Immediate Effects of a Brief Mindfulness Body Scan Meditation on the Nociceptive and Autonomic Nervous Systems

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School of Rehabilitation & Occupational Studies
Faculty of Health & Environmental Studies
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Attestation of Authorship

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

Signed

[Signature]

Neil Bossenger
September 2015
Abstract

Aim
To measure the immediate effects of a brief mindfulness body scan meditation on self-reported pain, the nociceptive system and autonomic nervous system.

Study design
A between-subject, repeated measures, double-blinded, randomised controlled trial, with experimental and control interventions. A 10-minute intervention period was followed by a 15-minute rest period.

Participants
Thirty adults with chronic pain (7 men and 23 women) recruited through advertising in local papers, web-based social media and professional networks.

Interventions
The experimental group followed a 10-minute audio recording of a mindfulness based body scan meditation. The control group listened to a 10-minute audio recording of text from an audio book in a pleasant, friendly voice whilst sitting quietly.

Main measures
The primary dependent variable for self-reported pain was rating of pain severity on a visual analogue scale. The primary dependent variables for nociception were: pressure pain threshold recordings at a painful site and pressure pain threshold recordings at a non-painful site. The primary dependent variables for the autonomic nervous system were: mean heart rate, heart rate variability, heart rate variability low frequency to high frequency power ratio, and skin conductance.

Results
There were no statistically significant differences between the group that listened to the experimental mindfulness tape and the group that listened to the control tape on any of the outcome measures.

Conclusion
In people with chronic pain, a brief mindfulness body scan meditation has no effect on rating of pain severity on a visual analogue scale, pressure pain thresholds, mean heart rate, heart rate variability, heart rate variability low frequency to high frequency power ratio, or skin conductance when compared to a control group. Further research is required before determining whether brief mindfulness interventions are helpful in people experiencing chronic pain.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<td>ACC</td>
<td>Anterior cingulate cortex</td>
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<td>ANS</td>
<td>Autonomic nervous system</td>
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<tr>
<td>BPM</td>
<td>Beats per minute</td>
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<td>BVP</td>
<td>Blood volume pulse</td>
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<td>CRPS</td>
<td>Complex regional pain syndrome</td>
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<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalogram</td>
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<tr>
<td>EPSP</td>
<td>Excitatory postsynaptic potentials</td>
</tr>
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<td>fMRI</td>
<td>Functional magnetic resonance imaging</td>
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<tr>
<td>HF</td>
<td>High frequency</td>
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<td>HR</td>
<td>Heart rate</td>
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<td>HRV</td>
<td>Heart rate variability</td>
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<tr>
<td>IBI</td>
<td>Interbeat interval</td>
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<tr>
<td>LF</td>
<td>Low frequency</td>
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<tr>
<td>LF/HF</td>
<td>Ratio between low frequency and high frequency band powers</td>
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<tr>
<td>MBSR</td>
<td>Mindfulness-based stress reduction</td>
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<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
</tr>
<tr>
<td>NMDA</td>
<td>N-methyl-D-aspartate</td>
</tr>
<tr>
<td>NTS</td>
<td>Nucleus tractus solitarius</td>
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<tr>
<td>nuHF</td>
<td>Normalised units of high frequency</td>
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<td>PAG</td>
<td>Periaqueductal grey</td>
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<td>PBN</td>
<td>Parabrachial nucleus</td>
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<td>PCS</td>
<td>Pain catastrophising scale</td>
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<tr>
<td>PEP</td>
<td>Pre-ejection period</td>
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<tr>
<td>PNS</td>
<td>Parasympathetic nervous system</td>
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<tr>
<td>PPT</td>
<td>Pressure pain threshold</td>
</tr>
<tr>
<td>RMSSD</td>
<td>Square root of the mean squared differences between successive RR intervals</td>
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<td>RSA</td>
<td>Respiratory sinus arrhythmia</td>
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<td>SCR</td>
<td>Skin conductance response</td>
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<td>SFMPQII</td>
<td>Short-form McGill pain questionnaire</td>
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<td>SNS</td>
<td>Sympathetic nervous system</td>
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<td>STD HR</td>
<td>Standard deviation of instantaneous heart rate</td>
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<td>VAS</td>
<td>Visual analogue scale</td>
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<td>VLF</td>
<td>Very low frequency</td>
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<td>VLM</td>
<td>Ventrolateral medulla</td>
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Chapter 1: Objectives

1.1. Aim

The aim of this study was to measure the immediate effects of a brief mindfulness body scan meditation on self-reported pain, the nociceptive system and autonomic nervous system.

1.2. Hypotheses

A. A single session body scan meditation will reduce self-reported measures of pain.
B. A single session body scan meditation will reduce the sensitivity of the nociceptive system.
C. A single session body scan meditation will shift the autonomic nervous system to a less sympathetic dominant state.
Chapter 2: Literature review

2.1. Introduction

The purpose of this study was to measure the immediate effects of a brief mindfulness body scan meditation on self-reported pain, the nociceptive system and autonomic nervous system. Previous studies have shown that a body scan meditation can help to improve self-reported perceptions of pain [1] but no research has measured its direct influence on the nociceptive or autonomic nervous system. The following chapter will provide context for the role of mindfulness as a tool within pain management, nociception, and its relationship to the autonomic nervous system. The nociceptive system and autonomic nervous system (as it relates to pain) will be outlined in this chapter with rationale on how mindfulness might influence them together.

2.2. Pain and nociception

“Strictly speaking, pain is not in any organ, but in the mind, since only that can feel.”

Dorman Steele, Human Physiology, 1872.

2.2.1. Chronic pain in NZ

Chronic pain can be its own disease with a distinct pathology unto itself. An estimated one in six people suffer from chronic pain in NZ [2]. Two-thirds (67%) of those reporting chronic pain have lived with chronic pain for 5 or more years and a quarter (27%) have lived with chronic pain for 40% or more of their lives [3]. Despite conventional healthcare utilisation, nearly half of people with chronic pain report their pain as not under control [4]. Pain can be associated with changes in the nervous system that can worsen over time [5] and can give rise to significant psychological and cognitive deficits [6].

The impact of chronic pain on health related quality of life is dramatic. Those reporting chronic pain have much poorer health and the sequelae of chronic pain can be widespread across multiple aspects of biopsychosocial function [2, 7]. Living with chronic pain can be challenging and people frequently look for ways to better self-manage [2]. In NZ, nearly a third of those who reported chronic pain do not use any form of medical treatment (defined as medicines, pills, tablets, injections, or waiting for surgery) and it is suspected they have developed their own management approaches [2].

Whilst pain clinics and treatment services are available for people with chronic pain in NZ, these services can be costly and limited by the time of experienced personnel to deliver the services [7]. There are only a few multidisciplinary pain management
programs available in the country for the management of chronic pain and recent studies have highlighted the need for improvement in service delivery [2, 7].

2.2.2. Pain and the nociceptive system

Pain is a complex perceptual experience influenced by a wide range of psychosocial factors, including emotions, social and environmental context; the meaning of pain to the person; beliefs, attitudes, expectations; as well as biological factors [6]. The biopsychosocial view of pain provides an integrated model that incorporates mechanical, neurophysiological, psychological, as well as the social variables which may cause and perpetuate pain [8]. At its base, some form of damage to tissue generates nociceptive input to the brain. The person then interprets the signal such as determining whether the pain is sharp, dull, aching or burning. This cognitive interpretation (or appraisal) attaches meaning to the pain and influences subsequent behaviour [6].

A distinction between pain and the neural mechanisms of nociception is important. Physiological pain starts in the peripheral terminals and is initiated by specialised sensory receptors, called nociceptors, innervating peripheral tissues, activated only by noxious mechanical, thermal or chemical stimuli. Nociceptors can be found in skin, mucosa, membranes, deep fascia, connective tissue of visceral organs, ligaments and articular capsules, periosteum, muscles tendons and arterial vessels [9]. These receptors are first-order afferent neurons making up the peripheral aspect of the nociceptive system.

Activation of nociceptors by mild noxious stimuli generates fast excitatory postsynaptic potentials (EPSP) in second-order neurons that signal the onset, duration, intensity and location of the stimulus [10]. If the EPSP sends the second-order neuron in the spinal cord over its threshold, the signal is transmitted to the cortex and the sensation of pain will be elicited. Activation of nociceptive pathways is subject to activity-dependent plasticity, which manifests as a progressive increase in the response of the system to repeated stimuli [10]. This comprises the central component of pain sensitisation. Second-order neurons are distributed along the dorsal horn of the spinal cord, organised according to the Rexed laminae [9]. The specific nociceptive neurons responding exclusively to noxious stimuli are found in laminae I, II, V and VI [9]. The sources of input for these neurons are high threshold Aδ nociceptive fibres, and heat and C-polymodal nociceptive fibres [9]. The spinal cord also contains excitatory and inhibitory interneurons, mostly located in laminae I to III, which synapse locally [11].

Pain is experienced at a physical level and an affective level. A model put forward by Melzack and Casey in 1968 embodies pain as a sensory-discriminative dimension, relating to spatial and temporal properties, a motivational-affective dimension,
incorporating tension, fear and autonomic events, and a cognitive-evaluative dimension of pain as a whole [12].

The anatomical pathways and cortical regions involved in nociception can also be divided into distinct systems. The lateral system participates directly in the sensory-discriminative attribution of nociception and involves specific thalamic nuclei which project to the somatosensory cortex [9]. This system can differentiate between the locality and intensity of the incoming stimulus.

The medial system involves the frontal cortex, anterior cingulate cortex (ACC), insula and hypothalamus and evaluates the motivational-affective and cognitive-evaluative dimensions of pain perception [13]. In contrast to the lateral system, the medial nociceptive system has less defined projections from the thalamus to the somatosensory cortex, including limbic structures such as the insula and ACC [14]. The ACC and the anterior insulae function as integrative structures during the experience and anticipation of pain [15]. It is for this reason the medial system mainly contributes to the motivation-affective component of pain [9]. The ACC plays a deterministic role in pain modulation and analgesia. This analgesic effect is mediated through interaction with other structures such as the orbitofrontal cortex, the amygdala and the periaqueductal grey (an area around the cerebral aqueduct) [16]. The ACC has been marked as an area of the brain significant to the habituation and attenuation of pain in a descending manner, triggering downstream opioid-dependent mechanisms, and dysfunctional habituation to pain may represent a risk factor for the development of chronic pain states [17].

The brainstem is another anatomical site linked to pain modulation [5]. Many dorsal horn projection neurons, such as those in lamina I, have axons that cross the midline of the spinal cord and project rostrally in the contralateral white matter to terminate in various brainstem nuclei [11]. These pathways are thought to underlie pain and temperature perception [11]. Areas of pain regulation have been identified in the midbrain, pons, and the medulla, especially around the periaqueductal grey [18]. These areas of the brain are rich in endogenous opioids and opioid receptors and they also give rise to fibre tracts that project to the dorsal horn of the spinal cord, where serotonin, norepinephrine and acetylcholine are released [13]. The action of these tracts is to inhibit and facilitate nociceptive input from afferents and/or output by nociceptive second-order neurons. Activation of these tracts results in inhibition of dorsal horn nociceptive structures, which are mediated by the activation of opioid-releasing interneurons [17].

2.2.3. Mechanisms of chronic pain

Chronic (or persistent) pain is defined as pain that persists for 3 months or more beyond the expected period of healing [2, 19]. It is regarded as having no
physiological purpose or adaptive value, in comparison to acute pain which has protective functions [20]. Nociceptive pain is initiated by tissue damage, whereas chronic pain frequently arises through changes to the central processing mechanisms of nociception [21].

Persistent pain has characteristics that include increased amplitude of response to a given stimulus (hyperalgesia), pain elicited by normally innocuous stimuli (allodynia) and spontaneous pain in the absence of external stimuli [22]. Several changes to the nervous system underlie these phenomena, including alterations in nociceptor threshold for activation, plasticity in synaptic connections in the spinal cord dorsal horn, or changes in activation of descending inhibitory and facilitatory pathways [11]. Chronic pain also frequently presents with symptoms of paraesthesia, sensory deficits, autonomic disturbances, and motor disturbances [23], reflecting interaction of the nociceptive system with other central and autonomic components.

Neuroplastic changes can occur on multiple levels along nociceptive pathways, from peripheral nociceptors to the cortex [24]. Neuroplasticity takes on two forms: modulation and modification [10]. Modulation means reversible changes in the excitability of primary sensory and central neurons. Modification means longer lasting alterations in the expression of neurotransmitters, receptors and ion channels; or alterations in the structure, connectivity and long term potentiation of neurons, such that the system is grossly modified, distorting its normal stimulus-response characteristics.

Long term plastic changes are caused by repetitive afferent or incoming stimulation and can lead to hyperalgesia (exaggerated neuronal responses) for long periods after peripheral drive has subsided. Nociceptors exhibit peripheral sensitisation when there is a reduction in the threshold for activation, an increase in the response to a given stimulus, or the appearance of spontaneous activity [25]. After peripheral injury or sensitisation, peripheral nerves display ectopic discharge which may lead to an increased barrage of nociceptive signalling to the dorsal horn of the spinal cord with or without peripheral stimulus [26]. Increased nociceptive transmission is, in part, due to plasticity of the sodium channel populations in peripheral nerves after injury. They lower the threshold for activation to the spinal cord and this adds to the amplification of peripheral events. Nerve growth factor has also been shown to have a key role in the process of peripheral sensitisation [27]. Demyelination and abnormal trafficking of sodium channels occurs along the membrane of injured nerves as well as their uninjured neighbours. Ongoing peripheral sensitisation and activation causing persistent drive into the spinal cord can induce central hyperexcitability.

Central sensitisation is an amplification of neural signalling within the central nervous system that elicits pain hypersensitivity [28]. N-methyl-D-aspartate (NMDA) receptors
found in the brain and spinal cord have lowered thresholds for activation following repetitive noxious input due to removal of the magnesium blockade of the receptor channels [10]. Under conditions where the stimulus is maintained, NMDA receptors have been implicated in the spinal events where responses of the dorsal horn neurons are significantly increased after repetitive peripheral stimulation despite the input remaining constant [22]. Wind-up is one of the mechanisms responsible for the amplification of neuronal discharge in the spinal cord; hence the brain will receive greater and longer duration input for a given stimulus.

As the brain repetitively receives greater input of stimulus for longer duration, functional reorganisation of the cortex can occur [5]. For example, for people suffering with complex regional pain syndrome (CRPS), a shrinkage of the cortical representational field of the affected limb was found and the extent of the shrinkage correlated highly with the intensity of pain and the magnitude of mechanical hyperalgesia [29]. Irrespective of the location, nature or course of different pain syndromes, the most common finding is a decrease of grey matter in the cingulate cortex, the orbitofrontal cortex, the insula and the dorsal pons [5].

Pain may also be altered because of dysfunctional brain changes which impair descending inhibition or facilitation. Intact descending inhibition and facilitation is a key component of modulation of the barrage of sensory input from the periphery that ascends to the brain [30]. In a study by Apkarian et al [31], 17 people with chronic back pain demonstrated brain atrophy and that the pathophysiology of chronic pain includes thalamo-cortical changes in nociceptive processing.

Figure 1. Process of pain chronification

In summary, pain chronification can be due to numerous modulatory mechanisms, such as effects at the nociceptor level with peripheral sensitisation, the wind-up phenomenon, central sensitisation, and changes in descending central modulation [10] (Fig. 1). The decrease of grey matter in brain regions which are highly associated with pain modulation could potentially lead to dysfunction in effective pain modulation [5]. Abnormal modulation of brain nociceptive systems, at first transient, but possibly becoming permanent, could in part explain the shift from acute to chronic pain. The
transition from acute to chronic pain is also known to be influenced by psychological processes [32].

