The role of psychological flexibility on headache severity following mild traumatic brain injury

A thesis presented in partial fulfilment of the requirements for the degree of

Masters of the Arts

in

Psychology

at Massey University, Wellington, New Zealand.

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2022

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Abstract

Several studies have confirmed there is a relationship between psychological factors and the experience of ongoing symptoms following a mild traumatic brain injury (mTBI). However, there is mixed evidence about which psychological factors. One emerging factor is psychological flexibility, a concept with theoretical foundations and which can be increased through Acceptance and Commitment Therapy (ACT). While other studies have looked at psychological flexibility and overall symptoms, there is benefit in looking more specifically at one symptom. One of the most prominent symptoms following mTBI are headaches, however there is not, currently, a complete understanding of what factors may lead to the experience and severity of headaches following an mTBI. This study obtained data from 149 participants through concussion clinics throughout the North Island of New Zealand at clinic intake, and at six months follow up 100 participants continued to participate. We measured psychological flexibility through the AAQ-ABI(RA), psychological distress through the DASS-21, and headaches through one item on the RPQ. The findings from these individuals supported all our hypotheses. Specifically, we found significant associations through multiple regression analysis between lower psychological flexibility and higher severity headaches at two time points: baseline and six months follow-up. We also found an association between psychological flexibility at baseline and headaches at six months follow-up. We also completed mediation analysis which found a significant influence of psychological flexibility on the relationship between psychological distress and headache severity at the same time periods. These results indicate that psychological flexibility may be a key mechanism in the experience of headaches after mTBI. Our results suggest that targeting psychological flexibility through ACT may help to improve the severity of headaches after mTBI.

Key words: psychological flexibility, mild traumatic brain injury (mTBI), headaches, post concussive symptoms, psychological distress.

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Attestation of Authorship

"I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person (except where explicitly defined in the acknowledgements), nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning."

Signature: Lydia Johnston

Date: 12 November 2022

Acknowledgements

As with the finishing of anything, it is often just one point in time, and it would be too easy to forget all the people who in small and large ways have contributed to this research project. The biggest and most unfailingly supportive has been my supervisor, Dr Josh Faulkner. Your expert assistance and willingness to persist in helping me through this project, is unforgettable. A huge thank you for all the time you have taken to talk through and read through all the drafts, give thoughtful feedback, provoke my thinking and generally be a pleasure to work with. It is truly appreciated, and I am grateful for your ongoing support. There is no way this could have been done without you.

Acknowledgements must go my colleagues and manager who graciously let me adjust my work schedule and supported me with completing this research in any way that I needed. I am incredibly grateful for that flexibility and will not forget it. I promise I will now have more to talk about than the progress of this research.

Another huge thanks also must go to my friends and whānau. They have put up with continued discussions about this topic along with support during holidays to let me work on this research. It is very much appreciated and has helped lead to the progression through this research.

I would also like to express my gratitude for the people who took part in this research, along with people that continue to take part in research throughout the world. There are many ways we can learn about people's experiences but without people willing to take part in such research we would miss out on so much knowledge. I hope that this offers a small step towards greater understanding of what might help to make people's lives a little easier to manage.

Chapter 1: Introduction

Traumatic Brain Injuries (TBI's) are a common occurrence throughout the world, with some estimates indicating 69 million people worldwide are affected (Dewan et al., 2019). TBI's are diagnosed into three main categories, mild, moderate, and severe. Mild TBI (mTBI) is by far the most common, with estimates in New Zealand indicating they account for around 90% of all TBI's (Feigin et al., 2013). A TBI is defined as an external force that results in an alteration in brain functioning or other pathology of the brain (Iaccarino et al., 2021; Menon et al., 2010) and this can result from many different causes e.g., car crashes, assaults, etc. Following a mTBI people can experience a range of symptoms (termed 'Post Concussion Symptoms') which can be spilt into three categories; cognitive, emotional, and somatic (Katz et al., 2015). Traditionally, it was believed that only a small percentage would experience these symptoms past three months, however recent data indicates around 50% of people still experience symptoms one year after the injury (Barker-Collo et al., 2013; Dikmen et al., 2017; Nelson et al., 2019; Theadom et al., 2016).

The pathophysiology of mTBI includes cellular and metabolic changes, diffuse axonal injury and, in some cases, gross pathology or obvious lesion/s. However, these biological changes do not determine those individuals who go on to experience persistent post concussive symptoms (Carroll et al., 2004; Hou et al., 2012; Mikolic et al., 2021; Scheenen et al., 2017). This had led to some experts stating that the persistent symptoms people experience after a mTBI are not exclusively related to the injury itself and other factors may also contribute (Dean et al., 2012; Fear et al., 2009; Garden & Sullivan, 2010; Katz et al., 2015; Meares et al., 2008; Mickevičiene et al., 2004; Smith-Seemiller et al., 2003). Other explanatory factors have been investigated, including demographic factors (i.e. age, gender, education); injury related factors (i.e. whether or not an orthopaedic injury also occurred with the mTBI); medical history (i.e. a previous medical condition or the number of historical concussions); and post-injury factors (i.e. perceived social supports) (Iverson, et al., 2017; MacMillan et al., 2002; Meares et al., 2011; Ponsford et al., 2000; Snell et al., 2011; Stulemeijer et al., 2006, 2008; van der Naalt et al., 2017).

One risk factor that has become prominent in the literature is the impact of psychological factors on mTBI recovery (Belanger et al., 2013; Broshek et al., 2015; Faulkner et al., 2021b; Hou et al., 2012; Silverberg & Iverson, 2011; van der Naalt et al., 2017). Pre-injury mental health factors, (King, 1996; MacMillan et al., 2002; Meares et al., 2008; Ponsford et al., 2000), depression, anxiety, and stress (Broshek et al., 2015; Hou et al., 2012; Iverson et al., 2017; Meares et al., 2011; Ponsford et al., 2019; Scheenen et al., 2017; Snell et al., 2011; van der Naalt et al., 2017) and perception, attribution patterns and coping style (Anderson & Fitzgerald, 2020; Gregorio et al., 2014; Hou et al., 2012; Krpan et al., 2011; van der Naalt et al., 2017) have all been found to contribute to the ongoing experience of symptoms. Therefore, researchers are now taking a biopsychosocial approach to understand the experience of symptoms as this includes biological, social, and psychological factors (Register-Mihalik et al., 2020; Silverberg & Iverson, 2011; Silverberg et al., 2015; Young, 2020).

Headaches are one of the most common symptoms following a mTBI (Dikmen et al., 2010; Faux & Sheedy, 2008; Langer et al., 2022; Lucas et al., 2014; Nampiaparampil, 2008; Schwedt, 2021). Treating headaches may help to improve overall symptomology (Martelli et al., 1999) but evidence indicates current treatments are not always efficacious (DiTommaso et al., 2014; Fraser et al., 2017; Lucas, 2011; Silverberg, 2019; Watanabe et al., 2012). There are several explanations for the ongoing experience of headaches. Regarding the biological factors, the physiological mechanisms of mTBI are still being debated (Ashina et al., 2019; Packard, 1999; Schwedt, 2021). Research indicates that a historical or current affective disorders (post-traumatic stress disorder, depression, and anxiety) are linked to an increase in headaches (Ham et al., 1994; Jouzdani et al., 2014; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Mittenberg et al., 1992; Ruff & Blake, 2016; Silverberg et al., 2017; Solomon, 2009; Suhr & Spickard, 2012; Yilmaz et al., 2017). However, this does not tell us what specific mechanism is causing this association. More recently researchers have been focusing on transdiagnostic factors as these may offer more usefulness when it comes to treatment for the individual (Dalgleish et al., 2020; Harvey et al., 2004; Hayes et al., 1996). There has been some exploration of transdiagnostic mechanisms that might explain headaches after mTBI. For instance, expectations and beliefs about headaches, and fear avoidance (Ferrari et al., 2001; Fraser et al., 2017;

Gunstad & Suhr, 2001) have shown promise. However, treatments targeting some of these factors have yet to show consistent benefits (Baker et al., 2018; Kjeldgaard et al., 2014b; Silverberg, 2019).

Recently, Acceptance and Commitment Therapy (ACT) has been shown to be effective in pain populations and for headaches in the general population (Almarzooqi et al., 2017; Dindo et al., 2014; Grazzi, et al., 2019; Hughes et al., 2017; Lin et al., 2018; McCracken & Morley, 2014; Mo'tamedi et al., 2012; Vasiliou et al., 2021, 2022). ACT is underpinned by a mechanism called psychological flexibility (PF). This can be defined as acting in accordance with values despite the presence of pain or distress, which results in behaviour, which is open, aware, and active (Hayes et al., 2012; Kashdan & Rottenberg, 2010; McCracken & Morley, 2014). Improving PF is a core component of ACT, and this is accomplished through targeting six processes: acceptance, cognitive defusion, present focused awareness, self as an observer, values-based action, and committed action (Hayes et al., 2006, 2012; McCracken & Morley, 2014).

There is emerging evidence that treatment by ACT on TBI population is efficacious to improve overall outcomes (Sander et al., 2020; Whiting et al., 2020). We also know that the mechanism of psychological flexibility, or its inverse psychological inflexibility, has been shown to be related to psychological distress (Bohlmeijer et al., 2011; Masuda & Tully, 2011). It has been shown that psychological flexibility mediates the relationship between distress and symptoms following a mTBI (Faulkner et al., 2021b). Therefore, this study sought to understand the relation between the specific symptom of headaches after a mTBI and the transdiagnostic mechanism of psychological flexibility.

The first aim of this research is to explore the association between headaches and psychological flexibility for those with a mTBI. The prediction is that headache severity and psychological inflexibility will be positively associated at both acute (baseline) and persistent phases (six months follow-up). In addition, it is predicted that psychological inflexibility (immediately following the injury) will predict headache severity six months later.

The second aim is exploring if psychological inflexibility mediates the relationship between psychological factors (depression, anxiety and stress) and headache severity in those with a mTBI.

The prediction is that higher levels of depression, anxiety and stress experienced after a mTBI are associated with higher headache severity, and this relationship will be mediated by psychological inflexibility.

Chapter 2: Literature Review

Mild Traumatic Brain Injury

Worldwide around 69 million people are affected by a Traumatic Brain Injury (TBI) each year (Dewan et al., 2019). In New Zealand, the incidence of TBI is approximately 790 out of every 100,000 people, which equates to around 40,000 people in New Zealand annually (Feigin et al., 2013). In the United States it was estimated that 2% of the population live with a disability because of a TBI (Thurman et al., 1999), and given challenges with estimations this likely underrepresents the true incidence of burden. The associated cost associated with TBI is substantial. Lifetime estimates based on medical costs and loss of productivity, estimate that TBI costs approximately $60 billion annually in the US alone (Langlois et al., 2006). Additionally, TBI can be associated with a reduction in quality of life and cause a significant burden to friends and family (Langlois et al., 2006).

TBI is caused by an external force that results in an alteration in brain functioning or other pathology of the brain (Iaccarino et al., 2021; Menon et al., 2010). TBI can result from rotation, acceleration/deceleration of the brain, an external object entering the brain (for instance with a weapon), through blast-wave exposure, or any combination of these (Giordano & Lifshitz, 2021). The context that creates this external force can vary, but TBI commonly occurs because of car accidents, falls, assaults, and sports-related injuries (Katz et al., 2015; Menon et al., 2010). In New Zealand, the most common mechanisms of injury are falls (38%), followed by mechanical force (21%), transport accidents (20%) and assaults (17%) (Feigin et al., 2013).

TBI is classified into three categories (mild, moderate, and severe) based on the severity of the injury, (Blyth & Bazarian, 2010). Mild TBI (mTBI) is by far the most common (Dewan et al., 2019), with around 90% of all TBI's in New Zealand fitting into this category (Feigin et al., 2013). Current diagnostic classifications of mTBI generally agree that this injury is associated with either a

loss of consciousness[1] of 30 minutes or less, a Glasgow Coma Scale (GCS) between 13-15[2], a period of Post-Traumatic Amnesia (Dewan et al.)[3] of less than 24 hours, an impaired mental state at time of accident (confusion, disorientation etc.), or transient neurological deficit (including focal signs, epilepsy, or non-surgical intracranial injury).

It is worth noting other terms that appear in the mTBI literature. There is use of the term complicated mTBI which usually refers to the classification of mTBI (as above) but with the addition of visible brain lesions (such as haemorrhages or oedema) confirmed through neuroimaging, however there is mixed evidence of these being distinct diseases (Katz et al., 2015). As well as this, often the terms mTBI and concussion are used interchangeably, with the term concussion often associated with sports related injuries (Katz et al., 2015) and possibly at the milder end of the spectrum (Carter et al., 2021). From now on, we will continue to use the term mild traumatic brain injury (mTBI) to encompass both terms.

Following a mTBI, individuals can experience a myriad of difficulties that are classified under the umbrella term Post-Concussion Symptoms (PCS). These symptoms are highly variable given the number of factors involved in the injury combined with patient characteristics (Giordano & Lifshitz, 2021). Symptoms are generally classified into three areas; cognitive, emotional, and somatic (Katz et al., 2015). Cognitive deficits include issues with concentration, attention, memory, and processing speed. Somatic symptoms include fatigue, insomnia, headaches, dizziness, sensitivity to noise or light (phono and photophobia). Whereas emotional or affective complaints include irritability, anxiety, depression, and emotional liability (Bergersen et al., 2017; Katz et al., 2015).

[1] Loss of consciousness is defined as a state of unconsciousness caused by brain damage and requires either loss of the function of one or both cerebral hemispheres or the reticular activating system, which results in someone being unresponsive to stimuli (Blyth & Bazarian, 2010; Medicine, 2004, 2005).

[2] The Glasgow Coma Scale (GCS) is commonly used to determine these categories and the severity of head injury. It covers three parameters: eye opening, verbal response, and motor response. This results in a global score which ranges from 3 to 15, with a higher score indicating better functioning (Lefevre-Dognin et al., 2021).

[3] Post traumatic amnesia is defined as a state of altered consciousness where memory is impaired, and confusion and agitation may be present (Marshman et al., 2013).

Historically, it was believed that, in the majority of individuals who experience a mTBI, post-concussion symptoms would resolve within three months post injury. In a small percentage of people, approximately 10-20%, post-concussion symptoms would persist beyond these time frames (Ll Wood, 2004). However, recent evidence no longer supports this and indicates that up to 50% of people still experience symptoms 12 months after the injury (Barker-Collo et al., 2013; Dikmen et al., 2017; Nelson et al., 2019; Theadom et al., 2016). For example, a recent large cohort study (n = 1453) found that 53% of patients with a mTBI who presented to a US Level 1 Trauma Centre, reported continuing functional limitations, as per scores on the Glasgow Outcome Scale Extended (GOSE), at one year (Nelson et al., 2019).

Historically, the term 'post concussive syndrome' was used to classify these individuals who experienced ongoing symptoms (Katz et al., 2015; Mikolic et al., 2021). However, the use of this term has been criticized as a syndrome conceptualisation as it implies that all the symptoms are association with an aetiology i.e., mTBI. (Katz et al., 2015). However, it has been found that post-concussion symptoms are non-specific and consequently can be experienced by people with have not sustained a mTBI (Katz et al., 2015). Consequently, more recently the term persistent post concussive symptoms (PPCS) has been used (Cole & Bailie, 2016; Nelson et al., 2019; Ponsford et al., 2019). A recent consensus document defined PPCS as follows "the presence of any symptom that cannot be attributed to a pre-existing condition and that appeared within hours of an mTBI, that is still present every day three months after the trauma, and that has an impact on at least one sphere of a person's life" (Lagacé-Legendre et al., 2021 p. 99).

The impact of PPCS can be severe. A recent study found that 53% of people had ongoing functional impairments at 12 months post mTBI (Nelson et al., 2019). In addition, quality of life can also be affected, including a reduction in mental and physical health (Ponsford et al., 2019) and lower satisfaction with life (McMahon et al., 2014). PPCS has also been associated with depression (Barker-Collo et al., 2015) and longer time off work (Stulemeijer et al., 2006). These ongoing impacts highlight the significance of understanding the factors that contribute to PPCS. To do this we first

need to need to understand the pathophysiology of a mTBI and consequently identify how changes to the brain might initiate these symptoms.

Pathophysiology of mTBI

The pathophysiology of mTBI can be understood by cellular and metabolic changes, diffuse axonal injury and, in some cases, by gross pathology or obvious lesion/s. The next section will briefly discuss each of these pathophysiological mechanisms.

Due to the forces that are exerted onto the brain because of a mTBI, brain cells cannot maintain their stable energy states. This leads to the release of a large amount excitatory neurotransmitters, particularly glutamate (Blyth & Bazarian, 2010; Werner & Engelhard, 2007). Glutamate or glucose is the main energy source of the brain (Bigler & Maxwell, 2012) and hence these changes cause disruption in the functioning of neurons. More specifically, the release of glutamate has cascading implications on the balance of chemicals in the neuron, resulting in more calcium entering and more potassium being released (Giza & Hovda, 2001). However, neurons need to continue to function. The changes in the biochemistry of the cell result in more energy being required but these changes have implications on the ability of the cell to receive this energy. A reduction in calcium affects oxygen metabolism and the increase in potassium reduces the ability of blood vessels to bring glucose (energy) into cells (Werner & Engelhard, 2007). In addition, cerebral blow flow is often reduced (Werner & Engelhard, 2007). All of this results in what is termed an 'energy crisis' within the cells, in which more glucose is needed than can be supplied (Giza & Hovda, 2001). This can continue for days following the injury and can consequently precipitate post-concussion symptoms (i.e., fatigue, slowed processing speed, memory issues, headaches etc.) (Giordano & Lifshitz, 2021).

