Pediatric Traumatic Brain Injury – Neurocognitive and Psychosocial Outcome 6 Months Post-Injury

A Follow-Up Study in a Norwegian Pediatric Sample

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Abstract

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Title: Pediatric Traumatic Brain Injury – Neurocognitive and Psychosocial Outcome 6 months post-injury: A follow-up study in a Norwegian pediatric sample

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Background:

The most frequent cause of interruption to a child's normal course of brain development, is acquired brain injury. Norwegian data regarding long-term outcomes and rehabilitation needs following pediatric traumatic brain injury (pTBI) is limited and therefore much needed in order to design interventions and provide appropriate rehabilitation for this patient population. This study is one of the few Norwegian explorations of neurocognitive and psychosocial outcomes after pTBI and aims to contribute to a better understanding of this population. **Method:** Recruitment took place January 2015 throughout 2016, and participants were recruited from the South-Eastern Health Region of Norway. Fifty children aged 12 months to 15 years presenting to Oslo University Hospital (OUH) with verified traumatic brain injury (TBI) and CT scan within 24 hours were included. Acute medical and demographic data was collected shortly after inclusion. Assessment of neurocognitive and psychosocial symptom burden was performed 6 months post-injury through neuropsychological assessment and questionnaires (parental and self-report). **Results:** Transportation was the main cause of injury, with falls being most common for the youngest children, while the proportion of sports related injuries increased with age. The sample was skewed towards the severe end of the spectrum, with 64% of the injuries classified as mild, and 22% and 14% moderate and severe respectively, when classified based on Glasgow Coma Scale (GCS). A substantial amount of the mild injuries had intracranial abnormalities on CT/MRI, resulting in a subdivision of the mild group into uncomplicated (n=21) and complicated (n=11) mild TBI. Regarding the neuropsychological outcome, The WISC-IV Working memory index was significantly lower than the standardization sample mean values, and this domain specific vulnerability was further supported by the performance on this index being significantly lower than both the verbal comprehension and perceptual reasoning

indexes on paired samples t-tests. Although the remaining neuropsychological test results appeared to be largely within average levels, 44% of the sample had clinically significant WISC-IV index discrepancies, which covaried with head injury severity. Also, when examining a neuropsychological impairment variable, a 40% impairment rate appeared, with the impairment rate being highest in the complicated mild group. Of note was the significant increase in parent reported post concussive symptoms (PCS) post-injury. Aspects of injury severity, family functioning, and child pre-injury symptom levels were all correlated with PCS, everyday life measures of executive function, as well as aspects of behavioral and emotional functioning after 6 months, supporting the link between premorbid vulnerability and post-injury function. The sample reported few symptoms within the psychosocial domain. **Conclusion:** This study demonstrates how stratifying pTBI severity based exclusively on the GCS can result in a too wide spectrum of injuries falling within the mild classification and underscores the importance of integrating radiological findings when identifying children at risk for persisting symptoms after pTBI. If intra-individual performance variation is not investigated in this patient population, neurocognitive symptoms may go undetected, which can consequently can lead to these children not receiving the treatment and educational support they need.

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We only had one wish for our thesis, well, two. One, we wanted to write about neuropsychology, and two, we thought we would make a great team writing it together. Luckily, this project allowed us to do both.

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"Whenever times were tough, you came around with your adorable fluff
The most important dedication of them all, is one we can no longer stall
Our project has its very own mascot, thank you very much, wonderful Spot"

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1 Introduction

1.1 The burden of pediatric traumatic brain injury

The most frequent cause of interruption to a child's normal course of brain development, is acquired brain injury (Catroppa, Anderson, Beauchamp, & Yeates, 2016). Brain development is a continuous process, starting before birth, and progressing into young adulthood. Early experiences are essential in shaping the architecture of the developing brain and acquired injury or disease can be especially impactful when occurring during childhood and adolescence. Traumatic brain injury (TBI) is commonly defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force" (Menon, Schwab, Wright, & Maas, 2010, p.1637). A pediatric TBI (pTBI) can in and of itself have a detrimental impact on a child's development and later life, and as children are in a state of constant learning and development, TBI not only affects existing skills and abilities, but can also impair prerequisites for development of future skills that are yet to emerge (Giza & Prins, 2006; Ryan et al., 2016; Muscara, Catroppa, & Anderson, 2008). Thus, acquired difficulties may not be immediately evident, especially for higher order executive functions that have not fully developed, nor may they be correctly interpreted as sequelae, but rather explained as behavioral or social difficulties of other reasons (Ganesalingam, Sanson, Anderson, & Yeates, 2007). This highlights the importance of identifying children and families who are in need of assessment and rehabilitation services after pTBI.

Norwegian data regarding long-term outcomes and rehabilitation needs following pTBI is limited and therefore much needed in order to design interventions and provide appropriate rehabilitation. To achieve this, it is paramount that clinicians have knowledge about the unique challenges associated with sustaining a pTBI, as well as the heterogeneity of cognitive and psychosocial outcomes and how these might interact with the complexity of other relevant factors relating to the child itself, and the child's environment. The current study is one of few Norwegian explorations of neurocognitive and psychosocial outcomes after pTBI, and aims to contribute to a better understanding of this population as well as its challenges.

1.2 Definition and classification

Traumatic brain injury is commonly defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force" (Menon et al., 2010, p. 1637). By

"alteration in brain function", one of the following clinical signs must be present: any period of loss of, or decreased level of consciousness, any loss of memory, either retrograde or post traumatic amnesia, neurological deficits, or any alteration in mental state at the time of the injury. Further, "other evidence of brain pathology" may include visual, neuroradiological, or laboratory confirmation of damage to the brain. "Caused by an external force" includes events such as the head being struck by an object, the head striking an object, the brain undergoing an acceleration/deceleration movement without direct external trauma to the head, a foreign body penetrating the brain, forces generated from events such as a blast or explosion or other force yet to be defined. Although there is no universal consensus regarding severity definitions, TBI is commonly classified by considering either duration of post traumatic amnesia (PTA), duration of loss of consciousness (LOC), and Glasgow Coma Scale score, or a combination of the three (Iverson & Lange, 2011) as shown in Table 1. PTA is a state characterized by disorientation and/or confusion, with significant memory deficits present, particularly for storing new information (Russell & Nathan, 1946). LOC is depicted as "lack of awareness, with the person appearing to be in sleep-like state" (Iverson & Lange 2011, p. 666). GCS is a clinical scale consisting of three dimensions measuring different aspects of behavior: motor-responsiveness, verbal performance, and eye opening (Teasdale & Jennett, 1974).

Table 1.

Classification system for traumatic brain injury

Classification	Loss of Consciousness	Glasgow Coma Scale	Post Traumatic Amnesia
Mild	<30 minutes	13-15	<24 hours
Moderate	30 minutes - 24 hours	9-12	1-7 days
Severe	>24 hours	3-8	>7 days

Note. From "Moderate and severe traumatic brain injury," by G. L. Iverson & R. T. Lange, 2011, "Little black book of neuropsychology, p. 667.

Although the aforementioned GCS ranges are most frequently used in defining severity groups, a subdivision of the mild TBI construct into complicated and uncomplicated categories has received increased attention. The original definition proposed dividing mild TBI (mTBI) into uncomplicated mild (uMTBI) and complicated mild (cMTBI) categories based on differential injury characteristics (Williams, Levin, & Eisenberg, 1990). In this definition of uMTBI and cMTBI, the two are differentiated by the presence of one or both of the following criteria: (a) depressed skull fracture and/or (b) a trauma related intracranial

abnormality (e.g., hemorrhage, contusion or edema). If criteria (a) and/or criteria (b) is present, the injury is classified as complicated mild TBI (Williams et al., 1990). Some researchers have also proposed to drop criteria (a) entirely (Iverson et al., 2012). Support for this definition has been found in research on adult TBI, where a subset of patients with more severe injury characteristics suffer adverse outcomes despite the injury being classified as mild (Borgaro, Prigatano, Kwasnica, & Rexer, 2003). A similar pattern has also been demonstrated in the pediatric population (McKinlay, Dalrymple-Alford, Horwood, & Fergusson, 2002).

1.3 Incidence, prevalence and societal burden of pTBI

It has been estimated that about 50 million people sustain a TBI per year worldwide, and that approximately half of the world's population will sustain at least one TBI across their lifespan (Maas et al., 2017). In developed countries, TBI is the leading cause of morbidity and mortality for children and adolescents and represent an important public health issue with a significant economic, societal, and individual burden (Catroppa et al., 2016). For TBI in general, the economic burden is substantial, with societal costs estimated at $US400 billion annually (Maas et al., 2017). In the U.S population, moderate and severe TBI had the highest health care cost at the individual level. However, at a population level, the costs of mild TBI far exceed those in the moderate and severe groups, due to the vast majority of the injuries being classified as mild (Graves, Rivara, & Vavilala, 2015).

Available data regarding incidence and prevalence of pTBI has considerable limitations, and estimates vary substantially worldwide due to variations in data sources, modes of data collection, injury classification, and age range of the target population (Catroppa et al., 2016). A recent epidemiological review found that most countries report a range between 47 and 280 per 100 000 children. However, these numbers are largely based on hospitalized individuals with TBI (Dewan, Mummareddy, Wellons, & Bonfield, 2016). When taking into account children who consulted either the emergency room or a general practitioner, but were not admitted to hospital, the average incidence rate increased to 1750 per 100 000 per year (McKinlay et al., 2008). European epidemiological analyses present rough estimates ranging from 47.3 per 100 000, to 694 per 100 000 in country level studies, when including all age and severity groups (Brazinova et al., 2016).

Cause of injury varies by age, with the most common cause of pTBI being motor vehicle accidents, falls and assaults for the children below the age of ten (Catroppa et al., 2016). Falls

are the predominant injury mechanism for children <4 (Crowe, Babl, Anderson, & Catroppa, 2009) with this also being the group most at risk for TBI as a result of child abuse (Adelson & Kochanek, 1998; Keenan & Bratton, 2006). Sports injuries become more common for children school aged and older (Crowe et al., 2009). The breakdown of severity level in pTBI is similar to that of adults, with mild TBI comprising 89 % of cases, and moderate and severe TBI only 8 and 3 % respectively (Crowe et al., 2009), and of the 700 000 children hospitalized annually in the U.S, 80-90% are reported to have mild injuries (Emery et al., 2016).

1.4 Mechanisms and pathophysiology: Characteristics of the pediatric brain

An extensive review of pathophysiology and mechanisms of TBI is beyond the scope of this thesis, and this topic has been thoroughly covered by multiple authors (e.g. see Lezak, Howieson, Bigler, & Tranel, 2012). Knowledge of the pathophysiology and mechanisms of TBI in general, and factors that apply specifically to the pediatric population, is however essential in order to provide adequate and patient-oriented treatment for children with TBI (Werner & Engelhard, 2007).

TBI is classified into penetrating (PHI) or closed head injury (CHI), where PHI includes all injuries where the skull and dura are penetrated, and CHI includes injuries where the brain or the dural layer covering the brain remains undamaged (Andriessen, Jacobs, & Vos, 2010). The injury mechanisms that occur during and after TBI are further divided into primary and secondary, where primary injuries are the direct result of the mechanical forces affecting cerebral tissue, and includes pathologies such as contusions, hemorrhage and axonal shearing. Secondary injuries refer to indirect damage initiated by the trauma, including complications such as hemorrhage, hypoxia, ischemia, elevated intracranial pressure and changes in metabolic function as well as the cellular and molecular processes that are initiated by the primary injury (Lezak et al., 2012).

Brain injuries are further classified as focal or diffuse (Catroppa et al., 2016; Lezak et al., 2012), and both pathologies are often present simultaneously (Skandsen et al., 2010). Focal refers to localized pathologies and includes contusions, lacerations, brain herniations, epidural- or subdural hematomas. Focal damage may occur as a result of coup or contrecoup (Sayed, Mota, Fraternali, & Ortiz, 2008), with coup being the initial blow at the point where the brain impacts the skull, often causing a cerebral contusion at site of impact. After initial

impact, the mechanical force causes the brain to bounce back to the diametrically opposite side of the skull causing a contrecoup effect with a resulting lesion. These lesions most frequently occur in the frontal and temporal areas, as well as the sylvian fissures (Lezak et al., 2012). Diffuse and multifocal injury occur over a more widespread area. Diffuse injuries can occur without the head striking, or being struck by an object, and are frequently seen in accidents involving sustained acceleration and deceleration forces (Skandsen et al., 2010). One such diffuse injury is diffuse axonal injury (DAI), which is a predominant pathological feature in TBI. DAI is found in white matter tracts (Smith, Meaney, & Shull, 2003), and describes damage to axons as a result of shearing or rotational forces. DAI is further classified as mild, moderate or severe (grade 1-3). DAI grade 1 is characterized by lesions in both cerebral hemispheres, grade 2 includes lesions to the corpus callosum, and grade 3 demands additional lesions present in the rostral lateral–dorsal brainstem (Meythaler, Peduzzi, Eleftheriou, & Novack, 2001; Vik, Kvistad, Skandsen, & Ingebrigtsen, 2006).

Age specific biomechanical properties of the skull, face, brain and neck muscles lead to children being susceptible to specific injuries that are less frequently present in adults. Children have larger heads relative to their bodies with the head being relatively heavy, which makes them vulnerable to both sustaining a pTBI, as well as different dynamics in response to acceleration or other external trauma forces (Pinto, Meoded, Poretti, Tekes, & Huisman, 2012). Furthermore, the higher flexibility of the cranial bones enhances the skulls capacity to absorb the mechanical forces, and results in diffuse, rather than focal damage (Begali, 1992). Absorption of trauma forces differ in the different areas of the brain, depending in part on the stage of myelination. Frontal regions are the last to myelinate and are thus one of the more susceptible regions to TBI (Pinto et al., 2012). Consequently, a TBI sustained early in life can significantly complicate neurodevelopmental processes (McKinlay, Grace, Horwood, Fergusson, & MacFarlane, 2009).

In pediatric TBI, DAI has been shown to occur in all severity groups, with studies reporting up to 40 % in children suffering severe TBI. DAI is challenging to detect on standard imaging, and its impact and extent is likely underestimated (Tong et al., 2004). Diffuse axonal injuries have been associated with poor outcomes post-injury, and these disruptions to white matter integrity have been linked to persistent impairments in global intelligence, language and executive functions (Ewing-Cobbs, Hasan, Prasad, Kramer, & Bachevalier, 2006; Ewing-Cobbs et al., 2008).

