

Symptom Attribution, Injury Severity, and Catastrophic Ideation in Postconcussional Disorder

by

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Abstract

Background: Previous researchers have suggested that catastrophic misinterpretation of benign experiences or catastrophic reactions to minor injury may be the predominant risk factors for chronic post-concussion symptoms. The “Expectation as Etiology” (Mittenberg et.al., 1992) and “Good Old Days” (Gunstad and Suhr, 2004) models were both developed in support of this view. Despite being widely cited and broadly accepted, neither model has been adequately tested in non-litigating individuals with chronic post-concussion symptoms. The current study investigates the utility of these models in predicting chronic post-concussion symptoms and impairment due to concussive injury. Individuals with a history of concussion reported their current symptoms, and also rated their symptoms prior to the experience of concussive injury. Comprehensive information about emotional and physical health, background information, injury history, health anxiety and anxiety sensitivity were also collected. In contradiction to predictions made by the models, individuals with chronic post-concussion symptoms did not report the experience of fewer symptoms prior to injury than those who had recovered from their injuries. Further, regression modelling indicates that the current emotional distress of the respondent is the strongest predictor of self-rated impairment, over and above all other predictors, including measures of catastrophic ideation. These results are inconsistent with the idea that chronic symptoms are mainly maintained by hypochondriacal traits, catastrophic ideation, or misinterpretation of benign symptoms. As such, the utility of the “Good Old Days”, and “Expectation as Etiology” models for explaining chronic post-concussion symptoms is questioned. These results suggest that current symptom report is not due to misattribution of previously existing symptoms, or catastrophic ideation, and that the strongest predictor of self-rated impairment due to injury is the current emotional distress of the respondent. These findings have clear relevance for the design of treatment protocols, and suggest that targeting current emotional distress may be more efficacious than attempts to address catastrophic ideation in individuals experiencing chronic post-concussion symptoms

Keywords: Concussion; Good Old Days; Symptoms; Post-Concussional Disorder; Expectation as Etiology, Anxiety Sensitivity

*For Sarah and Will,
the best little family a guy could ask for.
I used your eyes to see what was possible
when mine were blinded by doubt,
leaned on you when I was weary,
and stood on your shoulders when
I needed to see what was over the next hill.
Without your endless patience, sacrifice and
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List of Acronyms

AS	Anxiety Sensitivity
GCS	Glasgow Coma Scale
LOC	Loss of Consciousness
PCD	Post Concussional Disorder
PCS	Post-Concussion Syndrome
PCSC	Post-Concussion Symptom Checklist
PTA	Post Traumatic Amnesia
TBI	Traumatic Brain Injury

Chapter 1.

Introduction

Recovery from concussive injury in general, and disordered recovery in specific, has been a highly controversial topic in the research literature for many years. The goal of this dissertation is to contribute to the understanding of this controversy, and to provide information that will be useful in resolving it. This work was conducted to help clarify our understanding of the risk factors and correlates of chronic post-concussion symptoms as they relate to the expression of impairment following a concussive injury. As will be illustrated in the following review, several factors have contributed to this controversy, including disagreements over the nature and definition of the injury itself, disagreements about the relative influence of risk factors, and the nature and etiology of symptoms. On a broad level, much of the controversy has been driven by philosophical differences between researchers. Some have asserted that chronic symptoms may be due to subtle changes in brain function as a result of injury, while others posit that chronic symptoms are purely “psychological” in nature and have very little to do with injury. Within both camps, there is disagreement about the risk factors and correlates of symptom development and chronicity. One such disagreement concerns the role of traits such as vulnerability to catastrophic ideation. Some researchers have suggested that these traits are predominantly responsible for the development and maintenance of chronic symptoms, while others disagree. This research has been specifically designed to shed light on this issue. Obtaining a clear understanding of these issues is vitally important, in that effective treatments for chronic symptoms have not yet been developed, and treatments are likely to vary based upon the identification of the most important predictors of chronic symptoms.

In order to achieve the goal of understanding chronic post-concussion symptoms, it is first important to conduct a thorough review of the topic. The review that follows will help clarify the issues at hand by placing the controversy in historical context, considering

nosological and methodological concerns, and reviewing the results of previous research with regard to the risk factors and correlates of post-concussion symptoms. This review will provide a knowledge base about the condition and what is known about it, and allow for critical thinking about the issues at hand. Then the “Good Old Days” and “Expectation as Etiology” models will be reviewed as explanations of the etiology of chronic symptoms. Through this review it will be apparent that the assumptions of these models have not been adequately tested, and that the current study may help us to better understand chronic-post concussion symptoms.

Historical Perspectives on Concussion Research

The study of concussion has deep historical roots. Although the amount of research on the subject has grown exponentially, especially within the last decade, there is still much to be learned. Many questions regarding the origin and proper classification of concussions and their symptoms have not yet been answered satisfactorily. The following review places the study of concussion within historical context in order to provide a better understanding of the controversies and major issues surrounding the topic. This review also provides a broad survey of attempts to classify and define the condition, in order to clarify the usefulness of these definitions in the scientific study of concussion.

The word concussion comes from the Latin *concussionem*, which developed from the stem *concutere*. The Latin stem is itself made up from *com*; meaning “together”, and *quatere*, which means “to shake”. Thus, the etymology of the term as we use it today can be translated as “to shake violently” or “to dash together” (Pearce, 2008). Observations of alterations in consciousness due to injury are among the oldest in modern medical science. Observations of the effects of head injury in general, and attempts to understand and classify the outcome of differing levels of injury are represented throughout recorded history. There is a general consensus that the Edwin Smith papyrus contains the first description of alterations in mental state, such as transient loss of consciousness and confusion. In addition to the original author's text (3000– 2500 BC), the papyrus contained a commentary added a few hundred years later which described 27 cases of head trauma (Breasted, 1930). Hippocrates described the

effect of non-penetrating head injury in “De Vulneribus Capitis” (Jones, 1995); which states “Shaking or concussion of the brain produced by any cause inevitably leaves the patient with an instantaneous loss of voice (read: unconsciousness; Aphorism no. 58, Section 7). In the 10th century, AD, the Persian physician Muhammad ibn Zakarīya Rāzi described head injuries that resulted in a transient alteration in consciousness without observable physical damage (McCrorry and Berkovic, 2001). Berengario de Capri (1518, as cited in Denny-Brown, 1941) and Ambroise Paré (1634, as cited in Denny-Brown, 1941) both provided descriptions of injuries incurred in the absence of skull fracture. These authors used the term “commotio cerebri” as a descriptor of these kinds of injuries. “Commotio” is derived from the latin stem commovere, which can be translated as “to throw into disorder” (Pearce, 2008). In 1674, Boirel observed that commotio cerebri was different from other cerebral trauma because of the limited duration and intensity of symptoms (Boirel, 1674, as cited in Denny-Brown, 1941). Boirel noted that the symptoms elicited after concussions were inconsistent with the outcomes he observed in connection to injuries that resulted in grossly observable cerebral lesions (Denny-Brown, 1941). Others described injuries with significant functional impairment, including death, in the absence of observable haemorrhage or structural lesions (Littré, 1705; Vance, 1927; as cited in Pearce, 2008). Thus, the historical definition of concussion has long been inconsistent with lasting structural damage or permanent “brain injury”. The idea that brain function could be disrupted by injuries that did not result in observable changes in brain structure was being experimentally tested as early as the 19th century (Pearce, 2008). These early investigations included the use of animal models to observe the effects of administering repetitive low impact blows to the skull that did not produce observable damage to brain structures (Koch and Filene, 1874; as cited in Pearce, 2008). Of course, these researchers often produced injuries that resulted in death and relied on autopsy evidence. As such, these early researchers were likely observing pathology greater than that expected from concussion today. They also lacked modern technologies with which to examine microstructural damage.

As the industrial revolution matured, basic researchers continued to conduct autopsy based investigations for the anatomical correlates of injuries of varying severity. Increased industrialisation and the propagation of rail travel were associated with injuries of all sorts. Individuals began experiencing injuries as a result of working more closely

with machines. Of particular interest, a subset of individuals began complaining of symptoms following injuries to the head and neck that appeared mild to the objective observer. In the late 1800's, John Erichsen observed commonalities between individuals who reported symptoms as a result of injuries sustained through indirect impulsive forces (Erichsen, 1867). Erichsen described these patients as suffering from blunt trauma, falls or what we might now label as "whiplash" type injuries as a result of railway shunting accidents. Although the mechanism of injury was somewhat different in many cases, Erichsen observed that these individuals presented with a relatively similar constellation of symptoms, which suggested a similar etiology. The label given to the injury sustained in these cases was "railway spine". Erichsen attempted to explain how minor injuries to the head and spine could result in catastrophic outcomes by hypothesizing "molecular disarrangement" in the central nervous system or "anemia" of the spinal cord (Erichsen, 1867).

Armed with Erichsen's description of the ailment, the medical community became aware of increasing numbers of individuals who presented with symptoms similar to "railway spine", even in the absence of actual contact with a railway. As these injuries were often associated with industry, individuals began seeking compensation for their injuries from the railways and other industrial agents. Given the poor understanding of the condition at the time, and in light of the empirical evidence that similar injuries were not associated with observable changes in brain structure, controversy arose with regard to the veracity of these kinds of injury. Indeed, several experts in the field suggested that "compensation seeking" might be a better explanation of the problem. Wordsworth (1881) commented on this view by stating that most physicians "believed that all recovered on the settlement of their claims, and had resumed their wonted occupation" (Ophthalmological Society of the United Kingdom, 1881). Further, this controversy was reflected in Erichsen's statement that; "There is indeed no class of cases in which medical men are now so frequently called upon to give evidence in the courts of law, as those which involve the many intricate questions that arise in actions for damages against railway companies for injuries of the nervous system, alleged to have been sustained by passengers in collisions; and there is no class of cases in which more discrepancy of surgical opinion may be elicited" (Erichsen, 1882 p17).

Thus, the debate surrounding the “organic” versus “psychogenic” cause of the syndrome was born, with some arguing for a biological explanation and others suggesting that the symptoms were representative of “general nervous shock”, “functional disorders”, or “compensation neurosis” (Page, 1883; Rigler, 1879). Others proposed terms such as “traumatic neurosis” (Oppenheim, 1892; as cited in Holdorff and Denning, 2011) or explained the symptoms in terms of “hysteria” or “neurasthenia” (Charcot, 1889; as cited in Goetz, 2001). In 1892, however, Friedmann (1892; as cited in Gasquione, 1998) noted that many of the symptoms reported were also associated with hemodynamic factors and proposed the term “vasomotor symptom complex” to suggest that alterations in blood flow within the brain were the cause of the syndrome.

Even at that early stage, however, some theorists were able to see beyond the simplistic “organic” versus “psychogenic” view. These rational empiricists suggested that the two sides of the debate are in fact indistinguishable as all things “psychogenic” must be a product of the “organic”. This view is summarised by Hodges (1881) who rejected any suggestion that “railway spine” had anything to do with the spine, and suggested that this was a disorder of brain functioning when he wrote; “For this state of affairs¹ the retention in medicine of the term ‘spinal concussion’ is certainly in part responsible in that it satisfies in a measure the imagination, and excuses the rational explanation of the symptoms which are brought to his notice...it is probable that in the production of many of the hysteroid symptoms it is a disturbance of cerebral rather than spinal functions which is at fault...” (p. 364). This view is again reiterated by Walton (1883), who observed that “. . . these symptoms are rarely unattended by irritability, fretfulness, emotional tendency, and inability to confine the attention. These can only be the result of derangement in the higher cerebral centres” (p.338). The acknowledgement that just as emotions are the product of brain function, emotional symptoms can be conceived as a product of brain dysfunction is a critical one. Although these ideas have been known for well over 120 years, debate continues as to the importance of this assertion.

The first use of the term “postconcussion syndrome (PCS)” is credited to Strauss and Savitsky (1934), who wrote: “In our opinion, the subjective post-traumatic syndrome,

¹ Hodges is referring to the adversarial nature of the debate and to the polarization of experts caused by increasing litigation.

characterized by headache, dizziness, inordinate fatigue on effort, intolerance to intoxicants and vasomotor instability, is organic and is dependent on a disturbance in intracranial equilibrium due directly to the blow on the head. We suggest the term “postconcussion syndrome” for this symptom complex.”(Strauss and Savitsky, 1934; as cited in Evans, 2010) Although this historical definition is somewhat limited and incomplete in comparison to modern definitions, it is again acknowledged that even though the “syndrome” is characterized as “subjective”, the symptoms are nonetheless thought to be the result of “organic” changes in brain function as a result of injury.

In support of the influence of organic injury on the expression of post-concussion symptoms, it has long been known that individuals who have sustained concussions are acutely vulnerable to further injury. For this reason, concussion has been identified as a cause for concern in sports for many decades. This idea is clearly evidenced by the following statement, made by the American Football Coaches Association in 1937: “During the past 7 years the practice has been too prevalent of allowing players to continue playing after a concussion...Sports demanding personal contact should be eliminated after an individual has suffered a concussion.” Further, the so called “three strike rule”, wherein an athlete who experiences three concussions in a season is out for the season in order to avoid poor outcomes was conceptualised by Dr. Quigley, a sports physician at Harvard in 1945 (Thorndike, 1952). It is interesting to note that issues surrounding the role of concussive injury in sport remain relevant and largely unresolved sixty to seventy years after these statements were made. However, recent developments in professional sports associations indicate that this may be changing, and that there is a growing acceptance of the potential consequences of concussion in athletes.

Arguments over the nature and legitimacy of lasting symptoms following a concussion continue to this day. This argument is no doubt fuelled to some degree by the adversarial nature of the legal system, which requires experts for both defence and prosecution to settle matters of compensation. The viewpoints on either side of the debate have not changed substantially, although the evidence available for presentation by either side has increased. For example, in the 1950’s medical-legal texts on the subject of concussion provide “descriptive references to the symptomatology of

concussion from legends, folktales and history” (Courville, 1953), which would not be acceptable in today’s courtrooms.

The debate surrounding the legitimacy of post-concussion symptoms continued through the sixties and it is clear that over time the disagreements in the literature became more and more polarized. In 1961, Miller summarized the viewpoint of those who believe that PCS is really a “compensation neurosis” when he wrote: “The most consistent clinical feature is the subject’s unshakable conviction of unfitness for work....” (Miller, 1961), while Symonds (1962) took an equally strong opposing position in 1962 when he wrote, “It is questionable whether the effects of concussion, however slight, are ever completely reversible.” A beneficial side effect of such heated debate was the identification of the need to clearly define what a concussion is, as opinions were highly dependent upon nosology. One of the earliest attempts at a modern definition of concussion was made in 1966 by the Congress of Neurological Surgeons (Committee on Head Injury Nomenclature of the Congress of neurological surgeons, 1966). In that definition, concussion was defined as “a clinical syndrome characterized by immediate and transient impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces.” It is noted that this definition does not make reference to emotional or psychiatric consequences, favouring classification based upon alterations to the sensorium alone. This was likely due to a desire to focus on objectively identifiable signs and symptoms, and discomfort with poorly understood subjective or emotional symptoms within the medical and neuropsychological community at the time.

In the 1970’s the ability to respond quickly to injury became a salient factor in the progression of understanding and treating the effects of concussion. It has been suggested that the ability to use helicopters to evacuate injured soldiers during the Vietnam War and the concomitant reduction in mortality rates lead to an adoption and implementation of rapid emergency response for individuals with head injuries in North America (Ruff, 2005). During the same time period, trauma physicians became more sophisticated in their ability to triage injuries, perhaps due to the increased demand for emergency services. A scale that had been developed in Glasgow, the Glasgow Coma Scale (Teasdale and Jennet, 1974), was beginning to be used across the United States to classify the severity of traumatic brain injury (TBI). This scale allowed for repeated

ratings of severity at the scene, during transport, in the emergency department (ED), and throughout hospitalization. Although the focus of the scale was on the detection and management of severe brain traumas, by providing a rating from three to fifteen based upon level of consciousness and responsiveness to stimuli, the GCS also included a classification for "minor head injuries." Scores that fell between 13 and 15, in the early phases of recovery, were indicative of these "minor" head injuries (Teasdale and Jennet, 1974). Although these milder injuries went largely untreated and were often viewed as insignificant, the adoption of a common severity rating scale allowed for the recording of epidemiological data pertaining to the incidence of these injuries for the first time.

Given the prevalence of injuries observed in medical databases as a result of better recording practices, interest in the pathophysiology of mild head injuries was again sparked. Animal models began to emerge based on observations of diffuse axonal injury as a result of rotational injury (Gennarelli et al., 1982).² Although the debate surrounding the significance of animal studies and early neuroimaging findings continues (see below), these studies were very important in bringing the attention and focus of the scientific community back to the effects of "mild" injuries. The suggestion that objective evidence for structural damage could be found using modern imaging led to increased interest in quantifying the extent of the damage and enhanced the legitimacy of the study of post-concussion sequelae.

With the introduction of managed care in the United States in the 1990's, outpatient services and rehabilitation were aggressively cutback. Concurrent to the cutbacks in available rehabilitative services, individuals complaining of post-concussion symptoms began entering litigation at an increasing rate (Dungworth and Pace, 1990; Gresenz et al., 1999). As such, the need for well-informed assessment of post-concussion symptoms based upon empirical research increased dramatically (Schatza et al., 2001). At the same time, George Bush declared the nineties to be the "Decade of the Brain" (Bush, 1990) and resources were made available for brain research at all levels. This increase in funding further added to the dramatic increase in research into the topic of

² These early models are perhaps best characterised as investigations of more severe TBI, despite being often cited as investigations of "mild" injury. Although the injuries produced did not involve penetrating injuries, skull fracture, or direct impact; the forces applied were not consistent with concussion.

concussion. Since that time, research on concussion, its effects, risk factors and proposed etiological models has grown rapidly. The following sections will focus on clarifying what is known and what remains to be understood about this complex topic. These sections will begin with an examination of terminology and nomenclature in order to clarify the nature of the injury of interest to the current investigation. The discussion of how best to classify these injuries naturally leads to a review of neuropathological investigations of the effects of concussions. This review will focus on investigations of the interplay between changes in brain function and the expression of symptoms. Next, the epidemiology and societal impact of concussion is examined, and the scope and magnitude of the problem posed by concussions will be illustrated. After clearly defining the injury, its pathology, incidence, prevalence, and impact; the discussion will turn to a consideration of natural course and recovery from these kinds of injury. Then various attempts to classify pathological recovery will be examined. A broad review of risk factors for poor recovery will then be conducted. Finally, the discussion will turn to a consideration of theories which have attempted to explain the genesis and maintenance of chronic symptoms, and the reasons for this study.

Nomenclature/Definitions

Over the past two decades of intense research on the subject, numerous definitions have been proposed to classify the nature of the injury. This difference in definitions has no doubt contributed to the difficulties in establishing good epidemiological data and replicating findings. These definitions have differed on their criteria for severity, as well as the suggestion of “Brain” versus “Head” Injury. Indeed, “Brain Injury” and “Head Injury” are often used interchangeably in the literature. It is argued that this is done at the risk of nonspecificity and confusion of the relevant factors. As previously stated, nosology is critical to better understanding and comparison of research results. In the review of classifications presented below, it is argued that the term “concussion” is likely best. Table 1 presents a sample of definitions including the term “Head Injury”.

Table 1. Definitions Including the Term “Head Injury”

Source	Term	Clinical Symptoms	Exclusion
Neurotraumatology Committee of the World Federation of Neurological Societies (2001)	"Low Risk Mild Head Injury"	GCS = 15	no LOC, PTA, vomiting or headache
	"Medium Risk Mild Head Injury"	GCS = 15 and one or more of: LOC, PTA, vomiting, or diffuse headache	skull fracture
	"High Risk Mild Head Injury"	GCS = 14-15 and one or more of: skull fracture, neurological deficits, coagulopathy, drug or alcohol consumption, history of neurosurgery, pre-trauma epilepsy, age over sixty	none
Williams et al., 1990	"Complicated Mild Closed Head Injury"	GCS 13-15; observable brain lesion and/or depressed skull fracture	lack of imaging evidence
	"Uncomplicated Mild Closed Head Injury"	GCS 13-15 and linear or basal skull fractures (not necessary)	no evidence of abnormality on CT or skull x-ray

Note: GCS = Glasgow Coma Scale; PTA = Post Traumatic Amnesia; LOC = Loss of Consciousness.

In consideration of the data presented in Table 1 above, the definition suggested by the Neurotraumatology Committee of the World Federation of Neurosurgical Societies (Servadei, Teasdale and Merry, 2001) is indicative of some degree of ambiguity. This classification system is highly inclusive and nonspecific, and allows events ranging from those associated with no symptoms at all to those with skull fractures, hematoma's and neurological impairments to be classified as “Head Injuries”, so long as the patient is relatively responsive, as indicated by their GCS score.

Along the same lines, some have suggested that the integrity of the skull is the most salient factor in determining the severity of injury. The term “Closed Head Injury” (Williams et al., 1990) was proposed as a result of this idea. As seen in Table 1, this definition is again somewhat broad and nonspecific. It is interesting to note that, even in the presence of a documented brain lesion, this classification system refers to “Head Injury”, so long as the GCS score remains above 13.

Table 2. Definitions Including the Term “Traumatic Brain Injury”

Source	Term	Clinical Symptoms	Exclusion
Menon et al., 2010	Traumatic Brain Injury	alteration in brain function, or other evidence of brain pathology	none
American Congress of Rehabilitation Medicine, 1991	MTBI	GCS 13-15 and one or more of: any LOC < 30 min., PTA < 24 hours, any alteration in mental state	LOC > 30 min., PTA > 24 hours
National Center for Injury Prevention and Control, 2003	MTBI	GCS 13-15 and any alteration in mental status, LOC < 30 min., and one or more of: seizures, irritability, lethargy, vomiting, headache, dizziness, fatigue, poor concentration	LOC > 30 min.
World Health Organisation Collaborating Centre for Neurotrauma Task Force, 2005	MTBI	GCS 13-15 and one or more of: LOC < 30 min., PTA < 24 hours, focal neurological signs, seizures, intracranial lesions not requiring surgery	symptoms not due to drugs, alcohol, medications, other injuries, or treatment for other injuries, penetrating craniocerebral injury, "other problems"
European Federation of Neurosurgeons, 2011	Category 0 MTBI	GCS 15	Any "Risk factors"
	Category 1 MTBI	GCS 15; LOC < 30 min., PTA < 1 hour	Any "Risk factors"
	Category 2 MTBI	GCS 15; Any "Risk factors"	none
	Category 3 MTBI	GCS 13-14; LOC < 30 min., PTA < 1 hour, "Risk factors"	none
Levin et al., 1987	Complicated MTBI	positive CT or MRI	negative CT or MRI
	Uncomplicated MTBI	negative CT or MRI	positive CT or MRI
Borgano et al., 2003	Complicated MTBI	space occupying lesions, LOC, more cognitive than emotional symptoms	none specified
	Uncomplicated MTBI	brief or no LOC, more emotional than cognitive symptoms	no evidence of space occupying lesions

Note: GCS = Glasgow Coma Scale; PTA = Post Traumatic Amnesia; LOC = Loss of Consciousness; MTBI = Mild Traumatic Brain Injury

* Risk Factors include: unclear accident history, continued PTA, retrograde amnesia >30 min.,

trauma above clavicles, severe headache, vomiting, focal neurological deficit, seizure, age <2, age>60, coagulation disorder, "high energy" accident, intoxication with alcohol/drugs.

Table 2 (above), presents a summary of definitions that include the term "Traumatic Brain Injury". With regard to the distinction between definitions that focus on head injury as opposed to brain injury, Kay et al. (1992) offered the following clarification: "head injury is defined as an injury to any part of the head (eg, face, skull) whereas brain injury denotes damage to the brain". It is noted that these terms do not refer to the same pathological processes, insofar as an individual can sustain an injury to the head without the brain being injured, while the converse is also possible. However, due to its exclusion of the brain, it would seem as though the term "head injury" is insufficient to describe an injury that results in any alteration of mental state, confusion, emotional symptoms or post traumatic amnesia. As such, the term "Mild Traumatic Brain Injury" has gained acceptance, although several definitions have been proposed using this label as well.

In consideration of the use of the term "traumatic brain injury" (TBI); the definition of TBI proposed by the Demographics and Clinical Assessment Working Group of the International and Interagency Initiative toward Common Data Elements for Research on Traumatic Brain Injury and Psychological Health is presented at the top of Table 2 (Menon et al., 2010). Using this broad definition, which does not specify the extent, duration, or lower limits of "alteration in brain function", it would seem permissible to include the term "Brain Injury" in the classification of any event that alters brain function. This is likely misleading, however, given what is currently known about the nature of neuroplastic responses to environmental stimuli, which suggest that the brain alters its function quite regularly in response to external forces without any injury or significant trauma having occurred (Kays, Hurley, and Taber, 2012).

As further outlined in Table 2, the definition provided by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (1991) does not actually require objectively identifiable markers of brain damage. Thus, no evidence of "brain injury" is necessary, despite the use of the term "Brain Injury" in the definition. This definition also lacks a

lower limit for length of alterations in consciousness, which is a common difficulty in definitions.

The definition offered by the Centers for Disease Control and Prevention (National Center for Injury Prevention and Control, 2003) also uses the term “brain injury”. Again, as illustrated in Table 2, this definition does not reference specific brain damage, although it does make reference to acute symptoms beyond alteration in consciousness.

The criteria suggested by the World Health Organisation Collaborating Centre for Neurotrauma Task Force (Holm, L., Cassidy, J.D., Carroll, L.J., Borg, J. 2005), places more emphasis on brain damage. As seen in Table 2, this definition references the possibility of lesions or neurological abnormalities, and makes statements regarding differential diagnosis based upon ruling out factors unrelated to injury. It is interesting to note that this definition prohibits skull fracture.

The graded classification system proposed by the European Federation of Neurosurgeons (Vos et al., 2011), is also presented in Table 2. This classification system proposes that severity varies according to length of PTA and LOC, as well as one to two point shifts in the GCS rating. Also noted is the influence of risk factors, some of which were classified as exclusion criteria in other definitions.

Part of the difficulty with labelling such events as “Traumatic Brain Injury” is that this choice of nomenclature has led some to rely on evidence of objective, or observable injury in order to specify the significance of the event. Indeed, various authors have attempted to categorize these types of events according to the nature and extent of brain damage incurred. These definitions that rely on neuroimaging data are also presented in Table 2. Both Levin and colleagues (Levin et al., 1987), and Borgano et al. (2003), proposed classification systems based upon the ability to visualise structural lesions or hematoma. In their early attempts at defining the condition, Levin et al. (1987) did not include any consideration of functional symptoms at all. This is contrasted somewhat by the newer neuroimaging based definition offered by Borgano et al. (2003), who included some reference to cognitive and/or emotional symptoms. However, it is unclear how Borgano et. al, (2003) arrived at their decisions regarding the severity of injuries. As seen in Table 2, the distinction between “complicated” and “uncomplicated” injury

appears to vary as a function of the preponderance of cognitive or emotional symptoms. Given the emphasis that this definition places on observable “brain injury”, the severity distinction offered by Borgano et al. (2003) reflects a sense that neurocognitive findings are indicative of more “brain injury” than emotional symptoms. There is little evidence to suggest that this is the case. Indeed, emotional and social difficulties are well known and widely accepted sequelae associated with all levels of brain injury (see Jennekens, de Casterle and Dobbels, 2010, for review).

Each of the preceding definitions would appear to vary as a function of the ideological stance taken on the nature and severity of these kinds of injuries. While nomenclature that cites the head and skull as foci of interest would seem to downplay the emotional and neurocognitive effects of injury, the inclusion of the term “Traumatic Brain Injury” (TBI) or even just “Brain Injury” connotes a level of severity that is likely not warranted in most cases. The vast majority of injuries that result in GCS scores greater than 13 are relatively non-traumatizing to the physical or structural neural substrate and do not produce observable gross structural injury (Ruff, 2005).

The inclusion of the term “Traumatic Brain Injury”, then, is likely best reserved for instances in which structural damage is readily observable. This approach is likely not useful for the majority of cases in which an individual experiences a transient alteration in consciousness followed by typically self-limiting symptoms. Regardless of nomenclature, these kinds of injuries are almost always diagnosed according to a clinical interview that evaluates LOC and PTA, as well as subjective cognitive, emotional, somatic and/or neurological symptoms (Alexander, 1995). Emphasis on observable brain injury and positive neuroimaging as a component of any diagnostic classification is likely premature, since a majority of patients who report similar injuries are not routinely exposed to neuroimaging. Neuroimaging is not generally recommended for patients who report injuries that result in transient alterations in consciousness, unless accompanied by significant loss of consciousness (Ruff, 2005; Giza et al., 2013). Also, it would be clinically not reasonable and also cost prohibitive to require neuroimaging before a diagnosis is rendered (Ruff, 2005). Thus, definitions are required that neither overemphasize neuropathology, nor downplay the role of the brain function. It has been suggested that definitions that focus on clinically identifiable injury parameters such as

PTA, LOC, as well as the severity of cognitive, affective, and somatic symptoms are likely best (Alexander, 1995).

