Maori, Pacific, and European Differences in Response to Pain and Worry

An Experimental Investigation

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ABSTRACT

Pain is one of the leading causes of health care utilisation in the world. However, the complex interactions between the physiological and psychosocial factors involved in the experience of pain are not well understood. This issue becomes more complex when individuals from different ethnic and cultural backgrounds are involved. It is well established that people of different ethnic backgrounds possess unique attitudes, perceptions and reactions to pain; yet whether differences in the physiological processing of pain between ethnic groups exists remains uncertain. Despite this issue being of particular relevance to New Zealand, where ethnic disparities in the reporting and management of clinical pain are found to exist, there is lack in research looking into the mechanisms driving these disparities.

The current study aimed to experimentally investigate both behavioural and physiological responses to a painful task and a worry induction task, in a sample of Maori, Pacific Island and European individuals. It also aimed to investigate the role of 'pain catastrophising' in the relationship between ethnicity and pain. Some have suggested that levels of 'general worry' may mediate the influences of catastrophic thoughts about pain on the pain experience. On this basis, the final aim of the study was to explore the relationship between general worry and pain catastrophising, and the impact these variables had on performance at the painful task and worry induction task.

Sixty-four healthy volunteers were recruited and each were separately exposed to a painful cold pressor task (physical stressor), and a worry induction task (mental stressor). Pain tolerance, pain threshold, and subjective pain ratings were assessed. Physiological responses to the pain and worry tasks were assessed by monitoring participants' Heart Rate

(HR) and Heart Rate Variability (HRV). HRV provides insight into the cardiovascular systems ability to effectively respond to stressors such as pain and worry. The relationship between these behavioural and physiological outcome measures were compared to participants' self-reported catastrophic thoughts about pain and other psychosocial variables found to impact the pain experience.

Maori, Pacific and European participants were not found to significantly differ in their behavioural and physiological responses to the pain and worry tasks. However, Maori and Pacific Island participants showed significantly healthier physiological recovery patterns from the pain task compared to European participants. Further, higher baseline 'high frequency' HRV was found to be significantly linked with higher pain tolerance. Pain catastrophising levels were not found to significantly differ between ethnic groups, hence conclusions about its mediating influences could not be established. Nonetheless, pain catastrophising was found to be significantly, positively associated with general worry.

This research suggests that factors outside of a controlled experimental context may be responsible for differences in clinical pain reports between Maori, Pacific Island and European peoples. The ethnic differences in HRV recovery from the pain task, suggest differences in autonomic cardiovascular stress modulation between these groups. This research provides a platform for future research aimed at making the treatment of pain in clinical settings more ethnically sensitive. It also provides a motive for further research looking into the characteristics of cardiovascular stress modulation in these groups.

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INTRODUCTION

The experience of pain is universal to human experience, yet individuals differ widely in how they perceive, understand and report pain. From a medical perspective, pain is one of the most commonly reported symptoms and compelling reasons for individuals to seek health care (Abbott & Fraser, 1998; Collett, 2005; Hider, Whitehurst, Thomas, & Foster, 2011). Though unpleasant in nature, pain has useful functions such as acting as a warning signal when one has injured themselves and prompts protective behaviours (Carr & Goudas, 1999). However, for some the experience of pain can become chronic and have a significantly debilitating impact on day-to-day functioning. Chronic pain conditions may occur in the absence of any overt tissue damage. Additionally, chronic pain conditions are maintained and exacerbated by biological, psychosocial, and personal factors (Melzack, 2008).

The ethnic background of an individual has been found to have a unique influence on how one understands and reports pain (Fabian, McGuire, Goodin, & Edwards, 2011). However, the mechanisms which drive ethnic differences in the experience of pain are not clearly understood. It is important to understand the mechanisms driving ethnic differences in the pain experience as these differences lead to how specific ethnic groups address, report and manage pain in clinical settings. This is of particular importance in New Zealand, where not only is there a highly multi-ethnic population but also where ethnic disparities in the reporting of clinical pain are found to exist (New Zealand Ministry of Health, 2008). Despite these disparities, there is a limited amount of studies investigating how specific ethnic minority groups in New Zealand, namely Maori and Pacific Islanders, experience pain.

As mentioned, the experience of pain is multifaceted and is influenced by both biological and psychosocial factors. Hence, to comprehensively characterise individual differences in the experience of pain, all dimensions of the pain experience should be considered. Experimental studies provide a controlled environment where an individuals' self reported psychosocial perceptions of pain can be compared to their behavioural and physiological responses to pain through various pain induction techniques. The current study is the first experimental study to compare and contrast the relationship between psychosocial perceptions of pain, and behavioural and physiological responses to pain, in healthy Maori, Pacific and European individuals in New Zealand.

The following three chapters introduce the current study by providing an overview of the key factors investigated in the study. The first chapter introduces the phenomenon of pain, highlights current understandings of pain, and discusses the physiological processing of pain. The second chapter extends the discussion on pain by discussing key psychosocial and individual factors which are found to impact the experience of pain. Particular emphasis is placed on the impacts of ethnicity, catastrophising and worry on pain, as they are the primary psychosocial variables of interest. The third chapter then introduces and describes the phenomenon of Heart Rate Variability, which is used as the primary physiological outcome measure of responses to a pain induction task in the current study.

CHAPTER ONE

PAIN: AN INTRODUCTION

Pain is a complex and perplexing phenomenon that has been both experienced and studied for centuries. The experience of pain can be overwhelming. It can capture the attention of, and cause significant disability in the most strong-willed individuals. What was once understood to be solely a somatic experience, is now understood to be multidimensional and influenced by numerous psychosocial factors. It is due to this complexity, that researchers have found value in exploring pain as a holistic phenomenon, including how various historical, contextual, and personal factors influence ones experience of pain, and conversely how pain influences these factors. Research into these factors is of critical importance to the development and enhancement of treatments aimed at improving pain and well-being for those who suffer pain-related conditions. This present study contributes to this body of research, and looks into the psychosocial and physiological associations of how healthy individuals respond to pain. This chapter introduces pain, its underlying theories, existing classifications, and describes current understandings.

1.1. Defining Pain

Initially, when the notion of mind body dualism was widely accepted, pain was viewed exclusively either as a psychological phenomenon or a physiological sensation (Turk & Rudy, 1986). Psychologically, pain was seen as a manifestation of feelings of unpleasantness and viewed as an emotion (Perl, 2007). Pain was not included in the historical doctrine of the

five senses proposed by Aristotle. Rather, pain was placed with "...pleasure among passions of the soul" (Dallenbach, 1939, p. 331). As a physiological process, pain was seen as a result of the stimulation of sensory endings by noxious stimuli, leading to impulses being transmitted directly to the brain (Wall, 1979). In more recent history, pain has also been viewed as a *behavioural* expression of distress (Beecher, 1959; Craig, 2009) and various personal factors such as emotional state, past experiences, contextual factors, and culture have been found to impact and modify the experience of pain (Gatchel, Peng, Peters, Fuchs, & Turk, 2007; Lumley et al., 2011; Melzack & Wall, 1982).

Though difficulties exist in finding a universal definition of pain that incorporates all the different dimensions of this phenomenon, a need for a structured conceptualisation exists for researchers to enhance validity in the study of pain, and enhance communications between pain researchers and clinicians. The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage" (Bonica, 1979, p. 250). This definition encapsulates both the psychological and physiological viewpoints earlier described. It also captures the notion that pain can exist in the absence of any identifiable pathology, and can be influenced by factors *outside* of the body. Such concepts were excluded from early models attempting to explain the experience of pain.

1.2. Development of the Current Understanding of Pain

Until the mid 20th century, the processing of pain was believed to be a hard-wired, one-way neural pathway from the periphery (site of noxious stimuli input) to the pain processing

centres of the brain (Melzack & Katz, 2003). The first model to embrace this viewpoint was the *specificity theory*. Specificity theory proposed that "pain is a specific modality like vision or hearing, with its own central and peripheral apparatus" (Melzack & Wall, 1965, p. 971). From this perspective it was suggested that the amount of noxious stimulus one exhibits (i.e. the amount of tissue damage) should be directly proportional to the pain one should feel. However, advancements in pain research did not reliably support this notion (Melzack & Katz, 2003). The *pattern theory* emerged in response to the inaccuracies of the specificity theory. Pattern theory proposed that both noxious and non-noxious signals are sent in patterns to the brain via the dorsal horn of the spinal column where processing of these signals take place (Melzack & Wall, 1965). Pain was suggested to be a result of the accumulation of noxious signals reaching a particular threshold in a set amount of time.

Both the specificity and pattern theories have provided valuable insight into the sensory dimensions of the pain experience. However, they are found to lack accountability for the subjective nature of the pain experience, lack of power in explaining how pain can act as a motivation for behaviour and learning, lack of insight into the role that the brain has in the processing and perception of the pain signal, and lack of insight into the affective influences on pain (Gatchel et al., 2007; Melzack & Wall, 1982). These weaknesses have lead to the development of new theoretical models in the field of pain.

1.2.1. Gate Control Theory

The gate control theory is the predominant theoretical framework upon which the current psychological understandings of pain are studied (IASP, 1997). It proposes that the experience of pain is modulated not only by peripheral afferents, but also by neural gates in the spinal cord. This gate is influenced by descending signals from the brain which control

whether, and how pain signals are processed (Melzack, 2008). The theory is based upon the functional interaction between three major processes involving, the *Substantia Gelatinosa organ*, the action system construct, and the central control trigger construct.

Substantia Gelatinosa

The *Substantia Gelatinosa* (SG) is the central organ of the gate control theory. Initially, when the body experiences some form of noxious input, the peripheral terminal of peripheral nociceptive afferents at the site of the noxious input are aroused. Subsequently, depolarisation of these afferents takes place and nerve impulses are generated (Woolf & Salter, 2000). Three primary types of peripheral afferents are involved in the processing of pain: (a) A-β fibres which carry sensory information and are highly myelinated allowing for fast signal conduction; (b) C-fibres which carry dull/aching pain signals and are unmylinated hence, have a slower conduction rate than A-β fibres of approximately 0.5 to 2 ms^{-1}; and (c) A-δ fibres, which carry sharp pain signals, are less mylinated and have slower conduction rates than A-β fibres. Yet, A-δ fibres conduct faster than C-fibres, with a conduction rate of approximately 20 ms^{-1} (Feilds, 1987; Skevington, 1995). The cell bodies of the peripheral afferents are located in the dorsal root ganglion of the spinal cord, which extend and synapse with 'Transmission-cells' (T-cells) at the SG (located at lamina II of the dorsal horn). The SG acts as a gating mechanism for sensory signals. The cells in the SG assess and modulate the balance of signals coming in from the A-β, A-δ, and C-fibres, before these signals are transmitted to the T-cells. Impulses from A-β fibres excite the cells in the SG and mediate a *negative* feedback loop resulting in the inhibition of signals being transmitted to the T-cells. Impulses from A-δ and C fibres excite cells in the SG and mediate a *positive* feedback loop resulting in the activation of the T-cells, enabling transmission of pain signals to the brain (Melzack & Wall, 1965). Both central and peripheral pain processing systems are malleable.

Prolonged nociceptive stimulation has been found to lead to the modification of these pain pathways, and this has become hypothesised as a possible mechanism underlying various chronic pain conditions (Lynn & Perl, 1996; Woolf, 2011).

Action System

Excitation of the T-cells beyond a specific threshold is responsible for the activation of an *action system*. Pain signals continue to travel along the axon of the T-cells via two primary pathways, the spinothalamic pathway and the spinorecticular pathway. Spinothalamic pathways transmit signals associated with the sensory dimension of pain, whereas spinorecticular pathways transmit signals associated with the affective dimensions of pain (Lynn & Perl, 1996). The pain signals pass the medulla, pons and midbrain and enter the ventromedian nucleus of the thalamus (Bear, Connors, & Paradiso, 2007; Chapman & Stillman, 1996; Rainville, 2002). The thalamus is where the characteristics of the pain signals are initially deciphered. This includes the discrimination of the motivational-affective components of the pain, emotional aspects of the pain, and pain memory. The third order neurons then project the pain signals to different areas of the brain depending on their characteristics (Patestas & Gartner, 2006). Various neuroimaging studies have suggested that the sensory component of pain is processed in the somatosensory thalamus, the primary somatosensory cortex, and the secondary somatosensory cortex (Auvray, Myin, & Spence, 2010). They have also suggested that the affective components of pain are processed in the cerebellum, amygdala, nucleus accumbens, nuclei of basal ganglia and anterior cingulate cortex (ACC) (Auvray et al., 2010; Rainville, 2002). It has been speculated that the insular cortex may have a role in pain related autonomic activity, including cardiovascular reactivity (Critchley, Corfield, Chandler, Mathias, & Dolan, 2000). In summary, activation of the action system marks the commencement of brain activities which are aimed at reducing the sensory

and affective components of the pain, such as rubbing the painful area, startle response, flexion response, and vocalization (Melzack & Wall, 1965).

Central Control Trigger

Melzack and Wall (1965) brought to light the 'top-down' influence of the central nervous system on pain perception. Specifically, they suggested that there are heavily mylinated fibres which send impulses down from the frontal cortex and hypothalamus to the dorsal horn via the periaqueductal gray area of the midbrain. Depending on the state of the brain, these signals influence the transmission of sensory inputs from the periphery, i.e. the pain gating mechanism in the SG. In particular, it is proposed that there are three dimensions which impact the state of the brain, and consequently the type of signals sent down the descending pathway: a motivational and affective dimension (general affective states, expectation of pain, and urge to escape the unpleasantness); a cognitive and evaluative dimension (appraisal of injury, influence of cultural factors, suggestion and distraction); and a sensory and discriminative dimension (location, duration, quality and intensity of the pain) (Melzack & Casey, 1968). The top-down pathway exhorts control over how peripheral sensory inputs are processed and hence, how pain is perceived. This central control trigger provides a scientific explanation of how various cognitive and psychosocial factors can impact the perception of pain, making it a completely subjective experience (Melzack & Katz, 2003).

Overall, the gate control theory provides a theoretical foundation for explaining how psychosocial factors including worry, anxiety, depression and cultural views of pain, may influence ones pain perception via top-down influences on the pain gate. The shift in focus to the spinal cord as the site of pain modulation has lead to various advancements in pain management, particularly in the fields of anaesthesia and analgesia (Yaksh, 1999). Advances

in neuroscience and the *biopsychosocial* movement have also formed a platform for the classification of various types of pain.

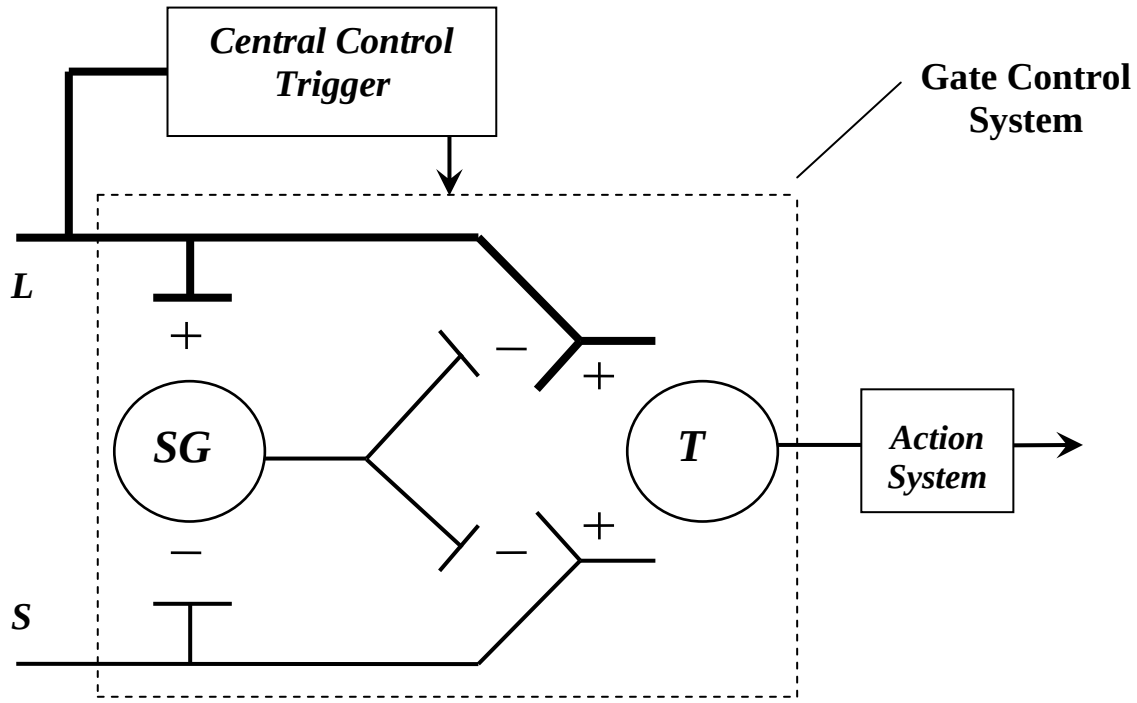


Figure 1. Schematic diagram of the Gate Control. Large (*L*) and small (*S*) fibres project from the periphery to the Substantia Gelatinosa (*SG*) and then to the central transmission (*T*) cells. The large fibres project to the central control trigger and then back into the Gate Control System. Signals T cells project to the primary cells of the action system. + represent excitation, and - represent inhibition. Adopted form Melzack and Katz (2003).

1.3. Classification of Pain

Some argue that due to the subjectivity of pain, forming classification systems may be impossible or simply undesirable (Merskey, 2007). Nonetheless, the benefits of having such classification systems in the field of pain are clear. Classifying pain based on its duration is found to provide vital information into the characteristics of one's experience of pain and provide a guideline of how the pain can be addressed.

1.3.1. Transient Pain

Individuals usually experience minor day-to-day sensations of pain which do not cause prolonged physical or emotional discomfort, and disappear quickly. Transient pain is characterised by initially being well defined and mild then intensifying for a few seconds with sensations such as throbbing or stabbing, and then quickly subsiding (Melzack & Wall, 1982). This type of pain is seldom associated with any real physiological consequence. In addition, no real demand is placed on an individual's attention and daily functioning is rarely affected (Loeser & Melzack, 1999; Melzack & Wall, 1982) .

1.3.2. Acute pain

Acute pain differs from transient pain in that it lasts longer and usually causes a temporary hindrance in day-to-day functioning. It is typically associated with some form of injury or tissue damage and may be anxiety provoking (Melzack & Wall, 1982). From a functional perspective, acute pain can act as an indicator of injury and consequently draws ones attention to the injury. It also prompts protective behaviours such as avoidance of excessive motion of the injured area which facilitates healing (Carr & Goudas, 1999). Acute pain is

usually alleviated with simple pain remedies and only lasts the duration of the corresponding injury. Acute pain influences, and is influenced by affect. This plays a role in how one defines the biological significance of the injury and what they attribute their injury to. In addition, acute pain can promote learning, by acting as an incentive to avoid future injury (Chapman & Stillman, 1996; Gatchel & Maddrey, 2004; Melzack & Wall, 1982). Acute pain can sometimes persist until its functional characteristics are no longer beneficial and can cause significant amounts of suffering and functional impairment, such as in *chronic pain*.

1.3.3. Chronic pain

Pain can persist beyond the normal time of healing of an injury, or of any useful function. The IASP (1994) define chronic pain as the subjective experience of persistent pain, in the absence of biomedical indicators such as tissue damage, or beyond the point of predicted healing. Chronic pain has generally been recognised as when pain persists beyond three to six months after an injury (New Zealand Ministry of Health [NZMoH], 2008). When pain becomes chronic, it can have long-term negative impacts on the sufferer's day to day living. However, often it is not only the pain itself that causes such debilitation, but also the myriad cognitive, behavioural, and physiological factors that commonly accompany it (Craig & Versloot, 2011). These factors contribute to why chronic pain conditions can have such debilitating effects in all facets of life including social function (e.g. withdrawal from social activities; Youssef, Atienza, Langseder, & Strauss, 2008), physical function (e.g. decreased mobility; Bishop, Meuleman, Robinson, & Light, 2007), psychological function (e.g. anxiety; Asmundson, Abrams, & Collimore, 2008; and depression; Bair, Robinson, Katon, & Kroenke, 2003) and can significantly reduce ones quality of life (Hunfeld et al., 2001; Wagner, Stenehjem, & Stanghelle, 1995).

Chronic pain conditions are usually accompanied by unsuccessful attempts to treat the pain with simple pain remedies (NZMoH, 2008). Major breakthroughs in research have established that it may be the amplification of neural signalling in the central nervous system that is responsible for prolonged episodes of pain which have no obvious organic origin (Woolf, 2011). This notion, referred to as 'central sensitisation', highlights that hyper-activation of the circuitry of the central nervous system may itself increase the duration and intensity of pain without direct involvement of any peripheral noxious stimuli. The emergence of this concept has added strength to the notion that pain can be 'centrally' driven, rather than exclusively peripherally mediated (Woolf, 2011).

1.4. Summary

Pain is a unique, multifaceted, and subjective perceptual phenomenon. This chapter explored current definitions and understandings of pain, the historical evolution of such understandings, and the various classifications of pain which have developed as a result. In the days of 'mind body dualism', the processing of painful sensations was thought to be a hardwired, one-way path from the site of the noxious stimulus to the brain. This idea has been firmly rejected with breakthroughs in research and the emergence of the gate control theory. The gate control theory has had an exceptional impact on the understanding of pain and has provided a scientific explanation of how psychosocial and individual factors influence the experience of pain. Through such developments, we now understand that though unpleasant in nature, pain can play a critical role in safety and survival, by acting as a precursor for learning, and prompting protective behaviours. Nonetheless, pain can become chronic,

through phenomenon such as 'central sensitisation', and persist beyond the point of any useful purpose. Chronic pain conditions can have debilitating effects on all facets of a person's life, and significantly hinder day to day functioning. The poor success of day to day analgesics in the control and treatment of chronic pain conditions have validated the importance of continuing to explore the impact of various psychosocial and individual factors on the pain experience.

CHAPTER TWO

The Multidimensional Nature of Pain

Pain is experienced universally, yet no two individuals have the same experience of pain. This is primarily because pain is a multidimensional phenomenon influenced by physiological (outlined in the previous chapter), psychosocial and individual factors. This multidimensional characteristic helps to explain the subjectivity of the pain experience. This chapter outlines some key psychosocial and individual factors proposed to influence pain. The psychosocial features and individual factors discussed will be those which are being explicitly measured in the current study. This includes discussions surrounding the impact of catastrophising, worry, stress, anxiety, attention, depression, ethnicity, gender and age on pain. Of these factors, catastrophising, worry and ethnicity will be discussed in more detail as they are the key variables of interest in the study.

2.1. Psychosocial Factors

Recent research in both clinical and non-clinical settings has shed light on the role of various emotional and psychological factors in the experience of pain. This section will discuss the influence of catastrophising, worry, stress, anxiety, attention, and depression on pain. Particular emphasis will be placed on catastrophising and worry as they are key variables of interest.

2.1.1. Catastrophising

Catastrophising has emerged as one of the most potent psychological factors to influence the pain experience. Pain catastrophising broadly refers to an "exaggerated negative mental set brought to bear during an actual or anticipated painful experience" (Sullivan et al., 2001, p. 53). The search for a valid measure to assess pain catastrophising led to the refinement of this definition. Sullivan and colleagues (1995) developed the Pain Catastrophising Scale (PCS), which is the most commonly used measure of pain catastrophising. Factor analysis revealed three main dimensions underlying pain catastrophising; *rumination*, *magnification*, and *helplessness*. Rumination, refers to intrusive, worrying, and repetitive thoughts, and inability to direct focus away from pain related thoughts. Magnification, refers to the unpleasantness of painful situations and the expectation of negative consequences. Helplessness, refers to perceived inability to cope with painful experiences (Sullivan et al., 1995). Each one of these factors has been found to impact the experience of pain in different ways.

With regards to chronic pain, pain catastrophising has been significantly correlated with increased pain reports and increased disability (Martin et al., 1996; Schanberg, Kredich, Keefe, Lefebvre, & Gil, 1996; Schanberg, Lefebvre, Keefe, Kredich, & Gil, 1997). Additionally, pain catastrophising is associated with heightened disability in those experiencing pain caused by acute injuries, regardless of the severity of the pain (Sullivan, Stanish, Sullivan, & Tripp, 2002; Sullivan & Neish, 1998). Various experimental studies have attempted to isolate and investigate catastrophising and help understand mediating and moderating factors which impact its influence on pain.

Individuals high in pain catastrophising tend to experience more intense pain, have reduced tolerance and lower thresholds to pain, have higher levels of pain related discomfort,

and exhibit increased pain behaviours while undertaking the cold pressor task (Campbell et al., 2010a; Edwards, Haythornthwaite, Sullivan, & Fillingim, 2004; Sullivan, Tripp, & Santor, 2000; Thastum, Zachariae, Scheler, Bjerring, & Herlin, 1997). Nonetheless, the relationship between pain catastrophising and pain severity is variable. Catastrophising is found to account for 7% to 31% of the variance in pain severity (Sullivan et al., 2001). The origin of pain catastrophising has been debated for a number of years. Various models have been proposed to explain the underlying mechanisms which drive the relationship between catastrophising and pain.

It has been suggested that high pain catastrophisers possess negative *pain schema.* They have overly negative, maladaptive beliefs about pain or lack self-efficacy hindering their ability to cope with pain. This has been referred to as the 'schema activation' viewpoint (Sullivan et al., 1995; Turk & Rudy, 1992). Once these negative schema are activated, through an actual or potential pain eliciting experience, they influence cognitive or emotional functioning which can lead to a worsening of the pain experience (Sullivan, et al., 2001).

Related to the Schema activation viewpoint is the Appraisal model of coping proposed by Lazarus and Folkman (1984). From this viewpoint, pain catastrophising manifests through attributions made about a pain related experience. It is suggested that the *magnification* and *rumination* dimensions of catastrophising are associated with primary appraisals (deciding whether a potential stressor is in need of attention and whether it is harmful of not). An example is the exaggeration of the threat value of a painful experience. The *helplessness* dimension of catastrophising is linked with the secondary appraisals of a pain related experience (beliefs about efficacy to cope, and perceived access to coping resources), an example being the excessively negative evaluative perceptions of one's ability

to cope with the pain (Sullivan, et al., 1995). Furthermore, it has been suggested that catastrophising may be linked to ones perceptions of their ability to control painful experiences (Geisser, Robinson, & Riley, 1999; Parker et al., 1989; Rosenstiel & Keefe, 1983; Turner & Clancy, 1986).

One common element of the Appraisal model and Schema activation viewpoint is that they both infer that high pain catastrophisers have an increased attentional focus on pain. Experimental studies have shown that catastrophic thoughts about pain contribute to an increase in attentional resources being directed towards pain (Van Damme, Crombez, & Eccleston, 2004a). Consequently, high pain catastrophisers are shown to have poorer disengagement from pain-related thoughts during the anticipation of a painful stimulus than from the anticipation of a non-painful stimulus (Van Damme, Crombez, & Eccleston, 2004b). In support of this attentional viewpoint, Sullivan and Neish (1998) found the *rumination* subscale of the PCS to be the strongest predictor of pain ratings during dental hygiene procedures. Highlighting that prolonged attentional investment or 'worry' may be a key contributing factor to the relationship between catastrophising and pain intensity.

Catastrophising has also been viewed as a means to *cope* with pain (Sullivan et al., 2001). However, this viewpoint is not without controversy as generally high pain catastrophisers and non-pain catastrophisers do not differ in the number and type of general coping strategies they employ (Spanos, Radtke-Bodorik, Ferguson, & Jones, 1979; Sullivan et al., 2001). Sullivan and colleagues (2001) noted that there may be a place for catastrophising as a coping mechanism if the goals of coping encompass ones' ability to effectively communicate their distress to others, leading to appropriate assistance or support being provided. This viewpoint has been firmly supported by other studies (Keefe et al.,

2003; Sullivan, Adams, & Sullivan, 2004). The models and viewpoints mentioned are not mutually exclusive, and they remain theoretical. Further, research has investigated other ways that catastrophising varies across individuals factors.

Pain catastrophising appears to emerge from an early age. Specifically, it has been hypothesised that the provision of excessive levels of support and physical comfort from an early age may augment the emergence of exaggerated reactions to pain (Sullivan, et al., 2001). Catastrophising has been found to be reasonably consistent over time in the absence of any intervention, yet evidence suggests that in adulthood it may decrease with age. Some have suggested this is due to lower levels of emotional rather than sensory processing of pain in older adults compared to youth (Jacobsen & Butler, 1996; Ruscheweyh et al., 2011; Sullivan & Neish, 1998). Females tend to report higher levels of pain catastrophising in both clinical-pain settings and non-clinical pain settings (Jensen, Nygren, Gamberale, Goldie, & Westerholm, 1994a; Keefe et al., 2000; Sullivan et al., 2000). Such findings have suggested that catastrophising may mediate gender differences in pain reports (Edwards et al., 2004; Sullivan et al, 2000).

Catastrophising tends to be an antecedent rather than a consequence of pain. In a pain free state, pain catastrophising has been shown to significantly predict pain levels during a dental hygiene procedure, when assessed one week prior to the procedure (Sullivan & Neish, 1999) and experimental pain levels at ten weeks (Sullivan, et al., 1995). These findings have lead to the viewpoint that catastrophising may have a *causal* influence on increased pain. Neurobiological investigations have provided support for this hypothesis. Various experimental studies have investigated the relationship between pain catastrophising, pain perception, and the diffuse noxious inhibitory control (DNIC) system in healthy individuals.