1.5 Children are not simply tiny adults

The developing brain has a great capacity for development and change and is therefore considered «plastic». Plasticity refers to the adaptive structural and functional changes that occur in the brain after lesions and during development (Anderson, Spencer-Smith, & Wood, 2011). Throughout childhood and adolescence, brain development is characterized by rapid growth followed by a decline in gray matter volume, with periods of rapid neurogenesis and synaptogenesis subsequently giving way to pruning and neuronal death. Unlike grey matter, myelination steadily increases throughout childhood and adolescence, and is not complete until the third decade of life. From birth, the brain volume increases four-fold and has by age 6 reached 90 % of adult volume (Teffer & Semendeferi, 2012).

Good outcomes reported in studies of focal lesions and hemispherectomies have resulted in some researchers arguing that the increased plasticity in children can lead to a higher capacity for recovery after insult, culminating in the "early plasticity" hypothesis. However, in these types of insults, much tissue remains undamaged, whereas in pTBI damage tends to be diffusely distributed. In these cases, the "early plasticity" model falls short in explaining recovery patterns. The "early vulnerability" hypothesis emerged as a response to this, proposing that the developing brain is in fact especially vulnerable to the effects of TBI compared to that of adults (Crowe, Catroppa, & Anderson, 2015). Current research favors the "early vulnerability" hypothesis in the understanding of pTBI outcomes, and low age in combination with severe injury is considered to represent a "double hazard" with particularly poor outcomes (Anderson, Catroppa, Morse, Haritou, & Rosenfeld, 2005).

1.5.1 Challenges in assessing functional recovery and residual deficits

Understanding and quantifying recovery is different in children compared to adults. In adults, cognitive functions are fully developed and remain relatively stable over time. If a given function is impaired as a result of TBI, the adult patient would be considered fully recovered when that function returns to estimated premorbid level. This is complicated when evaluating pediatric recovery from TBI, as development and skill acquisition must be considered in a dynamic fashion (Giza & Prins, 2006). The developing brain is less functionally committed and specialized than in adulthood (Crowe et al., 2015), and brain insult in children occurs during a time when neural networks underlying behavior and cognition are undergoing rapid maturation and consolidation, which can not only disrupt existing functions, but can also impede further learning processes and as such interfere with developmental milestones (Ryan

et al., 2016). This essentially means that even if the child returns to premorbid baseline, it could still represent a fall in function compared with age matched peers, who have already exceeded that point of skill acquisition (Anderson, Catroppa, Morse et al., 2005; Giza & Prins, 2006).

Although children may appear to function normally after initial recovery, the actual impairment might emerge over time. Due to the protracted development of the frontal lobes and high level social and cognitive functions, the full extent of post-injury deficits may only be discernible years after the initial brain insult, when these functions are expected to be fully developed (Giza & Prins, 2006; Ryan et al., 2016; Muscara et al., 2008). This concept of deficits emerging as the child's development progresses, or "growing into the lesion", was illustrated by Anderson, Bechara, Damasio, Tranel, and Damasio (1999). They reported two cases of lesions to the prefrontal cortex that were sustained before the age of 16 months. Despite initial recovery, as adults, both individuals displayed significant deficits in frontal lobe functions.

1.6 Outcome and predictors after pediatric TBI

Pediatric TBI can result in a broad range of sequelae that might be expressed as difficulties in cognitive, emotional and behavioral domains. Residual symptoms and recovery trajectories are influenced by complex interactions of different factors, few of which act in isolation. This warrants a biopsychosocial approach when attempting to understand and predict outcome in children where the interplay of the different biological, psychological and social factors is taken into account (Chapman & McKinnon, 2000), as demonstrated in Figure 1. This approach poses that features from all of the three domains may collectively contribute in producing a certain symptom picture or outcome. The biological aspect encompasses a multitude of factors, including the nature and severity of the head injury itself, potential complications or secondary injury processes, premorbid neurological conditions, illness or previous head injury, as well as chronological and developmental age at time of insult (Catroppa et al., 2016). The nature and severity of brain injuries have consistently been shown to impact functional outcome, with increasing injury severity being linked to poorer outcomes in predominantly cognitive, but also psychosocial domains (Anderson et al., 2014). Adding to this complexity is the fact that the child's premorbid levels of functioning also influence outcomes within the domains of behavioral and adaptive functioning (Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2008). These pre-injury factors may include

resources in form of cognitive status, behavioral adjustment and academic performance, as well as post-injury factors such as cognitive sequelae and coping mechanisms. Furthermore, both pre- and post-injury factors related to the child's caregivers exert an influence, such as family functioning, premorbid stressors, socioeconomic status, parental adjustment after injury and perceived family burden (Yeates, Taylor, Walz, Stancin, &Wade, 2010).

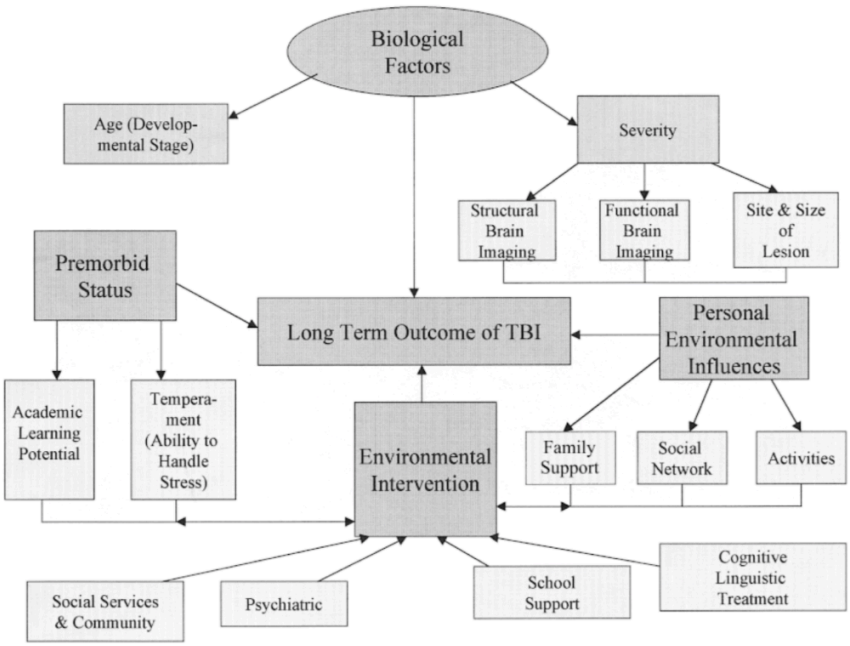

Figure 1. Factors that influence the long-term outcome of traumatic brain injury in children. Reprinted from "Discussion of developmental plasticity: Factors affecting cognitive outcome after pediatric traumatic brain injury", by S. B. Chapman & L. McKinnon, 2000, *Journal of Communication Disorders, 33*(4), p. 335. Copyright Elsevier Science Inc 2000. Reprinted with permission.

1.6.1 Cognitive outcome

Neurocognitive outcome in pTBI has been extensively researched, yet the findings are inconsistent (Babikian & Asarnow, 2009). Cognitive impairment after pTBI can be present in various degrees and can affect most neurocognitive domains with memory, attention and executive function being particularly vulnerable (Keenan & Bratton, 2006). In general, the research demonstrates a dose-response relationship with more severe injuries causing higher degree of neuropsychological impairment (Babikian & Asarnow, 2009).

Children with TBI are at increased risk of experiencing post-concussive symptoms (PCS) compared to children with other types of injuries (Emery et al., 2016). The symptom picture varies, but may include cognitive problems such as reduced attention, slowed response speeds, memory impairment, and somatic symptoms such as fatigue, headache and

dizziness (Ayr, Yeates, Taylor, & Browne, 2009). Although these symptoms are not specific to the mild group, they are commonly referred to as PCS specifically for the milder injuries (Iverson, 2005) as more pronounced deficits tend to be present for the moderate and severe injuries (Babikian & Asarnow, 2009). Furthermore, although these symptom constellations are indicative of TBI sequelae, they are not uniquely sensitive to TBI and must be differentiated from a range of other conditions, such as depression, chronic pain, and post-traumatic stress disorder (Iverson, 2005).

Post-concussion symptoms normally resolve during the first few weeks after mTBI, but may also persist for months, or in some cases, years in a minority of patients (Emery et at., 2016; Hessen, Nestvold, & Anderson, 2007). For the children who experience persistent symptoms, there may be additional risk factors present, such as young age at insult, preexisting behavioral or cognitive problems, or abnormalities during clinical assessment (Catroppa et al., 2016).

The following sections will present working memory, attention, processing speed and executive functions separately, however, these functions are in reality interrelated, and most neuropsychological tests tap into more than one cognitive domain (Lezak et al., 2012).

General intellectual functioning

Reduced scores on measures of intellectual functioning have consistently been demonstrated after pTBI, with poorer performance being tied to increasing severity (Babikian & Asarnow, 2009; Crowe et al., 2015). In cases of moderate or severe TBI prior to age six, persistent deficits have been documented in both intellectual and academic development 5 years after injury (Ewing-Cobbs, Prasad, et al., 2006). With regard to the mild injuries, the majority of studies report minimal deficits 2 years post-injury within the domains of intellectual functioning, attention and memory (Babikian & Asarnow, 2009). Although few statistically significant effects have been reported for mild pTBI, there does seem to be a subset of these children who do not adhere to this pattern, and who show adverse outcomes. A ten-year follow-up study reported intellectual functioning to be average at the group level, yet examination of individual results using impairment ratings yielded considerably higher impairment rates in the mild group compared to population expectations (Anderson, Brown, Newitt, & Hoile, 2011).

Attention, processing speed and working memory

Models of attention typically emphasize that attention relies on the efficient functioning of discrete but interacting cerebral regions. If any of these areas are exposed to damage or dysfunction, it may lead to an impairment which in turn will impact attentional skills, either in a global way, or on specific aspects of attentional processing (Anderson, Fenwick, Manly, & Robertson, 1998).

Attentional problems are often seen in children who sustain TBI, including poor concentration, distractibility, inability to complete tasks, and difficulties related to tasks that demand rapid switching. Deficits in both attention and speed of information processing have been demonstrated up to 5 years post-injury in pediatric samples. Although these deficits are seen across all severities, the severely injured children are at the highest risk for impairment (Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2007). However, one study examining long term outcomes in uMTBI and cMTBI reported that the complicated group performed more poorly on measures of divided attention, with younger age at injury and neurological symptoms being associated with poorer performance (Papoutsis, Stargatt, & Catroppa, 2014).

Working memory (WM) is perhaps best illustrated by the well-known definition launched by Alan Baddeley (2010) p. 136 "Working memory refers to the system or systems that are assumed to be necessary in order to keep things in mind while performing complex tasks such as reasoning, comprehension and learning". This system is considered to be limited in its capacity related to storage, monitoring, and manipulation of information (Knight & Stuss, 2002). Working memory deficits are some of the most frequent patient complaints after TBI (McAllister, Flashman, McDonald, & Saykin, 2006), and neuropsychological deficits in working memory and inhibition have repeatedly been demonstrated after pTBI (McAllister et al., 2006; Conklin, Salorio, & Slomine, 2008; Ewing-Cobbs, Prasad, Landry, Kramer, & DeLeon, 2004; Levin et al., 2004; Mandalis, Kinsella, Ong, & Anderson, 2007; Sinopoli & Dennis, 2012). WM is mediated by frontal lobe networks (Moscovitch & Winocur, 2002) and intact function of these networks is a prerequisite for the normal development of more complex cognitive skills in the maturing brain (Anderson, Levin, & Jacobs, 2002).

Memory and learning

Memory is not a unitary function, but an integrated system of cognitive processes that together contribute to facilitate the process of registering, encoding, storing and retrieving information. Impairment in one of the memory components may have downstream effects, in

the sense that it may cause malfunctions in other parts of the system (DeMaster, Johnson, Juranek, & Ewing-Cobbs, 2017). These different components are situated in different cortical and subcortical areas of the brain, such as limbic structures and anterior brain regions, all of which are vulnerable to the impact of TBI (Catroppa et al., 2016). Memory impairments can have a particularly detrimental effect when sustained during childhood, due to the disruption of normal brain development, and the exacerbating effect that memory and learning deficits can have on the attainment of new cognitive functions and mastery of normal learning (DeMaster et al., 2017). Memory deficits have been demonstrated after moderate and severe pTBI, and these difficulties may persist years after the injury. The findings are subtle for mild injuries, with some studies finding no differences between mild TBI and control groups (Babikian et al., 2011).

Executive function

Executive functions (EF) refer to the top-down cognitive processes that are necessary to achieve goal-oriented and purposeful behavior (Knight & Stuss, 2002; Stuss & Levine, 2002). Executive functions play a role in aspects of behavior such as goal formulation, initiating behavior, anticipation of consequences, planning and organizing, and adapting one's behavior to the task and context. Consequently, executive dysfunction (ED) can have a substantial impact on both development and functioning in everyday life (Cicerone et al., 2000).

The prefrontal cortex (PFC) is considered the primary neuroanatomical substrate of EF. The PFC modulates other neural systems in the brain through distributed connections putting prefrontal areas in a position to activate, inhibit and influence other structures of the brain (Knigh & Stuss, 2002). Therefore, impairment in EF can present after damage to other structures due to the interruption of distributed neural networks involving the prefrontal cortices (McAllister et al., 2006). In addition, the protracted development and maturation of the frontal lobes plays an important role in making EF especially vulnerable to insult during childhood (Pinto et al., 2012).

Executive dysfunction is a common neuropsychological sequela after TBI (Stuss & Levine, 2002), and a higher degree of EF deficits has been demonstrated for the moderate and severe injuries in long term assessment of outcomes after pTBI (Muscara et al., 2008). The results are less clear for mild TBI, as some studies do not find evidence for ED (Maillard-Wermelinger et al., 2009), while others report significant impairment in approximately 20-40 % within the first year post-injury (Sesma, Slomine, Ding, & McCarthy, 2008).

1.7 The importance of the current study

Up until recently, the understanding of pTBI has lagged behind that of adults, and the previously held assumption of children being more resilient to head injuries has largely been dismissed. The current understanding is that pTBI has to be viewed within a complex biopsychosocial developmental model (Muscara et al., 2008), and that early brain insult can both alter and disrupt developmental trajectories, making the pediatric brain especially vulnerable to adverse outcomes (Anderson et al., 2014; Catroppa et al., 2016).

