



## RESEARCH ARTICLE

# Mental Health Implications of Traumatic Brain Injury (TBI) in Children and Youth

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## Abstract

**Objective:** Traumatic brain injury (TBI) is the most common cause of death and disability in children and adolescents. Psychopathology is an established risk factor for, and a frequent consequence of, TBI. This paper reviews the literature relating psychopathology and TBI. **Method:** Selective literature review. **Results:** The risk of sustaining a TBI is increased by pre-existing psychopathology (particularly ADHD and aggression) and psychosocial adversity. Even among individuals with no psychopathology prior to the injury, TBI is frequently followed by mental illness especially ADHD, personality change, conduct disorder and, less frequently, by post-traumatic stress and anxiety disorders. The outcome of TBI can be partially predicted by pre-injury adjustment and injury severity, but less well by age at injury. Few individuals receive treatment for mental illness following TBI. **Conclusion:** TBI has substantial relevance to mental health professionals and their clinical practice. Available evidence, while limited, indicates that the risk for TBI in children and adolescents is increased in the presence of several, potentially treatable mental health conditions and that the outcome of TBI involves a range of mental health problems, many of which are treatable. Prevention and management efforts targeting psychiatric risks and outcomes are an urgent priority. Child and adolescent mental health professionals can play a critical role in the prevention and treatment of TBI through advocacy, education, policy development and clinical practice.

**Key Words:** *traumatic brain injury, mental illness, attention deficit hyperactivity disorder, children and adolescents*

## Résumé

**Objectif:** Le traumatisme crânio-cérébral (TCC) est la cause de décès ou d'incapacité la plus répandue chez les enfants et les adolescents. La psychopathologie est un facteur de risque établi du TCC, et en est aussi une conséquence fréquente. Cet article examine la littérature qui relie psychopathologie et TCC. **Méthode:** Une revue sélective de la littérature. **Résultats:** Le risque de subir un TCC est accru par une psychopathologie préexistante (en particulier le TDAH et l'agressivité) et l'adversité psychosociale. Même chez les personnes sans psychopathologie préalable au traumatisme, le TCC est souvent suivi d'une maladie mentale, spécialement le TDAH, un changement de personnalité, le trouble des conduites, et, moins fréquemment, le trouble de stress post-traumatique et le trouble anxieux. Le résultat du TCC peut être prédit en partie par l'adaptation avant le traumatisme et par la gravité du traumatisme, mais moins par l'âge au moment du traumatisme. Peu de personnes reçoivent un traitement pour maladie mentale après un TCC. **Conclusion:** Le TCC est substantiellement pertinent pour les professionnels de la santé mentale et leur pratique clinique. Les données probantes disponibles, bien que limitées, indiquent que le risque de TCC chez les enfants et les adolescents s'accroît en présence

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de plusieurs affections de santé mentale potentiellement traitables, et que le résultat d'un TCC comporte une série de problèmes de santé mentale dont bon nombre sont traitables. Les initiatives de prévention et de prise en charge ciblant les risques et les résultats psychiatriques sont une urgente priorité. Les professionnels de la santé mentale des enfants et des adolescents peuvent jouer un rôle essentiel dans la prévention et le traitement des TCC par la défense des intérêts, l'éducation, l'élaboration de politiques, et la pratique clinique.

**Mots clés:** *traumatisme crânio-cérébral, maladie mentale, trouble de déficit de l'attention avec hyperactivité, enfants et adolescents*

**T**raumatic brain injury (TBI) is the leading cause of death and disability globally. TBI occurs when the head is struck or undergoes a rapid movement or displacement without direct external trauma to the head. TBI initiates a trophic cascade of metabolic processes resulting in cell death from ischemia, oxidative stress, energy failure, excitotoxicity primarily due to excessive glutamate, axonal injury and other processes.

In Ontario, Canada, the rate of TBI among males and females, aged 15-24 years, is ~375/100,000 and ~175/100,000, respectively (Colantonio, Saverino, et al., 2010). TBI affects children disproportionately – one in thirty children will sustain a TBI by age 16 (Mitra, Cameron, & Butt, 2007). Almost half of those injured are younger than 19 years of age; of these, 40% are younger than nine years of age and 20% are younger than two years of age (Langlois, Rutland-Brown, & Wald, 2006).