The DNIC is the neurophysiological mechanism proposed to underlie the reduction of pain signal processing in the dorsal horn in response to a noxious stimulus being applied to other areas of the body. In other words, pain in one part of the body inhibits pain in another part of the body (Le Bars, 2002; Le Bars, Dickenson, & Besson, 1979; Schouenborg & Dickenson, 1985). Examples are the analgesic effects of certain forms of acupuncture or transcutaneous nerve stimulation (Le Bars et al., 1979). Findings have suggested that pain catastrophising is negatively associated with DNIC, thus indicating that catastrophising may precede increased ratings of pain through having antagonistic effects on endogenous pain inhibition mechanisms (Goodin et al., 2009; Weissman-Fogel, Sprecher, & Pud, 2008). This hypothesis has received support from numerous other studies (Campbell & Edwards, 2009; Campbell et al., 2010b; Severeijns, van den Hout, & Vlaeyen, 2005; Willer, Le Bars, & De Broucker, 1990). Nonetheless, these studies are limited by their cross-sectional nature. Additionally, findings exist which show no impact of induced pain catastrophising on expected pain, experienced pain, and performance at the cold-pressor task (Severeijns et al., 2005). Thus, it is clear that catastrophising plays a major role in the pain experience. Further research is required to establish firm conclusions about the underlying mechanisms driving this relationship.

2.1.2. Worry

Worry is experienced by most people at stages throughout their life. Worry usually involves a chain of intrusive thoughts and images which are potentially uncontrollable and characterised by negative-affectivity (Borkovec, Ray, & Stober, 1998). As Borkovec, Robinson, Pruzinsky, and DePree (1983) eloquently describe: “the worry process represents an attempt to engage in mental-problem solving on an issue whose outcome is uncertain but contains the possibility of one or more negative outcomes” (p. 10). A strength of this viewpoint is that it encapsulates

the potential for positive outcomes to be a consequence of worry (Mogg, Mathews, Bird, & Macgregor-Morris, 1990). One example is that worry may prompt engagement in problem-solving techniques to help an individual discover a solution to a problem. Nonetheless, excessive levels of worry are found to be detrimental to problem-solving (Borkovec et al., 1983; Eccleston & Crombez, 2007). Excessive worriers tend to have difficulty generating and implementing solutions to problems, particularly if the outcomes of the problem are ambiguous. This is generally due to a fixation with repeated attempts at identifying the problem and dwelling on its possible negative outcomes (Dugas, Letarte, Rhéaume, Freeston, & Ladouceur, 1995). During the development of the Worry Domains Questionnaire (WDQ; Tallis, Eysenck, & Mathews, 1992), the content of general day-to-day worries were investigated. Tallis and colleagues (1992) indentified that non-pathological worry revolved around five key domains. These included; relationships, lack of confidence, aimless future, work and financial issues. Worrying thoughts are attention grabbing and tend to leave an individual compelled to act. Importantly, *cognitive rehearsal* has been identified as a critical factor contributing to the maintenance of threat or concerns involved in the worry process (Wells & Morrison, 1994).

The notion of cognitive rehearsal, more recently termed 'rumination', has received considerable attention in research surrounding worry. Rumination and worry are both characterised by the experience of recurrent, intrusive, and negative cognitions (Papageorgiou & Siegle, 2003). Yet, worry tends to be about the future and rumination tends to be about the past (Watkins, Moulds, & Mackintosh, 2005). A central feature of these recurring thoughts is that they are cognitive representations of a psychosocial problem, or a stressor. Brosschot, Gerin, and Thayer (2006) devised the notion of *perseverative cognition* to refer to this central fundamental feature.

Perseverative cognition is believed to be the key maintaining factor for vigilance to threat. Vigilance to threat is a normal and adaptive response to threat, when the threat is transient and escapable. However, prolonged states of vigilance or 'action preparation' can have negative impacts on one's health (Brosschot, et al, 2006). For example, perseverative cognition, or prolonged stated of action preparation has been shown to lead to chronically enhanced heart rate (HR) and decreased heart rate variability (HRV). Further, the impacts of perseverative cognition on the immune system (immune suppression) and cardiac system (visceral damage) increase one's risk to a number of diseases such as cardiovascular disease and hypertension leading to premature mortality (Brosschot & Thayer, 1998; Brosschot et al., 2006; Linden, Earle, Gerin, & Christenfeld, 1997; Palatini & Julius, 1997; Ursin & Murison, 1983). A key contributor to perseverative cognition is the perceived inability to cope with the stressor, commonly reported as 'helplessness' (Brosschot et al., 2006). Considering the helplessness feature and ruminative nature of perseverative cognition, one can see how it may overlap with phenomenon such as catastrophising, and potentially influence pain.

The relationship between worry and pain is a relatively new area of research. However, the illness trajectory of chronic pain conditions are particularly uncertain, setting up an ideal environment for worry to thrive (Eccleston & Crombez, 2007; Eccleston, Crombez, Aldrich, & Stannard, 2001). Research has shown that those that experience chronic pain tend to persistently worry about the pain they are experiencing and its consequences (Eccleston et al., 2001). This type of *chronic* worry about pain, or 'pain related perseverative cognition' is found to be more distressing, attentionally demanding, intrusive, less pleasant and more difficult to disengage from than non-pain related worry for those suffering chronic pain (Eccleston et al., 2001). These worrying thoughts commonly result in misdirected

problem-solving leading to increased distress and consequently, heightened awareness to undesirable somatic sensations (Eccleston & Crombez, 2007; Eccleston et al., 2001). Lackner and Quigley (2005) conducted a cross-sectional investigation into the influences of catastrophising and trait-worry in patients with Irritable Bowel Syndrome (IBS). The authors revealed that worry was strongly associated with the negative affective component of pain, particularity impacting on long-term suffering. Importantly, the authors found that catastrophising was the key mediator of the relationship between worry and increased suffering in IBS patients. Further, it has been revealed that the *rumination* component of catastrophising may be the strongest predictor of increased pain reporting and disability in non-chronic pain cohorts (Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998). These findings substantiate the interrelated nature of worry and catastrophising in the context of pain.

In healthy individuals, worrying about pain is a normal response in a situation where the immediate escape from a painful event is not possible. This process functions to narrow or fix attention onto the object that threatens or causes pain, and drives efforts to reduce the threat. Melzack, Wall, and Ty (1982) assessed the characteristics of individuals presenting at an emergency clinic with acute pain-related injuries. Worry was found to be one of the most predominant emotions exhibited by these patients, particularly surrounding financial issues. Brosschot and Van Den Doef (2006) found that one-weeks total worry duration was positively associated with increases in somatic complaints such as lower back pain, neck pain, and stomach pains in a group of high school and college students. Hastie, Riley, and Fillingim (2005) conducted a telephone survey investigating differences in recent painful experiences between healthy young African Americans, Hispanics, and non-Hispanic whites. They found that pain-related worry was a significant, independent predictor of engagement in pain-reducing behaviours but only in Hispanics. This latter finding highlights the possible

moderating effects of ethnicity on the relationship between pain-related worry and pain behaviours.

Experimental studies have helped explore the influences of other possible moderators of the relationship between pain and worry. Boston and Sharpe (2005) found that level of worry about the cold pressor task was the primary mediating factor of the relationship between fear of pain, threat of manipulation, and pain tolerance during the task. Further, reductions in worry have been associated with improved tolerance and higher thresholds during the cold pressor task (Staats, Hekmat, & Staats, 2004). Though these studies are cross-sectional in nature, they paint a clear picture showing that the phenomenon of worry does impact the experience of pain, possibly by top-down alterations in pain processing.

In summary, it is clear that though a normal process, worry has a unique influence on the experience of both acute and chronic pain. *Chronic worry* about pain or 'pain related perseverative cognition' is intrusive, hard to dismiss and exacerbates pain-related suffering. This also leads to prolonged states of physiological arousal, which impacts both short-term and long-term health outcomes including those suffering chronic pain conditions. Therefore, the importance of investigating the phenomenon of worry in the context of pain is clear. Further exploration into factors speculated to influence the relationship between worry and pain such as catastrophising, would be of merit.

2.1.3. Stress

Pain can be viewed as a stressor, as the experience of pain disrupts the brains homeostatic regulatory systems and initiates processing of multiple systems to re-establish an *equilibrium* in the body (Melzack, 1999a). Importantly, it is the characteristics of a painful experience

that determine its ‘stressful’ effects on the body, such as the severity and duration of pain. Transient, acute or mild pain tends to lead to adaptive physiological reactions, whereas severe or prolonged pain can result in maladaptive or harmful physiological responses (Karas, Danneman, & Cadillac, 2008).

When stress occurs, including the perception of pain, a series of processes take place throughout the body which are necessary for actions such as tissue repair, activating the 'fight or flight' response, and driving body systems toward the reestablishment of homeostasis (Melzack, 1999b). Initially, cytokines are released into the bloodstream which travel up to the brain and are taken up by the hypothalamus, activating the hypothalamic-pituitary-adrenal (HPA) system. This leads to the release of corticotrophin releasing hormone (CRH) into the blood stream. CRH causes the release of adrenocorticotropic hormone (ACTH), and ACTH activates the adrenal gland to release the hormone, cortisol. Cortisol is essential in the stress response as it is responsible for sustaining sufficient levels of glucose for the stress response and acts on systems such as the immune system and endogenous opioid system.

At the same time the HPA system is carrying out these processes, the locus coeruleus-norepinepinephrine (LC-NE)-sympathetic system, located in the brainstem, acts upwards on various areas of the brain and then down the descending autonomic nervous system pathways (parasympathetic and sympathetic branches). During stressful experiences such as pain, activation of the sympathetic pathways dominate and prepare the cardiovascular system, blood vessels and other viscera for various procedures to re-establish homeostasis (Melzack, 1999a; Melzack, 1999b).

The release of cortisol and activation of the LC-NE-sympathetic system are essential in preparing the body for an efficient response to stressors such as pain, although prolonged activation of the LC-NE-sympathetic system and excessive release of cortisol is found to have detrimental effects on the body. These factors, particularly abnormal patterns of cortisol release have been linked to muscle, bone and neural tissue destruction leading to a number of chronic pain conditions such as Osteoporosis (Melzack, 1999b). Furthermore, these factors suppress immune system function, possibly leading to a number of chronic pain conditions, as well as decreasing the body's natural defence system against other medical conditions (Melzack, 1999a, 1999b; Sapolsky, 1992).

A key support for the link between pain, stress and immune system malfunction is the finding that many autoimmune diseases also produce pain, such as scleroderma and RA (Melzack, 1999a). Chrousos & Gold (1992) hypothesised that pain syndromes linked to stress may be caused and maintained as a result of decreased levels of cortisol, i.e. *hypocortisolism* due to prolonged stress. Recent correlational studies have tended to support this hypothesis (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Kuehl, Michaux, Richter, Schächinger, & Anton, 2010; Riva, Mork, Westgaard, Rø, & Lundberg, 2010).

Repeated maladaptive physiological activation such as that associated with a natural tendency to exhibit prolonged stress, e.g. individuals with high levels of trait worry (Brosschot et al., 2006), can *rewire* ones neurological control systems. This can lead the brain to produce a predictive, maladaptive, and damaging response pattern to any type of stress, which in-turn, may give rise to chronic pain conditions (Melzack, 1999b). In summary, though pain and stress are separate phenomenon, they possess overlapping properties. Prolonged stress may be sufficient enough to produce pain syndromes. These abnormal and

damaging responses to stress carry the potential to become part of an individual's 'signature' physiological response to stress, increasing ones risk of developing chronic pain conditions.

2.1.4. Anxiety

Considerable amounts of research have been conducted surrounding the association between pain and anxiety. Chronic pain sufferers tend to have elevated levels of non-pathological and pathological anxiety when compared to the general population (Asmundson et al., 2008; Beesdo et al., 2009; Gormsen, Rosenberg, Bach, & Jensen, 2010; Jensen, Turner, & Romano, 1994b; McWilliams, Cox, & Enns, 2003; McWilliams, Goodwin, & Cox, 2004). Further, it has been established that 'anxiety for pain' is the most prominent type of anxiety experienced by chronic pain patients (McCracken, Gross, Aikens, & Carnrike, 1996; McCracken, Zayfert, & Gross, 1992), and those that suffer from anxiety disorders are found to have heightened levels of pain reports (Campo et al., 2004; Carter & Maddock, 1992; Lautenbacher, Spernal, Schreiber, & Krieg, 1999; Schmidt & Telch, 1997; Spear, 1967). The question of whether pain leads to anxiety or anxiety leads to pain has also been investigated. At present, it is understood that there is a bi-directional relation between the two factors. That is, that pain can increase the likeliness that one will experience anxiety, and anxiety can increase the likeliness one will experience pain (Atkinson, Slater, Patterson, Grant, & Garfin, 1991).

Experimental studies have highlighted that it is important to consider what the anxiety is about, to understand its influence on pain. For example, Al Absi and Rokke (1991) induced subjects with either pain-relevant anxiety (about the pain they would experience in the following cold pressor task) or irrelevant anxiety (suggestion of ‘electric shock’) prior to the cold-pressor task. They found that subjects that were highly anxious about the cold-pressor task experienced more pain during the task than those that were highly anxious about a

possible electric shock. Similarly, Marlow (1981) found that experimentally induced non-pain related anxiety reduced subjects bias to report a pain-inducing pressure stimulus as painful. These findings indicate that the relationship between pain and anxiety may not always be unidirectional or positive. Further, they suggest that pain-relevant anxiety may enhance pain responsiveness, whereas irrelevant anxiety may inhibit pain responsiveness. This notion is supported by other studies indicating that heightened levels of non-pain related anxiety increases tolerance to the cold-pressor task, whereas heightened levels of pain-relevant anxiety decreases tolerance (Bobey & Davidson, 1970; Dougher, Goldstein, & Leight, 1987; Rhudy & Meagher, 2000).

Other laboratory based pain-anxiety studies have found different results. For example, some studies have failed to find a significant difference between the impacts of pain-relevant anxiety compared to non-pain related anxiety on pain tolerance (Cornwall & Donderi, 1988; Weisenberg, Aviram, Wolf, & Raphaeli, 1984). Some have suggested that it may not be whether anxiety is pain relevant or not that is of importance, rather it is whether a persons' *attention* is focussed on the pain or not (Arntz, Dreessen, & De Jong, 1994; Arntz, Dreessen, & Merckelbach, 1991). Thus, the relationship between anxiety and pain remains confounded and definite conclusions have yet to be established.

2.1.5. Attention

Attention has been recognised as one of the most prominent factors to modulate the experience of pain (Crombez, Van Damme, & Eccleston, 2005; Villemure & Bushnell, 2002). Pain places a demand on attentional resources which is critical in prompting appropriate behaviours to enable one to escape from the source of the pain. It also drives rest and recuperative behaviours allowing healing of painful injuries (Crombez, Eccleston,

Baeyens, & Eelen, 1996; Legrain et al., 2009). At the same time, the demanding impact that pain has on attention is one of the most debilitating aspects of the pain experience. It is found to make pain difficult to disengage from, leading to a reduction in attentional resources for other ongoing tasks (Crombez et al., 1996; Eccleston & Crombez, 1999). Eccleson and Crombez (1999) suggested that the relationship between pain and attention is not linear and is influenced by a number of factors related to pain such as intensity, threat value, novelty, and predictability.

Those experiencing high-intensity chronic pain are found to exhibit more disruption in attentionally demanding tasks compared to those with low-intensity chronic pain or no-pain (Eccleston, 1994; Eccleston, 1995). Furthermore, the higher one places the threat-value of pain, the more pain interferes with ones attention. This interference is found to be greater in those that also possess high levels of catastrophic thoughts about pain (Crombez, Eccleston, Baeyens, & Eelen, 1998a).

It is well established that heightened fear of pain increases the likeliness that one will become more vigilant to bodily sensations, and as a result, makes one more aware of painful sensations (Crombez, Eccleston, Baeyens, & Eelen, 1998b; Eccleston, Crombez, Aldrich, & Stannard, 1997; Peters, Vlaeyen, & Kunnen, 2002). Heightened pain related fear is shown to make pain harder to ignore and more difficult to disengage from (Crombez, Eccleston, Baeyens, Van Houdenhove, & Van Den Broeck, 1999; McCracken, 1997).

The novelty of a particular stimulus increases the likeliness that the stimulus will demand ones attention, and this process also applies to pain. Various studies have provided support for the notion that the more novel the pain appears, the higher its impact will be on

ones attention toward the pain. This is found to be particularly evident during the onset of pain (Crombez, Baeyens, & Eelen, 1994; Crombez et al., 1996; Crombez, Eccleston, Baeyens, & Eelen, 1997; Pavlov, 1927). Similar to the novelty of pain, it has been found that the expectation of a painful experience, results in a lowered disruption of attention and focus during ongoing tasks (Dawson, Schell, Beers, & Kelly, 1982). This indicates that attention is less disrupted when the experience of pain is anticipated. In summary, it is evident that attentional factors play a critical role in the experience of pain, though there are numerous moderating factors impacting this relationship. Therefore, the attention one places towards pain is an important factor to consider when investigating pain responses.

2.1.6. Depression

Pain and depression have a dynamic, bi-directional relationship, feeding off each other in a compounding cycle (Gureje, Simon, & Von Korff, 2001). In a chronic pain context, as the severity of pain increases, depressive symptoms and diagnosis of depression become more prevalent. Further, as depressive symptoms increase in severity, pain complaints are reported more frequently (Gambassi, 2009). Bair, Robinson, Katon and Kroenke (2003) estimated that 65% of patients clinically diagnosed as 'depressed' experience pain and that depression is present in 5-85% of pain conditions. The authors attributed the wide range of the latter finding to differences in the setting in which data was collected. Other studies have found similar trends (Gureje, 2008). Bair and colleagues (2003) also noted that CP patients with depression had increased amounts of pain related complaints and longer pain duration than patients not suffering depression.

Tang and colleagues (2008) investigated the effects of experimentally manipulating mood on a pain eliciting task, in chronic pain patients. They found that induced depressed

mood increased self-reported pain and decreased tolerance during the task. Conversely, induced positive mood resulted in significantly lower pain reports and greater pain tolerance. It has been suggested that it is low *self-efficacy* found in patients with depression which is associated with increases in catastrophising, leading to increases in pain intensity (Keefe, Lefebvre, Maixner, Salley, & Caldwell, 1997).

Research has shown that the impact of depression on chronic pain differs from the impact it has on acute forms of pain. For example, Dickens, McGowan, and Dale (2003) found that subjects with clinical depression had higher pain thresholds than subjects not suffering from depression. The authors mentioned that the contrasting findings between clinical and non-clinical settings may be due to the alterations in central and cortical processing found in chronic pain patients, compared to non-chronic pain patients, as well as contextual differences, leading to differences in pain perception. Conversely, more recent studies have shown that the positive correlation found between depression and chronic pain, also exists in the relationship between depression and pain in non-clinical settings (Euteneuer et al., 2011). It is clear that depression and pain impact each other, highlighting the importance of assessing depressive levels in research looking into pain responses, although further research is warranted before any firm conclusions can be established about the direction of this relationship.

2.2. Individual Factors

The characteristics of an individual have been found to influence both bottom up and top down aspects of the pain experience. Therefore, it is important to take account of these

features as well as other contextual factors found to contribute to the uniqueness of the pain experience for each individual. Ethnicity, gender and age will be discussed in this section. Particular emphasis will be placed on ethnicity as it is a key variable of interest.

2.2.1. Ethnicity

Ethnicity is a multidimensional concept which encapsulates the cultural, ancestral, historic, beliefs, biological features and physical characteristics of an individual (Campbell et al., 2008; Edwards, Fillingim, & Keefe, 2001a). Ethnicity encompasses physiological, psychological, and social dimensions hence, one can see how it may influence the pain experience at various levels, and fit well with the biopsychosocial view of pain. Pioneering work in the area of ethnicity and pain was done by Zborowski (1969), who found that Italian-Americans, Jewish-Americans, Irish-Americans, and 'White-Americans' each expressed unique attitudes and reactions to pain. Investigations following this pioneering work have added support for the notion that the ethnicity of an individual impacts their experience and attitudes towards pain in both clinical and non-clinical settings (Bates, 1987; Encandela, 1993; Fabian et al., 2011; Faucett, Gordon, & Levine, 1994; Green et al., 2003; Riley III et al., 2002; Thomas & Rose, 1991; White, Asher, Lai, & Burton, 1999).

The majority of literature investigating the relationship between ethnicity and pain has been based on African American, European American, and Hispanic ethnic groups (Hsieh, Tripp, Ji, & Sullivan, 2010). Riley and colleagues (2002) conducted an investigation into the differences in the experience of chronic pain in a large sample of Caucasian and African American individuals. Their key findings were that African American individuals reported significantly higher levels of pain unpleasantness, increased emotional reactions to pain, and exhibited a greater number of pain related behaviours than their Caucasian counterparts.

Interestingly, no differences were found in pain intensity levels between groups. This latter finding has also been found in various other studies (Edwards, Moric, Husfeldt, Buvanendran, & Ivankovich, 2005; Greenwald, 1991). A number of other studies have reported that African-Americans experience pain conditions, both acute and chronic, more severely than European-Americans (Chibnall, Tait, Andresen, & Hadler, 2005; Edwards, Doleys, Fillingim, & Lowery, 2001b; Selim et al., 2001; Sheffield, Biles, Orom, Maixner, & Sheps, 2000). These studies suggest a unique role of ethnicity in the experience of chronic pain.

In a review of experimental studies, Zatzick and Dimsdale (1990) suggested that there was no definite neurophysiological differences in pain detection across ethnic groups. However, subsequent research has revealed that there may be *genetic* differences by way of ethnicity that influence pain sensitivity and pain thresholds (Kim et al., 2004b; Skevington, 1995). Although, findings remain inconclusive and continue to be a topic of debate (Morris, 2001). Further research in experimental settings has revealed that African-American individuals tend to have higher experimental pain sensitivity leading to higher pain ratings, exhibit lower experimental pain threshold and tolerance, and rate experimentally induced pain as more unpleasant (Campbell, Edwards, & Fillingim, 2005; Edwards et al., 2001a; Edwards & Fillingim, 1999; Forsythe, Thorn, Day, & Shelby, 2011; Kim et al., 2004a; Rahim-Williams et al., 2007). A number of experimental studies have also been conducted comparing individuals of Asian ethnicity to other ethnic groups and have provided further support for the notion that ethnic-specific differences exist in the experience of experimentally induced pain (Clark & Clark, 1980; Fabian et al., 2011; Gazerani & Arendt-Nielsen, 2005; Knox, Shum, & McLaughlin, 1977; Komiyama, Kawara, & De Laat, 2007; Nayak, Shiflett, Eshun, & Levine, 2000; Watson, Latif, & Rowbotham, 2005). Nonetheless, as with research in clinical settings, experimental studies have shown conflicting findings

regarding the impact of ethnicity on specific dimensions of the pain experience. For example, Campbell and colleagues (2005) found that, though African Americans exhibited lower tolerances for various forms of experimentally induced pain compared to European Americans, these groups did not differ in pain threshold measures.

Some researchers have suggested that overall, responses to pain between ethnic groups may be quite similar but the factors which influence each ethnic group's pain experience are different and may contribute to the inconsistencies in the literature (Lipton & Marbach, 1984). Firstly, it is apparent that ethnic differences in pain are variable depending on which dimension of pain is being assessed. Studies have suggested that ethnicity has a greater impact on the motivational-affective dimension of pain than the sensory dimension (Edwards et al., 2001a; Edwards & Fillingim, 1999; Fabian et al., 2011; Greenwald, 1991; Lipton & Marbach, 1984). Secondly, closely related to which dimension of pain is assessed, is which measures are used to assess pain. For example, different effects of ethnicity on pain are found depending on whether pain is assessed using self-report measures or behavioural expressions of pain are being assessed (Campbell et al., 2005; Edwards et al., 2001a; Edwards & Fillingim, 1999; Greenwald, 1991; Hastie et al., 2005; Lipton & Marbach, 1984; Riley III et al., 2002). Thirdly, ethnic differences in pain are found to vary depending on the site or area of the body being assessed (Hastie et al., 2005; Moore & Brodsgaard, 1999; Riley III et al., 2002). Fourthly, it has been identified that various psychological factors such as hypervigilance and worry contribute to, and possibly moderate, the influence of ethnicity on pain thus highlighting the importance of accounting for such factors (Campbell et al., 2005; Hastie et al., 2005; Lipton & Marbach, 1984). To summarise, it is clear that the relationship between ethnicity and pain is *moderated* by a number of factors including the dimension of

pain being assessed, measures used to assess pain, and the site on the body the pain is being experienced.

Various mechanisms have also been proposed to *mediate* the relationship between ethnicity and pain. As highlighted by Bates (1987), an individual's general attitudes, expectations, emotional self-expression and experiential meanings are in part, developed through observing the reactions and behaviours of others around them who are similar in identity. This notion, termed 'observed learning' is the key feature of Bandura's (1977) *social learning theory*, and has been found to be a strong factor in predicting health behaviours (Conner & Norman, 2005; Rosenstock, Strecher, & Becker, 1988). Therefore, it has been suggested that it is through social learning, that ethnic-specific cultural beliefs, ideas, and values regarding pain, may influence ones experiences of pain (Bates, 1987; Davidhizar & Giger, 2004; Lasch, 2002; MacGregor, Griffiths, Baker, & Spector, 1997; Tomasello, Kruger, & Ratner, 1993). For one to learn and adopt ethnic-specific cultural views about pain from their own ethnic group, the individual would have to be exposed to people of similar ethnic identity. Thus, there is an obvious need to assess how much one actually affiliates, or identifies, with their ethnic group when attempting to draw conclusions on the influence of ethnicity on pain (Edwards et al., 2001a; Rahim-Williams et al., 2007). This critical component was left out of the majority of the studies previously discussed and hence is a limitation in the literature.

Pain catastrophising has also been suggested to be a mediating factor of ethnic differences in the pain experience (Fabian et al., 2011). Specifically, studies have shown that higher catastrophising levels in African Americans and Chinese individuals are associated with lower pain tolerance, and higher pain reactivity (particularly in the affective-dimension

of pain) in these ethnic groups compared to European individuals (Forsythe et al., 2011; Hastie, Riley III, & Fillingim, 2004; Hsieh et al., 2010).

Other researchers have suggested that ethnic differences in pain may be mediated by differences in endogenous noxious inhibitory systems. Campbell and colleagues (2008) experimentally tested the DNIC in African Americans and European Americans. They found that the impact of the DNIC was higher in European Americans than African Americans, contributing to greater verbal expressions of pain in this group. At this stage, research surrounding ethnic differences in pain inhibitory systems is still in its early days and thus, whether pain inhibitory systems are or are not mediators of ethnic differences in pain remains inconclusive (Campbell et al., 2008).

Findings such as African Americans having higher cardiovascular (HR and BP) reactivity during painful medical procedures than European Americans, have sparked speculation about other physiological mechanisms which may mediate ethnic differences in the pain experience (McNeilly & Zeichner, 1989). Ethnic minorities are more likely to have experienced longer periods of distress than majority groups, due to discrimination over time (Clark, Anderson, Clark, & Williams, 1999). Therefore, it has been proposed that they are more likely to have persistently high levels of sympathetic nervous system activation, in addition to other physiological factors, leading to excessive cardiovascular reactivity to pain, resulting in a poorer ability cope with pain (Clark et al., 1999; Edwards et al., 2001a).

The recognition of findings surrounding the impact of ethnicity on the pain experience have particular importance to New Zealand, due to the high level of ethnic diversity of New Zealand's population (Statistics New Zealand: Tatauranga Aotearoa, 2006). Epidemiological

data suggests that within New Zealand, minority groups exhibit differences in the experience of pain when compared to each other, and when compared to the majority (people of European ethnicity). Specifically, the New Zealand health survey conducted in 2006/2007 reported that Maori men were significantly more likely to present with chronic pain conditions compared to men in the total population. Additionally, that Pacific women, were significantly less likely to present with chronic pain conditions, compared to the total population (New Zealand Ministry of Health, 2008). Coggan, Norton, Roberts and Hope (1994) revealed that Pacific Island nurses had a higher point prevalence and average annual prevalence of nursing related back pain than Maori and European individuals. These findings suggest that there are ethnic differences in the experience of pain within the New Zealand population.

Few studies investigating the relationship between pain and ethnic groups in New Zealand have been published, and hence a gap in the literature remains. Although, from the few investigations that have taken place, it is apparent that differences in the understanding and perception of pain may exist between European, Maori, and Pacific peoples of New Zealand. Magnusson and Fennell (2011) conducted a qualitative investigation looking at how Maori people perceive and express pain. The authors interviewed kaumatua (Maori tribal leaders/elders) and Maori healthcare providers. They found that Maori view and experience pain holistically, in line with the biopsychosocial understanding of pain. Reference was made to the 'Te Whare Tapa Wha' model when discussing pain. Te Whare Tapa Wha is a holistic model of health and well-being from a Maori perspective, and incorporates a mental and emotional component (te taha hingaro), a family and community component (te taha whanau), a physical component (te taha tinana) and a spiritual component (te taha wairua).

Each component is expressed in terms of four supporting walls that make up a complete house, or 'whare', representing health and well-being (Durie, 1985).

Words used to describe pain emphasised the universality of the pain experience for Maori. Descriptions were found to encompass specifics about the pain, e.g. the location, intensity, and temporal qualities of pain. Emotions linked to the pain were also described as well as the possession of the pain e.g. 'my pain' or 'his pain'. This latter feature was found to be of importance as participants placed emphasis on helping others in pain, when describing the experience of pain. The universality of the pain experience was also captured by Thomson (1989) who noted that, for Maori, pain is intertwined with the surrounding environment. For example, if a person falls over on the ground and injures themselves, they must not only consider their own pain, but also the pain they have caused to the ground they fell on, and show it respect. Magnusson and Fennell (2011) also highlighted that elderly Maori were less likely to report pain or seek help for pain, regardless of being aware of the benefits of seeking help. Additionally, males were reportedly less likely to respond to pain in general, than females.