The historical term “concussion” is more often used in the sports medicine community, whereas MTBI is sometimes the preferred term in other medical specialties (Tator, 2009). Many authors use the term “concussion” in reference to injuries that result in only transient neurological deficits. Others have argued that the term concussion should be used to place an emphasis on impaired functional status following injury, and that “mild brain injury” should be used to place an emphasis on subsequent pathophysiology (Anderson et al. 2006). DeMatteo et al. (2010) found that using the term concussion when a patient is admitted to the hospital may downplay the functional consequences of injury, resulting in less than adequate follow-up with appropriate healthcare providers. Therefore, recommendations were made for using “MTBI” instead of “concussion” (DeMatteo et al. 2010). However, others have noted that the use of the term “brain injury” may be likely to induce anxiety which has been associated with poor outcomes (Weber and Edwards, 2010; Suhr and Gunstad, 2005).

For the reasons outlined above, a recent consensus statement on concussion in sports suggested that the term mild traumatic brain injury (MTBI) and concussion ‘referred to different injury constructs and should not be used interchangeably...’ (McCrary et al., 2009). Concussion is distinguished from more severe injuries, such as Moderate and Severe Traumatic Brain injuries by the exclusion of evidence of abnormality on standard structural neuroimaging studies, as well as the relatively milder and generally self-limiting presentation of acute symptoms. In this way, Concussion is defined as a functional, rather than structural injury (McCrary, Meeuwise, Johnston, et. al, 2009). However, numerous definitions using the term “concussion” have also surfaced. Table 3 (following page) presents several definitions of injury which include the term “concussion”.

In consideration of the information presented in Table 3, it is clear that each of the definitions proposed primarily stipulates the existence of transient alterations in mental state as a result of injury. It has been suggested that a concussion can be said to have occurred when impact, acceleration/deceleration, or rotational forces to the head lead to alteration of mental state (including feeling dazed, confused, disoriented) at the time of

the incident. There is also a general consensus that GCS scores above 13 are consistent with the acute effects of concussion. (American Congress of Rehabilitation Medicine, 1993). The American Academy of Neurology, Colorado Medical Society Guidelines suggested a graded severity rating based upon the duration of alteration in consciousness or the existence of LOC (Colorado Medical Society, 1991). The usefulness of this classification system has been called into question, however, as duration of PTA and LOC have been shown to be poorly associated with recovery (Caroll et. al, 2004; Ponsford et al., 2000).

Table 3. Definitions Including the Term “Concussion”

Source	Term	Clinical Symptoms	Exclusion
American Congress of Rehabilitation Medicine, 1993	Concussion	alteration in mental state, GCS >13; PTA <24 hours, LOC (if any) < 30 min.	PTA >24 hours, LOC > 30 min.
American Academy of Neurology, 1991	Grade 1 Concussion	transient alteration in mental state, post-concussion symptoms	no LOC, mental state abnormalities and symptoms >15 min..
	Grade 2 Concussion	transient alteration in mental state, post-concussion symptoms >15 min.	no LOC
	Grade 3 Concussion	transient alteration in mental state (any length), LOC	none
McCrory et. al, 2009	Concussion	transient impairment in neurologic function, graded set of clinical symptoms that may or may not involve LOC	no abnormality on standard structural imaging
American Academy of Neurology, 2013	Concussion	memory, orientation, may involve LOC	none specified

Note: GCS =Glasgow Coma Scale; PTA = Post Traumatic Amnesia, LOC = Loss of Consciousness

The Concussion in Sports Group, (McCrory et al., 2009) defined Concussion as a “Complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces” which “may be caused by either direct blow to the head, face, neck or elsewhere on the body with an “impulsive” force transmitted to the head”. They went on to stipulate that this force “typically results in the rapid onset of short-lived impairment of neurologic function that resolves spontaneously, may result in

neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury.” This group went on to suggest that the forces involved do not result in “abnormality on standard structural neuroimaging studies” but do result in “a graded set of clinical symptoms that may or may not involve LOC” and that “resolution of the clinical and cognitive symptoms typically follows a sequential course however it is important to note that in a small percentage of cases, post-concussive symptoms may be prolonged” (McCrory et al., 2009).

Finally, in a recent update of their guidelines for the management of concussion in sport, the American Academy of Neurology (2013) emphasized the role of memory and orientation difficulties as potential indicators of concussion. It should be noted, however, that this update does not include a formal definition as such. These statements are likely best viewed as addendums to their previous definition.

The term “concussion” is preferred in the current work. For the purposes of this research, the necessary conditions for concussion are defined according to the criteria proposed by Mc Crory et al. (2009). This definition is inclusive enough to capture the multiple mechanisms through which concussions are sustained, as well as the heterogeneity of experiences reported following these types of injury. With regard to further operationalization of the term, the exclusion criteria suggested by the American Congress of Rehabilitation (1993) are also employed. Thus, concussion is defined as the occurrence of an event such as is described by McCrory et al. (2009), that results in transient clinical symptoms, less than 24 hours of PTA, and less than 30 minutes of LOC, if any. Having reviewed various nomenclatures and classification for concussive injury in order to select the most appropriate term for the current research, the discussion now turns to the incidence, prevalence and impact that these injuries have on society.

Epidemiology and Societal Impact

As previously stated, the diversity of definitions regarding these kinds of injuries has contributed to difficulties with obtaining clear epidemiological data. Given the fact that estimates are likely to vary as a result of nomenclature and idiosyncratic definitions,

epidemiological data must be viewed with these caveats in mind. Where possible, the following review utilises objective data such as GCS scores, and length of hospitalisation as indicators of concussion. At times, educated estimates are made based upon data gleaned from recorded incidents of moderate and severe brain injuries. This is because data pertaining to more severe injuries is recorded more reliably than data pertaining to mild events, due to the potential involvement of the medical system. Estimates about the rate and impact of concussions that are made in this way will be imprecise as a result.

Concussions are far more prevalent than moderate or severe traumatic brain injuries. It has been estimated that the annual incidence of this type of injury in the general population varies from 100 to 300 cases per 100,000 inhabitants (Cassidy, Carroll, Peloso, Borg, et. al, 2004; Hirtz et. al, 2007). In the United States, around 1.7 million individuals present to emergency departments or are hospitalised due to injuries involving alteration in consciousness or brain trauma each year (Center for Disease Control and Prevention, 2006). Some estimates suggest that approximately 80% of these injuries could be classified as concussion, because they were discharged on the same day of their initial visit, while the remaining 20% of cases are split relatively equally between moderate and severe TBIs (Kraus et al., 1996). According to the Centers for Disease Control and Prevention (Langlois et al., 2003; Mellick et al., 2005) around 5.3 million people in the United States were living with a disability caused by a TBI in 2003. The portion of these cases that are disabled as a result of concussion are unknown. In 2000, the CDC tallied direct medical costs and indirect costs such as loss of productivity and estimated that this summed to \$76.5 billion spent annually on the effects of TBI (Finkelstein, et al., 2006; Coronado et al., 2012). Again, this figure includes injuries of all severity levels, and the proportion of the costs due to concussive injuries is unknown. It should be noted, however, that these estimates rely on data derived from hospital visits, and as such do not include information about individuals who sought care from a family physician, or sought no care at all. As such, the majority of concussed individuals are likely underrepresented in these figures.

It has been suggested that only 20% to 25% of all individuals who suffer concussions report their injuries to hospital (Sosin et. al, 1996). Others have estimated that for every 100,000 cases that present to hospital with injuries involving alterations in

consciousness, around 392 do not result in hospitalisation, and these maybe best classified as concussions (Guerero, Thurman and Sniezek, 2000). With these caveats in mind, the CDC has extrapolated that somewhere between 1.4-3.8 million concussions occur each year in the United States (Center for Disease Control and Prevention, 2006).

The breakdown of mechanisms of injury associated with mild injuries and concussions is extremely variable across studies with between 11-81% attributed to motor vehicle accidents; 10 to 46% as a result of falls; 6 to 30% from assaults or being struck by falling objects; and between 19 and 71% due to sporting activities (Jennett and Frankowski, 1990; Bazarian et al. 2010; Bordignon and Arruda 2002; Iverson et al. 2000; Kashluba et al. 2008; Kraus et al. 2005; Lee et al. 2008; Lipton et al. 2009; Lundin et al. 2006; Meares et al. 2008; Thornhill et al. 2000). Again, rates will vary according to definition.

With regard to local epidemiology, it has been estimated that there are 6000 new traumatic brain injury cases in British Columbia annually (Ministry of Health Services and Ministry of Health Planning, 2002). As around 20% percent of these traumatic brain injury patients are predicted to die on the way to the hospital, there are an estimated 4800 survivors of traumatic brain injury in British Columbia annually. The vast majority (3840 of 4800) of these survivors are estimated to present with concussion type injuries (Ministry of Health Services and Ministry of Health Planning, 2002). In Canada, it has been estimated that approximately six out of every 1000 worker compensation claims are the result of concussion, although concerns exist about the likelihood of underreporting (Kristman, Cote, Van Eerd, Vidmar, et. al, 2008). Martin (2003) reports data on workman's compensation claims in British Columbia. In his report, Martin (2003) defines these injuries as resulting in GCS between 13 and 15, brief LOC, brief PTA and negative CT scans. Using these criteria Martin (2003) reports that the average incidence rate of compensation claims due to concussion annually in British Columbia between 1987 and 2001, was about 0.3% of the total claims. However, the average cost of settlements paid as a result of concussion related claims was at least twice the average cost of all other claims in the same year, combined. Further, the median cost of claims settlements were tripled in comparison to other claims during the same year (Martin, 2003). As such, it is clear that the impact of concussions on society is nontrivial. Concussive injuries, and the management of symptoms associated with concussions

have a significant impact on our society. The next sections examine what is known about these post-concussion symptoms.

Post-Concussion Symptoms

The acute experience of concussion is phenomenologically variable and idiosyncratic. Common reactions to concussions can include a wide array of nonspecific symptoms. These symptoms include transient loss of consciousness, confusion, disorientation, limited post traumatic amnesia, balance difficulties, headaches, dizziness, nausea, subjective memory and concentration difficulties, irritability, tinnitus, fatigue, visual disturbances, hypersensitivity to noise, restlessness, anhedonia, sleep disturbance, vertigo, orientation difficulties, depression and anxiety (Binder, 1986; Fox, Lees-Haley, Earnest & Dolezat-Wood, 1995; Kay, Newman, Cavallo, Ezrachi & Resnick, 1992; Mittenberg, DiGiulio, Perrin & Bass, 1992, Giza et al., 2013). Studies have suggested that 86% of patients will have at least one “post-concussion” symptom on the first day after injury (Lundin et al, 2006). Symptoms of concussion typically present immediately after injury, but may be delayed by several hours (Patel, Reddy, 2010; EleMBERG, Henry, Macciochi, et al., 2009). These “Post-Concussion Symptoms” usually last less than 72 hours (EleMBERG, Henry, Macciochi, et al., 2009), and most concussions resolve spontaneously within 7 to 10 days (McCrorry, Meeuwse, Johnston, et. al, 2008).

Pathophysiology of Acute Symptoms

The transient nature of the majority of post-concussion symptom experiences has been related to findings from animal studies, which suggest that these symptoms arise as a result of a complex neurometabolic cascade (Giza and Hovda, 2001, Barkhoudarian et al., 2011; Kan et. al, 2012). The currently accepted understanding of the underlying pathology involves a paradigm shift away from an anatomic damage model to a neuronal dysfunction model. Under this model, symptoms are produced as a result of a complex cascade of ionic, metabolic and physiologic events (Giza and Hovda, 2001, Barkhoudarian et al., 2011; Kan et. al, 2012). Experimental brain injury induces several metabolic consequences. These consequences include shifts in ionic concentrations of

magnesium, potassium and calcium; indiscriminate release of excitatory amino acids such as glutamate; altered brain glucose metabolism (first hyperglycolysis, then hypoglycolysis); and reduced cerebral blood flow resulting in changes in neurotransmission. The acute clinical signs and symptoms of concussion are thought to be manifestations of this underlying neurometabolic cascade. This is in part due to the observation that the metabolic depression resulting from oxidative and glycolic debt due to the acute excitation of neurons following experimental brain injury lasts around 7 to 10 days in adult rats (Giza and Hovda, 2001). This time period is similar to the expected time course of recovery from concussive injury in humans (McCrory, Meeuwse, Johnston, et. al, 2008). It should be noted however, that these experimentally induced events may or may not be similar to the types of injuries incurred in concussion. Indeed, there is reason to believe that these models may be more appropriate for the understanding of moderate to severe brain injury. Animal studies are typically performed using a “fluid percussion” model which involves removal of a portion of the skull and forcing a directed quantity of saline directly onto the dura for a limited time period and with limited force (Reger et al., 2012). Alternate models include applying rotational forces to the head and skull with experimentally defined parameters for acceleration (Browne, Chen and Meaney, 2011). The force applied in these types of experiments is often based upon estimates of the biomechanical forces needed to induce a concussive injury. These studies attempt to induce injuries with observable behavioural and metabolic effects in animal models, which are constrained to some degree by the inability of the animals to communicate the nature of any symptoms induced. As such, these studies may apply forces beyond those typically incurred in naturally occurring concussions, in order to induce measureable effects. A wide range of estimates for force threshold values exist in the literature and the association between the strength of the forces applied and clinical outcome in humans is not well understood (Guskiewicz and Mihalik, 2011).

Although experimentally controlled inducement of injury is subject to criticism on the basis of injury severity and generalisation beyond the laboratory, the current animal model is broadly accepted. The inducement of the metabolic crisis described above is particularly attractive in its ability to explain the acute and transient nature of the majority

of post-concussion symptoms. However, as illustrated below, these symptoms sometimes persist beyond the period of metabolic recovery predicted by these models.

Course and Prognosis

Among the more than 1 million individuals who sustain concussions each year, the literature suggests that 80% to 90% make a favorable recovery (Binder et al., 1997; Wong, Regennitter and Barrios, 1994; Bigler, 2008). Although the majority of individuals who suffer a concussion recover without persistent disability, there is valid concern that the consequences of concussion are not transient for some (Iverson, 2005; Holm et. al, 2005). Indeed, it has been well established that a proportion of individuals who suffer a concussion experience lingering symptoms, which can lead to significant disability (Carroll et. al, 2004; Ponsford et. al, 2000; King, Kirkwilliam, 2011; Kirsch et. al, 2010; Lundin et. al, 2006; Nolin and Heroux, 2006). The size of the minority who become chronically symptomatic varies across studies from 7% (Binder, 1997) to approximately 33% (Alexander, 1995, Rimel, Giordani and Barth, 1981). Variability in the definition of concussion, as well sampling issues and outcome measures contribute to the variability in incidence rates reported for poor outcome (see Bernstein, 1999 for review). Some have suggested that upwards of 49% of individuals with a history of concussion will continue to report at least one symptom 3 months after injury, (Lundin et al, 2006). However, it is generally accepted that only around 15% experience a significant social or occupational impact from symptoms beyond a three month time period of recovery (Uzzell, 1999).

The nature and etiology of these persisting symptoms is a subject of intense debate and investigation. The following sections review the current diagnostic frameworks that are used to identify those with persisting symptoms. Also, attempts to understand the observable neuropathological changes that may result in poor recovery will be reviewed.

Diagnostic Considerations

Research into persistent symptoms, including epidemiological studies, treatment studies and reviews of symptomatology are somewhat clouded by a lack of conformity in the categorisation of pathological responses to injury. Benton, (1989) summarized the dilemma and provided a useful definition of Post Concussional Disorder when he wrote:

“It is generally understood to refer to a condition in which a person who has sustained a concussion complains of a variety of somatic, cognitive, emotional, motor, or sensory disabilities which he or she ascribes to the concussion. At the same time, convincing historical and clinical evidence of significant brain injury cannot be elicited (p. 3)”.

Research to date has suggested that the experience of post-concussion symptoms beyond three months post injury is indicative of pathological recovery (Binder, Rohling, & Larrabee, 1997, Faux, Sheedy, Delaney and Riopelle, 2011, Begaz et al., 2006; Chamelian et al., 2004; Iverson, 2006; Rees, 2003; Satz. et al, 1999, Stalnacke et al., 2005; Willer and Leddy, 2006; Belanger et al., 2005). Individuals with such a prolonged course of recovery have been variously referred to as having Chronic Post-Concussion Syndrome, Persistent Post-Concussion Symptoms, or Post Concussional Disorder (Benton, 1989; American Psychiatric Association, 1994; Iverson, 2006).

There are currently two widely used diagnostic rubrics that address the occurrence of chronic symptoms following concussion. Each defines the problem quite differently from the other, leading to some confusion in the literature (Barlow et al. 2010; Ruff 2011; Yeates 2010). The International Statistical Classification of Diseases and Related Health Problems–10th revision (ICD-10; World Health Organization 1992), describes “Postconcussional Syndrome” as a condition which “occurs following head trauma (usually sufficiently severe to result in loss of consciousness)”. The ICD-10 suggests that the set of symptoms following concussion are representative of a “syndrome”. This “syndrome” is defined as including “headache, dizziness (usually lacking the features of true vertigo), fatigue, irritability, difficulty in concentrating and performing mental tasks, impairment of memory, insomnia, and reduced tolerance to stress, emotional excitement, or alcohol.” The ICD-10 criteria go on to note that, “these symptoms may be accompanied by feelings of depression or anxiety”. The ICD-10 further appears to take a rather strong stance on etiology by stating that these emotional issues result, “from

some loss of self-esteem and fear of permanent brain damage... (which) enhance the original symptoms". The theoretical stance becomes even more apparent in the statement that "some patients become hypochondriacal, embark on a search for diagnosis and cure, and may adopt a permanent sick role". The diagnostic criteria are then listed as a positive endorsement of "at least three of the features described above". (WHO, 1992; section F07.2).

These criteria have been criticised for their ambiguity as well as the subjectivity involved in determining whether or not an individual can be said to be experiencing the "symptoms" provided (Iverson & Lange, 2003; Carroll et al., 2004). The ideological stance taken on the likely etiology of symptoms requires the clinician to make theoretical inferences about the cause and progression of symptoms. Some of these criteria cannot be established solely according to the report of the patient (Kashluba et al. 2006). Further, references to reduced tolerance to stress, emotional excitement, or alcohol are seldom found in the literature as postconcussive symptoms. Of the symptoms and conditions listed, nine can be assessed directly through self-report. These symptoms include; headache, dizziness, fatigue, irritability, difficulty in concentrating, impairment of memory, insomnia, depression, and anxiety. Only these nine symptoms are consistent with the symptoms commonly assessed in research (e.g., Gouvier et al., 1988; Iverson & Lange, 2003; Paniak et al., 2002; Ponsford et al., 2000). Further, given the well-established timeframe of expected recovery from symptoms following a concussive event, the ICD-10 classification is not useful in separating those who have experienced a concussion and recover normally from those who suffer from chronic symptoms and poor outcomes. (Kashluba, et al., 2006). Finally, and perhaps most relevant to the current research, the empirical basis for the assertion that symptoms are generated by hypochondriacal ideation is unclear.

An alternative classification is provided in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994).³ The DSM-IV includes criteria for "Postconcussional Disorder" as a diagnostic category for additional study, in order to aid in research into the topic. The DSM-IV criteria are unique in their

³ The author has been unable to find any reference to post-concussion symptoms in the recently published DSM-V.

specification that the symptoms must last beyond the three month limit established by the literature. For this reason, these criteria are better at distinguishing between chronic versus acute presentations than other classifications. The working diagnostic criteria, according to the DSM-IV, include a history of cerebral concussion with at least three of the following symptoms, lasting three months or more: fatigue, disordered sleep, headache, dizziness, irritability, anxiety-depression, personality changes, and anhedonia (American Psychiatric Association, 1994). Also included in the proposed diagnostic criteria is evidence of neuropsychological impairment, although the utility of cognitive testing in PCD has been called into question (Thornton, et al, 2008; Ponsford et al., 2012). Indeed, the literature is marked by mixed findings concerning the role of neurocognitive dysfunction in the expression of chronic symptoms. It has been well established that concussion has a demonstrable impact on neurocognitive functioning within the acute phase of recovery, but there is little support for objective neurocognitive impairment in the chronic phase. A recent multi-centre emergency room study administered measures of immediate and delayed recall in the acute phase of recovery. These measures were reported to predict Post Concussional Disorder at 3 months post injury with 80% sensitivity and 76% specificity (Faux, Sheedy, Delaney and Riopelle, 2011). However, these authors did not measure neurocognitive functioning in their 3 month follow-up and as such this data cannot speak to the benefit of using neurocognitive performance as diagnostic criteria. Indeed, although most research supports the notion that concussions are associated with neurocognitive dysfunction acutely, these mild impairments are generally reported to resolve completely by 1 to 3 months post injury (Binder, Rohling, and Larrabee, 1997; Dikmen, McLean and Temkin, 1986; Dikmen Machamer, Winn and Temkin, 1995; Gronwall and Wrightson, 1974; Hinton-Bayre, Geffen, Geffen, McFarland and Friis, 1999, Levin et al., 1987; Macciocchi, Barth, Alves, Rimel and Jane, 1996; Ponsford et al., 2000; Belanger et al., 2005). A recent meta-analysis of sports related concussions concluded that there was no evidence of neuropsychological dysfunction in any cognitive domain if testing was completed 10 days or more post-injury (Belanger and Vanderploeg, 2005). It has also been reported that athletes with a history of multiple concussions were unimpaired on a test of information processing speed given one month post injury, when compared to athletes with a history of only one concussion. Indeed, the athletes with a history of prior concussions actually performed significantly better than their matched controls

(Echemendia et al, 2001). Thus, the literature is not conclusive about the relationship between neurocognitive dysfunction and the endorsement of chronic symptoms (Thornton et. al, 2008; Dikmen, Machamer, Winn, and Temkin, 1995; Ponsford et. al, 2000, 2012; Belanger and Vanderploeg, 2005, Faux et. al, 2011). Given the weak relationship between neurocognitive performance and chronic outcomes, the specification of positive objective evidence of neurocognitive dysfunction in the DSM-IV criteria may have limited utility.

Neither the ICD-10, nor DSM-IV diagnostic criteria are considered without flaw with respect to their sensitivity to persistent post-concussion symptoms. Further, neither diagnostic rubric is considered perfect with respect to its specificity in separating those with disorder from those who are recovering normally. The inconsistency between these two widely used diagnostic manuals leads to poor diagnostic agreement (Boake et al. 2004, 2005). In addition, the criteria for post-concussion difficulties in both the DSM-IV and ICD-10 have been shown to have limited specificity. The “post-concussion” symptoms listed in each overlap significantly with symptoms expected in numerous other conditions (Boake et al. 2005).⁴ With respect to nosology, it has been suggested that the term Postconcussion Syndrome be consistently replaced with Postconcussional Disorder (PCD; Ruff, 2005). This is because the use of the term “disorder” reflects the growing consensus that there is no single uniform “syndrome” following a concussive injury. Also, the DSM-IV terminology allows for the emphasis of atypical recovery through the specification that disorder is apparent only after three months of unremitting symptoms. This time frame is based upon empirical research on recovery and the recognition that acute post-concussion symptoms are normal and expected. Symptoms should only be classified as evidence for “disorder” when they fail to resolve and become chronic (American Psychiatric Association, 1994).

Further, the DSM-IV PCD classification is somewhat atheoretical, and broad and inclusive in its consideration of etiological factors. It has been stated that PCD can be

⁴ As a side note, the observation that postconcussional disorder shares symptoms with other disorders is somewhat mundane, as illustrated by Buckholtz and Meyer-Lindenberg (2012) who wrote; “comorbidity between mental disorders is the rule rather than the exception...covariation among psychiatric diagnoses is so prevalent, and so extensive, that it alone belies the artificial nature of phenomenologically based categorical classification.”

viewed as being the result of: neuropathology, and/or psychopathology, and/or secondary gain in the form of consciously reduced effort or malingering, and *any combination thereof* (Ruff, 2005). This conceptual framework is preferred in the current work as it acknowledges the fact that multiple etiological pathways exist to the expression of symptoms and that these pathways are not mutually exclusive but are quite likely interdependent. By referring to the injury as a “Concussion” and by classifying pathological recovery as “Disorder”, the term “Post Concussional Disorder” avoids becoming included in the “Psychogenic versus Organic” debate. This is attractive because the “Psychogenic versus Organic” debate is largely based upon a rather naïve conceptualisation of the evidence based upon a false dichotomy rooted in Cartesian dualism.

There is no compelling evidence that any “psychogenic” phenomena can exist independently of its organic substrate. Given increased acknowledgement of this fact, the question of etiology has shifted from considerations of “biological” versus “psychological”. Modern, sophisticated researchers have begun to ask; “How do factors related to injury interact with pre-existing biopsychosocial systems to influence the expression and duration of symptoms?” (Rose, 2005). In attempts to answer this question, much research has been done to clarify the neuropathological correlates of persisting symptoms, if any, while others have focussed on the biological, demographic, psychological and social risk factors associated with poor outcomes.

Neuropathological Investigations

The following review of neuropathological investigations illustrates that results are sometimes mixed and a definitive understanding of the neuropathological correlates of chronic post-concussive symptoms is not yet clear. Despite this, much has been learned through the careful application of neuroimaging technologies to the problem, and this field is growing rapidly. A comprehensive review of the entire imaging literature is beyond the scope of this review, although the studies reviewed have been chosen for their representativeness and quality. To be clear, the review that follows will focus on investigations of brain changes in the post-acute phase of recovery from concussive injury, as this is somewhat different from investigations of acute effects, or in more

severe injuries. Changes in brain function are certain to be ultimately responsible for the subjective experiences and objectively observable behavioural changes that characterise post-concussional disorder. However, a clear understanding of the relationship between neuropathology and chronic symptoms has not yet been established. As evidenced in the review that follows, this lack of clarity is driven in part by the relative insensitivity of some imaging techniques, and in part by inconsistent findings. Further, where positive indicators of neuropathology have been found, it has been difficult to establish clear causative relationships between the concussion event and neuropathological outcome measures. This difficulty likely arises from the fact that the subtle neuropathological changes reported are not readily dissociable from those associated with psychiatric and emotional difficulties, such as depression and anxiety. As investigators in this field develop new and more sensitive technologies and methods, these investigations are likely to provide a wealth of information about the biological correlates of chronic post-concussion symptoms.

Structural Imaging

Studies utilizing traditional structural imaging techniques have largely failed to produce strong evidence that chronic symptoms are associated with identifiable macrostructural brain lesions or brain abnormalities. It has been reported that the vast majority of individuals who present with transient alterations in consciousness as a result of injury have normal CT scans. Indeed, the presence of structural abnormalities on CT or MRI is generally considered indicative of injury that is perhaps better conceptualised as a moderate to severe TBI (see McAllister, Sparling, Flashman and Saykin, 2001, for review). A recent study which compared individuals with post-concussion symptom complaints to controls found no association between acute CT findings, and symptom reporting between 5 and 7 years post injury (Jakola, Muller, Larsen, Waterloo, Romner and Ingebrigtsen, 2007). Bigler and Snyder (1995) reported no significant changes in MRI after experiencing a concussion in a small sample of individuals with preinjury scans (Bigler and Snyder, 1995). These authors report no significant differences between pre and post injury MRI or differences from control subjects in their group, despite the finding that the concussed group showed mild neurocognitive and emotional sequelae in the acute phase of recovery (Bigler and Snyder, 1995). Other researchers

have also reported that they have been unable to correlate abnormal findings on MRI with either post-concussion symptoms or long term outcome (Hughes, Jackson, Mason, et al., 2004; Schrader et. al, 2009). Lannsjö and colleagues (2012) performed acute CT scans on a large sample of concussed individuals who presented to emergency departments. These authors reported no association between the results of scans and the report of post-concussion symptoms at three months post injury. Thus, it is generally accepted that macro-structural imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI) are of limited utility in the evaluation of chronic post-concussion symptoms (Ryan and Warden, 2003; Kurca et al., 2006).

Diffusion Tensor Imaging

Diffusion Tensor Imaging (DTI) is an MRI technique that capitalises on the directional diffusion of water molecules along white matter tracks in order to provide images of brain structures. In healthy white matter tracts, water molecules are lined up in a more organized fashion than the water in the surrounding grey matter, which is more diffuse. DTI studies vary with regard to the strength of the field applied, as well as the measurements used. Despite this variability, studies utilising this technique typically report on the “Fractional Anisotropy” (FA) value within a predefined brain region of interest. This review will focus on reports of FA, as it is most the most commonly reported outcome measure, and is intuitively understood. FA values range from zero to one. A zero FA value is indicative of isotropic diffusion, indicating no difference between the diffusion of water molecules in the region. An FA value of one would represent complete anisotropy, which would indicate that all the water molecules are organised tightly within tissues. (Le Bihan et al., 2001). FA is generally thought to represent several factors related to brain tissue composition, including the degree of myelination of fibres, and axonal integrity and/or density (Arfanakis et al., 2002). As such, reductions in FA observed in white matter tracts have been considered to provide evidence of microstructural injury.

