Behavioural activation and inhibition systems in relation to pain intensity and duration in a large chronic musculoskeletal pain sample

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Declaration

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This thesis entitled “Behavioural activation and inhibition systems in relation to pain intensity and duration in a large chronic musculoskeletal pain sample” is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy

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Candidate’s declaration:

I confirm that:

- This thesis represents my own work;
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: UREC 2016-1069

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Abstract

Background: Pain related complaints form one of the most common, and most costly, presentations of patients in healthcare in New Zealand and worldwide. Theories of pain such as the biopsychosocial and fear-avoidance models aim to provide a multidimensional framework from which pain can be approached, considering various aspects of how pain affects people. The most effective pain management approaches seem to be multimodal and patient-specific. The Reinforcement Sensitivity Theory proposes two neurophysiological systems that regulate impulsivity and anxiety in human behaviour: the behavioural activation system and the behavioural inhibition system. It has recently been suggested that sub-grouping individuals affected by pain based on their levels of activation and inhibition could facilitate the allocation of more effective management strategies.

Design: Cross-sectional survey design.

Aim: To establish which of the following best predicts average intensity and duration of musculoskeletal pain: fear avoidance beliefs, kinesiophobia, and levels of behavioural activation or inhibition system.

Methods: Surveys were made available online to adults in New Zealand with pain complaints, and to patients at the Unitec Osteopathic Clinic, Clinic 41. Data were gathered over a three-month period and analysed using Spearman’s rho correlations, linear regressions and a between groups analysis assessing for differences between high and low intensity pain and levels of behavioural activation or inhibition.

Results: Correlational analyses showed significant positive relationships between pain intensity and fear-avoidance beliefs, kinesiophobia, and disability, as well as between pain duration and fear-avoidance, kinesiophobia and perceived disability. Regression analyses showed fear-avoidance beliefs, kinesiophobia and disability accounted for 31% of pain intensity variance. Disability alone accounted for 5% of the pain duration variance. Neither
behavioural activation nor inhibition systems significantly related to or predicted pain intensity or duration.

**Conclusion:** This study provides further support for the inter-relationships between fear-avoidance beliefs, kinesiophobia, disability and pain duration and intensity. The results do not show explicit support for the behavioural activation or inhibition systems relating to pain intensity or duration. It is suggested that this may be due to the measurement instrument, which could be explored in further studies.
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# Table of Contents

Nina Sanson ......................................................................................................................................... i

Abstract ............................................................................................................................................... iii

Acknowledgements .............................................................................................................................. v

Table of Contents ................................................................................................................................ vi

Section 1: Literature Review .............................................................................................................. 1

Pain ....................................................................................................................................................... 2

Mechanisms of Pain ........................................................................................................................... 2

  Sensitisation and Plasticity .................................................................................................................. 3

Classification of Pain States ............................................................................................................... 4

Burden of Pain ................................................................................................................................... 8

Models of Pain .................................................................................................................................. 11

Interventions: Managing Pain .......................................................................................................... 16

  Pharmaceutical pain management ..................................................................................................... 16

  Manual therapy for pain management ........................................................................................... 18

  Psychology .................................................................................................................................... 20

Pain and Behaviour ........................................................................................................................... 25

  BIS-BAS Model of Chronic Pain .................................................................................................... 27

  Previous Studies ............................................................................................................................... 29

Conclusion .......................................................................................................................................... 32

References .......................................................................................................................................... 33
<table>
<thead>
<tr>
<th>Section</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Section 2: Manuscript</td>
<td>1</td>
</tr>
<tr>
<td>Abstract</td>
<td>3</td>
</tr>
<tr>
<td>Introduction</td>
<td>4</td>
</tr>
<tr>
<td>Methods</td>
<td>8</td>
</tr>
<tr>
<td>Measures</td>
<td>8</td>
</tr>
<tr>
<td>Data Collection</td>
<td>8</td>
</tr>
<tr>
<td>Data reduction</td>
<td>11</td>
</tr>
<tr>
<td>Statistical Analysis</td>
<td>12</td>
</tr>
<tr>
<td>Results</td>
<td>14</td>
</tr>
<tr>
<td>Discussion</td>
<td>21</td>
</tr>
<tr>
<td>BIS and BAS</td>
<td>21</td>
</tr>
<tr>
<td>Intensity Regression</td>
<td>23</td>
</tr>
<tr>
<td>Duration Regression</td>
<td>24</td>
</tr>
<tr>
<td>Correlations</td>
<td>25</td>
</tr>
<tr>
<td>Comparison of current study with previous literature</td>
<td>26</td>
</tr>
<tr>
<td>Limitations</td>
<td>31</td>
</tr>
<tr>
<td>Future Research</td>
<td>32</td>
</tr>
<tr>
<td>Conclusions</td>
<td>32</td>
</tr>
<tr>
<td>References</td>
<td>34</td>
</tr>
<tr>
<td>Supplementary</td>
<td>55</td>
</tr>
<tr>
<td>The Survey</td>
<td>55</td>
</tr>
<tr>
<td>Ethical Approval Letter</td>
<td>60</td>
</tr>
</tbody>
</table>
Section 1: Literature Review
Literature Review

The aim of this review of the literature is to provide a rationale for investigating the predictive value of behavioural tendencies on pain intensity and duration, and the relationship between these variables. This literature review will explore (1) pain mechanisms and models, (2) literature assessing current interventions for pain management, and (3) what is currently known about the relationship between activation and inhibition behavioural tendencies in relation to chronic pain.

Pain

Mechanisms of Pain

Pain is a complex phenomenon involving an array of neurological processes, both in the peripheral and central nervous systems. Threatening, or noxious, stimuli are conveyed to the brain via nociceptive neurons indicating tissue damage (Dubin & Patapoutian, 2010). Nociceptive pain is generated by mechanical or thermal stimulation of nociceptive nerve fibres in the periphery and is conducted to the central nervous system by two main fibre types: unmyelinated C-fibres, and myelinated Aδ-fibres (Das, 2015; Dubin & Patapoutian, 2010). These fibres transmit the noxious stimulus to the dorsal horn of the spinal cord, and to the cerebral cortex via the spinothalamic tract (Das, 2015; Gebhart et al., 2009). Pain is the perception that is subsequently generated by several cortical structures of the brain in response to this nociceptive input. These structures include the prefrontal, anterior cingulate, somatosensory and supplementary motor cortex, insula, amygdala, posterior cingulate and posterior parietal cortex (Moskowitz & Golden, 2013). The brain then
responds with up- or down-regulation processes, which may highlight or dampen nociceptive signals from the spinal cord (Moskowitz & Golden, 2013). This process of initial pain experience is essential to survival as it allows us to form a decision around how to react to the threatening stimulus (Dubin & Patapoutian, 2010; Siddall & Cousins, 1997).

**Sensitisation and Plasticity**

In addition to supra-threshold stimuli causing pain, pain can also be caused or exacerbated by inflammatory mediators secondary to tissue injury (Ji, Xu, & Gao, 2014; Reichling & Levine, 2010). Inflammatory mediators, chiefly pro-inflammatory cytokines, can alter the responsiveness of peripheral nociceptors. That is, nociceptors can become more sensitive meaning non-threatening stimuli can be received as pain. The mechanism by which this occurs is a lowering of the nociceptor’s activation threshold (Reichling & Levine, 2010) which is commonly referred to as peripheral sensitisation (Siddall & Cousins, 1997). A state of hyperalgesic priming, in which a body area remains overly sensitive to inflammatory mediators for weeks after the initial insult (Reichling & Levine, 2010), may also occur.

Similarly, prolonged nociceptive input to the central nervous system can ‘wind-up’ nociceptive pathways (Latremoliere & Woolf, 2009). The increased activity of peripheral nociceptive neurons associated with peripheral inflammatory mediators has been suggested to contribute to an increased sensitivity of central neurons. This is referred to as central sensitisation and occurs through a subsequent rise in the release of neurotransmitters, neuromodulators and brain-derived neurotrophic factors (Ji et al., 2014). Once in a state of sensitisation, non-painful stimuli are perceived as being painful (allodynia), and stimuli are perceived as being more painful than the stimulus warrants (hyperalgesia) (Ji et al., 2014).
High threshold or long-term stimulation of nociceptive neurons can lead to modulation of neural pain pathways (Ji et al., 2014; Woolf & Salter, 2000). If these modulatory changes occur, excitatory synaptic responses become facilitated and synaptic inhibition is depressed, thereby amplifying responses to both noxious and innocuous inputs. Neuro-modulatory changes are more likely to occur in pain complaints of longer durations. These changes also contribute to the development of allodynia; therefore, chronic pain complaints are often perceived by the patient as being more intense than the amount of tissue damage warrants (Woolf & Salter, 2000).

