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A research report

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Investigating the relationship between mild traumatic brain injury and memory performance in a multilingual paediatric cohort.

by

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submitted to the Faculty of Humanities as partial fulfilment of the requirements for the Master of Arts (Social and Psychological Research) degree in the field of Research, University of the Witwatersrand, Johannesburg, <May 2020>.

Through sub-classification supervisor: Enid Schutte

Abstract

The long-term sequalae of paediatric mild traumatic brain injuries (mTBI) is abound with controversy and deficient research. A history of outdated frameworks, arbitrary definitions, and varying methodologies have largely resulted in depicting mTBI in children as benign and inconsequential, culminating in negligible treatments or follow-up visits. However, recent strives in research have demonstrated a small but substantial percentage of children adversely affected by mTBI, particularly as it applies to typical cognitive development. That being said, investigations on these findings are still scarce and widely dispersed, especially within the diverse South African context. The present study aimed to examine cognitive impairments – memory and learning specifically – within a multilingual mTBI-cohort. Archival data on a battery of neuropsychological tests (i.e. NEPSY-II and WISC-IV) administered to a sample of 50 children (ages 6-12) were used to compare neurotypical- and mTBI-groups. Children's overall performance on various memory (verbal, visual, and associative) and auditory-attention (maintenance, shifting, inhibitory) tests were compared to establish whether any statistically significant group differences were present. Inferential statistics found an overall statistical nonsignificance between the two groups, despite a clear advantage in the neurotypical group on tests with a timed-component, in contrast to a numerically better performance by the mTBI group on untimed-tests. These findings suggest an underlying mechanism related to working memory (i.e. speed of processing) may be adversely affected in the clinical group, but not the neurotypical group. Further, multiple regression analyses of the clinical group found sex-differences, mainly negative associations between test performance and number of languages/ age at assessment, as well as a hypothesised negative association between test performance and time elapsed since injury. However, multiple inconsistencies, as well as design and sample limitations warrant further investigation into this matter.

Keywords: mild traumatic brain injury, paediatric, multilingual, memory performance, assessment, South Africa,

University of the Witwatersrand, Johannesburg

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SENATE PLAGIARISM POLICY

Declaration by Student

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I confirm that ALL the work submitted for assessment for the above course is my own unaided work except where I have explicitly indicated otherwise.

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I understand that the University of the Witwatersrand may take disciplinary action against me if there is a belief that this is not my own unaided work or that I have failed to acknowledge the source of the ideas or words in my writing.

Signature:  Date: 28-05-2020

Heinrich R Zentgraf

Acknowledgments

I would like to thank the following people:

- Firstly, the two principle investigators, Dr Vered Lack and Dr Miriam Close, who graciously made this study, my inclusion in it, and access to the data possible. Thank you very much!
- Secondly, my supervisor, Ms Enid Schutte, for her tenacity in seeing this project through in its entirety. From early morning proof reading to being a brilliant soundboard when I needed a shoulder to cry on.
- Thirdly, Brett Gregory, for being incredibly diligent and patient in explaining data-related queries.
- To Dr Jerome Loveland, Lauryne Lokothwayo, Dr Kate Cockcroft, and everyone from the department directly or indirectly involved in this final product.
- To my family and friends, for being understanding about my absence these past few years of study.
- Last but not least, my loving wife Leilani, the rock in my life. Your unwavering support and constant reassurance have meant the world for me. I would not have made it this far without you by my side.

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Much controversy remains in the literature regarding the long-term sequelae of mild traumatic brain injuries (mTBI) in children. On the one hand, many researchers have found virtually no long-term neuropsychological effects (Babikian et al., 2011; Frencham et al., 2005; Goldstrohm & Arffa, 2005), whilst on the other hand, a growing body of research seems to suggest that this conclusion is not as clear-cut and that as many as 15% of the paediatric mTBI population is adversely affected following the sub-acute phase (i.e. 1-3 months post-injury) with cognitive impairments such as memory and learning deficits, attention difficulties, and speed of processing related problems (Araki et al., 2017; Burns & Yosick, 2013; Giza, 2006; Prince & Bruhns, 2017; Rose et al., 2015) lasting more than 12 months post-injury (Lumba-Brown et al., 2018; McInnes et al., 2017), or occasionally as long as 3-5 years (Iverson, 2005; Katz et al., 2015).

Moreover, context-specific factors such as low socioeconomic status in tandem with a polyglot society may further exacerbate the impact of paediatric-mTBI on multilingual-cytoarchitecture, as some authors have suggested that injuries to the vulnerable developing brain during sensitive and critical development phases may disrupt its typical growth trajectory (Cannella et al., 2019; Giza, 2006; Greco et al., 2019; Sturman & Moghaddam, 2011). Furthermore, the multilingual brain – which is believed to contain distinctly different neurological networks (Higby et al., 2013; Kuhl et al., 2016; Nichols & Joanisse, 2016) – may be subject to worse neurocognitive prognosis in comparison with their non-injured counterparts. Thus, this study investigates the cognitive outcomes of traumatic brain injury in multilinguals, particularly memory and learning performance in a paediatric mTBI group, as compared to their neurotypical cohort.

Background & significance

Traumatic brain injury (TBI) typically refers to the broad and comprehensive term associated with an insult or injury to the head, caused by acute external acceleration/ deceleration forces (Cristofori & Grafman, 2019; Dewan et al., 2016). The consequential injury results in a varying degree of neurological damage to the concealed brain, which may manifest an array of temporary or permanent pathologies in somatic, cognitive, or psychosocial capacities (Xiong et al., 2013). Current literature reports TBI as the global leading cause of deaths and disabilities, accounting for approximately 10 million fatalities and/ or hospitalisations in persons under 45 years of age (Naidoo, 2013; Pretorius, 2013; Torres et al., 2019; Xiong et al, 2013), the main reason for hospital visits in the general population (Torres et al, 2019), and cites paediatric-TBI as the most frequent emergency

room admission in low- and middle-income countries (Appenteng et al, 2018). By comparison, estimates for TBI-related incidence in South Africa approximate close to 89 000 new cases annually, which according to Pretorius suggests it as one of the highest countries-of-incidence. However, Naidoo argues this figure likely underestimates the actuality, premised on the fact that a TBI-bank in South Africa is non-existent, present-day studies on adult and paediatric TBI-prevalence are grossly lacking, which is further exacerbated by overcrowded yet understaffed hospitals, as well as poorly maintained medical records.

The structural brain damage sustained during a TBI is generally indicative of its neuropsychological prognosis, and as such, brain injuries range from mild to severe as do their correlating neurocognitive sequelae. Although research is more or less in agreement concerning TBI on the severe and moderate end of the spectrum, much controversy exists with regards to mild traumatic brain injuries (mTBI) and concussions. For moderate and severe TBI, discouraging long-term effects have consistently been demonstrated, whereas many scholars regard the effects on mild and post-concussive brain damage as trivial and benign. This is especially true in past studies on paediatric-mTBI, which often cite the young neuroplastic brain and its malleability to recover from injury.

However, some researchers found contrary evidence to these claims, and have demonstrated moderate neurocognitive sequelae in children with mTBI, such as difficulties in concentration and attention, as well as poor memory and speed of processing performance (Katz et al., 2015; Lumba-Brown et al., 2018). Consequently, TBI has been labelled the ‘silent epidemic’ (Goldstein, 1990; Langlois & Sattin, 2005) due to the unknown nature of its societal impact. Not only may this pose an excessive social and economic burden in terms of educational and medical costs, but also a decreased quality of life for the involved individual with regards to their academic and developmental progress (Prince & Bruhns, 2017; Shetty et al., 2018).

Literature review

Mild traumatic brain injury

Mild traumatic brain injury (mTBI) has classically been defined as an acute neurophysiological brain dysfunction caused by the impact of abrupt external acceleration/ deceleration forces to the head which lead to a temporary change of consciousness, as well as possibly accompanied by anterograde amnesia (Katz et al., 2015; Torres et al., 2019; Xiong et al., 2013). Several groups (e.g. American Congress of Rehabilitation Medicine [ACRM] or World Health Organization [WHO]) have attempted to delineate the parameters that envelope

change of consciousness, and despite a lack of consensus the following criteria are generally agreed upon: (1) loss of consciousness lasting no longer than 30 minutes, (2) post-traumatic amnesia for less than 24 hours, and (3) altered mental state such as confusion or disorientation (Katz et al., 2015). It is one of the most commonly occurring types of brain injuries in children which include symptoms such as headaches, fatigue, irritability, as well as an array of impaired cognitive functions (McInnes et al., 2017). The diagnosis and classification of a brain injury typically involve several observations to be made.

Foremost, and on a basic level, an evaluation of the nature of the physical injury ensues, i.e. whether it is a closed or blunt, penetrating, or a blast head injury. Although this provides minimal information to clinicians regarding the extent of brain damage, it does serve as shorthand for immediate surgical requirements as well as expected patient treatment (Hawryluk & Manley, 2015). Secondly, various scales are often used to distinguish brain injuries based on their severity. For instance, the Glasgow Coma Scale (GCS) score (Teasdale & Jennett, 1974) is one of the more established scoring systems used at medical facilities (Hawryluk & Manley, 2015).

The GCS measures three features of central nervous system function (Middleton, 2012) scored according to the best response on each domain – i.e. motor responsiveness (low of 1 and high of 6), verbal responsiveness (1-5), and eye-opening (1-4; Teasdale & Jennett, 1974) – in order to appraise the present level of consciousness, presence of anterograde amnesia, level of pain, and command responsiveness (Cristofori & Grafman, 2019). The accumulative score of all behaviours measured in conjunction with the parameters of consciousness mentioned above classifies the respective brain injury as either mild (13-15), moderate (9-12), or severe (3-8). This functional assessment is usually also followed by a structural assessment obtained through neuroimaging, as a means to help determine the extent of physical damage to the skull and concealed brain.

Information provided by neuroimaging techniques is vital in understanding the structural and functional changes that follow brain injury, and thereby informs our knowledge regarding prognosis. Computer tomography (CT) and magnetic resonance imaging (MRI) are two structural neuroimaging techniques that form part of routine clinical examination following brain injury (Cristofori & Grafman, 2019). CT persists as the standard of care during the acute phase to detect bleeding in the dura, brain, and ventricles (Wu et al., 2016). However, CT does not seem to clearly identify abnormalities in mild brain injuries as it does for moderate or severe kinds. This is exemplified in cases where fewer than 10 per cent of mild brain injuries had positive CT scans (Jeret et al., 1993). On the other hand, MRI

provides comparable information to CT, albeit more sensitive to subtle injuries (Lee & Newberg, 2005; Rose et al., 2015) and short-term prognoses (Wu et al., 2016). Improved sensitivity in MRI allows the detection of small brain contusions, high precision hematomas (Mittl et al., 1994), axonal haemorrhages, and other haemorrhagic trauma. For instance, Rose et al. (2015) found in 135 patients with mild brain injury and negative CT scans, that MRI identified approximately 28% with an intracranial injury. That being said, MRI often provides negative mTBI results, despite the presence of cognitive impairments (Rutland-Brown et al., 2006).

Mechanisms

mTBIs differ extensively in their aetiology, pathophysiology, and clinical presentation. Typically, one means by which to categorize the multitudinous brain injuries sustained is according to either primary or secondary aetiologies. That is, the long-term sequelae manifested in traumatic brain injuries are mainly contingent on the severity of primary and outcome of secondary injury (Lozano, 2015). When the concealed brain is exposed to a violent external force – whether directly or indirectly – the abrupt mechanical disturbance within brain tissue produces a primary injury, consequences which include damaged blood vessels (i.e. haemorrhages), as well as axonal shearing – a traumatic outcome in which axons are cut from their neurons (Hawryluk & Manley, 2015).

A primary injury comprises of various mechanisms, such as laceration, rotation, shearing, compression and others, which can be further dichotomized into contact/ impact injury or acceleration-deceleration forces (Xiong et al., 2013). All forms of brain injuries likely contain one or a multitude of mechanisms, and although incomplete, descriptions of these mechanisms allow for some degree of prediction with regards to intracranial pathology. For instance, epidural hematomas (e.g. skull fractures that result in arterial bleeding) are most commonly the consequence of impact injuries, whereas subdural hematomas (i.e. venous bleeding) are often linked to acceleration-deceleration injuries (Gennarelli et al., 1998; Hawryluk & Manley, 2015; Xiong et al., 2013). As such, the widespread tissue damage found in primary injuries and caused by the shearing and tearing of neural substructures such as neurons, blood vessels, and glia, may lead to subsequent cell death (Cristofori & Grafman, 2019; Hawryluk & Manley, 2015). Thus, mTBI should not be viewed as a distinct pathophysiological incident but rather an intricate disease process that results in structural

brain damage and functional deficits from both focal and diffuse injuries (Torres et al., 2019; Xiong et al., 2013).

Indeed, although previously controversial, it has now become widely accepted that primary injury causes progressive secondary damage as a result of cascading molecular injuries, lasting weeks after initial pathology (Belur et al., 2013). The diffuse (micro) injuries are believed to be associated with mitochondrial dysfunction, free radical production, diffuse axonal injury (DAI), excitotoxicity, and various other mechanisms (Hawryluk & Manley, 2015; Torres et al., 2019). For instance, studies have shown abnormalities in the integrity of white matter tracts associated with DAI, even in cases of single mTBI (McInnes et al., 2017). Similarly, part of secondary injury inflammation and mitochondrial dysfunction are attributed to an atypical cascade of neurometabolic complications that start with the disturbance of neuronal cells and glutamate discharge, followed by cellular metabolic surge which culminates in diminished biological energy and depressed neuronal activity (Lobo et al., 2010; Rose et al., 2015). Research on the prevalence of metabolic mismatch from mechanical damage in both adult and child mTBI-cases serves as the basis for immediate rest and recovery recommendations (e.g. diaschisis; Stein & Hoffman, 2003) following brain injury (Corwin et al., 2017). Although most mTBI cases have transitory symptoms dissipating within weeks, many studies have found a small percentage of patients who remain symptomatic post-injury (Torres et al., 2019), and in certain cases may deteriorate past the expected recovery-period (Lumba-Brown et al., 2018).

Sequelae & debate

Unsurprisingly, a large amount of research demonstrates that most mTBI-patients show good recovery and view somatic and cognitive deficits as short-lived symptoms which normalise within several weeks post-injury (Araujo et al., 2014; Prince & Bruhns, 2017; Torres et al., 2019) for approximately 90% of cases irrespective of the country (Dewan et al., 2016). For instance, evidence broadly suggests that overall improvement in neuropsychological functions recuperate to premorbid levels within a 1 to 3-month period, with non-significant deficits observed in cognitive capacities (Katz et al., 2015). These findings also corroborate what meta-analytical studies have found in uncomplicated-mTBI for both adult and paediatric cohorts by way of performance-based assessments (Kirkwood et al., 2014). However, ‘good’ recovery does not imply ‘full’ recovery; as Xiong et al. (2013) note that even an mTBI absent of noteworthy cellular degeneration and secondary-DAI, may still result in onerous cognition.

Several such cognitive impairments, theoretically related and behaviourally observable include sustained attention, memory and learning, and speed of processing; findings which have been extensively corroborated (Corwin et al., 2017; Lajiness-O'Neill et al., 2010, Rose et al., 2015; Wu et al., 2016). One study in specific has suggested that paediatric-mBTI in children between the ages of 7-8 years were more indicative of cognitive impairments within 18 months post-injury (Lumba-Brown et al., 2018). Similarly, de Koning and colleagues (2017) have found mTBI-patients immediately discharged from the emergency department – anticipated to have better recovery compared to those hospitalised – had more serious implications 6 months later, and 15% on average had complaints of troubling aftereffects one year later (Faul & Coronado, 2015; Prince et al., 2017; Rose et al., 2015; Torres et al., 2019). Thus, McInnes et al., (2017) argue that despite the dominant view of complete recovery 3-months following injury, as many as 50% of patients demonstrate various cognitive impairments following a single mTBI, and approximately 15% of children show persistent symptomatic-experiences 1-year past the initial injury (Schmidt et al., 2018).

Much of the divergence in data may be symptomatic of methodological variations in measuring mTBI-related pathologies; for instance, sensitive chronic-measures relative to insensitive theory-driven assessments (McInnes et al., 2017). Consequently, because of the incongruent evidence and widespread debate, it remains relatively inconclusive not only to what degree but also how pervasive cognitive impairments can be attributed to mTBI. Katz et al., (2015) suggest that cognitive deficits such as poor memory performance, atypical concentration levels, and lowered attention-span ascribed to the enduring symptoms and dysfunctional limitations of prolonged mTBI, are perhaps a potentially multidimensional phenomenon. That is, it may be an interrelated mixture of physical injury, psychological elements, and social aspects.

Memory & Attention

Memory is conceived as a multidimensional construct, present already early in life, and subject to protracted development. Several key aspects of memory should be noted at the outset of this section: (1) memory is not a singular concept, in the sense that at any developmental age its performance is multiply determined (i.e. the mnemonic process is contingent on more than one variable), (2) encoding, consolidation, and retrieval serve as basic processes of memory, (3) different types of memory undergo extensive development and serve different functions, and (4) several brain regions (e.g. the hippocampus) have been identified as critical neural structures associated with conscious memory (Bauer, 2013).

Although this list is not meant to be exhaustive, it provides an outline of some central elements when discussing memory.

Overview

The basic cognitive processes associated with the formation, storage, and retrieval of information are collectively known as memory. Although it is difficult and somewhat artificial to separate these mechanisms from each other (i.e. to delineate exactly when consolidation starts after encoding stops), it seems evident that these processes follow some sequential order and are therefore depicted accordingly: short-term maintenance of information is encoded, which leads to consolidation and storage of the encoded information for future retrieval (Bauer, 2013). The amount of information maintained and the efficiency of its encoding process is believed to improve according to a gradual increase in developmental age, indicative of the faster processing speeds of information as well as growth in short-term memory (STM) span (Cowan & Alloway, 2009). Essentially, memory is the outcome of learning new information. That is, learning is said to have occurred when a new memory is created, whether by single or repetitive exposure to information. However, not all memories undergo similar processes. Rather, findings suggest there are several different memory types, each facilitated through an assortment of neural circuitry and mechanisms.

Although numerous models of memory are prevalent within research literature, a majority of researchers tend to agree on certain fundamentals. As mentioned, the processes from inception to end are (1) encoding, (2) storage, and (3) retrieval. Encoding entails processing new information which produces memory traces for (mental) storage. Two steps constitute encoding, the first being acquisition – in which a sensory buffer 'selects' pertinent stimuli from the sensory systems for maintenance in short-term memory (STM). The second step, known as consolidation, involves the stabilization of neural changes in the brain which ultimately leads to long-term memory (LTM) or storage. By comparison, retrieval refers to the accessing of information in storage, and also its utilization in consciously creating a representation or performing a learned behaviour.

The concept of STM has been extended on throughout several models which conceptualize working memory (WM), Baddeley and Hitch's theory (1974; Baddeley, 2000) being the most dominant (Gazzaniga, Ivry & Mangun, 2014; Henry, 2012;). According to Baddeley, working memory is best understood as "*... a temporary storage system under attentional control that underpins our capacity for complex thought.*" (2007, p. 1).

Essentially, WM refers to the process by which information is temporarily and flexibly held 'in mind' (Bouchacourt & Buschman, 2019), for manipulation, storage, and reasoning tasks. Therefore, it seems a bi-directional relationship between WM and LTM is apparent (Cockcroft, 2015), whether new information is being stored into LTM, or retrieved for goal-directed behaviour (Mansouri et al., 2015).

The three-part WM model (Baddeley & Hitch, 1974) proposed two sub-systems for the rehearsal of phonological- and visuospatial-information, regulated by a third central executive mechanism. These two sub-systems (the phonological loop and visuospatial sketch pad respectively) operate as STM stores, which interacts with LTM as coordinated by the central executive system. Furthermore, each sub-system is hypothesized to be modality-specific; that is, the phonological loop acoustically codes information in WM, whereas the visuospatial sketch pad parallels this mechanism into either visual or visuospatial storage codes. An amendment to the original model included the addition of a fourth component, a multimodal short-term store; the episodic buffer (Baddeley, 2000). One hypothesized feature of the episodic buffer is that it stores information from many different modalities (Henry, 2012), thereby enabling the simultaneous maintenance of many different types of information. A second hypothesized feature is that the episodic buffer 'binds' different information sources together into semantic or meaningful information, as well as integrates and consolidates information into coherent episodes (Baddeley, 2003; Cockcroft, 2015).

Attention

Although memory and attention have traditionally been thought of and indeed researched as distinct concepts (Kiyonaga & Egner, 2013), it seems intuitive that memory encounters processing restrictions as well as necessitates selection. For instance, not all environmental aspects can be encoded into LTM, neither are we during retrieval processes able to bring all stored information to mind. Instead, attentional-selection is a requirement within the processing constraints of all memory stages (Kuhl & Chun, 2014). However, although the exact nature of this exchange remains unsettled, it seems evident that memory and attention operate bi-directionally (Kuhl & Chun, 2014).

According to Oberauer (2019**)**, the taxonomy of attention can be divided into two main categories, controlled and automatic, which further function on either perceptual or non-perceptual bases. For instance, a controlled perceptual task would comprise of selectively attending objects or features to monitor the outcomes of ongoing actions. By comparison, an automatic perceptual task would entail salient stimuli capturing attention, such as errors or

unexpected difficulties. At the other end, non-perceptual control refers to items attended to in WM, a response selection for example, whereas non-perceptual automatic attention retrieves information from LTM, often as an involuntary selection or intrusive thought (Oberauer, 2019). Thus, based on the aforementioned, attention (i.e. as an umbrella term) is ordinarily categorized as selective and sustained attention, distractibility, as well as also inhibitory control (Chun & Johnson, 2011).

Neuroanatomy of memory & attention

Research and neuroimaging studies on individuals with brain damage or particular kinds of disease, as well as the replicated animal models thereof, have contributed enormously to our understanding and knowledge that effortful attention and the consequential memory traces involves a multistage activity which is contingent on wide a network of neural structures (Bauer, 2013). For instance, it has been demonstrated that declarative memories are encoded, stored, and later retrieved within a multicomponent neural network comprised of several brain regions, such as the hippocampus, entorhinal, perirhinal, and parahippocampal (collectively part of the medial temporal lobes [MTL]), but also processed in the prefrontal cortex and association areas (Bauer, 2013; Eichenbaum & Cohen, 2001; Zola & Squire, 2000). Similarly, different brain regions across different age ranges also support more executive features of working memory processes (Bathelt et al., 2018). For example, research on WM tasks in younger children suggests that large white matter connections are indicative of a greater distributed network (Dean et al., 2014).

Furthermore, different grey matter volumes (i.e. decreased cortical thickness; Sowell, 2004) within frontal, parietal (Rossi et al., 2013), temporal and parietal connections (Treble et al., 2013), were statistically significant in predicting an individual's memory capacity, relative to their developmental age (Bathelt et al., 2018). Hence, the gradual development of WM in early to middle childhood reflects the pronounced biological change brain structures supporting these functions undergo (Gathercole et al., 2004; Tamnes et al., 2013). Moreover, these findings have been replicated in fMRI studies that show reorganization throughout brain networks is related to an increased capacity in memory and specifically WM (Bathelt et al., 2018; Houde et al., 2010).