DTI has demonstrated good sensitivity to diffuse axonal injury in traumatic brain injury, and has recently been used to investigate white matter changes in concussion. Studies utilizing this technique have reported inconsistent results. While some studies report no change, or even increased FA in concussed individuals (Henry et al., 2011, Wilde et al.,

2008), others report significant reductions. Smits et. al (2010), report that severity of post-concussion symptoms one month post injury in nineteen subjects were correlated with reduced white matter integrity. These authors report findings of reduced white matter integrity in the uncinate fasciculus, inferior fronto-occipital fasciculus, internal capsule and corpus callosum. Despite obtaining their data within the acute recovery phase, Smits et. al (2010), interpret their data as “evidence of microstructural injury as a neuropathological substrate of post-concussion syndrome”. However, others have reported contradictory findings. For example, Lange et. al (2012) measured FA values in the genu, splenium, and body of the corpus callosum of sixty individuals six to eight weeks after concussion, and compared these values to thirty-four trauma controls. These authors report no differences between the FA values of the concussed and non-concussed groups in any of the regions of interest (Lange et al., 2012). This is particularly interesting in that the concussed sample were reported to be rather more severe than is typical, as evidenced by the fact that they all reported to hospital as a result of their injuries. Indeed, several of the concussed individuals used in this sample had documented abnormalities on CT investigations (Lange et. al, 2012). Further, Lange et. al (2012) report no difference in FA between individuals who met ICD-10 criteria for post-concussional disorder, and those who did not.

With regard to the use of DTI in chronic post-concussional disorder, very few studies have been attempted to date, and these results have been mixed as well. Kraus et al., (2007), applied DTI to twenty individuals who had sustained concussion an average of 7.6 years prior to their study. In comparison to controls, individuals with a history of concussion showed reduced FA in the superior longitudinal fasciculus, saggital stratum and corticospinal tract (Kraus et al., 2007). However, Rutgers et al. (2008), report that twenty-four concussed individuals displayed reductions in FA in the genu of the corpus callosum if they were imaged less than three months post injury, but that no abnormalities were noted in any DTI measure if the imaging was conducted three months or more post-injury. In contrast, Lo et. al (2009) report a significant loss of white matter integrity relative to controls in the genu of the corpus callosum on the left and internal capsule bilaterally. These authors demonstrated these differences in individuals who complained of cognitive symptoms lasting over two years following concussion, suggesting that loss of white matter integrity may play a role in chronic symptoms. This

finding is potentially important, although the authors note that it might not be readily generalized to the population, as only ten subjects were imaged (Lo et al., 2009).

Regardless of the time frame post-injury, there are inconsistent findings surrounding the nature and extent of microstructural changes that can be attributed to concussion. In an attempt to clarify the literature, a recent meta-analysis was conducted on the subject. The meta-analysis included thirteen DTI studies, with fifteen independent comparisons on a total of 280 “mTBI” patients and 244 controls. This meta-analysis did not separately analyse studies that included individuals in the acute phase of recovery from those whose injuries occurred long ago (mean time between scan and injury ranged from three days to eight years). The results of the meta-analysis suggest that FA was only reliably reduced in the splenium of the corpus callosum (Aoki et al., 2013), although this is reported as being a very large effect (effect size, reported as a Z score, is listed as hovering around -2.24, Heterogeneity I square = 65.87%). No reliable associations between concussion and FA were obtained for any other region of interest, including the genu of the corpus callosum, internal capsule, and corona radiata. Further, when the analyses were constricted to studies which excluded medication use and/or substance abuse, no significant differences were obtained in any region (Z values ranged between .09 and .1; Aoki et al., 2013).

As previously stated, it has been difficult to interpret positive associations between DTI findings and concussion history in some cases due to methodological constraints. Within the limited literature on the subject, very few studies attended to psychiatric and emotional factors. This omission is considerable, given emerging data which indicates that emotional difficulties such as depression are associated with similar reductions in white matter integrity, in similar regions as those outlined above (Liao et al., 2013; Xian et al., 2010). Thus, when DTI does provide evidence of decreased FA, it is difficult to know if the reduction is due to injury, or to psychiatric illness, or both. The picture that appears to be emerging is that alterations in white matter integrity associated with concussion appear to be similar to those associated with mood disorder. More research is needed in order to investigate whether the neuropathological changes associated with the experience of concussion can be dissociated from those associated with mood disorder.

Functional Imaging

The field of functional neuroimaging has grown rapidly in the past few years, and has the potential to yield very important information about recovery from concussion. Research in this field has made significant contributions to our understanding of changes in brain function following concussion. However, the difficulty with dissociating observable brain changes due to injury from those associated with emotional functioning is also apparent in studies which have utilised functional imaging techniques. Measures of brain function such as functional magnetic resonance imaging (fMRI), and single photon emission computed tomography (SPECT) have been used widely to investigate the effects of concussive injury (McAllister, Sparling, Flashman and Saykin, 2001; McAllister et.al, 2006; Ryan and Warden, 2003; Gaetz and Weinberg, 2000). As with other investigations of neuropathology, results have been mixed. A recent study by Stulemeijer et al., (2010) found no difference in the brain activation patterns of individuals who had experienced concussions up to six weeks prior to scanning, when compared with healthy controls. In contrast, the majority of studies utilizing functional imaging techniques to assess functional abnormalities in concussed patients without structural abnormalities on CT or MRI scans have reported some evidence of hypometabolism primarily in frontal and temporal regions during the performance of working memory tasks (Ruff et al, 1994, McAllister, Sparling, Flashman and Saykin, 2001; Ryan and Warden, 2003). These studies have also suggested that a correlation exists between hypometabolism and poor neuropsychological performance, on tests of working memory (McAllister, Sparling, Flashman and Saykin, 2001; Davalos and Bennet, 2002, Chen et. al, 2004; 2007; 2008). Mayer et. al, (2009), report hypo-activation in several cortical and subcortical areas in association with auditory orientation and attentional inhibition tasks. It is difficult to interpret these results, however, as hypometabolism in these areas is also strongly implicated in emotional difficulties, which may have resulted from injury, but may have also been pre-existing (Ochsner, Ludlow, Knierim and Hanelin et. al, 2006; Robillard, 2004; Mattson and Levin, 1987). Indeed, Lewine et al., (2007), demonstrated reduced activation in the dorsolateral prefrontal cortex, anterior cingulate cortex and striatum of concussed athletes, in comparison to noninjured athletes. These authors went on to report that their findings of attenuated activation were dependent on the severity of depressive symptoms reported by the concussed athletes (Lewine et al., 2007). Along these same lines, Chen et. al (2008),

report that fMRI activation during a working memory task was reliably decreased in concussed athletes who endorsed significant depressive symptoms, but that non-depressed athletes with a history of concussion did not display any differences in activation as compared with controls.

Another source of interpretive difficulty with regard to fMRI findings is the meaning of changes in regional blood flow. Whereas some studies take reduced blood flow as an indicator of injury, others posit that injury is most associated with increases in blood flow. Under this model, more resources are needed to perform cognitive tasks when the neural substrate becomes inefficient due to injury. For example, Jantzen et al., (2010) compared fMRI scans of individuals before and after injury. These authors reported injury related increases in activation within the parietal, lateral frontal and cerebellar regions in the absence of any observable changes in cognitive performance (Jantzen et al., 2010). To date, it is unclear whether less activation indicates impaired function, or whether greater activation of particular regions suggests a compensatory mechanism. It is also possible that some combination of the two processes can be occurring concurrently. For example, it may be that initial disruption within the network leads to the observation of hypoactivation, which then converts to hyperactivation due to an inefficient attempt to compensate for the disrupted or poorly functioning system. The pattern of activation observed may also depend on individual differences in connectivity, as well as factors related to the severity and foci of injury.

Along the same lines, a review of the literature on the use of SPECT in the investigation of concussion sequelae suggests that there is not a strong relationship between neuropsychological impairment and SPECT abnormalities, and that emotional factors are a more reliable predictor of SPECT abnormalities than neurocognitive indices (Davalos and Bennet, 2002). This is especially relevant to fronto-temporal findings. Indeed, throughout this review it will become apparent that emotional distress is not easily ruled out as a contributor to outcome as measured by either neuropathological investigations and/or psycho-social studies.

Metabolic Markers

Another fascinating and growing area of research that holds much promise in increasing the understanding of recovery from concussion is the investigation and identification of metabolic indices of damage and repair to the neural substrate. An increasing number of studies have been conducted to identify metabolic markers for concussion and recovery (Bergsneider et al., 2000; Giza and Hovda, 2001). Several researchers have utilized Magnetic Resonance Spectroscopy (MRS) to quantify cellular metabolites in vivo (Shekdar, 2011). The metabolites most often studied are N-acetylaspartate (NAA), Choline (Cho), and Creatine-phosphocreatine (Cr) (Cecil, 1998; Belanger, 2007; Govind, 2010). NAA levels are thought to be associated with axonal injury and neuronal loss, as decreased levels have been associated with injury, metabolic dysfunction, and/or myelin repair (Gasparovic et al., 2009). Choline levels have been used as a marker for cell membrane repair (Shekdar, 2011). Creatine and Phosphocreatine levels have also been studied as indicators of cell energy metabolism (Signoretti et. al, 2009). Vagnozzi et. al, (2010) report significant alterations in the ratios of these metabolites in a group of head injured athletes. These metabolic changes were observed relatively quickly after injury, and resolved to a level consistent with noninjured controls after 30 days. Similarly, Johnson (2012), report significant reductions in NAA/Cr and NAA/Cho ratios in concussed athletes who were seen in the acute recovery phase. Henry et. al (2010) report significant injury related decreases in the NAA/Cr ratio in concussed athletes as compared with nonconcussed controls. Interestingly, these authors also report that the resolution of these neurometabolic changes was correlated temporally with the resolution of self-report of post-concussive symptoms (Henry et al., 2010). Thus, this research has proven productive in its ability to provide an index of biological injury that is sensitive to symptom levels in the acute phase of recovery, although the utility of metabolic markers in indexing chronic symptoms is as yet unclear.

In addition to the metabolites referred to above, many proteins have also been investigated as biomarkers for injury. Proteins such as S-100, cleaved tau and neuronspecific enolase, have been examined. Of these S-100 has been the most frequently studied. This is a protein normally found in cells derived from the neural crest which can be elevated in brain pathology and functions as a regulator of inflammatory response and cell growth. Unfortunately such studies are frequently beset with

difficulties of poor sensitivity and specificity, and small samples (Begaz et al, 2006). Also, a recent study which compared individuals with post-concussion symptom complaints to controls found no association between biological markers, such as S100B serum levels and long term expression of chronic symptoms (Jakola, Muller, Larsen, Waterloo, Romner and Ingebrigtsen, 2007). Indeed, although these investigations are useful in their ability to identify injury and link it to observable changes in brain chemistry, they are unable as yet to make determinations about recovery from that injury, beyond the acute recovery phase.

Electrophysiological Investigations

Another promising area of research into the effects of concussive injury on brain function is the use of eletrophysiological techniques to measure changes in brain function associated with injury and recovery. In consideration of studies employing electrophysiological techniques, the literature is consistent with findings reported using other assessment paradigms. Namely, there appears to be strong evidence for acute abnormalities following concussive injury but evidence for persisting indicators of brain dysfunction is mixed. Further, it is difficult to dissociate effects related to the concussive injury itself, from those associated with emotional distress. Again, electrophysiological investigations have determined that concussion produces similar effects as mood disorder and emotional distress.

EEG studies are based upon recordings of weak electrical signals at the scalp. The signals recorded at the scalp level are modified to some degree by the conductivity of the tissues between the recording site and the neuronal generator of the signal. The orientation of the neuronal generator is also a factor (Olejniczak, 2006). Studies employing electrophysiological techniques in the acute stages of recovery indicate that attentional functions may be particularly vulnerable to the immediate effects of concussion (Mendez et. al, 2005). Early electrophysiological studies focused on either auditorily or visually evoked event related potentials (ERP's). ERP's assess functional brain activity and have been considered to be excellent indices of subtle alterations in information processing such as those associated with concussion (Gaetz and Bernstein, 2001). As the methods and technology associated with this research are further refined, and the specific nature of the waveform components elicited are identified and

characterised, differences in the amplitude, latency, or scalp distribution of ERP's have the promise of elucidating the locus and extent of brain changes due to injury (Rugg et al., 1989). It has been suggested that the "long-latency" ERP components, which are elicited more than fifty milliseconds after stimulus onset, and specifically the "endogenous" components such as the N200 and P300, vary as a function of cognitive processes such as attention and expectancy (Duncan et al., 2005). While research has been conducted in consideration of many ERP components across the spectrum of brain injury severity (Duncan et. al, 2005), the ERP component that is most often investigated in concussion is the P3, or P300 waveform. The P3, which is purported to reflect processing of contextual cues, or memory updating, (Coles and Rugg, 1995) is considered a measure of attentive functions and short term memory, and variations in latencies in the P3 wave are thought to indicate modifications in the rapidity of these cognitive processes (Alberti, Sanrchielli, Mazzotta, and Gallai, 2001).

With regard to the acute phase of recovery, attenuation in the P3 ERP component has been reported to be related to the severity of post-concussion symptoms in college athletes at one week post injury (Dupuis, Johnson, Lavoie, et al., 2000). However, these authors further report that the ERP abnormalities found in their sample had completely resolved by 4 weeks after the concussive event (Dupuis, Johnson, Lavoie, et al., 2000). Interestingly, the same group of researchers later reported findings of an inverse relationship between the severity of post-concussion symptoms endorsed in the acute phase of recovery and P3 amplitude (Lavoie, Dupuis, Johnston, 2004).

The literature pertaining to the relationship between electrophysiological abnormalities and endorsement of chronic post-concussion symptoms beyond the acute phase of recovery is mixed. A series of studies has suggested that visual P3 latencies can distinguish between individuals who have experienced a concussion and those who have not (Gaetz and Weinberg, 2000), and that auditory P3 latency can differentiate between individuals who have had only one such event and those who have experienced 3 or more events (Gaetz, Goodman and Weinberg, 2000).

In a study which compared symptom endorsement and P3 latency of junior hockey players with 3 or more concussions and those with only one such injury, Gaetz, Goodman and Weinberg (2000), report that while P3 latency was increased in

individuals with three or more concussions relative to those with only one, only cognitive (and not emotional) symptom endorsement was associated with P3 latency. These authors use this data to argue that the “P3 is an index of changes in cognitive, and not emotional symptoms”, (Gaetz, Goodman and Weinberg, 2000; pp. 1085), and that “the hypothesis that chronic post-concussion symptoms are in part, or completely, psychogenic in all cases is not supported (Gaetz, Goodman and Weinberg, 2000; pp. 1086). In support of this conclusion, Broglio et. al (2009) report a significant decrement in P3 amplitude in athletes with a history of concussion that occurred an average of 3.4 years prior to recording.

However, this conclusion is challenged by the findings of Alberti et al. (2001), who also report a significant increase in P3 latency in adults with chronic symptoms following concussion. These authors administered measures of anxiety and depression along with their electrophysiological indices and found strong correlations between self-reported anxiety and depression and P3 latency. In support of this finding, Granovsky et al.,(1998) also reported significant associations between the characteristics of the P3 wave and self-reported anxiety in a sample of individuals with persisting symptoms following concussion (Granovsky, Sprecher, Hemli, Yarnitsky, 1998). It should be noted that increases in P3 latency have also been implicated in various emotional disorders, including depression (Vandoolaeghe, van Hunsel, Nuyten and Maes, 1998; Schlegel, Nieber, Herrman and Bakauski, 1991) and Post-Traumatic Stress Disorder (Karl, Malta and Maercker, 2006). Thus, the abnormalities in the P3 component associated with concussion history appear to be similar to those elicited by mood disorder and emotional distress.

More recently, Barr et al. (2012) reported abnormalities in quantitative EEG (qEEG) in athletes when recordings were conducted eight days post injury. However, these authors further report that the EEG waveforms had normalised and were indistinguishable from non-injured athletes at 45 days post-injury. Similar qEEG results are reported by McCrea et. al (2010). These authors provide important information about the acute effects of concussion on subtle changes in brain function.

Another arm of investigation has focussed on EEG coherence patterns to illustrate functional differences in brain region connectivity between concussed and non-

concussed individuals. Kumar et al. (2009), used spectral EEG analysis techniques to examine connectivity between frontal, temporal and parietal regions during both verbal and visual working memory tasks. These authors report significant decrements in interhemispheric connectivity, as evidenced by differing coherence patterns, in a group of concussed individuals, compared with non-injured controls. The applicability of this finding to individuals with chronic symptoms following concussion is somewhat unclear however. Kumar et. al (2009) tested their subjects an average of two months after injury, although the time since injury ranged from one to nine months. Further, 40% of their sample had documented intracranial abnormalities, including contusions and hematoma. Also, as with other EEG related findings, emotional distress cannot be ruled out as a potential explanation for alterations in EEG waveform (Nuwer et al., 2005).

The preceding review of neuropathological investigations suggests that there is much benefit in attempts to understand how concussion affects the structure and function of the brain. These technologies have the potential to reveal important information about recovery and etiology of symptoms, as they become more sophisticated and methods are refined further. In consideration of the neuropathological investigations reviewed above, several common patterns emerge. The first is that findings are predominantly inconclusive, and the literature contains many seemingly contradictory results. This is likely the product of heterogeneity in the definition of injury, as well as differences in methods and outcome measures. Secondly, it is clear that the majority of research has been conducted on individuals in the acute phase of recovery, and that more research into chronic or delayed recovery is required. Finally, neuropathological indicators are almost always subtle and nonspecific, and it is difficult to ascribe etiological significance to the findings. That is to say, the proposed indicators of brain changes associated with concussion are often similar to those widely known to be associated with psychopathological states such as depression and anxiety, and the existence of these conditions is not often controlled for. Given the similarity between the behavioural manifestations and associated neuropathology of mood disorder and concussion, it is difficult to be clear about the relative contributions that each make to the overall clinical picture, or to make clear statements about the etiology of symptoms. Without clear temporal information about the onset of symptoms, it is impossible to determine if the observed neuropathology is due to pre-existing mood disorder, or injury, or some

combination of both. Given this confound, it is not surprising that research has identified pre-injury psychiatric functioning as a significant risk factor for the development of chronic symptoms. As will be evidenced in the review below, the emotional status of the concussed individual is undoubtedly related to recovery. However, numerous other individual difference variables have also been examined, in addition to factors related to the injury itself. A review of research pertaining to these risk factors follows.

Risk Factors and Correlates

With regard to the assessment of factors associated with outcome, it is very important to be clear about the terms that are used when describing associations. As Kraemer et al., (2001) point out, “risk” refers to the probability of an outcome, a “correlate” is a factor somehow associated with outcome, a “risk factor” is best thought of as a correlate which has been shown to precede outcome, and a “causal risk factor” is a risk factor that has the ability to change the outcome. Under these definitions, all causal factors are risk factors, but the reverse is not true. Indeed, many risk factors are not causal, and to call a risk factor “causal” requires the ability to establish not only correlation and temporal precedence, but the ability to alter outcome based upon their measured values. With this in mind, many of the variables discussed below are likely best considered to be correlates with post-concussion outcome, some may be risk factors, but none have been experimentally manipulated in such a way as to meet the criteria for being “causal risk factors”. For example, emotional distress can be thought of as both an effect of injury, and perhaps a cause of further symptom reporting. As it is difficult to envision an acceptable experimental manipulation that would be able to clearly delineate causes from correlates and risk factors in an ethical manner, the question of variable importance is perhaps more tenable. A review of the research considering variable importance follows.

In attempting to understand how individual difference variables might predispose some to poor outcomes following concussive injuries, researchers have identified several variables as being potential risk factors. Factors such as age, genetic make-up, and sex of the injured person have been examined. Further, research has identified factors related to the socio-economic status of the injured person and vulnerability to being

motivated by secondary gain as potential risk factors. The emotional status and psychiatric history of the concussed individual has also been implicated in outcomes (Dischinger, Ryb, Kufera and Auman, 2009; Meares et. al, 2006, 2008; Ponsford et al., 2005, 2000, 2012; Thornhill et. al, 2000; Stulemeijer, Vos, Bleijenberg, et. al, 2007; Thornton et. al, 2008; Belanger, Spiegle and Vanderploeg, 2010; Carroll et. al, 2004; Binder and Rohling, 1996; Paniak et. al, 2002). Mixed results are also associated with investigations of factors relating to the history of previous injuries, as well as the mechanism of injury, including the nature of the forces involved and acute measures of injury severity such as length of PTA or LOC (Carroll et. al, 2004, Ponsford et. al, 2000). The relevant findings related to each of these factors are reviewed below.

Age

The age of the injured person appears to have some influence on both the prevalence of concussive injury and recovery from such injuries. On a developmental timescale, the prevalence of non-pediatric concussive injuries has a bimodal distribution, with two clear peaks observable in individuals between the ages of 15 and 24, and then again in people over the age of 65. (Abdel-Dayem et al. 1998; Kraus and Nourjah 1988; Mosenthal et al. 2004; Shukla and Devi 2010; Stapert et al. 2006; Sterr et al. 2006). Some have suggested that pediatric injuries are associated with longer recovery times and a greater likelihood of persistent sequellae than injuries which occur in adulthood (Field et al. 2003; Blinman et al. 2009; Grady 2010; Maddocks et al. 1995). Further, elderly individuals tend to suffer worse outcomes after TBI in general and concussion in particular (Stapert et al. 2006; Mosenthal et al. 2004; McCauley et al. 2001). Increased age has also been associated with delays in return to work after concussive injury (Paniak et al. 2000). However, despite these observations of the impact of age on outcome of TBI in general at the extremes of the lifespan, no clear associations have been reported between recovery from concussive injuries and age in adulthood and middle age.

Genetics

Some have suggested that genetic vulnerabilities may exist within certain populations that predispose them to poor recovery following injury. In particular the ApoE4 allele has

been investigated due to its association with the development of neurohistopathology. The ApoE protein is mapped onto chromosome 19 and is polymorphic. Around 14 percent of the general population will express the apolipoprotein E epsilon-4 isoform (Corder et al., 1993) which has been strongly associated with the development of Alzheimer's Disease. Research has suggested that this association may be due to inefficiency with which the E4 isoform catalyzes proteolytic breakdown of beta-amyloid, as compared with the two other isoforms of the allele, which can lead to the development of the amyloid plaques that are pathognomic for Alzheimer's Disease. (Wisniewski and Frangione, 1992; Jiang et al., 2008).

As similar neurohistopathology is also often observed in individuals with a history of chronic involvement in contact sports, the APOE-4 polymorphism is also generally thought to be associated with an increased risk of chronic traumatic encephalopathy in boxers (Jordan, Relkin and Ravdin, 1997; Jordan, 1998). Much of the evidence for the influence of this genetic factor on outcome derives from investigations of more severe injuries, and the association with milder injuries is less clear. For example, in a non-boxing population, ApoE polymorphism was significantly associated with death and adverse outcomes following acute traumatic brain injury as seen in a neurosurgical unit (Teasdale, Nicol and Murray, 1997). Further, in a prospective study of TBI outcome, ApoE genotypes were reported to be able to predict days of unconsciousness and functional outcome after six months (Friedman et al., 1999). In animal studies of moderate to severe TBI, transgenic mice that are bred to express the ApoE4 allele have been observed to react to laboratory induced injury with a higher degree of neuronal damage, inflammation and glial cell activation in comparison with controls (Zhou et al. 2008). However, while the literature clearly implicates these genetic factors as a contributor to poor outcome after more severe injuries (Ariza et al. 2006; Teasdale et al. 1997; Zhou et al. 2008), information concerning the impact of this polymorphism on mild injuries is less convincing. Indeed, reports vary between suggesting that E4 results in greater risk of poor outcomes (Lieberman et al. 2002; Moran et al. 2009), to no risk at all (Chamelian et al. 2004; Moran et al. 2009; Sundstrom et al. 2004) after concussive injuries. Some have even suggested that the expression of APOE4 might be a protective factor with respect to mild injuries (Pruthi et al. 2010). Still others have linked the occurrence of concussions sustained during sports to derangements in the promoter

region of APOE (Terrell et al. 2008; Tierney et al. 2010). These mixed results may be the result of methodological differences in the classification of injury severity and outcome measures, as well as epigenetic factors, but is likely also reflective of the heterogeneity in etiological pathways to the expression of symptoms (Moran et al. 2009).

Sex

The sex of the injured person has also been implicated. It has been observed that females have greater likelihood of suffering from prolonged recovery (Meares et al. 2008) at 1 month (Bazarian and Atabaki, 2001), 3 months (Dischinger et al. 2009; McCauley et al. 2001), and 1 to 5 years after injury (Bohnen et al. 1994). Women are also more likely to be concussed while playing sports than men (Dick 2009). The greater report of symptoms in women following a concussion is most pronounced with respect to the report of somatic symptoms (Farace and Alves 2000; Taylor et al. 2010). Conversely, males have been observed to be less likely to seek treatment (Demakis and Rimland, 2010), report persistent symptoms, (Bazarian et al. 2010; Demakis and Rimland, 2010) and take time off work due to symptom complaints (Bazarian et al. 2010). The extent to which these apparent sex differences are related to biological susceptibility to injury as opposed to the influence of gender roles on reporting bias is somewhat unclear. Indeed, it has been observed that women are more comfortable reporting symptoms of any sort than men are (Bazarian et al. 2010). The previous statements notwithstanding, some have speculated that sex differences in the organisation and development of the brain, such as bilaterality in the distribution of brain function, and neuroendocrinological factors might predispose women to these kinds of injuries (Farace and Alves 2000; Stein and Hoffman 2003). However, others have noted that ovarian hormones such as estrogen and progesterone are neuroprotective and enhance certain cognitive abilities (Whitfield, 2005), although these effects have not been tested with regard to recovery from concussion. Women are also more prone to depression than men (Parker and Brotchie, 2010), and again it is often difficult to differentiate the symptoms of primary mood disorder from post-concussion symptoms.

Social support

Apart from biological factors such as those outlined above, many researchers have emphasized the role of social factors in the development of poor outcomes following concussion. As even typical and nonpathological recovery from concussion can involve periods of decreased ability to function independently, time off work to recover, and even acute changes in vulnerability to emotional dysfunction, it is understandable that poor social support is a risk factor for persistent postconcussional disorder and depression when measured three months post-injury (McCauley et al. 2001). Measures of social support have been associated with quality of life following concussion as well as duration of time off work (Webb et al. 1995). Further, marital status has been suggested as a salient factor, with married individuals being less likely to report persistent symptoms than unmarried individuals (Ponsford et al. 2000). Of course, social support is also a significant factor in the development and maintenance of depression and anxiety (Grav et. al, 2011).

Financial/Secondary Gain

Economic status has also been identified as a risk factor, as individuals who have difficulty paying for healthcare or affording time off work for recovery report lower quality of life and less improvement in functional independence at both one and two years post-injury (Webb et al. 1995). The financial situation of the injured party is also relevant to considerations of compensation incentives and secondary gain. Many researchers have noted that involvement in litigation and compensation seeking are likely potent motivations to report symptoms following injury (Binder et al. 1993; Suhr et al. 1997; Youngjohn, Burrows & Erdal, 1995; Miller, 1961, Binder and Rohling, 1996; Kashluba et. al, 2008; Paniak et. al, 2002). Despite these strong claims, the effect of compensation seeking cannot be viewed as the only salient risk factor, as many individuals report chronic and persistent symptoms in the absence of compensation claims or other types of litigation (Ponsford et. al, 2012, Thornton et. al, 2008). Further, involvement in litigious proceedings may have effects beyond those attributable to compensation seeking, as the increased stress, repetitive retelling of the injury story, and pressures from lawyers and caregivers may also increase symptom reporting. In support of this, it has been observed that the effect size related to the impact that litigation plays on the

expression of persistent symptoms is not eliminated when symptom validity indices are applied (Belanger et al. 2005). Others have reported no association between involvement in litigation, receipt of insurance payments and persistent PCD at three months post-injury (Bohnen et al. 1994; McCauley et al. 2001).