Another key finding was that particular emphasis was placed on the differentiations and overlap of *spiritual* pain and *physical* pain. Others have also noted the strong spiritual component of pain as described by Maori. For example, Thomson (1989) noted that for Maori "...mamae (pain) may be regarded as an attacking force directed at the mauri (life force) or directed toward the wairua (spiritual force)...". This feature was also captured in Magnusson and Fennell's (2011) study, as it was found that pain was commonly associated with disease, and described as a demon preying on one's life force. Further, it was found that

Maori had a reluctance to express that they were in pain to their whanau, as they did not want to burden others around them with their own issues.

Another theme that emerged was the 'privacy' of the Maori people in regards to expressing health concerns, including issues regarding pain. These topics were reported to be discussed only with close whanau. Non-disclosure of health concerns, including pain to others outside of whanau, was seen as a sign of positive coping. Bassett (2002) reported that Maori physiotherapy clients had a common perception of 'no pain, no gain', hence did not feel the need to report pain to their physiotherapist during physiotherapy. Magnusson and Fennell (2011) also found that the themes which emerged linked back to the way participants preferred treatments for pain. Magnusson and Fennell's (2011) study was a small scale investigation and results are not generalisable to all Maori of New Zealand. However, it did capture some key features which may be unique to the Maori view and experience of pain.

No such study has been published on Pacific people. Hence, the experience of pain in this group remains unclear. Various models have been developed which have been proposed to encapsulate Pacific people's views of health. One of the predominant models is the Fonofale model (Pulotu-Endemann, 2001). The Fonofale model proposes a holistic view of health, encompassing family, culture, physical, spiritual, mental, and other (age, gender, sexual-orientation, and socioeconomic status) features that contribute to an individuals' health and well-being (Pulotu-Endemann, 2001). These dimensions are similar to the dimensions expressed in Te Whare Tapa Wha, particularly the presence of a spiritual dimension. Furthermore, pain is found to be a part of many cultural and traditional rituals found in the Pacific Islands. For example, in Samoan culture withstanding the pain of traditional body tattooing for a young male is part of the overall transition into manhood (Mead, 1961).

Therefore, it likely that Pacific people also have unique views of pain, leading to distinctive ways of coping with pain and differences in the pain experience.

From what has been discussed, it is clear that ethnic differences in the perception and experience of pain exist. Yet, these differences are perhaps, specific to the characteristics or dimension of pain being investigated. Some evidence suggests that Maori, Pacific and European ethnic groups in New Zealand each have unique, ethnic-specific views on pain, leading to differences in how pain is experienced and reported within these groups. Nonetheless, the data surrounding differences in the pain experience between these groups is still in its speculative stages and further investigation is warranted.

2.2.2. Gender

It is well established that there are differences between the way men and woman experience pain. Women have been found to have a higher pain prevalence compared to men (Keogh, 2009). Specifically, woman are found to experience more severe and frequent pain, pain that lasts for a longer duration, and are more at risk of suffering recurrent pain (Unruh, 1996). Further, it has been found that women tend to report higher pain levels in chronic pain conditions than men (Robinson, Wise, Riley, & Atchison, 1998). Gender differences in pain levels have been found to be most pronounced in head, facial and abdominal regions of the body (Von Korff, Dworkin, Le Resche, & Kruger, 1988).

Experimental studies have revealed that woman tend to have lower pain thresholds, lower pain tolerance, and experience higher pain levels during experimentally induced pain when compared to men (Fillingim & Maixner, 1995; Forsythe et al., 2011; Riley III, Robinson, Wise, Myers, & Fillingim, 1998). Other research suggests that pain catastrophising

may account for gender differences in pain sensitivity, pain reporting, pain tolerance and behavioural expressions of pain in both laboratory settings (Dixon, Thorn, & Ward, 2004; Edwards et al., 2004) and clinical settings (Keefe et al., 2000; Keogh & Eccleston, 2006; Keogh, McCracken, & Eccleston, 2005; Sullivan et al., 2000).

2.2.3. Age

Studies investigating the relationship between age and pain have produced mixed findings. At present, clinical studies tend to support the notion that there is an increase in pain perception with age. It has been shown that there is an increase in the prevalence and disabling effects of chronic pain as age advances (Gibson, 2003; Helme & Gibson, 2001; Manchikanti, Singh, Datta, Cohen, & Hirsch, 2009; Thomas, Peat, Harris, Wilkie, & Croft, 2004; Tsang et al., 2008; Verhaak, Kerssens, Dekker, Sorbi, & Bensing, 1998). Experimental studies have produced less conclusive results, with findings showing that pain perception either increases, decreases, or remains unchanged as age advances (Gibson & Farrell, 2004; Helme, Meliala, & Gibson, 2004; Lautenbacher, Kunz, Strate, Nielsen, & Arendt-Nielsen, 2005). It has been suggested that methodological differences between studies, particularly the type of pain used (e.g. thermal, pressure, or electric), duration of pain induction, and site on the body the pain is induced, contribute to these equivocal findings (Cole, Farrell, Gibson, & Egan, 2010; Lautenbacher et al., 2005).

Despite equivocal experimental findings it has been noted that in general, elderly individuals tend to have slight increases in pain threshold and a moderate to high decrease in pain tolerance compared to younger individuals (Edwards, Fillingim, & Ness, 2003; Gibson, 2003; Kerns, Sellinger, & Goodin, 2011). Edwards and colleagues (2003) suggested that this was due to the deterioration of endogenous pain inhibitory mechanisms as individuals age.

Other studies have suggested that it may be the result of age related alterations in pain processing fibres. Specifically, that older adults rely less on fast acting A-δ pain fibres, and more on slower acting C-fibres (Chakour, Gibson, Bradbeer, & Helme, 1996). Additionally, there is some evidence which indicates that these changes in pain perception are partially the result of age related reductions in the activation of the central nervous system during pain processing (Ferrell, 1995; Helme & Gibson, 1999).

2.3. Summary

The experience of pain is influenced by a number of psychosocial and individual factors. A number of these factors have been discussed in this chapter, including catastrophising, worry, stress, anxiety, attention, depression, ethnicity, gender, and age. Particular emphasis is placed on the influences of ethnicity, catastrophising, and general worry on pain, as they are the key variables of interest in the current study. It is important to note that, though these factors were discussed independently, they are not mutually exclusive and have interactive impacts on pain. In particular, worry, anxiety and stress are found to be closely related, each possessing a negative emotional component as well as having similar physiological influences on pain (Brosschot et al., 2006; Lovibond & Lovibond, 1995; Muris, Roelofs, Rassin, Franken, & Mayer, 2005). Furthermore, worry tends to lead to anxiety, and is found to be a prominent feature of Generalised Anxiety Disorder (Brown, Antony, & Barlow, 1992; Gana, Martin, & Canouet, 2001). These factors help explain how two people that experience the same amount of injury can have vastly different perceptions and experiences of pain thus, leading to differences in the way they report pain. Therefore, continual investigation into how these factors interact with each other and influence the physiological mechanisms driving pain, and vice versa, is of merit.

CHAPTER THREE

Physiological Measure of Pain: Heart Rate Variability

Movement away from the theoretical viewpoint of 'mind-body dualism' has led to the emergence of fields such as, *psychophysiology* which have adopted electrocardiographical measures including Heart Rate Variability (HRV). HRV is commonly used as an index of cardiovascular function as well as providing insight into the body's ability to effectively respond environmental stressors such as pain. The current chapter introduces HRV by firstly explaining its underlying mechanisms, followed by a summary of various individual factors which impact HRV and that are relevant to the present study. At present, research surrounding the relationship between HRV and pain is scarce and findings remain inconclusive. However, the chapter will conclude with an outline and summary of the current findings surrounding this relationship.

3.1. Defining Heart Rate Variability

HRV can be described as the temporal variation between sequences of consecutive heart beats, i.e. the variation in time of the inter-beat-intervals in heart rate (HR) (Karim, Hasan, & Ali, 2011). In healthy individuals, HR is not uniform and rhythmic fluctuations exist between heart beats (Malik & Camm, 1995). This variation can also be viewed as the amount of fluctuation around a mean HR (Conny, Kollee, Hopman, Stoelinga, & Herman, 1993). The fluctuations are oscillatory in nature and are regulated by the two main branches of the autonomic nervous-system: the sympathetic nervous system and the parasympathetic nervous

system. Information is sent via these systems from the brain to the sinoatrial (SA) and atrioventricullar (AV) nodes of the heart where it is processed. The SA node is of primary importance to HRV as it plays a critical role in modulating the pace at which the heart beats (Levy & Pappano, 2007). The parasympathetic nervous system tends to decelerate HR, and the sympathetic nervous system tends to accelerate HR (Malik & Camm, 1995).

At rest, the input from the parasympathetic nervous system dominates cardiovascular control (Levy, 1990). This predominance of HR by the parasympathetic nervous system has been termed *accentuated antagonism* (Uijtdehaage & Thayer, 2000). Early research supporting this phenomenon was conducted by Levy and Zieske (1969) who experimentally manipulated the sympathetic and parasympathetic branches of the autonomic nervous system in dogs. As expected, they found that increasing sympathetic activity accelerated HR. However, they also found that the increase in sympathetic activity was largely suppressed by a powerful, simultaneous increase in parasympathetic activity. Other supporting evidence has been provided by studies which have shown that blockage of the parasympathetic input to the cardiac system results in an almost complete disappearance of HRV (Dellinger, Taylor, & Porges, 1987). In the absence of any autonomic control, the heart beats around 100 beats per minute (bpm), however due to the inhibitory effects of the vagus (the primary nerve fibre of the parasympathetic nervous system), resting HR averages at around 70bpm. This inhibition is essential for cardiovascular flexibility, responsiveness, and stability (Berne & Levy, 2001; Levy, 1990; Verrier, 1987).

Vagal outflow is found to be suppressed during autonomic and behavioural responses to stress, periods of sustained attention, and states of increased mental effort. In general, short term vagal suppression has been related to better performance in attention-demanding tasks

(Porges & Byrne, 1992; Porges, Doussard-Roosevelt, & Maiti, 1994). Vagal suppression is different from vagal reactivity. Vagal reactivity is the ability of the parasympathetic nervous system to efficiently respond to short term cognitive, sensory or visceral demands placed on the cardiac system. It can be indicative of the adaptability of the nervous system. Both reduced levels of vagal outflow (leading to low HRV) and atypical vagal reactivity and recovery are linked to poorer cardiovascular regulation (Porges & Byrne, 1992; Sztajzel, 2004).

As mentioned earlier, input from the sympathetic nervous system and the parasympathetic nervous system create the oscillatory rhythms in HRV. These oscillatory rhythms are the product of separate, superimposed frequency components which are measured in cycles per second (Hertz; Hz) (van Ravenswaaij-Arts, Kollee, Hopman, Stoelinga, & van Geijn, 1993). Research has indicated that the sympathetic nervous system and the parasympathetic nervous system make power-specific contributions to the HR 'power spectrum'. These power contributions are used as an index to assess the amount of variance in HR caused by each branch of the autonomic nervous system (Akselrod et al., 1981). The vagus nerve acts fast and exerts its influence predominantly over the high frequency (HF; 0.15 - 0.40 Hz) power spectrum of HRV. The sympathetic mediators of HR, including activity of the beta-adrenergic and alpha-adrenergic systems, are slower acting than the parasympathetic mediators and exert their influence over longer periods of time. Thus, they are reflected in the low or middle frequency (LF; 0.01 - 0.15 Hz) power spectrum of HRV (Akselrod et al., 1985). Importantly, the LF component of HRV has also been found to consist of vagal input and thus, is proposed to reflect modulation of the cardiac system by both branches of the autonomic nervous system. On this basis, HRV analysis is a better indication of parasympathetic modulation of HR than sympathetic modulation of HR. Higher

HRV at rest is generally associated with healthier cardiovascular systems, whereas reduced HRV at rest is associated with decreased emotional functioning, delayed physiological recovery from stress, hindered autonomic functioning, lower cognitive functioning, and increased morbidity and mortality (Malik, 1996; Thayer, Ahs, Fredrikson, Sollers III, & Wager, in press; Thayer & Lane, 2007; Weber et al., 2010).

Theoretical Underpinnings

The neural modulation of the cardiovascular system has been found to be influenced by both 'top-down' and 'bottom-up' regulatory mechanisms. One of the most prominent bottom-up mechanisms to influence HRV is the *baroreflex*. The functional purpose of the baroreflex is to uphold cardiovascular function by stabilising Blood Pressure (BP) through alterations in HR (Bristow, Honour, Pickering, Sleight, & Smyth, 1969). The baroreflex is a closed-loop feedback system in which information is relayed back and forth from the cardiovascular system to the central control systems in the brain (Ringwood & Malpas, 2001). Specifically, changes in BP are detected by baroreceptors, located in the transverse arch of the orta, and the carotid sinus. Information about the rise or fall in BP and the rate of change are sent to the central processing areas of the brain. Cortical structures in the brainstem then process this information and transmit signals to the heart and associated blood vessels via parasympathetic and sympathetic pathways to adjust HR and stabilise BP accordingly (Vaschillo, Vaschillo, & Lehrer, 2006).

Various top-down mechanisms which influence the modulation of the cardiovascular system have also been revealed. Fundamental work in this area was conducted by Benarroch

(1993), who proposed the idea of a 'central autonomic network', a regulating system based in the central nervous system. The central autonomic network is proposed to control neuroendocrine, visceromotor, pain, and behavioural responses to environmental demands in order for the system to respond efficiently to ensure survival (Benarroch, 1993; Thayer & Lane, 2000; Thayer & Lane, 2009). The key brain structures involved in the autonomic network include the insular and anterior cingulate cortices, amygdala, hypothalamus, PAG, parabrachial nucleus, the nucleus of the solitary tract, ventrolateral reticular formation and raphe nuclei (Benarroch, 2006). Output of the autonomic network is transmitted primarily via the sympathetic and parasympathetic nerves to various areas of the body including the heart. Specifically, signals are sent from the brain via the stellate ganglion and vagus nerve to the SA node of the heart, subsequently innervating the heart and influencing HRV. Importantly, sensory information from the heart is also found to feedback into the autonomic network. Thus, HRV is not only seen as a product of the interaction between the parasympathetic and sympathetic controls of the heart, but also more broadly as an indicator of the bi-directional feedback system between the central nervous system and autonomic nervous system (Thayer, Hansen, Saus-Rose, & Johnsen, 2009).

Thayer and Lane (2000) expanded the central autonomic network model, and presented the model of neurovisceral integration. The neurovisceral integration model encompasses the central autonomic network, and the central autonomic network makes up the major physiological centres involved in top-down autonomic regulation. The model integrates brain structures which control autonomic, attentional, and affective systems (Thayer & Lane, 2000). It also proposes that under normal circumstances, the activity of the amygdala is under tonic inhibition by prefrontal cortical areas such the orbitofrontal cortex and the medial prefrontal cortex. It is this inhibition that is believed to mould neural

responses to environmental demands (Thayer et al., 2009). Support for the association between prefrontal cortical areas of that brain and HRV has been provided by both neuroimaging research and pharmacological blockage studies (Ahern et al., 2001; Lane et al., 2009; Lane, Reiman, Ahern, & Thayer, 2001; Thayer et al., in press). Furthermore, it has been suggested that the disinhibition of the amygdala leads to an increase in HR and reduction in HRV (Thayer & Lane, 2000). Such disinhibition is believed to occur in periods of stress or other states of emotional arousal (Thayer & Lane, 2007). Supporting evidence has been provided by way of clinical studies which show that a range of anxiety disorders have been associated with chronically low HRV and the development of cardiovascular conditions such as hypertension (Cogiamanian et al., 2010; Gorman & Sloan, 2000). Thus, the neurovisceral integration model provides a scientific explanation to how factors such as worry, anxiety, and other psychosocial variables my indirectly impact HRV, and reinforces the idea that HRV may serve as an index of the brains integrative systems. Thus, given the brain-heart connection one's capability to appropriately respond to environmental demands and other stressors is reflected in HRV. Therefore, HRV may be used as a measurement of how well the upper and lower brain centres communicate with each other, and hence reflect physiological function.

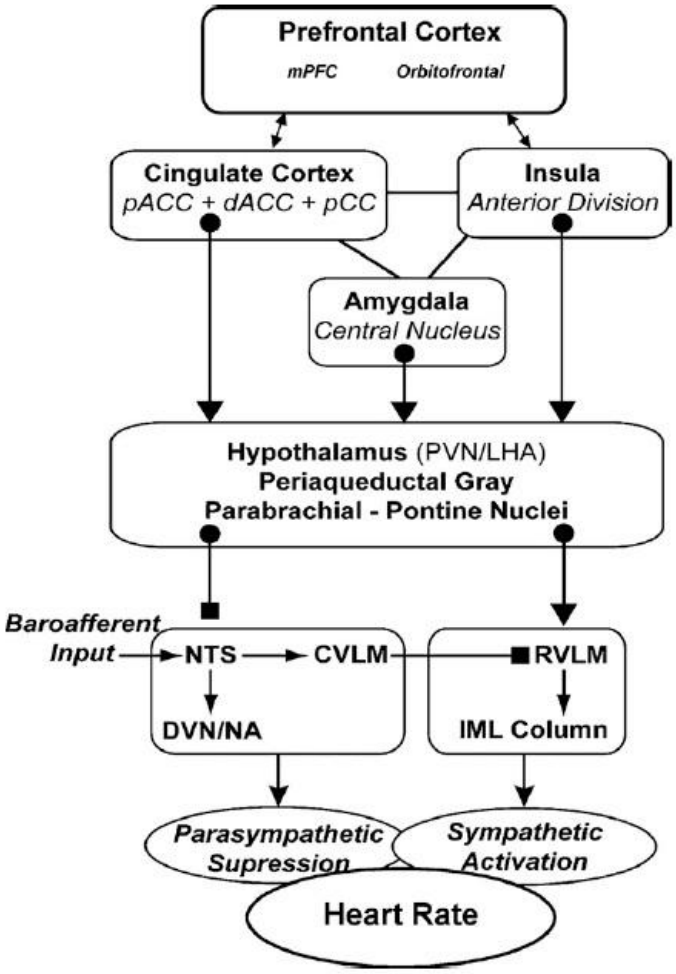


Figure 2. Schematic diagram of the systems involved in the central autonomic network as captured by the neurovisceral model. These are the proposed pathways by which the prefrontal cortex influences HRV, and subsequently HR. Reprinted with permission from Thayer and Lane (2009).

3.3. Individual Factors Influencing Heart Rate Variability

3.3.1. Age

Aging, particularly beyond adulthood has been found to be associated with reductions in parasympathetic modulation of HR, naturally leading to a decline in HRV over time (Jensen-Urstad et al., 1997; Lipsitz, Mietus, Moody, & Goldberger, 1990; Ryan, Goldberger, Pincus, Mietus, & Lipsitz, 1994). Specifically, it has been found that in those aged 65 years and over, there was a marked reduction in the fluctuation of HR, and decreased LF and HF power in HRV (Lipsitz et al., 1990; Stein, Ehsani, Domitrovich, Kleiger & Rottman, 1999). This decline was found to be steepest at ages 65 to 70 years and level out around the age of 75 years (Stein, Barzilay, Chaves, Domitrovich, & Gottdiener, 2009). The aging process has also been found to cause decreased compliance of the arterial walls, leading to a reduction in the sensitivity of the baroreflex mechanism leading to a further decrease in the parasympathetic modulation of HR (Laitinen et al., 1998; Shi et al., 2008).

3.3.2. Gender

Disparities in HRV have also been found to exist between males and females, although the direction of this difference remains unclear. The majority of literature supports the notion that HF power (parasympathetic modulation of HR) is higher, and LF power is lower, in women compared to men at rest and during task (Antelmi et al., 2004; Carter, Banister, & Blaber, 2003; Huikuri et al., 1996; Ryan et al., 1994). Various other findings have indicated that total HRV power is lower in women compared to men (Jensen-Urstad et al., 1997; Ramaekers,

Ector, Aubert, Rubens, & Van de Werf, 1998; Umetani, 1998). However, it is suggested that such HRV disparities are age specific. Agelink and colleagues (2001) found that young and middle aged women had lower LF power and LF/HF ratio compared with age-matched men, whereas no gender differences were observed in the total HF power. In addition, Stein, Kleiger, and Rottman (1997) found that for men, initially levels of overall HRV were higher compared to females, although as they became older there was a global reduction in HRV, particularly in the naturally occurring circadian fluctuations in HRV. Furthermore, the authors found that for women, aging was primarily associated with declines in shorter term fluctuations in HRV rather than circadian fluctuations. Kuo and colleagues (1999) found that such disparities in HRV tended to diminish after the age of 60 years. Other findings have indicated that women have larger decreases in HRV in response to a short-term stressor than men (Li et al., 2009). This suggests that women have an enhanced vagal responsiveness to stress (vagal withdrawal) compared to men, supporting the notion of enhanced parasympathetic regulation of HR in females over males (Porges, 1995). In sum, though gender differences in HRV are apparent, they are a product of one's age and are dependent on the characteristics of the HRV measure used.

3.3.3. Physical Activity

Exercise levels have also been found to impact HRV. A recent meta-analysis by Sandercock, Bromley and Brodie (2005) analysed 13 studies which investigated the effect of exercise on HF power of HRV. Overall, they found that these studies provided support for the concept that regular, long-term aerobic exercise led to a healthier cardiovascular system. They established that these benefits can for the most part be attributed to alterations in the neuroregulation of the heart, specifically through enhanced vagal (parasympathetic)

modulation of HR. This inference was drawn from studies which revealed that enhanced exercise levels directly increased HF power of the HRV power spectrum.

3.3.4. Ethnicity

The majority of research surrounding the relationship between ethnicity and HRV has taken place in the United States of America. Nonetheless, findings suggest that disparities do exist, although the direction of these disparities remains inconclusive. Some literature suggests that African Americans have lower HRV levels compared to European Americans (Lampert, Ickovics, Horwitz, & Lee, 2005). This lowered HRV in African Americans has been attributed to the chronic stress experienced by this group, associated with being both an ethnic minority (e.g. racial discrimination of minority ethnic groups) and being of a lower socioeconomic status compared to the general population (Anderson, McNeilly, & Myers, 1992; Lampert et al., 2005). Conversely, other studies have provided evidence that AAs have higher HRV levels than European Americans, particularly at rest (Gutin et al., 2005; Guzzetti et al., 2000; Li et al., 2009; Liao et al., 1995; Urbina, Bao, Pickoff, & Berenson, 1998; Wang, Thayer, Treiber, & Snieder, 2005). Furthermore, a recent study conducted by Martin and colleagues (2010) revealed being African American, Hispanic, or Asian, was predictive of higher HRV levels than those of European American ethnic group. The authors noted that African American ethnicity was the most consistent predictor of high HRV levels. Wang and colleagues (2005) revealed that these ethnic differences were present from a young age. Though the findings discussed are conflicting and are only applicable to the ethnic groups they involve, they provide support for the notion that ethnicity may have an impact on autonomic function, particularly HRV. Further research into autonomic differences between ethnic groups is warranted (Thayer, Wang, & Snieder, 2006).

Health disparities and links between racism and poorer health outcomes exist when looking at major ethnic groups in New Zealand (Chan et al., 2008; Harris et al., 2006). For example, cardiovascular disease, one of the leading causes of mortality in NZ is highest in Maori and Pacific Island peoples of NZ. Specifically, all forms of cardiovascular disease are higher in Maori people compared to the general population. Also, Pacific Island people have been found to have the highest rate of mortality for cerebrovascular disease and hospital discharge rate for stroke (MacDonald, 2009). Thus, any research which aims to provide insight into the physiological functioning of various ethnic groups in NZ would be of merit.

3.4. Heart Rate Variability and Pain

A link between the pain processing and the human nervous system was proposed over a century ago by William James (1884). Since HRV is an index of autonomic function there is obvious potential for a relationship to exist between HRV and pain. James (1884) suggested that pain sensations are partly due to changes in BP and blood flow. Further investigations into this relationship have revealed that the areas of the brainstem and cortical regions that modulate the cardiovascular system closely overlap with those which control pain (Lovick, 1993; Oberlander & Saul, 2002; Randich & Maixner, 1984). An early experimental study conducted by Dowling (1983) revealed that HR during the warning period of the cold pressor task significantly predicted pain tolerance during the task. Loggia, Juneau, and Bushnell (2011) conducted an experimental study which found that HR correlated highly with pain perception when subjects were exposed to a painful heat stimulus. The authors noted that gender of the experimenter and subject had a major role in influencing the autonomic response to pain. Moltner, Holzl, and Strain (1990) found that initial HR responses to experimental pain (three to six seconds) were highly correlated with stimulus intensity,

suggesting a strong link between initial autonomic responses with the sensory component of pain. In addition, longer lasting HR responses (after six seconds) were highly correlated with subjective judgements of the stimulus, indicating a strong link between longer-lasting autonomic responses with the affective/motivational or cognitive dimensions of pain. Researchers have inferred that these autonomic reactions to pain are in line with typical autonomic reactions to general stressors, and act as an adaption mechanism to maintain homeostasis (Appelhans & Luecken, 2008; Heller et al., 1984; Terkelsen, Molgaard, Hansen, Andersen, & Jensen, 2005). The mechanisms underlying fluctuations in autonomic activity caused by general stress and pain responses have been found to be associated with intrinsic adjustments of the sympathetic nervous system and parasympathetic nervous system, which can be indexed by HRV (Tousignant-Laflamme & Marchand, 2009).

Mean HRV has been found to decrease in situations of stress and pain as it becomes predominantly sympathetically mediated in these situations (Karas et al., 2008). Research into pain treatment has shed light on what is known as ‘sympathetically maintained pain', i.e. pain caused by sympathetic nerve supply to an affected area (Baron & Janig, 2003). This is supported by findings which showed that, in some cases, blockage of sympathetic nerve supply to a painful area eliminated pain (Treede, Davis, Campbell, & Raja, 1992).

Nonetheless, in chronic pain the over-activation of the sympathetic nervous has been found to be occasionally accompanied by an under-activation of the parasympathetic nervous (Cohen et al., 2000; Furlan et al., 2005). Further, Bruehl, Chung, Ward, Johnson, and McCubbin (2002) found that in the chronic pain population, pain sensitivity positively correlated with blood pressure. These findings further substantiate a link between pain and the autonomic nervous system and some researchers argue that it may actually be

malfunctions in the autonomic nervous that generate and maintain chronic pain (Janig, 1992; Passatore & Roatta, 2006; Schott, 1999). That notion has been supported by evidence showing that increases in parasympathetic nervous activity can lead to muscular pain relief in individuals suffering chronic pain (Cottingham, Porges, & Lyon, 1988a; Cottingham, Porges, & Richmond, 1988b; Ishii, Niioka, Watanabe, & Izumi, 2007; Sakai et al., 2007) . It has been postulated that this muscular pain relief is a result of increases in peripheral blood flow caused by increases in parasympathetic activity (Ishii et al., 2007). Conversely, Terkelsen, Andersen, Molgaard, Hansen, and Jensen (2004) found that the analgesic effects of distraction on experimentally induced nerve pain, was associated with decreased LF, HF, and total HRV power. These studies reinforce that the mechanisms associated with the autonomic nervous system, particularly in the brain, may also have a role in the perception of pain. Various researchers have investigated this notion and found support for common mechanisms underlying both the autonomic nervous system and the perception of pain.

Green and colleagues (2006) prospectively investigated the analgesic effects of deep brain stimulation of the PAG area of the midbrain (important in both cardiovascular control and pain modulation) in chronic pain patients. Their results showed that the amount of analgesia induced by deep brain stimulation was linearly associated to the amount of reduction in BP. Further, this reduction in BP was related to a decrease in sympathetic nervous system activity. This finding highlighted the involvement of the midbrain, particularly the PAG region in the relationship between chronic pain and autonomic nervous system function. Additionally, it elucidated the fact that that the reduction of sympathetic activity may be important in controlling pain.

Nonetheless, conflicting evidence exists which shows no difference in HRV parameters between chronic pain patients and healthy individuals. For example, Harman (1997) found that HRV patterns across sleep cycles did not differ in patients suffering chronic low back pain when compared to healthy individuals. Additionally, Storella and colleagues (1999) found that chronic pain patients who experienced significant pain reduction after treatment for a hemiated disk, had no differences in standard measures of HRV compared to those who did not experience a reduction in pain. However, when using non-standard measures of HRV (point correlation dimension), HRV was found to be higher in those patients who reported a pain reduction than those who did not.

Appelhans and Luecken (2008) conducted an experimental study investigating the relationship between resting frequency domain measures of HRV and pain sensitivity in healthy young adults. Their findings revealed that higher resting LF measures of HRV were significantly predictive of lower pain unpleasantness and higher pain thresholds during exposure to a painful thermal stimulus. The authors believed that this result is consistent with the viewpoint that the negative emotionality associated with pain, together with broader affective components, drive homeostatic adjustments to a painful situation. On this basis, they propose that pain may act as a functional mechanism to elicit endogenous baroreflex mediated pain inhibition (Appelhans & Luecken, 2008). This concept is firmly supported by the findings which showed that states of high negative arousal usually elicited acute increases in BP (Bruehl & Chung, 2004; Mini, Rau, Montoya, Palomba, & Birbaumer, 1995). Nonetheless, researchers have also found conflicting results regarding the relationship between pain and HRV in healthy individuals. In an experimental study, Meeuse and colleagues (2010) found no relationship between pain intensity and HRV parameters when exposing healthy individuals to a painful heat stimulus, although, this study did have limited

insight into various mediator and moderator variables that may have contributed to such effects. Therefore, though HRV is commonly used as a measure of physiological stress reactivity such as that experienced during pain (Karas et al., 2008), more investigation is warranted to assess various mediator and moderator variables before any definitive conclusions can be made about this relationship.

3.5. Summary

Non-uniform, oscillatory fluctuations in HR (HRV) exist in all healthy cardiovascular systems. These are associated with the cardiac systems ability to respond and recover from situational demands placed on it. HRV is an electrocardiographical measure which reflects the communication between the central nervous system and the peripheral autonomic receptors. This chapter has introduced HRV and discussed its underlying components. HRV is influenced by inputs to the heart from the two primary branches of the autonomic nervous system including the sympathetic nervous system and parasympathetic nervous system. However, it is predominantly modulated by the parasympathetic nervous system. Activation of the parasympathetic nervous system decelerates HR, whereas activation of the sympathetic nervous system accelerates HR.