Regarding Norwegian data, many studies from the past decade have provided valuable information about the epidemiology, outcome and health care needs following adult TBI (Andelic et al., 2012; Andelic et al., 2010; Andelic, Soberg, Berntsen, Sigurdardottir, & Roe, 2014; Andelic, Ye, et al., 2014; Sigurdardottir, Andelic, Roe, & Schanke, 2009). This has formed the basis for improved clinical management and rehabilitation services for adults with TBI, yet research on long-term outcomes and health service utilization in children with TBI is somewhat lacking. This Pediatric TBI-study is the one of the few Norwegian prospective, follow-up studies of children with TBI, and the first sample from the South-Eastern Health Region.

1.7.1 Aims and research questions

The aim of this thesis is to explore the neurocognitive and psychological effects of pediatric traumatic brain injury through analysis of data on the demographic and medical characteristics, and 6-months outcome data in a cohort of hospitalized children with TBI in South-Eastern Norway Health Region.

The following research questions will be addressed:

1) What characterizes the sample with regard to demographic and injury related variables and acute treatment efforts?
2) What are the neurocognitive and psychosocial outcomes 6 months after injury?
3) Do premorbid child or family factors, or injury specific characteristics have a subsequent impact on outcome?

2 Method

2.1 The study

This study was conducted in collaboration between Oslo University Hospital (OUH) and Sunnaas Rehabilitation Hospital (SunHF). The current pTBI study is an extension of a large-scale CENTER-TBI study (Maas et al., 2015). CENTER-TBI aims to study injury characteristics, clinical care and outcome of over 5000 mainly adult patients across Europe, and collects information on medical variables, radiological data and outcome after TBI. In the CENTER-TBI study, neuropsychological and extensive psychological follow-up is not performed for the children. The Oslo pTBI study is an ongoing PhD-project with pediatric neurologist Hilde M. Dahl as the PhD candidate. It includes assessment of functional and neurocognitive outcomes for the children enrolled and assesses health care service needs over the first two years after injury. The current thesis makes use of a subset of the data collected from this pTBI study.

2.2 Participants and procedures

The pTBI study includes children aged 12 months to 15 years who presented at OUH with a verified TBI. Inclusion criteria were as follows: a) children age 1-15 years, b) qualifying for the ICD-10 (2004) diagnosis S06.0-S06.9 (pathologies such as concussions, cerebral contusions, subarachnoid-, subdural-, and epidural hematomas and diffuse axonal injuries), and c) head trauma requiring CT scans performed within 24 hours after injury. Although a prerequisite for inclusion was meeting the criteria for ICD-10 (2004) diagnosis s.06.0-s06.9, some also had additional diagnosis of s02.00-s02.9 (different types of fractures to the skull and facial bones). Exclusion criteria were a) having pre-existing neurological disorders, and b) patient residing outside the South-Eastern Norway Health Region.

Recruitment of participants took place from January 2015 throughout 2016, with follow-up assessment being completed between 5 and 8 months after injury. Recruitment and inclusion was handled by a pediatric neurologist (Dr. Med. Hilde M. Dahl) in collaboration with the research team connected to the CENTER-TBI study in the time period January 2015 - March 2016 (Figure 2). In order for the project to reach an acceptable number of participants, inclusion continued beyond the recruitment period of CENTER-TBI (ended march 2016), that is throughout 2016. Therefore, a detailed Center TBI-based register data of those not included for various reasons is only available until March 2016, with details for the

remaining nine months being logged in the Norwegian Trauma registry. In 2015, 71 % of eligible children were included, and preliminary inspection suggest a fairly equal rate for 2016 (H. Dahl, personal communication, September 4, 2018). Exact numbers for 2016 have not yet been made available from the Norwegian Trauma registry. Therefore, the recruitment phase is illustrated with numbers based on 2015, with an expected comparable inclusion rate in 2016.

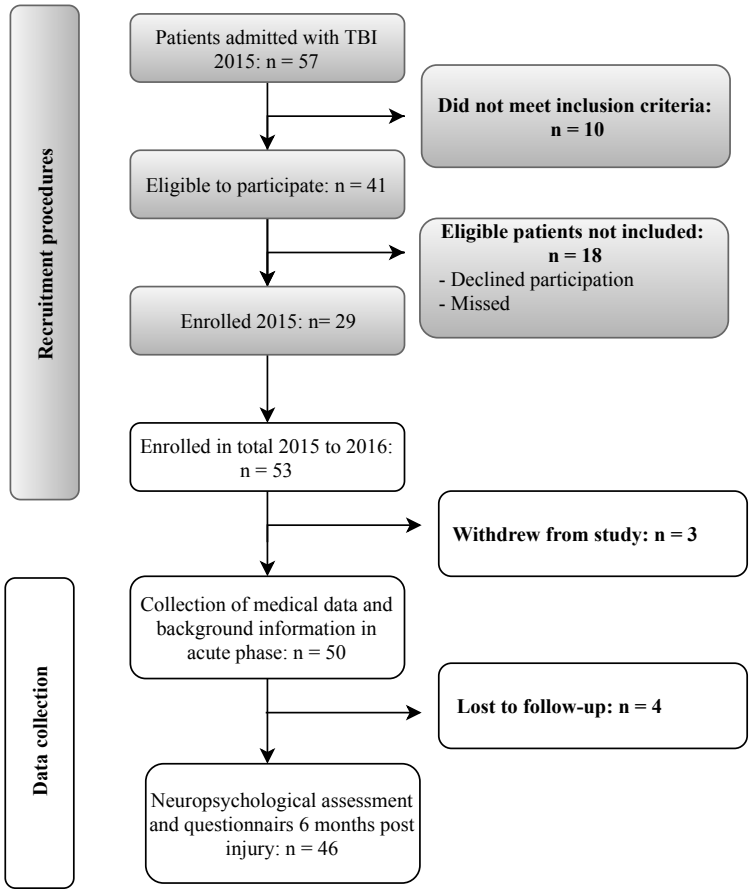

Figure 2. Overview of screening procedures and assessment points. Inclusion for 2015 is presented in detail (grey area), whereas the data collection and follow-up are based on total numbers from the entire inclusion period (white area).

TBI patients from the South-Eastern parts of Norway, residing outside of Oslo who are in need of neurosurgical evaluation/treatment are referred directly to OUH, as OUH is the Trauma Referral Hospital for the South-East of Norway. In Oslo, children presenting with signs of concussion or TBI, are directed to OUH if there is clinical indication for a CT scan. This ensured inclusion of the whole spectrum of injury severity, with the exception of the injuries where CT was not required.

The study was approved by the Regional Committees for Medical and Health Research Ethics (REK) and has been conducted in accordance with the Helsinki declaration (World Medical Association, 2013) and Vancouver rules (International Committee of Medical Journal Editors, 2018). Informed consent was provided by the children's legal guardian. Children older than 7 years were asked for their opinion on study participation before a parental decision was made.

2.3 Acute medical and demographic data

2.3.1 Demographic, injury and trauma variables

Parental length of education was recorded. Data regarding cause of injury, length of hospitalization, and whether the children were transferred to their home or to a rehabilitation institution was collected. Information related to surgical interventions was obtained, and the types of procedures were divided into the following categories: intracranial pressure monitoring (ICP), intracranial- and extracranial surgery, and facial surgery. Procedures not related to the head-region was not recorded for this study.

Trauma scores were extracted from medical journals by dr. med. Hilde M. Dahl. The severity of TBI was classified based on the lowest Glasgow Coma Scale score within 24 hours of hospital admission. The Abbreviated Injury Scale head (AIS_{head}) sub-score of the Abbreviated Injury Scale (AIS), version 1998 (Association for the Advancement of Automotive Medicine, 1990) was used to classify the anatomical severity of the brain injury. AIS_{head} is one of nine chapters with corresponding body parts, which together constitute the AIS. The Abbreviated Injury Scale classifies the injury in these 9 parts of the body in line with its relative importance on an ordinal scale ranging from 1 (minor) to 6 (maximum). An AIS_{head} score between 3 and 5 indicates intracranial pathology, and takes into account size, location and multiplicity of lesions (Foreman et al., 2007).

Overall injury severity was assessed with the Injury Severity Scale (ISS). ISS ranges from 1-75, with higher scores indicating more severe injury. To calculate ISS, each injury must be rated with AIS, then the squares of the highest AIS rating for the three most severely injured body areas are added together (Baker, O´Neill, Haddon, & Long, 1974; Baker & O´Neill, 1976). An ISS of 15 or above is defined as a severe trauma (Palmer, Gabbe, & Cameron, 2016).

2.3.2 Radiology

As part of the inclusion criteria, all participating children completed a CT head scan at admission, while MRI head scans were only performed if participants either a) consented to MRI as part of the CENTER-TBI study, or b) clinical indication of an MRI scan was present. Pathology on CT and/or MRI was defined as the presence of skull fractures, cerebral contusions, diffuse axonal injuries, subarachnoid-, epidural-, or subdural hematomas. Pathology was further dichotomized into presence/absence of abnormality. Incidental findings that were not injury-related were not recorded as a positive finding in this study.

2.3.3 Classification of severity

Injury severity was classified according to standard GCS criteria (Teasdale & Jennett, 1974). Mild TBI as defined by GCS 13-15 was further classified as uncomplicated (uMTBI) or complicated (cMTBI) depending on whether there was evidence of trauma-related intracranial abnormalities on either CT or MRI. The moderate (GCS 8-12) and severe (GCS <8) injuries were collapsed into one group due to the small number of severe injuries. Preliminary analyses were conducted with the moderate and severe groups remaining independent, and there were no differences to contraindicate combining the two groups. The combined moderate and severe group is referred to as m/sTBI.

2.4 Neuropsychological outcome measures

2.4.1 Performance based neuropsychological measures

Neuropsychological assessment instruments were selected in accordance with the recommendations provided by the NIH Pediatric TBI Common Data Elements Outcomes Workgroup (McCauley et al., 2012). The following domains were assessed: General intellectual capacity, motor functions, visuo-motor functions, working memory, attention, executive functions, learning and memory.

General intellectual capacity was assessed using the core tests that provide an IQ estimate in the Wechsler Scale suited for the child's age. For children age 2,5 to 7 years the Wechsler Preschool and Primary Scale of Intelligence (WPPSI-III) was used (Wechsler, 2002). Children 7 years and older were assessed using the Wechsler Intelligence Scale for Children (WISC-IV) (Wechsler, 2003). Core tests are summed to four indexes: perceptual reasoning index (PRI), verbal comprehension index (VCI), processing speed index (PSI), and working memory index (WMI), and the full scale IQ (FSIQ) is comprised of these four

indexes. In cases of missing subtest or index scores, GAI was calculated when possible, as GAI only consist of the indexes VCI and PRI. Particular emphasis was placed on the WISC-IV for the analyses as the majority of participants fell within the age range of WISC-IV as opposed to WPPSI-III. *Motor functions* were assessed with the Grooved Pegboard Test (Model 32025, Lafayette Instrument, USA) from the age of seven. Newly published Norwegian norms were applied (Skogan, Oerbeck, Christiansen, Lande, & Egeland, 2018). For measures of *visuo-motor functioning* in all age groups, Beery-Buctencia Developmental Test of Visual-Motor Integration (BVMI) was administered (Beery, Buktenica, & Beery, 2010). *Working memory* was assessed by numbers subtest from the Children's Memory Scale (CMS) (Cohen, 1997) for those between age 5 - 7 years, and Digit Span Test from WISC-IV (Wechsler, 2003) above age of 7. *Learning and memory* was assessed using Children's Auditory Verbal Learning Test-2 (CAVLT-2) (Talley, 1992) above 7 years. *Attention* was measured using Conners Continuous Performance Test (CCPT-II) (Conners, 2002) from age 8. *Executive functions* were assessed from 8 years, using the following subtests from Delis Kaplan Executive Function System (Delis, Kaplan, & Kramer, 2001); The Trail Making Test (TMT), conditions 2 & 4, and The Color Word Interference Test (CWI), all conditions.

2.4.2 Neuropsychological impairment index

As a consequence of the broad age range, a restricted number of tests were completed by all participants. A dichotomized impairment variable was created in an effort to quantify neuropsychological impairment levels for the total sample. Scores below one standard deviation from the normative mean occur frequently in healthy adult samples, as at least two abnormalities are typically seen in most participants on test batteries with at least 20 measures (Binder, Iverson, & Brooks, 2009). In order to account for performance variability and minimize the likelihood of false positives, neuropsychological impairment was defined as two or more subtests equal to, or more than 1,5 standard deviations below the normative mean.

2.4.3 WISC-IV index and composite score discrepancies

Intraindividual index and composite score discrepancies were examined for each participant, in order to allow investigation of test variation that was not evident by looking exclusively at the sample average for each index. Discrepancies were considered clinically significant if a comparable variation is present in ≤ than 10 % of the WISC-IV normative sample (Wechsler,

2003). There were however some cases where significant discrepancy was present, yet none of the index scores were below score 90. This was hypothesized to potentially be the result of a) normal variation in cognitive strengths and weaknesses, or b) a relative fall in previous high functioning. To further examine this, two separate groups were created; 1) all clinically significant discrepancies, and 2) clinically significant discrepancies remaining when excluding participants with all index scores above 90.

In order to further explore whether individual discrepancies were evident at a group level, paired-samples t-tests were used to determine if there was a statistically significant mean difference between indexes (PRI, VCI, PSI and WMI) as well as between the two composite scores (FSIQ and GAI). Of note, these comparisons require the same participant to have scores on both metrics, which was not always the case. As such, mean index scores presented in Table 3 deviate slightly due to some participants not being eligible for these analyses.

2.4.4 Self- and informant reported measures of cognition and executive symptoms

The participants and their parents completed questionnaires measuring post-concussive symptoms and executive functioning in everyday life.

Post-concussive symptoms (PCS) were assessed using the Health and Behavior Inventory (HBI) (Ayr et al., 2009), which has been translated to Norwegian by the research group involved. Children above 7 years reported their current symptoms at the 6 months follow-up, while the parents reported symptom levels retrospectively (4 weeks pre-injury), as well as at 6 months follow-up. HBI consists of 20 items measuring cognitive and somatic symptoms, where the cognitive scale contains 11 items, and the somatic scale is based on 9 items. The cognitive scale seeks to measure symptoms relating to attention, concentration, distractibility, problem solving, memory and learning. The somatic scale relates to headaches, dizziness, nausea, visual disturbances, and fatigue. The scale uses a 4-point Likert scale ranging from 0 “never” to 3 “often”.