Injury History

Individuals with a history of previous concussions are generally thought to be at greater risk for poor outcome following subsequent injury (Baugh et al., 2012). It has been reported that athletes with a history of three or more concussions are more likely than those with no prior history of concussion to become concussed again (Guskiewicz et al. 2003), experience acute symptoms of PTA and LOC, (Collins et al. 2002), and take longer to recover from these acute symptoms (Covassin et al. 2008; Guskiewicz et al. 2003; Iverson et al. 2004). The research regarding the influence of prior concussion on long term outcomes is somewhat mixed. Some have reported that the differences seen between those with a history of concussions and those with only one concussion do not persist beyond the acute phase of recovery (Echemendia et al, 2001; Guskiewicz et al., 2002), while others have documented differences in neurocognitive functioning at three months post injury (Wall et. al, 2006). In non-athlete or retired athlete populations, studies have demonstrated a cumulative effect of prior concussion history on the severity and duration of postconcussion symptoms complaints as well as acute cognitive outcomes (Ponsford et al. 2000; Teasdale and Engberg 2003). With regard to long term outcomes, others have noted a dose response relationship between post-concussion symptom reporting and even remote history of injury, while history of injuries was not associated with increased cognitive dysfunction on objective testing (Thornton et al., 2008).

Injury Specific Variables

Within the literature on predictors of chronic symptoms, markers of injury severity such as duration of loss of consciousness and post traumatic amnesia have not shown consistent associations with recovery (Carroll et. al, 2004; Ponsford et. al, 2000). However, the context in which the injury occurs may have particular relevance in explaining some of the variability in symptom reporting. While acute manifestations of

injury are somewhat similar across different mechanisms of injury, injuries incurred as a result of falls, sports concussion, assault, motor vehicle accident, or combat are each associated with unique attributes (Pertab et al. 2009). For example, motor vehicle accidents are associated with intense traumatic reactions and fear to a greater extent than sports injuries. Further, motor vehicle accidents may result in injuries related to direct head impact as well as “whiplash” type acceleration/deceleration type injuries injury without impact. Because acceleration-deceleration forces are much greater than in sports, motor vehicle accidents (MVA) may cause more severe injuries (Williams et al. 2010). It has been observed that symptoms such as headache and concentration difficulties are more common after motor vehicle accidents as compared to falls, cycling and sports concussions (Bazarian and Atabaki 2001; McCauley et al. 2001; Ponsford et al. 2000), even at 1 year post-injury (Sterr et al. 2006).

While it is intuitively understood that injuries sustained in potentially life threatening and traumatic situations such as motor vehicle accidents or armed combat are likely qualitatively different from those incurred at sporting events or recreational or domestic accidents, research to date has frequently chosen to aggregate findings from individuals who have sustained injuries in different contexts⁵. This is likely because a similar prognostic outlook is assumed based upon classification of the injury as “mild” and the evidence that injuries with similar acute manifestations generally resolve according to established timelines (EleMBERG, Henry, Macciochi, et al., 2009, McCrory, Meeuwse, Johnston, et. al, 2008, Binder et al., 1997; Wong, Regennitter and Barrios, 1994; Bigler, 2008, Faux, Sheedy, Delaney and Riopelle, 2011, Begaz et al., 2006; Chamelian et al., 2004; Iverson, 2006; Rees, 2003; Satz. et al, 1999, Stalnacke et al., 2005; Willer and Leddy, 2006; Belanger et al., 2005). While it has not been the case historically, modern research into postconcussion symptom expression tends to list mechanism of injury as illustrative of the make-up of the sample being used; however overt investigation of differences in symptom report between injury mechanisms is rare. Consequently, studies that ignore the circumstances surrounding the injury will be insensitive to differences that are a function of mechanism. Conversely, research that narrows its

⁵ Of course this is not always the case. For example, much research has been published regarding injuries sustained only during sporting events.

focus to injuries sustained in a homogenous manner runs the risk of lacking generalizability to the explanation of symptoms incurred through injuries with different mechanisms. Of course, consideration of mechanism of injury must also acknowledge that each individual has his or her own unique physiological and biochemical “context” with which injury related factors can interact to produce symptoms. Numerous variables are considered to contribute to the body's ability to dissipate forces incurred during injury, including individual differences in cerebrospinal fluid levels and function, vulnerability to brain tissue injury, relative musculoskeletal make-up, and the influence of forewarning, bracing, or anticipation of the injury event (Guskiewicz and Mihalik, 2011).

As previously reviewed, traditional investigations of the effects of impulsive or rotational forces may have limited generalizability to the understanding of concussive injury. These studies have been criticised for utilizing methods which are likely to result in more severe injuries than would be consistent with most concussions (Guskiewicz and Mihalik, 2011). Laboratory studies of the biomechanics of injury have tended to focus on singular force patterns, such as linear or rotational acceleration (Gennarelli et al., 1982, Kleiven, 2003), and/or impact versus impulsive force (Giza and Hovda, 2001; Shultz et al., 2011). It has been observed that rotational forces which cause the cerebrum to revolve with respect to the brainstem produce shearing and tensile strains which are associated with axonal injury in animal models (Browne, Chen and Meaney, 2011). Because activities in the midbrain and upper brainstem are responsible for alertness and responsiveness, it has been suggested that injuries involving rotational forces are more likely to result in loss of consciousness than predominantly linear forces. However, some have challenged this idea (Guskiewicz and Mihalik, 2011). The relative contributions of angular and linear accelerations are not clearly understood with respect to concussion. (Guskiewicz and Mihalik, 2011).

Injury mechanisms can also be categorized in terms of the relative contributions of impact versus impulsive force. During impact loading, the head strikes a surface, as in a fall or blow, and both contact and inertial loading takes place. In contrast, in purely impulsive head motion, as in “whiplash” type injuries, the head is not hit by an object and only inertial and acceleration loading occurs. In general, impact loading produces forces much greater than that produced by impulsive loading (Meaney and Smith 2011; Ommaya et al. 2002). It has been observed that impact loading is more likely to result in

skull fracture, brain contusion and epidural hematoma whereas inertial loading is more likely to result in injuries such as traumatic axonal injury (TAI) and subdural hematoma (Saatman et al. 2008).

Some have sought to identify a threshold of forces beyond which concussive injury is likely to occur. The *Wayne State University Concussion Tolerance Curve*, (Gurdjian et al., 1964), was created using modelling based upon a “standard model” head and skull subjected to different forces. This scale, which was the basis for force modelling for many subsequent animal studies, suggested that impacts up to 80 g should not result in injury, whereas impacts greater than 90 g could result in a concussion. Zhang et al., (2004) suggested that rotational accelerations of 4600 rad·s⁻², may be associated with a 25% chance of concussion, while 7900 rad·s⁻², would result in an 80% probability of concussion. However, others have challenged the notion that a singular threshold for concussive forces is possible to determine, due to the vast degree of heterogeneity in injury processes and individual differences in vulnerability to injury (Guskiewicz and Mihalik, 2011).

Outside of carefully controlled laboratory conditions, injuries often entail simultaneous and sequential acceleration in multiple directions and at multiple angles of rotation. Often injuries can also be characterised by both impact related and impulsive force patterns. Further, it is often the case that other injuries are sustained to the body at the same time as the concussion, which might also influence recovery. These variable mechanisms, in combination with location and duration of injury, can result in highly unique injuries, which might cause dramatically different patterns of injury distribution and severity. In general, investigations into the nature of the forces involved in injury have not shown clear associations with recovery from concussive injury (Guskiewicz and Mihalik, 2011).

Indeed, markers of injury severity are not consistently associated with outcome. The literature is mixed with regard to the influence of injury severity on the expression of post-concussion symptoms, with some suggesting that post-concussion symptom report is related to injury severity (Roe et al., 2009; Ponsford et al., 2012) and others going so far as to say that “the phenomenon of (post-concussion syndrome) does not show an association with mild traumatic brain injury” (Meares et al., 2011; p. 463). In our lab, we

have observed that the severity of exposure to injury, defined by the number of grades two or three concussions sustained, has a “dose response” relationship with the number of symptoms reported, but not objective neuropsychological measures of cognitive functioning (Thornton et. al, 2008). This observation has prompted questions about the interaction between injury characteristics and the experience of symptoms (Thornton et. al, 2008).

Psychiatric History/Emotional Distress

Current and premorbid emotional or psychiatric status has emerged as a consistent predictor of postconcussional disorder (King, 1999; Ponsford, 2012). Patients with chronic symptoms after concussion have been observed to have experienced twice as many adverse life events as have those with remission of symptoms (Fenton et al. 1993). Further, patients with a history of pre-injury life stressors are more likely to have more severe symptoms at 3 months after concussion (Kashluba et al. 2008). Depression has been identified as a common consequence of head injury in general (Gualtieri & Cox, 1991; Hanks, Temkin, Machamer, & Dikmen, 1999), and of concussion in particular, (Busch & Alpern, 1998). Depression has been shown to influence symptom reporting, but to be largely unrelated to neurocognitive performance after concussion (Rimel, et. al, 1981; Ponsford, Willmott, Rothwell, et al., 2000; Levin et. al, 1987; Ruttan and Heinrichs, 2003). Indeed, several studies have reported strong associations between negative affective states and self-reported cognitive complaints in both samples with a history of concussive injury (McCauley, Boake, Levin, Contant, & Song, 2001; Santa Maria, Pinkston, Miller & Gouvier, 2001), and samples with no such history (Binder, et. al, 1999; Errico et al., 1990; Gfeller, Gripshover, & Chibnall, 1996; Schwartz et al., 1996; Tiersky, Johnson, Lange, Natelson, & DeLuca, 1997). It has also been suggested that individuals who have sustained concussions are at greater risk for the development of anxiety disorders (Moore, Terryberry-Spohr and Hope, 2005). Anxiety in general has been reported at rates as high as 70% in mixed TBI samples (Rao and Lyketsos, 2002). In support of the role of anxiety in maintaining chronic symptoms, Harvey and Bryant (2000) estimated that 17-33% of “mTBI” patients will develop Post-traumatic Stress Disorder (PTSD). Also, in their study of the risk factors and co-morbidities of mild to moderate head injury, McCauley, Boake, Levin, Contant and Song,

(2001) found that not only did 37.5% of their participants with a diagnosis of Post-concussion syndrome have a concurrent diagnosis of PTSD, but that the odds of meeting the criteria for Post-concussion Syndrome at 3 months post-injury was 3.1 times higher in those who met PTSD criteria than in those who did not (McCauley, Boake, Levin, Contant, Song, 2001). Indeed, it has been suggested that acute emotional reactions to the experience of concussive injury can prospectively distinguish between those who recover without complication and those who will go on to develop chronic post-concussion symptoms (King, 1996, 1999). Further, Ponsford et. al (2012) report that premorbid psychiatric status and post injury anxiety were the strongest predictors of continuing symptoms at three months post injury in a prospective study which collected information regarding cognitive functioning, sex, education, history of previous head injury, injury severity, litigation, pain and medication.

Etiological Theories

As previously stated, it has been difficult to dissociate the structural and functional brain changes associated with concussion from those associated with emotional distress. It has also been difficult to determine causal risk factors for chronic symptom development. Perhaps as a result of these difficulties, it has been suggested that post-concussion symptom expression is best characterised as a psychosomatic, or “functional” illness. Two distinct models have been proposed to explain how this process might lead to the development of chronic symptoms. These models, known as “Expectation as Etiology” (Mittenberg et al., 1992) and “Good Old Days” (Gunstad and Suhr, 2001), have received wide acceptance in the literature. However, these models have not been adequately tested with regard to their ability to predict the symptom report profiles of individuals with chronic post-concussional disorder. A review of these models follows, including a comparison of their similarities and differences. As will become evident, these models both posit the importance of vulnerability to catastrophic ideation. These models differ, however, with regard to two important dimensions; the role of injury characteristics, and the development of symptoms.

Expectation as Etiology

Over two decades ago, it was hypothesized that post-concussion symptom report was highly influenced by individual beliefs, or expectations about the consequences of concussive injury. This hypothesis was coined the “expectation as etiology” hypothesis (Mittenberg et al., 1992). Mittenberg, (1992) suggested that the general population has some expectation of “post-concussion like” symptoms following injury, and that individuals reattribute commonly occurring experiences as symptoms of injury, in order to fulfill their expectations of impairment. Evidence for this model comes from two sources. The first source of evidence is based upon the relatively high incidence of “post-concussion” symptom report in healthy, non-injured individuals. For example, mild daily difficulties with memory and attention are prevalent experiences in healthy individuals, as is the occasional experience of headache, nausea, or transient dizziness. The second source of evidence for this model comes from research comparing the retrospective symptom report of concussed individuals to the current symptoms reported by non-injured controls. Specifically, individuals with a history of concussion endorse fewer symptoms when asked to reflect on their level of functioning prior to the injury, than would be predicted based upon the rate of current symptom endorsement in non-injured samples (Mittenberg et. al, 1992; Ferguson et al., 1999; Hilsabeck, 1998; Gunstad and Suhr, 2004; Lange, Iverson and Rose, 2010). Mittenberg and colleagues (1992) proposed that the reframing of common, every day complaints as symptoms of injury leads to the report of “supranormal” functioning when people are asked to report on their functioning prior to their injury. They went on to suggest that “...by underestimating the degree of premorbid symptom experience (compared with non-injured, healthy controls) ... participants overestimated the degree of change in symptoms pre-to post injury...” (Ferguson, Mittenberg, et. al, 1999; pp. 583). Under this model, perceived change in symptom levels is due to an underestimation of the experience of symptoms prior to injury. Mittenberg et. al, (1999), then propose that once this perceived change in symptoms due to injury has occurred, subsequent anxiety about the consequences of these symptoms leads to a worsening of symptoms, and eventually to full blown Post Concussional Disorder.

Good Old Days

Gunstad and Suhr (2001, 2002, 2004) extended Mittenberg's (1992) model by suggesting that the expected "syndrome" is not unique to concussion, but rather that any negative event, such as an injury or onset of illness leads to the activation of expectations of a nonspecific syndrome, which arises as a result of a "nocebo" effect. The "nocebo" effect is defined as the experience of symptoms as a direct result of expectations of symptoms (Kennedy, 1961; Hahn, 1999). In his excellent treatment of the topic, Hahn (1999), outlines three elements that are important in the explanation of the nocebo phenomenon. These elements include the expectation of poor outcome, susceptibility to catastrophic ideation, and the translation of expectations into expected experiential, behavioural and physiological outcomes. Hahn (1999), presents a model wherein expectations of outcome are influenced by susceptibility to catastrophization, which in turn result in the activation of psycho-physical processes that alter outcome. In Hahn's model, expectations of outcome vary as a function of the degree of catastrophic ideation, suggesting that catastrophic ideation is primary to the development of the "nocebo" effect. Hahn further states that the nocebo effect must arise from sincerely held conscious beliefs about illness or injury, such that "Hypochondria might be a nocebo effect...malingering, however cannot be nocebo phenomenon..." (Hahn, 1999; pg. 334). In referencing Hahn (1999), as part of their explanation of post-concussion symptom development, Gunstad and Suhr, (2004), suggest that post injury symptom report will be greater than "preinjury" reporting, due to expectations of poor outcome, and will vary according to the perceived severity of the event, illness, or injury, and the degree of vulnerability to catastrophic ideation possessed by the individual (Gunstad and Suhr, 2004). This general tendency to view oneself as healthier in the past than in the present following a negative event like an injury or illness was coined the "Good Old Days" effect (Gunstad and Suhr, 2001).

Motivation for Current Research

Both of the models presented above have been accepted on the basis of the apparent strength and reproducibility of the effects that they are based upon. However, upon closer examination of the foundational studies on which these models are based, some

methodological issues arise as potential threats to their viability. These threats include sampling issues and study design, as well as a lack of direct measurement of susceptibility to catastrophic ideation, lack of investigation of the role of injury severity, differential predictions about the development of symptoms, and the lack of consideration of the relative importance of the current emotional distress of the concussed individual to symptom report.

Sampling Issues and Study Design

Numerous researchers have cited the “Good Old Days” or “Expectation as Etiology” hypotheses interchangeably as important factors to consider in the development of post-concussional disorder (Iverson et al, 2010, Lange et. al, 2010; Garden, Sullivan and Lange, 2010; Ozen and Fernandes, 2011; Dean, O’Neil and Sterr, 2012; Heilbronner et al., 2009). The extension of these models to those who are chronically symptomatic may be preliminary however, as these models have not been adequately tested in a sample of individuals who are experiencing chronic symptoms. An examination of the original research on which the “Expectation as Etiology” and “Good Old Days” models is based reveals that sampling issues may have affected their results. Neither Mittenberg (1992), nor Gunstad and Suhr (2001, 2002, 2004) sampled from concussed subjects who were currently symptomatic. In fact, Gunstad and Suhr’s (2001, 2002, 2004) concussed group endorsed no more current symptoms than their control group, and Mittenberg’s (1992) concussed subjects actually endorsed fewer current symptoms than the control group. As such, the generalizability of these models to the prediction of chronic symptom complaints is questionable.

Recent replications of “supranormal” symptom reporting for recollections of “pre-injury” functioning in a sample of subjects with post-concussion symptoms (Iverson et al., 2010, Lange et al., 2010) have provided support for the Expectation as Etiology (Mittenberg, 1992) model. These recent investigations are important in understanding the relationship between symptom report and post-concussion syndrome, because they sampled from individuals with clear post-concussion symptoms. The applicability of these findings to individuals with chronic symptoms is limited, however, as symptom reports were collected within 3 months of injury prior to the development of chronic symptoms (Iverson et al., 2010, Lange et. al, 2010). As previously stated, it is widely

accepted that the normative recovery period for concussion can last up to three months (Binder, Rohling, & Larrabee, 1997, Faux, Sheedy, Delaney and Riopelle, 2011, Begaz et al., 2006; Chamelian et al., 2004; Iverson, 2006; Rees, 2003; Satz. Et al, 1999, Stalnacke et al., 2005; Willer and Leddy, 2006; Belanger et al., 2005).

Because the understanding of the development of chronic symptoms is most clinically important, it is necessary to test the utility of proposed etiological models such as the “Good Old Days” (Gunstad and Suhr, 2001) and “Expectation as Etiology” (Mittenberg, 1992), by sampling directly from those experiencing persistent symptoms. Given the very high prevalence of concussion, it is not realistic or warranted to provide comprehensive treatment to all people who suffer such an injury (Ponsford et. al, 2012). It is those individuals with lasting impairments, be they subjective or objective, who are most important to understand, because these individuals have a different prognostic profile, and the reason for this is not well understood (Iverson, 2005; Carroll et. al, 2004; Ponsford et. al, 2000; King, Kirkwilliam, 2011; Kirsch et. al, 2010; Lundin et. al, 2006; Nolin and Heroux, 2006). Further, because not every concussion leads to persisting symptoms, the difference between individuals with persisting symptoms and non-injured controls is less informative than a comparison of individuals with persisting symptoms and individuals with similar injuries, who have recovered in the expected timeframes. Indeed, Lange, Iverson and Rose, (2010) acknowledge this when they write “Future studies are encouraged to compare pre and post injury ratings in PCD present and PCD absent groups...”p. 449. Thus, there is a need to replicate and extend the findings associated with the Good Old Days and Expectation as Etiology models in a sample of chronically symptomatic individuals.

Another important methodological factor related to the sample composition is the influence of litigation. Iverson et. al (2010, Lange et. al, 2010) note that litigation and motivation may have had significant influences in the pattern of symptom reporting that they observed. Specifically, they present data which suggests that individuals, who are motivated to emphasize changes in functioning from the pre-injury to post-injury state, may be vulnerable to minimising the report of pre-injury symptoms. Thus, any differences in symptom reporting measured under such conditions may represent a pattern of behaviour that is due to litigation, as opposed to concussion per se.

In order to avoid confounds due to the influence of secondary gain, it would be useful to obtain a sample that is not actively litigating. Despite the inherent difficulty in obtaining such a sample, the clarity of results that is possible given the minimisation of these confounds is considered to be ideal. Perhaps due to the logistic difficulty in obtaining such a sample, to our knowledge no study has been conducted using such a rigorous sampling method.

Role of Catastrophic Ideation

Both of the models described above assert that catastrophic ideation plays a primary etiological role in the development of chronic symptoms. However, none of the original research upon which these models were based included any measure of susceptibility to catastrophic ideation. In order to make clear assertions about the role of catastrophic ideation in the development of symptoms, it would be important to directly measure vulnerability to catastrophic misinterpretation of symptoms, or the tendency to react catastrophically to adverse events.

With regard to health related symptom reporting, catastrophic ideation has been defined as the result of an interaction between a set of predisposing beliefs and the experience of interoceptive stimuli (Alloy and Riskind, 2005). Within the anxiety literature, there is some consensus that vulnerability to catastrophic ideation can be conceived of as an individual difference variable. Two measures of catastrophic ideation have been considered in the literature as relevant to the development of chronic symptoms. As outlined below, measures of Health Anxiety and Anxiety Sensitivity have been suggested as indices of the catastrophic ideation referenced in both the Expectation as Etiology and Good Old Days models.

Health Anxiety

Health Anxiety has been examined as a potential contributor to symptom report. Health Anxiety is characterised as a major contributor to the clinical diagnosis of Hypochondriasis, which is defined as the persistent preoccupation with the fear of having a serious disease or illness (American Psychiatric Association, 2000). Individuals with high levels of Health Anxiety are sensitive to even mild changes in bodily states (Weck, Bleichhardt and Hiller, 2010). As previously reviewed, many researchers

believe that Health Anxiety is a major determinant of post-concussion symptoms (World Health Organization 1992). Under this view, perceptions of illness and the adoption of the “sick role” following concussion contribute to the chronicity and severity of symptoms reported. Health Anxiety has been identified as a factor of interest in the development and maintenance of Post Concussional Disorder, and has been shown to be highly correlated with post-concussion symptom report (Stogner, 1999). This idea has received some support in the literature. For example, Whittaker and Kemp (2007), report on a longitudinal study of concussed individuals who presented to hospital as a result of their injuries. These authors obtained data on illness perceptions, depression, anxiety, post traumatic distress and other post-concussion symptoms in the emergency room, and then again at a three month follow-up (Whittaker and Kemp, 2007). Whittaker and Kemp (2007) report the results of hierarchical logistic regression analysis, which used these variables to predict whether or not individuals would meet criteria for PCD at the three month follow up. One of the illness perception variables that they measured was characterised as beliefs about the consequences of injury on social, physical, and psychological functioning. Whittaker and Kemp (2007), report that this variable emerged as an independent predictor of diagnostic status. The more individuals believed that their injury would result in negative consequences; the more likely they were to meet DSM-IV criteria for PCD at the three month follow up. Further, the addition of information regarding the severity of the injury, symptoms of post-traumatic distress, anxiety and depression did not improve the prediction model. That is to say, these variables were unable to account for more of the variance associated with diagnostic status, as compared with individual beliefs about the functional consequences of injury (Whittaker and Kemp, 2007). These findings are somewhat difficult to interpret, however, as no mention is made regarding the degree of colinearity between constructs which are known to be highly correlated. Further, the power of the model to detect contributions by other factors may have been limited, given the fact that a large number of predictors were included and the number of participants sampled from was relatively low. Also, given that the diagnosis of postconcussional disorder relies on the attribution of catastrophic consequences to the experience of injury, it is not surprising that this emerged as a significant predictor, due to the circularity of the research design. Despite these interpretive difficulties, it has been suggested that individual traits which

predispose individuals to catastrophic interpretations of symptoms should be examined further in the study of post-concussion outcomes.

Anxiety Sensitivity

Anxiety Sensitivity has also been considered in the literature, as a measurable variable which allows for the assessment of the role of susceptibility to catastrophic ideation in the expression of chronic post-concussion symptoms. Reiss and McNally, (1985) proposed the concept of Anxiety Sensitivity (AS, Reiss and McNally, 1985), which they described as an individual difference variable which captures the degree to which symptoms of anxiety may be misinterpreted as indicators of catastrophic social, somatic or psychological impairment (Reiss, Peterson, Gursky and McNally, 1986). For instance, an individual with high levels of Anxiety Sensitivity might believe that the physical experience of a fast heartbeat signals an impending heart attack, or that feelings of nervousness are a sign of severe mental illness (Reiss, Peterson, Gursky and McNally, 1986). Anxiety Sensitivity is most commonly assessed with the Anxiety Sensitivity Index (ASI; Peterson and Reiss, 1992), which assesses fears of common anxiety sensations and cognitions.

Some studies have supported the idea that AS is both empirically and conceptually distinguishable from trait anxiety (e.g. Holloway and McNally, 1987; McNally, 1989), although some have suggested that there may be some overlap between the two variables (Lilienfield, Turner and Jacob, 1998). Anxiety Sensitivity has been implicated in the expression of a wide array of anxiety disorders, including Panic Disorder (e.g. Schmidt, Lerew and Jackson, 1997), Post-traumatic Stress Disorder (e.g. Fedoroff, Taylor, Asmundson, & Koch, 2000), Agoraphobia (McNally and Lorenz, 1987) and Hypochondriasis (Bravo and Silverman, 2001). In prospective studies, high Anxiety Sensitivity has been shown to predict the occurrence of anxiety disorders and panic attacks (Maller and Reiss, 1992; Phlen and Peterson, 2002; Schmidt, Lerew and Jackson, 1997; Weems, Hayward, Killerf and Taylor, 2002). Participants high in Anxiety Sensitivity have been found to be five times more likely to develop an anxiety disorder than those low in Anxiety Sensitivity, (Maller and Reiss, 1992). Anxiety Sensitivity can also predict anxious responding to biological challenges in individuals with and without diagnoses of panic disorder (McNally, 2002). Anxiety Sensitivity has also been shown to

differentially predict anxiety, but not depressive symptoms (Schmidt, Lerew and Joiner, 1998). Thus, the Anxiety Sensitivity construct (Reiss and McNally, 1985) is an ideal candidate for the empirical investigation of the role of catastrophic ideation in the development of chronic post-concussion symptoms. This is because it is readily and reliably quantified (Peterson and Reiss, 1992), and has been shown to be empirically distinguishable from other risk factors, such as trait anxiety (Holloway and McNally, 1987; McNally, 1989) and depression (Schmidt, Lerew and Joiner, 1998).

Further, functional imaging studies suggest that the neural processes associated with Anxiety Sensitivity correlate to some degree with those implicated in recovery from concussion. Individuals high in AS have been shown to have differential patterns of PET imaged regional cerebral blood flow to the anterior cingulate, as compared with individuals low on this variable (Robillard, 2004). Further, in a study which utilized fMRI to evaluate the neuroanatomical regions associated with pain related fear and anxiety, Ochsner, Ludlow, Knierim and Hanelin et. al (2006) reported that Anxiety Sensitivity scores are predictive of medial prefrontal cortex activation in response to noxious thermal stimulation (Ochsner, Ludlow, Knierim and Hanelin et. al, 2006). These authors also report that in their sample, ASI scores were not significantly correlated with a measure of fear of pain, or with trait anxiety. Thus these authors support the contention that Anxiety Sensitivity represents a distinct construct, and suggest that higher ASI scores predict the activity of self-focused attentional processes used to monitor one's internal state (Ochsner, Ludlow, Knierim and Hanelin et. al, 2006).

Individuals who obtain high scores on the Anxiety Sensitivity Index believe that the experience of physiological symptoms is likely to result in dire consequences. As a result, it has been suggested their attentional focus is extremely rigid (Reiss, Peterson and Gursky, 1988). Individuals high in AS thus become excessively vigilant for the manifestation of such symptoms. Theoretically, this increased vigilance has the effect of increasing the detection of such stimuli, which in turn elevates anxiety levels and consequently increases the physiological manifestation of anxiety symptoms (Weems, Hayward, Killarf and Taylor, 2002). In contrast, individuals who endorse low levels of Anxiety Sensitivity do not believe that the experience of physiological symptoms has implications beyond the acute discomfort of the sensations and remain more versatile in their attentional focus (Borden and Lister, 1994).

It has been hypothesized that Anxiety Sensitivity may act as a diathesis for post concussional disorder (Wood, McCabe and Dawkins, 2011). Wood et. al (2011), compared ASI ratings and endorsement of post-concussion symptoms in a group of individuals experiencing acute symptoms following a concussion⁶ and another group who had sustained orthopedic injury. These authors reported that the concussed group endorsed higher ASI scores than those with orthopedic injury, as well as moderate positive correlations between ASI reporting and endorsement of post-concussion symptoms (Wood, McCabe and Dawkins, 2011). This exploratory study provides some evidence that Anxiety Sensitivity may be a risk factor for the development of Post-concussional Disorder, although several limitations in the study make it difficult to make strong assertions about this. These researchers did not sample from individuals with chronic symptoms, and as such the generalizability of these findings beyond the acute phase of recovery is unclear. Further, as outlined by the authors (Wood, McCabe and Dawkins, 2011), this exploratory study did not collect information regarding the influence of potentially confounding emotional factors such as depression and anxiety.

The role of catastrophic ideation in the expression of chronic symptoms remains unclear. Further research is called for in order to attempt to parse out the relative contributions of factors such as Anxiety Sensitivity and Health Anxiety, in comparison to the influence of other risk factors. The role that catastrophic ideation plays in the expression of chronic symptoms is perhaps best investigated through the direct measurement of variables such as Health Anxiety and Anxiety Sensitivity in the population of interest.