The neurovisceral integration model provides a scientific explanation of how HRV is influenced by both bottom up and top down mechanisms. Bottom up mechanisms include the baroreflex, and top down mechanisms include the influence of various psychosocial variables which alter the state of the brain. Each of these factors alters the neural output from the autonomic nervous system, which in turn alters HRV subsequently changing HR. On this

basis, fluctuations in HRV are found to be a window into the bi-directional feedback pathways between prefrontal cortical areas and lower hindbrain autonomic centres. Hence, HRV can also be used as an index of physiological regulatory function in response to environmental stressors such as pain. Age, gender, level of physical activity and ethnicity have also been found to influence an individual's HRV and each provide insight into the cardiovascular function of specific groups.

This chapter concluded with a discussion outlining research which has investigated the relationship between pain and HRV. At this stage, it is clear that areas of the brain which are responsible for the modulation of the cardiovascular system are also involved in the modulation of pain. Chronic pain patients have been found to have reduced HRV due to an over-activation of sympathetic nervous system, and occasionally an under-activation of the parasympathetic nervous system. In healthy individuals, higher resting LF measures of HRV have been found to be predictive of lower pain unpleasantness and higher pain thresholds. Nonetheless, research surrounding the relationship between pain and HRV is still in its early days and conflicting findings, such as evidence showing no relationship between HRV and pain, suggests that further investigation is needed. This includes investigation into various mediating and moderating factors which may contribute to the discrepancies in the current literature surrounding HRV and Pain.

CHAPTER FOUR

Proposed study, Aims, Study Design and Hypotheses

4.1. Proposed Study

As presented in the review, chronic pain has debilitating effects on social, physical, and psychological function, and can significantly reduce quality of life. It is also one of the leading causes of health care utilisation in the world. However, understandings of the complex interactions between the physiological and psychological factors involved in pain remains incomplete. This issue becomes more complex when individuals from different ethnic and cultural backgrounds are involved. It is well established that people of different ethnic background possess unique attitudes, perceptions and reactions to pain (Fabian et al., 2011; Riley III et al., 2002). However, it remains uncertain whether differences in the physiological processing of pain between ethnic groups exist. Practitioners and researchers who predominantly operate from a western viewpoint of pain, may not understand the unique characteristics of the pain experience for individuals of different ethnicities. In addition, they may lack understanding of how these factors influence report of pain. Therefore, to enhance understanding and improve treatments for pain it is important to recognise ethnic differences. This issue is of particular importance in New Zealand as differences in clinical pain reports exist between the major ethnic groups of New Zealand.

Pain catastrophising is one of the most strongly associated factors with increased pain intensity and emotional distress in both chronic and acute pain (Sullivan et al., 2002). It is also suggested to be a key mediating factor for ethnic differences in the experience of pain (Fabian et al., 2011). However, pain catastrophising has not been explored in a New Zealand context, despite the ethnic disparities in pain report (Coggan et al., 1994; New Zealand Ministry of Health, 2008). Catastrophising and 'general worry' share similar affective properties and some research has suggested that pain catastrophising may be a mediating factor linking general worry and pain reporting (Keogh, Book, Thomas, Giddins, & Eccleston, 2010; Lackner & Quigley, 2005). Thus, considering the overlap between pain catastrophising and general worry, exploring these variables in a multi-ethnic sample within New Zealand may help shed light on various mechanisms underpinning the epidemiological data showing ethnic differences in pain reports. In the long term, an investigation such as this carries the potential to enhance chronic pain management by making it more ethnically sensitive.

4.2. Aims

The purpose of the current study is threefold. Firstly, to explore behavioural performance at, and physiological responses to, a pain inducing task and a worry inducing task in a sample of Maori, Pacific and European individuals. Secondly, to investigate the role that pain catastrophising plays in the relationship between ethnicity and pain. Thirdly, to explore whether it is general worry, or pain specific worry (i.e. pain catastrophising) that contributes to poorer outcomes and higher physiological reactivity to both a pain inducing task and a

worry inducing task. The third aim also includes the exploration of the impact of pain catastrophising and general worry on recovery from both the pain task and worry task.

4.3. Study Design

An experimental design investigation employing a phasic-design protocol is proposed. The two major independent variables are; ethnic group and experimental phase. Ethnic group consists of three levels: Maori, Pacific, and European. There were six experimental phases: a baseline phase, a pain preparation phase, a painful task phase, a pain recovery phase, a worry induction phase and a worry recovery phase. The major outcome variables are performance at the pain task, including pain tolerance, pain threshold, and subjective pain ratings, as well as physiological adaption and physiological changes across the experimental phases (as measured using HR and HRV). Outcomes will be investigated both between groups (comparing differences between ethnic groups at each phase) and within the sample (looking at the sample as a whole across each phase).

Considering the biopsychosocial nature of pain, assessing both behavioural and physiological responses to the pain task, and matching these to psychosocial perceptions of pain is of merit. HRV was chosen as the major physiological outcome variable, alongside HR. HRV is used commonly as a measure of physiological stress reactivity such as that experienced during pain. It is seen as an index of the interaction between the parasympathetic and sympathetic controls of the heart, and also seen more generally as an indicator of the bi-directional feedback system between the central nervous system and autonomic nervous system. Therefore, HRV is suggested to be an accurate assessment of physiological function.

Thus, considering the cardiovascular health disparities between major ethnic groups in NZ (MacDonald, 2009), research looking into the physiological functioning of various ethnic groups in NZ using of cardiovascular measures such as HRV is of merit.

4.4. Hypotheses

Primary hypotheses are tailored around investigating outcomes between Maori, Pacific, and European ethnic groups, whereas secondary hypotheses are tailored around investigating outcomes for the study sample as a whole.

4.4.1. Primary hypotheses (Differences between ethnic groups)

Primary hypothesis 1.

According to the literature, Maori, Pacific, and European ethnic groups may possess unique views of pain and report pain differently (Coggan et al., 1994; Magnusson & Fennell, 2011; New Zealand Ministry of Health, 2008). On this basis it is hypothesised that there will be significant differences in pain tolerance levels, pain threshold levels, and subjective pain intensity ratings between Maori, Pacific and European ethnic groups during the pain task (cold pressor task). No direction of differences is specified as no reliable direction of differences has been suggested in the literature.

Primary hypothesis 2.

Research from outside of NZ suggests that ethnic differences in HRV levels exist between certain ethnic groups, although the direction of this relationship remains inconclusive (Li et al., 2009; Wang et al., 2005). Disparities in cardiovascular health between major ethnic

groups in NZ suggest differences in physiological functioning of the heart between ethnic groups (MacDonald, 2009). Thus, it is hypothesised that significant differences between Maori, Pacific, and European ethnic groups will be found in HR and HRV measures (Square-root of the Mean Squared Differences of successive N-N intervals, *Proportion* of interval differences of successive N-N intervals greater than 50ms, and natural log of high frequency HRV during both the pain task and the worry task.

Primary hypothesis 3.

On the basis of the explanation provided in the previous hypothesis, it is also hypothesised that significant differences between Maori, Pacific, and European ethnic groups will be found in the nature of recovery of HR and HRV, from the pain task to the pain recovery phase, and from the worry task to the worry recovery phase.

Primary hypothesis 4.

Research suggests that pain catastrophising (Pain Catastrophising Scale scores) may mediate ethnic differences in pain reports (Fabian et al., 2011). Thus, it is hypothesised that any differences in pain tolerance levels, pain threshold levels, and pain intensity ratings between these ethnic groups will be mediated by pain catastrophising.

4.4.2. Secondary hypotheses (Differences within entire sample)

Secondary hypothesis 1.

To provide further support for the suspected link between general worry and pain catastrophising, it is hypothesised that pain catastrophising and each sub-category of pain catastrophising (*rumination*, *helplessness*, and *magnification* as measured using the PCS) will be significantly positively associated with general worry.

Secondary hypothesis 2.

High levels of general day-to-day worry have been linked with lower HRV and it has been suggested that this also provides a link between stressful events and delayed cardiovascular reactivity (Brosschot et al., 2006). Therefore, it is hypothesised that higher levels of general worry (Penn State Worry score) will be significantly associated with lower levels of HRV mean change during the pain task and the worry task compared to baseline.

Secondary hypothesis 3.

Worry and catastrophising share similar negative affective properties (Keogh et al., 2010). Considering this overlap between these two constructs it is suggested that there may also be a link between pain catastrophising and HRV. Recent research has indicated that high pain catastrophising may be associated with lower cardiovascular reactivity (Wolff et al., 2008). Thus, it is hypothesised that higher levels of pain catastrophising will be significantly associated with lower levels of HR and HRV mean change during the pain task compared to baseline.

Secondary hypothesis 4.

Finally, studies have shown that lower HRV at rest is associated with decreased emotional functioning, hindered autonomic functioning, and lower cognitive functioning (Thayer et al., in press; Thayer & Lane, 2007). Taking into consideration that pain is a multidimensional phenomenon which is impacted by cognitive and emotional factors (Asmundson & Wright, 2004), it was hypothesised that lower baseline HRV will be significantly associated with lower pain tolerance, lower pain threshold, and higher ratings of pain intensity during the pain task.

CHAPTER FIVE

Methods

5.1. Participants

A convenience sample of 'healthy' adult volunteers of Maori, Pacific, or European ethnicity aged 18 years and over were recruited from the wider Auckland area. A total of 125 screening questionnaires were sent out to individuals who expressed an interest in taking part in the study. Potential participants were excluded if they (a) had any history of epilepsy; (b) were currently using any type of medication that would prevent accurate measurement of HRV or that may have influenced the sensation of pain; (c) had a known health condition/injury that might have put the participant at risk of harm during the experiment, impact performance at the pain task, or prevent accurate measurement of HRV; and (d) scored above 55 on the trait subscale of the State-Trait Anxiety Inventory. Participants who completed the entire study each received $30 in Motor Trade Association (MTA) vouchers as compensation for their time and effort.

The study received ethical approval from the University of Auckland Human Participants Ethics Committee on the 30th of May 2011 (see Appendix A). Recruitment commenced on the 30th of May 2011 and concluded on the 25th of October 2011.

Sample Size

The sample size required for the current study was calculated using the software G*Power (Faul, Erdfelder, Lang, & Buchner, 2007). Previous studies similar in nature have reported medium to large effect sizes (Appelhans & Luecken, 2008; Campbell et al., 2005; Forsythe et al., 2011). Therefore, an effect size of $f = .4$ was chosen for the current study. With an α-level set to .05 and a power of .80, a sample size of 64 was deemed appropriate for the current study. Overall, data from 64 participants who completed the entire study was used for analysis.

5.2. Procedure

The study consisted of two stages, an initial assessment/screening phase and an experimental phase.

Stage One: Initial assessment and recruitment

Individuals from the Auckland area received information regarding the study via advertisements through e-mail, posters, flyers and word of mouth. Those who were interested were directed to contact the one of the researchers running the study to organise the initial screening questionnaire (Questionnaire A; see Appendix B) to be sent out to them. The participants were requested to complete the questionnaire, which contained a consent form, a demographics questionnaire and a medical checklist. Questionnaire A took approximately 15 to 20 minutes to complete, and a free-post envelope was provided for individuals to send the questionnaire back upon completion. Responses to Questionnaire A were assessed and

individuals who were found to be eligible for the second phase of the study were contacted by e-mail or telephone. An appropriate time was scheduled for each potential participant to take part in the experimental part of the study. During this contact, participants were provided with directions as to where the experimental session would take place. They were also asked, to the best of their ability, to abstain from alcohol, caffeine and cigarettes 12 hours prior to the experiment.

Stage Two: Experiment

Experimental sessions took place in an experimental room based on the level 12 of the Auckland City Hospital support building, Grafton, Auckland city. Participants took part in the experiment individually at separate times. Upon arrival, each participant was greeted and thanked for their participation. The structure of each experimental session is outlined below.

Step 1: Introduction

Upon entry into the experimental laboratory each participant was seated on a comfortable adjustable seat. The session commenced by verbally providing the participant with a brief outline of the experimental protocol. Important information such as the participants dominant hand and cold pressor task past experiences were also recorded at this stage. A white coat was worn by the experimenter to standardise administration.

Step 2: Questionnaire B and equip HR chest belt

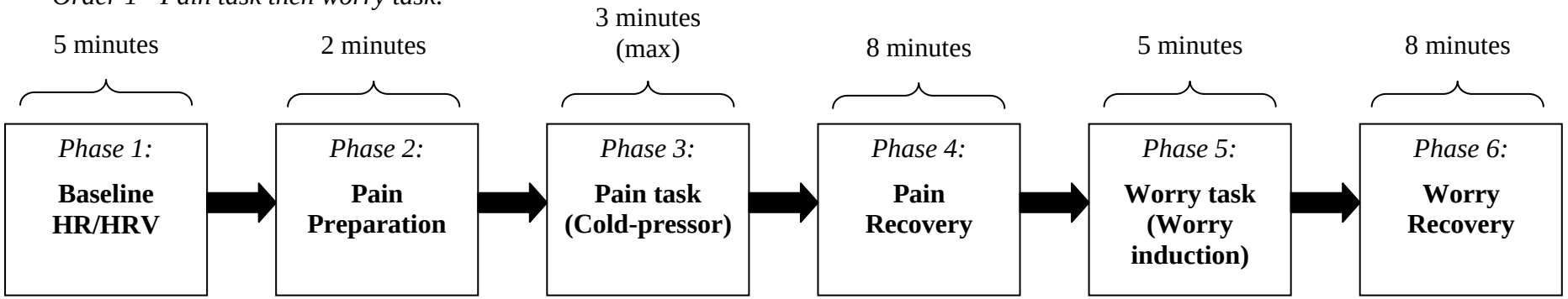
Participants were then asked to fill in the pre-experimental section of Questionnaire B (see Appendix C). Once the pre-experimental part of Questionnaire B was completed, participants were asked to put on the HR chest belt. Once the chest belt was equipped, participants were

asked to sit in a comfortable, relaxed position and were given a chance to relax before the commencement of the experimental phases.

Step 3: Experimental Phases

The primary component of the experimental session consisted of six phases. Each participant was exposed to each of the phases which took place simultaneously. Participants' HR and HRV was recorded continuously throughout each phase of the experiment. A diagram of the experimental phases is presented below in Figure 3, followed by an explanation of each phase. It is important to note that to account for 'order effects' (i.e. whether the participant received the painful task first or the worry induction first), the experimental phases were administered in two different orders. Each administration order was matched for ethnicity, age, and gender of the participant.

Order 1 - Pain task then worry task:



Order 2 - Worry task then pain task

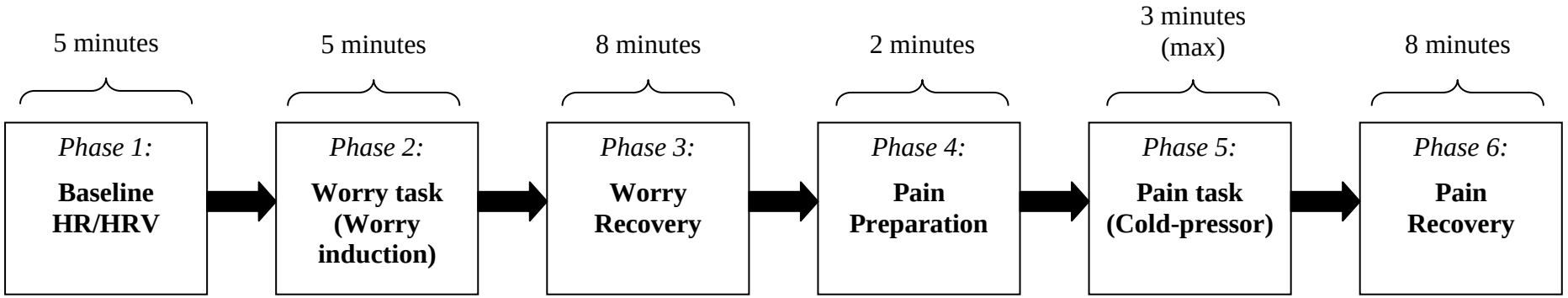


Figure 3. Experimental phases - counter balanced (order 1 and order 2)

Phase 1: Baseline HR/HRV

The initial phase consisted of a five minute recording of participants baseline HR/HRV. During this phase the participants sat in a relaxed position and were engaged in an innocuous filler task (watching a DVD). The primary purpose of the DVD was to direct the participants' attention away from 'distracting thoughts' which may have impacted accurate baseline HR/HRV measurement. The DVD used was titled '*Country Calendar: Directors Pick*' (see Appendix E), and was judged by the experimenters to be suitable for use during baseline measurement of HR/HRV. This DVD was also used during the recovery phases (detailed later).

Phase 2: Pain Preparation

During the pain preparation phase, the participants were asked to immerse their left hand, to the wrist, into a 160mm by 300mm by 500mm thermostatically controlled adapting bath set at 37°C. All participants were specifically asked to use their left hand during this phase and the next phase (painful task) in order to control for differences in pain-sensitivity between the left and the right hand (Murray & Hagan, 1973; Murray & Safferstone, 1970). The participants were asked to keep their hand immersed in the water for two minutes while verbal instructions were given for the next phase of the experiment (painful task), which followed immediately. The primary reason why the participants were asked to place their hand in body temperature water during the pain preparation phase was to standardise hand temperature for the pain task.

Phase 3*: *Painful task (Cold pressor task)

Pain will be induced using the cold pressor task. The cold pressor task has been utilised for over 80 years as a moderate, tonic, nociceptive stimulus to induce pain and cause autonomic nervous system arousal (Allen, Obrist, Sherwood, & Growell, 1987; Hatch, Klatt, Porges, Schroeder-Jasheway, & Supik, 1986; Knepp & Friedman, 2008; Mitchell, MacDonald, & Brodie, 2004; von Baeyer, Piira, Chambers, Trapanotto, & Zeltzer, 2005). For the task, the participants were instructed to immerse their left hand, to the wrist, into a 165mm by 265mm by 460mm bath containing ice-cooled water set at a temperature of 3°C (± 1°C). The ice was contained in one end of the bath by a plastic grid and the temperature of the water was maintained via a mechanical circulation mechanism. Participants were asked to submerge their hand at the opposite end of the bath for as long as they could, up to three minutes (180 seconds). Three minutes was selected as the maximum time of immersion based on previous literature (Mizushima et al., 1998; Wolf & Hardy, 1941). Timing commenced directly after the participants hand entered the bath and was recorded by the experimenter using a stop watch. Pain threshold was recorded as the amount of time it took for the participant to feel pain (self reported by participant). Pain tolerance was recorded as the amount of time the participant was able to keep their hand immersed in the ice water. Subjective pain intensity ratings were assessed directly after the task. Participants were asked to rank the pain that they felt while their hand was in the ice water from, zero (completely no pain) to ten (completely unbearable/extreme pain).

Figure 4. Left: pain preparation bath. Right: cold-pressor task bath

Phase 4: Pain recovery

Directly after the painful task, participants were asked to sit in a still, relaxed position and turn their attention once again to the DVD (as during the baseline phase) while their HR was recorded. The pain recovery phase lasted eight minutes. The purpose of this phase was to allow for investigation of the characteristics of HR and HRV *recovery* after the experience of pain (physical stressor).

Phase 5: Worry task (Worry induction)

Worry will be induced with a standard, brief, worry induction task. The worry induction task utilized was adopted from McLaughlin, Borkovec, and Sibbrava (2007), and has been successfully used as a method of experimentally inducing worry (Ruscio & Borkovec, 2004). The purpose of including a worry induction task was to provide further insight into

physiological characteristics of general worry, rather than assessing self-reports alone. It also serves as a comparison condition, as well as a control measure to assess whether differences in HRV responses are related specifically to pain stimuli. For the task, participants were asked to sit at a desk, and bring to their attention three of their most prominent current worries. They were asked to close their eyes and worry about the most worrisome thought, in a way that they normally worry about it but as intensely as they could. To ensure that all participants understood what 'worry' meant, the following definition was provided to them both verbally and in writing:

"Worrying involves thinking about a subject that has or can have negative consequences for you, and for which there is no, or not yet, a solution; it often, but not always, consists of a chain of negative thoughts, about the same or different topics, and often concerns something in the future, and the thought often takes shape as 'Imagine that X' or 'what would happen if …Y'. The same thoughts often return; when you are engaged in worrying it is difficult to stop or hold. It definitely occupies your mind and is often disturbing and intensive." (McLaughlin et al., 2007, p. 27).

The worry induction phase lasted five minutes. A five minute period has been found to be optimal to induce worry but not too long to allow worry to lessen (Ruscio & Borkovec, 2004). Once the five minutes had elapsed, participants were asked to open their eyes and shift back to their original location in front of the DVD player.

Phase 6: Worry recovery

For the worry recovery phase, participants were asked once again to sit in a relaxed position and turn their attention to the DVD. The worry recovery phase lasted eight minutes. The

purpose of this phase was to allow for investigation of the characteristics of HR and HRV *recovery* after the experience of experimentally induced worry (mental stressor).

Step 4: Experiment conclusion

After participants had completed all six experimental phases, they were asked remove the HR chest belt. They then proceeded to complete the post-experimental section of Questionnaire B, which asked about how they felt (primarily their worry levels) throughout the experiment. Once participants had completed the final section of Questionnaire B, they were asked if they had any more questions of concerns which were addressed accordingly. Participants then received their MTA vouchers and were thanked once again for their participation.

5.3. Measures and Materials

Screening Questionnaire (Questionnaire A)

Questionnaire A (see Appendix B) included a consent form and was used to screen potential participants for eligibility to take part in the experimental component of the study, provide insight into various aspects of the individuals' psychosocial orientation toward pain, and provide other psychosocial information about the individual. The variables which were assessed included, demographic data (including medical status), ethnic-identity, trait-anxiety, pain catastrophising, general worry, attention to pain, and depressive levels. Pain catastrophising and general worry were assessed as the primary variables of interest. Attention to pain, anxiety and depressive levels were also assessed due to their possible confounding effects on both catastrophising and HRV (Crombez, Eccleston, Baeyens, & Eelen, 1998b; Eccleston et al., 1997; Geisser, Robinson, Keefe, & Weiner, 1994; Knepp &

Friedman, 2008; Peters et al., 2002; Quartana, Campbell, & Edwards, 2009). The assessment of these variables are detailed below.

i. Demographic data

Demographic data was collected at two separate time points, during the initial screening questionnaire and again, for those who were found eligible, at the start of the experiment. During Questionnaire A, potential participants were asked to state their age, gender, education level, and what ethnicity they primarily identified with. A medical checklist was included which assessed suitability for the experiment. Individuals were asked if they had any history of epilepsy, and other cardiac or pain conditions which may have hindered accurate experimental recording or put the participant at risk of harm.

ii. Ethnic Identity

Ethnicity is a multidimensional concept, and varies from person to person. Thus, in research investigating ethnic-specific outcomes, assessing ethnic identity levels is recommended and is of merit (Fabian et al., 2011; Nelson, 2006). In the current study, the amount that one identified with their ethnic group was assessed using Multi-group Ethnic Identity Measure (MEIM; Phinney, 1992). The MEIM contains 23 items which are broken up into three subscales assessing three separate constructs: *ethnic identity* (14 items), *other-group orientation* (6 items) and *ethnic self-identification* (3 items). For the purposes of the current study, only the 14 items assessing 'ethnic identity' were used in data analysis, as *ethnic identity* was the primary construct of interest in the current study. The ethnic identity construct was broken down into three sub-constructs including, affirmation and belonging (5 items), ethnic identity achievement (7 items), and ethnic behaviours (2 items). Two of the items were reverse scored and were expressed as characteristics showing lack of ethnic

identity rather than its presence. Responses to each of the 14 items yielded a total score ranging from 14 to 56, with higher scores indicating higher levels of identity with ones' ethnic group. The *ethnic identity* subscale of the MEIM is found to have strong internal consistency with Chronbach's alpha (α) coefficients ranging from .81 to .92 (Dandy, Durkin, McEvoy, Barber, & Houghton, 2008; Goodstein & Ponterotto, 1997; Phinney, 1992; Ponterotto, Gretchen, Utsey, Stracuzzi, & Saya, 2003; Taub, 1995). It is also found to be a valid measure of ethnic identity as it correlates well with other measures assessing a similar constructs such as ethnic self-concept and racial identity development (Ponterotto et al., 2003). In the current study, the MEIM was found to have an excellent internal consistency with a Chronbach's α coefficient of .92.

iii. Trait-Anxiety

Individuals high in trait-anxiety are those who exhibit the tendency to react more anxiously to threatening situations in general and in a predictable manner (Spielberger & Gorsuch, 1983). Due to its relationship with pain and worry, trait-anxiety was assessed as a measure of the participants suitability for the experiment. Participants level of trait-anxiety was assessed using the trait-anxiety subscale of the State-Trait Anxiety Inventory (STAI; version Y-2; Spielberger & Gorsuch, 1983). The trait-anxiety subscale comprises of 20 items, each item is rated on a 4-point likert scale ranging from 1 (almost never) to 4 (almost always). 10 items from the trait-anxiety subscale were reverse coded in scoring. These items were expressed as emotional states which indicated a lack of trait-anxiety rather than its presence. The responses to the trait-anxiety sub-scale were summed to yield total scores ranging from 20 to 80, with higher scores indicating higher levels of trait-anxiety. The trait-anxiety subscale of the STAI has shown strong internal consistency with Chronbach's α coefficients ranging from .88 to .92. The subscale has also correlated well with other scales assessing trait-like anxiety

(Speilberger & Vagg, 1984; Spielberger & Gorsuch, 1983; Vigneau & Cormier, 2008). In the current study, the trait-anxiety subscale of the STAI was found to have a good internal consistency with a Chronbach's α coefficient of .85.

iv. Pain Catastrophising

Participants 'general' catastrophic thoughts and feelings about the experience of pain were assessed using the Pain Catastrophising Scale (PCS; Sullivan et al., 1995). The PCS consists of 13 items which are categorised to assess three separate subscales; *Rumination* (4 items), *Magnification* (3 items), and *Helplessness* (6 items). Each item is rated on a 5-point likert scale ranging from 0 (not at all) to 4 (all the time) based on the extent to which the participant perceives the item to be reflective of their reaction to pain. The responses to the 13 items are summed to produce a total scores ranging from 0 to 52, with higher scores indicative of higher pain catastrophising levels (Sullivan et al., 1995). The PCS has shown good to excellent internal consistency with Chronbach's α coefficients for the overall PCS ranging from .87 to .95 in non-clinical samples. The *Rumination*, *Magnification*, and *Helplessness* subscales have shown acceptable to excellent internal consistency with Chronbach's α coefficients ranging from .87 to .95, .60 to .88, and .79 to .91 respectively, in non-clinical samples (De Vlieger, Crombez, & Eccleston, 2006; Osman et al., 2000; Osman et al., 1997; Sullivan et al., 1995). The measure has also shown concurrent validity with other measures measuring similar negative emotional responses to pain such as the Inventory of Negative Thoughts in Response to Pain (r = .59) (Osman et al., 1997). Additionally, the PCS has shown to be reliable for use in an adult population making it suitable for the current study (Quartana et al., 2009). The PCS was found to have a good internal consistency in the current study with a Chronbach's α coefficient of .86.

v. General Worry

The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) was used to assess participant's dispositional tendency to worry that is independent of anxiety or depression (Brown et al., 1992; Meyer et al., 1990). It was also used to assess whether catastrophising has a unique relationship with pain compared to general worry. The PSWQ consists of 16 items asking participants to rate the degree to which various statements reflecting 'worrying' was typical of them. Each item is rated on a 5-point likert scale ranging from 1 (not at all typical of me) to 5 (very typical of me). Five of the items were reverse scored, representing a lack in trait worry rather than its presence. The responses to each item are summed up to yield total scores ranging from 16 to 80, with higher scores indicating higher levels of trait worry. The PSWQ has shown excellent internal consistency in non-clinical samples with Chronbach's α coefficients ranging from .88 to .93. Additionally, the scale has found to exhibit high levels of construct validity, temporal stability and has found to be unrelated to social desirability (Brown et al., 1992; Fresco, Heimberg, Mennin, & Turk, 2002; Meyer et al., 1990; van Rijsoort, Emmelkamp, & Vervaeke, 1999; Verkuil, Brosschot, & Thayer, 2007). In the current study, the PSWQ was found to have a strong internal consistency with a Chronbach's α coefficient of .89.

vi. Attention to Pain

The Pain Vigilance and Awareness Questionnaire (PVAQ; McCracken, 1997) was used to measure participants attention to pain. The PVAQ was originally developed for use in chronic pain patients, yet a slightly modified version was created for use in non-clinical samples. As a non-clinical sample was recruited in the present study, the latter version of the PVAQ was used. The measure broadly assesses two major dimensions of attention to pain including, *monitoring pain* (e.g. "I focus on sensations of pain"), and *awareness changes in pain* (e.g. "I

am quick to notice changes in pain intensity") (McCracken, 1997, p. 277). A three factor structure has also been suggested with the addition of *intrusion of pain* as a dimension of attention to pain (McWilliams & Asmundson, 2001). The PVAQ consists of 16 items, and each item is rated on a 6-point likert scale ranging from 0 (never) to 5 (always). Two of the items are reverse coded in scoring as they are expressed as states which indicate a lack of attention to pain rather than its presence. Responses to each item are summed to yield total scores ranging from 0 to 80, with higher scores indicating higher attention to pain. The PVAQ has shown excellent internal consistency with a Chronbach's α coefficient of .92 in a non-clinical sample (McWilliams & Asmundson, 2001). The *Monitoring Pain, Awareness Changes in* Pain, and *Intrusion* constructs also show good internal consistency with Chronbach's α coefficients of .88, .88, and .65 respectively, in a non clinical sample (McWilliams & Asmundson, 2001). The PVAQ has good construct validity and was developed using adults, making it a suitable measure for the current study (McCracken, 1997). Furthermore, the scale was found to have an acceptable internal consistency in the current study with a Chronbach's α coefficient of .75.

vii. Depressive levels

The Centre for Epidemiological Studies Depression scale (CES-D; Radloff, 1977) was used to assess the amount and regularity of depressive symptoms experienced by participants. The CES-D consists of 20 items primarily assessing the affective component of depression, i.e. low mood (Radloff, 1977). Each item asks the participants to rate how often they experienced emotional states or thoughts related to depression in the past week. Each item is rated on a 4-point likert scale ranging from 0 (rarely; less than one day) to 3 (most/all; 5-7 days). 4 of the items on the CES-D were reverse coded in scoring as they were expressed as emotional thoughts and states indicating the lack of depressive symptomology. The responses to the 20

items are summed to yield a total score ranging from 0 to 60, with higher scores indicating higher levels of depressive symptomology. The CES-D has shown good internal consistency with Chronbach's α coefficients ranging from .82 to .88, and is found to be a valid measure of depressive symptomology in non-clinical samples (Knight, Williams, McGee, & Olaman, 1997; Manson, Ackerson, Dick, Baron, & Fleming, 1990; Radloff, 1977). In the current study, the CES-D was found to have a strong internal consistency with a Chronbach's α coefficient of .89.

