09                  | 0.520           |
| RAVLT A6- Retention      | -.31                  | 0.033*          |
| RAVLT A7- Delayed Recall | -.28                  | 0.050*          |
| RAVLT- Total             | -.24                  | 0.097           |
| CAFT -RDS                | -.17                  | 0.248           |
| CAFT- Size               | .24                   | 0.104           |
| COWAT                    | -.11                  | 0.465           |
| TMTB                     | .14                   | 0.326           |
| TMTB- Errors             | -.01                  | 0.962           |
| Stroop- C/W              | -.22                  | 0.138           |
| WISC IV-PRI              | -.15                  | 0.313           |
| VMI                      | -.23                  | 0.105           |
| WISC IV-PSI              | -.19                  | 0.192           |
| PPVT                     | -.09                  | 0.533           |
| WISC IV-VCI              | -.01                  | 0.968           |

\*  $p < 0.05$  WISC IV-FSIQ= Wechsler Intelligence Scale for Children IV- Full Scale Intelligence Quotient, WISC IV-WMI= Wechsler Intelligence Scale for Children IV- Working Memory Index, SSST=Swanson Sentence Span Task, RAVLT -Retention= Rey Auditory Verbal Learning Test retention after interference trial score, RAVLT -Delayed Recall= Rey Auditory Verbal Learning Test delayed recall trial score, RAVLT- Total= Rey Auditory Verbal Learning Test total learning score of five trials, CAFT- Size= Controlled Animal Fluency Test Animals by Size Score, CAFT-RDS= Controlled Animal Fluency Test Relative Difficulty Score, COWAT= Controlled Oral Word Association Test, TMTB= Trail Making Test part B completion time in seconds, TMTB errors= number of errors on Trail Making Test part B, Stroop- C/W= Stroop colour/word score, WISC IV- PRI= Wechsler Intelligence Scale for Children IV- Perceptual Reasoning Index, VMI= Beery-Buktenica Visuo-motor Integration Test, Wechsler Intelligence Scale for Children IV - PSI= Wechsler Intelligence Scale for Children IV-Processing Speed Index, WISC IV-VCI= Wechsler Intelligence Scale for Children IV-Verbal Comprehension Index, PPVT= Peabody Picture Vocabulary Test score.

With alpha set at 0.05, Pearson's Bivariate correlations showed that there were significant low strength associations between abuse duration and performances on measures of RAVLT recall following interference  $r=-.31, p=0.03$ , and RAVLT delayed recall,  $r=-.28, p=0.05$  for the Secure Welfare group. These results indicate that as abuse duration increases, performances on the measures of learning and memory decrease. However, the strength of these associations was low. Given that there were a large number of correlation coefficients tested for significance in the analysis, these statistically significant correlations should be interpreted as exploratory.

### *3.5 Data Analysis for Cognitive and Affective Variables*

Analysis of differences in cognitive functioning between the secure welfare and control groups was conducted using Multivariate Analysis of Variance (MANOVA) and Independent t-tests. Assumption testing was conducted to ensure the data could be appropriately analysed with parametric statistical procedures. Normality was assessed using the Kolmogorov-Smirnov test, skewness and kurtosis values and examining histograms for each of the dependent variables. Statistical normality assessments for each of the variables are reported under their relative domains in the analysis (see Tables 8, 10, 12, 14, 16, 18 and 19). According to the Kolmogorov –Smirnov test some of the variables significantly deviated from normality. A small number of variables were significantly skewed. Stevens (2002) suggests that non-normal and significantly skewed distributions have a marginal effect on Type I error and power in MANOVA. It has also been reported that MANOVA analysis remains robust in conditions where the assumption of normality has been violated, furthermore, replacing MANOVA with non-parametric tests was shown to have very little effect on significance values (Seo, Kanda, & Fujikoshi, 1995).

Instances of Type I and Type II error often occur in MANOVA when there are outliers in the data set as MANOVA is particularly sensitive to outliers (Tabachnick & Fidell, 2007). Examination of box-plots and z-score conversions indicated that there were no significant outliers in the data set. Scatter-plots of the dependent variables were observed for linearity, indicating that this assumption was met. The assumption of homogeneity of variance-covariance matrices required of MANOVA

was not met for some variables according to Box's *M* test. However, it has been suggested that the SPSS MANOVA version of this test is extremely sensitive, and should be disregarded in the case of relatively equal sample sizes (Tabachnick & Fidell, 2007). According to Tabachnick and Fidell, Pillai's criterion should be used to evaluate multivariate significance when Box's *M* is significant as it is the most robust.

Effect sizes were calculated using Cohen's *d* in order to document the magnitude of the differences between the two groups on specific dependent variables. Effect sizes are reported as small, medium and large with corresponding *d* values of 0.2, 0.5, and 0.8 respectively, following the classification scheme developed by J. Cohen (1988; J. Cohen, 1992).

### 3.5.1 Overall cognitive functioning

Means (*M*) and standard deviations (*SD*) of FSIQ scores for both the control and Secure Welfare group are given in Table 7. An independent samples *t*-test was used to compare performance between the two groups on overall cognitive functioning (FSIQ). Normality analysis is presented in Table 8, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on the memory and learning measures.

Table 7

WISC IV- FSIQ scores (M, SD) for the Control and Secure Welfare Groups

|                            | CO group |           | SW group |           |           |                 |          |
|----------------------------|----------|-----------|----------|-----------|-----------|-----------------|----------|
|                            | (n=52)   |           | (n=49)   |           |           |                 |          |
| Measures:                  | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>t</i>  | <i>p</i> -value | Cohen's  |
| Overall Cognitive Function |          |           |          |           | Statistic |                 | <i>d</i> |
| WISC IV-FSIQ               | 96.46    | 9.16      | 87.18    | 9.91      | -4.89     | 0.001***        | 1.41     |

\*\*\*  $p < 0.001$  WISC IV-FSIQ=WISC-IV Full Scale Intelligence Quotient

The results showed that there was a significant difference between the Control group and Secure Welfare group on FSIQ. The effect size estimate for overall cognitive function,  $d=1.41$  can be considered very large according to Cohen's (1988) effect size framework. Normality analysis is presented in Table 8, showing the

Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups for WISC IV-FSIQ.

Table 8

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on WISC- FSIQ

| Cognitive Measures | CO (n=52)          |          |          | SW (n=49)          |          |          |
|--------------------|--------------------|----------|----------|--------------------|----------|----------|
|                    | Kolmogorov-Smirnov | Skewness | Kurtosis | Kolmogorov-Smirnov | Skewness | Kurtosis |
| WISC IV-FSIQ       | 0.86               | 0.04     | 0.39     | 0.11               | 0.36     | -0.51    |

WISC IV-FSIQ=Full scale intelligence quotient

### 3.5.2 Memory and learning

MANOVA was used to compare the performances of the Control and Secure Welfare groups on measures of learning and memory. Means (*M*) and standard deviations (*SD*) of the scores for the measures of learning and memory are given in Table 9. Normality analysis is presented in Table 10, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on the memory and learning measures.

Table 9

Memory and learning measures (M, SD) for the Control and Secure Welfare Groups

|                       | CO group |           | SW group |           |                       |                 |                     |
|-----------------------|----------|-----------|----------|-----------|-----------------------|-----------------|---------------------|
|                       | (n=52)   |           | (n=49)   |           |                       |                 |                     |
| Measures:             | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>F</i><br>Statistic | <i>p</i> -value | Cohen's<br><i>d</i> |
| Memory and Learning   |          |           |          |           |                       |                 |                     |
| RAVLT-Retention       | 12.08    | 2.56      | 9.69     | 3.15      | 17.51                 | 0.001***        | 0.84                |
| RAVLT- Delayed Recall | 12.00    | 2.26      | 9.18     | 4.01      | 19.17                 | 0.001***        | 0.88                |
| RAVLT-Total           | 55.27    | 6.90      | 45.75    | 11.24     | 26.60                 | 0.001***        | 1.04                |

\*\*\*  $p < 0.001$  RAVLT -Retention= Rey Auditory Verbal Learning Test retention after interference trial score, RAVLT –Delayed Recall= Rey Auditory Verbal Learning Test delayed recall trial score, RAVLT- Total= Rey Auditory Verbal Learning Test total learning score of five trials.

Using the Pillai's trace statistic, there were significant differences between the two groups on the combined learning and memory measures,  $V=0.22$ ,  $F(3,97)=9.18$ ,  $p=0.001$ . The univariate tests revealed significant differences for RAVLT retention, RAVLT delayed recall and RAVLT total (see Table 9), where the Secure Welfare group performed significantly more poorly than the control group on all the learning and memory measures. The Cohen's  $d$  values for all three measures were in the large effect size range.

Table 10

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on measures of memory and learning

| Cognitive Measures      | CO ( $n=52$ )      |          |          | SW ( $n=49$ )      |          |          |
|-------------------------|--------------------|----------|----------|--------------------|----------|----------|
|                         | Kolmogorov-Smirnov | Skewness | Kurtosis | Kolmogorov-Smirnov | Skewness | Kurtosis |
| RAVLT A6-Retention      | 0.18**             | -0.64    | -0.75    | 0.15               | -0.39    | -0.54    |
| RAVLT A7-Delayed Recall | 0.19**             | -0.59    | -0.31    | 0.12               | -0.31    | -0.79    |
| RAVLT- Total            | 0.12               | -0.47    | -0.51    | 0.08               | -0.15    | -0.16    |

\*\*  $p<0.01$

### 3.5.3 Working memory

MANOVA was used to compare the performances of the Control and Secure Welfare groups on measures of working memory. Means ( $M$ ) and standard deviations ( $SD$ ) of the scores for the measures of working memory are given in Table 11. Normality analysis is presented in Table 12, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on the working memory measures.



Table 11

Working memory measures (M, SD) for the Control and Secure Welfare groups

|                | CO group |           | SW group |           |           |                 |          |
|----------------|----------|-----------|----------|-----------|-----------|-----------------|----------|
|                | (n=52)   |           | (n=49)   |           |           |                 |          |
| Measures:      | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>F</i>  | <i>p</i> -value | Cohen's  |
| Working Memory |          |           |          |           | Statistic |                 | <i>d</i> |
| WISC IV-WMI    | 96.77    | 12.43     | 91.49    | 9.97      | 5.50      | 0.021*          | 0.47     |
| SSST           | 3.40     | 1.26      | 2.18     | 1.51      | 19.58     | 0.001***        | 0.89     |

\*  $p < 0.05$  \*\*\*  $p < 0.001$  WISC IV-WMI=WISC-IV Working Memory Index, SSST=Swanson Sentence Span Task total score

Pillai's trace indicated that there were significant differences between the two groups on the combined working memory measures,  $V = 0.17$ ,  $F(2,98) = 9.84$ ,  $p = 0.001$ . The univariate tests revealed significant differences between the groups with a large effect size on the Swanson Sentence Span Task (see Table 11), where the Secure Welfare group performed significantly more poorly on this task compared to the Control group. Performance on the WISC IV- WMI was also significantly different between the two groups with the Secure Welfare group performed at a lower level, though the yielded effect size is considered to be small.

Table 12

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on measures of working memory

| Cognitive<br>Measures | CO (n=52)              |          |          | SW (n=49)              |          |          |
|-----------------------|------------------------|----------|----------|------------------------|----------|----------|
|                       | Kolmogorov-<br>Smirnov | Skewness | Kurtosis | Kolmogorov-<br>Smirnov | Skewness | Kurtosis |
| WISC IV-<br>WMI       | 0.18**                 | 0.74     | 2.22     | 0.13*                  | 0.39     | -0.35    |
| SSST                  | 0.24**                 | -0.45    | 0.28     | 0.15**                 | 0.36     | -0.47    |

\* at  $p < 0.05$  \*\* at  $p < 0.01$

### 3.5.4 Executive functioning and attention

MANOVA was used to compare the performances of the Control and Secure Welfare groups on measures of executive functioning and attention. Means (*M*) and standard deviations (*SD*) of the scores for the measures of executive functioning and attention are given in Table 13. Normality analysis is presented in Table 14, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on the executive function/attention measures.

Table 13

Executive functioning and attention measures (*M*, *SD*) for the Control and Secure Welfare groups

|                                     | CO group |       | SW group |       |                |          |              |
|-------------------------------------|----------|-------|----------|-------|----------------|----------|--------------|
| Measures:                           | (n=52)   |       | (n=49)   |       |                |          |              |
| Executive Functioning and Attention | M        | SD    | M        | SD    | F<br>Statistic | p-value  | Cohen's<br>d |
| CAFT-Size                           | 12.33    | 2.73  | 11.45    | 3.67  | 1.87           | 0.174    | -            |
| CAFT-RDS                            | 40.86    | 16.19 | 42.63    | 18.77 | 0.26           | 0.612    | -            |
| COWAT                               | 34.92    | 7.38  | 31.57    | 11.00 | 3.27           | 0.074    | -            |
| TMTB completion time (sec)          | 66.24    | 24.96 | 92.43    | 35.80 | 18.37          | 0.001*** | 0.86         |
| TMTB-Errors                         | 0.54     | 0.90  | 1.41     | 2.54  | 5.39           | 0.022*   | 0.47         |
| Stroop-C/W                          | 42.79    | 9.67  | 32.96    | 8.90  | 28.16          | 0.001*** | 1.07         |

\*  $p < 0.05$  \*\*\*  $p < 0.001$  CAFT- Size= Controlled Animal Fluency Test Animals by Size Score, CAFT-RDS= Controlled Animal Fluency Test Relative Difficulty Score, COWAT= Controlled Oral Word Association Test, TMTB= Trail Making Test part B completion time in seconds, TMTB errors= number of errors on Trail Making Test part B, Stroop- C/W= Stroop colour/word score

Using MANOVA Pillai's trace, there were significant differences between the two groups on the combined executive function/ attention measures,  $V = 0.25$ ,  $F(6,94) = 5.14$ ,  $p = 0.001$ . The univariate tests revealed significant differences between the groups with large effect sizes on TMTB and Stroop-CW performance (see Table 13), where the Secure Welfare group performed significantly more poorly on these tasks compared to the Control group. Performances on CAFT-Size, CAFT-RDS and COWAT were not significantly different between the two groups, however TMTB-Errors was significant at  $p < 0.05$ , with a medium effect size.

Table 14

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on measures of executive function

|                           | <b>CO (<i>n</i>=52)</b>   |                 |                 | <b>SW (<i>n</i>=49)</b>   |                 |                 |
|---------------------------|---------------------------|-----------------|-----------------|---------------------------|-----------------|-----------------|
| <b>Cognitive Measures</b> | <b>Kolmogorov-Smirnov</b> | <b>Skewness</b> | <b>Kurtosis</b> | <b>Kolmogorov-Smirnov</b> | <b>Skewness</b> | <b>Kurtosis</b> |
| CAFT - RDS                | 0.12                      | 0.06            | -0.67           | 0.11                      | 0.20            | -0.10           |
| CAFT- Size                | 0.11                      | -0.17           | -0.04           | 0.12                      | 0.37            | 0.11            |
| COWAT                     | 0.08                      | 0.43            | -0.49           | 0.09                      | 0.58            | -0.20           |
| TMTB                      | 0.14*                     | 1.29            | 2.16            | 0.13*                     | 0.96            | 0.49            |
| TMTB-Errors               | 0.36**                    | 2.09            | 4.68            | 0.34**                    | 2.98            | 9.38            |
| Stroop-C/W                | 0.12*                     | 0.65            | 0.92            | 0.09                      | -0.03           | -0.39           |

\*  $p<0.05$  \*\*\*  $p<0.001$

### 3.5.6 Language

MANOVA was used to compare the performances of the Control and Secure Welfare groups on measures of language. Means ( $M$ ) and standard deviations ( $SD$ ) of the scores for the measures of language are given in Table 15. Normality analysis is presented in Table 16, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on the language measures.

Table 15

Language measures (M, SD) for the Control and Secure Welfare groups

|                  | CO group<br>( $n=52$ ) |       | SW group<br>( $n=49$ ) |       | $F$<br>Statistic | $p$ -value | Cohen's<br>$d$ |
|------------------|------------------------|-------|------------------------|-------|------------------|------------|----------------|
|                  | $M$                    | $SD$  | $M$                    | $SD$  |                  |            |                |
| <b>Measures:</b> |                        |       |                        |       |                  |            |                |
| <b>Language</b>  |                        |       |                        |       |                  |            |                |
| WISC IV-VCI      | 91.06                  | 8.33  | 87.33                  | 10.18 | 4.08             | 0.046*     | 0.41           |
| PPVT             | 98.85                  | 13.40 | 92.65                  | 12.13 | 5.90             | 0.017*     | 0.49           |

\*  $p < 0.05$  WISC IV-VCI= Wechsler Intelligence Scale for Children IV-Verbal Comprehension Index, PPVT= Peabody Picture Vocabulary Test score.

Using MANOVA Pillai's trace, there were significant differences between the two groups on the combined language measures,  $V = 0.17$ ,  $F(2,98) = 9.84$ ,  $p = 0.001$ . The univariate tests revealed that there were significant differences between the on both WISC IV-VCI and PPVT, yielding small effect sizes. The Secure Welfare group performed more poorly on both language measures in comparison to the control group.

Table 16

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on measures of language

|                           | CO ( <i>n</i> =52)         |                 |                 | SW ( <i>n</i> =49)         |                 |                 |
|---------------------------|----------------------------|-----------------|-----------------|----------------------------|-----------------|-----------------|
| <b>Cognitive Measures</b> | <b>Kolmogoro v-Smirnov</b> | <b>Skewness</b> | <b>Kurtosis</b> | <b>Kolmogoro v-Smirnov</b> | <b>Skewness</b> | <b>Kurtosis</b> |
| WISC IV-VCI               | 0.06                       | -0.01           | -0.38           | 0.15**                     | 0.30            | -1.01           |
| PPVT                      | 0.15**                     | -0.55           | 0.79            | 0.13*                      | -0.15           | -0.58           |

\*  $p < 0.05$  \*\*\*  $p < 0.001$

### 3.5.7 Visuo-perceptual functioning

MANOVA was used to compare the performances of the Control and Secure Welfare groups on measures visuo-perceptual function. Means (*M*) and standard deviations (*SD*) of the scores for the measures of visuo-perceptual function are given in Table 17. Normality analysis is presented in Table 18, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on visuo-perceptual function measures.

Table 17

Visuo-spatial and perceptual reasoning measures (*M*, *SD*) for the Control and Secure Welfare groups

|                              | CO group |           | SW group |           |           |                 |          |
|------------------------------|----------|-----------|----------|-----------|-----------|-----------------|----------|
|                              | (n=52)   |           | (n=49)   |           |           |                 |          |
| Measures:                    | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>F</i>  | <i>p</i> -value | Cohen's  |
| Visuo-Perceptual Functioning |          |           |          |           | Statistic |                 | <i>d</i> |
| WISC IV-PRI                  | 97.96    | 9.60      | 89.59    | 12.60     | 14.21     | 0.001***        | 0.76     |
| VMI                          | 107.67   | 10.64     | 98.22    | 12.47     | 16.87     | 0.001***        | 0.83     |

\*\*\*  $p < 0.001$  WISC IV-PRI= Wechsler Intelligence Scale for Children IV- Perceptual Reasoning Index, VMI= Beery-Buktenica Visuo-motor Integration Test

The Pillai's trace statistic indicated that there were significant differences between the two groups on the combined visuo-perceptual function measures,

$V=0.18$ ,  $F(2,98)=10.37$ ,  $p=0.001$ . The univariate tests revealed significant differences between the groups with large effect sizes on the WISC IV-PRI and the VMI (see Table 17), where the Secure Welfare group performed significantly more poorly on these tasks compared to the Control group.

Table 18

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on measures of language

|                           | CO ( $n=52$ )             |                 |                 | SW ( $n=49$ )             |                 |                 |
|---------------------------|---------------------------|-----------------|-----------------|---------------------------|-----------------|-----------------|
| <b>Cognitive Measures</b> | <b>Kolmogorov-Smirnov</b> | <b>Skewness</b> | <b>Kurtosis</b> | <b>Kolmogorov-Smirnov</b> | <b>Skewness</b> | <b>Kurtosis</b> |
| WISC IV-PRI               | 0.11                      | -0.82           | 1.77            | 0.08                      | 0.06            | 0.22            |
| VMI                       | 0.13*                     | -0.04           | -1.34           | 0.09                      | -0.02           | -0.46           |

\*  $p<0.05$

WISC IV-PRI= Perceptual Reasoning Index, VMI=Beery-Buktenica Visuomotor integration test score

### 3.5.8 Processing speed

An independent  $t$ -test was used to compare performance between the two groups on processing speed. The results showed that there was a significant difference between the Control group ( $M=106.21$ ,  $SD=13.80$ ) and Secure Welfare group ( $M=91.98$ ,  $SD=15.05$ ) on WISC IV- PSI,  $t(99)= -4.96$ ,  $p=0.001$ , indicating that the Secure Welfare group had significantly slower processing speed when compared to the Control group. The effect size estimate of  $d=1.0$  can be considered as large. Normality analysis is presented in Table 19, indicating Kolmogorov-Smirnov Statistic and skewness and kurtosis values for both groups on the processing speed measures.

Table 19

Distribution characteristics for the Control (CO) and Secure Welfare (SW) groups on processing speed

| Cognitive Measures | CO (n=52)          |          |          | SW (n=49)          |          |          |
|--------------------|--------------------|----------|----------|--------------------|----------|----------|
|                    | Kolmogorov-Smirnov | Skewness | Kurtosis | Kolmogorov-Smirnov | Skewness | Kurtosis |
| WISC IV-PSI        | 0.09               | 0.04     | -1.07    | 0.11               | 0.68     | 2.18     |

WISC IV- PSI= Wechsler Intelligence Scale for Children IV-Processing Speed Index

### *3.6 Cognitive variables that significantly predict group membership*

A binary logistic regression using a forward stepwise method (Field, 2009) to elucidate which cognitive variables significantly predicted group membership was used in the analysis. Group membership (i.e. Control and Secure Welfare) was the dependent variable, whilst all the cognitive variables and the affective variables (depression, anxiety and PTSD) were entered as independent variables. The significant predictors in the model, Beta ( $\beta$ ) and Standard Error of Beta ( $SE \beta$ ) are reported and other statistics associated with binary logistic regression analysis are listed in Table 20.

Table 20

Significant predictor variables of group membership using binary logistic regression

| Predictor              | $\beta$ | $SE \beta$ | Wald's $\chi^2$ | $df$ | $p$      | $e^\beta$<br>(odds ratio) |
|------------------------|---------|------------|-----------------|------|----------|---------------------------|
| Constant               | 11.48   | 3.11       | 13.63           | 1    | 0.001*** | -                         |
| SSST                   | -0.45   | 0.22       | 4.32            | 1    | 0.038*   | 0.64                      |
| RAVLT - Delayed Recall | -0.28   | 0.11       | 6.91            | 1    | 0.009**  | 0.76                      |
| WISC IV- PRI           | -0.07   | 0.03       | 4.95            | 1    | 0.026*   | 0.94                      |
| Stroop C/W             | -0.09   | 0.04       | 5.28            | 1    | 0.022*   | 0.92                      |
| TSCC -Dep              | 0.28    | 0.07       | 15.60           | 1    | 0.001*** | 1.32                      |

\*  $p < 0.05$  \*\*  $p < 0.01$  \*\*\*  $p < 0.001$ **Note:**  $R^2 = .47$  (Hosmer & Lemeshow),  $.48$  (Cox & Snell),  $.64$  (Nagelkerke).

SSST=Swanson Sentence Span Task total score, interference trial score, RAVLT-Delayed Recall= Rey Auditory Verbal Learning Test delayed recall trial score, WISC IV-PRI= WISC IV-Perceptual Reasoning Index, Stroop- C/W= Stroop colour/word score, , TSCC-Dep= Trauma Symptom Checklist for Children Depression score.

With alpha set at 0.05, the results of the binary logistic regression were significant, indicating that the Swanson Sentence Span Task, RAVLT A7- Delayed Recall, WISC IV-PRI, Stroop C/W, and TSCC Depression contributed significantly to the model. The model,  $\chi^2(1) = 66.37$ ,  $p = 0.001$ , demonstrated that these variables significantly predicted group membership, with the Secure Welfare group performing considerably lower on these variables in comparison to the Control group. The final model was able to correctly predict group membership for 80.2% of participants on the basis of the significant predictor variables listed above. A total of 78.8% of participants could be correctly identified as being in the control group, whilst 81.6% of participants could be correctly identified as Secure Welfare participants.

### 3.7 Affective Functioning

MANOVA was used to compare the results of the Control and Secure Welfare groups on measures of the Trauma Symptom Checklist (TSCC). Means ( $M$ ) and standard deviations ( $SD$ ) of the scores for the measures of affective function are given in Table 21.



Table 21

Trauma Symptom Checklist for Children (TSCC) scores for the Control and Secure Welfare groups

|                  | CO group<br>(n=52) |      | SW group<br>(n=49) |       | F<br>Statistic | p-value  | Cohen's<br>d |
|------------------|--------------------|------|--------------------|-------|----------------|----------|--------------|
|                  | M                  | SD   | M                  | SD    |                |          |              |
| <b>Measures:</b> |                    |      |                    |       |                |          |              |
| <b>Affective</b> |                    |      |                    |       |                |          |              |
| TSCC-Dep         | 46.71              | 7.77 | 56.47              | 16.23 | 15.12          | 0.001*** | 0.78         |
| TSCC-Anx         | 47.77              | 7.50 | 51.63              | 13.31 | 3.28           | 0.073    | -            |
| TSCC-PTS         | 48.52              | 9.23 | 56.08              | 13.56 | 10.84          | 0.001*** | 0.66         |

\*\*\*  $p < 0.001$  TSCC-Dep= Trauma Symptom Checklist for Children Depression score, TSCC-Anx= Trauma Symptom Checklist for Children Anxiety score, TSCC-PTS= Trauma Symptom Checklist for Children Post Traumatic Stress score.

The Pillai's trace statistic showed that there were significant differences between the two groups on the combined affective measures,  $V = 0.17$ ,  $F(3,97) = 6.69$ ,  $p = 0.001$ . The univariate tests showed that there were significant differences between the groups on the TSCC measures of depression and posttraumatic stress, with the Secure Welfare group indicating significantly higher scores on these measures in comparison to the Control group. The effect sizes for TSS-Dep and TSCC-PTS were considered large and medium respectively.

### 3.7.1 Relationship between affective functioning and cognitive performance

Pearson's Bivariate correlations were used to examine whether there were relationships between cognitive performance and affective functioning for the Control Group and Secure Welfare Group. Tables 22 (Control group) and 23 (Secure Welfare group) represent all the cognitive and affective measures and their associated correlation coefficients ( $r$ ).

Table 22

Bivariate correlations between the cognitive and affective measures for the Control group (n=52)

| Cognitive Measures       | TSCC-Dep |                 | TSCC-Anx |                 | TSCC-PTS |                 |
|--------------------------|----------|-----------------|----------|-----------------|----------|-----------------|
|                          | Dep      | <i>p</i> -value | Anx      | <i>p</i> -value | PTS      | <i>p</i> -value |
| WISC IV-FSIQ             | -.24     | 0.081           | .03      | 0.849           | .07      | 0.612           |
| WISC IV-WMI              | -.07     | 0.603           | .15      | 0.287           | .12      | 0.407           |
| SSST                     | -.13     | 0.364           | .17      | 0.227           | .05      | 0.704           |
| RAVLT A6- Retention      | -.14     | 0.326           | .05      | 0.703           | .05      | 0.730           |
| RAVLT A7- Delayed Recall | .05      | 0.735           | .18      | 0.194           | .07      | 0.619           |
| RAVLT- Total             | -.31     | 0.025*          | -.05     | 0.741           | -.08     | 0.598           |
| CAFT -RDS                | -.31     | 0.025*          | -.10     | 0.465           | -.23     | 0.100           |
| CAFT- Size               | .18      | 0.211           | .13      | 0.365           | .29      | 0.039*          |
| COWAT                    | -.38     | 0.006**         | -.25     | 0.075           | -.31     | 0.027*          |
| TMTB                     | .04      | 0.755           | .01      | 0.934           | .01      | 0.927           |
| TMTB- Errors             | -.03     | 0.860           | -.04     | 0.797           | -.01     | 0.980           |
| Stroop- C/W              | -.15     | 0.294           | -.08     | 0.573           | -.01     | 0.970           |
| WISC IV-PRI              | .06      | 0.688           | .10      | 0.468           | .28      | 0.046*          |
| VMI                      | -.03     | 0.862           | .22      | 0.121           | .15      | 0.282           |
| WISC IV-PSI              | -.29     | 0.036*          | -.18     | 0.195           | -.07     | 0.643           |
| PPVT                     | -.04     | 0.778           | .02      | 0.905           | -.08     | 0.586           |
| WISC IV-VCI              | -.11     | 0.448           | .08      | 0.552           | -.12     | 0.396           |

\*  $p < 0.05$  \*\*  $p < 0.01$

Pearson's Bivariate correlations showed that there were statistically significant but low strength associations between TSCC depression and performances on the RAVLT-total,  $r = -.31, p = 0.03$ , CAFT-RDS,  $r = -.31, p = 0.03$ , WISC IV-PSI  $r = -.29, p = 0.04$  and the COWAT,  $r = -.38, p = 0.01$  for the Control group. These results indicate that as the level of depression increases, performances on these measures of learning and executive function decrease. Significant low strength relationships were also

found between TSCC- Post Traumatic Stress and CAFT-Size,  $r=.29$ ,  $p=0.04$  and WISC IV-PRI,  $r=.28$ ,  $p=0.04$ . However these relationships were in the opposite direction, suggesting that perceptual reasoning and executive function performance increases with PTSD symptomatology. A negative low strength relationship was found between TSCC-PTS and the COWAT, suggesting that verbal fluency performance decreases as levels of posttraumatic symptoms increase. Given the large number of correlation coefficients, Bonferroni corrections were applied to avoid inflating Type I error. The use of Bonferroni corrections for tests of significance resulted in the more conservative alpha level of 0.003. When applying this alpha level, none of the correlation coefficients between the cognitive and affective variables were significant for the Control Group.

