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A review of post-concussion syndrome and psychological factors associated with concussion

Donna K. Broshek, Anthony P. De Marco, & Jason R. Freeman

Department of Psychiatry and Neurobehavioral Sciences, University of Virginia School of Medicine, Charlottesville, VA, USA

Abstract

Objective: This study reviewed several topics related to post-concussion syndrome and psychological factors associated with concussion. Topics include neurobiological perspectives, psychological predictors of post-concussion syndrome including pre-morbid anxiety, anxiety sensitivity and cognitive biases and misattribution. In addition, the iatrogenic effects of excessive rest are reviewed and treatment options are discussed briefly.

Main results: Animal models of concussion and mild traumatic brain injury suggest that a concussion can result in anxiety and fear reactions. The pathophysiology of depression following a concussion appears to be consistent with the cortico-limbic model of depression. Additionally, some individuals may be at risk for neurobiological depression and/or anxiety following a concussion. The literature also demonstrates that pre-morbid and concurrent anxiety increases the risk for prolonged concussion recovery. Cognitive biases and misattribution of symptoms contribute to lengthy recovery from concussion. In addition, medically prescribed excessive cognitive and physical rest may contribute to a protracted concussion recovery. Supervised and graduated physical activity, the introduction of anxiety reduction techniques and cognitive-behavioural therapy of cognitive biases and misattribution are effective means of shortening the length of post-concussion syndrome.

Conclusions: Understanding, assessing and treating the psychological factors associated with concussion are effective means of preventing or shortening the length of post-concussion syndrome.

Keywords

Anxiety, depression, mild traumatic brain injury

Introduction

For some individuals, recovering from concussion is prolonged and difficult to move beyond. While elite athletes are often intrinsically motivated to deny large and small physical discomforts in the pursuit of their goals, other athletes and recreational participants are often functionally disabled by their concern about their symptoms. The focus of this article is primarily on the non-elite athlete who has difficulty recovering from sports-related concussions and will review pre-morbid and co-morbid factors that can complicate recovery. While elite athletes may experience significant cognitive and psychological sequelae from multiple concussions, they are more likely to minimize the short- and long-term consequences of repeated head trauma. The willingness of elite athletes to risk their health has been illustrated by an interesting scenario devised by Goldman et al. [1]. They posed the following question to a group of elite athletes:

‘Would you take an illegal performance enhancing drug that was undetectable and guaranteed you would win an Olympic gold medal, if it would kill you in 5 years?’ The same survey was repeated bi-annually from 1982–1995 and consistently over half of elite athletes responded affirmatively [2]. In contrast, the same question was posed to the general population in 2009 and only one of 125 respondents said yes. Goldman et al.’s research demonstrates that highly competitive athletes willingly take significant risks with their health in their pursuit of athletic goals. In sharp contrast to the findings of Goldman et al., a recent study by other authors [3] found that only two athletes out of 212 male and female athletes would accept this ‘Faustian’ option. The authors hypothesized that changes in the social and political context have reduced the risk athletes are willing to take for victory and that the Goldman et al. dilemma is no longer relevant. The results of an anonymous survey by NFL Nation [4], however, argue against this optimistic perspective. When 320 NFL athletes were asked if they would play with a concussion in the 2014 Super Bowl, 85% responded affirmatively [4]. These studies demonstrate that there is significant variability in the risk that athletes willingly undertake to compete at various levels and across sports. These attitudes about health risks likely translate into differences in psychological...
response to concussion symptoms. The focus of this article is to review the psychological factors that can complicate recovery in some individuals after sustaining a concussion.

Although there has been a renewed focus on concussion in the media and among the general public and much recent discussion by professionals about lingering recoveries, the concept of functional disability after a concussion or mild traumatic brain injury (mTBI) was identified many years ago. Kay et al. [5], at the Research and Training Centre on Head Trauma and Stroke at the New York University Medical Centre, published their proposed neuropsychological model of functional disability after mTBI in 1992. They noted that most individuals recover quite well and they identified multiple factors that can complicate recovery, including neurologic findings, physical factors, psychological and personality factors, psychosocial issues and being involved in litigation. This model includes a sub-set of individuals with concussion who may have neurologic factors, including axonal shearing or pre-morbid history affecting integrity of the brain, such as previous neurologic damage or significant substance abuse. Physical factors include pain, sleep disturbance and peripheral injury. The hallmark of concussions is some degree of cognitive compromise, but when cognitive symptoms persist the individual may experience a shaken sense of identity [5]. This model states that psychological distress can create further cognitive compromise by suppressing attention, mental efficiency, learning and memory, thus creating cognitive symptoms above and beyond those accounted for by the concussion. These cognitive symptoms create frustration and additional distress, leading to anxiety and avoidance of anxiety provoking situations, such as those requiring optimal cognitive abilities. The anxiety and avoidant behaviours may also lead to depression. As the complex inter-play between anxiety and depression increases, they further distract the individual and cause additional cognitive compromise, which may result in additional emotional distress. The psychological overlay accumulates and intensifies and may become more disabling than the initial injury. Personality variables, such as high achievement goals, dependent personality or insecurity, can also affect the way an individual interprets their symptoms and recovery, as well as how others are reacting to and interacting with them. Kay et al. [5] also identified psychosocial factors that can affect recovery, including work or school demands, as well as pressure from teammates and coaches to return to competition. The literature on the association between symptoms of post-concussion syndrome (PCS) and litigation involvement is beyond the scope of this article, but factors associated with the legal process can also result in prolonged symptoms.