**Classification of Pain States**

Pain is divided into two main pain types depending on the duration: pain that is of less than three-month duration is classified as acute pain, while pain that lasts longer than three months is classified as chronic, or persistent, pain (Merskey & Bogduk, 1994). For research purposes, chronic pain is often classified as being longer than six months' duration (Merskey & Bogduk, 1994). Subacute and subchronic pain states also exist, with subacute pain referring to a pain duration more than five to seven weeks but not more than 12 (Bogduk, 1999). Subchronic pain may similarly begin at five to seven weeks duration but, in contrast to subacute pain, is defined as lasting longer than 12 and not more than 52 weeks (Fryer, Alvizatos, & Lamaro, 2005).

Acute pain is typically the result of a specific injury or insult to the body. This pain serves to evoke a reaction, such as reflexive avoidance of the painful stimulus and is, therefore, necessary for survival. Although there is no absolute correlation between pain and tissue damage, in acute pain states the pain sensation experienced is generally in proportion to the amount of tissue damage sustained (Eccleston & Crombez, 1999; Gebhart et al., 2009).
Once the nociceptive stimulus is removed and the injury heals, the pain subsides (Grichnik & Ferrante, 1991). As acute pain is more likely to be indicative of tissue damage, the development of avoidance behaviours and fear of movement or re-injury (kinesiophobia) can be helpful in minimising any further damage to the injured area. In chronic pain states, however, these aversive behaviours can become mal-adaptive, affecting physical functioning, e.g. in work, recreation and mood (Zale & Ditre, 2015).

In contrast to acute pain, chronic pain is often suggested to outlast the expected time of healing. Perhaps a key factor in the presentation of chronic pain complaints is that the pain can exist or worsen without any external stimulus at all (Apkarian, Hashmi, & Baliki, 2012). It has been noted in cases of post-traumatic stress disorder that this occurs as a result of central sensitisation (Latremoliere & Woolf, 2009). Apkarian and colleagues' (2012) reviewed functional and structural magnetic resonance imaging studies of chronic pain. Results showed a decrease in grey matter density and some alterations in white matter connectivity. In addition, chronic pain was shown to be elicited in different areas compared to acute pain states: the medial anterior cingulate cortex rather than the anterior cingulate were implicated in chronic pain, and there was an absence of insula activation unless a spike in the intensity of chronic pain was experienced. The authors further discussed how unremitting pain stimuli could contribute to a learning state whereby normal environments become associated with pain. Chronic pain sufferers are then not able to unlearn these environmental pain associations, as the pain does not cease long enough or often enough for them to not reinforce these negative associations. This in turn impacts on the limbic system, which contributes to the emotional response to pain, and the descending modulation as the brain continues to remind the body that this pain is negative and threatening.
This theory can be applied to cases of chronic low back pain. Low back pain is a common complaint in both acute and chronic states (Hoy et al., 2012). In the acute phase it can be self-limiting (Patrick, Emanski, & Knaub, 2014). However, complaints of low back pain often recur and are then classified as chronic conditions (Hoy, Brooks, Blyth, & Buchbinder, 2010). A fMRI study compared neuronal activation responses to pain, as well as subjective pain report, in chronic low back pain sufferers, fibromyalgia sufferers and healthy control subjects (Giesecke et al., 2004). Compared to controls exposed to the same pressure, chronic low back pain and fibromyalgia patients reported higher pain intensity and exhibited more neuronal activation in pain areas in the brain (specifically the contralateral primary and secondary somatosensory cortices, inferior parietal lobule, cerebellum, and ipsilateral secondary somatosensory cortices). This implicates central processes rather than peripheral as the driver of these chronic pain states.

It has also been proposed that there may be a genetic component contributing to individuals susceptibility to developing chronic pain (Hartvigsen et al., 2009; Van Hecke et al., 2017; Vehof, Zavos, Lachance, Hammond, & Williams, 2014). This proposal has particularly featured in discussions of chronic pain syndromes such as fibromyalgia and irritable bowel syndrome (Vehof et al., 2014; Voscopoulos & Lema, 2010). The heritable component of chronic pain conditions is most easily demonstrated in populations of twins (Hartvigsen et al., 2009; Vehof et al., 2014), but has also been shown in generational groups (Van Hecke et al., 2017). Some of the key genes that appear to be common to musculoskeletal pain include catechol-O-methyltransferase (COMT), and beta-2 adrenergic receptor (ADRB2) as part of the catecholaminergic system, as well as serotonin receptor 2A (HTR2A) and serotonin transporter (SLC6A4) as part of the serotonergic system (Zorina-Lichtenwalter, Meloto, Khoury, & Diatchenko, 2016). Also, not specific to musculoskeletal pain, mu
opioid receptor (OPRM1), guanosine triphosphate cyclo-hydrolase (GCH1) and all human lymphocyte antigen (HLA) groups have been implicated in pain genetic research (Mogil, 2012).

Psychosocial factors such as fear-avoidance beliefs, fear of re-injury, catastrophic thoughts and traits of anxiety and neuroticism are also linked with both the development and maintenance of chronic pain states – though it is not clear whether these pre-date the pain complaint, or develop as a result of persistent pain (Voscopoulos & Lema, 2010). The term fear-avoidance encompasses a range of beliefs that centre around a fear of pain and the belief that movement is detrimental (Parr et al., 2013; Gordon Waddell, Newton, Henderson, Somerville, & Main, 1993). Fear-related avoidance of movement in response to pain may be a beneficial adaptation in acute pain states as a self-preservation mechanism, however these beliefs enduring into chronic pain states become mal-adaptive (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Kinesiophobia is a subset of fear-avoidance that is specific to fear of injury, or re-injury – fear of movement itself, as opposed to fear relating to beliefs about movement in general (Parr et al., 2013). Catastrophising can also contribute to prolonging and worsening the pain experience, as it is characterised by over-exaggerating of the negative aspects of pain, and inability to disengage from ruminating on the pain experience (Sturgeon & Zautra, 2013). The presence of these mal-adaptive psychosocial variables has been shown to relate to longer pain duration and more disability (Denison, Åsenlöf, & Lindberg, 2004; Parr et al., 2013; Vranceanu et al., 2014).

The number of pain sites, rather than the specific location of pain, is yet another factor that has been suggested to increase the risk of chronicity (Mallen, Peat, Thomas, Dunn, & Croft, 2007). It has been suggested that considering the primary site of pain (for example, low
back or shoulder) does not lend much predictive ability to prognosis. Rather, the number of pain sites has been found to relate to prognosis, with more sites of pain indicating longer duration (de Vos Andersen, Kent, Hjort, & Christiansen, 2017). Having multiple sites of pain has also been shown to relate to decreased ability to work, a higher number of sick days and an increased intention toward early retirement (Miranda et al., 2010).

**Burden of Pain**

Pain is common to all genders, age ranges and ethnicities. In the section that follows, trends between these determinants and pain will be summarised. There appears to be a consensus that women report more chronic widespread pain (such as fibromyalgia) severe and frequent pain and pain of longer durations as compared to men (Henschke, Kamper, & Maher, 2015; Mansfield, Sim, Jordan, & Jordan, 2016). However, low back pain specifically is more commonly reported in men (Driscoll et al., 2014). The age group most affected by pain is reported to be slightly different in different studies. Some suggest that pain increases throughout adulthood (Fayaz, Croft, Langford, Donaldson, & Jones, 2016), others that there is a peak around 60 years of age, after which the prevalence of pain reaches a plateau (Mansfield et al., 2016). Another suggestion is that there is a spike in the prevalence of pain in middle-aged adults (35 – 55 years) and again in old age (Henschke et al., 2015). As the points of highest pain prevalence are in middle and late adulthood, there has been some concern raised over the ageing population that will see the level of pain-related complaints continue to rise (Fayaz et al., 2016).

