Exploring factors that influence academic and behavioural outcome and the specific role of premorbid functioning, in a sample of children with severe TBI.

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COMPULSORY DECLARATION: This work has not been previously submitted in whole or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, cited, and referenced.

Signature: Date:

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Abstract

Children who have sustained severe traumatic brain injury (TBI) demonstrate a range of deficits in neurocognitive and behavioural domains (Anderson, Northam, Hendy, & Wrennall, 2001; Babikian & Asarnow, 2009; van't Hooft, 2010). These impairments may have adverse effects on academic and behavioural outcomes and can therefore interfere with school re-entry, educational progress, and ultimately, quality of life of the injured child (Anderson & Yeates, 2010; Keenan & Bratton, 2006; Lallo & van As, 2004). Pre-injury characteristics may increase risk for, and play a role in, TBI outcome, however, many studies exclude children with known adverse premorbid functioning (Dennis et al., 2007; Farmer et al., 2002). There are also dearth of published studies incorporating a variety of factors affecting outcome (e.g., socio economic status (SES), age at injury, time since injury, premorbid functioning, family environment) as well as academic and/or behavioural outcome variables in the same study generally. The broad aim of the study was therefore to contribute to the existing pediatric TBI (pTBI) literature on outcomes and factors influencing outcomes. In this study, I focused on investigating both academic and behavioural outcomes in a group of South African children of school-going age who had sustained a severe TBI.

This study includes two parts. For the first part of the study, the aim was to explore the relationship between commonly reported factors that influence outcome after TBI (premorbid functioning, age at injury, time since injury, family environment and SES), and academic and behavioural outcome. For the second part of the study, the aim was to investigate the specific role of premorbid functioning in academic and behavioural outcome.

The sample included 27 children who had been admitted to Red Cross War Memorial Children's Hospital (RXH) and received intracranial monitoring for closed severe TBI between 2006-2011, who were of school-going age at the time of the injury.

In terms of part one of the study, the results show elevated problems with academic outcome, and behavioural and executive functioning in the sample. The results also show that in this sample, factors such as family environment and premorbid functioning are particularly important with regards outcome in the home environment; while factors such as age at injury, family environment and SES play more of a role within the classroom environment.

In terms of part two of the study, the data did not confirm that participants with evidence of premorbid problems have a poorer academic outcome than those with no evidence of premorbid problems. The data did confirm that caregivers reported participants with evidence of premorbid problems as having a poorer outcome in terms of behaviour regulation and overall executive dysfunction than participants with no evidence of premorbid problems.

The current study adds weight to the advocacy for increased awareness on preventative measures and for identifying children, and families, at greater risk for dysfunction and poorer outcomes following a TBI. This in turn will assist in informing rehabilitation needs and strategies for intervention to facilitate reintegration into mainstream schooling and placement in learner support services where appropriate.

Keywords: pediatric, traumatic brain injury, premorbid functioning, academic, behaviour, outcome

Traumatic brain injury (TBI) refers to a "traumatic insult to the brain, capable of producing brain damage and associated with functional impairment" (Anderson, Northam, Hendy, & Wrennall, 2001, p. 129). TBI is often used synonymously with the word head injury (HI), the latter being a nonspecific term usually referring to external scalp and skull injuries, which may not necessarily imply an injury to the brain (Bruns & Hauser, 2003).

TBI is a leading cause of death and acquired childhood disability. Survivors of TBI experience impairments in a range of neurocognitive and behavioural domains (Anderson et al., 2001; Babikian & Asarnow, 2009; van't Hooft, 2010). These impairments may have adverse effects in terms of academic and behavioural outcomes. Changes in both academic performance and behaviour can interfere with school re-entry, educational progress, and ultimately, quality of life of the injured child. Additionally, consequences of TBI affect both the child's family and those within the child's social environment (Anderson & Yeates, 2010; Donders, 1994; Keenan & Bratton, 2006; Lalloo & van As, 2004). These neurocognitive and behavioural impairments, and their effects on a range of outcomes, are particularly evident in children who have sustained a severe TBI (Anderson et al., 2001; Babikian & Asarnow, 2009; Taylor, 2010).

While children with severe TBI do show recovery over time following the injury, they nevertheless seem to lag behind their peers in many neurocognitive domains and are therefore at risk of not achieving developmentally appropriate gains. In addition, subsets of children with severe TBI appear to show significant and persistent neurocognitive impairments, while other children with severe TBI appear to recover remarkably well (Abelson-Mitchell, 2007; Babikian & Asarnow, 2009; Fay et al., 2009). It may be that these differences in outcome reflect the heterogeneity of injuries, as well as other injury-related factors, or the influence on recovery by a range of social, developmental, or environmental factors, for example, premorbid ability and family environment. In addition, the differences in methodological design in studies looking at the impairments experienced after TBI may further account for the variation in outcome (Babikian & Asarnow, 2009; Taylor, 2010). It is this diverse range of outcomes in children who have sustained severe TBIs that highlights the importance of continuing research into the nature of severe TBI and its outcomes, as well as the factors that influence degree of recovery in this population.

Classification and pathophysiology of TBI

TBI occurs when the brain is damaged mildly, moderately or severely, due to mechanical forces acting on the skull which are transmitted to the brain (Rao & Lyketsos, 2000). This mechanism of impact allows one to classify TBI into two main classes, namely open and closed TBI. An open TBI occurs due to the mechanical forces resulting from an object, such as a bullet or knife, penetrating the skull; while a closed TBI occurs due to the brain undergoing rapid acceleration or deceleration (Anderson et al., 2001; Zillmer, Spiers, & Culbertson, 2008). The current study focuses on closed TBI, therefore this review will be limited to discussion on this type of TBI.

Closed TBI. Closed TBIs account for the majority of pediatric TBIs (pTBIs; Anderson et al., 2001; Zillmer et al., 2008). In closed TBI, the brain is shaken within the skull cavity due to acceleration and/or deceleration forces acting on the brain. Deceleration forces experienced due to MVAs are the most frequent cause of closed TBI. While focal injuries do occur, diffuse injuries are more common in closed TBIs. Diffuse injury occurs when axons and blood vessels are sheared, stretched and torn due to forces causing the brain to move within the skull cavity. Diffuse axonal injury frequently occurs due to acceleration/deceleration forces, which result in the brain impacting against the surface texture of the tentorial plates (Rao & Lyketsos, 2000; Yeates, 2010; Zillmer et al., 2008). Damage can lead to degeneration of the axon and other cellular-level changes, which may affect frontal sub-systems resulting in associated cognitive deficits (Bamdad, Ryan, & Warden, 2003; Yeates, 2010; Zillmer et al., 2008). It is the frontal areas of the brain that are most likely to be injured during TBI due to its anatomical-vulnerability.

The pathophysiology of TBI, which is said to begin at the time of the impact but continues over a period of time, can be classified according to two categories of injuries: primary and secondary injuries (Yeates, 2010).

Primary injuries. Primary injuries are those that occur at the time of insult and as a direct result of the applied mechanical forces, such as acceleration (including both translational and rotational) and deceleration forces, causing trauma to the brain (Anderson et al, 2001; Naidoo, 2013; Yeates, 2010). Trauma is caused through mechanical injury to the brain tissue and through disruption of the brain vasculature (Naidoo, 2013). Primary injuries may include skull fractures, lacerations, contusions, and diffuse axonal injury (DAI). It is, however, not only the initial injuries that cause damage, but also the pathophysiological events occurring thereafter that play a role in damage sustained in a TBI (Figaji, 2010). Primary injuries initiate a cascade of physiological and biomolecular mechanisms, that further contributes to injury and which leads to further serious potential consequences (Anderson et al, 2001; Naidoo, 2013; Yeates, 2010).

Secondary injuries. Secondary injuries are those that occur after the primary insult and further contribute to the damage incurred by the brain as a result of primary injuries.

Secondary injuries may arise due to the vulnerability of the injured brain or may be pathophysiological events brought about by primary injuries. Secondary injuries include hypotension, hypoxia, brain swelling, and increased intracranial pressure, as well as mass lesions such as haematomas resulting from vascular disruption (Anderson et al, 2001; Figaji, 2010; Rao & Lyketsos, 2000; Yeates, 2010).

TBI can therefore potentially result in considerable damage to the brain structures, thereby disrupting function, leading to a variety of resulting effects. It is therefore not surprising that TBI is an important contributor to mortality and morbidity among children and adults, globally (Naidoo, 2013). The various epidemiological attributes of TBI will be discussed below.

Epidemiology of TBI

Incidence. TBI is a worldwide public health problem, with an annual estimated 10 million people requiring hospitalization or dying as a result (Hyder, Wunderlich, Puvanachandra, Gururaj, & Kobusingye, 2007; Langlois, Rutland-Brown, & Wald, 2006). In high-income countries (HICs), the general incidence of TBI is about 200 per 100 000 of the population (Bruns & Hauser, 2003). Faul, Xu, Wald, and Coronado (2010) reported that in the United States (US), almost half a million children under 14 years of age sustained TBIs annually, between 2003 and 2006. Higher incidence rates are usually seen in low- and middle-income countries (LAMICs), for example, India and South Africa, with a large proportion of TBIs resulting from traffic-related accidents (Corrigan, Selassie, & Orman, 2010). The accident rates in LAMICs exceed that of HICs, and continue to increase as the volume and density of traffic increases (Tabish, Lone, Afzal, & Salam, 2006). Not only is South Africa, in particular, regarded as having one of the highest rates of MVAs in the world, but the country's exceptionally high number of road traffic injuries is double that of the global rate (Levin, 2004; Norman, Matzopoulos, Groenewald, & Bradshaw, 2007).

Incidence of TBI in the South African context. Very little literature exists on epidemiological statistics for the South African population, particularly for children, and there is no nationwide TBI database (De Villiers, Jacobs, Parry, & Botha, 1984; Naidoo, 2013; Levin, 2004; Schrieff, Thomas, Dollman, Rohlwink, & Figaji, 2013). An additional problem exists in South African literature, as in international literature, that studies reporting incidence rates often do not differentiate between TBI and HI in their samples (Abelson-Mitchell, 2007; Bruns & Hauser, 2003; Hyder et al., 2007). This section of the review, nevertheless, includes statistics referring to head-injured populations primarily due to the scarcity of available South African epidemiological literature on TBIs and the population of interest for this research.

In their Johannesburg-based study, Nell and Brown (1990) reported an incidence of TBI of 360 per 100 000 population, in individuals between the ages of 15 and 24 years. Besides this study, there are no other studies reporting on the incidence rate of TBI in children/adults with TBI. A profile study of the 37,610 under-13 year old head-injured children that attended Red Cross War Memorial Children's Hospital (RXH), during the period 1991 – 2001, documented a frequency of 8.6% (3,234) for closed TBIs and 0.3% (113) for open TBIs, with respect to the type of injury (Lalloo & van As, 2004). Schrieff et al. (2013) reported in their epidemiological study, that 137 children were admitted to RXH during 2006 – 2011 and received intracranial monitoring for severe TBI. A frequency of 94.02% (110) for closed TBIs and 5.13% (6) for open TBIs was reported for the 117 (%) survivors of the 137 admitted, with this datum missing for one child. There is no other more recently published literature with regards to incidence rates on TBI in South Africa.

Mechanisms of injury. There are various mechanisms of injury that are associated with TBI in children. The most common causes are motor vehicle accidents (MVAs), in which the child is either a passenger or pedestrian, and falls (Anderson et al., 2001; Goldstein & Levin, 1987; Yeates, 2010). Other mechanisms of injury include child abuse and various forms of violence such as assaults and gun shot wounds (Anderson et al., 2001: Keenan & Bratton, 2006).

Mechanisms of injury of pTBI in the South African context. In South Africa, the most common causes of TBI reported for children admitted to hospital are traffic-related accidents and falls (De Villiers et al., 1984; Lalloo & van As, 2004; Levin, 2004; Semple, Bass, & Peter, 1998; Schrieff et al. 2013). Semple et al. (1998) reported in their Cape Town study of severely head-injured children that 83% of the sample had been involved in pedestrian vehicle accidents (PVA). Thereafter, both Lalloo and van As' (2004) profile study of head-injured children, and Miller and Stander's (2009) unpublished pTBI profile study, found falls to be the most common cause of head injury of varying severity, followed by traffic-related accidents. Among the traffic-related injuries, a greater majority of head injuries resulted from MVAs where the child was injured as a pedestrian. In South Africa, there is little regard for, and enforcement of, laws related to traffic safety as well as a lack of education regarding road safety (Levin, 2004; Norman et al., 2007). Road safety continues to remain a challenge faced by government in South Africa, and by society (du Toit, 2013). In particular, a large majority of children travel in minibus taxis in which there are no forms of child safety within the vehicle (Levin, 2004). A sample of children with severe TBI admitted to RXH had mechanisms of injury consistent with the above literature (Schrieff et al., 2013). MVAs (including both motor and pedestrian related accidents) accounted for 54.74% of the injuries, while falls accounted for 5.11%.

The incidence and aetiology for pTBI is said to vary according to several demographic factors including age and sex, as well as psychosocial context (Anderson et al., 2001, Yeates, 2010).

Epidemiological trends in pTBI.

Age. Various epidemiological trends are seen with respect to age, both internationally and in South Africa (De Villiers et al., 1984; Goldstein & Levin, 1984; Keenan & Bratton, 2006). With regards to South African studies, Semple et al. (1998) reported that severe head injury became more common with increased age in their sample of under 14-year olds admitted to RXH between 1990-1993, with the majority of the sample being older children of school-going age. Contrary to this, Miller and Stander (2009) reported in their two-year unpublished demographic profile of pTBI admissions to RXH during 2005 and 2007-2008, that incidence decreased as age increased, with the 9-12 year age group having the lowest incidence, compared to younger age groups. This study, however, included data for children across groups: mild, moderate, and severe, as indicated by the Glasgow Coma Scale (GCS) score. Schrieff et al. (2013) reported peak admissions by age, for children with severe TBI to RXH between 2006-2001, as occurring at 4, 6, 7 and 10 years.

Age versus mechanism of injury. Literature from the U.S. indicates that TBIs in infants are likely to occur due to falls or child abuse. Children younger than three years have the greatest incidence of TBI (Anderson et al., 2001; Keenan & Bratton, 2006). Young children of school-going age are likely to be injured due to falls or traffic-related accidents, which may be due to the fact that they are becoming more independent and their awareness of road safety is limited (Anderson et al., 2001; Keenan & Bratton, 2006; Semple et al., 1998; Yeates, 2010). As children become older and reach adolescence, more TBIs are seen that relate to sporting activities and traffic-related accidents, particularly as pedestrians (Anderson et al., 2001; Keenan & Bratton, 2006, Yeates, 2010). De Villiers et al. (1984) reported that in their Cape Town study of children under 14 years of age that aetiology differed with age, with transport-related accidents overtaking falls as the main cause of head injury as age increased. Here, head injury referred to an injury that was severe enough to lead to hospital admission.

Sex. Trends in incidence and mechanism of injury are also are seen with regards to sex. According to international literature, the risk of experiencing a TBI differs between males and females, with males having a higher risk (Anderson et al., 2001; Bruns & Hauser, 2003; Klonoff, 1971). Incidence data from the U.S. showed that males had an overall 1.4 times greater rate of TBI compared to females, across all age groups (Faul et al., 2010). Peak incidence rates among males and females appear to differ according to age, with males having an increase in incidence as they get older, and females having a declining incidence until around 15 years of age (Goldstein & Levin, 1984).

South African studies have found similar trends with regards to sex, with studies reporting a slightly greater proportion of males than females in their samples (Laloo & van As, 2004; Miller & Stander, 2009; Schrieff et al., 2013; Semple et al., 1998).

Psychosocial factors.

Environmental influences. Important contributing risk factors for pTBI resulting from PVAs include adverse environmental conditions, for example, a lack of transport accessibility (and therefore an increased likelihood of being a pedestrian as opposed to a passenger) and limited play areas (Demellweek, Baldwin, Appleton & Al-Kharusi, 2002; Hensen, Hadfield, &Cooper, 1999). PTBI also appears to occur more frequently at times when children are involved in play and other recreational activities, such as afternoons, during holidays, and on weekends (Anderson et al., 2001). An increased frequency during these times may partly be due to lack of suitable recreational facilities and parental care or supervision, which is often associated with low socioeconomic status (SES). Low SES areas also often lack adequate street lighting and have high traffic density, which further increases the risk for MVAs (De Villiers et al., 1984; Semple, Bass, & Peter, 1998). In South Africa, SES is likely to be one of the most influential contributing etiological factors to TBI. Indeed, there exists a high incidence of TBI in low SES circumstances (Levin, 2004; Schrieff et al., 2013).

Premorbid functioning. Pre-existing behavioural and learning problems can influence the likelihood of an accident, and therefore a TBI, occurring (Demellweek et al., 2002). For example, children who exhibit behavioural characteristics such as hyperactivity and aggression, as well as risk-taking behaviour associated with impulsivity, may indulge in thrill-seeking activities such as climbing tall trees or jumping off obstacles (Bijur, Stewart-Brown, & Butler, 1986; Demellweek et al., 2002). Likewise, symptoms such as distractibility, poor judgment, and an inability to plan and anticipate situations may lead to increased vulnerability to accidents, due to reduced safety awareness (Haas, Cope, & Hall, 1987). Kronenfeld and Glik (1995) report that behaviour is a major determining factor in pedestrian-related injuries, for example, where a child may impulsively run into the street. Therefore, researchers often state that individuals (including children) who sustain a TBI are not a random sample (Anderson & Yeates, 2010; Taylor & Alden, 1997).

Given high prevalence of TBI, and the number of factors that may lead to and increase the risk of an accident that can result in TBI, it is not surprising that there are a vast range of outcomes following a TBI.

Consequences of pTBI

Early findings. TBI in children and its consequences is a longstanding problem that has been extensively covered in literature. Early studies focused on identifying and evaluating the long-term effects of pTBI. Researchers then began looking at factors predicting outcome and subsequent recovery patterns (Anderson et al., 2001). Key early studies included those by Klonoff and colleagues (see, e.g., Klonoff, 1971; Klonoff, Low, & Clark, 1977). These researchers looked at the effects of TBI through investigating a sample of head-injured children in Vancouver at different time periods, findings of these early studies included memory, learning, and emotional problems. Studies by Rutter and colleagues extended the knowledge base concerning the effects of pTBI (see, e.g., Brown, Chadwick, Shaffer, Rutter, & Traub, 1981; Chadwick, Rutter, Brown, Shaffer, & Traub, 1981; Rutter, Chadwick, Shaffer, & Brown, 1980). Their findings show that there are frequent and persistent disabilities associated with severe TBI in children, a dose-response relationship between cognitive outcome and injury severity, as well as a significant increase in post-injury onset psychiatric problems following severe TBI.

Since these early studies, there have been numerous other studies investigating outcome in different domains following pTBI. These studies have reported a wide range of neurocognitive and behavioural sequelae that have implications for educational success (Anderson et al., 2001; Babikian & Asarnow, 2009; van't Hooft, 2010). The following section of the review highlights findings from the literature in terms of the most commonly reported cognitive impairments, that being in the domains of intellectual functioning, attention, executive function, memory and learning, and language.

Cognitive outcomes. Literature on outcomes documents a broad range of cognitive sequelae following pTBI. Cognitive sequelae include deficits in general intellectual functioning, attention, executive function, memory and learning, and language skills (Anderson et al., 2001; Anderson & Yeates, 2010; Babikian & Asarnow, 2009; Mayfield & Homack, 2005; Rao & Lyketsos, 2000; Yeates, 2010).

General intellectual functioning. Non-verbal IQ may be more vulnerable to TBI than verbal IQ, however, impairment occurs in both of these measures of general intellectual functioning (Anderson, Morse, Catroppa, Haritou, & Rosenfeld, 2004; Yeates, 2010). Significant recovery is seen in intellectual functioning, although IQ scores may continue to remain lower than at premorbid levels (Babikian & Asarnow, 2009; Yeates, 2010).

Attention. Literature has documented impairments in specific domains of attention following TBI, however, it appears that outcome often involves more global deficits in attention. In addition, these deficits may persist over an extended period of time, particularly with increased severity of TBI (Anderson et al., 2001; Yeates, 2010; Yeates et al., 2005). In an overview of pTBI literature, Ginstfeldt and Emanuelson (2010) found deficits in sustained and divided attention to persist over time. New learning within a classroom setting may be affected in children who have attention deficits (Mayfield & Homack, 2005).

Executive function. Executive functions are those that play a role in the coordination involved in carrying out goal-directed behaviour. These abilities play an important role in academic achievement, as well as behavioural and adaptive functioning. Working memory and inhibition in particular are important in the process of learning (Anderson, Anderson, Northam, Jacobs, & Mikiewiez, 2002; St Clair-Thompson & Gathercole, 2006). Executive function deficits may include impairments in working memory and planning, and as with other cognitive deficits, are associated with the nature and location of the pathology (Anderson et al., 2002; Bamdad et al., 2003; Levin & Hanten, 2005; Yeates, 2010; Yeates et al., 2005). Deficits occur when damage is sustained to the frontal areas in which the neural circuits mediating execution functioning are located (Anderson et al., 2002). For children who have sustained a severe TBI, there may be significant impairments in executive functioning and related domains, particularly in skills related to processing speed, attention, problem solving, and fluency (Babikian & Asarnow, 2009). Impairments in executive function may hinder the child’s ability to function in a classroom setting, both academically and socially. Problems with inhibition of behaviours may lead to a hostile environment in the classroom, particularly when these problems are expressed through agitation or inappropriate comments or actions. Lack of impulse control can result in unwanted behaviours such insulting comments, conduct problems, and getting out of one’s chair, which can be disruptive to other learners in the classroom (Mayfield & Homack, 2005).

Memory and learning. Memory difficulties, such as problems with encoding, storage and retrieval, present following TBI. Verbal memory appears to be particularly impaired in children who have sustained severe TBIs. Memory deficits can interfere with the processes of new learning, and skill and knowledge acquisition. Therefore there are substantial implications for school performance (in learning and retaining educational material) and everyday tasks (such as remembering to complete assignments or take medication) when memory processes are impaired, which may lead to decreased rate of age-appropriate skill development (Anderson et al., 2001; Babikian & Asarnow, 2009; Catroppa & Anderson, 2002; Farmer, Clippard, Luehr-Wiemann, Wright, & Owings, 1996; Kinsella et al., 1997; Lajiness-O'Neill, Erdodi, & Bigler, 2010; Reid & Kelly, 1993). Kinsella et al. (1997) found verbal learning to be a predictor of educational progress, in that children with learning deficits, two-years post-TBI were more likely to require remedial or special needs education.

Language. Deficits in pragmatic, receptive and expressive language skills may occur following TBI (Farmer et al., 1996; Savage et al., 2005; Yeates, 2010). Subtle persistent language difficulties are more likely in children with more significant TBIs. In particular, receptive language impairments may persist in children with severe TBI (Anderson et al., 2004; Yeates, 2010). However, recovery of certain language skills, such as those that are overlearned and automatic, often does occur (Farmer et al., 1996; Catroppa & Anderson, 2004). Decreased language performance hinders communication and interaction, and is particularly evident when the child encounters stressful situations. Communication skills are an important contributor to school success due to the language-based nature of learning and therefore deficits in pragmatic language skills may lead to academic difficulties (Savage et al., 2005; Yeates, 2010).