Independently of Kay et al. [5], Montgomery [6] also proposed a multi-dimensional model of disability after brain injury. His model described personal and situational factors that could prolong symptoms and contribute to neuropsychological disability. The personal factors included negative cognitions, tension-avoidance, physical symptoms and fatigue. Situational factors consisted of rapid processing demands, need for complex cognitive attention and external distractions. Montgomery recommended that rehabilitation efforts be focused on working with patients to modify these factors, including identification and management of ‘non-organic’ factors.

**Neurobiological perspectives of psychological consequences of traumatic brain injury**

In the most recent reiteration of the Consensus Statement on Concussion in Sport, the Concussion in Sport Group (CISG) recognized that psychological issues are commonly reported as a consequence of concussion and, therefore, should be considered when managing individuals who have sustained sports-related concussions [7]. While adjustment difficulties and psychological symptoms are not necessarily an uncommon reaction to traumatic brain injury of all severities, this is especially true in sports-related concussion, largely due to situational circumstances (e.g. cognitive difficulties, pain, removal and/or possible retirement from play, limited support from team/coaches). However, there is evidence to also suggest that neurobiological/pathophysiological changes associated with the brain injury, regardless of the severity level, may be directly related to the onset of psychological symptoms [8–10].

**Mayberg’s cortical-limbic model of depression**

Over the years, researchers have identified numerous brain regions and cortical networks that are associated with the pathogenesis of depression. It is general consensus that the onset of depressive symptoms is not necessarily the direct result of an insult to a particular cortical area or neurotransmitter system, but rather a multi-dimensional disruption to underlying functional pathways and the failure of reciprocating systems to maintain homeostatic emotional control [11]. For instance, frontal-limbic-subcortical pathways repeatedly have been found to mediate various aspects of human behaviour [12]. Structural and functional imaging studies have further confirmed that limbic, subcortical and frontal regions are critical neural substrates in the pathophysiology of depression [13–15]. A recent study examining connectomes in major depression using diffusion tensor imaging (DTI) found alterations within the default mode network, which is a network of cortical regions associated with self-referential activity and within the frontal-thalamocaudate network. The authors found that patients diagnosed with major depressive disorder exhibited reduced connectivity within both of these regions [16]. Mayberg’s limbic-cortical model of depression suggests that depressive symptoms and sadness are mediated by increased blood flow in the ventral limbic and paralimbic regions, which include anterior insula and subgenual cingulate and decreased blood flow in neocortical and limbic regions (e.g. prefrontal, inferior parietal, dorsal anterior cingulate, posterior cingulate) [11,17,18]. This is convergent with functional studies of depression demonstrating hyperactivity within the ventromedial prefrontal cortex and hypoactivity in the dorsolateral prefrontal cortex [14]. Finally, treatment of depression, including pharmacology, psychotherapy and surgery, is geared toward facilitating the ‘synchronized modulation of these dysfunctional cortical-limbic pathways’ ([11], p. 195). Structurally, decreased hippocampal volume has been associated with depressive episodes, with some
Traumatic brain injury and depression

Following an mTBI, it is estimated that 12–44% of individuals experience some degree of depression within the first 3 months [20]. Given that intracranial abnormalities following traumatic brain injury are likely to involve frontal and temporal lobes [21] and the above-mentioned fronto-limbic-subcortical structures implicated in depression, it is not surprising that depression is a commonly-observed biological consequence of traumatic brain injuries of all severities [22]. Subsequent studies of depression following traumatic brain injury continue to provide support for the cortical-limbic model of depression. In an athlete sample, Chen et al. [8] reported attenuated task-related activity in the dorsolateral prefrontal cortex, dorsal anterior cingulate cortex, insular cortex, thalamus and striatum in those with post-concussive and depressive symptoms relative to concussed-athletes without depressive symptoms and healthy controls. Furthermore, the reductions in these regions of interest were more pronounced in athletes reporting a higher severity of depressive symptomatology. The authors suggested that these findings may reflect dopaminergic dysfunction within the cortico-striato-thalamic system in those athletes reporting comorbid post-concussive and depressive symptoms. Regarding morphometric findings, Chen et al. found that, while concussed athletes without depression exhibited grey matter loss in the insular cortices, likely due to the trauma, the concussed and depressed group demonstrated further grey matter volume loss in the medial frontal and temporal regions. Furthermore, severity of depressive symptoms was negatively associated with rostral anterior cingulate cortex volume, such as greater degrees of depressive symptoms were associated with less grey matter volume in this particular region of interest. The authors concluded that, while the structural abnormalities observed in their sample were likely secondary to the concussion, the depressive symptoms were attributed to the medial prefrontal dysfunction related to the head injury [8].