Once this initial energy crisis has reduced, the brain can go into a period of reduced cerebral metabolism (a lack of glucose in the brain) (Giza & Hovda, 2001), which has been associated with a greater degree of injury severity (Tavazzi et al., 2005). This can lead to impairments in mitochondrial oxidative metabolism (the process cells use to release chemical energy) which leads to reduced

adenosine triphosphate (ATP) production (Werner & Engelhard, 2007). ATP enhances and delivers nutrients and oxygen to the brain, so this reduction can lead to a worsening of the energy crisis and can ultimately lead to cell death (Werner & Engelhard, 2007). These cellular and metabolic changes have been associated with lower executive functioning and emotional disturbances following mTBI (Gasparovic et al., 2009).

As well as these initial cellular and metabolic changes, injuries (especially from falls or sudden impacts) will often result in diffuse damage, specifically tissue distortion and shearing of axons, commonly referred to as diffuse axonal injury (Giordano & Lifshitz, 2021). This often affects the frontal and temporal lobes (see Figure 1) based on the way the brain is situated within the skull and the movement that occurs with the force (Bigler, 2007). Diffuse axonal injury refers to changes at the axon level (the nerve fiber) which disrupt the function of the axons, whose normal job is to transport impulses to other neurons (Giordano & Lifshitz, 2021) (see Figure 2 below). These disruptions can lead to flow on effects in the form of secondary transsynaptic injury (which is the transmission from one neuron to another) (Katz et al., 2015; Niogi & Mukherjee, 2010). Diffuse axonal injury is associated with executive dysfunctions and processing speed (Niogi & Mukherjee, 2010) and specifically in the frontal lobes (Lipton et al., 2009). Furthermore, diminished white matter tracks have also been associated mTBI (Hayes, et al., 2015) and specifically with headaches (Leung et al., 2018), a frequent symptom in mTBI.

Figure 1

Picture of the Human Brain Noting the Four Lobes and Key Gyrus's

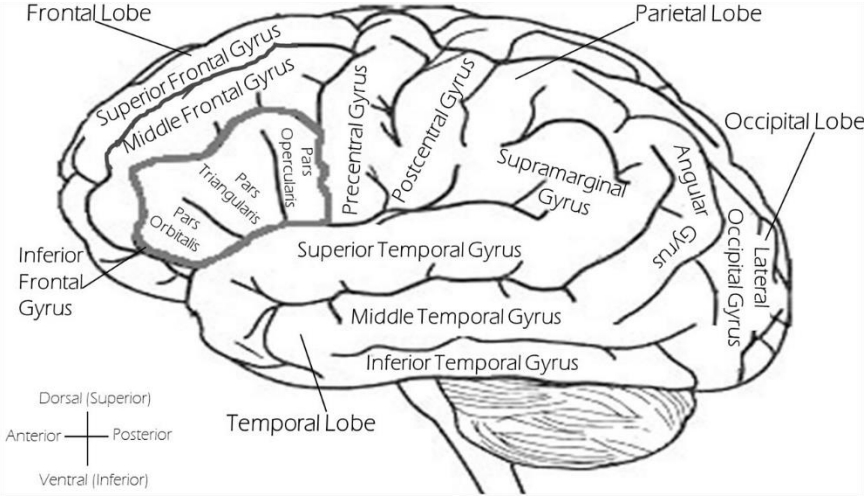
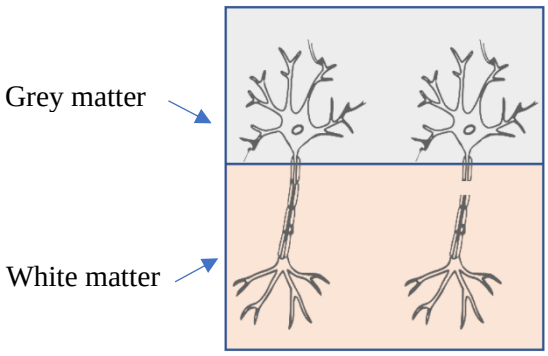


Figure 2

A Depiction of Axon Shearing Which Occurs in Diffuse Axonal Injury



As noted earlier, some people with mTBI have minor intracranial abnormalities (Bigler & Maxwell, 2012). These can include petechial haemorrhages (small bleeds in the brain), mild oedema (build-up of excess fluid) and/or small surface contusions (bruises in the brain) (Smits et al., 2008). In the literature the term complicated mTBI, is at times used to dissociate those individuals that have experienced these intracranial abnormalities (Katz et al., 2015). Currently, evidence regarding the implications of complicated versus uncomplicated mTBI on recovery and long-term outcomes is mixed (Lange et al., 2009; Nelson et al., 2019; Theadom et al., 2016). For instance, Lange et al.

(2009) found differences only in 3 out of 13 cognitive measures between those with and without imaging abnormalities approximately 3.5 days after their injuries in a sample of 20 mTBI participants and 20 controls. Similar to this, a recent study (Karr et al., 2020) found no differences in either cognitive or psychological measures or in symptom reporting between complicated or uncomplicated mTBI. However, in contradiction to this, Voormolen, et al. (2020) looked at a sample of over 1100 patients and found that those with complicated mTBI reported lower health related quality of life and functional outcome compared to those with uncomplicated mTBI at three- and six-months post injury. This provides some evidence of the mixed results when it comes to effects of intracranial abnormalities on recovery.

To summarise, the force associated with mTBI has implications on the biochemistry, metabolism, and integrity of white matter pathways in the brain. In addition, in some individuals a complicated mTBI can arise due to lesions and gross pathological changes occurring to the cerebral cortex. All these biological changes can precipitate the constellation of post-concussion symptoms that can arise after mTBI.

While these pathological changes are present there is increasingly inconsistent evidence that injury severity predicts recovery outcomes (Carroll et al., 2004; Hou et al., 2012; Mikolic et al., 2021; Scheenen et al., 2017). That is, the level of severity and thus the extent of brain pathology is not always associated with the number or persistence of symptoms. There are several aspects of a mTBI which can indicate severity; degree of loss of consciousness (Nelson et al., 2019), post traumatic amnesia (Dewan et al., 2019), the GCS score, and the presence of intracranial abnormalities. Research has explored these various indices of injury severity and inconsistent evidence continues to arise regarding its ability to predict outcomes and recovery after mTBI (Carroll et al., 2004; Hou et al., 2012; Lovell et al., 1999; Mikolic et al., 2021; Meares et al., 2008; Nelson, et al., 2019; Ponsford, et al., 2019; Scheenen et al., 2017). For example, in a prospective study of 90 participants with mTBI, Meares et al. (2008) found that in the acute period (up to 14 days post-injury) the presence of PTA was not predictive of a later diagnosis of Post-Concussion Syndrome. Instead, they found the strongest predictor of symptoms was pre-injury affective or anxiety disorders. It is worth noting that

they used self-report measures of PTA which may be subject to recall biases or inaccurate reporting, although they did match these up with hospital records. Furthermore, the impact of intracranial abnormalities on the number and persistence of post concussive symptoms is also mixed (Nelson et al., 2019; Theadom et al., 2016; van der Naalt et al., 2017; Voormolen, et al., 2020). Nelson et al. (2019) recruited a large sample of 1453 participants who experienced a mTBI and tracked them over a 12-month period. They divided those with a mTBI into two groups with either acute intracranial findings present (CT+) or absent (CT-). They found no differences in symptoms or cognitive functioning between the two groups at 12 months post injury.

As well as injury severity not consistently predicting post concussive symptoms, there is evidence that other populations, for example, those with chronic pain, depression, trauma, non-head related injuries, as well as healthy populations show similar symptoms (Dean et al., 2012; Fear et al., 2009; Garden & Sullivan, 2010; Katz et al., 2015; Meares et al., 2008; Mickevičiene et al., 2004; Smith-Seemiller et al., 2003). Fear et al. (2009) looked at a large (n=5869) random sample of military personnel who served in the Iraq war. They looked at symptoms and severity and found that those subjected to blasts (with an assumption they likely sustained a mTBI) had similar levels of post-concussion symptoms compared to those exposed to other events including aiding the wounded and exposure to uranium (where no mTBI is likely to have occurred). They concluded that post concussive symptoms are not only related to those who have sustained a head injury. It is worth noting that because of the self-report nature of this study along with no formal diagnosis of mTBI, some caution should be taken. However, in the study by Meares et al. (2008) discussed above, they also found that both mTBI and their trauma controls had similar reports of post-concussion symptoms. Post-concussion symptoms have consequently been described as non-specific as the symptoms experienced after mTBI are not unique to mTBI and have been found in other populations (Dean et al., 2012; Fear et al., 2009; Katz et al., 2015; Meares et al., 2008). This might lead to a conclusion that there is no specific disorder of post concussive symptoms following an mTBI, however, people are still experiencing the symptoms. What is needed, therefore, is a greater understanding of what other factors, aside from the injury itself, play a part in the expression of post-concussion symptoms.

As we have noted, post-concussion symptoms are not consistently related to injury severity and can also be present in other populations. Consequently, to provide accurate explanations for the presence and persistence of post-concussion symptoms following mTBI, prognostic models now include additional explanatory factors. These aetiological explanatory factors may include demographic factors such as age, gender, education; injury related factors for example whether or not an orthopaedic injury also occurred with the mTBI; medical history such as a previous medical condition or the number of historical concussions; and post-injury factors such as perceived social supports (Iverson, et al., 2017; MacMillan et al., 2002; Meares et al., 2011; Ponsford et al., 2000; Snell et al., 2011; Stulemeijer et al., 2006, 2008; van der Naalt et al., 2017). One risk factor that has become prominent in the literature is the impact of psychological factors on mTBI recovery (Belanger et al., 2013; Broshek et al., 2015; Faulkner et al., 2021b; Hou et al., 2012; Silverberg & Iverson, 2011; van der Naalt et al., 2017). The next section will provide a more detailed summary of the role of psychological factors on the severity and persistence of symptoms after mTBI.

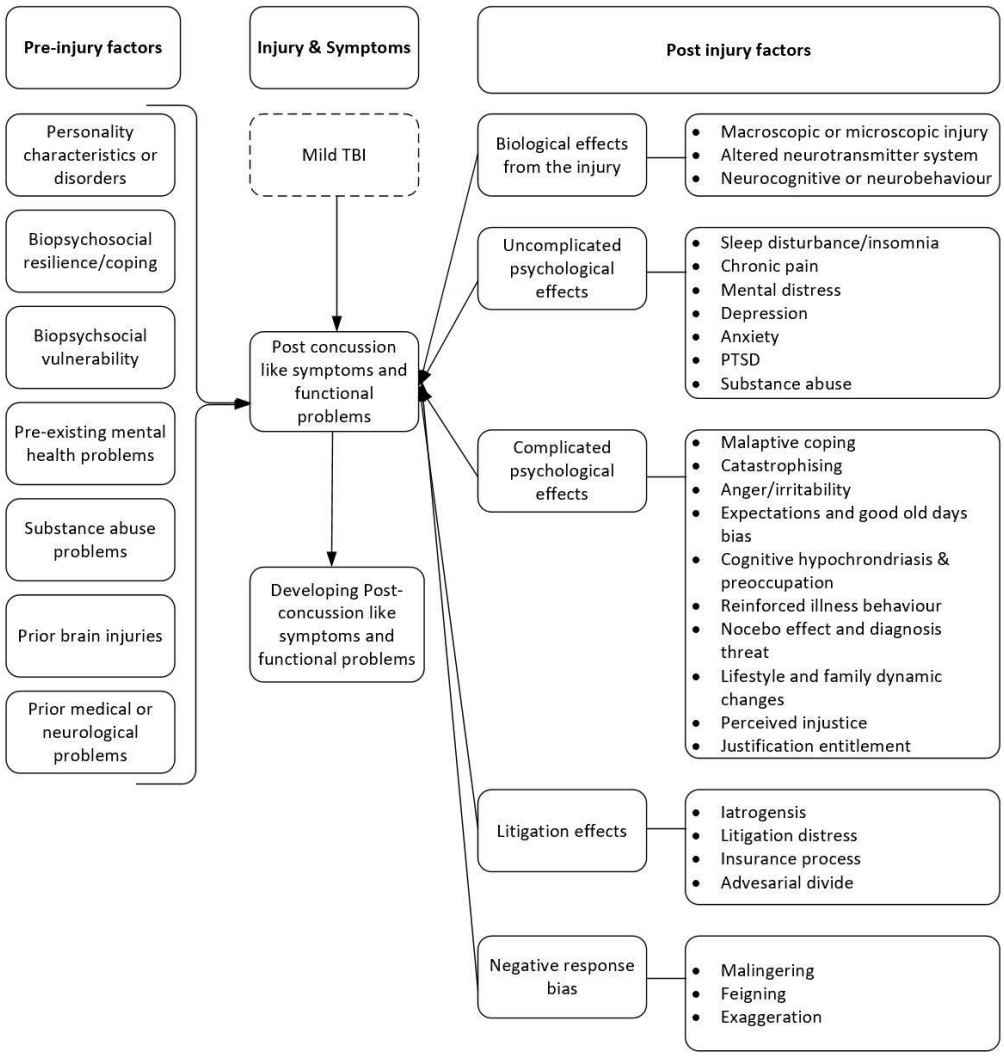
Psychological Factors and mTBI Recovery

Several psychological factors have been found to predict outcomes in post-concussion symptoms (Belanger et al., 2013; Broshek et al., 2015; Faulkner et al., 2021b; Hou et al., 2012; Silverberg & Iverson, 2011; van der Naalt et al., 2017). Firstly, pre-injury mental health factors, such as a clinical mental health diagnoses prior to the occurrence of the injury, are associated with a longer duration of post-concussive symptoms (King, 1996; MacMillan et al., 2002; Meares et al., 2008; Ponsford et al., 2000). Related to this, the emotional distress someone feels after the injury, including feelings of depression, anxiety, and stress, contribute to post-concussion symptom severity and duration (Broshek et al., 2015; Hou et al., 2012; Iverson et al., 2017; Meares et al., 2011; Ponsford et al., 2019; Scheenen et al., 2017; Snell et al., 2011; van der Naalt et al., 2017). In a recent systematic review of prognostic models of mTBI recovery, Silverberg and colleagues (2015) found that pre-injury mental health and acute psychological distress were the most robust predictors of persistent

post-concussion symptoms following mTBI. Furthermore, there is also evidence that an individual's perception, attribution patterns and coping style (i.e., adaptive vs. avoidant coping) are associated with post-concussion symptoms (Anderson & Fitzgerald, 2020; Gregorio et al., 2014; Hou et al., 2012; Krpan et al., 2011; van der Naalt et al., 2017). Accordingly, researchers have argued that explanatory models of persistent post-concussion symptoms adopt a biopsychosocial approach (Register-Mihalik et al., 2020; Silverberg & Iverson, 2011; Silverberg et al., 2015; Young, 2020). These approaches acknowledge the interconnection between biological, psychological, and socio-environmental factors (Iverson, 2019; Kenzie et al., 2017; Register-Mihalik et al., 2020). One such example of a biopsychosocial model of mTBI recovery can be visualised in Figure 3. Essentially, this model outlines that several pre-injury or predisposing factors (mental health, other brain injuries, coping styles etc.) lead to the experience of symptoms along with the injury itself (precipitating factor). The persistence of symptoms is also exacerbated by other factors such as the pathophysiology associated with mTBI, mood disorders after the injury, and how one responds to the injury (perpetuating factors). All this combined (or several of these factors combined) can lead to the ongoing persistence of symptoms (Young, 2020).

Figure 3

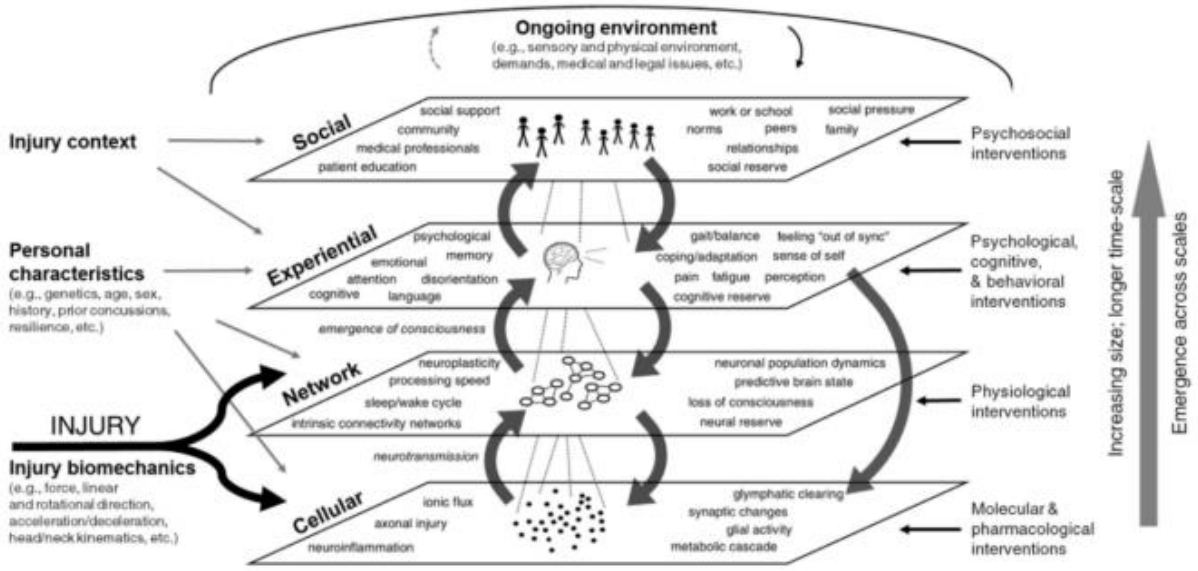
The Biopsychosocial Model adapted from Young (2020, p. 430).