Experimental Questionnaire (Questionnaire B)

A second questionnaire, Questionnaire B, was administered at the start of the experimental session for those who qualified for the experimental component of the study. Questionnaire B was designed to provide further details and characteristics of the participants who were eligible for the experimental component of the study. As mentioned earlier, Questionnaire B contained two main sections, a pre-experimental section and post-experimental section.

Pre-experimental Component - Questionnaire B

The pre-experimental component involved completing a (second) consent form, assessed further demographic data (including a second medical status check and assessment of health behaviours), and measured pre-experimental anxiety levels. Assessments of these variables are detailed below.

i. Demographic data

Demographic data collected in Questionnaire B included height, weight, physical activity levels, smoking status, average alcohol consumption and average caffeine intake. Assessment of caffeine consumption, alcohol consumption and cigarette use 12 hours prior to the

experiment was also recorded. The participants also completed the same medical check-list they completed in Questionnaire A, as a confirmation that they were still medically fit to take part in the experiment.

ii. State-Anxiety

State-anxiety refers to transient periods of anxiety, characterised by physiological arousal, feelings of apprehension, dread and tension related to an event or situation (Spielberger & Gorsuch, 1983). Participants state-anxiety levels at the start of the experimental protocol was assessed using the state-anxiety subscale of the State-Trait Anxiety Inventory (STAI; version Y-2; Spielberger & Gorsuch, 1983). The state-anxiety subscale comprises of 20 items in total, each of the items are rated on a 4-point likert scale ranging from 1 (almost never) to 4 (almost always). Nine items from the state-anxiety subscale were reverse coded in scoring. These items were expressed as emotional states which indicated a lack of state-anxiety rather than its presence. The responses to the state-anxiety sub-scale were summed, to yield total scores ranging from 20 to 80, with higher scores indicating higher levels of anxiety at the beginning of the experimental session. The state-anxiety subscale of the STAI has shown strong internal consistency with Chronbach's α coefficients ranging from .88 to .94. The subscale is also shown to have good construct validity (Speilberger & Vagg, 1984; Spielberger & Gorsuch, 1983; Vigneau & Cormier, 2008). In the current study, the state-anxiety subscale of the STAI was found to have a good internal consistency with a Chronbach's α coefficient of .86.

Post-experimental Component - Questionnaire B

Questionnaire B also contained a post-experimental section. This included six items that asked the participants to estimate their level of worry before the experiment, levels of worry during the experiment, ability to worry during the worry task, level of worry during the worry

recovery phase, worry levels during the pain preparation phase, and worry levels during the pain recovery phase. This post-experimental information was used as background information.

Physiological Data Collection

HR and HRV were measured continuously throughout the experiment using the *Polar RS800CX* HR chest belt and watch. The chest belt worn by the participant contained two electrodes and a transmitter which sat just below the inferior point of the sternum. The transmitter sent HR data from the chest belt to the watch worn by the experimenter, where the data was stored. After data collection, the data was transferred to a Personal Computer (PC) for analysis. Initially, data was uploaded to the software programme *Polar ProTrainer* (version 5.0). The Polar Pro Trainer programme provided a database to store the HR data and produced various forms of output such as HR tachograms. The HR data files were divided into six separate phases representing each phase of the experiment (baseline, pain preparation, pain task, pain recovery, worry task, and worry recovery), converted to text-files, and saved as six separate HR files. These files were then used to investigate HRV at each phase.

The text files were transferred to the software programme Kubious HRV (version 2.0; Tarvainen & Niskanen, 2008). Kubious HRV programme enabled analysis of inter-beat-intervals embedded in the HR data file and the production of HRV output for each phase. Accurate analysis of the inter-beat-intervals was critical and was dependent on the sampling frequency of the HR signal. Hence, as recommended by the Task Force (1996) the sampling frequency was set to 1000Hz. The inter-beat-interval data from each HR data file was presented as a time series. Each data file went through an artefact correction phase in

Kubious. The 'cubic spline interpolaton' was the algorithm used to approximate and replace any outlier or missing inter-beat-intervals in each data set. Kubious allowed for five different levels of sensitivity to be used for data correction, ranging from very low to very strong (Kaufmann, Sütterlin, Schulz, & Vögele, 2011). A 'medium' level of sensitivity was used for all data correction in the present study.

After data correction, the inter-beat-intervals for each phase were analysed to produce HRV output. In the present study, inter-beat-interval data was analysed using two separate methods including both time-domain and frequency-domain analysis. Time-domain analysis provided the overall amount of variance present in HR (Stein et al, 1994). The Task Force (1996) recommended various methods of time-domain analysis, two of the most frequently used were utilised in the present study. Firstly, the square-root of the mean squared differences of successive Inter-beat-intervals (*RMSSD*). Secondly, the proportion of interval differences of successive inter-beat-intervals greater than 50ms (*pNN50*). Both of these methods were presented in milliseconds (ms).

Frequency-domain analysis allowed for a more accurate assessment of the underlying mechanisms contributing to the variance in HR (Stein et al, 1994). In the current study, 'High Frequency' (HF) HRV rhythms (0.15-0.4 Hz) were analysed. HF rhythms are found to be primarily modulated by the parasympathetic nervous system (Barbieri, Triedman, & Saul, 2002; Pomeranz et al., 1985; Task Force, 1996). Analysis of HF HRV was carried out using power spectral density (PSD) analysis. PSD used information from the inter-beat-intervals to determine the separate 'power' contributions of autonomic nervous system to the overall variance in HR (Porges & Byrne, 1992). PSD can be conducted using parametric and non-parametric methods. Parametric methods were used for the present study rather than non-

parametric, as they produce more precise spectral components and provide an accurate estimation of PSD in small samples and over short time periods. The parametric algorithm used in current study was the Auto-Regressive (AR) algorithm with a model order of 16. The AR method has been shown to be advantageous over other methods as it automatically calculates HRV power contributions, and graphically produces better a spectral resolution (Kay & Marple, 1981; Task Force, 1996). The output of the frequency domain analysis was expressed as 'power' (ms^2).

5.4. Data Analysis

All data was analysed using Statistical Package for Social Sciences (SPSS) version 19.0 for Windows. All data were tested to ensure that the assumptions necessary for parametric statistical analysis were fulfilled. To assess the assumption that the spread of scores on each variable of interest was normally distributed, skewness and kurtosis values were evaluated, and histograms of score distribution were visually inspected. The distribution of scores for age, average weekly alcohol consumption, and average daily caffeine consumption were each found to be positively skewed. Square-root transformations ($\sqrt{X_i}$) were applied to each of these variables to correct for the positive skew. Data transformation was not found to significantly improve the distribution of scores for each variable, hence analysis on these data sets was conducted in their original form. Distributions for the frequency domain HRV data (HF) were found to be skewed. Thus, all HF HRV data was transformed using natural logarithm and expressed as *lnHF* HRV. *Levene's* tests were used for each between-group ANOVA conducted to ensure equality of variances of different groups. No missing data was found in the current study.

All analysis conducted on hypotheses which involved only behavioural and psychosocial outcome variables were conducted using the data sampled from all 64 participants (N = 64). However, it is suggested that baseline HR readings of above 100 beats per minute (bpm) indicates improper autonomic regulation of the cardiovascular system (Berne & Levy, 2001; Levy, 1990; Verrier, 1987). Hence, participants with a mean baseline HR of 101bpm or higher were considered unsuitable for data analysis involving physiological outcome variables. This exclusion criteria reduced the overall sample size from N = 64 to N = 55 for analysis of hypothesis involving HR and HRV.

Demographic data analysis was conducted for the sample as a whole (N = 64), and for each different ethnic group in the study. Hypotheses were tested using appropriate between groups and within groups analyses. The former was used to compare outcomes for different ethnic groups, as well as to compare outcomes for those categorised as exhibiting differing levels of each psychosocial variable assessed. The latter was used to compare performance at different phases of the experiment, for the entire sample. Post-hoc analyses were conducted where necessary. Correlational analysis was also used to investigate the associations between various psychosocial, behavioural, and physiological outcome variables. Specific details regarding the statistical tests and methods used are provided in the results section.

CHAPTER SIX

Results

This chapter presents the demographic characteristics for the entire study sample. Each hypothesis will then be addressed including details of the statistical tests utilised to investigate each hypothesis, followed by brief presentations of the corresponding findings. The chapter will begin by outlining the participants' progression through study using the Consolidated Standards of Reporting Trials (CONSORT) guidelines (Schulz, 1997).

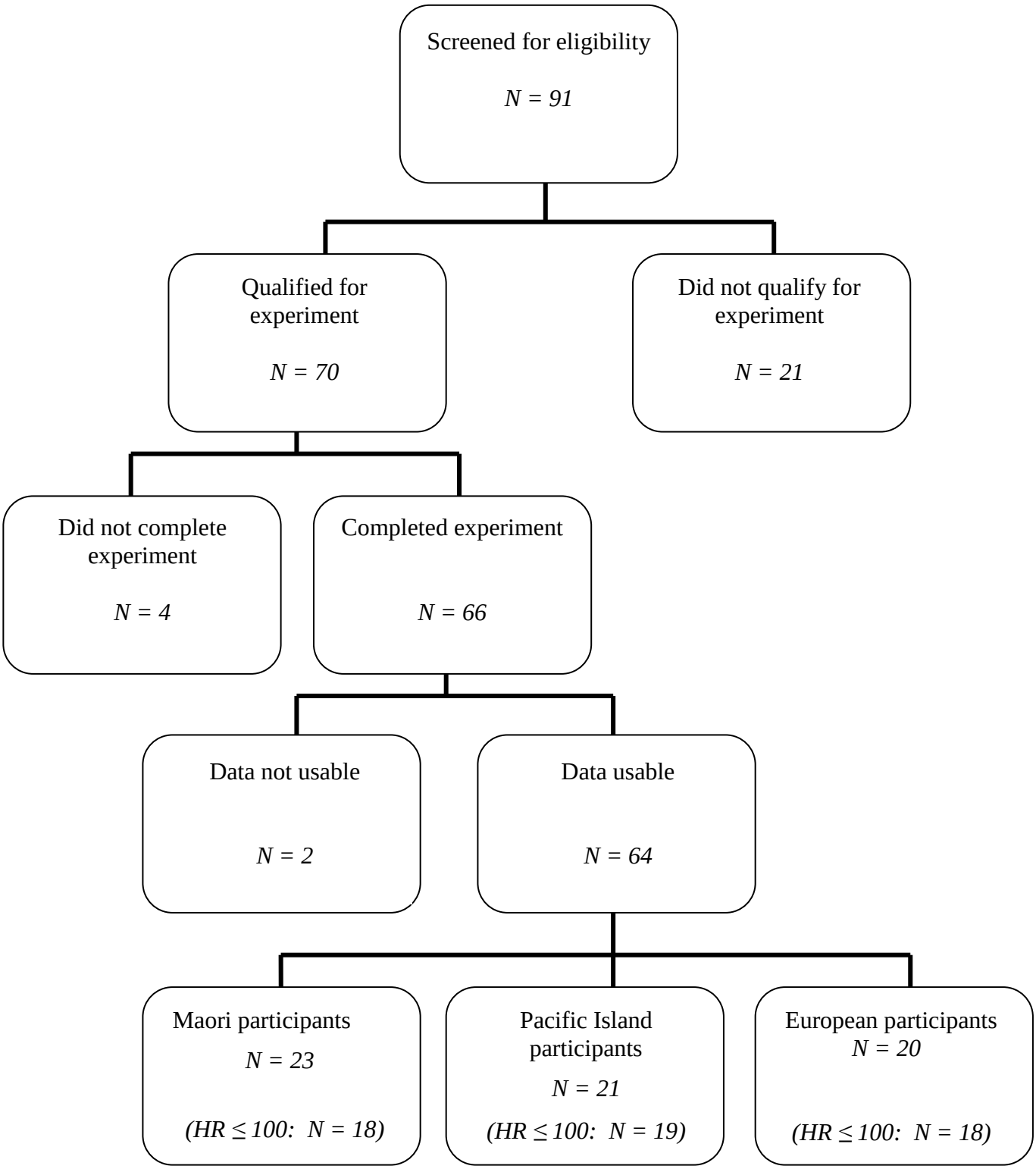


Figure 5. CONSORT diagram detailing participant progression through the study

A total of 91 potential participants were screened for eligibility. Of those 91, 21 did not qualify, 11 because they had health conditions that made them unsuitable for the experiment, four scored above the cut-off threshold of the STAI-trait set for the study (score of 55 or above), three were using medications that may have hindered accurate HRV measurement or influenced performance at cold pressor task, two had injuries that may have hindered performance at the cold pressor task, and one identified with an ethnicity which was not Maori, Pacific or European. Of the 70 participants that did qualify, four did not complete the experiment, three pulled out for undisclosed personal reasons and one did not attend the experiment and was unable to be contacted. This left 66 participants who completed the experiment, although data from two participants was not usable due to faults in the recording equipment. Thus, there were 64 data sets were used from participants who completed the entire study.

These 64 participants were categorised based on their identification of ethnic group for the data analysis (Maori, N = 23; Pacific, N = 21; European, N = 20). As mentioned previously, those with mean baseline HR of 101 or over were excluded from all analysis involving physiological outcome variables. This exclusion criteria reduced the overall sample size from 64 to 55 for analysis involving HR and HRV (Maori, N = 18; Pacific, N = 19; European, N = 18).

6.1. Demographic Data

29 males and 35 females were included in the study. No significant differences were found in age between males (M = 30.21, SD = 10.35) and females (M = 30.97, SD = 12.60), $F_{2,61}$ = .07, p > .05. The majority (48%) of the participants reported to be *moderately* physically

active. The ethnic makeup of the entire sample consisted of 23 Maori participants, 21 Pacific participants, and 20 European participants. As ethnicity was the main independent variable for the current study, the demographic characteristics of each ethnic group are presented below, in Table 1.

Table 1.

Demographic Data for Entire Study Sample (N = 64) and each Ethnic Group

		Ethnic Group		
	Entire sample (N = 64) ***M* (*SD*)**	**Maori (*N* = 23)** ***M* (*SD*)**	**Pacific (*N* = 21)** ***M* (*SD*)**	**European (*N* = 20)** ***M* (*SD*)**
Age	30.63 (11.56)	31.35 (12.09)	30.29 (11.98)	30.15 (11.03)
Gender (*N*):				
Male	29	9	10	10
Female	35	14	11	10
Ethnic Identity	43.88 (8.69)	47.00 (8.16)	45.90 (8.51)	38.15 (6.78)
Physical Activity (*N*):				
Inactive	6	2	1	3
Mildly Active	16	8	6	2
Moderately Active	31	6	10	15
Very Active	11	7	4	0
Education (*N*):				
NCEA Level 1	2	2	0	0
NCEA Level 2	1	0	1	0
NCEA Level 3	18	8	4	6
Tertiary Qualification	40	11	15	14
Not Specified	3	2	1	0

As can be seen in Table 1, Maori, Pacific and European ethnic groups did significantly differ in their levels of identity with their own ethnic group, $F_{2,61} = 7.78$, $p = .001$. After running a Tukey post hoc analysis, it was found that European participants (68.1%) identified significantly less with their own ethnic group than both Pacific (82.0%) and Maori (83.9%) participants, $p = .001$. With regards to the ethnic origin of the Pacific Island participants, 61.9% (13 out of 21) identified as Indo-Fijian, 19.0% (4 out of 21) identified as Samoan, 9.5% (2 out of 21) identified as Tongan, 4.8% (1 out of 21) identified as Niuean, and 4.8% (1 out of 21) identified as Cook Islander. No significant differences were found in age, gender, physical activity levels and education levels between ethnic groups. Table 2 shows health status and health behaviour for the three ethnic groups

Table 2.

Health Status and Health Behaviour Data for each Ethnic Group (N = 64)

	Ethnic Group				
	Maori ***M* (*SD*)**	**Pacific** ***M* (*SD*)**	**European** ***M* (*SD*)**	***F* - Statistic**	***p* - *Value***
Body Mass Index	25.82 (3.69)	26.47 (4.58)	22.90 (2.90)	5.14	.01
Hypertension *(N)*	0	2	1		
Days of troubled sleeping (In Past 30 Days)	7.20 (9.54)	6.43 (7.10)	3.48 (3.91)	1.49	.23
Current cigarette Smoker *(N)*	4	1	0		
Years Ever Smoked Cigarettes	12.94 (8.35)	11.00 (3.61)	8.33 (4.51)	.47	.64
Average weekly Alcohol Consumption (Standard Drinks)	1.14 (1.66)	1.55 (1.88)	3.58 (3.77)	5.39	< .01
Average Daily Caffeine Consumption (mg)	96.91 (109.84)	64.22 (51.23)	61.67 (72.50)	1.24	.30

Body Mass Index (BMI) scores for European participants (M = 22.90, SD = 2.90) were significantly lower than both Maori (M = 25.82, SD = 3.69) and Pacific (M = 26.47, SD = 4.58) participants, $F_{2,61}$ = 5.14, p < .05. No Maori participants presented with hypertension, however one European and two Pacific participants did present with hypertension. Four Maori participants were *currently* smokers, one Pacific participant was currently a smoker, and no European participants were currently smokers. The average years of smoking was also analysed, including participants that were not *current* smokers. There was no significant difference in the average number of years the participants in each ethnic group had been smoking. European participants (M = 3.58, SD = 3.77) were found to consume more alcohol (standard drinks) per week compared to both Maori (M = 1.14, SD = 1.66) and Pacific (M = 1.55, SD = 1.88) participants, $F_{2,61}$ = 5.40, p < .05. No differences were found between ethnic groups on their daily caffeine intake.

The differences in BMI and average alcohol consumption between ethnic groups was investigated to assess the impact they may have on any dependant (outcome) variables assessed later on in this section. Pearson product-moment correlation coefficient was used to assess the relationship between BMI, average alcohol consumption, HRV outcome measures (baseline, tasks, recovery phases, change scores), and pain task outcomes. No significant correlations were found between alcohol consumption and any outcome variables. However, correlations were found between BMI and HR. A medium, negative correlation was found between BMI and baseline HR, $r = -.40, n = 55, p < .01$., with higher BMI levels associated with lower baseline HR. Further, medium, negative correlations were found between HR and BMI at each phase of the experiment, including pain preparation ($r = -.39, n = 55, p < .01$), pain task ($r = -.42, n = 55, p < .01$), pain recovery ($r =$

$-.35, n = 55, p < .01$), worry task ($r = -.37, n = 55, p < .01$), and worry recovery ($r = -.43, n = 55, p < .01$). BMI did not correlate with any other outcome variables.

6.2. Ethnic differences in performance at pain task

A one-way between group multivariate analysis of variance (MANOVA) was conducted to assess whether there were differences in pain tolerance, pain threshold, and pain ratings between Maori, Pacific, and European participants during the cold-pressor task. Preliminary assumption testing for MANOVA was conducted and no serious violations were noted.

Table 3.

Outcomes for Cold Pressor Task - Comparison between Ethnic Groups (N = 64)

	Ethnic Group				
Outcome Variable	**Maori** ***M (SD)***	**Pacific** ***M (SD)***	**European** ***M (SD)***	***F* - Statistic**	***p* - *Value***
Pain Tolerance (Seconds)	153.13 (54.56)	140.86 (60.75)	150.72 (60.60)	0.27	.15
Pain Threshold (Seconds)	33.96 (26.10)	22.24 (17.64)	34.60 (23.52)	1.96	.77
Pain Ratings (0 to 10)	6.56 (1.79)	7.12 (1.44)	6.08 (1.72)	2.03	.14

As can be seen on Table 3, no significant differences for pain tolerance levels, pain threshold levels and pain ratings between ethnic groups. Thus, the hypothesis that Maori, Pacific and European ethnic group would exhibit differences in pain tolerance, pain threshold and pain intensity ratings was not supported.

6.3. Ethnic differences in HRV at baseline, in response to the pain task and in response to the worry task

As noted earlier, all participants with a baseline HR of above 100bpm (N = 9) were excluded from analysis involving HR and HRV measures.

Manipulation check

Before any physiological analysis was conducted, a manipulation check was conducted to verify the utility of the methodology used in the experiment, i.e. that the pain and worry tasks validly elicited physiological change. Thus, a one-way repeated measures ANOVA was used to assess changes in HR across each condition in the experiment. Results are presented in Table 4.

Table 4.

Experimental Manipulation Check - Change in HR Across Experimental Conditions (N = 55)

Phase	**HR (bpm)** ***M*** **(*SD*)**	***F* - Statistic**	***p* - *Value***
1. Baseline	74.00 (12.21)	24.18	< .001
2. Pain Preparation	76.98 (12.37)		
3. Pain Task	77.06 (12.31)		
4. Pain recovery	70.58 (13.38)		
5. Worry Task	75.52 (18.20)		
6. Worry Recovery	72.38 (13.31)		

Results from the ANOVA revealed that there was a significant effect of experimental condition on HR, Wilks' Lambda = .29, $F_{5,50}$ = 24.18, p < .001, multivariate partial eta squared = .71. Further investigation into the trend of HR through each phase revealed a significant *Order 5* trend across the experimental conditions, $F_{1,54}$ = 53.32, p < .001.

Baseline assessment of physiological variables between ethnic groups

A series of one-way, independent measures ANOVAs were conducted to see whether ethnic groups differed in HR or HRV measures at baseline. Results revealed no significant differences in any of the physiological variables between ethnic groups at baseline. Results are presented below, in Table 5.

Table 5.

Changes in HR and HRV(RMSSD, pNN50, and lnHF) Throughout Experimental Phases - Comparison between Ethnic Groups (N = 55)

		Ethnic Groups				
Experimental Phase	**Outcome Variable**	**Maori** *M (SD)*	**Pacific** *M (SD)*	**European** *M (SD)*	***F* - Statistic**	***P* - Value**
Baseline:	**HR**	70.61 (11.39)	75.53 (12.26)	75.80 (12.92)	1.04	.36
	RMSSD	52.62 (34.01)	40.23 (29.18)	36.33 (16.25)	1.57	.22
	pNN50	28.32 (23.94)	17.42 (20.21)	15.99 (15.50)	2.02	.14
	lnHF	6.63 (1.22)	5.97 (1.41)	5.98 (1.05)	1.70	.19
Pain Preparation:	**HR**	74.55 (12.76)	79.00 (12.43)	77.29 (12.20)	.59	.56
	RMSSD	51.26 (32.34)	35.36 (22.35)	41.25 (21.73)	1.59	.22
	pNN50	27.36 (23.69)	13.57 (15.07)	18.13 (16.73)	2.56	.09
	lnHF	6.20 (1.24)	5.81 (1.05)	5.73 (0.89)	1.00	.38
Pain Task:	**HR**	73.62 (12.95)	79.87 (12.09)	77.54 (11.74)	1.22	.30
	RMSSD	51.08 (35.60)	39.53 (29.59)	45.58 (24.13)	.68	.51
	pNN50	24.83 (23.46)	15.37 (17.83)	22.02 (18.66)	1.09	.34
	lnHF	6.30 (1.41)	5.89 (1.70)	6.19 (1.30)	.38	.68
Pain Recovery:	**HR**	67.73 (16.10)	72.64 (12.73)	71.25 (11.13)	.65	.53
	RMSSD	61.23 (43.23)	45.83 (32.94)	37.72 (16.58)	2.39	.10
	pNN50	32.96 (26.20)	20.83 (19.11)	16.96 (15.57)	2.92	.06
	lnHF	6.69 (1.26)	6.10 (1.35)	5.76 (0.83)	2.88	.07
Worry Task:	**HR**	74.28 (25.97)	75.68 (13.58)	76.59 (13.41)	.07	.93
	RMSSD	51.12 (34.34)	36.99 (27.44)	35.89 (20.63)	1.67	.12
	pNN50	26.18 (23.80)	16.01 (19.93)	15.21 (17.56)	1.60	.21
	lnHF	6.59 (1.45)	5.95 (1.24)	6.00 (0.98)	1.52	.23
Worry Recovery:	**HR**	69.37 (13.90)	74.45 (13.12)	73.21 (13.12)	.72	.49
	RMSSD	50.52 (29.70)	39.82 (28.79)	36.28 (16.55)	1.50	.23
	pNN50	26.58 (21.62)	16.46 (16.22)	15.90 (15.52)	2.03	.14
	lnHF	6.46 (1.18)	5.92 (1.30)	5.74 (0.92)	1.93	.16

6.4. Differences in physiological response to pain task and worry task between ethnic groups

The hypothesis that Maori, Pacific, and European participants would have different physiological responses to the pain and worry task was tested by conducting separate one-way, independent samples ANOVAs for each task and outcome.

As Table 5 indicates, no significant differences were found *HR*, HRV measured as *RMSSD*, HRV measured as *pNN50*, and HRV measured as *lnHF* in response to the pain task, *between* Maori, Pacific and European participants. Furthermore, no significant differences were found *HR*, HRV measured as *RMSSD*, HRV measured as *pNN50*, and HRV measured as *lnHF* in response to the worry induction task, *between* Maori, Pacific and European participants. In summary, the results do not support the hypothesis that Maori, Pacific and European participants differed physiologically in their response to both the pain and the worry tasks, by way of HR and HRV.

6.5. Ethnic differences in the nature of physiological recovery from the pain task and the worry task

The hypothesis that Maori, Pacific and European participants would show differences in the nature of their HR and HRV recovery from the pain task and worry task was tested using a series of two by three mixed between-within measures ANOVAs. The within subjects factor being task (two levels: pain/worry task and pain/worry recovery), and the between subjects factor being ethnicity (three levels: Maori, Pacific, and European). The nature of any significant overall findings was investigated using a series of follow-up, two by two, mixed measures ANOVAs, comparing each ethnic group to another indicating where any differences lay (i.e. establish whether difference lies between Maori and Pacific, Pacific and European, or European and Maori groups). The within subjects factor being task (two levels: pain/worry task and pain/worry recovery) and the between subjects factor being the ethnicity (two levels, dependant on which two ethnic groups are being compared).

Recovery from the pain task

Tests revealed that there was a significant main effect of task in HR, Wilks' Lambda = .55, $F_{1,52}$ = 42.81, p < .001, partial eta squared = .45. All ethnic groups were found to have reduced HR during the pain recovery phase compared to the pain task. However, there was found to be no significant interaction effect between task and ethnicity, Wilks' Lambda = .99, $F_{2,52}$ = 1.67, p > .05, partial eta squared = .01. Thus, indicating that there was no significant differences in change in HR from the pain task to pain recovery between Maori, Pacific, and European participants.

Results showed that there was no significant main effect of task in *RMSSD* HRV, Wilks' Lambda = .98, $F_{1,52}$ = 1.07, p > .05, partial eta squared = .02. However, there was

found to be a significant interaction effect between task and ethnicity, Wilks' Lambda = .87, $F_{2,52}$ = 3.56, p < .05, partial eta squared = .13. A series of repeated measures ANOVAs were conducted to investigate the nature of this interaction. No significant difference was found in *RMSSD* change scores between Maori and Pacific participants, Wilks' Lambda = .99, $F_{1,35}$ = .32, p > .05, partial eta squared = .01. However, a significant difference in *RMSSD* change scores was found between European and Pacific participants, Wilks' Lambda = .85, $F_{1,35}$ = 6.20, p < .05, partial eta squared = .15. Additionally, a significant difference in *RMSSD* change scores was found between European and Maori participants, Wilks' Lambda = .86, $F_{1,34}$ = 5.35, p < .05, partial eta squared = .14. Therefore, these results indicate that European participants (M = -7.86, SD = 20.44) differed from Maori participants (M = 10.15, SD = 25.95) and Pacific participants (M = 6.30, SD = 13.69) in the nature of their *RMSSD* change scores from the pain task to the pain recovery.