Traumatic brain injuries may be associated with atrophic changes that may be progressive in nature [23]. Hudak et al. [9] reported on the association between degree of atrophy following a traumatic brain injury, most of which were resultant of motor vehicle collision, across 16 regions of interest and depressive symptoms, as assessed by the Beck Depression Inventory-II. Individuals underwent magnetic resonance imaging (MRI) studies during the acute period of the injury and 6-months post-injury. As the reported median Glasgow Coma Scale for the sample was 8, approximately half of the sample had sustained a severe traumatic brain injury. Injury severity was correlated with depression severity, with ~20% of the sample reporting depressive symptoms. After employing false discovery rate to correct for multiple comparisons (FDR 0.05), the authors found that regional atrophy, specifically in the left rostral anterior cingulate and bilateral orbitofrontal cortex, was significantly correlated with depressive symptoms [9].

Limbic-medial prefrontal model of anxiety-related negative emotion

Anxiety disorders, as a group, are characterized by a disproportionate degree of fear, avoidance and worry in response to environmental or internal stimuli. In conceptualizing anxiety-related disorders, distinctions have been made between those related to fear, such as social anxiety disorder, vs. those characterized by anxiety/misery, such as generalized anxiety disorder. However, it should be noted that these categories are not necessarily mutually exclusive, as some disorders are characterized by both fear and anxiety/misery features.

The limbic-medial prefrontal model, as outlined by Etkin [24], describes the processing of fear-related emotions. Emotional processing, as it relates to fear, has been associated with hyperactivation of the amygdala and insula [24,25]. These limbic structures, as well as additional subcortical structures (e.g. hypothalamus and periaqueductal grey), encode and register emotional stimuli and initiate the co-ordination of behavioural responses. Exaggerated or excessive activation of the amygdala, in particular, has been implicated across anxiety-related disorders, as this structure is thought to control fear-based reactions [26]. The contribution of the insula, while less studied than the amygdala, is based on its regulation of the autonomic nervous system and its integral role in the process of interoception. The dorsomedial prefrontal cortex and dorsal anterior cingulate then further evaluate and monitor emotional stimuli and are responsible for the expression of fear, while the ventromedial prefrontal cortex and ventral anterior cingulate cortex are involved in the regulation, modulation or inhibition of these reactions [24,25]. The ventromedial prefrontal cortex has been found to play a critical role in regulating amygdala activity in humans, with hypoactivity in this region leading to pathological mood states. This is primarily due to the failure of this region to serve its regulatory role [27]. In fact, dysfunction within dorsomedial and ventromedial prefrontal structures has also been implicated across several anxiety-related disorders. Finally, through its vast interconnections with the amygdala and hypothalamus, the ventral hippocampus (in animal studies), which is involved in the regulation of endogenous anxiety-related behaviour, likely serves a role in the manifestation of anxiety. Damage to the ventral hippocampus, in rodents, has been associated with a reduction of anxiety behaviour. However, the functional contribution of the hippocampus in anxiety-related disorders is less understood in humans [24,25].

In addition to the aforementioned functional studies, volumetric studies of individuals who have been exposed to trauma, especially those who exhibited symptoms of post-traumatic stress disorder (PTSD), have documented structural abnormalities. More specifically, several studies have demonstrated reduced hippocampal volumes, whether unilateral or bilateral diminution, in trauma-exposed populations [28]. While it is difficult to determine whether these abnormalities are related to chronic symptomatology (e.g. PTSD) or simply due to trauma histories, other studies have demonstrated that elevated levels of glucocorticoids may lead to hippocampal diminution in non-human primates [29]. In a non-clinical
sample, Levita et al. [30] found a positive relationship between hippocampal volume (lateralized to the right hemisphere) and behavioural inhibition and suggested that any change to hippocampal volume, structural or functional, may serve as a diathesis for anxiety-related symptomatology.