Table 23

Bivariate correlations between the cognitive and affective measures for the Secure Welfare group (n=49)

| Cognitive Measures       | TSCC-Dep |                 | TSCC-Anx |                 | TSCC-PTS |                 |
|--------------------------|----------|-----------------|----------|-----------------|----------|-----------------|
|                          | Dep      | <i>p</i> -value | Anx      | <i>p</i> -value | PTS      | <i>p</i> -value |
| WISC IV-FSIQ             | -.37     | 0.009**         | .21      | 0.158           | .18      | 0.210           |
| WISC IV-WMI              | -.04     | 0.779           | -.08     | 0.602           | -.13     | 0.360           |
| SSST                     | -.25     | 0.089           | .18      | 0.203           | .15      | 0.301           |
| RAVLT A6- Retention      | .10      | 0.491           | .16      | 0.281           | .13      | 0.370           |
| RAVLT A7- Delayed Recall | .10      | 0.518           | .16      | 0.276           | .12      | 0.399           |
| RAVLT- Total             | .18      | 0.227           | .24      | 0.097           | .11      | 0.435           |
| CAFT -RDS                | -.43     | 0.002**         | .21      | 0.141           | .32      | 0.025*          |
| CAFT- Size               | .24      | 0.105           | .28      | 0.055           | .20      | 0.180           |
| COWAT                    | .35      | 0.015*          | .32      | 0.025*          | .27      | 0.066           |
| TMTB                     | -.30     | 0.036*          | -.24     | 0.101           | -.16     | 0.254           |
| TMTB- Errors             | -.16     | 0.261           | -.12     | 0.422           | -.14     | 0.350           |
| Stroop- C/W              | .11      | 0.457           | .05      | 0.761           | -.05     | 0.719           |
| WISC IV-PRI              | .35      | 0.015*          | .13      | 0.371           | .15      | 0.308           |
| VMI                      | -.02     | 0.657           | .16      | 0.173           | .14      | 0.287           |
| WISC IV-PSI              | .15      | 0.288           | .17      | 0.239           | .18      | 0.299           |
| PPVT                     | .29      | 0.045*          | .17      | 0.238           | .16      | 0.264           |
| WISC IV-VCI              | .29      | 0.046*          | .22      | 0.324           | .15      | 0.437           |

\*  $p < 0.05$  \*\*  $p < 0.01$

Pearson's Bivariate correlations showed that there were statistically significant but low strength associations between TSCC depression and performances on WISC IV-IQ,  $r = -.37$ ,  $p = 0.01$ , COWAT,  $r = .35$ ,  $p = 0.02$ , TMTB,  $r = -.30$ ,  $p = 0.04$ , WISC IV-PRI,  $r = .35$ ,  $p = 0.02$ , PPVT,  $r = .29$ ,  $p = 0.05$  and WISC IV-VCI,  $r = .29$ ,  $p = 0.05$ , for the Secure Welfare group. These relationships were in the unexpected direction, suggesting that as TSCC-depression score increases, so too does performance on these

cognitive variables. A significant moderate strength relationship was found between TSCC- Dep and performance on the CAFT-RDS,  $r=-.43$ ,  $p=0.002$ , indicating that higher scores on the depression scale are significantly related to greater difficulty on the CAFT- Animals by Size subtest. A significant relationship between the TSCC-PTS scale CAFT-RDS score was found  $r=.32$ ,  $p=0.03$ , however this was in the opposite direction. As mentioned previously, the inflation of alpha needs to be considered in these analyses due to the large number of correlation coefficients computed. When applying the alpha level of 0.003, none of the relationships between the cognitive and affective variables were significant for the Secure Welfare Group.

### *3.8 Gender Differences in Cognitive Function*

Analyses were conducted to examine whether gender differences existed within each of the groups in relation to performance on the cognitive variables. MANOVAs and Independent t-tests were used. These results need to be interpreted with caution due to the large differences in sub-sample sizes, in which there were more females than males in both the Control and Secure Welfare Groups. In order to guard against an inflated Type I error rate, Bonferroni corrections were applied and a more conservative alpha level of 0.02 was set to test significance.

#### *3.8.1 Overall cognitive function*

Independent *t*-tests were used to examine whether there were significant differences in overall cognitive performance within the two groups based on gender. Means (*M*) and standard deviations (*SD*) of the scores for overall cognitive function in relation to gender are given in Table 24 for the Control group and the Secure Welfare group

Table 24

Performances on WISC IV-FSIQ as a function of gender for the Control and Secure Welfare groups

| CO group (n=52) |        |      |         |      |       |        |       |         |      |       | SW group (n=49) |  |  |  |  |
|-----------------|--------|------|---------|------|-------|--------|-------|---------|------|-------|-----------------|--|--|--|--|
| Measures:       |        |      |         |      |       |        |       |         |      |       |                 |  |  |  |  |
| Overall         | Males  |      | Females |      |       | Males  |       | Females |      |       |                 |  |  |  |  |
|                 | (n=17) |      | (n=35)  |      |       | (n=13) |       | (n=36)  |      |       |                 |  |  |  |  |
| Cognitive       |        |      |         |      |       |        |       |         |      |       |                 |  |  |  |  |
| Function        | M      | SD   | M       | SD   | p     | M      | SD    | M       | SD   | p     |                 |  |  |  |  |
| WISC IV-FSIQ    | 93.82  | 8.76 | 97.74   | 9.20 | 0.150 | 85.38  | 10.42 | 87.83   | 9.79 | 0.451 |                 |  |  |  |  |

The independent t-test analysis showed that there were no gender differences in overall cognitive function for both the Control and Secure Welfare groups.

### 3.8.2 Memory and learning

MANOVA was used to compare performances based on gender for the Control and Secure Welfare groups on measures of learning and memory. Means (*M*) and standard deviations (*SD*) of the scores for the measures of learning and memory are given in Table 25.

Table 25

Memory and learning performances as a function of gender for the Control and Secure Welfare groups

|                         | CO group ( <i>n</i> =52) |           |                |           |          | SW group ( <i>n</i> =49) |           |                |           |          |
|-------------------------|--------------------------|-----------|----------------|-----------|----------|--------------------------|-----------|----------------|-----------|----------|
| Measures:               |                          |           |                |           |          |                          |           |                |           |          |
| Memory and Learning     | Males                    |           | Females        |           |          | Males                    |           | Females        |           |          |
|                         | <i>(n</i> =17)           |           | <i>(n</i> =35) |           |          | <i>(n</i> =13)           |           | <i>(n</i> =36) |           |          |
|                         | <i>M</i>                 | <i>SD</i> | <i>M</i>       | <i>SD</i> | <i>p</i> | <i>M</i>                 | <i>SD</i> | <i>M</i>       | <i>SD</i> | <i>p</i> |
| RAVLT A6-Retention      | 11.35                    | 2.91      | 12.43          | 2.33      | 0.157    | 8.54                     | 2.93      | 10.11          | 3.16      | 0.124    |
| RAVLT A7-Delayed Recall | 11.12                    | 2.32      | 12.43          | 2.13      | 0.058    | 7.69                     | 3.71      | 9.72           | 4.03      | 0.119    |
| RAVLT- Total            | 53.24                    | 6.82      | 56.26          | 6.82      | 0.140    | 41.54                    | 9.64      | 47.28          | 11.51     | 0.116    |

MANOVA Pillai's Trace showed that there were no significant gender differences in memory and learning skills for both the Control and Secure Welfare groups

### 3.8.3 Working memory

MANOVA was used to compare performances based on gender for the Control and Secure Welfare groups on measures of working memory. Means (*M*) and standard deviations (*SD*) of the scores for the measures of working memory are given in Table 26.

Table 26

Working memory performances as a function of gender for the Control and Secure Welfare groups

| CO group ( <i>n</i> =52) |                 |           |                 |           |          | SW group ( <i>n</i> =49) |           |                 |           |          |
|--------------------------|-----------------|-----------|-----------------|-----------|----------|--------------------------|-----------|-----------------|-----------|----------|
| Measures:                | Males           |           | Females         |           | <i>p</i> | Males                    |           | Females         |           | <i>p</i> |
|                          | ( <i>n</i> =17) |           | ( <i>n</i> =35) |           |          | ( <i>n</i> =13)          |           | ( <i>n</i> =36) |           |          |
| Working Memory           | <i>M</i>        | <i>SD</i> | <i>M</i>        | <i>SD</i> |          | <i>M</i>                 | <i>SD</i> | <i>M</i>        | <i>SD</i> |          |
| WISC IV-WMI              | 96.00           | 11.61     | 97.14           | 12.96     | 0.759    | 92.85                    | 12.29     | 91.00           | 9.14      | 0.573    |
| SSST                     | 3.29            | 1.40      | 3.45            | 1.20      | 0.665    | 2.08                     | 1.80      | 2.22            | 1.42      | 0.770    |

The MANOVA Pillai's Trace statistic showed that there were no significant gender differences in performances on working memory tasks for both the Control and Secure Welfare groups.

### 3.8 Executive functioning and attention

MANOVA was used to compare performances based on gender for the Control and Secure Welfare groups on measures of executive functioning and attention. Means (*M*) and standard deviations (*SD*) of the scores for the measures of executive functioning and attention are given in Table 27.



Table 27

Executive cognition/attention performances as a function of gender for the Control and Secure Welfare groups

| CO group ( <i>n</i> =52)                  |                |           |                |           |          | SW group ( <i>n</i> =49) |           |                |           |          |
|---|----------------|-----------|----------------|-----------|----------|--------------------------|-----------|----------------|-----------|----------|
| Measures:                                 |                |           |                |           |          |                          |           |                |           |          |
| Executive<br>Functioning and<br>Attention | Males          |           | Females        |           | <i>p</i> | Males                    |           | Females        |           | <i>p</i> |
|   | <i>(n</i> =17) |           | <i>(n</i> =35) |           |          | <i>(n</i> =13)           |           | <i>(n</i> =36) |           |          |
|   | <i>M</i>       | <i>SD</i> | <i>M</i>       | <i>SD</i> |          | <i>M</i>                 | <i>SD</i> | <i>M</i>       | <i>SD</i> |          |
| CAFT -RDS                                 | 38.90          | 15.03     | 41.81          | 16.86     | 0.548    | 39.00                    | 20.62     | 43.95          | 18.19     | 0.422    |
| CAFT- Size                                | 13.12          | 2.42      | 11.94          | 2.83      | 0.148    | 11.69                    | 3.61      | 11.36          | 3.74      | 0.784    |
| COWAT                                     | 33.53          | 7.99      | 35.6           | 7.08      | 0.347    | 30.46                    | 11.06     | 31.97          | 11.11     | 0.676    |
| TMTB                                      | 69.77          | 27.66     | 64.53          | 23.78     | 0.483    | 109.69                   | 45.99     | 86.20          | 29.69     | 0.041*   |
| TMTB- Errors                              | 0.82           | 1.29      | 0.40           | 0.60      | 0.110    | 2.54                     | 3.38      | 1.00           | 2.07      | 0.061    |
| Stroop- C/W                               | 40.53          | 10.92     | 43.89          | 8.96      | 0.244    | 30.54                    | 9.38      | 33.83          | 8.69      | 0.257    |

\*  $p < 0.05$

The Pillai's Trace statistic showed that there was a significant gender difference in TMTB performance for the Secure Welfare group. The females performed significantly better than the males on this task.

### 3.8.5 Language

MANOVA was used to compare performances based on gender for the Control and Secure Welfare groups on tasks of language function. Means (*M*) and standard deviations (*SD*) of the scores for the measures of language are given in Table 28.

Table 28

Language performances as a function of gender for the Control and Secure Welfare groups

| CO group (n=52) |          |           |          |           |          | SW group (n=49) |           |          |           |          |
|-----------------|----------|-----------|----------|-----------|----------|-----------------|-----------|----------|-----------|----------|
| Measures:       | Males    |           | Females  |           | <i>p</i> | Males           |           | Females  |           | <i>p</i> |
|                 | (n=17)   |           | (n=35)   |           |          | (n=13)          |           | (n=36)   |           |          |
| Language        | <i>M</i> | <i>SD</i> | <i>M</i> | <i>SD</i> |          | <i>M</i>        | <i>SD</i> | <i>M</i> | <i>SD</i> |          |
| WISC IV-VCI     | 90.24    | 9.21      | 91.46    | 7.99      | 0.625    | 85.23           | 11.42     | 88.08    | 9.76      | 0.392    |
| PPVT            | 101.41   | 17.19     | 97.6     | 11.20     | 0.341    | 94.77           | 10.40     | 91.89    | 12.75     | 0.469    |

The MANOVA Pillai's Trace statistic showed that there were no significant gender differences in performances on language measures for both the Control and Secure Welfare groups.

### 3.8.6 Visuo-perceptual functioning

MANOVA was used to compare performances based on gender for the Control and Secure Welfare groups on tasks of visuo-perceptual function. Means (*M*) and standard deviations (*SD*) of the scores for the measures of visuo-perceptual function are given in Table 29.

Table 29

Visuo-perceptual performances as a function of gender for the Control and Secure Welfare groups

| CO group ( <i>n</i> =52)  |                |           |                |           |          | SW group ( <i>n</i> =49) |           |                |           |          |
|---------------------------|----------------|-----------|----------------|-----------|----------|--------------------------|-----------|----------------|-----------|----------|
| Measures:                 |                |           |                |           |          |                          |           |                |           |          |
| Visuo-perceptual Function | Males          |           | Females        |           |          | Males                    |           | Females        |           |          |
|                           | <i>(n</i> =17) |           | <i>(n</i> =35) |           |          | <i>(n</i> =13)           |           | <i>(n</i> =36) |           |          |
|                           | <i>M</i>       | <i>SD</i> | <i>M</i>       | <i>SD</i> | <i>p</i> | <i>M</i>                 | <i>SD</i> | <i>M</i>       | <i>SD</i> | <i>p</i> |
| WISC IV-PRI               | 96.53          | 12.58     | 98.66          | 7.89      | 0.459    | 89.46                    | 11.07     | 89.64          | 13.25     | 0.966    |
| VMI                       | 109.24         | 12.04     | 106.91         | 9.99      | 0.466    | 95.23                    | 15.16     | 99.31          | 11.40     | 0.318    |

MANOVA Pillai's Trace showed that there were no significant gender differences in performances on visuo-perceptual measures for both the Control and Secure Welfare groups

### 3.8.7 Processing speed

MANOVA was used to compare performances based on gender for the Control and Secure Welfare groups on processing speed. Means (*M*) and standard deviations (*SD*) of the scores for the measure of processing speed are given in Table 30.

Table 30

Processing speed performances as a function of gender for the Control and Secure Welfare groups

| CO group (n=52)  |        |       |         |       |       | SW group (n=49) |       |         |       |       |
|------------------|--------|-------|---------|-------|-------|-----------------|-------|---------|-------|-------|
| Measures:        |        |       |         |       |       |                 |       |         |       |       |
| Processing Speed | Males  |       | Females |       | p     | Males           |       | Females |       | p     |
|                  | (n=17) |       | (n=35)  |       |       | (n=13)          |       | (n=36)  |       |       |
|                  | M      | SD    | M       | SD    |       | M               | SD    | M       | SD    |       |
| WISC IV-PSI      | 101.59 | 14.66 | 108.46  | 12.99 | 0.093 | 86.77           | 15.50 | 93.86   | 14.64 | 0.147 |

An independent samples *t*-test showed that there were no significant gender differences in performances on processing speed for both the Control and Secure Welfare groups.

### 3.8.9 Affective functioning

MANOVA was used to examine whether there were significant differences in affective functioning within the two groups based on gender. Means (*M*) and standard deviations (*SD*) of the scores for all the measures of affective function in relation to gender are given in Table 31 for the Control group and Table 32 for the Secure Welfare group.

Table 31

Affective measures as a function of gender for the Control group

| CO group (n=52) |        |       |         |      |                |         |              |
|-----------------|--------|-------|---------|------|----------------|---------|--------------|
| Measures:       | Males  |       | Females |      | F<br>Statistic | p-value | Cohen's<br>d |
|                 | (n=17) |       | (n=35)  |      |                |         |              |
|                 | M      | SD    | M       | SD   |                |         |              |
| Affective       |        |       |         |      |                |         |              |
| TSCC-Dep        | 50.47  | 8.80  | 44.89   | 6.60 | 6.56           | 0.013** | 0.77         |
| TSCC-Anx        | 51.24  | 7.51  | 46.09   | 6.98 | 5.92           | 0.019** | 0.73         |
| TSCC-PTS        | 51.00  | 10.48 | 47.31   | 8.46 | 1.86           | 0.179   | -            |

\*\*  $p < 0.01$ 

The Pillai's trace statistic from MANOVA showed that there were significant differences between males and females on the combined affective function measures for the Control group,  $V = 0.15$ ,  $F(3,48) = 2.80$ ,  $p = 0.050$ . The univariate tests revealed significant differences between males and females with large effect sizes on TSCC measures of depression and anxiety (see Table 31), where the males showed significantly higher scores on these aspects of affective function in comparison to females. No significant gender differences were found in relation to affective functioning for the Secure Welfare group (Table 32).

Table 32

Affective measures as a function of gender for the Secure Welfare group

| SW group ( <i>n</i> =49) |                |           |                |           |                       |                 |                     |
|--------------------------|----------------|-----------|----------------|-----------|-----------------------|-----------------|---------------------|
| Measures:                | Males          |           | Females        |           | <i>F</i><br>Statistic | <i>p</i> -value | Cohen's<br><i>d</i> |
|                          | <i>(n</i> =13) |           | <i>(n</i> =36) |           |                       |                 |                     |
|                          | <i>M</i>       | <i>SD</i> | <i>M</i>       | <i>SD</i> |                       |                 |                     |
| Affective                |                |           |                |           |                       |                 |                     |
| TSCC-Dep                 | 53.31          | 12.75     | 57.61          | 17.34     | 0.67                  | 0.418           | -                   |
| TSCC-Anx                 | 47.92          | 7.34      | 52.97          | 14.75     | 1.39                  | 0.245           | -                   |
| TSCC-PTS                 | 51.38          | 10.14     | 57.78          | 14.35     | 2.17                  | 0.147           | -                   |

## Chapter 4: Discussion

The results of this study suggested that adolescents with histories of severe child maltreatment performed worse relative to controls on a number of dimensions of cognitive function. Deficits were found in several cognitive domains, including overall cognitive function (FSIQ), memory and learning, working memory, executive functioning and attention, language, visuo-perceptual function and processing speed. These results are discussed in the framework of the particular hypotheses tested in this study.

### *4.1 Hypothesis One: Overall Cognitive Function*

The current study examined the neuropsychological profiles of adolescents with histories of abuse at the severe end of the spectrum. Based on previous studies indicating that experiences of abuse may interfere with the normal developmental progression of the central nervous system (CNS) which relate to deficits in cognitive function, the Secure Welfare (SW) group were expected to have overall cognitive function impairments, as measured by the FSIQ. The results of this study suggested that adolescents with a history of maltreatment (SW group) showed poorer performances on FSIQ in comparison to the demographically matched control (CO) group. The literature of maltreated infants and young children strongly supports a relationship between maltreatment history and poor overall cognitive function (Appelbaum, 1977; De Bellis et al., 2009; Dubowitz et al., 2002; Hoffman-Plotkin & Twentyman, 1984; Mackner et al., 1997; Pears & Fisher, 2005; Sandgrund et al., 1974; L. Singer, 1986; Strathearn et al., 2001). In studies including groups of older children and adolescents which are more reflective of the current sample, significant differences in FSIQ between maltreated and non- maltreated groups were also reported (Carrey et al., 1995; Palmer et al., 1999; Porter et al., 2005). However, in the overall analysis of the Porter et al study, FSIQ was treated as a covariate rather than a dependent variable. The mean FSIQ score for the SW group was almost one standard deviation below the population mean for FSIQ of 100, falling within the low average range. This finding is supported by research showing that abused infants scored

approximately 15 points lower on overall cognitive function as measured by the Bayley Scales of Infant Development (Mackner et al., 1997) and in older children measured by the WISC III- FSIQ score (Palmer et al., 1999).

Issues relating to premorbid cognitive function need to be considered when interpreting these findings. Information about premorbid cognitive function of the sample was not available; therefore it is unknown whether these adolescents were born with limited cognitive skills. The child(ren)'s level of cognitive ability may be a function of parental capacity. There have been reports that parents who abuse their children are likely to have poor cognitive capacities themselves (Kirkham, Schinke, Schilling, Meltzer, & Norelius, 1986). It is also likely that parents who maltreat their children have been maltreated themselves, further demonstrating the complexities of understanding genetic predispositions of intellectual capacity in maltreated samples (De Bellis et al., 2009). De Bellis and colleagues did take measures to examine the overall cognitive performance of biological parents of the participants, however only a small proportion of the neglected samples were residing with their biological parents. The limited sample size made it difficult to interpret the associations in a statistically sound manner.

There is also research to suggest that children with intellectual deficiencies are more susceptible to maltreatment experiences (Herrenkohl, Herrenkohl, Rupert, Egolf, & Lutz, 1993). There is a strong possibility that many of these adolescents were born with lower than average cognitive abilities, however there is substantial evidence to suggest that the experience of maltreatment relates to a number of neurophysiological processes that have degenerative effects on brain structure and function.

#### *4.1.1 Intellectual Disability*

Studies have also reported that a high proportion of maltreated children demonstrate FSIQ scores that fall within the intellectually disabled (ID) range (i.e. FSIQ score below 70). In the current study 12.5% of the SW participants fell within the ID range, whilst none of the CO participants obtained a score below 70 on FSIQ. Although this data was excluded from the overall analysis, it is important to consider why such a high proportion of abused adolescents fall within the ID range. These



participants, although suspected of having cognitive difficulties, were not identified as being ID before being assessed for the purposes of participating in this study. As a result, it could be assumed that they have struggled through various aspects of life, particularly school, without the supports available to intellectually disabled individuals from government and community agencies.

Estimated prevalence of ID has been reported to be up to a figure of three percent in the general population internationally, and up to two percent in the Australian population (Wen, 1997). This suggests that intellectual disability is overrepresented in maltreated populations. The discrepancy is quite important as these data suggests that there is approximately 10 percent more cases of ID in this maltreated sample when compared to the general community. One explanation is that infants and children born with an ID are at greater risk of being abused (Wolfe, 1985), which may be the reason why there is an overrepresentation of ID in abused samples. Alternatively, this may be the consequence of an altered developmental trajectory in response to child maltreatment early in life and further repeated incidences of maltreatment may affect brain development to an extent that leads to severe cognitive impairment representative of ID. These adolescents, in the past, may have had a capacity above the ID range, though over time, with prolonged experiences of maltreatment, may have encountered adverse brain development that resulted in cognitive decline that formally put them into the ID range.

There is a substantial body of evidence to suggest that the development of the brain is largely experience-dependent (e.g. R. J. Davidson, 1994; Glaser, 2000; Perry, 2002; Perry et al., 1995; W. Singer, 1995; M.H Teicher et al., 2004). It could be posited that all humans are born with equal potential for brain development, where interaction with the environment is responsible for both the initiation and inhibition of developmental sequences. Some would argue that genetic inheritance is an important determinant of intellectual capacity, however it has also been suggested that the genetic potential of the person cannot be unlocked without the influences of the environment (Lerner, 2002). Taking this position that humans have an 'equipotentiality' for brain development means that the limited intellectual capacities of these adolescents may be largely attributed to the maltreatment experience.

#### *4.1.2 Education and FSIQ*

It has been suggested that years of education strongly correlates with FSIQ (Matarazzo & Herman, 1984). In the Carrey et al (1995) study, level of education wasn't reported, whilst the Porter et al (2005) group, included participants who were matched on education, though were not matched on SES. SES has also been identified as an important determinant of FSIQ, particularly in poorer populations (Turkheimer et al., 2003).

Most studies in this area have not identified years in formal education as a demographic variable for matching, and also have not reported information regarding group differences in years of formal education (Beers & De Bellis, 2002; Carrey et al., 1995; Mezzacappa et al., 2001). The current study showed significant differences between the SW and CO groups based on years in formal education, with the SW group having fewer years in education than the CO group. The reason why many studies fail to report information regarding education may be due to the difficulties in obtaining reliable information relating to educational history from this population. Furthermore, matching based on years in formal education is a major difficulty when working with abused samples as it has been identified that they are a group that show high levels of truancy and extended periods of absence from school in comparison to non-abused groups (Nugent et al., 1998).

Another issue pertaining to education history is the notion of formal education versus non-formal education. Many adolescents that present to secure welfare abscond from placements, and may be deemed homeless, or engage in prostitution type work over an extended period of time. When placed in such circumstances, these adolescents may be exposed to experiences of 'non-formal education', providing skills that are required to adapt to the pressures of these environments. Such experiences are difficult to quantify in terms of what constitutes the common definition of education. In addition, how this type of education reflects in skills that are examined using traditional measures of cognitive function is largely unknown.

#### *4.2 Hypothesis 2: Memory and Learning*

Previous research findings have shown that maltreated children and adolescents demonstrate impaired performances on measures of memory and learning (Beers & De Bellis, 2002; De Bellis et al., 2009; Porter et al., 2005). The impact of maltreatment on these capacities may be related to the effect these experiences have on normal processes of brain development during childhood and adolescence.

It has been shown that the myelination of nerve fibres has a protracted development (Thatcher et al., 1987; Yakovlev & Lecours, 1967). Furthermore, it has also been shown that various regions of the brain experience increases and decreases of volume due to neuron production and synaptic pruning respectively, from infancy to adulthood (Giedd, 2004; Gogtay et al., 2004; Huttenlocher, 1979; Huttenlocher & Dabholkar, 1997; Jernigan et al., 1991; Sowell et al., 1999; Sowell et al., 2004; Sowell et al., 2002). The brain structures responsible for learning and memory capacity have been identified in the medial temporal lobes and the lateral prefrontal cortex (Canli et al., 2000; Fell et al., 2003; Kirchhoff et al., 2000; Otten et al., 2002a, 2002b; Squire & Zola-Morgan, 1991).

It would be expected that improvement in memory and learning skills occurs alongside the neurodevelopment taking place in these structures during childhood and adolescence. Research has shown that memory and learning capacity increases with age in child and adolescent samples (Anderson & Lajoie, 1996; Kramer et al., 1997; Schneider & Pressley, 1997; Simcock & Hayne, 2003). Anderson and Lajoie (1996) reported that children experienced two developmental spurts in the capacity to take in new information, the first at approximately eight years of age and the second at 12 years of age.

As the brain is in a significant period of plasticity during childhood and adolescence, the development of these skills is also largely dependent on environmental experiences that trigger neural activity and enhance synaptic connections within the brain (S. J. Martin & Morris, 2002; Perry et al., 1995). It has been suggested that the brain is most vulnerable to trauma during periods of significant brain plasticity (M.H Teicher et al., 2003). Repeated high levels of stress (such as that experienced in relation to child maltreatment) during these periods may

lead to neuronal changes in regions of the brain most sensitive to stress hormones (De Bellis, Baum et al., 1999). The hippocampus is one of the brain structures most commonly associated with the crucial memory processes of consolidating experiences into long term memory, and has been identified as the region most vulnerable to damage from excessive glucocorticoids (Packan & Sapolsky, 1990; Sapolsky et al., 1985; Sapolsky et al., 1990). Brain imaging studies of individuals with histories of abuse have shown that they have significantly reduced hippocampal size in comparison to controls (Bremner & Narayan, 1998; Bremner, Randall, Scott, Capelli et al., 1995; Bremner et al., 1997; Bremner et al., 2003). Altered hippocampal development in relation to traumatic stress may be related to the learning and memory deficits identified in the SW group.