2.2.4. Psychological factors

The biopsychosocial model has been instrumental in the development of treatment approaches for chronic pain [33]. There is no one distinct model to encompass all aspects of pain and dysfunction, however the biopsychosocial view takes into account biomechanical, biochemical and psychological factors that contribute to the experience of pain (Fig. 2). The extent of the involvement of each factor may differ from person to person, depending on variables such as age, chronicity and personality [34].

Psychological models propose that the treatment for chronic pain also needs to address the cognitive aspects of pain. This is based on the principle that a prerequisite for pain perception is that attention is drawn toward a noxious stimulus, but once it has been attended to, cognitive processes have to interpret what that stimulus means. This cognitive process is highly intertwined with emotional processes and can influence how pain is experienced and described by a person [35]. For example, attention to pain can become linked to fear and anxiety, making the person hyper-vigilant and acutely aware of pain signals [32]. Therefore, attempting to suppress or attend to thoughts about pain actually increases the pain experience [36].

“Cognitive errors” are beliefs about oneself and situations that are distorted in a way which over-emphasises negative consequences. An example of such a cognitive error is catastrophising, which can be defined as an exaggerated, negative orientation toward pain where a relatively neutral event is irrationally made into a catastrophe [37]. In essence, the person imagines the worst possible outcome and accepts the worst outcome as a given result. Catastrophising has been reported to be associated with elevated pain reporting in clinical settings and poor outcomes in cognitive-behaviourally oriented programs [33].

Findings have shown that higher levels of catastrophising are uniquely related to greater pain intensity in chronic pain patients [38]. While the precise mechanism underlying the relationship between cognitive modulation and pain intensity is still unclear, catastrophic thinking about pain is significantly associated with increased activity in brain regions related to anticipation of pain, attention to pain, emotional

Figure 2. The biopsychosocial model

Biomechanical e.g. joint, disc, soft tissue

Biochemical e.g. neurochemistry

Pain experience

Psychological e.g. anxiety, social aspects
aspects of pain and motor control [39]. Conversely, neuroimaging studies have shown that distraction techniques can reduce both sensory and affective components of pain due to reductions in activity of the insula, ACC, thalamus, somatosensory cortex as well as increases in pain modulation by the prefrontal cortex and brainstem [40]. Additionally, cognitive therapies have been shown to have small to moderate effects on pain and catastrophising immediately post-treatment [41].

Heightened focus on possible signals of pain might explain why seemingly small injuries result in intense pain. People with a high fear of pain show greater attentional bias toward pain-related information compared with those classified as having low fear of pain [42]. Therefore, attentional factors are important clinically because there are techniques that address such factors and could therefore reduce the perceived experience of pain. Distraction techniques, for example, can teach patients to shift their attention to stimuli other than pain, whereas interceptive exposure shifts attention toward the pain so that they become habituated to the pain signal [43].

People with chronic pain often develop negative expectations about their ability to exert control over their pain because expectations of resolution are not fulfilled [32]. The pain is continually competing for attentional resources and while sometimes long term sufferers develop strategies to manage their pain, people new to chronic pain are more susceptible to attentional disruption and higher levels of perceived pain [42]. They frequently terminate efforts to develop new strategies to manage pain and instead turn to passive coping strategies such as inactivity or self-medication to reduce emotional distress [19].

While chronic pain may be a somatic representation of psychological distress, or may represent a heightened sensitivity to bodily sensations, people with chronic pain usually demonstrate poor coping mechanisms and struggle to adapt and manage their pain [44]. In a study that compared the psychological characteristics of people with discrete pain versus chronic idiopathic pain, the latter reported higher levels of anxiety, fear of pain, helplessness and magnification of the problem [44].

Psychological factors have been reported to be predictive of long-term disability for many pain syndromes as well as for pain severity, emotional distress and treatment seeking [45]. The findings of these studies provide support for the biopsychosocial model and suggest that addressing how people perceive physical sensations can influence the experience of pain. This was demonstrated in a NZ study investigating mindfulness and chronic physical illnesses. Participants’ experience of pain and discomfort lessened as a result of mindfulness training and they showed significant positive changes around rumination, catastrophising and helplessness, hence being better able to manage their pain [46].
Psychological therapies are commonly offered after orthodox treatments have failed and the treatment goal shifts from one of removing the pain to managing the pain [41]. Cognitive methods such as mindfulness-based stress reduction are aimed at assessing the thoughts of pain and changing associations with them. It is usually a component of cognitive behavioural therapy delivered by experienced staff trained in these protocols. Mindfulness-based stress reduction can cost up to $600 per course and take up to 2 hours a week for 8 weeks to learn [47]. For some people this might not be achievable, precluding access to the intervention. Brief adaptations of the intervention could increase affordability and accessibility and assist people to improve their ability to control responses to stimuli, or come to accept them.

2.3. Autonomic nervous system

2.3.1. Pain-autonomic interactions

The autonomic nervous system (ANS) is structurally and functionally positioned to be an interface between the internal and external environment, co-ordinating bodily functions to ensure homeostasis and adaptive responses to stress [48]. Recent research points toward an association between dysfunction of the ANS and chronic pain [49], suggesting that dysregulation of the ANS (increased sympathetic and/or decreased parasympathetic tone) has a critical role in initiating and perpetuating central sensitisation [50].

The ANS and nociceptive systems work in tandem as part of a central network to deal with environmental challenges, interacting at multiple levels, including the periphery, dorsal horn, brainstem and forebrain [51]. These areas receive convergent nociceptive and visceral input, containing groups of neurons that initiate autonomic and behavioural responses to various stimuli, and inhibit or facilitate responses to pain [51].

Primary visceroceptive afferents terminate mainly in lamina I and V [48] of the spinal cord and project to the cerebral cortex via the spinothalamic tract, the major pathway for transmission of nociceptive information [51]. Lamina I neurons arise from progenitors of autonomic interneurons, and ascending projections of lamina I synapse strongly to sympathetic cell columns of the thoracolumbar spinal cord, thus forming loops for somato-autonomic and viscera-autonomic reflexes [52].

Lamina I neurons also project to brainstem areas involved in autonomic responses [53]. Lamina I neurons extend into the spinal trigeminal nucleus caudalis and then projects to the nucleus tractus solitarius (NTS) in the medulla and parabrachial nucleus (PBN) in the pons, which is the main integration site for all homeostatic afferent activity [54]. From here, dense projections go to the periaqueductal grey (PAG) in the midbrain, and then on to the hypothalamus and central nucleus of the
Afferent information then reaches the thalamus, ACC (considered the limbic motor cortex) and the insula (considered the limbic sensory cortex) [54]. A key feature of the spinal and trigeminal nociceptive pathways is that they provide collaterals which converge at every level of brainstem visceral pathways [55].

Nociceptive inputs may trigger autonomic responses via the NTS, PBN, amygdala, hypothalamus and ventrolateral medulla (VLM) because nociceptive afferents activate neurons in laminae I and V of the thoracic and upper lumbar spinal cord, which project monosynaptically to preganglionic sympathetic neurons at the same spinal level [55]. This provides a basis for segmental sympathetic reflexes. Cell groups in the VLM and pons project to the hypothalamus, intermediomedial cell column and dorsal horn, and modulate autonomic responses; the PBN projects to the amygdala and thalamus which activates emotional and arousal responses.

Two important locations for interaction between the nociceptive system and the ANS are the PAG and hypothalamus. The dorsolateral, lateral and ventrolateral columns of the PAG have reciprocal connections with autonomic centres of the lower brainstem and hypothalamus that regulate activity of peripheral autonomic pathways [56]. Stimulation of the PAG has been known to provide long term effective pain relief in certain populations [57] and the hypothalamus has a central role in the integration of autonomic responses and pain modulation, necessary for homoeostasis and adaptation to internal and external stimuli [48]. The hypothalamus can initiate nociceptive modulation and participate in autonomic control when activated by nociceptive inputs, having neurons that project to multiple areas including the PAG, PBN, NTS, VLM, raphe nuclei, as well as the dorsal horn and preganglionic nuclei [55], all of which are involved in nociceptive modulation.

The balance between inhibition and facilitation of nociception is dynamic and can be altered in and by different behavioural, emotional and pathological states [58]. The lateral and dorsolateral columns of the PAG receive well localised, superficial nociceptive inputs from the spinal and trigeminal dorsal horns and initiate sympathoexcitatory (fight/flight) responses mediated by neurons of the VLM, which activate sympathetic preganglionic neurons controlling cardiovascular effects, such as tachycardia, hypertension and redistribution of blood flow [59]. In contrast, neurons of the ventrolateral PAG column, receiving poorly localised somatic, visceral and muscular inputs, initiate sympathoinhibitory responses like hypotension and bradycardia by projecting to the region of the medullary raphe containing cardiac vagal premotor neurons [48]. The PAG, via neurons in the VLM, including serotonergic raphe magnus neurons, exerts a dual modulatory control on nociceptive relay at the level of the spinal and trigeminal dorsal horns [55].
Numerous brain areas including the ACC, insula, prefrontal cortex, somatosensory cortices, thalamus, basal ganglia, amygdala and brainstem structures such as the PAG, exert influence on autonomic control during nociceptive processing [60]. These areas may not be activated simultaneously in all painful conditions, but will be more or less activated depending on the biomechanical, biochemical and psychological factors influencing the subjective experience of pain. Their activation will also be dependent upon the intensity, duration and location of nociceptive input. Cerebral arousal, alertness, attention and sensory processing of environmental stimuli are promoted by the ANS [60] and it plays a crucial role in pain modulation.

2.3.2. Cortical-autonomic modulation

In both lateral and medial pain system activation, the ANS plays a major role in developing the most appropriate immediate physiological reaction and long term adaptation [30]. Central control of sympathetic and parasympathetic output involves several interconnected levels of the neuraxis, including spinal, bulbopontine, pontomesencephalic and forebrain [61].

The forebrain level includes the hypothalamus and components of the anterior limbic circuit such as the insula, ACC and amygdala, which are involved in the integration of bodily sensation and emotion-related autonomic responses [48]. The pontomesencephalic level integrates autonomic control with pain modulation and behavioural responses to stress, hence the PAG is a critical component of the emotional motor system [62]. This brainstem network receives input from the medial prefrontal cortex and ACC, and projects to other brainstem areas that control behaviour-specific patterns of motor and autonomic responses, which modulates dorsal horn excitability to nociceptive input and the gain of spinal reflexes [62].

The prefrontal cortex provides the major forebrain input to the PAG. The medial wall of the prefrontal cortex projects to the dorsolateral column; the anterior cingulate gyrus to the lateral, ventrolateral and dorsomedial columns; and the posterior orbitofrontal and anterior insular cortices project to the ventrolateral column of the PAG [63]. The prefrontal cortex is concerned with maintaining expectations and modulating the anticipation of pain. It has been shown that activity in the PAG region is enhanced during the anticipation of pain [60]. This activity correlates significantly with activity in the dorsolateral prefrontal cortex, which triggers opioid release within the brainstem so that the descending pain modulating system inhibits transmission of nociceptive signals from the dorsal horn [60].

Attention can influence brainstem activity and hence nociceptive processing [60]. In a study by Tracey et al [64], high-resolution functional magnetic resonance imaging (fMRI) was used to investigate brain activation within the PAG to painful stimuli in healthy people. The participants were asked to either focus on or distract themselves
from the painful stimuli. During distraction, pain intensity ratings were reduced while PAG activation was significantly increased. The study [64] suggested that the PAG is a site for higher cortical control of pain modulation in humans and will also influence the ANS with its autonomic connections to the lower brainstem.

The insula and ACC are both engaged in pain processing and high-level control of autonomic function [51]. The lamina I spinothalamocortical pathway projects to the viscerosensory insular cortex [51, 60] and is part of the lateral pain system involved in discriminative aspects of pain sensation. The insular cortex projects to many components of the central autonomic network including the amygdala, hypothalamus, PBN and NTS [65]. The right anterior insula is involved in sympathetic arousal associated with mental tasks [66] as well as changes in arterial pressure, heart rate, respiration, gastrointestinal motility, salivation, pupil dilation and piloerection [51]. This brain region receives numerous sensory inputs including touch and nociception [67]. A similar somatotopic organisation of pain processing has been shown in the basal ganglia, which is involved in cognitive, affective, motor and autonomic states [68] and therefore also serves the function of coupling pain with the most appropriate autonomic response.

The ACC receives input from the medial pain system and is also directly involved in control of autonomic functions, such as arousal during volitional behaviour and effortful cognitive processing [60]. ACC pyramidal neurons project directly to subcortical brain regions associated with autonomic control, including the hypothalamus, PAG and pons [69]. The genual aspect of the ACC receives input from motivational regions, such as the prefrontal cortex and amygdala, and is a main source of input for the PBN, NTS, nucleus ambiguus, VLM and interomedialateral cell column [65, 69]. These areas elicit a variety of visceromotor responses when stimulated, including changes in blood pressure, heart rate and respiration; facial flushing, salivation, nausea, vomiting and bowel and bladder evacuation [51]. Although different tasks engage areas of the ACC, fMRI findings indicate that a common dorsal cingulate region is involved in autonomic control during cognitive processing [69].

2.4. Mindfulness

2.4.1. Mindfulness based stress reduction

Mindfulness has been broadly conceptualised as a state in which one is highly aware and focused on the reality of the present moment, accepting and acknowledging it, without getting caught up in thoughts that might catastrophise an experience or an emotional reaction to a situation [70].
Mindfulness can be defined as a state of consciousness in which the person maintains a single pointed awareness focused on mental, interoceptive and exteroceptive experiences, distinct from outcome-based self-management strategies that aim for a definitive endpoint [71]. Mindfulness is a process devoid of striving or attachment to any goal [72].

The construct of mindful awareness or mindfulness meditation originated in early Buddhist documents but is neither religious nor esoteric in nature [73]. Western researchers and clinicians who have introduced mindfulness practice into health treatment programmes usually teach these skills independently of the religious or cultural origins because of Western society's unfamiliarity with Buddhist traditions or vocabulary [74]. Nevertheless, mindfulness meditation could be considered a component of ancient practices such as Vipassana meditation and Zen meditation [75]. These meditations belong to the pole of mindfulness at one end, opposite to concentrative meditations such as Transcendental Meditation [76]. Concentration-based approaches train people to restrict their focus of attention to a single stimulus such as a word, sound, object or sensation and when attention wanders, it is redirected to the object of meditation [77]. Mindfulness meditation, in contrast, only involves observation of changing stimuli as they arise, often directed toward the inner experiences of the person, like thoughts and emotions, and emphasizes a less goal-oriented, non-judgmental observation [77].

Mindfulness-based stress reduction (MBSR) programmes were first developed in 1979 by Jon Kabat-Zinn at the University of Massachusetts Medical Centre and are now the most frequently cited method of mindfulness training [77]. Typically such programmes entail an 8-week intensive intervention and there have been a number of controlled and uncontrolled studies performed assessing specific and nonspecific effects of mindfulness interventions [70, 78]. Originally the programmes were used alongside medical treatment for the management of chronic pain and stress-related disorders but have since been found to potentially benefit those with cancer, anxiety, depression, fibromyalgia and eating disorders amongst other conditions [79].

MBSR courses were first introduced in NZ in 2005 by Jim Carmody, a psychologist also from the University of Massachusetts Medical Centre, who trained health professionals to offer MBSR [46]. The courses are both effort and time intensive with eight 2½ hour night classes over 8 weeks, plus an additional full day retreat on a Sunday between weeks 6 and 8. In between sessions, trainees are also asked to complete a workbook and practice their learnings for up to 60 minutes a day, 6 days a week [46].

