SOCIAL INFORMATION PROCESSING AND WORKING MEMORY FOLLOWING PEDIATRIC STROKE

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Abstract

There is a dearth of research examining social competence following pediatric arterial ischemic stroke (AIS). Previous studies suggest that working memory (WM) and social information processing (SIP), two related individual characteristics that are critical for this construct, are compromised following pediatric AIS. However, research is limited and little is known about the effects of age at stroke and lesion location. Study 1 assessed the WM and SIP patterns of 32 children, aged 6 to 14, with histories of unilateral AIS and 32 controls using paradigms based on Baddeley and Hitch's three-component WM model and Crick and Dodge's six-step SIP model. Study 2 investigated the effects of age at stroke (i.e., perinatal, 1 month-5 years, 6-14 years) and lesion location (i.e., cortical, subcortical, and combined) on WM and social outcomes as rated by parents using a large, retrospective sample of children with unilateral AIS. Evidence was found for isolated, subtle difficulties in social cue encoding and decoding in children with AIS compared to controls. Children with AIS also consistently scored significantly lower than controls across WM indices and performance correlated with many SIP measures. Children who suffered a stroke between 1 month and 5 years were rated more favourably by parents on social skills than children who suffered a stroke between 6 and 14 years. No evidence was found for an effect of lesion location on WM and social outcomes, nor was there an effect of age at stroke on WM. These findings suggest that social competence in children with AIS may be compromised by subtle, interrelated difficulties in WM and SIP. Continual follow-up is recommended, as the subtle nature of these challenges increases the likelihood that they will go undetected. Moreover, the long-term trajectories of WM and social outcomes in the pediatric stroke population remains unknown.

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Introduction

Stroke is an important cause of acquired brain injury in the pediatric population. Strokes are diagnosed in approximately 1 in every 4000 live births per year and have an annual incidence of 0.6 – 13 per 100,000 in children between 1 month and 18 years, with a recurrence rate of 6-40% (Hartel, Schilling, Sperner, & Thyen, 2004). Stroke is defined as "the abrupt onset of a focal neurologic deficit, such as disruption in strength, speech, vision, or cognition that is consistent with a vascular distribution" (Festa, Lazar, & Marshall, 2008, p. 364). Stroke occurs as a result of a disruption in the normal blood flow in the brain, which deprives the brain of oxygen and nutrients and may ultimately lead to permanent tissue damage (referred to as an "infarction") (Festa et al., 2008). Strokes are classified according to three types: arterial ischemic stroke (AIS; stroke that results from an obstruction of blood flow caused by a narrowing or blood clot in an artery), cerebral sinovenous thrombosis (CSVT; stroke caused by a blood clot formed in the venous system of the brain), and intracranial hemorrhage (stroke caused by bleeding in the brain or subarachnoid space) (Festa et al., 2008). Investigations into neuropsychological impairment in children with histories of strokes have revealed subtle deficits in a range of domains, including intelligence (Allman & Scott, 2011; Ballantyne, Spilkin, Hesselink, & Trauner, 2008; Hartel, et al., 2004; Max, 2010; Nass & Trauner, 2004; Westmacott, MacGregor, Askalan, & deVeber, 2009; Westmacott, Askalan, MacGragor, Anderson, & deVeber, 2010), language (Avila et al., 2010; Bates et al., 1997; Chapman, Max, Gamino, McGlothin, & Cliff, 2003; Gout et al., 2005; Marchman, Miller, & Bates, 1991; Nass & Trauner, 2004; Stiles et al., 2010), memory and learning (Block, Nanson, & Lowry, 1999; Lansing et al., 2004), and visual-spatial processing (Akshoomoff, Feroleto, Doyle, & Stiles, 2002; Nass & Trauner, 2004; Schatz, Craft, Koby, & DeBaun, 2004; Schatz, Ballantyne, & Trauner, 2000; Stiles, Trauner, Engel, &

Nass, 1997; Stiles, et al., 2008; Stiles et al., 2010). Surprisingly, few studies have investigated the social domain. This is a critical gap in the literature, given that social functioning has been reported to be an important predictor of quality of life (Bohnert, Packer, & Warschausky, 1997).

A key construct guiding research on children's social behaviour is social competence. Although many definitions have been proposed, there is a growing consensus that social competence encompasses three components: individual characteristics and social skills, social performance and interaction, and social adjustment (Yeates et al., 2007). The first component refers to a range of abilities that are required in order to interact effectively with others (e.g., attentional control, working memory, goal-setting, problem-solving, planning, etc.) while the second component refers to the child's actual behaviour in social settings (Yeates et al., 2007). The third component reflects the quality of the child's relationships with others (Yeates et al., 2007). The proposed study will focus on social information processing (SIP) in particular, as past evidence has indicated that this individual characteristic is critical for social competence (Crick & Dodge, 1994; Yeates et al., 2007). A key theoretical model guiding research in this area was devised by Crick and Dodge (1994). This model construes SIP as a form of "on-line brain performance" involving six steps (i.e., encoding, interpretation, clarification of goals, response construction, response decision, and enactment) (Crick & Dodge, 1994). The extant research on the social behaviour of children with histories of stroke has shown that this population commonly experiences impairments in this domain (Ballantyne & Trauner, 1999; Boni, Brown, Davis, Hsu, & Hopkins, 2001; Mosch et al., 2005; Trauner, Panyard-Davis, & Ballantyne, 1996). To date, no studies have directly assessed each of the SIP steps in children with histories of stroke.

There is an increasing amount of literature linking SIP with working memory (WM). For example, research has shown that low WM capacity is associated with deficits in social competence (Anderson & Knight, 2010; Channon & Crawford, 2010; Hecker & Dutke, 2004; Hommel, Miguel, Naegele, Gonnet, & Jaillard, 2009). WM is defined as the mental system that allows us to keep information active for a brief amount of time while performing a task (Khan & Muly, 2011). The theoretical model guiding research in this domain was devised by Baddeley and Hitch (1974) and later extended by Baddeley (2000). This model is composed of three components (i.e., phonological loop, visuospatial sketchpad, and central executive). Although the current literature on WM in the pediatric stroke population is sparse, emerging evidence indicates that children with stroke demonstrate subtle WM deficits (Lansing et al., 2004; Westmacott et al., 2009, 2010). No studies to date have examined each of the components of WM in children with histories of stroke. Moreover, no studies have examined the relation between SIP and WM in this population.

The *primary* objective of this project was to characterize the WM and SIP profiles of children with histories of stroke. Utilizing the models put forward by Baddeley and Hitch (1974) and Crick and Dodge (1994), the WM and SIP indices of children aged 6-14 with unilateral AIS were compared to those of typically developing controls. The *secondary* objective was to compare the WM and SIP scores of children with cortical, subcortical, and combined (i.e., cortical and subcortical) lesion involvement and those with histories of stroke during the perinatal period, early childhood (i.e., 1 month-5 years), and late childhood (i.e., 6-14 years). This comparison of lesion locations was chosen because (1) lesions affecting cortical and subcortical regions have been associated with worse cognitive outcomes (Westmacott et al., 2010); and (2) because research findings emphasize the importance of the cortical and

subcortical connections of the prefrontal cortex (PFC) for WM and SIP (Baier et al., 2010; Beauchamp & Anderson, 2010; Khan & Muly, 2011; Ward, 2006; Yeates et al., 2007). In contrast, past studies have been unable to detect differences in cognitive performance in children based on the lateralization of the stroke (Everts et al., 2008; Lansing et al., 2004; Westmacott et al., 2009). The age comparisons were selected because (1) earlier age at injury has been associated with worse cognitive outcome (Lansing et al., 2004; Westmacott et al., 2010); and (2) because lesion location has been identified as an important moderator for the relationship between age at stroke and cognitive outcomes (Westmacott et al., 2010). The findings of this study will be critical for characterizing groups of children that are at high-risk for developing SIP and WM impairments following focal brain injury. Toward this end, the findings will be important in directing intervention efforts.

Pediatric Stroke

Mechanisms

Risk Factors.

Embolus (i.e., blood flow is blocked by a piece of material that formed in one place and travelled to another) and thrombosis (i.e., blood flow is blocked by a blood clot formed locally on the blood vessel wall) are the basic mechanisms of ischemic stroke (Blumenfeld, 2010). A number of risk factors have been identified for pediatric stroke. Many questions remain concerning the underlying mechanisms of pediatric stroke, as one-third of cases are idiopathic (Friedman, 2009) and half of cases present with more than one risk factor (Levine, 2012). Risk factors for pediatric stroke may be categorized according to the following: cardiac disorders, arteriopathies, infections, and hematologic disorders.

Cardiac Disorders.

Congenital heart disease (i.e., CHD; structural heart defect present at birth) accounts for about 15-30% of cases of pediatric stroke (Ciceri et al., 2011; Friedman, 2009). Children with CHD are particularly at high risk if they present with right-to-left shunt (Kirton, Westmacott, & de Veber, 2007; Shellhaas & Smith, 2010). Other cardiac conditions associated with risk of stroke include cardiomyopathy (i.e., chronic disorder of the heart muscle), cardiac tumors (i.e., abnormal growths in the heart), carditis (i.e., inflammation of the heart), endocarditis (i.e., inflammation of the endocardium), artificial valves (i.e., device used to repair/replace damaged heart valves), and arrhythmias (i.e., abnormal heart rhythm) (Freidman, 2009).

Arteriopathies.

Arteriopathies are a heterogeneous group of disorders of the arteries that account for about 50% of non-neonatal strokes (Chabrier, Husson, Lasjaunias, Landrieu, & Tardieu, 2003). Commonly identified arteriopathies in pediatric stroke include transient cerebral arteriopathy (i.e., stenosis shows regression or non-progression on follow-up imaging), Moyamoya disease (i.e., cerebrovascular disorder characterized by occlusion of the distal internal carotid arteries), Sickle cell disease (i.e., SCD; genetic blood disorder in which red blood cells are abnormally shaped), and dissection (i.e., tear on the inner surface of the artery) (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009; Kirton, Westmacott, & de Veber, 2007). Arteriopathies may also be caused by genetic conditions, such as Down syndrome, neurofibromatosis (i.e., genetic disorder that causes tumors to form on nerve tissue), and PHACE (i.e., posterior fossa brain malformations, facial hemangiomas, arterial abnormalities, coartation of the aorta and cardiac defects and eye abnormalities) syndrome (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009; Kirton, Westmacott, & de Veber, 2007).

Infections.

Infections account for about one-quarter of pediatric strokes (Pavlakis & Levinson, 2009). Common infections associated with pediatric stroke include varicella (i.e., chicken pox), encephalitis, and meningitis (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009). Infections are thought to lead to stroke via a systemic inflammatory response or infection of the endothelium (Pavlakis & Levinson, 2009).

Hematologic Disorders.

Common hematologic disorders linked to pediatric stroke include SCD, thrombophilias, and iron deficiency anemia (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009; Pavlakis & Levinson, 2009). SCD is the most important hematologic disorder associated with pediatric stroke, as the risk of stroke is 200 times greater in children with SCD than other children (Ciceri et al., 2011). Thrombophilias are a group of genetic or acquired conditions associated with an increased risk of thrombosis (Friedman, 2009).

Other.

Additional risk factors for pediatric stroke include head trauma, malignancy, chemotherapy, toxins/drugs, and dehydration (Friedman, 2009; Pavlakis & Levinson, 2009). A variety of maternal risk factors have been associated with perinatal stroke, including history of infertility, preeclampsia (i.e., condition in pregnancy characterized by hypertension and kidney problems), chorioamnionitis (i.e., placental infection), emergency cesarean section, oligiohydramnious (i.e., low levels of amniotic fluid), infection during pregnancy, coagulation disorders, and older maternal age (Pavlakis & Levinson, 2009; Shellhass & Smith, 2009; Stiles, 2010).

Vascular Supply.

The arterial supply to the brain consists of the anterior circulation stemming from the internal carotid arteries (i.e., carotid system) and the posterior circulation stemming from the vertebral arteries (i.e., vertebral-basilar system) (Blumenfeld, 2010). The anterior circulation supplies 80% of the brain and originates at the common carotid artery, which branches off from the aortic arch and splits into the internal and external carotid arteries (Festa et al., 2008). The external carotid artery supplies blood to the face, skull, and meninges, while the internal carotid artery branches into the opthalamic, posterior communicating, anterior choroidal, anterior cerebral, and middle cerebral arteries (Blumenfeld, 2010). The vertebral arteries arise from the subclavian arteries, enter the skull through the foramen magnum, and join to form the basilar artery (Blumenfeld, 2010). The anterior and posterior circulations meet at an arterial ring known as the circle of Willis, which is located in the subarachnoid space ventral to the frontal cortex (Festa et al., 2008).

The three main arteries supplying the brain are the anterior cerebral arteries (ACAs), middle cerebral arteries (MCAs), and posterior cerebral arteries (PCAs) (Blumenfeld, 2010). The ACA travels medially in the interhemispheric fissure and then travels back over the corpus callosum (Blumenfeld, 2010). The ACAs are connected by the anterior communicating artery. The two major branches of the ACA are the pericallosal and callsomoarginal arteries (Blumenfeld, 2010). The ACA supplies the anterior medial surface of the brain, covering territory spanning from the frontal to the anterior parietal lobes (Blumenfeld, 2010). The MCA splits into superior and inferior divisions in the Sylvian fissure, which supply the lateral cortex above and below the Sylvian fissure, respectively (Blumenfeld, 2010). Specifically, the superior division supplies the insula, frontal lobe, and superior parietal regions, while the inferior division

supplies the temporal and inferior parietal lobes (Festa, Lazar, & Marshall, 2008). The PCA arises from the basilar artery and sends branches supplying the inferior and medial temporal lobes and occipital cortex (Blumenfeld, 2010).

Vascular Distributions of Pediatric Stroke.

In general, the majority of perinatal strokes involve the territory of the left MCA (Stiles et al., 2010; Venkataraman et al., 2004). Term infants often have large MCA infarcts, resulting in damage to cortical and subcortical regions (Stiles et al., 2010). As with adults, the predominance of MCA infarcts appears to be largely due to the large territory supplied by the MCA. Likewise, the high incidence of left hemisphere infarcts is related to anatomical differences between the left and right carotid arteries (Coker, Beltrain, Myers, & Hmura, 1988). More specifically, Coker et al. (1988) note that a large majority of perinatal strokes result from emboli arising from the placenta. The embolus travels to the heart via the umbilical veins and then flows to the right heart across the foramen ovale into the left heart and then out to the aorta (Coker et al., 1988). The embolus is then more likely to travel to the left common carotid artery, as it is exposed to turbulent flow from the ductus (Coker et al., 1988). Next, vertebral dissections are more common than carotid dissections in children due to the higher susceptibility of the vertebral arteries to trauma (Riel-Romero, Kalra, & Gonzalez-Toledo, 2009). Thus, infarcts resulting from dissections often affect the posterior circulation (Riel-Romero et al., 2009). Moyamoya disease and SCD are associated with blockage of the distal internal carotid arteries and the proximal ACAs and MCAs (Riel-Romero et al., 2009). Finally, children with strokes resulting from varicella are more likely to have infarcts involving the basal ganglia (Riel-Romero et al., 2009).

Summary.

The basic mechanisms of ischemic stroke include embolus and thrombosis. Although numerous risk factors have been identified in pediatric stroke, an understanding of the underlying etiology remains unclear in many cases, with one-third being idiopathic (Friedman, 2009) and half presenting with more than one risk factor (Levine, 2012). The most common etiologic categories for pediatric stroke include cardiac disorders, arteriopathies, infections, and hematologic disorders. Cardiac disorders associated with pediatric stroke include CHD, cardiomyopathy, cardiac tumors, carditis, endocarditis, artificial valves, and arrhythmias, with CHD being the most common (Ciceri et al. 2011; Freidman, 2009). Arteriopathies represent an important etiologic group of pediatric stroke, accounting for approximately 50% of non-neonatal strokes (Chabrier, Husson, Lasjaunias, Landrieu, & Tardieu, 2003). Common arteriopathies identified in pediatric stroke include transient cerebral arteriopathy, Moyamoya disease, SCD, and dissection (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009; Kirton, Westmacott, & de Veber, 2007). Infections account for about one-quarter of pediatric strokes (Pavlakis & Levinson, 2009), with the commonest being varicella, encephalitis, and meningitis (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009). Hematologic risk factors include SCD, thrombophilias, and iron deficiency anemia (Ciceri et al., 2011; Fox & Fullerton, 2010; Friedman, 2009; Pavlakis & Levinson, 2009). The vascular territory most commonly implicated in pediatric stroke is the left MCA (Stiles et al., 2010; Venkataraman et al., 2004). Given that the MCA supplies blood to brain structures that are critical for language (i.e., Broca's area, Wernicke's area), motor functioning (i.e., motor cortex, striatum, globus pallidus, internal capsule), sensation (i.e., sensory cortex), it is not surprising that blockages affecting this

circulation can result in a wide variety of neurologic sequelae. The proceeding section will provide a brief overview of the neuropsychological profile of pediatric stroke.

Neuropsychological Profile

Intelligence.

Recent studies on global intellectual outcomes after perinatal and childhood stroke indicate that overall intelligence is generally within the broad range of average, although compromised compared to normative samples (Allman & Scott, 201; Ballantyne et al., 2008; Hartel, et al., 2004; Max et al., 2010; Nass & Trauner, 2004; Westmacott et al., 2009, 2010). The prognostic value of findings of intellectual outcomes in this population is limited by the mixed findings produced by longitudinal analyses. That is, some studies report that intellectual functioning after pediatric stroke remains relatively stable over time (Aram & Eisele, 1994; Ballantyne et al., 2008), while other studies reveal emerging cognitive deficits during the school-age years (Levine et al., 2005; Westmacott et al., 2009). Sample characteristics may have led to these conflicting findings, as several of the studies included children with histories of seizure disorders (Ballantyne et al., 2008; Levine et al., 2005), while others excluded children with neurological comorbidities (Aram & Eisele, 1994; Westmacott et al., 2009). The test-retest intervals also varied between studies, with the minimum time between assessments ranging from 6 months (Aram & Eisele, 1994) to 18 months (Ballantyne et al., 2008; Levine et al., 2005; Westmacott et al., 2009) and the maximum time ranging from about 8 years (Aram & Eisele, 1994; Ballantyne et al., 2008; Westmacott et al., 2009) to 15 years (Levine et al., 2005). The clinical utility of findings on cognitive outcomes after pediatric stroke is further limited by the large amount of inter-subject variability in intellectual functioning. For example, closer examination of studies examining cognitive functioning after pediatric stroke reveals intelligence

quotients ranging from the very limited range (i.e., Full Scale IQ ≤ 70) to superior range (i.e., Full Scale IQ > 120) (Allman & Scott, 2011; Ballantyne et al., 2008; Everts et al., 2008; Max et al., 2004). Multiple factors likely account for this diversity in intellectual outcomes, including: age at stroke, age at assessment, lesion characteristics, and presence of seizure disorders.

Language.

Studies have consistently reported an overall delay in the onset of language after perinatal stroke regardless of the side of the lesion, followed by a normal trajectory of receptive and expressive language development (Avila et al., 2010; Bates et al., 1997; Marchman et al., 1991; Stiles et al., 2010). Functional neuroimaging findings indicate the reorganization of language areas to the right hemisphere following left perinatal stroke, regardless of the extent of the lesion (Tillema et al., 2008). Although the immature brain appears to have the plasticity to sufficiently overcome the effects of early injury with regard to functional language abilities, emerging evidence reveals persisting difficulties in higher-order language abilities (e.g., discourse, semantics, etc.) (Avila et al., 2010; Chapman et al., 2003; Reilly, Losh, Bellugi, & Wulfeck, 2004). In contrast to the perinatal stroke group, strokes acquired later in childhood result in acute aphasia when lesions involve the left hemisphere (Gout et al., 2005; Kirton et al., 2007; Nass & Trauner, 2004). As with data reported on global intellectual functioning (see above), the presence of seizure disorders is associated with compromised language functioning (Avila et al., 2010).

Memory and Learning.

Although there is a paucity of research in this area, the limited data suggest that memory and learning are vulnerable to the effects of pediatric stroke. Studies report subtle, non-lateralized verbal memory deficits in children with histories of stroke relative to controls (Block et al., 1999; Lansing et al., 2004). The verbal learning and memory profiles of children with

histories of stroke is characterized by reduced encoding, less use of learning strategies to enhance recall, and reduced delayed free recall and recognition (Lansing et al., 2004). Further, early stroke (i.e., occurring before age 1) is associated with poorer performance (Lansing et al., 2004). In addition, recent evidence indicates that children with stroke secondary to SCD display deficits in prospective memory (McCauley & Pedroza, 2010). Clearly, more research is needed to explore a broad range of memory functioning, determine the effects of lesion characteristics and age of onset, and examine the relation between memory performance and other domains of functioning (e.g., social, academics, etc.).

Visual-Spatial Processing.

Children with histories of stroke demonstrate subtle, persistent deficits in visual-spatial processing (Akshoomoff et al., 2002; Nass & Trauner, 2004; Schatz et al., 2000, 2004; Stiles et al., 1997, 2008, 2010). On drawing tasks, young children with perinatal stroke show evidence of impairment regardless of the side of the lesion (Akshoomoff et al., 2002; Stiles, et al., 1997). With development, performance on drawing tasks improves, but subtle deficits remain and distinctive profiles among children with left versus right hemisphere strokes emerge (Akshoomoff et al., 2002; Stiles, et al., 1997). A double-dissociation in global-local processing has consistently been reported in the pediatric stroke literature, with children with damage to the right hemisphere showing difficulty with processing overall visual patterns and children with damage to the left hemisphere showing deficits in processing details (Schatz et al., 2000; Stiles, 2008). These findings confirm the hemispheric specialization of visual-spatial processing present early in development. Of note, given that the majority of studies consist of samples of children with perinatal stroke, caution is warranted in extending these findings to children with stroke occurring in later childhood.

Motor Outcomes.

Motor impairment is a common consequence of pediatric stroke, with an occurrence of 30-60% of cases (Golomb, Bhuwan, Saha, Azzouz, & Williams, 2008; Hartel et al., 2004). In fact, perinatal stroke has been reported to be the most common cause of congenital hemiplegic cerebral palsy (CP) (Kirton et al., 2007). The hemiplegic CP seen in children with histories of perinatal stroke is marked by weakness, spasticity, impaired dexterity, apraxia, and a circumducting gait (Stiles et al., 2010). The upper extremities are affected more often than the lower extremities, as the majority of strokes involve the MCA (Boardman et al., 2005; Kirton et al., 2007). Mixed infarctions in the MCA region and corticospinal tract involvement have been found to be predictive of hemiplegia following neonatal stroke (Husson et al., 2009). Moreover, asymmetrical wrist movements and reduction of digit movements has recently been found to be a predictor of hemiplegia following neonatal stroke (Guzzetta et al., 2009). In neonatal stroke, delayed presentation (i.e., neurologic symptoms emerging after 1 month of age), large-branch infarction, and male sex have also been identified as predictors of CP (Golomb et al., 2008). Observations of infants with neonatal stroke reveal that they spend less time manipulating, touching, and grasping toys with both hands, suggesting deficits in bimanual task performance (Chen, Lo, & Heathcock, 2013). Concomitant involvement of the basal ganglia, posterior limb of the internal capsule, and cerebral cortex is associated with hemiparesis in neonatal unilateral MCA stroke (Boardman et al., 2005). In contrast, involvement of any one of these structures increases the likelihood of hemiparesis in older children with unilateral MCA stroke (Boardman et al., 2005). Dystonia has also been found to be more common in older children with histories of stroke (Boardman et al., 2005). Finally, bilateral strokes are associated with triplegia/ quadriplegia (Boardman et al., 2005).

Age at Onset, Lesion Location, and Cognitive Outcomes.

Research on pediatric stroke suggests that there is a non-linear relationship between age at stroke and cognitive outcomes. Recent evidence indicates that there is a protective effect for strokes occurring during mid-childhood (Allman & Scott, 2011; Everts et al., 2008). It is proposed that the period of mid-childhood has the benefits of increased plasticity compared to the older brain (i.e., > 10 years) and reduced vulnerability compared to the immature brain (i.e., < 1 year) (Allman & Scott, 2011; Everts et al., 2008). Researchers suggest that the capacity for reorganization in the immature brain is not realized very early in development due to the rapid changes in brain development occurring during this time (Everts et al., 2008; Westmacott et al., 2010). Early injury is thought to disrupt the course of myelinization, particularly in the frontal lobes, thereby rendering the developing brain less capable of supporting higher-level cognitive skills, such as WM (Max, 2004). Moreover, Westmacott et al. (2010) point out that, because later-maturing brain regions rely on the development of early-maturing brain regions, damage in one brain region early in development has the potential to disrupt the maturation of other brain regions later in development. Consequently, children often "grow into" their deficits later in development and show difficulties in high-order cognitive skills (Westmacott et al., 2009). Conversely, although brain networks may be less vulnerable in the older brain, the potential for plasticity is also lower (Allman & Scott, 2011). Of note, mixed findings have been reported regarding the precise age range associated with the best cognitive outcomes. For example, some researchers have reported that the best outcomes are associated with stroke occurring between 5 and 10 years of age (Everts et al., 2008; Nass & Trauner, 2004) while others have found that the best outcomes are associated with stroke occurring between 1 and 5 years (Allman & Scott, 2011).