Some have suggested that the considerable development of memory and learning skills that takes place during early childhood coincides with the initiation of formal education, where memory strategies are required to perform successfully at school (Schneider et al., 2002; Sharp et al., 1979). Furthermore, when compared to adults, children who experienced training in the use of mnemonic strategies were more readily able to utilise the strategies and showed associated enhancement in memory and learning skills (Brehmer et al., 2007; Brehmer et al., 2008). As stated previously, adolescents with histories of maltreatment have inconsistent educational histories, characterised by frequent truancy and large absences from school. The impact of this on development of memory and learning is unknown, though it could be speculated that it may be an important factor contributing to the poorer performances on memory and learning measures in the SW group. Disrupted schooling and poor attendance may limit a child's access to experiences of memory strategy use, thus not having the stimulation necessary to initiate neurodevelopment in brain structures associated with memory and learning.

The literature has also indicated that gender differences in learning and memory performances are evident in child and adolescent populations (Kramer et al., 1997). The results of this study demonstrated that no such differences existed for both the SW and CO groups. In support of this finding, other studies have also suggested that differences in memory and learning performance between boys and girls were

minimal and making gender based distinctions was unnecessary in child and adolescent populations (Anderson & Lajoie, 1996).

#### *4.3 Hypothesis 3: Executive Functioning and Attention*

The findings of this study showed that the SW group performed at a significantly lower level compared to the CO group on specific measures of executive function and attention. These measures included: (i) Trail Making Test Part B (TMTB), which provided a measure of attention, mental flexibility, and cognitive set shifting, (ii) Stroop Colour/Word Test (Stroop), which provided a measure of perceptual set shifting and inhibitory control. Examination of the data showed significant correlations between performances on these measures, suggesting that they tap into similar skills of executive function. Similarly, performances on the verbal fluency tasks also correlated well, however no significant differences were found between the two groups on these measures including the (iii) Controlled Oral Word Association Test (COWAT) and the (iv) Controlled Animal Fluency Test (CAFT), animals by size score and relative difficulty score. These findings indicate that the SW and CO group showed comparable abilities in relation to basic level skills of executive function. Impairments were pronounced in the SW group for those tasks that were higher in complexity, requiring the use of a number of cognitive skills simultaneously.

Attentional difficulties and executive impairment have been identified in maltreated populations (Beers & De Bellis, 2002; De Bellis et al., 2009; DePrince, Weinzierl, & Combs, 2009; Mezzacappa et al., 2001; Navalta et al., 2006; Porter et al., 2005). The results of Beers and De Bellis indicated that children with abuse-related PTSD demonstrated significantly poorer performances on the Stroop test, Digit Vigilance Test, Wisconsin Card Sorting Test categories and the animal naming version of the COWAT. These measures examine the executive skills of inhibitory control, attention, problem solving and verbal fluency. The findings, with the exception of verbal fluency deficits are consistent with the current study. Although it needs to be considered that alongside PTSD, Beers and De Bellis' sample also had a number of comorbid psychiatric conditions (e.g. major depression) and behavioural

disorders (e.g. oppositional defiant disorder), which might be one explanation for the additional deficits in executive function not identified in the current sample. These aspects were considered part of the exclusionary criteria of a recent study investigating the effects of PTSD and childhood neglect experiences on cognitive performance. It was shown that neglected children with and without PTSD had deficits on measures of attention and executive function as well as a number of other cognitive domains, corroborating with the findings of this report (De Bellis et al., 2009).

Porter et al (2005) found significant differences on the attention and concentration index of the Test of Memory and Learning (TOMAL), however this finding was not significant after controlling for FSIQ and SES. If we follow the previously articulated position that it is not appropriate to covary for variables such as FSIQ, particularly when there are large differences between groups based on these measures (Adams et al., 1985, 1992; Dennis et al., 2009), then treating FSIQ as a dependent variable as in the Porter et al study would not have been problematic, however their samples were significantly different on SES, with the maltreatment group having a statistically lower mean SES. The lower scores of the maltreated group on measures of attention and concentration may have been related to lower SES, which has been related to cognitive performance (Turkheimer et al., 2003).

In line with the current findings, other studies have also found that maltreated groups demonstrate deficits on measures of inhibitory control (Mezzacappa et al., 2001; Navalta et al., 2006). These reports used measures termed Stop Signal Tasks or GO/NO-GO/STOP tasks where participants were required to react to specified target stimuli presented on a computer screen. These tasks have been associated with frontal brain function, and particularly the orbitofrontal cortex using functional neuroimaging (Casey, Giedd, & Thomas, 2000; Fuster, 1989; Kawishama, Satoh, Itoh, Yanagisawa, & Fukuda, 1996). The development of the right orbitofrontal cortex has been strongly associated with the mother-infant attachment relationship (Schorre, 2001b, 2001c; Seigal, 1999). Children who do not have access to an attachment figure during infancy and early childhood have been shown to develop interrelational difficulties and poor self-regulatory behaviours (Perry, 2001; van der Kolk & Fisler, 1994). Given that many maltreated children are deprived of this early attachment relationship, poor

inhibitory control and self regulation as measured by formal neuropsychological tasks may be a manifestation of limited orbitofrontal development.

There is substantial evidence to suggest that skills of executive functioning and attention undergo a considerable level of development beginning in childhood and continuing to the early years of adulthood (Isquith et al., 2004; Kray et al., 2004; Luna et al., 2004; Luna & Sweeney, 2001). The observed improvement in executive function capacity has been associated with maturation in the frontal lobes of the brain (Anderson et al., 2001; Blakemore & Choudhury, 2006; H. S. Levin et al., 1991; Luciana et al., 2005; Thatcher et al., 1987). The development of executive function occurs over a longer time span as the brain appears to mature in a posterior-anterior progression, where the frontal regions are the last to develop (Gogtay et al., 2004). The prefrontal cortex has been identified as one of the major mediators of executive function, organising neural input from a range of brain structures (Blakemore & Choudhury, 2006; Fuster, 1989; Sowell et al., 2002; Stuss et al., 2002). This suggests that executive functioning capacity is not exclusive to the frontal regions, but is a product of a complex interrelated network of various structures of the brain. Executive function impairment is commonly associated with frontal lobe pathology; however it can also result from a disruption within the networks that connect to the prefrontal regions (M. Alexander & Stuss, 2000).

It appears that the progressive improvement in some aspects of executive capacity is restricted in maltreated populations. In this study, the skills of perceptual set shifting, cognitive flexibility and impulse control were impaired in the SW group when compared to controls. The developmental literature has shown that set shifting skills are established as early as seven years of age (Klenberg et al., 2001), whilst others suggest that they do not reach maturity until 13 years of age (M. C. Davidson et al., 2006). It has been indicated that skills of inhibitory control mature at age 11 years (Brocki & Bohlin, 2004) although others have argued that they are not established until 15 years of age (Luna et al., 2004). The average age of the SW group was approximately 14.5 years, which would suggest, according to previous research, that these skills of executive function and attention would be largely developed in these individuals. The mechanism underlying this lack of development is difficult to ascertain, however there is evidence to suggest that repeated episodes of stress can

alter neural development and can even lead to cell death in various brain regions, including the frontal lobes (Arnsten & Shansky, 2004; Sapolsky, 1996). Navalta et al (2006) made reference to data indicating that sexual abuse experienced during early childhood affected the grey matter volume of primitive brain structures, whilst abuse experienced during late childhood and adolescence altered the neocortex. There is considerable evidence to suggest that the SW group has had repeated experiences of maltreatment in early childhood and adolescence. Although neuro-imaging methods were not utilised in this study, it could be argued based on Navalta et al's findings, that the SW group have experienced volumetric brain changes in both primitive and neocortical regions. These neocortical changes, if present, may be one explanation for the executive function impairments identified in the SW group.

#### *4.4 Hypothesis 4: Language*

As expected the hypothesis that SW participants would perform significantly more poorly on measures of receptive and expressive language was supported. The research of maltreated children and adolescents in this area has consistently reported language deficits using both formal and observational methods (Alessandri, 1991; Allen & Wasserman, 1985; Coster et al., 1989; Culp et al., 1991; De Bellis et al., 2009; Dubowitz et al., 2002; Fox et al., 1988; Hoffman-Plotkin & Twentyman, 1984; McFayden & Kitson, 1996; Oates et al., 1995; Pears & Fisher, 2005).

It has been theorised for some years that significant language development occurs during the early years of childhood known as the critical period. Evidence to support this theory has come from case studies of children severely deprived of language input during this period (Fromkin et al., 1984; Koluchova, 1972). Such case studies demonstrate that children deprived of language stimulation show very little ability in spoken language, and show very little improvement after being removed from their home environments. Studies of individuals who try to learn a second language also show that those who start before the adolescent period are more competent in the new language than those who begin after this time (Collier, 1987; J. Johnson & Newport, 1989; Oyama, 1976). This notion of a critical period for language development can also be associated with the research that suggests that



neuronal structural changes and interconnectivity are dependent on environmental input (Perry, 2002; Pfefferbaum et al., 1994; Schore, 2001c).

Parents of abused children commonly experience episodes of substance use, domestic violence and psychopathology (Dunlap et al., 2002; Widom, 1989; Wolfe, 1985). As a result, their children are deprived of the normal parent-child interactions that foster communicative development. It has been shown that maltreated infants with mothers who did not respond to their needs and provided limited verbal stimulation showed significant deficits in their communicative ability (Allen & Wasserman, 1985; Coster et al., 1989; Koluchova, 1972). Such deficits may be a result of limited language interactions during the critical period for language development.

At the structural level, functional MRI studies of children and adolescents have indicated areas supporting language functioning in both the left and right hemispheres became activated when listening to speech and using skills of expressive language (Molfese & Betz, 1988; Szaflarski et al., 2006; Wood et al., 2004). However there is wide literature support for the view that the left hemisphere is primarily responsible for expressive language function (e.g. Szaflarski et al., 2002). Progressive activation of these areas coincides with considerable changes in neural developmental and synaptic refinement up to the age of 13 years (Hahne et al., 2004; Huttenlocher & Dabholkar, 1997; A. B. Scheibel, 1990). Maltreated children may not experience the same level of structural changes that occur in the brain associated with language input compared to children who come from non-abusive environments. The adolescents that made up the SW sample in this study, share maltreatment experiences that are considered to be most severe, with many having a significant number of notifications of abuse reported for over 15 years. These intermittent experiences of maltreatment and the unavailability of caregivers provide very little opportunities to experience normal social interaction. It could be stated that these adolescents have had very little stimulation necessary to support language development during the critical period. This can be related to the considerable impairments in expressive and receptive language skills identified following assessment.

#### *4.5 Hypothesis 5: Visuo-perceptual Function*

Significant differences emerged between the SW group and the CO group on measures of visuo-perceptual function of the visual analysis type. As predicted, the SW group performed at an inferior level in comparison to the CO group. Pears and Fisher (2005) also demonstrated that preschool children with experiences of maltreatment had significant difficulty in completing measures of visuo-perceptual function on the NEPSY. Similar deficits on NEPSY measures of visuospatial performance of neglected children with and without PTSD have been identified by De Bellis et al (2009). Others also reported differences in visuospatial performance using Rey-Osterreith Complex Figure (ROCF), however they did not remain significant after corrections were made for multiple comparisons (Beers & De Bellis, 2002). The ROCF was also included in the Palmer et al (1999) study, however no significant differences were shown between the two groups on this measure.

The localisation of visuo-perceptual function in the brain, guided by the available literature, has been difficult to ascertain. It is still considered to be largely mediated by the right hemisphere, however it is also understood that it relies on communication with the left hemisphere and structures of the hindbrain (Damasio, 1985; Delis et al., 1986; Schmahmann & Sherman, 1998; Wallesch & Horn, 1990). In adult patients with lesions of the cerebellum, prominent impairments of visuo-perceptual function were identified, however this was not the case in children and adolescents with similar injuries (Schmahmann & Sherman, 1998; Wallesch & Horn, 1990). Visuo-perceptual deficits in children and adolescents have been associated with damage to the corpus callosum (Verger et al., 2001). It has been suggested that visuo-perceptual function is established quite early in childhood (Giudice et al., 2000; Stiles & Stern, 2001; Tada & Stiles, 1996; van Mier, 2006), whilst others have argued that it continues to develop through adolescence (Diamond, 2000; Rueckriegel et al., 2008).

Neuroimaging studies have shown that children with abuse related PTSD have significantly reduced corpus callosal size in comparison to controls (De Bellis, Keshavan et al., 1999). Furthermore, it has also been indicated that poor attachment relationships early in life correspond with poor right hemisphere development

(Schoe, 2000a, 2001b, 2001c, 2005). Together, these findings provide some evidence for the visuo-perceptual difficulties identified in maltreated children and adolescents.

#### *4.6 Hypothesis 6: Relationship between Cognitive Performance and Affective Functioning*

As disorders of affective functioning are commonly reported in maltreated samples, it was important to consider how these issues related to cognitive performance in the SW group. The relationship between the presence of psychopathology such as depression and anxiety and poor cognitive functioning has been well documented (Bremner, Randall, Scott, Capelli et al., 1995; Bremner et al., 2004; Geuze et al., 2009; Jelinek et al., 2006; Jenkins et al., 1998; Johnsen & Asbjornsen, 2008; Johnsen et al., 2008; Yehuda et al., 2004; Yehuda et al., 1995a). It has also been described in abused samples with PTSD (Bremner, Randall, Scott, Capelli et al., 1995; Bremner et al., 2004).

As expected, the results of this study showed that the SW group showed significantly higher levels of depression and posttraumatic stress (PTS) related symptomatology in comparison to controls. There were a few relationships between the cognitive and affective variables for both the CO and SW groups although these were not particularly strong, and were no longer significant once Bonferroni corrections were applied. The literature suggests that clinically depressed adult patients show evidence of impaired cognitive functioning, particularly in skills of memory, attention and executive function (N. F. Gould et al., 2007; R. L. Levin et al., 2007; MacQueen et al., 2003; Taylor- Tavares et al., 2007). The minimal affects that variables of depression, anxiety and PTS have on the cognitive functioning of maltreated participants in this sample may indicate that the mechanisms underlying cognitive impairments in abused populations may also be responsible for higher levels of psychopathology. In support of this theory, although unexpected, De Bellis et al (2009) found that PTSD diagnosis did not differentiate the cognitive performances (with the exception of one measure of delayed recall) of neglected children. However, PTSD symptomatology was significantly associated with poorer performance on a

number of cognitive variables included in the analysis. The authors suggested that methodological issues may have been responsible for these results.

Using neuroimaging methods, reductions of hippocampal size have been identified in depressed patients and individuals with PTSD (S. Becker & Wojtowicz, 2006; Bremner, Randall, Scott, Bronen et al., 1995; Gilbertson et al., 2002; Gurvits et al., 1996; Lindauer et al., 2006). Similar deficits of hippocampal structure have been observed in PTSD patients with histories of child abuse and victims of domestic violence (Bremner et al., 1997; Bremner et al., 2003). As symptoms of anxiety commonly co-occur with depression and PTS, it has been suggested that stress hormone release in response to activation of the HPA axis may be responsible for these hippocampal deficits (Brunsen et al., 2001; Moghaddam, 2002; Sapolsky et al., 1990). The neurobiological changes that occur in the presence of stress hormones may also lead to further degeneration of brain structures (Duric & McCarson, 2005; E. Gould et al., 1997; C. Li et al., 2005). This indicates that child maltreatment related stress may be associated with structural brain changes, particularly in regions of the hippocampus. Furthermore, the degeneration of the hippocampus may not only account for the memory and learning deficits in victims of abuse, but also the depressive and PTS symptoms. Thus, there is a strong possibility that the brain impairments seen in abused populations are also responsible for psychopathological symptoms, rather than vice versa.

One of the limitations of this study was that, due to limited resources, the presence of psychopathological disorders was not formally diagnosed in the sample. Although diagnosis of psychiatric or behavioural disorders formed part of the exclusionary criteria, it may be that some of the participants had clinical issues that were unidentified. The Trauma Symptom Checklist for Children (TSCC) (Briere, 1996), was used to examine symptomatology related to depression, anxiety and PTS, however, the information derived from it is not substantial enough to form the primary basis of diagnosis. Rather, the manual suggests that it should be used alongside other measures of trauma related symptomatology, including direct interview regarding DSM-IV criteria for the diagnosis of specific psychiatric conditions such as PTSD. Diagnosis of PTSD according to DSM-IV criteria was undertaken by a similar study of the effects of maltreatment and PTSD on cognitive

functioning (De Bellis et al., 2009). The Kiddie Schedule for Affective Disorders and Schizophrenia- Present and Lifetime Version (K-SADS-PL) was also used. Despite these rigorous methods of clinical assessment, the results indicated that there were only minimal statistically significant differences between the neglected participants diagnosed with PTSD and those without. However, statistically significant negative associations between the cognitive and PTSD variables were found. The findings presented in this report are inconsistent as the affective variables were not associated with poor performance on the cognitive variables, possibly related to the measure of affective functioning (TSCC) used in this study.

The TSCC is a self report measure of symptom domains associated with traumatic experiences. According to clinical reports, one criticism of the TSCC is that children and adolescents with histories of maltreatment have a tendency to under-report their experiences. The TSCC does have scales to identify those that under-report and over-report their symptoms; however the profiles of those that score above the cut off for under or over reporting are deemed invalid. Although profiles identified as invalid do not suggest that the individual has not experienced maltreatment, or other trauma, it does affect the ability to form a meaningful interpretation about the individual's symptoms (Briere, 1996). For younger children, aged eight to twelve, it has been suggested that both child and parent/caregiver complete alternate versions of the TSCC in order to overcome problems of under- and over-reporting (Lanktree et al., 2008). This would have been near impossible in the current study as it was difficult to access the caregivers of the SW group participants, although should be attempted in future research. Despite its limitations the TSCC has been shown to correlate well with other measures of trauma symptomatology and affective functioning (X. Li et al., 2009; Praver, DiGiuseppe, Pelcovitz, Mandel, & Gaines, 2000; Sadowski & Friedrich, 2000). It has also been recognised for its respectable normative sample of 3008 non-clinical children and adolescents aged seven to seventeen from various ethnic and socioeconomic groups (Drake, Bush, & van Gorp, 2001). Other trauma measures of this type have been criticised for using small normative samples that were not representative of the general population.

Considering these issues, other forms of understanding affective functioning and psychopathology such as clinical interview would be desirable. However this was

beyond the scope of this study and it appears from the available literature, that the TSCC, despite its pitfalls, is an appropriate measure of trauma related symptoms in abused and non-abused populations. Briere (1996) asserts that the TSCC is not diagnostic, although a number of studies support the construct validity of the TSCC (D. M. Elliot et al., 1995; Evans et al., 1994; Lanktree & Briere, 1995), suggesting that reasonable conclusions regarding affective functioning can be made from this measure.

#### *4.7 Deficits in other Domains of Cognitive Function*

A significant finding of this study was that the SW group showed impairments in other domains of cognitive function not previously identified by research in this area. The SW group had significant deficits in working memory capacity and processing speed when compared to the control group, these impairments have not been previously reported in maltreated samples. One explanation for these findings is that the SW group are considered to represent adolescents with histories of maltreatment at the most severe end of the spectrum, with many having experienced all four defined types of maltreatment. Research suggests that there are a number of factors associated with greater maltreatment severity, these include; abuse experiences associated with greater degrees of harm (Carrey et al., 1995) such as sexual penetration and involvement in interactions of domestic violence between caregivers, number of abuse episodes, maltreatment duration and perpetrator-child relationship (Palmer et al., 1999; Porter et al., 2005) and multiple out-of-home care placements (Carrey et al., 1995; Goodman, 1996; Pears & Fisher, 2005). Clinical reports would suggest that all of these factors have been experienced at arguably the greatest degree by participants who made up the SW group.

Maltreatment severity for each of the participants in the SW group was ascertained using a measure included in the Take 2 Harm Consequences Assessment (Take 2 HCA), where it is indicated as concerning, serious and extreme (mild, moderate and severe respectively). One of the major shortcomings of the maltreatment literature is that there is no commonly agreed upon standard of rating

maltreatment severity. Thus it is difficult to compare how cognitive performance differs between samples in terms of maltreatment severity. As almost 94 percent of the SW group fell in the severe category of maltreatment, it was not possible to investigate how variable levels of severity differentially impacted cognitive performance. Studies that have included measures of severity have identified poorer cognitive performances in those samples rated as having experienced greater degrees of maltreatment severity (Carrey et al., 1995; Palmer et al., 1999). Others that have not reported severity factors have shown evidence of cognitive impairment (Beers & De Bellis, 2002), whilst others have not (Samet, 1997).

The issues underlying difficulties in determining severity of maltreatment would also be apparent when attempting to ascertain maltreatment onset, duration and frequency. This study primarily relied on Department of Human Services (DHS) child protection files and Take 2 case records for the purposes of obtaining information regarding maltreatment history. A major limitation of this method is that all information regarding maltreatment history is recorded from the time at which the first notification of child abuse to DHS child protection was made. A number of cases may have been identified after the first experience of abuse, although, for the majority, maltreatment was likely to be present for a considerable period before it came to the attention of DHS. As a result there is a strong possibility that duration of maltreatment was underestimated for the SW group. The findings showed minimal strength associations between maltreatment duration and measures of learning and memory, however these were deemed only exploratory and should be interpreted with caution. Similarly, other studies that included measures of maltreatment duration found no relationships with cognitive performance (Carrey et al., 1995; Porter et al., 2005). Mezzacappa et al (2001) suggested that obtaining reliable reports of maltreatment history was difficult and accordingly did not consider maltreatment duration as a variable relating to cognitive performance in the main analysis.

#### *4.7.1 Working memory*

Working memory capacity has been described in terms of three component processes governed by an attentional controlling system known as the central executive (Baddeley, 1986, 1992, 2000; Baddeley & Hitch, 1974). Structurally, working memory has been associated with the frontal lobes and the parietal regions of

the brain (Collete & van der Linden, 2002; Owen et al., 1999; R. S. Scheibel & Levin, 2004; E. E. Smith & Jonides, 1998). The prefrontal cortex in particular has been identified as one of the major structures related to working memory performance (Curtis & D'Esposito, 2003; Curtis et al., 2000; Owen et al., 1999).

Functional neuroimaging of children and adolescents has shown progressive activation of the frontal and parietal regions associated with working memory performance (Klingberg et al., 2002; Kwon et al., 2002; Luciana & Nelson, 2002). Developmentally, working memory capacity appears to mature over an extended period, and has been shown to continue into the early years of adulthood (Luna et al., 2004).

According to Arnsten and Shansky (2004), during adolescence, the prefrontal cortex (PFC) is especially susceptible to the effects of stress hormones, and neurotransmitters released during the stress response. Even mild episodes of stress have been associated with working memory deficits related to impaired PFC functioning in adolescents (Arnsten, 1999; Arnsten & Goldman-Rakic, 1998; Arnsten & Shansky, 2004). From the data of animal studies, these authors propose that in the stage of adolescence, high levels of dopamine are released in response to stress that relates to impaired PFC function. Furthermore, oestrogen has been reported to have similar effects on PFC function suggesting that female adolescents have even greater susceptibility to impaired working memory capacity as a result of stress (Shansky et al., 2003). Repeated episodes of traumatic stress related to maltreatment may be the mechanism underlying poor working memory performance in abused samples. Maltreatment related trauma would be considered a significant source of stress for the adolescents in the SW group as they represent the most severe of maltreatment cases. They are also highly likely to experience stressors related to their lifestyle, where issues of highly transient placements, periods of homelessness and engaging in high risk behaviours such as prostitution and other criminal behaviours predominate. Such circumstances would relate to insurmountable levels of stress that may coincide with continuous elevated secretion of stress hormones resulting in impaired functioning of the PFC. At the extreme, levels of stress hormones considered to be neurotoxic may damage sensitive brain structures, such as the PFC, resulting in permanent working memory impairment.



#### *4.7.2 Processing speed*

It was found that the SW group performed significantly slower on tasks of processing speed in comparison to the control group. This was identified in terms of performance on the WISC IV Processing Speed Index (PSI). Beers and De Bellis (2002) included other measures of processing speed and found no significant differences in performance between the control and maltreated group with PTSD. However, the small number of participants in the Beers and De Bellis study may have been related to the insignificant differences found in this cognitive domain.

The most significant development in processing speed has been suggested to occur between the ages of approximately seven and nine years (Anderson et al., 2001; Brocki & Bohlin, 2004). Processing speed capacity has been related to performance on a variety of cognitive tasks (Bull & Johnston, 1997; DiLalla, 2000; Fry & Hale, 1996; Kail, 2000; Kail & Hall, 1994; Wechsler, 2003). The extent of myelination within the brain has been suggested to coincide with speed of processing (Posthuma et al., 2003). Evidence of slowed processing ability has been shown in children and adolescents with lesions to the corpus callosum, one of the largest myelinated tracts in the brain (Verger et al., 2001).

The process of myelination may be affected in individuals who experience repeated episodes of stress, and maltreated populations in particular are at risk of experiencing prolonged intermittent episodes of stress. It has been shown in animal studies that high levels of stress hormones interfere with the development of Schwann cells and oligodendrocytes, the cells that are responsible for producing the myelin sheath surrounding nerve tracts in the peripheral nervous system and the central nervous system respectively (Bohn, 1980). Whether this occurs in humans who experience high levels of stress is largely unknown, although it could be speculated that the processing speed deficits shown in the maltreated group may be a result of disrupted myelination within the brain. It has been suggested that maltreatment duration, rather than age of abuse onset, may have a strong relationship with white matter development as it is a process that continues over a long period of the lifespan (Navalta et al., 2006). A number of brain structures, including the corpus callosum undergo continuous myelination from childhood to young adulthood (Schaefer et al., 1990). In humans, disrupted myelination as a result of stress may be evidenced by the

identification of smaller corpus callosum size in maltreated participants (De Bellis, Keshavan et al., 1999). The corpus callosum has been associated with impaired processing speed in individuals who have experienced damage to this area (Verger et al., 2001). In abused participants, deficits in processing speed may be explained by limited myelination of the corpus callosum, and possibly other nerve tracts within the brain.

#### *4.8 Gender Differences in Cognitive and Affective Functioning*

The literature examining gender differences in cognitive functioning shows mixed results. Although there is a body of evidence to suggest that females perform better on measures of language and males have greater visuospatial abilities (Gur et al., 2000; Gur et al., 1999; Halpern & Wright, 1996; Hedges & Nowell, 1995; Voyer et al., 1995; Weiss et al., 2003), others have suggested that no such gender differences exist in these cognitive skills (Hyde & Linn, 1988). Interestingly, the results of the present study showed that there was only a significant gender difference on the TMTB task for the SW group. No other gender differences were found on the cognitive variables for both the SW and CO groups. However, these findings need to be interpreted with caution as there were approximately twice as many females than males in both the SW and CO groups, thus sampling issues may be an explanation for these findings.

Scores on affective measures as a function of gender was also examined in this study. For the CO group, significant differences between males and females were found on measures of depression and anxiety, with males demonstrating higher levels of symptoms than males. This was an unexpected finding and may be related to the two to one ratio of females to males in the sample. Although in the SW group, no significant differences in aspects of affective functioning were found. In normal adolescent populations it had been shown that females have a greater susceptibility to internalising problems in comparison males (Hankin & Abramson, 2001; Lewinsohn, Lewinsohn, Gotlib, Seeley, & Allen, 1998; Nolen-Hoeksema & Girgus, 1994). The difference is even more pronounced in maltreated populations, particularly in child sexual abuse cases, where females show significantly higher levels of internalised

psychopathology, including depression, anxiety and PTS (Cutler & Nolen-Hoeksema, 1991; Feiring, Taska, & Lewis, 1999; MacMillan et al., 2001). Contrastingly, males with histories of abuse are more likely to show externalising behaviours, such as criminality, aggression and overt sexualisation (Feiring et al., 1999; Garnefski & Diekstra, 1997). The similar levels of depression, anxiety and PTS shown in males and females of the SW group may suggest that they share comparable experiences of abuse and family environment that relate to the development of psychopathology. A high proportion of males in the SW group experienced all types of abuse, including sexual abuse, which has been shown to relate to greater levels of psychological symptoms.

#### *4.9 The Relationship between Child Maltreatment and Cognitive Function*

There are a number of hypotheses regarding the mechanisms underlying the cognitive deficits in maltreated populations. The SW group showed a range of impairments in all cognitive domains, suggesting that a number of different processes related to the abuse experience may be affecting brain development and function.