Role of Injury Severity

Apart from their agreement on the influence of catastrophic ideation, the Expectation as Etiology (Mittenberg, 1992) and Good Old Days (Gunstad and Suhr, 2004) models differ with regard to the impact of the injury on the expression of symptoms. Despite making assertions about the influence of injury severity, traditional markers of severity such as length of post-traumatic amnesia or loss of consciousness are not expressly investigated in the foundational research. Under the Expectation as Etiology model, post-concussion

⁶ The concussed group was assessed seven days after injury, which is well within the expected range for non-disordered recovery.

symptoms are not associated with the injury event per se, but are actually pre-existing, normative experiences that have been relabelled as markers of injury. The Expectation as Etiology model states that, for mild injuries that are consistent with the definition of concussion, the role of the injury is constrained to its ability to activate a pre-existing schema about the consequences of head injury in general. Any event that falls under the rubric “head injury” is sufficient to activate these expectations. Under the Expectation as Etiology framework, symptom report will be insensitive to differences in injury severity.

In contrast, the Good Old Days model suggests that symptom report is reactive to the injury event, insofar as the severity of the injury leads to the experience of greater distress. Again, it is worth noting that both of these models are specific to mild injuries which are consistent with the definition of concussion, and as such do not make predictions about injuries associated with moderate or severe brain injury. The differences in severity discussed here are all constrained by the limits imposed by the definition of concussion discussed previously. Recall that the “nocebo” effect has been characterised as reliant on not only pre-existing expectations of outcome, but is also highly influenced by susceptibility to catastrophic ideation. In his explanation of the nocebo effect, Hahn (1999), suggests that the severity of the effect will vary as a function of the perceived seriousness of the illness or injury. With regard to this topic, Hahn summarises findings provided by Barsky et al., (1993), when he writes; “Unwantedness comes in degrees, and individuals may have different thresholds regarding just how serious a condition must be to qualify as sickness” (Barsky et al., 1993; as cited in Hahn, 1999; pg. 334). As such, the nocebo effect is theoretically influenced by the interaction between the objective severity of the ailment or condition, and individual variability in catastrophic ideation. This sentiment is evident in the statement made by Gunstad and Suhr (2002), who wrote; “It is possible that a greater level of subjective undesirability may be associated with increased symptom report...” (Gunstad and Suhr, 2002; pp. 44). This idea has been recently revisited by Iverson et. al (2010), who wrote; “theoretically, having a more serious injury should be considered a greater adverse event, which should result in a stronger Good Old Days bias phenomenon” (Iverson et al., 2010; pp. 448). No research to date has investigated the

role that objective measures of injury severity might play in the expression of the “Expectation as Etiology” or “Good Old Days” models, with regard to chronic symptoms.

Misattribution of Pre-existing Symptoms versus Development of New Symptoms

Another difference between these two models is their position on the etiology and timing of symptom development. The “Expectation as Etiology” account favours the idea that while pre-existing symptoms may become more salient, new symptoms are not developed as a result of the injury. Mittenberg, (1992), cites the observation that pre-injury symptom report is relatively decreased as evidence for this hypothesis. This finding has been replicated recently in a series of investigations. Iverson et. al (2010) compared “premorbid” and post injury symptom report in individuals with a history of concussion as compared with healthy controls. Iverson et al., (2010; Lange, Iverson and Rose, 2010) observed that individuals who were deemed to be at risk for developing Post-Concussion Syndrome recalled fewer “pre-injury” symptoms, in comparison to the current symptom report of a “healthy” control group. This finding lends support to the idea that pre-existing symptoms are reattributed to injury, which is consistent with the Expectation as Etiology framework. This is contrasted with the predictions made by the “Good Old Days” model, which suggests that the experience of the injury generates a set of symptoms that have not been experienced previously. Indeed, while the “...Good Old Days model anticipates the nonspecificity of symptoms... (it) does not predict the underestimation of premorbid symptoms relative to healthy controls...”(Gunstad and Suhr, 2004; pp.400;). As previously stated, the “Good Old days” model asserts the influence of a “nonspecific nocebo effect”, which leads to the active generation of new symptoms, and has relatively little to do with perceptions of “pre-injury” symptom levels.

As such, these models can be differentiated by their predictions about the profile of retrospective and current symptom report. The Expectation as Etiology model predicts that increases in post-concussion symptoms are directly related to decreases in the report of pre-injury symptom levels, such that no new symptoms are generated. The Good Old Days model suggests that increases in post-concussion symptom report are due to catastrophic reactions to the injury event, which cause new symptoms to be expressed. Despite this fundamental difference in the predictions made by these two

models, researchers continue to cite them interchangeably, and more research is required in order to clarify the extent to which either model fits the symptom report profiles of individuals with chronic post-concussion symptoms.

Role of Emotional Distress

Throughout the review above, evidence has been presented which suggests that emotional distress is highly associated with the expression of chronic symptoms following concussion. Emotional functioning has been consistently identified as a potent correlate and risk factor with regard to the development of chronic symptoms (Ponsford, 2012; McCauley, Boake, Levin, Contant, & Song, 2001; Santa Maria, Pinkston, Miller & Gouvier, 2001). Indeed, despite suggesting the primacy of catastrophic ideation in the development of symptoms, Gunstad and Suhr, (2001), noted that depressed individuals reported more “post-concussion” symptoms than did individuals with a history of head injury, and also that depression largely accounted for elevations in current PCS symptom report (Gunstad and Suhr, 2002). As such, in order to obtain a clear understanding of the relative importance of catastrophic ideation versus current emotional disposition to the expression of symptoms, it is important to evaluate how current emotional distress influences the symptom report profiles of individuals with chronic post-concussion symptoms.

Subjective symptom report in general is highly influenced by the emotional disposition of the respondent. Indeed, a substantial literature has developed concerning the influence that emotional factors have on the report of symptoms, across a wide range of illnesses and conditions, including what has been coined “medically unexplained symptoms”. This literature suggests that the emotional disposition of the individual who is rating their symptoms affects not only the nature of the symptoms endorsed, but also contributes to the existence of observable biases in the approach to symptom report in general. Over two decades ago, Watson and Pennebaker (1989) observed that “negative affectivity” was highly associated with self-report of symptoms, and that emotional distress sometimes leads to the report of more symptoms than are supported by objective medical evidence. “Negative Affectivity” (sometimes referred to as “neuroticism”) is a general term, however, which subsumes several different emotional states, including anxiety, depression, and catastrophic ideation which, although intercorrelated, can be

differentiated from one another (Watson and Clark, 1992). These authors put forth what has come to be known as the “symptom perception hypothesis”, which suggested that individuals with high levels of distress, or “negative affectivity” report more symptoms because they are more vigilant and attentive to their bodies, more likely to notice bodily sensations, and more likely to misinterpret a wide range of sensations as indicators of illness (Watson and Pennebaker, 1989). This broad framework subsumes and homogenizes the influence of qualitatively different affective states, such as depression and anxiety. The “negative affectivity” construct also does not distinguish between the influence of affective state and cognitive biases such as vulnerability to catastrophic ideation. In order to be clear about the ways in which the emotional state of the respondent might influence symptom report, it may be instructive to take a somewhat more nuanced approach.

In a recent study by Howren and Suls (2011), the “symptom perception hypothesis” proposed by Watson and Pennebaker (1989) was further investigated, under the hypothesis that anxiety and depression have differential influences on symptom report. These authors observed that depressed individuals tend to recall more negative than positive information, especially when the information to be recalled is self-referential in nature (e.g. Mineka and Nugent, 1995; Matt, Yazquez and Campbell, 1992; as cited in Howren and Suls, 2011). Further, they noted that highly anxious individuals do not demonstrate this negative recall bias (Coles and Heimberg, 2002; Mathews and Macleod, 1994; Mineka and Nugent, 1995; as cited in Howren and Suls, 2011). Howren and Suls (2011) report that anxious individuals tend to report more current symptoms, but that anxiety does not have an impact on retrospective recall of symptoms. Others have observed that depressed individuals have a consistently negative bias in their retrospective recall of symptoms (Ben-Zeev and Young, 2010), and that anxious and nonanxious individuals are equally accurate in their retrospective report of symptoms (Rabin et. al, 2001). In light of these findings, it is important to consider the impact of affective state on the symptom report profiles of individuals who have suffered concussive injury.

Again, it has been difficult to establish a chain of causality between emotional dysfunction and the report of chronic post-concussion symptoms, as one may be thought to cause the other, and/or vice versa. This is in part due to the significant overlap

between the neuropathology and behavioural manifestations of concussion, and those of mood disorder. Also it is obviously difficult to design studies which can experimentally manipulate both affective state and the experience of concussive injury. Further, studies which attempt to comment on the association between affective state and either the expression of post-concussion symptoms, or the risk of meeting criteria for Post-concussional Disorder are vulnerable to criticism on the grounds that they are employing circular logic. Because Post-concussional Disorder and post-concussion symptoms in general are in part defined by the expression of emotional symptoms, it is difficult to make clear assertions about the influence of one on the other. For example, one may observe that depressed affect is a significant predictor of chronic post-concussion symptoms such as difficulty concentrating, and conclude that depression is a risk factor for Post-concussional Disorder, however, this conclusion may be erroneous due to the fact that difficulty concentrating, while traditionally thought of as a “cognitive” symptom in the concussion literature, is also considered to be a symptom of depression. In this example, the researcher is left able to state only that symptoms of depression are highly correlated with symptoms of depression. In order to contribute to the understanding of the relative influence of affective state on post-concussion symptom reporting, a different method may be more useful.

With regard to establishing the relative contributions of current emotional distress and catastrophic ideation to the development of Post-concussional Disorder, it is ideal to avoid circularity. The defining feature of poor recovery or Post-concussional Disorder is arguably not the experience of symptoms. While the experience of cognitive, affective, and somatic symptoms are necessary for the characterisation of pathological recovery in any diagnostic rubric, they are not sufficient. Most individuals experience some degree of symptoms following concussion, and these symptoms may even linger longer than expected, but only the subset who are rated as subsequently impaired in their social and occupational functioning meet established criteria for PCD. Indeed, the diagnostic criterion suggested in the DSM-IV requires that “the disturbance causes significant impairment in social or occupational functioning...” in order for a diagnosis to be made. As with any other psychological diagnosis then, it is not the experience of symptoms, but the degree of impairment that ultimately defines whether or not the individual meets criteria for diagnosis. As a thought experiment, consider the hypothetical situation in

which two individuals, A and B, present with similar symptoms. The symptoms are rated by both individuals as similar in terms of frequency and severity; however, individual A also reports that they are significantly impaired in social and occupational functioning, whereas individual B does not associate these symptoms with impairment. Under this scenario then, one individual meets criteria for disorder, whereas the other does not, despite the similarity in symptom report. With this distinction in mind, it is possible to assess the relative contributions of risk factors such as the emotional disposition of the respondent and catastrophic ideation to the development of disorder while avoiding the circularity that occurs in using symptoms to predict symptoms.

With respect to the reliance on ratings of social and occupational functioning in the determination of disorder, a debate is beginning to emerge in the literature. Some have pointed out that the definition of “impairment” has not been sufficiently operationalized to allow for reliable measurement (Usten and Kennedy, 2009). These authors have also pointed out that medical diagnosis separates criteria pertinent to the existence of disorder (i.e. signs and symptoms) from evaluation of the severity of the disorder (i.e. disability or functional impairment), and suggest that evaluation of mental or emotional disorders should also separate these constructs. Of course, it is true that severity of symptoms is often correlated with the degree of disability associated with any condition, but it is also important to note that severity of symptoms is conceptually and phenomenologically separate from impairment or disability as a result of those symptoms. Usten and Kennedy (2009), argue that the DSM assertion that symptoms must be associated with impairment in order to be classified as evidence of disorder conflates and confounds the existence of disorder with effects of disorder and suggest that these constructs should be separated.

Another view is specified by Wakefield (2009), who points out that consideration of the level of impairment has a place in diagnosis to the extent that it can characterise the harm caused by the underlying pathological process, and separate the experience of symptoms that are not indicative of disorder from those that are qualitatively different. In support of this view, Wakefield (2009) provides the example of medical and biological dysfunctions, such as benign angiomas, which are not classified as disorder because they do not result in harm. Wakefield (2009) suggests that all individuals have biological flaws and dysfunctions that would not be classified as disorder due to the lack of

observable harm or disability associated with the condition. He further points out that sometimes there is no independent way to establish that there is a dysfunction versus a non-disordered problem of living other than by evaluation of social and occupational impairment.

These considerations are important in the investigation of disordered recovery from concussion because the symptoms, as many researchers have pointed out, are not specific to concussion, and are experienced with relative frequency in “healthy” or non-injured controls. As such, it is important to define some threshold which can distinguish between the existence of disorder, and experiences that are largely normative. Again, it is recognised that disorder likely exists along a continuum which ranges from normative symptom experiences to severe disability, and several authors have pointed out that the threshold model may not adequately capture the variability in presentation and comorbidity observed across mental and emotional disorders. However, it is also clear from the literature that a subgroup of individuals exists, whose recovery from concussive injury is phenomenologically different from most others, and some method for distinguishing between these groups is important in order to research how best to help those who do not recover normatively. The DSM specification that symptoms be associated with significant social and occupational impairment is considered a viable way to classify disordered versus non-disordered individuals and is widely accepted and used in current diagnostic practice, despite some acknowledgement that it is not without flaw.

Further, as the degree of impairment experienced by the individual is arguably the most important factor in the assessment of the efficacy of clinical interventions, it is useful to understand how risk factors contribute to this outcome. By not relying on symptom report as the outcome variable of interest, it is possible to avoid the circular reasoning that has plagued this research to date. Using ratings of social and occupational impairment due to injury as the outcome variable would allow for the assessment of the relative contributions of emotional, cognitive and somatic symptoms, as well as other risk factors such as injury severity and catastrophic ideation to the development of disorder. To date, no study has utilised such a design to address these issues.

Another potential difficulty inherent in investigations of depression, anxiety, and catastrophic ideation is that these variables are typically highly correlated with each other. Previous researchers have taken several approaches to this problem, including producing compound variables which do not distinguish between factors, or merely not including correlated factors in analyses. It is possible, however, to assess the relative importance of emotional state and catastrophic ideation through careful study design and analysis. For example, although health anxiety and anxiety sensitivity both include the term “anxiety”, neither of these theoretical constructs is primarily affective, or emotionally based. Both anxiety sensitivity and health anxiety reference specific cognitive biases, or thought processes, rather than affective states. Indeed, as reviewed above, previous researchers have observed that these cognitive biases are dissociable from the affective states of depression and state anxiety (Holloway and McNally, 1987; McNally, 1989; Schmidt, Lerew and Joiner, 1998). However, although susceptibility to catastrophic ideation has been identified as dissociable from affect, it is noted that significant correlations are still often observed between constructs. Further, despite being conceptually different states, depression and anxiety also have been shown to have significant commonalities, and to be highly correlated with one another.

Two methods in particular can aid in the investigation of the relative contribution of these factors to chronic impairment. The first method is to carefully review the measures of each factor in order to ensure that they are as free from contamination by related constructs as possible. To the extent that construct measures include items that index similar states, sensations, or biases, these measures will be difficult to dissociate from one another. Numerous factor analytic studies and item analyses can be consulted in order to make measures as distinct as possible. The second method is to use certain techniques of statistical analysis that allow for the clear assessment of variable importance and influence when variables are somewhat correlated. In particular, recent statistical methods are capable of quantifying the degree to which variables provide unique explanatory power to the expression of impairment, as well as the degree to which constructs work together to produce an outcome. To date, no study has employed these techniques to address the differential contributions of emotional distress and catastrophic ideation to chronic impairment following concussion.

Objectives and Hypotheses

The current study was designed to investigate the viability of the “Good Old Days” and “Expectation as Etiology” models with respect to the generation of symptoms and whether they arise from misattribution and catastrophic ideation. A further objective was to determine the relative importance of risk factors such as susceptibility to catastrophic ideation, emotional state, and injury severity to self-reported ratings of social or occupational impairment due to injury. These objectives fall into three categories:

Objective 1) “Test of Misattribution Hypothesis”. The current research was designed to test the viability of the predictions made by the Expectation as Etiology and Good Old Days models with regard to the way in which current symptom report is generated in individuals with chronic symptoms. Both the Expectation as Etiology and Good Old Days models predict that current symptom reporting will be greater than retrospective symptom reporting (Mittenberg, et al., 1992; Gunstad and Suhr, 2004). The Expectation as Etiology model, however, suggests that the increase in current symptom report is due to a misattribution of pre-existing symptoms, whereas the Good Old Days model suggests that new symptoms are developed after the injury has occurred. In order to test these models it was hypothesized that individuals with chronic symptoms would endorse fewer “pre-injury” symptoms than those who had recovered from their injuries. Thus, the association between current symptoms ratings and estimates of “pre-injury” symptom levels, across disordered and non-disordered groups, are of interest. Support for this hypothesis would be evident if individuals with chronic symptoms reported the experience of fewer symptoms when they recalled the period prior to being injured, than recovered individuals did when recalling the same time period. This finding would lend credence to the idea that chronic symptoms are the result of a reattribution of pre-existing symptoms, as outlined by the Expectation as Etiology model.

Objective 2) “Test of Injury Severity Hypothesis”. The Expectation as Etiology and Good Old Days models also make dissociable predictions with respect to the influence of the injury event on self-reported ratings of social or occupational impairment. Specifically, the Good Old Days model predicts that report of impairment will increase as a function of the severity of the injury, because the “nocebo” effect is thought to be sensitive to changes in event severity. Conversely, under the Expectation as Etiology

framework, differences in injury severity are unrelated to outcome. In order to test these models, it was hypothesized that increased injury severity, as defined by the duration of alteration in consciousness and/or number of prior injuries would be associated with increases in ratings of impairment. Support for this idea would be found in the association between measures of the impact of injury on the central nervous system, as indexed by degree of alteration in consciousness and loss of consciousness, and the self-rated social and occupational impairment. A strong positive association would support the hypothesis that disordered recovery varies as a function of injury severity, which is consistent with the Good Old Days model.

Objective 3) “Test of Relative Importance of Catastrophic Ideation”. As previously stated, both the Expectation as Etiology and Good Old Days models assert that vulnerabilities to catastrophic ideation are primary influences on the development of post-concussion symptoms. However, a review of the literature consistently implicates affective distress as a highly potent risk factor for chronic post-concussion symptom complaints and impairment. As a final test of the utility of these models, it was hypothesized that measures of catastrophic ideation such as Health Anxiety and Anxiety Sensitivity would emerge as the most powerful predictor of social and occupational impairment, and that its influence would be greater than the contributions of other risk factors. This finding would suggest that catastrophic ideation is indeed a pivotal factor in determining chronic impairment, and provide support for the “Expectation as Etiology” and “Good Old Days” models. The relative contribution of measures of catastrophic ideation, as compared with other known risk factors, in determining ratings of social and occupational functioning is the focus of this portion of the investigation.

Chapter 2.

Method

Participants

Participants were recruited from the community through a broad advertising campaign including newspaper, radio, internet, magazine, and poster advertisements, as well as group presentations and talks by the principal investigator⁷. Participants all reported the experience of at least one concussive injury, which occurred at least three months prior to interview. In order to avoid errors due to difficulty with recall of the details of injuries, participants were excluded if they reported that their most recent concussion was twenty or more years prior to involvement in the study. In order to ensure that results were applicable to adults with concussive injuries, participants with pediatric injuries (occurring under age 12), or with a history of moderate to severe head injury, including “complicated” mild injuries⁸, were excluded. All participants were between the ages of 18 and 64 years of age. None of the participants were actively litigating. Recruitment took place over the course of four years. One hundred and twenty four individuals responded to the advertisements and were screened. Thirty declined participation after being screened, 19 were deemed to have sustained moderate to severe brain injuries based upon medical documentation and self-report of injury parameters, seven were excluded because they had sustained concussions prior to age twelve, four reported that their most recent concussion was over 20 years prior to being screened, one responder was under the age of 18, one was excluded due to active involvement in litigation, and one was determined to have never experienced a

⁷ Note that recruitment methods used for this study included broad advertising; hence the study response rate could not be calculated.

⁸ Complicated injury was defined as any injury that resulted in skull fracture or positive signs of structural damage or hematoma on neuroimaging.

concussion. Thus, 61 subjects met inclusion criteria. Participants were paid \$20.00 for their time. Participants were fully informed about the nature and purpose of the study and gave consent in writing. Medical records pertaining to the reported injuries were obtained and reviewed in cases where such records existed. Individuals with chronic symptoms were offered treatment through the Clinical Psychology Centre at Simon Fraser University. All data was collected in accordance with the Simon Fraser University Ethics Review Board policies and procedures. The study methods were approved by the Simon Fraser University Ethics Review board. All participants provided informed consent and were debriefed upon completion of the study.

Measures

Determination of Concussion History and Recovery Course

Brief Concussion Interview Form

All participants were pre-screened using the Brief Concussion Interview Form (BCIF, Whitfield, 2007; see Appendix A) to determine group membership and assess for exclusion criteria. The Brief Concussion Interview Form was developed in our lab to collect information about history of head injury and symptoms experienced as a result of concussion. Further, the BCIF collects information about the social and occupational impact of changes in functioning due to head injury. The BCIF asks questions that allow potential participants to be classified according to a modified version of the proposed symptom reporting criteria for Post Concussional Disorder as stated in the Diagnostic and Statistical Manual of Mental Disorders (4th edition, TR; American Psychiatric Association, 2000).

Sports Head Injury Questionnaire

Participants were also administered the first two pages of the Sports Head Injury Questionnaire (SHIQ, Thornton and Cox, 2002; Available on request) in interview form. The 'Sports-related Head Injury Questionnaire' (SHIQ) was designed to retrospectively document parameters associated with concussion, including the number and frequency of concussive events and the severity of such events. This measure has demonstrated

adequate reliability in the assessment of concussion history (test-retest $r = .79$) (Thornton, Cox & Powter et al., 2002).

Assessment of Physical and Psychological Health History

ADIS-IV

The Anxiety Disorders Interview Schedule for the DSM-IV (ADIS-IV) was used to assess for comorbid psychological difficulties, including anxiety disorders and depression, as well as general health and psychiatric difficulties. The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV) is a structured interview designed to assess for current episodes of anxiety disorders, and to permit differential diagnosis among the anxiety disorders according to DSM-IV criteria. The ADIS-IV includes worksheets that allow clinicians to not only determine whether or not the individual meets criteria for disorder, but also to provide ratings concerning the severity of the social and occupational impact of psychological difficulties associated. In addition, sections to assess current mood, somatoform, and substance use disorders are included. The ADIS-IV also contains screening questions for psychotic and conversion symptoms and familial psychiatric history, as well as a detailed section about the participant's medical and psychiatric treatment history. The ADIS-IV has shown good to excellent reliability in the diagnosis of anxiety disorders, with inter-rater reliabilities that range between .67 and .86, depending on the diagnostic category under consideration (Brown, DiNardo, et al, 2001).

Post-Concussion Symptom Reporting

Pre-injury and current levels of post-concussion symptoms were obtained by asking participants to complete the Post-Concussion Symptom Checklist (PCSC). The Post-Concussion Symptom Checklist (PCSC; Gunstad & Suhr, 2001) is a 97-item instrument consisting of several subscales which measure the frequency of cognitive, emotional and somatic symptoms. The checklist also contains a set of "Distractor" items which are not commonly associated with post-concussion symptoms. Symptom severity is rated on a 5-point Likert scale with the choices of never, rarely, occasionally, often, and always. This checklist has excellent internal reliability (.97) and test-retest reliability at two weeks (.88; Gunstad & Suhr, 2001). The sum of the frequency ratings obtained for

each of the cognitive, emotional, and somatic and distractor subscales on the PCSC (Gunstad & Suhr, 2001) were calculated. An omnibus Total score was also calculated as the sum of the cognitive, emotional and somatic subscales. The distractor subscale was not included in the Total score as it is not considered representative of post-concussion symptoms.

Emotional Distress

Zung Self-Rating Depression Scale

Current levels of depressive symptoms were measured using the The Zung Self Rating Depression scale (SRDS; Zung, 1965), a widely used instrument that contains 20 items which are rated on a 4 point scale. Responders are told to rate the frequency with which they experienced depressive symptoms in the week prior to filling out the questionnaire. Several studies have established the SRDS as a reliable (.88, Gabrys and Peters, 1985) and valid instrument for measuring depressive symptoms (Biggs et al, 1978; Gabrys and Peters, 1985; Agrell and Delhin, 1989; Thurber, Snow and Honts, 2002). As with many other measures of depressive symptoms, the SDRS includes items that do not directly index depressive affect per se. Factor analysis of the scale has indicated that it contains three subscales, pertaining to somatic symptoms, cognitive symptoms, and affective symptoms (Sakamoto et al., 1998). Because the factor of interest to the current study is the influence of depressive affect, only this subscale was included in the analyses⁹. For example, items such as “I feel downhearted and blue” were taken as more representative of depressive affect than “I find it easy to make decisions” (cognitive) and “My heart beats faster than usual” (somatic).

State-Trait Anxiety Inventory

Participants also completed the State Trait Anxiety Inventory. The State-Trait Anxiety Inventory (Spielberger et. al, 1983) is a widely used instrument which measures the degree to which individuals are currently experiencing a “state” of anxiety, as well as the individual’s general level of “trait” anxiety. The measure consists of 20 “state” items (e.g. I feel calm; I am tense; etc.). The participant is asked to rate the representativeness of

⁹ The reliability of this subscale is unknown.

that statement to their current state on a four point scale from “not at all” to “very much so”. The measure also consists of 20 “trait” items (e.g. I feel nervous and restless; I am happy; etc.), which are rated in frequency on a four point scale which ranges from “almost never” to “almost always”, with reference to the participant’s general experience of these feelings. According to the test-retest correlations provided by Spielberger et al. (1983), the measure has demonstrated adequate reliability for both state (.54) and trait (.86) level measurements. The STAI has been criticised for its inclusion of items that measure depressive symptoms, and it has been suggested that these items be omitted in order to clarify the measurement of symptoms of anxiety (Bieling et al., 1998). Further, the scales of the STAI include items which ask about the presence of anxiety, as well as items which ask about the absence of negative affect. Numerous researchers have suggested that items which ask only about the absence of negative affect are not useful in detecting the presence of anxiety (Bernstein and EVELAND, 1982; Benson and Hocevar, 1985; Pilotte and Gable, 1990; Schriesheim and Hill, 1981). Factor analyses of the entire STAI have consistently yielded results which suggest that questions which ask about the presence of anxiety load on different scales than those that ask about the absence of negative affect (Vigneau and Cormier, 2008; Matschinger and Angermeyer, 1992; Pilotte and Gable, 1990; Vautier and Pohl, 2009). Cox et al., (1993) performed a factor analysis of responses the State anxiety subscale of the STAI, and the Beck Depression inventory. These authors report that a three factor solution emerged from their data, and that items could be characterised as measuring depression, absence of negative affect, and state anxiety (Cox et al., 1993). For the purposes of this research, it was important to only include items thought to be directly related to the experience of state anxiety, in order to minimise construct overlap. Thus, the state anxiety subscale identified by Cox et al., (1993) was used¹⁰. These items were as follows; 3, 4, 6, 7, 9, 12, 13, and 17.

¹⁰ The reliability of this subscale is unknown.

Vulnerability to Catastrophic Ideation

Illness Attitudes Scale

Health anxiety was measured using the Illness Attitudes Scales (IAS; Kellner, Abbott, Winslow, & Pathak, 1987). The IAS is one of the most-used questionnaires for the evaluation of hypochondriacal attributes and is currently the gold standard in self-rated assessment of health anxiety (Sirri, Grandi, & Fava, 2008). The IAS has been shown to be one of the most suitable screening measures for hypochondriasis (Speckens, 2001). The Illness Attitudes Scales consist of twenty-eight true/false questions that address worry about illness, concern about pain, health management behaviours, hypochondriacal beliefs, thanatophobia, disease phobia, bodily preoccupation, treatment experience, and impact of symptoms on daily functioning (Bravo and Arrufat, 2005). The measure has been shown to have adequate validity, reliability (.84) and consistency (Bravo and Arrufat, 2005; Bravo and Silverman, 2001). The IAS has been examined using factor analytical techniques, which have identified loadings on four main subscales (Stewart and Watt, 2000). The subscales include; a) Fears, including fear of illness and/or death (eg. “Does the thought of serious illness scare you?”), b) Behaviours, including health related behaviours (eg. Do you avoid foods that may not be healthy?), c) Beliefs about the consequences and meaning of symptom experiences (If you have a pain that lasts a week or more, do you believe that you have a serious illness?), and d) Effects of the effects of illness on one’s life (Do your bodily symptoms stop you from enjoying yourself?). As the influence of susceptibility to catastrophic ideation is of the most interest in the current study, only items from the subscales loading on hypochondriacal fears and beliefs were included¹¹. Information regarding health related behaviours and the effects of illness were not considered indicators of the vulnerability to catastrophic ideation proposed above.