The relationship between age at stroke and cognitive outcomes has been reported to be moderated by lesion location (Westmacott et al., 2010; Nass & Trauner, 2004). Overall, cognitive sequelae are more severe in children with stroke involving both cortical and subcortical regions (i.e., infarct involving the cortex plus basal ganglia and/or thalamus) compared to stroke affecting either cortical or subcortical regions alone, even after accounting for lesion size (Westmacott et al., 2010). Studies have revealed different periods of vulnerability for cortical and subcortical infarcts, with subcortical strokes being most detrimental to cognitive outcomes during the perinatal period and cortical strokes being most detrimental in early childhood (i.e., 1 month-5 years) (Nass & Trauner, 2004; Westmacott et al., 2010). The earlier period of peak vulnerability for subcortical infarcts is proposed to be due to (a) the possible role of subcortical tissue in very early skill acquisition, (b) the rapid changes occurring in subcortical structures during early development; and/or, (c) the possible disruptive effect of subcortical pathology to later cortical and white matter maturation (Nass & Trauner, 2004; Westmacott et al., 2010). In summary, optimal recovery may be observed following stroke in mid-childhood because this period has the advantages of reduced vulnerability compared to the very young brain and enhanced plasticity compared to the older brain. Moreover, different periods of peak vulnerability are observed with strokes involving cortical and subcortical regions.

Summary.

The cognitive profiles of children with histories of stroke are marked by subtle deficits in overall intellectual functioning, language, visual-spatial processing, and memory and learning. Studies of intellectual functioning suggest that children with stroke generally perform in the low end of the average range, with outcomes varying according to age at stroke and lesion location (Allman & Scott, 201; Ballantyne et al., 2008; Hartel, et al., 2004; Max et al., 2010; Nass &

Trauner, 2004; Westmacott et al., 2009, 2010). Research on the language profiles of this population indicate that perinatal stroke affecting either hemisphere is associated with normative functional language development following an initial delay (Avila et al., 2010; Bates et al., 1997; Marchman, Miller, & Bates, 1991; Stiles et al., 2010), whereas childhood stroke leads to language impairments after left hemisphere damage (Gout et al., 2005; Kirton, Westmacott, & deVeber, 2007; Nass & Trauner, 2004). Both perinatal and childhood stroke profiles are marked by deficits in higher-order language abilities (Avila et al., 2010; Ballantyne, Spilkin, & Trauner, 2007; Chapman et al., 2003; Reilly et al., 2004). Verbal memory and learning profiles in children with stroke are marked by subtle, non-lateralized deficits (Block, Nanson, & Lowry, 1999; Lansing et al., 2004). In contrast, children with perinatal stroke demonstrate subtle, lateralized deficits in visual-spatial processing (Akshoomoff et al., 2002; Nass & Trauner, 2004; Schatz et al., 2004; Schatz, Ballantyne, & Trauner, 2000; Stiles et al., 1997, 2008, 2010). More specifically, evidence has been found for a double-dissociation in global-local processing (Schatz et al., 2000, 2004; Stiles et al., 2008). Motor impairment is the most commonly recognized consequence of pediatric stroke, affecting 30-60% of cases (Golomb, Bhuwan, Saha, Azzouz, & Williams, 2008; Hartel et al., 2004) and compromising deficits such as hemiplegic CP, hemiparesis, dystonia, and triplegia/quadriplegia (Boardman et al., 2005). Overall, evidence has been found for a non-linear relationship between age at stroke and cognitive outcomes, suggesting a protective effect for strokes occurring during mid-childhood (Allman & Scott, 2011; Everts et al., 2008). The relationship between age at stroke and cognitive outcomes is moderated by lesion location, as different periods of vulnerability have been identified for cortical and subcortical infarcts (Westmacott et al., 2010). WM and SIP represent two key domains that have remained largely unexplored in children with histories of stroke, thereby

rendering our understanding of the neuropsychological profiles of this population incomplete. The proceeding sections will provide a comprehensive overview of the theoretical models, neural correlates, normative development, and outcomes following pediatric stroke of WM and SIP.

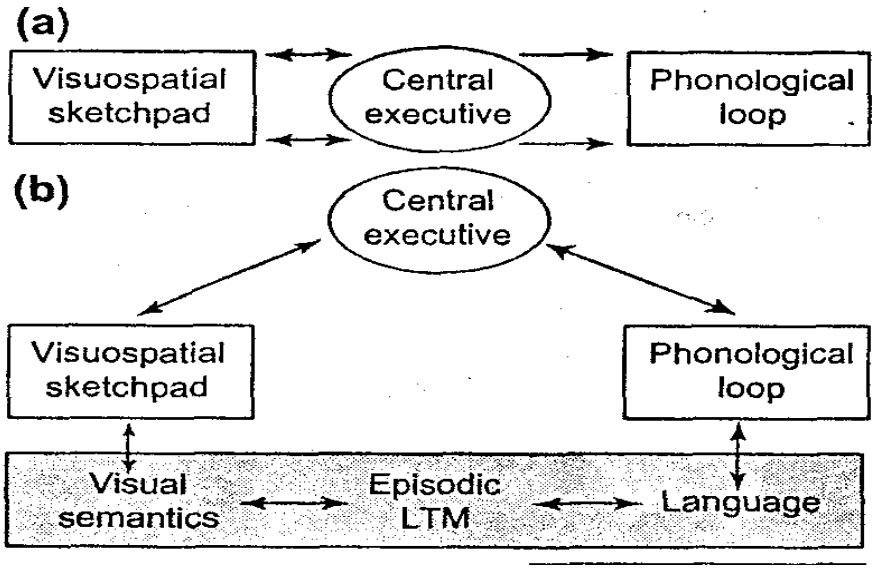
Working Memory

Theoretical Model

The multi-component model proposed by Baddeley and Hitch (1974) and later extended by Baddeley (2000) has been one of the most influential models of WM to date (see Figure 1). The original model put forward by Baddeley and Hitch (1974) consists of three components: the phonological loop, the visuospatial sketch pad, and the central executive. The phonological loop refers to the storage and rehearsal mechanisms of verbal material. The phonological loop consists of a phonological store that is commonly known as "short-term memory." The capacity of the phonological store is typically evaluated through verbal recall tasks that require the individual to retain arbitrary information (e.g., digits, words) (Alloway et al., 2004). The capacity of the phonological store is dependent on the amount of information that needs to be held in mind and rehearsal of the material. Information in the phonological store is refreshed by subvocal rehearsal of the information. The visuospatial sketchpad represents a similar system that is specialized for the storage and processing of visual information. Collectively, the visuospatial sketchpad and the phonological loop are conceived of as "slave systems" that subserve the central executive. The central executive component is responsible for the activation of long-term memory and the coordination between the phonological loop and the visuospatial sketchpad. The capacity of the central executive is typically assessed by complex tasks that require participants to simultaneously store and process information (e.g., recalling a series of digits in reverse order) (Alloway et al., 2004). In the latest revision of the model, Baddeley (2000) proposed that the

WM system consists of a fourth component – the episodic buffer. This component is responsible for integrating the separate components of WM and long-term memory into a single representation.

Figure 1: (a) The original multi-component model of WM proposed by Baddeley and Hitch (1974) and (b) the revised model proposed by Baddeley (2000)



Note: From "The episodic buffer: a new component of WM?," by A. Baddeley (2000), *Trends in Cognitive Sciences, 4,* p. 418.

Alloway et al. (2004) found strong support for the multi-component model of WM (Baddeley & Hitch; 1974; Baddeley, 2000) after examining the functional organization of WM in 633 children between the ages of 4 and 6 years. Participants were tested on measures that were designed to correspond to the central executive, phonological loop, and episodic buffer components of the multi-component model. Specifically, participants were evaluated based on their performance on tasks of verbal short-term memory (i.e., phonological store component of the phonological loop), complex memory span (i.e., central executive), sentence repetition (i.e.,

episodic buffer), phonological awareness, and non-verbal ability. After investigating the underlying factors contributing to variations in performance using regression analyses, Alloway et al. (2004) discovered that the data fit best with the three constructs of the multi-component model. In other words, the children's ability to temporarily store verbal information was distinct from both their abilities to engage in more complex verbal memory tasks and to repeat sentences.

Neural Correlates

The pivotal role of the prefrontal cortex (PFC) for WM has been established from electrophysiological, lesion, and imaging studies (see review, Khan & Muly, 2011). However, the functional organization of the PFC for WM remains a topic of debate. Levy and Goldman-Rakic (2000) offer a domain-specific account of WM, in which there is a distinction between the content processed in the dorsolateral and ventrolateral regions of the PFC. Based on findings drawn from experimental studies with monkeys, it is suggested that the ventral region is specialized for object recognition during visual WM tasks while the dorsal region is specialized for spatial WM. In contrast, Petrides (1996, 2000) argues that the dorsolateral and ventrolateral regions are specialized for different processes (rather than different types of material). Petrides (1996, 2000) proposes a hierarchical model of WM in which the posterior cortex is viewed as the storage site for information and the ventrolateral PFC is responsible for retrieving and maintaining information stored in the posterior cortex. The type of information retrieved and maintained in the ventrolateral PFC is thought to be lateralized, such that the right hemisphere is specialized for visual-spatial information and the left hemisphere is responsible for verbal information. The right dorsolateral PFC is critical for manipulating the content of WM, while the left dorsolateral PFC is responsible for monitoring the information that is being held in mind. Support for this model was found in a positron emission tomography (PET) study of spatial WM

conducted by Owen, Evans, and Petrides (1996). The findings showed that maintenance of the spatial information was associated with activity in the ventrolateral PFC, while manipulation and monitoring of the information was associated with dorsolateral activity (Owen et al., 1996).

In a recent review of the neurological underpinnings of WM, Khan and Muly (2011) emphasize that WM depends on the interactions between the PFC and cortical and subcortical brain regions. In terms of its subcortical connections, the thalamus has been identified as a major source of incoming information to the PFC. Lesions to the mediodorsal thalamic nucleus have been shown to impair performance on WM tasks. The basal ganglia is also a key source of output of the PFC, and has been proposed to play a role in selective attention to relevant stimuli during WM tasks. After examining the WM performance of patients with a history of stroke, Baier et al. (2010) reported that damage to the basal ganglia resulted in increased vulnerability to the influence of distracting stimuli. A number of cortical connections of the PFC have been implicated in WM. The PFC receives input from the inferior temporal and posterior parietal cortical areas, which play a crucial role in encoding visual information. These areas are associated with the dorsal and ventral visual pathways, which process objects and visual-spatial information, respectively. The PFC is also connected to the medial temporal lobe. Activation of entorhinal, perirhinal, and hippocampal neurons have been detected during WM tasks that involve novel stimuli. These findings indicate that an intact medial temporal lobe is necessary for WM performance specifically when the task involves unique stimuli. Finally, the PFC is also connected to the anterior cingulate cortex, which has been linked to monitoring processes during WM tasks. For example, the anterior cingulate cortex has been associated with error detection, and activity in this area has been associated with reward expectations. These findings highlight

the importance of the PFC and its interactions with cortical and subcortical brain regions in the neural network underlying WM.

Development

WM capacity develops across childhood and well into adulthood (Swanson, 1999). In an effort to determine the mechanisms underlying age-related changes in WM, Swanson (1999) examined verbal and visual WM performance in 778 participants between the ages of 6 and 57 years. Swanson (1999) assessed age-related differences in processing capacity and efficiency by evaluating performance levels under conditions of initial recall, cued recall, and maintenance (recall of the highest item on the cued recall condition but without the presentation of cues). The results showed a continuous age-related pattern of improvement in performance in both verbal and visual WM across recall conditions that reached a peak at age 45. These results suggest that age-related changes in the amount of information that can be maintained and manipulated occur throughout childhood and well into adulthood.

In line with these findings, rapid changes in the brain's organization of visual WM have been documented during the school-age years (Farber & Beteleva, 2011). For example, Farber and Beteleva (2011) reported age-related differences in the activation of a number of cortical areas after using an event-related potential (ERP) analysis to compare 7-8 and 9-10 year old children during a visual WM task. Both groups showed activation in the visual cortical areas during the task. However, differences were found in activation patterns at various stages of the task. During the initial encoding stage, the 9-10 year old group showed an increase in the activation of the inferior frontal cortex that was not detected in the 7-8 year old group. These results indicate an age-related change in the processing of visual information. During the cognitive part of the task, only the 9-10 year old group showed activation of the PFC. After

comparing the results to data collected from adults, Farber and Betelava (2011) discovered that the overall pattern of ERP changes was similar between the groups. Of note, although the overall pattern between adults and 9-10 year olds was similar, the dorsal regions of the PFC were found to be less involved during all stages of visual WM in the 9-10 year old group. It was concluded that the selective attention and regulatory mechanisms required for WM are formed between the ages of 9-10, and are associated with maturational changes in the prefrontal cortex. However, the authors cautioned that it remains unclear whether any age-related changes in the dorsal regions of the PFC may occur beyond the age of 10.

Age-related changes in brain connectivity during verbal WM tasks have been reported during childhood and adolescence. In a recent fMRI study, Booth, Mehdiratta, Burman, and Bitan (2008) compared patterns of connectivity in children between the ages of 9 and 15 years during a verbal WM task. Children were required to hold information in mind in order to determine whether two words were spelled the same. The children performed the task under separate visual and auditory conditions. The results showed an age-related increase in the connections between Heschl's gyrus and the dorsal inferior frontal gyrus during the auditory task. Similarly, an age-related increase in the connectivity from the calcarine and the superior temporal gyrus was found during the visual task. These results indicate that the connectivity between cortical regions and regions involved in phonological processing increase during childhood and adolescence.

WM Outcomes Following Pediatric Stroke

Several studies in adults indicate that WM is compromised following stroke (Hommel, Miguel, Naegele, Gonnet, & Jaillard, 2009; Philipose, Alphs, Prabhakaran, & Hillis, 2007; van Geldorp, Kessels, & Hendriks, 2013). Moreover, hemispheric side and lesion location are

associated with different WM deficits. Left cortical damage has been shown to result in impairment in the phonological loop (Philipose et al., 2007; Vallat, Azouvi, Hardisson, Meffert, Tessier, & Pradat-Diehl, 2005). Conversely, right cortical damage results in deficits in both the phonological loop and visuospatial sketchpad, thus indicating the critical role of the right cortex for multimodal WM processes (Philipose et al. 2007).

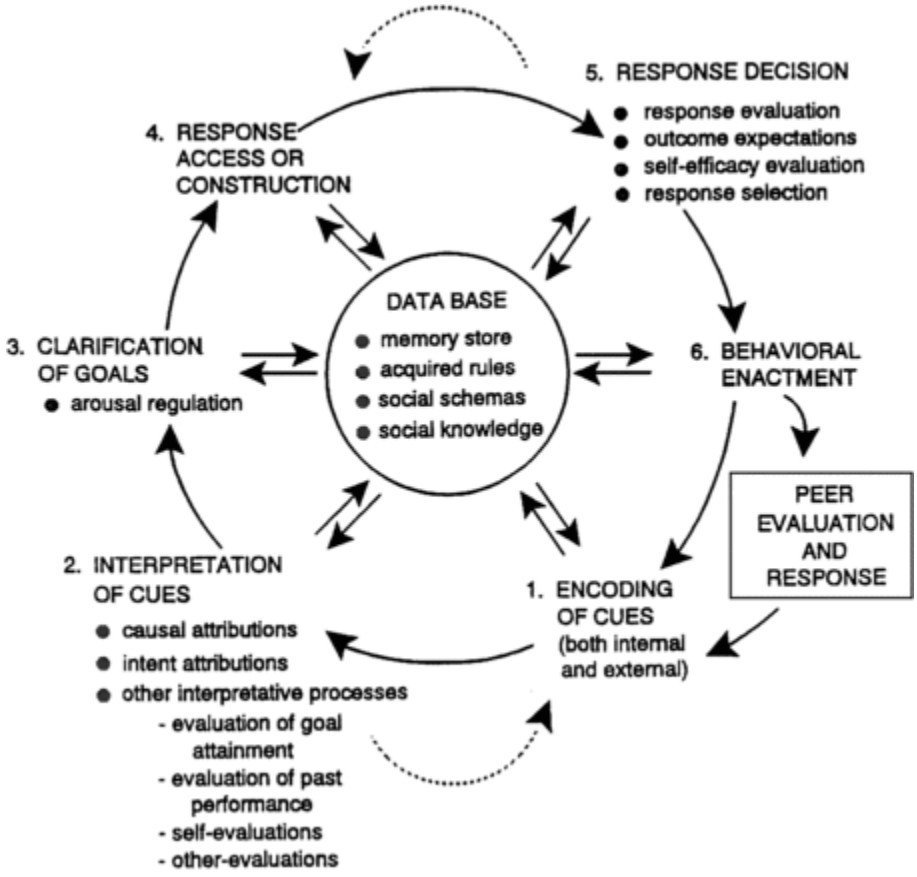
In contrast, much less is known about the consequences of pediatric stroke on WM performance. Emerging evidence indicates that children with stroke demonstrate subtle WM difficulties, with performance typically falling within the low average range (Lansing et al., 2004; Westmacott et al., 2009, 2010). These subtle deficits are associated with younger age at stroke (Lansing et al., 2004; Westmacott et al., 2010) and are reported to emerge during the school-age years (Westmacott et al., 2009). Different lesion locations may result in different patterns of WM performance in children. For example, White, Salorio, Schatz, and DeBaun (2000) found differential patterns of WM among children with anterior, posterior, and diffuse infarcts after examining performance on a word span task. Specifically, children with anterior infarcts demonstrated a reduced word length effect (i.e., performance did not improve with shorter words), despite the fact that their overall memory span was not significantly different than controls. Children with diffuse infarcts recalled fewer words but showed an intact word length effect. In contrast, the performance of children with posterior infarcts was comparable to controls. White et al. (2000) hypothesized that anterior infarcts caused dysfunction in the phonological loop while diffuse infarcts impaired the central executive. Of note, these findings are difficult to interpret due to the fact that the sample of participants with stroke was small (n = 20) and was comprised only of children with stroke secondary to SCD.

Social Information Processing

Theoretical Model

The model proposed by Crick and Dodge (1994) is considered the "gold standard" for conceptualizing SIP (see Figure 2). Crick and Dodge (1994) conceive of SIP as a form of "on-line brain performance" (p. 77) consisting of six problem-solving steps. Each step is influenced by the child's cognitive capabilities and database of past experiences. The model is proposed to be non-linear, such that the processing at each step occurs simultaneously. The child encodes social cues (step 1) and then engages in a number of interpretive processes in order to understand the situation (step 2). These interpretative processes are guided by the experiences stored in long-term memory, and may include an analysis of the cause of the event, an assessment of the perspectives of others, an evaluation of past performance, and an evaluation about the meaning of the event for the self and others. The child then decides upon a goal for the situation (step 3) and proceeds to generate possible responses (step 4). The child selects a specific response after considering the outcomes they expect to occur, their confidence in their own ability to successfully carry out the response, and the appropriateness of the response (step 5). Finally, the child enacts their chosen response (step 6). The child's ability to successfully carry out the steps involved in SIP depends upon their ability to temporarily hold social cues in mind while simultaneously accessing past events from their long-term memory and engaging in social reasoning.

Figure 2: Model of social information processing



Note: From "A review and reformulation of social information-processing mechanisms in children's social adjustment," by N. R. Crick & K. A. Dodge (1994), *Psychological Bulletin, 115,* p. 74.

Neural Correlates

SIP is mediated by a complex network of brain regions. Different brain regions are important for different aspects of SIP. The amygdala, basal ganglia, insula, and superior temporal sulcus are critical for encoding social cues (Beauchamp & Anderson, 2010; Ward, 2006; Yeates et al., 2007). For example, bilateral lesions to the amygdala have been shown to impair the processing of fearful stimuli and facial expressions of sadness, while damage to the ventral regions of the basal ganglia has been shown to impair the processing of anger

(Beauchamp & Anderson, 2010; Ward, 2006; Yeates et al., 2007). fMRI studies have reported selective activation in the insula in response to facial expressions of disgust (Ward, 2006). Lesions to the superior temporal sulcus have been reported to result in impaired detection of eye gaze direction (Ward, 2006). Thus, multiple brain regions are involved in the encoding of social cues (i.e., step 1 in the SIP model).

The PFC is critical for the higher-order thinking involved in SIP (Anderson & Beauchamp, 2010; Powell et al., 2010; Willis et al., 2010). The orbital PFC has been linked with the capacity to judge the approachability of emotional faces (Willis et al., 2010) and understand the mental states of others (i.e., theory of mind) (Powell et al., 2010). The ventromedial region of the PFC has been implicated in the perception of social interactions between others (referred to as "offline" processes of SIP), while the dorsomedial PFC has been linked with the perception of social interactions involving the self (referred to as "online" processes of SIP) (Schilbach et al., 2006). Notably, the PFC matures gradually throughout childhood and adolescence (Anderson & Beauchamp, 2010). Thus, damage to the PFC of the developing brain can potentially disrupt a variety of higher-order processes involved in SIP.

Development

The socio-cognitive integration of abilities model (SOCIAL) proposed by Beauchamp and Anderson (2010) provides a useful framework for understanding the development of social skills by demonstrating the biological, psychological, and environmental underpinnings of social behaviour. Attention-executive, communication, and socio-emotional abilities are identified as the key cognitive functions for social skills. Attention-executive skills include a broad range of higher-order abilities, such as attentional control, WM, goal-setting, problem-solving, and planning. Communication skills refer to a range of abilities that allow individuals to understand

or express messages in social interactions, such as expressive and receptive language skills. The socio-emotional component of the model includes affective processes that are critical for social skills, such as face/emotion perception, intent attribution, theory of mind, and moral reasoning. The emergence of these skills is mediated by brain development and integrity as well as internal/ external factors. Internal and external factors include a range of individual and environmental influences, such as personality and temperament, physical attributes, family function, socioeconomic status (SES), and culture. Thus, the emergence of the cognitive functions required for successful social outcomes relies on normal brain development within an optimal environment.

SIP develops from the acquisition of cognitive skills and an increase in information processing speed throughout childhood and adolescence (Crick & Dodge, 1994). Early social experiences create synaptic pathways in the brain, which allow for more efficient SIP (Crick & Dodge, 1994). An innate bias for social interactions is observed early in development, with infants demonstrating a preference for social stimuli at 2-3 months of age (Kelly et al., 2005). Affect recognition of faces and voices is achieved at 7 months of age (Grossman, Striano, & Friederici, 2006). By 9 months of age, infants demonstrate the ability to share attention with another individual in reference to a third event (i.e., joint attention) (Grossman et al., 2006). Beauchamp and Anderson (2010) emphasize that joint attention is a key milestone in the development of social skills because it is theorized to underlie the ability to understand the perspectives of others (i.e., theory of mind). The preschool years are marked by a shift from egocentric thinking to perspective taking. Children develop an understanding of false beliefs and intentions of others between 4-5 years of age (Baron-Cohen & Ring, 1994). Children continue to develop SIP skills throughout development as they gain exposure to a range of social

experiences. Social encounters supply children with knowledge regarding the intents of others, appropriate goals and responses, cause of events, etc. These experiences allow children to develop more efficient methods of organizing and understanding social information. Rapid development of social skills occurs in adolescence as a result of neurostructural and environmental changes. Adolescents are exposed to novel social situations as they attempt to increase their independence. At the same time, a decrease in grey matter and increase in white matter in the frontal and parietal cortices occurs in adolescence, which functions to increase the speed of processing (Beauchamp & Anderson, 2010). Essentially, the acquisition of cognitive skills and increased processing speed seen throughout development allow children and adolescents to become more efficient social information processors.

Milch-Reich et al. (1999) found clinical and developmental differences in children's SIP after examining 79 boys between 5 and 10 years with and without Attention Deficit Hyperactivity Disorder (ADHD). A 4-phase experimental approach was used to examine children's accessibility to social schemes, on-line representation of social events, recall of social events, and social reasoning. Participants were presented with a series of stories depicted through pictures and asked to explain what was happening, recall it from memory, and problem-solve about the social situations shown in the stories. Children's on-line processing of the events was assessed by measuring the time they spent looking at the pictures and by analyzing their verbal descriptions of the events. The results showed developmental differences in SIP, with younger children showing less accessibility to social schemes, reduced looking time and encoding of social cues, and less integrated representations of events. Clinical differences were also detected, with the ADHD group showing poorer recall and social reasoning skills. This study highlights

the rapid changes in SIP that occur during the elementary school years and the negative impact of attentional problems on SIP.