##### *4.9.1 Attachment*

The quality of the early attachment relationship between caregiver and child has been strongly implicated in the developmental processes that occur in specific regions of the brain. The development of the right hemisphere, and particularly the right orbitofrontal cortex, has been associated with the presence of early attachment experiences (Schore, 2001b, 2001c; Seigal, 1999). The right orbitofrontal cortex is a region of the brain that forms part of a larger network that mediates social, emotional and self regulatory function (Balbernie, 2001; Schore, 1994, 2001b). Evidence of executive function deficits were found in the SW group, particularly those skills associated with self regulatory behaviours and cognitive flexibility. The SW group also demonstrated higher levels of depressive symptomatology. These impairments may be associated with the integrity of the orbitofrontal cortex.

It has been identified that children who have been maltreated develop maladaptive attachment styles due to the unavailability of consistent caregivers

(Carlson et al., 1989; Schore, 2001a). These insecure attachment styles are characterised by difficulties in regulating relational stress, emotional behaviour and higher levels of psychopathological symptoms (Beijersbergen et al., 2008; Briere & Elliot, 1994; Morton & Browne, 1998; Perry, 2001; van der Kolk & Fisler, 1994). More recently, it has been shown that maltreated adolescents with insecure attachments perform lower on measures of attention, working memory and cognitive efficiency (Webster, Kist-Hackett, & Joubert, 2009). It could be argued that maltreated children have limited right brain development, particularly in the orbitofrontal region, due to poor attachment relationships early in life. This corresponds with the executive function impairments and depressive symptoms found in maltreated samples. The interpersonal difficulties demonstrated by maltreated children and adolescents may be associated with these executive deficits. Skills of self-regulation, inhibitory control and cognitive flexibility are fundamental to successful relational processes, and it is quite clear that abused children and adolescents are lacking in these abilities. This may provide further evidence that deprivation of attachment experiences during infancy results in impaired right brain development and function.

#### *4.9.2 Stress and cognitive development*

The impact of stress hormones on cognitive development has been one of the major theories relating the experience of maltreatment to impaired brain development and function. It has been suggested that repeated episodes of maltreatment coincide with recurring activation of the hypothalamic-pituitary-adrenal (HPA) axis (De Bellis, 2004). The HPA axis is responsible for releasing glucocorticoids which have target receptors in many parts of the body, including the brain. These activate physiological processes that enhance responses in the face of threatening stimuli (De Bellis, Baum et al., 1999). The mothers of maltreated children are highly likely to be involved in abusive relationships (Kolbo, 1996), thus also experiencing persistent activation of the HPA axis. In animal studies, high levels of circulating glucocorticoids in the blood stream during pregnancy have been shown to affect the developing foetus (Dunlop et al., 1997).

Clinical tests have shown that victims of abuse and trauma demonstrate significantly higher levels of glucocorticoids, suggestive of dysregulation of the HPA

axis (Davies et al., 2008; De Bellis et al., 1994; Kaufman et al., 1997; Lemieux & Coe, 1995; Perry, 2001; Perry & Azad, 1999; Perry & Pollard, 1998). It has been shown that high levels of glucocorticoids can have deleterious effects on neuronal structure and function (De Bellis, Keshavan et al., 1999; Sapolsky, 1996), especially during periods of neuronal migration, differentiation and synaptogenesis (De Bellis, Baum et al., 1999). Animal studies have also provided some evidence to indicate that glucocorticoids affect myelination of nerve tracts (Bohn, 1980). Infant animals exposed to high concentrations of glucocorticoids prior to birth have shown reduced myelination of the CNS (Dunlop et al., 1997), reduced brain weight at term (Huang et al., 1999) and neuronal loss within the hippocampus (Uno et al., 1990). Neuroimaging studies of maltreated children with PTSD have shown that they have significantly smaller brain sizes, particularly in the corpus callosal region (De Bellis, Keshavan et al., 1999). The hippocampus has been shown to be particularly sensitive to high concentrations of glucocorticoids (Armanini et al., 1990; Packan & Sapolsky, 1990; Sapolsky et al., 1985; Sapolsky et al., 1990; Uno et al., 1989). Neuroimaging studies have shown that adult combat war veterans with PTSD and women with histories of sexual abuse have significantly reduced hippocampal volumes in comparison to controls (Bremner, Randall, Scott, Bronen et al., 1995; Bremner, Randall, Scott, Capelli et al., 1995; Bremner et al., 1997; Gurvits et al., 1996).

Individuals at risk for high levels of stress, such as maltreatment victims, adult war veterans and normal adolescents have shown evidence of various cognitive impairments. The cognitive domains reportedly affected include; learning and memory (Bremner et al., 1996; Yehuda et al., 1995a), executive functioning , attention and working memory (Arnsten, 1999; Arnsten & Goldman-Rakic, 1998; Barrett et al., 1996; Gunnar, 1998) and visuospatial function (Barrett et al., 1996).

The evidence provided by the stress hypothesis provides a compelling argument for the various deficits seen in maltreated populations. These children and adolescents are at great risk for being exposed to high levels of stress hormones before and after being born. Animal studies have indicated that this has detrimental effects on CNS development, processes of myelination and neuronal structures. These findings support reports that maltreated individuals have significantly smaller intracranial volumes, corpus callosa and hippocampi. Some of the cognitive deficits

seen in the SW may be explained by the effect that stress hormones have on brain structure and function. The significantly lower FSIQ scores of the SW group may be a consequence of reduced intracranial volumes due to glucocorticoid related deterioration. Hippocampal degeneration has also been commonly reported in maltreatment victims and combat war veterans, providing a possible explanation for the memory and learning deficits seen in the SW group. It has been suggested that in normal adolescents, stress hormones alter neurotransmitter release resulting in altered prefrontal cortical function. The SW group would be expected to have experienced levels of stress beyond those of teenagers dealing with the normal pressures of adolescence. These high levels of stress may have produced permanent deficits in prefrontal function leading to the executive and working memory impairments identified in this study. Processes of myelination are thought to be affected by stress hormones, the corpus callosum is one of the largest collections of myelinated tracts in the CNS. Visuo-spatial performance has been associated with activation of the corpus callosum, therefore the visuo-perceptual deficits seen in the SW group may be a consequence of reduced myelination of the corpus callosum, however such conclusions can only be tentative.

#### *4.9.3 Traumatic brain injury*

Although history of head injuries were questioned in this study, the idea that adolescents in the SW group may have had injuries of this nature during early childhood, needs to be strongly considered. Injuries that possibly occurred in infancy may have not been recalled due to the inability to access memories that occurred prior to age two and a half years, commonly known as ‘infantile amnesia’. The severity of abuse experienced by the SW group also provides some indication that significant physical injuries are likely to have occurred.

The literature of physically abused children and adolescents indicates that a large proportion experience forces to the head leading to skull fractures and intracranial injuries (Leventhal et al., 1993; Merten et al., 1984; Merten et al., 1983; J. A. O'Neill et al., 1973). Studies have also shown that physically abused children who have come to the attention to medical services commonly have indications of previously unidentified head injuries (Ewing-Cobbs, Kramer et al., 1998; Jenny et al., 1999). Thus it is sensible to assume that a large number of maltreated children with

head injuries remain unknown, particularly if they haven't received medical attention. Making such assumptions has important implications for the relationship between childhood abuse and cognitive functioning.

Traumatic brain injuries (TBI) sustained during childhood and especially those that are characteristic of Shaken Baby Syndrome (SBS) are associated with significant deficits of cognition. The deficits reported in the childhood TBI literature include; attention, processing speed, memory (Ewing-Cobbs, Prasad et al., 1998; van Heugten et al., 2006), working memory, inhibitory control (Ewing-Cobbs, Prasad et al., 1998; Ewing-Cobbs, Prasad et al., 2004) and verbal learning (Roman et al., 1998). Children with SBS have profound neuropsychological difficulties over all the major cognitive domains, which has also been represented in FSIQ scores that fall one standard deviation below the mean (Stipanovic et al., 2008). Therefore, the impairments seen in severely maltreated populations may be a manifestation of TBIs experienced during infancy and early childhood. It could, however, be suggested that the presence of these injuries and the added effects of the abuse experience may produce cognitive deficits beyond those expected of children with TBIs and no maltreatment history. As a result, the presence of TBI or SBS in addition to poor early attachment relationships and repeated experiences of stress may have cumulative effects on brain development, structure and function, leading to significant cognitive difficulties. Studies that have examined the cognitive effects of inflicted TBI in children suggest that those from an environment of abuse and neglect have significantly poorer outcomes (Landry et al., 2004). Others have also shown that family environment and SES had a significant impact on long term outcomes following severe TBI (Schwartz et al., 2003; Taylor et al., 2002).

This study was limited in that accurate information regarding developmental and medical history that may have provided some evidence of childhood TBI was unavailable. The disorganised and transient lifestyles characteristic of abused populations makes it extremely difficult to collect complete information of this type. Parents are often unreachable, and may not have complete recollections of their child's history due to issues of psychopathology, substance abuse and domestic violence (Dunlap et al., 2002; Widom, 1989; Wolfe, 1985). Another problem is that adolescents who come to the attention of Secure Welfare services are likely to have

had frequent out-of-home placement changes, with many experiencing moves to different geographical locations. In these instances, medical and developmental records do not necessarily follow the child with each placement change. Only information reported in DHS child protection and Take 2 case files were available for the purposes of determining developmental history. Generally, the information provided in these documents was only available for the period after the first notification of abuse. Information about the young person's development and medical history prior to the first notification was in most cases unreported. This has significant implications, as it is unknown what number of the adolescents involved in this study had experiences of childhood traumatic brain injuries and prenatal drug exposure, experiences that are known to affect cognitive functioning.

#### *4.10 Substance Use and Cognitive Function*

Developmentally, the adolescent period is characterised by experimentation and risk taking (Spear, 2002, 2004a, 2004b). This coincides with the increased use of drug and alcohol seen during the adolescent years (Melchior et al., 2008). The level of use is even more pronounced in adolescents with histories of maltreatment (De Bellis, 2002; Malinosky-Rummell & Hansen, 1993). Large proportions of adolescents with histories of maltreatment have been shown to engage in the abuse of a range of substances, and initiate use at a younger age in comparison to age matched peers (Harrison et al., 1997). The entire SW group reported that they engaged in some type of substance abuse, and approximately 90 percent suggested that they had used a number of drugs concurrently. Given that behaviours of illicit substance abuse are characteristic of the SW group, it is essential to consider the role that these substances play in brain development and function. As parents of child maltreatment victims commonly share experiences of substance abuse (Jaudes et al., 1995) it is also very possible that these adolescents have been exposed to substances during gestation. Therefore, the relationship between prenatal drug exposure and cognitive functioning will also be examined.

A common finding reported in the research is that children who have been exposed to drugs prenatally show evidence of impaired arousal and regulatory



systems (Eiden et al., 2009; Mayes, 2002). Prefrontal deficits coincide with these difficulties, which have also been evidenced by impaired executive functioning (Azuma & Chasnoff, 1993; Chang et al., 2004; Frank et al., 2001; Mattson & Riley, 1998; Noland et al., 2005; Rasmussen & Bisanz, 2009). It has also been shown that children exposed to substances in utero show poorer performances on measures of overall cognitive function (Alessandri et al., 1998; Behnke et al., 2002; Huizink & Mulder, 2006; Jacobson et al., 2004; Mattson & Riley, 1998; L. Singer et al., 1997; L. T. Singer et al., 2004), motor and visuospatial function (Behnke et al., 2002; Chang et al., 2005; Frank et al., 2001; Mattson & Riley, 1998; L. Singer et al., 1997), language skills (Bandstra et al., 2002), memory and learning (Bandstra et al., 2002) and processing speed (Burden et al., 2005; Jacobson et al., 1993). The range of cognitive impairments identified in the research relating to children and adolescents exposed to prenatal substance use are very similar to those presented by the SW group. Thus, there is a strong possibility that the impairments identified in the SW group are a consequence of being exposed to drugs prior to being born. However, it has also been suggested that environmental factors bear great influence on the extent of cognitive impairment (Rasmussen & Bisanz, 2009). This means that children and adolescents with histories of prenatal drug exposure may have cognitive deficits that are further compounded by environmental influences, including childhood abuse and neglect.

As a large majority of the adolescents in the SW group engaged in polysubstance abuse, the following section will consider how such practices relate to impaired brain structure and function. It has been suggested that victims of maltreatment are likely to abuse a variety of substances as a means of coping with emotional disturbances, particularly if those disturbances are associated with psychopathology (Labouvie, 1986; Rosselli & Ardila, 1998; Ullman et al., 2006). Although, it has been reported that polysubstance abuse is also common in normal adolescent populations (Australian Institute of Health and Welfare, 2007).

Polysubstance abuse has been associated with the most extensive of cognitive impairments as different drugs interact with different regions of the brain (Rogers & Robbins, 2001; Selby & Azrin, 1998). The limited data available suggests that adolescents who use multiple substances show evidence of abnormalities of brain structure and function. A functional MRI study indicated that polysubstance abusing

adolescents showed impaired activation of frontal regions associated with completing a working memory task (Schweinsburg et al., 2005). Learning, memory and attentional difficulties have also been shown in adolescents who abuse a number of substances concurrently, and these deficits were still apparent at eight years follow up (Tapert & Brown, 1999; Tapert et al., 2002). Polysubstance abuse may be an explanation for some of the deficits in abused populations, however, the number of deficits related to multiple substance abuse do not account for all those seen in the SW group involved in this study. Similar to previous reports, it is highly likely that these substances are influencing brain structure and function, however the additional pressures of the maltreating environment may be the reason for the number and severity of cognitive impairments exhibited by the SW group.

Issues of substance abuse appear to be characteristic of individuals who present to Secure Welfare. Controlling for these factors would be ideal in order to separate the role of maltreatment history from substance effects on cognitive performance. However, this poses major practical difficulties, and including substance use in the exclusionary criteria of this study would have eliminated almost the entire sample. Obtaining a comparable group of adolescent participants without experiences of abuse and comparable drug use history also has its complications. Community samples of adolescents who engage in the use of illicit substances would be difficult to recruit due to the issues of legality associated with drug use of this type. Furthermore, attempting to match participants on type, extent and history of drug use would be particularly challenging. Adolescents who make up the Secure Welfare group have greater access to 'harder' drugs as many of them are known to engage with individuals who can obtain such substances. Many of the female SW participants in particular, have had experiences of engaging in prostitution type work, a profession which is notorious for illegal drug use practices (Potterat, Rothenberg, Muth, Darrow, & Phillips-Plummer, 1998). Attempting to engage a community sample of adolescents with similar levels of drug use seen in the SW group in this kind of research would be most difficult, although should be considered for the purposes of future research in this area.

Access to accurate information regarding substance use history of the SW and CO groups was another limitation of this research. Participants were asked what

substances they had used for a period of longer than three months over the course of their lives. Given that detail regarding substance use was largely dependent on self report, reliability of this information is questionable. For the SW group, information about substance use history was also obtained from the DHS CPD document; however for many of the cases this type of information was not reported. The study lacked an accurate measure of substance abuse information that included prenatal drug exposure, total lifetime and frequency of use; therefore it was difficult to examine how differing experiences of substance use affected the cognitive variables. Some studies examining the effects of abuse history on cognitive performance have failed to include information regarding participant substance use history (Mezzacappa et al., 2001; Porter et al., 2005), whilst others included history of substance use and prenatal substance exposure in their exclusionary criteria (Beers & De Bellis, 2002; Navalta et al., 2006; Porter et al., 2005). It is important to consider how substance abuse interacts with brain structure and function in abused samples, therefore appropriate measures to obtain this information is necessary. However, excluding participants on the basis of substance using history would significantly limit sample sizes, and more importantly, would exclude a special group of participants with abuse histories. It has been shown that individuals with severe abuse experiences are at high risk for developing substance use issues. Excluding these participants would limit the ability to show how experiences of severe abuse affect brain structure and function. Furthermore, it has been suggested that the neurotoxic effects of drugs and alcohol are further compounded by the abusive environment, thus it could be assumed that the cognitive deficits seen in individuals who have maltreatment and substance use histories cannot solely be attributed to substance abuse alone.

#### *4.11 Assessment and Referral Bias*

There is a large body of psychological literature about the subtle effects of unintentional experimenter influences on research assessment protocols (e.g. Forster, 2000; Kaplan & Saccuzzo, 2001; Rosenthal, 1966; Sheldrake, 1998), however a number of procedures were undertaken to ensure that such effects were largely minimised in the current study. It was ensured that the assessment protocol was performed in a standardised manner with all research participants by an experimenter

who had previous training of conducting neuropsychological assessment protocols. A number of practice assessments with individuals not included in the research sample were also undertaken, and observed by the research supervisor to ensure standard methods were being employed.

Kaplan and Saccuzzo (2001) suggest that individuals who test participants should not be aware of the participants group membership (i.e. experimental or control). This knowledge, although unintentional, may influence how the experimenter interacts with the participants (Kaplan & Saccuzzo, 2001). However it is well known that abused children and adolescents have less capacity to focus on tasks for an extended period of time, particularly when there is evidence of psychopathological symptoms and previous substance abuse. Special considerations were made for the SW group in this study, with many having to complete the assessment protocol over a number of sessions due to motivation and concentration issues. Given these circumstances, it would be evident to the experimenter which participants belonged to each group. Furthermore, a blind study including participants from Secure Welfare would be largely impossible, unless special considerations were made. Adolescents in Secure Welfare reside in a locked facility where leaving the premises is strictly unauthorised, unless the individual has to attend a court hearing, or requires specialised medical care. Similar restrictions are placed on people entering the facility, therefore control participants would not be allowed to enter. As the nature of the study circumstances precluded attempts to follow a blind design, the experimenter took great care in conforming to correct test administration procedures, and the tests utilised were both valid and reliable and have been used effectively in research as well as clinical practice.

Referral bias is another issue related to this research, and it has been identified as an integral part of maltreatment research in general (Sidebotham & Heron, 2006). Those that agreed to participate in the study may represent a select sample of maltreated adolescents that may not characterise the general population of abused youth. It has been suggested that such biases pose some limitations in terms of generalisability of findings, however difficulties in overcoming such issues have been noted (Drotar, 2000).

#### *4.12 Future Research Challenges*

The cognitive effects of child maltreatment definitely need to be investigated in future research. The long-term sequelae of childhood maltreatment is difficult to ascertain, as a number of interacting factors appear to influence cognitive capacity and performance in these populations. Idealistically, obtaining a better understanding of how abuse affects cognitive performance could only be well established using longitudinal designs, where children are assessed prior to being abused, or after first signs of maltreatment (De Bellis et al., 2009). It would be most valuable, if children for example maltreated in the preschool years, were assessed over a period of five to ten years. This would provide a better picture of how child maltreatment affects the developmental trajectory of cognition over time, and would also provide clearer evidence of whether intellectual disability is the cause or effect of maltreatment. Such research could also inform the implementation of interventions to enhance the cognitive skills of maltreated children and adolescents (De Bellis et al., 2009). There are a number of difficulties associated with the development of longitudinal designs. Longitudinal studies are arduous, not only because following up participants over a long period of time is fraught with complications, but also difficult in relation to getting adequate funding and resources in order to complete such long term research, as the majority of research grants last between two to three years (Eskenazi et al., 2005; Kinard, 1994).

Based on the observation that a large proportion of adolescents in the SW group reported histories of substance abuse, it is important to try to control for these issues. It is well known that drugs and alcohol have neurotoxic effects. Although difficult, it is recommended that control samples with similar histories of substance abuse are recruited in order to delineate the effects of child abuse on cognitive function. In the absence of a comparable control group of substance abusing adolescents, measures should be taken place to include questionnaires that provide detailed information about duration, frequency and use of multiple substances over the life span in order to compare how these issues differentially impact cognition.

Determining maltreatment related experiences and developmental history is a particularly difficult task in samples of severely abused children who commonly experience multiple placements. Better measures of these experiences, where possible, should be included in research designs to ensure a greater understanding of how these factors relate to cognitive performance. A number of studies have used the History of Victimization Questionnaire (HVF) in order to obtain information about abuse history (Palmer et al., 1999; Porter et al., 2005), however it is limited in that it requires the participants caregiver or therapist to complete the form, which may not be appropriate for some study samples.

More sophisticated measures of affective function may also be beneficial in order to separate the cognitive effects of these issues from child abuse history. Clinical interviews conducted by psychologists/psychiatrists may provide clearer indications of psychopathology. Careful psychiatric and neuro-radiological evaluation is required to gain a better understanding of abuse related cognitive impairments. A battery of cognitive tests is also necessary to detect specific cognitive deficits that, if present, could subsequently be the focus of more detailed investigations. Supplementary neuroimaging data would provide indications whether the neuropsychological deficits present accompany the expected organic changes in regions specific to particular functions. This would impart significant evidence for the neuropathological mechanisms that possibly underlie the child abuse experience.

#### *4.13 Implications and Conclusion*

It is quite clear that most adolescents with histories of severe maltreatment have profound cognitive difficulties, limiting their ability to perform a range of tasks required of everyday functioning. The level of cognitive impairment seen in this sample may be a function of maltreatment severity and possibly significant psychopathology, and thus needs to be considered in this aspect when attempting to generalise the findings. It is unknown, from this study whether similar cognitive deficiencies are present in individuals with less severe histories of maltreatment. It appears, from the literature, that those who have experienced maltreatment to a lesser

extent show a limited range of cognitive impairments (Mezzacappa et al., 2001; Navalta et al., 2006; Porter et al., 2005).

The lack of data regarding premorbid cognitive functioning, and the possibility that these adolescents were born with lower than average abilities makes it difficult to make firm conclusions about the impact of maltreatment on cognitive performance. However, the multiple levels of converging evidence to support an association with childhood maltreatment experiences and impaired brain structure and function would suggest that it is highly unlikely that the range of deficits seen in maltreated populations are solely a function of premorbid ability.

The hypotheses for the relationships between child maltreatment and cognitive performance suggest that these experiences affect the normal developmental progression of brain structures. There is evidence to suggest that language development occurs within a critical period, where opportunities for communication need to be available in order to establish these skills (Collier, 1987; J. Johnson & Newport, 1989; Oyama, 1976). Maltreated children are commonly neglected of these opportunities, explaining the consistent reports of language deficiencies in maltreated populations. It has been shown that the early attachment relationship has a significant role in the development of the right brain and particularly the orbitofrontal structures (Schoore, 2001b, 2001c; Seigal, 1999). Impaired functioning of these structures relates to deficient emotional regulatory behaviours and visuo-perceptual ability (Balbernie, 2001; Schoore, 1994, 2001b) which coincide with the limited performances on measures of self-regulation and visuo-perceptual function observed in this study.

The theory that maltreated populations have disproportionate levels of stress hormones, known to be toxic to specific brain structures also lends weight to the association between child abuse and cognitive impairment. Stress hormones are thought to disrupt processes of myelination (Bohn, 1980), and also relate to deterioration of brain structures including the hippocampus, frontal lobes (Armanini et al., 1990; Packan & Sapolsky, 1990; Sapolsky et al., 1985; Sapolsky et al., 1990; Uno et al., 1990; Uno et al., 1989) and the corpus callosum (De Bellis, Keshavan et al., 1999). These structural changes may explain the deficiencies in processing speed,

memory and learning, executive function and visuo-perceptual deficits identified in maltreated populations.

The strong possibilities that maltreated populations have had experiences of traumatic brain injury also need to be taken into account when examining the reasons for cognitive impairment. Severely maltreated children are likely to have been subject to multiple types of abuse, including physical abuse. Childhood physical abuse is commonly characterised by; in older children, direct forces to the head, or violent shaking of the upper body in infants (Leventhal et al., 1993; Merten et al., 1984; Merten et al., 1983; J. A. O'Neill et al., 1973; Stipanivic et al., 2008; Talvik et al., 2007). These injuries coincide with both gross and microscopic organic brain changes that are associated with significant cognitive difficulties. This suggests that the cognitive impairments observed in abused children and adolescents may be a direct manifestation of physical trauma, far beyond those explained by the developmental effects of neglect, poor attachment relationships and stress hormones. However, the research that has examined inflicted brain injuries in child populations has proposed that the additional stressors that maltreated children are exposed to, such as maternal capacity, family conflict, parental substance use and neglect, relate to further cognitive deficiencies not explained by the neuropathological effects of the injury itself (Landry et al., 2004; Schwartz et al., 2003; Taylor et al., 2002).

The results of this study can be used to inform the provision of clinical neuropsychological assessment of adolescents who present with a history of childhood maltreatment. This information is particularly beneficial for caregivers of adolescents with histories of maltreatment and the professionals working with them. Anecdotal reports would suggest that these adolescents have been, in the past, identified as problem children with behavioural difficulties. This study provides evidence to suggest that these manifestations are a result of significant impairments of cognitive capacity rather than of character. Routine cognitive assessments of children and adolescents who have experienced maltreatment may allow professionals to have a greater understanding of the behaviours demonstrated by these individuals. Furthermore, this may also allow for early targeted interventions in order to promote further skill development. For example, young children may be provided with language based tasks to help develop their communicative abilities.



As history of child maltreatment is a risk factor for developing substance use and psychiatric disorders, it is also important to identify and treat these problems as early as possible. Substance abuse in particular is known to have significant neurotoxic effects that deteriorate brain structures, leading to deficits of cognition. Psychiatric disorders such as depression, anxiety and PTSD, also affect cognitive performance.

The neuropsychological effects of child maltreatment, still remains a largely undeveloped field of enquiry. There is an apparent need for the comprehensive assessment of large samples of children and adolescents with histories of maltreatment. In order to clearly understand the effects of child maltreatment on cognitive performance, it is important for future research to recruit highly comparable control groups. One of the major shortcomings of this study was that issues of substance could not be controlled for. Despite this limitation, the current study found that the SW group performed very differently to a demographically highly comparable control group on a number of cognitive measures. The SW group showed significantly poorer functioning over a number of cognitive domains, including, overall cognitive function, language, memory and learning, executive functioning and attention, working memory, processing speed and visuo-perceptual function. A follow up study with a similar sample should be conducted with a different range of cognitive tasks to examine whether the same results, in relation to the affected cognitive domains, are replicated. The difficulty in recruiting a comparable control group in terms of substance use may be possible by targeting clinical and community organisations working with substance abusing adolescents. Given that a number of interacting factors are associated with the outcomes of victims of child abuse, it is important for future research to examine how these aspects differentially impact cognitive outcomes. Longitudinal designs, where possible, should be employed to track the progression of cognitive function in maltreated populations, particularly in those who have been taken out of the maltreating environment.

Child abuse prevention is the only way to avoid the significant psychological, cognitive and interpersonal effects of childhood maltreatment. Early detection for those at risk for committing child maltreatment is needed; unfortunately, this is not always possible, as many don't present to services where detection is likely, whilst

some may not show the early warning signs of abuse. In many circumstances, social policy prevents organisations from intervening before substantial evidence of maltreatment has been reported.

Sadly, the prevention of child abuse is an unlikely occurrence, though with early detection and intervention, the outcomes of these children can be significantly improved. Professionals working with maltreated populations need to consider the psychological, social and cognitive impacts of maltreatment, in order to fully understand the experience of the maltreated child.