Foremost, prefrontal structures and associations areas are primarily involved with encoding and consolidation processes, the latter which has been linked to the medial temporal structures and LTM tasks (Bauer, 2013; McGaugh, 2000). From a developmental perspective, the linear increases in white matter and curvilinear changes in grey matter volumes are not

only linearly associated with age (Giedd et al., 1999), but also indicative of greater brain region connectivity as well as myelination processes (Klingberg, 2008), believed to serve as the neural substrate of memory (Bauer, 2013). Due to the widespread nature of the various brain regions responsible for supporting distinct memory functions, mnemonic processes are believed to operate in an integrated manner once individual components reach functional maturity (Bauer, 2013; Fuster, 2002).

As such, the processes that retrieve LTM information from MTL-structures are thought to rely primarily on the prefrontal cortex (Ofen, 2012), as well as executive WM contribution from the left temporal cortex (Bathelt et al., 2018) in older children. By comparison, studies on memory load (for instance, retrieval from LTM to STM) have reported that WM-activity in young children is associated with lateral prefrontal brain regions (Brodmann area 46) (Moriguchi & Hiraki, 2013; Tsujimoto et al., 2004). Additionally, Tsujimoto et al. found significantly stronger brain activation during memory and working memory-related tasks in the frontal and parietal regions, such as when presenting three items formed in a cue array, as opposed to two items or less.

Memory and attention performance after brain injury

As previously discussed, some of the most debilitating effects that accompany brain injuries often translate into cognitive and neuropsychological impairments (Silver, 2000). Of these impairments, deficits in memory function are more prevalent and documented effects following mTBI (Jaffe et al., 1992; Lajiness-O'Neill et al., 2010; Yeates et al., 1995). For instance, in a meta-analysis by Karr et al. (2014), visual and verbal memory were reported as two mnemonic processes that were most severely compromised following mTBI. Further, when comparing immediate, delayed, and general (or global) scores for verbal and visual memory tasks, mTBI groups had lower index scores on average in comparison with matched control groups (e.g. Fisher et al., 2000).

Similar results were demonstrated by Yeates et al. (1995) in a paediatric group with blunt head injuries, who performed more poorly on verbal memory than the comparative control group, especially on delayed recall tasks. In fact, both performance tests (Baillargeon et al., 2012) and accuracy tasks on memory and working memory have demonstrated the impact of mTBI on neurophysiological functions in youth (Green et al., 2018); where adolescents showed worse performance outcomes and higher false alarms on non-verbal WM tasks when compared to young children (younger than 7 years) and young adults (older than 18 years).

Functional neuroimaging on memory tasks have also demonstrated greater brain activation in the prefrontal and frontotemporal regions when comparing children and adolescents at 12 months post-mTBI, relative to a matching control group (Westfall et al., 2015). Westfall and colleagues' results showed similar cognitive performance between control and contrast groups, however, brain activation in the mTBI cohort was significantly more pronounced than in the non-mTBI group. These results were interpreted as an indication that compensatory brain-regions activated to support post-mTBI memory performance (Westfall et al., 2015). By comparison, other researchers have suggested that the poor performance demonstrated in mTBI groups can be explained by low effort, rather than necessarily a deficit in ability (Flaro et al., 2007; L'Ecuyer-Giguère et al., 2019). This is evident in studies on executive function tasks, where decreased performance was found in paediatric mTBI participants, as far as six (Baillargeon et al., 2012) and twelve months (Catale et al., 2009) post-injury, relative to matching control groups. Westfall and colleagues also cite higher proactive interference as a possible source for these findings. Consequently, Lajiness-O'Neill et al. (2010) have found anterior cerebral lesions to correlate highly with proactive interference effects and demonstrated that speed of information processing may be a mediating factor.

Similarly, studies have found mild deficits on attention tests several years after mTBI (Hanten et al., 2013). For instance, children who incurred an mTBI 10 years earlier showed significant deficits on tests of attention, freedom from distractibility (Anderson et al., 2012), as well as cognitive flexibility (Catale et al., 2009) in comparison with their non-mTBI peers. Lastly, Catale and colleagues demonstrated decrease selective attention and switching on visual and verbal modality tasks in 6 to 12-year-old children with mTBI in comparison with non-mTBI matched controls, at a period of more than 12 months post-injury.

Other developmental factors; cohort-specific characteristics

An amalgam of connected ideas constitutes the following section. Mainly, this is to limit the scope of discussion and delve into each aspect succinctly. As such, this section attempts to provide an overlap between the previous two sections, notably – mild traumatic brain injury and memory and attention performance – in consideration of two overarching topics: (1) what the possible effects of mTBI are on developmental trajectories according to current literature, and (2) what research currently shows regarding the relationship of multilingual-brains and memory performance. The first instance will look at the literature on both experience-expectant (e.g. it is anticipated that speech is spontaneously learned; i.e. no

formal education is required to speak) and experience-dependent (i.e. skill development such as reading or writing requires appropriate environmental input) plasticity in relation to sensitive- and critical-periods, with a focus on the timeline between *age at injury* and *age at neuropsychological assessment*. The second part explores our current understanding with regards to the relationship between multilingual-cytoarchitecture and memory performance. Collectively, these considerations attempt to characterize the typical *Baragwanath child*.

Development is classically characterized by changes associated with chronological age, that is, morphological, chemical, and cell functions are transformative indicators of development (Carlier & Roubertoux, 2010). This biological unfolding forms part of a fundamental genetic blueprint (Kolb et al., 2013), but is also shaped through various experiences. Accordingly, different environmental factors such as diet, injury, or social relationships tend to correlate with idiosyncratic developmental trajectories. Furthermore, studies indicate that structural and functional trajectories are nonlinear processes (Gogtay et al., 2004), which may prove vital to understanding neurological disorders. That being said, development is not static but rather a function of underlying neuroplasticity (Kolb et al., 2012, 2013) and as such, brain changes that occur throughout life should be analysed at many levels, dependent only on the question being asked.

By comparison, neuroplasticity is generally defined as a structural reorganization as well as the strengthening of synaptic connections within the brain, a consequence of experience and often a precursor to noticeable behavioural and psychological changes (Kolb et al., 2012). Researchers also differentiate among three neural plastic types, suggested to run concurrently through lifelong stages of brain development. These are, experience-independent (occurs largely in prenatal development), experience-expectant (generalized nervous system exposure which drives post-natal development), and experience-dependent plasticity (changes on existing brain structures which occur at a neural and synaptic level based on environmental/ experiential influences) (Blake et al., 2002; Kolb et al., 2013).

Specifically, experience-independent and experience-expectant plasticity associated with structural and functional brain-maturity have been demonstrated to be analogous to critical periods (a special subclass of sensitive periods which results in the formation of critical neural circuits; often irreversible) as demonstrated throughout development. Similarly, experience-dependent plasticity has been shown structurally and therefore functionally linked to sensitive periods (a temporary period in which experiential influences have particularly strong effects on brain development) (Knudsen, 2004). Thus, the notions of typical development, its underlying neuroplasticity, combined with the addition of sensitive-

and critical-periods essentially provide us with an integrated ‘lens’ through which we can attempt to theoretically interpret the effects of mTBI on the trajectory of childhood brain development; that is, it serves as a guideline of a child’s expected development at certain ages, if not functionally disrupted. This helps in retrospectively looking at the age at injury, age at neuropsychological assessment, and importantly, what developmental transitions one would expect to have occurred in the time between these two points.

However, according to Spencer-Smith and Anderson (2009), an important caveat to be reminded of is that the relationship between mastery of neurobehavioral skills and the influence of brain injury on childhood development is not simple, and various trajectories have been observed by researchers. Consequently, the findings on children with mTBI typically report two developmental aspects; transient impairments (i.e. delayed developments expected to recover over time), and emerging deficits (i.e. failure to detect a compromise in undeveloped skills) (Anderson et al., 2011). Although subacute tests may show normal function, an increase in environmental demands over time (e.g. social roles, academic demand, etc) may result in ineffective developmental adjustments (Dennis, 1989), as well as the possibility of ‘growing into’ deficits (Anderson et al., 2011; Banich et al., 1990; Kolb et al., 2013).

For instance, studies on paediatric brain injury have shown normal behaviour at the time of insult, however, when daily demands increase (e.g. organizing chores, planning homework) difficulties related to executive functions emerge (Kolb et al., 2000a, 2004; Westmacott et al., 2009). Comparatively, delayed developmental milestones, due to an onset of neuropathology following early brain injury, have also been demonstrated by researchers (Reilly et al., 1998; Stiles et al., 2009). As an example, one case of brain injury during infancy resulted in a delayed pathology 2 years post-injury, where the gap in developmental expectations (i.e. functional behaviour) progressively widened (Anderson et al., 2011). Furthermore, (Crowe et al., 2012) found moderate support for IQ deficits in testing children with very early brain damage (mild to severe type).

Lastly, studies have also found cerebral (re)organization for language specialization at various ages in children who incurred brain damage. For instance, small contusions during infancy may result in an interhemispheric reorganization, while focal lesions in preschool years have shown intrahemispheric transfer and intrahemispheric maintenance caused by bilateral lesions as well as diffuse injuries result in the poorest functional outcomes, including speech and language delay (Anderson et al., 2004a; Anderson et al., 2005a).

Findings on multilingual memory performance

Cytoarchitecture refers to the multitude of cellular architecture (i.e. neurons and glial cells) of which the heterogeneous brain regions are comprised (Petrides, 2013). Further, current research estimates approximately 50 distinct regions from what can be demonstrated in terms of fMRI localization studies (Amunts et al., 2007; Carlier & Roubertoux, 2010). Moreover, multilingualism refers to a person's capacity to speak and thereby understand a multitude of languages (Higby et al., 2013). This is often analogously used with bilingualism. However, the latter more closely coincides with proficiency in only two languages. Thus, as the phrase suggests, multilingual-cytoarchitecture refers to the neural basis of language.

According to Cenoz and colleagues (2003b), a multilingual brain would most likely be the result of a slightly different network of brain regions than in comparison with bilingual counterparts. However, research on this is still scarce, and as such, how multilinguals contrast from bilinguals remain unclear. Larger contrasts have been drawn between monolinguals and multilinguals. For instance, studies have found that multilinguals have a higher grey matter density in their right-hemispheres and differ in white matter density when compared to monolinguals, as well as differing from bilinguals who acquired a second language later in life (Higby et al., 2013). Furthermore, although the precise mechanisms used to control and switch several languages are yet to be confirmed, findings suggest the same brain regions used in domain-general cognition are recruited (Branzi et al., 2016), such as the prefrontal cortex (Costa et al., 2009), anterior cingulate cortex (ACC), as well as the basal ganglia and associated regions used in memory and planning (Hayakawa & Marian, 2019).

Moreover, the ACC and left caudate which subserve language switching may also be involved in executive functions such as attention and inhibitory control (Abutalebi & Green, 2008). Likewise, when testing mono- and multilinguals on tasks of phonological competition (i.e. simultaneous activation of target word phenome), the monolingual group showed significant activation in brain regions associated with executive functions in comparison to the multilingual group. Hayakawa and Marian (2019) interpret the findings of lower brain activation in multilinguals as more efficient processing in linguistic demands, based on a decreased reliance on cognitive control networks substituted by wider distributed neural networks instead (Higby et al., 2013).

In terms of research on memory performance in multilinguals, various studies have shown a strong relationship between WM and language acquisition (Baddeley et al., 1998; Engel, 2009; Masoura & Gathercole, 2005). Likewise, studies specifically on multilingualism

in children have demonstrated that linguistic expertise strongly interacts with attentional abilities (Videsott et al., 2012), and suggests that language proficiency moderates executive control in multilinguals; that is, in tasks of attention, multilingual children have a higher level of attention and control than matched controls. Additionally, findings on the capacity of working suggest a direct relation in the ability to speak a second or third language (Hummel, 2002). That being said, researchers have also found results contradictory to the above.

For instance, inhibitory control tasks indicate that multilinguals are subject to higher cognitive demands in order to prevent non-target (secondary) language interference (Schroeder & Marian, 2017). By the same token, multilingualism is believed to place more demand on mnemonic processes; requiring not only an increased amount of information to be encoded, stored, and retrieved, but also retrieved from different contexts than monolinguals (Schroeder & Marian, 2017). As such, despite the apparent role of working memory in language processes, results are mixed on the exact nature of this relationship.

Rationale & aim

According to precedent literature, the rationale for the study was based on the following. Although a majority of research on the sequelae of mTBI seem to suggest that approximately 90% of patients (if not more) fully recover, other researchers argue that 10-15% (on average) remain symptomatic of cognitive impairments such as memory and attention difficulties. Moreover, the optimal development of these neuropsychological skills is another cause for concern as studies seem to indicate that mTBI at an early age may result in their delayed development and/ or emerging deficits. Finally, the pertinent nature of memory and attention conceptualised in aspects such as working memory, and its requisite relation to multilingualism on a neurostructural and neurocognitive level as suggested by some researchers, may further complicate the effect(s) of an early mTBI on the developmental trajectory. Therefore, the overall purpose of this study was to investigate memory performance in multilingual-children who have sustained an mTBI.

Specifically, the study aimed to investigate children with a single mTBI at 1-year or more post-injury, by means of archival neuropsychological data which assessed children on memory- and attention-based tasks. In line with the literature, auditory-verbal, visual-spatial, and associate memory aspects were tested on instructed and incidental learning, as well as immediate and delayed recall. Similarly, tasks specifically related to sustained auditory attention (maintenance), shifting, and inhibition formed part of this archival data. Beyond the direct empirical goals of this study, a further aim was to contribute theoretically to its global

study, to assist the development of diagnostic and follow-up assessments (e.g. decision-tree) for rural clinics.

Research methodology

Purpose and objectives

Based on the literature reviewed, and in line with the preceding rationale, the overall purpose of this study was to compare the effects of mTBI on memory performance in a paediatric-cohort with the memory performance of a neurotypical (i.e. non-mTBI matched contrast group) paediatric-cohort using archival data. Thus, the objectives were:

1) Use neuropsychological tests that elicit different aspects of memory and attention function within a paediatric-cohort who have sustained a single mTBI.
2) Use neuropsychological tests that elicit different aspects of memory and attention function within a neurotypical (NT) paediatric-cohort.
3) Use statistical analyses to compare and identify (statistically) significant differences in memory and attention function between the two groups.
4) Use appropriate statistical models to describe and further explore the relationship between the variables of interest and participant demographics.

Research questions

Based on the purpose and objectives, the following questions and their respective hypotheses were used to guide this study:

1. Does psychometrically quantified *verbal learning* for an mTBI cohort differ significantly from a neurotypical cohort in terms of: (a) learning trajectory (trials 1-5), (b) memory span, (c) susceptibility of information-interference (trials 6 & 7), and (d) memory decay (trial 8), on the NEPSY-II List Memory/ Delayed subtests?

H_0: The average test score in verbal-learning for the mTBI-sample equals that of verbal-learning for the neurotypical-sample on the NEPSY-II List Memory/ Delayed subtests.

H_1: The average test score in verbal-learning for the non-mTBI-sample is significantly different than that of verbal-learning for the neurotypical-sample on the NEPSY-II List Memory/ Delayed subtests.

2. Does psychometrically quantified *associated learning* for an mTBI cohort differ significantly from a neurotypical cohort in terms of: (a) immediate recall (trials 1-3), and (b) delayed recall (trial 4), on the NEPSY-II Memory for Names/ Delayed subtests?

H_0: The average test score in associated memory for the mTBI-sample equals that of associated memory for the neurotypical-sample on the NEPSY-II Memory for Names/ Delayed subtests.

H_1: The average test score in associated memory for the mTBI-sample is significantly different than that of associated memory for the neurotypical-sample on the NEPSY-II Memory for Names/ Delayed subtests.

3. Does psychometrically quantified *visual memory* for an mTBI cohort differ significantly from a neurotypical cohort in terms of: (a) learning trajectory (trial 1), (b) incidental recall (trial 2), and (c) delayed recall (trial3), on the WISC-IV Coding subtests?

H_0: The average test score in visual memory for the mTBI-sample equals that of visual memory for the neurotypical-sample on the WISC-IV Coding subtests.

H_1: The average test score in visual memory for the mTBI-sample is significantly different than that of visual memory for the neurotypical-sample on the WISC-IV Coding subtests.

4. Does psychometrically quantified *auditory attention* for an mTBI cohort differ significantly from a neurotypical cohort in terms of: (a) shifting, (b) inhibition, and (c) maintaining on the NEPSY-II Auditory Attention and Response Set subtests?

H_0: The average test score in auditory attention for the mTBI-sample equals that of auditory attention for the neurotypical-sample on the NEPSY-II Auditory Attention and Response Set subtests.

H_1: The average test score in auditory attention for the mTBI-sample is significantly different than that of auditory attention for the neurotypical-sample on the NEPSY-II Auditory Attention and Response Set subtests.

The preceding hypotheses have been proposed as current literature suggests mTBI may adversely impact memory and attention-related functions (Corwin et al., 2017; Lajiness-O'Neill et al., 2010; McInnes et al., 2017, Rose et al., 2015; Wu et al., 2016).

Research design

The study was based within a positive paradigm, and conducted according to a cross-sectional, exploratory, ex post facto, between-subjects design. The mTBI group was not manipulated (i.e. it was a pre-existing condition). Furthermore, the archival data used in this study was drawn from its umbrella project (THINK study) which employed convenience sampling (non-probability) based on volunteer availability.

Criteria: Inclusion & Exclusion

The inclusion and exclusion criteria used to recruit participants is listed here.

Inclusion criteria:

Clinical group (mTBI cohort):

1) Medical records to show prior classification of mTBI.
2) Post-injury recovery (i.e. time elapsed since injury) of 12 months or more.

Entire research sample (both mTBI and contrast groups):

1) All participants to be between the ages of 5-12 years old.
2) All participants admitted (previously or at time of recruitment) to Chris Hani Baragwanath Academic Hospital (CHBAH).

Exclusion criteria:

1) Participants with a history of multiple head injuries (of any severity).
2) Head injuries related to domestic violence or abuse.
3) Participants diagnosed with any psychological or psychiatric disorder, such as a mood (i.e. depression or anxiety) or post-traumatic stress disorder, specific barrier to learning or the likes, following the Diagnostic Statistical Manual of Mental Disorders (DSM-V) criteria, or any clinically diagnosed or other disorder or treatment or procedure (e.g. pre-operation anaesthesia instructions) with the potential to artificially impact on psychometric test performance.

With regards to the inclusion criteria, the age-range was determined based on findings within the literature review concerning the importance of adequate early development, as well as those factors related to developmental delays or emerging deficits. Equally important is demographic equivalence, an assumption this study makes for any participant admitted to CHBAH, as a varying degree of socioeconomic status (SES) has demonstrated an influence

on developmental aspects (Brito & Noble, 2014). Furthermore, as previously discussed, the subacute phase following a head injury tend to show recovery varying between 1-6 months. Thus, in an attempt to control for this possible confounder at study design level, only mTBI participants with head injuries 12 months prior (or more) were included in the study.

Regarding the exclusion criteria, participants with multiple head injuries were not included because repeated concussions are more likely associated with worse prognoses than a single mTBI (Prins et al., 2013), and multiple head injuries become an arbitrary measurement of brain-damage and its neuropsychological sequelae (Katz et al., 2015). Similarly, all participants with head injuries as a result of domestic violence as well as those previously diagnosed with a mental disorder were automatically excluded, as abnormal psychological distress or neuropsychological disorders may profoundly undermine assessment validity and reliability. Lastly, as this study is nested within a larger clinical trial based on the CHBAH-population, no demographic exclusions are applicable, and participants are only excluded under the aforementioned medical criteria that could prove to be confounding.

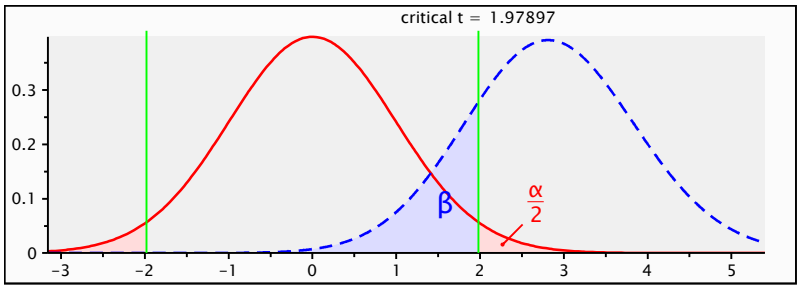
Sample

This study made use of archival records, where an initial sample (N = 60) divided into two groups (ages 5-12 years), was drawn. The clinical group consisted of participants who had sustained a single mTBI at least 12 months prior to assessment, and a contrast group of participants with no medical history of (m)TBI or neuropathological conditions (e.g. cerebral palsy, down syndrome etcetera). The clinical group was selected from the Chris Hani Baragwanath Academic Hospital medical database provided by the Principal Investigator (Dr Vered Lack), per the inclusion and exclusion criteria discussed above. The neurotypical contrast group sample comprised of volunteers, pre-surgery patients who had been admitted to the Paediatric Outpatient Department at CHBAH and were awaiting consultation and/ or treatments that would not jeopardize their neurological functioning in any way.

An *a priori* calculation was performed in G*Power 3.1.9.4 to determine the required number of cases needed to perform independent t-tests (as part of Objective 3 mentioned above) (Kang et al., 2008; Suresh & Chandrashekara, 2012). It was calculated that a two-tailed independent t-test, with an effect size of d = 0.5 (medium), alpha level of $\alpha = 0.05$, and power of $1 - \beta = 0.8$ (80%) would require 128 cases, an even 64 cases in each group.

Figure 1

A priori power calculation of required sample size for independent t-tests.

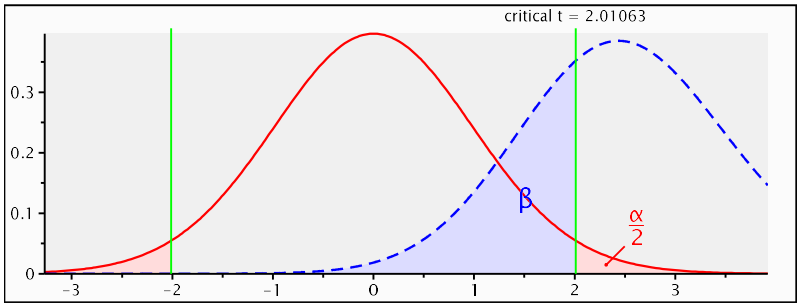


Sample description

However, the final sample acquired had several cases removed based on the inclusion/ exclusion criteria (e.g. mTBI recency, age at assessment, etcetera), and thus a total of 50 cases (25 per group) remained, far less than expected or suggested by the power calculation. Consequently, a post hoc analysis was conducted to revaluate statistical power based on this final sample which revealed the power had shrunk to 0.679 (68%), as shown in Figure 2. It may be worthwhile to mention at this juncture that this was a highly specialised group, subject to an extensive and time-consuming battery of neuropsychological tests, and was difficult to recruit sufficient participants, especially given the advent of precautionary measuring imposed by COVID-19.

Figure 2

Post-hoc power calculation of acquired sample for independent t-tests.