Culturally, pain is perceived in varying ways. Western cultures tend to take a biomechanical approach where pain is considered to be the result of a pathological process that can be repaired, thereby resolving pain. If no pathological cause can be identified, the
cause is assumed to be psychological in nature (Ho & Johnson, 2013). Eastern cultures, specifically traditional Chinese medicine, take a more holistic approach. Ailments are thought to be caused by both external pathogenic factors and internal imbalances specific to the individual (Ho & Johnson, 2013). In New Zealand, Māori culture takes a similar stance whereby pain is perceived to affect physical, psychological, social, and spiritual aspects of life (Magnusson & Fennel, 2011). Alongside varying cultural views on pain, there are differences in pain prevalence between different ethnic groups. The largest burden of disability secondary to work-related low back pain was found to be in East Asia, South East Asia, and, on a per capita basis, Oceania (Driscoll et al., 2014). There also may be more chronic widespread pain in a Southeast Asian population than in the European population (Mansfield et al., 2016). There have been reports of higher pain prevalence in American Indians, Alaska Natives, and Aboriginal Canadians compared to a general United States population (Henschke et al., 2015). Additionally, prevalence reports in Africa are slightly higher than those in Western countries (Henschke et al., 2015).

Statistics demonstrating the incidence and prevalence of pain complaints are variable. However, there is no doubt that pain is common, costly and has a negative impact, both on a large scale and on an individual level (Henschke et al., 2015). Reports of new incidences of low back pain, for example, can range from 1.5% to 38.9% over one year, with recurrence at one year ranging from 24% to 80%. Low back pain is one of the more commonly documented and studied pain complaints with the estimated prevalence of low back pain ranging from 24-49.5% (Henschke et al., 2015). Comparably, the prevalence of thoracic pain is estimated to be between 1.4-34.8%, and neck pain between 15.4-45.3%. Prevalence of chronic low back pain is lower than other low back complaints, but still remarkably common between 5.9-18.1%. It is estimated that chronic pain (not specific to
the low back) affects 16% of New Zealand adults (Swain & Johnson, 2016). The high variability in these numbers has been suggested to be due to lack of clarity in reporting regarding definitions of episodes, time periods assessed and prevalence (Henschke et al., 2015).

The financial cost of pain is multifactorial and includes aspects such as the national cost of healthcare, the loss of productivity through work absenteeism and personal cost of healthcare and support work (Henschke et al., 2015; Pearce et al., 2004). Managing chronic pain is one of the highest healthcare costs on a global scale (Henschke et al., 2015). Considering statistics gathered by New Zealand’s Accident Compensation Corporation (ACC), the cost of all pain complaints brought on as a result of injury among New Zealanders was approximately $2.2 billion for 1.7 million new claims and 1.5 million pre-existing claims active in 2016-2017 (ACC, 2017). An Australian report estimated the cost of chronic pain per annum amounted to $10,847 per person (Access Economics, 2007). Another report estimated that older adults (ages 45-64 years) not working due to poor health decreased Australia's gross domestic product by as much as $14.7 billion per annum (Schofield, Shrestha, Passey, Earnest, & Fletcher, 2008). Comparing the cost of pain-related complaints to that of other health complaints, the total cost of chronic pain in the USA in 2010 was between $560 - $635 billion; the cost of heart disease was closer to $309 billion, cancer $243 billion, and diabetes $188 billion (Gaskin & Richard, 2012).

In addition to the financial cost of pain, there is also often a personal cost to consider. Chronic pain can affect a number of psychosocial variables including mental health, physical ability, ability to work and ability to contribute to society (ACC, 2004; Henschke et al., 2015). Common mental health issues associated with chronic pain include
depression, anxiety, low self-efficacy and poor general emotional functioning (Burke, Mathias, & Denson, 2015). A meta-analysis by Burke, Mathias, and Denson (2015) found that while depression is a common psychological problem in adults experiencing chronic pain, they consistently report higher levels of anxiety. This suggests anxiety to be the foremost mental health variable affected by chronic pain. A review by Froud et al. (2014) highlighted some key themes in assessing the personal impact of low back pain. The first theme was that people affected by low back pain were no longer able to carry out all their activities of daily life, such as domestic chores and leisure activities. The second theme detailed the negative impact pain had on the participant's relationships, including familial relationships, sexual relationships and social life, leading to feelings of isolation and being burdensome on others. The third theme was work-related. Not only were there concerns about the number of sick days and fear for the loss of employment, there was also anxiety relating to workplace socialising and concerns regarding finance.

Models of Pain

There are several models conceptualising pain, some of the more prominent models include the biomedical, biopsychosocial, and fear avoidance models of pain.

The biomedical model of pain is one of the older models of pain, being most popular prior to the 1980s (Engel, 1980). It focuses primarily on providing a biological explanation for pain by considering the nociception and physiology that contributes to pain as a sensation that can be measured (Engel, 1980). This pathoanatomical consideration of pain is still widely used and can be clinically convenient as it provides a starting point from which the patient can be further examined. However, it has been argued that this model is reductionist as it fails to encompass the complexity of the pain experience (Bendelow, 2013).
Additionally, when a diagnosis focuses on an injured tissue or structural abnormality, it has been suggested that any negative beliefs the patient holds can be further reinforced (Darlow, 2016). In the case of chronic and idiopathic pain, the argument against use of the biomedical model is that it does not account for psychosocial contributions, such as fear, to an individual’s pain experience (Bendelow, 2013; Leeuw et al., 2007). In response to this, the biomedical model of pain was expanded by Engel (1977) into the biopsychosocial model of pain which includes psychological and social contributors to pain experience (Crombez, Eccleston, Damme, Vlaeyen, & Karoly, 2012).

It is now widely accepted that pain experience is influenced by a multitude of factors ranging from the structure and sensitivity of the nervous system to emotional state and societal norms (Moseley, 2007; Voscopoulos & Lema, 2010). The “bio” element of the biopsychosocial model relates to the physiological processes involved in the perception of pain. These include genetic predisposition to different pain phenotypes (such as pain location and duration) (Hartvigsen et al., 2009), sensitisation processes of peripheral and central neurons, as well as alterations to brain structure in response to persistent pain (Apkarian et al., 2012; Ji et al., 2014).

There are many possible elements that could be considered important within the biopsychosocial model of pain. Some of the more prominent ones include catastrophising, fear-avoidance beliefs, and self-efficacy (Gatchel et al., 2007). Denison, Åsenlöf, and Lindberg (2004) found that high pain intensity and low self-efficacy were both significantly predictive of disability in a sub-acute and chronic musculoskeletal pain population (n=371). They also found that the presence of disability had significant positive relationships with fear-avoidance beliefs of catastrophising and kinesiophobia, and a significant negative
relationship with self-efficacy. Vranceanu et al. (2014) found strong interrelationships between catastrophising, pain anxiety, post-traumatic stress and depression in 136 patients after skeletal trauma. Furthermore, all these variables were also significantly related to pain intensity and disability (Vranceanu et al., 2014).

The biopsychosocial model also incorporates how social connection is important when considering pain and pain management. As an example, positive social interaction, either verbal or non-verbal, has been found to reduce pain, while negative or ambiguous interaction increases pain (Krahé, Springer, Weinman, & Fotopoulou, 2013). Similarly, active and passive support have both been found to reduce the intensity of experimentally induced pain, regardless of whether it is provided by a known or unknown person (Brown, Sheffield, Leary, & Robinson, 2003). Beyond singular interactions, having a social support network has been shown to decrease depression and moderate pain in a longitudinal study of elderly persons (Lee, Kahana, & Kahana, 2016). These demonstrate the important positive effects of having social support and social interaction on pain. Socialisation has recently been found to directly relate to mortality. People with good quality and larger quantities of social interactions demonstrated 50% increased likelihood of survival compare to those lacking in substantial social relationships (Holt-Lunstad, Smith, & Layton, 2010). However, despite socialisation being helpful for reducing pain intensity and an important factor in contributing to length of life, social interactions can also have a negative effect on pain.