SIP Outcomes Following Pediatric Stroke

Recent findings indicate the presence of social impairments in children and adolescents with stroke (Ballantyne & Trauner, 1999; Boni, Brown, Davis, Hsu, & Hopkins, 2001; Mosch et al., 2005; Trauner, Panyard-Davis, & Ballantyne, 1996). The hemispheric side of the lesion may not correlate with social functioning following pediatric stroke. In a study pairing adults and children with similar unilateral stroke lesions, Mosch et al (2005) found laterality effects in the social functioning of adults, but not children. Adults with right hemisphere lesions showed significantly worse social functioning compared to adults with left hemisphere lesions. In contrast, children showed mild to moderate deficits in social functioning, regardless of lesion location. Trauner, Panyard-Davis, and Ballantyne (1996) documented that children with perinatal stroke were rated by parents as having more social impairments compared to controls on the Personality Inventory for Children. Although hemispheric side of the lesion was not found to influence social functioning, children with posterior lesions were reported to have higher rates of social deficits compared to children with anterior lesions. Similarly, studies examining quality of life in children with stroke have documented lower socialization scores compared to normative samples (Hurvitz et al., 2004; O'Keeffe et al., 2012). Finally, findings suggest that childhood stroke has a negative impact on social information encoding (Ballantyne & Trauner, 1999; Boni et al., 2001). Ballantyne and Trauner (1999) observed subtle deficits in facial recognition in children and adolescents with pre- or perinatal stroke, regardless of hemispheric side of the lesion. Right and left parietal lobe involvement was found to correlate with worse facial recognition performance, thus highlighting the pivotal role of this region for facial discrimination

early in development. Boni et al. (2001) similarly found that children with stroke secondary to SCD performed worse on measures of facial and vocal social cue encoding compared to children with SCD without stroke. Closer examination of the patterns of performance revealed that the stroke group performed worse when social cues were ambiguous. Conversely, children with stroke achieved greater success when the social situations were straight-forward and well-learned. Taken together, these findings suggest that children with histories of stroke are at increased risk for developing social difficulties. It will be crucial for future studies to conduct more detailed analyses of the precise nature of these social impairments and to examine this in relation to lesion characteristics and age at injury.

The Relationship between Working Memory and Social Information Processing

Recent evidence indicates that WM plays a critical role in SIP. The link between WM and SIP has been demonstrated in typically developing children (McQuade, Murray-Close, Shoulberg, & Hoza, 2013) and adults (Hecker & Dutke; 2004; Hills & Pachur, 2012; Thornton & Conway, 2013), children with ADHD (Kofler et al., 2011), and adults with brain injury (Anderson & Knight, 2010; Channon & Crawford, 2010; Hommel et al., 2009). For example, after examining the associations between WM components and teacher ratings of multiple measures of social functioning in 116 fourth- and fifth-grade typically developing children, McQuade et al. (2013) discovered that reduced central executive ability was linked with peer rejection, physical and relational aggression, impaired conflict resolution skills, and lower overall social competence. Poor phonological loop ability was also correlated with greater peer rejection, whereas no associations were found between visuospatial sketchpad ability and social functioning measures. The central executive has also been found to have a direct impact on the social functioning of children with ADHD, whereas the phonological loop and visuospatial

sketchpad have been found to influence social functioning in this population indirectly through their influences on hyperactive/impulsive and inattentive symptoms in this population (Kofler et al., 2011). Taken together, these findings indicate that the central executive plays an important role in social competence for typically developing children and children with ADHD.

WM has been shown to be a key cognitive determinant underlying social deficits following brain injury. After examining the cognitive and social functioning of 74 adults 6 months after stroke, Hommel et al. (2009) reported that 70% of the patients reported social impairment. After adjusting for stroke severity and depression, regression analyses revealed that reduced WM predicted worse social functioning. Consistent with these findings, Channon and Crawford (2010) showed that social problem-solving impairments were related to WM functioning in a group of 20 adults with acquired brain injury (10 resulting from a traumatic brain injury and 10 resulting from stroke). Compared to controls, the ABI group was poorer at interpreting social situations, generating solutions to social problems, and identifying the awkward elements in social situations. Further, WM performance was significantly correlated with all three of these social-problem-solving measures. Similarly, after examining WM and interpersonal functioning in 45 adults who had sustained traumatic brain injuries, Anderson and Knight (2010) reported an association between impaired functioning of the central executive component and reduced social competence. Taken together, these findings suggest that WM capacity may underlie the social deficits observed in adults after brain insult.

Research findings from studies in typically developing adults (Hills & Pachur, 2012; Hecker & Dutke, 2004; Thornton & Conway, 2013) suggest several potential mechanisms for the importance of WM for social functioning. First, as proposed by Hills and Pachur (2012), retrieving social memories recruits WM. This claim is supported by the finding of a significant

and positive correlation between central executive ability and performance on a computerized social recall task that required undergraduate participants to recall people that they knew as quickly as possible and to answer questions about the recalled individuals (Hill & Pachur, 2012). According to the SIP model proposed by Crick & Dodge (1994), the ability to quickly and efficiently retrieve social memories is critical for SIP, as one's long-term database of social memories ultimately guides one's attribution, goal, and response decision-making steps in social interactions. Second, as demonstrated by Hecker and Dutke (2004), central executive ability plays a key role in social perception, particularly when tasks are cognitively demanding and time-restricted. After dividing participants into high and low WM groups, Hecker and Dutke (2004) presented participants with a social perception task that involved identifying subgroups within a mental clique model. More specifically, participants were presented with relations on a computer screen presented one after another that indicated mutual liking (denoted by a plus sign) or disliking (denoted by a negative sign) between two people and asked to identify the number of subgroups within the model. In order to determine the influence of high versus low WM resources, the task was given under conditions that varied according to the time allotted, WM load, and assistance provided. The results showed that low WM individuals performed worse than high WM individuals when the WM load increased and when speed was a requirement. Low WM individuals also requested more additional information about the relations during the task. Thus, the results indicate that individuals with reduced WM are at a disadvantage during SIP tasks that are complex and time-limited. Finally, WM has been shown to be more efficient for social information than for non-social information and this effect has been linked to "chunking" retrieval processes (Thornton & Conway, 2013). For example, after manipulating sociality and WM load in an fMRI experiment, Thornton and Conway (2013) observed that

social information was less burdensome than non-social information on the frontoparietal network and that this finding was attributable to efficient chunking of social information. These findings indicate that not only is WM vital for social functioning, but it appears to be uniquely equipped to process social information. The current study is the first to empirically test whether WM performance predicts SIP in children with histories of stroke.

Purpose

The overall objectives of the current study were twofold. The *primary* goal was to examine the WM and SIP profiles of children with histories of stroke and to explore the relation between these two constructs. The *secondary* objective was to examine the effects of age at stroke (i.e., perinatal, 1 month-5 years, 6-14 years) and lesion location (i.e., cortical, subcortical, and combined) on WM and social outcomes. These objectives were addressed through two separate studies. Study 1 addressed the primary objective by investigating WM and SIP patterns of children aged 6 to 14 years with and without histories of stroke, utilizing the Baddeley and Hitch (1974) and Crick and Dodge (1994) theoretical models as guides. Study 2 was a retrospective study in which the secondary objective was addressed by examining parent report measures of WM and social skills in a large sample of children age 6 to 14 years with histories of stroke, looking at age of stroke and lesion location. The assessment of WM and SIP patterns in the pediatric stroke population is important for several reasons. First, this population offers a unique opportunity to examine the impact of early focal injury on development, thus increasing our knowledge of brain-behaviour relationships and plasticity of the immature brain. Second, a better understanding of the WM and social outcomes in children with histories of stroke is critical for directing therapeutic interventions and assisting with academic planning. Finally, this

research serves an important role in providing guidance and support to families, teachers, and clinicians.

Study 1: Assessment of Working Memory and Social Information Processing Patterns

Objectives and Hypotheses

The purpose of this study was to investigate WM and SIP patterns in children with histories of stroke and to explore the relationship between these processes. More specifically, it examined a) what components of WM (i.e., phonological loop, visuospatial sketchpad, or central executive) are impaired in children with histories of stroke and whether parent reports indicated WM impairment in everyday life; b) what steps of SIP (i.e., encoding, interpretation, clarification of goals, response construction, response decision, or enactment) are impaired in children with histories of stroke and whether parent reports indicated deficits in everyday life; and c) if WM performance predicts SIP performance. These aims were addressed by comparing the WM and SIP scores of children with histories of stroke to healthy controls using the Working Memory Test Battery for Children (WMTB-C; Pickering & Gathercole, 2001), Social Information Processing Interview (Keil & Price, 2009), Mind in Eyes Task (Baron-Cohen et al., 2001), Wechsler Abbreviated Scale of Intelligence-Second Edition (WASI-II; Wechsler, 2011), Behaviour Rating of Executive Function (BRIEF; Gioia et al., 2000), and Behaviour Assessment System for Children-Second edition (BASC-2; Reynolds & Kamphaus, 2004). Based on prior findings examining WM in children with histories of stroke (Lansing et al., 2004; Westmacott et al., 2010; White, Salorio, Schatz, & DeBaun, 2000), it was hypothesized that children with histories of stroke would demonstrate deficits in the three components of WM outlined by Baddeley and Hitch (1974) relative to controls. Moreover, it was expected that parent reports would indicate that children with histories of stroke display WM deficits in everyday life. Based

on the pediatric stroke literature examining social functioning (Boni et al., 2001; Coelho et al., 2005; Trauner, Panyard-Davis, & Ballantyne, 1996), it was anticipated that children with histories of stroke would demonstrate lower SIP performance relative to controls. Further, it was expected that the parent reports of children with histories of stroke would indicate social deficits in everyday life. Finally, on the basis of findings showing an association between WM and social competence (Anderson & Knight, 2010; Channon & Crawford, 2010; Hecker & Dutke, 2004; Hommel et al., 2009), it was hypothesized that WM performance would predict SIP performance for both children with histories of stroke and controls.

Method

Participants.

Two groups of children (aged 6-14) were included in this study: children with histories of stroke (n = 32) and a typically developing control group (n = 32). Children with histories of stroke were recruited based on their enrollment in the Children's Stroke Outcome Study at The Hospital for Sick Children in Toronto. A telephone recruitment procedure was used to contact children with unilateral AIS seen at The Hospital for Sick Children whose parents had provided consent to be contacted for research purposes. Controls were recruited via local advertisement. Recruitment criteria for the stroke group included the following: (1) a single AIS documented on magnetic resonance imaging (MRI) or computed tomography (CT); (2) AIS before the age of 14 years; (3) at least 6 month post-stroke; and, (4) fluency in English. Exclusion criteria included the following: (1) bilateral lesions; (2) multiple strokes; (3) seizure disorders; (4) preterm birth (less than 36 weeks gestation); (5) hypoxic-ischemic encephalopathy; (6) SCD; (7) psychosis; (8) moya moya disease; and, (9) any other neurological disorders (e.g., brain injury, malignancy, etc.). The criteria did not exclude children with learning disabilities or Attention-Deficit

Hyperactivity Disorder (ADHD), as the literature has shown that attention and academic problems are common in the pediatric stroke population (e.g. Max et al., 2002, 2010). Seven participants from the stroke group declined to take part in the study when they were contacted due to lack of interest.

Procedure.

Testing was completed at The Hospital for Sick Children, York University, and the homes of participants (depending on the convenience for the family). Informed consent was obtained from caregivers and consent/assent was obtained from the child prior to beginning testing (see Appendices A, B, C, and D). Participants completed subtests from the WMTB-C (Pickering & Gathercole, 2001), SIP Interview (Keil & Price, 2009), Mind in Eyes Task (Baron-Cohen et al., 2001), and the WASI (Wechsler, 1999). Parents/caregivers were asked to complete the BRIEF (Gioia et al., 2000), BASC-2 (Reynolds & Kamphaus, 2004) and a demographic questionnaire while their children were engaged in testing. At the end of testing, children and caregivers were provided with compensation, which included a small prize for the child and transportation reimbursement. The child and family were provided with a verbal and written debriefing reviewing the goals of the study (see Appendix E). Parents were provided with written reports describing their children's performance upon the completion of the study.

Measures.

WMTB-C (Pickering & Gathercole, 2001).

The WMTB-C (Pickering & Gathercole, 2001), which consists of a battery of 10 tests standardized for individuals ranging in age from 5 to 15 years, was used to assess the central executive, phonological loop, and visuospatial sketchpad components of WM. Scaled scores (M = 10, SD = 3) are calculated for each subtest. Adequate levels of internal validity have been found for the WMTB-C, as significant correlations ($p < .05$) have been found between the

phonological loop (r range = 0.54-0.72), visuospatial sketchpad (r range = 0.30-0.68), and central executive (r range = 0.27-0.63) measures. Furthermore, factor analyses confirmed that the measures corresponded to the three-factor WM model. High levels of external validity have also been found, as indicated by evidence for a strong association between phonological loop and performance and vocabulary scores in children aged 7 and 8 (r = 0.53 and 0.50, respectively, both $p < .01$). Moreover, strong associations in children aged 7 and 8 were found between central executive performance and vocabulary (r = 0.61 and 0.52, respectively, both $p < .01$), literacy (r = 0.56 and 0.49, respectively, both $p < .01$), and arithmetic (r = 0.56 and 0.55, respectively, both $p < .01$) scores. The following subtests were administered:

1. Backwards Digit Recall – This subtest is a verbal measure of the central executive component of WM. Participants were required to recall the sequence of digits spoken aloud by the examiner in reverse order. Initial trials begin with two digits and each level increases by one digit. Testing is discontinued once the child is unable to recall four trials at a level. Test-retest reliability coefficients range from 0.53 (years 1 and 2) to 0.71 (years 5 and 6).
2. Counting Recall – This subtest is a non-verbal measure of the central executive. Participants were presented with a booklet displaying 1-6 dots in an array and asked to count the number of dots. They were then asked to recall the number of dots presented in the arrays previously displayed. Initial trials begin with one dot array and each level increases by one dot array. Testing is discontinued once the child is unsuccessful at four trials within a level. Test-retest reliability coefficients range from 0.48 (years 5 and 6) to 0.74 (years 1 and 2).

3. Word List Recall – This subtest was used to assess the phonological loop. Participants were asked to recall words in the order that they were presented. The initial level begins with one word and increases until the child is unable to recall four trials of words within a level. The number of words the child is able to recall in their last successful span is taken to indicate their phonological loop capacity. Test-retest reliability coefficients range from 0.64 (years 5 and 6) to 0.80 (years 1 and 2).
4. Block Recall – This subtest measures the functioning of the visuospatial sketchpad. The examiner taps a set of blocks, and the child is asked to tap the blocks in the same sequence. The initial level begins with one block to recall, and continues until the child is unsuccessful at four trials within a level. The number of blocks the child is able to recall in their last successful span is taken to indicate their visuospatial sketchpad functioning. Test-retest reliability coefficients range from 0.43 (years 5 and 6) to 0.63 (years 1 and 2).

Social Information Processing Interview (Keil & Price, 2009).

The SIP Interview (Keil & Price, 2009) was used to assess the six SIP steps outlined by Crick and Dodge (1994). Each child was presented with the same four video vignettes of social situations and asked to imagine that they were the main characters in the vignettes. Sex was equally distributed across the four videos, with two males and two females as the focal characters. The videos depicted two domains of social situations: peer group entry and response to provocation. Each domain was composed of two videos. In the videos depicting peer group entry situations, the main character is shown approaching peers involved in a social activity (e.g., playing with sand). In the videos depicting response to provocation, the main character experiences a negative social outcome (e.g., hit by a ball). The intent of the peers in the videos was either benign/ambiguous or hostile (one video of each intent per domain). Following the

video, each child was asked to complete a standardized interview corresponding to each of the steps in Crick and Dodge's (1994) model. All interview protocols were scored by examiner A.F. Evidence has been found for strong internal consistency for the measure, with alpha coefficients ranging from 0.53 to 0.84 (M α = 0.71) for the provocation domain and from 0.56 to 0.84 (M α = 0.77) for the group entry domain. The interview consisted of the following stages:

1. Encoding of cues: Children's encoding of social cues was assessed by asking, "What happened in the story?" Responses were coded according to the number of details provided and summed to arrive at a total encoding score for a possible maximum of 6 points. Children received 2 points if they describe the actions of the other child toward the main character and 1 point for each of the following: actions of the main character, description of activity, mention of other children, and description of the setting.
2. Interpretation of cues: In order to assess children's interpretive processes of the social outcomes depicted in the video, they were asked why the child acted the way they did. They were asked to choose from the following forced-choice options: (1) being mean, (2) not being mean, and (3) hard to tell. The "being mean" option was taken to reflect a hostile attribution while the "not being mean" option was taken to reflect a non-hostile attribution. The "hard to tell" choice was taken to reflect uncertainty.
3. Clarification of goals: Children's goal selection processes was assessed by asking, "What do you want to happen next?" Children were asked to provide only one goal. Responses were coded according to the following: pro-social (e.g., wanting the other child to apologize, playing together), non-social (e.g., wanting nothing to happen), negative/aggressive (e.g., hitting the other child), and irrelevant (e.g., responses unrelated to the story).

4. Response access or construction: Children's social problem-solving skills were assessed by asking, "What would you say or do if this happened to you?" Children were prompted by the examiner to generate responses until either six responses were obtained or the child was unable to provide any additional responses. Responses were coded according to the following: aggressive (e.g., hit the child), competent (e.g., ask the child why they did what they did), or inept (e.g., cry).
5. Response evaluation: Children's evaluation processes were assessed by presenting them with three video clips depicting aggressive, competent, and inept responses. The children were then asked questions to determine whether they viewed the responses shown as being affiliation-oriented (i.e., whether the other child would like them) and instrumental (i.e., whether the response would be successful in obtaining the desired goal). Evaluation variables were the frequencies of each participant's endorsement of a strategy type (either aggressive, competent, or inept), separately for affiliation and instrumental outcomes.
6. Enactment: Each child was asked to role-play their behavioural response to the situation. Responses were rated on a 4-point scale according to the following criteria:
    - 4 points: Role-play includes relevant content and impeccable facial expression and tone of voice.
    - 3 points: Role includes relevant content and appropriate eye contact and tone of voice. Appropriate eye contact is defined as maintaining eye contact with the examiner during the enactment. Appropriate tone of voice is defined as speaking in a loud, clear voice that would be appropriate for a conversation.

- 2 points: Role-play includes relevant content. Child's does not display either appropriate eye contact or tone of voice.
- 1 point: Child uses inappropriate or irrelevant content, or lacks both eye contact and tone of voice.
- 0 points: Child provides an inappropriate response or does not respond.

Mind in Eyes Task (Baron-Cohen et al., 2001).

This task was used to further assess participants' skills at interpreting facial expressions. Participants were asked to view 28 pictures of male and female faces, showing only the eye area. They were then required to choose one of four words/phrases choices that best captured what the person was thinking or feeling. Support for the validity of this measure is indicated by documentation of a significant correlation ($p < .05$) with the conceptually similar Empathy-Quotient measure ($r = 0.56$; Chapman, Baron-Cohen, & Auyeung, 2006) and evidence suggesting that it is able to differentiate between children with Autism Spectrum Disorder (ASD) and typically developing children (Baron-Cohen et al., 2001). Furthermore, a recent neuroimaging study conducted by Moor and colleagues (2012) demonstrated that the brain regions involved in social perception (i.e., posterior superior temporal sulcus, medial PFC, inferior frontal gyrus, temporal pole) were activated in adolescents and young adults completing the task. Although more studies are needed to evaluate reliability, adequate internal consistency ($\alpha = 0.53$) was recently reported in a sample of 596 typically developing adolescents (Muller & Gmunder, 2014).

Wechsler Abbreviated Scale of Intelligence – Second Edition (Wechsler, 2011).

The WASI-II (Wechsler, 2011), which consists of a battery of four subtests standardized for individuals ranging in age from 6 to 90 years, was used to assess the global intellectual

functioning of participants. This measure is linked with the Wechsler Intelligence Scales for Children – Fourth Edition (WISC-IV; Wechsler, 2003) and Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV; Wechsler, 2008). Due to time constraints, only two subtests of the WASI-II (i.e., Matrix Reasoning and Vocabulary) were administered to estimate general intellectual functioning (i.e., Full Scale IQ-2). Therefore, it was not possible to obtain estimates of the Verbal Comprehension and Perceptual Reasoning Indices. The two subtest version was selected due to the fact that several of the stroke participants presented with motor impairments, thereby excluding them from completing the subtest requiring fine motor skills (i.e., Block Design). *T* scores ($M = 50$, $SD = 10$) are calculated for each subtest and the composite scores (i.e., VCI, PRI, FSIQ-4, FSIQ-2; $M = 100$, $SD = 15$) are calculated based on the sums of the corresponding *T* scores. Adequate test-retest reliability has been found for this measure, with the average reliability coefficients ranging from 0.79 to 0.90 for the subtests and 0.87 to 0.95 for the composite scores in the child sample. Adequate content validity has also been reported, as indicated by moderate to strong and significant ($p < .01$) intercorrelations between subtests (*r* ranges from the 0.40s to the 0.70s). Furthermore, exploratory and confirmatory factor analyses provided support for the structure of the measure. Importantly, moderate to strong and significant ($p < .01$) correlations were found between the WASI-II and the WISC-IV (*r* range = 0.73 to 0.91), thereby providing evidence for strong construct validity. The following provides a description of each subtest:

1. Matrix Reasoning: Participants were asked to complete visual patterns by selecting responses from an array. Reliability coefficients of the child sample for this subtest range from 0.85 to 0.91

2. Vocabulary: Participants were asked to orally define words presented by the examiner. Reliability coefficients of the child sample for this subtest range from 0.86 to 0.94.

Of note, ten of the participants from the stroke group had recently undergone intellectual testing as part of their assessment at The Hospital for Sick Children. In these cases, the test results collected from the previous assessment were used to estimate intellectual functioning in order to avoid potential practice effects. Therefore, the WASI-II (Wechsler, 2011) was administered to all of the participants in the control group and to those children in the stroke group who had not received an intellectual assessment for more than one year.

Demographic Questionnaire.

Parents were asked to complete demographic questionnaires in order to collect information regarding participants' developmental, medical, and family histories (see Appendix F).

Behaviour Assessment System for Children, Second Edition (Reynolds & Kamphaus, 2004).

The BASC-2 was used to assess social functioning in everyday life. The BASC-2 is a standardized parent questionnaire designed to measure behavioural problems in children between the ages of 2-5 (preschool form), 6-11 years (child form), and 12-21 years (adolescent form). Parents are asked to rate current behavioural problems on a 4-point scale: 0 (not true), 1 (sometimes), 2 (often), 3 (almost always). The BASC-2 produces 15 primary scales (activities of daily living, adaptability, aggression, anxiety, attention problems, attitude to teachers, atypicality, conduct problems, depression, functional communication, hyperactivity, leadership, social skills, somatization, and withdrawal), 7 content scales (anger control, bullying, developmental social disorder, emotional self-control, executive functioning, negative

emotionality, and resiliency), and 4 composite scales (adaptive skills, behavioural symptom index, externalizing problems, and internalizing problems). *T* scores (*M*=50, *SD*=10) are provided for each scale. Internal consistency reliabilities range from 0.70 to 0.88. The primary scale of interest for the current study was the Social Skills scale. Items of the Social Skills scale assess social-emotional reciprocity (e.g., "shows interest in others ideas"), adhering to social norms (e.g., "saying 'please' and 'thank you'"), and cooperating with others (e.g., "encouraging others to do their best"). The BASC-2 has satisfactory content, construct, and criterion-related validity. Lower scores are indicative of a higher degree of social impairment.

Behaviour Rating Inventory of Executive Function (Gioia, et al., 2000).

The BRIEF was used to evaluate the behavioural manifestations of executive functioning and WM in everyday life. The BRIEF is a standardized parent questionnaire designed to evaluate executive functions in children between the ages of 5 and 18 years. The BRIEF consists of 86 items. Parents are asked to evaluate behavioural problems on a 3-point scale: 1 (never), 2 (sometimes), and 3 (often). The BRIEF produces 7 clinical scales (Inhibit, Shift, Emotional Control, Monitor, WM, Plan/Organize, and Organization of Materials), 2 validity scales (Inconsistency and Negativity), and 3 composite scales (Behavioural Regulation Index, Meta-cognition Index, and Global Executive Composite). *T* scores (*M*=50, *SD*=10) are provided for each scale. Internal consistency reliabilities range from 0.80 to 0.98. The WM scale was the scale of primary interest for the current study.

Results

Group Differences on Demographic Variables.

Demographic characteristics of participants were determined from the demographic questionnaires completed by parents. Maternal and paternal education was rated on an eight-

point scale (1 = some elementary school; 2= completed elementary school; 3 = some high school; 4 = completed high school; 5 = some college; 6 = completed college; 7 = university degree; 8 = postgraduate degree). Family income was rated on a six-point scale (1 = under $20,000; 2 = $20,001-$30,000; 3 = $30,001-$40,000; 4 = $40,001-$50,000; 5 = $50,001-$80,000; 6 = over $80,000). Neurological characteristics of the stroke participants were determined from a review of health records.

Group differences for sex, ethnicity, maternal education, paternal education, family income, and parental marital status were examined through Chi-Square tests. Prior to analysis, demographic variables were examined for accuracy of data entry and missing values. No missing values were found for sex, ethnicity, age, and parental marital status. A single missing value (1.6% of cases) was found for family income and four missing values (6.2% of cases) were found for both maternal and paternal education. These missing values were the result of incomplete information on the demographic questionnaires. Groups did not differ significantly with respect to sex, X^2 (63, N = 64) = 64.0, p = .44, ethnicity, X^2 (5, N = 64) = 6.28, p = .28, maternal education, X^2 (5, N = 60) = 10.03, p = .07, paternal education, X^2 (6, N = 60) = 8.47, p = .21, family income, X^2 (4, N = 63) = 2.61, p = .63, or parental marital status, X^2 (2, N = 64) = 1.02, p = .60. Group differences for age were examined through one-way between-subjects analysis of variance (ANOVA). The results indicated that the groups did not differ significantly with respect to age, F (1, 62) = 2.24, p = .14. A summary of the demographic and neurological characteristics of participants is presented in Table 1.