Anxiety Sensitivity Index

Finally, Anxiety Sensitivity was measured using the Anxiety Sensitivity Index (ASI; Peterson and Reiss, 1992). The ASI is a 16 item self-report questionnaire that measures the severity of the respondent's concerns about psychological, physiological,

¹¹ The reliability of this subscale is unknown.

and socially relevant symptoms of anxiety. Items are scored from 0 to 4 and the total score is obtained by summing all item scores (higher scores indicate greater anxiety sensitivity). The ASI has demonstrated adequate test-retest reliability in individuals prone to panic attacks at 3 year intervals (.71; Maller and Reiss, 1992). The ASI has also been subjected to factor analysis, which has revealed that the items are best categorized by a three factor model (McWilliams and Cox, 2001). The three factors identified include fears of physical sensations (eg. It scares me when I feel faint); fears of psychological instability (eg. When I am nervous, it scares me that I might be mentally ill), and fears of social embarrassment (It is important for me not to appear nervous). In a comprehensive review of previous research into the validity of the instrument, as well as original investigation of the convergent validity of the items on the ASI, it has been observed that several items detract from the usefulness of the scale in measuring Anxiety Sensitivity (Blais et al., 2001). These authors found that the removal of five items resulted in a more precise measurement of Anxiety Sensitivity (Blais et al., 2001). The items recommended for removal are likely to measure other constructs, such as social anxiety (items one, five and thirteen; eg. Other people notice when I am shaky); and emetophobia (items seven and eight; eg. It scares me when I am nauseous). Thus, the recommended eleven item version¹² was utilised in the current research.

Procedure

Participants were self-referred in response to a broad advertising campaign. Individuals telephoned the clinical psychology centre at SFU if they were interested in participating, had sustained a concussion at least three months prior to participation and were not currently involved in litigation or compensation seeking. Upon initial contact, each participant was pre-screened by telephone using the BCIF, and invited to attend the clinical psychology centre at SFU if they met the inclusion criteria for the study. Upon arrival at the CPC, participants were provided with an information document outlining the nature of the study and were allowed to ask sufficient questions to be able to give informed consent. All participants were then interviewed in person by a doctoral student

¹² The reliability of this subscale is unknown.

in clinical psychology, using the ADIS-IV. Interviewers were trained in psychological interviewing and assessment in general and given specific instruction and observed practice in administration and scoring of the ADIS-IV using materials provided by the publishers. Upon completion of the ADIS-IV, the interviewer continued the interview using the SHIQ. Each participant then completed the rest of the self-report questionnaires under supervision by the interviewer, who was trained to be able to answer any questions relevant to the completion of these forms. All participants completed the PCSC twice, once to report current symptoms, and once to report on symptoms that may have been experienced prior to injury. In order to avoid response bias due to presentation order, the order of presentation of the PCSC's was counterbalanced across participants. Data collection sessions lasted an average of 2.5 to three hours per participant.

Demographic Information and Risk Factors

Information regarding age, education and sex of the participants were recorded, as well as injury parameters necessary to determine severity of each event. Both present versus absent counts and severity ratings for each of the psychological disorders covered in the ADIS-IV were recorded for each participant and these severity scores were summed to create variables reflecting psychological health across all of the disorders surveyed as well as those pertaining to only anxiety disorders.¹³ Potentially confounding physical ailments were assessed by the presence or absence of self-reported involvement in medical treatment as well as endorsement of a history of health problems. The list of health problems included cardiac, respiratory, neurological, endocrinological, gastrointestinal, hematological and immunocompromising conditions.

Participants rated their confidence in the details they provided about injury history on a five point scale, with five representing "Extreme" confidence. Endorsement of the presence or absence of a set of "life stressors" within the year immediately prior to participation was also recorded for each participant, including perceptions of

¹³ Counts reflect endorsement of sufficient symptoms to qualify for diagnoses, if those symptoms were endorsed as detrimental to the individual's current social and occupational functioning. Severity ratings reflect the rater's perception of the degree to which these symptoms are endorsed as problematic.

significant changes or difficulties in relationships, legal or criminal matters, occupational or scholastic endeavours, financial affairs, personal health and the health of significant others. These questions are part of the ADIS-IV pre-interview and were used to characterise the sample, and not as outcome measures.

Classification of Chronically Symptomatic and Recovered Groups

For investigations pertaining to differences between “pre-injury” and current symptom report, participants were grouped according to their responses to the BCIF. As previously stated, the BCIF allows for the classification of individuals based upon modified DSM-IV criteria for Post-Concussional Disorder, which does not require objective evidence of neurocognitive impairment. Specifically, individuals were classified as Chronically Symptomatic if they met the following criteria: a) current experience of three or more symptoms such as fatigue, disordered sleep, headache, dizziness, irritability, anxiety-depression, personality changes, and/or anhedonia; b) symptoms reported to have been unremitting since injury and to have lasted at least three months post injury; c) attribution of current symptoms to the experience of a concussion; d) self-rated social and occupational impairment greater than four on an eight point scale, with eight representing severe impairment. Specifically, individuals were asked to “Please rate the degree to which your symptoms have impacted your social or occupational functioning.” Ratings were made on an eight point scale that ranged from “no impact”, to “severe impact”. Ratings of four or greater on this scale were considered to represent significant social or occupational impairment sufficient to meet the DSM-IV diagnostic criteria pertaining to impairment. The ratings of perceived social and occupational impairment due to injury for all participants were also used as a continuous variable for analyses involving the relative importance of risk factors, and served as the main outcome variable for objectives related to injury severity and the relative importance of catastrophic ideation. Self-rated social and occupational impairment was considered to be a more useful index of disordered recovery than diagnostic status for hypotheses surrounding the relative importance of risk factors, because ratings of social and occupational impairment are not necessarily dependent on the values of those risk factors. Using this variable as the outcome measure in this way avoids confounds due to circularity in the research design.

Age matched subgrouping

In order to minimise the possibility of confounds created by differences in age, participants were subjected to a second grouping which separated groups according to diagnostic status as determined by the BCIF, and matched these groups according to age. Age matching was achieved by systematically removing the youngest subjects from the sample until relatively equal diagnostic groups were formed that did not differ in age.¹⁴

Composition of Groups

Table 4 on the following page presents data pertaining to group differences between the chronically symptomatic and recovered groups. Detailed information regarding current psychiatric and health concerns is listed in Appendix C.

For the full sample analysis, the groups were well matched on education, sex, number of injuries sustained, severity of injuries, the amount of time that had passed between the injury and the interview, subject confidence in their report of injury parameters and measures of overall psychological health and life stress. As is apparent in the table, the chronically symptomatic group was older than the recovered group $F(1,59) = 5.44$, $p < .05$. The chronically symptomatic group was also more likely to have incurred other injuries at the same time as their concussion $\chi^2(2, 61) = 7.10$; $p < .05$, as well as to have sought out medical intervention as a result of their injury $\chi^2(2, 61) = 4.73$; $p < .05$.

¹⁴ Other classification criteria were examined as well, including the use of a modified version of the ICD-10 diagnosis for post-concussional syndrome which excluded lowered tolerance for alcohol, stress and emotional excitement. A further classification based whether or not the individual had ever met the proposed modified criteria for PCD was also assessed. Neither of these alternate classifications for susceptibility to persisting symptoms resulted in different results during hypothesis testing.

Table 4. Composition of Chronic and Recovered Groups

	Chronic		Recovered			Age Matched Recovered		
Sample size (F/M)	20 (10/10)		41 (12/29)		$\chi^2 = 2.51$	20 (8/12)		$\chi^2 = .40$
Variable	M	SD	M	SD	F (U)	M	SD	F (U)
Age	42.10	11.70	33.90	13.40	5.44*	45.60	10.30	0.88
Education	14.10	2.80	14.60	2.30	0.46	14.60	3.10	0.25
Total Concussions	5.53	4.88	7.44	8.72	(354.00)	7.05	9.11	(178.00)
Grade 2 or 3 concussions	3.00	3.13	4.42	6.14	(359.50)	4.95	7.11	(159.00)
Length of PTA for most severe event (mins)	583.06	673.81	194.18	391.57	(236.50)*	244.47	454.79	(129.50)
Time since injury (mos)	49.60	65.54	60.25	63.46	(322.50)	93.11	72.45	(101.00)
Self-rated report accuracy (max 5)	4.10	0.57	3.90	0.76	1.65	3.90	0.89	0.42
Medical Involvement	N	% of sample	N	% of sample	χ^2	N	% of sample	χ^2
Went to hospital/doctor	18.00	90.00	26.00	63.00	4.73*	17.00	85.00	0.78
Medical Verification Reviewed	10.00	50.00	9.00	22.00		6.00	30.00	
Other Injury	13.00	65.00	12.00	29.00	7.1*	8.00	40.00	2.51
CT (+/-)	10 (0/10)	50.00	13 (0/13)	32.00		8(0/8)	40.00	
EEG. (+/-)	5 (0/5)	25.00	6 (0/6)	15.00		3(0/3)	15.00	
Psychological Health	M	SD	M	SD	F (U)	M	SD	F (U)
Current Life Stressors (max 6)	2.60	1.60	2.40	1.50	0.39	2.80	1.50	0.06
Total Severity of All ADIS-IV Ratings	8.21	7.40	3.15	4.13	(196.00*)	4.35	4.83	(127.50)
Total Severity of ADIS-IV Anxiety Ratings	5.68	4.44	2.64	3.57	(205.00)*	3.35	4.08	(126.00)
Physical Health	M	SD	M	SD	F	M	SD	F
total physical health score (max 18)	2.11	1.64	1.21	1.06	6.31*	1.55	1.00	1.66

* $p < .05$

Finally, the chronic group also endorsed significantly more difficulties with their current physical health $F(1, 59) = 6.31; p < .05$. When groups were matched more closely by

age, group differences on variables of interest were effectively nullified, such that the two groups were well matched on each variable, apart from the length of time since injury, which was somewhat longer in the recovered group $F(1, 39) = 7.58, p < .05$.

Injury Severity

Participants reported the length of Post Traumatic Amnesia and acute confusion in minutes. For those individuals with a history of multiple concussions, the length of PTA for the most severe event was used. All participants but one reported at least fifteen minutes of Post Traumatic Amnesia. This variable was then dichotomised using sixty minutes as a threshold marker for injury severity. As such, individuals whose most severe event was associated with less than sixty minutes of PTA were deemed to have incurred less severe injuries than those who reported sixty minutes of PTA or greater. As the lower limits of concussion are not well defined, the sixty minute PTA threshold has been suggested to be a reasonable transition point between mild and more severe events (Hannay, Howieson, Loring, Fischer and Lezak, 2004; Rao and Lyketsos, 2000). Presence or absence, as well as length of loss of consciousness were also recorded, as this has been suggested to be associated with injury severity (American Academy of Neurology Practice Parameter Grading System, 1997). Injuries were classified as relatively more severe if they were associated with loss of consciousness of five minutes or greater, and/or a period of post-traumatic amnesia lasting longer than sixty minutes.

Initial data preparation and Statistical Considerations

Data analysis was conducted using SPSS 17, and statistical significance was reported at the 0.05 level. All variables were each inspected for normality and the presence of outliers using the method proposed by Tabachnick and Fidell (2007). Univariate normality was assessed through the calculation of standardised scores for skewness and kurtosis of each variable, and comparison of these scores to a critical value representing significant difference from normality at the .01 level. Using this method, several variables were identified as non-normally distributed and were subjected to transformation. Transformation was undertaken in an iterative and stepwise manner, as per Tabachnick and Fidell (2007) in order to identify the appropriate level of transformation. As a result of this process, square root transforms were applied to the

affective subscale of the Zung Depression scale and the eleven item Anxiety Sensitivity Index scores, as well as “premorbid” emotional symptoms subscale of the PCSC, and “premorbid” somatic symptoms subscale of the PCSC. Log10 transforms were applied to the state anxiety measure, length of time since injury, as well as the severity of ADIS-IV total and anxiety diagnoses. Following the example of Thornton et. al (2008), total number of concussions reported, total number of grades two and three concussions reported, and the continuous variable of length of PTA for the most severe event were all subjected to rank order transforms. Finally, the total score for current health difficulties was normalised by assigning a value representing the second highest score plus one to one case which was identified as an outlier. Following these transformations each variable was assessed to be normally distributed. Linearity was confirmed through visual inspection of bivariate scatter plots.

In consideration of planned multivariate analyses, the assumptions of homoscedasticity, sphericity and homogeneity of the variance-covariance matrices were all met for each analysis as verified through the inspection of Box’s M, Levene’s statistic and the calculation of the Fmax statistic (Tabachnick and Fidell, 2007; pg. 86).

For analyses regarding the association between variables, one-way Pearson correlations were performed, as the direction of association was expressly hypothesized for each case. Variables under consideration for the regression analyses were also subjected to preliminary correlations using Pearson’s r in order to examine the intercorrelations between variables as there was a need to reduce the number of variables included in the model and to minimise multicollinearity. Conservative guidelines were adopted for the assessment of multicollinearity, with tolerance values less than .2 and variance inflation factors of greater than 2.5 taken as indicators of significant issues with multicollinearity. In consideration of power, according to Green (1991), a sample size of fifty six subjects is sufficient for detecting medium size effects of up to four independent variables in a sequential regression model predicting a single predictor. As such, there was a need to limit the amount of independent variables in the model. As there was a need to limit the amount of predictors entered into the regression models in order to ensure adequate power, only variables with significant correlations to ratings of social and occupational impairment were considered for inclusion in the final analyses. Further, in light of the observed pattern of intercorrelation among variables,

and as the main hypotheses concerned the relative importance of susceptibility to catastrophic ideation, as opposed to emotional distress, it was determined theoretically acceptable to combine measures. Health Anxiety and Anxiety Sensitivity were significantly correlate with each other $r(61) = .53, p < .01$. State anxiety and Depression were also highly correlated; $r(61) = .45, p < .01$. Thus, Health Anxiety as measured by the IAS and Anxiety Sensitivity as measured by the ASI were combined into one variable indexing catastrophic ideation, and state anxiety as measured by the STAI and depression as measured by the Zung were combined into another variable indexing the emotional disposition of the respondent. Combined variables represent the standardised sum of standard scores for the constructs that were combined. The final variables deemed suitable for analysis in this manner were the age of the participant, sex of the participant, combined catastrophic ideation, and combined current emotional distress.

Multivariate outliers and normality were assessed through inspection of standard normality plots of the standardized residuals, as well as inspection of Mahalanobis distances, Cook's D, and Leverage values for each variable in the model. Effect sizes were calculated for each result and the strength of each effect size is reported according to the guideline provided by Cohen (Cohen, 1988).

As mentioned above, analyses considering variable importance in the determination of ratings of social or occupational impairment were performed with the entire, ungrouped, data set. Risk factors and correlates, including the combined variables specified above were compared with regard to their association with the continuous, non-dichotomised impairment ratings. In order to do so, the procedure outlined by Nathans, Oswald and Nimon (2012) was employed. Specifically, the direct effects, total effects and partial effects of each variable as a contributor to the model were assessed using a variety of techniques, in order to obtain a clear picture of variable importance from multiple perspectives. Thus, in addition to traditional measures such as zero order correlations, beta weights and changes in the overall R-square due to insertion of a variable at a particular step, further procedures were employed. These measures included analysis of the Product Measure (Pratt, 1987), which enables rank orderings of variable importance based on partitioning of the regression effect. A benefit of the product measure is that it is representative of not only the variable's relationship with the independent variable in isolation from other variables, but also of the relationship

between the variable and the outcome measure while accounting for the contributions of all the other variables. The product measure thus partitions the overall regression effect, such that the sum of the product measures for each variable adds up to the total R square, allowing for a rank ordering of variable importance. Relative Weight Analysis (Fabbris, 1980; Genizi, 1993; Johnson, 2000) was also conducted. Relative Weight Analysis allows for the computation of the percentage of predictable variance accounted for by the variable in presence of all other variables. Thus, RWI statistics represent the importance of the predictor as a percentage of total variance explained in the model, when all other variables are present in the model. Also calculated were structure coefficients, which are useful in not only representing the amount of variance than an independent variable shares with the variance from predicted y scores, but also allows for a clear assessment of suppression effects. The structure coefficient is sensitive to discovering if a variable's contribution to the regression equation was "distorted" or not given precedence during the beta weight calculation process because variance that it shares with another variable was assigned to a different beta weight, and not to its own. In addition, a commonality analysis was conducted (Mayeske et al., 1969; Mood, 1969, 1971; Newton and Spurrell, 1967; Onwuegbuzie and Daniel, 2003) in order to quantify the amount of unique variance that each variable contributes the regression equation that is not shared by other variables (Zientek and Thompson, 2006). There are two types of commonality coefficients, labelled Unique Effects and Common Effects. Unique Effects reflect how much variance an independent variable contributes to a regression equation that is not shared with other independent variables. Common Effects, however, provide information about the extent to which independent variables "overlap" in their ability to predict variance in the dependent variable. A unique property of commonality analysis is that the statistics generated allow for an investigation of whether a variable contributes more to a regression equation when operating in combination with other variables, or independently from them. Finally, a Dominance Analysis was also performed (Azen and Budescu, 2003) in order to determine variable importance based upon comparisons of the unique variance contributions of all pairs of predictors to regression equations involving all possible subsets of predictors. Dominance weights also allow for the rank ordering of an individual variable's contribution to the overall regression effect, and can be useful in determining variable importance by examining these weights across all possible subsets of independent variables. Thus, if a variable is

said to exhibit “General Dominance” over other variables, this means that the variable contributes the most variance to the regression equation, regardless of the order in which variables are entered into the equation. General Dominance represents the average difference in fit between all subset models of equal size that include the variable and those that do not. Further, a variable is said to express “Complete Dominance” over another variable if that variable’s contribution to the regression equation is always greater than the variable it is compared with, across all possible regression subsets. This method of considering variable importance through multiple different “lenses” allows for a deep understanding of how the risk factors and correlates under consideration contribute both unique and shared variance to the prediction of the independent variable, and provides a clear path to assessing variable importance in the presence of correlated predictors (Nathans, Oswald and Nimon, 2012).

Chapter 3.

Results

Test of Misattribution Hypothesis

Full Sample Analysis

In order to test the hypothesis that individuals with chronic post-concussion complaints would endorse fewer symptoms as being present prior to injury than those who had recovered from their injuries, a 2x2 repeated measures MANOVA was conducted. Post-concussion symptom report type (pre- injury¹⁵ versus current) served as the within subjects variable, and recovery status (chronic versus recovered) was the between subjects variable. Results from the MANOVA indicated a significant main effect of recovery status, according to Pillai's trace (.388), $F(4, 55) = 8.73$, $p < .01$; Partial eta squared = .388. The main effect of report type was also significant, as illustrated by Pillai's trace (.850), $F(4, 55) = 77.83$, $p < .01$; Partial Eta Squared = .850. Further, the multivariate interaction was also significant, Pillai's trace (.444), $F(4, 55) = 10.98$, $p < .01$; Partial Eta Squared = .444. Table 5 on the following page presents the results of both the univariate repeated measures effects, as well as follow-up one way ANOVA's conducted separately for retrospective and current symptom reports, in order to deconstruct the multivariate interaction.

¹⁵ Throughout this text, retrospective ratings of symptoms experienced before the experience of any injury are labelled as "pre-injury" ratings. As outlined in the methods above, this does not mean that individuals gave ratings before they were injured, but rather that these ratings were given at the same time as current ratings, but participants were asked to reflect on their level of symptoms prior to injury.

Table 5. Results of Univariate Repeated Measures ANOVA (Full Sample)

Effect	Measure	F	df	sig.	Partial Eta Squared
Report Type	Cognitive	144.53	1.00	0.00	0.71
	Emotional	257.50	1.00	0.00	0.82
	Somatic	170.47	1.00	0.00	0.75
	Total	171.61	1.00	0.00	0.75
Recovery Status	Cognitive	12.66	1.00	0.00	0.18
	Emotional	35.35	1.00	0.00	0.38
	Somatic	11.05	1.00	0.00	0.16
	Total	20.38	1.00	0.00	0.26
Recovery Status by Report Type	Cognitive	34.40	1.00	0.00	0.37
	Emotional	36.65	1.00	0.00	0.39
	Somatic	25.53	1.00	0.00	0.31
	Total	38.67	1.00	0.00	0.40

One Way Anova Between Group Effects (Full Sample)

Report Type	Measure	F	df	sig.	Partial Eta Squared
Current Symptom Report	Cognitive	25.34	1.00	0.00	0.30
	Emotional	39.58	1.00	0.00	0.40
	Somatic	18.75	1.00	0.00	0.24
	Total	35.79	1.00	0.00	0.38
Pre-Injury Symptom Report	Cognitive	1.04	1.00	0.31	0.02
	Emotional	3.00	1.00	0.09	0.05
	Somatic	0.09	1.00	0.77	0.00
	Total	0.03	1.00	0.87	0.00

As can be seen in Table 5 above and Figures 1 and 2 on the following pages, no significant differences were evident across groups on any of the pre-injury symptom report measures, while the chronically symptomatic group endorsed significantly more current symptoms than the recovered group.

Figure 1. Full Sample Pre-injury Symptom Reporting by Recovery Status

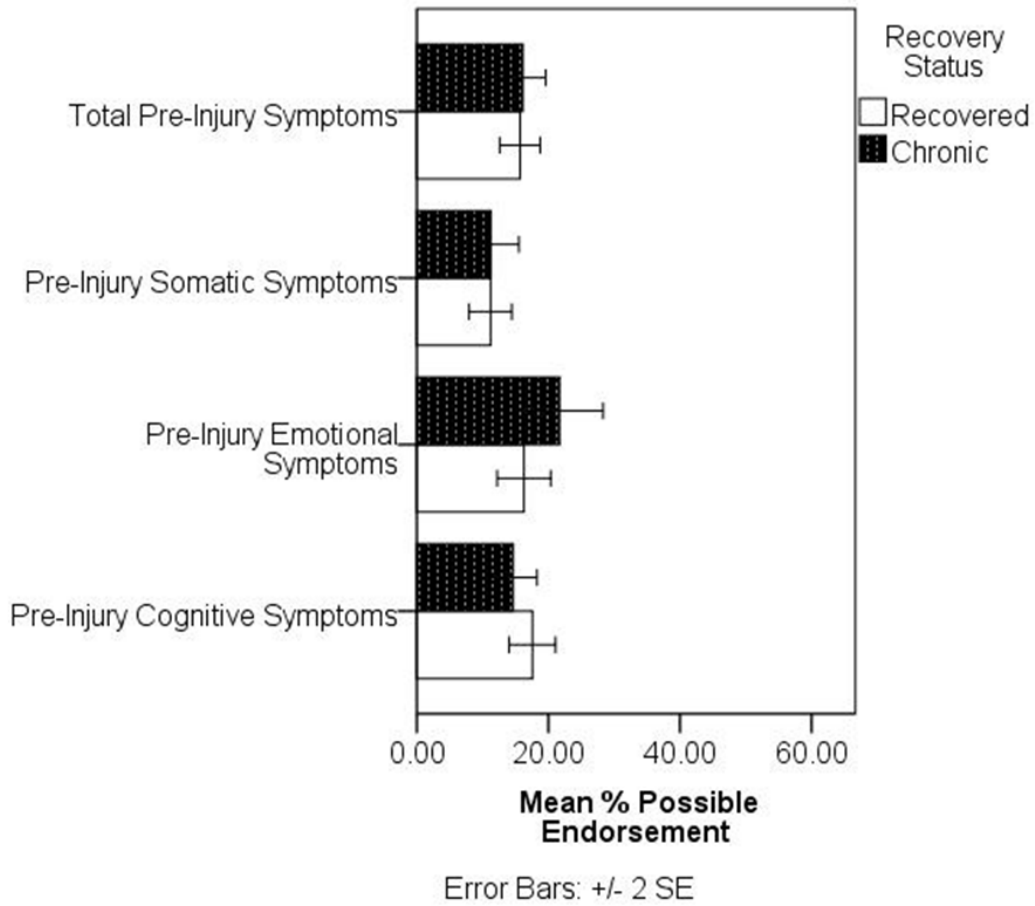
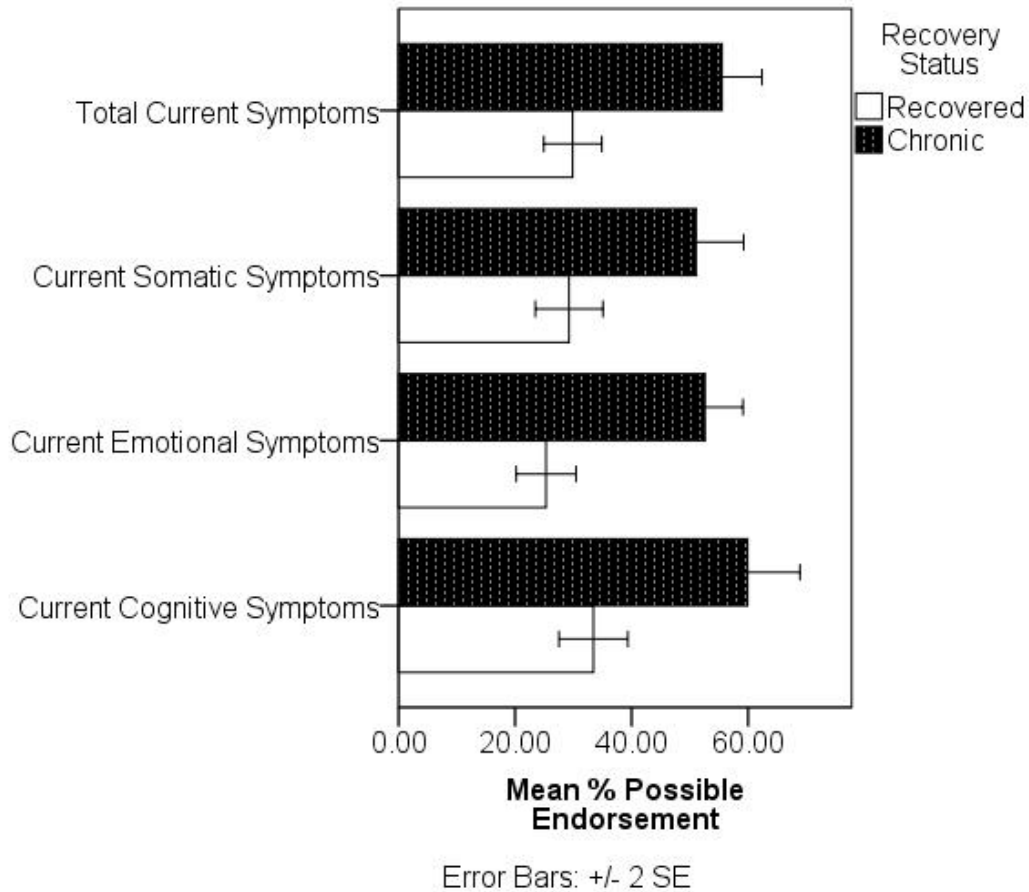


Figure 2. Full Sample Current Symptom Reporting by Recovery Status



Age Matched Sample Analysis

Similar results were obtained in the age matched analysis. Results from the MANOVA performed on the age matched sample also indicated a significant main effect of recovery status, according to Pillai's trace (.404), $F(4, 34) = 5.76$, $p < .01$; Partial eta squared = .404. The main effect of report type was also significant in the age matched sample, as illustrated by Pillai's trace (.892), $F(4, 34) = 70.53$, $p < .01$; Partial Eta Squared = .892. Again, the multivariate interaction was also significant, Pillai's trace (.424), $F(4, 34) = 6.26$, $p < .01$; Partial Eta Squared = .424. Table 6 presents the results of both the univariate repeated measures effects, as well as follow-up one way ANOVA's conducted separately for retrospective and current symptom reports, in order to deconstruct the multivariate interaction for the age matched sample.

Table 6. Results of Univariate Repeated Measures ANOVA (Age Matched Sample)

Effect	Measure	F	df	sig.	Partial Eta Squared
Report Type	Cognitive	102.99	1.00	0.00	0.74
	Emotional	225.73	1.00	0.00	0.86
	Somatic	149.23	1.00	0.00	0.80
	Total	127.40	1.00	0.00	0.78
Recovery Status	Cognitive	10.85	1.00	0.00	0.23
	Emotional	22.83	1.00	0.00	0.38
	Somatic	3.53	1.00	0.07	0.09
	Total	12.10	1.00	0.00	0.25
Recovery Status by Report Type	Cognitive	19.73	1.00	0.00	0.35
	Emotional	23.48	1.00	0.00	0.39
	Somatic	12.13	1.00	0.00	0.25
	Total	22.45	1.00	0.00	0.38
One Way Anova Between Group Effects (Age Matched Sample)					
Report Type	Measure	F	df	sig.	Partial Eta Squared
Current Symptom Report	Cognitive	16.69	1.00	0.00	0.31
	Emotional	25.53	1.00	0.00	0.40
	Somatic	7.11	1.00	0.01	0.16
	Total	20.92	1.00	0.00	0.36
Pre-Injury Symptom Report	Cognitive	0.06	1.00	0.82	0.00
	Emotional	0.54	1.00	0.47	0.01
	Somatic	0.00	1.00	0.96	0.00
	Total	0.00	1.00	0.97	0.00

As can be seen in Table 6 above and Figures 3 and 4 below, the age matched sample results were mainly identical to those obtained in the full sample, except that somatic symptom endorsement was no longer significantly different between groups. With regard to the follow up one way analyses, however, the same pattern emerged as above. Again, no significant differences were evident across groups on any of the pre-

injury symptom report measures, while the chronically symptomatic group endorsed significantly more current symptoms than the recovered group.