The burden of TBI on the health care system is enormous (Mitra et al., 2007; Colantonio, Croxford, Farooq, Laporte, & Coyte, 2009). Worldwide, an estimated 57 million individuals have been hospitalized with TBI (Langlois et al., 2006). In the USA, 500,000 people under 14 years of age visit the emergency room annually because of head injury, of which 40% are admitted. The severity of a TBI can range from mild to severe (Association, 2013). Severe TBI is characterized by unconsciousness, an absence of meaningful response and spontaneous activity. Mild TBI (mTBI) causes a brief change in mental state characterized by confusion, disorientation, feeling dazed, loss of memory for events immediately before or after the injury, or loss of consciousness for less than 30 minutes (Table 1). Although, the majority of head injuries (80%) are mTBIs, many receive medical attention for physical, cognitive, and behavioral consequences from their injury in the short and long term (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Bigler, Abildskov, et al., 2013; Bigler, Yeates, et al., 2013). At the other end of the severity scale, 12-15% of TBI cases are judged to be severe. Hospitalization and serious post-injury impairment is found in about half of severe cases (Selassie et al., 2008). About 1% of the population lives with consequences of TBI (Zaloshnja, Miller, Langlois, & Selassie, 2008).

In descending order, motor vehicle collisions, falls, sports and recreational injuries (including bike related injuries),

and assaults are the most common reasons for childhood TBI (Colantonio et al., 2009). The burden of childhood TBI falls disproportionately on children of lower socio-economic status (SES). The risk of TBI, particularly those involving pedestrians or cyclists struck by a vehicle, is the highest for children of lower SES and minority status (Dennis, Simic, Agostino, et al., 2013; Howard, Joseph, & Natale, 2005; Langlois, Rutland-Brown, & Thomas, 2005; McKinlay et al., 2010; Parslow et al., 2005; Yates, Williams, Harris, Round, & Jenkins, 2006).

As a consequence of the high prevalence, resulting impairment and cost, disproportionate burden on individuals with lower SES as well as its typical occurrence during a period of rapid neural development, childhood TBI poses considerable challenges to policy makers, scientists and practitioners. Moreover, TBI has substantial although frequently overlooked relevance to mental health. This review addresses key issues regarding mental health and TBI in children and adolescents.

## Pre-injury psychopathology and psychosocial risk factors

Pre-injury mental illness as indexed by psychiatric diagnosis in particular ADHD, aggression, prescription for psychiatric medication or utilization of mental health services doubles the risk for TBI (Bijur, Golding, Haslum, Kurzon, 1988; Fann et al., 2002; Eme, 2012). For example, ADHD is found in 20-30% of TBI cases prior to injury compared with a population prevalence of 5% (Gerring et al., 1998; Max, Wilde, et al., 2012; Yeates & Taylor, 2005).

Other risk factors for TBI include low SES, overcrowded households, disadvantaged neighborhoods, high incidence of adverse life events, young maternal age, many older and few younger siblings in the home as well as a history of previous TBI (Bijur, Golding, & Kurzon, 1988; Max, Schachar, et al., 2005a; Max, Wilde, et al., 2012). Among children younger than age five with one head injury (4.5%), 15% will have an additional head injury before age ten, 2.8% will have two, and 0.4% will have three (Keenan, Hall, & Marshall, 2008). Parental psychopathology also increases risk for TBI and its adverse consequences (McAllister, 2010). For example, children of mothers categorized as problem drinkers compared to children of mothers who are

**Table 1. Severity ratings for traumatic brain injury (TBI) from the DSM-5**

Injury characteristic	Mild TBI	Moderate TBI	Severe TBI
Loss of consciousness	< 30 minutes	30 minutes – 24 hours	> 24 hours
Posttraumatic amnesia	< 24 hours	24 hours – 7 days	> 7 days
Disorientation and confusion at initial assessment (Glasgow Coma Scale Score)	13 – 15 (not below 13 at 30 minutes)	9 – 12	3 – 8

nondrinkers have twice the risk of various, serious injuries including TBI.

In summary, the risk for TBI is increased in children and youth who have pre-injury psychiatric disorders and those who are experiencing high psychosocial risk. These factors likely operate in concert to alter the child's behavior and environment, to initiate and in turn, perpetuate a cycle of risk through TBI-related changes in behaviour. Given the high prevalence of ADHD (5%), aggression (4%) and psychiatric disorder of any type (20%) in children and youth, the importance of these conditions in conferring risk in the population for TBI is considerable.