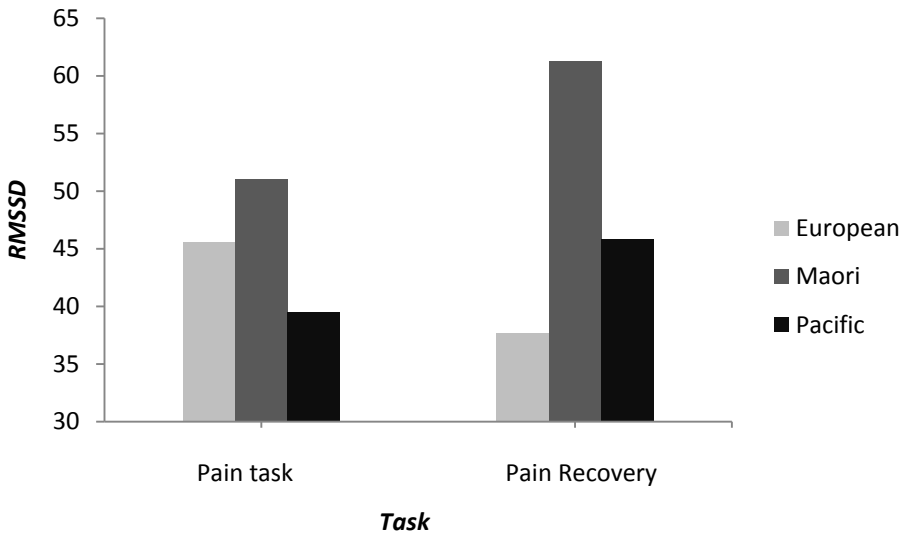


Figure 6. Change in HRV measured as *RMSSD* from pain task to pain recovery for each ethnic group

Results showed that there was no significant main effect of task in *pNN50* HRV, Wilks' Lambda = .94, $F_{1,52}$ = 3.06, p > .05, partial eta squared = .06. However, there was found to be a significant interaction effect between task and ethnicity, Wilks' Lambda = .81, $F_{2,52}$ = 6.06, p < .01, partial eta squared = .19. A series of repeated measures ANOVAs were conducted to investigate the nature of this interaction. No significant difference was found in *pNN50* change scores between Maori and Pacific participants, Wilks' Lambda = .98, $F_{1,35}$ = .56, p > .05, partial eta squared = .02. However, a significant difference in *pNN50* change scores was found between European and Pacific participants, Wilks' Lambda = .80, $F_{1,35}$ = 8.52, p < .05, partial eta squared = .20. Additionally, a significant difference in *pNN50* change scores was found between European and Maori participants, Wilks' Lambda = .81, $F_{1,34}$ = 7.87, p < .05, partial eta squared = .19. Therefore, these results indicate that European participants (M = -5.06, SD = 14.20) differed from Maori participants (M = 8.12, SD = 13.98) and Pacific participants (M = 5.46, SD = 6.57) in the nature of their *pNN50* change scores from the pain task to the pain recovery.

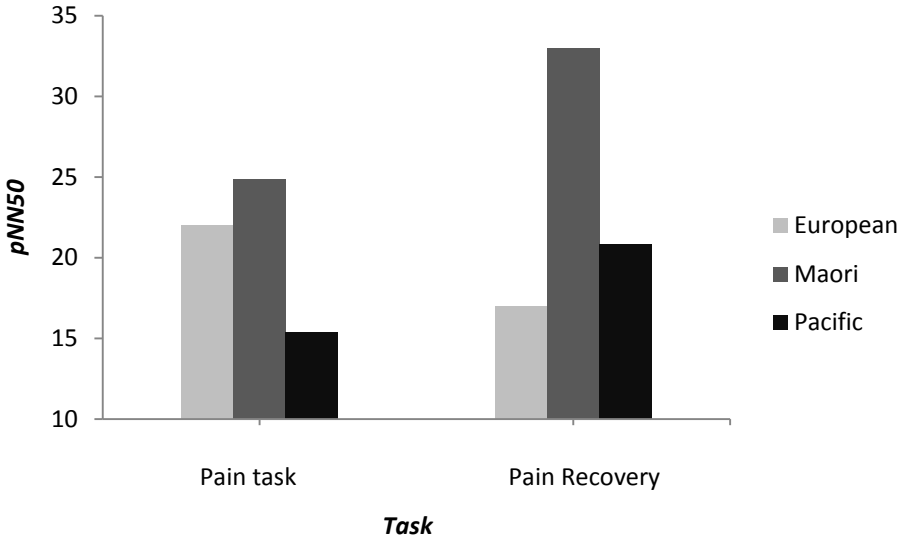


Figure 7. Change in HRV measured as *pNN50* from pain task to pain recovery for each ethnic group

Tests revealed that there was no significant main effect of task in *lnHF* HRV, Wilks' Lambda = 1.00, $F_{1,52}$ = .21, p > .05, partial eta squared < .01. However, there was found to be a significant interaction effect between task and ethnicity, Wilks' Lambda = .88, $F_{2,52}$ = 3.51, p < .05, partial eta squared = .12. A series of repeated measures ANOVAs were conducted to investigate the nature of this interaction. No significant difference was found in *lnHF* change scores between Maori and Pacific participants, Wilks' Lambda = .99, $F_{1,35}$ = .33, p > .05, partial eta squared = .01. However, a significant difference in *lnHF* change scores was found between European and Pacific participants, Wilks' Lambda = .89, $F_{1,35}$ = 4.31, p < .05, partial eta squared = .11. Additionally, a significant difference in *lnHF* change scores was found between European and Maori participants, Wilks' Lambda = .86, $F_{1,34}$ = 5.47, p < .05, partial eta squared = .14. Therefore, these results indicate that European participants (M = -.42, SD = 1.04) differed from Maori participants (M = 39, SD = 1.04) and Pacific participants (M = .21, SD = .81) in the nature of their *lnHF* change scores from the pain task to the pain recovery.

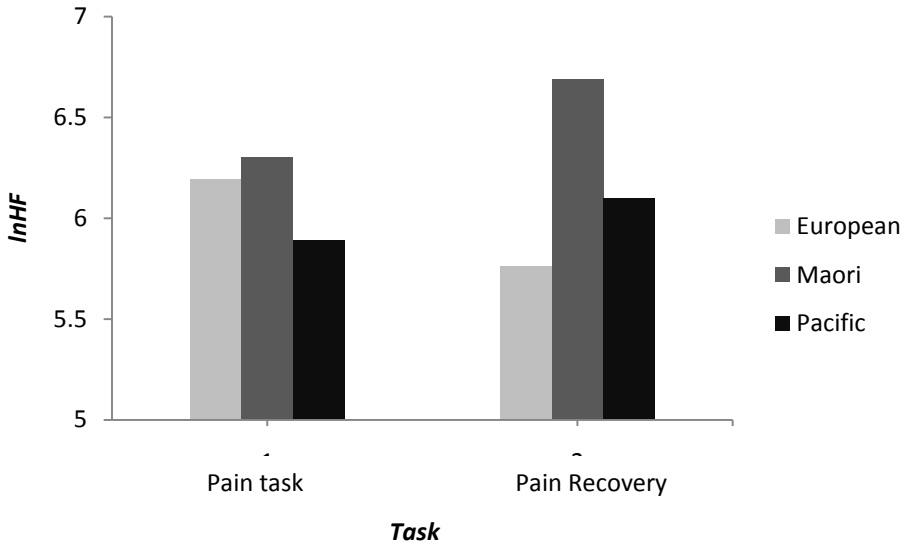


Figure 8. Change in HRV measured as *lnHF* from pain task to pain recovery for each ethnic group

Recovery from the worry task

Results showed that there was a significant main effect of task in HR, Wilks' Lambda = .87, $F_{1,52}$ = 7.68, p < .05, partial eta squared = .13. All ethnic groups were found to decrease HR from the worry task to worry recovery. However, there was found to be no significant interaction effect between task and ethnicity, Wilks' Lambda = .97, $F_{2,52}$ = .88, p > .05, partial eta squared = .03. Thus, indicating that there was no significant difference in change in HR from the worry task to worry recovery between Maori participants (M = -4.91, SD = 14.02), Pacific participants (M = -1.23, SD = 3.62), and European participants (M = -3.39, SD = 3.21).

Tests revealed that there was no significant main effect of task in *RMSSD* HRV, Wilks' Lambda = 1.00, $F_{1,52}$ = .28, p > .05, partial eta squared = .01. Additionally, there was found to be no significant interaction effect between task and ethnicity, Wilks' Lambda = .99,

$F_{2,52}$ = .39, p > .05, partial eta squared = .02. Thus, indicating that there were no significant differences between Maori, Pacific, and European participants in their *RMSSD* change scores from the worry task to worry recovery.

It was revealed that there was no significant main effect of task in *pNN50* HRV, Wilks' Lambda = 1.00, $F_{1,52}$ = .15, p > .05, partial eta squared < .01. Additionally, there was found to be no significant interaction effect between task and ethnicity, Wilks' Lambda = 1.00, $F_{2,52}$ = .01, p > .05, partial eta squared = .00. Thus, indicating that there were no significant differences between Maori participants (M = .40, SD = 8.57), Pacific participants (M = .45, SD = 9.33), and European participants (M = .69, SD = 11.07) in their *pNN50* change scores from the worry task to worry recovery.

Additionally, tests revealed that there was no significant main effect of task in *lnHF* HRV, Wilks' Lambda = .97, $F_{1,52}$ = .58, p > .05, partial eta squared = .03. Additionally, there was found to be no significant interaction effect between task and ethnicity, Wilks' Lambda = .99, $F_{2,52}$ = .38, p > .05, partial eta squared = .01. Thus, indicating that there were no significant differences between Maori participants (M = -.13, SD = .58), Pacific participants (M = -.03, SD = .96), and European participants (M = -.26, SD = .87) in their *lnHF* change scores from the worry task to worry recovery.

In summary, the results show that changes in HR from the both tasks (pain and worry) to the corresponding recovery phase (pain recovery and worry recovery) did not significantly differ between ethnic groups. Further, from the worry task to worry recovery no significant difference in change scores for any measures of HRV were found (*RMSSD*, *pNN50*, and *lnHF*) between ethnic groups. However, from the pain task to pain recovery significant

differences in change scores were found in all HRV measures between ethnic groups. Specifically, European participants differed in their change scores across all measures of HRV from Pacific and Maori participants. Hence, partial support was provided for the hypothesis that there are differences in the nature of physiological recovery from the pain task between ethnic groups, yet this was specific to HRV measures and not HR. No such support was provided for the worry task and worry recovery.

6.6. Impact of pain catastrophising and other psychological variables on ethnic differences in pain task outcomes

Pain catastrophising: Total sample

Pain catastrophising (as measured by the PCS) was initially investigated by looking at the influences of catastrophising across the total sample on performance at the pain task and subjective ratings of pain. Pearson product-moment correlation coefficient analysis was conducted to explore the association between pain catastrophising (including each subcategory of pain catastrophising), pain tolerance, pain threshold, and pain intensity ratings.

Table 6.

Correlation between pain catastrophising (and components of catastrophising) and pain task outcomes (N = 64)

Variable	**Pain Tolerance**	**Pain Threshold**	**Pain Intensity**
Pain catastrophising (*Overall*)	.30*	-.07	-.23
Pain catastrophising: *Rumination*	.23	.01	-.27*
Pain catastrophising: *Helplessness*	.28*	-.01	-.21
Pain catastrophising: *Magnification*	.27*	-.21	-.07

Note. * $p \leq .05$, two tailed. ** $p \leq .001$, two tailed.

Table 6 shows that medium, positive correlations were found between pain tolerance and pain catastrophising as well as the helplessness and magnification components of catastrophising. A small, negative correlation was found between the *rumination* component of pain catastrophising and subjective reports of pain intensity.

Pain catastrophising: Ethnic groups

A one-way ANOVA was conducted to assess whether Maori, Pacific and European participants differed in their levels of pain catastrophising. Results revealed that there were no differences in levels of pain catastrophising between Maori, Pacific and European participants (data presented in Table 5). Thus, the hypothesis that pain catastrophising would be different between ethnic groups, and that this difference would mediate differences in pain task outcomes was not supported.

Table 7.

Ethnic Differences in Baseline Psychosocial Variables (N = 64)

	Ethnic Group				
	Maori ***M* (*SD*)**	**Pacific** ***M* (*SD*)**	**European** ***M* (*SD*)**	***F* - Statistic**	***p* - *Value***
Pain Catastrophising	15.09 (8.45)	15.52 (8.47)	14.78 (7.34)	.04	.96
Trait Anxiety	37.04 (7.13)	35.48 (7.83)	35.75 (7.16)	.29	.75
State Anxiety	29.83 (7.16)	26.90 (6.07)	28.35 (6.49)	1.07	.35
Pain Vigilance and Awareness	41.04 (10.64)	42.43 (9.13)	36.30 (9.10)	2.22	.12
General Worry	44.22 (9.40)	43.33 (12.53)	38.50 (9.52)	1.77	.18
Depression	15.61 (9.02)	13.14 (9.82)	11.15 (9.00)	1.25	.30

Other psychological variables' impact on pain outcomes

Analysis was also conducted to assess differences between ethnic groups in the level of other psychological variables assessed at baseline, including trait anxiety, state anxiety (at start of experiment), pain vigilance and awareness, general worry, and depression. This was investigated using a series of one-way, independent measures ANOVAs. No differences between ethnic groups were found in any psychosocial variables assessed at baseline. Results are presented above (Table 7).

To assess the association between the other psychosocial variables assessed and the pain task outcomes on the entire sample (*N = 64*), a Pearson product-moment correlation coefficient was conducted. No significant correlations were found between any of the psychological variables and pain task outcomes, apart from state anxiety. A medium, positive correlation was found between state anxiety and pain tolerance, $r = .34,\ n = 64, p < .05$.

In order to further investigate the relationship between psychological variables and pain task outcomes, median splits were also conducted on all the psychosocial variables assessed, and a series of one-way independent measures ANOVAs were conducted to investigate whether there were differences in pain task outcomes between those who exhibited 'higher' levels of each variable, compared to those who exhibited 'lower' levels of each variable. Participants were categorised into 'higher' or 'lower' levels of each variable based on a median split.

Table 8.

Mean Score for Psychological Variable Median Grouping Splits (N = 64)

	Mean score on each corresponding questionnaire
	M (SD)
Pain Catastrophising:	
Higher (*N* = 32)	22.02 (3.85)
Lower (*N* = 32)	8.25 (4.18)
Trait Anxiety:	
Higher (*N* = 30)	42.37 (4.73)
Lower (*N* = 34)	30.62 (3.89)
State Anxiety:	
Higher (*N* = 31)	34.03 (4.79)
Lower (*N* = 33)	23.12 (2.29)
Pain Vigilance and Awareness:	
Higher (*N* = 30)	48.43 (5.94)
Lower (*N* = 34)	32.59 (6.02)
General Worry:	
Higher (*N* = 30)	51.47 (6.91)
Lower (*N* = 34)	33.91 (5.24)
Depression:	
Higher (*N* = 28)	21.71 (7.73)
Lower (*N* = 36)	6.94 (3.48)

As shown below, on Table 9, those who were categorised as exhibiting higher levels of trait anxiety, pain vigilance and awareness, general worry, or depression, did not differ from those categorised as exhibiting lower levels of each variable, at any of the pain task outcomes. However, those with higher levels of state anxiety had higher levels of pain tolerance, higher

pain thresholds, and reported lower pain intensity ratings than those exhibiting lower levels of state anxiety. Further, higher pain catastrophisers were found to have lower subjective ratings of pain intensity than lower pain catastrophisers.

Table 9.

Cold Pressor Task Outcomes for Median Split Categorisation of Entire Sample for Each Psychosocial Variable (N = 64)

	Pain Tolerance			Pain Threshold			Pain Intensity		
	***M* (*SD*)**	***t* - Statistic**	***p* - *Value***	***M* (*SD*)**	***t* - Statistic**	***p* - *Value***	***M* (*SD*)**	***t* - Statistic**	***p* - *Value***
Pain Catastrophising:									
Higher (*N* = 32)	162.16 (43.70)	1.95	.06	30.41 (21.61)	0.03	.97	6.14 (1.77)	-2.21*	.03
Lower (*N* = 32)	134.54 (67.08)			30.22 (24.94)			7.05 (1.49)		
Trait Anxiety:									
Higher (*N* = 30)	155.53 (51.62)	.93	.36	27.37 (22.50)	-.96	.34	6.47 (1.79)	-.56	.58
Lower (*N* = 34)	142.01 (62.93)			32.91 (23.73)			6.71 (1.61)		
State Anxiety:									
Higher (*N* = 31)	169.32 (34.64)	2.98**	.00	37.84 (26.60)	2.64*	.01	6.15 (1.62)	-2.12*	.04
Lower (*N* = 33)	128.65 (68.11)			23.24 (16.89)			7.02 (1.67)		
General Worry:									
Higher (*N* = 30)	148.27 (56.42)	-.01	.99	25.47 (19.96)	-1.59	.12	6.83 (1.74)	1.07	.29
Lower (*N* = 34)	148.42 (59.94)			34.59 (25.16)			6.38 (1.63)		
Pain Vigilance and Awareness									
Higher (*N* = 30)	144.70 (60.99)	-.47	.64	29.60 (23.75)	-.23	.82	6.92 (1.78)	1.45	.15
Lower (*N* = 34)	151.57 (55.66)			30.94 (22.94)			6.31 (1.58)		
Depression									
Higher (*N* = 28)	149.50 (56.26)	.14	.89	31.32 (27.96)	.31	.76	6.68 (1.91)	.35	.73
Lower (*N* = 36)	147.46 (59.84)			29.53 (18.98)			6.53 (1.52)		

Note. * $p \leq .05$, two tailed. ** $p \leq .001$, two tailed.

6.7. Association between pain catastrophising and general worry

The hypothesis that a positive association exists between pain catastrophising and general worry was tested using correlation analysis. The entire sample (N = 64) was used for this analysis.

Table 10.

Correlation between pain catastrophising (and components of catastrophising) and general worry (N = 64)

Variable	***General worry***
Pain catastrophising (*Overall*)	.39**
Pain catastrophising: *Rumination*	.25
Pain catastrophising: *Helplessness*	.39*
Pain catastrophising: *Magnification*	.39**

Note. * $p \leq .05$, two tailed. ** $p \leq .001$, two tailed.

The relationship between pain catastrophising (as measured using the PCS) and general worry (as measured using the PSWQ) was investigated using Pearson product-moment correlation coefficient. There was found to be a medium, positive correlation between overall pain catastrophising level and levels of general worry, r = .39, n = 64, p < .01, with higher levels of general worry associated with higher levels of pain catastrophising. There was also found to be a medium, positive correlation between the helplessness component of pain catastrophising and general worry, r = .39, n = 64, p < .05, with higher levels of general worry associated with higher levels of *pain catastrophising related helplessness*. Finally, there was found to be a medium, positive correlation between the magnification component

of pain catastrophising and general worry, r = .39, n = 64, p < .01, with higher levels of general worry associated with higher levels of *pain catastrophising related magnification*.

Thus, partial support was provided for the hypothesis that pain catastrophising is positively associated with levels of general worry.

6.8. Association between general worry levels and HRV change from baseline to the pain task and baseline to the worry task

The hypothesis that higher levels of general worry will be associated with lower levels of HRV change from baseline to the pain task and from baseline to the worry task was tested using a series of two by two, mixed between-within measures ANOVAs. The within subjects factor being phase (two levels: baseline and pain/worry task), and the between subjects factor being level of general worry (two levels: higher general worry and lower general worry). The mean amount of change in each HRV measure from baseline to the pain task and baseline to worry task was compared between participants with higher levels of general worry and participants with lower levels of general worry. The descriptive data for each general worry group are presented in Table 11. A reminder that the descriptive data for 'higher' and 'lower' general worry groups presented in Table 11. are for the N = 55 sample, rather than the entire sample (N = 64).

Table 11.

Mean PSWQ score for higher and lower worrying participants (N = 55)

		PSWQ Score
	N	***M* (*SD*)**
Higher general worry	27	50.74 (6.23)
Lower general worry	28	33.68 (5.54)

When comparing baseline to the pain task, there were no significant main effect of phase in *RMSSD* HRV, *pNN50* HRV, and *lnHF* HRV. Additionally, there was no significant interaction effect between phase and general worry level for any of these outcome variables. Thus, indicating that there was no significant difference in *RMSSD* HRV, *pNN50* HRV, and *lnHF* HRV change from baseline to the pain task between participants with higher levels of general worry compared to participants with lower levels of general worry.

Tests also revealed that when comparing baseline to the worry induction task there were no significant main effect of phase in *RMSSD* HRV, *pNN50* HRV, and *lnHF* HRV. Additionally, there was no significant interaction effect between phase and general worry level for any of these outcome variables. Thus, indicating that there was no significant difference in *RMSSD* HRV, *pNN50* HRV, and *lnHF* HRV change from baseline to the worry induction task between participants with higher levels of general worry compared to participants with lower levels of general worry.

Thus, no support was provided for the hypothesis that higher levels of general worry (as measured using the PSWQ) was associated with lower levels of HRV change during both the pain and worry task.

6.9. Association between pain catastrophising levels and physiological change from baseline to pain task

The hypothesis that higher pain catastrophising will be associated with lower levels of HR and HRV change from baseline to pain task was tested using a series of two by two, mixed between-within measures ANOVAs. The within subjects factor being phase (two levels: baseline and pain task), and the between subjects factor being pain catastrophising level (two levels: higher pain catastrophisers and lower pain catastrophisers). The mean amount of change in HR and each HRV measure from baseline to the pain task was compared between pain catastrophising groups. The descriptive data for each pain catastrophising group are presented in Table 12. The descriptive data for 'higher' and 'lower' pain catastrophising groups presented in Table 12 are for the N = 55 sample, rather than the entire sample (N = 64).

Table 12.

Mean PCS score for higher and lower pain catastrophising participants (N = 55)

		PCS Score
	N	***M* (*SD*)**
Higher pain catastrophisors	29	22.12 (3.75)
Lower pain catastrophisors	26	8.58 (4.22)

Tests revealed that there was a significant main effect of phase in HR, Wilks' Lambda = .87, $F_{1,53}$ = 7.94, p > .05, partial eta squared = .13. All participants were found to increase HR from baseline to the pain task. However, there was found to be no significant interaction effect between phase and pain catastrophising level, Wilks' Lambda = 1.00, $F_{1,53}$ = .29, p > .05, partial eta squared = .01. Thus, indicating that there was no significant difference in HR change from baseline to pain task between higher pain catastrophising participants and lower pain catastrophising participants.

It was shown that there was no significant main effects of phase in *RMSSD* HRV, *pNN50* HRV, and *lnHF* HRV. Additionally, there was found to be no significant interactions effect between phase and pain catastrophising level for these variables. Thus, indicating that there was no significant difference in *RMSSD* HRV, *pNN50* HRV, and *lnHF* HRV change from baseline to pain task between higher pain catastrophising participants and lower pain catastrophising participants.

Therefore, no support is provided for the hypothesis that higher pain catastrophising levels are associated with lower levels of HR and HRV change from baseline to the pain task.

6.10. Association between baseline HRV and pain task outcomes

The hypothesis that lower baseline HRV measures (time domain and frequency domain) were associated with poorer performance at the pain task was investigated by initially screening to see if any relationship existed between baseline HRV measures and pain task outcomes. The nature of any significant associations was investigated with follow-up analyses.

Table 13.

Correlation between baseline HRV measures and pain task outcomes (N = 55)

Variable	Pain Tolerance	Pain Threshold	Pain Intensity
RMSSD (baseline)	.19	.02	.06
pNN50 (baseline)	.22	.04	.04
lnHF (baseline)	.33*	.01	-.04

Note. * $p \leq .05$, two tailed.

The relationship between baseline HRV measures (*RMSSD, pNN50,* and *lnHF*) and pain task outcomes (pain tolerance, pain threshold, and pain intensity) was investigated using Pearson product-moment correlation coefficient. No significant correlation was found between both baseline *RMSSD* or *pNN50* HRV measures and any pain task outcomes. Further, no significant correlation was found between baseline *lnHF* and pain threshold, and pain tolerance. However, there was a medium, positive correlation between baseline *lnHF* HRV and pain tolerance, $r = .33$, $n = 55$, $p < .05$.

A simple linear regression was performed where *lnHF* HRV was regressed on pain tolerance. The model was found to be a good fit for the data ($R^2 = .11$, $F_{1,53} = 6.24$, $p < .05$). *lnHF* HRV predicted approximately 11% of the variance in pain tolerance (from R^2). A one unit increase in *lnHF* HRV resulted in a significant increase in pain tolerance by 14.85 seconds (*Unstandardised Beta* = 14.85), controlling for age and gender.

In summary, some support is provided for the hypothesis that lower baseline HRV leads to poorer pain task performance. However, this was only found for baseline *lnHF* HRV levels and pain tolerance. This finding and the other findings in this section are discussed in more detail and contextualised in the following chapter.

CHAPTER SEVEN

Discussion

This study was conducted to investigate the impact of ethnicity on pain tolerance, pain threshold, subjective pain ratings, and physiological responses to a cold-pressor task. Three major ethnic groups in New Zealand were assessed, namely Maori, Pacific, and European. This selection was based on suggested differences in the understanding of pain, and epidemiological data indicating differences in clinical pain reports between these groups (Coggan et al., 1994; Magnusson & Fennell, 2011; New Zealand Ministry of Health, 2008). Psychosocial variables such as pain catastrophising and general worry were also assessed to investigate the influence of these variables on the relationship between ethnicity and pain. This chapter commences by reporting and contextualising the findings of the present study. This is followed by a discussion of the theoretical and clinical contributions of these findings. Limitations and strengths of the study are then addressed. Finally, a discussion of the general conclusions and possibilities for future research bring the chapter to a close.

7.1. Ethnic Differences

Ethnic differences in performance at pain task

There were no differences between Maori, Pacific and European participants in their experimental pain tolerance levels, pain threshold levels, and subjective pain reports when exposed to a painful cold pressor task. This was unexpected given that previous studies have reported that ethnicity influences pain, including literature specifically concerning the ethnic

groups investigated in the current study (Campbell et al., 2005; Coggan et al., 1994; Forsythe et al., 2011; Magnusson & Fennell, 2011; Rahim-Williams et al., 2007). That said, the current literature on ethnic differences in the experience of pain are considered to be still at a largely theoretical stage. Furthermore, it is important to consider that findings may differ depending on context.

The experimental findings in the current study diverge from epidemiological data on the general population which show that there are differences in the frequency of clinical pain reports between Maori, Pacific and European (New Zealand Ministry of Health, 2008). There are a number of possible reasons for these conflicting findings. As outlined in the introductory chapters, it is apparent that a number of factors, such as the part of the body in which the pain was induced or the characteristics of the experimenter, may influence ethnic differences in pain response in experimental settings (Hastie et al., 2005; Moore & Brodsgaard, 1999; Riley III et al., 2002). As suggested by Magnusson and Fennell (2011), Maori see the non-expression of pain, particularly to unfamiliar persons (for example, non-family members), as a beneficial coping strategy. The experimenter in the current study can be considered to be an unfamiliar person who may have contributed to a level of non-expression of pain in Maori participants leading to lower pain ratings and increased pain thresholds and tolerances than would be expected in this group.

Edwards and colleagues (2001a) highlighted that it is important to consider the ethnicity of the experimenter. It has been reported that individuals may feel more comfortable reporting pain to those of the same ethnicity, compared to those of different ethnicity (Green et al., 2003). In this study, the experimenter was of Pacific Island ethnicity so Pacific Island participants may have experienced a certain degree of comfort expressing their pain during

the experiment. This may have led this group to exhibit lower pain tolerances, report lower pain thresholds, and report higher pain ratings than expected. Finally, the 'ethnic identity' levels and ethnic makeup of each group may have also impacted the pain task outcomes. The ethnic identity levels of the groups in the current study may not have been reflective of the ethnic identity levels of these groups in the general population. It is also important to note that in the Pacific Island ethnic group, a diverse sample of Pacific Island participants were obtained. The group consisted of 13 Indo-Fijians, four Samoans, two Tongans, one Niuean, and one Cook Islander. Though each of these Pacific Island origins were classed as one group, each may possess unique attitudes and responses to pain. These factors may explain why the absence of differences in pain task outcomes between groups conflicted with epidemiological findings, suggesting that differences in pain reports do exist between these groups.

Reliable ethnic differences in both pain thresholds and subjective pain ratings, in an experimental context have not been established. Therefore, the absence of differences in pain thresholds and subjective pain ratings found between groups in the current study acts more as a contribution to the experimental literature (Campbell et al., 2005; Edwards et al., 2005; Greenwald, 1991; Riley III et al., 2002). As mentioned previously, pain thresholds and subjective pain ratings may have been impacted by ethnic-specific reporting biases. Further experimental research regarding ethnic differences in pain thresholds and subjective pain reports is needed to develop firm conclusions. The findings of the current study conflict with the majority of the experimental literature on pain tolerance which suggests that ethnic differences exist (Campbell et al., 2005; Rahim-Williams et al., 2007; Woodrow, Friedman, Siegelaub, & Collen, 1972). There are a number of possibilities for why this occurred. For example, it was found that 75% of the participants kept their hand immersed in the cold water

for the entire three minute task duration. Thus, it is likely that the lack of ethnic differences in pain tolerance is a reflection of possible 'ceiling effects' due to the provision of verbal instructions to try and keep hands immersed in the water for the entire three minutes (von Baeyer et al., 2005). Before the participants were exposed to the painful cold pressor task, they sat through a 'pain preparation phase' in which they were also verbally informed that they were going to experience a fair level of pain during the task which was to follow. The provision of this information may have increased both the expectation and predictability of pain and as a result, contributed to the ceiling effects and subsequent lack of differences in pain tolerance between ethnic groups.

Ethnic differences in HR and HRV, in response to the pain task and in response to the worry task

In line with behavioural responses to the cold pressor task, no differences were found in physiological responses (HR and HRV) to either the cold pressor task or the worry induction task between Maori, Pacific and European participants. This is the first known study to assess HRV as an outcome measure in the Maori, Pacific and European ethnic groups within New Zealand. It is therefore not possible to say whether or not this finding was expected. However, ethnic differences in HRV have been reported in studies outside of New Zealand (Gutin et al., 2005; Lampert et al., 2005). Thus, ethnic differences in HRV in response to both the pain and worry tasks may have been expected. Nonetheless, studies have shown that HRV is age specific and it is possible that the young age of the sample in the present study may have contributed to the non-significant differences in HRV in response to both the pain and worry tasks (Jensen-Urstad et al., 1997; Lipsitz et al., 1990; Ryan et al., 1994).