Table 1: Demographic and neurologic information for controls and stroke participants

	Controls	Stroke Participants
Total Number	32	32
Males/Females	16/16	21/11
Maternal education[1], mean (SD)	6.84 (1.08)	6.00 (1.44)
Paternal education[1], mean (SD)	6.81 (1.17)	5.89 (1.64)
Family Income[2], mean (SD)	5.47 (1.08)	5.26 (1.57)
Ethnicity		
African origin	2	3
Asian Am./Asian Pacific Islander	6	3
Latino-a/Hispanic	3	0
European origin/White	12	19
Bi-racial/Multi-racial	4	4
Other	5	3
Age at stroke, mean (SD)		1.37y (2.51)
Perinatal		22
1 month-5 years		7
6-14 years		3
Time since stroke, mean (SD)		8.20y (3.91)
Age at test, mean (SD)	8.25y (0.43)	9.16y (0.43)
Lesion location		
Cortical		17
Subcortical		6
Combined		9

[1]Maternal and paternal education is rated on an eight-point scale (see text for details)
[2]Family income is rated on a six-point scale (see text for details)

Group differences for Full Scale IQ, Vocabulary, and Matrix Reasoning scores (as measured by the WASI-II or WISC-IV) were examined through one-way between-subjects ANOVAs. Prior to analysis, Full Scale IQ, Vocabulary, and Matrix Reasoning were examined for accuracy of data entry, missing values, and fit between their distributions and the assumptions of univariate analysis. The variables were examined separately for the 32 participants with history of stroke and 32 controls. A single missing value (3.1% of cases) for Full Scale IQ, Vocabulary, and Matrix Reasoning was found in the stroke group. This missing value resulted after a stroke participant cancelled their scheduled appointment for a

psychological evaluation at The Hospital for Sick Children. With respect to Full Scale IQ, one case in the stroke group was a univariate outlier because of their low score, while two cases in the control group were univariate outliers because of their high scores. It was determined that these outliers were sampled from the target population, and therefore they were retained in the data set. Kolmogorov-Smirnov's test of normality indicated non-normal Full Scale IQ distributions for both the stroke group (p = .04) and the control group (p = .009). Graphical checks indicated that the Full Scale IQ distribution for the stroke group was slightly negatively skewed, while the Full Scale IQ distribution for the control group was slightly positively skewed. Transformations were not applied to normalize the data due to the fact that the distributions were skewed in different directions. Kolmogorov-Smirnov's test of normality for Vocabulary *T* scores indicated a normal distribution for the stroke group (p = .20) and a non-normal distribution for the control group (p = .007). No univariate outliers were found in either distribution. A transformation was not applied to the distribution for controls due to the fact that graphical checks indicated that it was not markedly skewed. Kolmogorov-Smirnov's test of normality indicated normal Matrix Reasoning *T* score distributions for both the stroke group (p = .20) and the control group (p = .20). Further, no univariate outliers for Matrix Reasoning *T* scores were found in either group. Evaluation of the assumption of homogeneity of variance for Full Scale IQ, Vocabulary, and Matrix Reasoning was satisfied. Groups were found to differ significantly on Full Scale IQ [F (1, 61) = 13.47, p = .001, partial eta squared = .181, power = .951], with children in the stroke group having lower scores than children in the control group. These findings are consistent with previous studies (Allman & Scott, 201; Ballantyne et al., 2008; Hartel, et al., 2004; Max et al., 2010; Nass & Trauner, 2004; Westmacott et al., 2009, 2010). It is noteworthy that, despite this group difference, the mean Full Scale IQ scores for both groups fell

within the average range. In order to ascertain the influence of the outliers (noted above), a separate analysis examining group differences in Full Scale IQ was also conducted with the outliers omitted. However, groups continued to differ significantly on Full Scale IQ [F (1, 68) = 9.62, p = .003, partial eta squared = .142, power = .862), despite the omission of the outliers. Groups also differed significantly on Vocabulary [F (1, 61) = 11.4, p = .001, partial eta squared = .157, power = .914], with children in the stroke group have lower scores than children in the control group (albeit, still within the average range). No significant group difference was found for Matrix Reasoning [F (1, 61) = 3.57, p = .06, partial eta squared = .055, power = .461]. Information regarding the IQ scores of participants can be seen in Table 2.

Table 2: IQ scores for controls and stroke participants, mean (SD)

Measure	Controls (n = 32)	Stroke Participants (n = 31)
Full Scale IQ Composite	108.72 (17.37)	92.55 (17.60)
Vocabulary T-score	56.56 (11.00)	46.81 (11.92)
Matrix Reasoning T-score	53.25 (10.96)	47.74 (12.14)

Group Differences on Working Memory.

Working Memory Test Battery for Children (WMTB-C).

A one-way multivariate analysis of variance (MANOVA) was performed on the WMTB-C (Pickering & Gathercole, 2001) variables (Word List Recall, Block Recall, Counting Recall, and Backward Digit Recall). Prior to analysis, the variables were examined for accuracy of data entry, missing values, and the assumptions of multivariate analysis. The variables were examined separately for the 32 participants with stroke and the 32 controls. No missing values were found for Word List Recall and Block Recall. A single missing value (3.1% of cases) was found for Counting Recall in the stroke group. This missing value occurred as a result of this

subtest having to be discontinued for one stroke participant after it became evident that he was unable to comprehend the instructions. Two missing values (6.25% of cases) were found for Backward Digit Recall in the stroke group. One of these missing values occurred after the participant refused to engage in the task and the other occurred as a result of the task needing to be discontinued for one participant once it became clear that he was unable to follow the instructions. One univariate outlier was found in the Word List Recall distribution of the stroke group, three univariate outliers were found in the Block Recall distribution of the control group, and two univariate outliers were found in the Backward Digit Recall distribution of the stroke group. It was determined that these outliers were sampled from the target population and therefore they were retained in the data set. Kolmogorov-Smirnov's test of normality indicated normal distributions for Word List Recall (p = .20 for both stroke group and control group), Block Recall (p = .20 for stroke group and p = .18 for control group), Counting Recall (p = .20 for both stroke group and control group), and Backward Digit Recall (p = .19 for stroke group and .09 for control group). Results of evaluation assumptions of homogeneity of variance-covariance matrices, linearity, and multicollinearity were satisfactory.

With the use of Wilks' criterion, the combined DVs were found to differ significantly between groups, F (4, 56) = 3.61, p = .011, partial eta squared = .205. Power to detect the effect was .845. Univariate analysis was carried out to examine the main effect of group membership using the Bonferroni approach to adjust for multiple comparisons. The results showed significant effects for Word List Recall, [F (1, 59) = 11.83, p = .001, partial eta squared = .167, power = .923], Block Recall, [F (1, 59) = 8.42, p = .005, partial eta squared = .125, power = .814], Counting Recall, [F (1, 59) = 5.48, p = .023, partial eta squared = .085, power = .634], and Backward Digit Recall, [F (1, 59) = 6.00, p = .017, partial eta squared = .092, power = .673],

with the control group performing significantly better than the stroke group in each case. Again, it is noteworthy that despite these group differences, the mean scores for both groups fell within the average range. Table 3 presents the means and standard deviations of the Word List Recall, Block Recall, Counting Recall, and Backward Digit Recall scores for both groups.

Table 3: WMTB-C scores for controls and stroke participants, mean (SD)

Measure	N	Controls	Stroke Participants
Word List Recall	CO = 32; ST = 32	114.13 (16.18)	98.55 (19.18)
Block Recall	CO = 32; ST = 32	107.91 (14.39)	95.66 (18.51)
Counting Recall	CO = 32; ST = 31	101.16 (17.09)	90.31 (19.09)
Backward Digit Recall	CO = 32; ST = 30	101.38 (19.06)	90.55 (14.97)

Note. CO = Controls; ST = Stroke

BRIEF.

A one-way between subjects ANOVA was used to determine whether there was a group difference on the WM subscale of the BRIEF. Prior to analysis, the data were examined for accuracy of data entry, missing values, and fit between their distributions and the assumptions of univariate analysis. The variables were examined separately for the 32 participants with history of stroke and 32 controls. Two missing values (6.2% of cases) were found for the stroke group. These missing values resulted from failure of two families to complete questionnaires. There were no univariate outliers. Kolmogorov-Smirnov's test of normality indicated normal distributions for both the stroke group (p = .117) and control group (p = .200). The assumption of homogeneity of variance was determined to be satisfactory. Groups were found to differ significantly on the WM subscale [F (1, 60) = 18.45, p < .001, partial eta squared = .235, power = .988], with the stroke group being rated as having more difficulties than controls. The stroke group had a mean WM subscale T score of 61.6 (SD = 14.96) and the control group had a mean score of 48.16 (SD = 9.18). Although these results indicated a higher level of WM dysfunction

for stroke participants relative to controls, it is important to note that the mean score of the stroke group falls within the non-clinical range (clinical range on the BRIEF requires a *T* score ≥ 65 and a *T* score of 50 represents average).

Group Differences on Social Information Processing.

SIP Interview.

Prior to analysis, the encoding, attribution, goal, response generation, response evaluation, and enactment variables were examined for accuracy of data entry, missing values, and analysis assumptions. The variables were examined separately for the 32 participants with stroke and the 32 controls. A single missing value (3.1% of cases) was found in the stroke group for each variable. This missing value occurred due to the reluctance of one stroke participant to engage in the task. Given the fact that raw scores are used for this measure (rather that standardized scores), correlational analyses were first carried out to determine the relationships between age, sex, and each variable. When significantly correlated, age and sex were included in subsequent analyses as covariates. One-way ANOVAs were conducted to examine group differences in encoding and enactment. Poisson regression analyses were conducted to examine the main effect of group membership on attribution, goal, response generation, and response evaluation. This procedure was chosen due to the fact that these DVs consist of low frequency counting data (e.g., number of hostile vs. non-hostile attributions, number of pro-social goals generated, etc.). Descriptive statistics for the SIP variables is provided in Table 4.

Table 4: Descriptive statistics for stroke and control groups on SIP variables

Group	Variable	Mean (SD)	Minimum	Maximum
Stroke	Step 1: Encoding	13.71 (2.95)	8	19
	Step 2: Attribution			
	Hostile Attribution	.71 (.82)	0	2

	Non-hostile Attribution	1.35 (.88)	0	3
	Step 3: Goals			
	Pro-social	2.55 (1.56)	0	4
	Negative/Aggressive	.16 (.58)	0	3
	Non-social	.16 (.37)	0	1
	Inept	.13 (.43)	0	2
	Irrelevant	.13 (.34)	0	1
	Step 4: Response Generation			
	Total Responses	6.39 (2.10)	2	11
	Competent	4.10 (2.31)	0	10
	Aggressive	.32 (.83)	0	4
	Inept	2.00 (1.55)	0	5
	Step 5: Response Evaluation			
	Affiliation Competent	2.45 (1.15)	0	4
	Affiliation Aggressive	.26 (.51)	0	2
	Afflilation Inept	.94 (1.03)	0	3
	Instrumental Competent	2.19 (.91)	0	3
	Instrumental Aggressive	.29 (.46)	0	1
	Instrumental Inept	1.26 (.96)	0	3
	Step 6: Enactment	11.35 (.73)	1	16
Control	Step 1: Encoding	15.38 (3.02)	9	20
	Step 2: Attribution			
	Hostile Attribution	1.06 (.67)	0	2
	Non-hostile Attribution	.90 (.89)	0	3
	Step 3: Goals			
	Pro-social	2.88 (1.36)	0	4
	Negative/Aggressive	–	–	–
	Non-social	.09 (.39)	0	2
	Inept	.16 (.57)	0	3
	Irrelevant	.25 (.51)	0	2
	Step 4: Response Generation			
	Total Responses	7.63 (3.21)	1	17
	Competent	4.88 (3.00)	1	14
	Aggressive	.22 (.61)	0	3
	Inept	2.38 (2.09)	0	8
	Step 5: Response Evaluation			
	Affiliation Competent	3.00 (.88)	0	4
	Affiliation Aggressive	.09 (.29)	0	1

Affiliation Inept	1.09 (1.38)	0	4
Instrumental Competent	2.28 (.81)	0	3
Instrumental Aggressive	.06 (.25)	0	1
Instrumental Inept	.81 (.85)	0	3
Step 6: Enactment	12.41 (4.12)	0	16

Step 1: Encoding.

Data screening indicated that there were no univariate outliers. Kolmogorov-Smirnov's test of normality indicated normal distributions for both the stroke group (p = .149) and control group (p = .200). The assumption of homogeneity of variance was deemed satisfactory. Pearson product-moment correlations were first run to determine the relationships between age, sex, and total encoding. Although sex and total encoding were not significantly correlated, r = .052, N = 63, p = .681, there was a significant, positive correlation between age and total encoding, r = .255, N = 63, p = .044. Therefore, age was included as a covariate in the analysis of group differences in total encoding. A one-way between-subjects analysis of covariance (ANCOVA) revealed a significant difference between the stroke and control groups in total encoding, [F (1, 60) = 7.922, p =.007, partial eta squared = .117, power = .791], with the stroke group scoring lower than controls.

Step 2: Attribution.

Poisson regression analyses were conducted to examine the main effect of group membership on frequency of hostile and non-hostile attributions. Spearman's rank-order correlations were first run to determine the relationships between age, sex, and attributions. Of note, this procedure was selected rather than Pearson's product-moment correlation due to the fact that statistical and graphical checks of the data revealed that the distributions were markedly skewed (thus, violating the assumptions needed for Pearson's correlation). Age and hostile attribution were found to have a significant, positive correlation, r_s = .278, N = 63, p = .028,

whereas sex and hostile attribution was not significantly correlated, r_s = .081, N = 63, p = .530. Conversely, the opposite pattern of results was found for non-hostile attributions. That is, non-hostile attribution was significantly correlated with sex, r_s = -.259, N = 63, p = .040, but not age, r_s = -.106, N = 63, p = .407. Therefore, age was used as a covariate in the hostile attribution analysis and sex was used as a covariate in the non-hostile attribution analysis. Goodness of fit tests indicated that both the hostile attribution model and the non-hostile attribution model were a good fit (deviance value/df = .844 and .961, respectively). Graphical checks confirmed that the standardized deviance residuals approximated standard normal distributions. Results showed that there was no significant effect for group membership for hostile attribution (p = .083) or non-hostile attribution (p = .170). Tables 5 and 6 show the results of the Poisson regression analyses for hostile attribution and non-hostile attribution, respectively.

Table 5: Poisson regression analysis for hostile attribution

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	-.583	.497	-1.558	.392	1.374	.241
Age	.076	.054	-.03	.183	1.983	.159
Group	-.487	.281	-1.038	.064	2.999	.083

Table 6: Poisson regression analysis for non-hostile attribution

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	.463	.410	-.341	1.267	1.272	.259
Sex	-.386	.260	-.897	.124	2.202	.138
Group	.337	.245	-.144	.817	1.887	.170

Step 3: Goals.

Poisson regression analyses were conducted to examine the main effect of group membership on frequency of generation of pro-social goals, negative/aggressive goals, non-social goals, inept goals, and irrelevant goals. Spearman's rank-order correlations were first run to determine the relationships between age, sex, and attributions. Again, this procedure was selected rather than Pearson's product-moment correlation due to the fact that statistical and graphical checks of the data revealed that the distributions were markedly skewed (thus, violating the assumptions needed for Pearson's correlation). Age and sex were not significantly correlated with pro-social goals (r_s = .170, N = 63, p = .182, r_s = .153, N = 63, p = .230, respectively), negative/aggressive goals (r_s = .239, N = 63, p = .060, r_s = -.187, N = 63, p = .141, respectively), non-social goals (r_s = .203, N = 63, p = .110, r_s = .018, N = 63, p = .889, respectively), inept goals (r_s = -.056, N = 63, p = .663, r_s = -.059, N = 63, p = .665, respectively), or irrelevant goals (r_s = -.144, N = 63, p = .259, r_s = -.051, N = 63, p = .691, respectively). Thus, neither age nor sex were used as covariates. Goodness of fit tests indicated mild overdispersion for the pro-social goal model (deviance value/df = 1.188) and mild underdispersion for the non-social goal model (deviance value/df = .577), inept goal model (deviance value/df = .726), and irrelevant goal model (deviance value/df = .678). It was not possible to assess the negative/aggressive goal model due to the fact that very few participants generated this type of goal. Results showed that there were no significant effects for group membership for pro-social goals (p = .432), non-social goals (p = .458), inept goals (p = .775), or irrelevant goals (p = .280). Tables 7, 8, 9, and 10 show the results of the Poisson regression analyses for pro-social goals, non-social goals, inept goals, and irrelevant goals, respectively.

Table 7: Poisson regression analysis for pro-social goals

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	1.056	.104	.852	1.260	102.603	.000
Group	-.121	.153	-.421	.180	.618	.432

Table 8: Poisson regression analysis for non-social goals

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	-2.367	.577	-3.499	-1.236	16.810	.000
Group	.543	.730	-.889	1.974	.552	.456

Table 9: Poisson regression analysis for inept goals

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	-1.856	.447	-2.733	-.980	17.229	.000
Group	-.191	.671	-1.506	1.123	.081	.775

Table 10: Poisson regression analysis for irrelevant goals

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	-1.386	.354	-2.079	-.693	15.374	.000
Group	-.661	.612	-1.862	.539	1.167	.280

Step 4: Response Generation.

Poisson regression analyses were conducted to examine the main effect of group membership on number of total responses, competent responses, aggressive responses, and inept

responses. Spearman's rank-order correlations were first run to determine the relationships between age, sex, and attributions. Again, this procedure was selected rather than Pearson's product-moment correlation due to the fact that statistical and graphical checks of the data revealed that the distributions were markedly skewed (thus, violating the assumptions needed for Pearson's correlation). Age and sex were not significantly correlated with total number of competent responses (r_s = .228, N = 63, p = .073, r_s = .243, N = 63, p = .055, respectively), aggressive responses (r_s = .234, N = 63, p = .065, r_s = -.047, N = 63, p = .714, respectively), or inept responses (r_s = -.097, N = 63, p = .450, r_s = .095, N = 63, p = .459, respectively). Although sex was correlated with total number of responses (r_s = .300, N = 63, p = .017), age was not (r_s = .158, N = 63, p = .216, respectively). The mean total number of responses generated by males was 6.24 ± 2.05 and 8.12 ± 3.30 for females. Thus, sex was used as a covariate in only the total number of responses analysis. Age was not used as a covariate in any of the analyses. Goodness of fit tests indicated that the total number of responses model was an excellent fit (deviance value/df = .981), while mild overdispersion was found for the aggressive response model (deviance value/df = 1.06). A large amount of overdispersion was found for the competent response (deviance value/df = 1.61) and inept response (deviance value/df = 1.82) models, indicating that these models were not a good fit. This issue was addressed by using the negative binomial variant of Poisson regression to analyze the data. Goodness of fit tests using negative binomial regression indicated that the inept response (deviance value/df = .772) model was a good fit; however, moderate underdispersion was found in the competent response model (deviance value/df = .353). Hence, caution is warranted in interpreting the results of the competent response model. Results showed that there were no significant effects for group membership for total number of responses (p = .168), competent response (p = .533), aggressive

responses (p = .431), or inept responses (p = .573). A significant effect for the sex covariate was found on total number of responses (p = .014). Tables 11, 12, 13, and 14 show the results of the Poisson/ negative binomial regression analyses for total number of responses, competent responses, aggressive responses, and inept responses, respectively.

Table 11: Poisson regression analysis for total number of responses

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	1.668	.164	1.346	1.989	103.39	.000
Sex	.238	.097	.048	.428	6.030	.014
Group	-.134	.097	-.325	.056	1.905	.168

Table 12: Negative binomial regression analysis for competent responses

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	1.584	.194	1.204	1.964	66.63	.000
Group	-.174	.279	-.721	.373	.389	.533

Table 13: Poisson regression analysis for aggressive responses

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	-1.520	.378	-2.261	-.779	16.169	.000
Group	.388	.493	-.577	1.354	.621	.431

Table 14: Negative binomial regression analysis for inept responses

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	.865	.211	.452	1.278	16.849	.000
Group	-.172	.305	-.769	.425	.318	.573

Step 5: Response Evaluation.

Poisson regression analyses were conducted to examine the main effect of group membership on affiliation and instrumental evaluation variables (i.e., competent, aggressive, inept). Spearman's rank-order correlations were first run to determine the relationships between age, sex, and attributions. Again, this procedure was selected rather than Pearson's product-moment correlation due to the fact that statistical and graphical checks of the data revealed that the distributions were markedly skewed (thus, violating the assumptions needed for Pearson's correlation). Age and sex were not significantly correlated with affiliation aggressive (r_s = .110, N = 63, p = .390, r_s = -.103, N = 63, p = .420, respectively), affiliation inept (r_s = .176, N = 63, p = .168, r_s = .221, N = 63, p = .082, respectively), instrumental competent (r_s = .048, N = 63, p = .706, r_s = .195, N = 63, p = .126, respectively), instrumental aggressive (r_s = .207, N = 63, p = .103, r_s = -.046, N = 63, p = .721, respectively), or instrumental inept (r_s = .171, N = 63, p = .180, r_s = -.011, N = 63, p = .930, respectively) variables. Although sex was correlated with the affiliation competent variable (r_s = .280, N = 63, p = .024), age was not (r_s = .124, N = 63, p = .335). The mean number of affiliation competent responses endorsed by males was 2.49 (*SD* = .18) and 3.08 (*SD* = .17) for females. Thus, sex was used as a covariate in only the affiliation competent analysis. Age was not used as a covariate in any of the analyses. Goodness of fit tests indicated mild underdispersion for the affiliation competent (deviance value/df = .518) and

affiliation aggressive (deviance value/df = .634) models. A large amount of overdispersion was found for affiliation inept model (deviance value/df = 1.58) indicating that this models was a poor fit. This issue was addressed by using the negative binomial variant of Poisson regression to analyze the data. The goodness of fit test using negative binomial regression indicated that the affiliation inept response model was a good fit (deviance value/df = .910). Mild underdispersion was found for the instrumental competent (deviance value/df = .426) and instrumental aggressive (deviance value/df = .547) models. The instrumental inept model was found to be a good fit (deviance value/df = .986). Results showed that there were no significant effects for group membership for affiliation competent (p = .279), affiliation aggressive (p = .135), affiliation inept (p = .660), instrumental competent (p = .816), or instrumental inept (p = .084) variables. A significant effect for group membership was found for the instrumental aggressive variable (p = .049), with more stroke participants than controls rating aggressive responses as being effective in attaining a desired outcome. Tables 15, 16, 17, 18, 19, and 20 show the results of the Poisson/ negative binomial regression analyses for the affiliation competent, affiliation aggressive, affiliation inept, instrumental competent, instrument aggressive, instrumental inept response evaluation variables, respectively.

Table 15: Poisson regression analysis for affiliation competent response evaluation

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	.821	.261	.309	1.333	9.894	.002
Sex	.182	.155	-.122	.487	1.376	.241
Group	-.169	.156	-.475	.137	1.173	.279

Table 16: Poisson regression analysis for affiliation aggressive response evaluation

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	-2.367	.577	-3.499	-1.236	16.810	.000
Group	1.013	.677	-.314	2.339	2.237	.135

Table 17: Negative binomial regression analysis for affiliation inept response evaluation

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*P*
(Intercept)	.090	.245	-.390	.569	.134	.714
Group	-.156	.356	-.854	.541	.541	.660

Table 18: Poisson regression analysis for instrumental competent response evaluation

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	.825	.117	.595	1.054	49.652	.000
Group	-.039	.169	-.370	.291	.054	.816

Table 19: Poisson regression analysis for instrumental aggressive response evaluation

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	-2.773	.707	-4.158	-1.387	15.374	.000
Group	1.536	.782	.004	3.068	3.860	.049

Table 20: Poisson regression analysis for instrumental inept response evaluation

			95% Wald Confidence Interval		Hypothesis Test	
Parameter	B	Std. Error	Lower	Upper	Wald Chi-Square	*p*
(Intercept)	-.208	.196	-.592	.177	1.121	.000
Group	.437	.253	-.059	.933	2.982	.084

Step 6: Enactment.