A power of only 68% is considerably lower than the conventional 80%. One repercussion of this is such low power decreases the probability that a statistically significant result signifies a true effect. That being said, Button et al. (2013) conducted a meta-analysis on neuroscience-related research and determined that studies within this discipline often have, on average, 8 – 31% statistical power due to various factors including small sample sizes. More on this is discussed in the limitations and future research recommendations

chapters. An overview of the two groups and their respective participant divisions are presented in Table 1:

Table 1

Sample Description: Distribution of Participants

Demographic variables	Group (n = 50)	
	Contrast (n = 25)	Clinical (n = 25)
Gender distribution (n; %)		
Female (19; 38%)	11 (44%)	8 (32%)
Male (31; 62%)	14 (56%)	17 (68%)
Age[1] at testing		
Mean (SD)	103.72 (17.401)	107.64 (23.196)
Number of languages spoken		
Mean (SD)	2.86 (1.037)	2.72 (0.843)
Age[1] at injury		
Mean (SD)	--	67.52 (22.134)
Time[1] elapsed since injury		
Mean (SD)	--	40.12 (11.208)

[1]Age expressed in months.

As noted in Table 1, the final sample comprised of 50 children, divided into two groups. The contrast group (i.e. neurotypical) comprised of 11 females and 14 males (M_{age} = 103.72, *SD* = 17.401) and was comparable in terms of age at assessment (expressed in months) to the clinical group (i.e. the mTBI-participants) which consisted of 8 females and 17 males (M_{age} = 107.64, *SD* = 23.196). Similarly, with regards to number of languages spoken, the contrast group (M_{age} = 2.86, *SD* = 1.037) and clinical group (M_{age} = 2.72, *SD* = 0.843) were approximately analogous on this variable. However, as indicated, the sample was expected to be skewed with respect to its gender distribution. Lastly, age at injury for the clinical group was approximately five and half years (M_{age} = 67.52, *SD* = 22.134) with an average time elapsed since injury of three years and three months (M_{age} = 40.12, *SD* = 11.208).

Measuring instruments

The following instruments were selected to address the research questions according to the design and inclusion/ exclusion criteria provided above. It is worthwhile to mention that although it may have been beneficial to include instruments that also measure neurological disorders related to depression, anxiety, and attention deficit hyperactivity

disorder (ADHD), this was beyond the scope of the current study and was investigated by another principal investigator within the THINK study.

Demographic Record

The demographic record (DR) is a modified version of the demographic questionnaire prescribed by the THINK study that was used to compile participant information such as age, sex, home language and any other languages in which participants were proficient. Additionally, the DR also allowed the researcher to determine the age at injury and the time elapsed since injury (in months) for participants within the mTBI-group. The purpose of extracting these variables was to see which factors best predicted memory performance within the cohort.

A Developmental Neuropsychological Assessment, Second Edition (NEPSY-II)

The NEPSY-II is a battery assessment used to individually evaluate the neuropsychological development within children and adolescents between the ages of 3 and 16 (Korkman et al., 2007). Its development is based on a combination of Lurian theory and contemporary neuropsychological research in child development (Davis & Matthews, 2010) and comprises of 32 subtests, divided into six theoretical cognitive domains, 1) Attention and Executive Functioning, 2) Language, 3) Memory and Learning, 4) Sensorimotor, 5) Social Perception, and 6) Visuospatial Processing (Korkman et al., 2007).

The NEPSY-II has been standardised according to a Western-representative sample of 1200 children and adolescents, based on a 2003 census in the United States, that were divided into 12 groups ranging from 3 to 14 years old and stratified demographically. Findings in a study by Mulenga et al. (2001) on Zambian-children aged 9 to 11 years, showed the NEPSY (first edition) demonstrated generally lower normative-scores on domains related to Attention and Executive Functions and Memory and Learning, where the younger group performed better in the *Memory for Faces* subtest, in contrast to a better performance in the *Narrative Memory* subtest by older children. They reported that the Zambian cohort performed only marginally lower than the US normative sample on a majority of subtests as well as reporting no age differences in the Language domain, despite English being a non-native language, and thus concluded that there was a test-language familiarity within their sample group. As such, one aim of this study is to investigate how comparable the specific cohort is between-subjects, that is, in relation to each other. Thus, participant-scores will be age-corrected as per

the NEPSY-II manual (i.e. scaled scores), rather than using the cohort as a comparison against the standardised sample.

The following NEPSY-II subtests were used within this study. For the Memory and Learning domain, *List Memory* and *List Memory Delayed* were used to measure the memory span, learning effect, distractibility (i.e. interference), and memory-decay of verbal-memory by using a list of target-words for participants to memorize, ignore, and recall (immediately and delayed). *Memory for Names* and *Memory for Names Delayed* were used to measure associative-visual memory by learning the names of faces (i.e. cards with drawings of children).In terms of the Attention and Executive Function domain, the *Auditory Attention and Response Set* (AARS) subtests were used to measure selective and sustained attention, inhibition, and distractibility. In the former subtest, participants listen to an audio recording and respond appropriately when the target word is read.

Wechsler Intelligence Scale for Children, Fourth Edition: Coding subtest

The WISC-IV is an intelligence test for children aged 6-16, and assesses a child's general intellectual ability based on five indices; Verbal Comprehension Index, Visual Spatial Index, Fluid Reasoning Index, Working Memory Index, and Processing Speed Index (Wechsler, 2003). According to Gomez, Vance, and Watson (2016), one main requisite for assessment and comparisons is that participants are regarded equivalent in terms of demographics (e.g. age, home language and SES). A subtest within the Processing Speed Index used for this study is the *Coding* test (versions A and B).

For version A, the child (aged 6-7) is provided with a key of shapes with symbols which they must use to match similar blank shapes with the appropriate symbols. For version B, the child (aged 8-16) is provided with a key where different symbols are paired to numbers ranging 1 to 9; this key is then used by the child to match as many of the designated symbols according to the list of appropriate numbers within a time limit of 120 seconds (Wechsler, 2003). For the purpose of the THINK study, participants were required to complete three rows of symbol coding (that is, regardless if this takes longer than 120 seconds) to have sufficient exposure to the symbol stimuli. The main objective of this assessment within the current study is to measure a participant's visual memory through incidental learning (i.e. not specifically instructed to memorise the material, but tested on immediate and delayed recall of symbols). According to Wechsler (2003), although this assessment mainly tests speed of processing abilities, auxiliary cognitive domains namely memory, attention, and learning may affect task performance.

Procedure

The following section describes the retrospective procedures that produced the archival records used in this study.

All mTBI-participants (clinical group) identified from the preceding criteria were drawn from a database provided by the Principal Investigator and contacted telephonically by the clinic's administrative staff to invite their participation in the study. In terms of establishing this initial contact, participants were informed of the anticipated procedures and their ethical rights were explained (i.e. confidentiality agreement, rights to withdraw at any time). Prospective mTBI-participants were notified that transportation costs on the day of assessment will be reimbursed and that lunch is provided – this was only communicated in person on their arrival to pre-empt coercion at initial contact. The contrast group was recruited on a voluntary-basis, pre-surgery patients already admitted to the Paediatric Outpatient Department and awaiting consultation and/ or treatments that would not jeopardize neurological functioning. At the time of assessment, the appropriate assent and consent were acquired from all participants and parents/ legal guardians respectively. Potential participants were informed that assessments take place in a designated room in the Glaxo-Kline-Smith Paediatric Clinic at CHBAH, for approximately 2 hours and 30 minutes per participant, conducted select days per week.

Designated subtests from the NEPSY-II and WISC-IV batteries were used to perform assessments on the cohort. A Latin Square Design was utilized on the administered subtests to mitigate possible influences related to test-wiseness and fatigue, as well as minimise any administrative dynamic-effects between the investigator and participant across the sample. For each of the subtests administered, the researcher ensured the participant had understood the instructions clearly before commencing with the assessment.

Administration of the battery started with the List Memory immediate recall subtest. In this assessment, the researcher read from a target-list and the participant was asked to remember as many words as they can. Trial 1 was used as a means to gauge initial memory span (*LM-Memory span*), whereas trials 2-5 were used to gauge the rate of learning (*LM-Learning effect*). Following this, trial 6 featured an interference-list (*LM-Interference*) which the participant was asked to recall, and trial 7 again used the target-list, but without it being read by the researcher. In both the target- and interference-lists, words were changed to remain ethnically-appropriate (e.g. store to shop, pupil to learner). As this subtest examines memory-related functions only the provision of instructions was advised for administrative

purposes as practice trials may adversely have affected the evaluation, validity and final interpretation of test scores. The List Memory delayed-recall (*LM-Delayed*) subtest (trial 8) followed 25-35 minutes after the LM trial 7 subtest, in which the participant was reminded that a list of words (target-list only) was read earlier, and asked if they could recall any of those words. The LM-Delayed subtest attempts to gauge memory decay (i.e. degree of consolidation).

Next, the Memory for Names immediate recall subtest was administered, where an initial learning-trial (i.e. card and associated name presented for 10 seconds) was carried out. Either 6 or 8 cards (participant age-dependent) were presented over 3 trials, and should participants provide the wrong name, errors were corrected with the proper name to facilitate learning. To ensure the test remained ethnically-suitable, two names were changed (e.g. Carl to Sipho, Tonya to Zanele). The Memory for Names delayed-recall (MND) subtest followed 25-35 minutes after the immediate-recall subtest, wherein the researcher showed the participant the age-appropriate number of cards and asked to name the drawn child.

Either one or both AARS tasks were administered, dependent on participant's age. If the child was younger than 6 years of age, only the Auditory Attention task was administered, which involved four coloured paper circles and an audio recording of a list of words. Participants were instructed to perform the response-task when the target-word is heard, but also instructed to elicit no-response when any other (non-target) words were read. Children 6 years and older completed a second task; the Response Set (RS). This added complexity to the first by inverting instructions related to response-tasks, in addition to having different target-words for non-responses. For instance, where in the first subtask (AA) a participant had to inhibit any response upon hearing the word "blue", the second ruleset in the RS required the participant to touch the blue circle upon hearing the word "blue".

Lastly, one of two symbol coding subtests (Coding A and B), was administered depending on the participant's age group (Coding A for 6-7 years, Coding B for 8-16 years). In version A, the participants matched symbols according to distinct shapes based on a key. An initial practise trial was performed, followed by the completion of three rows (trial 1, incidental learning), to ensure sufficient exposure to stimuli. In trial 2 (immediate visual-recall), participants had to draw the appropriate symbol in the corresponding shape. Trial 3 (delayed visual-recall) was administered 25-35 minutes after trial 2, in which participants had to draw all the symbols they could remember. Coding B largely changes the complexity of the symbols presented, and associate numbers with each symbol rather than a distinct shape

as per Coding A. Both versions included a practise (trial 0), incidental learning (trial 1), immediate recall (trial 2), and delayed recall (trial 3).

It is imperative for validity and reliability purposes that the delayed-recall subtests followed 30 minutes after their respective immediate-recall subtests. Where possible, the researcher would administer the entire battery, however, if the need arose, a multilingual-translator was available to assist with test-facilitation.

The collected archival data were scored according to the respective NEPSY-II and WISC-IV manuals, cleaned, coded, and compiled into a spreadsheet using Microsoft Excel, for further statistical analysis in IBM SPSS version 26. As part of this consolidation process, the DR (Appendix A) was completed on a case-by-case basis for the purpose of extracting possible factors (e.g. participant demographic information such as the age of injury or number of languages spoken) which may serve as predictors of memory performance within this cohort.

Ethics

As mentioned, this was a sub-study nested within the THINK project, a registered clinical trial at CHBAH which permitted the retrieval of patient history from CHBAH database, and ethics approval from Human Research Ethics Committee (Medical; M1611113). As the final sample was derived from archival data, the researcher applied for ethics-approval (non-medical) within the above stated nested study.

Furthermore, according to the protocol approved under ethical clearance (M1611113), each participant-case had to sign an assent form, and had their parent or legal guardian sign the appropriate consent form (Appendix B). Moreover, participants were recorded only according to a case number, thereby ensuring strict confidentiality and anonymity. Additionally, the researcher signed a confidentiality agreement to further protect any participant information within the THINK project (Appendix E).

The tests were administered on days convenient for the participant, at the Glaxo-Kline-Smith Paediatric Clinic, CHBAH, with the assistance of Surgeons for Little Lives (Paediatric Surgery Outpatient Clinic). All test-materials were stored within a safe located in an access-restricted boardroom at CHBAH. The hospital has copies of the study, and a summary of each case can be made available to participants and/ or their parents/ legal guardians through administrative staff as well as the Principal Investigator. Any inquiries or request for more information/ concerns regarding the study please address this to the WITS Ethics committee (0117171888).

Data analysis

Analysis of data comprised of the following steps. The original transcripts were scored in accordance with the respective administration and interpretation manuals (i.e. the NEPSY-II and WISC-IV). As part of this process, relevant test scores (e.g. LM-L and MN) were age-corrected to produce scaled scores of the corresponding outcome variables. By comparison, other variables (e.g. Coding immediate and Coding delayed) utilized raw test scores, as ultimately these two groups were directly compared, and not against standardized norms.

Upon completion of this aforementioned process, all data were captured onto a Microsoft Excel spreadsheet for preliminary analysis. All age-related variables were converted into months to be directly comparable, while dichotomous variables were coded accordingly (e.g. '0' = Control group, '1' = Head Injury group).

Cases with grossly incomplete data were removed, while an analysis of cases with only some missing observations revealed a total of 13.73% variables across the dataset were missing values. Although conventional practice in statistical analysis might be to simply delete or eliminate cases with missing values (by means of listwise deletion for example), many researchers argue that there are more suitable alternatives in such instances. For example, Kang (2013) promulgate that maximum likelihood or multiple imputation may serve as more appropriate solutions. Multiple imputation (MI) replaces missing values with tenable values "which contain the natural variability and uncertainty of the right values" (Kang, 2013, p. 405). Essentially, it accomplishes this through performing predictive modelling on existing data and the relative missing data, in order to create a full dataset (i.e. the imputed set). Multiple iterations of this predictive modelling are performed (hence the name, multiple imputation), until various full sets are available for statistical analysis. A final pooled set is the result of combining all the analysed results of imputed datasets to produce an 'aggregate' dataset. One advantage to MI is its ability to model the variability and thereby uncertainty of missing values, which contributes to credible inferential analyses (Kang). Similarly, despite large numbers of missing values or small sample sizes, MI is able to generate appropriate statistical results. That being said, MI can be complex and laborious, especially without the appropriate computer software.

For this study, MI was employed because it (a) reinstates variances of missing values to the predictive model, (b) is robust against violations of normality, (c) and can produce appropriate results despite smaller sample size restrictions. However, to avoid potential bias

in drawing conclusions based on only imputed values, the original data is reported alongside the imputed (pooled) results.

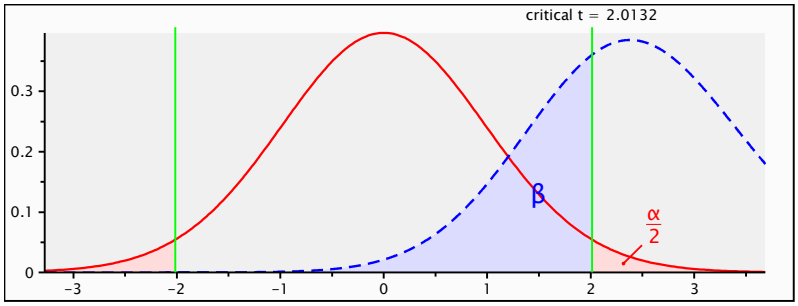
All analyses were conducted in IBM Statistical Package for the Social Sciences (SPSS) version 26. The order of analysis, reported in the next chapter, comprised of the necessary assumption checks, followed by descriptive statistics, group differences, and finally inferential statistics.

Initial assumptions tested to compare the two groups were (a) tests of normality, (b) detection of outliers or extreme values, and (c) the assumption of homogeneity of variance. Shapiro-Wilks test of normality was used for the first assumption, while a combination of box plots and the median absolute deviation (MAD) was used to detect outliers. Levene's test was performed in order to test for homogeneity of variances across all dependent variables.

Due to the parametric assumption of normality being violated to such a large degree, it was argued that a nonparametric alternative, the Mann-Whitney U test be used instead, accompanied with either rank-biserial (*r*), or common language (*f*) as indicative of effect sizes, depending on the distribution shape similarity. This is because the transformation of data would increase interpretation difficulty unnecessarily (Leech & Onwuegbuzie, 2002). By the same token, according to Bridge & Sawilowsky (1998) the nonparametric Mann-Whitney U test is up to four times more powerful than the t-test when used under highly skewed conditions. Figure 3 provides the revised power calculation for the Mann-Whitney U test, calculated at 68%.

Figure 3

Post-hoc power calculation of acquired sample for Mann-Whitney U tests.



Lastly, due to a high degree of similarity found between the two groups, multiple regression analyses focused on the clinical group, in order to model which predictor variables (i.e. participant demographics) contributed the most towards the observed variances in outcome variables.

Results

This section serves to report results on the various statistical analyses conducted. Its structure is as follows. First, the assumption checks necessary to run independent t-tests are provided. Normality assumption on a majority of variables was violated, although outliers were mostly mitigated (or argued to be practically negligible), and homogeneity of variance assumptions passed. Despite positive results from the latter two assumptions, the degree of normality violation raised concerns with regards to possible interpretation and subsequent conclusions drawn. Thus, it was decided to use a nonparametric alternative, the Mann-Whitney U test, which does not rely on assumptions about the population distribution (a limitation of the study discussed later on). Next, descriptive statistics are reported, followed by the results from the inferential statistics; Mann-Whitney U tests and multiple regression analyses.

Assumption checks

In order to assert normality within the sample distribution, Shapiro-Wilk tests were performed on all relevant variables within the two groups, presented below:

Table 2

Assumption Testing: Test of Normality – Shapiro-Wilk Results

		Contrast		Clinical	
Dataset	Variable	*W*	*p-value*	*W*	*p-value*
Original	*AA-C*	.953	.639	.939	.248
	RS-C	.780	.004*	.818	.035*
	AARS-C	.831	.016*	.909	.044*
	LM-C	.832	.017*	.902	.585
	LM Memory span	.859	.038*	.972	.233
	LM Learning effect	.775	.003*	.776	.041*
	LM Interference effect	.785	.004*	.760	.000*
	LM Delay effect	.784	.004*	.821	.001*
	MN (immediate)	.876	.063	.932	.170
	MND (delayed)	.894	.112	.886	.074
	MN-C	.861	.040*	.926	.320
	CO-I	.708	.001*	.783	.012*
	CO-D	.749	.002*	.807	.040*
Imputed (Pooled)	*AA-C*	.939	0.142	.956	.336
	RS-C	.818	.000*	.911	.033*
	AARS-C	.909	.028*	.944	.181

LM-C	.902	.020*	.947	.217
LM Memory span	.972	.688	.915	.040*
LM Learning effect	.776	.000*	.889	.011*
LM Interference effect	.760	.000*	.760	.000*
LM Delay effect	.821	.001*	.795	.000*
MN (immediate)	.932	.094	.931	.093
MND (delayed)	.886	.009*	.920	.050
MN-C	.926	.070	.949	.236
CO-I	.783	.000*	.846	.002*
CO-D	.807	.000*	.853	.002*

* p < .05 demarcates departure from normality; AA-C = Auditory Attention combined; RS-C = Response Set combined; AARS-C = Auditory Attention Response Set combined; LM-C = List Memory combined; LM =List Memory; MN = Memory for Names; MND = Memory for Names Delayed; MN-C = Memory for Names combined; CO-I = Coding immediate; CO-D = Coding delayed.

As per the results in Table 1, Shapiro-Wilk normality tests on the original dataset indicated that *AA-Combined* ($p = .639$), *MN (immediate)* ($p = .063$) and *MND* ($p = .112$) passed the assumption in the contrast group, whereas the remaining ten dependent variables violated the assumption of normality ($p < .05$). Despite the imputation of missing values, the sample's overall normality was not greatly affected, as only one additional dependent variable (i.e. *LM Memory span*) passed this assumption ($p = .688$). By comparison, the clinical group had a total of six variables that passed normality assumptions ($p > .05$) irrespective of which dataset was used (i.e. original or imputed), whereas seven of the dependent variables did not pass assumptions ($p < .05$), as demarcated in the table by an asterisk (*).

As a result of the aforementioned non-normality, two methods were employed to detect any significant outliers; boxplots and median absolute deviation. Although the visual inspection of boxplots is common practice in data analysis, Dawson (2011) suggests that many instances of normal as well as non-normal samples will erroneously flag outliers. Consequently, a complementary alternative was to include the median absolute deviation (MAD), as it is more robust against departures of normality. Table 3 reports on the outlier detection results, where outliers were found in the contrast group, irrespective of the dataset (i.e. original or imputed) utilised, but none in the clinical group:

Table 3

Assumption Testing: Outlier detection

		Contrast		Clinical	
Dataset	Variable	Boxplot	*MAD*	Boxplot	*MAD*

Original	*AA-C*	0	0	0	0
	RS-C	0	0	0	0
	AARS-C	0	0	0	0
	LM-C	2	0	0	0
	LM Memory span	0	0	0	0
	LM Learning effect	0	0	0	0
	LM Interference effect	0	0	0	0
	LM Delay effect	0	0	0	0
	MN (immediate)	1	0	0	0
	MND (delayed)	0	0	0	0
	MN-C	2	0	0	0
	CO-I	0	0	0	0
	CO-D	0	0	0	0
Imputed (Pooled)	*AA-C*	1	0	0	0
	RS-C	0	0	0	0
	AARS-C	1	0	0	0
	LM-C	0	0	0	0
	LM Memory span	2	0	0	0
	LM Learning effect	0	0	0	0
	LM Interference effect	0	0	0	0
	LM Delay effect	0	0	0	0
	MN (immediate)	1	0	0	0
	MND (delayed)	1	0	0	0
	MN-C	0	0	0	0
	CO-I	0	0	0	0
	CO-D	0	0	0	0

AA-C = Auditory Attention combined; RS-C = Response Set combined; AARS-C = Auditory Attention Response Set combined; LM-C = List Memory combined; LM = List Memory; MN = Memory for Names; MND = Memory for Names Delayed; MN-C = Memory for Names combined; CO-I = Coding immediate; CO-D = Coding delayed.

Since tests of normality already indicated distortion in the distribution of the contrast group, the outliers presented in Table 3 were to be expected. Comparatively, MAD indicated no outliers present.

To test the assumption of homogeneity of variance, Levene's test was performed within both groups and for each dependent variable (for the full tabled results, see Table 7 in Appendix G). Overall, non-significant results were found on almost all variables in the original dataset, with the exception of one significant variable, *MN Scaled* $F(48) = 7.954$, $p = .007$. By comparison, values after imputation yielded an overall non-significance on all variables except for two, again *MN Scaled*, $F(48) = 7.954$, and *LM Delay*, $F(48) = 4.109$, $p =$

.048, as well as a borderline case for variable *MN & MND*, F(48) = 4.048, p = .05. As such, there were some heterogenous variances within the sample, but none of analytical concern.