**Traumatic brain injury and anxiety-related symptoms**

Similar to depression, anxiety-related symptomatology is often observed in individuals following a sports-related concussion or mTBI. As previously mentioned, traumatic brain injuries often involve damage to the prefrontal cortex, ventral frontal lobe and anterior temporal lobe, areas which are heavily implicated in the recognition of emotionally-relevant stimuli and regulation of the reactions to those stimuli [24,25,31]. To examine the neurobiological relationship between mTBI and the acquisition of anxiety-related symptoms, more specifically fear, rodent models typically employ lateral fluid percussion injury and water maze paradigms. Studies employing this methodology, however, have yielded somewhat inconsistent results. Shultz et al. [32] found that rodents who received a mild fluid percussion injury had a tendency to spend more time in the open arms of the behavioural apparatus (i.e. water maze) immediately following the impact (i.e. 24 hours). This finding was interpreted as the injury having a short-term anxiolytic effect. An earlier study, on the other hand, found an increase in anxiety-related behaviour 1 month following the injury [33]. It is important to note that severity of injury (mild vs. severe) and time of assessment (24 hours vs. 1 month) are two factors that may have contributed to the inconsistency between findings. Bolouri et al. [34] found that rodents, who were administered multiple impacts, engaged in less spontaneous exploratory behaviour 2–4 days and at 1 and 2 weeks following injury, suggestive of increased anxiety, despite intracranial pressure returning to baseline after 7 days post-injury. Finally, Reger et al. [10], who used induced lateral fluid percussion injuries and Pavlovian fear conditioning in their rodent model, found that injured rodents demonstrated increased fear conditioning and an over-generalization of learned fear to conditioned and novel conditions. Reger et al.’s findings parallel the acquisition of post-traumatic symptomatology following mTBI, suggesting that these injuries may increase one’s vulnerability to psychological distress and trauma.

The studies reviewed above vary in terms of samples, mechanism of injury and severity of injury, but collectively provide foundational support regarding the pathophysiology of post-traumatic brain injury depression and anxiety. While there is significant overlap between the underlying pathophysiology observed in these psychological disorders and that observed in psychological disorders within the context of traumatic brain injury, including those at the mild end of the spectrum (i.e. sports-related concussion or mTBI), there are inconsistencies that need to be elucidated further by future research. Nevertheless, some of these findings may assist in explaining the development of psychological symptoms following the injury. For instance, one plausible explanation is that the aforementioned neurobiological abnormalities may serve as a pre-disposition for the expression of affective distress following a traumatic brain injury (i.e. diathesis/stress model). Another plausible explanation is that the structural/functional changes directly related to the brain injury may, in turn, place individuals at greater risk for resultant depression and anxiety symptoms, especially after sustaining repetitive injuries. This assertion is partially supported by literature demonstrating that a history of repeated concussions in early adulthood may place individuals at higher risk for depression later in life than the general population and counterparts without a concussion history [35–38].

**Post-concussion syndrome**

The term ‘post-concussion syndrome’ (PCS) often refers to a set of non-specific symptoms that are reported following a concussion or mTBI and that persist beyond the expected recovery period. Symptoms can include: headaches, fatigue, vertigo/dizziness, irritability, emotional lability or irritability, cognitive difficulty (e.g. concentration), sleep disturbance and/or depression and anxiety. Symptoms associated with PCS also have been found in other clinical and non-clinical groups, including healthy adults [39,40], chronic pain patients [41], spinal injury patients [39], non-brain injured trauma patients [42], psychological distress [43,44] and orthopaedic injuries [45]. Therefore, the lack of specificity associated with this particular constellation of symptoms increases the potential for misdiagnosis and clinicians should avoid simply attributing the presence of these symptoms to a remote mild brain injury [46,47]. While it is often estimated that 10–20% of patients who sustain an mTBI will experience PCS, an accurate estimate is unlikely given the lack of specificity in the symptom constellation [46]. Furthermore, it is important to bear in mind that the majority of individuals who sustain an mTBI or concussion experience a relatively quick, full and uneventful recovery from their injury [5,48,49].

**Aetiology of PCS**

As one may imagine, the aetiology of PCS has been a longstanding and controversial subject of debate. In his 1988 description of PCS, Lishman [50] distinguished between the ‘physiogenesis’ and ‘psychogenesis’ of symptomatology, noting that the ‘interplay [between organic and non-organic contributions] is time-dependent’. Lishman’s aetiological model hypothesized that neurobiological factors are relevant to the acute presentation of symptoms. As these factors resolve over time, psychological factors contribute to persisting symptoms [50]. Silverberg and Iverson [51] revisited and re-evaluated this hypothesis based on a review of the literature and concluded that both neurobiological and psychological factors, in fact, have an influence on symptoms during the early stages of recovery. The authors concluded that psychological disturbances: (1) contribute to symptoms during the acute stages of PCS, (2) are stable and persistent in prolonged presentations of post-concussive symptoms and (3) are predictive of late outcomes of PCS.