Executive functioning in everyday life was assessed using Behavior Rating Inventory of Executive Function (BRIEF) (Gioia, Isquith, Guy, & Kenworthy, 2000), with parental reports from 5 years, and self-report from 11 years. The BRIEF parent report consists of 86 items, whereas the child-report consists of 80 items. These generate 8 subdomains of executive functioning. The subdomains Inhibit, Shift and Emotional Control comprise the

Behavioral Regulation Index (BRI), and the subdomains Initiate, Working Memory, Plan/Organize, Organization of Materials and Monitor constitute the Metacognition Index (MI). BRI and MI combined constitute the overall Global Executive Composite (GEC). The scale uses a 3-point Likert scale with "never", "sometimes", and "often". The generated scores are considered to be within clinical range if $t \geq 65$, whereas higher scores denote poorer function.

2.5 Psychosocial outcome measures

Both the participants and their parents completed questionnaires regarding prosocial behavior and psychopathology, quality of life and family function.

Prosocial behavior and psychopathology was assessed with the Strengths and Difficulties Questionnaire (SDQ) (Goodman, 2001). Parental report was collected for children from 3 years and up, and a self-report version is available for children aged 11 and above. SDQ consists of 25 items, some positive and other negative. The items comprise five subscales, with five items each: emotional symptoms, conduct problems, hyperactivity, peer problems and prosocial behavior. The sum of the four first sub-scales give a total difficulties score, ranging from 0-40 where higher scores indicate more difficulties. The prosocial scale is not included in the total difficulties score as higher numbers indicate better outcomes on this particular scale. Internalizing scale is given by adding together the subscales for emotional symptoms and peer problems. Similarly, the externalizing scale is provided by adding conduct problems with hyperactivity. The scale uses a 3-point Likert scale ranging from "incorrect" to "completely correct".

Quality of life was assessed using the parent version of the Pediatrics Quality of Life questionnaire version 4.0 (PedsQL) from 2 years, and self-report version from 5 years (Varni, Seid, & Kurtin, 2001). PedsQL consist of 23 items, which generates sub-scales of Physical (8 items), Emotional (5 items), Social (5 items), and School Functioning (5 items). All but the first are summed to generate a Psychosocial Health score. For children self-report for ages 8-18 and for parental-reports, a 5-point Likert scale is utilized, ranging from 0 "never a problem" to 4 "almost always a problem". For the youngest children (age 5-7), there is a simplified 3-point Likert scale, ranging from 0 "not at all a problem", 2 "sometimes a problem", to 4 "a lot of a problem", with each response choice anchored to a happy to sad faces scale. Items are transformed to a scale ranging from 0 to 100, whereas higher scores indicate better quality of life.

Family functioning was assessed using the parentally reported 12 item General Family Functioning (GF) subscale from the McMaster Family Assessment Device (FAD) (Byles, Byrne, Boyle, & Offord, 1988). FAD consist of 7 subscales, where the GF-subscale provides a measure of overall family function, and correlates strongly with all of the subscales in FAD. Six of the items in GF describes healthy family functioning, the other 6 items describe unhealthy family functioning, and the GF can thus be used as a brief version of FAD. The scale uses a 4-point Likert scale ranging from "highly agree" to "highly disagree". Items reflecting negative family function are reverse scores, and an average score is calculated, with scores ≥2 indicating concern about family functioning (Miller, Epstein, Bishop, & Keitner, 1985).

2.6 Correlational analysis

The following measures will be used as background variables in the correlational analyses; length of parents' education, age at time of injury, family function, and HBI retrospective evaluations of cognitive, somatic symptom load, and injury characteristics quantified by GCS, AIS_{head}, ISS, and days in hospital. Outcome variables are the performance on neuropsychological tests, and questionnaire scores.

2.7 Data analysis

Statistical analyses were performed using the Statistical Package for Social Sciences (IBM SPSS statistics, version 25). Sample demographics and outcomes are presented descriptively. Due to the use of both parametric and non-parametric methods, and the restricted sample size, both mean and standard deviations, range, and median with 1st and 3rd quartiles are reported for demographic and injury related variables. A range of statistical analyses were conducted depending on the characteristics and distributions of each variable. Variables were explored for violations of assumptions for each statistical test conducted, and these are reported consecutively in the results section.

One sample t-tests were used to determine whether the neuropsychological results for the study sample deviated from the normative sample (e.g. Kahalley, Winter-Greenberg, Stancel, Ris, & Gragert, 2016). One sample t-tests compare the sample mean with a given test value, which was based on the normative mean in the standardization sample. For the WISC-IV index- and composite scores, the test value was set to 100, for subtests 10, and 50 for tests using T-scores.

Paired sample t-tests were used to investigate differences between FSIQ and GAI as well as between the four indexes. All WISC-IV composite and index scores were normally distributed, as assessed with the Shapiro-Wilks´s test ($p>.05$). Categorical variables were assessed using the Pearson X^2 test, reporting Chi Square or Fisher's Exact depending on cell count.

For comparisons between groups, nonparametric tests were performed if assumptions of normality or sample size recommendations ($n<30$) for the parametric equivalent tests were not met. Kruskal-Wallis H tests were performed when comparisons were made between 3 groups (Laerd Statistics, 2015). In cases where Kruskal-Wallis H was statistically significant it was followed up with Mann-Whitney-U tests in order to determine which pairwise group differences were significant. Subsequent pairwise comparisons can be made using a post-hoc analysis utilizing the Dunn's procedure with Bonferroni adjustment, but it is also possible to follow up with multiple Mann-Whitney-U tests for each of the groups, which is what was done here. There is no consensus regarding which of these procedures are considered superior, and if the two methods provide largely discordant results, it warrants replications in larger samples (Laerd Statistics, 2015). Of note, however, no such differences were present here. When two distributions are the same shape, the Mann-Whitney U test is used to determine whether there are differences in the medians of two groups. However, comparing medians requires the additional assumption of comparable shapes of the distributions, an assumption that was not met here. When distributions have a different shape, the Mann-Whitney U test is used to determine whether there are differences in the distributions of groups. As such, scores are determined to be higher or lower between groups based on the use of mean ranks to describe the group differences (Laerd Statistics, 2015).

The relationship between outcomes and demographic and medical variables was investigated using correlational analyses, not regressions, due to the limited sample size. Pearson correlations were conducted when preliminary analysis showed the relationship to be linear with both variables normally distributed, as assessed by Shapiro-Wilks ($p >.05$). When preliminary analysis showed the relationship to be monotonic, as assessed by visual inspection of a scatterplot, Spearman correlations were completed.

Correlational strength is expressed as weak (r/s = 0.1-0.29), moderate (r/s = 0.30-0.49) or strong (r/s >0.50). Effect sizes (Cohen's d) are interpreted as small (0.2), medium (0.5) or large (0.8) (Cohen, 1988). Results are presented with a conservative significance level of $p< .02$.

Due to the exploratory nature of this project, some findings will be mentioned despite not reaching the set level of significance, as they are considered of interest for future research employing larger samples, as well as potentially informing clinical decision making.

3 Results

3.1 Demographic and injury characteristics

Demographic and injury characteristics are presented in Table 2. The total study sample consisted of 50 children, with a majority (n=34) of male participants. Although the lowest registered age was 1 year, only 13 of the children were 6 years or younger. There were no significant differences in injury severity between the age groups <7, 7-12, and >12, nor was there any significant difference in parent´s length of education according to severity level.

The majority of the injuries were transportation related, with falls and sports-related injuries second and third most prominent, respectively. Cause of injury did not differ significantly according to injury severity. For the age group <7 years, 53.8 % of the injuries were caused by falls, while the age group >12 had the highest percentage of sports injuries (29.4 %) (Figure 3).

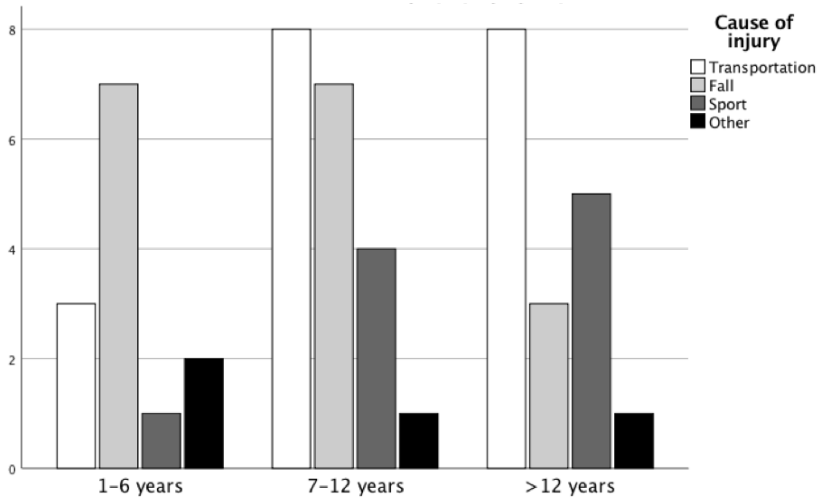

Figure 3. Cause of injury by age group.

Mean GCS score for the whole sample corresponds to moderate brain injury severity, however, the majority (n=32) of the injuries were classified as mild, with the most prevalent overall score being 15. As groups were divided based on GCS scores, it was unsurprising to find an overall effect of severity ($H(2) = 35.79$, $p<.001$), with lower GCS in m/s TBI group (mean rank = 9.50), compared to both uMTBI (mean rank = 29.00, U = .00, z = -5.42, p <.001) and cMTBI (mean rank = 24.00, U = .00, z = -4.49, p <.001). More interesting was the

fact that GCS score did not differ significantly between the uMTBI and cMTBI groups, nor did the distribution of scores.

AIS_{head} mean scores for the total sample fell within the severe range, as the most frequent score was 3 (n=27). Only two participants had a score below this, one being in the uMTBI and one being in the m/sTBI group. Twenty-one children had AIS_{head} scores of 4 or 5, indicating severe and critical head trauma, respectively. The majority of these scores (n=14) were found in the m/sTBI group with the remaining 7 in the complicated MTBI group, and none in the uncomplicated MTBI group. There was an overall effect of AIS_{head} severity ($H(2) = 22.43$, $p<.001$), as AIS_{head} scores were significantly higher in the m/s TBI group (mean rank = 27.69), compared with the uMTBI group (mean rank = 13.40, U= 50.50, z= -4.40, $p< .001$), and between cMTBI (mean rank = 23.36) and uMTBI (mean rank = 12.90, U= 40.00, z= -3.96, $p< .001$). Of note is that these differences likely reflect the fact that groups were stratified based on presence of abnormalities on neuroimaging, something that is closely tied to AIS_{head} scores. Interestingly, there was no significant difference between the cMTBI and m/sTBI groups with respect to AIS_{head} scores.

The ISS scores displayed a broad range and variability of overall injury severity, as the mode was 9 which was also the lowest registered score in the sample. Twenty-seven children (54 %) had an ISS score equivalent to severe injury with a score of 15 or above. ISS scores were significantly different across severity groups ($H(2) = 8.97$, $p=.011$), with scores in the uMTBI group (mean rank = 14.93) being significantly lower than for m/sTBI (mean rank = 25.92), U= 82.50, z= -3.04, $p= .002$.

Twenty-five children had positive imaging results, based on CT and MRI scans. Fourteen of these were within the GCS range of moderate and severe TBI. Contrary to expectations, eleven of the children with mild TBI had positive radiological findings, constituting 34 % of the injuries within the mild GCS range. A total of 16 surgical procedures relating to ICP, intra- and extracranial pathology were performed.

There was substantial variability in length of acute hospitalization. Although the longest stay was 60 days, only six individuals had hospital stays exceeding 20 days, and approximately half of the sample (n=26) were hospitalized for less than five days. Mode for the total sample was 1 day. In the two mild groups combined, the median length of hospitalization was 4 days. Hospitalization length varied between severity groups ($H(2) = 16.86$, $p<. 001$). The uMTBI group had an average stay of 2.55 days (mean rank = 12.25), and the cMTBI group 7.09 days (mean rank = 22.82), which was a significant difference (U= 35.00, z= -3.17, $p= .002$). In comparison, the m/sTBI group had an average stay of 14.94

days (mean rank = 26.36), which was higher than uMTBI (mean rank = 13.33), U= 56.50, z= -3.68, $p< .001$. No significant difference was seen between the cMTBI and m/s TBI with regard to length of hospitalization.

Thirty-eight children were discharged to the home, and 10 were sent to a rehabilitation institution. The children who were discharged to a rehabilitation setting had more severe injuries overall, as quantified by significantly more pathological scores on all of the trauma measures. GCS was lower in the group sent to rehabilitation (mean rank = 12.77), than for the children discharged to home (mean rank = 28.54, U= 74.50, z= -3.30, $p= .001$). Similarly, AIS_{head} scores were higher if further rehabilitation was required (mean rank = 37.27), compared to those sent home (mean rank = 21.45, U= 74.00, z= -3.56, $p< .001$). Similarly, for ISS in the group discharged to a rehabilitation institution (mean rank = 37.41), had higher scores compared with the group discharged to the home (mean rank = 21.41, U= 72.50, z= -3.32, $p= .001$).

Table 2

Demographic- and Injury Characteristics of the Study Sample

	Total sample (n=50)		Uncomplicated mild (n=21)		Complicated mild (n=11)		Moderate/severe (n=18)	
	Mean (SD)	Median [Q_1,Q_3]	Mean (SD)	Median [Q_1, Q_3]	Mean (SD)	Median [Q_1, Q_3]	Mean (SD)	Median [Q_1, Q_3]
Demographic characteristics								
Age at injury	9.64 (4.13)	10.00 [6, 14]	9.67 (3.52)	10.00 [7, 13]	8.55 (4.90)	7.00 [4, 14]	10.28 (4.40)	10.50 [7.5, 14]
Gender, f/m	19/34		6/15		3/8		9/9	
Parental education in years	14.95 (2.44)	15.00 [13, 17]	15.55 (1.93)	16.00 [13.62, 17]	14.68 (2.42)	14.00 [13, 17]	14.36 (3.00)	14.00 [12.50, 15.50]
Trauma characteristics								
GCS	12.26 (3.64)	14.00 [11, 15]	14.43 (.74) c***	15.00 [14, 15]	14.36 (.67) c***	14.00 [14, 15]	8.44 (3.65) a***, b***	10.00 [4.5, 12]
AIS_{head}	3.52 (.78)	3.00 [3, 4]	2.95 (.21) b*** c***	3.00 [3, 3]	3.73 (.64) a***	4.00 [3, 4]	4.06 (.87) a***	4.00 [3.75, 5]
$AIS_{head} \geq 3$ (n)	48		20		11		17	
ISS	18.78 (11.86)	16.00 [9, 25]	13.67 (5.97) c**	13.00 [9, 17.5]	21.00 (15.31)	16 [9, 32]	23.39 (12.95) a**	18.00 [15.75, 32]
ISS ≥ 15 (n)	27		5		7		15	
Hospitalization in days	8.12 (11.03)	4.00 [1.50, 9]	2.55 (2.21) b**, c***	1.00 [1, 4.75]	7.09 (4.59) a**	5.00 [4, 11]	14.94 (15.48) a***	8.50 [2, 23.5]

Note. Significant level is presented as: *$p < .02$, **$p < .01$, ***$p < .001$. The severity groups are labeled as a = uncomplicated mild, b = complicated mild, and c = moderate/severe, with this indicating a significant difference from said group.