Darlow (2016) discusses the effect clinician and community beliefs and behaviours can have on patient’s beliefs and outcomes, specific to low back pain. Clinicians with fear beliefs about pain, or who advise rest and caution in activities are more likely to reinforce
these beliefs in patients. The effect of community beliefs can also be very impactful (Darlow, 2016). Wider community beliefs about low back pain are not constructive, with many believing that the low back is inherently weak, that pain indicates damage and will lead to increased weakness of the injured part, that exercise can be detrimental, and that pain during exercise indicates those movements should be avoided (Darlow et al., 2014; Gross et al., 2006; Ihlebæk & Eriksen, 2003; Munigangaiah, Basavaraju, Jadaan, Devitt, & McCabe, 2016). This indicates a global deficit in informed and constructive beliefs about low back pain, management and prognosis which, in turn, has negative ramifications for those experiencing low back complaints (Darlow, 2016). Negative social influences on pain have also been shown on a more personal level. By assuming a role of ‘carer’, the beliefs of spouses and partners can reinforce fear of reinjury and disability in the pain sufferer (Brooks, McCluskey, King, & Burton, 2013).

Pain can have a significant impact on social factors as well. A systematic review of 42 studies by Froud et al. (2014) considered how pain affected patient’s lives. The themes reported include disruption of domestic and recreational activities, negative impact on relationships, negative impact on work, coping with social stigma, and the need to change their outlook on life to accommodate for the pain that is seemingly not changing. They overlap with the aforementioned psychological variables, as many of them result in the patient having fears about their pain or future with pain, having catastrophising thoughts and depression or feelings of hopelessness.

The fear-avoidance model of pain builds further onto the role that fear can have on pain experience. It centres around the idea that fear of pain, movement and injury, and associated beliefs about pain appear to be associated with chronic musculoskeletal pain
complaints (Lucchetti, Oliveira, Mercante, & Peres, 2012). These mal-adaptive beliefs, for example, fear-avoidance, catastrophising, and kinesiophobia have been suggested to act in a cycle of perpetuating chronic pain (Crombez, Eccleston, Damme, Vlaeyen, & Karoly, 2012; Vlaeyen & Linton, 2000). Fear of pain may lead to avoidance of activity, in turn reinforcing disuse and disability as well as depression, which potentially negatively moderates the pain perception, serving to increase fear of pain (Leeuw et al., 2006). It is not unreasonable to surmise that such cycle could be detrimental to the physical health of patients, and indeed could potentially exacerbate pain (Jensen, Ehde, & Day, 2016). Measuring these factors may be relevant in a chronic pain scenario as addressing fears around movement and (re)injury may help to break the cycle.

Considering patient’s complaints from a biopsychosocial or fear-avoidance perspective allows for a more complete assessment of the pain experience. Acknowledging these different elements of pain, such as patient’s beliefs, coping strategies and socio-economic status, results in a more patient-centred approach to treatment and pain management interventions (Vlaeyen et al., 2007). Some common pain management interventions are discussed below.
Interventions: Managing Pain

Though the prevalence of pain-related complaints appears to be rising, there are a number of methods commonly in use to treat pain. The first and perhaps most common is pharmaceutical management. Secondly, there are several modes of manual therapy that are often used in injury rehabilitation and pain management. Thirdly, psychological techniques such as cognitive therapy and educational approaches, among others, have become popular – particularly in managing chronic pain complaints. In the following, each of these approaches is outlined briefly, and recent research examining the mechanisms and effectiveness of the approaches is summarised.

Pharmaceutical pain management

Pharmaceutical drugs are commonly used in cases of acute, post-operative and persistent pain primarily for symptomatic management (Machado et al., 2015; Machado et al., 2017). There are several approaches utilised to achieve a reduction in pain. Non-opioids, including non-steroidal anti-inflammatories (NSAIDs) and Paracetamol, are often the first line of treatment. These drugs aim to have analgesic, anti-inflammatory and antipyretic effects. Reducing inflammation, and subsequently inflammatory mediators such as cytokines, helps to limit the development of hyperalgesia and allodynia (Gebhart et al., 2009). The next level of pharmaceutical pain management involves opioid drugs. These induce a stronger analgesic effect, however, due to a large number of adverse side effects, are only utilised in more severe cases (Park & Moon, 2010). Antidepressants including selective serotonin re-uptake inhibitors and norepinephrine-serotonin re-uptake inhibitors a more commonly used in chronic or centralised pain scenarios as analgesic effects are targeted at the spinal cord and may take several weeks to occur. A secondary mode of action is the anti-depressive effect that leads to small decreases in pain perception (Park & Moon, 2010).
Antidepressants are commonly used in conjunction with anticonvulsants such as Gabapentin where pain is believed to be of neuropathic origin. Finally, muscle relaxants such as Baclofen are utilised where muscle spasticity is assumed to take a primary role in pain generation (Park & Moon, 2010). A major role of pharmaceutical management of pain is to disrupt the nociceptive pathways. This can help to reduce the risk of developing peripheral and central sensitisation which can contribute to chronic pain complaints (Gebhart et al., 2009).

Although pharmaceutical management has been considered a superior approach, more recent evidence suggests that it may not always be effective (Machado et al., 2015). For example, Paracetamol may not constitute the best course of first-line treatment for non-specific spinal pain complaints. A systematic review and meta-analysis by Machado et al. (2015) provide high quality evidence to suggest that Paracetamol is not effective in reducing pain in those with low back pain complaints (both acute and chronic presentations). Furthermore, regular users of Paracetamol were four times more likely to have abnormal liver function test results compared to those not using Paracetamol, which should be considered in cases of high dose and long-term usage (Machado et al., 2015). Similarly, there is recent research suggesting non-steroidal anti-inflammatory drugs (NSAIDs) are no more effective than placebo in treating acute or chronic spinal pain (Machado et al., 2017). A review by Machado et al. (2017) found that the analgesic effects of NSAIDs were not significantly different from the pain reduction gained by taking only the placebo. Machado et al. (2017) note concerns about adverse events, mainly relating to the cardiovascular safety of using NSAIDs that target cyclooxygenase-2 (COX-2) inhibitors, and common gastrointestinal damage.
The rate of opioid prescription has been rising over the past years (Levy, PauloZZi, Mack, & Jones, 2015; Olsen, Daumit, & Ford, 2006), and subsequently the associated risks are becoming increasingly discussed (Chou et al., 2015). There are specific concerns relating long term use with addiction and death following opioid overdose (Levy et al., 2015), as well as reported adverse effects such as increased reported fractures, myocardial infarctions and endocrinological harm (Chou et al., 2015).

These findings suggest that, although pharmaceutical management of pain is common and may be effective in the short-term, long-term use of any of the aforementioned drugs is not helpful in reducing pain. Patients aiming to treat persistent pain complaints must, therefore, look to alternative avenues to find relief. One such avenue that is becoming more popular is that of manual therapies.

**Manual therapy for pain management**

Physical interventions are often used in the management of pain complaints alongside or as an alternative to pharmaceutical interventions. While these treatment modalities have grown considerably in popularity, they are still commonly termed complimentary treatments (Fredin & Lorås, 2017; Licciardone, Brimhall, & King, 2005). Treatment can include a range of approaches, from exercise prescription and ergonomic adjustments to manual treatment such as osteopathy, chiropractic and physiotherapy (Keeffe et al., 2016; Tsertsvadze et al., 2014). These manual treatments typically involve some mixture of spinal manipulation, joint articulation or mobilisation and soft tissue massage type techniques, amongst others, that are selected based on findings of a physical examination (Tsertsvadze et al., 2014).
A systematic review by Hidalgo, Detrembleur, Hall, Mahaudens, & Nielens (2014) evaluated 23 randomised controlled trials and found general manual therapy and combination treatments to be more beneficial than usual medical care or exercise alone for non-specific low back pain. Specifically, spinal manipulation was more effective compared to sham treatment and resulted in short-term (1-3 month) pain reductions for acute, subacute and chronic pain. Moderate quality evidence suggested that soft tissue techniques and spinal manipulation combined with either exercise or other usual medical care was better for improving pain, function, and quality of life in both the short and long term when compared to exercise interventions alone in cases of chronic pain. Finally, the authors report limited evidence of soft tissue and joint mobilisation techniques combined with usual medical care improving pain and function when compared to usual medical care alone.