Another way of conceptualising the biopsychosocial model is to look at it from a network or systems approach (Iverson, 2019; Kenzie et al., 2017). Iverson (2019) argues that instead of thinking about one singular event as casual to the persistence of symptoms after mTBI, it is the symptoms themselves which constitute the syndrome. That is, symptoms are often correlated, interactive, amplifying and mutually reinforcing each other (Kenzie et al., 2017). They also occur within a person's own context, life events, historical factors, and biological changes and it is this network that leads to the continuation of symptoms. An example of this approach can be visualised in a model by Kenzie et al. (2017) as shown in Figure 4.

Figure 4

Multiscale System Approach to mTBI adapted from Kenzie et al. (2017, p.6)



Biopsychosocial models have previously considered PCS in its entirety, that is they have looked at the overall total number of symptoms, rather than looking specifically at individual symptoms (Iverson, 2019; Kenzie et al., 2017; Silverberg et al., 2015). However, given the heterogeneity of the symptoms, in that they include cognitive, affective, and physical symptoms and within these broad categories include further variability, there is benefit in looking at the individual symptoms to better understand what might be occurring. For instance, some symptoms may increase or decrease over time, but these effects are not obvious when we consider PCS in totality. We also know that there is high variability with regards to the frequency and impact of specific symptoms in individuals (Bigler, 2008; Iverson, 2019; Register-Mihalik et al., 2020). This variability may be easier to unpack if we look at one specific symptom.

One of the most frequent and painful symptoms following an mTBI, are headaches (Martelli et al., 1999; Martins et al., 2012). Headaches are often experienced more in those with a mild TBI rather than in those with moderate or severe brain injuries (Couch & Bearss, 2001), adding complexity to their occurrence. They have also been shown to be associated with mTBI even when accounting for PTSD and depression (Hoge et al., 2008). As headaches are frequent in this population,

it is possible that being able to cope with this key symptom could be important in supporting overall recovery from mTBI (Martelli et al., 1999). Therefore, headaches present as a worthwhile individual symptom to further investigate.

Headaches

Headaches are one of the most common symptoms following a mTBI (Dikmen et al., 2010; Faux & Sheedy, 2008; Langer et al., 2022; Lucas et al., 2014; Nampiaparampil, 2008; Schwedt, 2021). A headache is defined as a pain in the head, neck, or face (Mirriam Webster, n.d.). Headaches after a mTBI, are often referred to as Post Traumatic Headaches (PTH). Estimates of the cumulative incidence of headaches after mTBI has been estimated at 71- 91% over one year (Hoffman et al., 2011; Lucas et al., 2014) and the lifetime prevalence of headaches following any head trauma is estimated at 4% (Rasmussen & Olesen, 1992). The impact of PTH can be significant (Dumke, 2017; Martins et al., 2012; McMahon et al., 2014; Vos et al., 2012; Yilmaz et al., 2017) and the mean headache frequency can be high, with one study estimating 25.4 days (+/- 7.1 days) per month (Ashina et al., 2020). The implications of PTH can also be significant. Those with PTH are less likely to return to work (Dumke, 2017; Kjeldgaard et al., 2014a; Yilmaz et al., 2017), have a lower quality of life (Martins et al., 2012), and rate their health more poorly (Kjeldgaard et al., 2014a). Therefore, given the frequency that people experience post-traumatic headaches and the impact they can have on people's quality of life and ability to work, headaches constitute a significant individual symptom following mTBI.

Post Traumatic Headaches are classified as a secondary headache condition by the International Classification of Headache Disorders – 3rd Edition ((IHS), 2018). A secondary headache condition is a headache that is associated with a specific injury or factor, rather than a primary headache which is not linked to any other cause ((IHS), 2018). If PTH resolve by themselves within three months post injury, they are considered *Acute*. However, if they continue for greater than three months after mTBI, they are defined as *Persistent*. According to the IHS, headaches need to start within seven days of the injury and be accompanied by symptoms of mTBI such as memory loss,

nausea, visual disturbances etc. ((IHS), 2018). Full diagnostic criteria can be found in Table 1. Estimates of the prevalence of persistent PTH vary from 15% - 78% (Evans, 2006; Faux & Sheedy, 2008; Ingebrigtsen et al., 1998; Kraus et al., 2005; Minderhoud et al., 1980; Nampiaparampil, 2008). This large variation is likely due to methodological differences in the ways headaches are defined, measured, and the time frame post injury when they are assessed (e.g., 3 vs. 6-months post injury). It is also worth noting that similar to PCS, the PTH literature is considered controversial, given it implies that it stems from the injury which may or not may not be the case (Obermann et al., 2010; Seifert & Evans, 2010; van Reekum, 2013).

Table 1.

Diagnostic Criteria of Acute and Persistent Headaches Following TBI and Specifically mTBI.

	Acute Headache due to TBI diagnostic criteria 5.1	**Persistent Headache due to TBI criteria 5.2**
Criteria for headaches after Mild TBI	A. Headache fulfilling criteria for 5.1 Acute headache attributed to traumatic injury to the head B. Injury to the head fulfilling both of the following: 1. associated with none of the following: a) loss of consciousness for >30 minutes b) Glasgow Coma Scale (GCS) score <13 c) post-traumatic amnesia lasting >24 hours1 d) altered level of awareness for >24 hours e) imaging evidence of a traumatic head injury such as skull fracture, intracranial haemorrhage and/or brain contusion 2. associated with one or more of the following symptoms and/or signs: a) transient confusion, disorientation or impaired consciousness b) loss of memory for events immediately before or after the head injury	A. Headache fulfilling criteria for 5.2 Persistent headache attributed to traumatic injury to the head B. Head injury fulfilling both of the following: 1. associated with none of the following: a) loss of consciousness for >30 minutes b) Glasgow Coma Scale (GCS) score <13 c) post-traumatic amnesia lasting >24 hours d) altered level of awareness for >24 hours e) imaging evidence of a traumatic head injury such as skull fracture, intracranial haemorrhage and/or brain contusion 2. associated with one or more of the following symptoms and/or signs: a) transient confusion, disorientation or impaired consciousness b) loss of memory for events immediately before or after the head injury

	c) two or more of the following symptoms suggestive of mild traumatic brain injury:	c) two or more of the following symptoms suggestive of mild traumatic brain injury:
	i. nausea	i. nausea
	ii. vomiting	ii. vomiting
	iii. visual disturbances	iii. visual disturbances
	iv. dizziness and/or vertigo	iv. dizziness and/or vertigo
	v. gait and/or postural imbalance	v. gait and/or postural imbalance
	vi. impaired memory and/or concentration.	vi. impaired memory and/or concentration.

Table adapted from (IHS, 2018)

Headaches also occur in the general population (primary headache) and by understanding the different phenotypes (categories of headaches) this can provide insights into the types of headaches that can arise following mTBI. In the general population, the IHS (2018) defines four primary headache types: 1) migraine; 2) tension-type headache; 3) trigeminal autonomic cephalalgias; and 4) other primary headache disorders ((IHS), 2018). A migraine type is generally a recurrent headache, resulting in attacks lasting 4–72 hours, which normally occur in a unilateral location, with a pulsating quality, and of moderate or severe intensity. They can be aggravated by routine physical activity and associated with nausea. A tension-type headache is typically bilateral, with a pressing or tightening quality, and of mild to moderate intensity which can last minutes to days. The pain does not worsen with routine physical activity and is not associated with nausea. A trigeminal autonomic cephalalgia (TAC) is often a shorter duration headache, which lasts several minutes to hours but with a severe, unilateral pain which can be orbital, supraorbital, temporal or in any combination of these sites, occurring from once every other day to eight times a day. Lastly, the final type, are headaches which do not fit the other criteria and are often associated with other occurrences e.g., cough headache or exercise headaches. It is also worth noting a combination of these types of headaches can occur (Packard, 1999).

PTH are not further defined into these categories, according to the IHS, but some recent studies have applied this classification system to better understand the characteristics of PTH (Ashina et al., 2020; Lucas et al., 2014; Schwedt, 2021). Ashina et al. (2020) studied 100 people with

persistent PTH and using a semi-structured interview found that the most common type of headache was chronic migraine-like headache. This was followed by combined migraine and tension type headache, and a small number reported a chronic (frequent and ongoing) tension-like headache. This is consistent with the findings of Schwedt (2021) who summarised the literature, finding the most common types after mTBI were migraine or tension type headaches. This shows the heterogeneity of PTH and illustrates that there is not a unique symptom experience of headaches following mTBI. To understand more about headaches, the next section will summarise what is currently known about their aetiology after mTBI.

Aetiology of Post Traumatic Headaches

Biopsychosocial explanatory models, which were described earlier, can also be applied to the experience of post-traumatic headache (Evans, 2010; Fraser et al., 2017; Kjeldgaard et al., 2014a; Packard, 1999; Register-Mihalik et al., 2020; Solomon, 2009). Regarding the biological factors, the physiological mechanisms of mTBI which contribute to PTH are still being debated (Ashina et al., 2019; Packard, 1999; Schwedt, 2019, 2021). However, some explanations have been investigated in the literature. First, the neurometabolic changes (i.e., the energy crisis and cellular metabolic changes) have been implicated as a causal factor in PTH, possibly by their effects on the trigeminovascular system (a system implicated in migraines in the general population) (Ashina et al., 2019; Benromano et al., 2015; Packard, 1999; Schwedt, 2021). The trigeminovascular system consists of small sensory neurons which are located within the trigeminal ganglion and are associated with pain pathways (May & Goadsby, 1999). Second, anatomical changes including diffuse axonal injury and grey and white matter abnormalities (also described earlier) have been implicated in the experience of PTH, hypothesized to be through its effect on descending pain modulation systems (Ashina et al., 2019; Burrowes et al., 2020; Chong, et al., 2018; Leung et al., 2018; Ofoghi et al., 2020; Schwedt et al., 2017). The descending pain modulation systems is a network of brain regions, which help modulate and control pain responses (Ashina et al., 2019). These studies provide some initial evidence that the pathological changes to the architecture of the brain following mTBI may be associated with PTH.

The neurometabolic and anatomical changes, just described, offer some of the explanation for why headaches might occur and persist after mTBI, but taking a biopsychosocial approach, it is likely, other factors also contribute. Dynamic (changeable) and static (fixed) factors both contribute to our understanding of PTH. However, as static factors are not able to be changed, they may only be useful to understand who may be more at risk of developing PTH, but not how we might treat people with PTH. The static factors found to contribute to the likelihood of experiencing PTH include a prior history of headaches and female gender (Hoffman et al., 2011; Lieba-Samal et al., 2011; Schwedt, 2021; Yilmaz et al., 2017). Aside from these static factors there are several dynamic psychological factors which have been linked to PTH. These include, affective disorders (post-traumatic stress disorder, depression, and anxiety), expectations and beliefs about headaches, and fear avoidance (Ferrari et al., 2001; Fraser et al., 2017; Gunstad & Suhr, 2001; Ham et al., 1994; Jouzdani et al., 2014; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Mittenberg, et al., 1992; Ruff & Blake, 2016; Silverberg et al., 2017; Solomon, 2009; Suhr & Spickard, 2012; Yilmaz et al., 2017). The following section will outline these factors in more depth.

Post-traumatic stress disorder, depression and anxiety have been found to be associated with PTH (Fraser et al., 2017; Ham et al., 1994; Jouzdani et al., 2014; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Ruff & Blake, 2016; Yilmaz et al., 2017). Jouzdani et al. (2014) completed a prospective study in a military sample, where 30 males were diagnosed with mTBI. They looked at a range of characteristics to better understand PTH in this population. They measured headaches using a combination of clinical interviews and questionnaires and used the diagnostic criteria from ICHD-2. They completed their measures at two time points, approximately three months apart. They found that headaches were not associated with any demographic or injury factors, but headaches were higher in those with PTSD and depression. One possible explanation for this association is the experience of stress within the brain (Nash & Thebarge, 2006). Specifically, psychological stress can affect the nociceptive neurons in the trigeminovascular system and the hypothalamic-pituitary-adrenal axis (HPA axis) which can decrease effectiveness of the pain systems and influence the experience of pain. If this occurs repeatedly (through ongoing stress) it may be one explanation for the chronicity of

headaches (Fraser et al., 2017; Nash & Thebarge, 2006). Supporting this idea are studies which looked specifically at PTSD and headaches which have found that PTSD and headaches are highly correlated (Afari et al., 2009; Balaban et al., 2012). As well as the way stress effects the brain, there are other explanations such as the shared vulnerability and mutual maintenance model, as outlined by Asmundson et al. (2002). The shared vulnerability model posits that an underlying mechanism is responsible for both affective disorders and headaches, for example anxiety sensitivity (fear of anxiety symptoms (Reiss, 1991)), which has been shown to be present in both PTSD and pain (Asmundson et al., 2000). On the other hand, the mutual maintenance model outlines that those with affective disorders may be more prone to negative interpretations (finding a negative view of things which are ambiguous) of their head pain. These negative interpretations create a greater degree of focus on their pain, leading to more distress and this cycle leads to the persistence of headaches (Sharp & Harvey, 2001). Unpacking and confirming what is occurring here is complex and it is possible a combination of these explanations is occurring.

To help understand this complexity, the wider research community is now exploring the role of specific psychological mechanisms (Frank & Davidson, 2014; Kazdin, 2007; Tryon, 2016). While traditionally there has been a focus on the broad diagnostic categories i.e., depression and anxiety, it has been acknowledged that there are mechanisms within these disorders, which also occur in other disorders (Dalgleish et al., 2020; Harvey et al., 2004; Hayes et al., 1996). These are termed transdiagnostic processes (or mechanisms) and are defined as cognitive or behavioural mechanisms which contribute to and maintain psychopathy across multiple disorders (Harvey et al., 2004; Morris & Mansell, 2018; Sauer-Zavala et al., 2017). Understanding the underlying processes which maintain different symptoms or disorders, will ultimately improve treatment outcomes for the individual (Frank & Davidson, 2014; Hayes et al., 2019; Sauer-Zavala et al., 2017). For example, someone may have anxiety, but this does not tell us what specific process is driving *this* individual's anxiety. For instance, they may have anxiety related beliefs such as, *I am fearful of pain*, or *I am fearful I will not be able to control my anxiety*. Identifying these unique processes can then become the target for treatment in specific individuals. With regards to PTH, it is possible that it is being maintained by a

psychological process or processes, which could also explain the co-occurrence in affective disorders after mTBI. If this is the case, understanding what this process is, will help to pinpoint the best treatment approach to alleviate this symptom.

There is evidence that certain transdiagnostic processes are associated with overall PCS, headaches and PTH specifically (Ferrari et al., 2001; Fraser et al., 2017; Gunstad & Suhr, 2001; Mittenberg et al., 1992; Silverberg et al., 2017; Solomon, 2009; Suhr & Spickard, 2012). Firstly, expectation as aetiology refers to the idea that our belief that symptoms are expected to occur after mTBI lead to us experiencing more symptoms (Ferrari et al., 2001; Fraser et al., 2017; Gunstad & Suhr, 2001; Mittenberg et al., 1992; Solomon, 2009). Mittenberg et al. (1992) outlined this in their original study which compared 100 head injured participants and 223 control participants. They provided control participants with an imagined scenario that they had received a head injury. They then measured the number of anticipated versus actual symptoms in both groups. They found the control group expected headaches to occur slightly more than they occurred in the mTBI sample. Given the number of symptoms was so similar, this led to the conclusion that our expectations about what might occur are having an influence on what does occur. There are similar ideas in the primary headache literature, which relate to peoples' beliefs about the cause and maintenance of their headaches (their perceived locus of control, which is either focused on external or internal explanations) (French, et al., 2000). French et al. (2000) found in a sample of 329 people presenting with primary headache disorder, that an external locus of control and lower self-efficacy contributed to the disability associated with headaches, albeit they only accounted for 21% of the variance in disability.