3.2 Neuropsychological outcome

3.2.1 Neuropsychological test performance: Group level statistics

Neuropsychological data are presented in Table 3. Thirty-seven children completed the Wechsler Intelligence Scale for Children. Full scale IQ was possible to calculate for 32 of these. When possible, GAI was calculated instead (n=36). All composite and index scores were normally distributed, as assessed by Shapiro-Wilks´s test (p> .05).

There were no significant differences in FSIQ, GAI, VCI, PRI, or PSI scores compared to the standardization sample. A relatively large difference was seen for the WMI, with the WMI scores for the pediatric TBI sample being lower with a mean of 93.18 (SD: 9.95), 95 % CI [3.29 to 10.35], compared to the normal WMI score of 100, t(32) = -3.93, p< .001, d= .68. The WMI difference was particularly driven by low performance on the subtest digit span, with these scores being significantly lower than those obtained in the standardization sample (mean: 8.39, SD: 2.11, 95 % CI [.90 to 2.33], t(35) = -4.57, p< .001, d= .760). CAVLT showed significant differences compared to the normative sample with respect to delayed recall (mean 55.97, SD: 10.45, 95 % CI [2.49 to 9.46], t(36) = 3.47, p= .001, d= .570), surprisingly in the direction of patients performing better than the normative sample.

No significant differences were identified between severity groups regarding the effect of injury severity on neuropsychological performance.

Table 3

Neuropsychological measures

Tests	Total (n=42[a]) Mean (SD)	uMTBI (n=19)[b] Mean (SD)	cMTBI (n=10)[c] Mean (SD)	m/sTBI (n=14)[d] Mean (SD)
Wechsler Intelligence Scale for Children				
Full scale IQ	100.50 (9.73)	101.93 (7.34)	100.67 (6.02)	98.45 (12.40)
General ability index	103.11 (13.92)	104.44 (13.61)	107.00 (11.61)	99.38 (15.50)
Verbal comprehension index	100.94 (17.13)	102.63 (17.18)	108.00 (16.37)	95.08 (16.82)
Perceptual reasoning index	104.78 (12.20)	105.38 (10.63)	104.29 (11.20)	104.31 (15.15)
Working memory index	93.18 (9.95) ***	96.40 (8.50)	85.86 (11.83)	93.45 (8.88)
Processing speed index	97.54 (13.22)	95.88 (12.78)	97.67 (14.37)	99.54 (14.02)
Coding	8.86 (2.91)	8.81 (2.81)	8.57 (2.76)	9.08 (3.30)
Symbol search	10.23 (2.05)	9.75 (1.73)	10.33 (2.80)	10.77 (2.08)
Digit span total	8.39 (2.11) ***	8.75 (2.08)	7.00 (2.00)	8.69 (2.05)
Digit span forward	8.86 (2.14)	9.13 (1.99)	8.00 (3.26)	9.00 (1.58)
Digit span backwards	9.03 (2.41)	9.20 (2.24)	7.86 (1.86)	9.46 (2.81)
Letter number sequencing	9.36 (1.90)	9.93 (1.79)	8.14 (2.47)	9.36 (1.36)
Vocabulary	9.44 (3.14)	9.94 (2.95)	9.57 (3.82)	8.77 (3.14)
Similarities	10.64 (2.87)	10.69 (2.89)	12.43 (1.51)	9.62 (3.07)
Comprehension	10.43 (3.58)	10.69 (3.70)	12.00 (3.26)	9.17 (3.43)
Matrix reasoning	10.64 (2.71)	10.69 (2.75)	10.43 (3.86)	10.69 (2.13)
Picture span	10.64 (2.01)	10.88 (1.58)	9.57 (2.22)	10.92 (2.32)
Block design	10.81 (2.69)	10.81 (2.34)	11.86 (2.47)	10.23 (3.19)
D-KEFS: Trail Making Test				
TMT 2	10.87 (2.38)	11.69 (1.70)	11.20 (2.58)	9.83 (2.72)
TMT 4	9.55 (2.13)	9.69 (1.65)	9.80 (2.38)	9.27 (2.64)
D-KEFS: Color Word Interference Test				
CWI 1	8.14 (3.07)	9.31 (2.98)	7.20 (2.04)	7.18 (3.31)
CWI 2	9.21 (2.69)	10.62 (1.98)	7.40 (2.30)	8.36 (2.90)
CWI 3	8.79 (2.99)	9.31 (2.92)	8.80 (1.64)	8.18 (3.60)
CWI 4	8.93 (2.82)	9.31 (2.89)	9.20 (1.30)	8.36 (3.35)
The Conners Continuous Performance Test				
Omissions	49.29 (5.75)	50.24 (7.30)	47.97 (3.67)	49.05 (5.14)
Commissions	51.70 (12.24)	50.31 (13.43)	54.28 (7.74)	51.70 (13.86)
HitRT	53.21 (10.50)	53.53 (9.00)	53.01 (5.39)	52.98 (14.57)
ISI change	49.55 (7.92)	50.44 (6.75)	49.68 (5.75)	48.49 (10.48)
Childrens Auditory Verbal Learning Test				
Immediate memory span	52.39 (10.85)	55.00 (10.72)	41.83 (7.65)	53.85 (9.91)
Level of learning	54.24 (11.50)	54.65 (12.85)	51.29 (8.69)	55.31 (11.51)
Interference	53.39 (11.10)	56.29 (12.21)	44.67 (9.93)	53.62 (8.35)
Immediate recall	54.92 (9.88)	57.18 (6.77)	50.50 (10.27)	54.00 (12.71)

Delayed recall	55.97 (10.45) **	58.35 (8.68)	51.86 (11.32)	55.08 (12.01)
Beery-Buktenica Developmental Test of Visual-Motor Integration				
BVMI total	50.45 (10.24)	52.83 (7.87)	48.20 (9.35)	49.00 (13.23)
Grooved Pegboard				
Dominant Hand	50.85 (10.62)	53.84 (9.61)	51.71 (10.01)	46.00 (11.33)
Non-Dominant Hand	51.00 (11.03)	50.95 (12.26)	55.00 (9.55)	48.92 (10.02)

Note. Significant level is presented as: *p< .02, **p< .01, ***p< .001.

[a] Highest n is reported in each severity group as n varied on the different tests.

[b] Uncomplicated mild traumatic brain injury.

[c] Complicated mild traumatic brain injury.

[d] Moderate and severe traumatic brain injury.

3.2.2 WISC-IV discrepancies and differences between index and composite scores

Two thirds of the children (n=24) had clinically significant discrepancies between index scores, according to the WISC-IV manual (Wechsler, 2003). Of the 24 children with discrepancies, 8 had all of their indexes above score 90, resulting in an approximate 44 % discrepancy rate when these were excluded. When all discrepancies were included in the analysis, injury severity did not differ between the children with and without discrepancies. Interestingly, when those with all index scores above 90 were excluded, there was a significant difference with respect to AIS_{head} scores, as the children with discrepancies (mean rank = 23.19) had significantly more severe injuries than the children with no discrepancies (mean rank = 14.75), U = 85, z = -2.65, p= .008.

The variability in performance within the different domains was also evident when examining the WISC index discrepancies using paired sample t tests. The scores on the GAI were significantly higher (M = 104.69, SD = 13.01) than the full scale IQ (M = 100.50, SD = 9.07), a significant mean difference of 4.18, 95 % CI [2.16, 6.21], t(31) = 4.22, p< .001, d= .745. No significant difference was found between perceptual reasoning or verbal comprehension indexes, the two index scores which constitute the general ability composite score. The significant, albeit small difference between the FSIQ and GAI is likely a result of the relatively poorer patient performance on both the working memory and processing speed indexes. As such, VCI scores (M = 102.61, SD = 16.71) were higher than the WMI (M = 93.18, SD = 9.95), with a mean difference of 9.42, 95 % CI [3.81, 15.03], t(32) = 3,42, p= .002, d= .595. The largest effect was seen between PRI (M = 105.55, SD = 10.82) which was significantly higher than the WMI (M = 93.18, SD = 9.95), with a mean difference of 12.36, 95 % CI [7.83, 16.89], t(32) = 5.56, p< .001, d= .968. Furthermore, PRI performance (M = 105.17, SD = 12.15) was significantly higher compared to the PSI (M = 97.54, SD = 13.22), a mean difference of 7.62, 95 % CI [2.41, 12.84], t(34) = 2.97, p= .005, d= .502.

3.2.3 Neuropsychological impairment

Forty-five participants had sufficient neuropsychological data to be included in calculations of the dichotomized impairment variable. Of these, 18 were classified as having neuropsychological impairment (40 %). The majority of children (n=10) with neuropsychological impairment were in the m/sTBI group, resulting in a 62,5 % impairment

rate in this group. Of the remaining 8, 5 were in the cMTBI group, and 3 in the uMTBI group.

The relative occurrence of impairment was higher in the cMTBI group (45.5 %) compared to the uMTBI group (16.7 %), however not at a significant level. For the mild groups combined, there was a 27.6 % impairment rate, with 8 of 29 children being categorized as having impairment. Of the children in the impaired group, 72 % (13/18) had injury-related abnormalities on neuroimaging, however that was also the case for 37 % (10/27) of the non-impaired group. There was not a statistically significant association between radiological findings and neuropsychological impairment as assessed by Fisher's exact test, $p = .033$. No significant differences were found between the impaired versus the non-impaired group regarding GCS, AIS_{head}, ISS, age or parental education. However, the children who were classified as impaired (mean rank = 28.28) were found to have longer hospital stays than the non-impaired group (mean rank = 18.50, U= 130.00, z= -2.50, p< .012). Fifteen of the 18 children with neuropsychological impairment had also completed the WISC-IV. When looking at neuropsychological impairment and discrepancies together, 10 of the 15 with neuropsychological impairment had a significant discrepancy between index scores, whilst 5 did not. Of the 21 children classified as having no neuropsychological impairment, 14 still had significant index discrepancy, whereas only 7 did not. When excluding the cases with significant discrepancy, but with all indexes above score 90, only 6 of the non-impaired participants still had index discrepancies. Although a relatively higher number of impaired children appeared to have concomitant discrepancies, this difference did not reach a significant level.

3.2.4 Self- and informant reported cognitive and executive symptoms

Self- and informant reported measures of cognitive and executive symptoms are presented in Table 4. The BRIEF scores were largely within average range, with some variation between severities, albeit not at a significant level. Similar results were obtained for the HBI, where no significant differences were identified across severity groups.

When comparing the retrospective and concurrent parent reported HBI total symptoms, there was an increase from retrospectively reported premorbid symptom levels (M = 11.50, SD = 7.97), and the same measures post-injury (M = 17.30, SD = 9.08), with a mean difference of 5.80, 95 % CI [3.19, 8.40], t(29) = 4.56, p< .001, d=.832. In order to differentiate which of the two subscales accounted for this difference, a similar procedure

was conducted for the cognitive and somatic scales. The scores on the cognitive scale at 6 months post-injury were significantly higher (M = 12.03, SD = 6.69) than those reported retrospectively (M = 9.00, SD = 6.60), with a mean difference of 3.03, 95 % CI [1.03, 5.03], t(29) = 3.10, p= .004, d=.567. Similarly, the somatic scale (M = 5.27, SD = 4.25) was also increased post-injury (M = 2.50, SD = 2.94), with a mean difference of 2.76, 95 % CI [1.52, 4.00], t(29) = 4.56, p< .001, d= .833.

Table 4

Self-reported measures of cognitive and executive symptoms

	Total sample Mean (SD)	uMTBI[a] Mean (SD)	cMTBI[b] Mean (SD)	m/sTBI[c] Mean (SD)
Health and Behavior Inventory				
Parent report retrospective	(n=33)	(n=14)	(n=8)	(n=11)
Cognitive	8.91 (6.39)	9.93 (5.78)	7.50 (7.15)	8.64 (6.96)
Somatic	2.64 (3.21)	2.64 (3.22)	3.63 (4.43)	1.91 (2.11)
Total	11.55 (7.85)	12.57 (7.68)	11.13 (9.06)	10.55 (7.77)
Parent report 6m	(n=33)	(n=12)	(n=8)	(n=13)
Cognitive	11.24 (6.88)	10.83 (6.65)	13.50 (8.19)	10.23 (6.48)
Somatic	5.03 (4.18)	3.83 (2.98)	7.38 (4.47)	4.69 (4.66)
Total	16.27 (9.35)	14.67 (8.92)	20.88 (9.96)	14.92 (9.14)
Self-report 6m	(n=35)	(n=15)	(n=7)	(n=13)
Cognitive	8.62 (5.54)	6.00 (5.30)	10.71 (5.46)	10.53 (4.90)
Somatic	4.09 (4.08)	3.60 (2.82)	5.14 (3.71)	4.08 (5.48)
Total	12.71 (8.11)	9.60 (6.66)	15.86 (8.74)	14.62 (8.65)
Behavior Rating Inventory of Executive Function				
Parent reported 6m	(n=36)	(n=15)	(n=10)	(n=11)
BRI	46.81 (11.12)	43.20 (10.68)	51.60 (10.85)	47.36 (11.16)
MI	48.59 (9.94)	46.20 (8.82)	53.37 (11.95)	48.36 (9.51)
GEC	48.15 (10.23)	45.67 (8.92)	53.25 (12.17)	47.82 (9.99)
Self-report 6m	(n=21)	(n=10)	(n=4)	(n=7)
BRI	44.76 (9.63)	44.40 (10.53)	46.75 (5.56)	44.14 (11.14)
MI	46.71 (10.46)	43.80 (9.23)	51.50 (9.67)	48.14 (12.58)
GEC	45.52 (10.00)	43.60 (9.33)	49.25 (7.63)	46.14 (12.56)

Note. Significant results from paired sample t-tests are described in the text above and are not highlighted in this table specifically.

[a] Uncomplicated mild traumatic brain injury.

[b] Complicated mild traumatic brain injury.

[c] Moderate and severe traumatic brain injury.