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## Appendices



## Appendix 1: Demographic questionnaire for the Control group

## Demographic Questionnaire

- Participant Name: \_\_\_\_\_ Research I.D: \_\_\_\_\_
- Date of Testing: \_\_\_\_\_
- Date of Birth: \_\_\_\_\_
- Age: \_\_\_\_\_
- Gender: *M F*
- Current School Year Level: \_\_\_\_\_
- Occupation (parent/guardian):  
\_\_\_\_\_
- Medical History:

*Have you ever had any serious illness?*

*-Major injury? (eg. head injury)*

\_\_\_\_\_

*-Major infection?*

*(eg. Meningitis)* \_\_\_\_\_

*-Period of*

*hospitalization?* \_\_\_\_\_

*-Prolonged period of*

*medication?* \_\_\_\_\_

*Other significant events/issues?* \_\_\_\_\_

## Appendix 2: Demographic questionnaire for the Secure Welfare group

## Demographic Questionnaire

▪ Participant Name: \_\_\_\_\_ Research I.D: \_\_\_\_\_

▪ Date of Testing: \_\_\_\_\_

▪ Date of Birth: \_\_\_\_\_

▪ Age: \_\_\_\_\_

▪ Gender: *M F*

▪ Current School Year Level: \_\_\_\_\_

▪ Occupation (parent/guardian): \_\_\_\_\_

▪ Medical History: \_\_\_\_\_

*Have you ever had any serious illness?*

*-Major injury? (eg. head injury)*

\_\_\_\_\_

*-Major infection?*

*(eg. Meningitis)* \_\_\_\_\_

*-Period of*

*hospitalization?* \_\_\_\_\_

*-Prolonged period of*

*medication?* \_\_\_\_\_

*Other significant events/issues?* \_\_\_\_\_

▪ Abuse History

*Type/s:*      Physical   ☐   Sexual   ☐   Emotional   ☐   Neglect   ☐

*Severity:*      Mild   ☐   Moderate   ☐   Severe   ☐

*Duration (years)* \_\_\_\_\_

*Any further*

*information?* \_\_\_\_\_

\_\_\_\_\_

### Appendix 3: Rey Auditory Verbal Learning Test record form

Participant No:  
Date:

# RAVLT Scoring Sheet

| Recall Trials  |    |    |    |    |    | Recall Trials   |    |    |    |
|----------------|----|----|----|----|----|-----------------|----|----|----|
| List A         | A1 | A2 | A3 | A4 | A5 | List B          | B1 | A6 | A7 |
| <i>Drum</i>    |    |    |    |    |    | <i>Desk</i>     |    |    |    |
| <i>Curtain</i> |    |    |    |    |    | <i>Ranger</i>   |    |    |    |
| <i>Bell</i>    |    |    |    |    |    | <i>Bird</i>     |    |    |    |
| <i>Coffee</i>  |    |    |    |    |    | <i>Shoe</i>     |    |    |    |
| <i>School</i>  |    |    |    |    |    | <i>Stove</i>    |    |    |    |
| <i>Parent</i>  |    |    |    |    |    | <i>Mountain</i> |    |    |    |
| <i>Moon</i>    |    |    |    |    |    | <i>Glasses</i>  |    |    |    |
| <i>Garden</i>  |    |    |    |    |    | <i>Towel</i>    |    |    |    |
| <i>Hat</i>     |    |    |    |    |    | <i>Cloud</i>    |    |    |    |
| <i>Farmer</i>  |    |    |    |    |    | <i>Boat</i>     |    |    |    |
| <i>Nose</i>    |    |    |    |    |    | <i>Lamb</i>     |    |    |    |
| <i>Turkey</i>  |    |    |    |    |    | <i>Gun</i>      |    |    |    |
| <i>Colour</i>  |    |    |    |    |    | <i>Pencil</i>   |    |    |    |
| <i>House</i>   |    |    |    |    |    | <i>Church</i>   |    |    |    |
| <i>River</i>   |    |    |    |    |    | <i>Fish</i>     |    |    |    |
| # correct      |    |    |    |    |    | # correct       |    |    |    |

Total: A1 to A5= \_\_\_\_\_

Trial: A6 - A5= \_\_\_\_\_

Recognition # targets correctly identified: \_\_\_\_\_

The test consists of fifteen nouns which are presented verbally to the participant for five consecutive trials, each trial followed by a free recall test, where the participant is required to reproduce as many words as possible from the list presented. Upon completion of the fifth trial, an interference list of fifteen words is presented, followed by a free recall test of that list. After a twenty-minute delay period, without further presentation of those words, the participant is required to recall the nouns from the first list presented. Finally, a recognition task, where participants are required to identify the nouns from the first list within a larger list of words is completed.

Participant No:  
Date:

Word List for RAVLT Recognition

|         |          |          |          |          |
|---------|----------|----------|----------|----------|
| Bell    | Home     | Towel    | Boat     | Glasses  |
| Window  | Fish     | Curtain  | Hot      | Stocking |
| Hat     | Moon     | Flower   | Parent   | Shoe     |
| Barn    | Tree     | Colour   | Water    | Teacher  |
| Ranger  | Balloon  | Desk     | Farmer   | Stove    |
| Nose    | Bird     | Gun      | Rose     | Nest     |
| Weather | Mountain | Crayon   | Cloud    | Children |
| School  | Coffee   | Church   | House    | Drum     |
| Hand    | Mouse    | Turkey   | Stranger | Toffee   |
| Pencil  | River    | Fountain | Garden   | lamb     |

#### Appendix 4: Swanson Sentence Span Task record form



## Swanson Sentence Span Task

### Instructions:

In this task I will be reading you a series of unrelated sentences to you. Your job is to remember the LAST word of each sentence in the order in which they are read.

First, I will read you a set of sentences. Then I will ask you a question about one of the sentences. Then I will say “Remember” and you are to tell me the last word of each sentence in correct order.

So it’s LISTEN, QUESTION, REMEMBER.

Let’s do some practice ones first. LISTEN as I say the sentences. Then I’ll ask you a QUESTION and then you REMEMBER the last word in each sentence in order.

Ready for the first set?

*NB: Remember to pause for 2 seconds after each sentence in the practice and testing sessions*

### PRACTICE SET 1 (provide feedback)

#### LISTEN

1. Many animals live on a farm.
2. People have used masks since early times.

#### QUESTION

What have been used since early times?

#### REMEMBER

### PRACTICE SET 2

1. The baby’s toy rolled under the bed.
  2. They walked around to the back of the house.
- Q. What rolled under the bed?

### PRACTICE SET 3

1. The squirrel hid the acorns in the hollow tree.
  2. It was so cold, the snow crunched under his feet.
- Q. What crunched?

Now I think you have the idea. Try to remember as much as you can and don’t be afraid to guess about the words or the answers to the questions. But listen carefully

START ALL SUBJECTS AT LEVEL 2. CEILING = 2 SETS WRONG IN A LEVEL.

LEVEL 2

1. Sarah wants you to give her a dollar.
  2. Mary tried to tell her mother the right street.
- Q. Who did Mary tell?

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1. Both of the games were cancelled because of trouble.
  2. Jennifer says she doesn't have time.
- Q. What was cancelled?

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LEVEL 3

1. We waited in line for an hour.
  2. Sally thinks we should give the bird its freedom.
  3. My mother said she would write an excuse.
- Q. Where did we wait?

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1. The cheerleader does not seem to have friends.
  2. Beth can't go because she didn't get shoes.
  3. Bob doesn't want to tell the teacher.
- Q. Who can't go?

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LEVEL 4

1. My little brother went in the wrong restaurant.
  2. The teacher wanted to see me about my book.
  3. You will be sorry if you break the window.
  4. My friend wants to learn about snakes.
- Q. Who will be sorry?

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1. If you work hard you can make a discovery.
  2. We didn't buy the car because of cost.
  3. I would like to know your opinion.
  4. It is important to think about safety.
- Q. What didn't we buy?

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LEVEL 5

1. The broken doll was not my fault.
  2. Joe is having problems with his memory.
  3. I have talked to my parents about the idea.
  4. John is not in a very good mood.
  5. They were all happy to be at the event.
- Q. What was broken?

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1. I can study if you give me a pencil.
  2. Children like to read books about animals.
  3. I will give Cindy the candy in a bowl.
  4. The good news gave Ann a feeling of happiness.
  5. Jeff likes to do homework in ink.
- Q. What will I give Cindy?

---

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TOTAL SETS CORRECT:

\_\_\_\_\_/8

## Appendix 5: Controlled Animal Fluency Test record form

Date:

60 Sec for each category (If S is silent for 15" or more repeat basic instructions)

- [illegible]

## Appendix 6: Controlled Oral Word Association Test record form

Date:

60 sec for each letter (If S is silent for 15sec repeat basic instructions and letter)

I will say a letter of the alphabet. Then I want you to say as many words that begin with that letter as you can. For instance if I say “G”, you might say grass, garden, green. Please do not say any words that are names of places or people, or products. Also, do not give the same word with a different ending such as *run* and *running*.

[illegible]

Total words (F,A & S): \_\_\_\_\_ Total words per minute: \_\_\_\_\_

## Appendix 7: Trail Making Test Part B test sheet

*End*  
13

10

8

9

I

D

B

4

3

7

*Begin*

1

5

H

C

12

G

A

J

2

6

L

E

F

K

11



## Appendix 8: Take Two Harm Consequences Assessment (Blank form)

Client:



## HARM CONSEQUENCES ASSESSMENT

### Part 1 of Referral Tool

To be completed as an initial  
screening tool for the TAKE TWO Program

Version: 16/01/2004



A partnership between Berry Street Victoria and the Austin  
CAMHS, with the support of La Trobe University and Mindful.

Client:

## THE HARM CONSEQUENCES ASSESSMENT:

The Harm Consequences Assessment is based on the Children and Young Person's Act 1989, the Victorian Risk Framework and current literature regarding the impact of abuse and neglect on the wellbeing of children and young people.

The domains included in the Harm Consequences Assessment are derived from Section 63 of the Children and Young Person's Act:

- [a] and [b] Abandonment
- [c] Physical Harm and Injury
- [d] Sexual Abuse
- [e] Emotional and Psychological Harm
- [f] Developmental Harm/Medical

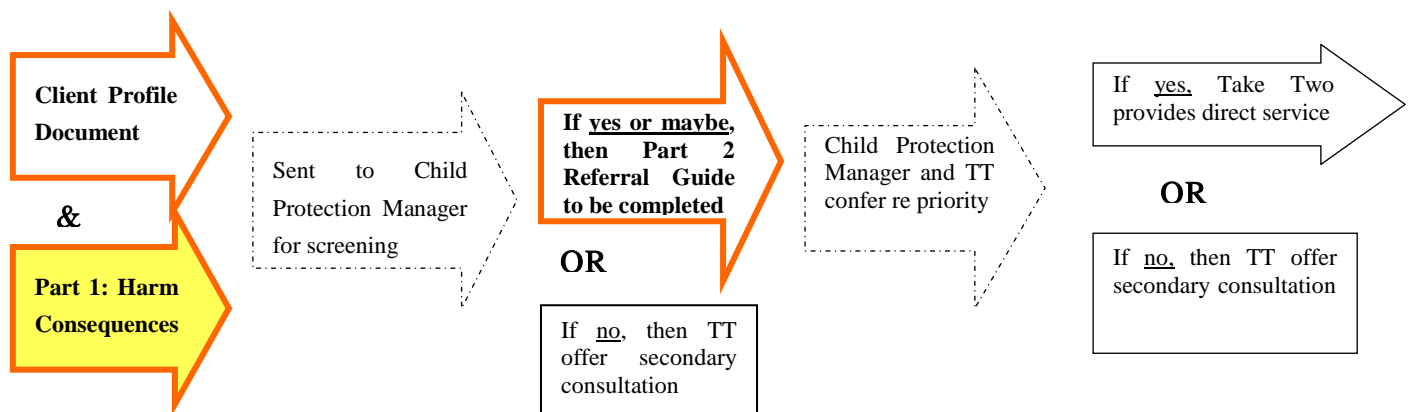
The Harm Consequences Assessment is divided into two sections:

- First section is a list of descriptors of abuse and neglect types and experiences. In other words, what happened to the child?
- Second section is a list of descriptors of the range of harms experienced by children and young people as a consequence of their experience or abuse and/or neglect. In other words, what was the impact on the child?

The Harm Consequences Assessment is used as the basic screen for assisting Child Protection workers/ CSO workers and Child Protection Managers to determine whether a client should be referred to Take Two.

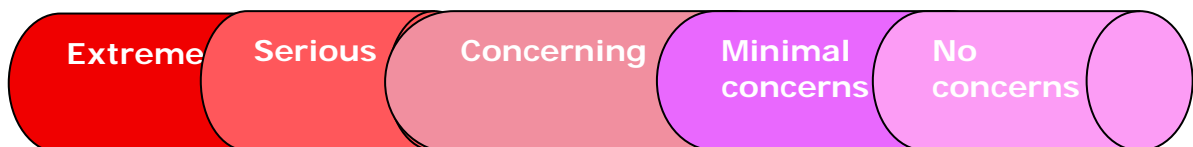
The DHS Client Profile Document should accompany the Harm Consequences Assessment.

If the decision is taken to refer the client to Take Two then the Take Two Referral Guide will need to be completed and the Harm Consequences Assessment and the Client Profile Document will be attached to the Referral Guide and sent to the Child Protection Manager for prioritisation.



## HOW TO COMPLETE HARM CONSEQUENCES ASSESSMENT:

- Tick the relevant abuse/neglect type(s) and experience(s) that have been confirmed or believed to have occurred at any time in the child's history or current situation.
- Tick the relevant impact/consequence(s) of these abuse/neglect experiences as you or others have observed or noted over time, eg mental health diagnoses, other assessments, feedback from carers, parents or school or direct observation.
- There is likely to be more than one consequence of harm and from more than one domain as a result of abuse and these should all be ticked as appropriate. Eg sexual abuse may lead to a range of sexual harms, emotional harms, physical harms and developmental harms.
- If one of the descriptors in the harm consequences section is present for the child, but clearly not a result of abuse or neglect, eg due to a medical condition or disability, then this should not be ticked.
- The comments section at the end of the Harm Consequences Assessment can be used if you believe a descriptor is more or less serious for this child than how it is listed, or if there is a descriptor missing which you believe is pertinent to understanding this child's experience and/or consequences of harm.
- The use of Extreme, Serious and Concerning headings have been derived from the Risk Judgment guide and are on the severe end of the continuum of abuse and neglect. Therefore describing an experience or impact as serious is considered very significant in this context.



- It is envisaged, but not required that clients referred to Take Two would have at least one **extreme** descriptor or perhaps several or many **serious** descriptors in the first section of the Harm Consequences Assessment (What was the abuse/neglect experience) and probably in the second section (What were the harm consequences). In some situations, the impact on the child may not yet be observable, but if extreme or serious abuse and neglect has occurred, it can be predicted that this will lead to harmful impact on the child if therapeutic intervention does not occur.

**Please refer to the User Guide or the Take Two Referral Tool for further instructions.**

Client:

## HARM CONSEQUENCES ASSESSMENT

**COMPLETE THE CLIENT DETAILS SECTION BELOW:**

|  |  |                      |                                |
|--|--|----------------------|--------------------------------|
| CASIS Number                                 | <input type="text"/>                           |                      |                                |
| Child's Name                                 | First Name                                     | Surname              | Age                            |
|  | <input type="text"/>                           | <input type="text"/> | <input type="text" value="1"/> |
| Worker's Name<br>(Person<br>completing tool) | <input type="text"/>                           |                      |                                |
| Date Completed                               | <input type="text" value="1/1/2008"/>          |                      |                                |
| Region                                       | <input type="text" value="Barwon South West"/> |                      |                                |

Client:

**Take Two - Harm Consequences Assessment**  
**Abuse/Neglect experiences of child/young person (What happened to the child/young person?)**

| Domains of abuse / neglect         | Extreme                                       | Serious                                       | Concerning                                    |
|------------------------------------|---|---|---|
| Abandonment / no appropriate carer | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of abuse / neglect         | Extreme                                       | Serious                                       | Concerning                                    |
| Physical Harm and Injury           | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of abuse / neglect         | Extreme                                       | Serious                                       | Concerning                                    |
| Sexual Abuse                       | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of abuse / neglect         | Extreme                                       | Serious                                       | Concerning                                    |
| Emotional and Psychological Harm   | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of abuse / neglect         | Extreme                                       | Serious                                       | Concerning                                    |
| Developmental and Medical Harm     | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |

Client:

**Take Two - Harm Consequences Assessment**

**Harm consequences to child/young person as result of abuse/neglect (What is impact of abuse/ neglect on child/young person?)**

| Domains of harm conseq's           | Extreme                                       | Serious                                       | Concerning                                    |
|------------------------------------|---|---|---|
| Abandonment / no appropriate carer | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of harm conseq's           | Extreme                                       | Serious                                       | Concerning                                    |
| Physical Harm and Injury           | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of harm conseq's           | Extreme                                       | Serious                                       | Concerning                                    |
| Sexual Harm                        | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of harm conseq's           | Extreme                                       | Serious                                       | Concerning                                    |
| Emotional and Psychological Harm   | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |
| Domains of harm conseq's           | Extreme                                       | Serious                                       | Concerning                                    |
| Developmental and Medical Harm     | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> | <a href="#">Click here to make selections</a> |

Client:

## Comments

Please add here any descriptors or comments you consider relevant.  
NB: For more detailed responses please provide in Part 2 (Referral Guide) of the Take Two Referral Tool.

Print a Printer-Friendly copy

*Clicking this button will print your document without the 'Click here to make selections' buttons*



Client:

## OFFICE USE ONLY

*This section is to be used by Berry Street  
Victoria staff only to collate and manage the  
information gathered by this form*

**Show XML**

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This Referral Tool is designed to provide information to assist decision making and planning and is based on the best information at the time of publication. This Referral Tool provides a general guide to appropriate practice, to be followed only subject to individual professional's or organisation's judgement in individual circumstances or contexts.

## Appendix 9: Take Two Harm Consequences Assessment user guide



## USER GUIDE FOR REFERRAL TOOL

User Guide for the Referral Tool for  
prospective TAKE TWO clients

Version: 16/01/2004



A partnership between Berry Street Victoria and the Austin CAMHS,  
with the support of La Trobe University and Mindful.

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## 1. AIM OF USER GUIDE:

This is an interim User Guide to provide a reference point for Child Protection and CSO workers whilst they are completing the TAKE TWO Referral Tool.

Training will be conducted in every region within Victoria from January to March 2004. Following this training, frequently asked questions will be added and final adaptations made to this User Guide. The regional TAKE TWO teams are also available to provide assistance.

## 2. BACKGROUND AND AIM OF TAKE TWO PROGRAM:

TAKE TWO is a new service funded by the Department of Human Services (DHS) and auspiced by Berry Street Victoria, in partnership with the Austin Hospital Child and Adolescent Mental Health Service (CAMHS), La Trobe University, Faculty of Health Sciences, School of Social Work and Social Policy, and Mindful (Centre for Training and Research in Developmental Health).

The aim of TAKE TWO is to significantly enhance the behavioural and emotional functioning, safety and wellbeing of infants, children and young people<sup>1</sup> subject to Child Protection intervention who have been identified as requiring specialist therapeutic and treatment interventions due to the aftermath of abuse and/or neglect. In other words, this program is to respond to Child Protection client's needs for safety; attachment; recovery from trauma; and promotion of their health and well-being, taking account of their context and history.

Children are eligible for the TAKE TWO program if they are substantiated Child Protection clients who have experienced severe abuse or neglect and who are at risk of or already demonstrating behavioural or emotional disturbance. They may be living at home or in any form of out-of-home care. They may or may not be on a Children's Court order.

The objectives of the TAKE TWO program are:

1. To improve outcomes for Child Protection clients through the provision of high quality services to the client group either directly and/or via work with significant others including family, carers, teachers and peers.
2. Working with service providers and planners to improve service provision

Some of the guiding principles underlying the TAKE TWO practice framework are:

- Abuse and neglect occurs along a continuum of severity and chronicity and occurs within a family and social context that needs to be understood, especially in terms of their meaning to the child.
- Significant abuse and neglect are traumatic experiences for children. These experiences place them at risk for developing emotional and behavioural disturbances, which in turn impacts on their ability to form positive relationships with others.
- Abuse and neglect of children within the family context represents a significant disruption to the child's attachment to his/her parents, siblings and significant others.
- Children are understood in their context and connections with their family and community including extended family, friends, day care, schools and service systems.

---

<sup>1</sup> The term 'child' will be used to refer to infant, child or adolescent.

## 4. OVERVIEW OF COMPLETING THE REFERRAL TOOL AND REFERRAL PROCESS:

### 4.1 Overview

The TAKE TWO Referral Tool is a combination of the **Harm Consequences Assessment** (HCA) and the **Referral Guide**. The **HCA** provides an initial screening mechanism to determine whether this is a possible referral for TAKE TWO, or indeed if the child is in need of therapy in general. The **Referral Guide** provides more questions about the child and other information to assist the final decision regarding priority for a specific referral to TAKE TWO. In other words, whilst a case may be screened as appropriate for therapy via the HCA, decisions regarding priority to TAKE TWO will need to be made depending on the number of cases competing for potential acceptance and the program's capacity at the time. The Referral Guide will be used for this prioritisation process.

The **DHS Client Profile Document** (CPD) needs to accompany the HCA as this provides essential information regarding family details, Aboriginality, protective and placement history and health and education information. The availability of this document has avoided the necessity of adding these questions into the TAKE TWO Referral Tool. It is also requested that a **genogram** be attached to the Referral Tool.

If another agency is involved such as VACCA, a contracted CSO, a CSO providing a placement and/or a therapeutic service, it is recommended that this organisation be involved in the consideration of a referral to TAKE TWO. TAKE TWO staff are available to provide assistance or consultation at any stage of the referral process.

Once these documents have been completed on the Microsoft Word templates provided they can also be saved on to the Child Protection CASIS file and/or CSO client file. Within DHS these tools can be emailed to the Child Protection Manager. In order for information to be sent to TAKE TWO the documents have been password protected so as to ensure security of information and are mailed or handed to TAKE TWO on a floppy disc.

The HCA in conjunction with the Client Profile Document is used as the initial screen for assisting workers and Child Protection Managers to determine whether a client should be referred to TAKE TWO. The result of this screening in relation to a referral to TAKE TWO could be a 'yes', 'no' or 'maybe'.

If the Child Protection Manager or delegate decides 'no' then TAKE TWO would be able to provide secondary consultation if required.

If the Child Protection Manager or delegate decides 'yes' or 'maybe' then the worker(s) will be required to complete the Referral Guide. The Referral Guide, HCA, Client Profile Document, genogram and other relevant reports would then be sent to the Child Protection Manager or delegate who, in consultation with the TAKE TWO Senior Clinician, will make a final decision regarding prioritisation for referral to TAKE TWO or secondary consultation including other possible options for therapeutic intervention.

It is envisaged that this prioritisation of referral process will be a collaborative discussion between the Child Protection Manager or delegate and the TAKE TWO Senior Clinician, although the final decision for referral rests with the Child Protection Manager.

### 3. DEVELOPMENT OF THE TAKE TWO REFERRAL TOOL:

A practical referral tool was needed to assist in guiding entry into the TAKE TWO program and to aid the development of consistent and transparent approaches to assessment and treatment within TAKE TWO. It was considered that a component of the tool was required to screen for appropriate referrals, and that another component was then required to prioritise within the range of appropriate referrals.

The three main purposes of the TAKE TWO Referral Tool are:

- To guide and support Child Protection and/or Community Service Organisation (CSO) workers in their understanding of the impact of abuse and/or neglect on children and to understand what this may mean in relation to therapeutic intervention.
- To provide information to assist Child Protection Manager (CPM) and TAKE TWO Senior Clinician (SC) in screening for and prioritising referrals to TAKE TWO.
- To provide information in order for TAKE TWO to allocate the case and begin process of further assessment, engagement and planning.

The development of the TAKE TWO Referral Tool was a collaborative process spearheaded by Professor Shane Thomas (School of Public Health, La Trobe University) and a working group involving Margarita Frederico, (School of Social Work and Social Policy, La Trobe University), Carol Reeves (Community Care Manager, NMR), Karen O'Neill (Specialist Support Services, Child Protection & Juvenile Justice Branch, DHS) and Julie Boffa (Policy and Practice, Child Protection & Juvenile Justice Branch, DHS), Ric Pawsey (Director, TAKE TWO) and Annette Jackson (Research Manager, TAKE TWO).

The process was informed by reviewing the literature regarding predictive factors of behavioural and emotional disturbance, responses to trauma and attachment disruption and the impact of abuse and neglect on children's emotional wellbeing. Specific attention was given to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), International Statistical Classification of Diseases and Related Health Problems (ICD-10), Victorian Risk Framework (VRF) and the Royal Children's Hospital Mental Health Service Stargate program's Trauma and Attachment screen. The working group discussed a range of options in the developmental phase of the referral tool and reported along the way to the TAKE TWO Clinical Practice and Advisory Group (CPAG), the DHS Internal Reference Group, and the TAKE TWO Leadership Group and staff for further consultation.

The referral tool that developed as a result of this process was then piloted in every DHS region. Three to six Child Protection workers in each region completed Referral Tools and provided written and verbal feedback regarding the tools. A small number of CSO workers were also involved in this pilot.

For the purposes of the pilot, Child Protection workers identified cases likely for referral to the TAKE TWO program and completed a draft Referral Tool alongside TAKE TWO staff. Where possible, Child Protection Managers were then asked to meet with the TAKE TWO Senior Clinician to review these tools and provide feedback regarding the tool and its usefulness for prioritisation. Consultation occurred with the Child Protection Professional Development Unit and initial discussions occurred with the Victorian Aboriginal Child Care Agency (VACCA). Based on the pilot and resulting feedback changes were made to the Referral Tool leading to the current version dated 16/01/2004. This version of the Referral Tool will be reviewed in June 2004.

## 4.5 SPECIFIC STEPS IN THE REFERRAL PROCESS

**Step 1.** Child Protection worker to review CASIS-Client Profile Document and ensure health conditions/disabilities, education needs and custody/access details are entered and other information is accurate and up-to-date.

**Step 2.** Workers (Child Protection and CSO worker if contracted case) to become acquainted with Referral Tool (HCA and Referral Guide) to determine information required in order to complete the tool. If another service is involved with this child (e.g. VACCA, CAMHS, CSO providing placement or family support service, other services providing therapy) then the referral to be discussed with them and to include their information as required.

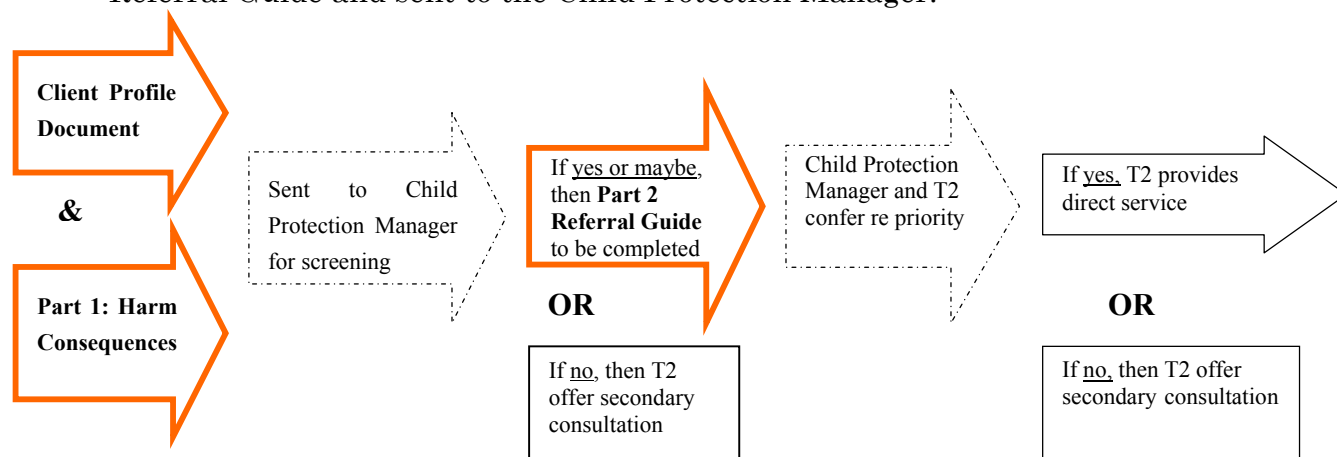
**Step 3.** Workers complete HCA, in consultation with Team Leader/ Unit Manager as appropriate. Workers send HCA and Client Profile Document to Child Protection Manager or delegate.

**Step 4.** Child Protection Manager or delegate decides whether this is an appropriate referral for therapy, and in particular, for TAKE TWO. This is the initial screening process. If no, then TAKE TWO may still be utilised for consultation if required. If yes, then next steps apply.

**Step 5.** Workers complete Referral Guide, in consultation with Team Leader/Unit Manager as appropriate. Child Protection worker sends all relevant documents to Child Protection Manager or their delegate.

**Step 6.** Child Protection Manager sends documents to TAKE TWO Senior Clinician, prior to their meeting where possible. Child Protection Manager and Senior Clinician meet to discuss priority for referrals. Senior Clinician advises Child Protection Manager on current capacity. Cases are then prioritised for referral. If Child Protection Manager decides not to refer client to TAKE TWO secondary consultation is available if required.