## Mental illnesses after TBI

A wide range of mental health problems emerge de-novo in the absence of pre-injury psychopathology, and most illnesses that were present prior to TBI will persist or worsen (Catroppa et al., 2015; Levin et al., 2007; Yeates et al., 2005).

### Secondary ADHD (S-ADHD)

S-ADHD is defined in the same way as developmental or primary ADHD with the exception of needing to be evident prior to 12 years of age. S-ADHD originates in 15-50% of individuals following TBI (Gerring, et al., 1998; Levin, et al., 2007; Max, Schachar, et al., 2005a; McKinlay, Grace, Horwood, Fergusson, & MacFarlane, 2009). The incidence of S-ADHD in TBI is greater than that found in healthy controls, or those hospitalized for a fracture not involving the head. In S-ADHD cases, inattentive subtype tends to predominate (Max, Schachar, et al., 2005b), and many cases exhibit affective lability and aggression. S-ADHD does not emerge in all cases immediately following the injury but can surface at various time-points: 15% of cases manifest S-ADHD after one year and 21% manifest S-ADHD after two years (Max, Schachar, et al., 2005a). The late emergence of disorders such as S-ADHD is poorly understood and little studied although it is known that experimental injury in animals can generate late effects. The incidence of ADHD after TBI may be even higher than 20% if one considers subthreshold cases (Eme, 2012).

Incidence of S-ADHD increases with TBI severity: S-ADHD occurs in 7% of individuals after mTBI and in 46%

following severe TBI (Max, Wilde, et al., 2012). Majority of S-ADHD cases following mTBI remit over the first year while in those with more severe injury, S-ADHD can persist for at least two years. S-ADHD rates in TBI are substantially higher than those found in individuals with orthopedic injuries (26%; Yeates et al., 2005; Yeates & Taylor, 2005) and controls (3%; Massagli et al., 2004) supporting a link with the actual brain trauma. Psychosocial adversity and low pre-injury adaptive function increase the likelihood of S-ADHD (Max, Schachar, et al., 2005b). Young age at time of injury does not confer any protection. Keenan et al. (2008) found that children who experienced a head injury before the age of two years were twice as likely as children in the general population to have S-ADHD after age two. Greater number of head injuries after age two demonstrated greater risk for S-ADHD. The association of TBI and S-ADHD in young persons is complicated by our inability to know which very young children would be likely to develop ADHD in the absence of TBI. Keenan et al. (2008) found that the risk of ADHD following TBI in two year olds was not substantially greater than the risk following burns suggesting that "risk-taking" traits rather than the head injury per se could well be the common factor for later ADHD in some young children. Nevertheless, TBI seems to increase substantially the likelihood of developing S-ADHD at any age with the actual brain trauma being the operative factor in many instances.

### Aggression

Aggression following TBI can manifest as aggression toward self, others or objects, behavioral or social disinhibition, lability of mood, verbal outbursts with minimal provocation as well as threatening behavior. Cole et al. (2008) found a significant increase in aggression one year after TBI among severely injured individuals. Increase in aggression was predicted by post-injury disability, pre-injury aggression, attention problems and anxiety, but not by socioeconomic status or family stress. Among adolescent psychiatric inpatients, a history of TBI predicts increased risk of criminality by 6.8-fold, conduct disorder by 5.7-fold, concomitant criminality and conduct disorder by 18.7-fold (Cole et al., 2008; Heverly-Fitt et al., 2014; Luukkainen, Riala, Laukkanen, Hakko, & Rasanen, 2012; McKinlay et al., 2014; Yeates et al., 2013).

TBI impedes social cognition and a child's ability to cope with every-day social tasks such as sharing, helping, and initiating relationships (Dennis, Agostino, et al., 2013; Dennis, Simic, Bigler, et al., 2013; Max, et al., 2000; Robinson, et al., 2014; Yeates, et al., 2013). Oppositional defiant disorder (ODD) is a pattern of defiant, disobedient, stubborn, argumentative and rude behavior with family and non-family adults, which occurs in 18% of cases following TBI. These outcomes are more common in those who have experienced a severe injury than a mild one, those who had high levels of pre-injury psychosocial adversity, ODD or aggression, and multiple brain injuries prior to the index injury (Heverly-Fitt, et al., 2014; Max, et al., 1998; Robinson, et al., 2014; Taylor, et al., 2015; Wolfe, et al., 2014; Yeates, et al., 2013).