3.3 Psychosocial outcome

Psychosocial questionnaire data are presented in Table 5 with the exception of PedsQL scores, which can be found in the appendix (Table 7), as no significant findings were revealed. Overall, symptoms reported were low to average for all the psychosocial questionnaires, and there were no significant differences between severity groups. FAD was largely below clinical cut off, indicating normal self-reported family functioning. PedsQL scores were comparable to those reported in other studies of children with acquired brain injury (Ilmer et al., 2016), where quality of life was overall reported to be good, with these scores being comparable to the healthy reference population. The parent reported SDQ scales measuring peer problems and emotional difficulties had the highest number of borderline and abnormal scores as defined by scoring criteria, with eight scores exceeding normal range on each of these scales. Interestingly, a similar pattern was not evident on self-reports, where hyperactivity was the scale with the highest number of borderline/abnormal scores, with only 3 participants within this range.

Table 5

Psychosocial measures

	Total sample Mean (SD)	uMTBI[a] Mean (SD)	cMTBI[b] Mean (SD)	m/sTBI[c] Mean (SD)
Strength and Difficulties Questionnaire				
Parent report	(n=43)	(n=16)	(n=11)	(n=16)
Total difficulties	7.23 (5.31)	7.25 (5.85)	8.55 (6.71)	6.31 (3.55)
Internalizing	2.98 (2.93)	2.88 (2.57)	3.91 (4.25)	2.44 (2.09)
Externalizing	3.93 (2.61)	3.75 (2.74)	4.64 (3.29)	3.63 (1.96)
Emotional problems	1.77 (2.15)	1.81 (1.97)	2.64 (3.07)	1.13 (1.36)
Conduct problems	1.07 (1.03)	1.00 (0.96)	1.36 (1.12)	0.94 (1.06)
Hyperactivity problems	3.00 (1.96)	2.75 (2.04)	3.27 (2.41)	3.06 (1.61)
Peer problems	1.47 (1.94)	1.69 (2.52)	1.27 (1.48)	1.38 (1.62)
Prosocial	8.21 (2.03)	8.31 (1.62)	8.18 (1.66)	8.13 (2.65)
Self-report	(n=23)	(n=10)	(n=4)	(n=9)
Total difficulties	6.26 (4.08)	6.90 (3.51)	9.00 (5.77)	4.33 (3.31)
Internalizing	2.39 (2.48)	2.90 (2.72)	3.25 (3.40)	1.44 (1.59)
Externalizing	3.96 (2.54)	4.00 (2.05)	5.75 (2.87)	3.11 (2.75)
Emotional problems	1.87 (1.93)	2.30 (2.26)	2.00 (2.16)	1.33 (1.50)
Conduct problems	1.00 (1.08)	1.00 (1.05)	1.75 (0.95)	0.67 (1.11)
Hyperactivity problems	3.13 (1.91)	3.00 (1.63)	4.00 (2.16)	2.89 (2.20)
Peer problems	0.61 (0.94)	0.60 (0.96)	1.25 (1.50)	0.33 (0.50)
Prosocial	8.39 (2.23)	8.70 (1.49)	8.50 (1.29)	8.00 (3.20)
General Family Function	(n=40)	(n=16)	(n=11)	(n=13)
Average score	1.40 (0.27)	1.42 (0.23)	1.30 (0.17)	1.49 (0.36)

Note. No significant differences were present between severity group

[a]Uncomplicated mild traumatic brain injury.

[b] Complicated mild traumatic brain injury.

[c] Moderate and severe traumatic brain injury.

3.4 Correlational analysis

Correlational tables can be found in the appendix (see Table 8, Table 9, and Table 10). All significant correlations are presented in the following sections.

3.4.1 Associations: Neuropsychological outcome

The impact of age at time of injury displayed few associations, with age being negatively associated with CWI 1 (r_s = -.539, p= .003), and BVMI (r_s = -.376, p= .014), indicating higher scores for the younger children. Family functioning was not significantly correlated with any of the neuropsychological measures, however, parental education length was associated with better performance on WISC-IV verbal comprehension index (r =.442, p= .008).

GCS was not significantly correlated with any neuropsychological measures, whereas the AIS_{head} scores were negatively associated with CWI 1 (r_s = -.439, p= .017), indicating that the children with more severe head injuries performed more poorly on this test. Similarly, more severe trauma as indexed by ISS, was negatively associated with performance on the CWI 1 (r_s = -.536, p= .001), BVMI (r_s = -.372, p= .015), and GP dominant hand (r_s = -.481, p= .002). Number of days in hospital correlated significantly with CWI 1 (r_s = -.447, p= .017) and GP Dominant hand (r_s = -.418, p= .009), suggesting lower performance on these measures for the children who required longer hospitalization.

3.4.2 Associations: Self-reported cognitive and executive symptoms

Unsurprisingly, there was a correspondence between parent-reported HBI retrospectively reported symptom levels and those reported post-injury. Pre-injury levels of cognitive symptoms were positively associated with post-injury levels of both cognitive symptoms (r =.677, p< .001) and total symptom score (r_s =.559, p= .001), but not somatic symptoms. Similarly, retrospectively reported somatic symptoms were positively associated with all three parent-reported HBI scales at 6 months: cognitive (r_s =.456, p= .011), somatic (r_s =701, p< .001), and total symptom levels (r_s =.659, p< .001). In summary, parent ratings were consistent pre- and post-injury, however, no associations were evident between parents' evaluations of premorbid symptoms and children's self-reported symptoms post-injury.

Both the general executive composite and the metacognitive index of the BRIEF were positively associated with parent rated HBI premorbid cognitive symptoms, with this link proving moderate for the GEC (r_s =.463, p= .010), and fairly strong for the MI (r =.536, p=

.002), both reported by parents. In a similar fashion, pre-injury parent-reported HBI somatic symptoms were positively associated with all of the parent-reported BRIEF composite and index scores: GEC (r_s =.488, p= .006), BRI (r_s =.438, p=.015), and MI (r_s =.515, p= .004). Of note, very few of the children had scores exceeding the BRIEF manual threshold of clinical significance, similarly, the premorbid levels of HBI cognitive symptoms as reported by parents were not particularly high compared to other studies (Fay et al., 2010). Nonetheless, these associations point in the direction of premorbid difficulties being associated with higher levels of executive symptoms 6 months after TBI.

3.4.3 Associations: Psychosocial outcome

Contrary to expectations, the GCS and AIS_{head} were not significantly associated with any of the psychosocial outcome measures. However, ISS was associated with parent-reported SDQ total difficulties score (r_s = .362, p= .017) and peer problems (r_s = .372, p= .014), implying a higher degree of problems reported for the more severely injured children. Family functioning was associated with parent-rating of SDQ externalizing problems (r_s = .442, p= .005) indicating higher rates of externalizing problems in families with poorer family function. The children with longer hospital stays were also reported by their parents to have more peer problems as illustrated by the positive association with this SDQ scale (r_s = .375, p= .013).

There was a link between premorbid cognitive symptoms and subsequent hyperactivity post TBI, as premorbid cognitive symptoms were positively associated with the parent-reported SDQ hyperactivity scale (r_s =.439, p= .011). Furthermore, premorbid somatic symptoms were positively associated with SDQ total difficulties score (r_s =.416, p= .016), and conduct problems (r_s =.460, p= .007), both of which parent-reported. Similar associations were not evident for the SDQ self-report, where ratings of pre-injury somatic symptoms correlated positively with self-reports of both internalizing problems (r_s = .558, p= .016) and emotional problems (r_s =.577, p= .012).

4 Discussion

Considering how few Norwegian studies have addressed outcomes after pediatric TBI, the first aim was to give a detailed description of the sample with respect to demographic and injury related characteristics. The second aim was to provide a description of how children in this sample performed 6 months post-injury, both on neuropsychological and psychosocial outcome measures, as well as to explore whether the aforementioned demographic and injury related variables were associated with outcomes.

Gender distribution, cause of injury, or length of parental education did not vary according to TBI severity. The sample included a severe spectrum of injuries, with a substantial amount of the mild injuries having intracranial abnormalities on neuroimaging. For the overall sample, working memory was significantly lower compared to the standardization sample. Although the remaining neuropsychological tests appeared to be largely within average, nearly half the sample had clinically significant WISC-IV index discrepancies, with this being associated with head injury severity. Examining a neuropsychological impairment variable resulted in a 40 % impairment rate.

The sample reported few self-reported cognitive and psychosocial symptoms at follow-up. There was however a significant difference in post-concussive symptoms between retrospective and post-injury parental scores. Factors related to injury severity, family function, and child preinjury symptom levels were all shown to be associated with post-injury reported PCS, everyday life measures of executive function, as well as aspects of behavioral and emotional functioning.

4.1 Demographic and injury characteristics

Consistent with existing knowledge, a higher number of males than females were found in this sample (Bruns & Hauser, 2003). Cause of injury appeared to adhere to familiar patterns, as falls were the most prevalent cause of injury for the youngest children, with an increased occurrence of sports injuries with higher age (Crowe et al., 2009).

In terms of severity distribution this sample was skewed towards the severe end of the spectrum, with only 64 % of the injuries classified as mild, 22 % moderate and 14 % severe. Severity estimates vary substantially depending on how studies classify severity, instruments and trauma measures utilized, and characteristics of the recruited patient population (Bruns & Hauser, 2003), with some studies reporting 90 % mild injuries, and 8 % and 3 % moderate

and severe, respectively (Catroppa et al., 2016). Additionally, in the present study a relatively large proportion (34 %) of the injuries classified as mild based on GCS scores had positive radiological findings, constituting the cMTBI group. Although being clinically considered in need for a CT scan was a criterion for inclusion, other studies with similar criteria have reported a lower proportion of intracranial abnormalities detected in the GCS range 14-15 after pTBI (Kuppermann et al., 2009). However, estimates have been reported to be higher when including GCS ranges 13-15 (Lumba-Brown et al., 2018), with the latter being comparable to the range used in the present study.

The severity pattern seen here was not uniquely surprising, as children in need of a CT scan will be hospitalized in the Oslo region, thus leading to a bias toward including the more severe mTBI cases in hospitalized samples. The children included likely had features or circumstances surrounding the trauma, or symptoms at the site of injury that were of a sufficient severity to warrant the child being brought to hospital. Furthermore, the relatively low number of mild injuries may in part be attributable to bias in the recruitment process. This is supported by the fact that the missed cases displayed lower ISS and AIS_{head} than those included in the sample (H. M. Dahl, personal communication, September 4, 2018). As these children displayed less severe injuries, they were also likely to be discharged early, probably resulting in lower level of inclusion. Importantly, this does not negate the effects seen in the complicated mild group, as the uncomplicated mild injuries included displayed little to no impairment overall.

AIS_{head} scores being higher in the complicated mild group was not unexpected, as positive radiological findings were part of the definition of cMTBI, and higher AIS_{head} scores reflect intracranial pathology. However, the fact that AIS_{head} scores did not differ significantly between the complicated mild group and the moderate and severe group indicated a fairly severe spectrum of injuries in the cMTBI group, which was not reflected in the GCS score. Although the complicated mild TBI construct is still somewhat contentious, there is increasing recognition of GCS being inadequate for accurately identifying mTBI, with a risk of injury severity being higher than what is reflected in the GCS score (Kennedy et al., 2006). In a similar fashion, for TBI sustained in early childhood, GCS score has been reported to be an unreliable indicator of severity, as a substantial number of children displayed radiological markers of TBI, despite GCS being within the mild range (Heather et al., 2013). This is in accordance with the findings of this study, which demonstrates how stratifying injury groups based solely on the GCS can result in a too wide spectrum of

injuries falling within the mild classification, and thus potentially lead to undertreatment of long-term functional disability.

4.2 Neuropsychological outcome

At a superficial level, this sample appeared to be relatively unimpaired in most domains assessed, as even the lowest mean scores did not deviate more than one standard deviation from the normative sample. However, upon closer examination some notable findings emerged. Within the domain of working memory, the pTBI survivors obtained significantly lower scores than expected for their age when compared with the standardization sample. Working memory deficits are reasonable to expect after TBI and has also been tied to injury severity (Levin et al., 2004). Other reports utilizing comparable methodology to the present study found little to no impairment on the WMI, with a relatively larger impairment of the processing speed index (Allen, Thaler, Donohue, & Mayfield, 2010; Donders & Janke, 2008; Rackley, Allen, Fuhrman, & Mayfield, 2012). The current study did not show evidence of significantly affected processing speed. The observed difficulties could be related to impairment in aspects of attention, as attention is required for the majority of working memory tasks (Lezak et al., 2012), and attentional difficulties are commonly seen after pTBI (Yeates et al., 2005). However, noteworthy findings were not present within other aspects of the attentional domain in the current study.

4.2.1 Identifying those at risk: Unveiling the hidden deficits

Neuropsychological impairment

One of the main issues encountered when analyzing these data was the large variability, which obscured the relative deficits. By looking at the defined neuropsychological impairment variable, neuropsychological impairment was present for more than a third (40 %) of the children in the sample. A similar pattern was reported in a 10-year follow-up study, where intellectual functioning displayed average results at the group level, yet the establishment of impairment ratings yielding considerably higher impairment rates (Anderson, Brown, et al., 2011). Interestingly, the relative impairment rate in this sample appeared to be higher in the cMTBI group as opposed to the uMTBI group, albeit not at a significant level.

Methodologically rigorous studies have demonstrated significant heterogeneity within the mTBI range (Satz et al., 1997). Children with mTBI and additional intracranial findings

have been demonstrated to display poorer performance within several cognitive domains, particularly on tasks with processing speed demands, when compared to children without intracranial lesions (Levin et al., 2008). A comparable pattern of heterogeneity was evident in this sample as the cMTBI group resembled the m/sTBI group, with the uMTBI group performing largely within average levels, which is in accordance with previous reports (Babikian et al., 2011; Papoutsis et al., 2014).

A frequently cited drawback with dichotomizing a continuous variable, is that variability is lost (Farrington & Loeber, 2000; Fedorov, Mannino, & Zhang, 2009). As such the "non-impaired" and "impaired" participants are considered distinct from each other, and all the "non-impaired" participants are considered alike. One might then miss the children with a) relative deficits b) multiple scores just above the threshold of the established cutoff. Similarly, there is also a risk of identifying some children as having impairment with only two low scores, which may have been a result of random performance variation. Although these limitations are relevant, they have been addressed in several ways; in order to reduce the risk of false positives, cut point was set at a minimum of two tests deviating at least -1,5 SD from the normative mean. Analyses were also performed on actual outcome data with the neuropsychological impairment variable acting as a supplementary analysis (Fedorov et al., 2009).