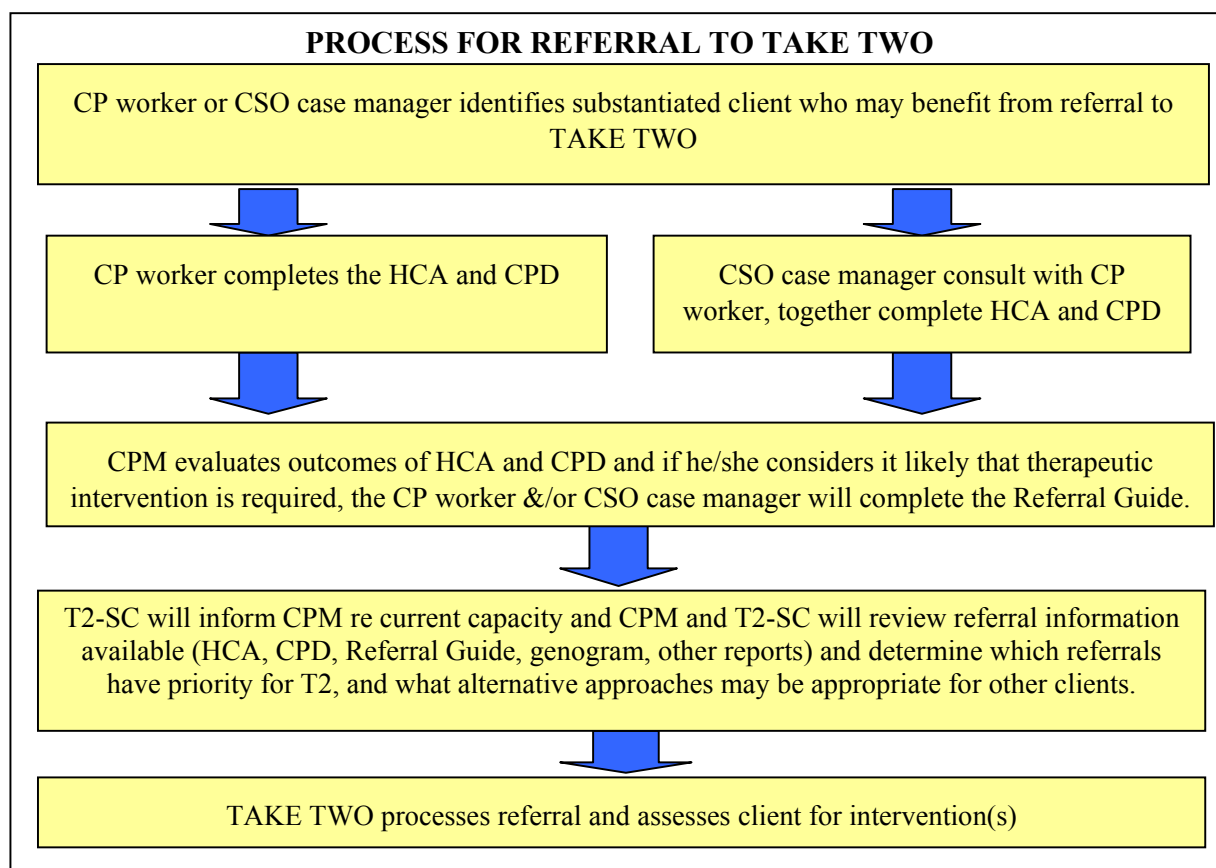
**Step 7.** If Child Protection Manager decides to prioritise referral to TAKE TWO, then direct service provision is able to begin once completed Referral Tool received by TAKE TWO (i.e. HCA, Referral Guide, Client Profile Document, and other relevant documents). Referral Guide and sent to the Child Protection Manager.





## 4.2 REFERRAL PROCESS

The following diagram provides a flow chart of the steps involved in referring a child to TAKE TWO.



## 4.3 CONTRACTED CASES

If case management has been contracted to a CSO, the completion of the Referral Tool will need to be a collaboration between the CSO case manager and the Case Contracting contact within Child Protection. For example the CSO case manager will most likely have more information regarding the details of the child's presentation and relationships, whereas both the CSO case manager and Child Protection worker will have a perspective on the harm consequences and only the Child Protection worker can produce the Client Profile Document from CASIS. It is then envisaged that the referral process would go via the Case Contracting contact within Child Protection through to the Child Protection Manager, as per other referrals to TAKE TWO.

## 4.4 SIBLING REFERRALS

If a referral is being made in relation to more than one child from a sibling group, then the tool needs to be completed for each child including the Client Profile Document. As the tool will be entered as a Word document, some of the information can be copied and pasted from one sibling's file to another, with distinguishing information filled in separately.

## 5. INSTRUCTIONS FOR COMPUTER USE:

You will have received the HCA and Referral Guide Word templates via email or disc. In addition to sending the referral through to TAKE TWO, this will enable workers to save completed Referral Tools on CASIS or the CSO client file. There are plans to have these on the DHS KnowledgeNet

Follow the same steps separately for the HCA and the Referral Guide template:

1. Double click on to the attachment in the email or disc.
2. Type in the password. This will have been provided to each region.
3. Click '**Enable Macros**'. It will then take a few seconds to open.  
[If you inadvertently click 'Disable Macros' it will not enable you to enter data into the document. If this is the case you will need to close the document and open again, this time clicking 'Enable Macros.']
4. This will take you into the HCA or Referral Guide where you type information as required and forward on to Child Protection Manager or delegate. Do not be concerned if it looks as if some information is no longer on the screen as it will all print out.
5. When you wish to print the document there will be a "**Print a Printer-Friendly copy**" button at the end of the template. This will print the key information and remove some of the extraneous information buttons.
6. To save the document on your computer you click file/save as and rename the document, eg HCAjones or RGjones. (eg. if Jones is client name)
7. If you want to save this document on CASIS (*after you have sent it to TAKE TWO via the Child Protection Manager or delegate*) you will need to remove the password protection. The following steps can do this.
  - a. Whilst the document is open, click **Tools**, then **Options**, then go into the **SAVE** tab.
  - b. Delete the password at the bottom of that page.
  - c. Save the document under the relevant name and save on to CASIS as you would any other Word document. E.g. create a new case note on CASIS, open it as a Word document and then select the TAKE TWO document by holding down the Control key and clicking on 'A'. Then copy and paste the TAKE TWO document into the case note on CASIS and save as usual
8. If you wish to password protect the Client Profile Document then follow similar steps in point 7, but this time enter the password where it states '**Password to open**'. This should be the same password as the other TAKE TWO document templates.
9. Next time you wish to make a referral you can either go back to the email where the documents were attached or if you have saved the template on your hard drive, you can click on to the document template there. Eg under '**My Documents**' or '**Desktop**'. Either way you then begin at step 1.
10. If you have any difficulties with the computer aspects of this tool, the functions relate to Microsoft Word and may vary depending on your version of Microsoft Word. If so, your own IT section may be able to assist with this or you can contact TAKE TWO.

## 6. THE CLIENT PROFILE DOCUMENT:

### 6.1 OVERVIEW

The Client Profile Document was developed by the Child Protection and Juvenile Justice Branch, DHS. It is essentially a summary document of the CASIS record that is used for referral to placement services, such as in relation to Looking After Children processes. It is serendipitous that the Client Profile Document is available as if it were not a similar tool would have needed to be developed.

Each referral to TAKE TWO is to be accompanied by a copy of the Client Profile Document. It should be noted that not all of this document is automatically populated by data and it is not necessary for the worker to complete all sections of it for the purposes of referral to TAKE TWO. The areas that do need to be completed are health conditions/disabilities, education and custody and access.

### 6.2 HOW TO ACCESS AND COMPLETE THE CLIENT PROFILE DOCUMENT

The Client Profile Document on CASIS needs to be accessed and updated if necessary by the Child Protection worker before printing. For more information regarding the Client Profile Document refer to Child Protection and Care Practice Bulletin 2003/1. A summary of steps to use the Client Profile Document for the purposes of referral to TAKE TWO are as follows:

1. From the Case Document Summary screen, click on the **Case Document Menu** tab.
2. From the Case Document Menu screen, select **"all"** from drop down menu.
3. Scroll through options until you find **Client Profile** – highlight, and click **"Open doc"**.
4. The Client Profile Document will open as a Word Document in CASIS, and information will be automatically merged from information previously recorded in CASIS.
5. If any information is inaccurate, it needs to be updated on the relevant CASIS screens.
6. All information is not automatically populated by CASIS and as such, some sections require the worker to enter additional information. Some of these sections are more appropriate for out-of-home care such as immunisation history and therefore, unless already updated, do not need to be entered for purposes of referral to TAKE TWO. Additional information to be entered into the Client Profile Document for purposes of referral to TAKE TWO are as follows:
  - Section 4.1: Health Conditions/Disabilities
  - Section 5: Education
  - Section 7: Custody and Access.
7. If you wish to password protect this document, refer to the previous page in this User Guide on Use of Computer, point 8.

## 7. THE HARM CONSEQUENCES ASSESSMENT:

### 7.1 OVERVIEW

The HCA is based on the Children and Young Person's Act 1989, literature regarding impact of abuse and neglect on the wellbeing of children and young people including attachment, trauma and permanency planning literature, and the Victorian Risk Framework (VRF).

The Victorian Risk Framework is the framework used by Child Protection workers to develop a risk of abuse and harm profile of clients and their families. The Risk Judgment Guide is a component of the VRF addressing harm consequence factors of abuse and neglect for the client. These factors are evident within the legislation governing the provision of Child Protection Services in Victoria, the Children and Young Persons Act 1989.

*S63 When is a child in need of protection?*

*For the purposes of this Act a child is in need of protection if any of the following grounds exist –*

*(a) the child has been abandoned by his or her parents and after reasonable inquiries –*

- i. the parents cannot be found; and*
- ii. no other suitable person can be found who is willing and able to care for the child;*

*(b) the child's parents are dead or incapacitated and there is no other suitable person willing and able to care for the child;*

*(c) the child has suffered, or is likely to suffer, significant harm as a result of physical injury and the child's parents have not protected, or are unlikely to protect, the child from harm of that type;*

*(d) the child has suffered, or is likely to suffer, significant harm as a result of sexual abuse and the child's parent's have not protected, or are unlikely to protect, the child from harm of that type;*

*(e) The child has suffered, or is likely to suffer, emotional or psychological harm of such kind that the child's emotional or intellectual development is, or is likely to be significantly damaged, and the child's parents have not protected, or are unlikely to protect, the child from harm of that type;*

*(f) The child's development or health has been, or is likely to be, significantly harmed and the child's parents have not provided, arranged or allowed the provision of, or are unlikely to provide, arrange or allow the provision of, basic care or effective medical, surgical or other remedial care.*

The domains included in the HCA derived from S 63 are:

- [a] and [b] Abandonment/Parental Incapacity
- [c] Physical Harm and Injury
- [d] Sexual Abuse
- [e] Emotional and Psychological Harm
- [f] Developmental Harm/Medical

The HCA is divided into two sections:

- The first section is a list of descriptors of abuse and neglect types and experiences according to these five domains. In other words, **what happened to the child?** The descriptors are derived from the VRF Risk Judgement Guide, the Risk Factors Warning List and the Child Abuse types list in CASIS. Some were adapted as a result of the pilot of the tool.

- The second section is a list of descriptors of the range of harms experienced by children as consequences of their experience or abuse and/or neglect. In other words, **what was the impact on the child?** This list has been derived from the Risk Judgement Guide and from other literature regarding consequences of abuse and neglect, attachment, trauma and permanency planning. Some descriptors were adapted as a result of the pilot of the tool.

The use of 'Extreme', 'Serious' and 'Concerning' headings were derived from the Risk Judgment Guide and are on the severe end of the continuum of abuse/ neglect. Therefore describing an experience or impact as 'Extreme' or 'Serious' is very significant in this context.



## 7.2 KEY MESSAGES IN RELATION TO THE HARM CONSEQUENCES ASSESSMENT

1. The HCA is intended to capture cumulative experience and consequences of abuse and neglect, not just the most recent incident or child protection involvement. For example, if the child is eleven years old, then workers are asked to reflect on their knowledge of the child's eleven years of experience.
2. The focus of the HCA is not on risk of future abuse, but on reflecting and summarising the impact of abuse and neglect already experienced.
3. As the first section of the HCA is describing the abuse or neglect experienced by the child, the terms relate to the behaviour of others towards the child, eg parental figures. As the second section relates to the impact of this abuse or neglect on the child, the terms include behaviours of the child. For example 'pattern of extreme humiliation' listed in the first section, is a pattern of behaviour towards the child. In the second section 'criminal activity involving violence/threats (eg armed robbery)' and 'ongoing or frequent substance abuse by ch/yp' relates to the child's behaviour as an example of impact of abuse or neglect.
4. As the HCA aims to encapsulate information that is relevant for therapeutic intervention it is useful to include information about both confirmed or believed abuse or neglect. Therefore it is not limited to substantiated abuse or abuse proven in a court of law, but rather on the worker's reasonable belief that such abuse or neglect has occurred.
5. It is envisaged, but not required, that clients referred to TAKE TWO would have at least one **'extreme'** descriptor or perhaps several or many **'serious'** descriptors in the first section of the HCA (What was the abuse/neglect experience?) and probably in the second section (What were the harm consequences?). There is no designated scoring system proposed for the HCA. In some situations the impact on the child may not yet be observable, but if extreme or serious abuse and neglect has occurred it can be predicted that this will lead to harmful impact on the child if therapeutic intervention does not occur.

## 7.3 OUTLINE OF THE HARM CONSEQUENCES ASSESSMENT

(A copy of the descriptors in the Harm Consequences Assessment is in Attachment 1)

### Client Details section

CASIS number

Child's Name:

Age of child:

Name of Worker Completing the tool:

Date of completing tool:

Region:

### Harm Consequences Assessment

#### Cumulative Abuse/Neglect Experiences of Child/Young Person

(What happened to the child/young person?)

| Domains of abuse/neglect          | Extreme  | Serious | Concerning |
|-----------------------------------|--|---------|------------|
| Abandonment/ no appropriate carer | Descriptors of abuse/neglect types are listed in each of these cells |         |            |
| Physical Harm and Injury          |  |         |            |
| Sexual Abuse                      |  |         |            |
| Emotional and Psychological Harm  |  |         |            |
| Developmental and Medical Harm    |  |         |            |

#### Harm Consequences to Child/Young Person as result of Abuse/Neglect

(What is impact of abuse/ neglect on child/young person?)

| Domains of harm consequences      | Extreme   | Serious | Concerning |
|-----------------------------------|---|---------|------------|
| Abandonment/ no appropriate carer | Descriptors of impact of abuse or neglect are listed in each of these cells |         |            |
| Physical Harm and Injury          |   |         |            |
| Sexual Harm                       |   |         |            |
| Emotional and Psychological Harm  |   |         |            |
| Developmental and Medical Harm    |   |         |            |

Comments:



## 7.4 HOW TO COMPLETE THE HARM CONSEQUENCES ASSESSMENT

- Type identifying information in the Client Details section of the HCA.
- Scan each domain row within the 'Cumulative Abuse/Neglect Experiences of Child/Young Person' and tick relevant abuse/neglect type(s)/experience(s) that have been confirmed or believed to have occurred at any time in the child's history or current situation.
- Scan each domain row within the 'Harm Consequences to Child/Young Person as result of Abuse/Neglect' and tick relevant impact/consequence(s) of these abuse/neglect experiences as you or others have observed or noted over time, eg mental health diagnoses, other assessments, feedback from carers, parents or school or direct observation.
- There is likely to be more than one consequence of harm and from more than one domain as a result of an abuse experience and these should all be ticked as appropriate. For example, sexual abuse may lead to a range of sexual harms, emotional harms, physical harms and developmental harms.
- If one of the descriptors in the 'Harm Consequences to Child/Young Person as result of Abuse/Neglect' is present but clearly not a result of abuse or neglect, e.g., due to a medical condition or disability, then this should not be ticked.
- In the 'Extreme' category relating to 'Harm Consequences to Child/Young Person as result of Abuse/Neglect' some of the descriptors include diagnoses, such as anxiety disorder, disorganised attachment, ADHD, conduct disorder, depression and eating disorder. These should only be ticked if there has been a specific medical/mental health diagnosis. If there are symptoms relating to these areas but no formal diagnosis, then there are corresponding descriptors in the 'Serious' category that can be ticked instead.
- If there is a single incident of abuse, then the most appropriate descriptor in the 'Cumulative Abuse/Neglect Experiences of Child/Young Person' should be ticked, not a range of descriptors. If, however, there were a number of incidents of abuse, then the range of descriptors that describe these various incidents can be ticked.
- In relation to the 'Harm Consequences to Child/Young Person as result of Abuse/Neglect' many descriptors are on a continuum of harm consequences for the child. Therefore for those consequences only the most applicable descriptor should be selected. For example, 'minimal sense of belonging', 'limited sense of belonging' and 'unclear sense of belonging' form a continuum and only one of these should be ticked if applicable.
- The 'Comments section' at the end of the HCA can be used if you believe a descriptor is more or less serious for this child than how it is listed, or if a descriptor is missing which you believe is pertinent to understanding this child's experience of abuse or neglect and/or consequences of harm.
- When completed this document can be saved as a Word document, which can then be saved on to CASIS after the password protection has been deleted. Refer to the Use of Computer section within this User Guide on page 8. If you wish to print this document then click the Print a Print-Friendly copy at the end of the HCA.
- There is a glossary of terms relating to the HCA on page 17 of this User Guide.

## 8. THE REFERRAL GUIDE:

### 8.1 OVERVIEW

The Referral Guide was developed to inform eligibility and prioritisation of referrals to TAKE TWO. It provides more detailed information than the HCA and is only required when the Child Protection Manager or delegate has determined that a referral to TAKE TWO is likely to be accepted.

The Referral Guide has the following headings:

- Identifying Information
- Eligibility Criteria
- Prioritising factors & information relating to impact of abuse/neglect on child.
- Therapeutic Intervention/Treatment
- Protection and Care involvement
- Specific Referral Information
- Decision Regarding Referral to TAKE TWO

The first section is in relation to identifying information, which coupled with the Client Profile Document, provides necessary client and worker information for TAKE TWO. The second section relates to eligibility criteria for a TAKE TWO service and is limited to 3 questions regarding Child Protection involvement. If any of these three eligibility questions is answered in the negative, then you do not need to complete the rest of the Referral Guide.

The next section provides more information regarding the experience of child abuse, other trauma and indicators of resilience. It includes a question regarding age of the child at onset of different types of abuse. This is one of the key factors highlighted in the literature: the younger the child is at the onset of abuse, the greater the likelihood of serious emotional and behavioural consequences for that child.

The question relating to other traumatic experiences is also highlighted in the literature as significant. For example if a child has experienced severe abuse and also experienced other trauma such as witnessing a parent overdose, this will add to the cumulative experience of trauma for the child and heighten the need for intensive therapeutic intervention. Similarly indicators of resilience enable assessment of how the child is currently adapting to his/her experience and points the way to potential avenues of therapy.

The section regarding therapeutic involvement provides information relating to what previous or current services have been involved, which may also assist in considering why a referral to TAKE TWO as compared to another service is indicated. The section relating to Protection and Care involvement may also assist the Child Protection Manager to determine prioritisation of referrals. The specific referral information section then leads on to thinking more specifically about what is the focus of this particular referral to TAKE TWO.

The final page is to be completed by the Child Protection Manager or their delegate regarding conclusions drawn and the final decision relating to referral to TAKE TWO.



## 8.2 SUMMARY OF QUESTIONS IN REFERRAL GUIDE

### Eligibility Criteria:

1. Is this child a current client of Child Protection?
2. Has harm to this child been substantiated during this protective involvement?
3. Has the child experienced serious or extreme harm from abuse and/or neglect as indicated by the Harm Consequences Assessment?

### Prioritising Factors and Information Relating to Impact of Abuse/Neglect:

4. Describe what actually happened in terms of the history and current abuse and/or neglect. Please note what age the child was when each type of abuse experience began.
5. Describe the child's experience of any other known trauma.
6. Describe any factors or indicators of actual and potential resilience for the child.

### Therapeutic Intervention/ Treatment:

7. Is/has the child &/or family attended counselling, therapy or any other form of treatment?
8. Please list if child and/or family are on a waiting list for any form of counselling or therapy.
9. Outline information or opinions that indicate which aspects of the child's system (formal or informal networks) could be a focus of support and therapeutic intervention.

### Protection and Care Involvement:

10. What is the current Child Protection case plan for this child?  
Overall goal of case plan. Specific Goals/Tasks:
11. Is there a perceived risk of unplanned change of placement?
12. Is Child Protection involvement planned to finish in the near future? If yes describe if there are plans to refer child/family to another case management service?

### Specific Referral Information:

13. What are the desired outcomes for this child arising from TAKE TWO intervention?
14. Describe potential barriers or hurdles you believe may impede achieving the outcomes.
15. In addition to the information present elsewhere in the TAKE TWO Referral Tool; is there anything else we should be aware of in relation to this child?
16. Has this referral been discussed with the child and family at this time?  
If Yes, what has been their response?  
If No, any specific reasons or concerns?

## 8. 3 HOW TO COMPLETE THE REFERRAL GUIDE

The following are notes or suggestions that were developed as a result of piloting the tool and in response to some common questions.

- Please review the whole Referral Guide first including the examples listed in the “Click here for more information’ buttons. This will provide an overview of the information required before entering information and will assist in preparation and avoiding duplication of answers.
- Q.2 asks whether or not the case was substantiated. The substantiation level is not required in this answer, as it is not an eligibility criterion. This is because the focus for TAKE TWO is in relation to the child’s experience of severe abuse at any time over their childhood, whereas the substantiation level is a decision made at a specific time in relation to a specific protective intervention. For example it is not uncommon for further information to become available to Child Protection that leads to a risk assessment that more serious harm occurred than what was initially identified at the time of substantiation.
- Q. 4 is in relation to telling the story about the child’s experience of abuse or neglect. Workers do not need to repeat the notification history or protective involvement as this is provided via the Client Profile Document. Rather it is providing a brief synopsis or dot point summary of the child’s experience of abuse.
- Q. 5 is asking about trauma not already mentioned, in other words, trauma not specifically related to abuse or neglect. An example of this category is the child witnessing violence perpetrated by someone outside the family. This question does not relate to family violence as this has already been covered in the HCA and in Q 4.
- Q. 9 enables the worker to reflect on what aspects of the child’s informal and formal network might be involved with TAKE TWO in order to work towards the desired outcomes for the child. Examples include working with the child at school, in their placement, with their peer group and/or with their family.
- Q. 11 relates to whether there is a risk of placement breakdown or risk of child being removed from home in the near future. It does not relate to planned changes, such as planned reunification or planned move to permanent care. Such planned changes of placement would be discussed in question 10 relating to case planning.
- Q. 12 is relevant if Child Protection is considering closing a case in the near future. Whilst this case may still be appropriate as a referral to TAKE TWO, it will be limited by the fact that TAKE TWO can only work for up to 3 months after Child Protection has closed. It is also important to note that as TAKE TWO does not provide case management, an alternative case management service would need to be considered, such as family services or CAMHS.
- Q18 and 19 are to be completed by the Child Protection Manager or their delegate.

## 9. OTHER DOCUMENTS:

### 9.1 GENOGRAM

Where possible, it is requested that a genogram be attached to the referral tool as this enables a very useful way of understanding who is in the family and the various relationships.

### 9.2 OTHER ASSESSMENTS

Please include any written assessments that have been completed by other services including paediatric, mental health or educational assessments. If a child is in placement, relevant Looking After Children documents are also of significant benefit. DHS would follow their normal procedures regarding releasing this information.

### 9.3 OTHER DHS DOCUMENTS

If there are Case Plan documents, Core Assessment Documents or other relevant information already prepared by DHS, then these would be beneficial in considering the referral and in determining what type of therapeutic intervention TAKE TWO may need to provide.

## 10. GLOSSARY:

This glossary relates to terms/ phrases that may require clarification in the HCA.

### **Cumulative Abuse/Neglect Experiences of Child/Young Person**

#### **No effective guardian – some self-sufficiency** (concerning/ abandonment domain)

This item relates to when a young person is old enough to ensure most of his/her needs are met, but has no legal guardian.

#### **Medical or surgical procedures misuse on child** (extreme/ physical abuse domain)

Where the child is exposed to unnecessary or inappropriate medical or surgical procedures, (eg. Repeated sexual assault examinations), or where such procedures (whether necessary or not) are performed by persons who are not medically trained.

#### **Munchausen by proxy (MSBP)** (extreme/ physical abuse domain)

The intentional production or feigning of physical or psychological signs or symptoms in another person who is under the individuals care. The motivation for the perpetrator's behaviour is to assume the sick role by proxy. External incentives for the behaviour, such as economic gain, avoiding legal responsibility, or improving physical well-being, are absent. (DSM-IV) This requires a medical/mental health diagnosis.

#### **Forcing ch/yp to witness violence** (extreme/ emotional abuse domain)

This item relates to when a child or young person is purposefully made to witness someone being violent. Eg. A father forcing a child to watch whilst he beats his/her mother. This is distinct from 'exposure to family violence' where it is not believed to be intentional that the child has witnessed the violence.

#### **Deprivation** (extreme/ developmental harm domain)

This item relates to when a parent figure deprives a child of basic necessities (such as food, fluids, water, shelter, physical contact, etc) on an ongoing basis.

## **Harm Consequences to Child/Young Person as result of Abuse/Neglect**

### **Sense of Permanence** (abandonment domain)

This relates to a child's sense of confidence in knowing with whom they are living and how long they are likely to remain with that person. In other words if a child has a 'minimal sense of permanence' they may have their bags packed and ready to move on at any time, as they will have no certainty that they will remain where they are. A child with 'limited sense of permanence' may have periods of time where they are unsure whether they are staying or leaving, but it is not a constant lack of permanence. A child with an 'unclear sense of permanence' is one where the worker is concerned about the child's sense of permanence but has no clear indicators of concern at this time.

A sense of permanence is closely related but distinct from a sense of belonging, security and trust.

### **Haematoma** (extreme/ physical harm)

A collection of blood trapped (blood clots) in the tissues, skin or in an organ. If it presents in the skin it is identified as bruising. It can only be seen in organs after specific tests, such as CT scans. A subdural haematoma is bleeding within one of the layers surrounding the brain. (NSW Liverpool Trauma Website). In the TAKE TWO context a severe haematoma is defined as severe, extensive bruising or any other form of haematoma

### **Psychotic** (extreme/ emotional harm domain)

A condition in which a person is unable to tell what is real from what is imagined, as occurs with the experience of hallucinations (sensory perceptions that occur in the absence of actual sensory stimulation) or delusions (firmly held false beliefs based on incorrect inference about reality). Symptoms may also include disorganised speech, disorientation or confusion, restrictions in range and intensity of emotional expression, in fluency and productivity of thought and speech and in the initiation of goal directed behaviour. An example of a psychotic disorder is schizophrenia. (Mental Health Services in Victoria - A guide to mental health terminology) This requires a mental health diagnosis.

### **Post-traumatic Stress Disorder (PTSD)** (extreme/ emotional harm domain)

The development of particular symptoms following exposure to a traumatic event. The individual's responses to the traumatic event include intense fear, helplessness or horror, which in children may be expressed through disorganized or agitated behaviour. Symptoms include persistent re-experiencing of the event, avoidance of stimuli associated with the trauma and increased arousal. (DSM-IV). This requires a mental health diagnosis.

### **Other Diagnoses** (extreme/ emotional harm domain)

Other diagnoses listed include depression, eating disorder, conduct disorder, anxiety disorder. These diagnoses along with the ones listed above, require a mental health/ medical diagnosis.

**Disorganised Attachment** (extreme/ emotional harm domain)

The child has no organised strategy of behaviour and displays contradictory behaviour in the parent's presence, e.g. dazed behaviour - freezing upon parent's return. The infant displays disorganised and or disoriented behaviours in the parents' presence, suggesting a temporary collapse of behavioural strategy.

Children who are unable to organise their behaviour to achieve proximity or security find that their distress and arousal remains heightened or unregulated. They find it difficult to maintain a functional and developmentally positive relationship with their carer. Their attachment behaviour becomes increasingly incoherent and disorganised, showing a confused mix of avoidance, angry approach responses, behavioural disorientation and inertia.

**Insecure / ambivalent Attachment** (serious/ emotional harm domain)

May be wary or distressed even prior to separation, with little exploration. Fails to settle and take comfort from parent on reunion, and usually continues to focus on parent and cry. Fails to return to exploration after reunion.

**Insecure / avoidant Attachment** (serious/ emotional harm domain)

Children with avoidant attachments may appear unconcerned by separation from the parent but will show physiological signs of anxiety, ie. The child shows no sign of missing parent then actively avoids parent on reunion. The child's response to the parent appears unemotional. Focuses on toys or environment throughout procedure. Children who show avoidant attachment patterns experience their parents as rejecting, interfering and controlling. If these children display distress it seems to annoy or agitate their caregiver.