MBSR comprises mainly of three techniques. Firstly, the body scan meditation is a 45-minute technique that involves a gradual sweeping of attention through the entire
body from feet to head, focusing non-critically on any sensation and using periodic suggestions of breath awareness and relaxation [1, 77]. The process includes noticing, but not reacting to pain, emotions, urges, thoughts and other feelings in the body [80]. When the person notices their mind start to wander into thoughts, memories or fantasies, the content of these feelings is briefly noted and then attention is returned to breath or the present moment.

The second MBSR technique is sitting meditation. Participants are instructed to sit in a relaxed and wakeful posture with eyes closed, directing attention to the sensations of breathing [77]. Finally, the third technique is Hatha yoga practice, which includes breathing exercises, simple stretches, and posture designed to strengthen and relax the musculoskeletal system [1]. An important consequence of mindfulness practice with all three techniques is the realisation that most sensations, thoughts and emotions fluctuate, or are transient, passing by like waves in the sea [77].

Systematic reviews involving chronic conditions have shown that while there is preliminary evidence supporting the notion that MBSR may improve health related quality of life and coping mechanisms for people with chronic conditions, evidence is limited when it comes to specific reduction in pain [1, 70, 79, 81]. While there is evidence that MBSR can improve pain acceptance [81], there are few controlled studies supporting the reduction in specific pain symptomatology.

Jon Kabat-Zinn, who first introduced the use of mindfulness for people with chronic pain, hypothesised that training in mindfulness would attenuate pain by altering emotional responses to pain and enhancing acceptance-related coping strategies [82, 83]. A clinical trial of 90 people with chronic pain found that mindfulness meditation training in the context of a 10-week MBSR program can be effective in reducing self-reports of pain and pain-related behaviours [82]. In a subsequent study of 51 people with chronic pain, who were unsuccessfully treated by conventional methods, Kabat-Zinn reported significant decreases in pain and reductions in mood disturbances after a 10-week MBSR program [74]. Kabat-Zinn then did a 4-year follow up study of 225 people with chronic pain, who were enrolled in an 8-week MBSR programme, which showed maintenance of pain improvement among 60-72% of the cohort; however, this study did not include a comparison group [84].

In a New Zealand study investigating the health benefits of mindfulness-based stress reduction for people living with chronic physical illnesses, participants reported improvement in physical and social functioning, as well as improvements in mental health, energy, vitality and overall general health as a result of mindfulness training [46]. The majority of participants in the study had conditions which gave rise to pain such as fibromyalgia, arthritis, migraines and headaches [46], but no studies specific to mindfulness and chronic pain have been conducted in this country. Additionally,
whilst the results of current trials of mindfulness in people with chronic pain have been encouraging, the studies have been limited by small sample sizes and heterogeneous samples of people, including populations with different ages and sites or types of pain [1].

As described previously, MBSR programmes range from 20 to 26 hours of session time and were designed by Kabat-Zinn to be long enough that people could grasp the principles of self-regulation through mindfulness and develop the skills to practice meditation on their own [74]. However, for some people, the circumstances of their condition or demands on time may mean that MBSR in its standard form will exclude them from the possibility of participating fully in a programme [85, 86]. Recent studies have investigated the effects of a brief mindfulness meditation of 15-minutes or less to prevent time being a barrier to participation in mindfulness and found no evidence that shortened versions of the programme are less effective [85]. In studies related to mindfulness and psychological outcomes, Carmody and Baer [85] examined whether shorter programme times could produce similar results to the 8 to 10-week programme. Comparisons suggested that reductions in the number of MBSR in-class hours may not necessarily lead to compromised outcomes. Also, it is possible for short term results to be maintained in the long term [1]. In a study by Grossman et al. [87], the short term benefits observed in a group of people with fibromyalgia who underwent a brief mindfulness intervention were maintained at a 3-year follow-up. In other studies, short term changes were seen in pain [88], the cardiovascular system [89] and emotional regulation [90].

As the necessity of a full length MBSR programme is questioned [85], attention is now focusing on the effects of a shorter mindfulness intervention for people with chronic pain [91-94]. There is preliminary evidence that mindfulness may assist people with psychosocial adaptation to pain, which might be longer lasting than the impact on pain symptoms [70]; however, some studies are limited by the use of uncontrolled study designs [70]. Kingston et al [91] addressed this limitation by introducing an active control in a randomized, single-blind trial. They used a 6-session, 3-week meditation intervention and found increases in pain tolerance to a cold-pressor task. Control participants were trained in guided visual imagery – a self-relaxation strategy that directs attention away from the present moment. Divided attention provides a good control for mindfulness and results showed an increase in pain tolerance for mindfulness participants. The authors questioned though whether the length of the study over a 3-week time frame may have influenced results [91].

2.4.2. Brief mindfulness and chronic pain

Some researchers refer to brief mindfulness techniques as focused breathing [90] or acceptance-based intervention [93], rather than mindfulness. This is because some participants have had no previous training in all mindfulness techniques [90].
However, to be consistent with terminology used in the literature, and as the teachings are based on the underlying principles of mindfulness, it will be referred to here as brief mindfulness.

A literature search was undertaken to determine the effects of short-duration mindfulness interventions on pain and related outcomes using Medline, ScienceDirect, the Cochrane database, and references of retrieved articles for studies on brief mindfulness interventions of less than 3 days in people with chronic pain. Search terms included mindfulness, MBSR, mindfulness-based intervention, brief, short-term, and chronic pain. Inclusion criteria of the literature search comprised randomised control trials using a mindfulness meditation intervention specifically, as opposed other forms of meditation. Outcome measures included pain thresholds, pain tolerance, self-reported pain ratings, pain unpleasantness ratings, mindfulness awareness ratings, and distress scales. The search identified limited evidence specific to brief mindfulness interventions of 3 days or less and application for people with chronic pain [88]. Four controlled studies that met the inclusion criteria were identified and reviewed [88, 92-94]. One study was of 3 days duration while three were 10 to 20-minutes in length for the intervention. Two studies used a therapist trained in mindfulness and two studies used a therapist-free intervention delivery. A summary of comparison is provided in Table 1. Studies that did not relate specifically to pain or chronic pain, or looked at items such as psychological distress or quality of life, were excluded.

Zeidan et al [94] studied the effects of a 3-day mindfulness intervention on experimentally induced pain. This study examined the analgesic effect of meditation on pain ratings of electrical stimulation before and after intervention. A threshold procedure was used to determine stimulus intensities associated with cutaneous threshold, low pain threshold and high pain threshold, to make sure the same degree of sensory experience occurred for each person. Cutaneous threshold was defined as the intensity at which the person first detected a sensation, and subsequent pain thresholds were determined by progressively increasing electrical current by 5 to 10mA. Math distraction and relaxation intervention were used as active controls in a within-group comparison. Results showed that 3 days of mindfulness meditation was effective in reducing pain ratings to experimentally induced pain. Participants reported less pain to both low and high pain intensities when meditating compared with cutaneous threshold before meditation. The authors also reported reductions in state anxiety after each meditation session, which additionally has the ability to attenuate feelings of pain. Each training session was held in a group setting of up to 8 people and lasted approximately 20-minutes while an instructor taught different meditation skills. On the second day of the intervention, participants mediated to a standardised audio emphasising how moment to moment awareness can alter the experience of internal and external events. The mindfulness instruction was delivered
by a facilitator with more than 10 years of experience in mindfulness meditation techniques over 3 days. Whilst experienced therapists facilitate the quality of the intervention provided, this can lead to problems when trying to implement the intervention into everyday practice. For example, there are limited therapists available with the expertise to provide the intervention which can make it difficult for people access, together with accompanying costs and travel which may not be funded.

Two studies measuring the effects of mindfulness on pain reduced the time frame to a single session, in a therapist-free form, delivering the intervention instructions through pre-recorded voices [88, 92]. The studies were published a month apart in 2012 and were the first of their kind to test a therapist-free, short term intervention. The potential benefit of a therapist-free intervention is twofold: 1. to reduce the time a participant would have to spend in an MBSR course, and 2. to provide opportunity for people to learn mindfulness without the need for experienced trainers.

In the first study, Liu et al [92] explored the effects of a brief mindfulness intervention in healthy college students on pain tolerance and distress of pain, measured by their response to experimentally induced cold-pressor pain. This cold-pressor technique can be used to screen participants who are not distressed by pain by excluding those who are able to keep their hand in ice water for more than 5 minutes [95, 96]. The reason for this is to increase the power of the experimental intervention for those who are more distressed by pain [93]. Twenty people were excluded from Liu’s [92] study, leaving a final sample of 60 females with a mean age of 20 years.

The study was a double blind, randomised controlled trial involving healthy undergraduate students. The sample comprised female students only who were not currently experiencing pain from a medical condition. They compared three types of coping strategies on experimentally induced pain: mindfulness, distraction, and spontaneous coping. Each intervention was given using pre-recorded audios and lasted for 15 minutes. Results showed that compared with using spontaneous strategies, the mindfulness intervention significantly improved pain tolerance and reduced immersion distress. The distraction strategy also significantly improved pain tolerance; however, it did not have a significant effect on the level of distress during the immersion period.
<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Intervention</th>
<th>Outcome Measures</th>
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<tbody>
<tr>
<td>Li et al.</td>
<td>2012</td>
<td>Double-blind, randomized controlled trial</td>
<td>Median depression (throughout), Distress Tolerance (throughout), Cold pressor test, Coping</td>
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<tr>
<td>Zadlan et al.</td>
<td>2010</td>
<td>Double-blind, randomized controlled trial</td>
<td>Median depression (throughout), Distress Tolerance (throughout), Cold pressor test, Coping</td>
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<tr>
<td>Weeser et al.</td>
<td>2007</td>
<td>Single-blind, randomized controlled trial</td>
<td>Median depression (throughout), Distress Tolerance (throughout), Cold pressor test, Coping</td>
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Table 1. Summary of study comparison

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<tr>
<th>Study Design</th>
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<td>Single 15-minute session, 55 people with bipolar disorder</td>
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In the second therapist-free study by Ussher et al [88], which was also a randomised controlled trial, the sample group consisted of patients attending an outpatient pain clinic in south west London who were diagnosed with a chronic pain condition. Participants were randomly allocated to intervention and control groups. The intervention was delivered using a pre-recorded audio therefore removing any effects from a therapist. Fifty-five from 86 people were recruited to the study and over 75% of them were females with low back pain. Results showed a significant reduction in ratings for pain related distress and for pain interfering with social relations for the intervention group compared with the control group. The findings support the results of the study by Liu et al [92] and suggest that chronic pain can alter the way nociception is processed [10].

The final study by Masedo et al [93] compared acceptance versus suppression of pain in a brief mindfulness study. A much larger sample was used than in Liu et al’s [92] study, comprising 219 participants with a more or less equal distribution of males (48%) to females (52%). Acceptance is the same idea as mindfulness whereby a person is actively contacting physical and psychological experiences, behaving effectively and not avoiding feelings [97]. Acceptance means to change the target for control from uncontrollable events (pain) to controllable factors, such as behaviour during the experience of pain, to lead to better daily function [93]. Suppression implies thought-stopping, which paradoxically can lead to an increase in intrusive thoughts and the experience of pain [98]. Suppression is different to distraction and therefore is a good differential in mindfulness comparisons. Thought-stopping involves recognising an inappropriate thought, emotion or sensation, silently yelling stop, then breathing deeply and exhaling slowly [93].

Masedo et al [93] used a single session, randomised controlled trial design. They found that the cold-pressor task was a less distressing experience for the acceptance group than the suppression group and improved pain tolerance time. This finding is congruent with Liu et al’s [92] results and two other studies looking at the effects of brief mindfulness on pain [88, 94]. However, Liu et al’s study [92] failed to demonstrate a main effect on pain ratings for each intervention, which is in contrast to Masedo et al’s [93] finding that the acceptance strategy produced lower subjective pain and distress ratings than distraction or spontaneous coping. A possibility for this is due to direct therapist interaction during each intervention. Masedo et al [93] compared acceptance, spontaneous coping and suppression strategies in a single session to control pain, and even though the therapists were blinded to the hypothesis, each intervention was scripted word by word to each participant for approximately 20 minutes [93] and this can help participants to learn the strategies more effectively and make adjustments accordingly [92].
There are three fundamental differences among the final three studies. Firstly, the intervention used by Ussher et al [88] was specifically a body scan meditation, as opposed to a general mindful acceptance of feelings or sensations. A body scan is different to other common mindfulness techniques such as sitting or walking meditations [88]. A person’s attention is guided to specific body parts in turn and instructed to acknowledge sensations in each area without attempting to change them. Body scans are often used as an accessible entry point to mindfulness meditation [99]. Secondly, the mindfulness intervention was delivered twice over a 24-hour period in the Ussher et al [88] study: once in a clinic setting and once in the participant’s normal home environment. Results showed that in the clinic setting, for the body scan group compared with the control group, there was a significant reduction in ratings for pain related distress and for pain interfering with social relations; however, in the participants’ home environment, none of the scored changes were significantly different between the groups [88]. Thirdly, Ussher et al [88] made use of subjective measures by creating a series of 4 questions evaluating pain ratings and 6 questions examining underlying processes of mindfulness. These 10 items were drawn from the Brief Pain Inventory, the Chronic Pain Acceptance Questionnaire, and the Mindful Attention Awareness Scale. Participants completed ratings for these 10 items immediately before and after each intervention. Masedo et al [93] and Liu et al [92] utilised pain and distress ratings but did not include any ratings for mindful awareness. Ussher et al [88] reported that more than three quarters (78%) of people in their study rated the body scan as being a useful tool for helping them manage chronic pain. While this study was strong on subjective assessment and analysing the immediate effects of a mindfulness body scan meditation on pain ratings, little is known about the effects on the nociceptive system.

Two of the four studies compared a brief mindfulness intervention to distraction [93, 94]. Liu et al [92] found that distraction improves pain tolerance, but not as well as mindfulness. Distraction might have different effects on chronic pain in the short term and it is important that these effects were compared to mindfulness. Liu et al [92] reported that people who generally had longer pain tolerance times tried to distract their attention away from pain by creating a mental image of a happy scene. The authors decided to use this strategy in their study instead of the more common distraction technique of solving math problems, such as Zeidan et al [94] incorporated in their brief mindfulness study. Zeidan et al [94] found math distraction to reduce pain ratings when compared to relaxation, but mindfulness meditation was still shown to be more effective in attenuating pain ratings than both distraction and relaxation. Turning one’s attention away from pain and pain-related thoughts is in contrast to the basis of mindfulness meditation, which is to teach people to feel emotions and bodily sensations more fully and without avoidance [93], so therefore distraction is a good control when analysing coping strategies. Ussher et al [88] used distraction by having the control group listen to a 10-minute audio recording of natural history text read in a
pleasant voice. This has previously been found to be acceptable in controlling the effects of attention when used as a control condition in comparison with a body scan [100, 101].

In summary, the studies reviewed demonstrate that mindfulness meditation delivered in short form of 3 days or less, without the need for a trained therapist, can be beneficial in reducing tolerance time of pain under experimental conditions and reduce pain and distress ratings for people with chronic pain. This has potential widespread benefit for people in pain that cannot afford the time or cost of an 8-week MBSR programme. Outcome measures with regards to pain, however, have been subjective appraisals and to date little is known about the direct influence of brief mindfulness on the nociceptive system.