These cognitive deficits may account for the academic sequelae associated with TBI and may therefore interfere with a child's academic performance (Donders, 1994). Impairments in behavioural, social, and emotional functioning are also seen following TBI. These impairments emerge as a part of the above-reviewed cognitive deficits, secondary to adjustment and environmental difficulties, or as selective deficits (Anderson et al., 2001; Taylor, 2010; Yeates, 2009). Deficits stemming from the TBI therefore extend to the family and classroom environment. Outcomes relating to the family environment and academic achievement are discussed below, in addition to a review of outcomes relating to behavioural and psychiatric disturbance.

Behavioural outcomes. Persistent behavioural changes may begin to appear following the acute stages post- TBI (Fletcher, Ewing-Cobbs, Miner, Levin, & Eisenberg, 1990; Taylor, 2010). Patterns of behavioural problems following TBI may vary from one child to another and often include impulsivity, agitation, hyperactivity, distractibility, and aggression (Fletcher et al., 1990; Kinsella, Ong, & Murtagh, 1999; Mayfield & Homack, 2005; Savage, Depompei, Tyler, & Lash, 2005; Schachar, Levin, Max, & Purvis, 2004; Taylor, 2010). Impairments in adaptive and behavioural functioning, for example, everyday social and functional skills, have also been reported (Anderson et al., 2001; Clark, Prior, & Kinsella, 2002; Fletcher et al., 1990). Fay et al. (2009) report that behavioural problems, particularly in severe TBI children, are likely to deteriorate over time.

Behavioural problems not only interfere with the functioning and educational progress of the injured child, but can also be disruptive to others in the home, in the community, or in the classroom, particularly when these problem behaviours persist over time (Savage et al., 2005; Yeates & Taylor, 2006). Behavioural impairments can also negatively impact school performance, in both developing current skills and acquiring new skills (Babikian & Asarnow, 2009; Keenan & Bratton, 2006). Children with behavioural impairments appear to perform more poorly academically than their peers (Hawley, 2004). These behavioural impairments may occur as a direct result of damage to the brain and its associated cognitive deficits. In particular, damage to the frontal areas may lead to impaired executive functioning including the inability to initiate tasks, self-monitor behaviour, and inhibit responses. Parental stress and expectations, as well and problems relating to adjusting to deficits, may also indirectly lead to behavioural impairments (Bamdad et al., 2003; Donders & Strom, 2000; Max et al., 1999; Mayfield & Homack, 2005; Taylor et al., 2001). Difficulties in terms of regulation of behaviour and emotion frequently result as a consequence of new-onset behavioural problems following a TBI and are associated with increased risk for adverse psychiatric outcomes (Taylor, 2010).

Psychiatric outcomes. Behavioural and emotional self-regulation impairments may increase the risk of developing psychiatric symptoms and disorders, both internalizing and externalizing in nature, such as anxiety disorders and conduct disorders, respectively. Other psychiatric symptoms and disorders include mood disorders, post-traumatic stress disorder (PTSD), Secondary (onset) Attention Deficit/ Hyperactivity Disorder (SADHD), oppositional defiant disorder and changes in personality (Rao & Lyketsos, 2000; Taylor, 2010; Yeates, 2010). These disturbances may in turn cause impairment or distress in different areas of functioning, for example, social or adaptive functioning (Max, Robertson, & Lansing, 2001). Max et al. (2005) found SADHD to be a common consequence after pTBI, with children exhibiting hyperactive, impulsive, and inattentive symptoms. These researchers also found that SADHD occurred comorbidly with externalising disorders, for example post-TBI personality change.

Personality change in children after TBI may manifest as a deviation, of at least a year in duration, from the normal development of personality patterns. Children with personality change show difficulties with learning and this may be influenced by symptoms such as poor insight into their condition (Max et al., 2001). Max et al. (2000) found children with severe TBI, who show persistent personality change, to have poorer adaptive functioning, in terms of socialization and skills of daily living.

Symptoms of psychiatric disturbance following TBI may result from the primary and secondary injuries sustained to the brain. However, they may also arise due to problems associated with adjusting to the deficits resulting from the injury. For example, aggressive outbursts stem from frustration experienced related to poor communication skills (Anderson et al., 2001; Savage et al., 2005).

Academic outcomes. Academic performance depends on various basic cognitive skills, for example, memory, attention, and learning. Therefore, the above-mentioned post-TBI cognitive deficits may have an adverse effect on academic performance (Arroyos-Jurado, Paulsen, Merrell, Lindgren, & Max, 2000; Ewing-Cobbs, Fletcher, Levin, Iovino, & Miner, 1998; Hawley, 2004). Research has documented evidence of decreased academic performance following TBI. Specifically, children with severe TBI obtained lower scores on academic achievement tests compared to children with TBI of lesser severity and controls. This discrepancy in scores existed despite the children with severe TBI showing some initial recovery in academic performance (Ewing-Cobbs et al., 1998; Taylor, 2010). TBI affects all core academic skills such as reading, writing, mathematics, and spelling; however, individuals may show impairment in some academic skills more than others (Taylor, 2010). Children that exhibit poor academic functioning may need to be placed in special education programmes (Savage et al., 2005). Other factors that can result in poor academic outcome may include the effects of behavioural impairments following TBI, as well as the often long-absence from school during the immediate stages after the injury resulting in less opportunity for learning (Babikian & Asarnow, 2009; Ewing-Cobbs et al., 1998).

Measures of academic outcome can provide an indication of the cognitive deficits experienced by children with TBI in the real-world setting of a classroom environment (Savage et al., 2005). Knowledge of cognitive deficits experienced within a classroom environment is especially important because teachers may assume that children with TBI are fully recovered from their injuries when no obvious physical deficits are seen. Hawley (2004) reports that some teachers may be unaware that learners in their classroom may have sustained a TBI, particularly when the injury occurred more than a year prior to the child entering that class. This unawareness by teachers may result in the lack of academic assistance and modifications in the classroom required by children with TBI (Mayfield & Homack, 2005).

Family stress. The TBI event, particularly when severe in nature, and the child's subsequent return home and to school can lead to considerable stress and burden for the family, who must then attempt to provide the necessary care and adjust to the particular needs and limitations of their injured child (Anderson et al., 2001; Prigatano & Gray, 2007). The cognitive, behavioural, academic, and psychosocial consequences following the TBI lead to parental distress (Anderson et al., 2001; Prigatano & Gray, 2007; Taylor et al., 2001). A U.S. study on school-going age children who had sustained TBIs found that for parents whose children had sustained a severe TBI, subsequent academic difficulties were of particular concern and added to the distress that they were experiencing (Prigatano & Gray, 2007). In addition, concerns regarding the child's future, reduced social skills, reduced peer interaction, behavioural problems, and emotional well-being have been reported in the literature and such concerns further contribute to stress in the family (Anderson et al., 2001; Prigatano & Gray, 2007; Taylor et al., 2001).

As described above, children with TBI experience a range of deficits, with implications for education in particular. In addition, the effects of the injury have consequences for people within the child's environment. Patterns of impairment vary among children who have sustained TBIs and may be due to the influence of multiple factors that play a role in the child's recovery (Donders & Strom, 2000). Several of these factors, both injury-related and non-injury related, are described below.

Factors affecting outcome after pTBI

Injury-related factors.

Severity of injury. Severity of the TBI sustained is frequently reported as a significant predictor of outcome (see, e.g., Anderson, Catroppa, Morse, Haritou, & Rosenfeld, 2009; Anderson, Morse, Catroppa, Haritou, & Rosenfeld, 2004; Anderson et al., 1997; Anderson et al., 2001; Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2008; Arroyos-Jurado et al., 2000; Yeates, 2010). The Glasgow Coma Scale (GCS) has traditionally been used to classify severity of a TBI as mild, moderate or severe. Severe injury corresponds to a score of 8 or below on the GCS, moderate injury from 9 to 12, and mild injury a score of 13 or higher (Zillmer et al., 2008). Internationally, the proportion of injuries per category of severity is about 80% mild, 10% moderate, and 10% severe (Bruns & Hauser, 2003; Zillmer et al., 2008).

In general, a dose-response relationship exists between injury severity and outcome, with greater severity associated with worse outcomes in neurocognitive domains and behavioural functioning (Anderson et al., 1997; Anderson et al., 2009; Chadwick et al., 1981; Fay et al., 2009; Yeates, 2010). A greater risk of long-term impairments and slower rate of recovery has been demonstrated in children with more severe TBIs (Anderson et al., 2009; Anderson et al., 2004). Children who have sustained a severe TBI are more likely to require special educational services than those with a less significant TBI (Kinsella, 1997).

Neurosurgical intervention. In the acute stages following a severe TBI, intervention in the form of prevention and management of secondary injuries, such as raised intracranial pressure (ICP) and hypoxia, is of outmost importance. Not only does this help reduce mortality, it also improves the outcome and quality of life of survivors (Figaji, 2010; Giza, Mink, & Madikians, 2007; Jiang, Gao, Li, Yu, & Zhu, 2002, Ylvisake et al., 2005). Management of patients should focus on monitoring and maintaining appropriate levels of medical variables such as ICP, cerebral perfusion pressure (CPP), and brain tissue oxygen tension ($PbtO_2$). Electroencephalograph (EEG) monitoring of the presence of subclinical seizures and autoregulation monitoring of cerebral blow flow (CBF) are important as seizures and changes in CBF can lead to changes in ICP, CPP, and $PbtO_2$. Controlling the variables of ICP, CPP, and $PbtO_2$ may also include interventions such as osmotherapy or surgery such as a decompressive craniectomy (Figaji, 2010).

Maintaining appropriate higher levels of CPP and $PbtO_2$, as well as lower ICP, are associated with better outcome (Figaji, 2010; Jiang et al., 2002; Michaud, Rivara, Grady, & Reay, 1992; Woodward et al., 1999). In clinical studies of neurosurgical intervention, outcome is usually in reference to a gross measure of neurological outcome indicating mortality and degree of survival (e.g., severe disability or no disability). For example, the Glasgow Outcome Scale (GOS) is often used as a measure of outcome (Jiang et al., 2002).

In a study by Figaji et al. (2009), $PbtO_2$ was found to be a stronger predictor of clinical outcome than ICP and CPP, with an independent association between low $PbtO_2$ and poor outcome. Outcome was defined in terms of whether it was favourable (such as disability or no disability) or unfavourable (such as death) and measured according to the GOS, as well as the Pediatric Cerebral Performance Category Scale (PCPCS; Figaji et al., 2009).

Literature on outcome in terms of neuropsychological domains with respect to neurosurgical management variables is lacking. They are, however, a few studies in which these relationships are described. For example, Woodward et al. (1999) found critical care management, particularly maintaining adequate CPP, to significantly influence neurobehavioural development in a sample of young children to adolescents. They demonstrated CPP to be a significant predictor of neurobehavioural outcome, with higher CPP associated with better adaptive functioning. Slawik et al. (2009) reported persisting impairments in executive functioning and attention in a group of children, with a mean age of 11.9 years, who had sustained a severe TBI and had required intensive ICP reduction management. Meixensberger et al. (2004) reported poorer neuropsychological outcome, particularly in terms of intelligence and memory, in their adult sample with low levels of brain tissue oxygenation. An unpublished study by Schrieff (2013) documented that cognitive outcomes were adversely affected when levels of $PbtO_2$ reached a threshold commensurate with an hypoxic episode. However, children who experienced hypoxic episodes did not appear to differ on measures of behavioural functioning, compared to those who did not experience hypoxic episodes.

Time since injury. The most significant recovery from TBI takes place during the first year after the injury. Thereafter, recovery over time slows down and begins to stabilise (Jaffe, Polissar, Fay, & Liao, 1995; Taylor et al., 2002; Taylor, 2010). In their meta-analysis, Babikian and Asarnow (2009) looked at the magnitude of recovery over time after pTBI on various measures of neurocognitive outcome. For children who have sustained a severe TBI, rapid recovery during the first year is most evident for previously learned skills, however, these children later experience persistent residual deficits. Children with severe injuries also seemed to lag behind control groups over time and may not have made developmental gains, in terms of skill acquisition. The injury and its resulting effects may be responsible for this lag in developmental gains. Taylor (2010) noted that while recovery in cognitive domains, as well as an improvement in neurobehavioural sequelae is evident across various levels of injury severity as time goes on, this is more incomplete in children with more severe TBIs. This can be seen in the recovery trajectory, which Anderson et al. (2009) illustrate to have a strong and constant severity effect. Anderson et al. (2009) also reported, however, that after a period of five years, the recovery tended to stabilize and it was age-related factors that became less significant over time. Age at injury, however, is n contrast reported by Anderson, Spencer-Smith and Wood (2011) to become more apparent over time following the TBI.

Age at injury. According to literature, rate of recovery and outcome after TBI may be influenced by the age at which the injury occurred, particularly since the young brain is still in the process of development (Anderson et al., 2009; Anderson, Catroppa, Morse, Haritou,

& Rosenfeld, 2005; Anderson, Godfrey, Rosenfeld, & Catroppa, 2012; Dennis, Yeates, Taylor, & Fletcher, 2007; Taylor & Alden, 1997; Yeates, 2009).

Being of a younger age is a risk factor for more adverse persistent cognitive outcomes and so children with TBI sustained at a young age require extra support with respect to their impairments (Anderson et al., 2009; Taylor, 2010). However, despite injury occurring at a young age, varying outcomes are observed with normal or even better cognitive and academic outcome still possible (Taylor & Alden, 1997). In addition, the relationship between outcome and age at injury is not as certain when TBI occurs during the school-going and adolescents years (Taylor & Alden, 1997; Yeates, 2009).

The role of age at injury in predicting outcome following pTBI is characterised by a debate in literature regarding the theories of plasticity versus early vulnerability.

Plasticity versus early vulnerability. Plasticity of the brain can be viewed in terms structure and function. Structural plasticity refers to the changes that occur in the microstructure and macrostructure of the brain, whereas functional plasticity refers to cognitive and behavioural compensation that occurs following TBI (Dennis, 2000). A child's brain differs from an adult brain in that it is still developing. Therefore, it is more plastic, allowing for these structural changes and compensation to take place. In light of the plasticity of a child's brain, early literature supporting the plasticity theory suggested that a TBI at an earlier age would lead to better outcome, traditionally referred to as the 'Kennard Principle' (Giza & Prins, 2006). Better outcomes at a younger age at insult are therefore possible due to the plastic nature of the brain which allows for better functional recovery, through greater ability for reorganization and compensation, thereby implying that plasticity is advantageous (Giza & Prins, 2006; van't Hooft, 2010). Discrepancies in literature regarding plasticity for specific cognitive skills for example, inconsistencies in recovery, are fitting, according to Anderson et al. (2011). The discrepancies are fitting because Anderson et al. (2011) suggest that focal, lateralized regions of the brain have greater potential for reorganization and compensation compared to more diffuse neural systems.

Other studies have shown that disruptions to the process of cognitive development are detrimental in terms of outcome in neurocognitive and behavioural domains (Anderson et al., 2001). Theorists in support of a model of early vulnerability argue that the younger children are, the fewer skills they possess, and so TBI can affect the acquisition of new skills and knowledge (Anderson et al., 2005). Following from this, disruption of development early in life that is, injury at a time when the function is not yet developed, may lead to the injured child 'growing into their deficits'. 'Growing into deficits' is where deficits only begin to emerge at the time when the affected skills or abilities would have normally developed, or in response to increased demands from the environment (Anderson, et al., 2011; Giza & Prins, 2006). It is also worth noting that while literature supporting early vulnerability of the child's brain suggests that new deficits emerge over time, this emergence of deficits is not an indefinite process. Anderson et al. (2009) demonstrated that after 30 months following TBI in younger children, the brain begins to stabilize and is able to acquire new skills.

There is generally greater support for the model of early vulnerability, with studies showing that younger age at injury is associated with poorer outcomes, and inversely, that older age at injury is associated with better outcomes (Anderson et al., 2005; Anderson et al., 2009; Anderson et al., 1997; Anderson et al., 2004; Prigatano & Gray, 2008; Spencer-Smith & Anderson, 2010).

Owing to the fact that the child's brain is in a dynamic state, where there is interaction with the environment and adaptive responses to experience and learning, Anderson at al. (2011) proposed that neither the theories of plasticity nor early vulnerability can independently account for the variation in TBI outcome. Instead, plasticity and early vulnerability are extremes of recovery located along a continuum. Where a child's recovery falls along this continuum is influenced by various factors, such as those described in the above review.

To further add to this debate, plasticity and vulnerability can be better understood as explanations of functional outcome, where outcome following TBI depends on a variety of factors (van't Hooft, 2010). The role that various factors play in moderating functional outcome may be better illustrated by Dennis et al.'s (2007) model of individual difference that conceptualizes the relationships between the factors that play a role in functional outcome. Here, they propose that functional plasticity ultimately determines functional outcome. However, functional plasticity, and therefore the response of the brain to injury, is mediated by reserve capacities in the brain. These reserve capacities differ between individuals due to factors such as pre-and post-injury functioning, SES, family environment and brain factors such as volume and connectivity, and are themselves mediated by factors such as age at injury and time since injury. Reserve capacities decrease following TBI and it is the individual differences that play a role in determining at what point deficits in functioning are expressed (Spencer-Smith & Anderson, 2010).

Non-injury related factors.

Socioeconomic status (SES). Researchers of a number of studies have demonstrated that SES plays a role in predicting recovery after pTBI (e.g., Anderson et al., 2004; Taylor et al., 2002; Yeates et al., 2004). Fay et al. (2009) reported a consistent association between low SES and persistent functional deficits across a range of domains including outcomes on neuropsychological, behavioural, adaptive, and academic measures. Families of low SES backgrounds may have fewer resources to assist in the child's recovery, limited access to health services, limited interventions in both the home and school environment, and feel particularly burdened by the stressors resulting from the accident due to their disadvantaged circumstances (Levin, 2004; Taylor et al., 2002). Taylor et al. (2002) found that it was only children with severe TBI in their sample, who were from disadvantaged backgrounds that demonstrated long-term deterioration in academic performance, relative to children with less severe TBIs or orthopaedic injuries. The rate of short-term progress in socialization skills, as well as an increase in behavioural difficulties, was also associated with disadvantaged backgrounds. Taylor et al. (2002) suggested that disadvantaged family circumstances might have worsened the effects of TBI on behavioural functioning and skill development. This exacerbation of the effects of TBI may be due to having fewer resources or decreased input from parents in the midst of increased stressors. Similarly, disadvantaged environments may fail to promote recovery from TBI or may interfere with post-injury development, when there are few resources and lack of access to care and rehabilitation (Levin, 2004; Taylor, 2010).

On the other hand, the relationship between disadvantaged environments and post-injury recovery and development leads one to believe that an advantaged environment may provide more enrichment and opportunity for adjustment to deficits and learning of compensatory skills (Taylor et al., 2002). The SES circumstances of a family may therefore influence how the family responds to, and deals with, the consequences of TBI, and so it is linked to the burden and stress experienced within the home.

Family environment. The family's response forms part of a child's recovery from TBI and therefore the family environment plays an important role in predicting the child's outcome (Anderson et al., 2001; Taylor et al., 2001; Yeates, 2010; Yeates et al., 1997). Families with a greater degree of family dysfunction have reported greater behavioural problems with their child (Kinsella, Ong, Murtagh, Prior, & Sawyer, 1999). There may be less structure and consistency in dysfunctional families, with behaviour and functioning expectations that are not very clear to the child (Chapman, et al., 2010). Taylor et al. (2001) demonstrated that for behavioural outcomes, there exists a bidirectional influence between the child and the family. This is where the child, due to the TBI and associated deficits, influences the family environment, while the family environment also influences the child's outcome. The injury itself may lead to parental distress; however, over time, the resulting behavioural sequelae exhibited by the child can also become a contributing factor to parental stress and burden. The family environment fostered by parental stress and burden may then influence and further contribute to the child's behavioural problems. In the South African context, family environment is an important factor, particularly due to increased stressors such as lack of healthcare and rehabilitation resources (Levin, 2004). A sample of South African parents was found to have an equal or greater amount of stress than that which is reported in samples from HICs (Oosthuizen, 2010).

The influence on outcome by family environment is not only negative. Factors such a social support and a cohesive family environment have been shown to be associated with favourable outcomes (Anderson et al., 2001). Gerrard-Morris et al. (2010) reported a similarity in cognitive development amongst children with and without TBI in optimal family environments. An environment that provides stimulation, opportunities to facilitate adjustment, as well as adequate and appropriate parental support is beneficial for ensuring successful outcome (Taylor et al., 2002). In addition, the education and involvement of all relevant family members during the early stages of recovery is valuable (Beaulieu, 2002).

Access to rehabilitation. Pediatric neuropsychological rehabilitation following TBI involves a multidisciplinary process that seeks to promote recovery, assist in compensating for residual deficits, and improve daily functioning by providing a stimulating environment that is suited to the particular needs of the injured child (Anderson et al., 2001; Zillmer et al., 2008). It is the setting in which neuronal reorganisation potential is maximised through providing real-world experiences (Anderson et al., 2001; Beaulieu, 2002; Zillmer et al., 2008). It is a process that may take place over an extended period of time, depending on the specific needs and recovery stage of the child (Anderson et al, 2001).

Rehabilitation following pTBI is said to be a necessary step in achieving successful outcome. As such, recovery and long-term outcome is influenced by access to these resources (Anderson et al., 2001; Beaulieu, 2002; Yeates, 2010).

Access to rehabilitation in the South African context. Despite the knowledge of outcomes following TBI and the role that neuropsychological rehabilitation plays in facilitating recovery, there are limited neuropsychological rehabilitation resources available to children with TBI in South Africa (Levin, 2004). Access to resources for rehabilitation, however, plays a role in recovery along with other factors affecting outcome.

Premorbid functioning. Premorbid functioning, such as behavioural and academic ability before the injury, may be important contributors to outcome after pTBI. Fay et al. (2009) demonstrated in their study that premorbid functioning contributed to predicting the functional outcomes. For example, declining behavioural and adaptive functioning was predicted by poorer premorbid behavioural functioning. Fay et al. (2009) concluded that more favourable premorbid functioning may lead to an improved recovery over time in children who have sustained a TBI, while significant adverse premorbid functioning may increase the likelihood of such children exhibiting persistent deficits. Premorbid functioning may therefore act as a potential vulnerability factor that predicts post-injury behavioural functioning (Anderson et al., 2001). For example, Yeates et al. (2005) found premorbid attention problems to be a moderator of long-term post-TBI attention problems, where more significant premorbid difficulties were associated with larger differences in attention problems between the TBI groups and the control group with orthopaedic injuries. Max et al. (2005) notes post-injury psychiatric disorders such as SADHD as being linked with adverse pre-injury functioning. Children with less pre-injury learned skills, such as communication and socialization, may not recover as readily as those with greater skills. Therefore, these children with fewer pre-injury skills have poorer regulation of attention, as well as impulsive and hyperactive behaviours (Max et al., 2005; Schachar et al., 2004).

A significant predictor of academic achievement, particularly in reading and spelling, is premorbid academic ability, with pre-injury academic level relating to post-injury academic scores (Arroyis-Jurado et al., 2000; Ewing-Cobbs et al., 1998; Jaffe et al., 1993). Additional cognitive deficits in children with more significant TBI have been associated with premorbid learning difficulties (Donders & Strom, 1997).