## 11. ATTACHMENT ONE: TAKE TWO - Harm Consequences Assessment

### Cumulative Abuse/Neglect Experiences of Child/Young Person (What happened to the child/young person?)

| Domains of abuse/neglect                 | Extreme  | Serious   | Concerning  |
|--|--|---|---|
| <b>Abandonment/ no appropriate carer</b> | Abandonment/Absence of parent with no appropriate alternate carer<br>Both parents dead and no appropriate alternative Parental incapacity such that they cannot care for child and no appropriate alternative<br>Multiple placements<br>Extreme lack of supervision  | Abandonment/ absence of parent with significant concerns re alternative carer<br>Both parents dead and significant concerns re alternative carer<br>Parental incapacity with significant concerns re alternative carer<br>Small number of placements<br>Continuous inadequate supervision | No effective guardian-some self-sufficiency<br>Low-level supervision                            |
| <b>Physical Harm and Injury</b>          | <u>Biting</u><br><u>Born drug dependent</u><br><u>Burning or scalding</u><br><u>Enforced confinement</u><br><u>Drowning</u><br><u>Drugs misuse on child</u><br><u>Deliberate exposure to heat or cold</u><br><u>Frequent failure to ensure safety</u><br><u>Female genital mutilation</u><br><u>Frequent serious physical abuse</u><br><u>Hitting with objects</u><br><u>Any physical abuse to an infant</u><br><u>Medical or surgical procedures misuse on child</u><br><u>Multiple offenders of physical abuse</u><br><u>Munchausen by proxy</u><br><u>Poisoning</u><br><u>Strangulation</u><br><u>Suffocation</u><br><u>Throwing/ shaking</u><br><u>Torture</u> | <u>Excessive physical discipline</u><br><u>Exposure to heat and cold due to inadequate care</u><br><u>Failure to ensure safety</u><br><u>Exposure to physical harm from family violence</u><br><u>An incident of physical abuse leaving mark</u>  | Inappropriate physical discipline leaving no injury or slight injury (e.g. slap mark on bottom) |
| <b>Sexual Abuse</b>                      | <u>Sexual exploitation</u><br><u>Frequent serious sexual abuse</u><br><u>Multiple offenders of sexual abuse</u><br><u>Sexual penetration</u>   | <u>Fondling</u><br><u>Grooming behaviour</u><br><u>Sexual harassment</u><br><u>Involving ch/yp in masturbation</u><br><u>Sexual abuse (including lower tariff sexual offence)</u>   | Inappropriate/minimal sexual exposure (activity or material)                                    |

|   |   |  |   |
|---|---|--|---|
| <b>Emotional and Psychological Harm</b> | <p>Complete absence of affection</p> <p>Pattern of overt blaming of ch/yp</p> <p>Pattern of extreme humiliation</p> <p>Pattern of extreme rejection</p> <p>Direct/real threat to child's life made in child's presence</p> <p>Pattern of highly unreasonable expectations</p> <p>Pattern of extreme verbal abuse</p> <p>Forcing ch/yp to witness violence</p> <p>Exposure to ongoing, extreme violence</p> <p>Involving ch/yp in violence to others</p> | <p>Inadequate caring relationships</p> <p>Chaotic family lifestyle</p> <p>Exposure to family violence</p> <p>High criticism/low warmth</p> <p>Acting towards the child primarily negatively</p> <p>Exposure to parental psychiatric illness</p> <p>Scapegoated</p> <p>Exposure to parental substance abuse</p> <p>Frequent inappropriate threats</p> <p>Emotional unavailability of parent figures</p> <p>Unreasonable expectations</p> <p>Severe verbal abuse</p> | <p>Lack of boundaries</p> <p>Exposed to conflictual relationships</p> <p>Lack of discipline</p> <p>One-off/rare rejecting comments</p> <p>One-off/rare threats</p> <p>Infrequent verbal abuse</p>   |
| <b>Developmental and Medical Harm</b>   | <p>Extreme lack of basic care</p> <p>Deprivation</p> <p>Extreme lack of food or fluids</p> <p>Extreme lack of medical care</p> <p>Refusal to send child to school (&lt;15 yrs)</p> <p>Absence of stimulation</p>  | <p>Inadequate basic care</p> <p>Continuous inadequate provision of food or fluids</p> <p>Inadequate medical care</p> <p>Inconsistently sending child to school (&lt;15 yrs)</p> <p>Inadequate stimulation</p>  | <p>Chronic low-level basic care</p> <p>Isolated/minor lack of basic care</p> <p>Inadequate clothing</p> <p>Not following through re immunizations</p> <p>Slowness in responding to common medical problems</p> <p>Not supporting child at school</p> <p>Low-level stimulation</p> |



## Harm Consequences to Child/Young Person as result of Abuse/Neglect (What is impact of abuse/ neglect on child/young person?)

| Domains of harm consequences             | Extreme  | Serious  | Concerning   |
|--|--|--|--|
| <b>Abandonment/ no appropriate carer</b> | Minimal sense of belonging<br>Minimal sense of future<br>Minimal sense of permanence<br>Minimal sense of security<br>Minimal trust   | Limited sense of belonging<br>Limited and age-inappropriate view of the future<br>Limited sense of permanence<br>Limited sense of security<br>Limited trust in others  | Unclear sense of belonging<br>Lack of confidence in others<br>Some insecurity<br>Unclear sense of permanence   |
| <b>Physical Harm and Injury</b>          | Bites<br>Burns<br>Severe Haematoma<br>Head injury/brain damage<br>Any injury to an infant<br>Internal injuries<br>Life threatening physical harm<br>Other injury requiring medical intervention<br>Condition requiring medical intervention (e.g. pregnancy, STD)<br>Multiple injuries<br>Mutilation<br>Paralysis<br>Serious preventable illness | Observable injury or condition (e.g. welt, bruise)<br>Minor but preventable illnesses  | Minor injury not requiring medical intervention (e.g. slap mark on bottom of older child)  |
| <b>Sexual Harm</b>                       | Self-blame<br>Dangerous to self sexual activity<br>Extreme sexual harm<br>Prostitution<br>Repeated sexual harm<br>Distorted understanding of sexuality<br>Sexualized violence towards others   | Sexual harassment of others<br>Sexual harm/exposure (including lower tariff sexual offence)<br>Pattern of inappropriate sexualised behaviour towards others<br>Promiscuity<br>Shame<br>Confused understanding of sexuality   | An incident of inappropriate sexualised behaviour to others  |
| <b>Emotional and Psychological Harm</b>  | Severe changes in affect or mood<br>Killing or torturing animals<br>Anxiety disorder diagnosis<br>Extreme lack of attachment or disorganised attachment<br>Attention Deficit Hyperactivity Disorder diagnosis<br>Conduct disorder diagnosis<br>Criminal activity involving violence/threats (eg armed robbery)<br>Dangerous self-harm            | Lengthy/ continuous absconding<br>Significant changes in affect or mood<br>Hurting animals<br>Anxious/Fearful<br>Ambivalent or anxious/avoidant insecure attachment<br>Conduct/behavioural problems<br>Non-violent criminal activity<br>Eating difficulties<br>Incident of fire lighting | Non-dangerous acting-out/attention seeking<br>Minor alterations in affect or mood<br>Minor alterations in behaviour<br>Minor alterations in confidence<br>Passivity<br>Emotional or psychological harm limited to timeframe of incident<br>Occasional sleep difficulties |



|  |   |  |  |
|--|---|--|--|
|  | <p>Depression diagnosis</p> <p>Eating disorder diagnosis</p> <p>Constant emotional unavailability</p> <p>Repeated or dangerous fire lighting</p> <p>Overwhelming sense of helplessness</p> <p>Hypersensitivity</p> <p>Disturbed or no sense of identity</p> <p>Extremely negative sense of identity</p> <p>Highly indiscriminate</p> <p>Presents as numb despite threatening/difficult situation</p> <p>Experiences of panic or terror</p> <p>Highly parentified</p> <p>Ch/yp involved in parent's delusions</p> <p>Post traumatic stress disorder diagnosis</p> <p>Enduring/severe psychological impairment or condition</p> <p>Risk-taking</p> <p>No self-awareness</p> <p>No self-control</p> <p>Profound sleep disturbance</p> <p>Ongoing or frequent substance abuse by ch/yp</p> <p>Suicide attempts by ch/yp</p> <p>Repeated and severe violence to others</p> | <p>Feelings of helplessness</p> <p>Hyperactivity</p> <p>Confused sense of identity</p> <p>Impulsive</p> <p>Indiscriminate</p> <p>Physical or emotional isolation</p> <p>Parentified</p> <p>Ch/yp confused re parent's delusions</p> <p>Negative impact to peer relationships</p> <p>Post traumatic symptoms</p> <p>School refusal</p> <p>Limited age-appropriate self-awareness</p> <p>Altered/ negative impact to self-esteem/self-confidence</p> <p>Threats to self-harm</p> <p>Frequent sleep difficulties</p> <p>Soiling / enuresis</p> <p>Intermittent substance abuse by ch/yp</p> <p>Suicidal ideation</p> <p>Limited response to threatening/difficult situations</p> <p>Intermittent emotional unavailability</p> <p>Intermittent violence or threats of violence to others</p> <p>Very withdrawn</p> | <p>Occasional and/or minor substance misuse by ch/yp</p> <p>Tense/ apprehensive/ fretful</p> <p>Occasional threats of violence to others</p>                 |
| <p><b>Developmental and Medical Harm</b></p> | <p>Dehydrated</p> <p>Significant developmental delays</p> <p>No friendships</p> <p>Delayed growth</p> <p>Malnutrition</p> <p>No sense of morality/conscience</p> <p>No school attendance (&lt;15 yrs)</p> <p>Extreme social isolation</p> <p>Significantly impaired speech and language</p> <p>Failure to thrive</p>  | <p>Deterioration in attention/concentration</p> <p>Deterioration in cognition</p> <p>Some developmental delay</p> <p>Minimal friendships</p> <p>Often hungry</p> <p>Limited understanding of morality/conscience</p> <p>Frequently not at school</p> <p>Social isolation</p> <p>Speech and language difficulties</p>   | <p>Concerns re development</p> <p>Occasionally hungry</p> <p>Missing immunizations</p> <p>Some school absenteeism</p> <p>Concerns re speech and language</p> |

**Please add here any descriptors or comments you consider relevant.**

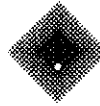
NB: For more detailed responses please provide in Part 2 (Referral Guide) of the TAKE TWO Referral Tool.

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This User Guide is designed to provide information to assist decision making and planning and is based on the best information at the time of publication. This User Guide provides a general guide to appropriate practice, to be followed only subject to individual professional's or organisation's judgement in individual circumstances or contexts.

## Appendix 10: Victoria University Human Research Ethics Committee approval letter



# MEMO

TO Dr Alan Tucker DATE 12/04/2006

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FROM Prof. Michael Polonsky  
Chair  
Victoria University Human Research Ethics  
Committee

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SUBJECT Ethics Approval - HRETH.05/110

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Dear Alan,

Thank you for your submission detailing amendments to the research protocol for the project titled "**Cognitive Functioning of Child Protection Clients in Secure Care: A Neuropsychological Study**" (HRETH.05/110)

The proposed amendments have been accepted by the Chair, Human Research Ethics Committee and approval for application HRETH.05/110 has been granted from 12/04/06 to 12/04/08.

Please note that the Human Research Ethics Committee must be informed of the following: any changes to the approved research protocol, project timelines, any serious or unexpected adverse effects on participants, and unforeseen events that may effect continued ethical acceptability of the project. In these unlikely events, researchers must immediately cease all data collection until the Committee has approved the changes.

If you have any queries, please do not hesitate to contact me on 9919 4625.

On behalf of the Committee, I wish you all the best for the conduct of the project.

  
Prof. Michael Polonsky  
Chair  
Victoria University Human Research Ethics Committee

Appendix 11: Department of Human Services Human Research Ethics Committee  
(Victoria) approval letter



## Department of Human Services

Incorporating: Health, Community Services, Aged Care and Housing

### **HUMAN RESEARCH ETHICS COMMITTEE**

**105/05**

**Ethics Committee Secretariat**

**Telephone: (03) 9096 5239 Fax: (03) 9096 9186**

**50 Lonsdale Street**

**MELBOURNE 3000**

**Email: [genevieve.nolan@dhs.vic.gov.au](mailto:genevieve.nolan@dhs.vic.gov.au)**

120 Spencer Street

GPO Box 1670N

Melbourne Victoria 3001

DX210081

[www.dhs.vic.gov.au](http://www.dhs.vic.gov.au)

Telephone: (03) 9637 4000

Facsimile: (03) 9637 4779

Our Ref:

Your Ref:

7 April 2006

Dr Alan Tucker  
Psychology Department  
Victoria University  
PO Box 14428  
MELBOURNE 8001

Dear Dr Tucker

**Re: 105/05 - Cognitive functioning of child protection clients in secure care: A neuropsychological study**

The Department of Human Services Human Research Ethics Committee, at its meeting on 5 April 2006, ratified the approval of the response dated 17 February 2006 for the above project.

Yours sincerely

**DR DIANE SISELY**  
**CHAIR**

## Appendix 12: Berry Street Victoria Policy and Practice Committee approval letter



28 August 2006

Dr Alan Tucker  
Senior Lecturer & Clinical Neuropsychologist  
School of Psychology  
Victoria University  
P.O Box 14428  
Melbourne 8001

Dear Dr Tucker,

Thank you for your research proposal Cognitive Functioning of Child Protection Clients in Secure Care: A Neuropsychological Study and the accompanying documentation from the Department of Human Services (Ethics Committee).

I would like to confirm that Berry Street Victoria's Policy and Practice Committee has endorsed your research proposal and understand that the research has commenced.

I do note on our file, that suggested amendments have been made to the original research proposal from Dr Chris Pullin, Acting Chair, University Human Research Ethics Committee and from Berry Street Victoria. Could you please confirm that that these suggestions have been incorporated into the final documentation and provide me with copies of the up-dated document.

Please feel free to contact me should you require any further assistance or have any queries.

I look forward to on-going contact with you and wish you well with this research.

Yours sincerely,

[Redacted Signature]  
Ms Zakia Ebrahim  
A/Manager  
ENQIRY Unit  
Berry Street Victoria  
Ph: 9429 9266  
Mobile: 0418 162 917  
Email: zebrahim@berrystreet.org.au



Appendix 13: Victorian Department of Education Human Research Ethics Committee  
approval letter



## Department of Education

Office for Education Policy and Innovation

2 Treasury Place  
East Melbourne, Victoria 3002  
Telephone: +61 3 9637 2000  
DX 210083  
GPO Box 4367  
Melbourne, Victoria 3001

SOS003551

Dr Alan Tucker, et al  
PO Box 14428  
MELBOURNE 8001

Dear Dr Tucker, et al

Thank you for your application of 26 March 2007 in which you request permission to conduct a research study in government schools titled: *Cognitive functioning of child protection clients in secure care: a neuropsychological study and a neuropsychological study of primary school-aged children with a history of maltreatment.*

I am pleased to advise that on the basis of the information you have provided your research proposal is approved in principle subject to the conditions detailed below.

1. Should your institution's ethics committee require changes or you decide to make changes, these changes must be submitted to the Department of Education for its consideration before you proceed.
2. You obtain approval for the research to be conducted in each school directly from the principal. Details of your research, copies of this letter of approval and the letter of approval from the relevant ethics committee are to be provided to the principal. The final decision as to whether or not your research can proceed in a school rests with the principal.
3. No student is to participate in this research study unless they are willing to do so and parental permission is received. Sufficient information must be provided to enable parents to make an informed decision and their consent must be obtained in writing.
4. As a matter of courtesy, you should advise the relevant Regional Director of the schools you intend to approach. An outline of your research and a copy of this letter should be provided to the Regional Director.

5. Any extensions or variations to the research proposal, additional research involving use of the data collected, or publication of the data beyond that normally associated with academic studies will require a further research approval submission.
6. At the conclusion of your study, a copy or summary of the research findings should be forwarded to Strategic Policy and Research, Department of Education, Level 2, 33 St Andrews Place, GPO Box 4367, Melbourne, 3001.

I wish you well with your research study. Should you have further enquiries on this matter, please contact Chris Warne, Project Officer, Strategic Policy and Research, by phone on (03) 9637 2272 or by email at <[warne.christine.p@edumail.vic.gov.au](mailto:warne.christine.p@edumail.vic.gov.au)>.

Yours sincerely



**John McCarthy**  
Assistant General Manager  
Strategic Policy and Research

19 / 6 / 2007

enc

## Appendix 14: Secondary School Principal's invitation letter

**VICTORIA UNIVERSITY**  
**School of Psychology**

**Research Study Description**

**TITLE: COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN  
SECURE CARE:  
A NEUROPSYCHOLOGICAL STUDY**

Dear Principal,

We are conducting a study into the impact that child abuse has on the cognitive functioning of adolescents in Secure Care. The primary group of participants involved in this study includes child protection clients residing in Secure Care. A crucial aspect of this research is to compare the cognitive functioning of adolescents who have experienced abuse and thus have been placed in a Secure Care facility with a group of adolescents **without such a history**.

The study aims to document how aspects of cognitive function including, memory, learning, language, visuospatial function, planning, organization and sequencing of behaviour are affected by trauma and abuse during childhood and adolescence, with particular emphasis on adolescents residing in secure welfare services.

Adolescents involved in the study need to be aged 12-16 years **without a history of abuse**. We would greatly appreciate your cooperation in gaining the participation of approximately 30 students, comprising around 6 students from each year levels 7 through to 11. Participation in this study involves a total of approximately one and a half hours of testing and interviews (which may be completed in two sessions if necessary). During this session/s the young person will be asked to complete some fairly simple memory, learning and other cognitive tasks. As part of the study we also need the young person and/or parent/guardian to complete a brief questionnaire on their educational and medical history. Adolescents with a history of major injuries/diseases affecting the central nervous system, reading and language difficulties and visual/auditory problems will not be included in the study. The young person will also be asked some questions related to how they are feeling at present. Testing may take place at the Victoria University Psychology Clinic or another mutually agreed upon location (including your school if that is convenient for you and the student). If it is convenient for your school, we will conduct the testing during school hours in a quiet room.

**Each student who participates will receive a free assessment report outlining their cognitive strengths and weaknesses.**

If you are willing for your school to participate in this study, we will provide you with participant information and consent forms for distribution to your students and their parent/guardians.

We welcome your queries in relation to this study. Please contact one of the undersigned.

Vidanka Ruvceska  
*PhD Candidate*  
Email: [vidanka.ruvceska@research.vu.edu.au](mailto:vidanka.ruvceska@research.vu.edu.au)  
Ph: (03) 9919 2221

Dr. Alan Tucker  
*Supervisor & Clinical Neuropsychologist*  
Email: [alan.tucker@vu.edu.au](mailto:alan.tucker@vu.edu.au)  
Ph: (03) 9919 2266

Appendix 15: Parent/guardian information and consent form for Secure Welfare participants



## **PARTICIPANT INFORMATION AND CONSENT FORM**

**VICTORIA UNIVERSITY  
School of Psychology**

### **Participant Information and Consent Form**

**Version 4 Dated 15/11/05**

**Site Take 2 Secure Welfare Service**

Full Project Title: **COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS  
IN SECURE CARE: A NEUROPSYCHOLOGICAL STUDY**

Principal Researcher: Dr. Alan Tucker

Associate Researcher(s): Vidanka Ruvceska

---

This Participant Information and Consent Form is **7** pages long. Please make sure you have all the pages.

### **1. Your Consent**

You and your child are invited to take part in the research project titled Cognitive functioning of child protection clients in secure care: A neuropsychological study.

This Participant Information contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

### **2. Purpose and Background**

The purpose of this project is to investigate and document the information processing skills (including memory and learning, language, organisation and planning and visuospatial function) of adolescents in a particular kind of protective care. That is, for those adolescents at immediate risk of harm who have been placed in a secure facility to establish safety, known as Secure Welfare.

In order to investigate this topic properly, we need to assess the functioning of a group of children in secure care **AND** a similar aged group of young people in the general community.



A total of 100 people will participate in this project, 50 children and adolescents living in Secure Welfare and 50 children and adolescents from Victorian primary and secondary schools.

Previous experience has shown that young persons who have had abusive histories are at risk for developing information processing problems, that is difficulties with functions such as learning and memory, organisation and planning of behaviour and visuospatial functioning. The types and number of information processing problems in adolescents are unclear and need to be explored. Such information will be useful as it will identify problems that can be improved with the aid of clinical services.

You are invited to participate in this research project because it will allow for a clearer understanding of the impact of abuse on information processing skills.

The results of this research may be used to help researcher *Vidanka Ruvceska* to obtain a degree.

### **3. Procedures**

Participation in this project will involve a total of approximately two hours of testing and interviews (which may be completed in two sessions if necessary). During this session/s your child will be asked to complete some fairly simple memory, learning and other cognitive tasks and some questions relating to how they are feeling at present. As part of the study we also need you to complete a brief questionnaire on your child's educational and medical history.

Information relating to your child's history with child protection will be collected from Department of Human Services case records.

A group of 50 children and adolescents who have not experienced any form of abuse will also complete the experimental procedure outlined above in order to observe whether child abuse has an impact on cognitive functioning.

### **4. Possible Benefits**

The study will be of great value to you as it will allow you to learn of your child's cognitive strengths and capabilities

Identification of these strengths as well as any weaknesses can be used to assist your child in their educational and career planning. It will also provide important information to your child's clinicians which will assist them in providing your child with appropriate clinical services.

### **5. Possible Risks**

Possible risks, side effects and discomforts include:

- Completion of the Trauma Symptom Checklist for Children (a measure of your child's emotional functioning) although highly unlikely may evoke some distressing emotion
- A negative emotional reaction may occur after learning of a cognitive deficit
- The completion of measures involved in the study may involve stress associated with unfamiliarity, fatigue and level of performance





If adverse reactions (although unlikely) during the testing procedure occur, your child will be referred to their managing Take 2 clinician and/or case worker for counselling.

If your child experiences any fatigue or distress associated with completion of the tests, they will be given the opportunity for breaks and the option to withdraw from testing at any time.

## **6. Privacy, Confidentiality and Disclosure of Information**

Any information obtained in connection with this project and that can identify you or your child will remain confidential. It will only be disclosed with your permission, except as required by law. If you give us your permission by signing the Consent Form, we plan to report your child's results to Take 2 in the form of a short summary outlining their performance on the cognitive tests mentioned earlier. Your child's individual results including their name and personal information will be held at the Take 2 offices in Flemington under lock and key for a minimum period of five years. Your child's managing Take 2 clinician and researchers Vidanka Ruvceska and Dr. Alan Tucker will have access to this information.

Your child's abuse history and important medical information will be taken from their DHS case records at Take 2.

Group results will also be reported in the form of a research thesis, however you and your child's confidentiality will be maintained in this publication as no individual results or names of individuals involved in the study will be reported. In any publication, information will be provided in such a way that you cannot be identified.

## **7. New Information Arising During the Project**

During the research project, new information about the risks and benefits of the project may become known to the researchers. If this occurs, you will be told about this new information. This new information may mean that you can no longer participate in this research. If this occurs, the person(s) supervising the research will stop your participation. In all cases, you will be offered all available care to suit your needs.

## **8. Results of Project**

After a short period following testing, you will be provided with a short report summarising your child's cognitive strengths and weaknesses and some recommendations in relation to their results. You can also have access to the group results published in the research thesis on completion of the study.

## **9. Further Information or Any Problems**

If you require further information or if you have any problems concerning this project, you can contact the principal researcher Dr. Alan Tucker (Ph. 9919 2266) or associate researcher Vidanka Ruvceska (Ph. 9919 2221).

## **10. Other Issues**

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact

Name: Ms Genevieve Nolan

Position: Executive Officer Human Services Human Research Ethics Committee

Telephone: 9637 4239

Name: The Secretary

University Human Research Ethics Committee, Victoria University

Telephone: (03) 9919 4710

Name: Dr. Alan Tucker

Position: Senior Lecturer, Victoria University School of Psychology

Telephone: (03) 9919 2221

### **11. Participation is Voluntary**

Participation in this research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Your decision whether to take part or not to take part, or to take part and then withdraw, will not affect your child's routine treatment, their relationship with those treating them or your/their relationship with Take 2.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw. This notice will allow that person or the research supervisor to inform you if there are any health risks or special requirements linked to withdrawing.

### **12. Ethical Guidelines**

This project will be carried out according to the *National Statement on Ethical Conduct in Research Involving Humans* (June 1999) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Department of Human Services and the Victoria University Human Research Ethics Committee.

### **13. Reimbursement for your costs**

You will not be paid for your participation in this project.

**CONSENT FORM****(Attach to Participant Information)**

VICTORIA UNIVERSITY

School of Psychology

**Consent Form****Version 4 Dated 15/11/05****Site Take 2 Secure Welfare**

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE:  
A NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me and I understand the Participant Information version 4 dated 15/11/05.

I freely agree to participate in this project according to the conditions in the Participant Information.

I will be given a copy of the Participant Information and Consent Form to keep

The researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .....

Signature

Date

Name of Witness to Participant's Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of information concerning the research project.

**THIRD PARTY CONSENT FORM** *(To be used by parents/guardians of minor children.)***(Attach to Participant Information)**

|  |
|--|
| On Institution's Letterhead or Name of Institution |
|--|

**Third Party Consent Form  
Version 4 Dated 15/11/05  
Site Take 2 Secure Welfare**

**Full Project Title:**

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

---

I have read, or have had read to me, and I understand the Participant Information version 4 dated 15/11/05.

I give my permission for \_\_\_\_\_ to participate in this project according to the conditions in the Participant Information.

I will be given a copy of Participant Information and Consent Form to keep.

The researcher has agreed not to reveal the participant's identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .....

Name of Person giving Consent (printed) .....

Relationship to Participant: .....

Signature

Date

Name of Witness to Parent/Guardian Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant's parent/guardian has understood that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of information concerning the research project.

*Note:* All parties signing the Consent Form must date their own signature.



## REVOCATION OF CONSENT FORM

*(To be used for participants who wish to withdraw from the project.)*

**(Attach to Participant Information)**

VICTORIA UNIVERSITY  
School of Psychology

### Revocation of Consent Form

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with ***Name of Institution.***

Participant's Name (printed) .....

Signature

Date

Appendix 16: Information and consent form for guardians of adolescents in Secure Welfare under the custody of Department of Human Services (Victoria)

**PARTICIPANT INFORMATION AND CONSENT FORM****VICTORIA UNIVERSITY  
School of Psychology****Participant Information and Consent Form****Version 5 Dated 15/11/05****Site Take 2 Secure Welfare Service**

Full Project Title: **COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS  
IN SECURE CARE: A NEUROPSYCHOLOGICAL STUDY**

Principal Researcher: Dr. Alan Tucker

Associate Researcher(s): Vidanka Ruvceska

This Participant Information and Consent Form is **6** pages long. Please make sure you have all the pages.

**1. Your Consent**

The young person (name) \_\_\_\_\_ has been invited to take part in the research project titled *Cognitive functioning of child protection clients in secure care: A neuropsychological study*.

This Participant Information contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to allow \_\_\_\_\_ to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to allow \_\_\_\_\_ take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent, allowing \_\_\_\_\_ to participate in the research project.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

**2. Purpose and Background**

The purpose of this project is to investigate and document the information processing skills of young people in a particular kind of protective care. That is, for those adolescents at immediate risk of harm who have been placed in a secure facility to establish safety known as Secure Welfare.



In order to investigate this topic properly, we need to assess the functioning of a group of adolescents in secure care **AND** a similar aged group of young people in the general community.

A total of 100 people will participate in this project, 50 from Secure Welfare and 50 from Victorian Primary and Secondary schools.

Previous experience has shown that children and adolescents who have had abusive histories are at risk for developing cognitive deficits, that is difficulties with various information processing skills including learning and memory, organisation, planning and sequencing of behaviour and visuospatial functioning. The pattern and extent of these deficits in such children and adolescents remain unclear and need to be explored. Such information will be useful as it will identify areas of deficit allowing for the provision of clinical services.

The young person is invited to participate in this research project because it will allow for a clearer understanding of the impact of abuse on information processing skills.

The results of this research may be used to help researcher *Vidanka Ruvceska* to obtain a degree.

### **3. Procedures**

Participation in this project will involve a total of approximately two hours of testing and interviews (which may be completed in two sessions if necessary). During this session/s the young person\_\_\_\_\_ will be asked to complete some fairly simple memory, learning and other cognitive tasks and some questions relating to how they are feeling at present. As part of the study we also need a brief questionnaire on the young person's educational and medical history to be completed. If possible, this information will be taken from the young person directly, however if further clarification or details are required, Department of Human services case records will be utilised.

Information relating to the young person's history with child protection will be collected from Department of Human Services case records.

A group of 50 children and adolescents who have not experienced any form of abuse will also complete the experimental procedure outlined above in order to observe whether child abuse has an impact on cognitive functioning.

### **4. Possible Benefits**

The study will be of great value to the young person as it will allow them to learn of their cognitive strengths and capabilities

Identification of these strengths as well as any weaknesses can be used to assist the young person in their educational and career planning. It will also provide important information to the young person's clinicians which will assist them in providing the young person with appropriate clinical services.