Ethnic differences in the nature of physiological recovery from the pain task and the worry task

As outlined earlier, HRV is a valid index of the autonomic nervous system's modulation of the heart and provides insight into the body's ability to effectively respond to environmental stressors (Thayer et al., 2009). It has been well established that to effectively assess individual differences in cardiovascular stress reactivity one must not only consider the physiological reactivity to a stressor itself, but also take into account the nature of physiological recovery from the stressor (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004). Thus, the nature of HR and HRV recovery from both the pain and worry task were also assessed.

All ethnic groups were found to have reduced HR during the pain recovery phase compared to the pain task. However, there were no differences in the nature of HR recovery between ethnic groups from the pain task. The finding that HR in the recovery phase was lower than HR during the pain task indicates that the pain task did elicit the expected typical autonomic response (Appelhans & Luecken, 2008; Heller et al., 1984; Kudielka et al., 2004; Terkelsen, Molgaard, Hansen, Andersen, & Jensen, 2005). A review of HRV found that there were no significant changes in HRV from the pain task to the pain recovery phase in the sample as a whole. However, upon further investigation it was found that European participants differed from both Maori and Pacific participants in the nature of their HRV change from the pain task, even while accounting for age, gender and physical activity levels. Specifically, European participants were found to have decreased *RMSSD, pNN50* and *lnHF* HRV levels from the pain task to the pain recovery. On the other hand, Maori and Pacific

participants were found to have increased levels of all three HRV measures from the pain task to the pain recovery.

A decrease in HRV is driven by an increase in sympathetic and a decrease in parasympathetic, neural input into the cardiovascular system. Thus, the decrease in HRV from the pain task to the pain recovery phase shown in European participants suggests that even after the pain task the cardiovascular system remained predominantly sympathetically mediated. This can be considered an atypical physiological recovery pattern following a stressful event such as the pain task (Porges & Byrne, 1992; Sztajzel, 2004). Decreased HRV typically occurs in times of physical or mental stress (Karas et al., 2008). Thus, European participants may have continued to experience stress or mental effort related to the painful task after it had ended. Interestingly, however, European participants were found to report the lowest levels of 'worry related to the painful task' after the painful task, compared to the other ethnic groups. Despite this unusual finding, it is possible that the increased sympathetic activity during the pain recovery phase was due to other forms of mental stress or worry unrelated to the painful task. Atypical forms of vagal recovery, such as that shown by European participants, have previously been linked to poorer cardiovascular stress responses (Porges & Byrne, 1992; Sztajzel, 2004).

The increase in HRV during the pain recovery phase seen in Maori and Pacific participants suggests an increase in parasympathetic activity during the recovery phase. This pattern of recovery from a stressful task has been linked with healthier cardiovascular systems (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999). Maori and Pacific participants appeared to demonstrate healthier cardiovascular autonomic regulation patterns during recovery from the stressful pain task compared to Europeans. This finding contradicts

epidemiological data in New Zealand which suggests that the prevalence of unhealthy cardiovascular conditions is higher in Maori and Pacific Islanders compared to the general population (MacDonald, 2009). Nonetheless, this contradiction has also been found in other recent studies suggesting that various ethnic minority groups demonstrate better autonomic cardiovascular control, yet have poorer cardiovascular health outcomes, compared to individuals of European descent. It has been suggested that in these minority groups, peripheral mechanisms such as those relevant to the vasculature, drive the poorer cardiovascular health outcomes rather than central autonomic mechanisms (Hill, Sollers III, & Thayer, 2010; Wang et al., 2005). This may explain why in the current study, Maori and Pacific participants showed better cardiovascular autonomic function compared to Europeans, yet have poorer cardiovascular health outcomes in general.

Patterns of recovery from the worry task served as a good comparison to the patterns of recovery from the pain task. As with the pain recovery, all ethnic groups were found to have reduced levels of HR during worry recovery compared to the worry task, yet the nature of recovery was not influenced by ethnicity. Again, the overall decrease in HR is a typical pattern of physiological recovery from a mental stressor such as the worry induction task (Linden et al., 1997). No differences were found in any of the HRV measures assessed, from the worry induction task to the worry recovery phase, in the sample as a whole and between ethnic groups. This finding is intriguing as, although differences in HR were found, they were clearly not driven by changes in HRV, thus supporting the notion that the relationship between HR and HRV is non-linear (Eckberg & Sleight, 1992). Again, this suggests that perhaps factors outside of the autonomic nervous system influence the cardiovascular systems ability to recovery from mental stress.

Impact of pain catastrophising and other psychological variables on ethnic differences in pain task outcomes

Research has indicated that catastrophising may mediate ethnic differences in pain response (Fabian et al., 2011). However, in the current study no differences were found in pain catastrophising levels between Maori, Pacific, and European ethnic groups. This finding, along with the absence of differences in pain task outcomes between ethnic groups, means that conclusions about the hypothesised mediating effects of pain catastrophising on ethnic differences in the pain experience cannot be drawn.

Nonetheless, due to the well established role that pain catastrophising has on the pain experience, the impact of pain catastrophising, along with the other psychosocial variables assessed (state anxiety, trait anxiety, pain attention and vigilance, general worry and depression) were further investigated. It was found that higher levels of pain catastrophising-related-rumination were associated with lower pain intensity ratings. Furthermore, higher levels of overall pain catastrophising were associated with higher pain tolerance, as were the helplessness and magnification subscales of pain catastrophising.

These pain catastrophising findings contradict the vast majority of the literature which has indicated that higher pain catastrophising levels tend to lead to a more intense pain experience, lower pain tolerances and reduced pain thresholds (Campbell et al., 2010a; Edwards et al., 2004; Sullivan, Tripp, & Santor, 2000; Thastum et al., 1997). Upon further analysis, higher pain catastrophising levels were found to be associated with higher levels of trait anxiety and higher levels of general worry. Hence, it may have been that those who were more characteristically anxious, or worried more in general, may have simply reported more

catastrophic thoughts about pain due to worries about the experiment or tasks, rather than truly exhibiting high levels of catastrophic thoughts specific to pain. Another possible explanation for response biases is differences in the understanding of pain between the ethnic groups of interest. The PCS questionnaire is based on catastrophic thoughts and feelings about physical experiences of pain. Yet this was not specified in the instructions. As mentioned earlier, evidence suggests that Maori and Pacific people encompass a universal view of pain and closely link physical pain to 'spiritual/emotional pain'. This may have caused confusion for some Maori and Pacific participants when answering the PCS questionnaire, hindering PCS scores. This possibility was reinforced by a number of Maori and Pacific participants who expressed confusion about whether the PCS was asking about physical pain or what they termed, 'emotional pain'. Finally, being an experimental study where participants were exposed to a painful task and were recruited on a volunteer basis, not many participants with high levels of catastrophic thoughts about pain were recruited. In fact, the mean PCS score was 15.13 (with a median score of 15.50), which may be considered low in light of the fact that, upon construction of the PCS, those that scored 15 or below were classed as 'non-catastrophisers' (Sullivan et al., 1995). Given these established norms, approximately half of the participants in the current study may have been considered 'non-pain catastrophisers'. This may have hindered the assessment of how pain catastrophising influenced performance at the painful task.

Median splits were conducted on all psychosocial variables of interest in order to investigate the influences of these variables on behavioural pain task outcomes. It was found that those who exhibited higher levels of trait anxiety, pain vigilance and awareness, general worry, or depression did not differ in pain task outcomes compared to those who exhibited lower levels of each variable. With regards to trait-anxiety and depression, again it is difficult

to say whether the findings surrounding these constructs are in line with what is expected. Some studies have indicated that higher levels of depression have been associated with a heightened pain experience (Euteneuer et al., 2011) and other studies have indicated that higher levels of depression have been associated with a lowered pain experience (Dickens et al., 2003). Comparably, some studies looking at general/trait-anxiety have suggested that higher levels of anxiety are associated with an increased likeliness that one will experience pain (Atkinson et al., 1991), whereas other studies have indicated that higher levels of anxiety may not necessarily be associated with a heightened pain experience (Malow, 1981).

Staats, Hekmat, and Staats (2004) found that levels of general worry were negatively associated with pain tolerance and pain thresholds. Thus, the non-significant difference in pain task outcomes between higher general worriers and lower general worriers is unexpected. Furthermore, the finding that those who exhibited higher levels of pain attention and vigilance did not differ in pain task outcomes compared to those with lower pain attention and vigilance contradicts a vast amount of literature. It has been found that those who attend more to pain and are more vigilant to somatic sensations tend to report heightened pain experiences and unpleasantness (Bantick et al., 2002; Miron, Duncan, & Catherine Bushnell, 1989). Nonetheless, the perceived 'threat value' of a painful task has been shown to moderate the impact of attention to pain on the pain experience (Crombez, Eccleston, Baeyens, & Eelen, 1998b; Eccleston et al., 1997; Peters et al., 2002). Thus, assessment of how threatening the participants found the pain stimulus may have provided insight into such findings.

Despite the absence of differences between the variables mentioned above, it was found that those with higher levels of state anxiety (assessed at the beginning of the

experiment) had higher levels of pain tolerance, higher pain thresholds, and reported lower pain intensity ratings, than those exhibiting lower levels of state anxiety. This finding indicates that the state anxiety experienced by participants may have been related to other factors, for example, anxiety about taking part in an experiment or 'white coat' anxiety (Jhalani et al., 2005). This interpretation is considered likely, as a large amount of literature indicates that non-pain relevant anxiety typically lessens the pain experience (Al Absi & Rokke, 1991; Bobey & Davidson, 1970; Dougher et al., 1987; Malow, 1981; Rhudy & Meagher, 2000).

7.2. Findings on Entire Sample

Association between pain catastrophising and general worry

In support of the hypothesised relationship, the current study found that higher levels of general worry were associated with higher levels of pain catastrophising. This finding is in line with literature which indicates that catastrophising shares similar affective properties with general worry (Keogh et al., 2010; Lackner & Quigley, 2005; Turner & Aaron, 2001). Upon further exploration it was found that higher levels of general worry were associated mostly with higher levels of both pain catastrophising-related-magnification and helplessness. No association was found between general worry and the rumination dimension of pain catastrophising. These findings are also consistent with literature indicating that those that exhibit high levels of worry commonly report negative expectations or consequences for day to day events (i.e. magnification), as well as a lack of confidence in their ability to cope with adverse life experiences (i.e. helplessness) (Brosschot et al., 2006; Turner & Aaron, 2001). The non-significant association between rumination and worry is in line with literature which

indicates that worry and rumination are two separate constructs, where 'worry' tends to be about the future and 'rumination' tends to be about the past (Papageorgiou & Siegle, 2003; Watkins et al., 2005). Only two participants reported having previously taken part in the cold pressor task prior to the current experiment, hence 'ruminative' thoughts (i.e. based on past experiences) about pain specific to the cold-pressor task were unlikely.

Association between general worry levels and HRV change from baseline to the pain task and baseline to the worry task

Contrary to the hypothesis, the current study did not find differences in HRV from baseline to the pain task, or from baseline to the worry task, across the entire sample and when comparing higher worriers to lower worriers. The finding that the worry induction task did not cause any difference in HRV compared to baseline in the total sample is in contrast with a typical HRV response to a mental stressor (Davis, Montgomery, & Wilson, 2002). Mean HRV is expected to decrease in times of stress as HRV becomes predominantly sympathetically mediated in these situations (Karas et al., 2008). Although, it must be noted that on average participants reported being unable to worry normally during the worry induction task. Thus, it may be possible that the level of worry exhibited by the participants during the worry induction task was not adequate to elicit any significant physiological arousal.

The finding that higher worriers and lower worriers did not differ in their physiological arousal levels from baseline to the pain and from baseline to the worry task, conflicts with literature surrounding the 'perseverative cognition' framework (Brosschot et al., 2006). It is suggested that prolonged states of vigilance resulting from the rehearsal of distressing thoughts, as seen in those who possess chronic levels of day-to-day worry, may

lead to chronically enhanced HR and decreased HRV. This chronic physiological activation is found to decrease the flexibility of the autonomic nervous system and hence extreme reactivity to stressors may not be observed (Brosschot et al., 2006; Wells & Morrison, 1994). Thus, the lack of difference in physiological arousal between higher and lower worriers may have been because participants in the current study did not have chronic levels of worry, and hence may not have experienced the physiological consequences of perseverative cognition. In support of this view it has been found that mean PSWQ scores of 56.57 or above are indicative of pathological levels of worry, yet in the current study those who were considered 'higher worriers' only reported a mean PSWQ score of 51.47(Fresco, Mennin, Heimberg, & Turk, 2003).

This is not the first study to report indifferent physiological responses to stressors between individuals exhibiting differing levels of general worry. Davis and colleagues (2002) conducted three experimental investigations, each investigating the relationship between worry and autonomic activity as indexed by HR and HRV. The researchers exposed healthy participants to a series of tasks including public speaking, relaxation, non-stressful cognitive tasks, worried thinking, and an aversive imagery condition. That study found no support for what they termed 'cognitive rigidity' i.e. less physiological reactivity, in those who were classed as 'worriers' when compared to those classed as 'non-worriers'. They also concluded that the effects of chronic worry on autonomic function may be a product of increasing age or may be dependent on the severity of the worry exhibited (Davis et al., 2002).

In the present study, though higher worriers and lower worriers did not significantly differ in their physiological reactivity to either the pain and worry task, baseline HRV levels were generally lower in the higher worrier group. Hence, it is possible that with a larger

sample and greater statistical power this trend may have reached statistical significance and influenced HRV reactivity outcomes.

Association between pain catastrophising levels and physiological change from baseline to pain task

Physiological change from baseline to the pain task was also compared between both higher and lower pain catastrophisers. Average HR was found to increase from baseline to the pain task for the entire sample. However, no difference was found in the nature of HR and HRV change between higher and lower pain catastrophisers. The increase in HR from baseline to the pain task fits with a typical physiological response to stress (Appelhans & Luecken, 2008; Heller et al., 1984; Terkelsen, Molgaard, Hansen, Andersen, & Jensen, 2005). However, the absence of differences in HR and HRV change between higher and lower pain catastrophisers is in contrast to research. It is suggested that increased levels of catastrophic thoughts about pain are associated with decreased cardiovascular reactivity to stressors such as pain (Wolff et al., 2008). Nonetheless, this blunted cardiovascular reactivity observed in high pain catastrophisers has often been shown in clinical settings. Chronic pain sufferers experience pain on a persistent basis, hence catastrophic thoughts about pain are likely to occur more frequently and for prolonged durations compared to a healthy sample. This may contribute to prolonged states of physiological activation and reduced reactivity to stressors in this population (Eccleston et al., 2001; Wolff et al., 2008). Hence, the physiological functioning in a clinical sample may not be comparable to those in a healthy sample such as that used in the present study.

Researchers have also identified that when assessing the influences of pain catastrophising on experimental pain, it is important to consider the time at which pain

catastrophising levels were assessed (George, Dannecker, & Robinson, 2006). For the current study, pain catastrophising levels were assessed at least a day prior to the experiment. Hence, it may be possible that social desirability influenced the reporting of pain catastrophising levels. Outcomes may have been different if this was assessed during or after the pain task. Additionally, participants in the current study were informed that the pain they were going to feel in the experiment had no negative, long term effects and that the task was going to take place in a controlled environment where they had control over how long they wanted to experience pain. This prior knowledge and perceived control over the process may have reduced the threat value of the pain and subsequently reduced the impact of catastrophic thoughts during the painful task, leading to the non-significant findings.

Another factor which may have also contributed to the unexpected finding is the accuracy of the baseline measurements of HR and HRV. In the current study, baseline HR was found to be higher than HR during both the pain recovery and worry recovery phases. Thus, it may be possible that baseline physiological measurements were influenced by various forms of physiologically arousing thoughts such as anxiety related to the experiment, white coat anxiety, or other arousing thoughts outside of the experiment. This slight degree of physiological arousal during the baseline phase may have reduced the amount of physiological reactivity to the pain task, and hindered comparisons between higher and lower pain catastrophisers.

Association between baseline HRV and pain task outcomes

A key finding of this study is that higher baseline *lnHF* HRV levels were associated with higher pain tolerance, while controlling for age and gender. However, baseline *RMSSD* HRV and *pNN50* HRV were not found to correlate with pain task outcomes. The significant

correlation between *lnHF* HRV and pain tolerance is fascinating and adds support to the body of literature suggesting a link between autonomic function and pain (Appelhans & Luecken, 2008; Loggia et al., 2011; Lovick, 1993; Oberlander & Saul, 2002; Randich & Maixner, 1984). Furthermore, a large majority of literature in this area indicates that higher resting HRV is associated with healthier cardiovascular systems, enhanced emotional regulation, better autonomic functioning, and increased cognitive functioning (Asmundson & Wright, 2004; Malik, 1996; Thayer et al., in press; Thayer & Lane, 2007). Hence, the finding that higher baseline *lnHF* HRV (indexing higher resting vagal activity) was associated with better pain tolerance converges with the literature. This finding also points in the same direction as clinical findings which have shown that increases in parasympathetic activity are linked to muscular pain relief in sufferers of chronic pain conditions (Cottingham et al., 1988a; Cottingham et al., 1988b; Ishii et al., 2007; Sakai et al., 2007). Nonetheless, there are still some studies which have shown no relationship between HRV and pain (Meeuse et al., 2010). This, along with the finding that time domain measures of HRV (*RMSSD* and *pNN50*) did not correlate with pain task outcomes, suggests that replications of the significant relationship found between *lnHF* HRV and pain tolerance are needed before any firm conclusions can be made. The theoretical and possible clinical contributions of this finding and the other findings discussed, are outlined in the following section.

7.3. Contributions

The first major contribution of the current study is that it was the first of its kind to assess specific behavioural and physiological responses to experimental pain in Maori, Pacific and European peoples in a controlled environment. No differences in both behavioural and

physiological responses to the pain task and to the worry task were found between these ethnic groups. These findings suggest that it may be external factors which are not present in laboratory settings which contribute to disparities in clinical pain reporting between these groups.

The second key contribution of this research is that it provided a snap-shot of the autonomic stress recovery patterns of Maori, Pacific, and European peoples. HRV was found to decrease in European participants after the pain task, whereas in both Maori and Pacific participants it was found to increase after the pain task. Despite these differences in HRV recovery patterns, all ethnic groups were found to have decreased HR after the pain task. Thus, though all participants showed a typical stress recovery pattern in terms of decreased HR after the pain task, the different ethnic groups may have different underlying mechanisms driving the decrease in HR.

Another contribution of this study is that it provides further support for the notion that pain catastrophising and general worry are overlapping constructs. Higher levels of pain catastrophising were found to be associated with higher levels of general worry. This association was found to be greatest for the 'helplessness' and 'magnification' components of pain catastrophising. This finding carries potential clinical relevance as it adds support to existing literature indicating that worry and catastrophising share similar characteristics (Keogh et al., 2010; Lackner & Quigley, 2005). It suggests that in terms of pain management, it may not only be a patient's catastrophic thoughts about pain that may need to be addressed, but also a patient's tendency to worry in general. Nonetheless, the correlational nature of this finding limits its applicability.

The final major contribution of the present study is that it provided support for a link between autonomic regulation and the pain experience. Higher resting parasympathetic activity was associated with increased pain tolerance during a cold pressor task. Enhanced vagal outflow at rest (indexed by increased HRV) is found to link to better cardiovascular regulation in times of demand and has been shown to enhance cognitive and emotional functioning (Malik, 1996; Porges & Byrne, 1992; Sztajzel, 2004; Thayer et al., in press; Thayer & Lane, 2007). This finding can be used to direct future research into how potential physiological mechanisms linked to the pain experience, specifically parasympathetic pathways, can be used to potentially enhance pain tolerance in clinical settings.

7.4. Limitations

The characteristics of the sample may be considered a limitation as they restrict the generalisability of the findings. The majority of the participants were university students and the entire sample had a mean age of 30 years. Both pain and HRV are influenced by age (Lautenbacher et al., 2005) and therefore the study's findings may have differed in an older or younger sample. Furthermore, though pain catastrophising was a major variable of interest in the present study, the pain catastrophising range was limited as it was found that there were a low number of 'high pain catastrophisers'. Nonetheless, studies that use pain induction as part of their protocol and recruit on a volunteer basis are exposed to the risk of recruiting participants who are more comfortable with pain, rather that those who possess high levels of catastrophic thoughts about pain.

It is important to note that the small sample size of the current study limits the applicability of the findings to only those included on the current study and are not

generalisable. The ethnic-specific findings are not representative of all Maori, Pacific, and European people. Furthermore, the participants self identified their ethnicity. No objective measures were used to confirm how genetically accurate the ethnic makeup of each participant was. Thus, any ethnic-specific conclusions need to be replicated in larger samples and researchers should endeavour to utilise more accurate measures of ethnicity, such as biomarkers (Davies, Villablanca, & Roderick, 1999). Psychosocial variables measured in the current study were assessed by using self report measures. Thus, the variables assessed were based on the participants' perceptions and may not have been an accurate representation of the participant. All measures used were written in English, therefore those who lacked understanding of words or phrases used in the questionnaires may have been hindered by their ability to accurately answer questions. Furthermore, self-report measures are exposed to the impacts of social desirability. For example, participants may have answered questions based on what they felt the 'correct answer' was, or what made them 'look good' (Crowne & Marlowe, 1960). Social desirability could have been assessed in the current study, yet this limitation was weighed against the potential risk of overburdening the participants with a high volume of questionnaires to complete. HR and HRV were chosen as the optimal physiological measures to assess the outcomes of interest, yet other measures could have been used to provide a more comprehensive picture of physiological responses to pain. This may have included the assessment of changes in blood pressure, which has been used in research of a similar nature (Peckerman et al., 1991).

In the current study, participants were asked to watch a DVD as an innocuous filler task during the baseline, pain recovery and worry recovery phases. The DVD was administered at all three phases to allow for standardised comparisons. Nonetheless, innocuous filler tasks are not typically used during the assessment of ‘physiological recovery’

from a stressor as it risks impacting the physiological recovery process. In this study the DVD may have 'dampened' the variability in HR and HRV during the recovery phases.

The limitations inherent in all experimental research are also important to consider. The findings in this study were obtained in a controlled environment and thus, any associations found cannot be directly applied to clinical settings such as to chronic pain patients. However, once the findings are replicated and links become more established, they can be used to guide future research or the development of psychological/physiological interventions to improve pain outcomes.

Despite these limitations, the study had a number of strengths. A major strength of the experimental protocol was that the experimental phases were counterbalanced. Participants either received the worry induction task first or the cold pressor task first. This was done to control for any order-effects of the experimental protocol, e.g. carry over effects of experiencing pain first or worry first. The study also assessed both subjective and objective markers of the pain. Not only were participants asked to report their experiences of pain, but behavioural (performance at pain task) and physiological (changes in HR and HRV) were also assessed to provide comprehensive insight into each participants' pain experience.

Another strength of the current study is that it addresses the lack of literature comparing the similarities and differences in the pain experience between Maori, Pacific and European people of New Zealand. As mentioned earlier, the few comparisons that have been made have compared the outcomes of different studies and are therefore exposed to the limitations of comparing different samples. The current study was also the first known study to compare the pain experiences of all three ethnic groups in one study. Another strength is

that the targeted sample size was obtained. Comparable numbers of both males and females in each ethnic group were obtained, and the groups were age matched. Finally, the study took into account factors such as age, gender, health behaviours, health status, cold pressor task hand use and past experience, as these factors are found to impact pain responses.

7.5. General Conclusions

The current study found no differences in both behavioural and physiological responses to the painful task between Maori, Pacific, and European ethnic groups. Thus, the findings are not in line with clinical research suggesting differences in the reporting of pain between these groups (New Zealand Ministry of Health, 2008). It is likely that the highly controlled experimental settings reduced social and environmental influences on the pain experience which are found to be of unique importance in Maori and potentially Pacific peoples. Despite the lack of ethnic differences in physiological reactivity to the pain task, European participants were found to have reduced levels of HRV during recovery from the pain task whereas Maori and Pacific participants were found to have higher levels of HRV during pain recovery. This suggests that European participants had poorer autonomic regulation of the cardiovascular system compared to both Maori and Pacific participants.

Pain catastrophising was a major variable of interest in the current study due to its potential impact on pain experience and its potential role as a mediator of ethnic differences in pain reports (Fabian et al., 2011; Sullivan et al., 1995). However, due to the absence of differences in pain task outcomes between ethnic groups, and the lack of differences in pain catastrophising levels between ethnic groups, conclusions about its mediating role could not be established. While not specifically investigated, higher pain catastrophising levels were

found to be associated with better pain task outcomes and lower pain ratings. This finding is unusual, yet may be attributable to biases in the reporting of pain catastrophising or the narrow pain catastrophising score range. Participants who were found to have higher levels of pain catastrophising were also found to worry more in general.

Surprisingly, the current study did not find differences in physiological reactivity to both the pain task and the worry task when comparing both higher pain catastrophisers to lower pain catastrophisers, and higher general worriers to lower general worriers. Thus, the study did not provide support for the well established framework of perseverative cognition (Brosschot et al., 2006). Despite this unexpected finding, higher baseline *lnHF* HRV was found to be associated with higher pain tolerance. This finding is of particular importance as it provides reinforcement for the link between autonomic function and regulation of the stress response (Benarroch, 1993; Thayer & Lane, 2000; Thayer & Lane, 2009).

The findings and design of the current study present some appealing future directions. The particular areas focused on were the impact of ethnicity on pain and the associations between key psychosocial variables found to impact pain, and physiological mechanisms underlying pain. Future research should be aimed at the replication and application of these findings into clinical settings.

7.6. Future Research

Being the first known study to experimentally assess responses to pain in Maori, Pacific and European peoples of New Zealand, any significant results are considered preliminary and

require replication. For example, the unexpected finding that European participants have poorer physiological recovery patterns than Maori and Pacific participants in response to the pain task is in need of replication. This finding may have been a product of the relatively young sample, hence replications in older sample would be of merit. Particularly, as in older samples the prevalence of cardiovascular conditions are higher and thus are likely to show different outcomes to the present study.

Replications should also aim to improve the limitations of the current study. Pain catastrophising was one of the major variables of interest in the current study. Yet due to the nature of the study there were not many high pain catastrophising individuals recruited. Hence, replications of these types of studies may need to recruit larger sample sizes to adequately compare varying levels of pain catastrophising. Further, ethnic differences in response to experimental pain are found to be influenced by the area of the body in which the pain is induced (Hastie et al., 2005; Moore & Brodsgaard, 1999; Riley III et al., 2002). Thus, despite the lack in ethnic differences during the pain task, future replications should also utilise other pain induction techniques, and induce pain at different sites on the body.

The absence of differences in pain response observed between Maori, Pacific and European participants may indicate that differences in the experience of pain reported between these groups may be due to factors which are not present in an experimental context. Thus, qualitative research may also be useful to enhance understandings of pain in Maori and Pacific peoples. A comprehensive study on Maori views and perceptions of pain was conducted by Magnusson and Fennell (2011). Not only do their findings need replication but no such study has been published on Pacific Island people of New Zealand, highlighting an area in need of research. The pain questionnaires used in the present study have not been

'normed' on Maori and Pacific peoples. Thus, qualitative research may also form the basis for the modification of existing questionnaires or the development of new questionnaires which assess aspects of the pain experience important to Maori and Pacific peoples.

The finding that Maori and Pacific participants had better autonomic control getting HR back down to a tonic level after the pain task compared to European participants, conflicts with epidemiological data suggesting poorer cardiovascular health in Maori and Pacific people compared to Europeans (MacDonald, 2009). This finding opens a door for future research looking at the mechanisms driving the ethnic disparities in cardiovascular health in New Zealand. Future investigations should aim to look at both central autonomic function and compare it to vascular function, perhaps in response to both induced physical and mental stress in Maori and Pacific groups. It may also be of merit to conduct such investigations in an older age sample, which may provide insight into when ethnic specific cardiovascular health disparities occur.

Finally, the finding that higher baseline *lnHF* HRV was associated with higher pain tolerance provided further support for the link between autonomic function and pain processing. Thus, future research may be directed towards developing new techniques, or improving current methods, aimed at increasing pain tolerance by potentially altering the function of the autonomic nervous system. Based on the current findings and previous studies, these techniques should be tailored towards increasing vagal activity.

The current study has presented findings which have shed light on ethnic specific characteristics of the pain experience and physiological mechanisms associated with pain. It has also provided insight into cardiovascular stress recovery patterns of Maori, Pacific and

European people of New Zealand. This research leaves us with ideas which, in conjunction with further research, carry the potential to be clinically applied to help individuals suffering chronic pain conditions live more functional and fulfilling lives.

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APPENDIX A

Ethical Approval, Participant Information Sheet

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MAORI, PACIFIC, AND EUROPEAN DIFFERENCES IN PAIN RESPONSE

PARTICIPANT INFORMATION SHEET

I am Sandeep Deo, a student researcher in the Department of Psychological Medicine. My supervisors are Malcolm Johnson and Dr. John Sollers, senior lecturers in the Department of Psychological Medicine. We are inviting *adults* (18 years and above) who identify primarily as *either* of Maori, Pacific Island or New Zealand (NZ) European ethnicity to participate in a study which looks at the variations in the time between your heart beats ("Heart rate variability") when you are experiencing pain, when you think about painful experiences, and when you are worrying in general. HRV gives us an indication of how your nervous system is working in your body.

We are searching for *adults* without pain or heart conditions that can speak and read English for the study. The study comprises of two stages: (1) initial assessment, and (2) the experimental phase. If you choose to participate in the study we would like you to complete stage one of the study now, which is to complete some questionnaires asking about your patterns of thinking and worry. This will take about 20 minutes. We would also like you to give us a contact telephone number.

Then, based on your questionnaire we will contact some of you to see if you are willing to participate in stage two of the study. If you agree, when you come to do the experiment we will ask you to fill in a short questionnaire which asks the same health questions to confirm that you are okay for the study, some more questions that relate to heart rate variability, and a second consent form.