### **Personality change (PC)**

One of the most common outcomes of TBI is PC as outlined in detail in the review by Max (2014). PC as a consequence of TBI is evident after both severe (40%; Max et al., 2000) and mTBI (5%). PC is not diagnosed when another diagnosis better accounts for the change in behaviour or when disorder occurs exclusively in the presence of delirium. PC can be transient or persistent. However, PC and externalizing disorders are commonly comorbid following TBI (Max, Levin, et al., 2005). Most commonly, one observes an increase in affective lability, aggression and disinhibition, and less commonly, a pattern of apathetic or paranoid behaviour (Max, Robertson, & Lansing, 2001). PC is more common with increasing severity of injury, but is not predicted by psychosocial adversity, pre-injury mental illness, family dysfunction, or age at injury. Lesions of the dorsal prefrontal cortex, specifically the superior frontal gyrus, are associated with personality change after controlling for severity of injury and the presence of other lesions (Max, Levin, et al., 2005).

Following TBI, many individuals complain of cognitive, somatic, and behavioral changes (inattention, difficulty thinking, trouble remembering, tiredness, headaches, photophobia, dizziness, trouble seeing, sensitivity to noise, insomnia, irritability, nervousness, sadness, anhedonia, decreased motivation, and personality change; Babcock et al., 2012; Moran et al., 2011). Many of these symptoms are found in individuals who have not had a traumatic brain injury or who have had an injury that did not involve the head suggesting that there may not be a specific link to TBI. Nevertheless, these syndromes are evident in 35-50% of TBI cases overall and there is a gradient with severe TBI cases being more commonly affected suggesting that the TBI does indeed play a role in the etiology of these symptoms (Hajek et al., 2010; Pickering, Grundy, Clarke, & Townend, 2012; Taylor et al., 2010). In addition, post-concussive symptoms are more frequent among those with loss of consciousness or abnormalities on neuroimaging following TBI. Persistent post-concussive symptoms in the absence of neurological markers is also common among individuals who sustain

mTBI. PCS is associated with significant decline in health-related quality of life.

### **Internalizing disorders**

Psychiatric consequences of TBI are not limited to "externalizing" disorders. Various *de novo* "internalizing" disorders such as mood (~25%) and anxiety (~20%) disorders are also common (Hawley, 2003; Kirkwood et al., 2008; Max et al., 2011; Max, Keatley, et al., 2012; Max et al., 2013). Although obsessive-compulsive disorder is relatively rare, obsessive-compulsive symptoms (OCS) occur in 26-35% of individuals after TBI with the most common symptoms including worry about disease, pre-occupation with cleanliness, excessive cleaning, ordering, and perseveration (Grados et al., 2008; Max et al., 2000). Internalizing disorders tend to remit more often than externalizing disorders although many persist (McKinlay et al., 2009). Psychosocial adversity and female sex are predictors of post-injury internalizing disorders.

### **Post-traumatic stress disorder (PTSD)**

The lifetime prevalence of PTSD in the general child population is estimated to be 7.8% but is found in 13% of individuals one year after severe TBI. Many individuals report sub-threshold PTSD symptoms such as re-experiencing and hyper-arousal following TBI. Both disorders may involve varying degrees of amnesia for the traumatic event. PTSD is more common in females than males and in those who experience anxiety directly after TBI. Pre-injury psychosocial adversity, anxiety, and injury severity are predictors for post-injury PTSD (Gerring et al., 2002; Hajek et al., 2010; Levi, Drotar, Yeates, & Taylor, 1999).

In summary, a wide range of psychiatric disorders including internalizing and externalizing disorders can appear *de novo* and persist for many years following TBI (Catroppa, Godfrey, Rosenfeld, Hearps, & Anderson, 2012; Max et al., 2013). The extent of persistence of impairment increases with injury severity, family dysfunction and pre-injury psychopathology (Ventura et al., 2010). Post-TBI mental illnesses are often accompanied by an overall decrease in general health, shorter life expectancy, academic underachievement and neurocognitive deficits in areas such as working memory and response inhibition (Ventura et al., 2010). Comorbidity tends to be the rule rather than the exception for children who have experienced TBI. Max et al. (2005b) reported that 60% developed more than one novel psychiatric disorder at some point after TBI.