Licciardone et al. (2005) reviewed six randomised controlled trials assessing the efficacy of osteopathic manual treatment specifically for low back pain complaints. Osteopathic treatment was found to significantly reduce pain at short and long-term follow-ups (three months) in the United Kingdom and the United States of America, compared to active treatment, placebo control, or no treatment. As with the pharmaceutical interventions, manual therapies for pain management do involve some element of risk. For example, adverse events recorded by Paige et al. (2017) included the manual treatment itself being painful, increases in pain for 1-2 days post-treatment and an increased sense of fatigue and stiffness, though these were all transient and only reported in the minority.

A further study assessing treatment outcomes of physiotherapy for musculoskeletal pain reported statistically and clinically significant benefits (de Vos Andersen et al., 2017). Participants who were more satisfied with their treatment outcomes were likely to be older
or retired persons with less pain intensity and pain for a shorter duration, less disability, less fear-avoidance, persons without compensations claims, non-smokers, and those in better mental health. Many of these are the type of psychosocial variables that have been found to relate to chronicity, disability and depression (Froud et al., 2014). Interestingly, de Vos Andersen et al. (2017) did not find any specific injury or pain site to be related to more intensity or longer duration of pain, which further supports the significant role that psychosocial variables can play in pain experience.

These articles outline that manual treatments can reproducibly reduce pain, at least over a short-term period of up to three months. Manual therapy has some risks associated, as do the pharmaceutical approaches for pain management, however these negative effects are not common and are transient in most cases. Given that the most burdensome form of pain is persistent or chronic pain (Henschke et al., 2015), a long-term solution is still sought after.

**Psychology**

Manual therapy sessions allow for an interplay of symptomatic changes, therapeutic relationship and health and illness beliefs, which may positively influence their efficacy (Bradbury, Bishop, Yardley, & Lewith, 2013). There is considerable evidence to suggest that fear and anxiety regarding pain, in fact, make the pain worse – both more intense and, in some, more debilitating (Denison et al., 2004; Vranceanu et al., 2014). Many cognitive-based pain management strategies are becoming more popular for treating chronic pain conditions, alongside the more traditional approaches (Beck & Dozois, 2011). Some of the more widely known psychological interventions include cognitive behavioural therapy, educational approaches such as Explain Pain, and mindfulness-based interventions.
Cognitive behavioural therapy is an activity-based psychological intervention for pain management first devised by Beck (1963). There is no specific protocol for this treatment; instead, any combination of strategies may be used based on the clinician's judgement (Ehde, Dillworth, & Turner, 2014). Cognitive behavioural activities are typically based on cognitive tasks, such as keeping a daily record of thoughts allowing patients to recognise automatic thoughts and critically assess the validity and helpfulness of those thoughts; or behaviour specific tasks, for example, the patient makes a statement about an activity, then carries out the activity, and finally reassess the initial statement for accuracy (Beck & Dozois, 2011). Other activities can include relaxation training, activity-based goal setting, problem solving and activity pacing guidance. There are often also activities that are given for between-session practice. A Cochrane review by Williams, Eccleston, & Morley (2012) found that cognitive behavioural therapy had the largest positive effect on patients' mood, both directly after treatment and at follow up. There was a small positive effect on catastrophising and disability when compared to active controls. A positive effect on pain, disability, mood and catastrophising was also reported when compared to usual treatment and wait list controls. This suggests that, although there is not a strong link between cognitive behavioural therapy and pain reduction, this treatment approach lessens psychosocial aspects that can worsen the impact of pain, such as catastrophising.

A more recent review by the same group, Eccleston, Morley, & Williams (2013) reviewing 42 studies with a cumulative total of 4788 adults experiencing musculoskeletal pain, found that cognitive behavioural therapy was effective in reducing pain and disability immediately after treatment. They were not able to ascertain how enduring such treatments were but did note that the initial effect size was small. Specific cognitive behavioural
treatments targeted at anxiety-driven thought patterns had more promising results, with reduced catastrophising thoughts and fear about current and future pain. Both these reviews concluded that, while there was some reduction in pain intensity, cognitive behavioural therapy provides an improvement in the quality of life through a reduction in catastrophising thought patterns and disability.

Another psychological intervention growing in popularity is the Explain Pain educational approach by Butler and Moseley (2003). Again, this does not refer to one specific intervention, but a group of different therapeutic approaches with the core goal of helping patients to understand their pain. More specifically, patients are guided in the understanding of the biological mechanisms of pain. The aim of this is to reduce the opinion that pain is inherently negative and threatening, but rather a protective biological mechanism (Moseley & Butler, 2015). A review of eight articles studied the efficacy of neuroscience education for adults suffering from chronic musculoskeletal pain (Louw, Diener, Butler, & Puentedura, 2011). Four of the included articles cited Butler and Moseley's Explain Pain text specifically in guiding the educational interventions. Regardless of the specific educational resource, the educational approach showed positive effects on reducing pain, disability, anxiety and stress across all of the reviewed studies (Louw et al., 2011).

Mindfulness is yet another cognitive based therapy that has been shown to be effective in managing pain complaints (Gotink et al., 2015). Developed by Jon Kabat-Zin in 1979, Mindfulness interventions promote an attention to internal and external experiences as they occur and without judgement (Gotink et al., 2015; Veehof, Trompetter, Bohlmeijer, & Schreurs, 2016). Acceptance and commitment therapy focuses on one aspect of
Mindfulness, that is acceptance of the pain and of the current state of ability rather than ruminating (Veehof et al., 2016).

Mindfulness-based interventions have been effective in reducing pain intensity in chronic pain populations (Reiner, Tibi, & Lipsitz, 2013). As well as acceptance therapy, the specific interventions analysed in this review of 16 studies included mindfulness-based stress reduction, mindfulness meditation and emotional regulation therapy. The positive results of reduced pain intensity were of moderate effect size. The authors hypothesised that the cause for pain reduction was not due to any effect on biological processes, but rather to the promotion of positive thought patterns helping to lessen the emotional impact of pain (Reiner et al., 2013).

Further review by Gotink et al. (2015) of 115 individual studies supports that mindfulness can be helpful in improving physical function and quality of life, while reducing stress and anxiety. Additionally, they suggest that undertaking mindfulness-based therapies can have a prophylactic effect against the development of chronic pain in healthy children and adults alike. In a comparison, cognitive-based therapies were found to provide more effective results in decreasing depression, pain interference and disability than mindfulness-based interventions, however, the authors support mindfulness as a useful adjunct to traditional treatment (Veehof et al., 2016).

These summaries of current literature regarding pharmaceutical, manual and psychological interventions are positive, suggesting that each intervention has a role to play in pain management. Elements of the psychological interventions can be incorporated into manual treatments either directly or indirectly. Directly, it is possible for manual therapists to
undertake additional training in the field of psychology (Hill et al., 2011). Indirectly, it is known that the clinician’s own beliefs surrounding pain, injury and disability can impact on patients’ health and illness beliefs (Bradbury et al., 2013; Darlow, 2016). Therefore, further education regarding the biopsychosocial influences on and impacts of pain could help promote more positive outlook on pain management and rehabilitation. Specifically, reassuring the patient that the cause of pain is not a serious injury and being very clear on which activities can be undertaken, rather than those that should be avoided. Appreciation of cultural background can positively impact the way patients interact with healthcare professionals and intervention outcomes (Swain & Johnson, 2014). Multidisciplinary or tailored treatment approaches are suggested to give patients the most appropriate management for them, balancing physical and psychological interventions as required (Burton et al., 2005; Hill et al., 2011; Keeffe et al., 2016).
Pain and Behaviour

Alongside biopsychosocial considerations of pain, there is an increasing awareness of how behavioural tendencies can influence the pain experience. The Reinforcement Sensitivity Theory, born out of attempts to understand anxiety disorders, proposes two neurobiological systems that regulate impulsivity and anxiety in human behaviour (Gray, 1970). The first of the two systems, the Behavioural Approach or Activation System (BAS), is thought to regulate impulsivity. The second, the Behavioural Avoidance or Inhibition System (BIS), purportedly regulates anxiety (Gray & McNaughton, 2000). This theory and its behavioural systems have since been linked to personality traits, positive and negative affect and pain (Jensen, Tan, & Chua, 2015; Kennis, Rademaker, & Geuze, 2013). The Reinforcement Sensitivity Theory further suggests that individual’s responses to external stimuli through either the activation or the inhibition system is partially innate and partially based on learnt experiences. Crucially, this means that the degree to which a response may be dominated by the activation or inhibition system may be responsive to education or cognitive therapies (Jensen et al., 2016).