2.4.3. Mindfulness and nociception

Mindfulness meditation has been shown to be effective in the reduction of pain symptoms [75], however the transformation of nociceptive information into the subjective experience of pain, and then the modulation of pain, is a complex process with overlapping sensory, cognitive and affective dimensions [12]. Little is known about how the brain, brainstem and spinal cord regions involved in mindfulness interact with nociceptive processing. Mindfulness meditation is also associated with the modulation of sensory representations via emotional regulation [90], so delineating direct nociceptive mechanisms involved in mindfulness-related pain modulation remains unclear.

The nociceptive system can be assessed using quantitative sensory testing and functional imaging. Pain measurement consists of psychophysical, psychological and physiological tools of measurement that evaluate pain thresholds, tolerance, pain-related behaviours, and pain-related autonomic events [102]. Quantitative sensory tests are psychophysical in nature, with an objective physical stimulus but a subjective report from the person as the response [103].

Mechanosensitivity is one of the most commonly used criterion to classify response properties of nociceptive neurons in the central nervous system and tenderness to blunt pressure may be due to peripheral or central sensitisation [104]. Most polymodal nociceptors have excitation thresholds below the pain threshold and exhibit adaptation within a few seconds of stimulation, but pain from noxious pressure typically increases with longer lasting stimuli [105], reducing the threshold for nociceptor activation, potentially leading to peripheral and central sensitisation [25]. People with chronic pain typically have lower pain thresholds than in healthy matched controls [106] and Imamura et al. [106] suggest that pain may not only be restricted to painful areas, with sensitivity to pressure also being present at sites distant to the painful area. Pain located in structures away from the source of pain is defined as
referred pain and, as a central phenomenon, has been recognised for years, frequently used for diagnostic purposes [107].

Pressure pain threshold (PPT) is defined as the minimal amount of mechanical pressure that produces pain [108]. Quantitative measurement of pain thresholds due to blunt pressure is performed most commonly using pressure algometry with the aid of a hand-held device [104]. The devices most often used for this purpose are the pressure threshold meter and the pressure pain algometer, which both have a circular rubber pad of 1cm² for contact with the skin. They can be either spring-loaded or electrical with a pneumatic pressure gauge. The pressure algometer is placed perpendicular to the skin and pressure is applied at a constant rate. Recommended pressure application rates range from 0.05 to 20N/s, applied to allow the person time to react when pain is felt [108]. The action of pressure is stopped when the person reports feeling pain.

No studies to date have examined the effects of mindfulness on nociception using PPT measurement; however, two studies used functional imaging to assess the neural mechanisms by which mindfulness meditation can influence pain [109, 110].

Zeidan et al [110] used fMRI to investigate how mindfulness meditation affects pain-related brain processes. In a controlled trial, brain activity was compared in 15 healthy adults in the presence of noxious thermal stimulation, before and after 4 days of meditation training. Prior to being taught mindfulness meditation, participants were familiarised with the sound of the MRI machine and the sensation of thermal stimuli. In the pre-meditation session, participants were tested by alternating application of noxious thermal stimuli (49°C) and neutral stimuli (35°C) to the calf, at rest, and with attention to breath, thereby providing a control for mindfulness meditation. After 4 days of meditation training, participants were tested again, yet this time instructed to meditate by focusing on the changing sensations of breath in the presence of thermal stimuli administered again in alternating patterns of heat and neutral temperatures of 12 seconds duration. Meditation produced a 40% reduction in pain intensity ratings compared with rest and significantly reduced pain unpleasantness ratings by 57%. Meditation reduced pain-related activation of the contralateral primary somatosensory cortex and increased activity in the ACC and anterior insula. Participants with the greatest reductions in pain intensity ratings exhibited the largest meditation-induced activation of the right anterior insula and bilateral ACC, while those with the greatest reductions in pain unpleasantness ratings exhibited greatest activation of the orbital frontal cortex and greatest deactivation of the thalamus.

These findings indicate a possible substrate for pain modulation. Meditation-related activation in these executive-level cortical areas may influence thalamic nociceptive processing [110]. Activation in higher order brain regions, such as the prefrontal
cortex, can regulate lower sensory processes, specifically in the thalamic reticular nuclei [83], thus inducing a gating mechanism which modulates ascending noxious information before accessing cortical regions implicated in conscious perception [110]. In a functional imaging study by Dickenson et al [109], it was shown that focused breathing significantly increased activity in the fronto-parietal regions, pre-supplementary motor area, anterior insula and ACC. The prefrontal cortex is likely to be a pivotal source of pain modulation as it receives sensory information from all modalities and is associated with limbic affective-motivational structures. The ACC also employs control mechanisms to modulate pain through activation of the descending opioid system via the PAG [83]. This aids opioid-mediated analgesia acting on the level of the spinal cord dorsal horn, which is why it is important to measure pressure pain thresholds using mechanosensitivity. Mindfulness might modulate pain perception via pain-specific opioid-sensitive descending modulatory pathways, reducing the excitability of dorsal horn neurons [111].

2.4.4. Mindfulness and the ANS

The autonomic nervous system plays a critical role in regulating cardiovascular responses to mental and physical stress. The balance between sympathetic and parasympathetic regulation of the cardiovascular system is crucial to blood pressure stability and long term health. The sympathetic nervous system (SNS) elevates heart rate and blood pressure via adrenergic activity whereas the parasympathetic nervous system (PNS) slows the heart through cholinergic actions focused at the sinoatrial node [112]. There is a close relationship between cortical functions and cardiovascular health, and clinical studies have shown networks in the brainstem and forebrain to influence autonomic outflow and cardiovascular control [112, 113].

Indices of autonomic function including heart rate, heart rate variability (HRV), skin conductance/resistance, respiratory rate, and electroencephalogram (EEG) activity have become biomarkers for monitoring meditative states [114]. HRV that is related to respiration is known as respiratory sinus arrhythmia (RSA) and is a good index of vagal activity at the cardiac sinoatrial node under most non-stressful conditions [115]. Hence, it is an indicator of PNS activity directed at the heart. RSA is a rhythmical fluctuation in heart periods at the respiratory frequency that is characterised by shortening and lengthening of heart periods in phase with inspiration and expiration respectively [116]. RSA is quantified in people using a vagal tone monitor with a moving polynomial filter to assess beat-to-beat HRV in the adult respiratory frequency band [89]. Estimates of low frequency (0.06 to 0.10Hz) and high frequency (0.12 to 0.40Hz) HRV are often used as an index of cardiac sympathetic activity and parasympathetic activity, respectively [117]. A number of researchers have noted that the magnitude of RSA is influenced not only by outgoing vagal activity but variables such as respiratory frequency, depth, and stimulation of oxygen and carbon dioxide sensitivity [89].
In a study by Phongsuphap et al [118], it was shown that HRV during concentration meditation can change from a normal state in a systematic way. During meditation, the power spectrum of RR (beat-to-beat) intervals tends to shift toward a specific location of frequency to form a resonant peak. This shift in power has a number of health benefits, such as resetting baroreflex sensitivity, increasing parasympathetic tone and improving efficiency of gas exchange in the lung [118]. The power spectrum for short time series can be classified into three ranges [119]: very low frequency, low frequency and high frequency, with the higher frequency being primarily modulated by PNS activity. In Phongsuphap et al’s [118] study, for some cases it was shown that the resonant peak appeared in the high frequency range during meditation, which means that HRV is synchronised to respiratory rhythm, indicating that meditation can increase parasympathetic tone [120]. A resonant peak during meditation reflects a state of coherence, meaning synchronisation among different physiological oscillatory systems such as heart, respiratory, and blood pressure rhythms [121]. Coherence during mediation has the potential benefit of resetting baroreceptor sensitivity involved in short-term blood pressure control [120].

Wallace [122] was among the first to report that conditioning procedures such as meditation can alter autonomic functions and provide evidence that heart rate, skin conductance response (SCR) and blood pressure can be controlled using meditation. Oxygen consumption, heart rate, skin resistance and electrocardiogram (ECG) measurements were recorded before, during and after a single 30-minute session of meditation in 15 healthy college students with experience in meditation. Significant changes were shown between the meditation and control period in all measurements. Oxygen consumption decreased by 20% and remained low during meditation, coupled with a decrease in frequency of breath or tidal volume; skin resistance increased markedly at the onset of meditation, with some rhythmical fluctuations during meditation; and ECG recordings showed a mean decrease of 5 beats per minute in heart rate, indicating an increased PNS activity directed at the sinoatrial node.

Different forms of meditation have different effects on autonomic function and heart rate dynamics [123]; therefore, understanding the physiological effects of various aspects of mindfulness meditation becomes important in deducing which forms are more beneficial for reducing sympathetic activity. Two recent studies compared the short term effects of mindfulness meditation on autonomic and cardiovascular function to relaxation techniques [89, 114]. Both investigated common autonomic indices in the first experiment of each study. In the second experiment, Ditto et al [89] measured pre-ejection period (a measure of cardiac sympathetic activity), while Tang et al [114] used EEG activity to monitor ACC activity.
Ditto et al [89] compared the body scan meditation to progressive muscular relaxation and to a wait-list control group in 32 healthy young adults with no previous experience in meditation. In the first part of the study, individuals participated in 2 sessions, scheduled 4 weeks apart. The body scan group were guided in a therapist-free form using a tape, guiding the listener to attend to various parts of the body, breathing and observing, allowing thoughts to recede for 20 minutes. The progressive muscular relaxation also listened to a tape and practiced techniques that involve tensing and relaxing different muscle groups, while the wait-list control group were tasked to sit quietly for an equal period of time. Autonomic indices measured included heart rate, HRV and RSA.

Ditto et al [89] found that individuals who practiced mindfulness meditation displayed significantly larger baseline-to-treatment increases in RSA than the wait-list control group and the muscular relaxation group. Thus, the body scan meditation appeared to have enhanced PNS activity and this result did not require extensive practice – results showed immediate physiological effects. Heart rate also decreased during the 20-minute treatment period; however, none of the interventions (meditation, muscular relaxation, or just sitting) reduced blood pressure either immediately or over the 1-month period. Another part to the study was then conducted where participants served as their own control. Fifteen of 30 healthy young adults practiced mindfulness meditation during the first session and then listened to a control audio tape in the second session, whereas the other half of the group listened to the audio tape first and then practiced mindfulness second. This within-subject design was aimed at reducing generalisation of results from the first study. Additional cardiovascular measures such cardiac pre-ejection period (PEP) were obtained using an Ambulatory Impedance Monitor.

PEP, a systolic time interval of ventricular depolarisation, is a non-invasive measure which reflects cardiac contractility, a function primarily controlled by beta-adrenergic mechanisms or cardiac sympathetic activity [124]. PEP is useful in psychophysiological research since measurement of heart rate alone does not indicate whether vagal or sympathetic influences are changing autonomic function [124]. Ditto et al [89] found reductions in PEP during meditation, suggesting that meditation may produce an increase in cardiac sympathetic activity. Sometimes heart rate is not altered during meditation, when it would be expected to decrease, and this might be due to the beta-adrenergic influence of PEP [89, 124]. In some cases, experienced meditators exhibit an increase in low frequency HRV reflecting increased sympathetic activity [118, 123]. These findings suggest that complex changes occur in the autonomic nervous system during meditation and that it is not simply a state of rest. Meditation involves active, arousal-promoting processes as well as relaxing processes [89].
Tang et al [114] investigated the short-term effects of meditation on central and autonomic nervous system interaction in 86 Chinese undergraduate students over 5 days, comparing meditation to relaxation. Forty-six people participated in the first experiment, measuring autonomic indices such as heart rate, SCR and respiratory rate; and in the second experiment, 40 people participated using the same autonomic indices plus EEG recordings. After 5 days of mindfulness training, results from experiment 1 showed a reduction in SCR, heart rate, chest respiratory rate, as well as more high-frequency HRV. According to the authors, these factors indicate better ANS regulation, especially more PNS activity during and following mindfulness training in comparison with relaxation [114].

Recent neuroimaging studies have demonstrated that sympathetic outflow to the heart is modulated by the activity of the ACC [112]. Tang et al [114] hypothesised that activity in the ACC increases during mindfulness training because it serves as part of an executive attention network involved in the control of cognition and emotion [125]. To explore these brain mechanisms during short-term meditation, Tang et al [114] recorded brain activity using EEG and single photon emission computed tomography (SPECT) to obtain temporal and spatial information on ACC activity. After 5 days of training, global brain activity was reduced in the meditation group, however more regional cerebral blood flow was shown in the right ACC. To show the relationship between brain activity and physiological indices, the authors performed correlations between changes in the frontal midline theta power and high-frequency HRV. High-frequency HRV is associated with parasympathetic control of the ANS, and ACC activation correlated significantly with this, suggesting ACC influence on PNS activity. In contrast, relaxation produced more frontal, temporal and parietal activations than mindfulness, possibly due to the high level of brain activation required during effortful control in relaxing different parts of the body [114]. Differentiating between cortical influence on PNS and SNS still remains unclear as the two systems are tonically active and operate in conjunction with each other. However the results from Tang et al [114] are consistent with other studies which indicate that mindfulness meditation and the medial prefrontal cortex is directly involved in human cardiovagal control [112], increases parasympathetic activity [114], and does not require a significant amount of training.

Mindfulness could be a useful strategy for chronic pain but little has been done to examine the short-term effects of mindfulness on the ANS of this population. Brief mindfulness techniques require limited training. Therefore, if chronic pain is driven or reinforced by alterations in autonomic function, then people with chronic pain could benefit from techniques that are able to mitigate these alterations.
Chapter 3: Method

3.1. Introduction

The purpose of this study was to measure the immediate effects of a brief mindfulness body scan meditation on self-reported pain, the nociceptive system and autonomic nervous system in people with chronic pain. The mindfulness intervention was compared to a control intervention. The following chapter outlines the method used in the research by describing the study design, participants, procedure, interventions, outcome measures, data management and statistical analysis.

3.2. Study setting and design

This study was undertaken at Spinewave Wellness Centre, a private practice in Remuera, Auckland, NZ. A between-subject, repeated measures, double-blinded, randomised controlled trial with experimental and control interventions was used.

3.3. Study participants

3.3.1. Sample size

The sample size calculation was undertaken using an alpha level of 0.05 and power of 0.8 using G*Power 3.1.3 [126]. A prior power calculation for an F test using G*Power revealed that for a moderate effect size of 0.3, with 2 groups, N = 24 participants will be required. An effect size of 0.3 was selected based on a similar previous study in this field that showed a significant reduction in pain after a 10-minute session of mindfulness in people with chronic pain [88]. An effect size of 0.3 will account for 9% total variance within the sample [127]. The sample size was increased to 30 participants (or 15 per group) as the effect size of the intervention on autonomic system outcome measures was uncertain but estimated to be lower.

3.3.2. Recruitment

Participants were sourced from advertising in local papers, web-based social media and through professional networks. The newspaper advertisement (Appendix D) included the study title, brief information on the study, eligibility criteria and an invitation to volunteer.

3.3.3. Inclusion and exclusion criteria

Participants were included in the study if they satisfied the following inclusion criteria:

- Aged over 18 years.
- Diagnosed by a clinician with a chronic pain condition such as fibromyalgia, complex regional pain syndrome, neuralgia, or general longstanding chronic pain like back, neck, leg or arm pain.
• Had experienced pain for at least 3 months.

Participants were excluded if they had any of the following:

• Previously engaged in mindfulness-based programmes because experienced meditators exhibit greater effect on the autonomic nervous system than novices [118].
• Unstable heart condition or pacemaker due to unwanted beat-to-beat signals [128].
• Unstable health condition as this could confound results of the experiment.
• Head pain, since pressure pain thresholds cannot be recorded on the head.
• Poor hearing or poor command of the English language, since the interventions required listening to and understanding an audio file.
• Prescribed and taking psychotropic medication as this could confound results of the experiment [88].
• Social issues that precluded them from attending the session, such as lack of transport or family commitments.
• Inability to provide informed consent.