A similar concept of base rate misattribution has been put forward to explain symptoms following mTBI (Gunstad & Suhr, 2001; Mittenberg et al., 1992). This refers to people overestimating their symptoms following their injury and incorrectly classifying everyday symptoms as related to the injury. For instance, a headache occurring after cessation of coffee drinking could be erroneously attributed to the brain injury. This is possibly more likely to occur with headaches given the larger incident of headaches in the general population (Rasmussen & Olesen, 1992). To support

this, Gunstad and Suhr, (2001) found in a sample of 190 undergraduates, that both primary headache sufferers and those with head injuries underestimated premorbid symptom rates relative to controls. This indicates that people are underestimating the occurrence of headaches in regular life. While this study is not specifically with people with PTH, it does include both people with mTBI and those with headaches, indicating that it is highly likely this phenomenon would also occur in the PTH population. Related to this, Anderson and Fitzgerald (2020) looked specially at those with PCS and divided symptoms into two clusters, one was three physical symptoms which included headaches which they called 'early type PCS', the other included the other 13 symptoms which they called 'late type PCS'. They showed that while controlling for psychological distress, the degree to which the participants believed their symptoms were due to the mTBI significantly predicted early and late type PCS symptoms, indicating again that it is the interpretation of symptoms which may be exacerbating their experience.

Another mechanism by which PTH may be maintained is through fear avoidance (Silverberg et al., 2017; Suhr & Spickard, 2012). Fear avoidance can be described as a pattern of avoidance, starting with negative thoughts about headaches. This leads to an avoidance of activities that people fear will trigger the headache. This avoidance is reinforcing as the perceived trigger does not occur, and nor do any negative consequences e.g., no headache (Cassetta et al., 2021; Vlaeyen & Linton, 2000). In relation to PTH, a specific term, cogniphobia is used to describe avoidance of activities which may cause mental exertion (e.g., avoidance of doing your taxes) for fear out of developing a headache (Silverberg et al., 2017). Interesting, cogniphobia has been found to be a unique headache trigger for mTBI, in that mental exertion has not been found to be a headache trigger in other headache populations (Silverberg, 2019; Silverberg et al., 2019).

Silverberg et al., (2017) investigated cogniphobia and whether this might influence the disability associated with headaches and cognitive functioning in those with mTBI. In a sample of 80 participants recruited through outpatient clinics and approximately 2-3 months post injury they measured cognitive test performance, cogniphobia (via The Cogniphobia Scale), post-concussion symptoms and fear avoidance of other activities. Headaches were measured using one headache item

from the British Columbia Post-Concussion Symptom Inventory. They found that participants with higher severity headaches reported higher levels of cogniphobia, in that they avoided mentally taxing activities for fear of developing or worsening a headache. Silverberg et al., (2017) also found that those who avoided mental exertion also avoided other things like stress triggers. Why this occurs it not specifically known, but it is possible that avoidance may be exacerbated by the information people receive after their brain injury, which is often about resting and avoiding strenuous activities (Silverberg et al., 2017), and specifically for headaches, people are often told to avoid activities which may trigger the headache (Friedman & De Ver Dye, 2009). This might be helpful advice at the beginning of recovery but if it continues, may become problematic.

These ideas about fear avoidance are also present in the wider pain research (Vlaeyen & Linton, 2000). This model suggests that people who are overly concerned and fearful of their pain, will avoid activities (especially any which might trigger pain) and this may start to (or exacerbate), depression and disability, which then causes more withdrawal and avoidance and ultimately a repetitive poor functioning cycle in which pain continues to be experienced (McCracken & Morley, 2014; Smitherman et al., 2015; Vlaeyen & Linton, 2000).

It becomes clear that there are still many theories and ideas related to the cause and maintenance of PTH with little consensus empirically. There is evidence of an association between PTH and affective disorders, as well as evidence that the specific psychological mechanisms of expectations and misattributions, as well as fear avoidance, may play a role in the continuation of headaches after mTBI. As per the biopsychosocial model, the factors discussed are likely to come together for each person in a myriad of different ways, which complicates investigation. However, another way to elucidate what may be occurring is through the research on the treatment of PTH.

It appears that traditional treatment methods (including medication) for headaches are not always effective in the PTH population (DiTommaso et al., 2014; Fraser et al., 2017; Lucas, 2011; Silverberg, 2019; Watanabe et al., 2012). Thus, people have begun to investigate treatments based on the factors previously noted. For example, the efficacy of targeting fear avoidance by exposure therapy for PTH was initially investigated by Silverberg (2019). Exposure therapy is a behavioural

approach in which participants are gradually exposed to their feared stimuli. Silverberg (2019) recruited a small sample of four people with persistent headaches following mTBI. Headaches were assessed using a structured interview, alongside measures of functioning, triggers, avoidance, and symptom burden. Participants also kept a headache diary for the duration of the experiment. Each participant completed eight weeks of a behavioural exposure therapy called "learning to cope with triggers". They found uneven improvement on their selection of measures, that is, two people showed reduced headache severity and fewer days with moderate to severe headaches, but the other two showed no such improvements. They also noted improvement in one measure did not correspond with improvement in other measures, further complicating the expected hypothesis that all measures would follow a similar pattern. These mixed results could be due to the small pilot sample size, or it is possible that other mechanisms are contributing to the experience of headaches following an mTBI.

In a more well controlled, randomised control study, for those with persistent PTH following mTBI, Kjeldgaard and colleagues (2014b) ran a group Cognitive Behavioural Treatment (CBT). This included nine group sessions with 35 participants and 37 waitlisted controls. Their treatment was based on the CBT model and focused on education and changing cognitive assumptions. This was coupled with relaxation techniques, progressive muscle relaxation and breathing techniques. Interestingly, they found no improvement in headaches, nor any improvement in quality of life, distress, or overall symptoms in those who completed the treatment, but they did find some spontaneous remission in the waitlist control group. However, other studies have found the opposite effect, with improvements noted in headache severity following a CBT based treatment after mTBI (Baker et al., 2018). However small sample sizes, and lack of control groups makes these results more tentative.

These studies have outlined that there is complexity in understanding the key mechanisms occurring in PTH and therefore in understanding what treatment approach is efficacious. It is therefore worth exploring other possible mechanisms and treatment approaches in PTH following mTBI. There are some emerging ideas from the pain and primary headache literature which might provide more insights.

A treatment which has received support in the pain literature is Acceptance and Commitment Therapy (ACT) (Hughes et al., 2017; Lin et al., 2018; McCracken & Morley, 2014). ACT is a third wave therapy, following traditional behavioural therapy (first wave) and cognitive-behavioural therapy (second wave). It differs from CBT as the focus is not on changing cognitions but rather changing the function of the cognitions, and the relationship to them, through mindfulness and other processes, with the goal of living well, rather than just eliminating suffering (Hayes et al., 2006). In a recent systematic review and meta-analysis of 11 studies looking at effectiveness of ACT for pain conditions, Hughes and colleagues (2017) found medium to large effect size improvements in pain acceptance. ACT was preferred over control treatments and significant small to medium effect sizes were found for functioning, anxiety, and depression. However, no significant changes were found for pain intensity or quality of life. It is worth noting that pain reduction is not the key goal of ACT, rather it is improving functioning which is the focus (Hayes et al., 2012; McCracken & Morley, 2014).

ACT is now showing promising results in the treatment of primary headaches (Almarzooqi et al., 2017; Dindo et al., 2014; Grazzi, et al., 2019; Mo'tamedi et al., 2012; Vasiliou et al., 2021, 2022). For example, Vasiliou et al. (2022) investigated ACT with 94 participants who were broken into two groups; one which received ACT and a waitlist control group. The treatment consisted of nine weekly group sessions of 1.5 hours each and was based on the ACT model with the addition of behavioural medicine practices (e.g., sleep hygiene). Headaches were diagnosed by a neurologist and based on ICD-II criteria. They found improvements in headache related disability for the ACT group compared to the controls. They also found that this was mediated by improvements in headache acceptance and decreases in avoidance of headaches, and these improvements were maintained after three months. This provides some initial evidence that suggests ACT may be beneficial for those experiencing headaches. The mechanism which ACT targets is a transdiagnostic process called psychological flexibility.

Psychological flexibility

Generally, psychological flexibility (PF) can be defined as acting in accordance with values despite the presence of pain or distress, which results in behaviour, which is open, aware, and active (Hayes et al., 2012; Kashdan & Rottenberg, 2010; McCracken & Morley, 2014). Recently, Cherry et al. (2021) completed a scoping review of the literature and proposed that psychological flexibility should cover these three areas; a) the ability to handle interference or distress (e.g., experiencing emotional discomfort); b) taking action to manage interference or distress (e.g., engaging in an emotion regulation strategy, persisting, accepting); and c) taking action which occurs in a manner that fits situational demands and facilitates the pursuit of personal goals or values. It is this definition which we shall use going forward.

Improving PF is a core component of ACT, and this is accomplished through targeting six processes: acceptance, cognitive defusion, present focused awareness, self as an observer, values-based action, and committed action (Hayes et al., 2006, 2012; McCracken & Morley, 2014). First, *Acceptance* involves active awareness of unwanted experiences without unnecessary attempts to change them, their frequency or form, particularly when doing so would cause psychological harm and it allows people to experience events more fully (Hayes et al., 2006, 2012; McCracken & Morley, 2014). For example, headache sufferers would be encouraged to let go of their struggle against the head pain. Its opposite process is *experiential avoidance*, which is the avoidance of emotions, thoughts, sensations, memories deemed to be negative. For example, headache sufferers might avoid thoughts about their head pain, or situations which they believe will trigger headaches. *Cognitive defusion* is a process to alter the function of thoughts (or other internal events) rather than trying to alter their form, frequency, or situational sensitivity. The result of defusion is usually a decrease in believability of, or attachment to thoughts rather than an immediate change in their occurrence (Hayes et al., 2006, 2012). For instance, one may be aware of a negative belief that eating ice-cream will lead to a headache, instead of believing this inherently, one might think ‘oh that’s an interesting thought I’m having’ but does not need to believe it. Its opposite is *cognitive fusion*, whereby verbal processes

or thinking are fused with behaviour (Hayes et al., 2006, 2012). *Present focused awareness* is a process of being in the moment and having direct non-judgemental contact with what is taking place. *Self as an observer*, reflects the idea of being in the flow of one's experiences and being content with whatever might occur, it is a letting go of any labels we might put on ourselves. *Values* refers to the pursuit of the values you believe are important in life and its opposite is *failures to pursue personal values*. *Committed action* is a continuous action taken towards clear and specific goals (which are based on values) which can be achieved, even if difficulties are present (Hayes et al., 2006). A summary of the psychological flexibility model adapted by Twohig (2012) is shown in Figure 5.

Figure 5

The Psychological Flexibility Model adapted from Twohig (2012, p.502).

Theory of Psychological Flexibility

Psychological flexibility has emerged out of a philosophical science called Functional Contextualism (Hayes et al., 2006; McCracken & Morley, 2014). Functional contextualism includes looking at behaviour within the context it occurs (including situationally and historically), with a focus on the achievement of goals (Hayes et al., 2006; McCracken & Morley, 2014). A key assumption of this viewpoint is that no thought or behaviour is inherently problematic but rather it is what this leads to (or our reaction to it), that may be problematic (Twohig, 2012). PF is also based on operant (traditional behavioural approaches) and relational frame theory (RFT). RFT is an account of language and cognition and explains how we come to associate and relate different things to one another (Hayes et al., 2006; McCracken & Morley, 2014). It assumes these three things; (1) human cognition is a specific kind of learned behaviour, (2) cognition alters the effects of other behavioural processes, (3) cognitive relations and cognitive functions are regulated by different contextual features of a situation (Hayes et al., 2006). In this way PF brings together both behavioural and cognitive components within a philosophical framework (McCracken & Morley, 2014).

Measures of Psychological Flexibility

One of the most frequent measures of psychological inflexibility is a self-report questionnaire, the Acceptance and Action Questionnaire (AAQ-II), and its predecessor AAQ-I (Bond et al., 2011). This is a general measure of psychological inflexibility, and it has seven items which are scored on a five-point Likert scale (Bond et al., 2011). There has been some controversy over this measure (Cherry et al., 2021) based on its psychometric properties including its construct validity, in which people have suggested it might be a measure of psychological distress rather than inflexibility or experiential avoidance (Tyndall et al., 2019; Vaughan-Johnston et al., 2017; Wolgast, 2014). However, other studies have found that it adds explanation to the variable studied above and beyond psychological distress (Gloster et al., 2011). There are some other emerging measures of PF including the Personalized Psychological Flexibility Index (PPFI) (Kashdan et al., 2020) and the

Multidimensional Psychological Flexibility Inventory (MPFI) (Rolffs et al., 2018). However, limited research has been conducted using these two measures.

Given PF is context specific, there are also specific measures of the AAQ-II which have been developed for different populations or contexts. Ong et al. (2019) found 28 different context specific measures of the AAQ-II which cover a huge range of areas from chronic pain to body image, parenting, substance abuse and many more. These measures are beneficial as they can be more specific and therefore more likely to get at the heart of psychological flexibility for a specific issue and, as a core tenet of PF is a focus on context, this can give us more useful information (Ong et al., 2019). For those with an acquired brain injury the Acceptance and Action Questionnaire for Acquired Brain Injury (AAQ-ABI) was recently developed (Whiting et al., 2015). This measure has been demonstrated to have three factors: Denial, Active Acceptance and Reactive Avoidance (Whiting et al., 2015). However only the third factor Reactive Avoidance has shown adequate psychometric properties (Faulkner et al., 2021a; Whiting et al., 2015). Therefore, some researchers have begun using the Reactive Avoidance factor of the AAQ-ABI e.g., the AAQ-ABI (RA) in mTBI research (Faulkner et al., 2022). Reactive avoidance in this context is thought to measure acceptance and experiential avoidance, specifically with regards to a person's acquired brain injury (Whiting et al., 2015).

Psychological flexibility and mTBI

Psychological inflexibility has been shown to be related to a wide range of different heath conditions including, depression, anxiety, chronic fatigue, and eating disorders (Bluett et al., 2016; Bohlmeijer et al., 2011; Densham et al., 2016; Masuda & Tully, 2011; Scott et al., 2016). Treating psychological inflexibility through ACT has shown promise in the pain population (Bailey et al., 2010; Scott et al., 2016; Veehof et al., 2011; Vowles et al., 2020) and more recently in the TBI population (Sander et al., 2020; Whiting et al., 2020). Specifically with regards to the TBI population, Sander et al. (2020) investigated ACT in a TBI population (which included mild, moderate, and severe TBI) who were at least six months post injury. They were specifically looking for improvements in psychological distress which they measured through the Global Severity Index. They

measured PF through the Acceptance and Action Questionnaire-II (AAQ-II). With a sample size of 44 in an ACT treatment group and 49 in a control 'care as usual' group they completed an 8-week ACT programme which focused on the six core ACT processes. The results showed improvements in the ACT group on measures of psychological distress as well as improvements in psychological flexibility and committed action, which were maintained at three months. While this study focused on psychological distress, other recent research has investigated post concussive symptoms and PF specifically.

Recently, it has also been shown that psychological flexibility can help to predict PCS outcomes in a mTBI sample (Faulkner et al., 2021b, 2022). Faulkner et al. (2021b) investigated psychological flexibility in a sample of 169 participants with mTBI who had been referred to concussion clinics in New Zealand. They used a range of measures including Acceptance and Action Questionnaire (AAQ-II) and the AAQ-ABI (RA). The Rivermead Post Concussion Symptom Questionnaire was used to understand PCS symptoms, and psychological distress was measured using the Depression, Anxiety and Stress Scale (DASS), as well a functional status measure was also included. They found that psychological flexibility significantly contributed to PCS and functional status. As well as this, they also found a mediation role of psychological flexibility, in that psychological flexibility mediated the relationship between stress, anxiety, and depression, on functional status and symptoms. This provides some initial evidence of the role of psychological flexibility as a mechanism which has an influence on post concussive symptoms following mTBI.

This section has provided an overview of the concept of psychological flexibility, its theoretical foundations and how it pertains to, and modified by the third wave therapy ACT. It has also shown some emerging research in how PF may be involved with functioning after a brain injury and what measures are psychometrically sound to measure this. The next chapter outlines the present study and the associated hypotheses which this is based upon.

Chapter 3: The present study

Research indicates that psychological factors are involved in the prediction of symptoms following mTBI (Belanger et al., 2013; Broshek et al., 2015; Faulkner et al., 2021b; Hou et al., 2012; Silverberg & Iverson, 2011; van der Naalt et al., 2017). As such, current research argues that a biopsychosocial model offers the best explanation of the aetiology of post concussive symptoms (PCS) (Register-Mihalik et al., 2020; Silverberg & Iverson, 2011; Silverberg et al., 2015; Young, 2020). One of the most common symptoms experienced following mTBI is headaches (Dikmen et al., 2010; Faux & Sheedy, 2008; Langer et al., 2022; Lucas et al., 2014; Nampiaparampil, 2008; Schwedt, 2021). They are so common they have their own category in the wider headache literature, being classified as Post Traumatic Headaches (PTH) (IHS, 2018). Treating headaches effectively may help to reduce the overall symptom burden of PCS (Martelli et al., 1999). At present however, there is very mixed evidence on what can help to reduce the experience of headaches after mTBI (DiTommaso et al., 2014; Fraser et al., 2017; Lucas, 2011; Silverberg, 2019; Watanabe et al., 2012). We know that depression, anxiety, and stress are prominent following a mTBI and in those with PTH (Ham et al., 1994; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Ruff & Blake, 2016; Yilmaz et al., 2017). There is emerging evidence that we should look toward transdiagnostic processes and mechanisms that might be present in PTH (Ferrari et al., 2001; Fraser et al., 2017; Gunstad & Suhr, 2001; Mittenberg et al., 1992; Silverberg et al., 2017; Solomon, 2009; Suhr & Spickard, 2012) and that these may also underlie psychological distress following the injury. One mechanism which has shown increasing promise in both the pain and headaches literature is psychological flexibility and the treatment associated with this, Acceptance and Commitment Therapy (Almarzooqi et al., 2017; Grazzi et al., 2019; Hughes et al., 2017; Lin et al., 2018; McCracken & Morley, 2014; Mo'tamedi et al., 2012; Vasiliou et al., 2021, 2022). However, this has not been investigated specifically in a sample of people following mTBI with acute, as well as persistent headaches.