### **Prediction of outcomes**

It is a truism that no two individuals with TBI are the same with regard to pre-injury risk and protective factors (Bigler, Abildskov, et al., 2013). Each individual experiences a unique neurotrauma arising from the mechanics of their injury, post-injury adjustment (anxiety, depression, guilt) and

resulting social consequences such as the amount of time that a child misses school (Richards & Carroll, 2012). Predictors include adverse environmental circumstances, low pre-injury adaptive function and scholastic ability, psychopathology, and family dysfunction.

Injury severity is a critical factor in predicting adverse mental health outcomes -the greater the TBI severity, the higher the likelihood of post-injury psychopathology. Even among those with mTBI, there seems to be a relationship between TBI severity and the risk of adverse outcomes (Massagli et al., 2004; McKinlay et al., 2009). Nonetheless, injury severity does not account for all of the variation in mental health outcome (Max, Schachar, et al., 2005a; Max, Wilde, et al., 2012; Yeates et al., 2005). The incidence of psychopathology, the number of lesions on CT or MRI scans and the extent of cognitive deficit after TBI are often unrelated to initial injury as typically assessed. Even among children with severe brain injury, half have no neuropsychological, behavioral, adaptive or academic impairment at follow-up (Fay et al., 2009). To some extent, the lack of a strong severity-outcome relationship could be explained by our poor understanding of the notion of severity. With every new development in imaging, we discover lesions that had not previously been detected but which predict outcome (Bigler, 2013; Levin et al., 2008).

The strength of the link between severity and prognosis also depends on what outcome is being addressed and possibly on the length of time since injury. For example, Anderson et al. (2012) found that poorer adaptive skills were evident for those with more severe injury ten years after injury, but that behavioural difficulties were present regardless of injury severity. In some studies, adaptive function at outcome was predicted primarily by pre-injury adaptive function (Anderson, Catroppa, Haritou, Morse, & Rosenfeld, 2005; Catroppa, Anderson, Morse, Haritou, & Rosenfeld, 2008; Yeates et al., 2004).

How outcome is related to injury characteristics over time is only just beginning to be understood. It was once assumed that brain recovery reached a stable state after TBI, but it is now clear that chronic neuroinflammation and white matter degeneration persist for years after a single traumatic brain injury while neurogenesis can occur throughout life (Smith et al., 2013). These processes provide a mechanism for the waxing and waning after injury of mental health, neurocognitive and behavioural outcomes.

Pre-injury psychopathology increases the likelihood of post-injury psychopathology (Levin et al., 2007; Massagli et al., 2004; Max et al., 2013; Yeates et al., 2005) and may interact with injury severity in predicting adverse outcomes; pre-injury psychopathology coupled with increasing injury severity predict greater risk of psychopathology and impairment after injury. The association of poorer pre-injury behavioral adjustment predicting greater distress and impairment after injury is also found in mTBI. This pattern

highlights the role of reserve capacity as a moderator of adverse effects of TBI (Ponsford et al., 2000; Satz, Cole, Hardy, & Rassovsky, 2011; Satz et al., 1999).

Contrary to what was once thought, young age at the time of injury confers no advantage for recovery for mental illness (Dennis, Spiegler, et al., 2013). Given that neural circuitry, as well as social and cognitive skills are developing during childhood in step with rapid neural growth, it is understandable that injury in young child could have greater adverse effects than an equivalent injury in an older individual (Gronwall, Wrightson, & McGinn, 1997). Younger age at injury predicts greater cognitive deficit (Leblanc et al., 2005), risk for developmental delay and epilepsy, as well as poorer academic and intellectual performance especially if the injury occurs before the age of two years (Anderson et al., 2009). Furthermore, TBI early in life affects a larger proportion of an individual's lifespan than does an injury later in childhood or in adolescence.

## Treatment implications

There is little solid evidence supporting the role of medication in neuroprotection and neurorecovery in children and adolescents (Pangilinan, Giacoletti-Argento, Shellhaas, Hurvitz, & Hornyak, 2010), or to guide mental health practice after a TBI. Few treatment trials of quality for TBI patients have been conducted (Frenette et al., 2012; Teasell et al., 2007). Only 28% of acquired brain injury treatment studies have employed a randomized clinical trial, and most studies have small sample sizes. Consequently, a minority of therapeutic conclusions are based on strong evidence. Most TBI patients (86%) do not receive any therapy targeting their psychological or psycho-educational needs either in the short or the long term (Colantonio, Howse, et al., 2010). This state of affairs is true even among the most severely injured (Catroppa et al., 2012).