3.4. Ethical and cultural considerations

Ethical approval was obtained from the AUT University Ethics Committee (AUTEC), approval number 13/354 (Appendix A). During the design and implementation of the study the principles of the Treaty of Waitangi, including partnership, participation and protection were applied, and the recruitment process ensured that all eligible participants had equal opportunity to take part in the study regardless of ethnicity. Each participant was fully informed with a participant information sheet (Appendix E) and consented to participating in the study by signing a consent form (Appendix F), being aware that they had equal chance of being in either the experimental or control group. The privacy of each participant was maintained at all times by assigning participant codes.

3.5. Study procedure

The following section describes the study procedure, including pre- and post-intervention outcome measures, experimental and control interventions (Fig. 4).

Volunteers contacted the researcher via e-mail or telephone after learning of the study through the recruitment procedures used, as outlined above (see 3.3.2). At this point, each volunteer was screened for inclusion and exclusion criteria, as well discussing the nature of their pain. Each participant was then e-mailed the participant information sheet (Appendix E) to be informed of the purpose of the study and its procedure in writing. Eligible participants were invited to attend one session at the
At this session participants were asked for personal details including age, gender, handedness and pain condition, and provided written informed consent (Appendix F). The nature of each participant’s pain was assessed prior to the intervention using the Pain Catastrophising Scale (see 3.5.1) and the Short-Form McGill Pain Questionnaire II (see 3.5.2).

The study was a double-blinded, randomised controlled trial; therefore, both participants and the researcher were blinded to the intervention received. Participants were informed during the screening process that they had equal chance of being in the intervention group or the control group. If a participant was randomised to the control group, they were offered an opportunity to undergo the real mindfulness body scan at the completion of the study if it was shown to be beneficial.

Participants were told an audio file would be played for them whilst they were sitting quietly and they were asked to listen to the instructions (Fig. 3). Participant grouping was randomised using a computer generated random list within Microsoft Excel (2010). The interventions were provided on an audio file following a standardised procedure which allowed the researcher to remain blinded. Two audio files, labelled Tape A and Tape B, were sent to the researcher and loaded onto a laptop. The audio file delivered to each participant through headphones contained either the experimental or control intervention and was played without the researcher present in the room.

Two outcome measures of pain were recorded at baseline (before the intervention), immediately following the intervention, and 15 minutes after the intervention. The first was a rating of pain severity on a visual analogue scale (VAS) (see 3.7.2) and the second was PPT at a site of pain on the body and at a control or non-painful site (see 3.7.1).

While the audio file was playing, physiological data was constantly recorded. Four autonomic indices were recorded continuously from baseline (before the intervention) through to 15 minutes following the intervention. These were blood volume pulse (see

Figure 3. Participant positioning
3.7.3), heart rate (see 3.7.4), heart rate variability (see 3.7.5), and skin conductance response (see 3.7.6).

![Procedure outline diagram]

**Figure 4. Procedure outline**

### 3.5.1. Pain Catastrophising Scale

As pain catastrophising affects how individuals experience pain, the Pain Catastrophising Scale (PCS) was used to assess pain catastrophising within the sample. The PCS is a 13-item tool derived from definitions of catastrophising and can be completed in less than 5 minutes [129] (Appendix B).

People who catastrophise tend to do three things, all of which are measured by this questionnaire: 1. They ruminate about their pain (“I can’t stop thinking about how much it hurts”); 2. They magnify their pain (“I’m afraid that something serious might happen”); and 3. They feel helpless to manage their pain (“there is nothing I can do to reduce the intensity of my pain”) [37].
A total PCS score of 30 out of 52 corresponds to the 75th percentile of PCS scores in samples of people with chronic pain and represents a clinically relevant level of catastrophising [129]. People who score between the 50th and 75th percentile on the PCS are considered at moderate risk for the development of chronicity. Catastrophising not only contributes to heightened levels of pain and emotional distress, but also increases the probability that the pain condition will persist over an extended period of time [129].

3.5.2. Short-Form McGill Pain Questionnaire

The nature and sensitivity of a pain condition can influence results. The Short-Form McGill Pain Questionnaire II (SFMPQII) helps describe the level of pain participants are experiencing. The main component of the SFMPQII consists of 22 descriptors of pain rated on a Likert scale from 0 to 10 (Appendix C).

The first Short-Form McGill Pain Questionnaire (SFMPQI), developed by Melzack [130], includes visual analogue and verbal rating scales, as well as 15 pain descriptors. The SFMPQII was expanded upon to include seven additional descriptors so that it would provide a comprehensive assessment and characterisation of the symptoms of both neuropathic and non-neuropathic pain [131]. Studies by Dworkin et al [131] suggest the SFMPQII provides reliable and valid support for four components of pain: continuous pain, intermittent pain, neuropathic pain, and affective descriptors.

The total Pain Rating Index score is obtained by summing the item scores from the SFMPQII. A higher score on the SFMPQII indicates worse pain [132]. The Pain Rating Index is interpreted in terms of quantity of pain, as evidenced by the number of words used and the rank of value for each word, as well as the quality of pain, defined by the particular words that are chosen [132].

3.6. Experimental and control interventions

3.6.1. Mindfulness body scan meditation

The body scan meditation intervention was a brief 10-minute audio recording that guided the attention of the participant through their entire body, focusing non-critically on any sensation and using periodic suggestions of breath awareness and relaxation [1, 77]. The instructions asked participants to acknowledge all sensations without attempting to change them by practising mindful acceptance [88]. The process included noticing, but not reacting to pain, emotions, urges, thoughts and other feelings in the body [80]. When the person noticed their mind start to wander into thoughts, memories or fantasies, the content of these feelings was briefly noted and then attention returned to breath or the present moment.
3.6.2. Control intervention

To control for the potential influences of pure relaxation as people take time out of the day to sit in a quiet room and listen passively to a person speaking in a pleasant voice, an audio recording of a narrator reading a section of Dan Brown’s book “The Lost Symbol” [133] was used as a control intervention. The section of the audio book used holds the listener’s attention but is not exciting. This type of control has previously been found to be acceptable in countering the effects of attention when used as a control condition in comparison with a body scan meditation [88, 89].

3.7. Outcome Measures

3.7.1. Pressure pain threshold

Pressure pain threshold (PPT) is defined as the minimal amount of mechanical pressure applied to a specific body that produces pain [108]. A simple handheld pressure algometer (Lafayette Manual Muscle Testing System, Model 01165, USA) was placed perpendicular to the tissue surface at two standard locations and pressure applied steadily at a constant rate. One location was a nominated site of chronic pain and the other a non-painful site on the opposite limb or trunk. When the participant reported feeling pain during application, the action of pressure was stopped and the maximum pressure achieved recorded. Each location was assessed twice before the intervention (baseline), immediately following the intervention, and 15 minutes after the intervention (Fig. 11). The two PPT readings taken at each interval were averaged and recorded, i.e. two PPT recordings at the painful site and two PPT recordings at the non-painful site.

3.7.2. Visual analogue scale

The visual analogue scale (VAS) is a subjective, psychometric measure of pain. A participant is asked to indicate his or her perceived pain intensity along a 100mm horizontal line. The left end of the line is marked with “no pain” and the right end “worst pain imaginable” (Fig. 5). The distance of the rating measured from the left edge produces the VAS score.

![Visual analogue scale](image)

*Figure 5. Visual analogue scale*

Changes in the VAS score represent a relative change in the magnitude of pain sensation. The VAS has properties consistent with a linear scale; thus, if the VAS score is halved after a clinical intervention, then the person’s pain is halved [134]. The VAS score correlates well with acute pain levels but has an error of
approximately 20mm [134]. Each participant recorded their VAS score before the intervention (baseline), immediately following the intervention, and 15 minutes post intervention. For the two post-intervention measures, the participants were not able to see their previous score(s) while rating their current score.

3.7.3. Blood volume pulse

Blood volume pulse (BVP) was measured using a photoplethysmograph and indicates dynamic changes in blood volume underneath the sensor. BVP is not an outcome measure itself but is used as the application to estimate heart rate and HRV. The BVP signal indicates relative changes in the vascular bed due to vasodilation or vasoconstriction (increases or decreases in blood perfusion), as well as changes in the elasticity of the vascular walls, which may correlate with changes in blood pressure [128]. The photoplethysmograph was placed over the ring finger (Fig. 6) of the non-dominant hand, as the index and middle fingers were used for recording the SCR. BVP was recorded at 128Hz using a NeXus-10 MKII and BioTrace software (MindMedia, Netherlands).

3.7.4. Heart rate

The volume of blood in the arteries and capillary beds increases with each arterial pulsation. Heart rate (HR) can be estimated from the BVP signal [128]. HR is the number of heart beats per minute (bpm) and is calculated by measuring the time interval between the heart beats, or the interbeat interval. The time of the interbeat interval is divided into 60 seconds to calculate the beat-by-beat HR.

![Figure 6. Photoplethysmograph (BVP) placement](image)

3.7.5. Heart rate variability

HRV is the amount of HR fluctuation around the mean HR [119]. HRV describes variations of both instantaneous HR and beat-to-beat intervals that can be analysed
using time or frequency domain methods [135]. Cardiac cycles centred on the tone of the sinoatrial node produce rhythms which are modulated by central oscillations (e.g. respiratory centres) and peripheral oscillations (e.g. blood pressure) that are influenced by autonomic activity [119]. Spectral analysis of HRV examines these frequency-specific oscillations. Estimates of low frequency (0.06 to 0.10Hz) and high frequency (0.12 to 0.40Hz) HRV are often used as an index of cardiac sympathetic activity and parasympathetic activity respectively [117]. Low frequency (LF) power divided by high frequency (HF) power produces an LF/HF power ratio, which is considered an indicator of sympathovagal balance [136] with relative normal limits falling between 1.0 and 2.0 [119]. Very low frequency (VLF) HRV is strictly greater than 0.00Hz but less than 0.04Hz [137]. The physiological explanation of the VLF component of HRV is not well defined [119] and is usually attributed to non-harmonic properties that reflect slow, regulatory mechanisms such as thermoregulation [138]. For this reason VLF is usually avoided when interpreting short term ECG recordings [119] and subtracted from the total power of LF and HF (Table 2). Normalised spectral HRV measures express quantities on a percentage scale basis and normalised units of high frequency (nuHF) HRV is the index of modulation of parasympathetic activity [137]. A higher score indicates greater parasympathetic activity; therefore, nuHF was used as the main HRV outcome measure in this study.

3.7.6. Skin conductance response

The SCR is a momentary increase in the electrical conductivity of the skin associated with increased sweat gland activity [139]. The SCR is typically measured from the palmar surface of the hand where eccrine sweat gland density is greatest. A pair of electrodes was placed on the palmar tips of the index and middle fingers of the non-dominant hand [140] after sites had been pre-treated with ethanol wipes (Fig. 7). A small direct electrical current passes through the electrodes and measures conductance through the skin. As sweat glands become more active, electrical conductivity of the skin momentarily increases resulting in a SCR, typically ranging between 0.1 and 2.0 micro Siemen (µS) units [139]. A lower score shows more parasympathetic activity [114]. The SCR is elicited by stimuli that are novel, unexpected, intense, complex or emotionally arousing [139]. The SCR was recorded at 32Hz using a NeXus-10 MKII and BioTrace software (MindMedia, Netherlands).
3.8. Data processing

Autonomic indices were recorded continuously without interruption during the 10-minute intervention and 15 minutes post intervention using NeXus-10 MKII and BioTrace software (MindMedia, Netherlands). A total of 25 minutes of raw data (SCR, BVP and HR) were recorded with BioTrace (Fig. 8). Autonomic variables were processed in 5 epochs of 5-minute intervals. These were then grouped to match the pain time epochs (Fig. 11). Epoch 1 and 2 spanned the 10-minute intervention, and epochs 3, 4 and 5 spanned the 15-minute rest period after the intervention.

Raw interbeat interval (IBI) data were obtained from the BVP trace (Fig. 8) using BioTrace’s HRV analysis function. IBI data, including any artefacts, were exported in ASCII text file format to Kubios HRV (Biosignal Analysis and Medical Imaging Group, Finland).
Kubios HRV is advanced software for studying the variability of heart beat intervals [141], including time-domain (Fig. 9) and frequency-domain (Fig. 10) variables of HRV.

**Figure 9. Time-domain variables of HRV in Kubios**

The following time-domain variables were recorded for analysis: Mean heart rate (mean HR), standard deviation of instantaneous heart rate values (STD HR), and the square root of the mean squared differences between successive RR intervals (RMSSD), which can be used as a measure of short term variability [141].

**Figure 10. Frequency-domain variables of HRV in Kubios**

The generalised frequency bands for short term HRV recordings are VLF (0-0.04Hz), LF (0.04-0.15Hz) and HF (0.15-0.4Hz) [141]. In frequency-domain methods, a power spectrum density estimate is calculated for RR interval series and frequency bands are extracted from this. Frequency bands extracted for this study were: absolute and relative powers of LF and HF bands; LF and HF band powers in normalised units (nuLF, nuHF); the LF/HF power ratio; and peak frequencies for each band (Table 2).

SCR data, measured in micro Siemens (µS), were exported in ASCII text file format to Microsoft Excel (2010). Data for the 25 minutes of total recording were divided into 5-minute epochs as discussed previously and the average of 9,600 samples was entered for each epoch. This provided an average of the SCR response over time.
### Table 2. Frequency domain measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Units</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak frequency</td>
<td>Hz</td>
<td>VLF, LF, and HF band peak frequencies</td>
</tr>
<tr>
<td>Absolute power</td>
<td>ms²</td>
<td>Absolute powers of VLF, LF, and HF bands</td>
</tr>
<tr>
<td>Relative power</td>
<td>%</td>
<td>Relative powers of VLF, LF, and HF bands</td>
</tr>
<tr>
<td></td>
<td></td>
<td>VLF (%) = VLF (ms²) / total power (ms²) x 100%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LF (%) = VLF (ms²) / total power (ms²) x 100%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HF (%) = VLF (ms²) / total power (ms²) x 100%</td>
</tr>
<tr>
<td>Normalised power</td>
<td>nu</td>
<td>Powers of LF and HF bands in normalised units</td>
</tr>
<tr>
<td></td>
<td></td>
<td>LF (nu) = LF (ms²) / (total power [ms²] - VLF[ms²])</td>
</tr>
<tr>
<td></td>
<td></td>
<td>HF (nu) = LF (ms²) / (total power [ms²] - VLF[ms²])</td>
</tr>
<tr>
<td>LF/HF</td>
<td></td>
<td>LF/HF Ratio between LF and HF band powers</td>
</tr>
</tbody>
</table>

Note: Hz = hertz; VLF = very low frequency; LF = low frequency; HF = high frequency; ms² = milliseconds squared; nu = normalised units.

PPT readings, recorded in Newtons, and VAS measurements, recorded in millimetres, were spread into 3 epochs: baseline, immediately post intervention (10 minutes), and 15 minutes post intervention (Fig. 11). Two PPT readings were taken at each interval and averaged for the painful site and non-painful site. One VAS measurement was recorded at each interval.

| Baseline | Immediately post intervention (10 minutes) | 15 minutes post intervention |

**Figure 11. Timeline of assessments**

### 3.9. Data analysis

All data, including autonomic and pain variables, was then entered into SPSS (SPSS 220.0 for Windows, SPSS Inc., USA) for analysis. Following completion of the study, the researcher was unblinded to the interventions. Tape A was the experimental intervention and Tape B was the control intervention.

Analysis involved two phases. Firstly, a descriptive analysis of the participants’ characteristics and baseline variables of interest were performed for each group.
Comparisons between the two groups for each variable were performed using t-test and chi-square test.