Figure 3. Age Matched Sample Pre-injury Symptom Reporting by Recovery Status

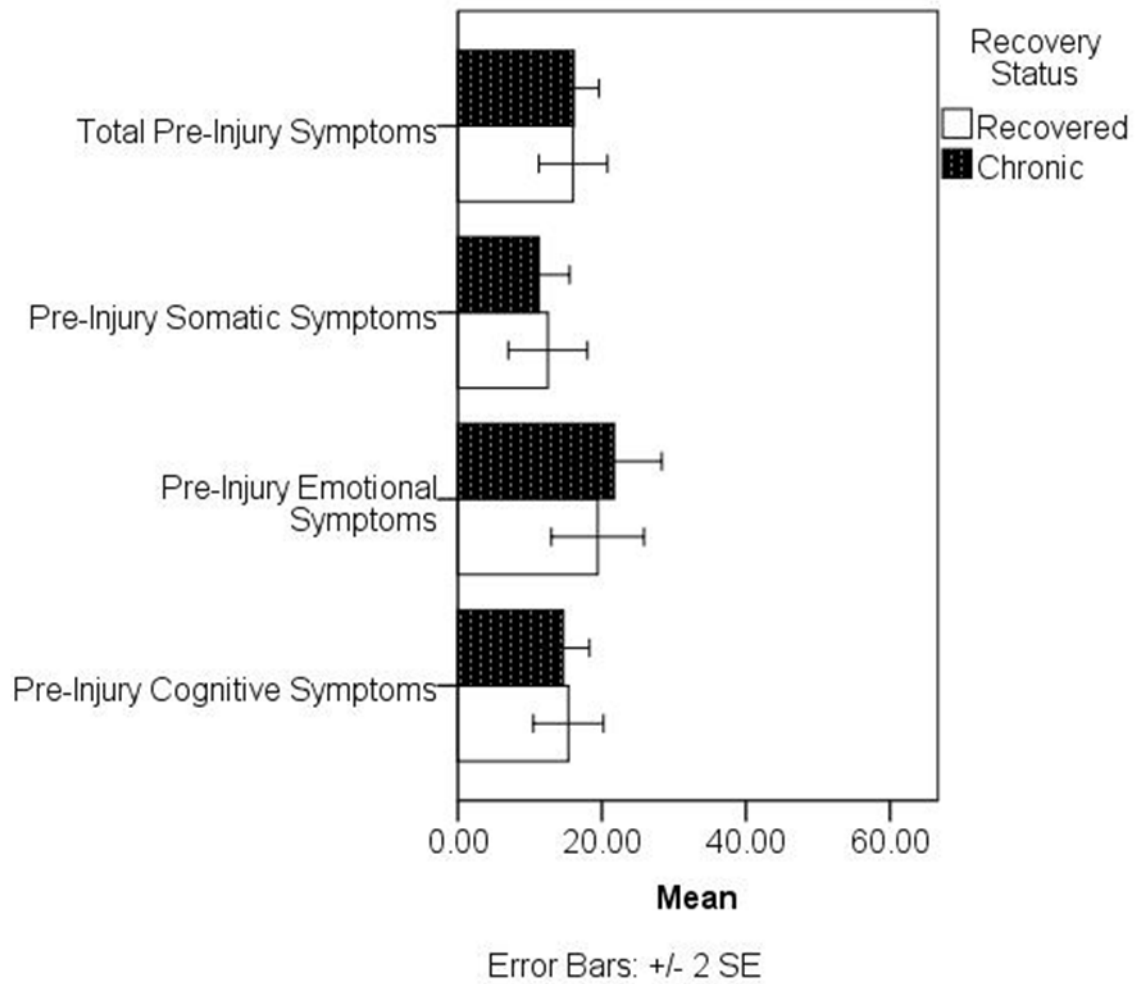
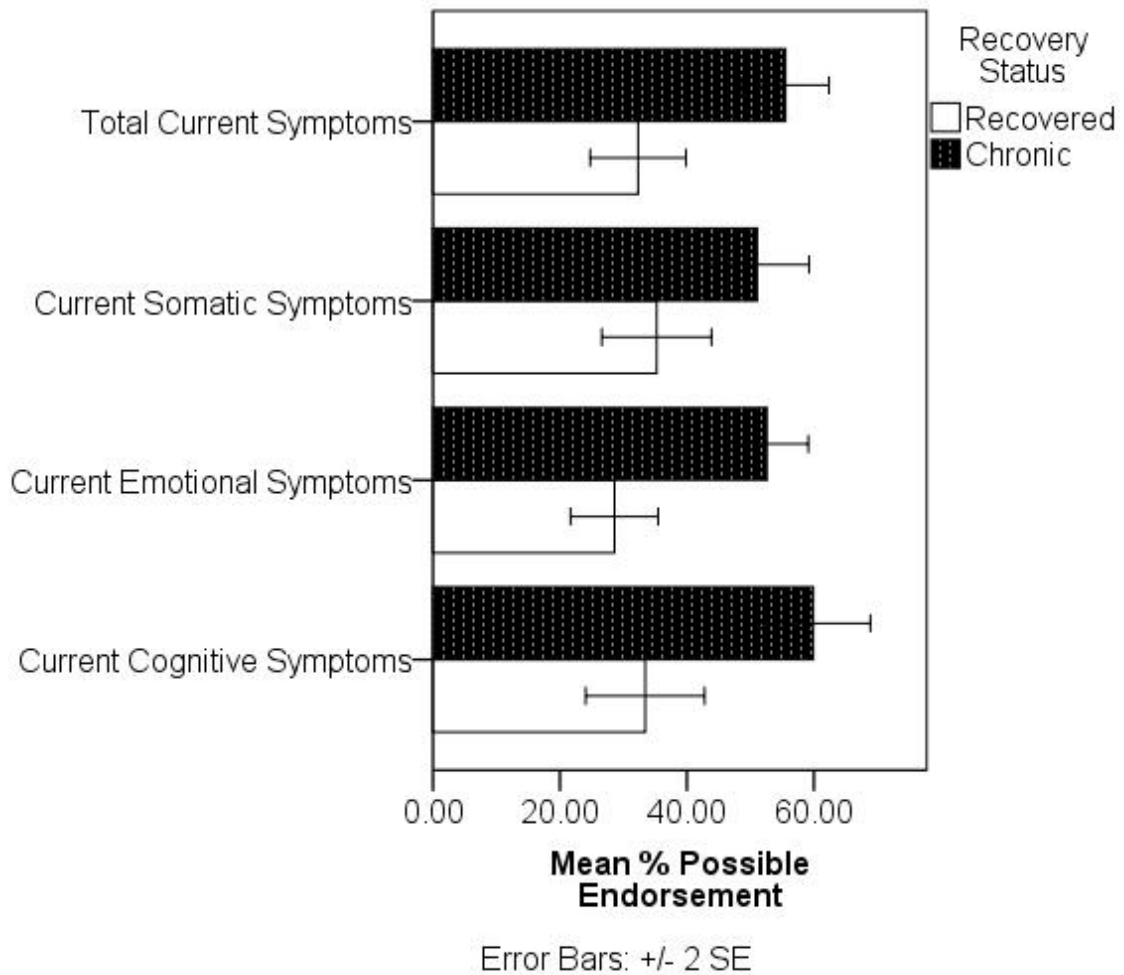


Figure 4. Age Matched Sample Current Symptom Reporting by Recovery Status



While both the recovered and chronically symptomatic groups endorsed more current than pre-injury symptoms, the chronically symptomatic group did not endorse fewer pre-injury symptoms than the recovered group.

Assessment of Risk Factors in the Prediction of Chronic Impairment

Test of Injury Severity Hypothesis

In contradiction to the hypothesis that ratings of impairment would increase as a function of injury severity, the severity of the CNS effect for the most severe injury ($r = .1, n = 61, p > .05$), was not significantly correlated with ratings of social and occupational impairment. Further, the correlation between the number of previous concussions experienced and ratings of social and occupational impairment was also not significant ($r = -.05, n = 61, p > .05$). Injury severity, defined as the degree of alteration in consciousness associated with the injury event, as well as history of exposure to similar injuries did not display a linear relationship with ratings of social and occupational impairment in this sample. To the extent that the length of alteration in consciousness or presence of loss of consciousness can be considered to index the subjective severity of the injury event itself, this outcome contradicts the predictions made by the Good Old Days model. Further, given the lack of reliable correlation observed between measures of injury severity and injury exposure to the ratings of social and occupational impairment specified as primary outcome measures, these variables were not included in the subsequent regression analyses for determining the importance of catastrophic ideation, as reported below.

Test of Relative Importance of Catastrophic Ideation

In order to test the hypothesis that catastrophic ideation is the single most important factor in the determination of impairment in social and occupational functioning due to injury, regression modelling was performed. Table 7 presents the results of regression modelling to determine the relative importance of age, sex, catastrophic ideation, and emotional state, to the prediction of ratings of social and occupational impairment due to injury. As illustrated in Table 7, vulnerability to catastrophic ideation did not explain significant variance in ratings of impairment, beyond the variance explained by participant variables such as age and sex, or the emotional state of the respondent. A comparison across all statistics presented in Table 7 indicates that the emotional state of the respondent was the strongest predictor of perceived impairment in social and

occupational functioning due to injury. Emotional state obtained the highest beta weight ($\beta = .37, p < .01$), demonstrating that it made the largest contribution to the regression equation, while holding all other predictor variables constant. The zero-order correlation of emotional state with perceived impairment ($r = .46$), when squared, illustrates that emotional state shared the largest amount (21%) of its variance with perceived impairment. The squared structure coefficient ($r_s^2 = .65$) demonstrated that emotional state explained the largest amount (65%) of the variance in y , the predicted values of impairment. Product measure results demonstrated that emotional state accounted for the largest partition of variance in impairment ratings ($Pratt = .171$, 52.1% of the regression effect), when multiplying the beta weight ($\beta = .37$) by the zero-order correlation ($r = .462$). The results of the relative weight analysis also support that emotional state explained the largest portion of the overall regression effect ($RWI = .127$, 38.7%) when partitioning that effect based upon a creation of variables' uncorrelated or independent counterparts.

Table 7. Hierarchical Regression Analysis predicting ratings of perceived impairment

	B	SE	β	p	R ²	ΔR^2
Step 1: Participant Variables:					0.18	.184**
Age	0.10	0.04	0.28	0.02		
Sex	2.79	1.22	0.28	0.03		
Step 2: Emotional State	1.90	0.55	0.39	0.00	0.33	0.144**
Step 3: Catastrophic Ideation	0.13	0.79	0.03	0.87	0.33	0.00

* $p < .05$; ** $p < .01$

Note. For each step we report only statistical details for the variable(s) entered on that particular step. Nonetheless, the statistics reported for each step are adjusted for the contribution of the other variables in the model.

Summary of Statistics Determining Independent Variable Contributions to Regression Effects

Variable	β	r_s	r_s^2	r	Pratt	Unique	Common	GDW	RWI	RWI%
Age	0.190	0.576	0.332	0.330	0.063	0.034	0.075	0.063	0.065	19.817
Sex	0.260	0.571	0.326	0.327	0.085	0.063	0.044	0.081	0.083	25.305
Emotional State	0.370	0.806	0.650	0.462	0.171	0.063	0.150	0.130	0.127	38.720
Catastrophic Ideation	0.026	0.611	0.373	0.350	0.009	0.000	0.122	0.054	0.053	16.159

Dominance analysis results (See Table 8) demonstrated the complete dominance of emotional state over age and sex, whereas the dominance of emotional state over catastrophic ideation was conditional on the values of sex. The general dominance pattern can be determined by looking across each row of the dominance weight table, under the heading “Additional Contribution of:” and noting which values are larger. As is evidenced in Table 8, each of the numbers listed in the column labelled “Additional Contribution of: Emotional State” is larger than the numbers in the columns representing the contribution of each of the other predictor variables, across all possible subsets, apart from the contribution of sex in one subset, when the values are equal. As such, it is clear that the prediction of ratings of impairment is optimised when the emotional state of the respondent is included in the regression equation. When these dominance values are averaged into an omnibus index of variable importance, as illustrated by the general dominance weights (*GDW*) listed in Table 7, the general overall dominance of emotional state is highlighted (*GDW* = .130). This statistic indicates that the emotional status of the respondent contributed the largest average contribution to the regression equation across all possible subsets. Because the *GDW* partitions the total *R* square (the numbers listed under the *GDW* column in Table 7 sum to the total *R* square), it is possible to rank the importance of the variables in the determination of values of the dependent variable. The *GDW* thus suggests that a rank order of variable importance would start with emotional state, followed by sex of the respondent, which is in turn closely followed by age, and that catastrophic ideation emerges as the least important factor in terms of optimising the regression equation, regardless of the order that variables are entered into the model, or which variables are entered together. Notably, the general dominance weight for emotional state was roughly equivalent to the relative weight (*RWI* = .127), indicating that its contribution to the regression effect assessed in terms of averages of unique variance contributions to all possible subsets or through the creation of uncorrelated counterpart variables was largely the same.

Conversely, and most pertinent to the predictions made by the Expectation as Etiology and Good Old Days models, the results of the regression analyses suggest that vulnerability to catastrophic ideation was in fact the poorest predictor of impairment ratings. Again, as seen in Table 7, catastrophic ideation had the lowest beta weight (β = .026) indicating that catastrophic ideation played a relatively minor role in the regression

effect while holding all other variables constant. The squared structure coefficient ($r_s^2 = .37$) for catastrophic ideation, however, was somewhat higher than expectations based upon the value of its beta weight, as was the zero-order correlation ($r = .35$) which suggested that catastrophic ideation explained twelve percent of the variance in the overall dependent variable. When partitioning the overall R^2 based on multiplying the beta weight by the zero-order correlation, however, product measure ($Pratt = .009$) results reflected that catastrophic ideation contributed very little variance to the regression effect. The relative weight for catastrophic ideation ($RWI = .053$) also demonstrates that catastrophic ideation contributed relatively little variance to the regression effect, when partitioning it based upon variables' uncorrelated counterparts.

The observation that catastrophic ideation contributed very little unique variance to the overall effect, despite being reasonably correlated with impairment ratings suggests that its variance overlapped more with one or more of the other predictors, than it did with the dependent variable. This is verified by inspecting the pattern of commonality coefficients presented in Table 8. As is evident in Table 8, under the heading "Commonality Coefficients" catastrophic ideation contributed very little unique variance (.09%) to the regression effect, whereas the common effects for catastrophic ideation and emotional state (24.5%) reflect that these variables contributed significant shared variance to the overall effect. Indeed, inspection of the unique and common effect columns in Table 7, under the headings "Unique" and "Common" illustrate how emotional state contributed both more unique and shared variance to the overall effect, whereas catastrophic ideation contributed essentially no unique variance whatsoever.

These results suggest that vulnerability to catastrophic ideation is not a primary predictor of ratings of social and occupational impairment due to injury, in the post-acute recovery period. These findings strongly contradict the hypothesis that chronic impairment is a product of hypochondriacal tendencies, or catastrophic misinterpretation of symptoms. In these results, the emotional state of the respondent emerged as the single most powerful predictor of self-reported ratings of social or occupational impairment

Table 8. Complete Dominance Weights and Commonality Coefficients

Variable(s)	Model <i>R</i> ²	Additional Contribution of:			
		Age	Sex	Emotional state	Catastrophic Ideation
Subset Containing No Predictors		0.109	0.107	0.213	0.122
Age	0.109		0.075	0.156	0.083
Sex	0.107	0.077		0.186	0.111
Emotional State	0.213	0.052	0.080		0.000
Catastrophic Ideation	0.122	0.070	0.096	0.092	
Age and Sex	0.184			0.144	0.081
Age and Emotional state	0.265		0.063		0.000
Age and Catastrophic Ideation	0.192		0.073	0.073	
Sex and Emotional State	0.293	0.035			0.001
Sex and Catastrophic Ideation	0.218	0.047		0.076	
Emotional State and Catastrophic Ideation	0.214	0.051	0.080		
Age, Sex and Emotional State	0.328				0.000
Age, Sex and Catastrophic Ideation	0.265			0.063	
Age, Emotional State and Catastrophic Ideation	0.265		0.063		
Sex, Emotional State and Catastrophic Ideation	0.294	0.034			
All predictors	0.328				
Commonality Coefficients					
Effect				Coefficient	Percent
Unique to Age				0.034	10.390
Unique to Sex				0.063	19.190
Unique to Emotional State				0.063	19.310
Unique to Catastrophic Ideation				0.000	0.090
Common to Age and Sex				0.017	5.270
Common to Age and Emotional State				0.012	3.780
Common to Sex and Emotional State				0.010	2.920
Common to Age and Catastrophic Ideation				0.000	0.140
Common to Sex and Catastrophic Ideation				0.000	-0.080
Common to Emotional State and Catastrophic Ideation				0.081	24.540
Common to Age, Sex and Emotional State				0.006	1.810
Common to Age, Sex and Catastrophic Ideation				0.000	-0.050
Common to Age, Emotional State and Catastrophic Ideation				0.030	9.170
Common to Sex, Emotional State and Catastrophic Ideation				0.003	0.850
Common to all predictors				0.009	2.660
Total				0.328	100

*Negative values represent suppression effects

Emotional State vs. Somatic and Cognitive Symptoms

Given the primacy of emotional state in the prediction of ratings of impairment in social and occupational impairment noted above, it was deemed important to further investigate the extent to which these symptoms were predictive, as compared with other types of post-concussion symptoms. In order to further investigate the role of current emotional state in the prediction of impairment in social and occupational functioning, in relation to the experience of other symptoms, such as cognitive and somatic symptoms, a series of multiple regression analyses were performed. In these analyses, the relative contributions of age, sex, emotional symptoms, somatic symptoms and cognitive symptoms to the prediction of perceived impairment were assessed. Table 9 presents the results of these analyses.

The results from the hierarchical regression analysis illustrate that emotional symptoms explained a significant amount of variance in perceived impairment, beyond that accounted for by all of the other predictors. A comparison across all of the statistics presented in Tables 9 and 10 indicates that emotional symptoms were the strongest predictor of impairment ratings. As seen in the “Summary of Statistics Determining Independent Variable Contributions to Regression Effects” below, emotional symptom report obtained the largest beta weight ($\beta = .427, p < .05$), demonstrating that it made the largest independent contribution to the regression equation, while holding all other variables constant. The zero-order correlation of emotional symptoms with impairment ($r = .679$), when squared, illustrates that emotional symptoms shared the largest amount of (46%) of its variance with impairment in social and occupational functioning.

Table 9. Hierarchical Regression Analysis predicting ratings of perceived impairment

	<i>B</i>	<i>SE</i>	β	<i>p</i>	<i>R</i> ²	ΔR^2
Step 1: Participant Variables					0.184	.184**
Age	0.103	0.044	0.282	0.024		
Sex	2.790	1.220	0.278	0.026		
Step 2: Somatic	0.174	0.046	0.483	0.000	0.352	.169**
Step 3: Cognitive	0.084	0.023	0.492	0.001	0.479	.127**
Step 4: Emotional	0.116	0.044	0.428	0.012	0.538	.059*

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

Note. For each step we report only statistical details for the variable(s) entered on that particular step. Nonetheless, the statistics reported for each step are adjusted for the contribution of the other variables in the model.

Summary of Statistics Determining Independent Variable Contributions to Regression Effects

Variable	β	<i>r</i> _s	<i>r</i> _s ²	<i>r</i>	Pratt	Unique	Common	GDW	RWI	RWI%
Age	0.032	0.450	0.203	0.330	0.011	0.001	0.108	0.028	0.029	5.390
Sex	0.213	0.446	0.199	0.227	0.048	0.041	0.066	0.056	0.060	11.152
Somatic	0.005	0.775	0.600	0.568	0.003	0.000	0.323	0.098	0.092	17.100
Cognitive	0.257	0.873	0.762	0.640	0.164	0.023	0.387	0.158	0.160	29.740
Emotional	0.427	0.926	0.857	0.679	0.290	0.058	0.403	0.199	0.196	36.431

The squared structure coefficient ($r_s^2 = .86$) demonstrated that emotional symptoms explained the largest amount (86%) of the variance in *y*, the predicted values of impairment ratings. Product measure results demonstrated that emotional symptoms accounted for the largest partition of the variance in perceived impairment (*Pratt* = .29, 54% of the regression effect) obtained by multiplying the beta weight by the zero-order correlation. Relative weight analysis also indicated that emotional symptoms explained a large portion of the overall regression effect (*RWI* = .196, 36%) when partitioning that effect based upon the creation of variables' uncorrelated or independent counterparts. Dominance analysis results (see Table 10) demonstrated the complete dominance of emotional symptoms over each other predictor, as it contributed more unique variance in the regression effect than each of the other variables across all of the multiple regression sub-models which that include this variable. This complete dominance can be determined by noting how each value for "The additional contribution of: emotional

symptoms” is larger than the values for each other variable, across the rows in Table 10. Emotional symptom report also displayed general dominance (Table 9; $GDW = .199$) over each of the other variables. Cognitive symptoms clearly emerged as the second strongest direct predictor of perceived impairment. In terms of the information presented in Table 9, inspection of the beta weight for cognitive symptoms ($\beta = .257, p < .05$), indicates that it made the second largest contribution to the regression equation when holding all other predictors constant. The zero order correlation ($r = .64$) between cognitive symptoms and impairment ratings was also the second largest in the model, which, when squared, demonstrated that cognitive symptoms shared the second largest amount (41%) of its variance with impairment. The squared structure coefficient ($r_s^2 = .76$) illustrated that cognitive symptoms shared the second largest amount (76%) of variance with y Product measure results ($Pratt = .16$) indicate that cognitive symptoms accounted for 30.5% of the total R^2 when it was partitioned based upon the product of the beta weight and the zero-order correlation. The relative weight of cognitive symptoms ($RWI = .16$) was identical to its product measure ($Pratt = .16$), demonstrating that cognitive symptoms accounted for 30% of the regression effect when partitioning it based upon creation of the independent variables’ uncorrelated counterparts. Thus, cognitive symptom accounted for the second largest amount of variance in the regression equation across multiple measures.

Complete dominance analysis results (see Table 10) supported the fact that cognitive symptoms were completely dominant over somatic symptoms, as scrutiny of Table 10 illustrates that each of the numbers in the column under the heading “Additional Contribution of: Cognitive Symptoms” are larger in magnitude than those under the corresponding column for Somatic Symptoms. Further, the general dominance weight also supports the fact that cognitive symptoms were dominant over somatic symptoms (Table nine; $GDW = .158$).

Once again, the general dominance weight of cognitive symptoms was nearly identical to its obtained relative weight, reflecting that cognitive symptoms explained the second largest, and a substantial 29.7% of the variance in R^2 when the variance is portioned according to this method.

Although other statistics clearly showed how emotional symptoms were the strongest direct contributor to the model, followed by cognitive symptoms, they did not show exactly how those variables contributed unique and shared variance to the regression equation. As previously stated, cognitive and emotional symptoms are often correlated and as such it is important to consider the degree of “overlap” shared by these predictors. Thus, commonality coefficients were consulted to obtain this information (see Table 10 – “Commonality Coefficients”). In consideration of the commonality analysis results, it was clear that emotional symptoms contributed more unique variance (10.9%) to the regression equation than did cognitive (4.3%) or somatic (0%) symptoms. The common effects for cognitive and emotional symptoms (19.3%, see “Common to Cognitive and Emotional) and cognitive, somatic, and emotional symptoms combined (29.1%, see “Common to Somatic Cognitive and Emotional) reflect the fact that these variables also contributed substantial shared variance to the regression effect. These results are indicative of the overlap between these symptom domains. Indeed, analysis of the unique and common effects presented in Table 9, under “Unique” and “Common” effects in the “Summary of Statistics Determining Independent Variable Contributions to Regression Effects” portion of the table leads to the conclusion that each of the variables under consideration contributed more shared than unique variance to the overall model. The Commonality coefficient findings presented in Table 10 also suggest that sex acted as a suppressor variable for cognitive and emotional symptom report.

The suppression effect due to sex is indicated by the negative values in commonality coefficients including sex and either, as well as both cognitive and emotional symptoms. As such, including the sex of the participant in the model allowed the influence of both emotional and cognitive symptoms on the prediction of impairment to be improved. Although the overall suppression was small (1.33%), partitioning the influence of sex on cognitive and emotional symptom report from the overall effect allowed emotional and cognitive symptoms to be better predictors, which is consistent with research suggesting that sex plays an important role in symptom reporting in general.

Table 10. Complete Dominance Weights and Commonality Coefficients

Variable(s)	Model <i>R</i> ²	Additional Contribution of:				
		Age	Sex	Somatic	Cognitive	Emotional
Subset Containing No Predictors		0.109	0.107	0.322	0.410	0.461
Age	0.109		0.075	0.218	0.310	0.359
Sex	0.322	0.077		0.241	0.358	0.403
Somatic	0.410	0.005	0.026		0.119	0.152
Cognitive	0.461	0.009	0.055	0.031		0.081
Emotional	0.327	0.007	0.049	0.013	0.030	
Age and Sex	0.419			0.168	0.285	0.329
Age and Somatic	0.468		0.025		0.116	0.150
Age and Cognitive	0.441		0.050	0.024		0.075
Age and Emotional	0.474		0.045	0.009	0.026	
Sex and Somatic	0.491	0.004			0.130	0.165
Sex and Cognitive	0.443	0.004		0.013		0.072
Sex and Emotional	0.477	0.003		0.003	0.027	
Somatic and Cognitive	0.494	0.002	0.037			0.055
Somatic and Emotional		0.003	0.039		0.022	
Cognitive and Emotional		0.003	0.046	0.005		
Age, Sex and Somatic					0.127	0.163
Age, Sex and Cognitive				0.010		0.069
Age, Sex and Emotional				0.002	0.025	
Age, Somatic and Cognitive			0.036			0.054
Age, Somatic and Emotional			0.038		0.020	
Age, Cognitive and Emotional			0.044	0.003		
Sex, Somatic and Cognitive		0.001				0.059
Sex, Somatic and Emotional		0.002			0.024	
Sex, Cognitive and Emotional		0.001		0.000		
Somatic, Cognitive and Emotional		0.001	0.041			
Age, Sex, Somatic and Cognitive						0.059
Age, Sex, Somatic and Emotional					0.023	
Age, Sex, Cognitive and Emotional				0.000		
Age, Somatic, Cognitive and Emotional			0.041			
Sex, Somatic, Cognitive and Emotional		0.001				
All predictors	0.538					

Commonality Coefficients

Effect	Coefficient	Percent
Unique to Age	0.001	0.150
Unique to Sex	0.041	7.580
Unique to Somatic	0.000	0.000
Unique to Cognitive	0.023	4.300
Unique to Emotional	0.058	10.860
Common to Age and Sex	0.001	0.100
Common to Age and Somatic	0.000	0.020
Common to Sex and Somatic	0.003	0.600
Common to Age and Cognitive	0.001	0.110
Common to Sex and Cognitive	-0.003	-0.480
Common to Somatic and Cognitive	0.002	0.340
Common to Age and Emotional	0.000	0.050
Common to Sex and Emotional	-0.005	-0.850
Common to Somatic and Emotional	0.010	1.930
Common to Cognitive and Emotional	0.104	19.270
Common to Age, Sex and Somatic	0.002	0.310
Common to Age, Sex, and Cognitive	0.000	0.010
Common to Age, Somatic and Cognitive	0.001	0.230
Common to Sex, Somatic and Cognitive	0.004	0.750
Common to Age, Sex and Emotional	0.000	0.000
Common to Age, Somatic and Emotional	0.003	0.600
Common to Sex, Somatic and Emotional	0.011	1.990
Common to Age, Cognitive and Emotional	0.002	0.420
Common to Sex, Cognitive and Emotional	-0.008	-1.530
Common to Somatic, Cognitive and Emotional	0.156	29.080
Common to Age, Sex, Somatic and Cognitive	0.001	0.240
Common to Age, Sex, Somatic and Emotional	0.003	0.480
Common to Age, Sex, Cognitive and Emotional	0.000	0.010
Common to Age, Somatic, Cognitive and Emotional	0.069	12.750
Common to Sex, Somatic, Cognitive and Emotional	0.032	5.910
Common to all predictors	0.026	4.770
Total	0.538	100

Overall, these findings support the assertion that emotional symptoms are the single most significant contributor to impairment for concussed individuals in the post-acute stage of recovery. Cognitive symptoms emerged as the second most important predictor, while somatic symptoms, sex and age were less potent predictors. These results are consistent with a view that when individuals with a history of concussion are

asked to make determinations about the degree of impairment in social and occupational functioning that they have experienced as a result of their injury, these individuals rely mainly on their current experience of emotional distress, and to a lesser extent, current cognitive difficulties. These results do not support the idea that catastrophic ideation plays a pivotal role in the perception of impairment in social or occupational functioning due to concussive injury.