Descriptive statistics

Descriptive statistics are presented in Table 4. For the purpose of remaining consistent with the imputation method employed, both the original and imputed (pooled) results are reported:

Table 4

Descriptive Statistics: Participant Performance Variables

Subtest variable	Contrast group					Clinical group				
Original set	Mean	SD	Median	Min	Max	Mean	SD	Median	Min	Max
AA-C	8.92	4.132	8.0	3	18	6.11	3.085	6.0	1	11
RS-C	9.62	4.556	12.0	1	14	9.83	3.761	12.0	2	14
AARS-C	10.0	4.564	12.0	1	15	10.83	3.839	12.5	4	16
LM-C	7.0	3.559	6.0	3	15	5.78	3.264	5.0	1	12
LM Memory span	4.85	1.864	4.0	2	7	3.83	2.093	4.0	0	7
LM Learning effect	2.0	1.354	1.0	1	5	2.72	1.364	3.0	1	5
LM Interference	2.54	1.613	2.0	1	5	2.22	1.629	1.0	1	5
LM Delay effect	3.92	1.382	5.0	1	5	2.94	1.798	3.0	1	5
MN (immediate)	6.38	3.254	6.0	2	15	6.89	1.875	7.0	4	10
MND (delayed)	6.92	2.753	6.0	4	12	6.17	2.684	6.5	3	11
MN-C	6.15	3.078	6.0	2	14	6.22	2.074	6.0	3	10
CO-I	.701	.367	1.0	.222	1.0	.648	.339	.778	.0	1.0
CO-D	.701	.394	1.0	.0	1.0	.673	.358	.778	.0	1.0
Imputed set										
AA-C	8.12	3.833	8.0	3	18	6.56	3.63	6.0	1	16
RS-C	10.28	3.703	12.0	1	14	9.2	3.606	9.0	2	14
AARS-C	10.68	4.059	12.0	1	16	10.12	3.563	10.0	4	16
LM-C	7.44	4.053	6.0	1	15	6.08	3.328	5.0	1	12
LM Memory span	4.8	2.255	4.0	0	10	3.88	2.333	4.0	0	7
LM Learning effect	2.12	1.394	2.0	1	5	2.64	1.381	3.0	1	5
LM Interference	2.64	1.63	2.0	1	5	2.48	1.686	2.0	1	5
LM Delay effect	3.68	1.345	4.0	1	5	3.12	1.74	3.0	1	5
MN (immediate)	6.88	3.383	6.0	2	15	6.84	1.772	7.0	4	10
MND (delayed)	6.4	2.739	6.0	2	12	6.64	2.531	7.0	3	11
MN-C	6.4	2.986	6.0	2	14	6.32	1.909	6.0	3	10
CO-I	.699	.327	.778	.222	1.0	.7	.32	.778	.0	1.0
CO-D	.7	.341	.889	.0	1.0	.658	.377	.778	.0	1.0

AA-C = Auditory Attention combined; RS-C = Response Set combined; AARS-C = Auditory Attention Response Set combined; LM-C = List Memory combined; LM = List Memory; MN = Memory for Names; MND = Memory for Names Delayed; MN-C = Memory for Names combined; CO-I = Coding immediate; CO-D = Coding delayed.

Inferential statistics: Mann-Whitney U test

As mentioned, a majority of dependent variables violated the assumption of normality in both groups, compromising the validity of inferences drawn. Thus, it was decided to conduct a nonparametric equivalent: the Mann-Whitney U test. The results reported below are divided into two sets; those variables which demonstrated similar distributions, and those which did not (i.e. had dissimilar distributions). Table 5.1 reports on the former:

Table 5.1

Mann-Whitney U test: median, mean rank & effect size – similar shaped distributions

Test variable	Contrast group			Clinical group					
Original dataset	n	Median	m-rank	n	Median	m-rank	U	*p*	*r*
AA-C	20	9.5	26.9	24	6.0	18.83	152	.037*	.314
AARS-C	17	12.0	19.82	21	12.0	19.24	173	.885	.026
LM-C	21	5.0	23.95	23	5.0	21.17	211	.47	.109
LM-M	22	5.0	26.45	24	4.0	20.79	199	.149	.213
LM-L	22	1.0	19.64	24	3.0	27.04	349	.051**	.288
LM-I	22	2.0	25.57	24	2.0	21.6	218.5	.294	.155
Imputed (pooled)									
AA-C	25	8.0	28.56	25	6.0	22.44	236	.136	.211
AARS-C	25	12.0	26.96	25	10.0	24.04	276	.476	.101
LM-C	25	6.0	27.7	25	5.0	23.3	257.5	.283	.152
LM-M	25	4.0	27.86	25	4.0	23.14	253.5	.247	.164
LM-L	25	2.0	22.6	25	3.0	28.4	385	.144	.207
LM-I	25	2.0	26.82	25	2.0	24.18	279.5	.502	.095

* $p < .05$ demarcates departure from normality. AA-C = Auditory Attention combined; RS-C = Response Set combined; AARS-C = Auditory Attention Response Set combined; LM-C = List Memory combined; LM-M = List Memory memory span; LM-L = List Memory learning; LM-I = List Memory interference; MN = Memory for Names; MND = Memory for Names Delayed; MN-C = Memory for Names combined; CO-I = Coding immediate; CO-D = Coding delayed.

Mann-Whitney U tests examined group differences on test performance and found a single dependent variable to be statistically significant. Specifically, *AA-Combined* was greater in the contrast group (*Mdn* = 9.5) than in the clinical group (*Mdn* = 6.0), $U = 152$, $p = .037$. However, *LM Learning effect* approximated statistical significance (i.e. a borderline case), which surprisingly, was greater in the clinical group (*Mdn* = 3.0), $U = 349$, $p = .051$, than in the contrast group (*Mdn* = 1.0), irrespective of which dataset was analysed. This is surprising, especially when one considers the mean ranks of all other LM-related tests (i.e. verbal-learning domain) were numerically greater (albeit not statistically significant) in the

contrast group than the clinical group. For instance, the contrast group, *LM & LMD* (*mean rank* = 23.95), $U = 211$, $p = .47$, *LM Memory span* (*mean rank* = 199), $U = 199$, $p = .149$, and *LM Interference effect* (*mean rank* = 25.57), $U = 218.5$, $p = .294$, had larger differences when compared to the clinical group, respectively, (*mean rank* = 21.17), (*mean rank* = 20.79), and (*mean rank* = 21.6). The aforementioned differences were apparent, irrespective of which dataset (i.e. original or pooled imputed) were used.

Results in Table 5.2 report on the dependent variables which violated the assumption of similarly shaped distributions (Appendix H). Consequently, only *mean ranks* are used to distinguish which group had higher or lower values on the respective variables.

Table 5.2

Mann-Whitney U test: mean rank – dissimilar shaped distributions

Test variable	Contrast group		Clinical group				
Original dataset	n	Mean rank	N	Mean rank	U	*p*	*f*
RS-C	17	20.82	21	18.43	156	.523	.437
LM-D	20	25.02	23	19.37	169.5	.127	.368
MN	25	24.44	25	26.56	339	.604	.542
MND	24	24.15	25	25.82	320.5	.679	.534
MN-C	24	23.65	25	26.30	332.5	.511	.554
CO-I	23	24.96	22	20.95	208	.294	.411
CO-D	21	23.55	24	22.52	240.5	.788	.477
Imputed (pooled)							
RS-C	25	27.26	25	23.74	268.5	.388	.430
LM-D	25	27.9	25	23.1	252.5	.225	.404
MN	25	24.44	25	26.56	339	.604	.542
MND	25	24.4	25	26.6	340	.591	.544
MN-C	25	24.68	25	26.32	333	.687	.533
CO-I	25	26.12	25	24.88	297	.757	.475
CO-D	25	26.58	25	24.42	285.5	.589	.457

AA-C = Auditory Attention combined; RS-C = Response Set combined; AARS-C = Auditory Attention Response Set combined; LM-C = List Memory combined; LM-M = List Memory memory span; LM-L = List Memory learning; LM-I = List Memory interference; MN = Memory for Names; MND = Memory for Names Delayed; MN-C = Memory for Names combined; CO-I = Coding immediate; CO-D = Coding delayed.

Results in Table 5.2 show the following. The contrast group often had numerically higher, albeit not statistically significant, test scores such as LM Delay effect (mean rank = 25.02) or CO immediate (mean rank = 24.96), in comparison to the clinical group (mean rank = 19.37), U = 169.5, p = .127, and (mean rank = 24.96), U = 208, p = .294, respectively. That

being said, in the Memory for Names subtest, the clinical group had higher test scores on all three modalities (i.e. immediate recall, delayed recall, composite score), albeit not statistically significant (mean rank = 26.56), than the contrast group (mean rank = 24.44), U = 339, p = .604. This pattern of results was consistent, irrespective of which dataset was employed.

Inferential statistics: Multiple Regression Analysis

In line with the fourth study objective (i.e. to describe and explore the relationship/s between demographic and response variables), several multiple regression analyses were performed. However, throughout the preliminary stage, numerous response variables were identified which violated assumptions necessary for regression, most prominently linearity and homoscedasticity were not met. Thus, in order to avoid analytical incongruencies as well as erroneous interpretations, only select response variables (i.e. those which met all assumptions) formed part of the final analyses.

Analysis of interest variables followed the same structure. First, step one was a regression analysis of the *base demographics* (i.e. corresponding factors present in both groups such as gender, age at assessment, and number of spoken languages [NOSL]) as independent (predictor) variables, and the relevant response variable as the dependent (outcome) variable. Step two of analyses included the mTBI condition – time elapsed since injury – as an additional predictor variable, again with the respective response variable as the dependent (outcome) variable.

As a precursor to the results that follow, as well as the discussion which ensues, it was anticipated, based on the preceding literature review, that age at injury would be a stronger predictor in the mTBI condition. However, this variable was found to have a high multicollinear relationship with age at testing, an independent variable included in all regressions/ steps. By the same token, multiple comparisons demonstrated zero change in neither the variance nor model-fit with the inclusion of the age at injury variable. As such, it was deemed superfluous to include age at injury in further analysis. A consequence of this is discussed in the limitations section below.

Table 6.1 reports the regression analyses on the response variable, AA-combined:

Table 6.1

Regression analyses with demographics as independent variables and AA-C as dependent variable

Model	Standard coefficients Beta	t	p	*F*	R^2	Adjusted R^2
Step 1		-	.088	2.49	.262	.157

	(Constant)	-	3.858	-	-	-	-
	Gender	-.376	-1.871	.075	-	-	-
	No. of spoken languages	-.224	-1.069	.297	-	-	-
	Age at assessment	-.170	-.820	.421	-	-	-
Step 2			-	.052	2.82	.361	.233
	(Constant)	-	4.323	-	-	-	-
	Gender	-.513	-2.479	.022	-	-	-
	No. of spoken languages	-.269	-1.332	.198	-	-	-
	Age at assessment	-.056	-.270	.790	-	-	-
	Time since injury	-.370	-1.756	.094	-	-	-

The results in Table 6.1 show that the group-equivalent demographic variables in step 1 predicted 15.7% of the variance in AA-combined ($F(3, 21) = 2.49$, $p < .088$). The beta coefficient of gender drew close to significance ($\beta = -.376$, $p < .075$), in comparison to the beta coefficients of number of languages spoken ($\beta = -.224$, $p < .297$) as well as age at assessment ($\beta = -.170$, $p < .421$) of which neither were statistically significant in the first model (step 1). Comparatively, the inclusion of mTBI-condition in step 2 predicted 23.3% variance in AA-combined ($F(4, 20) = 2.82$, $p < .52$) with a borderline statistical significance. The step 2 beta coefficient of gender ($\beta = -.513$, $p < 0.22$) was statistically significant, whereas neither the beta coefficients of NOSL ($\beta = -.269$, $p < .198$), age at assessment ($\beta = -.056$, $p < .790$), nor time since injury ($\beta = -.370$, $p < .094$) were statistically significant, albeit an approach to significance on the latter.

Table 6.2 reports the regression analyses on the response variable, RS-combined:

Table 6.2

Regression analyses with demographics as independent variables and RS-C as dependent variable

Model	(variable)	Standard Beta	t	p	*F*	R^2	Adjusted R^2
Step 1			-	.178	1.801	.205	.091
	(Constant)	-	3.269	-	-	-	-
	Gender	-.442	-2.121	.046	-	-	-
	No. of spoken languages	-.032	-.146	.886	-	-	-
	Age at assessment	.009	.041	.967	-	-	-
Step 2			-	.172	1.781	.263	.115
	(Constant)	-	3.435	-	-	-	-
	Gender	-.547	-2.463	.023	-	-	-
	No. of spoken languages	-.066	-.303	.765	-	-	-
	Age at assessment	.096	.430	.672	-	-	-
	Time since injury	-.284	-1.254	.224	-	-	-

Results from Table 6.2 show that in step 1, the base demographic variables predicted 9.1% of the variance in RS-combined ($F(3, 21) = 1.801$, $p < .178$). The beta coefficient of gender was statistically significance ($\beta = -.442$, $p < .046$), in comparison to the beta coefficients of number of languages spoken ($\beta = -.032$, $p < .886$) and age at assessment ($\beta = .009$, $p < .967$) of which neither were statistically significant in the first model (step 1). By contrast, the inclusion of the mTBI-condition in step 2 predicted 11.5% variance in RS-combined ($F(4, 20) = 1.781$, $p < .172$) with non-statistical significance. The step 2 beta coefficient of gender ($\beta = -.547$, $p < 0.23$) was statistically significant, whereas neither the beta coefficients of NOSL ($\beta = -.066$, $p < .765$), age at assessment ($\beta = -.096$, $p < .672$), nor time since injury ($\beta = -.284$, $p < .224$) were statistically significant.

Next, Table 6.3 reports results of the regression analyses on the response variable, LM-interference:

Table 6.3

Regression analyses with demographics as independent variables and LM-I as dependent variable

Model	(variable)	Standard coefficients Beta	t	p	*F*	R^2	Adjusted R^2
Step 1			-	.013	4.569	.395	.309
	(Constant)	-	4.521	-	-	-	-
	Gender	-.278	-1.526	.142	-	-	-
	No. of spoken languages	.247	1.301	.207	-	-	-
	Age at assessment	-.687	-3.656	.001	-	-	-
Step 2			-	.028	3.393	.404	.285
	(Constant)	-	3.328	-	-	-	-
	Gender	-.236	-1.180	.252	-	-	-
	No. of spoken languages	.261	1.339	.195	-	-	-
	Age at assessment	-.722	-3.591	.002	-	-	-
	Time since injury	.114	.560	.582	-	-	-

Results from Table 6.3 show that in step 1, the base demographic variables predicted 30.9% of the variance in LM-interference ($F(3, 21) = 4.569$, $p < .013$) which was statistically significant. The beta coefficients of gender ($\beta = -.278$, $p < .142$) and number of spoken languages ($\beta = -.247$, $p < .207$) were not statistically significant, in comparison to age at assessment ($\beta = -.687$, $p < .001$) which was statistically significant in the first model (step 1). By comparison, the inclusion of the mTBI-condition in step 2 predicted (a reduced) 28.5% variance in LM-interference ($F(4, 20) = 3.393$, $p < .0.28$) with statistical significance. Step 2 beta coefficients of gender ($\beta = -.236$, $p < 0.252$), number of spoken languages ($\beta = .261$, $p <$

.195), and time since injury (β = .114, p < .582) were not statistically significant, whereas age at assessment (β = -.722, p < .002) was statistically significant.

Furthermore, Table 6.4 presents results of the regression analyses on the response variable, MND:

Table 6.4

Regression analyses with demographics as independent variables and MND as dependent variable

Model	(variable)	Standard coefficients Beta	t	p	*F*	R^2	Adjusted R^2
Step 1			-	.002	6.936	.498	.426
	(Constant)	-	6.890	-	-	-	-
	Gender	-.118	-.711	.485	-	-	-
	No. of spoken languages	-.159	-.917	.370	-	-	-
	Age at assessment	-.640	-3.736	.001	-	-	-
Step 2			-	.006	4.986	.499	.399
	(Constant)	-	5.669	-	-	-	-
	Gender	-.135	-.738	.469	-	-	-
	No. of spoken languages	-.164	-.920	.368	-	-	-
	Age at assessment	-.625	-3.393	.003	-	-	-
	Time since injury	-.047	-.249	.806	-	-	-

Results from Table 6.4 show that in step 1, the base demographic variables predicted 42.6% of the variance in MND ($F(3, 21) = 6.936$, p < .002) which was statistically significant. The beta coefficients of gender (β = -.118, p < .485), and number of spoken languages (β = -.159, p < .370) were not statistically significant, in comparison to age at assessment (β = -.640, p < .001) which was statistically significant in the first model (step 1). By comparison, the inclusion of the mTBI-condition in step 2 predicted 39.9% variance in MND (F(4, 20) = 4.986, p < .0.06) with statistical significance. Step 2 beta coefficients of gender (β = -.135, p < 0.469), number of spoken languages (β = .625, p < .368), and time since injury (β = -.047, p < .806) were not statistically significant, whereas age at assessment (β = -.625, p < .003) was statistically significant.

Lastly, Table 6.5 reports the regression analyses on the response variable, MN & MND (composite) score:

Table 6.5

Regression analyses with demographics as independent variables and MN-C as dependent variable

Model		Standard coefficients Beta	t	p	*F*	R^2	Adjusted R^2
Step 1			-	.246	1.492	.176	.058
	(Constant)	-	4.568	-	-	-	-

	Gender	-.039	-.184	.856	-	-	-
	No. of spoken languages	-.058	-.260	.797	-	-	-
	Age at assessment	-.400	-1.824	.082	-	-	-
Step 2			-	.131	2.017	.287	.145
	(Constant)	-	4.947	-	-	-	-
	Gender	-.185	-.845	.408	-	-	-
	No. of spoken languages	-.105	-.492	.628	-	-	-
	Age at assessment	-.279	-1.268	.219	-	-	-
	Time since injury	-.394	-1.771	.092	-	-	-

Results in Table 6.5 show the base demographic variables (step 1) predicted 5.8% of the variance in MN & MND composite ($F(3, 21) = 1.492$, $p < .246$) which was statistically non-significant. The beta coefficients of gender ($\beta = -.039$, $p < .856$), and number of spoken languages ($\beta = -.058$, $p < .797$) were not statistically significant, in comparison to age at assessment ($\beta = -.400$, $p < .082$) which merely approached statistically significance in the first model (step 1). By comparison, the inclusion of the mTBI-condition in step 2 predicted 14.5% variance in MN & MND composite ($F(4, 20) = 4.947$, $p < .131$), again with statistical non-significance. Step 2 beta coefficients of gender ($\beta = -.185$, $p < .408$), number of spoken languages ($\beta = .105$, $p < .628$), and time since injury ($\beta = -.394$, $p < .219$) were not statistically significant, whereas age at assessment ($\beta = -.279$, $p < .092$) approached statistical significance.

Discussion

A central purpose of this study was to compare the memory and attention of multilingual children with mild traumatic brain injuries, against demographically matched children without such injuries. In order to investigate this comparison between the two groups, archival data on a battery of neuropsychological tests – which assessed different memory modalities (i.e. verbal, visual, and association) as well as auditory attention-based tasks – were analysed.

In accordance with the precedent literature review, it was believed that small cognitive differences would be found between the two groups. To clarify, that is not to say a lack of evidence, nor the suggestion that all differences found, however small, were expected to have strong statistical significance. Rather, as previous research suggests, that children with mTBI demonstrate up to 15% cognitive impairment in comparison to their neurotypical counterpart (McInnes et al., 2017, Prins et al., 2013).

Results from this study provided interesting findings, albeit somewhat expected,

inconclusive, as well as unexpected.

On the whole, the two groups were found to have few to no statistically significant differences in terms of memory and learning performances. This finding seemed evident despite the sample being matched demographically in terms of age, sex distribution (i.e. more males than females in both groups), as well as number of languages spoken. Furthermore, no statistically significant differences, with small to negligible effect sizes, were apparent in spite of utilizing multiple imputation to restore missing values and variances. Different task conditions (e.g. immediate recall, delayed recall, or incidental learning) also seemed nonsignificant with respect to revealing group differences. As such, the findings showed that on all three memory-modalities as well as attention-based tasks, there was little evidence of cognitive deficits present in the clinical group when compared to the control group.

As noted at the outset of this paper, many scholars have corroborated results such as this, in which mTBI-patients perform similar to their neurotypical counterpart (Hanten et al, 2013; Carroll et al., 2004). The findings from this study were consistent with previous research, in which little to no statistical significance, with only small to medium effect sizes, were reported on neuropsychological 'paper and pencil' tests between mTBI- and neurotypical-cohorts (Krivitzky et al., 2011). However, due to a much smaller than expected sample size, the resultant power analysis suggested that the results reported here may be due to statistical chance rather than true effect.

Still, it seems the overall nonsignificance found is in general alignment with contemporary research, most likely indicative of mTBI prognosis which estimates full recovery within 3-12 months post-injury (Dewan et al., 2016, 2019). Taken together, this suggests that the sample showed the expected cognitive recovery, at least outside the sensitivity of the neuropsychological measurements employed.

Notwithstanding the results congruence with other research, some findings were equally unexpected.

Specifically, a single subtask on the original dataset was statistically significant (AA-C, $p < .05$) in which the contrast group scored higher than the clinical on auditory attention (maintenance), whereas a second measurement had borderline significance (LM-L, $p = .051$), in which the clinical group performed surprisingly better than their counterpart on verbal learning. As a measurement of verbal-memory which involves rote learning (i.e. theoretically inclusive of learning strategies such as rehearsal), the LM-L is an untimed test condition, in stark contrast to the timed condition of the (verbal-auditory) AA-subtask (Korkman et al., 2007).

Using MEG measurements, Kaltiainen et al. (2019) detected similar measurable differences between mTBI- and control-subjects on sustained-attention tasks (e.g. the Code Transmission and Stroop Color Word tests), indicative they concluded of damaged white matter tracts, behavioural changes, and/ or different functional states within the clinical group. Of particular importance, a trend in this study showed the control group performed numerically higher on all tasks which had a timed-component (RS-C, AARS-C, LM-I, LM-D, CO-I, and CO-D), in comparison to a converse trend within the clinical group who performed numerically higher on all tasks which lacked a timed-element (MN, MND, MN-C).

Taken together, this finding was interpreted as implying an underlying mechanism of (working) memory function playing a larger role in performance difference. One tentative suggestion regarding the difference on timed- versus untimed-subtasks is that speed of processing, an executive function, may be a factor.

In line with similar research, an mTBI-cohort investigated 5 years post-injury demonstrated compromised processing speed and attentional skill deficits in comparison to the uninjured control group (Catroppa et al., 2007). Likewise, this finding was corroborated in a study linked to the THINK umbrella project; the same sample tested within this study was previously investigated on processing speed, and results suggested statistically significant differences, with the control group performing measurably better than the clinical group on processing speed tasks (Nair, 2020). By comparison, Maillard-Wermelinger et al. (2009) showed an mTBI-cohort performed unexpectedly better than a control group on visuospatial working memory tests, but demonstrated a noticeable trend towards problems on executive function-based tasks in their clinical group.