**Predictors of PCS**

It is understood that there are factors that likely predict the presence of post-concussive symptoms above and beyond
actually sustaining a brain injury. For example, Meares et al. [42] found that mTBI in the general population did not predict post-concussive symptoms. Instead the authors found that pre-injury depression or anxiety and acute post-traumatic stress (at ~5-days post-injury) were predictive of post-concussive symptoms at 1-week post-injury, this was not the case 3-months later when experiencing a traumatic brain injury did not significantly contribute to the regression model. Rather, the authors found that pre-morbid psychiatric history and gender (being female) predicted post-concussive symptoms at 1-week post-injury. Ponsford et al. [52] found that, while the presence of a mTBI, along with concurrent anxiety, pre-morbid psychiatric history and gender (being female) predicted post-concussive symptoms at 1-week post-injury, this was not the case 3-months later when experiencing a traumatic brain injury did not significantly contribute to the regression model. Rather, the authors found that pre-morbid psychiatric and physical history, concurrent anxiety and trauma-related symptoms, life stressors and pain were predictive of post-concussive symptoms at follow-up. In addition to affective factors (e.g. neuroticism, depression and anxiety) [39], coping style also has been found to contribute to the perception of post-concussive symptoms in children, where emotion-focused strategies were positively associated with symptom report and problem-focused strategies were negatively associated with symptom report across mTBI and orthopaedic groups [53]. Similarly, pre-injury resilience and depression were predictive of 1-month post-injury anxiety and post-concussive symptoms [54]. Along with prolonging recovery from a concussion, psychological distress has been shown to adversely influence performance on computerized neurocognitive tests which are commonly used to establish a pre-season baseline [55], thus further complicating how emotional distress can influence recovery or detection of clinical recovery.

Anxiety sensitivity, which is considered a personality trait, is a fearful response associated with an individual’s own bodily sensations that arises from the belief that these sensations are signs of impending harmful consequences [56]. Anxiety sensitivity, for example, has been demonstrated to influence how individuals perceive pain following a concussion [57]. In comparing two clinical groups, a mild head injury group and orthopaedic control group, Wood et al. [58] found that both groups reported symptoms consistent with PCS (although individuals who sustained a mild head injury reported more symptoms) and post-concussive symptoms were positively associated with anxiety sensitivity. With that being said, this relationship was stronger among the mild head injury group [58]. The authors concluded that heightened anxiety sensitivity may contribute to how clinical populations, especially those who experience an mTBI, perceive their injuries.

Cognitive misattribution and expectancy effects

Attribution or expectation may play an important role in the development of post-concussive symptoms for some individuals as they anticipate that these symptoms will be a natural part of their recovery from an mTBI and erroneously attribute any perceived difficulty to the injury. The tendency to attribute common current symptoms and health concerns to a past concussion has been called ‘expectation as aetiology’ and was first coined by Mittenberg et al. [59]. They recruited 223 healthy community volunteers and a group of 100 individuals clinically referred for head injury. All participants were given a checklist of cognitive, affective and somatic symptoms. The participants who had sustained head injuries were asked to indicate their current symptoms, as well as any symptoms they might have experienced prior to their injury. The control participants were asked to endorse any current symptoms they were experiencing and also to indicate what symptoms they might experience if they sustained a head injury as presented in a clinical vignette. The individuals with head injury reported 60% fewer symptoms pre-injury relative to the base rate in the healthy controls, but both groups reported a high degree of symptoms experienced or imagined (for the controls) after head injury. Mittenberg et al. proposed that the expectation of symptoms after head injury results in the misattribution of common symptoms and complaints (for example, headaches) to the injury and minimization of pre-injury symptoms and minimization of other aetiologies, such as stress.

In one of the earlier studies on how cognitive biases can affect perception of symptoms, researchers examined 209 male athletes participating in sports at the high school, college or postgraduate level [60]. The respondents were divided into those who acknowledged experiencing a sports-related concussion within the previous year but no other form of head trauma and a control group who reported no concussions or head trauma the previous year. Using a survey format, the athletes in the concussion group were asked to indicate which of 30 symptoms they had experienced after their concussion and which symptoms they experienced before their concussion. The control group was simply asked to identify any current symptoms they might be experiencing and to also indicate which symptoms they anticipated experiencing after a concussion. Of the 30 symptoms, only headache was reported more frequently by concussed athletes and there was no difference in the two groups on the other 29 symptoms. Notably, the concussion group under-estimated their pre-concussion symptoms by 97% relative to the control group. The authors suggested that education on concussion symptom base rates is an important intervention during the acute phase of recovery given the role that misattribution and expectation play in maintaining PCS.