WISC-IV discrepancies

One unexpected finding was the high number of clinically significant WISC-IV index discrepancies, with two thirds of the sample being affected. A third of these children had a clinically significant discrepancy between average scores and very high scores, indicating a high premorbid level of functioning. Although scores were still within normal range, the large interpersonal variance may represent a relative, but not global fall in cognitive functioning that may go undetected if internal profile variation is not considered. The fact that full scale IQ was lower than the GAI was not uniquely surprising as GAI is less affected by working memory and processing speed (Rowe, Kingsley, & Thompson, 2010), and WMI was one of the affected domains in this sample. The PRI displayed significantly higher scores than the WMI, with nearly one SD separating the two, this difference yielding a large effect size. Taken together with the fact that the PRI has been described as one of the less susceptible domains after TBI (Allen et al., 2010), the present findings support the notion of relative deficits. As such, using the full-scale IQ score when large discrepancies are present

can consequently obscure the full extent of the child's cognitive functioning in TBI populations (Fiorello et al., 2007; Koriakin et al., 2013).

This study included multiple performance-based measures, enabling detection of relative deficits. However, assessments utilizing less comprehensive assessment methods may struggle to detect these effects. Significant impairment and need for assistance in academic settings may be required, even in the absence of global intellectual impairment (Koriakin et al., 2013), underscoring the importance of looking past composite scores and examining all the indexes in order to get a comprehensive and accurate understanding of the child's relative cognitive strengths and weaknesses when assessing and evaluating further rehabilitation and educational needs.

4.2.2 Cognitive sequelae: Challenges in identification and detection

Some of the neuropsychological impairments found in the current study were not immediately evident as demonstrated by the analysis of intra individual discrepancies, as well as the relatively high number of impairment in the sample overall. Mild impairments may go undetected in early childhood, but as the school system and society gradually require a higher degree of self-regulation and strategic thinking, deficits could become evident (Alloway, Gathercole, Kirkwood, & Elliott, 2009).

Assessing executive difficulties may represent an added challenge, due to the multifaceted nature of these functions and the many ways in which ED can be expressed (Royall et al., 2002). Executive dysfunction is a very common symptom after TBI (Stuss & Levine, 2002), therefore, the limited findings within this domain was somewhat surprising. Particular susceptibility for ED has been demonstrated at younger age at injury (Anderson et al., 2014), as well as with increasing severity (Sesma et al., 2008). Considering the many important roles EF play in the initiation, inhibition and monitoring of behavior, there is a risk that impairment within this domain could be misinterpreted as unrelated behavioral and social difficulties (Ganesalingam et al., 2007), and as such could be challenging for parents and teachers to correctly identify as TBI sequelae.

Neuropsychological assessment is conducted in an environment that is inherently organized and free from external distractors, in contrast to the demands of everyday life, and standardized testing may be at risk for overestimating the child's level of executive functioning (Sesma et al., 2008; Vriezen & Pigott, 2002). Consequently, difficulties can be

present in more complex and demanding environments, even in the absence of deficits on performance-based tests (Anderson, 2002).

In this study, the majority of executive measures were only administered to the children above age 7, and the three severity groups were small, which may contribute to the limited findings within this domain. Most standardized tests of EF have been developed for adults, and appropriate normative materials lack for certain age groups (Anderson, 1998). Furthermore, aspects of EF emerge and consolidate at different stages of development in a non-linear fashion, necessitating knowledge about the differential development of these skills when assessing EF in children. This is further complicated in clinical populations, where the evaluation of residual deficits must take into account the child's developmental trajectory, premorbid attainment of specific skills, in addition to the effects the TBI may have had (Anderson, 2002). Taken together, this highlights the need for long term monitoring of this group, as well as the importance of giving parents and teachers sufficient information in order to correctly understand these difficulties, should they emerge.

4.2.3 Association between demographic and injury-related variables and neuropsychological outcome

As previously illustrated, a multitude of factors exert an influence on outcomes after pediatric TBI. One commonly reported finding is that younger children tend to have less favorable outcomes over time (Catroppa et al., 2016). Few age effects were seen in this study, yet age at injury was negatively associated with CWI 1 and BVMI. Of note is that the CWI 1 is restricted to the ages above seven, which makes it difficult to interpret this finding from a vulnerability perspective. Additionally, this follow-up was conducted at six months post-injury, whereas deficits may not necessarily be evident till years later, when functions are expected to be fully developed (Giza & Prins, 2006; Ryan et al., 2016; Muscara et al., 2008).

Children from families with greater levels of dysfunction have been demonstrated to display a higher degree of difficulties within the domains of attention and executive function, regardless of injury severity. Furthermore, lower socioeconomic status has been associated with both long-term executive and attentional problems (Kurowski et al., 2011), highlighting the complexity and multifactorial nature of recovery. There was however little family dysfunction reported in our sample, and therefore, FAD showing no correlations with neuropsychological outcome was not unexpected.

The association between parental length of education and the verbal comprehension index was not a novel finding, as a link between parental education and children's academic achievement is well established (Dubow, Boxer, & Huesmann, 2009). It is further supported by other research demonstrating a positive association between length of parental education and the child's neurocognitive functioning (Schoenberg, Lange, & Saklofske, 2007). A limitation is that length of parental education was the sole indicator of socioeconomic status in the present study, however, parental education has been shown to exert a direct effect on subsequent child achievement, even when controlling for other socioeconomic factors such as occupation and income (Dubow, et al., 2009).

Overall, the ISS displayed more associations with neuropsychological outcome than the more specific TBI severity measures GCS and AIS_{head}. GCS displayed no associations, whereas AIS_{head} scores were negatively associated with CWI 1 performance. This was initially understood as head injury severity affecting tasks requiring psychomotor speed, however AIS_{head} did not display any significant correlations with other measures of speed, thus giving less support to this notion. Furthermore, the ISS was associated with CWI 1, BVMI as well as GP, all in a negative direction. Adhering to this pattern, both the CWI 1 and the GP dominant hand were negatively associated with longer hospital stays, which is unsurprising as it indicates that the more severe injuries that require longer hospital stays also result in lowered performance on some aspects of cognitive and motor functioning. Injury variables such as GCS, LOC, and intracranial lesions have repeatedly been linked with outcomes after pTBI (Prasad, Ewing-Cobbs, Swank, & Kramer, 2002). Therefore, finding few associations between TBI severity indices and neuropsychological outcome was contrary to expectations (Catroppa et al., 2008).

4.3 Self- and informant reported cognitive, executive and psychosocial outcome

There was a marked increase in post-concussive symptoms (PCS) between the retrospective and post-injury evaluations, with this difference yielding a large effect size regarding total symptom load. Children with mTBI have been found to display both cognitive and somatic symptoms compared to other injury control groups, with symptom load being linked to severity as indicated by LOC and radiological findings (Taylor et al., 2010; Yeates et al., 2012). Increased PCS has also been shown in a mTBI sample compared to an orthopedic control group, even when controlling for preexisting symptom levels (Taylor et al., 2010). In

light of this, it seems unlikely that the reported PCS at six months follow-up are a result of either a general injury effect or symptom levels predating the TBI, but firm conclusions would require a control group. Furthermore, elevated PCS have been linked to an increased risk of functional impairment as well as reduced quality of life (Yeates et al., 2012), something that was not apparent in this sample, as the majority of PedsQL scores were within normal range.

Executive difficulties in everyday life are frequently seen after pTBI, and significant deficits on the BRIEF measure have been demonstrated 5 years post-injury, for parental and self-reported measures, with a higher degree of problems reported with increasing injury severity (Mangeot, Armstrong, Colvin, Yeates, & Taylor, 2002). Comparable findings were not evident in the current study, as none of the BRIEF index scores were above clinical cut off, nor did they differ significantly between severity groups. The highest levels of BRIEF symptoms were present in the cMTBI group for all three indexes, as the parent reported GEC score differed by almost 1 SD from the uMTBI group. Considering the rate of neuropsychological impairment as well as WMI being the most affected index, higher levels of BRIEF difficulties was expected. However, given research reporting little correspondence between parent reports and performance based executive functioning (Vriezen & Pigott, 2002), this was not uniquely surprising.

Norwegian normative data are not available for the BRIEF, but lower average scores for healthy controls have been demonstrated in some Norwegian studies of adults with TBI when compared to the U.S normative sample (Løvstad et al., 2016). A similar tendency has been reported on the parent and teacher reports in a Norwegian pediatric sample, with some scales being marginally lower than those reported in U.S norms. However, U.S norms have been validated for use in Norwegian populations, with the conclusion that the indexes are valid and reliable (Fallmyr & Egeland, 2011).

Premorbid individual and family factors have been shown to influence psychosocial outcome measures to a higher degree than injury related factors (Anderson, Catroppa, Haritou, Morse, & Rosenfeld, 2005). This pTBI sample appeared to have few exacerbating factors as the FAD scores gave no indications of dysfunctional home environments, in combination with the fact that high average education level indicated resourceful families (Ryan et al., 2016; Chapman et al., 2010; Yeates et al., 1997). Furthermore, the retrospective evaluations yielded little reason to suspect a significant amount of difficulties predating the injury, suggesting low levels of both pre- and co-morbid family problems. As such it should perhaps not be surprising to find little psychosocial difficulties post-injury.

Time of assessment may have affected the results, as other reports have found that difficulties may emerge over time. A pattern of gradual deterioration, or lack of expected development, may extend past the acute recovery time, where lingering problems within psychosocial and behavioral domains may emerge, as physical and cognitive recovery stabilizes and increasing attention is focused toward the child readjusting (Anderson, Catroppa, Haritou, et al., 2005). Children may be less exposed to taxing environments in the first months after recovery, and caretakers or other adults in the child's life may have developed compensatory strategies that mask these difficulties shortly after injury (Gioia & Isquith, 2004). The limited evidence for post-injury cognitive or psychosocial symptoms might also be an expression of these symptoms being too mild to detect on the measures employed (Fay et al., 2009), as self- and parent-reported difficulties are frequently cited in the pTBI literature (eg. Anderson, Brown, Newitt, & Hoile, 2009; Li & Liu, 2013; McKinlay et al., 2002).

4.3.1 Associations: Psychosocial outcome, self-reported cognitive and executive symptoms

None of the self or parent-reported outcome measures were associated with the trauma scores GCS or AIS_{head}. In contrast, the ISS was positively associated with total difficulties and peer problems on the parent reported SDQ, and length of hospitalization was positively associated with parent reported SDQ peer problems. A previous paper reported average symptom levels of emotional distress and peer-problems in a sample with pTBI to be comparable to a population of children receiving mental health care services (Tonks, Yates, Williams, Frampton, & Slater, 2010). Although similarly elevated mean scores were not evident here, an increased risk of peer-problems and emotional difficulties after head injury in childhood has repeatedly been demonstrated (Prigatano & Gupta, 2006; Tonks et al., 2010). Further, the number of close friends has been shown to be lower for the more severe injuries (Prigatano & Gupta, 2006), giving some support to the association described above.

Parental education level, which here acts as a proxy of socioeconomic status, showed no significant associations with psychosocial outcome measures. Family function was however positively correlated with externalizing difficulties on the parent-reported SDQ. The directionality of this association is complicated to interpret, as there likely is a bidirectional relationship between family function and child behavior. Child behavioral problems may be the cause of increased family distress and higher FAD scores, or the child's behavior may be

a result of, or exacerbated by, a dysfunctional family environment (Schwartz et al., 2003). Furthermore, there is also a chance that both the family function and the child's behavior predated the injury, as preinjury factors have been shown to influence behavior and family function post-injury (Anderson, Catroppa, Haritou et al., 2005). The current dataset does not allow conclusion regarding causality, but the association between family functioning and child externalizing behavior problems is nonetheless interesting.

The fact that pre-injury ratings of both cognitive and somatic PCS were strongly associated with parent reported BRIEF measures at follow-up support the notion that preexisting difficulties had an impact on executive symptoms post-injury in this sample. Furthermore, parentally reported premorbid levels of cognitive symptoms also correlated with parent reported hyperactivity problems on the SDQ. Similar associations were not evident for self-report data, however, parental rated somatic symptoms pre-injury correlated strongly with child reported emotional problems. These results are congruent with previously established research of the central contribution that preinjury functioning and vulnerabilities have on subsequent behavior and outcomes after pTBI (Anderson et al., 2001; Anderson, Catroppa, Haritou et al., 2005).

4.4 Study strengths and limitations

This is one of the few Norwegian studies that has explored the impact of pTBI on both neuropsychological and psychosocial outcomes. Standardized testing was used, and as this was the first assessment time-point, practice effects were not a confounding variable. Several pre-injury factors were taken into account, yet elaborate information regarding socioeconomic status, ethnicity, as well as premorbid levels of functioning would have been preferable, and the lack thereof can be considered a limitation. Furthermore, the trauma scoring systems were not specifically tailored to the pediatric population, due to the sample being included in a larger European study with a main focus on adults with TBI.

Psychosocial outcomes were based on questionnaire measures, with the majority of these being parentally reported. Using questionnaire measures is not a limitation in and of itself, yet these indirect measures have some inherent challenges. Response styles, or the tendency towards responding in a certain way, is rarely controlled for, and may represent a source of error (Vaerenbergh & Thomas, 2013). There is a chance that some measures are influenced by parents not wanting to portray their child or their family as dysfunctional. Considering the complexity of the child’s behavior and function, and how it interacts with the

environment, the study might have captured a more comprehensive picture of the child's functioning if other arenas were also quantified by, for example, teacher reports, information about special needs and academic achievement.

The current study lacked a control group. This is not uncommon, even though an age-matched orthopedic/other injury control group may have further delineated whether the results were attributable to a general injury effect, as previously reported (Babikian et al., 2009). As no control group was included, some of the comparisons were instead performed with scores directly derived from the standardization sample. As most clinicians utilize these same scores in daily practice when evaluating TBI patients, this methodology is considered relevant and informative for clinical practice.

No formal corrections were made for multiple testing, as utilizing Bonferroni corrections is not inconsequential. The intention with Bonferroni corrections is to lower the risk of type I errors, and as type I and type II errors are inversely related, this will inflate type II errors (Rothman, 1990). There is some evidence for the utility of such corrections when many tests are performed on the same subsample, similar to what was done here. However, in considerations of the aforementioned limitations it has been proposed that describing the nature of the analysis and why it was done, as well as discussing possible interpretations of results is preferable to formal corrections (Perneger, 1998). Given the current sample size, the main pitfall is related to type II errors, therefore, Bonferroni corrections were considered too conservative in this explorative study, and it was decided to use a conservative significance level of $p < .02$. Caution is warranted when interpreting findings, particularly null-findings, as lack of statistical significance does not necessarily signify no real effect.