Therefore, the aim of this study is to investigate the relationship between psychological flexibility and headaches following mTBI, as well as look at the possible mediating role of

psychological flexibility on the relationship between psychological distress and headaches following the mTBI. In doing so, the treatment approaches for those with headaches following mTBI may be able to be improved, which will reduce the burden this puts on individuals, their families, and the wider community.

Questions and Hypotheses

The first aim is to explore the association between headaches and psychological flexibility for those with a mTBI. The prediction is that headache severity and psychological inflexibility will be positively associated at both acute (baseline) and persistent phases (six months follow-up). In addition, it is predicted that psychological inflexibility (immediately following the injury) will predict headache severity six months later. These have been broken down into three key questions and hypotheses.

1. Is psychological inflexibility associated with higher headache severity at baseline?

Our hypothesis is that individuals with higher levels of psychological inflexibility will experience greater headache severity immediately following mTBI (at baseline) than those with lower psychological inflexibility.

2. Is psychological inflexibility associated with higher headache severity at six months follow-up?

Our hypothesis is that individuals with higher levels of psychological inflexibility at six-months follow-up, will also have greater headache at this time point when compared with those with lower psychological inflexibility.

3. Does psychological inflexibility at baseline predict headache severity at six months follow-up?

Our hypothesis is that higher levels of psychological inflexibility at baseline will be predictive of greater headache severity at six months follow-up.

The second aim is exploring if psychological inflexibility mediates the relationship between psychological distress (depression, anxiety and stress) and headache severity in those with a mTBI.

The prediction is that higher levels of depression, anxiety and stress experienced after an mTBI are associated with higher headache severity, and this relationship will be mediated by psychological inflexibility. These have been broken down into an additional three key research questions and hypotheses.

4. Does psychological inflexibility mediate the relationship between psychological distress and headaches at baseline?

Our hypothesis is that psychological inflexibility will mediate the relationship between psychological distress and headache severity at baseline.

5. Does psychological inflexibility mediate the relationship between psychological distress and headaches at six months follow-up?

Our hypothesis is that psychological inflexibility will mediate the relationship between psychological distress and headache severity at six months follow-up.

6. Does psychological inflexibility at baseline mediate the relationship between psychological distress and headaches at six months?

Our hypothesis is that psychological inflexibility at baseline, will mediate the relationship between psychological distress and headache severity at the persistent stage.

Chapter 4: Methods

This research sits within a larger study looking at the role of psychological flexibility and mTBI outcomes. The current research uses a quantitative approach which will be further outlined in this chapter. This research was supported by Proactive Rehabilitation Concussion services and the Health Research Council of New Zealand (20-041). Ethical approval was granted on 19 February 2020 (20/32).

Participants and Procedure

Between March 2020 and September 2020 individuals were recruited through Proactive Concussion Service Clinics throughout the North Island of Aotearoa/New Zealand. Specifically, the clinics were located in the following regions: Wellington (4 clinics), Manawatu and Whanganui (2 clinics), Hawke's Bay (3 clinics), Bay of Plenty (3 clinics) and Auckland (3 clinics).

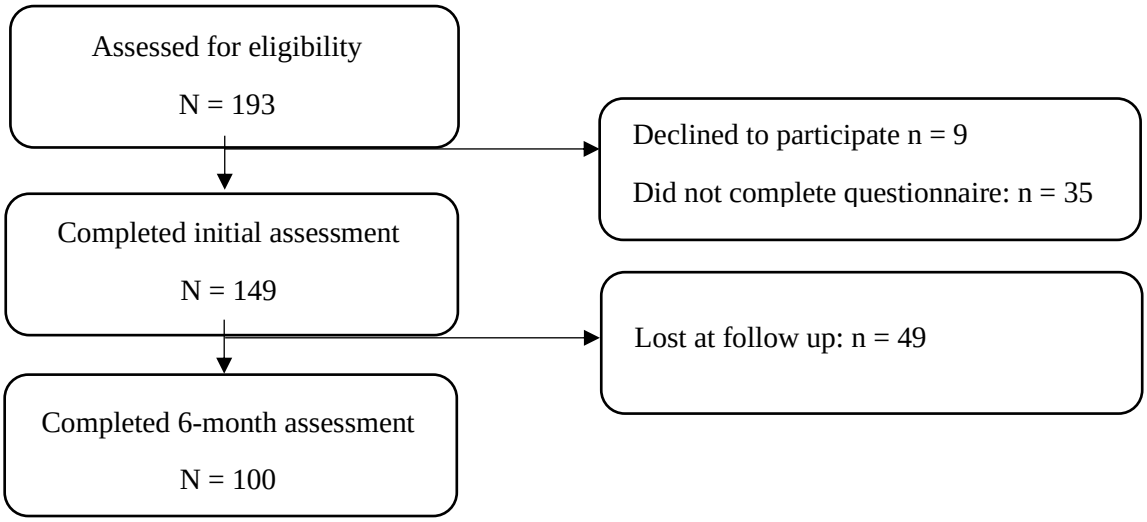
All individuals taking part in the study gave their written consent prior to beginning, and all were 18 years old or over. New Zealand has a nationwide injury insurance scheme called the Accident Compensation Corporation (ACC), and this meant all individuals automatically qualified for this scheme and thus were covered for treatment costs and any financial compensation if they were unable to engage in employment. All individuals with a history of mTBI were referred to concussive service clinics by their GP. The definition of the mTBI follows the World Health Organisation (WHO) Neurotrauma Task Force criteria which is also endorsed by ACC. This states that a mTBI is the result of an external, direct, or indirect, force that causes an altered brain function, or other evidence of brain pathology. It also states the LoC does not exceed 30 minutes and PTA is no greater than 24 hours (Menon et al., 2010). Participants were included if they were less than three months post injury and were excluded based on the following criteria: not being fluent in English, having a prior neurological condition, and/or having a severe unstable medical condition.

Upon being referred to the Proactive Concussion Service Clinics, individuals were seen by an occupational therapist (OT) for an initial assessment. All OT's had been briefed on inclusion criteria for this study and if an individual met the criteria, they provided basic information about the study along with an information sheet. If the individual was interested, consent was obtained for their details to be passed on to a member of the research team. Within four weeks of entry to the concussion service, a researcher contacted the individual to obtain formal consent to participate in the study. Eligible and consenting participants completed questionnaires via REDCap (Harris et al., 2009), a secure web-based platform, or via telephone (with data entered into the database manually by a researcher). Data collected at this time point is herein referred to as 'baseline'. Participants were then contacted and asked to complete the same questionnaires six months later; data collected at this time point will be herein referred to as 'six-month follow-up'.

A total of 193 individuals were eligible for the study. However, 9 individuals declined to participate and 35 did not complete the initial questionnaires, leaving a baseline sample of 149 participants. At six months, 49 participants were lost to follow up leaving a six-month follow up sample of 100 participants. Figure 6. shows an overview of individuals in the study at all time points.

Figure 6.

Overview of Eligible Individuals and Subsequent Participants Completing the Research at Intake and Six Months Follow-up.



Measures

Outcome Variable

Headaches

The Rivermead Post-Concussion Symptoms Questionnaire (RPQ) is a self-report measure which covers 16 of the most common symptoms following an mTBI within the past 24 hours (King et al., 1995). These are headaches, dizziness, nausea/vomiting, noise sensitivity, sleep disturbance, fatigue, irritability/easily angered, depressed/tearful, frustration/impatience, poor memory, poor concentration, slow to think, blurred vision, double vision, light sensitivity, and restlessness. They can be broken down into three categories, somatic (e.g., headaches, dizziness, nausea), cognitive (e.g., memory, concentration) and emotional/mood (e.g., feeling depressed, irritable) (Eyres et al., 2005). Each symptom is rated on a 5-point Likert scale (with higher scores indicating greater severity) as follows: 0 = not experienced at all; 1 = no more a problem than before injury; 2 = a mild problem; 3 = a moderate problem; 4 = a severe problem. This measure has shown good reliability and validity (Balalla et al., 2020; Eyres et al., 2005; King et al., 1995). Given the focus of this research we chose to look at a single item on the RPQ, "headaches", which is the first item on the RPQ. This approach has been adopted to operationalise headache severity in mTBI research (Faux & Sheedy, 2008; Silverberg, 2019). In the current research, a score of 1 ("no more of a problem than before injury") was recorded as zero, given the recommendation by King et al. (1995).

Predictor Variables

Psychological flexibility

The AAQ-ABI is a 15-item questionnaire (Whiting et al., 2015). The AAQ-ABI has a three-factor structure (factor 1 (9 items) = reactive avoidance; factor 2 (2 items) = denial; factor 3 (2 items) = active acceptance), however, factors 2 and 3 have been found to have poor psychometric properties (Whiting et al., 2015). Consequently, in the current study, only the AAQ-ABI (RA) was used, which is a context-specific measure of psychological flexibility. Specifically, it is a 9-item questionnaire covering questions to address experiential avoidance associated with reactions to having a brain injury

(Whiting et al., 2015). The AAQ-ABI (RA) uses a 5-point Likert scale (0= 'never true' to 4 = 'always true') with scores ranging from 0 to 36; higher scores indicate greater psychological inflexibility associated with an acquired brain injury. The measure has been developed and validated in an undifferentiated ABI sample (Cronbach's alpha 0.89; Whiting et al., 2015). In the present study, items within the questionnaire were modified by replacing "brain injury" with "concussion" in line with the characteristics of the sample and their engagement in an outpatient treatment programme that was referred to as a 'concussion service'. For instance, some of the items included are "I hate the way my concussion makes me feel" and "I stop doing things when I feel scared about my concussion". Faulkner and colleagues (2021a) found that the AAQ-ABI (RA), in an mTBI sample, had one distinct underlying factor with strong internal consistency (Cronbach's $\alpha = 0.87$). In line with their recommendations, in the current study, ordinal measures were converted to interval scores using their conversion table to improve the psychometric properties of the measure. It is worth noting this measure is a measure of psychological inflexibility, given higher scores indicate more inflexibility, however given the theoretical foundations of psychological flexibility it is assumed that the opposite of inflexibility is flexibility (Hayes et al., 2012; McCracken & Morley, 2014).

Reactive avoidance in this context is thought to measure acceptance and experiential avoidance, specifically with regards to a person's acquired brain injury (Sylvester, 2011; Whiting et al., 2015) and this subset of the AAQ-ABI has been used in other research (De Iorio, 2020; Faulkner et al., 2022). Reactive avoidance is deemed an adequate measure of psychological inflexibility as it captures and a rigid and inconsistent approach to dealing with a variety of situations (Rochefort et al., 2018).

The AAQ-ABI (RA) has shown adequate test-retest reliability, good internal consistency (Cronbach's $\alpha = 0.87$), good construct validity and association with general measures of psychological flexibility, as well as measures of psychological distress (although not enough to say they are measuring the same thing) (Faulkner et al., 2021a; Whiting et al., 2015).

Demographic and Clinical Variables

Demographic and injury characteristics were obtained at the beginning of the study. These included age, gender, ethnicity, education level, occupation, pre-injury employment status, relationship status, previous concussion history, prior history of medical conditions and mental health conditions. Injury characteristics included: mechanism of injury, time since injury, and if other injuries were sustained at the same time as mTBI.

Psychological Distress

The Depression Anxiety and Stress Scale-21 (DASS-21) is a self-report 21 one item measure of anxiety, depression and stress and has three corresponding subscales, with seven items in each (Antony et al., 1998; Lovibond & Lovibond, 1995). Each item is rated on a 4-point Likert Scale about the extent to which each item has been experienced over the past week, ranging from did not apply at all, to, applied all the time, and therefore higher scores indicate greater distress. The anxiety subscale relates mostly to feelings of hyperarousal e.g., panic attacks and fear and includes items such as, "I experienced difficulty breathing" and "I was worried about situations in which I might panic and make a fool of myself". The depression subscale looks at feelings of sadness, hopelessness, and unworthiness. An example of the items includes "I felt that life was meaningless" and "I felt that I had lost interest in just about everything". Lastly the stress subscale looks at tension and irritability as this relates to feeling stressed (Antony et al., 1998), for example "I was in a state of nervous tension" and "I found that I was very irritable". This measure is not intended to correspond to diagnosis of depression, anxiety or stress but instead provides a quick understanding of the levels of distress someone is experiencing in these three areas (Lovibond & Lovibond, 1995; Randall et al., 2017).

With regards to the psychometric properties of this measure, internal consistency has been shown to be high, with a Cronbach's alphas of 0.94 for Depression, 0.87 for Anxiety, and 0.91 for Stress (Antony et al., 1998). Concurrent validity was also good when compared to other similar measures (Antony et al., 1998; Lovibond & Lovibond, 1995). This is a measure commonly used in research and clinical settings (Crawford & Henry, 2003) and a recent meta-analysis has shown support for the three-factor model (Yeung et al., 2020), however it is interesting to note that they also found evidence of an underlying general psychopathology which may be present across all three

factors (Yeung, et al., 2020). This general factor has also been found in other research in the general population, alongside confirming the presence of the three-factor model (Henry & Crawford, 2005).

DASS-21 has also been investigated in the TBI population and has shown suitable psychometrics in this population (Randall et al., 2017). Randall et al. (2017) in a sample of moderate to severe TBI found that a quadripartite model fitted the best. This aligns with what Henry and Crawford (2005) found, three factors plus a general factor (psychological distress). They also confirmed high internal consistency of the DASS-21 in this population (Randall et al., 2017). Research has also been completed with a mTBI sample. Dahm et al. (2013), looked at how well the DASS and Hospital Anxiety and Depression Scale (HADS) could screen for mood disorder in 123 participants, with TBI's ranging from mild to severe. They found good psychometrics including, high concurrent validity between these two measures (both $R = 0.76$, $p < 0.001$), high internal consistency along with good specificity and sensitivity. These studies provide assurance that it is suitable to use the DASS-21 in the mTBI population as a measure of distress.

Data Analysis

For all statistical analyses the Statistical Package for Social Sciences (SPSS) Version 25 was used with a significant level set at .05 to signify statistical significance. Descriptive analyses were conducted to broadly characterise the sample (age, gender, ethnicity, education, medical and mental health history, injury characteristics, pre-injury employment and relationship status) and the outcome measures (headache severity, psychological flexibility, psychological distress). Descriptive statistics are reported as numbers and percentages for categorical variables, means and standard deviations for continuous variables.

To examine the role psychological flexibility on headache severity, linear regression analyses was used. This first involved an unadjusted linear regression model to examine the relationship between psychological flexibility and headache severity. An adjusted model was then used to include clinical and demographic variables known to influence headache severity or PCS following mTBI. These variables were selected a priori based on empirical evidence in accordance with the

recommendations of Sun and colleagues (1996). This included age (Iverson et al., 2017; van der Naalt et al., 2017; Yilmaz et al., 2017), gender (Hoffman et al., 2011; Iverson et al., 2017; Yilmaz et al., 2017), education (Snell et al., 2011; van der Naalt et al., 2017), concussion history (Hoffman et al., 2011), mental health history (Fraser et al., 2017; Iverson et al., 2017; Jouzdani et al., 2014; King, 1996; MacMillan et al., 2002; Meares et al., 2008; Ponsford et al., 2000), medical history (Lieba-Samal et al., 2011; Stulemeijer et al., 2008), other injury experienced (Stulemeijer et al., 2006) and mechanism of injury (Booker et al., 2019). In addition, the severity of psychological distress was also included in the adjusted model (Ham et al., 1994; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Ruff & Blake, 2016; Yilmaz et al., 2017). These analyses were computed at baseline (aim 1) and at six months follow up (aim 2). In addition, in accordance with aim three, the regression analyses were also repeated to examine the relationship between psychological flexibility at baseline and headache severity at six-month follow up.

A power analysis using the G*power software was used to ensure adequate sample size for the regression analysis. Assuming a medium effect size (*f2*) of 0.15, an α of 0.80, and a maximum of 8 predictors, power analysis suggested a minimum sample of approximately 100 participants (Faul et al., 2009).

Next, meditation analysis was used, given the role of psychological flexibility as a mechanism that exerts an influence on the relationship between two variables (psychological factors and recovery outcomes) (MacKinnon et al., 2007). PROCESS for SPSS (Hayes, 2012) was used to conduct a series of mediation analyses to evaluate the indirect effects of psychological flexibility on the relationship between the predictor (psychological distress) and the dependent variable (headache severity). We estimated the individual effect of each dependent variable on each mediator (i.e., psychological distress, AAQ-ABI(RA); path A), the effect of each mediator on the predictor variables (i.e., AAQ-ABI(RA), headache severity; path B), and the indirect effect of the dependent variables through each of the individual mediators (path A x B). We compared the direct effect (path C'; the effect of the dependent variable on the predictor variable minus each mediator) and the total effect (path C; the effect of the dependent variable on the predictor variable plus each mediator). We assessed the

magnitude of all mediation effects (paths A x B) by the standardized beta coefficients. We assessed the reliability of the sample mediation effects via the percentile (nonparametric) 95% confidence interval (CI) generated by a bootstrapping procedure with a resample rate of 5,000 to avoid inflation of type 1 error rate (Preacher & Hayes, 2008). Bootstrapping is a computational nonparametric resampling technique that enables population characteristics to be estimated from the existing sample (Mooney et al., 1993). We report variance in headache severity that is explained by each mediation model (overall R^2). Again, these analyses were computed at baseline (aim 1) and at six months follow up (aim 2). A third meditation analysis was also done to examine the indirect effects of psychological flexibility at baseline on the relationship between psychological distress and headache severity at six months.