It may be that premorbid problems exacerbate the effects of TBI through increased vulnerability of the brain. Children with premorbid dysfunction may have a decreased threshold for displaying effects of the injury. Increased vulnerability may result in a lower threshold capacity for coping with, and compensating for, these deficits due to decreased available resources (Schwartz et al., 2003; Taylor, 2010; Yeates et al., 2005).

This lower threshold may be further aggravated when the age of the child comes into play, when resources such as cognitive skills, have not yet developed.

Rationale for Research

Children who have sustained severe TBI demonstrate deficits in neurocognitive domains (e.g., executive functioning, attention, memory, language) that can have adverse effects on academic and behavioural outcomes. Educational and behavioural development in children rely on intact executive function, attention and learning abilities; therefore, disruption of these abilities can have significant consequences on the academic attainment and behavioural functioning of children who have sustained TBIs (Anderson, et al., 2001; Arroyos-Jurado et al., 2006). Behavioural impairments can also negatively impact school performance, in both developing current skills and acquiring new skills (Keenan, & Bratton, 2006; Babikian & Asarnow, 2009). Changes in both academic performance and behaviour can interfere with educational progress and quality of life of the injured child. The consequences of TBI can be burdensome to the child's family and others in their environment, such as teachers and peers (Anderson & Yeates, 2010; Keenan & Bratton, 2006; Lallo & van As, 2004).

Some children who have sustained a severe TBI appear to be more affected than others. There are various factors that influence the brain's vulnerability to the effects of severe pTBI and therefore the degree and range of resulting deficits. Exploring the relationships between these factors, such as time since injury, age at injury, family environment, SES and premorbid functioning, and investigating relationships between these factors and academic and behavioural outcomes, would allow for identification of children at risk for poorer outcomes. Recognizing which children are at risk for more adverse sequelae would allow for the identification of rehabilitation needs, as well as barriers to recovery, so that appropriate intervention strategies can be put in place (Kinsella et al., 1997).

While there is a vast amount of literature on pTBI, which focuses on outcome and factors affecting outcome, little literature exists on the pTBI population specifically within a South Africa context. In addition, while there is evidence that pre-injury characteristics may increase risk for, and play a role in, TBI outcome, many studies exclude children with known adverse premorbid functioning (Dennis et al., 2007; Farmer et al., 2002). Exclusion of participants with premorbid problems from studies is often done to eliminate this pre-injury factor from confounding the assessment of TBI outcome. This exclusion may be employed, for example, so that between-group differences are more likely to reveal and isolate the effects of the brain injury alone when controlling for pre-injury factors (Taylor, 2010; Taylor & Alden, 1997). However, it is important to include children with premorbid dysfunction within a study investigating TBI outcome, as premorbid functioning plays a role in predicting outcome. Premorbid functioning does indeed act as an important predictor of academic and behavioural outcome, as evidenced in studies that included participants with known premorbid problems (Arroyis-Jurado et al., 2000; Donders & Strom, 1997; Ewing-Cobbs et al., 1998; Fay et al., 1990; Jaffe et al., 1993; Max et al., 2005; Schachar et al., 2004). In addition, Spencer-Smith and Anderson (2010) reported premorbid functioning to be a vulnerability factor that decreases the brain's reserve capacities. Brain reserve capacity mediates functional plasticity, which they suggest plays a role in functional outcome following TBI.

There are limited studies in which a large number of factors affecting outcome, and outcome variables, in the same sample of participants, are investigated. Instead, studies usually look at the influence of a specific factor on the outcomes of the sample. One of the methodological issues in TBI research is the lack of multivariate studies (Kinsella et al., 1999; Fletcher et al., 1990). Noval, Bush, Meythauler, and Canupp (2001) stress the need for multivariate TBI studies to avoid overemphasis on particular aspects of recovery from TBI, while overlooking other factors that may be more informative. There are also a dearth of published studies incorporating a variety of factors affecting outcome (such as SES, age at injury, time since injury, premorbid functioning, family environment) as well as academic and/or behavioural outcome variables in the same study generally, and no published studies of this nature specifically in a South African pediatric population.

Specific Aims and Hypotheses

The broad aim of the study was therefore to contribute to the existing pTBI literature on outcomes and factors influencing outcomes. In this study, I focused on investigating both academic and behavioural outcomes in a group of South African children of school-going age who have sustained a severe TBI. In doing so, I explored a number of factors that affect academic and behavioural outcomes, with a particular focus on premorbid functioning.

Other factors that I explored included age at injury, time since injury, family environment and SES. Although researchers in the field report on a myriad of factors that affect outcome, the factors explored in this study are the most commonly reported ones.

This study includes two parts. The first part of the study is an investigation of the factors that influence academic and behavioural outcome after TBI. The specific aim was therefore to explore the relationship between commonly reported factors that influence outcome after TBI, and academic and behavioural outcome. Because this part of the study is exploratory in nature, there are no specific hypotheses.

The second part of the study focuses specifically on the factor of premorbid functioning within the sample. The specific aim was therefore to investigate the role of premorbid functioning in academic and behavioural outcome. In this section of the study, I tested the following two specific hypotheses:

Hypothesis 1: Participants with evidence of premorbid problems will have a poorer academic outcome than those with no evidence of premorbid problems.

Hypothesis 2: Caregivers[1] will report participants with evidence of premorbid problems as having a poorer outcome than those with no evidence of premorbid problems on behavioural measures.

[1] Caregivers in this study refers to the participants' primary caregivers, including parents, grandparents, and legal guardians.

Chapter 2: Research Methodology

Research Design and Setting

The study has a cross-sectional, quantitative design.

Part 1: Investigation of the factors that influence academic and behavioural outcome after TBI. The first part of the study is exploratory in nature and investigates factors that influence academic and behavioural outcome after TBI in a group of South African school-going children with severe TBI. There are both descriptive and correlational aspects included in this part of the study.

Part 2: The specific role of premorbid functioning. The second part of the study has a specific focus on the factor of premorbid functioning and its role in academic and behavioural outcome. The design included a between-group comparison of two independent groups: one group of children had evidence of premorbid problems (Premorbid Problems group), while the other group had no evidence of premorbid problems (No Premorbid Problems group). I compared the groups on the academic and behavioural outcome variables.

Caregivers completed the relevant measures in a conference room at the Development Clinic at Red Cross War Memorial Children's Hospital (RXH), in the waiting room of the RXH Head Injury Clinic, or at their home. I sent the teacher versions of the relevant measures to the participants' schools. The relevant teachers completed these forms at their earliest convenience.

Participants

The sample includes 27 children who had been admitted to RXH for closed severe TBI, over a five-year period, between 2006 and 2011, who were of school-going age at the time of their injury. TBI severity was determined using the GCS. Severe injury corresponds to a GCS score of 8 or below, while moderate and mild injury corresponds to a GCS score of 9 to 12, and 13 to 15, respectively (Zillmer et al., 2008). The participants had a post-resuscitation GCS score of ≤8 and received intracranial monitoring. Intracranial monitoring involves placement of intracranial catheters that monitor physiological variables such as $PbtO_2$ and ICP and is usually used with children who have sustained severe TBIs at the RXH. The Head of the Division of Pediatric Neurosurgery at RXH provided the clinical data for the children in the sample, including GCS scores.

Inclusion criteria. I included children who had been admitted to RXH for closed severe TBI (GCS score of ≤8) during the period 2006 – 2011. Further, it was required that participants be of school-going age[2] at the time of their injury and at the time of assessment. I was able to obtain pre-injury and post-injury academic data by only including children of school-going age. Participants had to be at least one year post-injury which meant that assessment would take place during the phase in which the recovery trajectory tends to stabilize (Jaffe, Polissar, Fay, & Liao, 1995; Taylor et al., 2002; Taylor, 2010). Parents or caregivers and teachers of the participants had to be able to speak and understand English, Afrikaans or isiXhosa, to minimize the confounding effects of potential language barriers.

Exclusion criteria. I excluded children who had sustained open TBIs because of its lower incidence, and differing pathophysiology and outcomes (compared to closed TBI; Anderson et al., 2001). I also excluded children who were not attending school at the time of injury and assessment, as this would have limited what academic data was available for those children.

Selection of participants. In total, 137 children were admitted to RXH for severe TBI between 2006 and 2011 (Schrieff et al., 2013). Of this number, 23 children died as a result of their injuries. Of the remaining 114, six children were excluded because they had sustained an open TBI. Of the remaining 108 children, 53 were not of school-going age at the time of injury, which left 55 children who were then eligible to take part in the study. Of the 55 potential participants, two caregivers failed to return the measures, three caregivers declined participation in the study,[3] and three of the children had not returned to school after their injury. Of the three that had not returned to school, one child had severe cerebral palsy and was unable to attend school, and two were awaiting placement in special needs institutions. Of the 47 potential participants that remained, I was unable to contact 20 of the caregivers because there were either no contact details for these participants or their contact details were outdated. This left a sample of 27 children participating in this study, as illustrated in Figure 1.

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[2] According to the South African Schools Act of 1996, all children between the ages of seven and 15 are required to attend school. A child can be registered for Grade 1 if they are seven years old or will be turning seven during their first year of schooling. A child may also be admitted to Grade 1 at a public school if they are five years old and turning six by 30 June of that year (Department of Education, 2006; 2013).

[3] One caregiver stated time constraints as the reason for declining, one caregiver did not give a reason for declining when she was contacted, while the third failed to attend the arranged data collection session and subsequently did not respond to further communication.

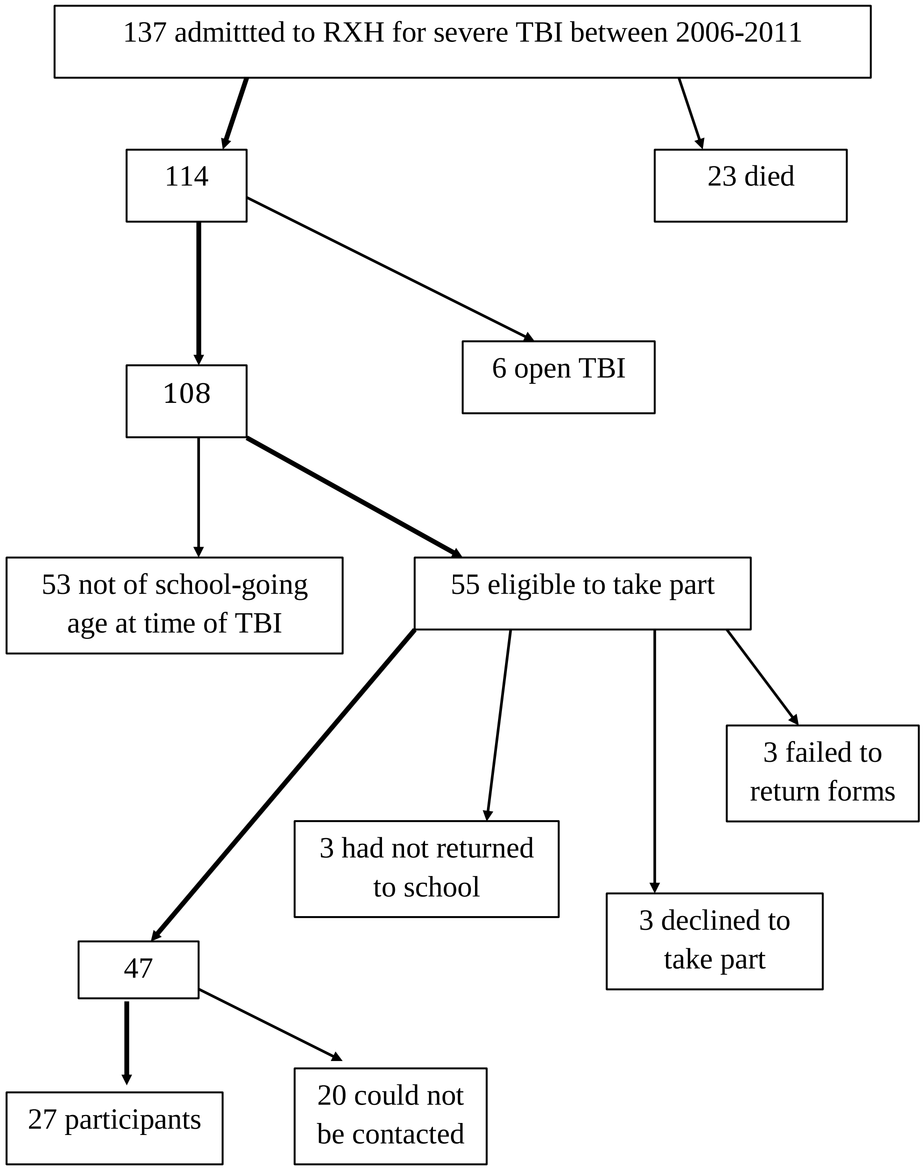

Figure 1. Flowchart of how the sample of 27 participants was obtained.

Measures

I had the original English versions of the following measures translated into Afrikaans and isiXhosa. These were linguistically validated through forward and back translations and authentication, for use by the Afrikaans and isiXhosa-speaking caregivers and teachers of the participants, by the Language Centre at the University of Stellenbosch (Cape Town, South Africa).

Parent information questionnaire and asset index. I administered this questionnaire to participants' caregivers to get an indication of their demographic and socioeconomic backgrounds (See Appendix A). It captures demographic information such as parental/guardian employment and education, home language and annual household income.

The questionnaire includes an asset index that groups asset ownership into three categories according to the aggregate score obtained on this measure: 0-5 (low), 6-12 (medium), and 13-17 (high). This asset index reflects the material and financial resources of the household, for example, appliances, a flushing toilet, running water and car, as well as whether or not the responder makes use of financial services, such as bank accounts and credit cards.

The asset index follows the index used by Myer, Stein, Grimsrud, Seedat, and Williams (2008) who reported that it showed excellent reliability (Cronbach's $\alpha = 0.92$). They included this measure of assets in addition to traditional indicators of SES (such as household income, employment and education level), as traditional SES indicators may not adequately represent socioeconomic variation in the setting of LAMICs (Myer, Ehrlich, & Susser, 2004).

Pediatric neuropsychology developmental questionnaire. I used this questionnaire to obtain information regarding the developmental history of the participants. Questions on this form fall under the categories of pregnancy and birth, development, family composition, parent information and medication (See Appendix B). I asked caregivers to complete the form to the best of their knowledge. I examined the responses to determine if there were indications of pre-injury problems, for example, complications during pregnancy or birth, or delayed developmental milestones. This questionnaire is used at the Pediatric Neuropsychology Clinic at RXH.

Child Behavior Checklist. The CBCL is a scale that is used to assess the capabilities and the behavioural and emotional functional domains of children between the ages of 6 and 18 years (Achenbach & Rescorla, 2001). The CBCL has both a parent and teacher version, which I used to get an indication of the child's behaviour in a home and school setting, when completed by the caregivers and teachers of the participants, respectively. In addition to demographic questions, there are items that investigate competency within the categories of activities, social and school on the CBCL parent version. The CBCL teacher version has items that investigates adaptive functioning and academic performance. Scores are then produced from a 112-item questionnaire that forms part of both the parent and teacher version of the CBCL, for eight syndrome scales. These include: Anxious/Depressed, Withdrawn/Depressed, Somatic Complaints, Social Problems, Attention Problems, Rule-breaking Behaviour and Aggressive Behaviour. There are also six Diagnostic and Statistical Manual (DSM)-oriented scales: Affective Problems, Anxiety Problems, Somatic Problems, Attention Deficit/Hyperactivity Problems, Oppositional Defiant Problems, and Conduct Problems. Scores can also be produced for two broad syndrome groups, namely Internalizing

Problems and Externalizing Problems. Internalizing problems refer to problems within the self, such as depression and anxiety, while externalizing problems refer to for example, aggressive, delinquent behaviours.

A Total Problems score is then also computed from the syndrome scales that form part of the Internalizing Problems and Externalizing Problems groups, as well as the remaining syndrome scales. An open-ended 113th item is included for additional problems. However, if multiple problems are recorded for this item, only one problem then forms part of the Total Problems score (Achenbach & Rescorla, 2001). A 3-point rating scale is used to record responses, that being “not true”, “somewhat or sometimes true”, and “very true or often true”.

Raw scores on the measure are converted to age-standardized T-scores, with lower scores truncated at T = 50. T-scores distinguish between normal, borderline clinical and clinical ranges. On the syndrome and DSM-Oriented scales, T-scores below 67 are within the normal range, while borderline clinical T-scores are between 67-70, and T-scores above 70 are indicative of clinical behavioural problems. For the Total, Internalizing and Externalizing Problems scales, T-scores below 60 are within the normal range, while borderline clinical T-scores are between 60-63, and T-scores above 63 are indicative of clinical behavioural problems (Achenbach, 1991; Achenbach & Rescorla, 2001).

Psychometric properties and cross-cultural use. The psychometric properties of the CBCL include that of validity and reliability, as well as test-retest reliability (Achenbach & Rescorla, 2001). Regarding Cronbach’s α, values vary between 0.72 to 0.97. Internal consistency values are reported as being between 0.72 and 0.95 for the CBCL parent and teacher version, respectively. Regarding test-retest reliability, *r*-values are reported as being between 0.95 and 1.00. Inter-rater reliability is reported as being between 0.93 and 0.96.

There are limited published (e.g., Cluver, Gardner & Operario, 2007; Palin et al., 2008; Shields, Nadasen, & Pierce, 2008; Van Gelder & Kraakman, 2007) and unpublished studies (e.g., Cheesman, 2011; Fischer, 2009; Schrieff, 2013) with South African samples that make use of the CBCL.

Behavior Rating Inventory of Executive Function. The BRIEF is a standardized rating scale assessing executive function behaviours in daily activities within the home (parent version) and school (teacher version) environment, for children aged 5-18 (Gioia et al., 2000; Vriezen & Pigott, 2002). Caregivers and teachers completed the parent and teacher versions of the BRIEF, respectively. An overall composite score of executive function, the Global Executive Composite (GEC), is produced from this 86-item questionnaire, as well as two index scores of executive processes, namely Behavioural Regulation and Metacognition. Scores for the eight clinical subscales within these two domains are also produced. The clinical subscales assess eight interrelated executive function domains. The Behavioural Regulation Index (BRI) includes the subscales of Inhibit, Shift, and Emotional Control, while Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor forms part of the Metacognition Index (MI).

A 3-point rating scale is used to record responses, that being “never”|, “sometimes”, and “often”. Raw scores are converted to T-scores for the composite, index and subscale scores. Higher T-scores indicate greater executive function difficulties experienced in each of the domains. The clinical cut-off is a T-score of 63 (Chapman et al., 2010; Mangeot, Armstrong, Colvin, Yeates, & Taylor, 2002; Vriezen & Pigott, 2002).

Psychometric properties and cross-cultural use. The BRIEF shows high levels of internal consistency and stability, as well as test-retest reliability (Chapman et al., 2010; Malloy & Grace, 2005). Gioia et al. (2000) reported Cronbach’s α values of between 0.80 – 0.98 for internal consistency for both parent and teacher versions of the BRIEF. Regarding test-retest reliability, r-values are reported as being 0.81 and 0.87, for parents and teachers, respectively. Moderate inter-rater reliability is reported for parents and teachers, with an average of $r = 0.32$.

Though the BRIEF has been extensively used in both research and clinical practice settings, there has been limited use outside English-speaking countries (Huizinga & Smidts, 2011). In addition, there is only unpublished literature on South African samples (e.g., Schrieff, 2013). It has however been used cross-culturally in published studies, for example, in the Han Chinese and Dutch populations (Huizinga & Smidts, 2011; Qian, Shuai, Cao, Chan, and Wang, 2010).

Parenting Stress Index. The Parenting Stress Index (PSI) is a parent self-report scale that is designed to evaluate stress within the parent-child system (Abidin, 1995). Specifically, the PSI is a 120-item scale that helps to identify (a) characteristics of the parents and family that do not promote normal child development and functioning, (b) behavioural and emotional problems in children, and (c) parents who may be at risk for using dysfunctional parental practices (Family Resource Information, Education and Network Development Services (FRIENDS), 2010).

Subscales of the PSI relate to the child and parent. The six child-related subscales include Mood, Adaptability, Demandingness, Reinforcement of the Parent, Distractibility/Hyperactivity and Acceptability. Scores from the child-related subscales are added to produce an overall child domain score. The seven parent-related subscales include Competence, Isolation, Attachment, Health, Role Restriction, Depression and Spouse. Scores from the parent-related subscales are added to produce an overall Parent Domain score. An overall Total Stress score is derived from adding the Child and Parent Domain scores. A Life Stress score is also derived from the PSI and is separate from the Total Stress score. This Life Stress score is based on stress experienced, external to the parent-child relationship, for example, as a result of marriage, legal problems, and deaths in the immediate family.

A 5-point rating scale is used to record responses, from “strongly disagree” to “strongly agree”. Greater stress corresponds to higher ratings (Abidin, 1995; FRIENDS, 2010). According to Abidin (1995), scores that fall at or above the 85th percentile are in the critical range above that which is considered normal. It is recommended that respondents that obtain a Total Stress score at or above 260 be referred for further professional investigation.

Psychometric properties and cross-cultural use. Internal consistency of this measure is high. Abidin (1995) reports Cronbach’s α values of between 0.70 and 0.83 for the Child Domain subscales, 0.70 and 0.84 for the Parent Domain subscales, and over 0.90 for the Total Stress scale. The PSI shows good test-retest reliability, with *r*-values reported as being 0.63, 0.91 and 0.96 for the Child Domain, Parent Domain and Total Stress scale, respectively.

This scale has been empirically validated cross-culturally to be able to predict child behaviour and adjustment, and parental behaviour. The PSI is described as a robust diagnostic measure, with its validity holding after translation and when used for diverse cultures (Family Resource Information, Education and Network Development Services (FRIENDS), 2010). The PSI has been used in published studies (Harris, Ellison & Clement, 1999; Potterton, Stewart & Cooper, 2007) and unpublished studies (e.g., Cheesman, 2011; Oosthuizen, 2007) on South African populations as a measure of parenting stress.

Procedure

Caregivers completed the measures during one session at the Developmental Clinic at RXH, in the waiting room of the Head Injury Clinic at RXH or at their home. I contacted caregivers of potential participants telephonically or approached them at the RXH Head Injury Clinic on the day of another scheduled appointment if contact details were unavailable. Research assistants, who are first language isiXhosa-speakers, contacted isiXhosa-speaking caregivers telephonically. The principal investigator or one of the research assistants (in the case of first language isiXhosa-speaking caregivers) explained the purpose of the study and the procedure to be followed and invited the caregivers to participate in the study. Once interest in participating in the study was expressed, the principal investigator or research assistant scheduled an appointment for completion of all the measures if contact had been made telephonically. Measures were completed immediately if the caregivers were approached at the RXH Head Injury Clinic.

On the day of assessment, I reiterated the purpose and procedure of the study and obtained informed consent from caregivers before the session commenced. This information was translated and repeated by a research assistant in the case of first language isiXhosa-speakers. First language Afrikaans-speaking caregivers were comfortable with English and did not require the purpose and procedure to be explained in Afrikaans. In addition, several of the first language Afrikaans-speaking caregivers opted to complete the English versions of the measures.

I administered the parent information questionnaire and asset index and a pediatric neuropsychology developmental questionnaire to the caregivers, followed by the CBCL, the BRIEF and the PSI. The duration of the session was approximately two hours and caregivers were allowed a short break when necessary.