Small sample size as well as uneven groups, reduced the statistical power and limited the available analyses. The small number of severe injuries was the main reason to collapse the moderate and severe injuries into a single group, which in turn limits the findings related to the most severe injuries. However, similar studies have comparable sample size (e.g. Catroppa et al., 2008; Muscara et al., 2008), and collapsing severity groups is also quite common (e.g. Muscara et al., 2008). Variability in age groups, and lack of uniform measures caused difficulties in comparing across the total sample, which resulted in variable number of participants on different neuropsychological tests. Due to the broad age range included, few of the measures used encompassed the entire age range, causing challenges in comparing the different groups on similar metrics.

4.5 Implications and future directions

As demonstrated, significant intraindividual variability in test performance was common. It is unlikely that all of the participants in the present study would have been offered a neuropsychological assessment outside of a research setting, which could result in many difficulties going undetected. Furthermore, if difficulties become present at a later point in time they can be misinterpreted as social or behavioral difficulties (Ganesalingam et al., 2007). Therefore, providing an understanding of the unique vulnerabilities of the child after TBI might have important implications for the child's developmental trajectory. Interpreting child behavior within this framework is important, as an accurate understanding leads to relevant interventions, as well as avoiding children unduly being labeled as disruptive, lazy or problematic.

Children sustaining a mTBI with few immediate symptoms might have an added risk, as they are rarely referred for formal neuropsychological assessment and their parents and teachers may have received limited information about the long-term sequelae of childhood TBI. In fact, unmet information needs have been reported for 70 % of parents, regardless of TBI severity (Hawley, 2003), and numerous studies have demonstrated that children with TBI often do not receive the therapeutic interventions they need post-injury (Yeates et al., 1997; Kirk, Fallon, Fraser, Robinson, & Vassallo, 2015; Slomine et al., 2006; Greenspan, & MacKenzie, 2000). Therefore, this thesis argues for the importance of educating parents and health care providers in order to ensure early identification of developmental deviations and TBI-related difficulties. It also requires teachers and special educational services to be aware of relative deficits, how to detect them, and how this can be an indicator TBI sequelae.

A substantial proportion of the mild head injuries had findings on neuroimaging, which underscores the need for acute hospitals to be aware of mild head injuries with radiological findings. Disruptions to white matter integrity has been shown to occur in all severity groups in pTBI (Tong et al., 2004), and standard CT scans commonly fail to detect this type of pathology (Gentry, Godersky, Thompson, & Dunn, 1988). As such, some patients can have negative CT findings, whilst MRI scans show evidence of DAI (Mittl et al., 1994). Studies utilizing more advanced neuroimaging protocols such as susceptibility-weighted imaging have reported consistent improvements in detecting hemorrhagic lesions, and as a result have suggested that the presence and extent of DAI is severely underestimated for survivors of pTBI (Tong et al., 2003).

Established Scandinavian treatment guidelines suggest CT only for mild GCS ranges 14-15 if at least one of the following are present a) focal neurological signs, b) spasms/seizures, and/or c) clinical signs of skull base or impression fracture (Astrand, Rosenlund, & Undén, 2016). Furthermore, these guidelines only state CT as an acute diagnostic approach, whereas MRIs are performed if the clinician considers it to be indicated (Ingebrigtsen, Romner, & Kock-Jensen, 2000). Therefore, patients with pathologies that are not immediately evident on CT, and without additional clinical signs, may still have disruptions that go undetected.

The aforementioned guidelines only address acute treatment and diagnostic efforts, whereas this thesis also underscores the need for monitoring this subset of patients over time, due to the risk of both neuropsychological and psychosocial problems. The current findings highlight the importance of future studies integrating radiological markers when quantifying severity, as they appear to be more closely associated with outcomes (Heather et al., 2013).

The impact of pTBI is extensively researched and documented, yet there is less research on long-term outcomes (Ryan et al., 2016). Studies following up participants into early adulthood are better able to detect deficits as they are evaluated at a time when affected skills are expected to have fully emerged, making findings less likely to be a result of developmental delays. Future research should ideally focus on long-term follow-up of this patient group, especially the mild complicated injuries, in order to investigate recovery patterns and potential deficits emerging over time.

4.6 Conclusion

The current follow-up study describes a sample consisting of a fairly severe spectrum of injuries, as a substantial proportion of the mild TBIs had intracranial lesions. The results suggest working memory as an area of particular vulnerability after pediatric traumatic brain injury. Furthermore, the subset of patients with complicated mTBI appeared to display less favorable outcomes and had comparable results to that of the moderate/severe group, which is similar to previous research on the topic of cMTBI (Borgaro et al., 2003). The present findings extend upon existing research by demonstrating the pervasive heterogeneity that characterizes this patient population (Levin et al., 2008), whilst at the same time illustrating how deficits may be elusive to detect at first glance, and that relative strengths may obscure actual impairment.

This study shows that despite the relatively high rate of intraindividual variation and neuropsychological impairment, the sample experienced few troublesome symptoms within the psychosocial domain. Of note was the marked increase in post-concussive symptoms relative to pre-injury levels. Injury severity, family function, and child preinjury symptom levels all seemed to impact on post-injury levels of PCS, everyday life measures of EF, as well as aspects of behavioral and emotional functioning, which supports the notion that premorbid vulnerability impacts upon subsequent functioning post-injury.

In summary, this study demonstrates how stratifying severity groups based exclusively on GCS might result in poor outcome prediction, as a too wide spectrum of injuries falls within the mild classification, integrating radiological findings is important when identifying children at risk for persistent symptoms after pTBI. Patients with significant intra-individual performance variation may have an added risk of having said deficits go undetected, which consequently can lead to these children not receiving treatment. Therefore, this thesis wishes to place an emphasis on the continued clinical monitoring of individuals sustaining a traumatic brain injury in childhood and adolescence.

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Appendix

Table 7

Pediatric Quality of Life Inventory (PedsQL)

Age group	Subdomain	n	Self-report Mean (SD)	n	Parent report Mean (SD)
2-4	Total score			5	79,65 (17,14)
	Physical health				79,86 (13,14)
	Psychosocial health				79,55 (20,54)
	Emotional functioning				72,00 (24,13)
	Social functioning				85,00 (16,58)
	School functioning				81,00 (22,36)
5-7	Total score	10	74,12 (13,03)	10	76,20 (15,09)
	Physical health		87,49 (10,61)		86,87 (7,62)
	Psychosocial health		69,66 (15,19)		72,75 (17,81)
	Emotional functioning		72,00 (22,50)		64,00 (26,33)
	Social functioning		82,00 (18,73)		85,50 (10,12)
	School functioning	8	66,25 (13,02)	9	76,38 (13,86)
8-12	Total score	12	86,40 (6,09)	11	87,08 (8,86)
	Physical health		91,88 (8,35)		94,60 (6,10)
	Psychosocial health		84,58 (6,89)		84,58 (10,51)
	Emotional functioning		81,67 (14,66)		82,38 (20,64)
	Social functioning		93,75 (6,44)		94,09 (7,68)
	School functioning		78,33 (11,34)		77,27 (13,29)
13-18	Total score	15	86,51 (8,45)	12	81,80 (12,78)
	Physical health		90,39 (12,59)		87,50 (10,65)
	Psychosocial health		85,22 (8,63)		79,90 (16,07)
	Emotional functioning		86,33 (13,15)		83,75 (16,25)
	Social functioning		95,33 (6,39)		86,25 (24,32)
	School functioning		74,00 (15,72)		69,72 (22,14)

Table 8

Neuropsychological outcome and its correlates

	Educational length parents	Age at injury	Family functioning	HBI retrospect Cognitive	HBI retrospect Somatic	GCS	AIS	ISS	Days in hospital
Wechsler Intelligence Scale for Children									
Full scale IQ	.296 $_p$	-.018	-.041 $_p$	.008	-.046	.142	-.275	-.319	-.350
General ability index	.371 $_p$	.181	-.124 $_p$	.023	-.076	.116	-.085	-.304	-.275
Verbal comprehension index	.442** $_p$	.256	.007 $_p$	-.022	-.140	.142	-.109	-.263	-.296
Perceptual reasoning index	.062	-.001	-.259	.103	.053	.100	-.055	-.248	-.176
Working memory index	.366 $_p$	-.067	-.150 $_p$	-.074	-.164	.011	-.230	-.136	-.343
Processing speed index	.030 $_p$	-.170	.021 $_p$	.139	.187	-.161	-.036	-.299	-.144
D-KEFS: Trail Making Test									
TMT 2	.178	-.306	-.020	.257	.061	.293	-.377	-.298	-.345
TMT 4	-.366	-.110	-.067	.275	.214	.085	-.206	-.133	.005
D-KEFS: Color Word Interference Test									
CWI 1	.083	-.539**	.209	.271	.223	.227	-.439*	-.563**	-.447*
CWI 2	.193 $_p$	-.023	.278 $_p$	.173 $_p$	-.149	.099	-.274	-.314	-.421
CWI 3	-.070	-.203	.318	.194	.413	-.041	-.305	-.389	-.398
CWI 4	-.140 $_p$	-.237	.167 $_p$	-.119 $_p$	.114	.107	-.201	-.265	-.211
The Conners Continuous Performance Test									
Omissions	.196 $_p$	-.177	.207 $_p$	.134 $_p$	.019	-.001	-.293	-.183	-.424
Commissions	-.182 $_p$	-.214	-.040 $_p$	.113 $_p$	.187	.117	-.340	-.134	-.266
HitRT	.329 $_p$	.273	.173 $_p$	-.330 $_p$	-.241	.029	.328	.075	.150
ISI change	-.005 $_p$	-.324	.350 $_p$	-.340 $_p$	-.104	.110	-.135	-.101	-.031

Childrens Auditory Verbal Learning Test									
Immediate memory span	.176 $_p$	.252	.199 $_p$	.351 $_p$	.030	-.199	-.134	-.102	-.355
Level of learning	.084 $_p$	.194	-.253 $_p$	.214 $_p$	.160	-.013	.003	-.107	-.309
Immediate recall	-.016 $_p$	.102	-.014 $_p$	.226 $_p$	.296	.003	-.093	-.105	-.252
Delayed recall	.040 $_p$	.186	-.210 $_p$	.238 $_p$	.291	.127	-.070	-.163	-.283
Beery-Buktenica Developmental Test of Visual-Motor Integration									
BVMI total	.216 $_p$	-.376*	.077 $_p$	.280 $_p$	-.038	.081	-.307	-.372*	-.300
Grooved Pegboard									
Dominant hand	.317 $_p$	.160	.028 $_p$	.084 $_p$	.058	.227	-.276	-.481**	-.418**
Non-dominant hand	.171	.188	.045	.127	-.041	-.010	.044	-.239	-.117

Note: * p <.02, ** p <.01, ***p <.001. Pearson correlations are denoted with $_p$, Spearman correlations remain unmarked.

Table 9

Self-reported cognitive and executive symptoms and their correlates

	Educational length parents	Age at injury	Family functioning	HBI retrospect Cognitive	HBI retrospect Somatic	GCS	AIS	ISS	Days in hospital
BRIEF[a]									
Parent report 6m									
BRI	-.099	.015	.269	.389	.438*	-.225	.215	.221	.199
MI	-.087 $_p$	-.006	.092 $_p$	.536** $_p$	.515**	-.043	.089	.101	.074
GEC	.115	.001	.256	.463**	.488**	-.157	.133	.145	.133
Self-report 6m									
BRI	.234 $_p$	.171	-.060 $_p$	.419 $_p$	.359	-.104	.081	.071	-.185
MI	.163 $_p$	.120	-.173 $_p$	.225 $_p$	.304	-.079	-.070	.116	-.143
GEC	.229 $_p$	.174	-.122 $_p$	.342 $_p$	.321	-.051	-.049	.083	-.208
HBI[b]									
Parent report 6m									
Cognitive	-.065 $_p$	-.208	.137 $_p$	.677*** $_p$	.456*	.066	-.067	-.035	.031
Somatic	-.327	.100	.113	.176	.701***	.054	-.016	.052	.096
Total	-.223 $_p$	-.116	.099 $_p$	.559**	.659***	.081	-.061	-.021	.038
Self-report 6m									
Cognitive	-.206	-.194	-.480	.255	.025	-.228	.231	.125	.012
Somatic	-.146	.077	-.148	.099	.276	.054	-.129	-.072	-.245
Total	-.185 $_p$	-.088	-.288 $_p$	.183 $_p$	.173	-.171	.132	.050	-.072

Note: * p <.02, ** p <.01, ***p <.001. Pearson correlations are denoted with $_p$, Spearman correlations remain unmarked.

[a] Behavior Rating Inventory of Executive Function

[b] Health and Behavior Inventory

Table 10

Correlations between psychosocial measures and outcome measures

	Educational length parents	Age at injury	Family functioning	HBI retrospect Cognitive	HBI retrospect Somatic	GCS	AIS	ISS	Days in hospital
SDQ[a]									
Parent report									
Total difficulties	-.193	-.222	.291	.180	.416*	-.115	.162	.362*	.307
Internalizing	-.358	-.245	.059	-.076	.313	-.019	.113	.292	.317
Externalizing	.033	-.202	.442**	.243	.353	-.126	.119	.200	.152
Emotional problems	-.283	-.341	-.041	.106	.382	.081	-.012	.061	.087
Conduct problems	-.125	-.054	.366	.104	.460**	-.010	-.010	.273	.157
Hyperactivity problems	.005	-.194	.317	.439*	.273	-.193	.195	.160	.183
Peer problems	-.251	.012	.097	-.085	.324	-.035	.101	.372*	.375*
Prosocial	-.004	-.037	-.164	-.212	-.258	-.082	.242	-.061	.003
self-report									
Total difficulties	.070 $_p$	.070	-.203 $_p$	.200 (p)	.454	.292	-.131	.053	-.243
Internalizing	-.100	-.076	.010	.031	.558*	.217	-.345	-.186	-.437
Externalizing	.178 $_p$	.150	-.262 $_p$	.277 $_p$	.347	.270	-.071	.114	-.022
Emotional problems	-.199	-.048	.006	.086	.577*	.093	-.304	-.275	-.460
Conduct problems	.087	-.057	-.214	.119	.194	.238	-.075	-.031	.048
Hyperactivity problems	.043 $_p$	-.128	-.234 $_p$	.262 $_p$	.338	.156	-.034	.122	.079
Peer problems	-.077	-.032	.164	.045	.451	.285	-.407	-.019	-.113
Prosocial	-.290	-.254	.006	.032	.196	-.070	.052	-.073	-.063

Note: * p <.02, ** p <.01, ***p <.001. Pearson correlations are denoted with $_p$, Spearman correlations remain unmarked.

[a] Strength and Difficulties Questionnaire.