Chapter 5: Results

Participants

At clinic intake the ages of participants varied from 19 to 69 years of age, with a mean of 35.26 year (SD = 12.669). The majority were female (66.4%) and NZ European (63.8%). Most people had a university education (67.1%), and majority were also in employment (83.2%). Regarding injury characteristics, most injuries were due to a fall (43%) and over half experienced another injury as well as the mTBI (59.1%). Lastly nearly half had a history of mental health diagnoses (48.3%). There were no statistically significant differences between the demographic, clinical, and injury characteristics at clinic intake and those at six-months follow-up. At clinic intak the time since injury ranged from between 2 to 12 weeks with the average being 5.91 weeks (SD = 2.291). At six-months follow-up the time since injury increased with the range from 28 to 40 weeks with a mean of 32.55 weeks (SD = 2.84756).

Table 2.

Demographic, Clinical and Injury Factors for the mTBI Sample at Clinic Intake and Six Months Follow-up

Demographic characteristics	Clinic Intake (n=149)	Frequency (%)	6 month follow up (n=100)	Frequency (%)
Gender				
Female	99	66.4	71	71
Make	50	33.6	29	29
Ethnicity				
Māori	18	12.1	9	9
NZ European	95	63.8	68	68
Other	36	24.2	23	23
Age				
18-29	67	45	40	40
30-45	46	30.9	36	36
45+	36	24.2	24	24

Education				
High School	49	32.9	29	29
University	100	67.1	71	71
Employment				
Working	124	83.2	82	82
Not working	25	16.8	18	18
Relationship				
In a relationship	91	61.1	62	62
Single	58	38.9	38	38
Mechanism of injury				
Motor vehicle accident	17	11.4	8	8
Fall	64	43	45	45
Assault	13	8.7	6	6
Other	55	27.5	41	41
Other injury				
Yes	88	59.1	60	60
No	61	40.9	40	40
History of concussion				
Yes	62	41.6	40	40
No	87	58.4	60	60
Medical history				
Yes	47	31.5	37	37
No	93	62.4	63	63
Missing	9	6	0	0
Mental health history				
Yes	72	48.3	49	49
No	77	51.7	51	51
Time since injury				
Less than 8 weeks	130	87.2		
8 weeks of more	19	12.8	100	100

Study Variables

The descriptive statistics of the study variables both at baseline and six months follow-up are outlined below in Table 3.

Table 3.

Descriptive Statistics of Study Variables at Baseline and Six Months Follow-up

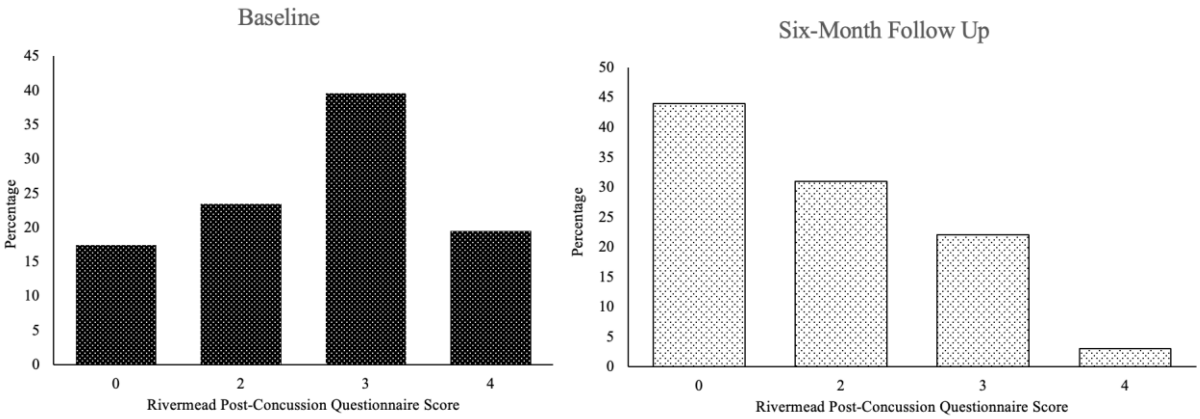
	Baseline (n=149)			Six Months (n=100)		
	Mean	Standard Deviation	Range	Mean	Standard Deviation	Range
Headache severity	2.44	1.30	0.00-4.00	1.41	1.32	0.00-4.00
Post concussion symptoms	31.79	13.59	0.00-64.00	18.75	11.81	0.00-54.00
Psychological flexibility	10.21	3.50	0.00-22.00	7.03	4.15	0.00-19.11
Psychological distress	21.50	14.49	0.00-66.00	19.98	13.93	0.00-64.00

Headache Severity Frequency

As shown in Figure 7, at baseline headaches were prevalent and were most likely to be rated as “a moderate problem” (39.6%) and then “a mild problem” (23.5%); “a severe problem” was present in 19.4% of the sample, with “not experienced at all” the least common (17.4%). This pattern was different at six months follow-up. Here, “not experienced at all” was the most common (43.0%), followed up a “a mild problem” (31.0%), and then a “moderate problem” (22.0%). At six-months follow-up, “a severe-problem” was only evident in 3.0% of the sample.

Figure 7.

Percentage of Headache Severity Endorsement based on the Rivermead Post-Concussion Questionnaire at Baseline and Six-Months Follow-up



Correlation Coefficients

Table 4 describes the correlations coefficients for the outcome and predictor variables at baseline and six-months follow-up.

Table 4.

Correlation Coefficients at Baseline and Six Months Follow-up

Baseline				
	Headaches	Psychological Flexibility	Psychological Distress	Post-Concussion Symptoms
Headaches		.335*	.309*	.677**
Psychological flexibility			.621**	.575**
Psychological distress				.649**
Six Months				
	Headaches	Psychological Flexibility	Psychological Distress	Post-Concussion Symptoms

Headaches	.284*	.174	.534**
Psychological flexibility	.	379**	.592**
Psychological distress			.403**

Multiple Linear Regression

Assumption Testing

The assumption of multicollinearity was assessed by examining the correlations between each of the predictor variables. Preliminary examination revealed correlations below the recommended .70 threshold as suggested by Pallant (2013). Additionally, analyses revealed that all tolerance values and variance inflation factors (VIF) for each of the predictor variables were above the recommended .10 threshold for tolerance, and below the recommended value of 10 for VIF (Pallant, 2013), thus meeting the assumption of multicollinearity. The assumption of independent errors was met as the Durbin-Watson test revealed a value of 2.20, indicating a lack of autocorrelation. Examination of scatterplots, histograms, and P-P plots were consistent with assumptions of homoscedasticity, linearity, and normality. Thus, the results of assumption testing support the use of multiple linear regression analyses.

Baseline

To explore the relationship between psychological flexibility and headache severity at baseline a multiple liner regression model was conducted. The unadjusted model was significant ($F(1,147) = 18.57$, $p < .01$, $R^2 = .11$). As shown in Table 5, in this model, psychological flexibility was significantly associated with headache severity at baseline ($\beta = 0.12$, $p < .001$). In the adjusted model, variables known to exert an influence on mTBI outcomes were included. The model, with gender, age, previous concussions, mechanism of injury, mental health, medical history, and psychological distress was significant ($F(7,141) = 2.67$, $p = .007$, $R^2 = .16$). As shown in Table 5, in this model, only psychological flexibility predicted headache severity at baseline ($\beta = 0.11$, $p = .01$).

Table 5.

Coefficients of Predictor Variables in the Unadjusted and Adjusted Linear Regression Models on Headache Severity at Baseline

	β	**SE**	***p***
Unadjusted Model			
Psychological flexibility at baseline	0.12	0.03	<.001
Adjusted Model			
Gender	0.10	0.28	.67
Age	-0.01	0.01	0.13
Concussion history	0.20	0.23	0.38
Medical history	-0.15	0.24	0.53
Mental health history	0.11	0.23	0.64
Mechanism of injury	-0.02	0.09	0.83
Psychological flexibility at baseline	0.11	0.04	0.01*
Psychological distress at baseline	0.01	0.01	0.48

*Significance p =<0.05

Six Months Follow-Up

To explore the relationship between psychological flexibility and headache severity at six months, linear regression analyses was also conducted. The unadjusted model was significant ($F(1,99) = 9.27$, $p = .003$, $R^2 = 0.08$). As shown in Table 6, psychological flexibility was significantly associated with headache severity ($\beta = 0.06$, $p = .003$). The adjusted model was also significant ($F(7,99) = 2.23$, $p = .040$, $R^2 = 0.14$). As shown in Table 6, in this model, psychological flexibility ($\beta = 0.06$, $p = .014$), and gender ($\beta = 0.59$, $p = .042$) predicted headache severity at six month follow up.

Table 6.

Coefficients of Predictor Variables in the Unadjusted and Adjusted Linear Regression Models on Headache Severity at Six Months Follow-up

	β	**SE**	***p***
Unadjusted Model			
Psychological flexibility at six-month follow-up	0.06	0.2	.003
Adjusted Model			
Gender	0.59	0.29	.042*
Age	-0.15	0.01	.177
Concussion history	-0.10	0.27	.710
Medical history	-0.09	0.29	.765
Mental health history	0.26	0.27	.349
Psychological flexibility at six-month follow up	0.06	0.02	.014*
Psychological distress at six-month follow up	0.01	0.01	.503

*Significance p =<0.05

Psychological Flexibility at Baseline on Headaches at Six Months

Finally, the predictive role of psychological flexibility at baseline on headache severity at six months was also examined using linear regression. The unadjusted model was significant ($F(1,99) = 18.13$, $p < .001$, $R^2 = 0.16$) with psychological flexibility at baseline significantly associated with headache severity at six months follow-up ($\beta = 0.15$, $p = .001$). The adjusted model was also significant ($F(7,93) = 3.32$, $p = .003$, $R^2 = 0.20$). As shown in Table 7, in this model, only psychological flexibility at baseline predicted headache severity ($\beta = 0.14$, $p = .007$) at six-months follow-up.

Table 7.

Coefficients of Predictor Variables in the Unadjusted and Adjusted Linear Regression Models with Psychological Flexibility at Baseline on Headache Severity at Six-Months Follow-up

	β	**SE**	***p***
Unadjusted Model			
Psychological flexibility at baseline	0.15	0.04	<0.001
Adjusted Model			
Gender	0.51	0.28	.074
Age	-0.01	0.01	.232
Concussion history	0.08	0.26	0.76
Medical history	-0.05	0.27	.848
Mental health history	0.34	0.27	.848
Psychological flexibility at baseline	0.14	0.05	.007*
Psychological distress at six month follow-up	0.01	0.01	.548

*Significance p =<0.05

Summary

Multiple linear regression analysis demonstrated that psychological flexibility made a significant and unique contribution to headache severity at both the baseline and six-months follow-up, with no other variable making a significant contribution, aside from gender making a significant and unique contribution to headache severity at six months. As well as this, psychological flexibility at baseline predicted headache severity at six-months follow-up. Each model showed between 16-20% of the variance in headaches was accounted for by the significant predictors.

Mediation Analysis

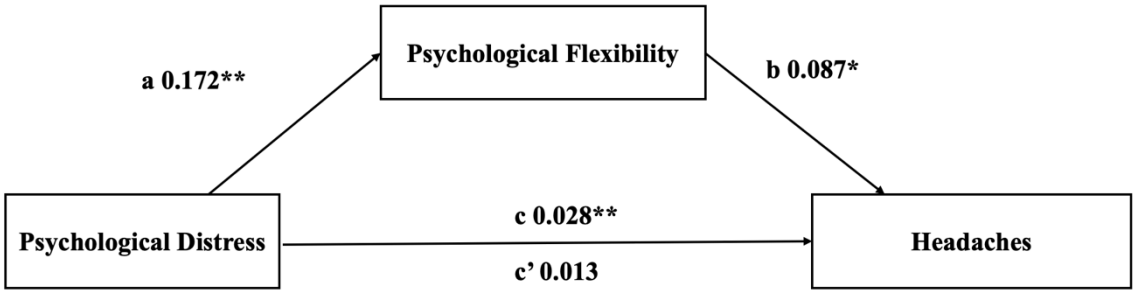
The bootstrapped method (PROCESS) with *n* = 5000 bootstrap resamples and 95% bias corrected and accelerated confidence intervals was used in a series of analyses to examine the indirect effects of psychological flexibility (M_1 = AAQ-ABI (RA)) on the relationship between psychological distress and outcomes (headache severity).

Baseline

Psychological distress at baseline had a total effect on headaches, β = 0.028, SE = 0.007, p<.001, 95% CI [0.014,0.042]. As shown in Figure 8, in this model, on each respective path, the AAQ-ABI (RA) illustrated significant coefficients in both a and b paths. There was a significant indirect effect of psychological distress on headaches through psychological flexibility, β = 0.015, SE = 1.850, p<.001, 95% CI [0.009,0.032], however, there was no significant direct effect, β = 0.013, SE = 0.001, p=.196, 95% CI [-0.001,0.030]. Approximately 12% of the variance in headaches was accounted for by the predictors (R^2 = 0.12).

Figure 8.

Mediation Analysis of Psychological Flexibility on the Relationship between Psychological Distress and Headaches at Baseline

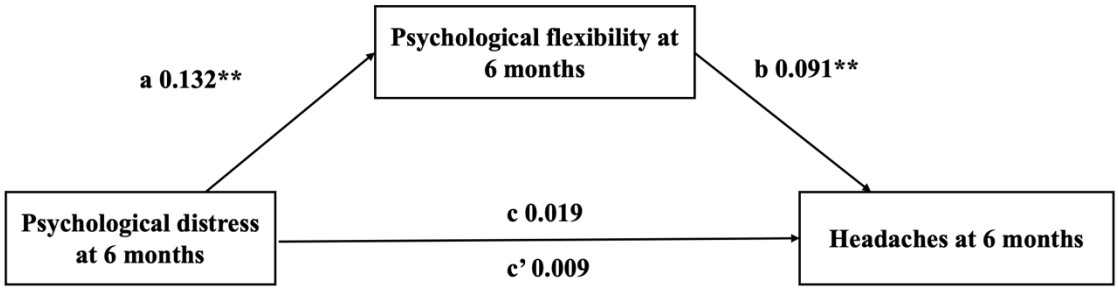


Six Months Follow-up

At six months, there was no significant total effect of psychological distress on headaches, β = 0.019, SE = 0.011, p=.083, 95% CI [-0.003,0.041]. As shown in Figure 9, in this model, on each respective path, the AAQ-ABI (RA) illustrated significant coefficients in both a and b paths. There was a significant indirect effect of psychological distress on headaches through psychological flexibility, β = 0.011, SE = 0.005, p<.001, 95% CI [0.015,0.023], however, there was no significant direct effect, β = 0.009, SE = 0.012, p=.461, 95% CI [-0.015,0.032]. Approximately 9% of the variance in headaches was accounted for by the predictors (R^2 = 0.09).

Figure 9.

Mediation Analysis of Psychological Flexibility on the Relationship between Psychological Distress and Headaches at Six-Months Follow-Up

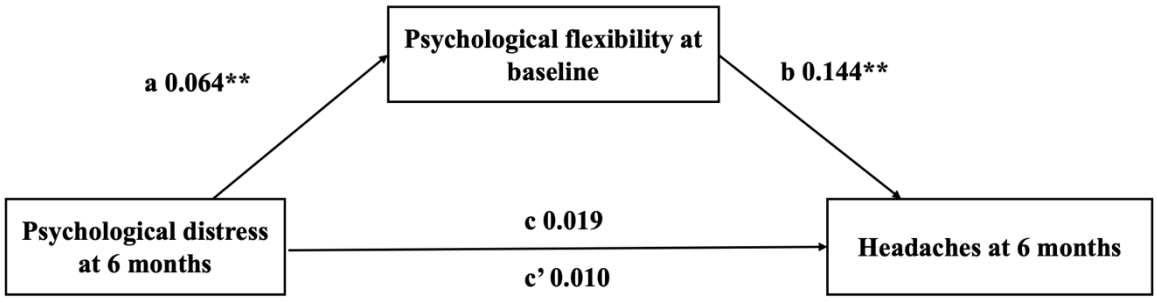


Psychological Flexibility at baseline on the relationship between psychological distress and headaches at six months.

As shown in Figure 10, in this model, on each respective path, the AAQ-ABI (RA) illustrated significant coefficients in both a and b paths. There was a significant indirect effect of psychological distress on headaches at six months through psychological flexibility at baseline, β = 0.009, SE = 0.005, p<.001, 95% CI [0.013,0.019], however, there was no significant direct effect, β = 0.011, SE = 0.011, p=.461, 95% CI [-0.011,0.031]. In this model, only 3% of the variance in headaches was accounted for by the predictors (R^2 = 0.03).