Consequently, part of the group differences observed here, whether statistically significant or only numerically, may be indicative of attention-based deficits in executive functions of the clinical group, rather than ‘memory deficits’. That being said, on the balance, a meta-analysis by McKinlay (2009) on TBI-studies calls attention to the substantial variability between the methodologies as well as measured mechanisms many of these studies employ. As such, it is difficult to conclusively separate one specific mechanism from others as it pertains to (working) memory and learning. More research on this is warranted.

This study also examined participant demographic variables (i.e. predictors) and their relations to test performances.

Expectantly, male was the stronger predictor in terms of gender contribution on measurement variance for all models. Considering gender distribution in the sample was

largely skewed (i.e. 68% males versus 32% females in the clinical group), this finding makes sense. Interestingly though, contribution of the gender predictor shrank by approximately two thirds (66%) on both Memory for Names subtasks (MND and MN-C). At face value, this effect seems to imply female performance increased substantially on these two subtasks. A similar trend of improved female performance, albeit a smaller effect size, was noted on the LM-I subtask. Consistent with a meta-analytic review, sex differences were present in face memory tasks with females remembering more faces than males, irrespective of whether stimuli comprised of one or both genders (Herlitz & Loven, 2013). By comparison, Lowe et al. found performance on verbal tasks were significantly higher in females than males, although the two groups had nearly equivalent performance on facial memory (2003).

Of particular interest was the negative relationship between the number of languages spoken (NOLS) predictor and outcome variables on all models except for LM-I, a measurement of proactive interference on memory consolidation (Korkman et al., 2007). Foremost, the negative relationship seems to suggest that more languages spoken are indicative of lower outcome performance. However, the inverse result on the interference subtask, seemed to suggest that more languages are indicative of a lower interference-effect (i.e. improved memory consolidation performance), although the effect size is most likely too small to be meaningful. At first glance, the findings may support what was initially hypothesized in accordance with scant literature on compromised developmental trajectories in multilingual cohorts following mTBI (Anderson et al., 2011; Schroeder & Marian, 2017).

However, the positive relationship on LM-I seems to imply, contrary to negative predictions, that language proficiency may serve as a possible buffer on verbal tasks, despite the inclusion of the mTBI-condition (see Table 6.3). In line with previous studies, Rosselli et al. (2019) explored whether a secondary language serves as a neuroprotective effect in a mild cognitively impaired cohort, on which their results suggested that bilinguals outperformed monolinguals on memory assessments – a finding they associated with more inhibitory control in the bilingual group. That being said, current literature suggests that multilinguals have wider distributed neural networks, which may be further complicated in diffuse brain injuries such as mTBI (Cenoz et al., 2003b; Higby et al., 2013). However, the dearth of research on cognitive assessments on multilingual children with mTBI repudiates the verbal-component result found herein, at least in terms of verbal IQ performance (Rosselli et al., 2019). More research on this topic is warranted.

Unexpectantly, findings on the age at assessment (AAA) predictor were largely inconclusive. On all models, barring RS-C, AAA showed a negative relationship with the

outcome variables, contrary to the expectation that higher ages are typically related to increased performance (Mous et al., 2017; Kolb et al., 2015). As such, results suggested the younger participants performed better than their older peers on all outcome variables except for the more complex attention-shifting subtask (RS-C). In line with this, previous research has found that fundamental executive functions (i.e. processing speed) and working memory functions that underly more complex abilities are disrupted followed (m)TBI (Gorman et al., 2016). Though, due to the small sample size, analysis did not include grouping participants into different age bands to explore this finding further, something future research may benefit from.

Lastly, as per the hypothesis that paediatric mTBI in a multilingual cohort may complicate recovery and the expected developmental trajectory, findings on the inclusion of the mTBI-condition in analysis suggest that an increase of time elapsed since injury was related to worse performance on all outcome variables, barring LM-I (a possible neuroprotective effect related to language proficiency mentioned earlier).

Studies on long-term sequalae of early mTBI report similar trends, specifically as cognitive performance relates to working memory and processing speed 5 years post-injury (Catroppa et al., 2007). Of particular interest was the finding that participants with both mTBI and post-concussive syndrome (PCS) conditions demonstrated significantly more errors than those without PCS, even past 1-year post-injury (Dean & Sterr, 2013). Although the results in this study are based on the assumption that ‘more time elapsed since injury’ implies ‘younger age at injury’, this did not form part of the final analysis. That being said, it seems plausible, despite analysis, that this is a reasonable assumption, as previous research demonstrated significantly larger white matter abnormalities in participants subjected to an mTBI at an earlier age (Tremblay et al., 2019).

To recapitulate, findings on intragroup differences for memory and learning performance are mainly statistically nonsignificant, with small to negligible effect sizes and low power, albeit differences were numerically present. The control group performed better on timed memory- and attention-tasks, which is suggestive of higher demand on executive functions, one possibility being processing speed. By contrast, the clinical group predominantly performed better on untimed memory- and attention-tasks, attributed to less demand on executive functions.

In terms of the clinical intergroup variance, some findings showed statistical significance (i.e. gender and age at assessment) with medium to small effect sizes, while other predictor variables were nonsignificant with small to negligible effect sizes.

Sex differences suggested better female performance by up to 66% on visual-associative memory (i.e. memory for names) and up to 33% on verbal-memory performance (specifically associated with an interference effect). Furthermore, the number of languages spoken was associated with worse performance on attention- and visual-memory based subtasks, but had a positive (small) effect on the verbal-memory subtask. Additionally, findings indicate that younger participants seem to perform better than their older peers on 'simple' tasks, but not on 'complex' tasks (e.g. RS-C is more demanding of executive functions). Lastly, as hypothesized, findings suggest that a 'longer history of' the mTBI-condition (indicated by a longer time elapsed since injury) was associated with worse performance on all outcome variables except for the verbal-interference effect, an earlier trend identified, possibly related to language proficiency (i.e. a covariate effect).

An alternative explanation for this pattern of results may be hypothesized by the presence of comorbidities in the sample, not adequately controlled for in the study's design or statistical analyses. In line with the literature, one likely candidate is the existence of ADHD, whether pre- or post-morbid.

Foremost, research on poorer working memory in children with ADHD is fairly ubiquitous. In a recent meta-analysis, Ramos et al. (2019) found verbal WM to be worse in ADHD-cohorts than healthy controls. Interestingly, a negative association between increased participant age and Digit Span Backwards (DSB) performance – a WM subtask of the WISC-IV which involves information manipulation and transformation (Kaufman, 1994 as cited in Wechsler, 2003) – was also evident in Ramos and colleagues' study. Likewise, Kofler et al. (2009) found that ADHD-children showed attention deficits once central executive (CE) processes are overloaded with cognitive demands, but that the healthy controls and ADHD-group performed similarly in terms of storage and rehearsal-processes when tested at normalized capacities. Particularly relevant for the current study, Karatekin (2004) investigated WM performance in ADHD-children within Baddeley's model, and found no substantial differences in STM (i.e. 'buffers') or rehearsal mechanisms (i.e. subvocal recital), but that ADHD-children performed worse on divided attention tasks in comparison to simple response time tasks.

Thus, similar to the trend of the current study, the aforementioned findings suggest that WM performance on certain processes (e.g. storage) are equivalent to healthy controls, but in stark contrast, ADHD-children perform worse on CE-based tasks. That being said, longitudinal studies and comorbidity research highlight the importance of a secondary concern for both mTBI and ADHD groups; pre- and post-morbidity.

A meta-analysis by Adeyemo et al. (2014) documented a statistically significant relationship between mTBI and ADHD. In a follow-up study, it was demonstrated that age of onset for ADHD was prior to mTBI occurrence, and that the rate of ADHD was substantially higher in the mTBI-group than controls (Biederman et al., 2015). Furthermore, Biederman and colleagues also showed that mTBI-subjects with ADHD comorbidity had more acute symptoms (e.g. fatigue or impaired concentration)than mTBI-subjects without ADHD. Lastly, longitudinal studies have also shown an increased risk to develop ADHD after a single mTBI (Yang et al., 2016) as well as multiple mTBI (Li et al., 2017).

Consequently, taken together with the results discussed earlier, it not only seems plausible, but strikingly reasonable to assume that the current study's sample may be subject to such comorbidity. Further investigation is warranted to explore this possibility.

Limitations

Some of the limitations apparent to this study may be found in terms of either theoretical or practical implications.

Foremost, in terms of sample size, the intention at proposal stage was to collect more data in the field to maximise the sample size. However, due to a world epidemic (i.e. Coronavius or COVID-19), research protocols were severely restricted, which ultimately led to the decision to use archival data instead. Consequently, a much smaller sample than was originally anticipated (and calculated for) was acquired, which decreased overall power. Moreover, due to these restrictions, the sampling strategy also did not allow for randomized sampling, which means there was no statistical basis for extending the findings beyond this isolated population. As such, due to a focus of sampling within a specific cohort (i.e. restricted to CHBAH patients), between-subject comparison (internal validity) and generalisability (external validity) will also be limited to individuals within this population. Additionally, although it was found theoretically relevant to group participants into different age-bands, in order to statistically account for growth-spurts and sensitive periods within this age group (Korkman et al., 2013), due to the low power of the acquired sample, it was not feasible to group participants accordingly.

Secondly, as noted by Naidoo (2013), medical records may be inaccurate concerning individuals who have sustained a single head injury. Thus, the possibility of the control group having had a previous head injury, or the clinical group having had more than one previous mTBI cannot be accounted for by any reliable means other than current medical history and parent/ legal guardian disclosure. That being said, additional factors could be present which

were not properly screened (i.e. due to lack of screening measures or availability of data) or statistically controlled for, such as behavioural (ADHD) or psychosocial disorders (depression or anxiety). Consequently, extraneous variables may be present in either one or both groups that influenced assessment performance and thereby the resultant findings. Future studies would benefit from including adequate screening measures or control for such confounds.

Thirdly, and in line with the previous limitation, diagnosis of mTBI are often based on arbitrary measures such as the Glasgow coma scale (Katz et al., 2015). Furthermore, the delimitation between post-concussive syndrome injuries, mTBI, and even moderate TBI are further complicated by the presence of open and closed skull fractures, hypoxia, and other brain-related insults. By the same token, despite decades worth of studies and theoretical advances on aspects related to memory and attention, some of the most prolific researches are still not in agreement on much of the literature, which isolates interpretations of a study such as this within one specific paradigm.

Finally, both the NEPSY-II and WISC-IV assessment batteries have been developed internationally (i.e. according to Westernised norms), and thus may be subject to bias and not cross-culturally appropriate to South Africa's diverse (i.e. non-English speaking) cultural groups (Shuttleworth-Edwards, Gaylard & Radloff, as cited in Laher & Cockcroft, 2013). Moreover, subtle instrument adaptations were made, to ethnically align test items to the appropriate cultural and linguistic setting. Although the items changed could be regarded as inconsequential (e.g. changed the word "store" to "shop" on the List Word subtask), (Olson & Jacobson, 2015) argue that construct validity in neuropsychological measures are often based on the assumption that the person undergoing assessment have been exposed and can relate to the task. Consequently, despite validity and reliability concerns on adapted instruments, this was also a low-SES population, which means this assumption may not be unequivocally warranted. Future research would benefit from developing culturally-appropriate norms and/ or instruments.

Contributions, implications, and future research recommendations

The current literature in South Africa on paediatric mTBI research is in short supply. As such, it is a hopeful aspiration that the results and their interpretations provided by this study, albeit somewhat limited in its generalisability to a unique population (i.e. the CHBAH child), have contributed to ongoing research. Not only was this a highly diverse group

comprised of numerous cultures, but the multilingual-aspect which underlies this population is as idiosyncratic as it is widespread. Thus, a theoretical neurodevelopmental assumption borne out of this setting that may assist and inform future studies is the perspective of a multilingual cytoarchitecture.

Practically, it is speculated that the contribution of this study and future replications/ improvements may assist in the development of decision trees for rural clinics, to appropriate diagnose, formalise treatment(s), and ensure follow-up visitations. That is, to not dismiss paediatric mTBI as a 'trivial' issue. In line with this, an implication of this study may be highlighted in the importance for medical practitioners to pursue longitudinal follow-up visitations on mTBI-patients. For instance, Naidoo (2013) notes the peak admission age for paediatric mTBI in Southern Africa was 6 years, which may have profound consequences on developmental trajectories as discussed in the literature review and in accordance with the interpretations here. Critically, age-appropriate treatments on acute, subacute, and long-term levels may be warranted. Similarly, a theoretical implication emphasized by this study is inherent in the need to develop culturally-appropriate norms for these age groups and respective populations.

Future research would benefit from acquiring a larger sample to assist with statistical analyses. This may be beneficial in terms of two different perspectives. Foremost, despite the opportunity to mitigate missing values as were inherent in this sample, a larger sample could have the added benefit of meeting all necessary parametric assumptions (e.g. such as normality which was highly violated in the current study), enabling a wider range of inferential statistics and consequently the conclusions drawn. However, assuming the population is non-normal, a larger sample would also allow researchers to utilize robust statistics (i.e. trimmed means) that are arguably more useful in decreasing type II errors, or able to better detect genuine associations among variables (Erceg-Hurn et al., 2013).

Future research could also benefit from adjusting units of measurement utilized here. For example, participants can be placed in accordance with different age-bands, so chronological developmental ages are more directly comparable, rather than operating on a continuum. By the same token, years could be used instead of months, as the latter may be too large to detect a true difference between and within groups. Lastly, in light of the alternate explanation, future research would also benefit from including appropriate screening measures to detect/ control comorbidities.

Conclusion

Besides unreported cases and scant literature, paediatric mTBI is still largely viewed as trivial and benign. Although statistically smaller than more severe counterparts, mTBI may adversely affect typical developmental trajectory necessary for optimal cognitive skills. Among many complaints, memory and attention are some key cognitive areas disrupted in these cohorts. The present study focused on investigating whether group differences were present in children with mTBI, in comparison to children without mTBI. It utilized an ex-post facto design on archival data of neuropsychological test batteries (comprising of NEPSY-II and WISC-IV subtests) across various memory-modalities as well as an auditory-attention task. Statistical analyses indicated – in line with much of contemporary literature – a numerical but nonsignificant advantage for control group performance over the clinical group. Additionally, results showed a statistically significant advantage on an auditory-attention (maintenance) task for the control group, in converse to an approximated significance for rote verbal learning in the clinical group. This finding suggested there may be an underlying (central executive) 'attention' component of working memory that resulted in better performance on timed tasks for the control group, indicative of a developmental delay or emerging deficit in the clinical group. Further regressions seemed to support this interpretation, as younger mTBI participants were predicted to perform better than older participants in a simple but not complex attention task, as well as predicting higher visual- and associative-memory performance. However, some apparent inconsistencies were associated between an increased number of languages (small effect) and verbal-memory performance, which warrant further investigation into paediatric polyglot cohorts affected by mTBI.

References

Abutalebi, J., & Green, D.W. (2008). Control mechanisms in bilingual language production: Neural evidence from language switching studies. *Language and Cognitive Processes, 23*, 557–582.

Adeyemo, B. O., Biederman, J., Zafonte, R., Kagan, E., Spencer, T. J., Uchida, M., Kenworthy, T., Spencer, A. E., & Faraone, S. V. (2014). Mild traumatic brain injury and ADHD: a systematic review of the literature and meta-analysis. *Journal of attention disorders, 18(7)*, 576–584. https://doi.org/10.1177/1087054714543371

Amunts, K., Schleicher, A., & Zilles, K. (2007). Cytoarchitecture of the cerebral cortex—More than localization. *NeuroImage, 37(4)*, 1061–1065. https://doi.org/10.1016/j.neuroimage.2007.02.037

Anderson, V., Anderson, P., Grimwood, K., & Nolan, T. (2004). Survivors of childhood bacterial meningitis: impact of neurological complications and age at illness 12 years post-illness. *J Pediatr Psychol, 29*, 67–81.

Anderson, V., Catroppa, C., Morse, S., Haritou, F., & Rosenfeld, J. (2005). Functional plasticity or vulnerability following early brain injury? *Pediatrics, 116*, 374–82.

Anderson, V., Godfrey, C., Rosenfeld, J.V. & Catroppa, C. (2012). 10 years outcome from childhood traumatic brain injury. *International Journal of Developmental Neuroscience, 30*, 217-224. doi:10.1016/j.ijdevneu.2011.09.008

Anderson, V., Spencer-Smith, M., & Wood, A. (2011). Do children really recover better? *Neurobehavioural plasticity after early brain insult. Brain, 134(8)*, 2197–2221. https://doi.org/10.1093/brain/awr103

Appenteng, R., Nelp, T., Abdelgadir, J., Weledji, N., Haglund, M., Smith, E., Obiga, O., Sakita, F. M., Miguel, E. A., Vissoci, C. M., Rice, H., Vissoci, J. R. N., & Staton, C. (2018). A systematic review and quality analysis of pediatric traumatic brain injury clinical practice guidelines. *PLOS ONE, 13(8)*. https://doi.org/10.1371/journal.pone.0201550

Araujo, G. C., Antonini, T. N., Monahan, K., Gelfius, C., Klamar, K., Potts, M., Yeates, K. O., & Bodin, D. (2014). The Relationship Between Suboptimal Effort and Post-Concussion Symptoms in Children and Adolescents With Mild Traumatic Brain Injury. *The Clinical Neuropsychologist, 28(5)*, 786–801. https://doi.org/10.1080/13854046.2014.896415

Araki, T., Yokota, H., & Morita, A. (2017). Pediatric Traumatic Brain Injury: Characteristic Features, Diagnosis, and Management. *Neurologia Medico-Chirurgica, 57(2)*, 82–93. https://doi.org/10.2176/nmc.ra.2016-0191

Babikian, T., Satz, P., Zaucha, K., Light, R., Lewis, R. S., & Asarnow, R. F. (2011). The UCLA Longitudinal Study of Neurocognitive Outcomes Following Mild Pediatric Traumatic Brain Injury. *Journal of the International Neuropsychological Society, 17(05)*, 886–895. https://doi.org/10.1017/S1355617711000907

Baddeley, A. (2007). Oxford psychology series: Vol. 45. Working memory, thought, and action. Oxford University Press. https://doi.org/10.1093/acprof:oso/9780198528012.001.0001

Baddeley, A. (2000). The episodic buffer: A new component of working memory? *Trends in Cognitive Sciences, 4(11)*, 417–423. https://doi.org/10.1016/S1364-6613(00)01538-2

Baddeley, A. (2003). Working memory and language: An overview. *Journal of Communication Disorders, 36(3)*, 189–208. https://doi.org/10.1016/S0021-9924(03)00019-4

Baddeley, A. D., Gathercole, S. E., & Papagno, C. (1998). The phonological loop as a language learning device. *Psychological Review, 105(1)*, 158-173.

Baddeley, A.D., & Hitch, G.J. 1974. Working memory. In: Bower G (ed), The psychology of learning and motivation (Vol 8). New York: Academic Press. 47–90.

Baillargeon, A., Lassonde M., Leclerc S., & Ellemberg D. (2012). Neuropsychological and neurophysiological assessment of sport concussion in children, adolescents and adults. *Brain Inj., 26(3)*, 211–220.

Banich, M., Cohen-Levine, S., Kim, H., & Huttnlocher, P. (1990). The effects of developmental factors on IQ in hemiplegic children. *Neuropsychologia, 28*, 35–47.

Bathelt, J., Gathercole, S. E., Johnson, A., & Astle, D. E. (2018). Differences in brain morphology and working memory capacity across childhood. *Developmental Science, 21(3)*. https://doi.org/10.1111/desc.12579

Bauer, P. J. (2013). Memory. In P. D. Zelazo (Ed.), The Oxford Handbook of Developmental Psychology, 1, 504–541. https://doi.org/10.1093/oxfordhb/9780199958450.013.0018

Belur, P.K., Chang, J.J., He, S et al. (2013). Emerging experimental therapies for intracerebral hemorrhage: targeting mechanisms of secondary brain injury. *Neurosurg Focus, 34*.

Biederman, J., Feinberg, L., Chan, J., Adeyemo, B. O., Woodworth, K. Y., Panis, W., McGrath, N., Bhatnagar, S., Spencer, T. J., Uchida, M., Kenworthy, T., Grossman, R.,

Zafonte, R., & Faraone, S. V. (2015). Mild Traumatic Brain Injury and Attention-Deficit Hyperactivity Disorder in Young Student Athletes. *The Journal of nervous and mental disease, 203(11)*, 813–819. https://doi.org/10.1097/NMD.0000000000000375

Blake, D., Strata, F., Churchland, A., & Merzenich, M.M., 2002. Neural correlates of instrumental learning in primary auditory cortex. *Proc. Natl. Acad. Sci. U. S. A. 99*, 10114–10119.

Bouchacourt, F., & Buschman, T. J. (2019). A Flexible Model of Working Memory. https://linkinghub.elsevier.com/retrieve/pii/S0896627319303770

Branzi, F.M., Calabria, M., Boscarino, M.L., & Costa, A. (2016). On the overlap between bilingual language control and domain-general executive control. *Acta Psychol., 166*, 21–30. https://doi.org/10.1016/j.actps y.2016.03.001.

Bridge, P. D., & Sawilowsky, S. S. (1999). Increasing physicians' awareness of the impact of statistics on research outcomes: comparative power of the t-test and and Wilcoxon Rank-Sum test in small samples applied research. *Journal of clinical epidemiology, 52(3)*, 229–235. https://doi.org/10.1016/s0895-4356(98)00168-1

Brita, N. H., Noble, K. G. (2014). Socioeconomic status and structural brain development. *Frontiers in Neuroscience, 8*. DOI=10.3389/fnins.2014.00276

Brooks, B. L., Sherman, E. M. S., & Strauss, E. (2009). NEPSY-II: A Developmental Neuropsychological Assessment, Second Edition. *Child Neuropsychology, 16(1)*, 80–101. https://doi.org/10.1080/09297040903146966

Burns, T. G., & Yosick, R. (2013). Mild Traumatic Brain Injury in Children and Adolescents: From Basic Science to Clinical Management. *Applied Neuropsychology: Child, 2(1)*, 78–79. https://doi.org/10.1080/21622965.2012.742793

Button, K., Ioannidis, J., Mokrysz, C. et al. (2013). Power failure: why small sample size undermines the reliability of neuroscience. *Nat Rev Neurosci 14*, 365–376. https://doi.org/10.1038/nrn3475

Cannella, L. A., McGary, H., & Ramirez, S. H. (2019). Brain interrupted: Early life traumatic brain injury and addiction vulnerability. *Experimental Neurology, 317*, 191–201. https://doi.org/10.1016/j.expneurol.2019.03.003

Carlier, M., & Roubertoux, P. (2010). Genetics and cognition: The impact for psychologists in applied settings. *European Psychologist, 15(1)*, 49–57. https://doi.org/10.1027/1016-9040/a000007

Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L., Paniak, C., Pépin, M., & WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury (2004). Prognosis for mild traumatic brain injury: results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. Journal of rehabilitation medicine, (43 Suppl), 84–105. https://doi.org/10.1080/16501960410023859

Catale, C., Marique, P., Closset, A., & Meulemans, T. (2009). Attentional and executive functioning following mild traumatic brain injury in children using the Test for Attentional Performance (TAP) battery. *Journal of Clinical and Experimental Neuropsychology, 31*, 331-338.