In a study of healthy athletes, individuals with depression and normal controls, Gunstad and Suhr [61] asked participants to indicate current symptoms, as well as symptoms they would expect to experience after an mTBI. Based on their results, they proposed the ‘good old days’ bias as an explanation for the tendency to under-estimate pre-injury problems and over-estimate pre-injury health after a concussion.

In a very interesting recent study of the ‘good old days’ bias, a group of 90 Canadian individuals who were receiving temporary worker’s compensation due to an mTBI and had been referred to a concussion clinic were surveyed [62]. A group of 177 healthy community and university controls were included as controls. Using the British Columbia Post-Concussion Symptom Inventory, the injured group was asked to estimate their pre-injury symptoms and to endorse their current symptoms. The control group was asked to indicate their current symptoms. Consistent with previous research, the concussed group endorsed far fewer symptoms pre-injury than the base rate reported by the healthy group. Of particular interest, this study also divided the concussed group into those
who failed \((n = 16)\) or passed \((n = 46)\) effort testing using the Test of Memory Malingering (TOMM). Those who failed the TOMM reported more symptoms than those passing the effort testing. In contrast, those who failed the TOMM reported far fewer symptoms pre-injury than those who passed the test, lending support to the 'good old days' bias within a group of concussed individuals receiving compensation for their injury. This study appears to be the first to also demonstrate that this cognitive bias varies within concussed individuals, depending on whether or not they pass effort testing, with those failing such testing demonstrating a stronger 'good old days' bias.

Belanger et al. [63] recently examined the role of self-efficacy, knowledge about mTBI and attribution of symptoms to mTBI as factors that might account for additional variance in post-concussion symptoms beyond that accounted for by demographic variables and psychiatric predictors. While self-efficacy did not uniquely add information about the severity of persisting symptoms, the most significant predictor was attribution. Individuals who sustained an mTBI in the past 4 years, who believed their symptoms were due to their injury, were more likely to report greater overall symptom severity. (For more on the psychological theories of PCS, readers are referred to McCrea [64]).

**Psychosocial factors in sports-related concussions**

As discussed, sports-related concussions are a neurobiological injury. The majority of those who experience single and uncomplicated concussions recover within days to weeks, but there can be psychiatric and psychological effects, including from well-intended recommendations for very lengthy periods of cognitive and physical rest. As sports-related concussions typically occur within team sports, psychosocial factors can also complicate recovery. The invisible, often subjective aspect of sports-related concussions creates vulnerability to multiple sources of fear. These include, but are not limited to a fear of re-injury, fear of being perceived as weak, fear of losing or not achieving a desired role, fear of isolation or loss of affiliation with the team and even fear of losing financial stability (income or scholarship).

Despite the great strides in public education about the importance of safe management of concussions, the poverty of objective findings for sports-related concussion can exacerbate a pre-existing 'play through pain' mentality and amplify social pressure for athletes with sports concussion to return to full participation prematurely. Left to their own preferences, such athletes often want to fast track return to activity, but they are influenced by many other individuals. Explicit or implicit messages from teammates, coaching staff and other sport-specific personnel may add additional stress and anxiety during the recovery from concussion. Coaching staff may grow impatient, with recoveries lasting more than a few days and add to the pressure, implicitly playing on fears of losing a starting position. Traditional media and social media may also weigh with vocal fans and commentators discussing the status of concussion recovery, contributing to the anxiety and stress surrounding recovery.

These external pressures can easily influence vulnerable athletes with reduced cognitive resiliency for managing stress. Thus, it is critical for the evaluation of sports concussion, management of the recovery process and return to full participation in sport to be under the purview of independent healthcare providers. Bias, real or perceived, creates too many risks for premature return to activity. Additionally, it may also be critical to provide pro-active psychological and even social buffers to athletes during the concussion management process. This can support challenging misattributions, misconceptions and multiple forms of pressure to accelerate return. Providing education to athletes after concussion can be a form of treatment and can address potential cognitive distortions and misattribution related to concussion symptoms [65].

**Iatrogenic psychological effects of medical management**

The development of the Zurich protocol that grew from the International Consensus Conferences on Sports Concussion [7] was instrumental in developing individualized concussion management, but most importantly it emphasized the need for concussed athletes to be removed from competition or other activity and given a period of rest. While many child neurologists were unaware of the original Zurich protocol as recently as 2012 [66], the release of the new guidelines published by the American Academy of Neurology [67] and recent publicity has increased awareness of these practise guidelines. As promoted by the International Consensus Conference on Sports Concussion, the period of rest with gradual return to activity has provided a formalized method of insuring rest, as well as return to activity based on symptom presentation.

As with many concepts in clinical practise, the pendulum has now swung widely in the other direction such that some athletes with concussion are held out of all physical activity for lengthy periods of time, resulting in iatrogenic effects due to inactivity. In some scenarios, well-meaning clinicians have held athletes—normally used to intense physical activity—out of all physical activity for lengthy periods of time. It may be that many symptoms of PCS, such as feeling sluggish, slowed down, tired and foggy, may be prolonged or intensified by lengthy inactivity.