Secondly, a generalised linear model using repeated measures over 3 time epochs (Fig. 11), with “tape” (intervention) as a between factor, was used to analyse the pain outcome measures. The primary dependent variables for pain were: PPT recordings at the painful site, PPT recordings at the non-painful site, and VAS measurements.

Additionally, a generalised linear model using repeated measures over 5 time epochs, with “tape” (intervention) as a between factor, was used to analyse the autonomic data. Note that epoch 1 and 2 spanned the intervention period and epochs 3, 4 and 5 spanned the rest period after the intervention. The primary dependent autonomic variables were: mean HR, nuHF, LF/HF ratio, and SCR.

The estimate of the intervention effect was adjusted for potential confounding effects of the following covariables: age, gender, PCS and SFMPQII scores. A $p$ value of less than 0.05 was considered significant.
Chapter 4: Results

4.1. Introduction

The following chapter presents the main findings of the study. It will provide an overview of recruitment and retention, followed by a description of the participants’ characteristics. Finally, analysis of the effect of the interventions on self-reported pain, the nociceptive system and autonomic nervous system will be presented.

4.2. Recruitment and retention

Thirty-six people volunteered for the study. One participant was excluded for having a pacemaker. Of the 35 meeting the inclusion criteria, four did not respond to follow-up calls to finalise participation and one did not show for her session. Thirty participants (n = 15 per group) completed the 10-minute intervention and 15-minute rest period (Fig. 12). Data collection took place from September 2014 to April 2015.

4.3. Sample characteristics

The age of participants ranged from 25 – 81 years, with 7 male and 23 female participants. Five people were left handed and 25 right handed. Eligible participants presented with the following chronic pain conditions: 9 with musculoskeletal pain, 5 with fibromyalgia, 3 with spinal pain, 9 with arthritis, 3 with neuropathy, and 1 with complex regional pain syndrome.

The mean PCS score of participants for the current study was 19 (48th percentile) with a maximum of 45 (97th percentile) and a minimum score of 2 (5th percentile). Eight participants scored between the 50th and 75th percentile and 4 participants scored above the 75th percentile. The PCS score was shown to influence self-reported measures of pain (4.4) and mean HR (4.6.1).
For the purpose of this study, participant's total SFMPQII score was summed to indicate the person's current level of pain. The mean SFPMQII score was 70, with a maximum score of 188 and minimum of 13. The SFMPQII score was shown to influence PPT of the painful site (4.5.1), PPT of the non-painful site (4.5.2), mean HR (4.6.1), high frequency HRV (4.6.2), and LF/HF power ratio (4.6.3).

As shown in Table 3, there were no significant differences between the groups at baseline before listening to either Tape A (experimental intervention) or Tape B (control intervention).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Tape A</th>
<th>Tape B</th>
<th>Statistical Test</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, Mean (SD)</td>
<td>59.13 (16.41)</td>
<td>60.47 (11.58)</td>
<td>T-Test</td>
<td>0.799</td>
</tr>
<tr>
<td>PCS, Mean (SD)</td>
<td>20.47 (9.43)</td>
<td>17.47 (11.72)</td>
<td>T-Test</td>
<td>0.446</td>
</tr>
<tr>
<td>SFMPQII, Mean (SD)</td>
<td>64.40 (36.63)</td>
<td>75.20 (43.60)</td>
<td>T-Test</td>
<td>0.469</td>
</tr>
<tr>
<td>Gender, N (%)</td>
<td></td>
<td></td>
<td>Chi-Square Test</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>5 (33)</td>
<td>3 (25)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>10 (66)</td>
<td>12 (75)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Handedness, N (%)</td>
<td></td>
<td></td>
<td>Chi-Square Test</td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>12 (80)</td>
<td>13 (87)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>3 (20)</td>
<td>2 (13)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Tape A = experimental intervention; Tape B = control intervention; SD = standard deviation; PCS = Pain Catastrophising Scale; SFMPQII = Short-Form McGill Pain Questionnaire II; N = number of participants.

4.4. Self-reported pain

Comparison of experimental (Tape A) and control (Tape B) interventions for self-reported measures of pain using VAS scores is represented in Figure 13. Results are presented in Table 4. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.

There was no significant main effect of intervention ($\beta = -0.081$, $p = 0.986$), of time post intervention ($\beta = -8.233$, $p = 0.145$), of time 15 minutes post intervention ($\beta = -6.833$, $p = 0.227$), or interaction effect of intervention x time ($\beta = 2.333$, $p = 0.846$) on VAS scores.
Figure 13. VAS self-reported measures of pain

Note: VAS = Visual Analogue Scale; error bars = standard error of the mean; mm = millimetres.

Table 4. Statistical model results for VAS scores showing intervention and time effects/interaction with covariables that were retained in the model

<table>
<thead>
<tr>
<th>Variable</th>
<th>β (SE)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>-0.081 (4.716)</td>
<td>0.986</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>-8.233 (5.651)</td>
<td>0.145</td>
</tr>
<tr>
<td>Time, 15 min post intervention</td>
<td>-6.833 (56.651)</td>
<td>0.227</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>2.333 (12.042)</td>
<td>0.846</td>
</tr>
<tr>
<td>PCS score</td>
<td>0.708 (0.225)</td>
<td>0.002</td>
</tr>
<tr>
<td>Gender, Male</td>
<td>-8.643 (5.279)</td>
<td>0.102</td>
</tr>
</tbody>
</table>

Note: VAS = Visual Analogue Scale; β = beta; SE = standard error; Tape A = experimental intervention; PCS = Pain Catastrophising Scale.
PCS scores were significantly associated with VAS scores ($\beta = 0.708$, $p = 0.002$), i.e. for each PCS point increase, the VAS score increased by 0.708mm. There appeared to be an influence of gender ($\beta = -8.643$, $p = 0.102$), i.e. males were associated with lower VAS scores across all time epochs. SFMPQII scores and age were not associated with VAS scores therefore not retained in this statistical model.

These results do not support the hypothesis that a single session body scan meditation will reduce self-reported measures of pain for people with chronic pain.

4.5. Nociceptive system

4.5.1. PPT of the painful site

Comparison of experimental (Tape A) and control (Tape B) interventions for pressure pain thresholds at the participant’s nominated painful site is represented in Figure 14. Results are presented in Table 5. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.

![Figure 14. PPT recordings of the painful site](image)

*Note: PPT = pressure pain threshold; error bars = standard error of the mean; N = Newton.*

There was no significant main effect of intervention ($\beta = 6.567$, $p = 0.184$), of time post intervention ($\beta = 6.433$, $p = 0.277$), of time 15 minutes post intervention ($\beta = 7.757$, $p = 0.190$), or interaction effect of intervention x time ($\beta = 4.233$, $p = 0.769$) on PPT of the painful site.
Gender was significantly associated with PPT ($\beta = 33.201, \ p < 0.001$), i.e. the mean PPT was $33.2\, \text{N}$ higher among males than females across each epoch. SFMPQII scores were significantly associated with PPT of the painful site ($\beta = -0.178, \ p = 0.004$), i.e. every increase in the Pain Rating Index was associated with a reduction of PPT by $0.178\, \text{N}$. PCS scores and age were not associated with PPT of the painful site therefore not retained in this statistical model.

### Table 5. Statistical model results for PPT of the painful site showing intervention and time effects/interaction with covariables that were retained in the model

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$ (SE)</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>6.567 (4.938)</td>
<td>0.184</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>6.433 (5.922)</td>
<td>0.277</td>
</tr>
<tr>
<td>Time, 15 min post intervention</td>
<td>7.757 (5.922)</td>
<td>0.190</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>4.233 (14.431)</td>
<td>0.769</td>
</tr>
<tr>
<td>SFMPQII score</td>
<td>-0.178 (0.062)</td>
<td>0.004</td>
</tr>
<tr>
<td>Gender, Male</td>
<td>33.201 (5.531)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: PPT = pressure pain threshold; $\beta$ = beta; SE = standard error; Tape A = experimental intervention; SFMPQII = Short-Form McGill Pain Questionnaire II.

These results do not support the hypothesis that a single session body scan meditation will reduce the sensitivity of the nociceptive system for people with chronic pain.

#### 4.5.2. PPT of the non-painful site

Comparison of experimental (Tape A) and control (Tape B) interventions for pressure pain thresholds at the participant’s nominated non-painful site is represented in Figure 15. Results are presented in Table 6. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.
There was no significant main effect of intervention ($\beta = 4.912, p = 0.428$), of time post intervention ($\beta = -3.997, p = 0.591$), of time 15 minutes post intervention ($\beta = -4.938, p = 0.507$), or interaction effect of intervention x time ($\beta = -0.740, p = 0.967$) on PPT of the non-painful site.

Gender was significantly associated with PPT ($\beta = 27.203, p < 0.001$), i.e. the mean PPT was 27.2N higher among males than females across each time epoch. SFMPQII scores were significantly associated with PPT of the non-painful site ($\beta = -0.390, p < 0.001$), i.e. every increase in the Pain Rating Index was associated with a reduction in PPT by 0.39N. PCS scores and age were not associated with PPT of the non-painful site therefore not retained in this statistical model.

![Figure 15. PPT recordings of the non-painful site](image-url)

**Figure 15.** PPT recordings of the non-painful site

*Note: PPT = pressure pain threshold; error bars = standard error of the mean; N = Newton.*
Table 6. Statistical model results for PPT of the non-painful site showing intervention and time effects/interaction with covariables that were retained in the model

<table>
<thead>
<tr>
<th>Variable</th>
<th>β (SE)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>4.912  (6.202)</td>
<td>0.428</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>-3.997 (7.438)</td>
<td>0.591</td>
</tr>
<tr>
<td>Time, 15 min post intervention</td>
<td>-4.938 (7.437)</td>
<td>0.507</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>-0.740 (17.871)</td>
<td>0.967</td>
</tr>
<tr>
<td>SFMPQII score</td>
<td>-0.390 (0.781)</td>
<td>0.001</td>
</tr>
<tr>
<td>Gender, Male</td>
<td>27.203 (6.946)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: PPT = pressure pain threshold; β = beta; SE = standard error; Tape A = experimental intervention; SFMPQII = Short-Form McGill Pain Questionnaire II.

These results do not support the hypothesis that a single session body scan meditation will reduce the sensitivity of the nociceptive system for people with chronic pain.

4.6. Autonomic nervous system

4.6.1. Mean heart rate

Comparison of experimental (Tape A) and control (Tape B) interventions for mean HR is represented in Figure 16. Results are presented in Table 7. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.

There was no significant main effect of intervention (β = -2.632, p = 0.100), of time post intervention (β = 0.301, p = 0.845), or interaction effect of intervention x time (β = -1.548, p = 0.682) on mean HR.

Those who listened to the experimental intervention showed a non-significant trend (p = 0.100) of reduced mean HR of 2.632bpm across all 5 time epochs. Gender was significantly associated with mean HR; males displayed a higher HR (8.946bpm) over
all epochs ($p < 0.001$). There was a significant decrease of 0.417bpm for an increase in age by one year ($p < 0.001$) and a significant increase of 0.075bpm for each Pain Rating Index point (SFMPSII; $p = 0.003$). There appeared to be an influence of PCS scores on mean HR ($\beta = -0.177$, $p = 0.056$).

**Figure 16.** Mean HR

*Note: Intervention = epoch 1 and 2; post intervention = epochs 3, 4 and 5; Hz = hertz; HR = heart rate.*
Table 7. **Statistical model results for mean HR showing intervention and time effects/interaction with covariables that were retained in the model**

<table>
<thead>
<tr>
<th>Variable</th>
<th>β (SE)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>-2.632 (1.599)</td>
<td>0.100</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>0.301 (1.539)</td>
<td>0.845</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>-1.548 (3.774)</td>
<td>0.682</td>
</tr>
<tr>
<td>PCS score</td>
<td>-0.177 (0.093)</td>
<td>0.056</td>
</tr>
<tr>
<td>SFMPQII score</td>
<td>0.075 (0.025)</td>
<td>0.003</td>
</tr>
<tr>
<td>Age</td>
<td>-0.417 (0.057)</td>
<td>0.001</td>
</tr>
<tr>
<td>Gender, Male</td>
<td>8.946 (1.732)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: HR = heart rate; β = beta; SE = standard error; Tape A = experimental intervention; PCS = Pain Catastrophising Scale; SFMPQII = Short-Form McGill Pain Questionnaire II.

These results do not support the hypothesis that a single session body scan meditation will shift the ANS to a less sympathetic dominant state for people with chronic pain.

**4.6.2. High frequency HRV**

Comparison of experimental (Tape A) and control (Tape B) interventions for percentage of change in the normalised unit of high frequency HRV (nuHF) is represented in Figure 17. Results are presented in Table 8. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.

There was no significant main effect of intervention (β = -1.355, p = 0.657), of time post intervention (β = -4.593, p = 0.137), or interaction effect of intervention x time (β = 1.553, p = 0.821) on nuHF.

It was found that age was significantly associated with nuHF (β = 0.546, p < 0.001), i.e. nuHF increased by 0.546% with an increase in age by one year. SFMPQII scores
were also significantly associated with nuHF ($\beta = 0.093$, $p = 0.019$), i.e. nuHF increased by 0.093% for each Pain Rating Index point. PCS scores and gender were not associated with nuHF therefore not retained in this statistical model.

Figure 17. High frequency HRV in normalised units

Note: Intervention = epoch 1 and 2; post intervention = epochs 3, 4 and 5; nuHF = normalised units of high frequency; HRV = heart rate variability.
Table 8. Statistical model results for nuHF showing intervention and time effects/interaction with covariables that were retained in the model

<table>
<thead>
<tr>
<th>Variable</th>
<th>β (SE)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>-1.355 (3.052)</td>
<td>0.657</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>-4.593 (3.085)</td>
<td>0.137</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>1.553 (6.861)</td>
<td>0.821</td>
</tr>
<tr>
<td>SFMPQII score</td>
<td>0.093 (0.039)</td>
<td>0.019</td>
</tr>
<tr>
<td>Age</td>
<td>0.546 (0.113)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: nuHF = normalised units of high frequency; β = beta; SE = standard error; Tape A = experimental intervention; SFMPQII = Short-Form McGill Pain Questionnaire II.

These results do not support the hypothesis that a single session body scan meditation will shift the ANS to a less sympathetic dominant state for people with chronic pain.

4.6.3. LF/HF power ratio

Comparison of experimental (Tape A) and control (Tape B) interventions for LF/HF power ratio is represented in Figure 18. Results are presented in Table 9. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.

There was no significant main effect of intervention (β = -0.464, p = 0.233), of time post intervention (β = -0.084, p = 0.831), or interaction effect of intervention x time (β = 0.199, p = 0.813) on LF/HF power ratio.

It was found that age was significantly associated with LF/HF (β = -0.052, p < 0.001), i.e. LF/HF decreased by 0.052 with an increase in age by one year. SFMPQII scores were also significantly associated with LF/HF (β = -0.010, p = 0.048), i.e. LF/HF decreased by 0.010 for each Pain Rating Index point. PCS scores and gender were not associated with LF/HF power ratio therefore not retained in this statistical model.
Figure 18. LF/HF power ratio

Note: Intervention = epoch 1 and 2; post intervention = epochs 3, 4 and 5; LF = low frequency; HF = high frequency.