Figure 10.

Meditation Analysis of Psychological Flexibility at Baseline on the Relationship between Psychological Distress and Headaches at Six-Months Follow-Up



Summary

The mediation analysis showed an indirect effect of psychological distress on headache severity through psychological flexibility. This was confirmed at all time periods: baseline, six months and psychological flexibility at baseline and psychological distress and headaches at six months. It is worth noting that the relationship between psychological distress and headache severity was not significant in any three time periods except at baseline when psychological flexibility was excluded. However, when psychological flexibility was included in the model there was a significant indirect relationship between psychological distress and headache severity through psychological flexibility at all three time periods.

Chapter 6: Discussion

The first aim of this study was to understand the role of psychological flexibility on headaches following mTBI, with the hypothesis that individuals with lower psychological flexibility (i.e., higher inflexibility) would have greater headache severity. We investigated this over two time periods, baseline, within several weeks of sustaining a mTBI and at six months follow-up, when headaches could be classified as persistent. We also investigated if psychological flexibility at baseline was predictive of headache severity at six-months follow-up. The results support our three hypotheses. Specifically, it was shown that those with lower psychological flexibility had greater headache severity at baseline and at six months follow-up. Additionally, it was also shown that lower psychological flexibility at baseline was predictive of higher headache severity at six-month follow-up. These findings remained even after factors known to exert an influence on mTBI were included in the model. These three analyses showed that between 16-20% of the variance in headache severity was due to the significant predictors.

The second aim of this study was to understand the possible mediating role of psychological flexibility on the relationship between psychological distress and headache severity. There is evidence that psychological distress is often present after an mTBI and for those with headaches (Ham et al., 1994; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Ruff & Blake, 2016; Yilmaz et al., 2017). However, the specific mechanism which accounts for this, is still in debate (Ferrari et al., 2001; Fraser et al., 2017; Gunstad & Suhr, 2001; Mittenberg et al., 1992; Silverberg et al., 2017; Solomon, 2009; Suhr & Spickard, 2012). One possiblity is psychological flexibility which is known to underlie and exacerbate psychological distress. As such we predicted that psychological flexibility would mediate the relationship between psychological distress and headache severity. The results support all our three hypotheses. Specifically, psychological flexibility was found to mediate the relationship between psychological distress (as measured through the DASS-21) and headache severity. This finding was evidence at baseline, six months follow-up and through the relationship between

psychological flexibility at baseline and psychological distress and headache severity at six months follow-up. These results will now be discussed in greater detail.

Hypothesis one: Higher psychological inflexibility will be associated with higher headache severity at baseline

Consistent with this hypothesis, this study found that at baseline (on average 5.9 weeks (SD = 2.291)) post mTBI, individuals with higher psychological inflexibility had significantly higher headache severity. These results were still statistically significant when accounting for other variables known to influence post concussive symptoms. This supports other research which has found that psychological factors have an influence on the severity of post-concussion symptoms immediately following the mTBI (Belanger et al., 2013; Broshek et al., 2015; Faulkner et al., 2021b; Hou et al., 2012; King et al., 1999; Silverberg & Iverson, 2011; van der Naalt et al., 2017).

This finding also supports the views of Silverberg and Iverson (2011), in their argument against Lishman's (1988) original article on the aetiology of post concussive symptoms (PCS). Lishman's original article stated that psychological factors were only present several months post mTBI, with biological factors accounting for the symptoms earlier, post injury. Silverberg and Iverson (2011) highlighted several studies which showed that psychological factors, specifically psychological distress was associated with PCS, early on following mTBI (King et al., 1999; Meares et al., 2011; Snell et al., 2011). The current study adds further support to this research, showing that several weeks post injury, psychological factors, in this case, specifically psychological flexibility, is present and is associated with one PCS symptom, headache severity.

These findings also provide support for the biopsychosocial model of PCS and headaches (Andrasik et al., 2005; Register-Mihalik et al., 2020). This model states that several biological, psychological, and social factors all combine to influence the experience of symptoms after a mTBI. In this case psychological flexibility at baseline (or the acute phase) accounted for 16% of the variance in headache severity in the adjusted model, where several other factors were considered. This leaves 84% unaccounted for. However, this is understandable within the biopsychosocial model, as biological and social factors, and other psychological factors, are likely to be contributing to the

unaccounted-for variance. For example, as noted earlier the neurometabolic changes may still be exerting an effect through their effects on the trigeminovascular system (Ashina et al., 2019; Packard, 1999; Schwedt, 2021) which may account for part of the ongoing experience of headaches. Coupled with this are social factors, for instance, the amount of care and concern partners have of their spouses has been found to result in increased pain perception (Flor et al., 1995).

These findings also support the primary headache literature outside of the mTBI context, which has shown emerging evidence that lower psychological flexibility is related to higher headache severity, disability, and distress (Dindo et al., 2014; Foote et al., 2016; Mo'tamedi et al., 2012). Specifically, Foote et al. (2016) found that in 103 adults who suffered from migraines, that acceptance of pain and values-based action, two of the elements of psychological flexibility, accounted for 10% of the variance in headache severity and 20% of headache-related disability. This is a similar degree of variance to the current research, suggesting psychological flexibility may be influencing both post traumatic headaches and primary headaches in similar ways to maintain the experience of headaches.

Our results are consistent with previous research in mTBI that illustrates that specific psychological factors contribute to headaches (Ferrari et al., 2001; Fraser et al., 2017; Gunstad & Suhr, 2001; Mittenberg et al., 1992; Silverberg et al., 2017; Solomon, 2009; Suhr & Spickard, 2012). One of these factors is fear avoidance, which is important to distinguish from the avoidance that can be part of psychological flexibility. In this study psychological flexibility was measured through a context specific measure, the AAQ-ABI (RA) which is a measure of psychological flexibility for those with acquired brain injury. In this study, only the Reactive Avoidance factor was used due to poor psychometric properties of the other two factors. In this context reactive avoidance captures acceptance and experiential avoidance as it relates to peoples' feelings about their concussion (Sylvester, 2011; Whiting et al., 2015). Related to reactive avoidance is fear avoidance. Fear avoidance, as noted earlier, has been described by Vlaeyen and Linton, (2000), as comprising four main steps, beginning with negative thoughts about pain; this causes avoidance of activities which might bring on pain, this results in reduced or no pain, which self-reinforces the avoidance behaviour. If this occurs over time it can have a detrimental impact on physical functioning (Vlaeyen & Linton,

2000). Reactive avoidance, on the other hand, is focused more on the internal sensations which are avoided, so people will avoid distressing thoughts and feelings. It is when this reactive avoidance is applied rigidly, that is, it is applied in many different contexts and nearly every time distressing thoughts/feelings appear, that this becomes psychological inflexibility (Hayes et al., 2012). It is important to understand the differences between these concepts but also to acknowledge that they may both present for individuals and can occur together in self-reinforcing ways.

To understand how psychological flexibility may influence headache severity, two possible pathways need to be considered. First, people with higher degrees of inflexibility or avoidance, may start avoiding thoughts and feelings associated with their head injury. We know that headaches are a symptom often present immediately following a mBTI (Anderson & Fitzgerald, 2020), so thoughts and feelings about headaches are likely to be present early on. If these thoughts and feelings cause distress, attempts will be made to avoid such thoughts and feelings, thus reducing distress in the short term (Hayes et al., 1996). When this avoidance strategy is applied rigidly (in the case of lower flexibility), there may be a greater degree of distress when they do appear, this distress then exacerbates the severity of the headache e.g. "I worry my headaches will not go away, every time I have this thought I worry more and try to think about something else, then I get a headache and of course it's really bad". It is also possible that those with lower flexibility interpret their pain as being more severe, as has been shown to occur in those with higher experiential avoidance (Chawla & Ostafin, 2007).

Second, another related possibility, is that psychological inflexibility results in rigid attempts to avoid the physical internal sensations of a headache (e.g., attempting to pretend one does not have a headache when one does). These attempts at avoidance often include thinking about the issue itself, e.g., "I can't think about my headache" includes thinking about headaches. This often results in a greater emphasis on headaches (or anything you are attempting to avoid) (Clark, et al., 1991; Hayes et al., 1996). When this avoidance is applied rigidly it begins to restrict the degree to which difficult thoughts or sensations are experienced; therefore, when these thoughts and sensations do arise there is a reduced ability to deal with them (Morris & Mansell, 2018).

Hypothesis two: Higher psychological inflexibility will be associated with higher headache severity at six months follow-up

Consistent with this hypothesis, this study found at six months follow-up (on average 32.55 weeks (SD = 2.84756) post mTBI), individuals with higher psychological inflexibility had significantly higher severity headaches. Also significant in this model was gender, with females having higher severity headaches. These results were still statistically significant when accounting for all other variables known to influence post concussive symptoms. This supports other research which has found that psychological factors have an influence on the severity of post-concussion symptoms in the long term (Belanger et al., 2013; Broshek et al., 2015; Faulkner et al., 2022; Hou et al., 2012; Silverberg & Iverson, 2011; Silverberg et al., 2015; van der Naalt et al., 2017).

It is also important to know that at this six-month follow-up timepoint, the prevalence of people experiencing any headache was 56% and while this has tapered off from baseline, it is still a significant number of people. It is comparable to other research findings, for example, a systematic review in 2008 found that approximately 57% of people experienced a chronic headache after mTBI (Nampiaparampil, 2008). It is also comparable to research which has indicated a higher prevalence of persistent of overall PCS than has been previously thought (Barker-Collo et al., 2013; Dikmen et al., 2017; Nelson et al., 2019; Theadom et al., 2016).

This finding adds to the wider literature on PTH as previous studies have either not looked specifically at the psychological factors related to PTH, instead focusing on primary headaches (French et al., 2000; Gunstad & Suhr, 2001; Mittenberg et al., 1992), or they have found inconclusive evidence when treating the mechanisms believed to underlie headache severity in the longer term, e.g., through CBT or exposure therapy (Kjeldgaard et al., 2014b; Silverberg, 2019).

However, at the six-month timeframe, psychological flexibility was shown to account for 17% of the variance in headache severity. This is still not a large amount of the variance, but interestingly, this is very similar to the amount of variance at baseline which points to the stability of psychological factors over time. Previous research has both supported (Anderson & Fitzgerald, 2020;

Snell et al., 2011) and negated the stability of factors over time (Meares et al., 2011). Snell et al. (2011) found very similar rates of psychological distress over time, however Meares et al. (2011) found an increasing association between PTSD and PCS over time. Our results suggest the stability of psychological flexibility in this population. This aligns with the rigidity concept of psychological flexibility, in that the avoidance strategies are likely to continue being applied by the individual unless there are attempts to change these (Hayes et al., 2012).

This finding also confirms our previous discussion about the biopsychosocial model, but this can now be applied in the context of persistent symptoms (Andrasik et al., 2005; Kenzie et al., 2017; Register-Mihalik et al., 2020). It is also possible that PF is co-occurring alongside other psychological mechanisms. Other research investigated different transdiagnostic factors for persistent PCS, for instance, Hou et al. (2012) looked at all or nothing behaviour and negative mTBI perceptions. They found that at six months, the most important factor was negative mTBI perceptions. It is possible that this could also occur alongside low psychological flexibility, and both mechanisms could be working alongside each other and be mutually reinforcing. For instance, it is possible that people have negative perceptions about their functioning following mTBI, this in turn could also manifest into a greater degree of concern about their symptoms, which leads to greater avoidance, and this is also maintained by the behavioural rigidity associated with lower PF. Thus, our results can be viewed alongside other evidence to start to build a fuller additive picture of what may be occurring with persistent headaches after mTBI.

Our results also add support to the literature on chronic pain and psychological flexibility. Generally, pain is considered chronic if it persists for six months or more (Russo & Brose, 1998). Studies have shown that psychological flexibility is predictive of chronic pain (Gentili et al., 2019; McCracken & Velleman, 2010; Vowles & McCracken, 2010). McCracken and Velleman (2010) found that in a sample of 239 participants in primary care, psychological flexibility accounted for on average 24% of the variance in physical, emotional, and social functioning along with healthcare use which was higher than the variance accounted for by pain intensity (on average 9.2%). While this was not an mTBI or headache sample, head pain and chronic pain are both pain-related conditions and

could be considered to share some similarities (Snell et al., 2018). Given these similarities is possible that PF is contributing in similar ways to both types of pain.

To understand further how headaches may be maintained chronically, we can turn back to the avoidance pattern described earlier. At this stage, the rigid way of responding has had time to embed and become a habit, alongside avoidance becoming a reinforcing negative feedback loop (Cassetta et al., 2021; Vlaeyen & Linton, 2000). For example, it is likely that individuals with high psychological inflexibility continue their pattern of avoiding emotions, thoughts and experiences associated with a headache. This may lead to a greater severity headache or a heightened focus on headaches over time. This in turn creates a greater degree of avoidance, and the pattern continues in a continuous feedback loop. The person may be unable to see another way of responding or is unaware of what is occurring. This is just an example of how PF may manifest with headaches, noting that given the different components of PF may come together for specific individuals in different ways (McCracken & Velleman, 2010).

Alongside PF being a significant predictor, at this six-month timeframe gender was also a significant predictor for headache severity. Specifically female gender was predictive of higher severity headaches. This has been shown in other mTBI research (Hoffman et al., 2011; Iverson et al., 2017; Yilmaz et al., 2017). There is also evidence that females have a larger number of headaches in the general population, specifically a greater degree of primary migraine headaches (Al-Hassany et al., 2020). Although the reasons for these gender differences are varied, one explanation is that this is due to hormonal influences (Al-Hassany et al., 2020). It is therefore plausible that headaches occurring after mTBI are due to the same cause but are misattributed as being related to the mTBI, in accordance with the base rate misattribution theory noted earlier (Gunstad & Suhr, 2001; Mittenberg et al., 1992). Other possible causes of this discrepancy have been noted such as females having weaker neck muscles, and females being more likely to report symptoms, especially in relation to sports related injuries (Dick, 2009; Iverson et al., 2017). Further research is needed to understand what is occurring here.

Hypothesis three: Higher psychological inflexibility at baseline will be result in higher headache severity at six months follow-up

Our findings supported the hypothesis that psychological inflexibility, measured at baseline, was predictive of headache severity at six months follow-up. That is, individuals with higher psychological inflexibility at baseline had greater severity headaches six months later. No other variables know to influence PCS were statistically significant in this model. In this analysis psychological inflexibility at baseline was shown to account for 20% of the variance in headache severity six months later. These findings are consistent with other research which has found that psychological factors immediately following an mTBI have an influence on the severity of post-concussion symptoms in the long term (Broshek et al., 2015; Faulkner et al., 2022; Hou et al., 2012; King et al., 1999; Silverberg & Iverson, 2011; van der Naalt et al., 2017).

The amount of variance is similar to the other two time periods discussed, with this being slightly higher, but it is comparable to other research in overall PCS. For instance, King et al. (1999) specially looked at 57 people with mild and moderate mTBI and found that three main factors were the best predictors of PCS severity six months after the injury; these included anxiety and depression (as measured by HADS), RPQ (at 7-10 days post injury) and PTA, accounting for 23% of the variance in symptoms at six months follow-up. Therefore, our amount of variance appears comparable to other research and may be indicative of the degree of influence psychological factors have on persisting symptoms after mTBI.

The factors noted by King et al., (1999) are also broad factors and our research has attempted to understand more deeply how these factors may exert their influence by looking at a transdiagnostic mechanism. Other studies have attempted to do the same and have found an influence of baseline factors on longer term outcomes (Snell et al., 2011). For example, similar findings were shown by Snell et al., (2011) who looked at the role of illness perceptions and coping on mTBI recovery. They found that unhelpful injury perceptions initially (e.g., beliefs that symptoms were attributed to the injury), and the severity of expected consequences, contributed to significantly worse outcomes six months later, as did measures of depression and anxiety. While these are different psychological

mechanisms, again it is possible they are also present alongside psychological flexibility, as per the biopsychosocial model, and that some of these may be mutually reinforcing.

Our finding also supports previous research which has looked at the long-term consequences of flexibility but in relation to psychological distress rather than headaches after mTBI (Bonanno et al., 2004). While this is different to headache severity, it shows the long-term impact that lower levels of psychological flexibility can have. For example, Bonanno et al. (2004) completed an interesting study in the wake of the September 2001 Terrorist attacks in New York, USA. They put together an experiment for 87 colleague undergraduates. They had three conditions, one in which people had to overtly express their emotions on seeing a set of pictures; another condition in which people had to suppress their emotional expression and a third condition where people acted as they normally would (e.g., not express or suppress). They found that the ability to flexibly respond (to both suppress and express) to the pictures was the biggest determinant of distress when measured again on average 1.5 years later. This was after controlling for initial distress, motivation, and cognitive resources. That is, the ability to flexibly respond was predictive of lower distress 1.5 years later. While this was not measuring psychological flexibility per se, it is possible that the ability to respond flexibly and therefore have lower levels of rigidity, is a similar process that occurs in the concept of psychological flexibility.