The activation system contributes to approach behaviours, goal achievement and pleasurable outcomes in response to innate and learnt positive stimuli. In contrast, the inhibitory/avoidance system contributes to a more cautious approach and withdrawal behaviours in response to threatening or negative stimuli. The inhibition system has also been suggested to have the ability to override the activation system if the perceived threat is great enough (Jensen et al., 2016). Dominance of the inhibition system has been linked with personality traits of negative affect, neuroticism and anxiety, whereas dominance of the activation system has been linked with extraversion and positive affect (Campbell-Sills, Liverant, & Brown, 2004; Carver & White, 1994).
Physiologically, the inhibition system exists primarily as a pathway through the amygdala, (Gray & McNaughton, 2000; McNaughton & Corr, 2004). This area is known to be involved in acute pain perception (Moskowitz & Golden, 2013), fear conditioning, emotional learning and memory (Balleine & Killcross, 2006). Other brain areas may become involved depending on the intensity of a perceived threat (Kennis et al., 2013). The activation system includes the basal ganglia (Gray & McNaughton, 2000; McNaughton & Corr, 2004), a group of nuclei that create a network within the forebrain (Utter & Basso, 2008). They are most notably involved in the dopaminergic circuit, which is linked with movement, and reward detection, response to positive stimuli and in motivation to achieve a goal (Corr, 2013; Gray & McNaughton, 2000; Jensen et al., 2016; McNaughton & Corr, 2004). In addition, the left prefrontal cortex, an area associated with “approach” emotions (such as happiness and anger) is considered part of the activation system. Conversely, right prefrontal brain areas associated with “withdrawal” emotions (such as anxiety and sadness) may form part of the inhibition system (Jensen et al., 2016).

The emotional and motivational relationship of each behavioural system has been demonstrated by Balconi, Falbo, and Conte (2011). Alongside Carver and White's BIS/BAS Scale (1994b), they assessed participants heart rate, skin conductibility, and muscle activity. They found, as predicted, that those who scored highly in BAS responded more strongly to positive images, whereas those who scored highly in BIS responded more highly to negative images or patterns. BIS participants were also found to be more sensitive to negative emotional cues.
BIS-BAS Model of Chronic Pain

Research by Jensen and colleagues (Jensen et al., 2015, Jensen et al., 2016, Jensen et al., 2017) has recently extended the Reinforcement Sensitivity Theory to the perception of pain. Specifically, the authors suggest that an individual’s pain response can result primarily from, and be modulated by, the activation or the inhibition system. This new theory, the BIS/BAS model of chronic pain, proposes that pain is experienced as an aversive or negative stimulus and therefore activates the inhibition system (Eccleston & Crombez, 1999; Jensen et al., 2016). This, in turn, may cause avoidance behaviour and could lead to anxiety and catastrophising (Jensen et al., 2016; Lucchetti, Oliveira, Mercante, & Peres, 2012). When individuals are in an anxious state, there is an increased tendency to focus on threat-related cues, and decreased tendency to disengage from those threat-related cues. In the presence of cues that signal pain, ‘safety cues’ receive less attention. It is in this way that pain can have an inhibitory effect on activation behaviours. In the presence or anticipation of pain, a decrease in BAS could be expected, because BIS has some inhibitory effect on BAS. It is further argued that when a painful stimulus is removed, a positive feeling of relief typically ensues and this triggers the activation system (Jensen et al., 2016). However, in people suffering from chronic pain, reward circuits in the brain can function be disrupted (Elvemo, Landrø, Borchgrevink, & Haberg, 2015).

The proposed BIS-BAS model of chronic pain builds on the Fear-Avoidance Model of pain. Jensen and colleagues (2016) argued that, while they could appreciate the mutual causation of pain relating to disability, avoidance beliefs, kinesiophobia, in a disuse cycle, they preferred a multidirectional over a unidirectional model, such as the Fear-Avoidance model. Thus, the BIS-BAS model of chronic pain is structured like a web, with more focus on an interactional perspective. The model proposes that there is more interaction between variables that cause, contribute to and result from the pain experience, such as
environmental cues, a person's emotional state or affect (including proneness to anxiety or depression) and cognitive cues (such as learnt behaviour that pain is dangerous, or conversely that pain will be short lived and therefore expecting relief). Furthermore, this model suggests that these variables are interchangeable as both mechanisms and outcomes. As an example, this model allows for anxiety to be 1) a personality trait that worsens an individual’s perception of their pain, 2) a symptom that is brought on as a result of pain, or 3) symptom that develops through the irresolution of pain affecting daily life.

Jensen et al. (2016) further argue that individuals whose primary response is that of activation may respond to a greater degree to a specific set of intervention approaches and that this differs from individuals whose primary response is that of inhibition. Patients who are characterised by predominant activation responses may adopt an active coping strategy, but in their bid to overcome their pain may tend to overact and re-aggravate the painful area. Here, most effective treatment approaches could include behavioural moderation and cognitive therapy (Jensen et al., 2016). Primarily inhibitive individuals may be more likely to interpret sensations as painful, be more fearful in general and more prone to catastrophising, thus fitting into a fear-avoidance model of (chronic) pain (Crombez et al., 2012; Jensen et al., 2016). Educational approaches aimed at diminishing anxiety around pain may be best suited to these individuals.

Sub-grouping individuals affected by pain based on their levels of activation and inhibition could, therefore, facilitate the allocation of more effective management strategies. Such a person-centric approach to patient management has been shown to be effective in reducing time and money spent in therapy and time off work, and is likely to promote a positive
patient experience of treatment (Bradbury et al., 2013; Constand et al., 2014; Paul-Savoie, Bourgault, Gosselin, Potvin, & Lafrenaye, 2015).

**Previous Studies**

At present, very little literature has examined a putative relationship between activation and inhibition systems and pain perception. Jensen et al.'s (2015) study was based on a large asymptomatic sample (n=563). The university sample was also relatively homogeneous regarding background and relatively young (average age 19). Jensen et al. (2015) included a standard scale assessing levels of inhibition and activation (BIS/BAS Scale, Carver & White, 1994), an assessment of the frequency of headaches, and a question relating to participants’ average pain intensity at ten main body areas. Results showed a significant relationship between activation of the inhibition system and pain intensity and headache frequency.

A fundamental limitation of the study by Jensen et al. (2015) was the young, asymptomatic sample population. First, generalisation to typical (chronic) pain populations from this sample is difficult, as the participants were not suffering chronic pain. Second, it has been shown that the scale used to assess levels of inhibition and activation (BIS/BAS Scale, Carver & White, 1994), is most consistent for age ranges beyond that of the Jensen et al. (2015) sample (Jorm et al., 1998). Third, sampling an asymptomatic population resulted in a broad and uneven distribution of pain sites. This resulted in limited power for statistical analyses. Instead, headache frequency was analysed, which is again not easily generalised to people with musculoskeletal pain as the cause of headaches can be widely varied (Stark et al., 2013). A final limitation of the study relates to the use of non-standardised
questionnaires for the assessment of pain site and duration of headaches. Replicability and comparability with other findings in the literature are limited as a result.

A second, more recent study by Jensen et al. (2017) built on their above-discussed work and aimed to assess pain intensity and behavioural inhibition in a chronic pain population (n=88). Kinesiophobia, catastrophising, pain interference, and depressive symptoms were also assessed. Hierarchical linear regressions gave interesting results about the relationships between behavioural inhibition and mal-adaptive cognitions. A significant effect of high behavioural inhibition on depressive symptoms was revealed after pain intensity was controlled for, as well as a moderating effect of behavioural inhibition on the relationship between kinesiophobia and depressive symptoms. There were no significant results pertaining to the relationship between behavioural inhibition and pain intensity directly, nor behavioural inhibition and kinesiophobia directly. The small sample size is a limitation of this second study. Having a small sample can negatively impact statistical reliability (Field, 2009). Also, the authors of this study state that the cross-sectional design was a limitation of this study (Jensen et al., 2017).