Initial data screening revealed two univariate outliers in the stroke group and one univariate outlier in the control group. These outliers were retained in the data set because it was determined that they were sampled from the target population. Kolmogorov-Smirnov's test of normality indicated non-normal distributions for both the stroke group (p = .016) and control group (p = .004). Graphical checks indicated that both distributions were slightly negatively skewed. The encoding variable was reflected and a square root transformation was applied. Following the transformation, Kolgomorov-Smirnov's test of normality revealed that the distribution for controls was normal (p = .119) and the distribution for the stroke group had approached normality (p = .026). Graphical checks confirmed that the transformation had improved the shapes of the distributions for both groups. The assumption of homogeneity of variance was deemed satisfactory. Given the fact that raw scores are used for this measure (rather that standardized scores), a Pearson product-moment correlation was first run to determine the relationships between age, sex, and total enactment. Although age and total enactment were not significantly correlated, r = -.225, N = 63, p = .076, sex and total enactment were significantly correlated, r = -.318, N = 63, p = .011. The mean total enactment score (prior to transformation) was 10.95 (SD = 4.15) for boys and 13.23 (SD = 6.66) for girls (measured on a 16-point scale). Therefore, sex was included as a covariate in the analysis of group differences in total encoding.

A one-way between-subjects ANCOVA revealed that the groups did not differ significantly in total enactment, [F (1, 60) = .724, p =.398, partial eta squared = .012, power = .133].

Mind in Eyes.

Prior to analysis, the Mind in Eyes variable was examined for accuracy of data entry, missing values, and the assumptions of univariate analysis. The variable was examined separately for the 32 participants with stroke and the 32 controls. There were no missing values. Two univariate outliers were found in the stroke group. These outliers were determined to be sampled from the target population and were therefore retained in the data set. Kolmogorov-Smirnov's test of normality indicated normal distributions for both the stroke group (p = .051) and control group (p = .200). Given the fact that raw scores are used for this measure (rather that standardized scores), a Pearson product-moment correlation was first run to determine the relationships between age, sex, and Mind in Eyes score. A significant, positive correlation was found between age and Mind in Eyes score, r = .380, N = 64, p = .002. Similarly, Mind in Eyes score and sex were significantly correlated, r = .309, N = 64, p = .013. The mean total Mind in Eyes score was 15.08 (SD = 3.81) for boys and 17.59 (SD = 3.96) for girls (measured on a 28-point scale). Therefore, sex and age were included as covariates in the analysis of group differences in Mind in Eyes score. A one-way between-subjects ANCOVA indicated a significant group difference in Mind in Eyes score, [F (1, 60) = 5.023, p =.029, partial eta squared = .077, power = .597], with the control group scoring higher (M = 17.00, SD = .73) than the stroke group (M = 15.28, SD = .68).

BASC-2.

A one-way between subjects ANOVA was used to determine whether there was a group difference on the Social Skills subscale of the BASC-2 (Reynolds & Kamphaus, 2004). Prior to

analysis, the data were examined for accuracy of data entry, missing values, and fit between their distributions and the assumptions of univariate analysis. The variables were examined separately for the 32 participants with history of stroke and 32 controls. A single missing values (3.1% of cases) was found for the stroke group. This missing value resulted from failure of one family to complete the questionnaire. There were no univariate outliers. Kolmogorov-Smirnov's test of normality indicated normal distributions for both the stroke group (p = .200) and control group (p = .052). The assumption of homogeneity of variance was determined to be satisfactory. Groups were not found to differ significantly on the Social Skills subscale [F (1, 61) = .626, p =.432, partial eta squared = .010, power = .122]. The mean Social Skills subscale T score was 46.74 (SD = 12.32) for the stroke group and 49.06 (SD = 10.94) for controls (hence, both within the average range).

Relationships between Working Memory and Social Information Processing Variables.

Correlation analyses were conducted to examine the relationships between the SIP variables (i.e., SIP Interview variables, Mind in Eyes score, BASC-2 Social Skills subscale) and the WM variables (i.e., WMTB-C variables and BRIEF WM subscale). Pearson's product-moment correlations were used to examine the relationships between the WM variables and SIP encoding, SIP enactment, Mind in Eyes, and BASC-2 Social Skills subscale. Spearman's rank-order correlations were used to examine the relationships between the WM variables and SIP attribution, goal, response generation, and evaluation variables. Again, this procedure was selected rather than Pearson's product-moment correlation due to the fact that statistical and graphical checks of the data revealed that the distributions were markedly skewed (thus, violating the assumptions needed for Pearson's correlation). Correlation analyses were run

separately for the stroke group and the control group in order to allow for comparisons between groups. For the stroke group, pro-social goal, affiliation competent response evaluation, and enactment scores were positively and significantly correlated with Block Recall and Backward Digit Recall scores. A significant and positive correlation was also found between encoding and Backward Digit Recall for the stroke group. The BRIEF WM scale was negatively and significantly correlated with the BASC-2 Social Skills scale for both groups, indicating that children who were rated more favourably by parents on social skills tended to be rated by parents as having less WM dysfunction. Finally, a significant positive correlation was found between instrumental competent response evaluation and the BRIEF WM scale for the control group. Table 21 summarizes the correlations for both groups.

Table 21: Correlations between WM and SIP variables for stroke and control groups

Group	SIP Variables	Word List Recall	Block Recall	Counting Recall	Backward Digit Recall	BRIEF WM scale
Stroke	Encoding	.168	.345	.133	.370*	-.053
	Non-hostile attribution	-.192	-.148	-.067	-.070	.148
	Hostile attribution	-.038	-.007	-.058	-.189	.054
	Pro-social goal	.202	.636**	.192	.447*	-.141
	Total responses	.172	.269	.030	.222	-.136
	Competent responses	.166	.171	.105	.037	-.158
	Affiliation competent	.222	.537**	.195	.393*	.085
	Instrumental competent	.127	.243	.071	.078	.068
	Enactment	.201	.416*	.425*	.585*	-.191
	Mind in Eyes	.224	.198	.244	.053	.004
	BASC-2 Social Skills	-.052	.139	.206	.034	-.386*
Controls	Encoding	-.085	-.113	.215	-.091	.219
	Non-hostile attribution	.093	-.096	-.157	-.126	.098
	Hostile attribution	-.02	.113	.155	-.004	-.209
	Pro-social goal	.141	-.033	.148	-.059	.149
	Total Responses	-.047	-.121	.219	-.094	.236
	Competent response	-.208	.041	.146	.016	.119
	Affiliation competent	-.035	-.145	.117	.237	.124
	Instrumental competent	-.173	.297	.138	-.146	.370*

Enactment	.113	.004	.274	.046	.149
Mind in Eyes	.261	.065	.189	.176	.094
BASC-2 Social Skills	-.178	.125	-.16	.215	-.522**

**correlation is significant at the 0.01 level (2-tailed)
*correlation is significant at the 0.05 level (2-tailed)

A series of regression analyses were carried out to predict the SIP variables from the WM variables. Regression analyses were run separately for the stroke group and the control group in order to allow for comparisons between the models. Standard multiple regression analyses were carried out for the SIP encoding, SIP enactment, Mind in Eyes, and Social Skills subscale analyses and Poisson regression analyses were carried out for the SIP attribution, goal, response generation, and evaluation analyses. Age and sex were included in the models if prior correlational analyses (described previously) indicated significant correlations. Results of evaluation of linearity and homoscedasity were satisfactory. Tests for multicollinearity indicated that a very low level of multicollinearity was present (*VIF* = 1.65 for Word List Recall, 1.69 for Block Recall, 1.58 for Counting Recall, 1.76 for Backward Digit Recall, and 1.16 for BRIEF WM scale).

SIP Interview.

Step 1: Encoding.

Separate standard multiple regressions were performed for the stroke group and control group with total encoding score as the dependent variable (DV) and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, BRIEF WM subscale, and age as the independent variables (IVs). Age was included in the model due to the significant, positive correlation between age and total encoding score (reported above). These variables did not significantly predict total encoding score for the stroke group, F (6, 19) = 1.670, p = .183, R^2 =

.345, or the control group, F (6, 25) = 1.126, p = .376, R^2 = .213. Table 22 displays the results of the regression analyses for each group.

Table 22: Multiple regressions of WM on encoding for stroke and control groups

Group	Variables	B	SE B	β	T	*P*	
Stroke	Word List Recall	-.001	.040	-.009	-.035	.973	
	Block Recall	.007	.063	.447	1.219	.238	
	Counting Recall	-.047	.044	-.303	-1.054	.305	
	Backward Digit Recall	.053	.067	.271	.789	.440	
	BRIEF WM scale	-.046	.047	-.237	-.993	.333	
	Age	.480	.262	.426	1.83	.083	R^2 = .345
Control	Word List Recall	-0.02	0.039	-0.106	-0.51	.615	
	Block Recall	-0.046	0.041	-0.218	-1.13	.269	
	Counting Recall	0.04	0.036	0.224	1.097	.283	
	Backward Digit Recall	-0.012	0.034	-0.074	-0.341	.736	
	BRIEF WM scale	0.019	0.067	0.059	0.289	.775	
	Age	0.47	0.295	0.321	1.591	.124	R^2 =.213

Step 2: Attribution.

A series of separate Poisson regression analyses for the stroke group and control group were conducted with attribution (i.e., hostile and non-hostile) as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, and BRIEF WM Subscale as the IVs. Age was also included as an IV in the analysis for hostile attribution, given its positive, significant correlation with this variable (previously noted). Similarly, sex was included as an IV in the analysis for non-hostile attribution, given its correlation with this variable (previously noted). Goodness of fit tests indicated mild overdispersion for the non-hostile attributions models for the stroke group and control group (deviance value/df = 1.057 and 1.130). Mild overdispersion was found for the hostile attribution model for the stroke group (deviance value/df = 1.391), but not the control group (deviance value/df= .639). The non-hostile attribution model was not significant for the stroke group (p = .893) or control group (p = .398).

Similarly, the hostile attribution model was not significant for the stroke group (p = .750) or control group (p = .913). Tables 23 and 24 show the results of the Poisson regression analyses for non-hostile attribution and hostile attribution, respectively.

Table 23: Poisson regressions of WM on non-hostile attribution for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	-.490	1.79	-3.99	3.019	.075	.784
	Word List Recall	-.009	.013	-.034	0.017	.462	.496
	Block Recall	-.010	.022	-.052	0.033	.194	.660
	Counting Recall	.010	.015	-.029	0.031	.003	.954
	Backward Digit Recall	.016	.022	-.028	0.06	.530	.467
	BRIEF WM scale	.009	.014	-.019	0.037	.436	.509
	Sex	0.404	.479	-.535	1.344	.712	.399
Control	Intercept	-1.044	2.299	-5.549	3.461	.206	.65
	Word List Recall	.020	.015	-.008	.049	1.93	.165
	Block Recall	-.009	.017	-.043	.024	.310	.578
	Counting Recall	-.008	.014	-.035	.019	.321	.571
	Backward Digit Recall	-.010	.013	-.035	.015	.617	.432
	BRIEF WM scale	.020	.023	-.026	.065	.707	0.40
	Sex	.827	.423	-.002	1.656	3.826	.050

Table 24: Poisson regressions of WM on hostile attribution for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	-1.308	2.773	-6.743	4.128	.222	.637
	Word List Recall	.004	.016	-.029	.037	.053	.818
	Block Recall	.007	.029	-.049	.064	.065	.799
	Counting Recall	.011	.022	-.032	.053	.240	.624
	Backward Digit Recall	-.025	.030	-.084	.034	.710	.400
	BRIEF WM scale	-.010	.019	-.049	.028	.280	.597
	Age	.179	.109	-.035	.392	2.678	.102
Control	Intercept	.343	1.976	-3.531	4.216	.030	.862

	Word List Recall	-.001	.013	-.026	.023	.013	.911
	Block Recall	.003	.014	-.023	.030	.051	.821
	Counting Recall	.005	.012	-.017	.028	.220	.639
	Backward Digit Recall	-.003	.011	-.025	.018	.096	.756
	BRIEF WM scale	-.029	.024	-.076	.018	1.439	.230
	Age	.087	.096	-.101	.274	.820	.365

Step 3: Goals.

Separate Poisson regression analyses for the stroke group and control group were conducted with pro-social goal as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, and BRIEF WM Subscale as the IVs. Given that pro-social problem-solving was the primary focus of analysis, regressions were not carried out for the other goal variables (i.e., negative/aggressive, inept, irrelevant, non-social) in order to reduce the number of analyses. Goodness of fit tests indicated mild overdispersion for the stroke group and control group (deviance value/df = 1.134 and 1.071). The model was not significant for the stroke group (p = .084) or control group (p = .922). However, a significant main effect was found for the Block Recall variable for the stroke group (p = .020). Table 25 displays the results of the Poisson regression analyses for pro-social goal.

Table 25: Poisson regressions of WM on pro-social goal for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	.663	1.142	-1.576	2.902	.337	.562
	Word List Recall	-.008	.008	-.024	.008	1.046	.306
	Block Recall	.032	.014	.005	.059	5.441	.020
	Counting Recall	-.010	.009	-.028	.008	1.221	.269
	Backward Digit Recall	-.003	.014	-.031	.025	.047	.829
	BRIEF WM scale	-.013	.009	-.032	.005	2.006	.157
Control	Intercept	.487	1.247	-1.957	2.930	.152	.696
	Word List Recall	.005	.008	-.010	.020	.388	.534

Block Recall	-.002	.008	-.018	.014	.042	.839
Counting Recall	.003	.007	-.011	.017	.160	.689
Backward Digit Recall	-.004	.007	-.017	.009	.344	.558
BRIEF WM scale	.006	.012	-.018	.030	.266	.606

Step 4: Response Generation.

Separate Poisson regression analyses for the stroke group and control group were conducted with total responses as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, BRIEF WM Subscale, and sex as the IVs. Sex was included in the model due to the fact that it was found to be significantly correlated with total responses (previously noted). Goodness of fit tests indicated that the model was a good fit for the stroke group (deviance value/df = .872). Mild overdispersion was found for the control group (deviance value/df = 1.064). The model was significant for the control group (p = .013), but not the stroke group (p = .768). Closer inspection of the results revealed that sex appeared to be driving the model effect for the control group, as it was the only variable with a significant effect (p = .009). Separate Poisson regression analyses for the stroke group and control group were also conducted with competent response as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, and BRIEF WM Subscale as the IVs. Given that pro-social problem-solving was the primary focus of analysis, regressions were not carried out for the other response variables (i.e., aggressive and inept) in order to reduce the number of analyses. Goodness of fit tests indicated overdispersion for the stroke group and control group (deviance value/df = 1.474 and 1.675, respectively). Although a negative binomial regression analysis was carried out in an attempt to correct this issue, goodness of fit tests then revealed notable underdispersion. Thus, caution is warranted in the interpretation of the results. The model was not significant for the stroke group (p = .529) or control group (p = .067). However, significant main effects were

found for the Word List Recall (p = .042), Block Recall (p = .034), and Counting Recall (p = .013) variables for the control group. Tables 26 and 27 display the results of the Poisson regression analyses for total responses and competent response, respectively.

Table 26: Poisson regressions of WM on total responses for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	2.049	.766	.548	3.550	7.162	.007
	Word List Recall	.000	.006	-.011	.010	.003	.954
	Block Recall	.005	.009	-.012	.023	.362	.548
	Counting Recall	-.006	.006	-.018	.006	1.021	.312
	Backward Digit Recall	.004	.009	-.014	.023	.233	.630
	BRIEF WM scale	-.008	.006	-.021	.005	1.575	.209
	Sex	-.085	.202	-.481	.312	.175	.675
Control	Intercept	2.398	.793	.843	3.952	9.139	.003
	Word List Recall	-.006	.005	-.015	.004	1.369	.242
	Block Recall	-.008	.005	-.018	.001	2.772	.096
	Counting Recall	.006	.005	-.002	.015	2.044	.153
	Backward Digit Recall	.003	.004	-.005	.011	.562	.453
	BRIEF WM scale	.008	.007	-.007	.022	1.109	.292
	Sex	-.369	.141	-.646	-.092	6.815	.009

Table 27: Poisson regressions of WM on competent response for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	2.019	.887	.279	3.758	5.175	.023
	Word List Recall	.002	.006	-.010	.015	.148	.701
	Block Recall	.006	.011	-.016	.027	.270	.603
	Counting Recall	-.008	.007	-.021	.006	1.256	.262
	Backward Digit Recall	.001	.011	-.021	.023	.010	.921
	BRIEF WM scale	-.012	.007	-.027	.002	2.850	.091
Control	Intercept	2.305	.976	.392	4.218	5.575	.018
	Word List Recall	-.012	.006	-.024	.000	4.148	.042

Block Recall	-.013	.006	-.025	-.001	4.487	.034
Counting Recall	.015	.006	.003	.027	6.108	.013
Backward Digit Recall	.005	.005	-.004	.015	1.284	.257
BRIEF WM scale	.006	.009	-.019	.019	.000	.995

Step 5: Response Evaluation.

Separate Poisson regression analyses for the stroke group and control group were conducted with attribution as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, and BRIEF WM Subscale as the IVs. Again, given that pro-social problem-solving was the primary focus, only those responses reflecting pro-social evaluations were considered for analysis. Hence, only the affiliation competent and instrumental competent variables were selected for analysis. Sex was also included as an IV in the affiliation competent model due to its significant correlation with this variable (reported previously). Goodness of fit tests indicated that the affiliation competent model was a good fit for the stroke group (deviance value/df = .756). Mild underdispersion was found for the control group (deviance value/df = .319). Mild underdispersion was found for the instrumental competent model for both the stroke group and control group (deviance value/df = .523 and .394, respectively). The affiliation competent model was not significant for the control group (p = .748) or the stroke group (p = .762). Similarly, the instrumental competent model was not significant for the control group (p = .930) or the stroke group (p = .913). Tables 28 and 29 display the results of the Poisson regression analyses for affiliation competent and instrumental competent, respectively.

Table 28: Poisson regressions of WM on affiliation competent response evaluation for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	-.539	1.286	-3.060	1.982	.176	.675
	Word List Recall	-.006	.009	-.023	.011	.490	.484
	Block Recall	.011	.015	-.017	.040	.592	.442
	Counting Recall	.008	.011	-.014	.030	.518	.472
	Backward Digit Recall	-.002	.016	-.032	.028	.015	.901
	BRIEF WM scale	.006	.011	-.015	.027	.350	.554
	Sex	-.031	.322	-.662	.600	.009	.923
Control	Intercept	.698	1.195	-1.645	3.041	.341	.559
	Word List Recall	-.004	.008	-.019	.010	.360	.548
	Block Recall	-.001	.008	-.016	.014	.006	.937
	Counting Recall	.002	.007	-.011	.016	.133	.716
	Backward Digit Recall	.006	.006	-.006	.019	.979	.322
	BRIEF WM scale	.005	.012	-.019	.029	.173	.677
	Sex	-.319	.220	-.750	.113	2.095	.148

Table 29: Poisson regressions of WM on instrumental competent response evaluation for stroke and control groups

				95% Wald Confidence Interval		Hypothesis Test Wald Chi-Square	
Group	Parameter	B	Std. Error	Lower	Upper		*p*
Stroke	Intercept	.354	1.320	-2.233	2.940	.072	.789
	Word List Recall	-.005	.009	-.023	.012	.356	.551
	Block Recall	-.001	.015	-.031	.029	.009	.925
	Counting Recall	.012	.012	-.011	.035	1.002	.317
	Backward Digit Recall	-.003	.016	-.036	.029	.041	.840
	BRIEF WM scale	.005	.011	-.016	.027	.244	.621
Control	Intercept	-.188	1.340	-2.814	2.437	.020	.888
	Word List Recall	-.003	.009	-.019	.014	.095	.757
	Block Recall	.004	.009	-.014	.021	.160	.689
	Counting Recall	.002	.008	-.014	.017	.047	.828
	Backward Digit Recall	.002	.007	-.013	.016	.048	.826

BRIEF WM scale	.012	.014	-.015	.039	.742	.389

Step 6: Enactment.

Separate standard multiple regressions were performed for the stroke group and control group with total enactment score as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, BRIEF WM subscale, and sex as the IVs. Sex was included in the model due to the significant correlation between sex and total enactment score (previously reported). These variables did not significantly predict total enactment score for the stroke group, F (6, 19) = 1.545, p = .217, R^2 = .328, or the control group, F (6, 25) = 1.138, p = .370, R^2 = .214. Table 30 displays the results of the regression analyses for each group.

Table 30: Multiple regressions of WM on enactment for stroke and control groups

Group	Variables	B	SE B	β	T	*p*	
Stroke	Word List Recall	-.021	.052	-.104	-.398	.695	
	Block Recall	.023	.086	.102	.267	.792	
	Counting Recall	.003	.060	.014	.048	.962	
	Backward Digit Recall	.124	.089	.485	1.402	.177	
	BRIEF WM scale	-.045	.061	-.173	-.727	.476	
	Sex	1.330	1.912	.157	.696	.495	R^2 = .328
Control	Word List Recall	-.010	.053	-.038	-.183	.856	
	Block Recall	.006	.057	.022	.108	.915	
	Counting Recall	.056	.048	.231	1.150	.261	
	Backward Digit Recall	.005	.046	.024	.114	.911	
	BRIEF WM scale	.043	.087	.096	.49	.622	
	Sex	3.002	1.531	.370	1.961	.061	R^2 =.214

Mind in Eyes.

Separate standard multiple regressions were performed for the stroke group and control group with Mind in Eyes score as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, BRIEF WM subscale, age, and sex as the IVs. Sex and age were

included in the model due to their significant correlations with Mind in Eyes score (previously reported). These variables significantly predicted Mind in Eyes score for the control group, F (7, 24) = 3.320, p = .013, R^2 = .492, but not the stroke group, F (7, 19) = 1.874, p = .131, R^2 = .408. Closer examination of the results revealed that sex and age were driving the model effect for the control group (p = .045 and .004, respectively). Although the overall model was not significant for the stroke group, Word List Recall and age were found to be significant predictors (p = .044 and .014, respectively). Table 31 displays the results of the regression analyses for each group.

Table 31: Multiple regressions of WM on Mind in Eyes score for stroke and control groups

Group	Variables	B	SE B	β	T	*p*	
Stroke	Word List Recall	.106	.049	.565	2.157	.044	
	Block Recall	.023	.057	.118	.407	.689	
	Counting Recall	.048	.054	.253	.899	.380	
	Backward Digit Recall	-.093	.065	-.380	-1.431	.169	
	BRIEF WM scale	-.058	.058	-.235	-.993	.333	
	Sex	2.927	1.641	.378	1.784	.090	
	Age	.825	.306	.594	2.698	.014	R^2 = .408
Control	Word List Recall	.053	.045	.208	1.194	.244	
	Block Recall	.011	.048	.039	.234	.817	
	Counting Recall	-.013	.041	-.056	-.332	.743	
	Backward Digit Recall	-.002	.039	-.007	-.042	.967	
	BRIEF WM scale	-.033	.076	-.073	-.436	.667	
	Sex	2.735	1.290	.336	2.120	.045	
	Age	1.089	.339	.544	3.215	.004	R^2 =.492

BASC-2.

Separate standard multiple regressions were performed for the stroke group and control group with the Social Skills subscale T score from the BASC-2 as the DV and Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, and BRIEF WM subscale as the IVs. These variables significantly predicted the Social Skills T score for the control group, F (5, 26) =

2.193, p = .014, R^2 = .406, but not the stroke group, F (5, 21) = 2.193, p = .094, R^2 = .343. Closer inspection of the results revealed that BRIEF WM *T* score was a significant predictor in the model for the control group (p = .003). Although the overall model was not significant for the stroke group, Block Recall and BRIEF WM *T* score were significant predictors (p = .037 and .033, respectively). Table 32 displays the results of the regression analyses for each group.

Table 32: Multiple regressions of WM on BASC-2 Social Skills T-score for stroke and control groups

Group	Variables	B	SE B	β	T	*p*	
Stroke	Word List Recall	-.218	.141	-.364	-1.541	.138	
	Block Recall	.400	.180	.642	2.223	.037	
	Counting Recall	.042	.168	.069	.248	.807	
	Backward Digit Recall	-.392	.203	-.506	-1.931	.067	
	BRIEF WM scale	-.377	.166	-.483	-2.277	.033	R^2 = .343
Control	Word List Recall	-.223	.119	-.330	-1.882	.071	
	Block Recall	.126	.125	.166	1.009	.322	
	Counting Recall	-.027	.110	-.042	-.243	.810	
	Backward Digit Recall	.106	.104	.184	1.019	.318	
	BRIEF WM scale	-.640	.195	-.537	-3.274	.003	R^2 = .406

Supplementary Analyses for IQ.

Given the finding of a significant group difference in IQ (previously reported), a series of hierarchical regression analyses were performed to evaluate the relative importance of IQ and group membership. Thus, the aim was to determine the importance of group membership beyond differences in IQ alone. Of note, this method of analysis was selected rather than covariance analysis because IQ does not meet the requirements for a covariate. As several researchers have pointed out (Dennis, 2009; Stevens, 2009), it is inappropriate to use IQ as a covariate because this removes important variance from the grouping variable, thereby violating the assumptions of covariance analysis and producing misleading results. Only the significant findings arising out of

this study were selected for further analysis using hierarchical regression. Hence, hierarchical regressions were carried out for the following DVs: Word List Recall, Block Recall, Counting Recall, Backward Digit Recall, SIP encoding, Mind in Eyes, and BRIEF WM T-score. IQ was assigned highest priority and entered into the first step of the regression and group membership was entered into the second step.

Working Memory Test Battery for Children (WMTB-C) Regression Analyses.

Word List Recall.