Teachers completed the CBCL and BRIEF teacher versions after the caregivers had completed the measures. I either took the relevant teacher forms that is, the teacher consent form, and the CBCL and BRIEF questionnaires directly to the participants' schools or sent them electronically. I encouraged the teachers to contact me if they had any queries. Teachers completed the measures at a time convenient for them and thereafter returned the forms to me electronically or I collected them from the school directly. I sourced the academic data from the responses on the measures and from the participants' RXH medical folders[4]. In the case where components of the academic data were unclear, I consulted the caregiver directly for this information.

Premorbid functioning information. To get a general sense of participants' academic and behavioural functioning before sustaining the TBI, I examined each participant's case file for documents detailing evidence of behaviour or academic problems, academic history as well as any relevant medical history for example, previous HIs and TBIs. I examined the developmental history form for information regarding the pregnancy and birth, developmental milestones, behaviour problems and schooling information. I examined the CBCL for details regarding academic history and evidence of academic problems. I also

[4] Hereafter referred to as 'case file' owing to the fact that, while the folder is primarily used to store medical information and documents, non-medical information such as demographics, correspondence and school results can also be found in it.

specifically investigated premorbid academic history of participants by looking at pre-injury grade failures and pre-injury school type.

Ethical Considerations

The study followed the ethical guidelines for research using human subjects as outlined by, and was approved by, University of Cape Town (UCT)'s Department of Psychology's Research Ethics Committee, as well as the Faculty of Health Sciences Ethics Committee (see Appendix C). This study adhered to the ethical principles for medical research involving human subjects as set out in the World Medical Association Declaration of Helsinki (Journal of the American Medical Association, 2000). I obtained permission from the Western Cape Education Department to access to school data and to contact teachers in order for them to complete the teacher measures (See Appendix D).

Consent. I obtained informed consent from the parent or caregiver before completing the measures (see Appendix E). At the same time, I requested permission to obtain relevant information from the child's school. I also obtained informed consent from class teachers that completed the teacher measures (see Appendix F). However, I did not obtain assent as the children in the study did not complete any measures.

Confidentiality. I assured caregivers and teachers that no identifying information would be kept in my data sheet or report.

Voluntary participation. Caregivers and teachers were assured that their participation was voluntary and that they were not obliged to participate in the study, even after signing a consent form. They were informed that they were allowed to withdraw from the study at any point in time, without penalty.

Risks and benefits for participants. There were no risks to the participants, or their caregivers, in this study. Caregivers were allowed to take rest breaks during completion of the measures if they experienced fatigue. There was no direct benefit from this study for the participants. However, it is hoped that the data obtained will contribute to knowledge concerning academic and behavioural outcomes in South African children who have sustained a TBI and furthermore, to potentially identify those at risk for more significant adverse outcomes in order to inform appropriate intervention strategies.

Statistical Analysis

I used the Statistical Package for the Social Sciences (SPSS) version 22.0 to complete the data analyses. The steps involved in these analyses are outlined below. Statistical significance was set at $\alpha = 0.05$, unless otherwise specified.

Descriptive statistics. Data analysis began with examination of the descriptive statistics of the sample. These were used to (a) provide an indication of the demographic and clinical characteristics of the participants; (b) investigate and describe the distribution of the data sets; (c) detect the presence of outliers; and (d) test the assumptions of parametric data.

To assess whether the data followed a normal distribution, I performed the Shaprio-Wilk test. I used Levene's test to assess for homogeneity of variance. The assumptions underlying the analyses were checked. These assumptions were upheld, unless otherwise specified.

Effect size. I measured effect size to assess the real-world significance of the relationships between the variables. In this study, the *r*-statistic provided a measure of effect size in both parametric and non-parametric analyses. Effect sizes are described as small, medium or large and are represented by *r*-values of 0.10, 0.30 and 0.50, respectively (Field, 2009). While the *r*-statistic and Cohen's *d* can both serve as a measure of effect size, they are not always equivalent such as when sample sizes are equal or not. McGrath and Meyer (2006) note several advantages to using the *r*-statistic over Cohen's *d*. For example, there is a more direct relationship between the *r*-statistic and statistical power, and is therefore preferable when sample sizes are unequal. The *r*-statistic can also be calculated for both dichotomous and quantitative variables in any combination.

Type I error. Though the multiple comparisons used in this study's analyses may increase the likelihood of making a Type I error, strict statistical control methods, such as the use of a Bonferroni correction, were not used. Conservative measures were not used as they themselves may increase the likelihood of making a Type II error. In the context of public health research, more concern is placed on missing effects that, although possibly subtle, are of clinical importance (Jacobson & Jacobson, 2005).

Independent variables. These are factors that affect outcome after TBI.

Premorbid functioning. The premorbid functioning variable is an average score that represents the participant's functioning prior to their TBI. I regarded pre-injury academic problems, pre-injury behavioural problems (behaviours which were regarded as problematic or abnormal e.g., hyperactivity) and problems associated with development as evidence of there being premorbid problems. A previously sustained TBI (other than the TBI which resulted in inclusion in this study) or HI (that resulted in hospital admission) was also classified as a premorbid problem, since there is extensive evidence of impairment of neurocognitive domains following such injuries, even those that are mild in nature (Anderson et al., 2001; Babikian & Asarnow, 2009; van't Hooft, 2010). Reported preterm births, according to birthweight and gestational ages, were also considered to be indicative of premorbid problems. This is because risk of poor cognitive performance during school years and behavioural problems is associated with preterm birth (Sutton & Darmstadt, 2013).

I defined *behaviour problems* as any documented accounts of behaviour described as being problematic or abnormal, and that pre-dated the TBI. I defined *academic problems* as any evidence of poor academic achievement, poor academic skills such as reading and writing, and general difficulties at school, all of which pre-dated the TBI and were documented in the sources that were examined. I defined *previous HIs and TBIs* as those that resulted in hospital admission, and other than the TBI that resulted in inclusion in this study, respectively. I defined *other* as any problems that did not fall under any of the categories of problems described above; this included any documented problems with pregnancy, birth and development as well as medical conditions.

I investigated the components of the premorbid functioning variable through various sources including the participant's case file, the developmental history form, and the CBCL. I assigned a value of 0 or 1 for each component when there was no evidence of any premorbid problems and evidence of premorbid problems, respectively, from the information obtained from the case file, developmental history form and the CBCL, as well as academic history. Components that comprise the premorbid functioning variable are detailed below and presented in Table 1.

Table 1
Components that Comprise the Premorbid Functioning Variable

Source	Value
Case file	0 no evidence of problems 1 evidence of problems
Developmental history form and CBCL	0 no evidence of problems 1 evidence of problems
Academic history	
Pre-injury grade failures	0 no pre-injury failure 1 pre-injury failure
Type of education	0 mainstream pre-injury education 1 special needs pre-injury education

Note. CBCL = Child Behavior Checklist

Case file. I looked through the case file of each participant and noted any relevant premorbid history. I was unable to access several of the case files as the RXH Medical Records Department was unable to locate them.

Developmental history form and CBCL. The developmental history form and the CBCL were grouped together as one source of information of premorbid functioning. I assessed the responses on these forms, particularly looking at responses regarding the pregnancy and birth, development, problems with behaviour and academics, use of remedial education services and grade failure history.

Premorbid academic history. The premorbid academic history includes two components: 1) whether or not the participant had failed any grades prior to the TBI that resulted in inclusion in this study, and 2) the type of education that they were receiving prior to the TBI, these being either mainstream education or special needs education. The components forming academic history and values assigned for each component are also shown in Table 1. In cases where academic premorbid history was unclear, for example if it was unknown if there were any pre-injury failures, it was directly queried with caregivers.

Premorbid functioning variable. In part one of the study, a total premorbid functioning score was calculated by adding the score on each source (i.e., the case file, developmental history form and CBCL, and the two academic history components) and dividing it by the available number of sources (i.e., by a maximum of four if the case file, developmental history form, and CBCL were assessable and if the two components of academic history were known). A higher score represents greater evidence of premorbid problems. For example, participants that did not have evidence of premorbid problems across any of the sources had a premorbid functioning score of 0, while participants who had evidence of premorbid problems in one of the four sources had a premorbid functioning score of 0.25.

No participant had more than one source was missing. However, in the cases where there was unknown information, for example, when the case file could not be accessed or if the caregiver failed to complete the developmental history or CBCL measures, that source was excluded from the equation when calculating the premorbid functioning score. This may have slightly increased the average score for participants with missing data. However, I used this method rather than assigning a negative value to unknown sources, which would have underestimated the premorbid problems already noted. It was also preferable than to assign the same value as one of the other sources since I found that many of the participants had evidence of problems noted in one source and not the other.

In part two of the study, I used the variable of premorbid functioning to split the sample into two groups: 1) Premorbid Problems versus, 2) No Premorbid Problems. In order to do this, a value of 0 was assigned to each source when there was no evidence of premorbid problems and a value of 1 was assigned to each source when there was evidence of premorbid problems (see Table 1). A participant with a value of 1 on any of the sources was placed in the Premorbid Problems group, while a participant with 0 on all sources was placed in the No Premorbid Problems group.

Time since injury. Time since injury is measured in months, from the time the TBI occurred until the time that the assessment took place.

Age at injury. The age at which the TBI occurred is measured in months.

Family environment. The Total Stress score derived from the PSI is used as a measure of the child's family environment.

SES. SES is represented by the asset index aggregate score taken from the parent information and asset index form. The asset index reflects the material and financial resources of the participant's household.

Dependent variables. These are the outcome scores that represent academic outcome and behavioural outcome.

Academic outcome. Academic outcome is represented by a composite score representing two variables: 1) that of the child's education type at the time of assessment and 2) whether or not the child has repeated any grades since returning to school after their injury. Both variables were assigned values to assist in computing a composite score. Table 2 presents the values assigned to the variables that comprise academic outcome. The variables of 'education type' and 'repeated grades' were each converted to z-scores, which represents results in terms of standard deviation units (Jaffe et al., 1995). A composite z-score was then derived through averaging the z-scores for these variables. Greater scores represent poorer academic outcome.

Table 2
Variables that Comprise the Composite Score of Academic Outcome

Variable	Value
Education type at time of assessment	0 mainstream school
	1 mainstream school with remedial help
	2 mainstream school with special needs school application or awaiting placement
	3 special needs school
Repeated grades since returning to school	0 no repeated grades since returning to school
	1 repeated a grade since returning to school

Behavioural outcomes. I derived the behavioural outcome variables from the parent versions of the CBCL and the BRIEF. I used the scores from the Total Problems, Internalizing Problems, and Externalizing Problems scales on the CBCL. I used the scores from the BRI, the MI, and the GEC score on the BRIEF. These same scores were taken from teacher versions of the CBCL and BRIEF. Greater scores represent a greater degree of problem behaviour as assessed by the CBCL and BRIEF.

Part 1: Investigation of the factors that influence academic and behavioural outcome after TBI. In this part of the study, descriptive statistics are provided for the factors affecting outcome variables, injury characteristics of the sample, parent and teacher ratings of the CBCL and the BRIEF, and the relevant academic history of the sample.

Correlation matrices. I used bivariate correlation matrices to explore factors affecting outcome (i.e., the independent variables) by investigating the relationships between these independent variables (premorbid functioning, time since injury, age at injury, family environment, SES) and the outcome variables (Total Problems, Externalizing Problems, Internalizing Problems, BRI, MI, GEC). In the correlational analyses, I used Pearson's correlation coefficient (r) for normally distributed, continuous variables and Spearman's correlation coefficient (r_s) for continuous variables that were not normally distributed. I used one-tailed tests in the correlational analyses based on established literature, which informed the direction of the expected relationships.

Intraclass correlations. I used the intraclass correlation coefficients to assess the consistency among responses given by caregivers and teachers on the same measure (Field, 2009; Rosenthal & Rosnow, 2008). Less than half of the sample (n =13) had returned teacher measures for both the CBCL and the BRIEF, therefore these correlations were only evaluated in case where teacher measures were returned.

Part 2: The specific role of premorbid functioning.

Independent samples t-tests. I used independent samples t-tests to explore the specific role of premorbid functioning. I could then determine whether the Premorbid Problems and No Premorbid Problems groups differed in academic and behavioural outcome, using the same outcome variables as described in part one of this study. I used one-tailed tests in the correlational analyses based on established literature, which informed the direction of the expected relationships.

Chapter 3: Results

Part 1: Investigation of the factors that influence academic and behavioural outcome after TBI

Missing data. I handled missing data on the PSI, BRIEF and CBCL according to the instructions given in their respective manuals (Abidin, 1995; Gioia et al., 2000; Achenbach & Rescorla, 2001). On the BRIEF, for example, a score of 1 is assigned if one or two item responses are missing in a subscale, however, if more than two responses are missing for a subscale, the score is not calculated (Gioia et al., 2000). On the PSI, an average score for that subscale is assigned when there is one missing item. Scores for subscales, domains and the total score may not be calculated under certain circumstances of missing responses, such as when there is more than one missing response in a domain or more than one domain score missing within a subscale (Abidin, 1995). In this sample, data for the BRIEF was missing for one participant. This caregiver had to take the measures home with him to complete; however, all measures were returned apart from the BRIEF. He was subsequently lost to follow-up due to a change in contact details after changing employment. One participant's scores are missing from the CBCL. The caregiver did not correctly complete the form and it was unable to be scored. The caregiver subsequently moved away and was lost to follow-up. Various scores were missing on the PSI, therefore various subscale and total scores could not be calculated. Missing data was excluded on a pairwise basis for correlational analysis or analysis-by-analysis basis for the between-group analyses. However, since less than half of the teacher versions of the CBCL and BRIEF were returned (n = 13), the analyses involving these measures included the data for those 13 participants only.

Demographic characteristics of the sample. Table 3 presents a description of the demographic characteristics of the sample (N = 27). IsiXhosa was the home language of just over half of the sample (51.85%; n = 14). Regarding sex, 62.96% of the sample was male (n = 17). The children were the participants in this sample, while their caregivers were the informants. The children themselves did not complete an formal measures. I collected data about the participant's premorbid functioning and academic and behavioral outcomes.

Table 3
Demographic Characteristics of Sample (*N* = 27)

Variable	
Sex	
Male: Female	17: 10
Age in months at Assessment	
Mean (*SD*)	151 (24.00)
Range	115-195
Home Language	
English	5
Afrikaans	5
English and Afrikaans	2
isiXhosa	14
Other	1

Note. The home language recorded as being "other", was in fact Swahili. However, the caregiver was fluent in English and therefore the participant was not excluded.

Injury-related characteristics. Table 4 presents the injury-related characteristics of the sample. The youngest participant was 6.5 years, while the oldest was 12.67 years at the time of injury. The range of time since injury was 1.25 – 5.83 years. The GCS scores, recorded post-resuscitation, show that all participants sustained a TBI classified as severe.

Table 4
Injury Characteristics of Sample (*N* = 27)

Variable	
Age at injury (months)	
Mean (*SD*)	114.11 (21.94)
Range	78-152
Time since injury (months)	
Mean (*SD*)	37.22 (15.19)
Range	15-70
GCS	
Mean (*SD*)	5 (1.62)
Range	2-8

Note. Means are presented with standard deviations in parentheses. GCS = Glasgow Coma Scale

Premorbid functioning. I used various sources in order to obtain information about participants' premorbid functioning. Table 5 shows the frequency and types of premorbid problems that were noted in the case files and on the developmental history and CBCL forms. Table 5 also includes the academic premorbid information.

Table 5
Frequency and Types of Premorbid Problems

Source	Types of problems	Frequency (%)
Case file[a] (*n* = 22)	Behavioural problems[c]	5 (22.73)
	Academic problems[d]	2 (9.09)
	Previous HIs or TBIs[e]	2 (9.09)
Developmental history form[b] (*n* = 26)	Behavioural problems[f]	2 (7.69)
	Other[h]	3 (11.54)
CBCL[b] (*n* = 26)	Academic problems[g]	2 (7.69)
Academic (*n* = 27)		
Pre-injury grade failures	No pre-injury grade failures: One or more pre-injury grade failures	23:4 (85.19: 14.81)
Pre-injury type of education	Mainstream: special needs	26:1 (96.30: 3.70)

Note. Frequencies are presented with percentages in parentheses. TBI = Traumatic Brain injury; CBCL = Child Behavior Checklist.
[a]Data missing for five participants because I was unable to locate their case files. [b]Data missing as one parent chose to not complete the developmental history form, with no reason given, and one CBCL was not correctly completed. [c] Pre-injury behavioural problems documented by medical personnel included hyperactivity and impulsivity. [d]One participant was noted by a doctor to be doing more poorly in school following a previous TBI which resulted in him failing a grade, while the other participant was noted by a speech therapist to be struggling generally at school. [e]One participant was previously involved in a motor vehicle accident (MVA) involving a bike (type of bike unknown), while the other was assaulted, sustaining an injury to his head. [f]Hyperactivity. [g]One participant had poor literacy skills and another had general difficulties (unspecified) in Grade 1. [h]The following problems were reported: early birth and low birth weight (36 weeks, 2 kilograms (kgs); 32 weeks, unknown weight; unknown gestation period, 1.9kgs); cerebral palsy; developmental delay.

Case file. The most frequently reported premorbid problems were that of behavioural complaints for example hyperactivity and impulsivity. Premorbid problems were mostly reported in the case files by medical doctors and/or occupational or physical therapists during their assessments.

Developmental history form and CBCL. Information regarding academic and behavioural problems, and developmental history such as delayed milestones, were taken from the developmental history form and CBCL. Due to the limited relevant items on the CBCL, I grouped the information taken from the developmental form and CBCL together as one source. Caregivers reported academic and behavioural problems, as well as other problems during the developmental period, such as low birth weight developmental delay. The developmental history form and the CBCL are separated in Table 5 for descriptive purposes.

Premorbid academic history. The majority of the participants (*n* = 23) had not failed any grades at school prior to their TBI. Only one of the participants attended a special needs school premorbidly.

Premorbid functioning variable. Participants received a premorbid functioning score that ranged between 0 and 1 (*M* = 0.16, *SD* = 0.24). A higher score represents greater evidence of premorbid problems.

Family environment. Table 6 presents the descriptive statistics for scores obtained on the PSI, which includes the Total Stress score, the Life Stress score, the Child Domain and Parent Domain, as well as the subscales that make up those domains. The frequencies of scores in the sample that fell into the critical range are indicated in the table. On the Child Domain, the subscale of Acceptability, according to Abidin (1995), is related to how child meets the prior expectations of their parent. As seen in Table 6, 85.19% (23/27) of the sample scored in the critical range on the Acceptability subscale, which implies that the majority of the participants do not meet the expectations that their parents had for them. On the Parent Domain, the Reinforces Parent subscale (74.07%; 20/27), Demandingness subscale (74.07%; 20/27), and the Adaptability subscale (65.38%; 17/26)) also had a high frequency of scores falling into the critical range. Regarding the Total Stress scores, 60% (25/27) of these scores fell in the critical range. However, there was a greater percentage of scores that fell in the critical range on the Child Domain (72%; 18/25), compared to that of the Parent Domain (44.44%; 12/27).

Table 6
Family Environment as Measured by the PSI (*N* = 27)

PSI outcome variable	Raw score *M* (*SD*)	Frequency of critical scores[c] (%)
Child Domain[a]	136.60 (29.09)	18 (72.00)
Distractibility/Hyperactivity[b]	26.88 (5.53)	11 (42.31)
Adaptability[b]	33.00 (7.52)	17 (65.38)
Reinforces Parent	14.07 (4.78)	20 (74.07)
Demandingness	27.52 (8.92)	20 (74.07)
Mood[b]	13.54 (4.22)	17 (65.38)
Acceptability	23.07 (5.27)	23 (85.19)
Parent Domain	146.89 (27.48)	12 (44.44)
Competence	35.56 (7.16)	13 (48.15)
Isolation	15.81 (4.57)	9 (33.33)
Attachment	16.00 (3.15)	14 (51.85)
Health	12.89 (3.72)	6 (22.22)
Role Restriction	22.00 (5.84)	10 (37.04)
Depression	25.30 (7.77)	14 (51.85)
Spouse	19.33 (4.44)	8 (29.63)
Total Stress[a]	282.24 (50.91)	15 (60.00)
Life Stress[b]	17.04 (11.13)	15 (57.69)

Note. Means are presented with standard deviations in parentheses. Frequencies are presented with percentages in parentheses. PSI = Parenting Stress Index.
[a]Data missing for two participants. [b]Datum missing for one participant.
[c]Represents scores ≥85%ile.

SES. Table 7 presents the SES information of the sample. Most of the caregivers, that is, the parents or guardian of the participants, reported on the Parent Information Questionnaire and Asset Index that they have at least eight to eleven years of education. All but one household reported having an income, despite a large number of caregivers reporting that they and/or their partners are unemployed, disabled or still studying (father, $n = 9$; mother, $n = 13$). Income may come from sources such as state child-care grants, disability grants or from other persons living in the household who are not the participants' parents or guardians (such as an aunt, uncle, grandparent or family friend).

The SES variable used in the analyses in correlational and between-group analyses in part 1 and part 2, respectively, is represented by the asset index aggregate score reported in Table 7. All participants fell into the medium (n= 14) or high asset (n =12) index bracket indicating that all the participants had at least 6 or more material and financial resources in their households. The mean number of assets was 12.15 (SD = 2.57), and these ranged between 7 assets and 17 assets.

Table 7

SES and Asset Index Data for the Sample (*N* = 27)

Variable	
Household income per year[a]	
0	1
1 – 5000	6
5001 – 25 000	8
25 001 – 100 000	10
100 001+	2
Parental education (father: mother: guardian)[b]	
0 years	1: 0: 0
1 – 6 years	0: 0: 0
7 years	5: 3: 0
8 – 11 years	10: 12: 1
12 years	3: 7: 0
13+ years	3: 3: 0
Unknown/incomplete	4: 1: 0
Parental employment (father: mother: guardian)[b]	
Higher executives, major professions	0: 0: 0
Business managers of medium businesses, lesser professionals	0: 1: 0
Administrative personnel, managers, minor professionals, owners / proprietors of small businesses	2: 0: 0
Clerical and sales, technicians, small businesses	2: 3: 0
Skilled manual (usually having had training)	4: 2: 1
Semi-skilled	3: 4: 0
Unskilled	0: 1: 0
Homemaker	0: 1: 0
Student, disabled, no occupation	9: 13: 0
Unknown/incomplete	6: 1: 0
Material and financial resources (Asset Index)	
0 – 5 assets (low)	0
6 – 12 assets (medium)	15
13 - 17 assets (high)	12

Note. [a]Household income presented in South African Rands (ZAR). [b]For one of the cases, information on education and employment was only provided for the participant's guardian and not for the parents.

Academic outcome. Table 8 provides information on the outcomes of the participants in terms of their academics. At the time of assessment, one third of the sample (*n* = 9) were being educated in a mainstream school and were not receiving any remedial help. Just over a quarter of the sample (*n* = 7, 25.93%) required remedial help within mainstream schooling. Remedial help was usually in the form of extra lessons or placement in a remedial-oriented class at the mainstream school. For six participants, school authorities were making arrangements to place them in a special needs school that catered for their specific learning or physical needs. These participants awaiting placement continued to attend mainstream schools while their applications to special needs schools were being reviewed or while they were awaiting placement once an opening became available at a specific special needs school. They may or may not have received remedial help in the interim. Of the sample, 18.52% (*n* = 5) were attending special needs schools. They were either placed directly into these schools following their TBI, or were placed in the schools at a later stage before commencement of the study. In sum, children who were receiving remedial help, were awaiting placement in special needs school, or were attending a special needs school at the time of assessment, comprised of two thirds of the sample (*n* = 18, 66.66%).