Both these studies used the BIS/BAS Scale to assess for behavioural inhibition and activation. This questionnaire appears to have uneven weighting between items assessing inhibition and activation, with three subscales reflecting different aspects of the activation system: Drive, Fun Seeking and Reward Responsiveness, and only one for behavioural inhibition. Despite this flaw, the BIS/BAS Scale is the most widely used measure for the Reinforcement Sensitivity Theory constructs of behavioural inhibition and activation (Corr, 2016). The BIS/BAS Scale has been found to be a valid and reliable tool, with all four subscales having internal consistencies ranging from .66 to .76, and test-retest reliabilities
of .59 to .69 (Carver & White, 1994a; Cogswell, Alloy, van Dulmen, & Fresco, 2006). The second most popular instrument assessing these factors is the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ) (Torrubia, Ávila, Moltó, & Caseras, 2001). Though still popular, it has been less widely used due to having only one factor each for behavioural inhibition and behavioural activation; the latter is now generally recognised to be multidimensional. Additionally, there are 48 items, compared to 24 for the BIS/BAS Scale, making it comparatively more arduous for participants to complete (Corr, 2016). A primary flaw of both of these instruments, particularly when utilised in studies examining behaviour and pain, is that neither includes items assessing pain or response to pain specifically (Jensen, Ward, Thorn, Ehde, & Day, 2017).

Presently, Jensen and colleagues (2017) are exploring the design of a tool to specifically examine behavioural pain response. This new instrument, while based on the behavioural activation and inhibition systems, assesses cognition and beliefs, emotion and behavioural intentions in relation to pain. The two subscales are therefore likely to be named Negative Responsivity to Pain and Positive Responsivity scales (Jensen et al., 2017). On initial assessment, these scales appear to demonstrate moderately strong psychometric properties, and clinical utility, with only 20 items in total. However, the authors state that further amendment and testing to the items within the Negative Responsivity subscale is warranted. Also, further research into the Positive Responsivity scale as a whole is recommended before this instrument is made available for use (Jensen et al., 2017).

Alongside further examination of the negative and positive responsivity to pain constructs, future research could also expand on the BIS/BAS model of chronic pain. Structural equation modelling should be considered, with a view to providing more detail about
weighting and causal aspects of the proposed relationships in the BIS-BAS theory of chronic pain.

**Conclusion**

Healthcare sectors worldwide appear to be leaning toward assessing and treating pain through a much more holistic biopsychosocial approach. Despite this, the incidence, prevalence and associated cost of pain-related complaints continue to rise. Multimodal interventions are showing the most promising results in pain management. The new theory of innate behavioural tendencies lends a fascinating new angle to a biopsychosocial approach, with specific treatment options outlined for individuals that tend toward inhibitory or approach behaviours. This theory has so far shown promising results in relating pain experience with these behavioural tendencies, although further study in a larger population of adults complaining of musculoskeletal pain is required. Future research could also expand on the scale to measure behavioural traits by tailoring the questions to pain experience.
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Section 2: Manuscript
Behavioural activation and inhibition systems in relation to pain intensity and duration in a large chronic musculoskeletal pain sample

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Running Head: Behavioural inhibition, activation and chronic pain

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Abstract

Pain experience may be linked to an individual’s behavioural approach and avoidance (inhibition) tendencies. There is potential clinical utility in tailoring patients’ pain management treatments based on their behavioural tendencies. The current study aimed to extend work showing a link between behavioural approach/inhibition and pain experience by examining this relationship in a large population (n=709) affected by musculoskeletal pain. Pain intensity and pain duration were included in correlational analyses with behavioural activation and inhibition, fear-avoidance beliefs, kinesiophobia, and disability. Regression analyses to determine any predictive value of these variables on pain intensity or duration were also run. Finally, putative differences in activation and inhibition tendencies between groups reporting high and low pain intensities were tested. Findings showed significant positive relationships between pain intensity and fear-avoidance beliefs, kinesiophobia, and perceived disability, as well as positive relationships between pain duration and fear-avoidance, kinesiophobia and perceived disability. Fear-avoidance beliefs, kinesiophobia and disability accounted for 31% of the variance of the pain intensity data. Disability alone accounted for 5% of the variance within the pain duration data. Neither BIS nor BAS significantly related to or predicted pain intensity or duration and no differences in activation and inhibition tendencies were evident between high and low pain intensity groups. The present results show relationships between pain intensity and duration and fear-avoidance, kinesiophobia and disability consistent with the current literature. However, no relationships between BIS or BAS and pain intensity or duration were found. A closer examination of the BIS/BAS Scale as a measure of pain-related complaints is warranted.

Key Words: BIS/BAS, behavioural inhibition, behavioural activation, pain, fear-avoidance, kinesiophobia, disability.
Introduction

Pain, particularly chronic pain, has a significant negative impact on social and financial resources regarding both the governmental and insurance spend as well as the personal cost and lost productivity (Damian Hoy et al., 2012; Pearce et al., 2004). Statistics from New Zealand’s Accident Compensation Corporation (ACC) indicate that both reported incidences of pain and the annual cost of pain is rising (ACC, 2016, 2017). Pain typically occurs in response to a noxious stimulus and, as a protective mechanism, can have an interruptive function on behaviour (Chris Eccleston & Crombez, 1999; Gatzounis, Schrooten, Crombez, & Vlaeyen, 2014). Further assessing the way in which pain and behaviour relate, measuring patient’s innate tendencies toward approach or avoidance (inhibitory) behaviours. This offers the potential for healthcare providers to offer a more tailored management for pain complaints and personalise recommendations based on this assessment (Jensen et al., 2016).

Several models of pain encompass the interrelationship between the various aspects of pain experience. The biopsychosocial model of pain acknowledges Melzack and Wall’s gate control theory (1965) as the physiologic mechanism of pain. Illness behaviours and humans’ naturally social tendencies are included in the social aspect, and the psychological aspect is considered through how attitudes and beliefs toward pain can alter the way patients cope (Waddell, 1992; Waddell, 1987). The Fear-Avoidance Model considers how an individual’s history and beliefs may contribute to how threatening they perceive their pain to be, and therefore how likely they are to enter the negative feedback cycle the model proposes (Vlaeyen & Linton, 2000). The BIS/BAS model of chronic pain is based on the Reinforcement Sensitivity Theory, which suggests that there are two related neurophysiological systems that modulate behaviour based on rewarding or aversive
stimuli. The behavioural activation system (BAS), comprised of the basal ganglia and left prefrontal cortex, is stimulated during a positive stimulus, such as seeking food when hungry (Gray & McNaughton, 2000). Avoidance of movement (fear-avoidance) or fear of movement (kinesiophobia) in response to negative stimuli is generated by activation of the behavioural inhibition system (BIS) (Jensen et al., 2016). This system is formed largely in the amygdala, and contributes to the “stop, look, listen” response to fearful or threatening stimuli (Gray, 1990; Gray & McNaughton, 2000).

The relationship between this two-part neurobiological system and pain perception has recently been explored in a couple of studies by Jensen, Tan, and Chua (2015), and Jensen et al. (2017). Using an asymptomatic student population, Jensen et al. (2015) found a positive correlation between the inhibition system, headache frequency and pain intensity, as well as a negative correlation between the activation system and headache frequency. The authors suggest that different approaches to pain management may result in greater effects for patients with dominance of either of the systems. This was expanded by Jensen et al. (2017) with a chronic pain population (n=83). They assessed pain intensity, behavioural inhibition, pain catastrophising, depressive symptoms, kinesiophobia, and pain interference. Their results showed that high behavioural inhibition predicted depression by 12% after intensity has been controlled for. Kinesiophobia and catastrophising predicted depression 27% after both inhibition and intensity had been controlled for. They also found a significant interaction between behavioural inhibition and kinesiophobia when predicting depression. However, there were no significant findings relating behavioural inhibition with pain interference, kinesiophobia or catastrophising. Further exploration of the relationship between pain perception and behavioural tendencies could, therefore, provide findings leading to more effective pain management. The current study aims to extend this work
examining the putative relationship between inhibition and activation systems and pain to a large sample of individuals affected by persistent musculoskeletal pain.