The regression analysis for Word List Recall revealed that R was significant at both steps, indicating that both IQ and group membership reliably predicted Word List Recall performance. After step 1, with IQ in the equation, $R^2 = .307$, F (1, 61) = 27.055, p = < .001. Thus, IQ accounted for 30.7% of the variance in Word List Recall performance. The addition of group membership to the regression model explained an additional 4.4% of the variance in Word List Recall performance and this change in R^2 was significant, F (1, 60) = 4.029, p = .049. Table 33 displays the results of the regression analysis at both steps.

Table 33: Hierarchical regression of IQ and group membership on Word List Recall performance

Model	Variables	B	SE B	β	t	*p*	
1	IQ	.653	.126	.554	5.201	.000	
							$R^2 = .307$
2	IQ	.538	.135	.456	3.97	.000	
	Group membership	10.336	5.149	.231	2.007	.049	
							$R^2 = .351$

Block Recall.

The regression analysis for Block Recall showed that R was significant only at the first step, suggesting that group membership did not reliably predict Block Recall performance above and beyond IQ. After step 1, with IQ in the equation, $R^2 = .315$, F (1, 61) = 28.075, p = < .001.

Thus, IQ accounted for 31.5% of the variance in Block Recall performance. The addition of group membership to the regression model explained only an additional 2.7% of the variance in Block Recall performance, which was not significant, F (1, 60) = 2.489, p = .120. Table 34 displays the results of the regression analysis at both steps.

Table 34: Hierarchical regression of IQ and group membership on Block Recall performance

Model	Variables	B	SE B	β	t	*p*	
1	IQ	.523	.099	.561	5.299	.000	
							R^2 = .315
2	IQ	.450	.108	.484	4.183	.000	
	Group membership	6.461	4.095	.182	1.578	.120	
							R^2 = .342

Counting Recall.

The regression analysis for Counting Recall showed that R was significant only at the first step, suggesting that group membership did not reliably predict Counting Recall performance above and beyond IQ. After step 1, with IQ in the equation, R^2 = .423, F (1, 60) = 42.953, p = < .001. Thus, IQ accounted for 42.3% of the variance in Counting Recall performance. The addition of group membership to the regression model explained only an additional 0.3% of the variance in Counting Recall performance, which was not significant, F (1, 59) = .351, p = .556. Table 35 displays the results of the regression analysis at both steps.

Table 35: Hierarchical regression of IQ and group membership on Counting Recall performance

Model	Variables	B	SE B	β	t	*p*	
1	IQ	.657	.099	.650	6.630	.000	
							R^2 = .423
2	IQ	.630	.109	.624	5.775	.000	
	Group membership	2.391	4.036	.064	.592	.556	
							R^2 = .426

Backward Digit Recall.

The regression analysis for Backward Digit Recall showed that R was significant only at the first step, suggesting that group membership did not reliably predict Backward Digit Recall performance above and beyond IQ. After step 1, with IQ in the equation, $R^2 = .269$, F (1, 59) = 21.752, p = < .001. Thus, IQ accounted for 26.9% of the variance in Backward Digit Recall performance. The addition of group membership to the regression model explained only an additional 1.3% of the variance in Backward Digit Recall performance and this change in R^2 was not significant, F (1, 58) = 1.017, p = .317. Table 36 displays the results of the regression analysis at both steps.

Table 36: Hierarchical regression of IQ and group membership on Backward Digit Recall performance

Model	Variables	B	SE B	β	t	*p*	
1	IQ	.489	.105	.519	4.664	.000	$R^2 = .269$
2	IQ	.439	.116	.466	3.795	.000	
	Group membership	4.511	4.473	.124	1.009	.317	
							$R^2 = .282$

SIP Encoding.

The regression analysis for SIP Encoding (i.e., first step of the SIP Interview) showed that R was significant only at the first step, suggesting that group membership did not reliably predict SIP Encoding performance above and beyond IQ. After step 1, with IQ in the equation, $R^2 = .099$, F (1, 60) = 6.76, p = .013. Thus, IQ accounted for 9.9% of the variance in SIP Encoding performance. The addition of group membership to the regression model explained only an additional 2.2% of the variance in SIP Encoding performance and this change in R^2 was

not significant, F (1, 59) = 1.504, p = .225. Table 37 displays the results of the regression analysis at both steps.

Table 37: Hierarchical regression of IQ and group membership on SIP Encoding performance

Model	Variables	B	SE B	β	t	*p*	
1	IQ	.051	.020	.314	2.564	.013	
							R^2 = .099
2	IQ	.040	.022	.246	1.829	.072	
	Group membership	1.012	.825	.165	1.226	.225	
							R^2 = .121

Mind in Eyes.

The regression analysis for Mind in Eyes score showed that R was significant only at the first step, suggesting that group membership did not reliably predict Mind in Eyes performance above and beyond IQ. After step 1, with IQ in the equation, R^2 = .197, F (1, 61) = 14.942, p = < .001. Thus, IQ accounted for 19.7% of the variance in Mind in Eyes performance. The addition of group membership to the regression model explained only an additional 0.1% of the variance in Mind in Eyes performance and this change in R^2 was not significant, F (1, 60) = .408, p = .827. Table 38 displays the results of the regression analysis at both steps.

Table 38: Hierarchical regression of IQ and group membership on Mind in Eyes performance

Model	Variables	B	SE B	β	t	*p*	
1	IQ	.094	.024	.444	3.866	.000	
							R^2 = .197
2	IQ	.092	.027	.432	3.378	.001	
	Group membership	.226	1.033	.028	.219	.827	
							R^2 = .197

BRIEF WM T score.

The regression analysis for BRIEF WM *T* score revealed that R was significant at both steps, indicating that both IQ and group membership reliably predicted BRIEF WM *T* score. After step 1, with IQ in the equation, $R^2 = .189$, $F\ (1, 60) = 14.028$, $p = < .001$. Thus, IQ accounted for 18.9% of the variance in BRIEF WM *T* score. The addition of group membership to the regression model explained an additional 10.9% of the variance in BRIEF WM *T* score and this change in R^2 was significant, $F\ (1, 59) = 9.178$, $p = .004$. Table 39 displays the results of the regression analysis at both steps.

Table 39: Hierarchical regression of IQ and group membership on BRIEF WM *T* score

Model	Variables	B	SE B	B	t	*p*	
1	IQ	-.315	.084	-.435	-3.745	.000	
							$R^2 = .189$
2	IQ	-.202	.087	-.279	-2.311	.001	
	Group membership	-10.134	3.345	-.366	-3.030	.004	
							$R^2 = .299$

Discussion

The aim of Study 1 was to assess the impact of pediatric unilateral AIS on WM and SIP patterns. This was a cross-sectional study comparing children with histories of stroke with typically developing children on a series of WM and SIP measures. This was also the first attempt to examine the association between these constructs in the pediatric stroke population. It was predicted that children with histories of stroke would demonstrate more deficits in WM and SIP relative to typically developing children, as assessed by task performance and parent ratings. Furthermore, it was expected that WM performance would predict SIP performance.

Working Memory Outcomes

As hypothesized, children with histories of stroke performed significantly worse than controls on all four WMTB-C (Pickering & Gathercole, 2001) measures. These results were corroborated by parent reports indicating significantly more WM dysfunction in the stroke group compared to controls. It is important to note that although significant group differences in WM were found, the mean group averages for the stroke group on the WMTB-C (Pickering & Gathercole, 2001) fell within the average range (mean composite scores ranged from 90.3 to 98.5). Similarly, the mean *T* score on the BRIEF (Gioia et al., 2000) fell within the non-clinical range (*M* = 61.6). These results are consistent with the existing literature suggesting subtle WM difficulties in children with histories of stroke relative to controls (Lansing et al., 2004; Westmacott et al., 2009, 2010; White et al., 2000). The current study adds to the existing literature by confirming subtle challenges not only in the verbal central executive component of WM (as previously demonstrated by Lansing et al., 2004, Westmacott et al., 2009, 2010, and White et al., 2000), but also in the phonological loop, visuospatial sketchpad, and visual central executive components. These results indicate that children with unilateral AIS are at risk for developing subtle challenges in all three components of WM, but not to the extent that they would be considered "impaired."

Supplementary analyses were also carried out to determine the impact of intellectual functioning and stroke diagnosis on WM variables. Consistent with the existing literature (Allman & Scott, 201; Ballantyne et al., 2008; Hartel, et al., 2004; Max et al., 2010; Nass & Trauner, 2004; Westmacott et al., 2009, 2010), the intellectual functioning of children with unilateral AIS was compromised relative to controls, although the mean of the stroke group fell within the low end of the average range (*M* FSIQ = 92.6). Hierarchical regression analyses

indicated that performances on the Block Recall, Counting Recall, and Backward Digit Recall subtests of the WMTB-C (Pickering & Gathercole, 2001) were in line with what would be expected based on intellectual functioning. In contrast, the difficulties displayed by the stroke group relative to controls on the Word List Recall subtest and the WM subscale of the BRIEF (Gioia et al., 2000) were above and beyond what would be predicted by intellectual functioning alone. In other words, group differences on the phonological loop and WM in everyday life were best explained by both stroke diagnosis and intellectual functioning, while group differences on the visuospatial sketchpad and the central executive were best explained by individual differences in intellectual functioning. The predictive power of intellectual functioning for WM is not surprising, especially in light of the fact that multiple studies have documented a strong relationship between WM and intellectual functioning for both children (Belacchi, Carretti, & Cornoldi, 2010; Engel de Abreu, Conway, & Gathercole, 2010; Swanson, 2011; Voelke, Troche, Rammsayer, Wagner, & Roebers, 2013) and adults (Ackerman, Beier, & Boyle, 2005). However, the fact that stroke diagnosis was able to contribute to the prediction of performance on both the phonological loop variable and the more behaviourally-oriented questionnaire variable indicates that stroke diagnosis has predictive power that goes beyond intellectual functioning alone. Thus, it will be important for future studies to untangle how stroke diagnosis and intellectual functioning are related to WM performance in children. In doing so, it will be critical for studies to take developmental differences into account by comparing different age groups, as it is well-known that WM develops from early childhood well into adulthood (Booth et al., 2008; Farber & Betelava, 2011; Swanson, 1999). In other words, the influence of intellectual functioning on WM could vary depending on the developmental stage of the child.

Social Outcomes

The stroke group performed significantly worse than controls on the stage of the SIP Interview (Keil & Price, 2009) that corresponds with the first step of the Crick and Dodge (1994) model – encoding. These results were complemented by the finding that children with histories of stroke fared significantly worse on the Mind in Eyes Task (Baron-Cohen et al., 2001) than controls, indicating difficulty in facial decoding. In contrast, children with unilateral AIS performed similarly to controls across the remaining steps of the Crick and Dodge (1994) model. Specifically, groups did not differ with respect to interpretation (i.e., hostile vs. non-hostile attributions), goals (i.e., pro-social, negative/aggressive, non-social, inept, irrelevant), response generation (i.e., total responses, competent, aggressive, inept), or enactment. Although the groups generally evaluated responses similarly, stroke participants were more likely than controls to evaluate aggressive responses as being effective. The finding of generally similar SIP profiles across groups was corroborated by parent reports, as groups did not differ significantly on the Social Skills subscale of the BASC-2 (Reynolds & Kamphaus, 2004). Moreover, the mean *T* scores on the Social Skills subscale fell within the non-clinical range for both the stroke group ($M = 46.7$) and the control group ($M = 49.1$). These findings add to the existing literature by providing the first comprehensive assessment of SIP skills in school-age children with unilateral AIS.

The fact that parents rated the stroke group similarly to controls on the Social Skills subscale is surprising given that previous studies have documented caregiver reports of social deficits in children with histories of stroke (Greenham et al., 2010; Hurvitz et al., 2004; O'Keeffe et al., 2012; Trauner et al., 1996). These differing results may be due to methodological factors, as a variety of caregiver report measures were used in previous studies, none of which included

the BASC-2 (Reynolds & Kamphaus, 2004). Differing sample characteristics may have also contributed to the conflicting findings, as different inclusion/exclusion criteria were used. For example, several of the studies included children with hemorrhagic stroke (e.g., Hurvitz et al., 2004; Trauner et al., 1996) and none excluded children with seizure disorders. Further, the sample analyzed by Greenham et al. (2010) consisted of children with a variety of brain insults (e.g., stroke, traumatic brain injury, infective, etc.). It is possible that the stroke participants in the current study were less vulnerable to social deficits due to the specific nature of the mechanism of brain insult (i.e., AIS) and by virtue of the fact that none had histories of seizure disorders, which has been shown to be a predictor of worse cognitive and behavioural outcomes (Ballantyne et al., 2008; Everts et al., 2008; Hartel et al., 2004; Nass & Trauner, 2004). Family environment may also account for the lack of social impairment. For example, previous studies of children with TBI have identified low SES and maladaptive parenting as correlates for poor behavioural outcomes (for review, see Li & Liu, 2013). Given that the majority of the families of the stroke group in the current study were found to have relatively high SES, this may have served as a protective factor for social outcomes. Future research is required to investigate specific family functioning variables (e.g., parenting styles, parental distress, parental warmness, etc.) associated with social outcomes following pediatric stroke. Finally, the choice of parent informants in the current study may also account for the finding of adequate social functioning. It is possible that social difficulties may have been reported by the children themselves or by their teachers. This point highlights the need for future studies to gather reports from multiple informants.

The finding of social cue encoding and decoding difficulties in children with histories of stroke is consistent with previous literature (Ballantyne & Trauner, 1999; Boni et al., 2001). This

finding is important because deficits in social cue decoding have been identified as a risk factor for social maladjustment (Bryan & Bryan, 1990; Crick & Dodge, 1994). In addition, social cue decoding has been found to predict popularity among peers (Boyatzis & Satyaprasad 1994; Nowicki & Duke, 1992). It has been suggested that SIP at the early encoding/decoding steps impacts processing at all subsequent steps (Crick & Dodge, 1994). For example, failure to encode an adequate amount of information about a social situation can potentially lead to inaccurate interpretation of social cues, followed by the generation of inappropriate ideas about how to respond, which can ultimately lead to inappropriate behavioural conduct. With this in mind, the lack of impairment found across the remaining SIP steps and in social functioning in everyday life (as indicated by parent reports) in the current study is particularly puzzling. In other words, it is unclear as to why children with AIS would demonstrate isolated difficulties in social encoding and decoding that would not impact other domains of social functioning. Several explanations may account for these results. First, the findings of compromised social encoding and decoding abilities relative to controls may reflect subtle difficulties in these areas, rather than genuine "deficits." In other words, these relative challenges may not be at a level that overtly interfere with overall social functioning. It is possible that a certain "threshold" of difficulties in these abilities must be reached before observing an impact on subsequent steps of SIP. Closer examination of the results provides support for this view, as although significant differences between the groups were found, there was not a large discrepancy between the mean scores for each group. Further work will be necessary in order to ascertain the specific nature of the relationship between overall social competence and social encoding and decoding abilities. An alternative explanation for adequate performance on the remaining steps in the SIP Interview (Keil & Price, 2009) despite difficulties on the first step may relate to the task itself. That is,

although designed to be ambiguous, the social situations presented in the task may have been simple enough that participants may not have had to rely as heavily on their social encoding and decoding abilities. As illustrated by the Crick and Dodge (1994) model, interpretational processes are guided by one's database of social information, which consists of the long-term memory store of previous social experiences, social knowledge, and social schemata. Thus, it is possible that even with limited information about the social situation encoded, one may be able to successfully navigate simple social situations by relying on this database as a guide. This view is in line with Boni et al.'s (2001) finding that children with stroke secondary to SCD demonstrated more errors on social decoding when the tasks increased in complexity (i.e., cues became less explicit). It will be important for future studies to examine the SIP profiles of children with AIS using more complex and cognitively demanding measures.

Supplementary analyses indicated that the group differences found in social encoding and decoding were attributable to IQ. That is, hierarchical regressions revealed that stroke diagnosis did not account for a significant amount of variance in these variables above and beyond IQ alone. This finding is not entirely surprising, given that several lines of converging evidence have revealed an association between IQ and social skills in children. For example, significant, positive correlations have been reported between IQ and teacher ratings of a variety of personality traits in typically developing children, including sociability, popularity, ambition, and maturity (Barnard Zimbardo, & Sarason, 1968). Similarly, verbal IQ has been shown to predict emotional intelligence in typically developing adolescents (Hogan et al., 2010). In addition, a recent study of children with ASD found that children with higher IQ scores had higher caregiver ratings of adaptive/appropriate social skills than children with low IQ scores (Tureck & Matson, 2012). Importantly, the association between IQ and social skills in children with histories of

stroke has not been directly evaluated in previous studies. Thus, more research is needed regarding the specific nature of the relationship between IQ and social skills in this population.

Relationship between Working Memory and Social Information Processing

Mixed support was found for the hypothesized association and predictive value of WM on SIP skills. This hypothesis was explored by conducting separate correlational analyses and multiple regression models for the stroke group and the control group to determine the associations between the variables, as well as the proportion of variance in SIP performance explained by the group of WM variables. The majority of the regression models were non-significant. The exception was a significant finding for the Social Skills *T* score model for the control group. Closer inspection of the results of this model revealed that only the BRIEF WM *T* score had a significant effect. Additional exceptions were findings of significant effects for several of the individual WM predictors (despite the fact that the overall models were non-significant). Correlational analyses revealed consistent significant and positive associations between verbal central executive ability (as indicated by Backward Digit Recall performance) and encoding, generation of pro-social goals, affiliation competent response evaluation, and enactment scores on the SIP Interview (Keil & Price, 2009) for the stroke group. Similarly, significant, positive correlations were found between visuospatial sketchpad ability (as indicated by Block Recall performance) and each of these measures, with the exception of encoding. Not surprisingly, the BRIEF WM scale was negatively and significantly correlated with the BASC-2 Social Skills scale for both groups, indicating that children who were rated more favourably by parents on social skills tended to be rated by parents as having less WM dysfunction. The only other significant correlation found for the control group was a positive association between

instrumental competent response evaluation and the BRIEF WM scale. This is the first study to date to explore the contribution of WM to social functioning in the pediatric stroke population.

The finding of significant relationships between central executive ability and SIP encoding, goal-setting, response evaluation, and enactment skills is consistent with previous studies documenting a significant relationship between the central executive and social outcomes in other populations of children (Kofler et al., 2011; McQuade et al., 2013) and adults (Anderson & Knight, 2010; Channon & Crawford, 2010; Hecker & Dutke, 2004; Hills & Pachur, 2012; Hommel et al., 2009; Thornton & Conway, 2013). However, unlike previous findings in children (Kofler et al., 2011; McQuade et al., 2013), visuospatial sketchpad ability was also associated with SIP. Given that the measure of SIP used in the current study required participants to view video vignettes, it is not surprising that there was an association between SIP performance and the ability to hold visual information in mind. Furthermore, it did not come as a surprise that, like previous studies (Hommel et al., 2009; Kofler et al., 2011; McQuade et al., 2013), an association was found between questionnaire measures of social functioning and WM. However, the relatively low number of associations found between WM and SIP in the control group is puzzling, particularly in light of the number of significant correlations found in the stroke group. This discrepancy raises a fundamental question about the relationship between SIP and WM as it pertains to typically developing children versus children with histories of stroke: whether SIP taxes WM capacity to a greater extent in the pediatric stroke population compared to typically developing children. In other words, it remains unclear as to whether children with histories of stroke must rely more heavily on WM during SIP compared to typically developing children.

Limitations of the current study may have contributed to the failure to find significant regression models. The current study likely lacked statistical power to detect these effects due to

the fact that each group consisted of only 32 children. Although it would have been possible to rectify this problem by combining the groups for the regression analyses (rather than running separate analyses), this type of analysis was not carried out in order to allow for comparisons between the models. In other words, it was unclear as to whether the WM variables would have the same predictive power for each group. Furthermore, it would have been meaningless to combine the groups given the inherent differences in their medical and developmental histories. Despite the lack of significant findings, WM explained a substantial percentage of the variance in SIP for both the stroke group (R^2 range = .328 to .408) and the control group (R^2 range = .213 to .492). Collectively, these results indicate that WM plays an important role in SIP and that future studies comparing the magnitude of the relationship between SIP and WM in large samples of children with histories of stroke and typically developing children will be necessary to further elucidate this complex relationship.

Study 2: Assessment of Effects of Age at Stroke and Lesion Location

Objectives and Hypotheses

This study was intended as an extension of the findings from Study 1. The objective of this study was to examine the effects of age at stroke (i.e., perinatal, 1 month-5 years, 6-14 years) and lesion location (i.e., cortical, subcortical, and combined) on WM and social outcomes. This issue was explored by comparing the WM and Social Skills *T* scores from the BRIEF and BASC-2 (respectively) among children with histories of stroke. Based on past findings that have indicated a non-linear relationship between age at stroke and cognitive outcomes (Allman & Scott, 2011; Everts et al., 2008), it was hypothesized that the early childhood stroke group (i.e., stroke between 1 month-5 years) would demonstrate more favourable WM and social outcomes compared to the perinatal and late childhood (i.e., stroke between 6-14 years) stroke groups.

Moreover, based on the Westmacott et al. (2010) findings, children with lesions in combined cortical and subcortical regions were expected to display poorer WM and social outcomes than children with lesions located in either the cortical or subcortical regions. Further, based on the Westmacott et al. (2010) findings, different periods of vulnerability for WM and social outcomes were expected for the cortical, subcortical, and combined cortical-subcortical stroke groups.

Method

Participants

All data consisted of a retrospective participant sample of patients evaluated as part of a routine, post-stroke neuropsychological assessment at The Hospital for Sick Children. Inclusion criteria included the following: (1) a single AIS documented on MRI or CT; (2) AIS before the age of 14 years; (3) at least 6 month post-stroke; and, (4) fluency in English. Exclusion criteria included the following: (1) bilateral lesions; (2) multiple strokes; (3) seizure disorders; (4) preterm birth (less than 36 weeks gestation); (5) hypoxic-ischemic encephalopathy; (6) SCD; (7) psychosis; (8) moya moya disease; and, (9) any other neurological disorders (e.g., brain injury, malignancy, etc.). These criteria yielded a sample of 125 children for the BRIEF data and 129 children for the BASC data. Participants were stratified by age at stroke as follows: *perinatal* (stroke in the prenatal period or first 28 days of life), *early childhood* (stroke occurrence between 1 month to 5 years), and *late childhood* (stroke occurrence between 6-14 years). Participants were also stratified by lesion location as follows: *cortical* (cortical infarct with no subcortical involvement), *subcortical* (infarct restricted to thalamus and/or basal ganglia), and *combined* (infarct involving cortex plus basal ganglia and/or thalamus). Table 40 displays the participant characteristics for the BRIEF data and Table 41 displays the participant characteristics for the BASC-2 data.

Table 40: Characteristics of participant group for BRIEF analysis, by age at stroke

	All participants	Perinatal	1 mo-5y	6-14y
Total number	125	49	53	23
Male/Females	74/51	29/20	30/23	15/8
Age at stroke, mean (SD)	2.94y (3.78)	0.002y (0.007)	2.66y(1.71)	9.75y (2.62)
Age at assessment, mean (SD)	9.34y (3.31)	9.26y (3.39)	8.46y (2.61)	11.57y (3.70)
Time since stroke, mean (SD)	6.33y (3.71)	8.96y (2.95)	5.80y (2.93)	2.13y (2.08)
Lesion Location				
Cortical	40	27	11	2
Subcortical	44	4	28	12
Combined	41	18	14	9

Table 41: Characteristics of participant group for BASC-2 analysis, by age at stroke

	All participants	Perinatal	1 mo-5y	6-14y
Total number	129	56	53	20
Male/Females	75/54	31/25	30/23	14/6
Age at stroke, mean (SD)	2.56y (3.58)	0.002y (0.006)	2.51y(1.70)	9.61y (2.76)
Age at assessment, mean (SD)	8.61y (3.93)	8.31y (4.08)	7.81y (3.51)	11.52y (3.39)
Time since stroke, mean (SD)	6.02y (4.01)	8.16y (3.71)	5.31y (3.52)	2.27y (2.39)
Lesion Location				
Cortical	42	29	11	2
Subcortical	46	6	30	10
Combined	41	21	12	8

Procedure

Parents completed the BRIEF and/or the BASC-2 as part of their child's routine neuropsychological evaluation. All testing was completed at The Hospital for Sick Children. As with Study 1, the WM scale was analyzed for the BRIEF data and the Social Skills scale was analyzed for the BASC-2 data (detailed descriptions of the BRIEF and the BASC-2 are provided on pages 43-44). All parents had provided consent for their child's assessment results to be used for research purposes.

Results

Age Group Characteristics.

A one-way between subjects ANOVA was used to determine whether there was a significant difference between the groups for age at assessment. With respect to the BRIEF data, a significant difference was found for age at assessment [F (2, 122) = 7.841, p = .001, partial eta squared = .114, power = .948], with children in the late childhood stroke group being significantly older at the time of assessment than the perinatal group (p = .013) and the early childhood stroke group (p = < .001). Similarly, a significant difference for age at assessment was found for the BASC-2 data, [F (2, 126) = 7.392, p = .001, partial eta squared = .105, power = .935], with children in the late childhood stroke group again being significantly older at the time of assessment than the perinatal group (p = .004) and the early childhood stroke group (p = .001). However, age was not used a covariate as both the BRIEF and BASC-2 are standardized measures.