Griesbach et al. [68] at UCLA have done a series of studies using an animal model of concussion. Using a fluid percussion injury methodology, these researchers investigated the effects of exercise on rodents after they were concussed. The results showed that introducing exercise during the acute period of 0–6 days post-injury exacerbated cognitive impairments (based on the Morris Water Maze task), providing support for the Zurich model of an initial rest period to prevent exacerbation of symptoms. A key finding, however, is that introducing exercise during the post-acute phase 14–20 days after injury actually improved learning and memory. In addition, rodents who had received a sham injury also demonstrated improved performance with exercise. Griesbach’s [69] work demonstrated that voluntary exercise in rats leads to an increase in brain-derived neurotrophic factor (BDNF), which plays a key role in hippocampal neuronal plasticity. Notably, only negligible amounts of BDNF cross the blood–brain barrier and, thus, attempts to use medication to increase BDNF have been unsuccessful.
Another interesting finding in this line of research on exercise and recovery is that the optimal window of exercise after concussive injury is severity dependent in the rodent model [70]. Rodents who received a moderate fluid percussion injury had exercise introduced at either 0–6, 14–20 or 30–36 days post-injury. An increase in BDNF was found only in those exposed to exercise 30–36 days post-moderate fluid percussion injury, demonstrating the importance of a longer rest period for those who received a greater force of injury.

Treatment

Education and reassurance

While there are few treatments for the early stage of concussion recovery, it is notable that education and reassurance are highly effective for reducing PCS. In a review of outcome studies, Miller et al. [71] found that a single psychoeducational session is a key factor in preventing or shortening PCS. Providing supportive reassurance during one therapeutic session as well as education about the symptoms of concussion, the expected recovery course from concussion and emphasizing the appropriate attribution of symptoms to benign aetiologies, as well as the gradual resumption of activities were found to be highly effective in reducing symptoms.

Exercise and return to activity

The positive effects of exercise on human health have been known for many years. In a review article, Powell et al. [72] summarized multiple pro-health benefits of a variety of forms of exercise. They report the known effects of exercise to increase immune function, mitochondrial volume and coronary artery size and decrease inflammation and blood coagulation, among many other positive health effects. Exercise is also often recommended as a method of managing and treating depression [73] and sleep disturbance [74], both of which may be problematic after concussion.

Using a retrospective review of medical records, Majerske et al. [75] discovered that moderate levels of exercise after sports-related concussion were associated. Another study investigated the effects of exercise on a small number of children (n = 16) who had PCS 4 weeks after injury [76]. The majority of the paediatric participants reported headaches, fatigue, depression and poor endurance prior to the introduction of a supervised aerobic exercise protocol. Upon introduction of the exercise programme, all showed a rapid resolution of symptoms. A similar study in adults with PCS [77] enrolled six athletes and six non-athletes in an exercise protocol. The concussion injuries occurred due to sports, motor vehicle collisions or work-related falls. Using a standard Balke treadmill protocol, participants began an exercise programme in which they were asked to exercise at 80% of their maximum heart rate as established during a baseline, while being monitored and stopping upon symptom exacerbation. During the study, all participants reached the success criterion of exercising to or near their age-predicted heart rate maximum without symptom recurrence and resulting in a significant decrease in post-concussion symptoms. Notably, at 3-month follow-up, 10 of the 12 participants were symptom-free and all had returned to athletics, school and/or work. While many of these are very small studies, they are intriguing and the potential benefits of structured and supervised exercise during the post-acute concussion recovery period merits further investigation with large scale studies.

In an extensive review of the existing literature, Silverberg and Iverson [78] suggest that rest may not be the ‘best medicine’ after concussion. While noting that many professionals advise rest after concussion, there is no clear definition of exactly what this entails or for how long. This review also notes that the recommendation for rest is not evidence-based, but grounded in clinical assumptions. The authors also note the sub-group of patients with chronic PCS symptoms who might not achieve symptom-free status and that lengthy physical inactivity is likely to do more harm than good. Silverberg and Iverson state that ‘Rest may be the most prescribed medical intervention in history’ (p. 251). According to Silverberg and Iverson, ‘complete bed rest beyond a few days is sufficient to cause post-concussion-like symptoms and may exacerbate symptoms after mTBI’ (p. 252). They note that those individuals with persisting PCS at rest are likely disabled by a combination of physical inactivity, avoidance behaviours and limited mastery opportunities. All of these likely contribute to the cognitive distortions and misattributions that may prolong concussion recovery.