A good example of the gaps in treatment literature can be found on the question of stimulant medication for post-injury ADHD. Experimental animal studies support treatment for S-ADHD (increase striatal dopamine transmission in injured rats; Wagner et al., 2009). A review of the few studies that evaluated stimulant treatment of childhood S-ADHD suggested that these drugs are effective in S-ADHD although they may have less effect than in primary ADHD. Typically, stimulants are used at a lower dose in S-ADHD than in primary ADHD in the absence of quality evidence (Jin & Schachar, 2004). In the general population, 56% of primary ADHD cases (Visser et al., 2014; Visser, Lesesne, & Perou, 2007) but only 7% of S-ADHD cases receive treatment with methylphenidate (Levin et al., 2007). The relative under-utilization of stimulants in the care of patients with TBI could reflect family or practitioner preferences, absence of clear practice guidelines, lack of access to service or insufficient education about the potential of drug therapy. Of note, half of the potential TBI subjects in one

study declined participation (Willmott & Ponsford, 2009). ADHD practice guidelines make no specific treatment recommendations for psychiatric disorders such as S-ADHD after TBI (CADDRA, 2011; see Pliszka & Issues, 2007).

There is some evidence that risk for TBI can be mitigated by prescription for psychostimulant medication for those with ADHD (Fann et al., 2002). However, retrospective study indicates that the majority of TBI patients who meet criteria for pre-injury ADHD have not received treatment for the disorder prior to their injury (Gerring et al., 1998; Max et al., 2004). There is a clear need for evidence-based TBI treatments and studies regarding strategies for reducing risk.

Similarly, evidence supporting non-pharmacological treatment for TBI is limited. Cognitive behavioural therapy can improve children's behavior (Pastore et al., 2011) and randomized control trials of family therapy based on problem solving can be effective. In a recent study by Wade et al. (2015), children 12-17 years who had been hospitalized for TBI in the previous seven months showed less impaired functioning after online counselor-assisted problem solving therapy. By and large, current treatments for mental health problems after TBI are based on what is known about the effects of these interventions in the un-injured population.

## Conclusion

This review of the psychiatric precursors and consequences of TBI clearly indicates the importance of mental health in prevention, assessment and treatment efforts for TBI. Mental illnesses are modifiable risk factors for TBI. Untreated ADHD, in particular, seems to add risk for TBI. Available evidence indicates that minority of TBI cases with pre-injury ADHD have received adequate treatment. Counseling about injury prevention ought to be a standard component of care for children and adolescents with psychopathology, particularly ADHD and aggression, as well as those who have already suffered a TBI.

TBI often results in impairing and persistent mental illnesses such as S-ADHD, aggression, internalizing disorders, personality change, and post-concussive symptoms, in addition to broad social cognitive and social function impairments. In many cases, post injury psychopathology is persistent and in some few cases mental health problems may appear after a period of apparent recovery. Clinical practice must be adjusted accordingly. These disorders must be identified among children with TBI and treated where possible. Acquired forms of psychiatric disorders might not be identical to developmental forms and thus the diagnostic criteria for acquired psychopathology and optimal therapeutic practices will require specific study.

Given the burden to affected families and the community, urgent research priorities must be set. First, there is a clear need for further research into mental illness and TBI.

Large-scale, multi-site cohorts of children, youth and young adults who have experienced traumatic brain injuries of various degrees of severity and mechanism could be studied longitudinally on a range mental health, neuropsychological and brain imaging parameters. Some research goals in these cohorts include parsing the effects of mediator and moderator variables such as injury severity, pre-injury mental health, age, lesions type and location, and environmental circumstances (Abo-Zaid, Sauerbrei, & Riley, 2012; Catroppa et al., 2012; Luoto et al., 2013), as well as identifying better biomarkers of evolving psychopathology, social and academic impairment (Washington et al., 2012). Second, clinical trials networks to evaluate a range of drug, non-drug and combination therapies along with patient, family, and practitioner education on medication such as stimulants must be developed (Ponsford et al., 2001). Mental health problems arising after TBI are likely to differ from those that arise in the absence of TBI and therefore require specific study to determine best treatment strategies for both short- and long-term periods following TBI.

There is an urgent need for evidence based practice guidelines for the full range of mental health consequences of TBI including non-pharmacological interventions aimed at improving cognitive, academic and psychosocial outcomes. Mental health practitioners of all disciplines can play a critical role in the prevention and treatment of TBI through advocacy, education, policy development and clinical practice.

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