Catroppa, C., Anderson, V. A., Morse, S. A., Haritou, F., & Rosenfeld, J. V. (2007). Children's attentional skills 5 years post-TBI. *Journal of pediatric psychology, 32(3)*, 354–369. https://doi.org/10.1093/jpepsy/jsl019

Cenoz, J., Hufeisen, B., & Jessner, U. (2003b). Why investigate the multilingual lexicon? In J. Cenoz, B. Hufeisen, & U. Jessner (Eds.), The multilingual lexicon (pp. 1–9). Dordrecht, the Netherlands: Kluwer Academic.

Chun, M. M. and Johnson, M. K. (2011). Memory: Enduring traces of perceptual and reflective attention. Neuron, 72, 520–535.

Cockcroft, K. (2015). The role of working memory in childhood education: Five questions and answers. *South African Journal of Childhood Education, 5(1)*, 18. https://doi.org/10.4102/sajce.v5i1.347

Corwin, D. J., Grady, M. F., Joffe, M. D., & Zonfrillo, M. R. (2017). Pediatric Mild Traumatic Brain Injury in the Acute Setting. *Pediatric Emergency Care, 33(9)*, 643–649. https://doi.org/10.1097/PEC.0000000000001252

Costa, A., Hernández, M., Costa-Faidella, J., & Sebastián-Gallés, N. (2009). On the bilingual advantage in conflict processing: Now you see it, now you don't. *Cognition, 113(2)*, 135–149.

Cowan, N., & Alloway, T. (2009). Development of working memory in childhood. In M. L. Courage & N. Cowan (Eds.), The development of memory in infancy and childhood (pp. 303–342). New York: Taylor & Francis.

Cristofori, I., & Grafman, J. (2019). Traumatic Brain Injury. In M. L. Alosco & R. A. Stern (Eds.), The Oxford Handbook of Adult Cognitive Disorders (pp. 503–525). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780190664121.013.24

Crowe, L.M., Catroppa, C., Babl, F.E., Rosenfeld, J.V. and Anderson, V. (2012) Timing of Traumatic Brain Injury in Childhood and Intellectual Outcome. *Journal of Pediatric Psychology, 37(7)*, 745–754. https://doi.org/10.1093/jpepsy/jss070

D'Amato, R.C. & Hartlage, L.C. (Eds.). (2008). Essentials of Neuropsychological Assessment: Treatment Planning for Rehabilitation. (2nd edn.) New York: Springer Publishing Company.

Davis, J.L and Matthews, R. N. (2010). NEPSY-II Review: Korkman, M., Kirk, U., & Kemp, S. (2007). "NEPSY--Second Edition" (NEPSY-II). San Antonio, TX: Harcourt Assessment. *Journal of Psychoeducational Assessment, 28(2)*, 175-182.

Dawson, R. (2011). How Significant is a Boxplot Outlier?. Journal of Statistics Education, 19(2). DOI: 10.1080/10691898.2011.11889610

De Koning, M.E., Scheenen, M.E., van der Horn, H.J., Hageman, G., Roks, G., Spikman, J.M. & van der Naalt, J. Non-hospitalized patients with mild traumatic brain injury: The forgotten minority. *J. Neurotrauma, 34*, 257–261.

De Koning, M.E., Scheenen, M.E., van der Horn, H.J., Hageman, G., Roks, G., Yilmaz, T., Spikman, J.M., & van der Naalt, J. (2017). Outpatient follow-up after mild traumatic brain injury: Results of the UPFRONT-study. *Brain Inj.*

Dean, D.C., O'Muircheartaigh, J., Dirks, H., Waskiewicz, N., Walker, L., Doernberg, E., … Deoni, S.C. (2014). Characterizing longitudinal white matter development during early childhood. *Brain Structure and Function, 220*, 1921–1933.

Dean, P. J., & Sterr, A. (2013). Long-term effects of mild traumatic brain injury on cognitive performance. *Frontiers in human neuroscience, 7*, 30. https://doi.org/10.3389/fnhum.2013.00030

Dennis M. (1989). Language and the young damaged brain. In: Boll T, Bryant BK, editors. Clinical neuropsychology and brain function: research, measurement and practice. Washington: American Psychological Association; 1989. p. 85–124.

Dewan, M. C., Mummareddy, N., Wellons, J. C., & Bonfield, C. M. (2016). Epidemiology of Global Pediatric Traumatic Brain Injury: Qualitative Review. *World Neurosurgery, 91*, 497-509. https://doi.org/10.1016/j.wneu.2016.03.045

Engel, P. M. J. (2009). Working memory and learning: A 3-year longitudinal study of children growing up in a multi-lingual environment. University of York, York.

Eichenbaum, H., & Cohen, N. J. (2001). From conditioning to conscious recollection: Memory systems of the brain. New York: Oxford University Press.

Erceg-Hurn, D. M., Wilcox, R. R., & Keselman, H. J. (2013). Robust Statistical Estimation. In T. D. Little (Ed.), The Oxford Handbook of Quantitative Methods (Vol. 1, pp. 388-406). Oxford University Press.

Faul, M., & Coronado, V. (2015). Epidemiology of traumatic brain injury. *In Handbook of Clinical Neurology, 127*, 3–13. Elsevier. https://doi.org/10.1016/B978-0-444-52892-6.00001-5

Fisher, D. C., Ledbetter, M. F., Cohen, N. J., Marmor, D., & Tulsky, D. S. (2000). WAIS-III/WMS-III profiles for mildly to severely brain-injured patients. *Applied Neuropsychology, 7(3)*, 126–132.

Flaro, L., Green, P., Flaro, L., Green, P., & Robertson, E. (2007). Word Memory Test failure 23 times higher in mild brain injury than in parents seeking custody: The power of external incentives. *Brain Injury, 21(4)*, 373–383.

Frencham, K. A. R., Fox, A. M., & Maybery, M. T. (2005). Neuropsychological Studies of Mild Traumatic Brain Injury: A Meta-Analytic Review of Research Since 1995. *Journal of Clinical and Experimental Neuropsychology, 27(3)*, 334–351. https://doi.org/10.1080/13803390490520328

Fuster, J. M. (2002). Frontal lobe and cognitive development. *Journal of Neurocytology, 31*, 373–385.

Gathercole, S. E., Pickering, S. J., Ambridge, B., & Wearing, H. (2004). The Structure of Working Memory From 4 to 15 Years of Age. *Developmental Psychology, 40(2)*, 177–190. https://doi.org/10.1037/0012-1649.40.2.177

Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2014). Cognitive neuroscience: The biology of the mind (Fourth edition). W. W. Norton & Company, Inc.

Gennarelli, T. A. (1993). Mechanisms of brain injury. *Journal of Emergency Medicine, 11*, 5–11.

Gennarelli, T. A., Thibault, L. E., & Graham, D. I. (1998). Diffuse Axonal Injury: An Important Form of Traumatic Brain Damage. *The Neuroscientist, 4(3)*, 202–215. https://doi.org/10.1177/107385849800400316

Giedd J, Blumenthal J, Jeffries N, Castellanos F, Liu H, Zijdenbos A, et al. (1999). Brain development during childhood and adolescence: a longitudinal MRI study. *Nat Neurosci, 2*, 861–3.

Giza, C. C. (2006). Lasting effects of pediatric traumatic brain injury. *The Indian Journal of Neurotrauma, 3(1)*, 19–26. https://doi.org/10.1016/S0973-0508(06)80005-5

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., Nugent, T. F., Herman, D. H., Clasen, L. S., Toga, A. W., Rapoport, J. L., & Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences, 101(21)*, 8174–8179. https://doi.org/10.1073/pnas.0402680101

Goldstein, M. (1990). Editorial. Traumatic brain injury: A silent epidemic. *Annals of Neurology, 27(3)*, 327.

Goldstrohm, S., & Arffa, S. (2005). Preschool children with mild to moderate traumatic brain injury: An exploration of immediate and post-acute morbidity. *Archives of Clinical Neuropsychology, 20(6)*, 675–695. https://doi.org/10.1016/j.acn.2005.02.005

Gomez, R., Vance, A., & Watson, S. D. (2016). Structure of the Wechsler Intelligence Scale for Children - Fourth Edition in a Group of Children with ADHD. *Frontiers in psychology, 7*, 737. doi:10.3389/fpsyg.2016.00737

Gorman, S., Barnes, M. A., Swank, P. R., Prasad, M., Cox, C. S., Jr, & Ewing-Cobbs, L. (2016). Does processing speed mediate the effect of pediatric traumatic brain injury on working memory? *Neuropsychology, 30(3)*, 263–273. https://doi.org/10.1037/neu0000214

Green, S. L., Keightley, M. L., Lobaugh, N. J., Dawson, D. R., & Mihailidis, A. (2018). Changes in working memory performance in youth following concussion. *Brain Injury, 32(2)*, 182–190. https://doi.org/10.1080/02699052.2017.1358396

Greco, T., Ferguson, L., Giza, C., & Prins, M. L. (2019). Mechanisms underlying vulnerabilities after repeat mild traumatic brain injuries. *Experimental Neurology, 317*, 206–213. https://doi.org/10.1016/j.expneurol.2019.01.012

Hanten, G., Li, X., Ibarra, A., Wilde, E.A., Barnes, A., McCauley, S.R., McCarthy, J., Hoxhaj, S., Mendez, D., Hunter, J.V., Levin, H.S. and Douglas H. Smith. (2013). Updating Memory after Mild Traumatic Brain Injury and Orthopedic Injuries. *Journal of Neurotrauma, 30(8)*, 618-624. https://doi.org/10.1089/neu.2012.2392

Hawryluk, G.W.J., & Manley, G.T. (2015). Classification of traumatic brain injury: past, present, and future. Handbook of Clinical Neurology, 127 (3rd series). Traumatic Brain Injury, Part I

Hayakawa, S., & Marian, V. (2019). Consequences of multilingualism for neural architecture. *Behavioral and Brain Functions, 15(1)*, 6. https://doi.org/10.1186/s12993-019-0157-z

Henry, L. (2012). The Development of Working Memory in Children. SAGE Publications Ltd. https://doi.org/10.4135/9781446251348

Herlitz, A. & Lovén, J. (2013). Sex differences and the own-gender bias in face recognition: A meta-analytic review. *Visual Cognition, 21(9-10)*, 1306-1336, DOI: 10.1080/13506285.2013.823140

Higby, E., Kim, J., & Obler, L. K. (2013). Multilingualism and the Brain. *Annual Review of Applied Linguistics, 33*, 68–101. https://doi.org/10.1017/S0267190513000081

Houde, O., Rossi, S., Lubin, L., & Joliot, M. (2010). Mapping numerical processing, reading, and executive functions in the developing brain: An fMRI meta-analysis on 52 studies including 842 children. *Developmental Science, 13*, 876–885.

Hummel, K. M. (2002). Second language acquisition and working memory. In F. Fabbro (Ed.), Advances in the neurolinguistics of bilingualism, 95–117. Udine, Italy: Forum.

Iverson, G. L. (2005). Outcome from mild traumatic brain injury. Current Opinion in Psychiatry, 18(3), 301–317.

Jaffe, K. M., Fay, G. C., Polissar, N. L., Martin, K. M., Shurtleff, H. A., Rivara, J. B., et al. (1992). Severity of pediatric traumatic brain injury and early neurobehavioral outcome: A cohort study. *Archives of Physical Medicine and Rehabilitation, 73*, 540–547.

Jeret JS, Mandell M, Anziska B, et al. (1993). Clinical predictors of abnormality disclosed by computed tomography after mild head trauma. *Neurosurgery, 32*, 9-15.

Kaltiainen, H., Liljeström, M., Helle, L., Salo, A., Hietanen, M., Renvall, H., & Forss, N. (2019). Mild Traumatic Brain Injury Affects Cognitive Processing and Modifies Oscillatory Brain Activity during Attentional Tasks. *Journal of neurotrauma, 36(14)*, 2222–2232. https://doi.org/10.1089/neu.2018.6306

Kang, M., Ragan, B. G., & Park, J. H. (2008). Issues in outcomes research: an overview of randomization techniques for clinical trials. *Journal of athletic training, 43(2)*, 215–221. https://doi.org/10.4085/1062-6050-43.2.215

Kang H. (2013). The prevention and handling of the missing data. *Korean journal of anesthesiology, 64(5)*, 402–406. https://doi.org/10.4097/kjae.2013.64.5.402

Karatekin, C. (2004). A test of the integrity of the components of Baddeley's model of working memory in attention-deficit/hyperactivity disorder (ADHD). *Journal of Child Psychology and Psychiatry, 45(5)*, 912–926. https://doi.org/10.1111/j.1469-7610.2004.t01-1-00285.x

Karr, J. E., Areshenkoff, C. N., & Garcia-Barrera, M. A. (2014). The neuropsychological outcomes of concussion: A systematic review of meta-analyses on the cognitive sequelae of mild traumatic brain injury. *Neuropsychology, 28(3)*, 321.

Katz, D. I., Cohen, S. I., & Alexander, M. P. (2015). Mild traumatic brain injury. *In Handbook of Clinical Neurology, 127*, 131–156. https://doi.org/10.1016/B978-0-444-52892-6.00009-X

Kirkwood, M. W., Peterson, R. L., Connery, A. K., Baker, D. A., & Grubenhoff, J. A. (2014). Postconcussive Symptom Exaggeration After Pediatric Mild Traumatic Brain Injury. *PEDIATRICS, 133(4)*, 643–650. https://doi.org/10.1542/peds.2013-3195

Kiyonaga, A., & Egner, T. (2013). Working memory as internal attention: Toward an integrative account of internal and external selection processes. *Psychonomic Bulletin & Review, 20(2)*, 228–242. https://doi.org/10.3758/s13423-012-0359-y

Klingberg, T. (2008). White matter maturation and cognitive development during childhood. In C. A. Nelson & M. Luciana (Eds.), Handbook of developmental cognitive neuroscience (2nd ed), 237–243.

Knudsen, E. I. (2004). Sensitive Periods in the Development of the Brain and Behavior. *Journal of Cognitive Neuroscience, 16(8)*, 1412–1425. https://doi.org/10.1162/0898929042304796

Kofler, M.J., Rapport, M.D., Bolden, J. et al. (2010). ADHD and Working Memory: The Impact of Central Executive Deficits and Exceeding Storage/Rehearsal Capacity on Observed Inattentive Behavior. *J Abnorm Child Psychol 38*, 149–161. https://doi.org/10.1007/s10802-009-9357-6

Kolb, B., Coie, J., & Wishaw, I. (2000). Is there an optimal age for recovery from motor cortex lesions. Brain Res, 882, 62–74.

Kolb, B., Mychasiuk, R., Muhammad, A., Li, Y., Frost, D. O., & Gibb, R. (2012). Experience and the developing prefrontal cortex. *Proceedings of the National Academy of Sciences, 109(Supplement_2)*, 17186–17193. https://doi.org/10.1073/pnas.1121251109

Kolb, B., Pellis, S., & Robinson, T. (2004). Plasticity and functions of the orbital frontal cortex. *Brain Cognition, 55*, 104–15.

Kolb, B., & Gibb, R. (n.d.). Brain Plasticity and Behaviour in the Developing Brain.

Kolb, B., Mychasiuk, R., Muhammad, A., & Gibb, R. (2013). Brain Plasticity in the Developing Brain. *In Progress in Brain Research, 207*, 35–64). https://doi.org/10.1016/B978-0-444-63327-9.00005-9

Korkman, M., Kirk, U., & Kemp, S. (1998). NEPSY: A developmental neuropsychological assessment manual. San Antonio, TX: The Psychological Corporation.

Korkman, M., Kirk, U., & Kemp, S. (2007a). NEPSY-II: A developmental neuropsychological assessment. San Antonio, TX: The Psychological Corporation.

Korkman, M., Kirk, U., & Kemp, S. (2007b). NEPSY-II: Clinical and interpretive manual. San Antonio, TX: The Psychological Corporation.

Korkman, M., Lahti-Nuuttila, P., Laasonen, M., Kemp, S. L. & Holdnack, J. (2013). Neurocognitive development in 5- to 16-year-old North American children: A cross-sectional study. *Child Neuropsychology, 19(5)*, 516-539. DOI: 10.1080/09297049.2012.705822

Krivitzky, L. S., Roebuck-Spencer, T. M., Roth, R. M., Blackstone, K., Johnson, C. P., & Gioia, G. (2011). Functional magnetic resonance imaging of working memory and response inhibition in children with mild traumatic brain injury. *Journal of the International Neuropsychological Society, 17*, 1143–1152.

Kuhl, B. A., & Chun, M. (2014). Memory and Attention (A. C. (Kia) Nobre & S. Kastner, Eds.; Vol. 1). Oxford University Press. https://doi.org/10.1093/oxfordhb/9780199675111.013.034

Kuhl, P. K., Stevenson, J., Corrigan, N. M., van den Bosch, J. J. F., Can, D. D., & Richards, T. (2016). Neuroimaging of the bilingual brain: Structural brain correlates of listening and speaking in a second language. *Brain and Language, 162*, 1–9. https://doi.org/10.1016/j.bandl.2016.07.004

Laher, S., & Cockcroft, K. (Eds.). (2013). Psychological Assessment in South Africa: Research and applications. Johannesburg, South Africa: Wits University Press. doi:10.18772/22013015782

Lajiness-O'Neill, R., Erdodi, L., & Bigler, E. D. (2010). Memory and Learning in Pediatric Traumatic Brain Injury: A Review and Examination of Moderators of Outcome. *Applied Neuropsychology, 17(2)*, 83–92. https://doi.org/10.1080/09084281003708837

Langlois, J. A., & Sattin, R. W. (2005). Traumatic brain injury in the United States: Research and programs of the Centers for Disease Control and Prevention (CDC). *Journal of Head Trauma Rehabilitation, 20(3)*, 187–8.

L'Ecuyer-Giguère, F., Greffou, S., Tabet, S., Frenette, L. C., Tinawi, S., Feyz, M., & de Guise, E. (2019). Visual memory performance following mild traumatic brain injury

and its relationship with intellectual functioning. *Applied Neuropsychology: Adult*, 1–13. https://doi.org/10.1080/23279095.2018.1528263

Lee, B., & Newberg, A. (2005). Neuroimaging in traumatic brain imaging. NeuroRx, 2(2), 372–83. doi:10.1602/neurorx.2.2.372

Li, L., Li, Y., McDonald, C., & Liu, J. (2018). Parent-Reported Mild Head Injury History in Children: Long-Term Effects on Attention-Deficit Hyperactivity Disorder. *Global Pediatric Health*. https://doi.org/10.1177/2333794X18756465

Lobo, V., Patil, A., Phatak, A. & Chandra, N. (2010). Free radicals, antioxidants and functional foods: Impact on human health. *Pharmacogn Rev, 4(8)*, 118–126. doi: 10.4103/0973-7847.70902

Lowe, P. A., Mayfield, J. W. & Reynolds, C. R. (2003). Gender differences in memory test performance among children and adolescents. *Archives of Clinical Neuropsychology, 18(8)*, 865-878. https://doi.org/10.1016/S0887-6177(02)00162-2.

Lozano, D et al. (2015) Neuroinflammatory responses to traumatic brain injury: etiology, clinical consequences, and therapeutic opportunities. *Neuropsychiatry Dis Treat 11*, 97-106

Lumba-Brown, A., Yeates, K. O., Sarmiento, K., Breiding, M. J., Haegerich, T. M., Gioia, G. A., Turner, M., Benzel, E. C., Suskauer, S. J., Giza, C. C., Joseph, M., Broomand, C., Weissman, B., Gordon, W., Wright, D. W., Moser, R. S., McAvoy, K., Ewing-Cobbs, L., Duhaime, A.-C., … Timmons, S. D. (2018). Diagnosis and Management of Mild Traumatic Brain Injury in Children: A Systematic Review. *JAMA Pediatrics, 172(11)*. https://doi.org/10.1001/jamapediatrics.2018.2847

Maillard-Wermelinger, A., Yeates, K. O., H. Taylor, G., Rusin, J., Bangert, B., Dietrich, A., Nuss, K. & Wright, M. (2009). Mild traumatic brain injury and executive functions in school-aged children. *Developmental Neurorehabilitation, 12(5)*, 330-341. DOI: 10.3109/17518420903087251

Mansouri, F. A., Rosa, M. G. P., & Atapour, N. (2015). Working Memory in the Service of Executive Control Functions. *Frontiers in Systems Neuroscience, 9*. https://doi.org/10.3389/fnsys.2015.00166

Masoura, E. V., & Gathercole, S. E. (2005). Contrasting contributions of phonological short-term memory and long-term knowledge to vocabulary learning in a foreign language. *Memory, 13(3-4)*, 422-429.

McGaugh, J.L., (2000). Neuroscience - memory - a century of consolidation. *Science, 80(287)*, 248–251.

McGraw, K. O., & Wong, S. P. (1992). A common language effect size statistic. *Psychological Bulletin, 111(2)*, 361–365. https://doi.org/10.1037/0033-2909.111.2.361

McInnes, K., Friesen, C. L., MacKenzie, D. E., Westwood, D. A., & Boe, S. G. (2017). Mild Traumatic Brain Injury (mTBI) and chronic cognitive impairment: A scoping review. *PLOS ONE, 12(4)*. https://doi.org/10.1371/journal.pone.0174847

McKinlay, A. (2010), Controversies and outcomes associated with mild traumatic brain injury in childhood and adolescences. *Child: Care, Health and Development, 36*, 3-21. https://doi.org/10.1111/j.1365-2214.2009.01006.x

Middleton P. M. (2012). Practical use of the Glasgow Coma Scale; a comprehensive narrative review of GCS methodology. Australasian emergency nursing journal : AENJ, 15(3), 170–183. https://doi.org/10.1016/j.aenj.2012.06.002

Mittl, R. L., Grossman, R. I., Hiehle, J. F., Hurst, R. W.,. Kauder, D. R., Gennarelli, T. A., & Alburger, G. W. (1994). Prevalence of MR evidence of diffuse axonal injury in patients with mild head injury and normal head CT findings. *AJNR American Journal of Neuroradiology, 15(8)*, 1583–9.

Mous, S. E., Schoemaker, N. K., Blanken, L. M. E., Thijssen, S., Ende, J., Polderman, T. J. C., Jaddoe, V. W. V., Hofman, A., Verhulst, F. C., Tiemeier, H. & White, T. (2017). The association of gender, age, and intelligence with neuropsychological functioning in young typically developing children: The Generation R study. *Applied Neuropsychology: Child, 6(1)*, 22-40. DOI: 10.1080/21622965.2015.1067214

Moriguchi, Y. and Hiraki, K. (2013). Prefrontal cortex and executive function in young children: a review of NIRS studies. *Front. Hum. Neurosci. 7(867)*. doi:10.3389/fnhum.2013.00867

Mulenga, K., Ahonen, T. & Aro, M. (2001) Performance of Zambian Children on the NEPSY: A Pilot Study. *Developmental Neuropsychology, 20(1)*, 375-383. DOI: 10.1207/S15326942DN2001_4

Naidoo, D. (2013). Traumatic brain injury: The South African landscape. *South African Medical Journal, 103(9)*, 613-614. doi:10.7196/SAMJ.7325

Nair, K. (2020). *Investigating the effects of Paediatric Mild Traumatic Brain Injury on Speed of Processing of South African Children within a polyglot society, at Chris Hani*

Baragwanath Academic Hospital. [Unpublished master's thesis]. University of the Witwatersrand, Johannesburg, Gauteng, South Africa.