After this extensive review of literature on the prescribed use of rest to treat concussion, Silverberg and Iverson [78] made several evidence-based recommendations. Specifically, they stated that there is no evidence that complete rest (defined as ‘recumbence in bed and avoidance of cognitive stimulation’) of any length results in positive or negative outcomes after concussion and they note that the negative consequences of inactivity can occur after 3 days. They do state, however, that there is an increased risk of an additional concussion when athletes return to competition within 1 week of concussive injury and that, in some individuals, ‘vigorous’ exercise within 2 weeks of concussion might result in persisting symptoms, especially for individuals who sustain more serious injuries. With the exception of competitive sports, gradual return to activity is more likely to enhance than complicate recovery. Participating in regular exercise may be protective against the development of depression and anxiety after concussion. Sub-symptom threshold exercise appears to be safe and may be beneficial to concussion recovery. Silverberg and Iverson [78] also noted that physical or cognitive exertion can temporarily increase post-concussion symptoms at any point in recovery, but that it is unclear if there are any long-term consequences from brief increases in symptoms.

Introducing graded exercise protocols under the supervision of an athletic trainer or other healthcare provider may also provide an in vivo method for anxiety desensitization. As many individuals with PCS develop anxiety around their symptoms, receiving reassurance and parameters for safe exercise may reduce anxiety symptoms. Identifying clearly defined goals for exercise, including heart rate maximum, often enable athletes experiencing PCS to relax with the knowledge that they are exercising safely under supervision. While the initial introduction of exercise is often anxiety-provoking, the positive
mental and physical health benefits of exercise may result in a relatively quick reduction of anxiety and PCS symptoms.

For some individuals, cognitive-behavioural therapy to target and modify cognitive biases and misattribution, as well as to teach anxiety reduction techniques, may also aid recovery. Psychotherapy can also be useful in identifying psychosocial factors contributing to symptom presentation and the teaching of specific coping skills for dealing with psychosocial pressures. With the athlete’s permission, psychotherapeutic intervention may require providing education to coaches, athletic trainers and other personnel, as well as addressing co-morbidities such as attention deficit hyperactivity disorders (ADHD) that may complicate recovery [79]. Athletes with ADHD may impulsively resume activity before they have been cleared to do so and may end up prolonging their recovery. Addressing their low frustration tolerance and impulsivity in therapy and acknowledging the difficulty of sitting out from their sport is especially important with this sub-group of athletes.

A dysfunctional cognitive feedback loop has been proposed to explain PCS and which can be targeted during psychotherapeutic intervention [80]. In this model, the initial concussion symptoms disrupt cognition and, in vulnerable individuals, these transient symptoms can cause anxious and fearful reactions, which can cause further cognitive disruption. This alternating pattern of cognitive slips followed by anxiety which exacerbates cognitive disruption creating greater levels of anxiety can create a dysfunctional feedback loop. The proposed treatment starts with validating the patient’s symptoms and sense of self without addressing their aetiology [80]. Next, therapy should progress to a functional analysis of the cognitive breakdowns and identifying factors contributing to dysfunction. The next phase of therapeutic intervention focuses on re-establishing a new and effective sense of self by focusing on gaining control over symptoms through compensatory strategies and modifying emotional responses.

For individuals with refractory post-concussion depression, psychopharmacologic treatment may be needed if they do not fully respond to psychotherapeutic interventions. Selective serotonin re-uptake inhibitors (SSRIs) have been suggested as the most likely category of medications to be effective in this population [81]. Other medication options include serotonin-norepinephrine re-uptake inhibitors (SNRIs), tricyclic antidepressants, novel antidepressants such as trazodone and psychostimulants.

Summary and conclusions

In summary, animal models of concussion and mTBI injury suggest that the injury itself can trigger anxiety and fear reactions and some vulnerable individuals may be at risk of neurobiological depression and/or anxiety. The pathophysiology of depression following a concussion appears to be consistent with the cortico-limbic model of depression. The literature also demonstrates that pre-morbid and concurrent anxiety increases the risk for prolonged concussion recovery. Some concussed individuals have anxiety sensitivity that predisposes them to be very focused on physiological symptoms, which creates additional anxiety related to misattribution of symptoms. Thus, anxiety sensitivity may be a risk factor for PCS. Numerous studies have identified cognitive biases, including the ‘good old days’ bias, which contributes to lengthy recovery from concussion. In addition, well-meaning healthcare professionals may contribute to protracted PCS by prescribing excessive amounts of cognitive and physical rest. A treatment model that incorporates supervised and graduated physical activity, the introduction of anxiety reduction techniques and cognitive-behavioural therapy of cognitive biases and misattribution, as well as identifying and teaching skills for coping with psychosocial factors complicating recovery, is likely to be the most efficacious means of returning concussed athletes back to activity and back to full participation in their daily life.

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Declaration of interest

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