Table 9. Statistical model results for LF/HF power ratio showing intervention and time effects/interaction with covariables that were retained in the model

<table>
<thead>
<tr>
<th>Variable</th>
<th>$\beta$ (SE)</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>-0.464 (0.389)</td>
<td>0.233</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>-0.084 (0.394)</td>
<td>0.831</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>0.199 (0.841)</td>
<td>0.813</td>
</tr>
<tr>
<td>SFMPQII score</td>
<td>-0.010 (0.005)</td>
<td>0.048</td>
</tr>
<tr>
<td>Age</td>
<td>-0.052 (0.014)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: LF = low frequency; HF = high frequency; $\beta$ = beta; SE = standard error; Tape A = experimental intervention; SFMPQII = Short-Form McGill Pain Questionnaire II.

For participants who listened to the experimental intervention, LF/HF power ratio diminished by 0.464 over all time epochs compared to the control, implying a shift in sympathovagal tone toward the parasympathetic over 25 minutes. This was not significant though ($p = 0.233$) and does not support the hypothesis that a single
session body scan meditation will shift the ANS to a less sympathetic dominant state for people with chronic pain.

4.6.4. Skin conductance response

Comparison of experimental (Tape A) and control (Tape B) interventions for SCR is represented in Figure 19. Results are presented in Table 10. The estimate of the intervention effect was adjusted for potential confounding effects of age, gender, PCS and SFMPQII scores.

There was no significant main effect of intervention ($\beta = 0.546, p = 0.318$) on SCR. SCR increased by 0.546\,\mu S across all time epochs for participants who received the experimental intervention. Time post intervention was significant ($\beta = 1.356, p = 0.014$) on SCR but does not support the hypothesis that a single session body scan meditation will shift the ANS to a less sympathetic dominant state. There was no significant interaction effect of intervention x time ($\beta = -0.470, p = 0.722$).

Age and gender were significantly associated with SCR (Table 10). An increase in age by one year reduced SCR by 0.104\,\mu S and males lowered SCR recordings by 3.52\,\mu S across all time epochs. There was a minor trend of decreased SCR during both interventions but this was not maintained. PCS and SFMPQII scores were not associated with SCR therefore not retained in this statistical model.

This result does not support the hypothesis that a single session body scan meditation will shift the ANS to a less sympathetic dominant state for people with chronic pain.
Table 10. **Statistical model results for SCR showing intervention and time effects/interaction with covariables that were retained in the model**

<table>
<thead>
<tr>
<th>Variable</th>
<th>β   (SE)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tape A</td>
<td>0.546 (0.548)</td>
<td>0.318</td>
</tr>
<tr>
<td>Time, Post intervention</td>
<td>1.356 (0.551)</td>
<td>0.014</td>
</tr>
<tr>
<td>Tape x Time interaction</td>
<td>-0.470 (1.323)</td>
<td>0.722</td>
</tr>
<tr>
<td>Age</td>
<td>-0.104 (0.020)</td>
<td>0.001</td>
</tr>
<tr>
<td>Gender, Male</td>
<td>-3.520 (0.620)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: SCR = skin conductance response; β = beta; SE = standard error; Tape A = experimental intervention.
Chapter 5: Discussion

The purpose of this study was to measure the immediate effects of a brief mindfulness body scan meditation on self-reported pain, the nociceptive system and autonomic nervous system. Results of this study showed no statistical differences between the group that listened to the experimental mindfulness tape and the group that listened to the control tape on any of the outcome measures. However, weak trends in reduced pain were observed for self-reported pain and pain sensitivity as measured by pressure pain thresholds for both groups.

The weak trends observed for self-reported pain and PPTs are comparable to previous research findings indicating that mindfulness may be effective in reducing self-reported pain [88, 92-94]. Whilst the findings did not reach statistical significance and were common in the two groups, evidence of an effect from such a brief intervention warrants attention. This study was the first to investigate the effects of a brief mindfulness intervention on PPT and associated changes in the autonomic nervous system. The small sample size of 15 per group may have been underpowered, affecting the ability of the study to detect any statistically significant differences in nociception and autonomic indices. The study was powered for changes in VAS as the nociceptive and ANS outcome measures have not been previously investigated following a brief mindfulness intervention. Previous studies involving brief mindfulness strategies used sample groups ranging from 55 to 219 participants and reported significant findings [88, 92, 93], suggesting that a larger number of participants may have been required to detect changes in the PPT and ANS variables. The trends in effect observed in PPTs and self-reported pain suggest the impact of brief mindfulness interventions is worthy of further investigation.

As people with chronic pain were likely to find some aspects of an 8-week MBSR intervention challenging, this study explored the effects of a brief 10-minute mindfulness body scan intervention. The brevity of the intervention, using only one mindfulness technique (the body scan), may have precluded any significant effects on the autonomic and nociceptive systems from arising. Effects on the nociceptive and autonomic nervous system may occur as people become more skilled in using mindfulness. Indeed, studies of mindfulness meditation of 3 days [94] to 10 weeks [142, 143] have demonstrated and maintained improvement in perceptions of pain. As this was the first study to explore effects of a 10-minute mindfulness intervention on the nociceptive and autonomic nervous systems in people with chronic pain, further investigation of these outcomes is required in order to quantify the effects of a full MBSR programme. It was noted that, even when using a brief mindfulness intervention, physically some people were in so much pain they could not sit still for any length of time without discomfort. In the context of pain, the needs of this group were unique and had to be adapted for. Certain arm and leg positions had to be
adjusted for comfort due to conditions such as inflammatory arthritis, neuropathy or complex regional pain syndrome. This made the process of the participant perceiving and accurately recording their current level of pain challenging.

To increase generalisability of the findings, participants with a range of chronic pain conditions were recruited for this study. There were six different pain conditions, including musculoskeletal and neurological conditions. Differences in the nature and presentation of these conditions may have influenced the results. For example, one participant who presented with Charcot-Marie Tooth Disease, an extremely sensitive demyelinating neuropathy, responded very differently to a person with a mild form of osteoarthritis. Indeed, for people with complex pain conditions, the physical act of mindfulness may have no impact or even cause negative effects [144]. The idea of mindfulness meditation is to become highly aware of the present moment, acknowledging and accepting feelings and sensations without getting caught up in their meaning or catastrophising them. Firstly, for people who might not even be able to sit comfortably, the act of directing focused attention may be too difficult when in chronic pain [144]. Secondly, for some people with chronic pain, mindfulness or focused attention is like shining a light on exactly what is wrong with them. The directed attention may make the experience of pain worse, heighten stress responses and lead to negative mood. These negative effects have been shown in a previous study involving mindfulness and cancer patients [144] but are generally the exception to the majority of participants’ responses. For the most part, mindfulness-based interventions of longer duration have been shown to support physical and social functioning [46], reduce pain-related behaviours [82] and improve and maintain chronic pain [84]. The effects of a brief mindfulness body scan meditation on nociception and the ANS observed across the conditions in this study had no effect but may be worth investigating in other conditions. It may be the case that mindfulness is more effective in some people and/or conditions than others. Unfortunately, due to the sample size in this study, this was not able to be explored, but exploration of the nature of responders and non-responders to mindfulness warrants investigation too.

Use of the mindfulness approach has often been criticised for just being another relaxation strategy. However, advocates of the approach suggest that mindfulness works based on a number of mechanisms such as helping people to focus their attention, becoming more aware of their physical body, regulating their emotions and helping people to be more accepting of themselves rather than self-critical [145]. To control for the relaxation response, the control group were offered an intervention that had a relaxation effect to show if mindfulness had a greater effect than just a relaxation response. The findings of this study show that as there was no statistically significant difference between the two interventions of 10 minutes, and as both groups demonstrated weak trends of reduced self-reported pain and pain sensitivity,
a relaxation response was evident in both groups. In Chiesa et al's [1] systematic review of mindfulness-based interventions for chronic pain, when mindfulness interventions are compared to active controls that exclude the active ingredient of mindfulness meditation, they usually show no significant advantage for the reduction of perceived pain. Indeed, other interventions revealing an effect of brief mindfulness used a distraction control group [92, 94]. The inclusion of a relaxation, usual care (non-treatment), or distraction control group may be helpful to explore this in more depth in future research.

In addition, it is likely that participants may have been nervous or a little anxious about the research initially. After sitting still in a quiet room for 25 minutes, participants may have become acclimatised to the environment and more familiar and at ease with the researcher. As participants were likely to feel more relaxed, this may have influenced their pain ratings. The 15-minute rest period following the intervention was planned to assess for any carry-over effect from the intervention. All participants were novices to mindfulness meditation and therefore it was important to assess for carry-over effect of immediate skills learned (e.g. breathing deeply and sensorial awareness) into the period following the intervention. The study showed no statistically significant evidence of carry-over effects; however, an initial task not related to the study procedures could be used to help people feel more comfortable in the study environment before undertaking the interventions to help reduce any study evoked relaxation effects in the future.

Both central and autonomic nervous system responses, including the electrodermal system, may be used as indicators for specific pain-related cognitive and emotional processing in people with various kinds of pain [146]. There was a significant effect of time on SCR, likely due to a large spike in SCR during epoch 3. When one is presented with a stimulus with a possible significant consequence, SCR increases are likely to occur in anticipation of that outcome. This is facilitated by descending cortical outputs to the ANS that mediate sympathetic SCR responses in anticipation of negative events [139]. Epoch 3 indicated the termination of the intervention and initiation of another assessment of the participant's pain. It is speculated this meant returning to the real-world environment from an altered state, not being distracted from the pain anymore if that were the case, drawing attention to the pain, and then having the researcher induce a nociceptive response with an algometer. The latter would induce both a sensory and affective pain response, which would most likely be the reason for the SCR spike at epoch 3.

The findings on self-reported pain may also have been influenced by the outcome measures used. Even though results indicated a steep decrease in VAS scores for both intervention groups, there are inherent errors and controversies with the use of VAS. VAS was chosen as an outcome measure for this study because it requires little
training to administer and score, and has been found to be adaptable to a broad range of populations and settings [132]. Research suggests though that people can find it difficult to judge how to rate their pain on the VAS line [147]. DeLoach et al [148] found that a single VAS measurement has an imprecision of 20mm when measuring acute pain and suggested that clinically significant changes in pain sensation would require a change in VAS score of similar magnitude. This should be considered when interpreting changes in VAS scores in clinical practice. A recent study [149] also highlighted the lack of sensitivity of the VAS pain scale to distinguish between groups of people with different pain levels and/or pain conditions. It is known that the margin of error increases non-linearly in VAS scores with an increase in severity of pain [134], while the scale remains more linear with mild to moderate pain. With the vast degree in difference of pain conditions in this study, VAS scores would most likely have changed non-linearly in this fashion. Pain is a difficult construct to measure due to its subjectivity and because it is comprised of both sensory and affective components.

Results of this study showed that PCS scores were significantly associated with VAS scores and therefore how people felt about their pain. Higher levels of pain catastrophising is known to influence the intensity of pain in people with chronic pain [38]. In this study, every PCS point increased VAS by almost a millimetre. The participants were a varied group of complex chronic pain syndromes with almost half the sample scoring above the 50th percentile on the pain catastrophising scale. Previous brief mindfulness studies had all used healthy students [92-94] and did not include cognitive evaluations of how the participants felt about their condition. Many people applied to this study looking for hope of long term resolution and may have responded differently to the interventions compared to healthy people. Including healthy participants as a control might also be a consideration for future research.

Gender was found to significantly affect PPT ratings. It is speculated that the possibilities for higher PPT ratings of males included, but were not limited to: the nature of their condition being less severe to them psychologically (low PCS score); possessing a less sensitive condition than other participants, e.g. musculoskeletal versus demyelinating neuropathy; or socially desirable responding when providing pain responses to the researcher. Socially desirable responding is the tendency for participants to present a favourable image of themselves on questionnaires, which can obscure results [150]. It is possible some of these males did not want to present themselves in a light of suffering and therefore offered scores that were higher than what they may truly have been. Social desirability reporting therefore needs to be taken into account when exploring the effects of mindfulness.

While PPT measurement has demonstrated reliability and validity as a diagnostic tool [104], it may also possess inherent errors as an outcome measure for people with
chronic pain. Ohrbach et al [151] proposed that examiner expectancy could affect PPT results. Firstly, a researcher may unintentionally and unconsciously manipulate the rate of pressure application of the PPT device, causing an increase or decrease in PPT. Secondly, if the researcher is expecting a certain value for a painful or non-painful site, he or she may increase or decrease the rate of pressure application based on this expectancy, especially if the participant has not given the signal to stop the pressure. For example, at a painful site, if the researcher is not watching for facial cues of pain and the participant has forgotten to signal stop, then the researcher may slow down the rate of pressure application because they believe to be reaching a PPT ceiling. Whereas at the non-painful site, the researcher may be more cavalier with the rate of pressure because they are less concerned with hurting the person. Both scenarios could potentially skew results. Despite these potential errors, there are still reliable differences in previous studies between PPT recordings of painful and control sites [151] and this has been demonstrated in this study with non-painful PPT site measurements being significantly higher than painful PPT site measurements.

Another potential PPT error is a reduced reading due to temporal summation. Temporal summation is defined as a progressive increase in pain perception caused by a sequence of somatosensory stimuli of the same intensity [107]. When applying pressure with an algometer at the same painful site twice in a row, a person with central sensitisation might exhibit a lower PPT on the second application of pressure due to the facilitated temporal summation of pain. This effect was accommodated for in this study by alternating PPT recordings between the painful site and the non-painful site when averaging two recordings.

Normalised spectral HRV measures were used in this study because the normalisation process expresses values on a more easily understood proportion (0-1), or percentage scale basis, and removes most of the large within and between-subject variability in the total raw HRV spectral power [137]. Furthermore, normalised HF was selected over normalised LF as an indicator of intervention effect on ANS activity because the higher frequency spectral bands are more indicative of parasympathetic activity only [114, 137]. This is because there is controversy surrounding the make-up of low frequency spectral bands with some studies considering LF to reflect both sympathetic and vagal activity [119]. Therefore, even though LF was recorded and analysed, it was not presented in the results of this study.

It is also common for research papers to present nuHF and the LF/HF ratio conjointly [137], with the former as an index of parasympathetic activity and the latter an index of sympathovagal balance between the two autonomic branches, implying that they are two independent variables each with distinct physiologic interpretation. However,
as Burr [137] points out, normalised spectral HRV indices nuLF and nuHF are each linked to the LF/HF ratio with simple reversible 1-to-1 mappings, i.e. nuLF and nuHF are each exactly predictable from the LF/HF ratio and vice versa. This is another reason why nuHF was chosen in this study over nuLF. The LF/HF power ratio was analysed and presented but produced no statistically significant differences between the groups.

Results for nuHF showed no statistical difference between the groups listening to either intervention tape. The weak trend observed though was that the experimental mindfulness group showed reduced parasympathetic activity, which is in contrast to a previous study [114] looking at meditation and nuHF. That study involved 5 days of integrative body-mind training with healthy people however, which is a significantly longer period of time than a 10-minute body scan meditation. No other brief mindfulness studies to date [88, 92-94] have looked specifically at autonomic activity as it relates to chronic pain therefore it is difficult to draw any comparisons. Ditto et al [89] has been the only one to look at the effects of a brief mindfulness body scan meditation on the ANS to date but, again, used healthy participants. Their results were also mixed because a decrease in respiration rate shifts parasympathetically mediated RSA to lower frequencies by increasing cardiac sympathetic activity. In our study, the body scan meditation intervention involved a slowing of respiration rate, which might explain the reduction in parasympathetic activity for the experimental group if meditation is known to increase cardiac sympathetic activity. A slowing of respiration rate would reduce mean HR and this was a weak trend observed in this study across all time epochs for the experimental group, consistent with Ditto et al’s [89] findings for a brief body scan meditation. A trend of decreased parasympathetic activity echoes Ditto et al’s [89] sentiment that meditation can involve active, arousal promoting processes; however, meditation alone cannot account for the reduction in parasympathetic activity for people with chronic pain. Cortical and psychological influences through midline structures such as the ACC can influence the ANS [112], as was observed in the third SCR epoch. The affective component of physically being in pain, and drawing attention to that pain, will most likely modulate the ANS to engage both sympathetic and parasympathetic activity. HRV frequency results of a complex pain group would be more challenging to interpret compared to a group of healthy individuals due to the complex outputs of the central and autonomic nervous systems.