Nichols, E. S., & Joanisse, M. F. (2016). Functional activity and white matter microstructure reveal the independent effects of age of acquisition and proficiency on second-language learning. *NeuroImage, 143*, 15–25. https://doi.org/10.1016/j.neuroimage.2016.08.053

Oberauer, K. (2019). Working Memory and Attention – A Conceptual Analysis and Review. *Journal of Cognition, 2(1)*, 36. https://doi.org/10.5334/joc.58

Ofen, N. (2012). The development of neural correlates for memory formation. *Neuroscience & Biobehavioral Reviews, 36(7)*, 1708–1717. https://doi.org/10.1016/j.neubiorev.2012.02.016

Olson, K., & Jacobson, K. (2015). Cross-Cultural Considerations in Pediatric Neuropsychology: A Review and Call to Attention. Applied neuropsychology. Child, 4(3), 166–177. https://doi.org/10.1080/21622965.2013.830258

Onwuegbuzie, A. J. & Leech, N. L. (2005). On Becoming a Pragmatic Researcher: The Importance of Combining Quantitative and Qualitative Research Methodologies. *International Journal of Social Research Methodology, 8(5)*, 375-387. DOI: 10.1080/13645570500402447

Petrides, M. (2013). Neuroanatomy of Language Regions of the Human Brain 1st Edition. Academic Press: United States.

Pretorius, C., & Broodryk, M. (2013). Misconceptions about traumatic brain injuries among South African university students. *South African Journal of Psychiatry, 19(3)*, 5. doi: https://doi.org/10.4102/sajpsychiatry.v19i3.436

Prince, C., & Bruhns, M. (2017). Evaluation and Treatment of Mild Traumatic Brain Injury: The Role of Neuropsychology. *Brain Sciences, 7(12)*, 105. https://doi.org/10.3390/brainsci7080105

Ramos, A. A., Hamdan, A. C. & Machado, L. (2020). A meta-analysis on verbal working memory in children and adolescents with ADHD. *The Clinical Neuropsychologist, 34(5)*, 873-898. DOI: 10.1080/13854046.2019.1604998

Reilly, J., Bates, E., & Marchman, V. (1998). Narrative discourse in children with early focal brain injury. *Brain Lang, 61*, 335–78.

Rose, S. C., Weber, K. D., Collen, J. B., & Heyer, G. L. (2015). The Diagnosis and Management of Concussion in Children and Adolescents. *Pediatric Neurology, 53(2)*, 108–118. https://doi.org/10.1016/j.pediatrneurol.2015.04.003

Rosselli, M., Loewenstein, D. A., Curiel, R. E., Penate, A., Torres, V. L., Lang, M., Greig, M. T., Barker, W. W., & Duara, R. (2019). Effects of Bilingualism on Verbal and Nonverbal Memory Measures in Mild Cognitive Impairment. *Journal of the International Neuropsychological Society : JINS, 25(1)*, 15–28. https://doi.org/10.1017/S135561771800070X

Rossi, S., Lubin, A., Simon, G., Lanoë, C., Poirel, N., Cachia, A., … Houde, O. (2013). Structural brain correlates of executive engagement in working memory: Children's inter-individual differences are reflected in the anterior insular cortex. *Neuropsychologia, 51*, 1145–1150.

Rutland-Brown, W., Langlois, J. A., Thomas, K. E., & Xi, Y. L. (2006). Incidence of traumatic brain injury in the United States, 2003. *Journal of Head Trauma Rehabilitation, 21(6)*, 544–8.

Schmidt, J., Hayward, K. S., Brown, K. E., Zwicker, J. G., Ponsford, J., van Donkelaar, P., Babul, S., & Boyd, L. A. (2018). Imaging in Pediatric Concussion: A Systematic Review. *Pediatrics, 141(5)*. https://doi.org/10.1542/peds.2017-3406

Schroeder, S. R., & Marian, V. (2017). Cognitive consequences of trilingualism. *International Journal of Bilingualism, 21(6)*, 754–773. https://doi.org/10.1177/1367006916637288

Shetty, T., Nguyen, J. T., Cogsil, T., Tsiouris, A. J., Niogi, S. N., Kim, E. U., Dalal, A., Halvorsen, K., Cummings, K., Zhang, T., Masdeu, J. C., Mukherjee, P., & Marinelli, L. (2018). Clinical Findings in a Multicenter MRI Study of Mild TBI. *Frontiers in Neurology, 9*, 836. https://doi.org/10.3389/fneur.2018.00836

Silver, C. H. (2000). Ecological validity of neuropsychological assessment in childhood traumatic brain injury. *Journal of Head Trauma Rehabilitation, 15*, 973–988.

Sowell, E. R. (2004). Longitudinal Mapping of Cortical Thickness and Brain Growth in Normal Children. *Journal of Neuroscience, 24(38)*, 8223–8231. https://doi.org/10.1523/JNEUROSCI.1798-04.2004

Spencer-Smith, M., & Anderson, V. (2009). Healthy and abnormal development of the prefrontal cortex. *Developmental Neurorehabilitation, 12(5)*, 279–297. https://doi.org/10.3109/17518420903090701

Stein, D., & Hoffman, S. (2003). Concepts of CNS plasticity in the context of brain damage and repair. *J Head Trauma Rehab, 18*, 317–41.

Stiles, J., Nass, R., Levine, S., Moses, P., & Reilly, J. (2009). Perinatal stroke: effects and outcomes. In: Yeates KO, Ris DM, Taylor HG, Pennington B, editors. Pediatric neuropsychology: research, theory and practice. New York: Guilford.

Sturman, D. A., & Moghaddam, B. (2011). The neurobiology of adolescence: Changes in brain architecture, functional dynamics, and behavioral tendencies. *Neuroscience & Biobehavioral Reviews, 35(8)*, 1704–1712. https://doi.org/10.1016/j.neubiorev.2011.04.003

Suresh, K., & Chandrashekara, S. (2012). Sample size estimation and power analysis for clinical research studies. *Journal of human reproductive sciences, 5(1)*, 7–13. https://doi.org/10.4103/0974-1208.97779

Tamnes, C.K., Walhovd, K.B., Grydeland, H., Holland, D., Ostby, Y., Dale, A.M., & Fjell, A.M. (2013). Longitudinal working memory development is related to structural maturation of frontal and parietal cortices. *Journal of Cognitive Neuroscience, 25*, 1611–1623.

Teasdale, G & Jennet, B. (1974). Assessment of coma and impaired consciousness. *The Lancet, 304(7872)*, 81-84. DOI: https://doi.org/10.1016/S0140-6736(74)91639-0

Torres, A. R., Shaikh, Z. I., Chavez, W., & Maldonado, J. E. (2019). Brain MRI in Children with Mild Traumatic Brain Injury and Persistent Symptoms in Both Sports- and Non-sports-related Concussion. *Cureus*. https://doi.org/10.7759/cureus.3937

Treble, A., Hasan, K. M., Iftikhar, A., Stuebing, K. K., Kramer, L. A., Cox, C. S., Jr, Swank, P. R., & Ewing-Cobbs, L. (2013). Working memory and corpus callosum microstructural integrity after pediatric traumatic brain injury: a diffusion tensor tractography study. *Journal of neurotrauma, 30(19)*, 1609–1619. https://doi.org/10.1089/neu.2013.2934

Tremblay, S., Desjardins, M., Bermudez, P., Iturria-Medina, Y., Evans, A. C., Jolicoeur, P., & De Beaumont, L. (2019). Mild traumatic brain injury: The effect of age at trauma

onset on brain structure integrity. *NeuroImage. Clinical, 23*, 101907. https://doi.org/10.1016/j.nicl.2019.101907

Tsujimoto, S., Yamamoto, T., Kawaguchi, H., Koizumi, H., & Sawaguchi, T. (2004). Prefrontal Cortical Activation Associated with Working Memory in Adults and Preschool Children: An Event-related Optical Topography Study. *Cerebral Cortex, 14(7)*, 703–712. https://doi.org/10.1093/cercor/bhh030

Videsott G, Della Rosa PA, Wiater W, Franceschini R, and Abutalebi J. How does linguistic competence enhance cognitive functions in children? A study in multilingual children with different linguistic competences. *Bilingualism: Language and Cognition, 15(4)*, 2012.

Wechsler, D. (2003). Wechsler Intelligence Scale for Children, 4th Edn. San Antonia, TX: Psych Corp.

Westfall, D. R., West, J. D., Bailey, J. N., Arnold, T. W., Kersey, P. A., Saykin, A. J., & McDonald, B. C. (2015). Increased brain activation during working memory processing after pediatric mild traumatic brain injury (mTBI). *Journal of Pediatric Rehabilitation Medicine, 8(4)*, 297–308. https://doi.org/10.3233/PRM-150348

Westmacott, R., MacGregor, D., Askalan, R., & de Veber, G (2009). Late emergence of cognitive deficits after unilateral neonatal stroke. *Stroke, 40*, 2012–2019.

Wu, X., Kirov, I. I., Gonen, O., Ge, Y., Grossman, R. I., & Lui, Y. W. (2016). MR Imaging Applications in Mild Traumatic Brain Injury: An Imaging Update. *Radiology, 279(3)*, 693–707. https://doi.org/10.1148/radiol.16142535

Xiong, Y., Mahmood, A., & Chopp, M. (2013). Animal models of traumatic brain injury. *Nature Reviews Neuroscience, 14(2)*, 128–142. https://doi.org/10.1038/nrn3407

Yang, L.Y., Huang, C.C., Chiu, W.T. et al. (2016). Association of traumatic brain injury in childhood and attention-deficit/hyperactivity disorder: a population-based study. *Pediatr Res 80*, 356–362. https://doi.org/10.1038/pr.2016.85

Yeates, K. O., Blumenstein, E., Patterson, C. M., & Delis, D. C. (1995). Verbal learning and memory following pediatric closed head injury. *Journal of the International Neuropsychological Society, 1*, 78–87.

Zola, S. M., & Squire, L. R. (2000). The medial temporal lobe and the hippocampus. In E. Tulving & F. I. M. Craik (Eds.), The Oxford handbook of memory (pp. 485–500). New York: Oxford University Press.

APPENDIX A: DEMOGRAPHIC RECORD



Demographic Record **Reference Number**_____

Section A

Background information

Date: …….

Information provided by: …….

Completed by: …….

Participant demographics

Case number	
Date of birth	
Sex	
Home language	
Other languages spoken	Proficiency: good (3)/ average (2)/ poor (1)

Participant's history

Age at injury	
Age at assessment	
Elapsed time (assessmentT – injuryT)	
Mechanism of injury	
Glasgow Coma Scale score (on arrival)	
Number of days hospitalised	
Notes	

Caregiver's rating on participant:	1	2	3	4	5
Concentration					
Forgetfulness					
Distractibility					

1 = poor; 2 = below average; 3 = average; 4 = above average; 5 = great

Caregiver's information

Relationship to participant	
Ethnicity	
Language of choice	
Highest educational qualification	
Employment status	

APPENDIX B: THINK STUDY INFORMATION SHEET

Psychology

School of Human & Community Development

University of the Witwatersrand

Private Bag 3, Wits, 2050

Tel: 011 717 4503 Fax: 011 717 4559

Principal Investigator: Dr Vered Lack

Name of Organization: University of the Witwatersrand

Information for caregiver

Introduction

We are researching mild traumatic brain injury, which is very common in this country. I am going to give you information about this research and invite you to be part of this research. Before you decide, you can talk to anyone you feel comfortable with about the research.

There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take my time to explain to you in whatever language you speak, if that makes you feel more comfortable. If you have questions later, you can ask them of me, the doctor, or the Head Injury Clinic staff.

What is the purpose of the research?

Mild brain injury is becoming one of the most common injuries in this region. The aftereffects of these types of injury are unknown and research wants to find out whether patients of mild traumatic brain injury have aftereffects from the injury or not. We also want to see how it affects cognitive performance in children who have sustained a mild traumatic brain injury.

Who are the participants invited to this study?

We are inviting all children who reported to Baragwanath Pediatric Surgery with a mild head injury and non-head injury children themselves to participate in the research on mild traumatic brain injury and the effects it has on cognitive performance. All parents must give consent for this participation.

Are you forced to participate in this study?

You and your child's participation in this research are entirely voluntary. It is your choice whether to participate or not. Whether you choose to participate or not, all the services you receive at this clinic will continue and nothing will change. You may change your mind later and stop participating even if you agreed earlier.

What will be done in this study?

We will start by informing you about the study, and thereafter you will be given an informed consent form to sign. The consent form will be for you, the parent/caregiver to consent to participate in the research and also to consent for your child to participate in this research. An assent form will be given to your child as well. Once both forms are signed, we will then proceed with the administration of specific tasks.

The tasks that will be administered to your child are: (1) List Memory, (2) Memory for Names, (3) Auditory Attention Response Task, (4) Inhibitory, (5) as well as the Coding tasks while simultaneously you will be filling out the demographic questionnaire in the next room. The test will be administered by a multilingual research colleague who is involved in the same umbrella project; however, the researcher will facilitate the process. Multilingual clinic staff will also be present in both rooms to help in cases of illiteracy or simply misunderstanding the task. The test administrator will give instructions from the Developmental Neuropsychological Assessment, and the Wechsler Intelligence Scale for Children. At completion, tasks and questionnaires from both you and your child will be collected and stored in a sealed box which will later be kept in a secure box which is locked in a doctor's boardroom, which will not leave the hospital.

How long will this study take?

The research takes place over 60 months in total. During that time, you will be asked to come to the Head Injury Clinic for only 1 scheduled Friday or Saturday, for approximately 2 hours.

Will I be reimbursed?

We will give you the amount of money to pay for your travel to the Head Injury Clinic. You will not be given any other money or gifts to take part in this study. This applies specifically to patients who have sustained a mild head injury.

Will the research protect confidentiality and anonymity?

Participants will be made aware that all data will be kept confidential, in a secure location. Anonymity can be guaranteed because the datasheet will only be identified by a participant number in cases where follow up is necessary. This is to ensure the patient's data can be placed in the patient's file.

Who will have access to the data?

Summary reports will be made available to the hospital and parents are welcome to consult the Principal Investigator with regards to the results on the participant's tasks. Any problematic case will be highlighted for the attention of the Principal Investigator.

Who to Contact?

Who can I contact regarding this research?

Enid Schutte: (011)-717-4521 or enid.schutte@wits.ac.za

Dr Lack: veredlack@yahoo.com

For more information should any issues arise, please contact the Wits Ethics committee on 011 717 1888.

Please contact us with further questions regarding this research

Information for child

Introduction

We are doing research work on head injury, which is very common in this country. I am going to tell you about this and ask you to be part of this research. Before you decide, you can talk to anyone you want to about the research.

There may be some words you do not understand. Please ask me to stop as we go through the information and I will take my time to explain to you in the language of your choice if you want me to. If you have questions later, you can ask them of me, the doctor, or the people that work at the clinic

What is the purpose of the research?

Head injury is becoming one of the most common injuries in this region. We do not know much about the effects of it and this research wants to find out whether children with head injuries have effects after the injury or not. We also want to see if it affects your cognitive performance.

Who are the participants invited to this study?

We are asking all those children who had head injuries and were admitted into the Baragawanth Hospital to take part in this research on head injury and the effects it has on cognitive performance.

Are you forced to participate in this study?

You must want to take part in this research. It is your choice. Whether you choose to take part or not, all the services you get at this clinic will still be given to you and nothing will change. You may change your mind later and stop taking part even if you wanted to earlier.

What will be done in this study?

We will start by telling you about the study. We will give you an assent form to sign. Once the form is signed, we will then give you activities to do.

The activities will be explained to you in your home language and you will be told what to do in each activity. There will be people to help if you do not understand. When you are done with the activity, it will be collected by me and closed up in a box and later on put in a locked cupboard.

How long will this study take?

You will be asked to come to the Head Injury Clinic for only 2 hours on 1 Friday or Saturday.

What will I get in return?

We will give you back the money that you used to come to the Head Injury Clinic. You will not be given any other money or gifts to take part in this study.

Who will know what I wrote and who I am?

No-one will know who you are, only me and the doctor, so no-one will know what you wrote in the activity because when you are done with it, I will close it up in a box and put it in a locked cupboard.

PARENTAL INFORMED CONSENT FORM

I _____ have read the foregoing information on the research project, "Investigating the relationship between mild traumatic brain injury and memory performance in a multilingual paediatric cohort", or it was read to me. I have asked all questions about the research and have been answered satisfactorily. I voluntarily give my consent to participate in this research.

I _____ have been invited to have my child participate in the research project "Investigating the relationship between mild traumatic brain injury and memory performance in a multilingual paediatric cohort". I voluntarily give my consent to the participation of my child in this research.

Participant's signature: _____

Date: _____

Researcher name: _____

Researcher /person signature: _____

Date: _____

This is an Informed Consent form for parents of children who were admitted into the Paediatric Surgery Unit and the child. We would like to take this opportunity to invite you to participate in the research on mild traumatic brain injury.

ASSENT FORM FOR CHILD

I understand the research is about Mild Traumatic Brain Injury and a test on cognitive performance will be given to me to complete under the instruction of a co-researcher.

I have had the information regarding the research read to me. I have asked all questions about the research and have been answered satisfactorily and any other questions can be asked later. I assent to participate in this research.

Child's name: _____

Child's signature: _____

Date: _____

Researcher's name: _____

Researcher's signature: _____

Date: _____

Has the participant received a copy of from? Yes / No

Has the Caregiver/Parent signed the informed consent form? Yes / No

Researcher's initial: _____

APPENDIX C: Umbrella project's ethics clearance

GAUTENG PROVINCE
HEALTH
REPUBLIC OF SOUTH AFRICA

CHRIS HANI BARAGWANATH ACADEMIC HOSPITAL
Directorate: Paediatric Surgery
Enquiries: Dr V. Lack
Tel. number: (011)933 8138; Email: veredlack@yahoo.com

Attention: Human Research Ethics Committee (Medical)

Re: Clearance Certificate no M1611113

To whom it may concern

Update on protocol: "Examining Neurocognitive, psychosocial and psychiatric sequelae following mild TBI: a potentially significant impact in a low socio-economic population"

Project start date: 25 March 2017
End Date: 5 year prospective study; ongoing (goal is 300 test subjects and 300 controls)

Progress (March 2017- end November 2017):

- 68 head injured patients tested
- 41 "control" non-head injured patients tested
- Stats not yet run on this group's results however there is an overrepresentation of left handed children in the head-injured group, suggesting the possibility of dominance switching (this may be more evident in moderate and severely head injured children and needs to be be explored).

Challenges:

- HADS: difficult to administer to children under 8 years of age and those with poor command of English. The test concepts do not translate well into African languages due to the grading system used (terms such as sometimes, occasionally etc result in ambiguity).
- "Clocks" component of NEPSY II testing "Attention and executive functioning" has yielded erratic results, possibly dependent on school syllabus rather than inherent abilities of the children.

Amendments:

- We would like to include in our test batches all children who experienced head injuries presenting to CHBAH.
- Add the WISC V as a measure of processing speed and working memory. Requires few instructions, eastly translated and is diagram based. This test is suitable for children aged from 6 years old.
- HADS only to be used in children aged 8 years or above comfortable to communicate in English.
- Remove "clocks" component of the NEPSY II
- Add "hand dominance" as a question on the Data Collection sheet.

Thank you for your attention and consideration.
Kind regards,

Dr Vered Lack

R14/49 Dr Vered Lack et al

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M1611113

NAME: (Principal Investigator)	Dr Vered Lack et al
DEPARTMENT:	General Surgery Chris Hani Baragwanath Academic Hospital Department of Paediatric Surgery
PROJECT TITLE:	Examining Neurocognitive, Psychosocial and Psychiatric Sequelae Following Mild TBI (Traumatic Brain Injury): A Potentially Significant Impact in a Low Socio-Economic Population
DATE CONSIDERED:	25/11/2016
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	
APPROVED BY:	Professor P Cleaton-Jones, Chairperson, HREC (Medical)
DATE OF APPROVAL:	20/02/2017

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary in Room 301, Third Floor, Faculty of Health Sciences, Phillip Tobias Building, 29 Princess of Wales Terrace, Parktown, 2193. University of the Witwatersrand. I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to resubmit the application to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. In this case, the study was initially reviewed in November and will therefore be due in the month of November each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature Date

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

APPENDIX D: Research permission letter

Application for Research Site Permission Letter 18 March 2020

Chris Hani Baragwanath Academic Hospital: Department of Paediatric Surgery

R68 Old Potchefstroom Road

PO Bertsham

Johannesburg

2013

South Africa

RE:	Head of Department:	Prof Jerome Loveland
	Principal Investigator:	Dr Vered Lack
	(Larger Study):	Examining Neurocognitive, Psychosocial and Psychiatric Sequelae Following Mild TBI (Traumatic Brain Injury): A Potentially Significant Impact in a Low Socio-Economic Population
	Student Investigator:	Heinrich Reinhard Zentgraf
	Title of Research Study	
	(Sub-study):	Investigating the relationship between mild traumatic brain injury and memory performance in a multilingual paediatric cohort

To: Dr Vered Lack/ Prof Jerome Loveland

My name is Heinrich Zentgraf, a student currently enrolled in the Master of Arts by Coursework and Research Report in the field of Psychology programme at the University of the Witwatersrand. Enid Schutte, my supervisor, has informed me of your investigation, titled: '*Examining Neurocognitive, Psychosocial and Psychiatric Sequelae Following Mild TBI (Traumatic Brain Injury): A Potentially Significant Impact in a Low Socio-Economic Population' (*Protocol Number: M1611113) which I find intriguing. I am writing this letter to convey my interest in joining your research team, as I would like to pursue a similar line of inquiry, specifically, one which investigates memory performance related sequelae following mild traumatic brain injury.

Accordingly, I hereby formally request permission to conduct my sub-study, Project Title: '*Investigating the relationship between mild traumatic brain injury and memory performance in a multilingual paediatric cohort*' within the parameters of your aforementioned study, and wish to enquire whether your research team could accommodate me for approximately 1 year, or the duration of my MA studies.

Should the above be met in affirmation, I further request access to pro/retrospective records containing the following patient/ clinical data:

1.1) Date of Admission

1.2) Gender

1.3) Age

1.4) Mechanism of Injury

1.5) Initial GCS.