The experiment lasts one hour and involves looking at your heart rate variability at rest, when you are worrying, and when you have your hand in icy cold water ("cold pressor task"; experiencing pain).
For the pain task, you put your hand in the water for up to three minutes and you can take your hand out whenever you want. For the worry task we ask you to write down three worries you have and then to think about them for a short time. For the measurement of heart rate variability we will connect you to a heart rate monitor just above your bottom ribs. For women we will have a female researcher to help. The experiment takes about 1 hour and we will give you a $30.00 voucher on completion to compensate you for any inconvenience.

You can withdraw from the study and have your data removed and destroyed at any time until completion of the experimental study. At that time we will transfer all your data anonymously onto computer files and will no longer be able to identify which data belongs to which participant. These computer files and your questionnaires will

be saved and stored for six years and then destroyed. The study is a Masters level thesis with costs from Department of Psychological Medicine, University of Auckland research funding, any information collected and results obtained will be specific to this thesis. The final project results may be submitted as a research paper in relevant scientific journals. We hope to build on our understanding of thinking patterns, worry, and pain and hope that this information will eventually benefit chronic pain sufferers.

If you would like to receive a report on the findings from the study please put an address on the consent form and we will send you a summary in about twelve months.

Thank you for reading this and considering our study.

Please keep this sheet for your information and return the consent form and questionnaire to us in the enclosed pre-paid return envelope.

Sandeep Deo, Dr John Sollers and Malcolm Johnson,

Head of Department Professor Robert Kydd
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For any queries regarding ethical concerns you may contact the Chair, The University of Auckland Human Participants Ethics Committee, The University of Auckland, Office of the Vice Chancellor, Private Bag 92019, Auckland 1142. Telephone 09 373-7599 extn. 83711.

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/202

APPENDIX B

Questionnaire A: Consent Form A, Screening, and Initial Psychosocial Data Collection

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PARTICIPANT CONSENT FORM A

This form will be stored for a period of 6 years.

MAORI, PACIFIC, AND EUROPEAN DIFFERENCES IN PAIN RESPONSE

A research study by: Sandeep Deo, Dr John Sollers, and Malcolm Johnson.

- I have read and understood the Participant Information Sheet and know why I have been asked to participate.
- I understand that participation in this study is voluntary and will take me about 20 minutes to complete the questionnaire now and 45 minutes to do the experimental study later.
- I know that I can do the questionnaire part and might not be contacted about the experimental part.
- I know that if I am contacted about the experimental part I will be able to choose again whether to participate and I am able to withdraw at any time and my data will be destroyed until I have completed the experimental study when all my data will be recorded anonymously on the research database.
- I am aware that the experimental study asks me to think and worry about something relevant to me and also that I will be exposed to painful stimulation.
- I am aware that I can receive a summary of the findings of the research when completed if I wish and provide an address below
- I understand that the research data will be stored for 6 years and then destroyed.

I agree to take part in this research

Name……Signature……
Date……

My phone number to arrange to send the questionnaires and later to arrange an appointment for the experimental study is ……..

If you wish for a summary of the findings of the study to be sent to you, please provide a physical contact address and/or an email address on the next page. If not, please go to page 3.

I would like summary of the findings to be sent to (please select method and provide details):

☐ Email: ……

AND/OR

☐ Physical Address: ……...

……..

……..

……..

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/202

QUESTIONNAIRE

PARTICIPANT ID #……

Today's date: …… Your age: ……..

Your Gender …….

Your Ethnicity (The ethnicity you ***primarily*** identify with): ……

Your highest academic qualification: (Please tick one)

	NCEA level 1 / 5th form certificate
	NCEA level 2 / Year 12 secondary school
	NCEA level 3 / Bursary
	Tertiary level qualification
	Other

Please answer the following 20 questions referring to the ethnicity you ***primarily* identify with (as answered above). Use the numbers given below to indicate how much you agree or disagree with each statement (Please circle the most appropriate number).**

1. *Strongly Disagree* **2.** *Somewhat Disagree* **3.** *Somewhat Agree* **4.** *Strongly Agree*

1. I have spent time trying to find out more about my own ethnic group, such as its history, traditions and customs…… 1 2 3 4

2. I am active in organizations or social groups that include mostly members of my own ethnic group…… 1 2 3 4

3. I have a clear sense of my ethnic background and what it means for me…….. 1 2 3 4

4. I like meeting and getting to know people from ethnic groups other than my own…… 1 2 3 4

5. I think a lot about how my life will be affected by my ethnic group membership…… 1 2 3 4

6. I am happy that I am a member of the group that I belong to…… 1 2 3 4

7. I sometimes feel it would be better if different ethnic groups didn't mix together…… 1 2 3 4

8. I am not very clear about the role of ethnicity in my life…… 1 2 3 4

9. I often spend time with people from ethnic groups other than my own…… 1 2 3 4

10. I really have not spent much time trying to learn more about the practices and history of my ethnic group…… 1 2 3 4

11. I have a strong sense of belonging to my own ethnic group…… 1 2 3 4

12. I understand pretty well what my ethnic group membership means to me, in terms of how to relate to my own group and other groups…… 1 2 3 4

13. In order to learn more about my ethnic background I have often talked to other people about my ethnic group…… 1 2 3 4

14. I have a lot of pride in my ethnic group and its accomplishments…… 1 2 3 4

15. I don't try to become friends with people of other ethnic groups…… 1 2 3 4

16. I participate in the practices of my own group such as special food, music or customs…… 1 2 3 4

17. I am involved in activities with people from other ethnic groups…… 1 2 3 4

18. I feel a strong attachment towards my own ethnic group…… 1 2 3 4

19. I enjoy being around people from ethnic groups other than my own…… 1 2 3 4

20. I feel good about my ethnic background……1 2 3 4

A number of statements which people have used to describe themselves are given below

Read each statement below and then circle the appropriate number to the right of the statement **to indicate how you <u>generally</u> feel.**

1 = Almost Never
2 = Sometimes
3 = Often
4 = Almost Always

	Almost Never	Sometimes	Often	Almost Always
21. I feel pleasant	1	2	3	4
22. I feel nervous and restless	1	2	3	4
23. I feel satisfied with myself	1	2	3	4
24. I wish I could be as happy as others seem to be	1	2	3	4
25. I feel like a failure	1	2	3	4
26. I feel rested	1	2	3	4

27. I am "calm, cool and collected"	1	2	3	4
28. I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
29. I worry too much over something that really doesn't matter	1	2	3	4
30. I am happy	1	2	3	4
31. I have disturbing thoughts	1	2	3	4
32. I lack self-confidence	1	2	3	4
33. I feel secure	1	2	3	4
34. I make decisions easily	1	2	3	4
35. I feel inadequate	1	2	3	4
36. I am content	1	2	3	4
37. Some unimportant thought runs through my mind and bothers me	1	2	3	4
38. I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
39. I am a steady person	1	2	3	4
40. I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

Thinking about when you have experienced pain, circle the number that indicates the degree to which you have experienced each of the following thoughts or feelings. 0 = not at all to 4 = all the time

1. I worry all the time about whether the pain will end.
   0 1 2 3 4
   not at all — all the time

2. I feel I can't go on.
   0 1 2 3 4
   not at all — all the time

3. It's terrible and I think it's never going to get any better.
   0 1 2 3 4
   not at all — all the time

4. It's awful and I feel that it overwhelms me.
   0 1 2 3 4
   not at all — all the time

5. I feel I can't stand it anymore.
   0 1 2 3 4
   not at all — all the time

6. I become afraid that the pain may get worse.
   0 1 2 3 4
   not at all — all the time

7. I think of other painful experiences.

0	1	2	3	4
not at all				all the time

8. I anxiously want the pain to go away.

0	1	2	3	4
not at all				all the time

9. I can't seem to keep it out of my mind.

0	1	2	3	4
not at all				all the time

10. I keep thinking about how much it hurts.

0	1	2	3	4
not at all				all the time

11. I keep thinking about how badly I want the pain to stop.

0	1	2	3	4
not at all				all the time

12. There is nothing I can do to reduce the intensity of the pain.

0	1	2	3	4
not at all				all the time

13. I wonder whether something serious may happen.

0	1	2	3	4
not at all				all the time

Please indicate by circling one of the below, how typical the following statements are of you.

1 = not at all typical, to 5 = very typical

		not at all typical of me				very typical of me
1	If I do not have enough time to do everything, I do not worry about it	1	2	3	4	5
2	My worries overwhelm me.	1	2	3	4	5
3	I do not tend to worry about things.'	1	2	3	4	5
4	Many situations make me worry.	1	2	3	4	5
5	I know I should not worry about things, but I just cannot help it	1	2	3	4	5
6	When I am under pressure I worry a lot.	1	2	3	4	5
7	I am always worrying about something.	1	2	3	4	5
8	I find it easy to dismiss worrisome thoughts.'	1	2	3	4	5
9	As soon as I finish one task, I start to worry about everything else I have to do	1	2	3	4	5

10	I never worry about anything.	1	2	3	4	5
11	When there is nothing more I can do about a concern, I do not worry about it anymore.	1	2	3	4	5
12	I have been a worrier all my life.	1	2	3	4	5
13	I notice that I have been worrying about things.	1	2	3	4	5
14	Once I start worrying, I cannot stop.	1	2	3	4	5
15	I worry all the time.	1	2	3	4	5
16	I worry about projects until they are all done.	1	2	3	4	5

Thinking about when you have experienced pain, circle the number that best describes your experience for each of the below statements.

0 = Never – 5 = Always

		Never					Always
1	I am very sensitive to pain.	0	1	2	3	4	5
2	I am aware of sudden or temporary changes in pain.	0	1	2	3	4	5
3	I am quick to notice changes in pain intensity.	0	1	2	3	4	5
4	I am quick to notice effects of medication on pain.	0	1	2	3	4	5
5	I am quick to notice changes in location or extent of pain.	0	1	2	3	4	5
6	I focus on sensations of pain.	0	1	2	3	4	5
7	I notice pain even if I am busy with another activity.	0	1	2	3	4	5
8	I find it easy to ignore pain,	0	1	2	3	4	5
9	I know immediately when pain starts or increases.	0	1	2	3	4	5
10	When I do something that increases pain, the first thing I do is check to see how much my pain was increased,	0	1	2	3	4	5
11	I know immediately when pain decreases.	0	1	2	3	4	5
12	I seem to be more conscious of pain than others.	0	1	2	3	4	5
13	I pay close attention to pain.	0	1	2	3	4	5
14	I keep track of my pain level.	0	1	2	3	4	5
15	I become preoccupied with pain.	0	1	2	3	4	5
16	I do not dwell on pain.	0	1	2	3	4	5

PARTICIPANT ID #……

Below is a list of the ways you might have felt or behaved. Please tell me how often you have felt this way during the past week.

0 = Rarely or none of the time (less than 1 day)
1 = Some or a little of the time (1-2 days)
2 = Occasionally or a moderate amount of time (3-4 days)
3 = Most or all of the time (5-7 days)

		Rarely			Most /all
1	I was bothered by things that usually don't bother me.	0	1	2	3
2	I did not feel like eating; my appetite was poor	0	1	2	3
3	I felt that I could not shake off the blues even with help from my family or friends	0	1	2	3
4	I felt I was just as good as other people	0	1	2	3
5	I had trouble keeping my mind on what I was doing	0	1	2	3
6	I felt depressed	0	1	2	3
7	I felt that everything I did was an effort	0	1	2	3
8	I felt hopeful about the future	0	1	2	3
9	I thought my life had been a failure.	0	1	2	3
10	I felt fearful	0	1	2	3
11	My sleep was restless	0	1	2	3
12	I was happy	0	1	2	3
13	I talked less than usual	0	1	2	3
14	I felt lonely	0	1	2	3
15	People were unfriendly	0	1	2	3
16	I enjoyed life	0	1	2	3
17	I had crying spells	0	1	2	3
18	I felt sad	0	1	2	3
19	I felt that people dislike me	0	1	2	3
20	I could not 'get going'	0	1	2	3

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/202

MEDICAL CHECKLIST

Participant name..... PARTICIPANT ID No……

The information from this checklist is to make sure the procedures do not present a risk to you. The information will not be included in the study or recorded with any of the other study data.
We will keep this medical checklist in a locked filing cabinet separate from the other information from this experiment accessible only to the researchers for six years.

Please answer the following questions:

1. Have you ever had any form of epilepsy? yes/no
2. Are you currently using medication of any type? yes/no
   (If you are we will ask you about this)
3. Do you have any known heart or circulatory condition? yes/no
4. Do you have hypertension? yes/no
5. Do you suffer from any skin disorders? yes/no
6. Are you in good general health? yes/no
7. In the past six months, have you suffered from any painful injury or condition lasting more than a week? yes/no
8. Have you ever had any injury or medical condition that may have affected your ability to sense pain? yes/no

Signature..... Date.....

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/202

APPENDIX C

Questionnaire B: Consent Form B, Pre- and Post-Experimental Questionnaire

Department of Psychological Medicine

THE UNIVERSITY OF AUCKLAND
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Facsimile: 64 9 3737013
Email: mh.johnson@auckland.nz

PARTICIPANT CONSENT FORM B

This form will be stored for a period of 6 years.

MAORI, PACIFIC, AND EUROPEAN DIFFERENCES IN PAIN RESPONSE

A research study by: Sandeep Deo, Dr John Sollers, and Malcolm Johnson.

- I have read and understood the Participant Information Sheet and know why I have been asked to participate.
- I understand that participation in this study is voluntary and will take me about 45 minutes to do the experimental study.
- I know I am able to withdraw at any time and my data will be destroyed, until I have completed the experimental study when all my data will be recorded anonymously on the research database.
- I am aware that the experimental study asks me to think and worry about something relevant to me and also that I will be exposed to painful stimulation.
- I understand that the research data will be stored for 6 years and then destroyed.

I agree to take part in this research

Name……Signature……
Date……

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/202

MEDICAL CHECKLIST AND QUESTIONNAIRE

Participant name..... Participant ID #.....

The information from this checklist is to make sure the procedures do not present a risk to you. The information will not be included in the study or recorded with any of the other study data. We will keep this medical checklist in a locked filing cabinet separate from the other information from this experiment accessible only to the researchers for six years.

Please answer the following questions by circling either yes or no:

9. Have you ever had any form of epilepsy? Yes No
10. Are you currently using medication of any type? Yes No
    If yes please indicate:.....
11. Do you have any known heart or circulatory condition? Yes No
12. Do you have hypertension? Yes No
13. Do you suffer from any skin disorders? Yes No
14. Are you in good general health? Yes No
    If no, please indicate the problem .....
15. In the past six months, have you suffered from any painful injury or condition lasting more than a week? Yes No
    If yes, please indicate what area is affected……..
16. Have you ever had any injury or medical condition that may have affected your ability to sense pain? Yes No

Signature..... Date.....

Below are some questions that are relevant in measuring heart rate variability. Please note that the information you provide will not be linked to you in any presentation of research findings.

H1. Height _____ H2. Weight _____

H3. Ethnicity (The ethnicity you *primarily* identify with): _____

H4. Please circle one of the below to indicate the most accurate description of the amount of activity in your day to day life

(1) Inactive (3) Moderately active
(2) Mildly active (4) Very active

H5. Please describe the **main type** of activity / exercise you get (e.g., sports, gym, housework, daily walking)

_____

H6. Do you smoke? _____ If yes, or you used to smoke, please indicate:

(a) how long have you ,or did you smoke for: _____

(b) on average, how many cigarettes per day did you smoke: _____

H7. Please describe your average *daily* caffeine intake (e.g., 3 cups of instant coffee).

_____

H8. Please indicate, on average, how many units of alcohol you consume *per week* _____

H9. Please indicate how many days in the *past month* you experienced disruptions in sleep, including trouble falling asleep, waking too early, trouble staying asleep: _____

H10. Have you consumed any caffeine **today**?
(Pease circle one and note the amount if applicable) NO / YES (Amount = ...)

H11. Have you consumed any alcohol **today**?
(Pease circle one and note the amount if applicable) NO / YES (Amount = ...)

H12. Have you smoked any cigarettes **today**?
(Pease circle one and note the amount if applicable) NO / YES (Amount = ...)

Read each statement below and then choose the appropriate number to the right statement to indicate how you feel <u>right now</u>, that is, at this moment.

1 = Not at all
2 = Somewhat
3 = Moderately so
4 = Very much so

	Not at all	Somewhat	Moderately so	Very much so
1. I feel calm ……	1	2	3	4
2. I feel secure ……...	1	2	3	4
3. I am tense …….....	1	2	3	4
4. I feel strained …….....	1	2	3	4
5. I feel at ease ……	1	2	3	4
6. I feel upset ……..	1	2	3	4
7. I am presently worrying over possible misfortunes ……	1	2	3	4
8. I feel satisfied ……..	1	2	3	4
9. I feel frightened ……	1	2	3	4
10. I feel comfortable ……	1	2	3	4
11. I feel self-confident ……..	1	2	3	4
12. I feel nervous ……	1	2	3	4
13. I am jittery ……	1	2	3	4
14. I feel indecisive ……	1	2	3	4
15. I am relaxed ……..	1	2	3	4
16. I feel content …….	1	2	3	4
17. I am worried …….	1	2	3	4
18. I feel confused ……..	1	2	3	4
19. I feel steady ……..	1	2	3	4
20. I feel pleasant ……	1	2	3	4

PLEASE STOP HERE
THE REMAINING PAGE TO BE COMPLETED AT THE END OF THE EXPERIMENT

POST EXPERIMENT QUESTIONS

THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND HEALTH SCIENCES

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Private Bag 92019
Auckland
New Zealand,

85 Park Road, Grafton
www.health.auckland.ac.nz

Telephone: 64 9 373 7599
Facsimile: 64 9 3737013
Email: mh.johnson@auckland.nz

Date:			
Participant Name:		ID#:	

Please answer each of the below questions by circling one number to the right.
0 = not at all, 5 = very much so.

	Not al all					Very much so
To what extent did you worry about participating in this experiment prior to taking part?	0	1	2	3	4	5
To what extent did you worry about the pain you might experience directly before the icy water / cold pressor task began (i.e. during the 2 minute temperature control condition).	0	1	2	3	4	5
To what extent did you continue to think about the pain associated with the cold pressor task after it had finished (i.e., while watching the DVD)	0	1	2	3	4	5
During the worry task, to what extent were you able to worry in a way that you normally worry?	0	1	2	3	4	5
To what extent did you continue to worry after the worry task had finished (i.e. while watching the DVD)?	0	1	2	3	4	5
To what extent did you find participating in this experiment worrisome (in general)	0	1	2	3	4	5

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/202

THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND HEALTH SCIENCES

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Email: mh.johnson@auckland.nz

MAORI, PACIFIC, AND EUROPEAN DIFFERENCES IN PAIN RESPONSE

RECEIPT

I, …….. (full name), have received $30 worth of petrol vouchers for participating in the Maori, Pacific, and European differences in Pain response.

Signature……..…Date……..

Thank you for participating in the study
Sandeep Deo, Malcolm Johnson, and John Sollers III

APPROVED BY THE UNIVERSITY OF AUCKLAND HUMAN PARTICIPANTS ETHICS COMMITTEE ON 30 May 2011 for (3) years, Reference Number 2011/20

APPENDIX D

Experimental Data Collection and Protocol Form

Department of Psychological Medicine

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MAORI, PACIFIC, AND EUROPEAN DIFFERENCES IN PAIN RESPONSE

EXPERIMENTAL PROCEDURES SHEET

Date:	
Experimenter:	SD
Participant Name:	
Participant ID:	
Condition:	1. P-W 2. W-P
Done Cold Pressor Before?	Y / N
Dominant Hand?	R / L
Start Time	
Pain Threshold	
Pain Tolerance	

CONDITION 1		Task time	Time Mark
1	HRV Time mark: Baseline HRV recording (play DVD) (* Write out petrol voucher receipt * Fill out page 4 of questionnaire)	5 mins	0
2	HRV time mark: end baseline. Get them to move over to water bath area	minimal	
3	HRV time mark – hand in room temperature water (*Explain the cold pressor task)	2 mins	Time started: ……
4	HRV Time mark: remove hand from body temperature water, move over to CPT. Stir cold water		
5	HRV time mark as they place hand in cold water (until bar clicks)	Until tolerance	Time started: ……
6	HRV time mark when they remove hand. Dry off hand. Turn to face DVD.	minimal	Time feel pain ..... Time removed hand:……..
7	Play recovery DVD	8 mins	Time started:……..
8	HRV time mark explain worry task	minimal	
9	HRV Time mark: worry induction	5 mins	Time started:……
10	HRV Time mark: tell them to stop. Ask them to move to position to watch DVD	minimal	
11	HRV Time mark: Recovery DVD	8 mins	Time started:……
12	HRV time mark. Stop.		

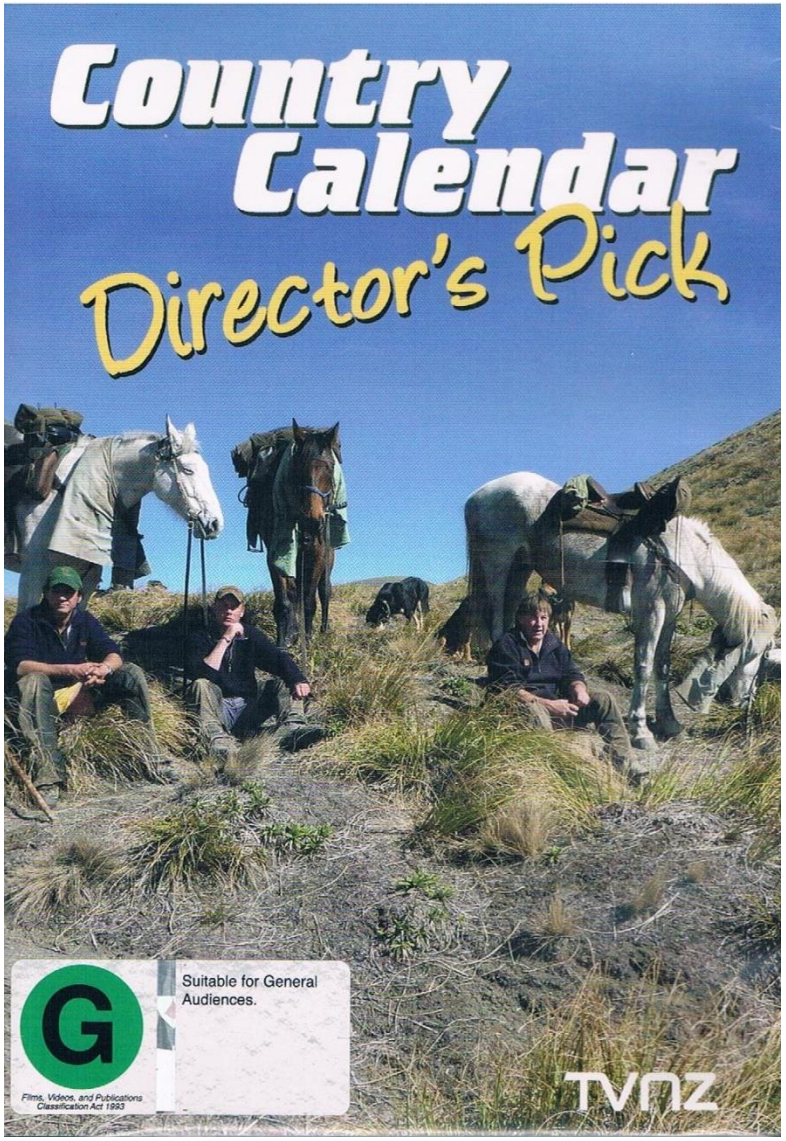
* Fill out last pages of questionnaire + receipt

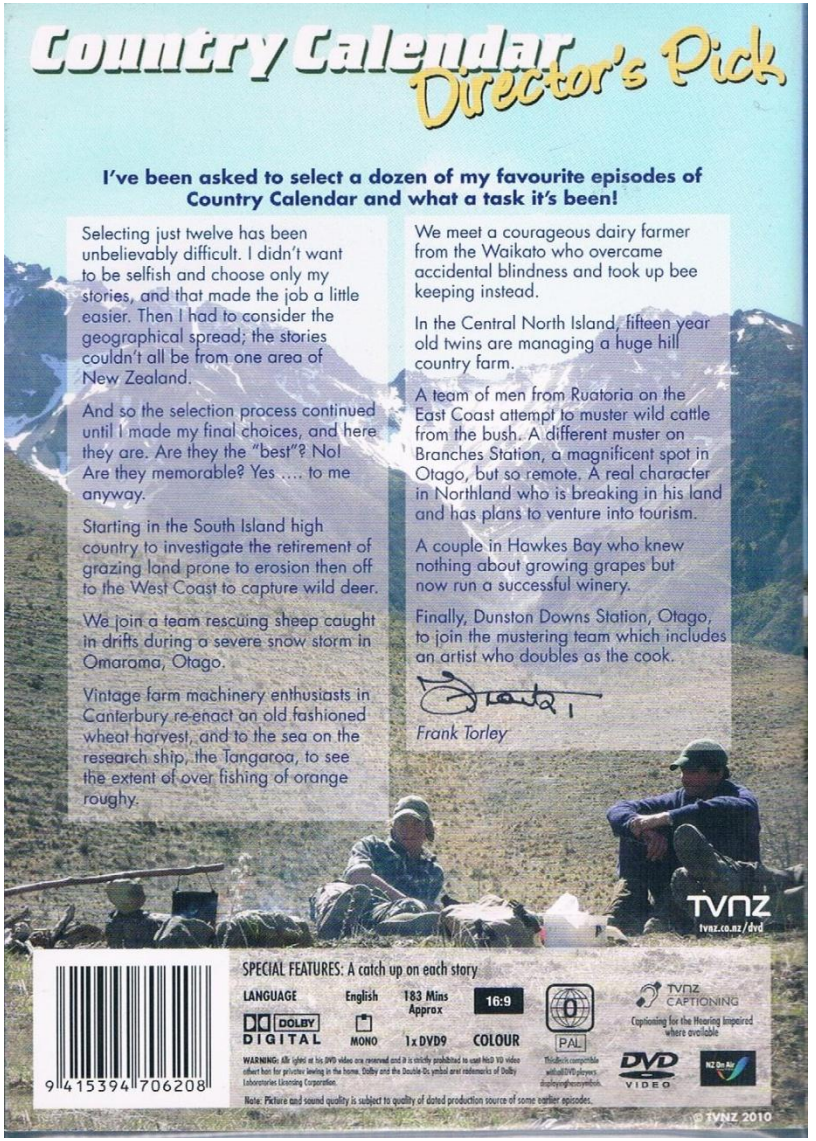
CONDITION 2		Task time	Time Mark
1	HRV Time mark: Baseline HRV recording (* Write out petrol voucher receipt * Fill out page 4 of questionnaire)	5 mins	0
2	HRV time mark: end baseline. Explain worry task	minimal	
3	HRV Time mark: worry induction	5 mins	Time started: ……
4	HRV Time mark: tell them to stop. Move to a position where they can see DVD.	minimal	
5	HRV Time Mark: play recovery DVD	8 mins	Time started:……..
6	HRV time mark – end recovery. Get them to move over to water bath area.	minimal	
7	HRV time mark hand into control water condition (*Explain the cold pressor task)	2 mins	Time started:……
8	HRV time mark. End control condition. Ask them to move over to put hand in icy water. Stir icy water	minimal	
9	HRV time mark as they place hand in icy water	Until tolerance	Time started:……
10	HRV time mark when they remove hand. Dry off hand. Turn to face DVD.	minimal	Time feel pain ..... Time removed hand:……..
11	HRV time mark play recovery DVD	8 mins	Time started:…….
12	HRV Time Mark. Stop.		

* Fill out last pages of questionnaire + receipt

APPENDIX E

DVD Used During Experimental Phases (Baseline , Pain Recovery, and Worry Recovery)


Country Calendar
Director's Pick
G
Suitable for General Audiences.
Films, Videos, and Publications Classification Act 1993
TVNZ


Country Calendar
Director's Pick
I've been asked to select a dozen of my favourite episodes of Country Calendar and what a task it's been!
Selecting just twelve has been unbelievably difficult. I didn't want to be selfish and choose only my stories, and that made the job a little easier. Then I had to consider the geographical spread; the stories couldn't all be from one area of New Zealand.
And so the selection process continued until I made my final choices, and here they are. Are they the "best"? No! Are they memorable? Yes .... to me anyway.
Starting in the South Island high country to investigate the retirement of grazing land prone to erosion then off to the West Coast to capture wild deer.
We join a team rescuing sheep caught in drifts during a severe snow storm in Omaroma, Otago.
Vintage farm machinery enthusiasts in Canterbury re-enact an old fashioned wheat harvest, and to the sea on the research ship, the Tangaroa, to see the extent of over fishing of orange roughy.
We meet a courageous dairy farmer from the Waikato who overcame accidental blindness and took up bee keeping instead.
In the Central North Island, fifteen year old twins are managing a huge hill country farm.
A team of men from Ruatoria on the East Coast attempt to muster wild cattle from the bush. A different muster on Branches Station, a magnificent spot in Otago, but so remote. A real character in Northland who is breaking in his land and has plans to venture into tourism.
A couple in Hawkes Bay who knew nothing about growing grapes but now run a successful winery.
Finally, Dunston Downs Station, Otago, to join the mustering team which includes an artist who doubles as the cook.
Frank Torley
TVNZ
tvnz.co.nz/dvd
9 415394 706208
SPECIAL FEATURES: A catch up on each story
LANGUAGE
English
183 Mins Approx
16:9
DOLBY DIGITAL
MONO
1 x DVD9
COLOUR
PAL
TVNZ CAPTIONING
Captioning for the Hearing Impaired where available
DVD VIDEO
NZ On Air
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This disc is compatible with all DVD players displaying the correct symbol.
Note: Picture and sound quality is subject to quality of dated production source of some earlier episodes.
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