A one-way between subjects ANOVA was also used to determine whether there was a significant difference between the groups for time since stroke. With respect to the BRIEF data, a significant difference was found for time since stroke [F (2, 118) = 45.68, p < .001, partial eta squared = .436, power = 1.0]. Unsurprisingly, the perinatal group had a significantly greater time between stroke and assessment than the early childhood and late childhood groups (both p < .001). The early childhood group also had a significantly greater time between stroke and assessment than the late childhood group (p < .001). Similarly, a significant difference was found for time since stroke for the BASC-2 data [F (2, 120) = 22.04, p < .001, partial eta squared = .269, power = 1.0]. Again, the perinatal group had a significantly greater time between stroke and assessment than the early childhood and late childhood groups (both p < .001). The early

childhood group also had a significantly greater time between stroke and assessment than the late childhood group (p = .004). Correlational analyses revealed no significant relationships between time since stroke and BRIEF WM *T* score, r = .107, p = .242, or BASC-2 Social Skills *T* score, r = -.117, p = .199.

Age at stroke and lesion location effects for the BRIEF.

A 3 x 3 ANCOVA was conducted to evaluate the effects of age at stroke and lesion location on the WM *T* score for the BRIEF, with time since stroke as the covariate. Prior to analysis, the WM *T* score variable was examined for accuracy of data entry, missing values, and the assumptions of univariate analysis. No missing values were found and there were no outliers. Kolmogorov-Smirnov's test of normality indicated normal distributions for both the early childhood stroke group (p = .200) and the late childhood stroke group (p = .200); however, the distribution for the perinatal stroke group was found to be non-normal (p = .015). Graphical checks indicated that the distribution for the perinatal group was slightly negatively skewed. It was not possible to transform the data because this resulted in violations of normality for the early childhood and late childhood stroke groups. The assumption of homogeneity of variance was determined to be satisfactory. The ANCOVA indicated that there were no significant main effects for age at stroke [F (2, 111) = .015, p = .985, partial eta squared = .000, power = .052] or lesion location [F (2, 111) = 1.002, p = .370, partial eta squared = .018, power = .221]. Further, there was no significant interaction between age at stroke and lesion location [F (4, 111) = .656, p = .624, partial eta squared = .023, power = .208]. The mean WM subscale *T* score was 58.47 (*SD* = 1.74) for the perinatal group, 58.17 (*SD* = 1.58) for the early childhood group, and 56.09 (*SD* = 2.99) for the late childhood group. With respect to the lesion location groups, the mean WM *T* score was 57.98 (*SD* = 1.84) for the cortical group, 59.36 (*SD* = 1.71) for the subcortical

group, and 56.27 (*SD* = 2.14) for the combined group. Of note, all of these mean *T* scores fall within the non-clinical range. Table 42 displays the proportions of participants who obtained a *T* score one standard deviation above the mean (i.e., *T* score > 60), indicating a higher degree of WM dysfunction.

Table 42: Proportion of participants with BRIEF WM *T* scores > 60

Group	Total Number
All	57
Age at stroke	
Perinatal	22
1 mo-5y	24
6-14y	11
Lesion location	
Cortical	19
Subcortical	19
Combined	19

Age at stroke and lesion location effects for the BASC-2.

A 3 x 3 ANCOVA was conducted to evaluate the effects of age at stroke and lesion location on the Social Skills *T* score from the BASC-2, with time since stroke as the covariate. Prior to analysis, the Social Skills *T* score variable was examined for accuracy of data entry, missing values, and the assumptions of univariate analysis. No missing values were found and there were no outliers. Kolmogorov-Smirnov's test of normality indicated normal distributions for the perinatal group (p = .200), early childhood group (p = .067), and late childhood group (p = .200). Graphical checks confirmed that the distributions were normal. The assumption of homogeneity of variance was determined to be satisfactory. The ANCOVA indicated a significant main effect for age at stroke [F (2, 113) = 3.301, p = .040, partial eta squared = .055, power = .616], but no significant effects for lesion location [F (2, 113) = 1.218, p = .300, partial eta squared = .021, power = .261], or the interaction between age at stroke and lesion location [F

(4, 113) = 1.627, p = .172, partial eta squared = .054, power = .487]. Using the Bonferroni approach to adjust for multiple comparisons, post-hoc analyses indicated a significant difference between the early childhood group and the late childhood group (p = .039), in favour of the early childhood group. However, no significant differences were found between the perinatal group and early childhood group (p = 1.00) or late childhood group (p = .084). The mean Social Skills subscale *T* score was 48.59 (*SD* = 1.47) for the perinatal group, 50.81 (*SD* = 1.34) for the early childhood group, and 46.50 (*SD* = 2.79) for the late childhood group. With respect to the lesion location groups, the mean Social Skills *T* score was 49.43 (*SD* = 1.75) for the cortical group, 50.20 (*SD* = 1.46) for the subcortical group, and 47.78 (*SD* = 1.76) for the combined group. Of note, all of these mean *T* scores fall within the non-clinical range. Table 43 displays the proportions of participants who obtained a *T* score one standard deviation below the mean (i.e., *T* score < 40), indicating a higher degree of social skill impairment.

Table 43: Proportion of participants with BASC-2 Social Skills T scores < 40

Group	Total Number
All	30
Age at stroke	
Perinatal	14
1 mo-5y	10
6-14y	6
Lesion location	
Cortical	10
Subcortical	9
Combined	11

Discussion

The aim of Study 2 was to investigate the impact of age at stroke, lesion location, and their interaction on social and WM outcomes in children with histories of stroke. This study analyzed parent rating data of social skills and WM in a large, retrospective sample of children with unilateral AIS. Comparisons were made among children who experienced stroke during the perinatal period, early childhood (i.e., 1 month-5 years), and late childhood (i.e., 6-14 years). In addition, comparisons were made among children with cortical, subcortical, and combined (i.e., cortical and subcortical) lesions. It was predicted that the early childhood stroke group would demonstrate more favourable WM and social outcomes compared to the perinatal and older age at stroke groups. Children with lesions in combined cortical and subcortical regions were expected to display poorer WM and social outcomes than children with lesions located in either the cortical or subcortical regions. Moreover, different periods of vulnerability for WM and social outcomes were expected for the cortical, subcortical, and combined cortical-subcortical stroke groups.

Age at Stroke and Lesion Location Effects for Working Memory

In contrast to hypotheses, children's WM *T* scores on the BRIEF parent report (Gioia et al., 2000) did not differ according to age of stroke (i.e., perinatal, early childhood, late childhood), lesion location (i.e., cortical, subcortical, combined cortical-subcortical), or their interaction. It is important to note that, as with Study 1, all of the mean *T* scores for the groups fell within the non-clinical range. The lack of effect for age at stroke is in stark contrast to previous studies indicating a nonlinear pattern of cognitive impairment, whereby stroke in early childhood is found to be a protective factor (Allman & Scott, 2011; Everts et al., 2008; Westmacott, 2010). The non-significant finding for lesion location also conflicts with prior

studies in adults highlighting the importance of cortical regions for the phonological loop and visuospatial sketchpad (Philipose et al., 2007; Vallat et al., 2005). Moreover, this finding contrasts with studies reporting an interaction between lesion location and age of onset, whereby cortical and subcortical infarcts have been reported to have different periods of peak vulnerability (Westmacott et al., 2010; Nass & Trauner, 2004). These differing results may be due to methodological factors, as the current analysis of age at stroke and lesion location effects relied on a parent report measure, while other studies utilized specific WM tasks (i.e., Allman & Scott, 2011; Everts et al., 2008; Nass & Trauner, 2004; Philipose et al., 2007; Vallat et al., 2005; Westmacott et al., 2010). Further, the inconsistency between the findings of the current study and those of Philipose et al. (2007) and Vallat et al. (2005) may relate to developmental factors, as these studies consisted of adult samples. In other words, the cognitive and behavioural implications of stroke on the developing brain are likely very different than those for the adult brain. In addition, the sample analyzed by Philipose et al. (2007) consisted of adults in the acute stage of stroke (i.e., 3 days within symptom onset), which may not extend to children assessed several years post-stroke. In other words, the longer recovery time may have allowed the developing brain to evolve neural networks to compensate for the damage sustained by the stroke. It will be important for future studies to identify the mechanisms predicting the subtle difficulties found in the phonological loop, visuospatial sketchpad, and central executive components of WM in children with histories of unilateral AIS reported in Study 1. Potentially fruitful avenues for future investigation may include analyzing other lesion characteristics such as lesion size and laterality. It would also be helpful to compare WM performance among children with anterior, posterior, and diffuse infarcts given that previous research has identified different patterns of performance associated with damage to these regions in children with stroke

secondary to SCD (White et al., 2000). Moreover, longitudinal analysis will be critical in order to gain insight into the developmental trajectories of the specific WM components in children with histories of unilateral AIS.

Age at Stroke and Lesion Location Effects for Social Skills

The findings provided limited evidence for effects of lesion location or its interaction with age at stroke on social skills outcomes in children with histories of stroke. Group differences were found only between the early childhood and late childhood stroke groups, with the early childhood group being rated more favourably. This finding provides partial support for the view that there is a protective effect for stroke occurring in early childhood (Allman & Scott, 2011; Everts et al., 2008). At first glance, an obvious potential explanation for this protective effect of stroke in early childhood may relate to the confounding variable of time since stroke. That is, it is plausible that the children in the late childhood stroke group may have been rated less favourably by parents on social skills due to the fact that they had less time to recover since the stroke and had therefore not yet reached an optimal level of functioning. However, correlational analyses indicated that this was not the case, as time since stroke was not significantly correlated with BASC-2 (Reynolds & Kamphaus, 2004) Social Skills scores. A more likely alternative explanation for this effect may relate to differences in developmental stages of the children. Analyses of age group characteristics revealed that the late childhood group was significantly older at the time of the assessment compared to the early childhood group. It is possible that children in the early childhood group may have been exhibiting less social difficulties by virtue of the fact that their younger age group is faced with less social demands compared with the older age group. That is, the younger age group would not yet be required to master the social intricacies and higher-order thinking skills needed for successful

social interactions with older children. It is important to note that all of the mean *T* scores for each group fell within the non-clinical range. Again, developmental factors may account for the failure to find clinically significant difficulties in social functioning. It may be the case that social impairments do not start to manifest until later development (e.g., late adolescence, early adulthood, etc.) as social situations become increasingly complex. On the other hand, in the case of stroke occurring in the perinatal or early childhood periods, social difficulties may be more evident during early development and then resolve by later childhood. These developmental factors would not have been captured in the current study, as the age range was restricted to 6-14 years. Longitudinal studies will be critical for providing insight into the developmental trajectories of social outcomes in children with histories of stroke. Regarding the failure to find an effect of lesion location on social outcomes, it is possible that the effect of lesion location on cognitive outcomes documented in children with histories of stroke (Allman & Scott, 2011; Everts et al., 2008; Nass & Trauner, 2004; Westmacott et al., 2010) simply does not translate to the social domain. In others words, there may not be a clear link between this factor and social outcomes in the pediatric stroke population. On the other hand, it is possible that an effect of lesion location on social outcomes does exist in the sample, but simply was not captured due to the reliance on caregiver reports. It will be helpful for future studies to further assess effects of age at stroke and lesion location on social functioning using a variety of measures and lesion characteristic variables.

General Discussion

Surprisingly little is known about the social outcomes of children with histories of stroke, despite the significant long-term impact of social functioning on quality of life (Bohnert et al., 1997). To this end, the SIP and WM profiles of this population have remained largely

unexplored, despite research evidence and theoretical models suggesting that these abilities are critical, interrelated cognitive determinants of social competence (Anderson & Knight, 2010; Channon & Crawford, 2010; Crick & Dodge, 1994; Hecker & Dutke, 2004; Hommel et al., 2009; Yeates, 2007). To address these gaps, the current study compared the SIP and WM patterns of 32 children with histories of unilateral AIS to 32 healthy controls. Further, the relationship between WM and SIP performance was explored. These findings were extended in a second study that evaluated parent reports assessing WM and social skills in a large, retrospective sample of children with histories of unilateral AIS sustained during the perinatal period, early childhood (i.e., 1 month-5 years), and late childhood (6-14 years). Moreover, the effects of lesion location were investigated by comparing these indices among children with cortical, subcortical, and combined cortical-subcortical strokes.

Children were assessed on a comprehensive range of measures which were selected on the basis of their correspondence with gold-standard theoretical models of WM (Baddeley & Hitch, 1974) and SIP (Crick & Dodge, 1994). SIP capacity was assessed using the SIP Interview (Keil & Price, 2009). This is an experimental measure of SIP designed for children. The SIP Interview (Keil & Price, 2009) is designed to assess all six steps of the Crick and Dodge (1994) model: encoding, interpretation, goal clarification, response generation, response evaluation, and enactment. SIP capacity was further assessed using an experimental measure of facial decoding (i.e., Mind in Eyes Task; Baron-Cohen et al., 2001) and standardized parent reports (i.e., BASC-2; Reynolds & Kamphaus, 2004). WM capacity was evaluated using four subtests selected from the WMTB-C (Pickering & Gathercole, 2001). The WMTB-C (Pickering & Gathercole, 2001) is a standardized measure of WM for children designed to assess all three WM components proposed by Baddeley & Hitch (1974): phonological loop, visuospatial sketchpad, and central

executive. WM capacity was further assessed using standardized parent reports (i.e., BRIEF; Gioia, et al., 2000). In addition, overall intelligence was assessed using the WASI-II (Wechsler, 2011).

A number of important findings resulted from this study. First, children with histories of stroke performed significantly worse than typically developing children across measures of the phonological loop, visuospatial sketchpad, and central executive components of WM, although mean group averages fell within the average range. These findings of subtle WM difficulties were corroborated by parent reports, which indicated significantly higher rates of WM dysfunction in everyday in the stroke group compared to controls, although still within the non-clinical range. Second, children with histories of stroke performed significantly worse than typically developing children on measures of social cue encoding and decoding, but no significant group differences were found on the remaining steps of SIP (e.g., goal-setting, response generation, response evaluation, etc.) or parent reports of social skills in everyday life. Thus, the isolated difficulties in social cue encoding and decoding appeared to be subtle in nature, such that they did not interfere with subsequent SIP steps and day-to-day social functioning. Third, visuospatial sketchpad and verbal central executive abilities were associated with performance across many SIP steps, including encoding, pro-social goal-setting, competent response evaluation, and enactment in the stroke group. An association between parent reports of social skills and WM was also found in both groups. However, regression models failed to indicate a predictive value of WM for SIP, likely due to reduced statistical power. Further, far fewer associations between SIP and WM were found for the control group, raising the question of whether SIP is more taxing on WM abilities for children with histories of stroke compared to typically developing children. Fourth, stroke occurring during late childhood (i.e., 6-14 years)

was more detrimental to social skill functioning than stroke occurring during early childhood (i.e., 1 month-5 years). In contrast, no effect for lesion location or its interaction with age at stroke was found for social outcomes. Moreover, no effect for age at stroke, lesion location, or their interaction was found for WM. Fifth, consistent with previous studies (Allman & Scott, 201; Ballantyne et al., 2008; Hartel, et al., 2004; Max et al., 2010; Nass & Trauner, 2004; Westmacott et al., 2009, 2010), children with histories of stroke performed significantly worse than typically developing children on measures of intellectual functioning, although mean group averages again fell within the average range. Lastly, supplementary analyses indicated that the majority of WM and SIP performances were attributable to intellectual functioning, with the exception of phonological loop ability and WM functioning in everyday life.

This is the first study to examine WM and SIP profiles and the relationship between these processes as well as the influences of lesion location, age at stroke, and their interaction in children with histories of stroke. Strengths of the current study include the use of a homogeneous sample of children with unilateral AIS, selection of measures based on their correspondence with gold-standard theoretical models of SIP (Crick & Dodge, 1994) and WM (Baddeley & Hitch, 1974), inclusion of parent-report measures, use of a control group, large sample of children studied (especially when considering the rare occurrence of pediatric stroke), consideration of the impact of intellectual functioning on outcomes, and exploration of the relationships between SIP, WM, age at stroke, and lesion location. The findings of the current study have important clinical and theoretical implications, in so far as contributing information to enhance our knowledge of outcomes following pediatric stroke, brain-behaviour relationships, and relationship between WM and SIP. Based on these findings, children with histories of stroke might be expected to display subtle difficulties in WM and social information encoding and

decoding. Given the number of associations found between WM and SIP, these difficulties appear to go hand-in-hand. Moreover, children who sustain a stroke between 6 to 14 years of age may be particularly at risk for demonstrating social difficulties. Given the subtle nature of these difficulties, it is likely that they will go undetected without careful evaluation. These findings therefore highlight the need to include comprehensive assessments of both SIP and WM in the neuropsychological evaluations of children with histories of stroke. Moreover, indications of WM difficulties should alert clinicians to the possibility of SIP difficulties. Intervention efforts should also take into account the relationship between SIP and WM. For example, WM training may be a useful added component to social skills training. Future research is now required to shed light on the long-term trajectories of WM and SIP in the pediatric stroke population, as well as their interactions with various neurological, cognitive, psychosocial, and demographic variables.

Limitations and Future Directions

Unique contributions of the current study include the investigations of the social and WM profiles of children with unilateral AIS using gold-standard theoretical models of SIP (Crick & Dodge, 1994) and WM (Baddeley & Hitch, 1974) as guides, analysis of the effects of lesion location, age at stroke, and their interaction on WM and SIP, and exploration of the relationship between WM and SIP. Nevertheless, several caveats should be noted when interpreting the results. The age range of the current study was limited to 6 to 14. Therefore, the generalizability of findings to preschool children or older adolescents is unknown. Future studies should examine the WM and SIP patterns of children with histories of stroke in a variety of age groups. The sample was also comprised largely of intact, high SES families. Given that home environment (e.g., parenting styles, parental distress, family functioning, etc.) has been shown to correlate

with behavioural outcomes following TBI (Liu & Liu, 2012), it is possible that this factor accounted for the overall lack of SIP and WM deficits found in the stroke group by acting as a protective mechanism. Future studies examining the implications of home environment on outcomes following pediatric stroke will be an important next step in this research. Next, this study used direct assessments and parent reports to assess children's WM and social functioning. It will be valuable for future research to consider alternative measures of WM and social functioning, such as teacher and self-reports. Although this study used standardized measures to assess the phonological loop, visuospatial sketchpad, verbal and visual central executive, and everyday WM, a measure of the episodic buffer was not included. It may therefore be useful for future research to consider the influence of the episodic buffer on social interactions. As previously mentioned, lack of statistical power may have limited the ability to detect the predictive value of WM for SIP. Large studies examining the predictive power of WM for SIP outcomes in children with histories of stroke and typically developing children will therefore be required. Given the discrepancy found in the current study between the number of correlations found between SIP and WM in the stroke group versus the control group, it will be particularly informative for studies to include analyses comparing the models between the two groups in order to shed light on the question of whether SIP is more taxing on WM resources for children with histories of stroke relative to controls. In addition, the cross-sectional nature of the current study precluded the ability to gather information about the long-term WM and SIP trajectories of pediatric stroke survivors. It will be crucial for future studies to examine long-term WM and SIP trajectories of this population, particularly in light of the ongoing development of the PFC throughout adolescence and adulthood and the subsequent changes in higher-order cognitive abilities. Due to sampling issues (i.e., the rare occurrence of childhood stroke compared to

perinatal stroke), it was necessary to utilize retrospective samples in order to address the secondary aims of the current study concerning the effects of age at stroke, lesion location, and their interaction. For this reason, this analysis relied on parent report measures and did not include a comprehensive assessment of direct SIP and WM tasks. Large studies utilizing a combination of questionnaire and direct measures will be require to enhance our knowledge of the effects of lesion location and age at stroke as they pertain to the social and WM domains. To this end, it will be helpful to analyze a variety of lesion characteristics (e.g., lesion size, laterality, etc.) and to utilize more detailed lesion classification systems when examining the influence of this variable on cognitive and social outcomes. Similarly, a comprehensive analysis of the effects of age at stroke will require comparisons among a variety of age groups. Lastly, the current study did not take into account the effect of participation in various rehabilitation and extra-curricular activities that may have influenced WM and SIP abilities (e.g., speech-language therapy, tutoring, social groups, clubs, sports teams, etc.).

Conclusion

This research provides a more detailed understanding of social competence following pediatric stroke by investigating two individual characteristics that are critical for this complex construct: WM and SIP. This study illustrates that children with histories of unilateral AIS are at risk for compromised WM and SIP abilities relative to typically developing children, although difficulties are subtle and do not represent genuine "deficits." The fact that only subtle difficulties relative to healthy controls were evident could be interpreted as evidence that the developing brain is generally able to compensate for early injury, but perhaps not to the point that a "complete" recovery is achieved. Furthermore, the results suggest that WM ability, age at stroke, and IQ are important variables associated with social outcomes, with stroke between 6

and 14 years, reduced WM ability, and lower IQ being linked with less favourable social outcomes. In contrast, the most influential variable for WM outcomes appears to be IQ. An additional important finding is that there were no differences in social and WM outcomes based on lesion location. These results suggest that age at stroke and lesion location may have different mediation and moderation effects on various cognitive and social outcomes, depending on the domain in question. Hence, broad conclusions concerning the influence of age at stroke and lesion location on overall outcomes following pediatric stroke should be avoided. Future research is now required to investigate the interactive relationships among a variety of demographic (e.g., age at stroke, age at assessment, sex, SES, etc.), stroke (e.g., stroke type, etiology, etc.), lesion (e.g., volume, laterality, location, etc.), neurologic (e.g., seizure status, neurologic impairment, etc.), and environmental (e.g., family environment, parenting style, etc.) variables in the developmental trajectories of cognitive and social outcomes of children with histories of stroke. To this end, a comprehensive evaluation of social competence will require a multilevel approach, such that abilities spanning across all three components of this construct (i.e., individual characteristics and social skills, social performance and interaction, and social adjustment) are considered (Yeates et al., 2007). This research will be critical for designing interventions to promote better cognitive and social outcomes in children with histories of stroke, improving our understanding of prognosis in this population, and enhancing our knowledge of brain-behaviour relationships.

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Appendix A

Parent Consent

Title of Study: Working Memory and Social Information Processing Following Pediatric Stroke

Investigators:

Principle Investigator:
Robyn Westmacott, Ph.D., C. Psych., Psychologist, The Hospital for Sick Children
Telephone: (416) 873-6376

Co-investigators:
Amanda Fuentes, M.A., Clinical-Developmental Psychology student, York University
Email: amanda19@yorku.ca

Mary Desrocher, Ph.D, C.Psych., Associate Professor, York University
Telephone: (416) 736-5115 ext. 33111

Gabrielle deVeber, M.D., Neurologist, The Hospital for Sick Children
Telephone: (416) 813-7721

Purpose of the Research

The purpose of this study is to explore the connection between working memory and social understanding in children with and without histories of stroke. Working memory is defined as the mental system that allows us to keep information active for a brief amount of time while doing a task. For example, problem-solving requires working memory because it requires you to hold all of the important information in mind while coming up with a solution at the same time. Social information processing is the process through which we interpret and respond in a social situation. For example, if you wish to watch a different television show than someone else, social information processing allows us to think about types of things we would say to the person holding the remote control and to decide how we would react to their responses.

We are particularly interested in better understanding how children with and without histories of stroke perform on working memory and social information processing tasks, and whether achievement on these tasks is in some way related. We are also interested in better understanding how the age at stroke and the area of the brain affected by the stroke affects performance on working memory and social information processing tasks.

This research study has been reviewed and approved by the ethics protocols by the Human Participants Review Committee (HPRC) of York University and The Hospital for Sick Children and conforms to the standards of the Canadian Tri-Council Research Ethics Guidelines.

Description of the Research

This study will assess working memory and social information processing in typically developing children and children with histories of stroke between 6 and 14 years of age. The assessment will be conducted in a quiet room at York University or The Hospital for Sick Children. The length of testing session will be approximately 2 to 2 ½ hours. Breaks and snacks

will be provided. Parents will be asked to complete three questionnaires prior to or during the assessment.

Participation is voluntary and you and your child is free to withdraw from the study or refuse to complete a task at any time. Refusal to participate in or withdrawal from the study will not jeopardize current or future relationships with the researcher, York University, and/or The Hospital for Sick Children. If you or your child decides to stop participating, you will still be eligible to receive the out-of-pocket expenses and token of appreciation for agreeing to be in the project.

All results are confidential, subject to examination only by the investigators. Confidentiality will be provided to the fullest extent possible to the law. All data are coded and your name will not appear on any forms. You will be provided with a more detailed description of what we are hoping to find at the end of the study. You will also be provided with an individual report summarizing the results of your child's assessment. We will be reviewing your child's health record at SickKids for research purposes.

Potential Harms

We know of no harm that taking part in this study could cause your child.

Potential Discomforts or Inconvenience

The tasks are administered in a game-like format and most require verbal responses. Some children may feel uncomfortable in this situation, especially when tasks become difficult. If your child gets upset during the testing period or does not wish to continue, we will stop testing.

Potential Benefits

To individual subjects

You will receive a brief report describing your child's performance. This report will provide you with a better understanding of your child's strengths and weaknesses in regards to working memory and social information processing.