Table 8

Components of the Academic Outcome Variable (*N* = 27)

Component	Frequency (%)
Post-injury type of education	
Mainstream	9 (33.33)
Mainstream with remedial help	7 (25.93)
Mainstream with special needs application in place or awaiting placement	6 (22.22)
Special needs education	5 (18.52)
Repeated grade post-injury: Did not repeat grade post-injury	16:11 (59.26: 40.74)

Note. Frequencies are presented with percentages in parentheses.

Table 9 compares some of the academic information obtained for the sample and presents the ratios of grade repetition pre- and post-injury, as well as the pre- and post-injury type of education. Table 10 presents a cross-tabulation of pre- and post-injury academic information.

Table 9

Comparison of Pre- and Post-injury Academic Information (*N* = 27)

Component	Pre-injury	Post-injury
Grade repetition		
Did not repeat grade: Repeated grade	23: 4	11: 16
Type of education		
Mainstream: Special needs	26: 1	22: 5

Note. Frequencies are presented with percentages in parentheses.

Table 10

Crosstabulation of Pre-injury Academic Information by Post-injury Academic Information (N = 27)

Component			Post-injury academic information			
			Grade repetition		Type of education	
			No	Yes	Mainstream	Special needs
Pre-injury academic information	Grade repetition	No	8 (29.6%)	15 (55.6%)	19 (70.4%)	4 (14.8%)
		Yes	3 (11.1%)	1 (3.7%)	3 (11.1%)	1 (3.7%)
		Total	11 (40.7%)	16 (59.3%)	22 (81.5%)	5 (18.5%)
	Type of education	Mainstream	11 (40.7%)	15 (55.6%)	22 (81.5%)	4 (14.8%)
		Special needs	0 (0%)	1 (3.7%)	0 (0%)	1 (3.7%)

In this sample, there was a three-fold increase in the number of children using special education services, from before to after the TBI. This increase reveals that for 44.44% of the sample (*n* = 12), the use of remedial services or special needs education was required only after the TBI, even when taking into account premorbid academic problems.

Pre-injury, 14.81% (*n* = 4) of the sample had repeated one or more grades, while more than half of the sample (*n* = 16; 59.26%) had to repeat a grade following their TBI which represents a 275% increase from before to after the TBI. Participants in both mainstream and special needs schools formed part of the sample that repeated a grade post-injury. Of the four participants (14.80%) that repeated a grade pre-injury, only one (3.70%) also repeated a grade post-injury. This participant attended a special needs school pre- and post-injury. The three participants (11.10%) that repeated a grade pre- but not post-injury, attended mainstream school pre- and post-injury, with only one requiring the use of remedial services at the time of this study.

Behavioural outcomes.

CBCL. *Parent measure.* Table 11 presents the descriptive statistics for scores obtained on the parent version of the CBCL. These include the scores for the broad syndrome groups of Internalizing Problems and Externalizing Problems, the eight syndrome scales and the Total Problems score for all syndrome scales, as well as the DSM-oriented scales. Age-standardized scores are used to distinguish between normal, borderline clinical and clinical ranges.

The sample on average scored in the clinically significant range on the Total Problems (*M* = 68.50, *SD* = 9.85), Internalizing Problems (*M* = 67.27, *SD* = 9.87) and Externalizing Problems (*M* = 66.19, *SD* = 11.92) scales. Within the Total Problems scale, the Internalizing Problems scale had highest frequency of participants whose scores fell in the clinical range (65.38%), followed by the Externalizing Problems scale (53.85%). These percentages of critical range scores indicate that more than half of the participants experienced clinically significant problems across a range of behaviours. Less than half of the sample scored within the clinical range on all other individual scales that form part of the CBCL, the highest frequency being on the Aggressive Behaviour (46.15%) and Affective Problems (46.15%) scales.

Table 11
Outcome Scores Obtained on the CBCL Parent Version (*N* = 27)

CBCL Syndrome Profile[a]	Mean (*SD*)	Range	Frequency of borderline clinical scores	Frequency of clinical scores
Total Problems	68.50 (9.85)[b]	47-88	3 (11.54)	17 (65.38)
Social Problems	67.77 (13.57)[c]	50-100	6 (23.08)	8 (30.77)
Thought Problems	63.72 (11.25)	50-90	5 (19.23)	8 (30.77)
Attention Problems	68.42 (11.87)[c]	51-100	3 (11.54)	10 (38.46)
Internalizing Problems	67.27 (9.87)[b]	48-89	4 (15.38)	17 (65.38)
Anxious/Depressed	65.31 (10.57)	50-94	3 (11.54)	8 (30.77)
Withdrawn/ Depressed	65.23 (10.20)	50-93	4 (15.38)	5 (19.23)
Somatic Complaints	66.08 (10.07)	50-90	8 (30.77)	7 (26.92)
Externalizing Problems	66.19 (11.92)[b]	46-88	4 (15.38)	14 (53.85)
Rule-breaking Behaviour	63.12 (11.22)	50-86	4 (15.38)	7 (26.92)
Aggressive Behaviour	69.58 (13.94)[c]	50-100	1 (3.85)	12 (46.15)
DSM-Oriented Scale				
Affective Problems	68.58 (9.64)[c]	50-87	1 (3.85)	12 (46.15)
Anxiety Problems	60.58 (8.51)	50-100	3 (11.54)	4 (15.38)
Somatic Problems	64.65 (9.93)	50-93	4 (15.38)	5 (19.23)
ADH Problems	65.96 (8.07)	52-80	6 (23.08)	6 (23.08)
Oppositional Defiant Problems	63.46 (10.74)	50-80	3 (11.54)	9 (34.62)
Conduct Problems	67.00 (12.63)[c]	50-93	5 (19.23)	8 (30.77)

Note. Means are presented with standard deviations in parentheses. Frequencies are presented with percentages in parentheses. CBCL = Child Behavior Checklist; ADH = Attention Deficit/Hyperactivity.
[a]Reported for *n* = 26. [b]Clinical range. [c]Borderline clinical range.

Teacher measure. Table 12 presents the descriptive statistics for scores obtained on the CBCL teacher version. Borderline clinical and clinical ranges are the same as for the CBCL parent version described above.

Table 12

Outcome Scores Obtained on the CBCL Teacher Version (*N* = 13)

CBCL Syndrome Profile	Mean (*SD*)	Range	Frequency of borderline clinical scores (%)	Frequency of clinical scores (%)
Total Problems	66.23 (11.35)[a]	45-84	0 (0.00)	9 (69.23)
Social Problems	66.23 (9.53)	50-82	1 (7.69)	3 (23.08)
Thought Problems	63.38 (10.29)	50-85	1 (7.69)	3 (23.08)
Attention Problems	64.31 (8.92)	50-79	3 (23.08)	3 (23.08)
Internalizing Problems	62.00 (12.24)[b]	38-82	2 (15.38)	7 (53.85)
Anxious/Depressed	61.92 (9.77)	50-86	2 (15.38)	1 (7.69)
Withdrawn/Depressed	61.08 (10.36)	50-79	0 (0.00)	3 (23.08)
Somatic Complaints	60.08 (9.37)	50-75	1 (7.69)	2 (15.38)
Externalizing Problems	64.08 (11.54) [a]	43-88	2 (15.38)	8 (61.54)
Rule-breaking Behaviour	63.46 (13.19)	50-100	2 (15.38)	2 (15.38)
Aggressive Behaviour	65.08 (10.50)	50-86	1 (7.69)	3 (23.08)
DSM-Oriented Scales				
Affective Problems	62.85 (9.63)	50-78	3 (23.08)	2 (15.38)
Anxiety Problems	60.31 (7.23)	50-73	2 (15.38)	1 (7.69)
Somatic Problems	58.77 (8.59)	50-69	4 (30.77)	0 (0.00)
ADH Problems	62.92 (8.60)	50-75	0 (0.00)	2 (15.38)
Oppositional Defiant Problems	62.69 (7.38)	50-75	3 (23.08)	1 (7.69)
Conduct Problems	62.46 (13.20)	50-100	1 (7.69)	1 (7.69)

Note. Means are presented with standard deviations in parentheses. Frequencies are presented with percentages in parentheses. CBCL = Child Behavior Checklist; ADH = Attention Deficit/Hyperactivity.
[a]Borderline clinical range. [b]Clinical range.

The mean score for each of the subscales fell in the normal range. Overall on Total Problems scale, the sample scored on average in the clinically significant range (*M* = 66.23, *SD* = 11.35). Within the Total Problems scale, the Externalizing Problems scale (*M*= 64.08, *SD* = 11.54), on average fell in the clinical range; while the Internalizing Problems scale (*M* = 62.00, *SD* = 12.24), on average fell into the borderline clinical range. The sample shows a greater degree of behavioural dysfunction in terms of externalizing problems, such as aggression and delinquent behaviour, than internalizing problems, such as depression and anxiety.

Within the Total Problems scale, the Externalizing Problems scale had highest frequency of participants whose scores fell in the clinical range (61.53%), followed by the Internalizing Problems scale (53.85%). These frequencies indicate that more than half of the sample scored in the clinically significant range on those scales. Nearly 70% of the participants (9/13) scored in the clinically significant range on the overall Total Problems scale, which indicates that they exhibited clinically significant problems across a range of behaviours in the school-setting.

Intraclass correlations. I assessed consistency among responses given by caregivers and teachers on their respective versions of the CBCL using intraclass correlation coefficients. Table 13 displays the intraclass correlation coefficients for the parent and teacher versions of the CBCL data. For both the Total Problems and Externalizing Problems scales, there were medium effect sizes for the intraclass correlations. The effect size of the intraclass correlation between caregiver and teacher raters on the Internalizing Problems scale was small.

Table 13
Intraclass Correlation Coefficients for the Parent and Teacher Versions of the CBCL (*N* = 13)

Variable	*r*
Total problems	.446
Internalizing problems	.307
Externalizing problems	.451

Note. CBCL = Child Behavior Checklist.

BRIEF. ***Parent measure.*** Table 14 shows that on the parent version of the BRIEF, the mean of all indices, apart from Initiate (*M* = 61.77, *SD* = 11.88) and Organization of Materials (*M* = 58.08, *SD* = 9.85), fell in the clinical range. Amongst the subscales, the Working Memory Index had the highest number of scores that fell in the clinical range (73.08% of the sample) followed by Emotional Control (65.38%), and Plan/Organisation (65.38%). Overall on the GEC, 69.23% of the sample scored in the clinical range. Looking specifically at the major index scales, more than half the sample scored within the clinical range on the BRI (61.54%) and within the clinical range on the MI (61.54%).

Table 14

Outcome Scores Obtained on the BRIEF Parent Version (*N* = 27)

Indices[a]	Mean (*SD*)	Range	Frequency of clinical scores (%)
GEC	69.19 (14.58)[b]	47-97	18 (69.23)
BRI	70.65 (17.47)[b]	45-101	16 (61.54)
Inhibit	67.62 (18.82)[b]	40-103	14 (53.85)
Shift	68.54 (15.85)[b]	40-98	14 (53.85)
Emotional Control	68.04 (14.97)[b]	43-91	17 (65.38)
MI	66.08 (12.07)[b]	45-87	16 (61.54)
Initiate	61.77 (11.88)	43-89	10 (38.46)
Working Memory	70.00 (10.00)[b]	50-87	19 (73.08)
Plan/Organization	64.46 (11.08)[b]	45-88	17 (65.38)
Organization of Materials	58.08 (9.85)	45-72	9 (34.62)
Monitor	63.35 (13.78)[b]	40-86	15 (57.69)

Note. Means are presented with standard deviations in parentheses. Frequencies are presented with percentages in parentheses. BRIEF = Behavior Rating Inventory of Executive Function; BRI = Behavior Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite

[a]Reported for *n* = 26. [b]Clinical range

Teacher measure. Table 15 shows that on the teacher version of the BRIEF, the mean of all indices fell in the clincal range. Amongst the subscales, the Working Memory (92.31%) and the Plan/Organisation (92.31%) indices had the highest number of scores that fell in the clinical range, followed by the Shift and Emotional Control indices, where 84.62% of the sample scored within the clinical range.

Looking specifically at the major index scales, the majority of the sample scored within the clinical range on the BRI (84.62%) and within the clinical range on the MI (84.62%), and therefore following on from this, the same frequnecy of clinical range scores is reported for the overall GEC.

Table 15
Outcome Scores Obtained on the BRIEF Teacher Version (N = 13)

Indices	Mean (*SD*) (*n* = 13)	Range	Frequency of clinical scores (%)
GEC	81.08 (19.26)[a]	42-118	11 (84.62)
BRI	76.54 (17.59)[a]	45-112	11 (84.62)
Inhibit	74.15 (17.08)[a]	45-99	8 (61.54)
Shift	80.00 (20.21)[a]	45-118	11 (84.62)
Emotional Control	75.46 (15.23)[a]	45-107	11 (84.62)
MI	79.54 (18.12)[a]	42-109	11 (84.62)
Initiate	73.85 (16.84)[a]	47-101	9 (69.23)
Working Memory	78.69 (16.10)[a]	43-108	12 (92.31)
Plan/Organization	76.69 (15.70)[a]	43-103	12 (92.31)
Organization of Materials	76.08 (26.71)[a]	44-123	8 (61.54)
Monitor	76.46 (14.83)[a]	42-94	11 (84.62)

Note. Means are presented with standard deviations in parentheses. Frequencies are presented with percentages in parentheses. BRIEF = Behavior Rating Inventory of Executive Function; BRI = Behavior Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite.
[a]Clinical range

Intraclass correlations. I assessed consistency among responses given by caregivers and teachers on the BRIEF using intraclass correlation coefficients. Table 16 displays the intraclass correlation coefficients for the BRIEF data. For both index scores, as well as the composite score, the effect sizes of the intraclass correlations between the caregiver and teacher raters were large.

Table 16
Intraclass Correlation Coefficients for the Parent and Teacher Version of the BRIEF (N = 13)

Variable	*r*
GEC	.733
BRI	.734
MI	.668

Note. BRIEF = Behavior Rating Inventory of Executive Function; GEC = Global Executive Composite; BRI = Behavior Regulation Index; MI = Metacognition Index.

Correlation matrices.

Parent measures. The first correlation matrix, presented in Table 17, shows the correlations between factors affecting outcome (i.e., family environment, SES, time since injury, age at injury, and premorbid functioning), academic outcome, and behavioural outcome obtained on the parent measures (BRIEF- and CBCL-related measures). The premorbid functioning variable used in the correlation matrix is that which has been previously described.

Correlations with premorbid functioning. Results show that there is a significant positive correlation between premorbid functioning and the GEC score obtained on the BRIEF, with a medium effect size (r_s =.348). This significant correlation between premorbid functioning and the GEC score suggests that greater evidence of poor premorbid functioning is associated with greater executive dysfunction. However, when an outlier on the premorbid functioning variable was removed, the relationship between premorbid functioning and the BRIEF GEC score was no longer significant (see Appendix G). All other correlations involving the premorbid functioning variable remained non-significant after removal of the outlying premorbid functioning score. However, it can be argued that it is important to include this score in the analyses, as it is a reflection of the poorer premorbid functioning of that particular participant.

Correlations with family environment. As shown in Table 17, family environment is significantly positively correlated with academic outcome, CBCL Total Problems, CBCL Internalizing Problems, CBCL Externalizing Problems, BRIEF GEC, BRIEF BRI, and BRIEF MI. With each of the correlations there was large effect size, except for academic outcome for which there was a medium effect size (r_s = .392). The positive correlations with family environment suggest that more stress experienced in the family environment is associated with poorer academic outcome and a greater degree of behavioural and executive problems as assessed with the CBCL and the BRIEF. There is a significant negative correlation between family environment and SES, with a medium effect size (r = -.407), indicating that when there are more material and financial resources available in the household, there is less stress within the family environment. There were no outliers on the family environment variable, or in fact on any other of the independent or dependent variables, apart from that on the premorbid functioning, which has already been discussed.

Correlations between outcome scores. There were strong positive significant correlations between academic outcome and the outcome scores (GEC, BRI, MI) on the BRIEF. These correlations involving the BRIEF suggest that the poorer the academic outcome, the greater the degree of executive function behaviours as assessed on the BRIEF. There were medium effect sizes associated with the significant positive correlations between academic outcome and Total Problems on the CBCL (r_s = .383), and between academic outcome and the Externalizing Problems scale on the CBCL (r_s = .423). These two correlations suggest that the poorer the academic outcome, the greater the degree of total behavioral problems and specifically externalizing problems as assessed on the Total Problems of the CBCL.

All the results of the relationships investigated here were in expected directions. Also as expected, were the significant positive correlations between outcome scores on the CBCL and the BRIEF. The effect sizes of these correlations ranged from medium to large.

Table 17

Correlation Matrix for Factors Affecting Outcome, Academic Outcome, and Behavioural Outcome Variables Obtained on Parent Measures (*N* = 27)

	Variable	1[a]	2	3	4[b]	5	6[a]	7[c]	8[c]	9[c]	10[c]	11[c]	12[c]
1.	Premorbid functioning[a]	-	-	-	-	-	-	-	-	-	-	-	-
2.	Time since injury	-.212	-	-	-	-	-	-	-	-	-	-	-
3.	Age at injury	.032	-.257	-	-	-	-	-	-	-	-	-	-
4.	Family environment[b]	.188	.192	.011	-	-	-	-	-	-	-	-	-
5.	SES	-.151	-.040	.108	-.407*	-	-	-	-	-	-	-	-
6.	Academic outcome[a]	.144	.314	-.011	.392*	.060	-	-	-	-	-	-	-
7.	CBCL Total Problems[c]	.032	.028	.022	.793**	-.045	.383*	-	-	-	-	-	-
8.	CBCL Internalizing problems[c]	-.158	-.052	.032	.629**	-.097	.056	.873**	-	-	-	-	-
9.	CBCL Externalizing Problems[c]	.076	.029	.033	.792**	-.061	.423*	.933**	.800**	-	-	-	-
10.	BRIEF GEC[c]	.348*	-.063	.107	.658**	-.024	.615**	.768**	.455*	.813**	-	-	-
11.	BRIEF BRI[c]	.320	-.048	.090	.681**	-.151	.549**	.819**	.557**	.861**	.945**	-	-
12.	BRIEF MI[c]	.254	-.054	.099	.600**	.068	.661**	.672**	.345*	.727**	.962**	.822**	-

Note. SES = socioeconomic status; CBCL = Child Behavior Checklist; BRIEF = Behavior Rating Inventory of Executive Function; BRI = Behavior Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite; Statistics presented are Pearson correlation coefficients (*r*) unless otherwise stated. All tests are 1-tailed.

[a]Statistics presented are Spearman correlation coefficients (r_s). [b]*n* = 25. [c]*n* = 26.

**p* < 05. ***p* < .01.

Teacher measures. The second correlation matrix, presented in Table 18, show the correlations between factors affecting outcome (i.e., family environment, SES, time since injury, age at injury, and premorbid functioning), academic outcome, and behavioural outcome obtained on the teacher measures (CBCL Total Problems, CBCL Internalizing Problems, CBCL Externalizing Problems, BRIEF GEC, BRIEF MI, and BRIEF BRI. The premorbid functioning variable used in the correlation matrix is that which has been previously described.

Correlations with premorbid functioning. There were significant positive correlations between premorbid functioning and family environment; and between premorbid functioning and academic outcome. The effect sizes of both correlations are large (r_s = .540; r_s = .565) and suggest that poorer premorbid functioning is associated with both greater stress in the family environment and poorer academic outcome.

Other significant correlations. There was a significant strong negative relationship between age at injury and Externalizing Problems on the CBCL (*r* = -.625), and age at injury and Total Problems on the CBCL (*r* = -.633). These two correlations suggest that the younger the age at injury, the greater the degree of externalizing problems and behavioral problems as assessed on the Total Problems of the CBCL, respectively. There was a medium significant positive correlation between SES and MI on the BRIEF (*r* = .481), suggesting that the more material and financial resources there are in the home, the greater the degree of executive function problems in terms of metacognition for example, working memory and organizing materials. There is a strong positive relationship between family environment and academic outcome (r_s = .571), and between family environment and CBCL Internalizing Problems (*r* = .561). These two correlations suggest that increased stress in the family environment is associated with poorer academic outcome, and greater internalizing problems. The scores on the CBCL and BRIEF were all significantly positively correlated as is expected.

*Removal of outliers***.** Due to the small sample included in this matrix (*N* = 13), it is likely that these results are influenced by the presence of outliers. Outliers were present for the variables of premorbid functioning, age at injury, SES, and CBCL Externalizing Problems. Indeed, after removal of outliers, the following correlations were no longer significant: premorbid functioning and family environment, premorbid functioning and academic outcome, age at injury and CBCL Externalizing Problems, and SES and BRIEF MI. After removal of outliers, premorbid functioning and age at injury were significantly positively correlated (large effect size; r_s = -.561), which suggests that greater evidence of poor premorbid functioning is associated with an younger age at injury. The converse would then also be true. The significant relationship between age at injury and CBCL Total Problems, remain, as do the significant correlations between the behavioural outcome scores.

Table 18

Correlation Matrix for Factors Affecting Outcome, Academic Outcome, and Behavioural Outcome Variables Obtained on Teacher Measures (*N* = 13)

	Variable	1[a]	2	3	4	5	6[a]	7	8	9	10	11	12
1.	Premorbid functioning[a]	-	-	-	-	-	-	-	-	-	-	-	-
2.	Time since injury	-.118	-	-	-	-	-	-	-	-	-	-	-
3.	Age at injury	-.447	-.285	-	-	-	-	-	-	-	-	-	-
4.	Family environment	.540*	.145	-.148	-	-	-	-	-	-	-	-	-
5.	SES	.018	.271	.003	-.146	-	-	-	-	-	-	-	-
6.	Academic outcome[a]	.565*	-.355	.023	.571*	-.280	-	-	-	-	-	-	-
7.	CBCL Total Problems	.413	.278	-.633*	.330	.232	-.121	-	-	-	-	-	-
8.	CBCL Internalizing Problems	.432	.204	-.266	.561*	.024	.169	.794**	-	-	-	-	-
9.	CBCL Externalizing Problems	.411	.249	-.625*	.288	.251	-.194	.876**	.559*	-	-	-	-
10.	BRIEF GEC	.179	.279	-.090	.314	.434	-.097	.720**	.626*	.557*	-	-	-
11.	BRIEF BRI	.321	.429	-.065	.366	.281	-.087	.705**	.644**	.571*	.922**	-	-
12.	BRIEF MI	.140	.174	-.133	.252	.481*	-.102	.701**	.573*	.538*	.973**	.810**	-

Note. SES = socioeconomic status; CBCL = Child Behavior Checklist; BRIEF = Behavior Rating Inventory of Executive Function; BRI = Behavior Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite; Statistics presented are Pearson correlation coefficients (*r*) unless other stated. All tests are 1-tailed.

[a]Statistics presented are Spearman correlation coefficients (r_s).

*p < .05. **p < .01.