Older age was shown to reduce mean HR, increase nuHF, decrease LF/HF, and decrease SCR. The median age of both sample groups was 60 years. Aging effects the plasticity of the ANS including loss of neurons, loss of axonal branches and alterations in neurotransmitter release [152, 153]. Cell loss has been demonstrated in the interomediolateral cell column of the spinal cord of the elderly, resulting in reduced density of sympathetic nerve endings [153]. It has also been suggested that
there is age-related reduction in sympathetic $\beta$-adrenergic drive to the heart, together with a decrease in $\beta$-adrenergic sensitivity in older people [152]. In fact, many cardiovascular reflexes show altered responsiveness with aging, including respiratory sinus arrhythmia and vagal baroreflexes [153]. This would most likely result in a reduction of HR and elevation of parasympathetic activity.
Chapter 6: Conclusion

The purpose of this study was to measure the immediate effects of a brief mindfulness body scan meditation on self-reported pain, the nociceptive system and autonomic nervous system. In people with chronic pain, a brief mindfulness body scan meditation has no effect on rating of pain severity on a visual analogue scale, pressure pain thresholds, mean heart rate, heart rate variability, heart rate variability low frequency to high frequency power ratio, or skin conductance when compared to a control group. Further research is required before determining whether brief mindfulness interventions are helpful in people experiencing chronic pain.

Previous evidence suggests that mindfulness may positively influence the experience of pain but results of this study suggest that individuals may need to become more skilled in mindfulness techniques over longer periods of time in order for mindfulness to potentially have greater, longer-lasting effects.

Brief mindfulness has been shown to have an effect on pain ratings and the autonomic nervous system of healthy people; however, its effect on complex pain conditions did not present with similar findings in this study. Parameters such as the nature of the pain condition, length of the intervention, sample size, outcome measures used, carry-over effect, environment, positioning, age, gender, social desirability and pain catastrophising are all complex factors that have contributed to the results of this study.
Chapter 7: Limitations

The following limitations apply to this study:

A. The sample size may have been underpowered to detect statistically significant differences in the ANS outcome measures. No previous brief mindfulness studies have thus far investigated the nociceptive and autonomic nervous system together.

B. Differences in the nature and presentation of the complex pain conditions in this study may have influenced results. Mindfulness meditation may be more effective in some people and/or conditions than others.

C. The ANS outcome measures were not assessed prior to the interventions. A longer baseline period would ensure that participants were suitably comfortable with the study environment and baseline recordings could be obtained for the ANS outcome measures.

D. Age was shown to influence the ANS outcome measures and may have influenced the efficacy of the interventions.
Chapter 8: Further research

A number of areas of further research have been identified during the course of this study which include, but are not limited to:

A. Determining the effect of an 8-week MBSR programme on the autonomic and nociceptive nervous systems.
B. Performing a randomised control trial of brief mindfulness on people with chronic pain; on sensory and affective self-reported pain; using both a relaxation and usual care control group.
C. Exploring the influence of age on the autonomic nervous system and how this may influence the response to pain treatments.
References


Appendix A: Ethical approval

10 December 2013

Gwyn Lewis
Faculty of Health and Environmental Sciences

Dear Gwyn

Re Ethics Application: 13/354 Immediate effects of mindfulness body scan meditation on the nociceptive and autonomic nervous systems.

Thank you for providing evidence as requested, which satisfies the points raised. I am pleased to confirm that the Chair and I have approved your ethics application for three years until 8 December 2016.

As part of the ethics approval process, you are required to submit the following to AUTEC:

- A brief annual progress report using form EA2, which is available online through http://www.aut.ac.nz/researchethics. When necessary this form may also be used to request an extension of the approval at least one month prior to its expiry on 8 December 2016;
- A brief report on the status of the project using form EA3, which is available online through http://www.aut.ac.nz/researchethics. This report is to be submitted either when the approval expires on 8 December 2016 or on completion of the project.

It is a condition of approval that AUTEC is notified of any adverse events or if the research does not commence. AUTEC approval needs to be sought for any alteration to the research, including any alteration of or addition to any documents that are provided to participants. You are responsible for ensuring that research undertaken under this approval occurs within the parameters outlined in the approved application.

AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to obtain this. If your research is undertaken within a jurisdiction outside New Zealand, you will need to make the arrangements necessary to meet the legal and ethical requirements that apply there.

To enable us to provide you with efficient service, please use the application number and study title in all correspondence with us. If you have any enquiries about this application, or anything else, please do contact us at ethics@aut.ac.nz.

All the very best with your research,

Kate O’Connor
Executive Secretary
Auckland University of Technology Ethics Committee

Cc: Neil Bossenger neil@spinewave.co.nz
Appendix B: Pain Catastrophising Scale

Mindfulness Data Collection

ID

Date

Pain Catastrophising Scale (PCS)

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery. We are interested in the types of thoughts and feelings that you have when you are in pain.

Listed below are 13 statements describing different thoughts and feelings that may be associated with pain. Using the following scale, indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

<table>
<thead>
<tr>
<th>Rating</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Not at all</td>
</tr>
<tr>
<td>1</td>
<td>To a slight degree</td>
</tr>
<tr>
<td>2</td>
<td>To a moderate degree</td>
</tr>
<tr>
<td>3</td>
<td>To a great degree</td>
</tr>
<tr>
<td>4</td>
<td>All the time</td>
</tr>
</tbody>
</table>

When I’m in pain...

<table>
<thead>
<tr>
<th>Number</th>
<th>Statement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I worry all the time about whether the pain will end.</td>
</tr>
<tr>
<td>2</td>
<td>I feel I can’t go on.</td>
</tr>
<tr>
<td>3</td>
<td>It’s terrible and I think it’s never going to get any better.</td>
</tr>
<tr>
<td>4</td>
<td>It’s awful and I feel that it overwhelms me.</td>
</tr>
<tr>
<td>5</td>
<td>I feel I can’t stand it anymore.</td>
</tr>
<tr>
<td>6</td>
<td>I become afraid that the pain will get worse.</td>
</tr>
<tr>
<td>7</td>
<td>I keep thinking of other painful events.</td>
</tr>
<tr>
<td>8</td>
<td>I anxiously want the pain to go away.</td>
</tr>
<tr>
<td>9</td>
<td>I can’t seem to keep it out of my mind.</td>
</tr>
<tr>
<td>10</td>
<td>I keep thinking about how much it hurts.</td>
</tr>
<tr>
<td>11</td>
<td>I keep thinking about how badly I want the pain to stop.</td>
</tr>
<tr>
<td>12</td>
<td>There’s nothing I can do to reduce the intensity of the pain.</td>
</tr>
<tr>
<td>13</td>
<td>I wonder whether something serious may happen.</td>
</tr>
</tbody>
</table>

Reference:

Appendix C: Short-Form McGill Pain Questionnaire II

Mindfulness Data Collection

ID

Date

Short-Form McGill Pain Questionnaire II (SFMPQII)

This questionnaire provides you with a list of words that describe some of the different qualities of pain and related symptoms. Please put an X through the numbers that best describe the intensity of each of the pain and related symptoms you felt during the past week. Use 0 if the word does not describe your pain or related symptoms.

<table>
<thead>
<tr>
<th>1. Throbbing pain</th>
<th>none</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>worst possible</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Shooting pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>3. Stabbing pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>4. Sharp pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>5. Cramping pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>6. Gnawing pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>7. Hot-burning pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>8. Aching pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>9. Heavy pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>10. Tender</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>11. Splitting pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>12. Tiring-exhausting</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>13. Sickening</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>14. Fearful</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>15. Punishing-cruel</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>16. Electric-shock pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>17. Cold-freezing pain</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>18. Piercing</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>19. Pain caused by touch</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>20. Itching</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>21. Tingling or “pins &amp; needles”</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
<tr>
<td>22. Numbness</td>
<td>none</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>10</td>
<td>worst possible</td>
</tr>
</tbody>
</table>

Reference:

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Dr Neil Bossenger • 0296388
Appendix D: Advertisement

AUT University is conducting a research study investigating the effects of a brief mindfulness strategy on chronic pain and the nervous system. Mindfulness has been found to improve chronic pain. The research team is aiming to test out whether a brief form of mindfulness may have similar benefits and also to see how mindfulness affects the nervous system.

If you have been diagnosed with a chronic pain condition, such as fibromyalgia, complex regional pain syndrome, neuralgia, general longstanding chronic pain like back, neck, arm or leg pain, and have experienced pain for at least 3 months, you may be eligible to participate in this study.

If you are interested and wish to obtain more information, please contact:

Dr Neil Bossenger 09 522 0025 or email neil@spinewave.co.nz
www.spinewave.co.nz
Appendix E: Participant information sheet

Participant Information Sheet

Study title: The effects of a mindfulness body scan on chronic pain and the nervous system.

Locality: SpineWave Wellness Centre
1/102 Remuera Road, Auckland
Ethics committee ref.: 13/NTA/105

Lead investigator: Dr Neil Bossenger
Contact phone number: 021 239 7623

You are invited to take part in a study on the effects of a mindfulness body scan on chronic pain and the nervous system. Mindfulness is about bringing awareness to the present moment without judgment. One session of mindfulness is shown to alter the nervous system and reduce pain related distress.

Whether or not you take part is your choice. If you don’t want to take part, you don’t have to give a reason, and it won’t affect the care you receive. If you do want to take part now, but change your mind later, you can pull out of the study at any time.

This Participant Information Sheet will help you decide if you’d like to take part. It sets out why we are doing the study, what your participation would involve, what the benefits and risks to you might be, and what would happen after the study ends. We will go through this information with you and answer any questions you may have. We expect this will take about 10 minutes. You may also want to talk about the study with other people, such as family, whānau, friends, or healthcare providers. Feel free to do this.

If you agree to take part in this study, you will be asked to sign the Consent Form on the last page of this document. You will be given a copy of both the Participant Information Sheet and the Consent Form to keep.

This document is 5 pages long, including the Consent Form. Please make sure you have all the pages.

Please note: only adults aged over 18 years will be recruited into the study.

Why are we doing the study?

An estimated one in six people suffer from chronic pain in NZ and nearly half of patients with chronic pain report their pain as not under control. It causes changes in the nervous system that often worsen over time and can give rise to significant psychological problems.

The purpose of this study is to see the effects that a brief mindfulness strategy has on people with chronic pain after a single session. The intervention will involve increasing your awareness to your body and feelings without judgment. The aim is that by increasing awareness of pain, people may feel more able to manage their symptoms.
Previous studies involving mindfulness in people with long-term conditions have shown improvements in quality of life over time. However, there is limited knowledge regarding pain reduction, especially after only a single session. This is what we aim to look at whilst also recording what is happening in the nervous system.

Participants in the study will be randomised into one of two groups. One group will undergo an audio recorded mindfulness body scan and the other group will listen to a 10-minute relaxation audio recording.

You will have equal chance of being in either group because we need to have a comparison group to determine the effects of the real mindfulness body scan on the nervous system. If you are randomised to the relaxation group, you will be offered the opportunity to undergo the real mindfulness body scan at the completion of the study if it is shown to be beneficial.

The study has been funded by AUT University and will be undertaken by researchers from the university. If you have any questions about the study you can contact the lead investigator, Dr Neil Bossenger on 09 522 0025 or neil@spinewave.co.nz

The study has received ethics approval from the Health and Disability Committee and the AUT Ethics Committee. Ethical approval was obtained on 22nd August 2013 and data collection is now underway.

What would your participation involve?

All participants will be required to attend a single session at Spinewave Wellness Centre in Remuera. Each session, including preparation and recordings after the intervention, should last no more than 2 hours. The actual treatment part of the session will last no more than 10 minutes.

At the session you will be required to complete questionnaires regarding your pain. The process will then involve gently placing electrodes on your skin to make recordings of your nervous system during the audio recordings.

We will also be looking at the effects of mindfulness on the pain system. This will involve gently pushing a metal probe into an arm muscle and a leg muscle until you report too much discomfort.

We expect to complete the study by June 2015. Participation in the study will not influence any current or future treatment of your pain.

What are the possible benefits and risks to you of participating?

The main benefit of the study is that participants who are randomised to receive the real intervention may have their pain reduced and also learn a new technique to manage their pain in the future.
Mindfulness is not invasive and not harmful. During the session, gel electrodes will be placed on your skin to make recordings of the nervous system. In a rare event this may create some skin irritation which is easily prevented by washing the area.

To assess your pain levels, a probe will gently be applied to an arm muscle and a leg muscle. The probe will be applied to your muscles until you experience pain and then immediately stopped.

You will be able to stop any of these procedures at any time.

For participating in this study, you will be given a $20 of petrol voucher to cover your time and travel costs.

**What would happen if you were injured in the study?**

If you were injured in this study, which is unlikely, you would be eligible for compensation from ACC just as you would be if you were injured in an accident at work or at home.

If you have private health or life insurance, you may wish to check with your insurer that taking part in this study won’t affect your cover.

**What are the rights of participants in the study?**

Participation in this study is voluntary. You are free to decline to participate, or to withdraw from the study at any time, without experiencing any disadvantage. You have the right to access information about you collected as part of the study. You will be told of any new information about adverse or beneficial effects related to the study that becomes available during the study that may have an impact on your health.

If you participate in the study you will be assigned a code. All information relating to you will be identified by your code and not your name. No one will be able to identify your individual results in the study data.

**What will happen after the study ends, or if you pull out?**

At the end of the study, we will not give you any further treatment if you received the real intervention. If the study shows that a mindfulness body scan is beneficial, you will be offered a session of mindfulness at AUT University if you were in the relaxation group. All written and electronic information obtained from the study will be stored in locked filing cabinets or password protected computers. The information will be stored for 10 years from age 16 years in accordance with Health (Retention of Health Information) Regulations 1996 and then shredded or deleted from the computers. If you elect on the Consent Form to receive a summary of the study findings, a 1-page report will be posted or emailed to you on completion on the study (by June 2015).

**Where can you go for more information about the study, or to raise concerns or complaints?**
If you have any questions, concerns or complaints about the study at any stage, you can contact:

Dr Gwyn Lewis, Principal Investigator
Phone: 09 921 9999 x7621
Email: gwyn.lewis@aut.ac.nz

If you want to talk to someone who isn’t involved with the study, you can contact an independent health and disability advocate on:

Phone: 0800 555 050
Fax: 0800 2 SUPPORT (0800 2787 7678)
Email: advocacy@hdc.org.nz

You can also contact the health and disability ethics committee (HDEC) that approved this study on:

Phone: 0800 4 ETHICS
Email: hdecs@moh.govt.nz
Appendix F: Consent form

Consent Form

Declaration by participant:

I have read, or have had read to me in my first language, and I understand the Participant Information Sheet. I have had the opportunity to ask questions and I am satisfied with the answers I have received.

I freely agree to participate in this study.

I have been given a copy of the Participant Information Sheet and Consent Form to keep.

Participant’s name: ____________________________________________

Signature: ___________________________ Date: ____________________

Declaration by member of research team:

I have given a verbal explanation of the research project to the participant, and have answered the participant’s questions about it.

I believe that the participant understands the study and has given informed consent to participate.

Researcher’s name: ____________________________________________

Signature: ___________________________ Date: ____________________