### **5. Possible Risks**

Possible risks, side effects and discomforts include:

- Completion of the Trauma Symptom Checklist for Children (a measure of your child's emotional functioning) although highly unlikely may evoke some distressing emotion





- A negative emotional reaction may occur after learning of a cognitive deficit
- The completion of measures involved in the study may involve stress associated with unfamiliarity, fatigue and level of performance

If adverse reactions (although unlikely) during the testing procedure occur, the young person will be referred to their managing Take 2 clinician and/or case worker for counselling.

If the young person experiences any fatigue or distress associated with completion of the tests, they will be given the opportunity for breaks and the option to withdraw from testing at any time.

## **6. Privacy, Confidentiality and Disclosure of Information**

Any information obtained in connection with this project and that can identify the young person will remain confidential. It will only be disclosed with your permission, except as required by law. If you give us your permission by signing the Consent Form, we plan to report the young person's results to Take 2 in the form of a short summary outlining their performance on the cognitive tests mentioned earlier. The young person's individual results with name and personal details will be held at the Take 2 offices in Flemington under lock and key for a minimum period of five years. The young person's managing Take 2 clinician and researchers Vidanka Ruvceska and Dr. Alan Tucker will have access to this information.

The young person's abuse history and important medical information will be taken from their DHS case records at Take 2.

Group results will be reported in the form of a research thesis, however the young person's confidentiality will be maintained in this publication as no individual results or names of individuals involved in the study will be reported.

In any publication, information will be provided in such a way that the young person cannot be identified.

## **7. New Information Arising During the Project**

During the research project, new information about the risks and benefits of the project may become known to the researchers. If this occurs, you will be told about this new information. This new information may mean that the young person can no longer participate in this research. If this occurs, the person(s) supervising the research will stop the young person's participation. In all cases, the young person will be offered all available care to suit their needs.

## **8. Results of Project**

After a short period following testing, the young person will be provided with a short report summarising their cognitive strengths and weaknesses and some recommendations in relation to their results. They can also have access to the group results published in the research thesis on completion of the study.

## **9. Further Information or Any Problems**

If you require further information or if you have any problems concerning this project, you can contact the principal researcher Dr. Alan Tucker (Ph. 9919 2266) or associate researcher Vidanka Ruvceska (Ph. 9919 2221).

## **10. Other Issues**

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact

Name: Ms Genevieve Nolan

Position: Executive Officer Human Services Human Research Ethics Committee

Telephone: 9637 4239

Name: The Secretary

University Human Research Ethics Committee, Victoria University

Telephone: (03) 9919 4710

Name: Dr. Alan Tucker

Position: Senior Lecturer, Victoria University School of Psychology

Telephone: (03) 9919 2221

## **11. Participation is Voluntary**

Participation in this research project is voluntary. If you do not wish for the young person to take part they are not obliged to. If you decide to allow the young person to take part and later change your mind, you are free to withdraw them from the project at any stage.

Your decision whether to allow the young person to take part or not to take part, or to take part and then withdraw, will not affect the young person's routine treatment, their relationship with those treating them or their relationship with Take 2.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw. This notice will allow that person or the research supervisor to inform you if there are any health risks or special requirements linked to withdrawing.

## **12. Ethical Guidelines**

This project will be carried out according to the *National Statement on Ethical Conduct in Research Involving Humans* (June 1999) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Department of Human Services and the Victoria University Human research Ethics Committee.

## **13. Reimbursement for your costs**

The young person will not be paid for your participation in this project.

**THIRD PARTY CONSENT FORM** *(To be used by parents/guardians of minor children.)***(Attach to Participant Information)**

VICTORIA UNIVERSITY

School of Psychology

**Third Party Consent Form**  
**Version 5 Dated 15/11/05**  
**Site Take 2 Secure Welfare**

**Full Project Title:****COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me, and I understand the Participant Information  
version 5 dated 15/11/05.

I give my permission for \_\_\_\_\_ to participate in this project  
according to the conditions in the Participant Information.

I will be given a copy of Participant Information and Consent Form to keep.

The researcher has agreed not to reveal the participant's identity and personal details if  
information about this project is published or presented in any public form.

Participant's Name (printed) .....

Name of Person giving Consent (printed) .....

Relationship to Participant: .....

Signature

Date

Name of Witness to Parent/Guardian Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its  
procedures and risks and I believe that the participant's parent/guardian has understood  
that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of  
information concerning the research project.

*Note:* All parties signing the Consent Form must date their own signature.



## REVOCATION OF CONSENT FORM

*(To be used for participants who wish to withdraw from the project.)*

**(Attach to Participant Information)**

VICTORIA UNIVERSITY  
School of Psychology

### Revocation of Consent Form

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with **Take 2**.

Participant's Name (printed) .....

Signature

Date

## Appendix 17: Secure Welfare participant information and consent form

**PARTICIPANT INFORMATION AND CONSENT FORM****VICTORIA UNIVERSITY  
School of Psychology****Participant Information and Consent Form****Version 1 Dated 12/11/05****Site Take 2 Secure Welfare Service**

Full Project Title: **COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS  
IN SECURE CARE: A NEUROPSYCHOLOGICAL STUDY**

Principal Researcher: Dr. Alan Tucker

Associate Researcher(s): Vidanka Ruvceska

---

This Participant Information and Consent Form is **7** pages long. Please make sure you have all the pages.

**1. Your Consent**

You are invited to take part in the research project titled Cognitive functioning of child protection clients in secure care: A neuropsychological study.

This Participant Information contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

**2. Purpose and Background**

The purpose of this project is to investigate and document the information processing skills (including memory and learning, language, organisation and planning and visuospatial function) of adolescents in a particular kind of protective care. That is, for those adolescents at immediate risk of harm who have been placed in a secure facility to establish safety, known as Secure Welfare.

In order to investigate this topic properly, we need to assess the functioning of a group of children in secure care **AND** a similar aged group of young people in the general community.



A total of 100 people will participate in this project, 50 children and adolescents living in Secure Welfare and 50 children and adolescents from Victorian primary and secondary schools.

Previous experience has shown that young persons who have had abusive histories are at risk for developing information processing problems, that is difficulties with functions such as learning and memory, organisation and planning of behaviour and visuospatial functioning. The types and number of information processing problems in adolescents are unclear and need to be explored. Such information will be useful as it will identify problems that can be improved with the aid of clinical services.

You are invited to participate in this research project because it will allow for a clearer understanding of the impact of abuse on information processing skills.

The results of this research may be used to help researcher *Vidanka Ruvceska* to obtain a degree.

### **3. Procedures**

Participation in this project will involve a total of approximately two hours of testing and interviews (which may be completed in two sessions if necessary). During this session/s you will be asked to complete some fairly simple memory, learning and other cognitive tasks. As part of the study we also need you to complete a brief questionnaire on your educational, medical history and some questions on how you are feeling at present. Information relating to your history with child protection will be collected from Department of Human Services case records.

A group of 50 adolescents who have not experienced any form of abuse will also complete the procedure outlined above in order to observe whether child abuse has an impact on cognitive functioning.

### **4. Possible Benefits**

The study will be of great value to you as the participant as it will allow you to learn of your cognitive strengths and capabilities

Identification of these strengths as well as any weaknesses can be used to assist you in your educational and career planning. It will also provide important information to your carers and clinicians which will assist them in providing you with appropriate clinical services.

### **5. Possible Risks**

Possible risks, side effects and discomforts include:

- Completion of the Trauma Symptom Checklist for Children (a measure of your emotional functioning) although highly unlikely may bring out some distressing emotion
- You may become concerned after learning that you have a cognitive deficit
- The completion of tasks within the study may cause you stress associated with being unfamiliar with the tasks, becoming tired and the level of your performance

If you are finding the testing procedure difficult, you will be referred to your Take 2 clinician and/or case worker for counselling.



If you experience any tiredness or distress associated with completion of the tests, you will be given the opportunity for breaks and the option to withdraw from testing at any time.

## **6. Privacy, Confidentiality and Disclosure of Information**

Any information obtained in connection with this project and that can identify you will remain confidential. It will only be reported to another with your permission, except as required by law. If you give us your permission by signing the Consent Form, we plan to report your results to Take 2 in the form of a short summary outlining your performance on the cognitive tests mentioned earlier. Data with your name and personal details will be held at the Take 2 offices in Flemington under lock and key for a minimum period of five years. Your Take 2 clinician and researchers Vidanka Ruvceska and Dr. Alan Tucker will have access to this information.

Your abuse history and important medical information will be taken from your DHS case records at Take 2.

Data that does not identify you will also be reported in the form of a research thesis, however your confidentiality will be maintained in this publication as only group results will be reported. In any publication, information will be provided in such a way that you cannot be identified.

## **7. New Information Arising During the Project**

During the research project, new information about the risks and benefits of the project may become known to the researchers. If this occurs, you will be told about this new information. This new information may mean that you can no longer participate in this research. If this occurs, the person(s) supervising the research will stop your participation. In all cases, you will be offered all available care to suit your needs.

## **8. Results of Project**

After a short period following testing, you will be provided with a short report summarising your cognitive strengths and weaknesses and some recommendations in relation to your results. You can also have access to the group results published in the research thesis on completion of the study.

## **9. Further Information or Any Problems**

If you require further information or if you have any problems concerning this project, you can contact the principal researcher Dr. Alan Tucker (Ph. 9919 2266) or associate researcher Vidanka Ruvceska (Ph. 9919 2221).

## **10. Other Issues**

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact

Name: Ms Genevieve Nolan

Position: Executive Officer Human Services Human Research Ethics Committee

Telephone: 9637 4239





Name: The Secretary

University Human Research Ethics Committee, Victoria University

Telephone: (03) 9919 4710

Name: Dr. Alan Tucker

Position: Senior Lecturer, Victoria University School of Psychology

Telephone: (03) 9919 2221

### **11. Participation is Voluntary**

Participation in this research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Your decision whether to take part or not to take part, or to take part and then withdraw, will not affect your routine treatment, your relationship with those treating you or your relationship with Take 2.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw. This notice will allow that person or the research supervisor to inform you if there are any health risks or special requirements linked to withdrawing.

### **12. Ethical Guidelines**

This project will be carried out according to the *National Statement on Ethical Conduct in Research Involving Humans* (June 1999) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Department of Human Services and the Victoria University Human Research Ethics Committee.

### **13. Reimbursement for your costs**

You will not be paid for your participation in this project.

**CONSENT FORM****(Attach to Participant Information)**

VICTORIA UNIVERSITY

School of Psychology

**Consent Form****Version 1 Dated 12/11/05****Site Take 2**

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE:  
A NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me and I understand the Participant Information version 1 dated 12/11/05.

I freely agree to participate in this project according to the conditions in the Participant Information.

I will be given a copy of the Participant Information and Consent Form to keep

The researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .....

Signature

Date

Name of Witness to Participant's Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of information concerning the research project.



**THIRD PARTY CONSENT FORM**

*(To be used by parents/guardians of minor children.)*

**(Attach to Participant Information)**

|  |
|--|
| On Institution's Letterhead or Name of Institution |
|--|

**Third Party Consent Form  
Version 1 Dated 12/11/05  
Site Take 2**

**Full Project Title:**

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me, and I understand the Participant Information  
version **1** dated **12/11/05**.

I give my permission for \_\_\_\_\_ to participate in this project  
according to the conditions in the Participant Information.

I will be given a copy of Participant Information and Consent Form to keep.

The researcher has agreed not to reveal the participant's identity and personal details if  
information about this project is published or presented in any public form.

Participant's Name (printed) .....

Name of Person giving Consent (printed) .....

Relationship to Participant: .....

Signature

Date

Name of Witness to Parent/Guardian Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its  
procedures and risks and I believe that the participant's parent/guardian has understood  
that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of  
information concerning the research project.

*Note:* All parties signing the Consent Form must date their own signature.



## REVOCATION OF CONSENT FORM

*(To be used for participants who wish to withdraw from the project.)*

**(Attach to Participant Information)**

VICTORIA UNIVERSITY  
School of Psychology

### Revocation of Consent Form

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with ***Name of Institution.***

Participant's Name (printed) .....

Signature

Date

## Appendix 18: Parent/guardian information and consent form for Control participants



## PARENT/GUARDIAN INFORMATION AND CONSENT FORM

### VICTORIA UNIVERSITY School of Psychology

#### Parent/Guardian Information and Consent Form

Version 3 Dated 15/11/05

Site Victoria University

Full Project Title: **COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS  
IN SECURE CARE: A NEUROPSYCHOLOGICAL STUDY**

Principal Researcher: Dr. Alan Tucker

Associate Researcher(s): Vidanka Ruvceska

---

This Participant Information and Consent Form is **7** pages long. Please make sure you have all the pages.

#### **1. Your Consent**

You and your child are invited to take part in the research project titled Cognitive functioning of child protection clients in secure care: A neuropsychological study.

This Participant Information contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to your and your child's participation in the research project.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

#### **2. Purpose and Background**

The purpose of this project is to investigate and document the information processing skills of young people in a particular kind of protective care. That is, for those adolescents at immediate risk of harm who have been placed in a secure facility to establish safety known as Secure Welfare.

In order to investigate this topic properly, we need to assess the functioning of a group of adolescents in secure care **AND** a similar aged group of young people in the general community. A total of 100 people will participate in this project, 50 from Secure Welfare and 50 from Victorian Secondary schools.



Previous experience has shown that children and adolescents who have had abusive histories are at risk for developing cognitive deficits, that is difficulties with various information processing skills including learning and memory, organisation, planning and sequencing of behaviour and visuospatial functioning. The pattern and extent of these deficits in such children and adolescents remain unclear and need to be explored. Such information will be useful as it will identify areas of deficit allowing for the provision of clinical services.

You and your child are invited to participate in this research project because it will allow for a clearer understanding of the impact of abuse on information processing skills. In order to obtain this understanding we need to compare the cognitive profiles of children with an abuse history (from Secure Welfare) to those who **have not** had a history of abuse (primary and secondary school students).

The results of this research may be used to help researcher *Vidanka Ruvceska* to obtain a degree.

### **3. Procedures**

Participation in this project will involve a total of approximately two hours of testing and interviews (which may be completed in two sessions if necessary). During this session/s your child will be asked to complete some fairly simple memory, learning and other cognitive tasks. Your child will also be asked some questions in relation to how they are feeling at present. As part of the study we also need you to complete a brief questionnaire on your child's educational and medical history.

A group of 50 children and adolescents who have experienced some form of abuse and are residing in Secure Welfare will also be involved in the research. They too will complete the experimental procedure outlined above in order to observe the differences in cognitive functioning between children who have and **have not** experienced child abuse.

### **4. Possible Benefits**

The study will be of great value to you as it will allow you to learn of your child's cognitive strengths and capabilities

Identification of these strengths as well as any weaknesses can be used to assist you in your child's educational and career planning.

### **5. Possible Risks**

Possible risks, side effects and discomforts include:

- Completion of the Trauma Symptom Checklist for Children (a measure of your child's emotions at present) although highly unlikely may evoke some distressing emotion
- A negative emotional reaction may occur after learning of a cognitive deficit
- The completion of measures involved in the study may involve stress associated with unfamiliarity, fatigue and level of performance

If adverse reactions, although highly unlikely, during the testing procedure occur, you will be given contact details of Dr. Alan Tucker (experienced clinician and supervisor) who can direct you to appropriate clinical services. Alternatively, your child may contact the



Kids Helpline on 1800 55 1800 if they become distressed by some of the questions asked and need someone else to talk to who is separate from this study.

If your child experiences any fatigue or distress associated with completion of the tests, they will be given the opportunity for breaks and the option to withdraw from testing at any time.

## **6. Privacy, Confidentiality and Disclosure of Information**

Any information obtained in connection with this project and that can identify you or your child will remain confidential. It will only be disclosed with your permission, except as required by law. If you give us your permission by signing the Consent Form, we plan to report your child's results only to yourself and your child in the form of a short summary outlining your performance on the cognitive tests mentioned earlier. Your child's individual results will be held at the Victoria University School of Psychology under lock and key for a minimum period of five years. Researchers Vidanka Ruvceska and Dr. Alan Tucker will have access to this information.

Group results will be reported in the form of a research thesis, however you and your child's confidentiality will be maintained in this publication as no individual results or names of individuals involved in the study will be reported.

In any publication, information will be provided in such a way that you cannot be identified.

## **7. New Information Arising During the Project**

During the research project, new information about the risks and benefits of the project may become known to the researchers. If this occurs, you will be told about this new information. This new information may mean that you can no longer participate in this research. If this occurs, the person(s) supervising the research will stop your participation. In all cases, you will be offered all available care to suit your needs.

## **8. Results of Project**

After a short period following testing, you will be provided with a short report summarising your child's cognitive strengths and weaknesses and some recommendations in relation to their results. You can also have access to the group results published in the research thesis on completion of the study.

## **9. Further Information or Any Problems**

If you require further information or if you have any problems concerning this project, you can contact the principal researcher Dr. Alan Tucker (Ph. 9919 2266) or associate researcher Vidanka Ruvceska (Ph. 9919 2221).

## **10. Other Issues**

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact

Name: Ms Vicki Xafis





Position: Executive Officer Human Services Human Research Ethics Committee

Telephone: (03) 9637 4239

Name: The Secretary

University Human Research Ethics Committee, Victoria University

Telephone: (03) 9919 4710

Name: Dr. Alan Tucker

Position: Senior Lecturer, Victoria University School of Psychology

Telephone: (03) 9919 2266

### **11. Participation is Voluntary**

Participation in this research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw. This notice will allow that person or the research supervisor to inform you if there are any health risks or special requirements linked to withdrawing.

### **12. Ethical Guidelines**

This project will be carried out according to the *National Statement on Ethical Conduct in Research Involving Humans* (June 1999) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Department of Human Services and the Victoria University Human research Ethics Committee.

### **13. Reimbursement for your costs**

You will not be paid for your participation in this project.

**CONSENT FORM****(Attach to Participant Information)**

VICTORIA UNIVERSITY

School of Psychology

**Consent Form****Version 3 Dated 15/11/05****Site Victoria University**

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE:  
A NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me and I understand the Participant Information version 3 dated 15/11/05.

I freely agree to participate in this project according to the conditions in the Participant Information.

I will be given a copy of the Participant Information and Consent Form to keep

The researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .....

Signature

Date

Name of Witness to Participant's Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of information concerning the research project.

**THIRD PARTY CONSENT FORM**

*(To be used by parents/guardians of minor children.)*

**(Attach to Participant Information)**

|   |
|---|
| VICTORIA UNIVERSITY<br>School of Psychology |
|---|

**Third Party Consent Form  
Version 3 Dated 15/11/05  
Site Victoria University**

**Full Project Title:**

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

---

I have read, or have had read to me, and I understand the Participant Information version **3** dated **15/11/05**.

I give my permission for \_\_\_\_\_ to participate in this project according to the conditions in the Participant Information.

I will be given a copy of Participant Information and Consent Form to keep.

The researcher has agreed not to reveal the participant's identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .....

Name of Person giving Consent (printed) .....

Relationship to Participant: .....

Signature

Date

Name of Witness to Parent/Guardian Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant's parent/guardian has understood that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of information concerning the research project.

*Note:* All parties signing the Consent Form must date their own signature.



**REVOCATION OF CONSENT FORM**

*(To be used for participants who wish to withdraw from the project.)*

**(Attach to Participant Information)**

|   |
|---|
| VICTORIA UNIVERSITY<br>School of Psychology |
|---|

**Revocation of Consent Form**

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with ***Name of Institution.***

Participant's Name (printed) .....

Signature

Date

## Appendix 19: Control participant information and consent form



## **PARTICIPANT INFORMATION AND CONSENT FORM**

**VICTORIA UNIVERSITY  
School of Psychology**

### **Participant Information and Consent Form**

**Version 2 Dated 15/11/05**

**Site Victoria University**

Full Project Title: **COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS  
IN SECURE CARE: A NEUROPSYCHOLOGICAL STUDY**

Principal Researcher: Dr. Alan Tucker

Associate Researcher(s): Vidanka Ruvceska

---

This Participant Information and Consent Form is **7** pages long. Please make sure you have all the pages.

### **1. Your Consent**

You are invited to take part in the research project titled Cognitive functioning of child protection clients in secure care: A neuropsychological study.

This Participant Information contains detailed information about the research project. Its purpose is to explain to you as openly and clearly as possible all the procedures involved in this project before you decide whether or not to take part in it.

Please read this Participant Information carefully. Feel free to ask questions about any information in the document. You may also wish to discuss the project with a relative or friend or your local health worker. Feel free to do this.

Once you understand what the project is about and if you agree to take part in it, you will be asked to sign the Consent Form. By signing the Consent Form, you indicate that you understand the information and that you give your consent to participate in the research project.

You will be given a copy of the Participant Information and Consent Form to keep as a record.

### **2. Purpose and Background**

The purpose of this project is to investigate and document the information processing skills of young people in a particular kind of protective care. That is, for those adolescents at immediate risk of harm who have been placed in a secure facility to establish safety known as Secure Welfare.

In order to investigate this topic properly, we need to assess the functioning of a group of adolescents in secure care **AND** a similar aged group of young people in the general community.



A total of 100 people will participate in this project, 50 from Secure Welfare and 50 from Victorian Secondary schools.

Previous experience has shown that children and adolescents who have had abusive histories are at risk for developing cognitive deficits, that is difficulties with various information processing skills including learning and memory, organisation, planning and sequencing of behaviour and visuospatial functioning. The pattern and extent of these deficits in such children and adolescents remain unclear and need to be explored. Such information will be useful as it will identify areas of deficit allowing for the provision of clinical services.

You are invited to participate in this research project because it will allow for a clearer understanding of the impact of abuse on information processing skills. In order to obtain this understanding we need to compare the cognitive profiles of children with an abuse history (from Secure Welfare) to those who **have not** had a history of abuse (primary and secondary school students).

The results of this research may be used to help researcher *Vidanka Ruvceska* to obtain a degree.

### **3. Procedures**

Participation in this project will involve a total of approximately two hours of testing and interviews (which may be completed in two sessions if necessary). During this session/s you will be asked to complete some fairly simple memory, learning and other cognitive tasks. As part of the study we also need you to complete a brief questionnaire on your educational, medical history and some questions on how you are feeling at present.

A group of 50 children and adolescents who have experienced some form of abuse and are residing in Secure Welfare will also complete the experimental procedure outlined above in order to observe whether child abuse has an impact on cognitive functioning.

### **4. Possible Benefits**

The study will be of great value to you as the participant as it will allow you to learn of your cognitive strengths and capabilities

Identification of these strengths as well as any weaknesses can be used to assist you in your educational and career planning.

### **5. Possible Risks**

Possible risks, side effects and discomforts include:

- Completion of the Trauma Symptom Checklist for Children (a measure of your emotional functioning) although highly unlikely may evoke some distressing emotion
- A negative emotional reaction may occur after learning of a cognitive deficit
- The completion of measures involved in the study may involve stress associated with unfamiliarity, fatigue and level of performance

If adverse reactions, although highly unlikely, during the testing procedure occur, you will be given contact details of Dr. Alan Tucker (experienced clinician and supervisor) who



can direct you to appropriate clinical services. Alternatively, you may contact the Kids Helpline on 1800 55 1800 if you become distressed by some of the questions asked and need someone else to talk to who is separate from the study.

If you experience any fatigue or distress associated with completion of the tests, you will be given the opportunity for breaks and the option to withdraw from testing at any time.

## **6. Privacy, Confidentiality and Disclosure of Information**

Any information obtained in connection with this project and that can identify you will remain confidential. It will only be disclosed with your permission, except as required by law. If you give us your permission by signing the Consent Form, we plan to report your results only to yourself and your parent/guardian in the form of a short summary outlining your performance on the cognitive tests mentioned earlier. Individual results with your name and personal information will be held at the Victoria University School of Psychology under lock and key for a minimum period of five years. Researchers Vidanka Ruvceska and Dr. Alan Tucker will have access to this information.

Group results will also be reported in the form of a research thesis, however your confidentiality will be maintained in this publication as no individual results or names of individual involved in the study will be reported.

In any publication, information will be provided in such a way that you cannot be identified.

## **8. New Information Arising During the Project**

During the research project, new information about the risks and benefits of the project may become known to the researchers. If this occurs, you will be told about this new information. This new information may mean that you can no longer participate in this research. If this occurs, the person(s) supervising the research will stop your participation. In all cases, you will be offered all available care to suit your needs.

## **9. Results of Project**

After a short period following testing, you will be provided with a short report summarising your cognitive strengths and weaknesses and some recommendations in relation to your results. You can also have access to the group results published in the research thesis on completion of the study.

## **10. Further Information or Any Problems**

If you require further information or if you have any problems concerning this project, you can contact the principal researcher Dr. Alan Tucker (Ph. 9919 2266) or associate researcher Vidanka Ruvceska (Ph. 9919 2221).

## **11. Other Issues**

If you have any complaints about any aspect of the project, the way it is being conducted or any questions about your rights as a research participant, then you may contact

Name: Ms Vicki Xafis

Position: Executive Officer Human Services Human Research Ethics Committee

Telephone: (03) 9637 4239





Name: The Secretary

University Human Research Ethics Committee, Victoria University

Telephone: (03) 9919 4710

Name: Dr. Alan Tucker

Position: Senior Lecturer, Victoria University School of Psychology

Telephone: (03) 9919 2266

## **12. Participation is Voluntary**

Participation in this research project is voluntary. If you do not wish to take part you are not obliged to. If you decide to take part and later change your mind, you are free to withdraw from the project at any stage.

Before you make your decision, a member of the research team will be available to answer any questions you have about the research project. You can ask for any information you want. Sign the Consent Form only after you have had a chance to ask your questions and have received satisfactory answers.

If you decide to withdraw from this project, please notify a member of the research team before you withdraw. This notice will allow that person or the research supervisor to inform you if there are any health risks or special requirements linked to withdrawing.

## **13. Ethical Guidelines**

This project will be carried out according to the *National Statement on Ethical Conduct in Research Involving Humans* (June 1999) produced by the National Health and Medical Research Council of Australia. This statement has been developed to protect the interests of people who agree to participate in human research studies.

The ethical aspects of this research project have been approved by the Human Research Ethics Committee of the Department of Human Services and the Victoria University Human Research Ethics Committee.

## **14. Reimbursement for your costs**

You will not be paid for your participation in this project.

**CONSENT FORM****(Attach to Participant Information)**

VICTORIA UNIVERSITY

School of Psychology

**Consent Form****Version 2 Dated 15/11/05****Site Victoria University**

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE:  
A NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me and I understand the Participant Information version 2 dated 15/11/05.

I freely agree to participate in this project according to the conditions in the Participant Information.

I will be given a copy of the Participant Information and Consent Form to keep

The researcher has agreed not to reveal my identity and personal details if information about this project is published or presented in any public form.

Participant's Name (printed) .....

Signature

Date

Name of Witness to Participant's Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its procedures and risks and I believe that the participant has understood that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of information concerning the research project.

**THIRD PARTY CONSENT FORM**

*(To be used by parents/guardians of minor children.)*

**(Attach to Participant Information)**

VICTORIA UNIVERSITY  
School of Psychology

**Third Party Consent Form****Version 2 Dated 15/11/05****Site Victoria University****Full Project Title:****COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I have read, or have had read to me, and I understand the Participant Information  
version **2** dated **15/11/05**.

I give my permission for \_\_\_\_\_ to participate in this project  
according to the conditions in the Participant Information.

I will be given a copy of Participant Information and Consent Form to keep.

The researcher has agreed not to reveal the participant's identity and personal details if  
information about this project is published or presented in any public form.

Participant's Name (printed) .....

Name of Person giving Consent (printed) .....

Relationship to Participant: .....

Signature

Date

Name of Witness to Parent/Guardian Signature (printed) .....

Signature

Date

Declaration by researcher\*: I have given a verbal explanation of the research project, its  
procedures and risks and I believe that the participant's parent/guardian has understood  
that explanation.

Researcher's Name (printed) .....

Signature

Date

\* A senior member of the research team must provide the explanation and provision of  
information concerning the research project.

*Note:* All parties signing the Consent Form must date their own signature.



## REVOCATION OF CONSENT FORM

*(To be used for participants who wish to withdraw from the project.)*

**(Attach to Participant Information)**

VICTORIA UNIVERSITY  
School of Psychology

### Revocation of Consent Form

Full Project Title:

**COGNITIVE FUNCTIONING OF CHILD PROTECTION CLIENTS IN SECURE CARE: A  
NEUROPSYCHOLOGICAL STUDY**

I hereby wish to WITHDRAW my consent to participate in the research proposal described above and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with ***Name of Institution.***

Participant's Name (printed) .....

Signature

Date