Part 2: The specific role of premorbid functioning

Independent samples *t*-tests. Table 19 presents the results of independent samples *t*-tests on factors affecting outcome (age at injury, time since injury, SES and family environment) and demographic variables. The table shows that the groups that is, Premorbid Problems vs. No Premorbid Problems, were relatively homogeneous on all these variables. The results show that there was no significant difference between the groups on the variables of age at injury, time since injury, SES and family environment. There was also no significant difference between the groups on the demographic variables of age at assessment. The Premorbid Problems group (male: female; 8: 4) and the No Premorbid Problems group (male: female; 9: 6) did not differ significantly in terms of sex distribution, χ^2 (1,27) = 1.342, p = .247. There was one outlier in each group on the SES variable. The between-group difference for SES remained non-significant after removal of the two outliers (see Appendix H).

Table 20 displays the results of the independent samples *t*-tests performed on the academic outcome data, as well as the GEC, BRI and MI scores on the BRIEF parent version and the Total Problems, Externalizing Problems and Internalizing Problems scores on the CBCL parent version.

There were no statistically significant differences between the two groups on the behavioural outcome measures of CBCL Internalizing Problems, CBCL Externalizing Problems, CBCL Total Problems, and the BRIEF MI. This data suggests that in this sample, problems with behavioural and emotional regulation, as well as with problem-solving and behaviour monitoring abilities, did not vary significantly between the Premorbid Problems group and the No Premorbid Problems group. Important to note, however, is that the between-group difference in BRIEF MI scores bordered on statistical significance (p = .050). There was a medium effect size for this comparison (r = .330), which implies that with a larger sample size, there may be a significant difference in BRIEF MI scores across the two groups. There was also no statistically significant difference between the groups in terms of academic outcome.

The between-group comparison revealed a statistically significant difference in GEC scores on the BRIEF, and in BRI scores on the BRIEF. In both cases, the Premorbid Problems group scored higher than the No Premorbid Problems group. There was a medium effect size associated with each of these comparisons. This indicates that there is greater reported executive dysfunction in terms of behaviour and emotional regulation in children who have evidence of premorbid problems. There were no outliers on any of the outcome measures

Table 19

Independent Samples t-test on Factors Affecting Outcome and Demographic Variables (*N* = 27)

	Premorbid Problems (*n* = 12)			No Premorbid Problems (*n* = 15)			Test Statistics		
Variable	*n*	Range	*M* (*SD*)	*n*	Range	*M* (*SD*)	*df*	*t*	*p*
Age at injury[a]	12	80-144	114.17 (13.30)	15	78-152	114.07 (25.12)	25	-.012	.991
Time since injury[a]	12	15-52	32.25 (12.79)	15	16-70	41.20 (16.19)	25	1.562	.131
SES[b]	12	7-16	11.67 (2.42)	15	7-17	12.47 (2.64)	25	.810	.425
Family environment[c]	11	200-368	295.91 (52.80)	14	125-367	271.50 (48.56)	23	-1.201	.242
Age at assessment[a]	12	118-194	144.5 (23.80)	15	115-195	157.07 (22.80)	25	1.396	.175

Note. Means are presented with standard deviations in parentheses. SES= socioeconomic status. All tests are 2-tailed.
[a]Age at injury, time since injury and age at assessment are presented in months. [b]Aggregate score taken from the parent information and asset index form. [c]Total Stress score derived from the PSI.

Table 20
Independent Samples t-test on Academic and Behavioural Outcomes using Parent Measures (*N* = 27)

	Premorbid Problems (*n* = 12)			No Premorbid Problems (*n* = 15)			Test Statistics			
Variable	*n*	Range	M (*SD*)	*n*	Range	M (*SD*)	*df*	*t*	*p*	*r*
CBCL										
Total Problems	12	47-88	69.83 (12.01)	14	54-77	67.36 (7.82)	24	-0.631	.267	.128
Internalizing Problems	12	50-89	66.50 (11.81)	14	48-81	67.93 (8.26)	24	0.362	.361	.074
Externalizing Problems	12	46-88	68.58 (13.57)	14	46-80	64.14 (10.38)	24	-0.945	.177	.189
BRIEF										
GEC	11	55-97	75.73 (13.57)	15	47-90	64.40 (13.78)	24	-2.084	.024*	.391
BRI	11	50-101	70.09 (17.99)	15	45-94	64.47 (14.73)	24	-2.279	.016*	.422
MI	11	56-87	70.64 (10.50)	15	45-83	62.73 (12.38)	24	-1.712	.050	.330
Academic Outcome	12	-1.15-1.18	0.14 (9.88)	15	-0.15-1.18	-0.11 (0.73)	25	-0.836	.206	.165

Note. Means are presented with standard deviations in parentheses. CBCL = Child Behavior Checklist; BRIEF = Behavior Rating Inventory of Executive Function; BRI = Behavior Regulation Index; MI = Metacognition Index; GEC = Global Executive Composite All tests are 1-tailed. The *r* value presents an estimate of effect size.
**p* < .05.

Chapter 4: Discussion

In this dissertation, I aimed to contribute to the pTBI outcome literature by exploring factors associated with outcome in group of school-going South African children who had sustained a severe TBI. I did so in two ways. In part one of this dissertation I explored some of the commonly reported factors affecting outcome after TBI, namely: premorbid functioning, time since injury, age at injury, family environment, and SES, and the influence of these factors on academic and behavioural outcome. In the second part of the dissertation, I focused specifically on the role of premorbid functioning on academic and behavioural outcome. I tested two hypotheses in this part of the dissertation: 1) participants with evidence of premorbid problems will have a poorer academic outcome than those with no evidence of premorbid problems, and 2) caregivers will report participants with evidence of premorbid problems as having a poorer outcome than those with no evidence of premorbid problems on behavioural measures. I will elaborate on these aims and their findings, and how they compare to that of relevant published literature. Finally, the limitations of the study and directions for future research are discussed, and the significance of the findings of the current study is put forward.

Part 1: Investigation of the Factors that Influence Academic and Behavioural Outcome after TBI

The aim of this part of the study was to explore the relationship between commonly reported factors that influence outcome after TBI, and academic and behavioural outcome. I will first present and discuss the descriptive results relating to the academic and behavioural outcome of the sample. The descriptive results illustrates what the outcome of the participants was at the time of their recruitment and also therefore provides a backdrop to the subsequent analyses. I will then present and discuss the findings of the correlation analyses regarding the relationships between the factors affecting outcome and the outcome variables.

Descriptive results.

Academic outcome. Two thirds (66.66%, 18/27) of the sample were, at the time of assessment, receiving some form of remedial or special needs education or were in the process of being placed in remedial or special needs education post TBI. Thus, there were more children requiring some form of specialized education following their TBI than those who were integrated back into mainstream schooling. This is consistent with literature on children with TBI being reintegrated into the schooling system; that the need for specialized educational services increases in children with TBI (Donders, 1994; Ewing-Cobbs, Fletcher, Levin, Iovino, & Miner, 1998; Kinsella et al., 1997; Savage et al., 2005). Taylor et al. (2003) found that 62% of the children with severe TBI in their sample were in programmes that catered for special education needs, even several years after their injuries. Placement in these programmes occurred soon after injury. Similar high rates were reported in a study by Kinsella et al. (1997), where 70% of children with severe TBI required special needs intervention or attended school part-time after their injury; while Donders (1994) reported a need for special education in over 48% of his sample, which represented a 40% increase over and above those that used these services prior to their injury. In the current study, there was a three-fold increase (n = 6; n = 18) from before to after the TBI in the number of children using special education services. More than half (59.26%, 16/27) of the sample had to repeat at least one grade after returning to school post injury. There was a four-fold increase in the number of children who had to repeat one or more grades from before to after the TBI. Only one of the participants that repeated a grade premorbidly, also repeated a grade after their TBI. This participant was reported as having cerebral palsy prior to his TBI and premorbidly attended a special needs school.

The reasons for these adverse academic outcomes are varied. The cognitive deficits associated with TBI, including difficulties in attention, memory, executive function, and essential skills such reading and writing, can impact on academic performance (Arroyos-Jurado, Paulsen, Merrell, Lingren, & Max, 2000; Donders, 1994; Ewing-Cobbs, Fletcher, Levin, Iovino, & Miner, 1998; Hawley, 2004; Taylor, 2010). Other factors such as behavioural impairments and absence from school may also play a role (Babikian & Asarnow, 2009; Ewing-Cobbs et al., 1998). In some cases, the child missed out on weeks or months of school during their recovery period, particularly when the TBI occurred during the school terms. Frequently, the child also missed periods of school to attend doctors' appointments. There are also a whole host of other factors affecting outcome, which I will further discuss.

It is worth mentioning that perusal of all accessible case files a year after participation in this study revealed that two of the participants were subsequently placed in a special needs school, and a further one had been recommended for placement (therefore 25.93% of the sample were in a special needs school). This change in academic outcome may reflect an increase of academic problems over the course of time post-injury, which can be expected according to the literature (Arroyos-Jurado et al., 2000; Ewing-Cobbs et al., 1998; Taylor, 2010). However, this delay of placement in special needs schools may also be a reflection of a delayed process of identification of needs, for example when deficits are not immediately evident or are only evident at a later stage when cognitive demands on the child increase. The delay in placement may also be due to a lack of suitable availability for placement, especially when resources are limited (Mayfield & Homack, 2005; Taylor et al., 2003). In South Africa in particular, the delay in placement is often due to it being a lengthy process in terms of bureaucracy and that the number of, and placement in, special needs schools is limited (B. Daniels, personal communication, February 19, 2014).

Behavioural outcomes.

Parent measures. CBCL. Total Problems, Internalizing Problems, and Externalizing Problems were on average in the clinical significant range, with these means being relatively homogenous. Elevated syndrome and DSM-Oriented scale scores, such as, Aggressive Behaviour, Affective Problems, Attention problems, Social Problems, and Conduct Problems, fell in the borderline clinical range. These problems are commonly reported sequelae following severe TBI and reflect difficulties associated with self-regulation of behaviour and emotions (Anderson & Yeates, 2010; Mayfield & Hormack, 2005; Taylor, 2010; Yeates, 2009). There was a high frequency of clinically significant problematic behaviours reported by caregivers of the participants in this study. In terms of the syndrome grouping on the CBCL, caregivers reported a greater frequency of clinically significant internalizing problems than externalizing problems. Internalizing problems may include anxiety, withdrawal, depression, and other emotional problems; while externalizing behaviour problems may manifest as aggression, irritability, disinhibition and rule-breaking (Achenbach & Rescorla, 2001; Dooley, Anderson, Hemphill, & Ohan, 2008; Mayfield & Homack; 2005; Taylor, 2010). This overall high occurrence of problem behaviours is consistent with numerous studies documenting the behavioural impairments in children after TBI. Such behavioural impairments may persist over time with varying patterns, which may include both internalizing and externalizing problems (see, e.g., Anderson et al., 2001; Fay et al., 2009; Fletcher et al., 1990; Kinsella et al., 1999; Mayfield & Homack, 2005; Savage et al., 2005; Schachar et al., 2004; Taylor, 2010; Yeates & Taylor, 2006).

On average, caregivers rated aggressive behaviours as being the most problematic. Dooley et al. (2008) reported that aggressive behaviours are likely a result of anger and distress in response to one's injury and deficits but may also, however, be associated with emotional lability and decreased frustration tolerance.

BRIEF. In more than half of the sample, caregivers reported clinically significant problems across a range of executive functioning subdomains. Inspection of the means show that apart from Initiate and Organization of Materials, all other indices on the BRIEF fell into the clinical range. Working Memory (i.e., the ability to manipulate information in the mind) appeared to be the most problematic subdomain of executive function.

The high frequency and proportion of executive dysfunction in the sample may reflect damage to the frontal areas of the brain in which the neural circuits mediating execution functioning are located (Bamdad et al., 2003). The results are consistent with literature documenting executive function deficits in children who have sustained a TBI (Anderson et al., 2002; Bamdad et al., 2003; Levin & Hanten, 2005; Yeates, 2010; Yeates et al., 2005). Specifically, working memory is crucial to various cognitive functions and is particularly susceptible to the effects of a TBI (Hillary, Genova, Chiaravalloi, Rypma, & DeLuca, 2006).

Problems with the behavioural and emotional regulation, such as assessed on the CBCL and the BRIEF, may occur as a direct result of damage to the brain's regulatory circuits and the associated cognitive deficits. In particular, damage sustained from a TBI to the frontal lobes, which are vulnerable because of their anatomical location, can result in deficits with initiating and self-monitoring behaviour, and inhibiting responses. Behavioural dysfunction can also be an indirect consequence in response to difficulties in adjusting to deficits and resuming previous activities, particularly as children become more aware of their deficits, and as a reaction to the accident and the response of their family. Behavioural dysfunction, as well as disturbances in social functioning, may then become evident and persistent (Andrews, Rose, & Johnson, 1998; Arroyos-Jurado et al., 2000; Bamdad et al., 2003; Max et al., 1999; Mayfield & Homack, 2005; Yeates, 2009).

Even in the absence of physical impairments, the child may be aware of a change in their abilities and therefore feel increased burden in response to such changes after their TBI, especially if they are unable to perform at their previous level. They may become more aware of their deficits upon their return to school and other activities as this places increased neurocognitive and psychosocial demand on them. In turn, the child may experience difficulties in coping at school and dealing with social interactions, which further leads to problematic behaviours such as withdrawal and aggression. Parental stress and expectations of recovery also play a role in contributing to difficulties in behaviour. In dysfunctional families where there is increased stress, there may be less structure and consistency, with expectations regarding behaviour and functioning that are not very clear to the child. The response of the family plays a role particularly when recovery is less obvious. When the recovery is less obvious or visible, this may lead to caregivers becoming more discouraged and less hopeful. Therefore, the quality of support given to the child by their caregivers, and the adjustments made for their behaviour by caregivers, may be affected (Chapman, et al., 2010; Donders & Strom, 2000; Mayfield & Homack, 2005; Taylor et al., 2001; Yeates et al., 2001).

Teacher measures. CBCL. The means of the broad syndrome groups (i.e., Internalizing Problems and Externalizing Problems), as well as the Total Problems grouping fell within the borderline clinical range or clinical range. More specifically, within the Total

Problems scale, the Externalizing Problems scale on average fell in the clinical range; while the Internalizing Problems scale on average fell into the borderline clinical range. More than half of the participants scored within the clinically significant range on both the Externalizing and Internalizing Problems scales.

BRIEF. The outcome scores obtained on the BRIEF teacher version suggest, on average, that problems are experienced across all aspects of executive functioning in the school setting. The means of all indices fell within the clinically significant range. However, the most problematic areas of executive functioning, descriptively, are those of working memory, and the ability to shift between problem-solving strategies.

Comparison with parent measures. CBCL. Inspecting the means reveal that teachers rated participants as having fewer problematic behaviours than caregivers on the CBCL, apart from on the subscale of Rule-breaking Behaviour. The Internalizing Problems group was descriptively lower than the Externalizing Problems group on the teacher CBCL, with the inverse on the parent CBCL. The differences in teacher and caregiver ratings are consistent with studies looking at the inter-rater agreement on such measures. The differences can be explained by the informants experiencing different expressions of the behaviour when in different environments (Achenbach & Rescorla, 2001; Youngstrom, Loeber, & Stouthamer-Loeber, 2000). It also may be that the classroom environment provides more opportunity for problematic externalizing behaviours to become evident, such that there are more rules and regulations that need to be followed. In the same way, problematic internalizing behaviours may be more evident to caregivers at home when there is less structure present or when the child experiences new situations (such as going to a new place). Of the internalizing and externalizing problem behaviours assessed on the CBCL, it was aggressive behaviours that teachers and caregivers rated as the most problematic type of behaviour. This rating was consistent with caregivers who also reported aggressive behaviours to be the most problematic. Inspecting the means also showed that Somatic Complaints, part of the Internalizing Problems grouping, appeared to be comparatively lower on the teacher version of the CBCL, than the parent version, with respect to scales on the Internalizing and Externalizing Problems syndrome grouping. It may be that children are more comfortable and open to expressing to their caregivers, rather than their teachers, that they are experiencing somatic complaints such as stomachache, for example.

BRIEF. Teachers rated participants at having on average more problems across all of the BRIEF indices than caregivers. This discrepancy may be explained by the fact that deficits in executive functioning may be more evident in the classroom environment where learning and classwork place increased demand on these abilities (Mayfield & Homack, 2005).

Intraclass correlations. The intraclass correlations between teacher and parent raters on the respective versions of the CBCL are moderate. These moderate correlations are not surprising since the different raters experience the manifestations of the child's behaviour in different settings. The behaviour may be in response to situations unique to those particular settings, such that certain behaviours for example, conduct problems as a whole, may be more evident to parents in the home environment, and less so to teachers within the more structured classroom environment. Another example is that rule-breaking, specifically, may be more evident to teachers in the school environment when there are more rules in place that the child needs to adhere to than at home (Achenbach & Rescorla, 2001). The intraclass correlations between teacher and caregiver scores on the BRIEF indices, were, however, larger, suggesting that executive function deficits in the subdomains as specifically assessed by the BRIEF are more consistent across the home and school environment, perhaps due to similar cognitive demands placed on child in these settings (e.g., when completing schoolwork in class or homework at home).

Correlational analyses. A vast number of factors affecting outcome, and measures of outcome, are reported in the literature, however, limited studies investigate these in the same sample of participants. In this study, I explored the relationships between the commonly reported factors (premorbid functioning, time since injury, age at injury, family environment, and SES), and variables of academic and behavioural outcome in one sample. I found that family environment significantly correlated with the premorbid functioning variable, as well as with outcome on both parent and teacher measures. I found significant correlations between age at injury and outcome scores on the teacher version of the CBCL. Family environment significantly correlated with SES when using the teacher measures. The outcomes scores significantly correlated with each other when using both parent and teacher versions. There were no significant correlations between time since injury and any of the variables.

Significant correlations.

Premorbid functioning. There were no significant correlations between premorbid functioning and the independent variables.

Parent measures. The positive significant relationship between the BRIEF GEC score and premorbid functioning suggests that greater evidence of poor premorbid functioning is associated with greater executive dysfunction. There was a medium effect size associated with this relationship. The role of premorbid functioning will be further explored in the discussion of the results of second part of the study.

Teacher measures. There were no significant correlations between premorbid functioning and any of the outcome measures.

Time since injury.

There were no significant correlations between time since injury and any other variable.

Age at injury.

There were no significant correlations between age at injury and the independent variables.

Parent measures. There were no significant correlations between age at injury and any of the outcome measures.

Teacher measures. There were a strong negative significant correlations between age at injury (the range being 6.5 - 12.67 years) and Total and Externalizing Problems on the CBCL suggesting that children who were younger at the time of their injury show greater problems in behavioural and emotional functioning, specifically externalizing behaviours such as rule-breaking and aggression. Conversely, those who were older at their age of injury show less of these types of problems. According to Wetherington and Hooper (2010), social and behavioural functioning could be impacted, when injury occurred at a younger age, by impairments resulting from the TBI that have a negative effect on the developmental trajectory of social-behavioral abilities. In addition, the developmental trajectory can also be altered if the TBI occurred during critical periods that is, when there is increased sensitivity of the brain's neural networks to influences from the environment, which can lead to functional consequences for the child (Anderson et al., 2011). Therefore teachers are more likely to be seeing, in the classroom environment, the consequences of altered development when the TBI occurred at a younger age, or less likely if the TBI occurred at an older age, where perhaps the child was able to develop better adaptive coping skills (Wetherington and Hooper, 2010).

Family environment. The results of the correlational matrix, using the full sample (n = 27), showed a negative significant relationship between family environment and SES, where greater stress in the family environment is associated with a lower SES. The relationship between family environment and SES had a medium effect size. This relationship is not surprising, since families with lower SES may have fewer resources, economically or in terms of access to services, to assist in the child's recovery. Such families may therefore feel burdened by the stressors following the accident due to their disadvantaged circumstances

(Levin, 2004; Taylor et al., 2002). Indeed, caregivers reported the extra economic burden associated with the injury and subsequent doctors appointments to be stressful.

Parent measures. Family environment, as measured by the Total Stress score on the PSI was significantly correlated with all of the outcome measures (academic; Total, Internalizing and Externalizing Problems on the CBCL; and the GEC, BRI and MI on the BRIEF). The direction of the relationships between family environment and the outcome variables were positive, with large associated effect sizes on behavioural outcomes and a medium effect size for academic outcome. Overall, these relationships between family environment and outcome suggest that the greater the stress within the family environment, the poorer the child's outcome both academically and behaviourally.

The relationships between family environment and academic outcome, and behaviour outcomes, are consistent with literature in that the behavioural and academic consequences of TBI, in addition to resulting cognitive and psychosocial impairments, lead to parental distress (Anderson et al., 2001; Prigatano & Gray, 2007; Taylor et al., 2001).

The relationships between family environment and academic outcome, and behavioural outcome, is also consistent with literature documenting the important role of family environment in predicting outcome after TBI (Anderson et al., 2001; Taylor et al., 2001; Taylor et al., 2002; Yeates, 2010; Yeates et al., 1997). Kinsella et al. (1999) also found positive correlations between child behaviour ratings and family functioning ratings and suggested that families reporting greater behaviour problems in their children, report greater dysfunction themselves. Such dysfunctional families may have less structure and consistency, with unclear expectations for the child regarding their behaviour (Chapman, et al., 2010).

Given the reported relationships between family environment and both academic and behavioural outcome, the high levels of stress reported by the caregivers is concerning. Of the sample, 60% of caregivers reported an overall level of stress within what is considered to be the critical range. A greater frequency of critical range scores was reported for the Child Domain as compared to that of the Parent Domain. This greater frequency of critical range Child Domain scores suggests that in this sample, more stressors associated with behavioural and emotional problems in children are experienced in the family environment than stressors associated with parental dysfunction, such as depression or feelings of incompetence.

More specifically, the PSI's Acceptability scale had the greatest frequency of critical range scores, indicating many of the participants were not meeting their caregivers' expectations in terms of the participants' emotional and intellectual characteristics. In other words, emotional functioning and intellectual abilities are below that which the caregiver thought they should or would be at. That the participants were not meeting the expectations of their caregivers, may be a reflection of the caregivers' struggle in coming to terms with their children's deficits and the subsequent challenges that are brought about by the TBI. It may also be because children with TBI may be assumed to have fully recovered from their injuries when no obvious physical deficits are seen (Hawley, 2004). Therefore, when the child is not able to function and cope as well as the caregiver may have expected, this can lead to the caregivers experiencing increased feelings of distress, disappointment, and guilt particularly as they start feeling less hopeful for that their child will recover completely (Mayfield & Homack, 2005).

In addition to the results obtained on this formal measure, anecdotal information obtained during assessment provided supportive evidence to the challenges facing this population of children with severe TBI and their families. It was quite clear that caregivers were concerned about the changes they have seen in their children, concerned about the children's academic standing, and worried about their care. For example, one mother stated that she worried about how her child will survive when she is no longer around. Another mother was concerned that even though her child was struggling academically, the school promoted the child to the next grade level every year. This is commonplace in South African schools – weak learners may under certain conditions be promoted to higher grades without fulfilling the pass requirement. For example, according to the Department of Basic Education (2013), learners not meeting the subject grade requirements for promotion to the subsequent grade can be nevertheless be progressed. Learners may be progressed under these circumstances to prevent them being retained for more than four years in a phase. The phases are Foundation Phase (Grades 1-3), Intermediate Phase (Grade 4-6), and Senior Phase (Grade 7-9).

Concern regarding academics is a shared worry among parents. For example, Prigatano and Gray (2007) reported problems with school performance as being a major source of stress. That poor academic performance may be contribute to increased parental stress, supports the demonstrated relationship in the current study between family environment and academic outcome in that the poorer the academic outcome, the greater the stress within the family environment.