Chapter 4.

Discussion

The current study was designed to test three important hypotheses that were generated by the Expectation as Etiology (Mittenberg, 1992), and Good Old Days (Gunstad and Suhr, 2004) models, with respect to the expression of chronic Post-concussional Disorder. These hypotheses included the hypothesis that chronic post-concussion symptoms are the result of misattribution of pre-injury symptoms to the effects of injury, the hypothesis that disordered recovery, as measured by ratings of impairment in social and occupational functioning would be positively correlated with injury severity, and the hypothesis that catastrophic ideation is a critical risk factor in the determination of chronic impairment following concussive injury. None of the hypotheses outlined above were supported by the results of the study. Individuals with chronic symptoms did not endorse fewer “pre-injury” symptoms than those who had recovered from their injuries, and measures of catastrophic ideation were not clearly associated with perceptions of impairment. These results call the viability of the Good Old Days and Expectation as Etiology models into question, with regard to their ability to predict the symptom report profiles and impairment ratings of individuals with chronic Post-concussional Disorder.

This dissertation has made two important contributions to the understanding of perceptions of chronic impairment following concussive injury. Perhaps foremost among these is the finding that catastrophic ideation, hypochondriacal ideation, and/or misattribution of symptoms are not well associated with self-rated impairment. Secondly, this dissertation clearly identifies post-injury emotional difficulty as the single most potent risk factor associated with chronic impairment. These contributions suggest that diagnostic systems and etiological models which postulate that catastrophic ideation is primary are questionable. Further, this research has implications for the development and administration of clinical treatment to individuals with chronic symptoms following

concussion, because it can help focus interventions on the emotional symptoms which are most associated with self-rated impairment.

The current study represents a first attempt to evaluate the “Expectation as Etiology” (Mittenberg, 1992) and “Good Old Days” (Gunstad and Suhr, 2004) models with individuals who are experiencing chronic symptoms and are not litigating. A review of the literature indicates that these models are not well understood, despite being widely cited as important in the explanation of post-concussion symptom report (Iverson et al, 2010, 2011; Garden, Sullivan and Lange, 2010; Ozen and Fernandes, 2011; Dean, O’Neil and Sterr, 2012; Heilbronner et al., 2009). The general acceptance of these models in the literature has likely been driven by the apparent strength and reproducibility of the “supranormalcy” of pre-injury symptom reporting in individuals with a history of concussion. Despite the apparent robustness of this effect, several methodological issues have been identified which call the applicability of this effect into question, with regard to the development and maintenance of chronic post-concussion symptoms. These issues include sampling factors such as the inclusion of nonsymptomatic or non-chronically symptomatic individuals in the study group, inappropriate selection of control subjects, and the inclusion of litigating samples. Further, previous research on the topic in general has suffered from design problems, such as circular predictive reasoning, wherein the predictors of outcome were confounded with the outcome itself. The current research was designed to address these issues, in order to evaluate the sufficiency of the “Good Old Days” and “Expectation as Etiology” models in explaining the impairment level of chronically symptomatic individuals. Further, and on a more general level, the current research was designed to extend these findings to an exploration of the importance of previously identified risk factors, in terms of their influence on ratings of impairment due to concussive injury. With these issues in mind, the current research explored chronic symptom report along three important axes; the applicability and usefulness of the misattribution theory, the reactivity of self-rated impairment in social or occupational functioning to injury severity, and the relative importance of catastrophic ideation in the prediction of self-rated perceptions of impairment.

Development of New Symptoms versus Misattribution of Pre-existing Symptoms

The results of the current study do not support the misattribution hypothesis, in that the “pre-injury” symptom report of individuals with chronic symptoms was not observed to be “supranormal” in comparison to an injured, but recovered control group. In review, Mittenberg (1992) proposed that post-concussion symptoms were not generated as a result of injury, but rather that the symptoms endorsed in the post-concussion period were really pre-existing symptoms that had been reattributed to the injury. Evidence for this theory came in the form of data suggesting that concussed individuals reported fewer pre-injury symptoms than nonconcussed individuals reported experiencing currently. This finding has been replicated recently by Iverson et al. (2010), and Lange et. al (2010), who presented data which indicated that individuals who are at risk for developing chronic symptoms endorsed significantly fewer symptoms than a non-injured control group, when asked to report on their experience of symptoms prior to injury. These findings would appear to contradict the results of the current study, although methodological differences between these studies may provide some explanation.

Iverson et. al (2010, Lange et. al, 2010) note that litigation and motivation may have had significant influences in the pattern of symptom reporting that they observed. Specifically, they present data which suggests that individuals, who are motivated to emphasize changes in functioning from the pre-injury to post-injury state, may be vulnerable to minimising the report of pre-injury symptoms. Thus, this may represent a pattern of behaviour that is due to litigation, as opposed to concussion per se. As the current research did not include any litigating individuals, the symptom report profiles obtained herein are not vulnerable to this potential confound. Further, it is noted that these studies did not sample from individuals in the chronic phase of recovery (Iverson et al., 2010; Lange et al., 2010). Indeed, the foundational research upon which the Good Old Days (Gunstad and Suhr, 2001, 2002, 2004) and Expectation as Etiology models (Mittenberg, 1992) were based did not sample from individuals with chronic symptoms either. As such, it is difficult to apply the findings reported in previous research to individuals with chronic impairment.

The data presented herein does not support the “Expectation as Etiology” (Mittenberg, 1992) hypothesis, in that premorbid symptom report was found to be unrelated to diagnostic status. It is worth noting that in the current study, “pre-injury” symptom report was not minimised in either the chronically symptomatic or recovered groups, and that both groups’ “pre-injury” symptom reporting hovered around twenty percent of the maximum score for each subscale, nor was current symptom report maximised in the chronic group. Both chronically symptomatic and recovered groups reported fewer “pre-injury” symptoms than current symptoms, which may be consistent with the hypothesis that the experience of concussion is sufficient to alter one’s view of past symptom levels, or that the experience of a concussion leads to the development of lingering symptoms regardless of whether or not the individual regards these symptoms as significantly impairing to their social or occupational functioning. However, other factors may also be at work, including a general response bias which may be unrelated to injury.

With regard to the hypothesis that the experience of chronic symptoms should be related to decreases in pre-injury symptom reports, the results of the current investigation are consistent with evidence from research in symptom reporting in other areas. Within the literature on symptom report and cognitive bias in general, previous research has consistently shown that the retrospective symptom reports of individuals who are experiencing distress are biased towards intensification of symptoms, when compared with current ratings (Csikszentmihalyi and Larson, 1987; Delespaul and deVries, 1987). This pattern, which suggests that current distress often leads to increased, rather than decreased reporting of past symptoms has been observed in various clinical populations, including eating disorders (Stein and Corte, 2003), panic disorder (deBeurs et al., 1992), obsessive compulsive disorder (Herman and Koran, 1998), borderline personality disorder (Ebner-Premier et al., 2006), and depression (Ben-Zeev et al., 2009, 2010). It has further been suggested that the difference between current and retrospective report of symptoms in general is due to the fact that current reporting can be based upon actual experience, but that retrospective report is largely influenced by beliefs about one’s health and identity as a healthy or ill individual (Houtveen et. al 2007).

Indeed, some have suggested that the experience of an adverse event or injury is not necessary in order to observe differences in retrospective versus current “post-

concussion” symptom reporting (Sullivan and Edmed, 2012; Panayiotou, Crowe and Jackson, 2011), and that the endorsement of fewer symptoms on retrospective reports may be due to a general recall bias. This is consistent with research which has suggested that healthy individuals exhibit a positive recall bias when recalling specific life events (Mitchell et al., 1997; Walker et al., 2003). This tendency to view the past through “rose coloured glasses” has been hypothesised to represent evidence of a “psychological immune system” that is protective against negative affectivity in healthy individuals (Gilbert et. al, 1998), but may be lacking for individuals who are experiencing emotional distress. A further implication of this is that individuals can be expected to report fewer symptoms in the past, in comparison to current symptom reports, regardless of the existence of injury or illness. This research suggests that the method of comparing retrospective or “pre-injury” symptom report of concussed individuals to the current symptom report of healthy individuals is misguided. Past research has demonstrated that current symptom reports are qualitatively different from retrospective reports, and are subject to biases in recall which are likely to influence results. Further, the use of a non-injured control group is problematic in that such a design does not allow for the investigation of factors related to recovery and chronic perceptions of impairment.

Panayiotou et al., (2011) present data suggesting that noninjured control subjects and noninjured subjects with a current psychiatric diagnosis both endorsed fewer PCS symptoms when asked to report on their experiences “6 to 12 months ago” than they did when asked to report current symptoms. Further, these authors present data that noninjured individuals who were asked to imagine that they had incurred a concussion actually reported more symptoms on the retrospective reporting condition than they reported currently (Panayiotou et. al, 2011). Thus, it would seem that the comparison of the retrospective symptom reports of concussed individuals and current symptom reports of noninjured controls is problematic. The current research avoids this issue by sampling from both recovered and chronically symptomatic individuals with similar histories of concussion, so that the main difference between groups is the fact that one group developed chronic symptoms, while the other did not.

Consistent with predictions of the “Good Old Days” model, the results presented herein indicate that the experience of concussion is associated with the report of more symptoms currently than prior to injury. Both the recovered individuals and those in the

chronically symptomatic group endorsed more current than “pre-injury” symptoms. This is interesting to note in that previous research into the topic has used healthy, non-injured control subjects, who have been presumed not to alter their report of symptoms over time. Given the suggestion that non-injured individuals might be expected to report fewer symptom retrospectively than currently, due to a general response bias, the methods utilised by previous researchers may have exaggerated the “supranormalcy” of pre-injury symptom reporting in their samples. As the difference between those who recovery normally and those who experience chronic symptoms is of more clinical significance than the difference between concussed and non-concussed individuals, the current research is uniquely positioned to test the validity of these models in the population of interest. Under the current research design, symptom chronicity was unrelated to ratings of pre-injury symptom levels, which calls the reattribution hypothesis into question. It should be noted that although the current research clearly suggests that current symptoms are not well characterised as reattributions of pre-existing symptoms, there are multiple pathways through which these symptoms may have been generated. Of specific relevance to the current research are theories surrounding the role of the injury itself, and the role of catastrophic ideation.

Reactivity to Injury Severity

With respect to the role of the injury, the current research found no association between the severity of injury, and ratings of impairment in concussed individuals in the post-acute phase of recovery. Again, it is worth repeating that injury severity in the current study was limited to injuries that would meet criteria for concussion. Certainly injury severity is an important determinant of outcome in moderate to severe brain injury. Further, it is important to note that the duration of alteration of consciousness and presence of loss of consciousness and/or history of injuries may be related to the development of symptoms acutely, and this study does not rule out the idea that injury parameters are important in the acute phase. What is apparent in these data is that self-rated impairment in social and occupational functioning in the post-acute phase of recovery is not associated with the severity of the injury. This finding is interesting in that it is somewhat inconsistent with the predictions of the Good Old Days model (Gunstad and Suhr, 2004) in the prediction of chronic symptoms. Gunstad and Suhr

(2004) posited that the expression of symptoms relied upon the influence of the “nocebo” effect, which they defined according to the definition made by Hahn (1999). Hahn (1999) defines the nocebo effect as the result of an interaction between the severity of the event, and a susceptibility to catastrophic beliefs about outcome. As such, it has been hypothesized that symptom report will vary as a function of injury severity (Gunstad and Suhr, 2002; Iverson, 2010). In the current research, injury severity was not associated with ratings of impairment due to injury. These findings weaken the argument that symptom generation occurs as a result of the nocebo effect. The lack of association between injury severity and chronic impairment observed in the current study is consistent with previous research which has also failed to find clear associations between measures of injury severity, and post-concussion symptom report (Meares et al., 2008; Ponsford et al., 2012). While injury severity may indeed be important for the expression of symptoms in the acute phase of recovery, the current research suggests that injury severity is no longer a determinant of outcome in the chronic phase. This result is consistent with previous theories which suggest that the effects of concussion on the functioning of the central nervous system predominate early in the natural history of recovery, but that psychological, motivational and emotional factors become more important as time goes on (e.g. Lishman, 1988). The nature of these factors is further discussed in the next section.

Vulnerability to Catastrophic Ideation versus Emotional State of the Respondent

With regard to the influence of psychological factors on the degree of impairment associated with injury, two themes have emerged in the literature. Some have suggested that chronic impairment may develop as a result of vulnerabilities to catastrophic ideation about the consequences of injury, and others have highlighted the influence of emotional factors. Both Mittenberg (1992), and Gunstad and Suhr (2002, 2004) posited models which suggested that vulnerabilities to catastrophic ideation were primary in the development of post-concussion symptoms. This sentiment has been mirrored in the very definition of Post-concussion Disorder proposed in the ICD-10, which suggests that symptoms are essentially hypochondriacal in nature (World Health Organization, 1992). One difficulty with previous research which has supported this view

is the fact that often circular reasoning is employed in support of this claim. Longitudinal studies have sought to measure beliefs about illness, as well as expectations of impairment, and then correlated these with outcome defined by meeting diagnostic criteria that is defined by these very same factors. The current study avoids this difficulty by allowing individuals to rate their level of impairment in social or occupational functioning, and then quantifying which factors are most related to those ratings. Further, previous research may have failed to distinguish between measures of state emotional disposition and measures of catastrophic ideation as a result of the correlation between these measures. Indeed, some have simply not included potentially collinear measures in their predictive models, perhaps due to difficulties with meeting the statistical requirements of the tests used. By carefully selecting and operationalizing measures based upon previous research, and by assessing variable importance using recent statistical techniques which are capable of partitioning effects when variables are somewhat correlated, the current research is able to speak to the ways in which risk factors overlap and work together in the prediction of perceived impairment, as well as the unique contributions made by each. The results presented herein do not support the hypothesis that chronic impairment due to injury is driven by catastrophic ideation. Indeed, in relation to the age, sex, and emotional disposition of the respondent, vulnerability to catastrophic ideation emerged as the least important predictor. These results suggest that individuals who are beyond the acute phase of recovery base their ratings of impairment most strongly on evaluations of their current emotional state, and that illness attitudes, hypochondriacal beliefs, and symptom misinterpretation are less important. Further, in this study, emotional symptoms such as depressed mood and anxiety appear to be driving ratings of impairment, above and beyond the current experience of any other symptom type, including cognitive and somatic symptoms.

Conclusion

It is important to consider these results in light of the method through which they were obtained. Participants were self-selected and as such may represent the most severe cases of Post-Concussional Disorder. This was not generally conceived as problematic to the research questions of interest, but may limit the generalizability of these findings to the population of concussed individuals. However, to the extent that the Post-

concussional Disorder group may have been motivated to seek treatment, this would theoretically suggest a predisposition to emphasize changes in symptoms from the pre to post injury state, rather than to minimise them. Further, none of the participants were involved in litigation or compensation seeking of any kind. Again, this was stipulated in the study design in order to clarify the pattern of post-concussion symptom report in the absence of motivation to highlight changes from pre to post injury functioning from sources other than the injury and the person themselves. However, it is noted that large numbers of those who experience concussions go on to be involved with legal claims or compensation claims, and as such these data may not reflect the symptom report profiles of such cases. Also, it may be that participants' ratings of their pre-injury state were influenced by misunderstanding of the time periods being referenced. For example, although individuals were instructed to give their ratings of symptoms before they ever experienced a concussion, some may have responded by giving ratings of their symptoms prior to their most recent concussion, or prior to their most severe concussion only. This possibility is considered unlikely, as all questionnaires were thoroughly explained and supervised by trained research assistants.

This study relied on self-report of symptoms and injury parameters, and as such results are somewhat dependent on the veracity of the participants' recollection of details of their lives and events that occurred to them in the past. Although participants generally endorsed high levels of confidence about their recall of events, and care was taken to review medical records where available, this study is correlational in nature and as such cannot speak directly to the influence of pre-existing factors and injury related variables. For example, although individuals reported on factors related to injury severity, such as length of PTA and LOC in minutes, the reliability of these reports is unknown. In order to address this issue, a threshold model was adopted for quantifying injury severity, which dichotomised severity into those with more than one hour of PTA or more than five minutes of LOC, as these types of events are likely more salient than injuries associated with relatively brief alterations in consciousness. This method was considered to an effective way of addressing issues pertaining to the reliability of recall of PTA and LOC. However, the results of the study are generally reflective of results obtained in longitudinal studies that have implicated emotional functioning as a strong predictor of outcome. As such, these results are thought to reflect corroboration of the idea that the

set of symptoms endorsed after experiencing a concussion are not well characterised as misattributions of previously occurring symptoms, or over-reactions to everyday sensations, and that chronic impairment is best predicted by emotional functioning.

Further, the current study did not utilise a “healthy” or non-injured control group, as the question of interest pertained to differences between those with persistent symptoms and those who had recovered from similar injuries. As such, the existence of a general response bias favouring idealisation of the past could not be further examined. It is possible that both the chronic and recovered groups’ ratings of symptoms prior to injury might be “supranormal” in comparison to a non-injured control. Future studies may wish to include a non-injured control group in a similar study, in order to quantify the extent to which a general recall bias might be at play, and if this bias is any different from that displayed by individuals with a history of concussion. What is clear from these results is that Post-concussional Disorder is not associated with decreases in “pre-injury” symptom report. Indeed, it is difficult to interpret the pattern of “pre-injury” symptom reporting in the current study without reference to a general response bias, as very few variables were associated with “pre-injury” ratings, and when associations were observed, they typically resulted in increases in “pre-injury” symptom report. Given that both the recovered and the chronically symptomatic groups endorsed fewer “pre-injury” than current symptoms, and the lack of differences between groups, future studies may wish to investigate if the experience of concussion, whether it results in long lasting symptoms or not, is sufficient to homogenize individuals beliefs about their overall health prior to the injury.

With further regard to sampling, it is noted that the sample size was somewhat smaller than would be optimal. Despite this, each analysis was determined to be adequately powered, based upon the relative size of the effects obtained, and the number of independent variables utilised. The current sample also provides a good representation of the different mechanisms of concussive injury reported in the literature. (Jennett and Frankowski, 1990; Bazarian et al. 2010; Bordignon and Arruda 2002; Iverson et al. 2000; Kashluba et al. 2008; Kraus et al. 2005; Lee et al. 2008; Lipton et al. 2009; Lundin et al. 2006; Meares et al. 2008; Thornhill et al. 2000). In this sample, twenty four percent of the concussions were the result of motor vehicle accidents, nineteen percent of the injuries were due to falls or domestic accidents, eighteen percent of the cases were due

to assaults, and thirty seven percent of the cases were the result of sporting injuries. Thus, the full range of injury mechanisms was represented.

It should also be noted that the current study is correlational in nature, and as such, a causal chain cannot be inferred from this data. It may be that the physiological experience of concussion leads to the development of emotional symptoms, or that the emotional difficulties arose independently of the concussive event. The current research suggests that participants perceived these emotional symptoms as being generated sometime after the concussion occurred, but this is based upon self-report, which is itself highly influenced by mood. As previously noted, structural and functional investigations of the sequelae of concussion bear strong resemblances to brain changes typically observed in primary mood disorder, and again it is difficult to determine if injury leads to a vulnerability to emotional dysfunction, or if the effects of emotional dysfunction are primary. In these data, the severity of the alteration in consciousness associated with the concussion was not directly related to impairment due to injury, while emotional difficulty was. Again, it is worth repeating that these ratings of impairment in social and occupational functioning were specifically made with reference to the influence of the concussion itself, and not broad ratings of impairment in general. Thus, in this sample, individuals consistently associated their current emotional difficulties with the influence of the injury itself, and suggested that it is these emotional symptoms that they perceived as being a result of injury, which led to the most impairment in social and occupational functioning. This suggests that the experience of emotional symptoms such as anxiety and depression is the most salient factor in distinguishing those who perceive themselves to be impaired in social or occupational functioning from those who do not feel as though their injury has resulted in lasting impairment, but does not rule out the possibility that the observed levels of emotional distress are due to neural pathology related to the concussion event. Further research is required to answer these questions.

Another potential limitation to the current study was the use of a single question to rate impairment in social and occupational functioning. While previous researchers have called for the increased inclusion of measures of social and/or occupational functioning in psychological research (McKnight et al., 2009), the measurement of this variable in the current research was limited to a single rating, and the reliability of this measure is unknown. This question was developed to satisfy the DSM-IV criteria for functional

impairment, but it is noted that the DSM-IV requires “significant impairment in social or occupational functioning”, which implies that impairment in either domain is sufficient to meet this criteria. Conflating “social” and “occupational” impairment into a single scale makes it difficult to know if one aspect of functioning is more affected than another, and to know if participants were primarily considering only one aspect of functioning, or both together. Both symptom report (Ruscio & Ruscio, 2000), and functional outcome measures (Bech, Lunde & Uden, 2002) have been criticised in the literature for lacking a unidimensional structure and factor analyses usually reveal more than one latent factor. This difficulty may not be only a product of the measure used, but also might arise due to the correlations between these underlying, or latent constructs, or to heterogeneity in the sample. While broad measures of functioning, such as the one used in this research are useful because they do not constrain the respondent to considering only subsets of symptoms, future researchers may wish to include measures which separate social and occupational functioning. Future researchers are encouraged to include measures of social and occupational functioning in models concerning outcome following concussive injury, in order to avoid circularity and widen the focus from symptom counts to measures of the effect that symptoms have on the functioning of the individual. Future researchers may wish to use more widely studies measures of functional impairment. For example, the most widely used measure of social functioning in Depression research is the Short Form Health Survey (SF-36), which includes two questions that ask about social functioning. Measures of employment status and absenteeism from work are typically thought to represent adequate in the evaluation of occupational functioning (McKnight et al., 2009).

It is unlikely that the pattern of results observed regarding the relative importance of emotional functioning on ratings of impairment is due to bias caused by concurrently rating emotions and impairment. This is because ratings of impairment were made first, and on a separate occasion than ratings of emotional functioning, and that not only emotional, but also somatic, and cognitive symptoms were asked about.

With further regard to the methods employed in the current research, it is noted that this data was collected before the implementation of DSM-V, which does not include post-concussional disorder. Post concussive complaints under the new diagnostic system would likely be best coded under “Mild Neurocognitive Disorder due to Traumatic Brain

Injury". It is interesting to note that this diagnosis does not acknowledge the influence of emotional symptoms at all, and that the diagnosis is based solely upon the report of cognitive symptoms. Further, it is noted that diagnosis can be made solely due to "concern of the individual...that there has been a mild decline in cognitive function". The data presented in this thesis suggests that attending to only cognitive complaints may not be the best way to characterise individuals with complaints after mild injuries such as concussion.

This research should help us to better understand chronic impairment following concussion. Further, the results of the current study are considered to have clinical relevance, in that they can be used to guide treatment planning. These data suggest that disordered recovery is best characterised by emotional dysfunction, as opposed to catastrophic ideation or hypochondriacal beliefs. Further, the results of this study indicate that the current emotional state of the respondent is the strongest predictor of impairment, above and beyond the influence of the current experience of cognitive and somatic symptoms. As such, treatments which focus on the alleviation of depressive symptoms and feelings of anxiety may be most effective in helping individuals with chronic symptoms recover.

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Appendix A. Brief Concussion Interview Form

Brief Concussion History Interview

Subject #: _____

Shiftworker: Please be aware that participants may be phoning the clinic inquiring about the "Concussion Study". If such an individual does phone please complete the following brief interview with them, and notify Kevin Whitfield at kwhitfie@sfu.ca. Please place each completed Interview in the file labeled "C710GRP". Please use the reverse of these pages as necessary. Thank-you.

Name: _____ Age: _____(discontinue if >65 or <19)

Home Tel. _____ Sex: (M F)

Other Tel. _____ English 1st Language? (Y N)

If first language is other than English: Years of education in English _____
Age when learned English _____

What number/time of day is the best to reach you? _____

How should we identify ourselves? CPC ___ SFU ___ Other (describe) _____

How did you hear about the study?

What was the date of your most recent concussion? _____(discontinue if < 3 mos. ago)
(month/year)

Are you currently involved in any legal proceedings? (Y N) (discontinue if YES)

How old were you when the concussion occurred? _____yrs.

Was the concussion sport related? (Y / N)

What happened? _____

How did you know that you had suffered a concussion?

Did you lose consciousness? (Y / N) If so – for how long? _____ mins
(discontinue if longer than 30 mins)

How long were you confused/groggy/unable to remember things for? _____ mins/hrs
(discontinue if longer than 24 hours)

If the concussion was sport related, did you continue playing? (Y / N)

Did you see a doctor/were you hospitalized? (Y / N) _____
(describe medical involvement)

Were there any other signs (other than loss of consciousness/confusion) that indicated to you that you had suffered a concussion? If so, how long did they last?

Did you experience any of the following symptoms after your head injury? How long did they last?

Symptom	Present/ Absent	Duration (mos.)
1) Becoming Easily Fatigued		
2) Disordered Sleep		
3) Headache		
4) Vertigo or dizziness		
5) Irritability or aggression on little or no provocation		
6) anxiety, depression or affective lability		
7) Changes in personality (e.g. social or sexual inappropriateness)		
8) Apathy or Lack of Spontaneity		

Did you experience any other symptoms that started after the immediate symptoms?

Do you feel as though you have experienced enduring symptoms as a result of this concussion? (Y / N) What symptoms would you say you still feel as a result of your concussion?

Please rate the degree to which your symptoms have impacted your social or occupational functioning.

1.....2.....3.....4.....5.....6.....7.....8
 no impact severe impact

Please rate the degree that your symptoms represent a worsening from your pre-injury level of functioning.

1.....2.....3.....4.....5.....6.....7.....8
 no impact severe impact

When the concussion occurred, did you hurt any other part of your body?
(neck/back/legs/broken bone/etc.) Explain:

Would you be interested in taking part in a group that was designed to lessen the intensity and frequency with which you experience these symptoms? (Y N)

Your responses indicate that you may be eligible for participation in our study. We would like to schedule a time when we could ask you some more questions. The interview, which will be conducted here at the Clinical Psychology Centre at SFU takes around 2 hours to complete. Participants are paid \$20.00 for their time. Can you suggest to us when would be a good time to phone you to schedule your interview?

Appendix B. Psychological and Physical Health Details

Current Psychological Health	PCD		Recovered		Age Matched Recovered	
	N	% of sample	N	% of sample	N	% of sample
Panic	2	10	2	5	1	2
Agoraphobia	1	5	0	0	0	0
Social Phobia	4	20	9	22	6	30
Generalised Anxiety Disorder	7	35	5	12	3	15
Obsessive Compulsive Disorder	1	5	1	2	0	0
Specific Phobia	7	35	10	24	5	25
Post Traumatic Stress Disorder	5	25	2	5	1	2
Major Depressive Episode	6	30	2	5	2	10
Bipolar Disorder	1	5	0	0	0	0
Hypochondriasis	0	0	1	2	1	2
Somatisation Disorder	0	0	0	0	0	0
Mixed Anxiety/Depression	0	0	1	2	1	2
Alcohol Abuse	0	0	0	0	0	0
Substance Abuse	0	0	1	2	1	2
Current Psychiatric Medication	4	20	3	7	3	15
Previous Psychiatric Medication	12	60	10	24	8	40
<i>Current Physical Health</i>	N	%	N	%	N	%
Currently in Medical Treatment	12	60	10	24	9	45
diabetic	2	10	2	5	2	10
heart problems	1	5	3	7	1	5
blood pressure issues	7	35	8	20	6	30
epilepsy	0	0	0	0	0	0
cancer	0	0	1	2	1	5
thyroid	2	10	2	5	2	10
other endocrinological problem	1	5	1	2	0	0
asthma	2	10	8	20	2	10
other respiratory problem	3	15	4	10	4	20
migraine	8	40	8	20	5	25
stroke	0	0	0	0	0	0
ulcer or gastrointestinal issues	7	35	7	17	6	30
other blood disorder	1	5	1	2	1	5
HIV/AIDS	0	0	0	0	0	0
other disease/disorder	6	30	2	5	1	5
allergies	10	50	20	50	11	55
smoker	3	15	6	15	4	20