To society

You will be contributing to the understanding of the relationship of working memory and social information processing, an area that has not been extensively studied and much in need of research.

Confidentiality

We will respect your privacy. No information about who your child is will be given to anyone or be published without your permission, unless required by law. For example, the law could make us give information about you if a child has been abused, if you have an illness that could spread to others, if you or someone else talks about suicide (killing themselves), or if the court orders us to give them the study papers. Should you or your child wish to withdraw from the study, all information collected about your child will be destroyed. All information collected will be kept for a minimum of 7 years following completion of the research study. This information will be kept in a locked filing cabinet or storage area. All material will be shredded upon disposal.

Sick Kids Clinical Research Monitors or the regulator of the study may see your health record to check on the study. By signing this consent form, you agree to let these people look at your

records. We will put a copy of this research consent form in your patient health record and give you a copy as well.

The results of the tests we describe in this form will be used only for this study. If another doctor or caregiver caring for you needs to see these results, you will have to give us your permission. We will ask you to sign a form saying that you agree that this person can see your child's results. We recommend that only a registered psychologist or doctor tell you what the results of these tests mean.

Reimbursement

We will reimburse you for all your reasonable out of pocket expenses for being in this study (e.g., parking). If you stop taking part in the study, we will pay you for your expenses for taking part in the study up until that point. Children will be given a small token of appreciation for participating.

Participation

If you choose to let your child take part in this study you can take your child out of the study at any time. The care your child gets at Sick Kids will not be affected in any way by whether your child takes part in this study.

If your child becomes ill or is harmed because of study participation, we will treat your child for free. Your signing this consent form does not interfere with your legal rights in any way. The staff of the study, any people who gave money for the study, or the hospital are still responsible, legally and professionally, for what they do".

Conflict of Interest

I, and the other research team members have no conflict of interest to declare.

Sponsorship

Funding for this study was provided by March of Dimes of Canada and Ontario's Ministry of Training, Colleges, and Universities.

Consent should be indicated on the following page.

Thank you for your time.

Consent Form – Working Memory and Social Information Processing Following Pediatric Stroke

I have read the information on the research information form and understand the purpose and goals of this study. I have been informed that my child's results will be confidential. I also realize that my child's participation is voluntary and that they are free to withdraw at any time without jeopardizing their relationship with the researchers, York University, and The Hospital for Sick Children. I have also been told that there are no risks or benefits to taking part in the study. I have also been informed that I may keep the first part of the consent form, on which is printed the name and phone number of persons to contact, in the event that I may have questions about the research at some later time.

By signing this form, I agree that:

1) You have explained this study to me. You have answered all my questions.
2) You have explained the possible harms and benefits (if any) of this study.
3) I know what I could do instead of having my child take part in this study. I understand that I have the right to refuse to let my child take part in the study. I also have the right to take my child out of the study at any time. My decision about my child taking part in the study will not affect my child's health care at Sick Kids.
4) I am free now, and in the future, to ask questions about the study.
5) I have been told that my child's medical records will be kept private except as described to me.
6) I understand that no information about my child will be given to anyone or be published without first asking my permission.
7) I agree, or consent, that my child_____ may take part in this study.

_____ _____

Printed Name of Parent/Legal Guardian Parent/Legal Guardian's signature & date

_____ _____

Printed Name of person who explained consent Signature of Person who explained consent & date

_____ _____

Printed Witness' name (if the parent/legal guardian does not read English) Witness' signature & date

If you have any questions about any aspect of this research, please contact Dr. Mary Desrocher at **(416) 736-5115 ext. 33111.** Questions may also be directed to the York University Graduate Psychology Office at **(416) 736-5290.** Any ethical concerns regarding this research project should be directed to the Manager of Research Ethics at York University, at **(416) 736-5914.** If you have questions about your rights as a subject in a study or injuries during a study, please call the Research Ethics Manager at **(416) 813-5718**.

Appendix B

Child Control Consent

Title of Study: Working Memory and Social Information Processing Following Pediatric Stroke

Investigators:

Principle Investigator:
Robyn Westmacott, Ph.D., C. Psych., Psychologist, The Hospital for Sick Children
Telephone: (416) 873-6376

Co-investigators:
Amanda Fuentes, M.A., Clinical-Developmental Psychology student, York University
Email: amanda19@yorku.ca

Mary Desrocher, Ph.D, C.Psych., Associate Professor, York University
Telephone: (416) 736-5115 ext. 33111

Gabrielle deVeber, M.D., Neurologist, The Hospital for Sick Children
Telephone: (416) 813-7721

Purpose of the Research

The purpose of this study is to explore the connection between working memory and social understanding in children with and without histories of stroke. Working memory is defined as the mental system that allows us to keep information active for a brief amount of time while doing a task. For example, problem-solving requires working memory because it requires you to hold all of the important information in mind while coming up with a solution at the same time. Social information processing is the process through which we interpret and respond in a social situation. For example, if you wish to watch a different television show than someone else, social information processing allows us to think about types of things we would say to the person holding the remote control and to decide how we would react to their responses.

We are particularly interested in better understanding how children with and without histories of stroke perform on working memory and social information processing tasks, and whether achievement on these tasks is in some way related. We are also interested in better understanding how the age at stroke and the area of the brain affected by the stroke affects performance on working memory and social information processing tasks. You do not have this disease. We would like to collect information from healthy subjects like you.

This research study has been reviewed and approved by the ethics protocols by the Human Participants Review Committee (HPRC) of York University and The Hospital for Sick Children and conforms to the standards of the Canadian Tri-Council Research Ethics Guidelines.

Description of the Research

This study will assess working memory and social information processing in typically developing children and children with histories of stroke between 6 and 14 years of age. The assessment will be conducted in a quiet room at York University or The Hospital for Sick Children. The length of testing session will be approximately 2 to 2 ½ hours. Breaks and snacks

will be provided. Parents will be asked to complete three questionnaires prior to or during the assessment.

Participation is voluntary and you are free to withdraw from the study or refuse to complete a task at any time. Refusal to participate in or withdrawal from the study will not jeopardize current or future relationships with the researcher, York University, and/or The Hospital for Sick Children. If you decide to stop participating, you will still be eligible to receive the out-of-pocket expenses and token of appreciation for agreeing to be in the project.

All results are confidential, subject to examination only by the investigators. Confidentiality will be provided to the fullest extent possible to the law. All data are coded and your name will not appear on any forms. You will be provided with a more detailed description of what we are hoping to find at the end of the study. You will also be provided with an individual report summarizing the results of your assessment. We will be reviewing your health record at SickKids for research purposes.

Potential Harms

We know of no harm that taking part in this study could cause you.

Potential Discomforts or Inconvenience

The tasks are administered in a game-like format and most require verbal responses. Some children may feel uncomfortable in this situation, especially when tasks become difficult. If you get upset during the testing period or do not wish to continue, we will stop testing.

Potential Benefits

To individual subjects

You will receive a brief report describing your performance. This report will provide you with a better understanding of your strengths and weaknesses in regards to working memory and social information processing.

To society

You will be contributing to the understanding of the relationship of working memory and social information processing in children, an area that has not been extensively studied and much in need of research.

Confidentiality

We will respect your privacy. No information about who you are will be given to anyone or be published without your permission, unless required by law. For example, the law could make us give information about you if a child has been abused, if you have an illness that could spread to others, if you or someone else talks about suicide (killing themselves), or if the court orders us to give them the study papers. Should you wish to withdraw from the study all information collected about you will be destroyed. All information collected will be kept for a minimum of 7 years following completion of the research study. This information will be kept in a locked filing cabinet or storage area. All material will be shredded upon disposal.

Sick Kids Clinical Research Monitors or the regulator of the study may see your health record to check on the study. By signing this consent form, you agree to let these people look at your records. We will put a copy of this research consent form in your patient health record and give you a copy as well.

The results of the tests we describe in this form will be used only for this study. If another doctor or caregiver caring for you needs to see these results, you will have to give us your permission. We will ask you to sign a form saying that you agree that this person can see your results. We recommend that only a registered psychologist or doctor tell you what the results of these tests mean.

Reimbursement

We will reimburse you for all your reasonable out of pocket expenses for being in this study (e.g., parking and getting you to and from SickKids). If you stop taking part in the study, we will pay you for your expenses for taking part in the study up until that point. You will also be given a small token of appreciation for participating.

Participation

If you choose to take part in this study you can stop participating at any time.

If you become ill or are harmed because of study participation, we will treat you for free. Your signing this consent form does not interfere with your legal rights in any way. The staff of the study, any people who gave money for the study, or the hospital are still responsible, legally and professionally, for what they do.

Conflict of Interest

I, and the other research team members have no conflict of interest to declare.

Sponsorship

Funding for this study was provided by March of Dimes of Canada and Ontario's Ministry of Training, Colleges, and Universities.

Consent should be indicated on the following page.

Thank you for your time.

Consent Form – Working Memory and Social Information Processing Following Pediatric Stroke

I have read the information on the research information form and understand the purpose and goals of this study. I have been informed that my results will be confidential. I also realize that my participation is voluntary and am free to withdraw at any time without jeopardizing my relationship with the researchers, York University, and The Hospital for Sick Children. I have also been told that there are no risks or benefits to taking part in the study. I have also been informed that I may keep the first part of the consent form, on which is printed the name and phone number of persons to contact, in the event that I may have questions about the research at some later time.

By signing this form, I agree that:

1) You have explained this study to me. You have answered all my questions.
2) You have explained the possible harms and benefits (if any) of this study.
3) I know what I could do instead of taking part in this study. I understand that I have the right to refuse to take part in the study. I also have the right to stop taking part in the study at any time. My decision about taking part in the study will not affect my health care at Sick Kids.
4) I am free now, and in the future, to ask questions about the study.
5) I have been told that my medical records will be kept private except as described to me.
6) I understand that no information about me will be given to anyone or be published without first asking my permission.
7) I agree, or consent to take part in this study.

_____	_____
Printed Name of Participant	Participant's signature & date
_____	_____
Printed Name of person who explained consent & date	Signature of Person who explained consent
_____	_____
Printed Witness' name (if the participant does not read English)	Witness' signature & date

If you have any questions about any aspect of this research, please contact Dr. Mary Desrocher at **(416) 736-5115 ext. 33111.** Questions may also be directed to the York University Graduate Psychology Office at **(416) 736-5290.** Any ethical concerns regarding this research project should be directed to the Manager of Research Ethics at York University, at **(416) 736-5914.** If you have questions about your rights as a subject in a study or injuries during a study, please call the Research Ethics Manager at **(416) 813-5718**.

Appendix C

Child Patient Consent

Title of Study: Working Memory and Social Information Processing Following Pediatric Stroke

Investigators:

Principle Investigator:
Robyn Westmacott, Ph.D., C. Psych., Psychologist, The Hospital for Sick Children
Telephone: (416) 873-6376

Co-investigators:
Amanda Fuentes, M.A., Clinical-Developmental Psychology student, York University
Email: amanda19@yorku.ca

Mary Desrocher, Ph.D, C.Psych., Associate Professor, York University
Telephone: (416) 736-5115 ext. 33111

Gabrielle deVeber, M.D., Neurologist, The Hospital for Sick Children
Telephone: (416) 813-7721

Purpose of the Research

The purpose of this study is to explore the connection between working memory and social understanding in children with and without histories of stroke. Working memory is defined as the mental system that allows us to keep information active for a brief amount of time while doing a task. For example, problem-solving requires working memory because it requires you to hold all of the important information in mind while coming up with a solution at the same time. Social information processing is the process through which we interpret and respond in a social situation. For example, if you wish to watch a different television show than someone else, social information processing allows us to think about types of things we would say to the person holding the remote control and to decide how we would react to their responses.

We are particularly interested in better understanding how children with and without histories of stroke perform on working memory and social information processing tasks, and whether achievement on these tasks is in some way related. We are also interested in better understanding how the age at stroke and the area of the brain affected by the stroke affects performance on working memory and social information processing tasks.

This research study has been reviewed and approved by the ethics protocols by the Human Participants Review Committee (HPRC) of York University and The Hospital for Sick Children and conforms to the standards of the Canadian Tri-Council Research Ethics Guidelines.

Description of the Research

This study will assess working memory and social information processing in typically developing children and children with histories of stroke between 6 and 14 years of age. The assessment will be conducted in a quiet room at York University or The Hospital for Sick Children. The length of testing session will be approximately 2 to 2 ½ hours. Breaks and snacks

will be provided. Parents will be asked to complete three questionnaires prior to or during the assessment.

Participation is voluntary and you are free to withdraw from the study or refuse to complete a task at any time. Refusal to participate in or withdrawal from the study will not jeopardize current or future relationships with the researcher, York University, and/or The Hospital for Sick Children. If you decide to stop participating, you will still be eligible to receive the out-of-pocket expenses and token of appreciation for agreeing to be in the project.

All results are confidential, subject to examination only by the investigators. Confidentiality will be provided to the fullest extent possible to the law. All data are coded and your name will not appear on any forms. You will be provided with a more detailed description of what we are hoping to find at the end of the study. You will also be provided with an individual report summarizing the results of your assessment. We will be reviewing your health record at SickKids for research purposes.

Potential Harms

We know of no harm that taking part in this study could cause you.

Potential Discomforts or Inconvenience

The tasks are administered in a game-like format and most require verbal responses. Some children may feel uncomfortable in this situation, especially when tasks become difficult. If you get upset during the testing period or do not wish to continue, we will stop testing.

Potential Benefits

To individual subjects

You will receive a brief report describing your performance. This report will provide you with a better understanding of your strengths and weaknesses in regards to working memory and social information processing.

To society

You will be contributing to the understanding of the relationship of working memory and social information processing in children with histories of stroke, an area that has not been extensively studied and much in need of research.

Confidentiality

We will respect your privacy. No information about who you are will be given to anyone or be published without your permission, unless required by law. For example, the law could make us give information about you if a child has been abused, if you have an illness that could spread to others, if you or someone else talks about suicide (killing themselves), or if the court orders us to give them the study papers. Should you wish to withdraw from the study all information collected about you will be destroyed. All information collected will be kept for a minimum of 7 years following completion of the research study. This information will be kept in a locked filing cabinet or storage area. All material will be shredded upon disposal.

Sick Kids Clinical Research Monitors or the regulator of the study may see your health record to check on the study. By signing this consent form, you agree to let these people look at your records. We will put a copy of this research consent form in your patient health record and give you a copy as well.

The results of the tests we describe in this form will be used only for this study. If another doctor or caregiver caring for you needs to see these results, you will have to give us your permission. We will ask you to sign a form saying that you agree that this person can see your results. We recommend that only a registered psychologist or doctor tell you what the results of these tests mean.

Reimbursement

We will reimburse you for all your reasonable out of pocket expenses for being in this study (e.g., parking and getting you to and from SickKids). If you stop taking part in the study, we will pay you for your expenses for taking part in the study up until that point. You will also be given a small token of appreciation for participating.

Participation

If you choose to take part in this study you can stop participating at any time. The care you get at SickKids will not be affected in any way by whether you take part in this study.

If you become ill or are harmed because of study participation, we will treat you for free. Your signing this consent form does not interfere with your legal rights in any way. The staff of the study, any people who gave money for the study, or the hospital are still responsible, legally and professionally, for what they do.

Conflict of Interest

I, and the other research team members have no conflict of interest to declare.

Sponsorship

Funding for this study was provided by March of Dimes of Canada and Ontario's Ministry of Training, Colleges, and Universities.

Consent should be indicated on the following page.

Thank you for your time.

Consent Form – Working Memory and Social Information Processing Following Pediatric Stroke

I have read the information on the research information form and understand the purpose and goals of this study. I have been informed that my results will be confidential. I also realize that my participation is voluntary and am free to withdraw at any time without jeopardizing my relationship with the researchers, York University, and The Hospital for Sick Children. I have also been told that there are no risks or benefits to taking part in the study. I have also been informed that I may keep the first part of the consent form, on which is printed the name and phone number of persons to contact, in the event that I may have questions about the research at some later time.

By signing this form, I agree that:

1) You have explained this study to me. You have answered all my questions.
2) You have explained the possible harms and benefits (if any) of this study.
3) I know what I could do instead of taking part in this study. I understand that I have the right to refuse to take part in the study. I also have the right to stop taking part in the study at any time. My decision about taking part in the study will not affect my health care at Sick Kids.
4) I am free now, and in the future, to ask questions about the study.
5) I have been told that my medical records will be kept private except as described to me.
6) I understand that no information about me will be given to anyone or be published without first asking my permission.
7) I agree, or consent to take part in this study.

_____	_____
Printed Name of Participant	Participant's signature & date
_____	_____
Printed Name of person who explained consent & date	Signature of Person who explained consent
_____	_____
Printed Witness' name (if the participant does not read English)	Witness' signature & date

If you have any questions about any aspect of this research, please contact Dr. Mary Desrocher at **(416) 736-5115 ext. 33111.** Questions may also be directed to the York University Graduate Psychology Office at **(416) 736-5290.** Any ethical concerns regarding this research project should be directed to the Manager of Research Ethics at York University, at **(416) 736-5914.** If you have questions about your rights as a subject in a study or injuries during a study, please call the Research Ethics Manager at **(416) 813-5718**.

Appendix D

Assent

Title of Study: Working Memory and Social Information Processing Following Pediatric Stroke

Investigators:

Principle Investigator:
Robyn Westmacott, Ph.D., C. Psych., Psychologist, The Hospital for Sick Children
Telephone: (416) 873-6376

Co-investigators:

Amanda Fuentes, M.A., Clinical-Developmental Psychology student, York University
Email: amanda19@yorku.ca

Mary Desrocher, Ph.D, C.Psych., Associate Professor, York University
Telephone: (416) 736-5115 ext. 33111

Gabrielle deVeber, M.D., Neurologist, The Hospital for Sick Children
Telephone: (416) 813-7721

Why are we doing this study?
We are trying to understand how children learn and remember things. We are also looking at what kinds of things children do and say when they are with their friends or with people they don't know. We would like you to help us better understand these things.

What will happen during the study?
I would like you to do some games for me. These include remembering numbers and words, working with blocks, watching some videos and telling me what could happen, and other games like this. I would like you to try your best when playing these games. If you find it hard, just let me know if you want to stop. You can stop at any time. This will take about 2 to 2 ½ hours. We will take a break and provide a snack for part of the way through. We will be reviewing your health record at SickKids for research purposes.

Are there good things and bad things about the study?
These games will not hurt you in any way. There are no risks involved. You may find some of the tasks difficult and I just want you to try the best you can. If you find something uncomfortable, we can stop.

There are no direct benefits to you for taking part in this study. However, you will be helping us to understand how other children think and learn in similar situations. This will help us figure out how to help children who have difficulties with these types of games.

Who will know what I did in the study?
We will respect your privacy. The results will be shared with your parents/legal guardians. No information about who you will be given to anyone or be published without your permission, unless required by law. If we feel your health may be in danger, we may have to report your results to your doctor.

Can I decide if I want to be in the study?
Nobody will be angry or upset if you do not want to be in the study. We are talking to your parent/legal guardians about the study and you should talk to them about it too.

Sponsorship

Funding for this study was provided by March of Dimes of Canada and Ontario's Ministry of Training, Colleges, and Universities.

If you have any questions, please ask. If you would like to continue, please write your name on the line below.

I was present when _____ read this form and said that he or she agreed, or assented, to take part in this study.

_____ _____

Printed Name of person who obtained assent Signature & Date

Appendix E

Debriefing Script

The goal of this study is to explore the relationship of complex cognitive processes and social information processing in children with and without histories of stroke. We are testing children from the ages of 6 through to 14 years old. We are interested in how children's memory for verbal and visual information affects their socially adaptive behaviour. This is important because children are presented with a significant amount of information every day that they must remember and interpret. This information is important for learning and we want to know how children remember and manipulate this information. The brain is constantly changing throughout childhood, and new connections are being formed while others are being strengthened.

Working memory is important in a social context. Children and adolescents must go through a process of encoding, interpreting and responding to situations that are presented to them. They must look at incoming information and decide how they will respond. This interpretation and response system is thought to involve a 'database' of information from previous social interactions that can be used in the current situation. Because social interaction is often a constant exchange of both verbal (words) and nonverbal (gestures) information, a child may be interpreting, encoding and responding to many things at the same time. Therefore, they may need to keep a lot of information 'online' while interacting.

We used many different types of tasks to assess your working memory and social information processing. We also have to control for certain things that may also affect social behaviour, such as language skills. These tasks will help us better understand how working memory is related to social information processing. We expect that children who have significant difficulties with these higher order cognitive processes will have more difficulty in social situations.

Thank you for being a part of an original research project. Please feel free to ask any other questions you may have at this point, or in the future by contacting Amanda Fuentes (amanda19@yorku.ca), Mary Desrocher (416-736-5115 ext. 33111), or Robyn Westmacott (416-813-6376). Once again, thank you for your time and consideration.

Appendix F

Demographic Questionnaire

SECTION 1: GENERAL INFORMATION

In your own words, describe any concerns that you might have pertaining to your child's physical or cognitive development, academic achievement, behavior, or social/emotional development:

1. _____

2. _____

3. _____

4. _____

5. _____

SECTION 2: DEVELOPMENTAL HISTORY

Has this child ever had or been diagnosed with (if yes, please provide further information):

ADD	[ ] no	[ ] yes – explain:_____
ADHD	[ ] no	[ ] yes – explain:_____
Allergies	[ ] no	[ ] yes – explain:_____
Anxiety	[ ] no	[ ] yes – explain:_____
Autism Spectrum Disorder	[ ] no	[ ] yes – explain:_____
Chronic sinusitis	[ ] no	[ ] yes – explain:_____
Depression	[ ] no	[ ] yes – explain:_____
Diabetes	[ ] no	[ ] yes – explain:_____
Epilepsy	[ ] no	[ ] yes – explain:_____
Febrile convulsion	[ ] no	[ ] yes – explain:_____
Gifted and Talented	[ ] no	[ ] yes – explain:_____
Head Injury	[ ] no	[ ] yes – explain:_____
Hearing problems	[ ] no	[ ] yes – explain:_____
Hospitalization	[ ] no	[ ] yes – explain:_____
Language difficulties	[ ] no	[ ] yes – explain:_____
Language disorder	[ ] no	[ ] yes – explain:_____
Learning Disability	[ ] no	[ ] yes – explain:_____

Multiple ear infections	[ ] no	[ ] yes – explain:_____
Operations	[ ] no	[ ] yes – explain:_____
Other chronic illness	[ ] no	[ ] yes – explain:_____
Other psychiatric illness	[ ] no	[ ] yes – explain:_____
Sensory difficulties	[ ] no	[ ] yes – explain:_____
Vision problems	[ ] no	[ ] yes – explain:_____
Other	[ ] no	[ ] yes – explain:_____

SECTION 3: FAMILY HISTORY

1. Parents' Marital Status:

Single	_____
Married	_____
Separated	_____
Divorced	_____
Common-Law	_____
Legal Guardian	_____

2. Number of children in the family:

	Name	Age	Sex	Grade	Health?
1.	_____	_____	____	_____	_____
2.	_____	_____	____	_____	_____
3.	_____	_____	____	_____	_____
4.	_____	_____	____	_____	_____
5.	_____	_____	____	_____	_____
6.	_____	_____	____	_____	_____

3. Does any member of the family have a history of academic, learning, or attention difficulties? If yes, please indicate who and the nature of the difficulties.

_____

_____

_____

_____

4. Does anyone in the family have a history of emotional or psychiatric illnesses?
If yes, please indicate who and the nature of the difficulties:
_____

_____

_____

5. LANGUAGE:

What is your child's native (first) language? _____

If not English, at what age did your child start speaking English? _____

What is the mother's native language? _____

What is the father's native language? _____

What other languages are used in your home? _____
Which language is used most with your child? _____

6. MOTHER'S EDUCATION:

What is the highest educational level of the mother?

- Some elementary school (grades completed): _____
- Finished elementary school
- Some high school (grades completed):_____
- Finished high school
- Some college (No. of years): _____
- Finished college
- University
- Postgraduate degree (i.e., M.A., Ph.D.)

What diploma, degree, or certificate did you receive? _____

Where did you study, if not in Canada? _____

What kind of work do you do? _____

What is your job title? _____

Age when child was born _____ Current age _____

7. FATHER'S EDUCATION:

What is the highest educational level of the father?

- Some elementary school (grades completed):_____
- Finished elementary school
- Some high school (grades completed):_____
- Finished high school
- Some college (No. of years): _____
- Finished college
- University
- Postgraduate degree (i.e., M.A., Ph.D.)

What diploma, degree, or certificate did you receive? _____

Where did you study, if not in Canada? _____

What kind of work do you do? _____

What is your job title? _____

Age when child was born _____ Current age _____

8. FAMILY INCOME: What is the family's annual household income? Please circle.

- Under $20,000
- $20,001 - $30,000
- $30,001 - $40,000
- $40,001 - $50,000
- $50,001 - $80,000
- Over $80,000

9. What is your child's ethnicity? Please circle.

- African Origin
- Asian Am./Asian Pacific Islander
- Latino-a/Hispanic
- Aboriginal Canadian/Am. Indian/Alaska Native
- European Origin/White
- Bi-racial/Multi-racial
- Other: _____