The demonstrated relationships between family environment and outcome also implies that decreased stress is associated with better outcome. Therefore, attempting to address and lower the stress within the family environment following TBI should be of utmost importance. The importance of a low-stress family environment is supported by literature, for example, demonstrating the association between favorable outcomes and factors such as social support and a cohesive family environment (Anderson et al., 2001).

Teacher measures. Family environment was significantly correlated with the outcome scores of Internalizing Problems on the CBCL, academic outcome, and premorbid functioning. The direction of the relationships between family environment and Internalizing Problems, academic outcome, and premorbid functioning were positive, with large effect sizes. Higher scores on the Internalizing Problems syndrome group, academic outcome and premorbid functioning variables indicate greater dysfunction. Therefore, these reported relationships suggest that children with greater stress in the family environment are likely to have more internalizing behaviour problems evident in the classroom environment, a poorer academic outcome, and that it is likely that they had poorer functioning prior to their TBI. Conversely, children with less stress in the family environment are less likely to display internalizing behaviour problems in the classroom, have a better outcome academically, and have had a better level of functioning (i.e., less premorbid problems as described above) prior to their TBI.

SES. I have already mentioned and discussed the significant correlation between family environment and SES. There were no other significant correlations between the independent variables and SES.

Parent measures. There were no significant correlations between SES and any of the outcome measures.

Teacher measures. A medium significant positive correlation between SES and MI on the BRIEF indicated that children in homes with more material and financial resources have increased reported problems with aspects related to problem-solving and behaviour monitoring, or conversely less material and financial recourses and less reported problems with aspects related to problem-solving and behaviour monitoring, evident in the classroom environment. The direction of this relationship is surprising, in light of literature suggesting disadvantaged circumstances are associated with deficits of behavioral measures and exacerbate the effects of TBI on behavioural functioning and skill development (Fay et al., 2009; Taylor et al., 2002). An explanation for the significant correlation could be that children with a higher SES may be attending a better resourced school, where there may potentially be an increased need for metacognition. Therefore one may be more likely to see problems with such cognitive functioning within the classroom environment.

Significant correlations between the outcome measures.

Parent measures. With regards to outcomes, both the Total Problems and Externalizing Problems groups showed a positive significant relationship with academic outcome, with medium effect sizes. These relationships suggest that children with increased problems associated with behavioural and emotional functioning, specifically showing more rule-breaking or aggressive behaviour, are more likely to be requiring or receiving remedial or special needs services, or are more likely to have to repeat grades subsequent to their injury. These relationships followed the expected directions consistent with literature that describes the impact of behavioural impairments on school performance (Babikian & Asarnow, 2009; Keenan & Bratton, 2006). The relationship between academic outcome and Internalizing Problems was not significant, and had a small effect size. The difference in the relationship between internalizing and externalizing behaviour problems with academic outcome is similar to that which Nelson, Benner, Lane, and Smith (2004) reported. These authors found that among students who had emotional or behavioural disorders, the students that exhibited externalizing behavioural problems were more likely than students with internalizing behavioural problems to have deficits in academic achievement. A longitudinal analysis looking at internalizing and externalizing problem behaviour, Malecki and Elliot (2002) reported negative correlations between problem behaviors and academic achievement (i.e., greater problem behaviours were associated with poorer academic achievement). While the construct of problem behaviours was said to include both internalizing and externalizing problems, the authors did not differentiate this in relation to academic achievement in their analyses. An explanation for the difference in academic outcome with regards to internalizing and externalizing behaviour problems may be that the externalizing behaviour problems may have more of an influence than internalizing behaviour problems with regards to coping with and progressing academically. Externalizing behaviours may interfere with the learning process more than internalizing behaviours.

Strong, positive, significant associations between academic outcome and the outcome scores (GEC, BRI, MI) on the BRIEF, suggests that children with increased executive dysfunction, including problems with regulation of behaviour and abilities related to problem solving, are more likely to be receiving or in need of more remedial help or who have failed grades subsequent to their injury. The relationships between academic outcome and behavioural outcome, as assessed by the BRIEF, would be expected given the role that executive functions play in goal-directed behaviour and problem solving. It is consistent with literature documenting the role of executive functions in the learning process and in academic achievement (Anderson et al., 2002; St Clair-Thompson & Gathercole, 2006). In their sample of children and adolescents with Attention-Deficit Hyperactivity Disorder (ADHD), St Clair-Thompson and Gathercole (2006) found that there was increased risk of repeating grades, learning disabilities and poor academic achievement amongst those with deficits in executive functioning.

The significant positive relationships between the CBCL and BRIEF were expected because (a) related scales on the CBCL and BRIEF should correlate with each other as a reflection of the construct validity of these measures; and (b) the Internalizing Problems and Externalizing Problems scales, and the BRI and MI form part of the overall outcome scores (Total Problems and GEC, respectively) obtained on these measures. The construct validity of the CBCL and BRIEF can be explained by the role of executive function in behavioural regulation, therefore behaviour rating scales such as the CBCL that measure aspects of behaviour impacted on by executive functions, should correlate with related indices on the BRIEF (Gioia et al., 2000).

Teacher measures. The scores on the CBCL and BRIEF were all significantly positively correlated. The significant positive correlations are expected for the reasons as detailed above for the parent measures.

These findings highlight factors that should be considered important indicators as to outcome after TBI when the child is reintegrated into the classroom environment. It may assist in identification of needs for intervention purposes (Wetherington and Hooper, 2010).

In summary, the discussion in part one focused on the academic and behavioural outcomes in the sample, and explored its relationship with factors affecting outcome. The results show elevated problems with academic outcome, and behavioural and executive functioning in the sample, and that these outcomes are experienced differently by informants across different settings. The results also show that in this sample, factors such as family environment and premorbid functioning are particularly important with regards outcome in the home environment; while factors such as age at injury, family environment and SES play more of a role within the classroom environment for these participants.

Part 2: The Specific Role of Premorbid Functioning

The aim of this part of the study was to investigate the role of premorbid functioning in academic and behavioural outcome. The sample was divided into two groups where one group had evidence of premorbid problems (as indicated by grade failure, attendance at a special needs school, academic and behavioural problems, developmental problems, previous HIs or TBIs, etc. all premorbidly), while the other group did not have evidence of premorbid problems. I investigated whether the two groups, that is, the Premorbid Problems group and the No Premorbid Problems group, differed in terms of academic and behavioural outcome. The findings of these investigations will be discussed.

The groups did not differ on other frequently reported factors predicting outcome (i.e., time since injury, age at injury, SES and family environment) other than premorbid functioning. These variables are commonly reported factors affecting outcome after TBI (see, e.g., Anderson et al., 1997; Anderson et al., 2001; Anderson et al., 2004; Anderson et al., 2005; Anderson et al., 2009; Babikian & Asarnow, 2009; Giza & Prins, 2006; Jaffe, Polissar, Fay, & Liao, 1995; Prigatano & Gray, 2008; Taylor et al., 2001; Taylor et al., 2002; Spencer-Smith et al., 2011; Taylor, 2010; Yeates et al., 1997; Yeates et al., 2004; Yeates, 2010). Therefore, it was important to have the groups homogenous as far as possible in terms of these factors to explore the specific role of premorbid functioning on the assessed outcomes. There were also no significant between-group differences on the demographic variables of sex and age at assessment.

The role of premorbid functioning in academic outcome.

As previously discussed, the number of participants using special education services increased three-fold from before to after the TBI. This increase, took into account premorbid academic problems (even when remedial services were not used) and indicates that for 44.44% of the sample (*n* = 12), the use of remedial services or special needs education was required only after the TBI.

Between-group comparison on academic outcomes. For hypothesis one of this part of the study, I predicted that participants with evidence of premorbid problems would have a poorer academic outcome than those with no evidence of premorbid problems. This hypothesis was not confirmed. There was no statistically significant difference between the groups in terms of academic outcome. The between-group difference had a small effect size. Nevertheless, inspecting the means show that the Premorbid Problems group had a larger academic outcome score compared to the No Premorbid Problems group indicating that the on average, participants with evidence of premorbid problems had a poor academic outcome and thus repeated a grade following their injury and/or had greater use of remedial or special education services. However, participants from both groups obtained the same range of academic outcome composite scores indicating varying levels of academic outcome within each group. The non-significant finding is inconsistent with literature reporting premorbid academic ability to be a significant predictor of academic achievement (Arroyis-Jurado et al., 2000; Ewing-Cobbs et al., 1998; Jaffe et al., 1993).

Literature also documents, however, that subsets of children with severe TBI show significant neurocognitive deficits, while other children with severe TBI appear to recover remarkably well (Abelson-Mitchell, 2007; Babikian & Asarnow, 2009; Fay et al., 2009). It may be that this discrepancy in outcome reflects the heterogeneity of injuries sustained or the influence of the various factors affecting outcome. While this study controlled for various factors, neurosurgical variables and the type (i.e. nature) of injury were not investigated due to lack of availability of data. In addition, a variety of factors, other than premorbid functioning, for example, cognitive deficits and absence from school during the acute and recovery period, also play a role in determining academic outcome, as discussed in part one of this study. This discrepancy in recovery from severe TBI may explain the range of academic outcomes across both groups.

The role of premorbid functioning on behavioural outcome.

Between-group comparison on behavioural outcomes. For hypothesis two of this part of the study, I predicted that caregivers would report that participants with evidence of premorbid problems as having a poorer outcome than those with no evidence of premorbid problems on behavioural measures.

The hypothesis was confirmed for the GEC and BRI, but not for the MI, of the BRIEF. The hypothesis was not confirmed for the Internalizing problems, Externalizing Problems, and Total Problems scales of the CBCL.

There was a significant between-group difference on both the BRIEF GEC and BRI with the Premorbid Problems group obtaining higher scores, which were also in the clinically significant range, than the No Premorbid Problems group. These significant between-group differences have a medium effect size and indicate that children with premorbid problems are more likely to exhibit greater executive dysfunction, particularly with regards to set-shifting, and regulating emotions and behaviour through inhibitory mechanisms (Gioia et al., 2000). The difference in outcome between the groups is consistent with literature documenting poorer behavioural and functional outcomes in children with premorbid dysfunction (Anderson et al., 2001; Donders & Strom, 2001; Farmer et al., 2002; Fay et al., 2009; Swartz et al., 2003). It may be that premorbid problems exacerbate deficits in executive function. One mechanism may be that premorbid problems increase the vulnerability of the brain to the effects of TBI, in that children with premorbid problems have a lowered threshold for the expression of TBI-related deficits. Vulnerability related to impaired coping with the effects of the TBI or having fewer resources for compensating for effects of TBI may also account for increased executive dysfunction when premorbid problems are present (Dennis et al., 2007; Schwartz et al., 2003; Spencer-Smith & Anderson, 2010; Taylor, 2010).

There was no significant between-group difference on MI of the BRIEF. However, the between-group difference bordered on significance with a medium effect size, and the means were in the expected directions. With a larger sample size, this comparison may therefore reach statistical significance.

There were no significant differences on the Total Problems broad syndrome group, or on the smaller groupings of Internalizing Problems and Externalizing Problems. Examination of the means revealed that both groups scored in the clinical range on each of these syndrome groupings. The groups were relatively homogenous on Total Problems and Internalizing Problems. With regards to Total Problems and Externalizing Problems, the means were in the expected direction, however, all comparisons on the CBCL were associated with small effect sizes. The results reported here were inconsistent with studies documenting that premorbid functioning plays a role in predicting outcome, such that poorer premorbid behavioural functioning predicts declining behavioural function (Anderson et al., 2001; Donders & Strom, 2001; Fay et al., 2009).

An explanation for the inconsistent results in this part of the study may be that grouping of the participants into Premorbid Problems and No Premorbid Problems groups did not differentiate between participants with premorbid behavioural problems and other premorbid problems such as previous TBIs, poor academic performance or developmental delay. Studies investigating premorbid functioning and outcome often focus on particular pre-injury characteristics, such as behaviour or academic functioning only, or the presence of mood disorders or learning difficulties. Pre-injury data is also obtained through varying measures that differ from those used in this study for example, studies use academic scores, neuropsychological test scores or pre-injury psychiatric history to provide a measure of premorbid functioning (see e.g., Arroyos-Jurado et al., 2000; Donders & Strom, 2001; Farmer et al., 2002; Kinsella et al., 1999; Schwartz et al., 2003).

In summary, the data from the current study did not confirm that participants with evidence of premorbid problems would have a poorer academic outcome than those with no evidence of premorbid problems. The data partially confirmed Hypothesis two with respect to the BRI and GEC of the BRIEF. The findings thus suggest that caregivers reported participants with evidence of premorbid problems as having a poorer outcome in terms of behaviour regulation and overall executive dysfunction on the BRIEF BRI and GEC, respectively, than participants with no evidence of premorbid problems.

Limitations and directions for future research

Study design. There are several limitations in terms of the design of the study. I used an exploratory design in the first part of the study, which included a correlational analysis, and between-groups analyses in the second part of the study to investigate the variables and outcomes of interest. Ideally, a hierarchical model of multiple regression would have been a more appropriate approach to investigating factors affecting outcome using the variables in the current study, as well as additional variables such neurosurgical variables as will be discussed below. This regression model was not implemented due to the small final sample size, due to difficulty in obtaining participants, which in itself was also limited by time constraints. Should a larger sample size be available, the regression model could be used to predict the academic and behavioural outcome variables as described in the current study. To explore the role of premorbid functioning, an interaction of time since injury x premorbid functioning variables could be entered into the model and used to test whether outcome differs between individuals with high premorbid functioning with a long time since injury and individuals with low premorbid functioning with a long time since injury. This interaction is illustrated in Figure 2.

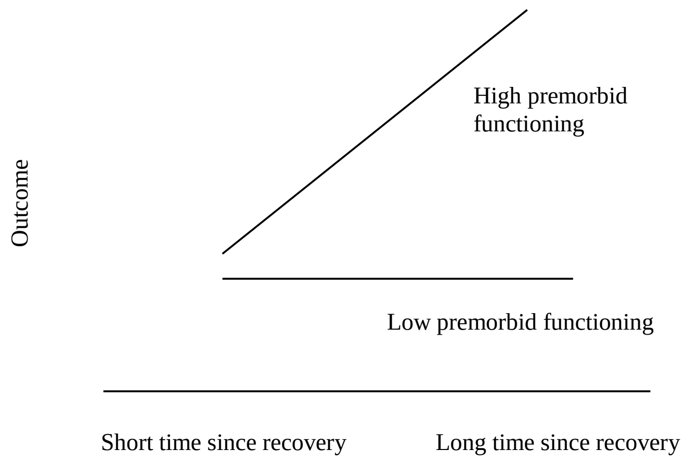

Figure 2. Interaction of time since injury and premorbid functioning.

I originally planned for this study to include a model of change in academic grade performance, which would have formed part of the academic outcome variable. For this model of change, I would have required academic grade results from the time prior to the injury, upon return to school, and at the time of assessment. The model of change was however not achievable due to the lack of complete academic history, in that school test and examination results, from before and after the TBI, were not obtainable for the majority of the sample. As previously mentioned, schools and caregivers alike, often do not keep complete records and there is a lack of record-keeping resources such as computerized records or a centralized database. Ideally, using a model of change in academic outcome, should the necessary data be accessible, would allow for the role of premorbid functioning to be examined, with respect to academic outcome, through a change in performance over three periods of time that is, the period before injury, the initial period upon return to school, and the current point in time. A change in performance could then be determined as a percentage recovery from the initial decline in academic performance and the total decline in academic performance. This model of change is illustrated in Figure 3.

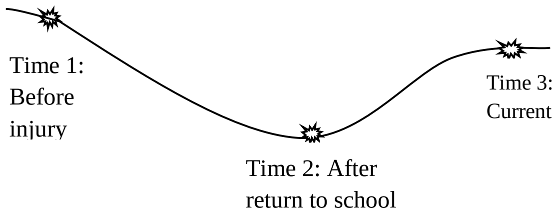

Figure 3. Model of change in academic performance.

While the current study did investigate a large number of factors in one sample, it did not take into account the influence of neurosurgical variables with respect to the intracranial monitoring received by the participants, as the complete data set was not available at the time of analysis. This data should be included in future studies owing to the important role of factors such as $PbtO_2$, ICP and CPP in TBI outcomes (Figaji et al., 2009a; Figaji, 2010; Jiang et al., 2002; Michaud, Rivara, Grady, & Reay, 1992; Schrieff, 2013; Woodward et al., 1999). Another factor that would be useful to include in future studies is that of the nature of injury, where the role of the location, extent and type of injury sustained is investigated, owing to the heterogeneity of pathologies associated with severe TBI (Figaji, 2010). However, detailed information on the nature of each TBI sustained was not available for the sample. It may be that the variation in outcome investigated in a study could be explained, in part, by these injury-related variables. For example, where focal injuries are present, there may be more potential for reorganization and compensation in that a greater region of the brain remains intact, than when more diffuse regions are affected (Anderson et al, 2001; Anderson et al., 2011).

To assist in further teasing apart the role of pre-injury and injury factors in outcome, future studies should including a sample of typically developing children, as well as those with premorbid problems who have not sustained a TBI.

Sample size. One of the main limitations in this study was the small sample size, and thus results should be viewed with caution. The statistical power of the current sample size was insufficient to perform regression analyses. In addition, a lack of power reduces the sensitivity and generalizability of the results. Though many of the effect sizes were in the medium to large range, increasing the sample size in future research will assist in increasing the generalizability of findings and the likelihood of finding real differences in between-group comparisons (Lipsey & Hurley, 1990). In addition, I would aim to increase the sample size in subsequent studies in order to carry out multiple regression analyses.

All reasonable measures were taken in order to expand the sample size in this study. These included checking the hospital's electronic patient information database on at least ten occasions, looking through the relevant medical folders and attending clinics at RXH in the hope of approaching the caregiver in person when they were there for a scheduled appointment. Unfortunately, records were not always complete or up to date, and appointments not always attended. In particular, telephonic contact details were more often than not incorrect or no longer in service. This problem of unreliable and often incomplete records is not uncommon in public hospitals (Naidoo, 2013). Clinical populations are also difficult to access, and added to this, severe TBI accounts for only 10% of TBIs. Therefore, severe TBI populations are considerably difficult to access (Adelson, 2010; Bruns & Hauser, 2003).

Regarding the teacher measures, the number of teacher-raters was half that of the caregivers due to a poor return-rate. Therefore the difference in sample size may play a role in the comparisons between these scores. Teacher measures were extensively followed up over a period of a year through direct visits to the schools, repeated faxing and emailing of measures and numerous phone calls. Teachers who responded reported being extremely busy with marking as well as coaching of extramural activities, while others did not respond at all. Several of the schools did not have available current contact details and were unreachable.

Children who were not currently attending school at the time when they were contacted for participation in the study were excluded, as no post-injury, current academic results would have been available for them. For example, one child that was excluded was not attending school at the time when contact was made as he was awaiting placement in a school for special needs, and he had not returned to his previous school. Another child excluded from this study was awaiting placement in a suitable skills training or ‘day-care’ centre at the time of recruitment, as she was considered low functioning both physically and mentally. The exclusion of children who were not attending school became a limiting factor in the size of the sample. In hindsight, children such as the two described in the examples above, should have been included in this study. The reasons for their exclusion formed part of their academic outcome that is, they were both awaiting placement in an appropriate special needs institution.

Missing data. The results of this study should be viewed with caution in light of missing data. In some cases, forms had to be dropped off with or posted to caregivers, when they were unable to take time off work or transport was problematic. This meant that responding could not be monitored and missing responses had to be followed up on, often unsuccessfully. In addition, details of important developmental history such as milestones and birth weight, particularly in the case of preterm babies, were unknown by some of the caregivers. There were instances where missing data was unavoidable, for example, when responses were deliberately left out at the discretion of the person completing the form since participation was voluntary.

Access to data. Other academic data, such as term and year-end results could not be included in the analyses as school records were incomplete or unobtainable for some of the participants. Exclusion of academic results data from the analyses was due to non-response from the schools, caregivers not having complete records, or schools not having complete records.

Reasonable measures were taken to get hold of these results including contacting the Western Cape Education Department. Schools also do not keep full academic records of current or past learners in many cases. A more comprehensive investigation with the inclusion of school results may identify problem areas specifically in relation to academic premorbid functioning, and assist in determining the role of premorbid functioning on academic outcome. School report results, if obtained, can be used as a more objective, uniform, measure of premorbid functioning in follow-up studies particularly if the results of standard national-based assessments can also be accessed.

Informant report measures. This study made use of parent and teacher self-report measures, which can be regarded as limiting due to issues with response sets, social desirability bias, and retrospective access from memory of the queried behaviours (Andrews et al., 1998; De Los Reyes & Kazdin, 2005; Hunsley, 2009; Williamson, 2006). Obtaining teacher ratings are known to be associated with logistical problems such as a poor return rate of forms (Kinsella et al., 1995; Yeates & Taylor, 2006). In this study, I encountered difficulties in contacting teachers outside of teaching hours. Teachers also reported a lack of available time in which to complete the measures, particularly when nearing or during the examination period. Nevertheless, informant measures were used in this study due to their psychometric properties and their benefits in terms of ecological validity (Anderson et al., 2002; Gioia & Isquith, 2004; Leathem, Murphy, & Flett, 2005; Swartz et al., 2003). Further studies should look to supplement informant measures, for example, by including structured interviews with caregivers and teachers, as well as direct observations of behaviour.

Summary and conclusion

Severe TBI in children often results in various neurocognitive and behavioural deficits. Such deficits may lead to adverse outcomes both academically and in behavioural functioning, and have therefore important implications for learning and educational achievement. I investigated factors influencing outcome, with a specific focus on the role of premorbid functioning, in a group of school-going South African children with severe TBI.

The results show that problems (e.g., increased need for special education services, behavioural and executive function behaviour problems) experienced by this sample of children are consistent with those reported for severe TBI in the literature (Anderson et al., 2001; Babikian & Asarnow, 2009; van't Hooft, 2010). In addition, the results highlight the importance of factors such as family environment and premorbid functioning in outcome.

The current study therefore adds weight to the advocacy for increased awareness in terms of identifying children, and indeed families, that are at greater risk for dysfunction and poorer outcomes following a TBI. This in turn will assist in informing rehabilitation needs and strategies for intervention to facilitate reintegration into mainstream schooling and placement in learner support services where appropriate. There are a limited number of special needs schools in South Africa, and none that specifically cater for the unique needs of children with TBI (Levin, 2004). It is possible that more children in this study would be placed in a special needs school if there was greater availability of and easier access to a suitable school. Thus it seems reasonable that increased availability of educational resources, particularly learner support that focuses on the unique sequelae that results from TBI, as well as interim support until placement can be made, should be of paramount importance.