Access a pro/retrospective database containing the following data:

2) A Developmental Neuropsychological Assessment: Second Edition (NEPSY-II)

2.1) List Memory Subtests and List Memory Delayed Subtests and

2.1.1) List Memory Total Correct Scores (Trials 1-7)

2.1.2) List Memory Repetitions Total Scores

2.1.3) List Memory Non-List Words (Novel) Total Scores

2.1.4) List Memory Wrong List Words (Interference) Total Scores

2.1.5) List Memory Learning Effect Scores

2.1.6) List Memory Interference Effects Scores

2.1.7) List Memory and List Memory Delayed Total Correct Scores

2.1.8) List Memory Delay Effect Scores

2.2) Memory for Names Subtest and Memory for Names Delayed Subtest and

2.2.1) Memory for Names Total Scores

2.2.2) Memory for Names Delayed Total Scores

2.2.3) Memory for Names and Memory for Names Delayed Total Scores

2.3) Auditory Attention and Response Set Subtests and

2.3.1) Auditory Attention Total Correct Scores

2.3.2) Auditory Attention Commission Error Scores

2.3.3) Auditory Attention Total Omission Error Scores

2.3.4) Auditory Attention Total Inhibitory Error Scores

2.3.5) Response Set Total Correct Scores

2.3.6) Response Set Commission Error Scores

2.3.7) Response Set Total Omission Scores

2.3.8) Response Set Total Inhibitory Error Scores

2.4) Inhibition Subtests and

2.4.1) Uncorrected Error Scores

2.4.2) Self-Corrected Error Scores

2.4.3) Total Errors Scores

2.4.4) INN Total Errors Scores

2.4.5) INI Total Errors Scores

2.4.6) INS Total Error Scores

2.4.7) IN Total Errors Scores

2.4.8) INN Total Completion Time Scores

2.4.9) INI Total Completion Time Scores

2.4.10) INS Total Completion Time Scores

2.4.11) INN Total Uncorrected Errors Scores

2.4.12) INI Total Uncorrected Errors Scores

2.4.13) INS Total Uncorrected Errors Scores

2.4.14) INN Total Self-Corrected Errors Scores

2.4.15) INI Total Self-Corrected Errors Scores

2.4.16) INS Total Self-Corrected Errors Scores

3) Wechsler Intelligence Scale for Children IV (WISC-IV)

3.1) Coding Test

3.1.1) Coding Total Correct Scores

3.1.2) Coding Commission Error Scores

3.1.3) Coding Total Omission Error Scores

3.1.4) Coding Total Inhibitory Error Scores

3.1.5) Coding Correct Scores

3.1.6) Coding Error Scores

3.1.7) Coding Total Omission Scores

3.1.8) Coding Total Inhibitory Error Scores]

Utilise the documentation provided by the larger study, namely:

6.1) Participant Information Sheet

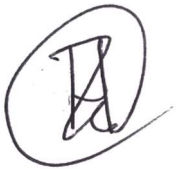
6.2) Consent Form

6.3) Assent Form

7) Invite participants to take part in the study, inform them about the study, their participation, their rights and how their information will be used.

Yours sincerely

H. R. Zentgraf



_____ _____

Principal Investigator Signature Date
Dr Vered Lack

_____ _____

Head of Department Signature Date
Prof Jerome Loveland

_____ _____

Supervisor Signature Date
Ms Enid Schutte

APPENDIX E: Confidentiality agreement

GAUTENG PROVINCE
HEALTH
REPUBLIC OF SOUTH AFRICA

CHRIS HANI BARAGWANATH ACADEMIC HOSPITAL
Directorate: Paediatric Surgery
HOD: Prof J Loveland
Enquiries: Dr V. Lack
Tel. number: (011)933 8138; Email: veredlack@yahoo.com

CONFIDENTIALITY AGREEMENT

THINK Study: Traumatic Head Injury-Neurocognitive and Behavioural Assessment and Management in Kids

Human Research Ethics Committee (Medical)
- Clearance Certificate No M1611113

DOH Trial No: DOH-27-0617-5762

THINK Study is founded on many years of work and data collection by the Principal Investigator and HOD of the Department of Paediatric Surgery. This work forms the backbone of the clinical trial (and is the intellectual property of the department).

Although THINK aims to answer important and clinically relevant questions, confidentiality and patient autonomy are of paramount importance to the Department. To this end, clinical information used in the study is highly protected, as per ethics requirements of the study.

As Principal Investigator of the above-mentioned, and holder of the Ethics Permissions it is incumbent on myself to enforce confidentiality prerequisites as stipulated by Ethics. To this end, I can only share clinical information relevant to your component of the study. This data is shared in confidence, and may only be shared with the student stipulated below and direct co-supervisor (stipulated below). Also, as Principal Investigator, and to ensure clinical information in accurately represented, myself and my supervisor are required to read all manuscripts that result from analysis of this data, and as PI, I am to be supervising author on all resulting publications.
Please sign below, to confirm acceptance of the above stipulations.

Student	**DATE** 2020/03/18	Research supervisor	**DATE**
Dr Vered Lack	**DATE**	Prof Jerome Loveland	**DATE**

APPENDIX F: Code of conduct

CODE OF CONDUCT
FOR RESEARCHERS / APPLICANTS FOR ETHICS APPROVAL AND / OR RESEARCH ETHICS MEMBERS

This Code of Conduct is applicable to all University of the Witwatersrand, Johannesburg (University) researchers/ applicants which includes staff and students of the University and third parties applying for ethics approval and research ethics members.

This Code of Conduct must be read in conjunction with the University's Code of Conduct document HRG/26 approved by Council, C2006/482.

As a member of the University Community, I agree and commit to the rules and regulations of the University as well as all national and international laws and regulations applicable to my field of study. Furthermore, I agree and commit myself to abide by the ethical principles and responsibilities as set out in the Singapore Statement on Research Integrity dated 22 September 2010, in any and all research endeavours that I undertake as a researcher or applicant of the University or research ethics member.

I acknowledge and agree that I have read the Terms of Reference and the Standard Operating Procedures for the relevant research ethics committee that I am applying for ethics approval and agree to follow them.

The four major values of research integrity to which I will agree to adhere to and that will guide my research are:

·Honesty and integrity in all facets of my research or decisions that I make as a researcher and/or ethics committee member;

·Accountability in the conduct of my research or decisions that I make as a researcher and/or research ethics member;

·Professional courtesy and fairness in working with other people; and

·Good stewardship of research on behalf of other people.

Therefore I will further agree to adhere to the following ethical responsibilities:

1. I will take responsibility for the originality and trustworthiness of my research.
2. I will stay abreast of and adhere to all institutional, national, and international laws, regulations, rules and policies applicable and related to my research.
3. I will at all times employ appropriate research methods, base my conclusions on critical analysis of the evidence and report my findings and interpretations fully and objectively.
4. I will keep clear and accurate records of all research that I have conducted in a manner that will allow verification and replication of my work by others, if applicable.
5. I will, where applicable, share my data and findings openly and promptly, in line with external funding rules. This will be done as soon as possible after I have had an opportunity to establish priority and ownership claims.
6. I will take responsibility for my own contributions to publications, funding applications, reports and other representations of my research. I will also and only include authors who meet valid authorship criteria which are set out in the University's Authorship Policy.
7. I will acknowledge the names and roles of those who made significant contributions to my research in publications, including writers, funders, sponsors, and others, but do not meet authorship criteria.
8. In my peer reviews, I will provide fair, prompt and rigorous evaluations and I will respect confidentiality when I review others' work.
9. I will disclose all conflicts of interest (financial and other) that could compromise the trustworthiness of my work in research proposals, publications, public communications, and in review activities.
10. When I publically address a community in the spirit of academic freedom, I will in all stages base my professional comments on research findings, if applicable, and my expertise. I will distinguish between professional comments and opinions based on personal views.

11. Should any irresponsible research practices and/or research misconduct become known to me or brought under my attention, I will report such irresponsible research activities to the appropriate University representatives, Research Ethics Committee and in line with the University's Research Integrity Policy.
12. I will respond to any irresponsible research practices or conduct, by taking prompt actions as set out in the procedures of the University. I will also protect those who report misconduct in good faith, to the best of my abilities.
13. I will endeavour to create and sustain an environment that encourage research integrity through education of students, research teams and peers, as well as abide by policies, and reasonable standards for advancement.
14. I will at all times weigh societal benefits against the risks inherent in my research.

The absence of a specific guideline in this Code of Conduct does not relieve and individual of the responsibility of applying the highest ethical standards when reacting to a situation. Ethical, respectful, responsible and diligent conduct of all members of the University community is essential for maintaining and enhancing the position of the University of the Witwatersrand, Johannesburg as a leading university in the Republic of South Africa, Africa and the world.

Researcher / Applicant / Research Ethics Member Name: Heinrich R Zentgraf

Signature: 

Date: 06.05.2020

Place: University of Witwatersrand

APPENDIX G: Independent t-test: group differences & Levene's Test

Table 7

Independent t-tests: group differences & Levene's Test

Dataset	Subtest variable	Levene's Test for Equality of Variances	t	df	Sig. (2-tailed)	Effect size (*d*)[1]
Original	*AA-Combined*	Equal variances assumed	2.083	42	.043*	.65
	RS-Combined	Equal variances assumed	.611	36	.545	.2
	AARS-Contrast	Equal variances assumed	.060	36	.953	.02
	LM-C	Equal variances assumed	.882	42	.383	.27
	LM Memory span	Equal variances assumed	1.602	44	.116	.48
	LM Learning effect	Equal variances assumed	-1.890	44	.065	.57
	LM Interference	Equal variances assumed	.811	44	.422	.24
	LM Delay effect	Equal variances assumed	1.548	41	.129	.48
	MN	Equal variances not assumed	.052	36.249	.959	.02
	MND	Equal variances assumed	-.186	47	.854	.05
	MN-C	Equal variances assumed	-.099	47	.922	.04
	CO-I	Equal variances assumed	.753	43	.456	.23
	CO-D	Equal variances assumed	.105	43	.917	.03
Imputed	*AA-Combined*	Equal variances assumed	1.478	48	.146	.43
	RS-Combined	Equal variances assumed	1.045	48	.301	.3
	AARS-Contrast	Equal variances assumed	.518	48	.607	.15
	LM-C	Equal variances assumed	1.297	48	.201	.37
	LM Memory span	Equal variances assumed	1.418	48	.163	.41
	LM Learning effect	Equal variances assumed	-1.325	48	.191	.38
	LM Interference	Equal variances assumed	.341	48	.735	.1
	LM Delay effect	Equal variances not assumed	1.273	45.143	.209	.37
	MN	Equal variances not assumed	.052	36.249	.959	.02
	MND	Equal variances assumed	-.322	48	.749	.09
	MN-C	Equal variances not assumed	.113	40.807	.911	.03
	CO-I	Equal variances assumed	-.011	48	.991	.003
	CO-D	Equal variances assumed	.42	48	.677	.12

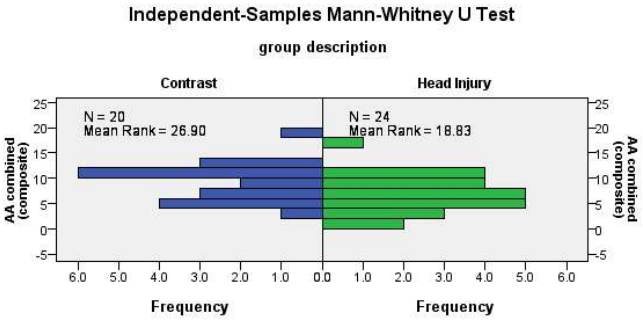
* Demarcates statistically significant value ($p < .05$)

[1] Denotes Cohen's *d*

APPENDIX H: Mann-Whitney U test: distribution shape inspection

Figure 4

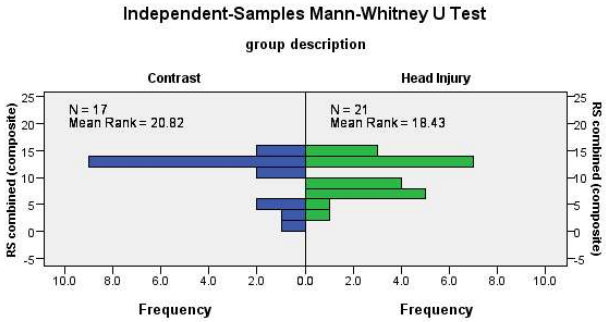
Mann-Whitney U test distribution shape inspection: AA-Combined.



Total N	44
Mann-Whitney U	152.000
Wilcoxon W	452.000
Test Statistic	152.000
Standard Error	42.239
Standardized Test Statistic	-2.083
Asymptotic Sig. (2-sided test)	.037

Figure 5

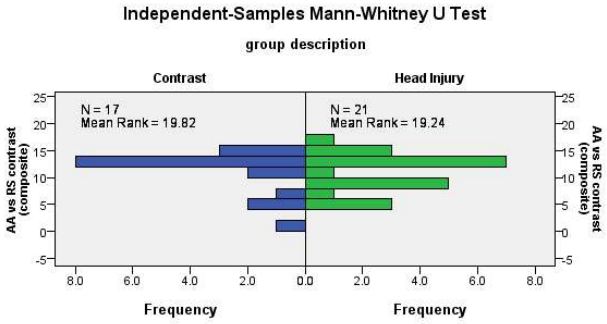
Mann-Whitney U test distribution shape inspection: RS-Combined.



Total N	38
Mann-Whitney U	156.000
Wilcoxon W	387.000
Test Statistic	156.000
Standard Error	33.633
Standardized Test Statistic	-.669
Asymptotic Sig. (2-sided test)	.504
Exact Sig. (2-sided test)	.523

Figure 6

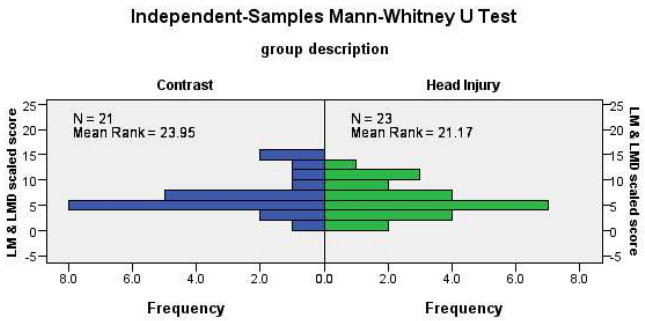
Mann-Whitney U test distribution shape inspection: AARS-Contrast.



Total N	38
Mann-Whitney U	173.000
Wilcoxon W	404.000
Test Statistic	173.000
Standard Error	33.678
Standardized Test Statistic	-.163
Asymptotic Sig. (2-sided test)	.870
Exact Sig. (2-sided test)	.885

Figure 7

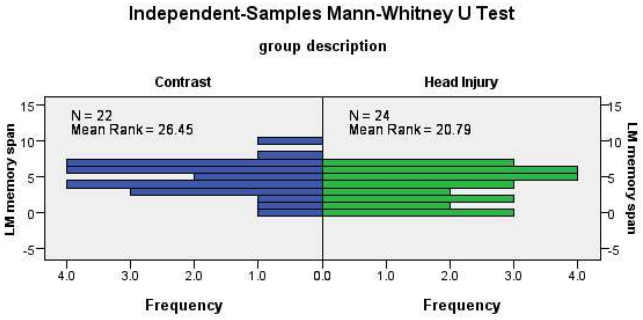
Mann-Whitney U test distribution shape inspection: LM-Composite.



Total N	44
Mann-Whitney U	211.000
Wilcoxon W	487.000
Test Statistic	211.000
Standard Error	42.190
Standardized Test Statistic	-.723
Asymptotic Sig. (2-sided test)	.470

Figure 8

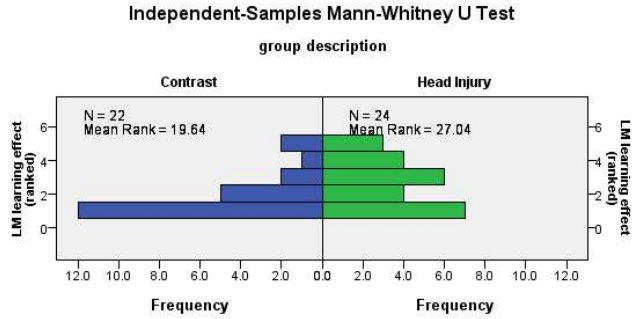
Mann-Whitney U test distribution shape inspection: LM-Memory span.



Total N	46
Mann-Whitney U	199.000
Wilcoxon W	499.000
Test Statistic	199.000
Standard Error	45.088
Standardized Test Statistic	-1.442
Asymptotic Sig. (2-sided test)	.149

Figure 9

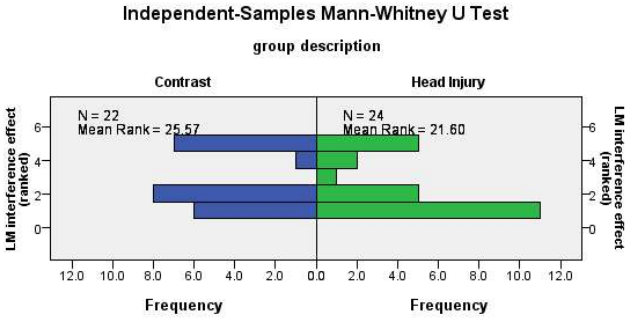
Mann-Whitney U test distribution shape inspection: LM-Learning effect.



Total N	46
Mann-Whitney U	349.000
Wilcoxon W	649.000
Test Statistic	349.000
Standard Error	43.491
Standardized Test Statistic	1.954
Asymptotic Sig. (2-sided test)	.051

Figure 10

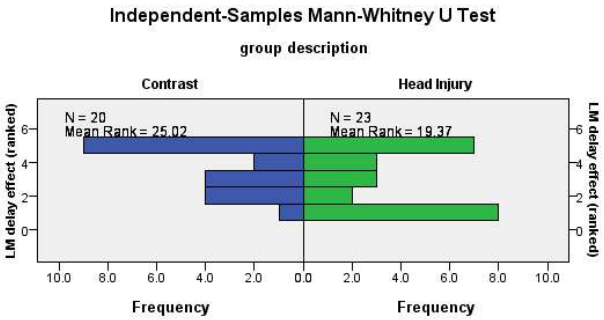
Mann-Whitney U test distribution shape inspection: LM-Interference effect.



Total N	46
Mann-Whitney U	218.500
Wilcoxon W	518.500
Test Statistic	218.500
Standard Error	43.365
Standardized Test Statistic	-1.049
Asymptotic Sig. (2-sided test)	.294

Figure 11

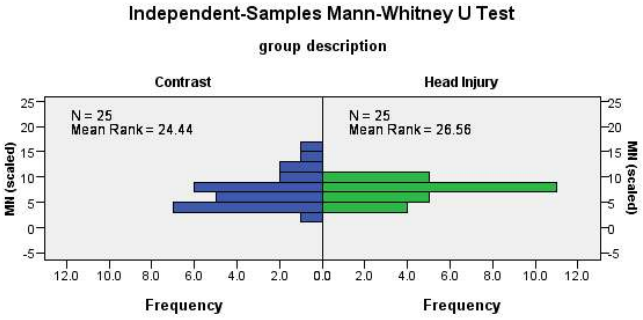
Mann-Whitney U test distribution shape inspection: LM-Delay effect.



Total N	43
Mann-Whitney U	169.500
Wilcoxon W	445.500
Test Statistic	169.500
Standard Error	39.631
Standardized Test Statistic	-1.527
Asymptotic Sig. (2-sided test)	.127

Figure 12

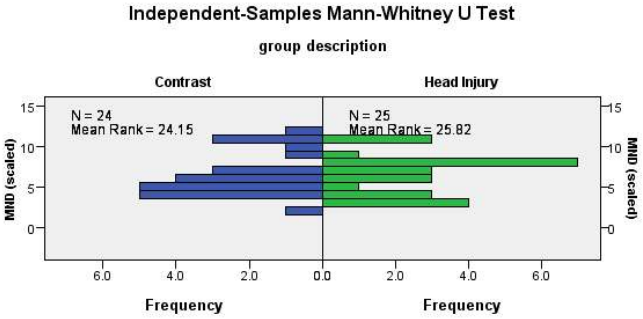
Mann-Whitney U test distribution shape inspection: MN.



Total N	50
Mann-Whitney U	339.000
Wilcoxon W	664.000
Test Statistic	339.000
Standard Error	51.051
Standardized Test Statistic	.519
Asymptotic Sig. (2-sided test)	.604

Figure 13

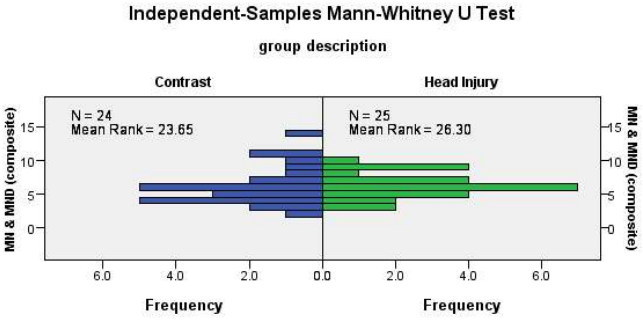
Mann-Whitney U test distribution shape inspection: MND.



Total N	49
Mann-Whitney U	320.500
Wilcoxon W	645.500
Test Statistic	320.500
Standard Error	49.600
Standardized Test Statistic	.413
Asymptotic Sig. (2-sided test)	.679

Figure 14

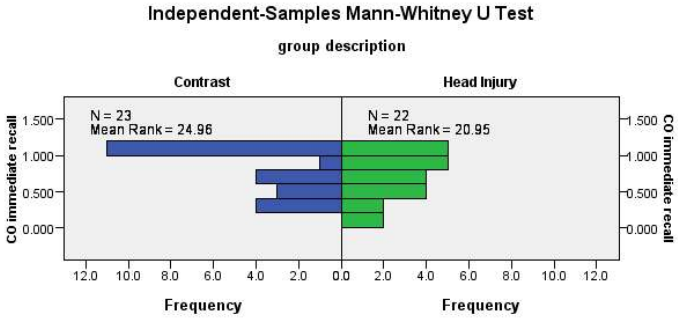
Mann-Whitney U test distribution shape inspection: MN-Composite.



Total N	49
Mann-Whitney U	332.500
Wilcoxon W	657.500
Test Statistic	332.500
Standard Error	49.402
Standardized Test Statistic	.658
Asymptotic Sig. (2-sided test)	.511

Figure 15

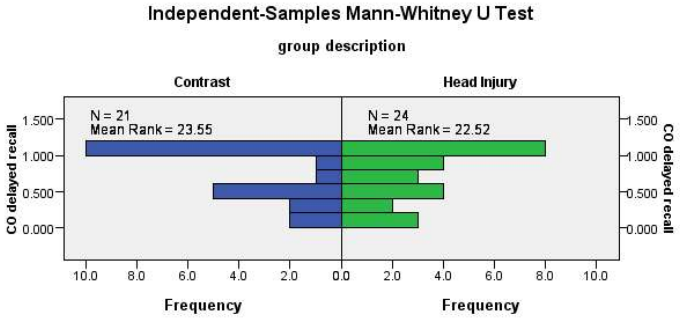
Mann-Whitney U test distribution shape inspection: CO-I.



Total N	45
Mann-Whitney U	208.000
Wilcoxon W	461.000
Test Statistic	208.000
Standard Error	42.906
Standardized Test Statistic	-1.049
Asymptotic Sig. (2-sided test)	.294

Figure 16

Mann-Whitney U test distribution shape inspection: CO-D.



Total N	45
Mann-Whitney U	240.500
Wilcoxon W	540.500
Test Statistic	240.500
Standard Error	42.667
Standardized Test Statistic	-.270
Asymptotic Sig. (2-sided test)	.788