Previous research on psychological flexibility has tended to focus on functional outcomes over symptom severity. That is, research has found that psychological flexibility has a positive effect on people's everyday functioning but limited or no effects of psychological flexibility on the degree of pain or severity of headaches (Foote et al., 2016; Mo'tamedi et al., 2012; Vasiliou et al., 2021). This is one of the tenants of ACT, that symptoms themselves are not the focus, rather functioning in everyday life is the focus (Hayes et al., 2012). Interestingly, the current research has specifically found an influence of psychological flexibility at baseline on headache severity six months later. This adds further weight to the understanding of PF, that not only has it been shown to impact functional outcomes, but it can also impact symptom severity. However, it should be noted that we cannot infer causality, as it could be that another factor is accounting for both PF and headache severity.

Hypothesis Four, Five and Six: The mediating role of psychological flexibility on headache severity

Consistent with the three mediation hypotheses, our results showed that psychological inflexibility made a unique contribution to the relationship between psychological distress and headache severity shortly after mTBI (on average 5.9 weeks (SD = 2.291) post mTBI), at six months follow-up and between psychological flexibility at baseline and the relationship between psychological distress and headache severity at six months follow-up (on average 32.55 weeks (SD = 2.84756) post mTBI). An initial significant direct relationship was found between psychological distress and headache severity at baseline. However, when psychological flexibility was included as a mediator there was no significant direct relationship between psychological distress and headache severity. Regarding the other two time periods, there was no initial significant direct relationship between distress and headache severity. When psychological flexibility was included in the model there remained no significant direct relationship, only the indirect relationship with psychological flexibility was significant. This indicates that psychological flexibility is approaching full meditation of the relationship between distress and headache severity at all three time periods.

The literature has shown that psychological distress is associated with headaches and their severity, specifically that greater levels of distress lead to greater headache severity (Fraser et al., 2017; Ham et al., 1994; Jouzdani et al., 2014; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Ruff & Blake, 2016; Yilmaz et al., 2017). However, limited other research has investigated the mechanisms within psychological distress to understand how this relationship occurs. Mostly this has been investigated through treatment research. Previously, Sander et al. (2020) found that psychological flexibility was modified through a brief ACT treatment within a TBI population, and that also corresponded with improvements in psychological distress. However, other research (Whiting et al., 2020) has shown improvements in depression and stress following ACT treatment but without corresponding changes in psychological flexibility. However, as the authors noted this was with a small population of 9 and 10 people in each treatment group and psychological flexibility was approaching significance. Our results suggest that psychological flexibility may explain some of the

association between psychological distress and headache severity. In other words, we know people who are distressed will have higher severity headaches, but we know more specifically that lower levels of psychological flexibility may explain the relationship between higher levels of psychological distress and its association with higher severity headaches.

Our findings also support the wider literature of psychological flexibility as a transdiagnostic mechanism. Several other studies have also found associations between psychological flexibility and a wide range of health conditions including, depression, anxiety, chronic fatigue, chronic pain, and eating disorders (Bluett et al., 2016; Bohlmeijer et al., 2011; Densham et al., 2016; Masuda & Tully, 2011; Scott et al., 2016). This study adds to the literature, showing that for those who have suffered an mTBI and experience headaches, psychological flexibility may be having an impact on headache severity. This provides further evidence that PF is a transdiagnostic factor, e.g., that it is present across a range of different conditions.

These findings are in line with other research over the long term, which looked at functional outcomes rather than symptoms or headache severity. Faulkner et al. (2022) found that PF mediates the relationship between psychological distress and functional disability at approximately six months post mTBI. Looking at this from the opposite viewpoint, a recent study by Elliott et al. (2019) looked at war veterans with and without mTBI to understand key personality or other factors that may contribute to resiliency. In a sample of 264 veterans who completed measures at 4, 8 and 12 months, they found that psychological flexibility mediated the relationship between resilience and adjustment in all their time periods. This indicates that resiliency coupled with higher psychological flexibility contributes to positive outcomes in the medium to long term. This is an opposite way of conceptualising psychological flexibility but ultimately these results are in line with our findings, that PF is a mediating variable between psychological factors and outcomes.

To help understand how PF is mediating the relationship between psychological distress and headache severity at this six-month period, we need to return to the idea of the rigidity and the inflexible way people may apply avoidance and other strategies (Morris & Mansell, 2018). Over time inflexible strategies may be maintained in those with lower PF, given it would appear as if there were

a benefit in strategies such as avoidance. For those with distress, it may be the persistent application of these rigid strategies (which may have been adaptive at the beginning) that contributes to an increase in headache severity.

Our third finding indicates that psychological flexibility at baseline may influence how psychological distress impacts headache severity at six months follow-up. Specifically, higher psychological distress coupled with high psychological inflexibility is associated with higher severity headaches at six months follow-up. This is in alignment with the other two mediation findings and points to the continued way that PF is influencing the relationship between distress and headaches. However, PF only accounts for approximately 3% of the variance in headaches at this time period. This is a notably low amount and indicates that the vast amount of variance is unexplained. In this case it is possible that the reduced amount of variance is because of the period between the baseline measure of psychological flexibility and psychological distress, and headaches measured six months later. There are likely other biopsychosocial factors which also mediate this relationship, over this extended time period. That is, six months is enough time for other factors to emerge and influence the relationship. For instance, other factors which may express themselves over a longer period, such as sleep (Wickwire et al., 2018), social support and community integration (Stalnacke, 2007), are possibly having an influence on this relationship. These, however, are just possibilities and future research would need to investigate other possible mediating factors to fully understand what is occurring here.

Implications

This research has implications for the treatment of headaches following mTBI. Traditional treatments used in primary headaches have not always been shown to be effective for PTH (DiTommaso et al., 2014; Fraser et al., 2017; Lucas, 2011; Silverberg, 2019; Watanabe et al., 2012). Our research has shown a link between higher psychological inflexibility and higher headache severity. This is promising as psychological flexibility can be increased through ACT (Hayes et al., 2006, 2012; McCracken & Morley, 2014). We know that research on ACT to increase psychological

flexibility has shown effectiveness in the pain population (Bailey et al., 2010; Scott et al., 2016; Veehof et al., 2011; Vowles et al., 2020), the primary headache population (Almarzooqi et al., 2017; Dindo, et al., 2014; Grazzi, et al., 2019; Mo'tamedi et al., 2012; Vasiliou et al., 2021, 2022), and more recently in the TBI population (Sander et al., 2020; Whiting et al., 2020). Our findings indicate that if we identify people early on after their mTBI who have lower psychological flexibility, we can start treatment with ACT straight away and this may lead to a reduction in the severity of headaches. The other option is that even if we do not identify someone in the acute phase after their mTBI, but we do up to six months later, we can still begin ACT as our findings illustrate that the relationship between PF and headaches is still evident in the chronic phase. This is important given we know headaches are one of the main symptoms following mTBI (Dikmen et al., 2010; Faux & Sheedy, 2008; Langer et al., 2022; Lucas et al., 2014; Nampiaparampil, 2008; Schwedt, 2021) and if ACT can improve PF and therefore headaches, this may also have a beneficial flow on effect to other symptoms, but perhaps more importantly overall functioning for the individual.

We also know that psychological distress is common after a mTBI (Broshek et al., 2015; Hou et al., 2012; Iverson et al., 2017; Meares et al., 2011; Ponsford et al., 2019; Scheenen et al., 2017; Snell et al., 2011; van der Naalt et al., 2017) and has an influence on symptoms and functioning. If we can influence its expression through increasing psychological flexibility (through ACT), we may also see an improvement in headache severity. This could also have flow-on effects to other symptom expressions. However, it is important to highlight that this is only speculative at this stage, and our findings provide theoretical justification to examine the role of ACT to improve headache severity, as well as psychological distress more generally following mTBI. A randomised clinical trial, that is replicated, would need to occur before ACT can be recommended as a treatment option.

Furthermore, our findings also suggest that there may be benefit in measuring psychological flexibility in concussion services to identify those who are at great risk of experiencing persistent headaches over time. We have shown that PF at baseline is predictive of headache severity six months later, which means PF is one way to identify people who are a risk of ongoing headaches. Measuring psychological flexibility can be done quickly and non-intrusively through a simple context specific

self-report measure. If we can identify them early enough, then we can begin appropriate treatment sooner, and hopefully see a reduction or elimination in persistent headaches following mTBI.

Strengths and Limitations

Strengths

This study had several strengths, the first is that we controlled for many factors which have been shown in other research to influence PTH or PCS. The factors included; age (Iverson et al., 2017; van der Naalt et al., 2017; Yilmaz et al., 2017), gender (Hoffman et al., 2011; Iverson et al., 2017; Yilmaz et al., 2017), education (Snell et al., 2011; van der Naalt et al., 2017), concussion history (Hoffman et al., 2011), mental health history (Fraser et al., 2017; Iverson et al., 2017; Jouzdani et al., 2014; King, 1996; MacMillan et al., 2002; Meares et al., 2008; Ponsford et al., 2000), medical history (Lieba-Samal et al., 2011; Stulemeijer et al., 2008), other injuries experienced (Stulemeijer et al., 2006), psychological distress (Ham et al., 1994; Kjeldgaard et al., 2014a; Lieba-Samal et al., 2011; Ruff & Blake, 2016; Yilmaz et al., 2017) and mechanism of injury (Booker et al., 2019). This increases the strength of our findings as we have attempted to mitigate the influence of these factors and therefore makes our findings regarding psychological flexibility more robust.

Another key strength of this study was the use of two time periods, baseline, and six months follow-up. Previous research has tended to choose one or the other (Belanger et al., 2013; Silverberg et al., 2015; Snell et al., 2011) but including both provides an ability to understand what is happening at two significant periods, acutely and in the long term. This is especially relevant for PCS symptoms given the previous history of believing these symptoms were only present for a short acute period whereas more recent evidence concludes that symptoms persist in the long term in a much larger percentage of the population (Barker-Collo et al., 2013; Dikmen et al., 2017; Nelson et al., 2019; Theadom et al., 2016).

Many different locations were included throughout the north island of New Zealand, so this is likely to capture a reasonable amount of diversity from both urban and rural populations. Noting the results may not be generalisable to other populations in the world. We also had a similar percentage of

ethnicities as is present in the overall New Zealand population, indicating our sample was a realistic representation. For instance, Statistics New Zealand (n.d.) most recent 2018 census shows that 70.2% of the New Zealand population is of European ethnicity, which compares with our average across the two time periods of 65.9%. Māori ethnicity from the 2018 census was 16.5%; in our study the average was 10.55% which is noted as slightly under the national average. Lastly encapsulating all other ethnicities from the 2018 census was 25.9% and in the current study the average was 23.6%.

Limitations and Future Research

There are several limitations of this research that need to be discussed. First, as noted earlier, psychological flexibility was measured using the context-specific measure, the AAQ-ABI (RA), and only the reactive avoidance items were included. In this context reactive avoidance captures acceptance and experiential avoidance as it relates to peoples' feelings about their concussion (Sylvester, 2011; Whiting et al., 2015). It has been put forward by others (Cherry et al., 2021; Kashdan et al., 2020), that this may not be the equivalent of psychological flexibility, in that reactive avoidance and psychological flexibility are not two sides of the same coin. This comes back to the definition of psychological flexibility suggested by Cherry and colleagues (2021) in which psychological flexibility encompasses three main components; handling interference or distress, taking action to manage the distress, and the action fitting the situational demands and facilitating the pursuit of personal goals or values. This definition may be broader than the concept of reactive avoidance and thus not be capturing the entirety of the concept of psychological flexibility (Kashdan et al., 2020). However, other arguments have acknowledged that it does capture some of the core of psychological flexibility, in that is captures the rigid way some people respond to a variety of situations (Bond et al., 2011; Faulkner et al., 2021b). Future research into understanding the best measure of psychological flexibility is needed. For instance, new measures have emerged such as the Personalised Psychological Flexibility Index (Kashdan et al., 2020) and the Multidimensional Psychological Flexibility Inventory (Rolffs et al., 2018), both of which have attempted to capture more aspects of psychological flexibility e.g., the six dimensions targeted in ACT to increase psychological flexibility, as outlined earlier.

Another limitation related to the AAQ-ABI (RA), is that it has been shown to be highly correlated with psychological distress and there is concern from others that they may not be measuring two distinct things (Cherry et al., 2021; Doorley et al., 2020; Tyndall et al., 2019). However, others have found that this measure adds further explanatory value above and beyond psychological distress (Gloster et al., 2011). Nevertheless, it is also possible that we have measured more aspects of psychological distress rather than the distinct concept of PF, in which case the usefulness of the current results could be limited. However, we have attempted to account for this by including psychological distress through our use of the DASS-21 in our regression model. In future, research could look at including different measures of PF to further validate our results.

Headaches following mTBI have their own classification system but in this research, we did not use the formal diagnostic criteria, mentioned earlier in Table 1. Including this would have helped to confirm the presence PTH and made the results more applicable to the wider research on PTH. It would have also confirmed the presence of Acute and Persistent PTH which again make the results consistent with other research on this symptom. Several other studies have used this official measure of headaches (Ashina et al., 2020; Couch & Bearss, 2001; Faux & Sheedy, 2008; Lieba-Samal et al., 2011; Metti et al., 2020) and it would be beneficial to include this in any further research so we can confirm these headaches are PTH and not headaches occurring for other reasons. However, it should be noted that the RPQ does specifically ask respondents to think about their symptoms in relation to their mTBI and to think about this as being different from before their injury.

Another limitation is that our only measure of headache severity was the one item on the RPQ with four levels of severity and it only captured headaches within the last 24 hours. This may not have captured peoples' full experience of headaches given the specific timeframe. As well, they may not have spent a large amount of time thinking about their headache severity given it was just one questions among several others. This may have limited the accuracy of their headaches; in that they may have over-estimated or underestimated the severity of their headaches. Other studies looking at headaches have often included a headache diary (Martin et al., 2014; Silverberg, 2019) in which participants record when they have a headache and how severe it was for a set period of time. This

may have led to a more accurate understanding of headaches for this population, and it would also provide information about the number of headaches, not just their severity.

Future research into headaches and psychological flexibility would benefit from including a greater range of measures for both constructs as this would add greater detail to the findings and provide further clarity that we have measured the constructs adequately. In future research it would also be helpful to replicate these findings to confirm this effect is present in other populations, including other populations throughout the world. Longitudinal research would also help to ascertain how the mechanism of psychological flexibility is working and how directly or indirectly this is influencing symptoms after an mTBI.

A further limitation was not including previous headaches as a confounding variable. Previous research has indicated that a history of headaches is a risk factor for the experience of headaches after mTBI (Hoffman et al., 2011; Lieba-Samal et al., 2011; Lucas, 2011; Schwedt, 2021; Yilmaz et al., 2017). This means that we cannot rule this out as something that could have influenced the experience of headaches above and beyond psychological flexibility and gender.

While our sample size was reasonable, with just over 100 people in our analysis, it is possible this sample size is still not representative of the larger mTBI population. We also did not recruit people from the general population, instead, we recruited those who had been referred to a concussion clinic. This may have biased the sample in that only those people referred and who attended a concussion clinic had the opportunity to take part in this research. Thus, it limits the generalisability of these results to a certain degree.

It is also noted that around 30% of the participants dropped out between our baseline and six-month follow-up. The reasons for this are not known; but a few things are possible. The population that dropped out may have had higher degree headaches or other symptoms which caused them not to want to continue to take part. It is equally possible that this population was feeling much better with fewer symptoms, and they did not want to take part as they felt this would not add any value or had moved on with their life. Furthermore, our population was largely women, 66.4% at baseline and 71% at six months follow-up. This again restricts the generalisability of the results.

While this research shows an association between psychological flexibility and headache severity, we cannot infer causation between the two variables. It is possible that a third variable can account for both lower flexibility and higher severity headaches, that we have not accounted for.

This research only measured our variables up to six months after individuals were recruited into the study. Future research would benefit from researching over a longer time period, including up to 12 months or 1-2 years in the future. This would help to understand the ongoing nature of headaches, but also if there are associations with psychological flexibility many years after the mTBI.

Furthermore, we didn't specifically look at what types of headaches people were experiencing e.g., migraine or tension type headaches. This would have helped to understand the types of headaches experienced by this population and if this corresponds with the previous research of this type. It would have also been interesting to understand if different headache types were more or less associated with psychological flexibility.

Lastly, future research would benefit from understanding if increasing psychological flexibility through ACT, does have an influence on the severity of headaches in the mTBI population. Therefore, research such as a randomised control trial for the mTBI population with ACT as the treatment, is suggested. This would provide evidence of the effect of PF on headache severity, and if proven this will ultimately improve outcomes for those people with symptoms, especially ongoing symptoms after their mTBI.

Conclusion

All of our results were statistically significant and supported all our hypotheses regarding the association between psychological flexibility and headache severity after mTBI. This suggests that psychological flexibility may have an influential role in the experience of headaches following mTBI. It provides theoretical justification to explore the role of Acceptance and Commitment Therapy for reducing the severity of headaches after mTBI. Given headaches are such a significant symptom that often continue long past the initial injury it is important to make progress in understanding what treatment approaches may be efficacious. More generally, this study also supports wider research on

psychological flexibility acting as a transdiagnostic mechanism across a range of conditions, including mTBI. Furthermore, it provides a framework to better understand the influence of psychological distress on the severity of symptoms. Future research to replicate these results would be beneficial as would research using ACT to target increasing psychological flexibility to see if this results in a decrease in headache severity.

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