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APPENDIX A

Parent Information Questionnaire and Asset Index

PARENT QUESTIONNAIRE AND ASSET INDEX

GENERAL INFORMATION

Full name (Parent):	
Telephone:	Work: ( ) Home: ( ) Cell:
Home Language:	
Full name (Child):	
Gender:	M F
Date of Birth:	
Grade:	

HOUSEHOLD INCOME: (Please circle appropriate number)

Household income per year:	1. R0 2. R1 – R5 000 3. R5001 – R25 000 4. R25 000 – R100 000 5. R100 001+

PARENTAL EDUCATION: (Please circle appropriate number)

	Biological mother	Biological father	Guardian
Highest level of education reached? Mark one response for each person as follows:			
1. 0 years (No Grades / Standards) = No formal education (never went to school)	1.	1.	1.
2. 1-6 years (Grades 1-6 / Sub A-Std 4) = Less than primary education (didn't complete primary school)	2.	2.	2.
3. 7 years (Grade 7 / Std 5) = Primary education (completed primary school)	3.	3.	3.
4. 8-11 years (Grades 8-11 / Stds 6-9) = Some secondary education (didn't complete high school)	4.	4.	4.
5. 12 years (Grade 12 / Std 10) = Secondary education (completed senior school)	5.	5.	5.
6. 13+ years = Tertiary education (completed university / technikon / college)	6.	6.	6.
7. Don't know	7.	7.	7.

PARENTAL EMPLOYMENT: (Please circle appropriate number)

Hollingstead categories:	Biological mother	Biological father	Guardian
1. Higher executives, major professionals, owners of large businesses)	1.	1.	1.
2. Business managers of medium sized businesses, lesser professions (e.g. nurses, opticians, pharmacists, social workers, teachers)	2.	2.	2.
3. Administrative personnel, managers, minor professionals, owners / proprietors of small businesses (e.g. bakery, car dealership, engraving business, plumbing business, florist, decorator, actor, reporter, travel agent)	3.	3.	3.
4. Clerical and sales, technicians, small businesses (e.g. bank teller, bookkeeper, clerk, draftsperson, timekeeper, secretary)	4.	4.	4.
5. Skilled manual – usually having had training (e.g. baker, barber, chef, electrician, fireman, machinist, mechanic, painter, welder, police, plumber, electrician)	5.	5.	5.
6. Semi-skilled (e.g. hospital aide, painter, bartender, bus driver, cook, garage guard, checker, waiter, machine operator)	6.	6.	6.
7. Unskilled (e.g. attendant, janitor, construction helper, unspedified labour, porter, unemployed)	7.	7.	7.
8. Homemaker	8.	8.	8.
9. Student, disabled, no occupation	9.	9.	9.

MATERIAL AND FINANCIAL RESOURCES (ASSET INDEX): (Please circle appropriate number)

Which of the following items, in working order, does your household have?

Items	Yes	No
1. A refrigerator or freezer	1.	1.
2. A vacuum cleaner or polisher	2.	2.
3. A television	3.	3.
4. A hi-fi or music center (radio excluded)	4.	4.
5. A microwave oven	5.	5.
6. A washing machine	6.	6.
7. A video cassette recorder or dvd player	7.	7.

Which of the following do you have in your home?

Items	Yes	No
1. Running water	1.	1.
2. A domestic servant	2.	2.
3. At least one car	3.	3.
4. A flush toilet	4.	4.
5. A built-in kitchen sink	5.	5.
6. An electric stove or hotplate	6.	6.
7. A working telephone	7.	7.

Do you personally do any of the following?

Items	Yes	No
1. Shop at supermarkets	1.	1.
2. Use any financial services such as a bank account, ATM card or credit card	2.	2.
3. Have an account or credit card at a retail store	3.	3.

APPENDIX B

NEUROPSYCHOLOGY RESEARCH HISTORY SHEET

Child's Name: ______________________________ Date of Birth: ___________________Age: _________

PREGNANCY AND BIRTH

What complications, if any, were there during the *pregnancy*?

What *medicines* (prescribed or non-prescribed) were taken during pregnancy?

If the birth was earlier or later than the *expected date*, please specify.

What complications, if any, were there during the *birth*? What *type* of birth?

Were there any difficulties with *bonding*?

What was your baby's *weight*? __________

What complications, if any, were there in the *newborn period*?

What *feeding* difficulties, if any, were there? Are there any currently?

What *sleeping* difficulties, if any, were there? Are there any currently?

DEVELOPMENT

At what age did your child:

sit unaided? ____________________

crawl? ____________________

walk unassisted? ____________________

dress and undress unassisted? ____________________

button own clothes? ____________________

tie shoe laces? ____________________

say their first word? ____________________

use 2 words together? ____________________

write own name? ____________________

Were there any problems with *motor, speech or co-ordination* development?

At what age was your child *dry by day*? ____________________

At what age was your child *dry by night*? ____________________

What problems, if any, were there with *bowel and bladder* control?

Were there any early *separations* from you?

Please list any *illnesses* and problems with *hearing* or *vision* that your child has/had.

What, if any, problems have there been with your child's *behaviour*?

Has your child ever been referred to a *Psychologist/Psychiatry* service?

What *type* of school does your child attend?

Do you currently have, or have you had, any concerns about your child's *performance* at school?

Have there been any *emotionally difficult* experiences for your child?

FAMILY COMPOSITION

Including names and ages of your *other children* (and state if any are half or step brothers or sisters).

Genogram:

Level of *attainment* at school of siblings:

PARENTS' DETAILS

	Mother	Father
Name		
Relationship (e.g. mother, step-mother)		
Age		
Occupation		
Highest educational level		

Please give any details of any medical or *mental health problems* you or your family of origin may have had.

MEDICATIONS

What *medication* is your child currently receiving?

Please feel free to mention anything else you would like to bring to our attention.

[Observations of child:]

Name of History-Taker: ___________________________ Date: __________ Signed: __________

APPENDIX C

UNIVERSITY OF CAPE TOWN

Health Sciences Faculty
Faculty of Health Sciences Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6338 • **Facsimile** [021] 406 6411
e-mail: sumayah.ariefdien@uct.ac.za

04 August 2011

HREC REF: 345/2011

Ms A Dollman
c/o Ms L Schrieff
Department of Psychology
Humanities Graduate School Building
Upper Campus

Dear Ms Dollman

PROJECT TITLE: INVESTIGATION OF THE ACADEMIC AND BEHAVIOURAL OUTCOMES OF A GROUP OF SCHOOL GOING SOUTH AFRICAN CHILDREN WHO HAVE SUSTAINED A SEVERE TRAUMATIC BRAIN INJURY.

Thank you for addressing the issues raised by the committee.

It is a pleasure to inform you that the Ethics Committee has **formally approved** the above-mentioned study.

Approval is granted for one year till the 15 August 2012.

Please submit a progress form, using the standardised Annual Report Form (FHS016), if the study continues beyond the approval period. Please submit a Standard Closure form (FHS010)if the study is completed within the approval period.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the REC. REF in all your correspondence.

Yours sincerely

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637.
Institutional Review Board (IRB) number: IRB00001938

sAriefdien

APPENDIX D

WESTERN CAPE
Education Department
Provincial Government of the Western Cape

RES
EAR
CH

awyngaar@pgwc.gov.za
tel: +27 021 476 9272
Fax: 0865902282
Private Bag x9114, Cape Town, 8000
wced.wcape.gov.za

REFERENCE: 20110803-0111
ENQUIRIES: Dr A T Wyngaard

Miss Aimee Dollman
Psychology Department
UCT

Dear Miss Aimee Dollman

RESEARCH PROPOSAL: INVESTIGATION OF THE ACADEMIC AND BEHAVIOURAL OUTCOMES OF A GROUP OF SCHOOL-GOING SOUTH AFRICAN CHILDREN WHO HAVE SUSTAINED A SEVERE TRAUMATIC BRAIN INJURY

Your application to conduct the above-mentioned research in schools in the Western Cape has been approved subject to the following conditions:

1. Principals, educators and learners are under no obligation to assist you in your investigation.
2. Principals, educators, learners and schools should not be identifiable in any way from the results of the investigation.
3. You make all the arrangements concerning your investigation.
4. Educators' programmes are not to be interrupted.
5. The Study is to be conducted from **1 August 2011 till 30 September 2011 and 17 January 2012 till 30 April 2012**
6. No research can be conducted during the fourth term as schools are preparing and finalizing syllabi for examinations (October to December).
7. Should you wish to extend the period of your survey, please contact Dr A.T Wyngaard at the contact numbers above quoting the reference number.
8. A photocopy of this letter is submitted to the principal where the intended research is to be conducted.
9. Your research will be limited to the list of schools as forwarded to the Western Cape Education Department.
10. A brief summary of the content, findings and recommendations is provided to the Director: Research Services.
11. The Department receives a copy of the completed report/dissertation/thesis addressed to:
    The Director: Research Services
    Western Cape Education Department
    Private Bag X9114
    CAPE TOWN
    8000

We wish you success in your research.

Kind regards.
Signed: Audrey T Wyngaard
for: **HEAD: EDUCATION**
DATE: 04 August 2011

MELD ASSEBLIEF VERWYSINGSNOMMERS IN ALLE KORRESPONDENSIE / PLEASE QUOTE REFERENCE NUMBERS IN ALL CORRESPONDENCE / NCEDA UBHALE IINOMBOLO ZESALATHISO KUYO YONKE IMBALELWANO

GRAND CENTRAL TOWERS, LAER-PARLEMENTSTRAAT, PRIVAATSAK X9114, KAAPSTAD 8000
GRAND CENTRAL TOWERS, LOWER PARLIAMENT STREET, PRIVATE BAG X9114, CAPE TOWN 8000

WEB: http://wced.wcape.gov.za

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VEILIGE SKOLE/SAFE SCHOOLS ☎ 0800 45 46 47

APPENDIX E

Informed Consent to Participate in Research and Authorization for Collection, Use, and Disclosure of Questionnaire and Other Personal Data

You are being asked to take part in a research study. This form provides you with information about the study and seeks your authorization for the collection, use and disclosure of questionnaire data, as well as other information necessary for the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand. By participating in this study you will not be penalized or lose any benefits to which you would otherwise be entitled.

1. Name of Participant ("Study Subject")

____

2. Title of Research Study

Exploring factors that influence academic and behavioural outcome and the specific role of premorbid functioning, in a sample of children with severe TBI.

3. Principal Investigator and Telephone Number(s)

Aimee Dollman
Masters Student
Department of Psychology
University of Cape Town
082 767 5564

Dr Leigh Schrieff
Supervisor
Department of Psychology
University of Cape Town
021 650 3708

Professor Anthony Figaji
Co-Supervisor
School of Child and Adolescent Health
University of Cape Town
Red Cross War Memorial Children's Hospital
021 658 5400

Dr Pedro Wolf
Co-Supervisor
Department of Psychology
University of Cape Town
021 650 3430

4. Source of Funding or Other Material Support
NRF Thuthuka Grant, Grant holder: Leigh Schrieff

5. What is the purpose of this research study?
The purpose of this research is to investigate the academic and behavioural outcomes of children who have sustained a severe traumatic brain injury (TBI). It will specifically investigate the relationship between your child's functioning before the TBI and these outcomes.

6. **What will be done if you take part in this research study?**
The purpose and procedure of the study will be explained to you. You will be asked to complete a parent information and asset index questionnaire, and a questionnaire about your child's developmental history.
You will then be asked to complete three additional questionnaires that will look at your child's behaviour, and at how you and your family have coped with your child's injury. You will be allowed to take breaks when necessary.

Your child's school will be contacted to get information regarding your child's school results.

If you have any questions now or at any time during the study, you may contact the Principal Investigator listed in #3 of this form.

7. **If you choose to participate in this study, how long will you be expected to participate in the research?**
Completing the questionnaires will place during one session, which should not last longer than two (2) hours.
If at any time during the session you wish to stop your participation, you are free to do so without penalty.

8. **How many people are expected to participate in the research?**

100

9. **What are the possible discomforts and risks?**
There are no known risks associated with participation in this study. Should you get tired during the study, you will be allowed to rest. Refreshments will be available to you.

If you wish to discuss the information above or any discomforts you may experience, you may ask questions now or call the Principal Investigator listed in #3 of this form.

10a. What are the possible benefits to you?

You or the child in your care may or may not personally benefit from participating in this study. Should behavioural problems be identified during the process of this study, you will be referred to the appropriate services.

10b. What are the possible benefits to others?

The information gained from this research study will help improve our understanding of the academic and behavioural consequences that result due to TBI, particularly their relationship to a child's functioning before a TBI.

11. If you choose to take part in this research study, will it cost you anything?

Participating in this study will not cost you anything.

12. Will you receive compensation for taking part in this research study?

You will receive R50 towards transport costs as well as a R50 food voucher.

13a. Can you withdraw from this research study?

You are free to withdraw your consent and to stop participating in this research study at any time. If you do withdraw your consent, there will be no penalty.

If you have any questions regarding your rights as a research subject, you may phone the Psychology Department offices at 021-650-3430.

13b. If you withdraw, can information about you still be used and/or collected?

Information already collected may be used.

15. Once personal and performance information is collected, how will it be kept secret (confidential) in order to protect your privacy?

Information collected will be stored in locked filing cabinets or in computers with security passwords. Only certain people have the right to review these research records. These people include the researchers for this study and certain University of Cape Town officials. Your research records will not be released without your permission unless required by law or a court order.

16. What information about you may be collected, used and shared with others?

This information gathered from you will be demographic information, information on your child's developmental history, and records of your responses to questionnaires regarding your child's behaviour, and the experience by your family in relation to your child's accident. If you agree to be in this research study, it is possible that some of the information collected might be copied into a "limited data set" to be used for other research purposes. If so, the limited data set may only include information that does not directly identify you. For example, the limited data set cannot include your name, address, telephone number, ID number, or any other numbers or codes that link you to the information in the limited data set.

17. How will the researcher(s) benefit from your being in the study?

In general, presenting research results helps the career of a scientist. Therefore, the Principal Investigator and others involved this research project may benefit if the results of this study are presented at scientific meetings or in scientific journals.

18. Signatures

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; and how the participant's performance and other data will be collected, used, and shared with others:

____ ______________________

Signature of Person Obtaining Consent and Authorization Date

You have been informed about this study's purpose, procedures, possible benefits, and risks; and how your performance and other data will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. You hereby authorize the collection, use and sharing of your performance and other data. By signing this form, you are not waiving any of your legal rights.

____ ______________________

Signature of Person Consenting and Authorizing Date

Please indicate below if you would like to be notified of future research projects conducted by our research group:
______________ (initial) Yes, I would like to be added to your research participation pool and be notified of research projects in which I might participate in the future.

Method of contact:

Phone number: __________________________
E-mail address: __________________________
Mailing address: ________________________________
____
____

APPENDIX F

Teacher Informed Consent to participate in research

_______________ has been asked to take part in a research study. The study requires the involvement of his/her parents and teacher. His/her parents have given consent for this participation. This form provides you with information about the study and seeks your permission to partake in some of the activities involved in the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand.

5. Name of Participant ("Study Subject" – the child)

____

6. Title of Research Study

Exploring factors that influence academic and behavioural outcome and the specific role of premorbid functioning, in a sample of children with severe TBI.

7. Principal Investigator and Telephone Number(s)

Aimee Dollman
Masters Student
Department of Psychology
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082 767 5564

Dr Leigh Schrieff
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University of Cape Town
021 650 3708

Professor Anthony Figaji
Co-Supervisor
School of Child and Adolescent Health
University of Cape Town
Red Cross War Memorial Children's Hospital
021 658 5400

Dr Pedro Wolf
Co-Supervisor
Department of Psychology
University of Cape Town
021 650 3430

8. Source of Funding or Other Material Support

NRF Thuthuka Grant, Grant holder: Leigh Schrieff

6. What is the purpose of this research study?

The purpose of this research is to investigate the academic and behavioural outcomes of children who have sustained a severe traumatic brain injury (TBI). It will specifically investigate the relationship between a child's functioning before the TBI and these outcomes.

7. What will be required if you take part in this research study?
Firstly, the primary caregiver of the child will complete three questionnaires designed to give an indication of the behavioural skills and problems in the child. You, as the teacher, will be asked to complete two teacher's report forms. These forms are designed to get an indication of the child's behaviour in a school setting. The Principal Investigator will arrange a session time that is most convenient for you.

8. If you choose to participate in this study, how long will you be expected to participate in the research?
One session of approximately 40 minutes in duration is needed to complete the forms. However, you may decide to withdraw your participation, without penalty, at any point in time.

8. Will you receive compensation for taking part in this research study?
No

9. Can you withdraw from this research study?
You are free to withdraw your consent and to stop participating in this research study at any time. If you do withdraw your consent, there will be no penalty.

If you have any questions regarding you or the child's rights as a research subject, you may phone the Psychology Department offices at 021 650 3430.

11. Signatures

As a representative of this study, I have explained to the participant's (child's) teacher the purpose of this research study and their involvement in it:

____ ____________________
Signature of Person Obtaining Consent Date

You have been informed about this study's purpose and your involvement in. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. By signing this form, you are not waiving any of your legal rights.

____ ____________________
Signature of Person Consenting Date

APPENDIX G

BIVARIATE CORRELATION BETWEEN PREMORBID FUNCTIONING AND THE BRIEF GEC SCORE AFTER REMOVAL OF OUTLIER

Figure G1 shows there was one outlier in the premorbid functioning data. The association between premorbid functioning and the GEC score on the BRIEF was no longer significant once this outlier was removed (r = .255; p = .109).

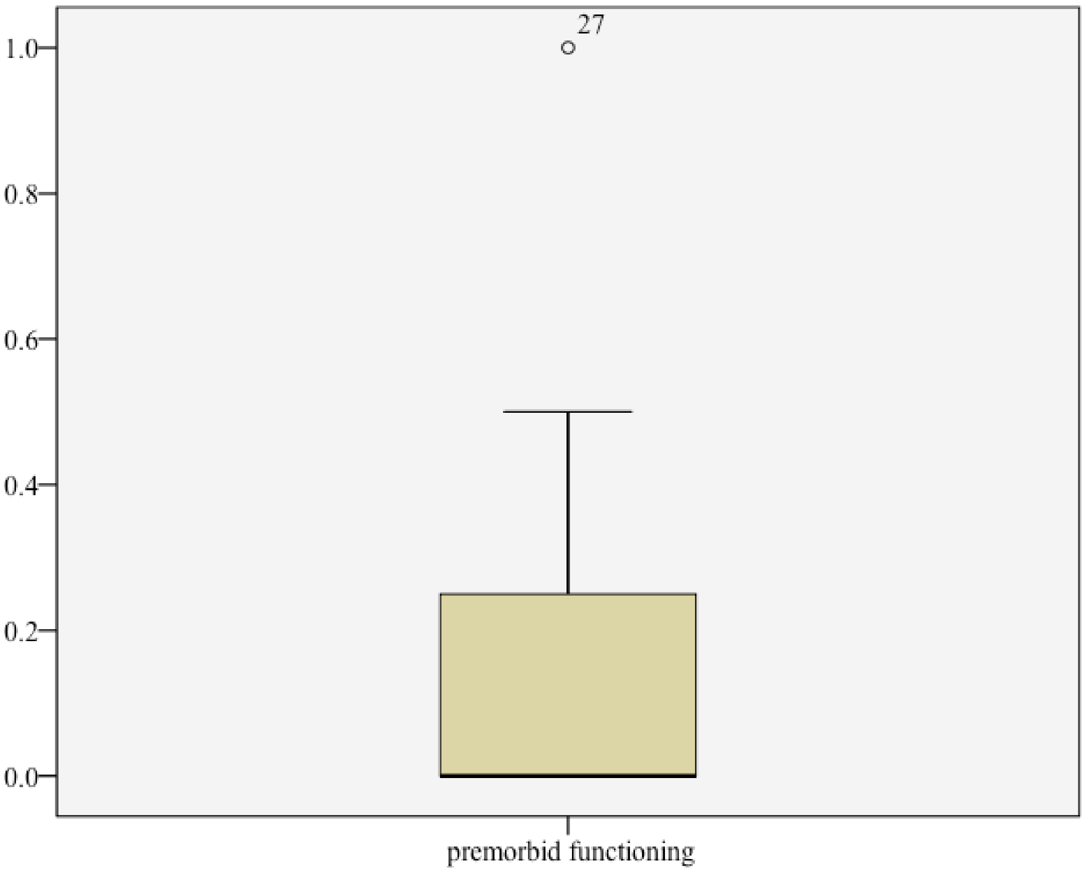

Figure G1. Box plot for *premorbid functioning* variable.

APPENDIX H

BETWEEN-GROUP DIFFERENCES IN SES AFTER REMOVAL OF OUTLIERS: PREMORBID PROBLEMS VS. NO PREMORBID PROBLEMS GROUPS

Figure H1 shows there was one outlier in the Evidence of Premorbid Problems group and one outlier in the No Evidence of Premorbid Problems group for the SES variable. The between-group difference remained non-significant even after the two outliers were removed, $t(1, 23) = .883$; $p = .286$.

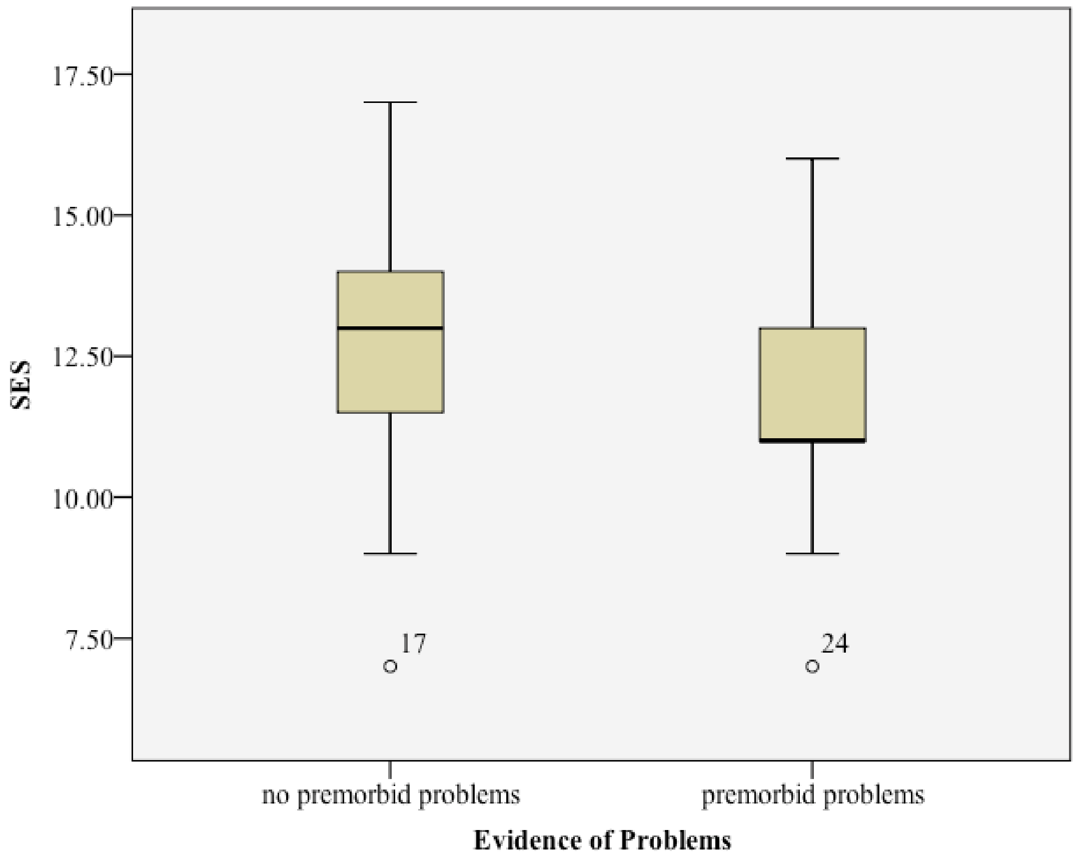

Figure H1. Box plot for *SES* variable.