The intensity of a person's pain, although not necessarily indicative of tissue state, may indicate how much the pain is affecting that person, physically or emotionally (Chris Eccleston & Crombez, 1999), therefore it remains a key variable in this study. Furthermore, this study aims to expand on the research undertaken by Jensen et al. (2015) and Jensen et al. (2017) by examining a larger and more diverse sample population and by including the variables: duration, disability, fear avoidance beliefs and kinesiophobia. Jensen et al. (2015) found a significant relationship between headache frequency and BIS. Here, the sample has been expanded to include a symptomatic population of people experiencing musculoskeletal pain, excluding headache. Under the framework of the biopsychosocial model of pain, psychosocial variables such as fear-avoidance beliefs and kinesiophobia are often also assessed for when considering how a patient is being affected by their pain (Brox, 2014; Wertli, Rasmussen-Barr, Held, et al., 2014). In addition to these psychological variables, the impact pain has on individuals’ daily function and physical ability is vital to assess and, in line with recommendations by Dworkin et al. (2005) and Lee et al. (2015), we chose to include it here. Unlike Jensen et al. (2017), measures for depression and catastrophising were not included, as this study aimed to explore primarily movement-related factors concerning pain experience and the behavioural systems, rather than focusing on emotional aspects. Further, rather than frequency of pain, duration has been selected as it is more common to report the pain duration in a clinical setting (Das, 2015; Rainville, 2002).
Associations between variables of pain intensity, pain duration, disability, kinesiophobia, and fear-avoidance beliefs have been demonstrated in previous studies (Denison, Åsenlöf, & Lindberg, 2004; Vranceanu et al., 2014; Wideman & Sullivan, 2011). However, to the authors’ knowledge, Jensen et al. (2017) is the only study to have examined the predictive power of behavioural tendencies on these established factors. Therefore, the aim of this study was to extend previous work by establishing whether average intensity and duration of musculoskeletal pain could be predicted by behavioural activation and inhibition tendencies and other known variables including fear-avoidance beliefs, kinesiophobia and disability in a sample of people reporting persistent musculoskeletal pain.
Methods

Measures

A web- and paper-based cross-sectional descriptive survey design using a convenience sample was implemented to identify the relationship between behavioural inhibition and activation (as measured by the Behavioural Inhibition and Activation Scale, BIS/BAS scale), pain-related fear and avoidance (Fear-Avoidance Beliefs Questionnaire), kinesiophobia (Tampa Scale for Kinesiophobia), disability (Pain Disability Index), pain site (as assessed with a Body Map) and pain level (Quadruple Visual Analog Scale, QVAS). A basic demographic section preceded the above instruments and pilot surveys were found to be acceptable. The study was approved by the local ethics committee and informed consent was implied by submission of the completed survey (UREC 2016-1069).

Data Collection

A composite questionnaire of the collated measures was prepared for administration using an online application (SurveyMonkey, Palo Alto, CA, USA) and advertised widely via Facebook Advertising to appeal to a range of demographics. In addition, printed questionnaires were distributed to all patients attending our clinical centre over a three-month data collection period. Assessment of pain symptomology relied on participants’ self-report. Completion of the questionnaire took approximately 10 minutes, and no inducements were offered.

BIS/BAS Scale

Carver and White's BIS/BAS Scale (1994) is one of the most widely used measures to assess dominance of an individual’s inhibition and activation systems (Demianczyk,
Jenkins, Henson, & Conner, 2014). The 24-item Likert-type scale provides a numerical rating on activation of both systems with higher scores indicating more activation and lower scores indicating less activation (Carver & White, 1994a). However, the two subscales are not inversely related (Jensen, Ehde, & Day, 2016). The BIS/BAS Scale has been found to be a valid and reliable tool, with all four subscales having internal consistencies ranging from .66 to .76, and test-retest reliabilities of .59 to .69 (Carver & White, 1994a; Cogswell et al., 2006). Currently, there does not appear to be a consensus in other literature regarding threshold values for a high or low score (Carver & White, 1994; Demianczyk et al., 2014; Gray, Hanna, Gillen, & Rushe, 2016; Jorm et al., 1998).

**Tampa Scale for Kinesiophobia**

The Tampa Scale for Kinesiophobia (TSK-11) is a widely used instrument for assessing kinesiophobia in back pain patients (Woby, Roach, Urmston, & Watson, 2005). It is an 11-item scale developed by removing items that demonstrated poor validity in the original TSK (Miller, Kori, & Todd, 1995). The TSK-11 shows improved psychometric properties compared to the original TSK (internal consistency: TSK α=0.76 versus TSK-11 α=0.79; reliability: TSK ICC=0.82 versus TSK-11 ICC=0.81) with the advantage of being shorter to implement. Scoring for the TSK-11 ranges from 11 to 44, the higher score representing more kinesiophobia/fear of re-injury (Woby et al., 2005).

**Fear Avoidance Beliefs Questionnaire**

The Fear-Avoidance Beliefs Questionnaire (FABQ) is a 16-item measure capturing fear-avoidance beliefs in patients affected by low back pain (Waddell, Newton, Henderson, Somerville, & Main, 1993). There are two subscales within the FABQ: Physical Activity (FABQ-PA) and Work (FABQ-W), which assess the potential for fear in physical activity
and the individual's work respectively. The initial study of this questionnaire by Waddell et al. (1993) found the measure to be reliable (k=0.61, p=0.001). Further psychometric evaluation by George, Valencia, and Beneciuk (2010) found concurrent validity between the FABQ-PA subscale and kinesiophobia (TSK-11, r=0.62), but weak concurrent validity between the FABQ-W subscale and TSK-11 (r=0.38). In order to minimise overlap with items from the kinesiophobia scale, the current study includes the FABQ-W subscale only.

**Visual Analogue Scale**

The Visual Analogue Scale (VAS) is a widely used tool to measure the intensity of patient's pain (Hawker, Mian, Kendzerska, & French, 2011; Woodforde & Merskey, 1972). The VAS has good test-retest reliability (r=0.94) and has demonstrated sensitivity to changes in pain over as little as four hours (p<0.001). There are several versions of this scale, one widely used version of which is the Quadruple Visual Analogue Scale (Q-VAS). This instrument presents the patient with four identical scales, asking them to report intensity of the 1) pain right now, 2) average pain, 3) pain level at its best, and 4) pain level at its worst. The answers are then averaged, giving a more precise indication of the average pain levels. Due to an absence of differences between the total pain score and Q-VAS score for Pain Right Now (Q-VAS RN), the predominant use of current pain ratings in clinical practice and in Jensen et al.’s (2017, 2015) work, only values for “pain right now” (Q-VAS RN) were included in the final analysis of this study.

**Body Map**

The Body map is a drawing of a body, front, back and sides, on which patients can indicate the area(s) in which they perceive pain (Margolis, Tait, & Krause, 1986). Body maps are widely used both clinically and in research for assessing painful areas, as they are simple to
understand and quick to administer (Herr & Garand, 2001; von Baeyer, Lin, Seidman, Tsao, & Zeltzer, 2011). Body mapping has demonstrated high inter-rater reliability (0.96-1) (Margolis et al., 1986).

**Pain Disability Index**

The Pain Disability Index (PDI) was designed by Pollard (1984) as a subjective measure of disability to complement findings of physical examination. The PDI has seven scales pertaining to various aspects of life, such as home responsibilities, social, and life-support activities. The total scoring indicates the level of disability ranging from “no disability” to “worst disability”. Psychometric evaluation found the PDI to be internally consistent ($\alpha=.86$), valid, and with a moderate test-retest reliability (Tait, Pollard, Margolis, Duckro, & Krause, 1987; Tait, Chibnall, & Krause, 1990). It also correlates well with the well-known Oswestry Disability Questionnaire (Grönblad et al., 1993).

**Data reduction**

On completion of data collection, raw data were downloaded and responses from printed questionnaires entered before data checking and cleaning. Inclusion criteria stipulated that only responses from adults currently living in New Zealand between the ages of 16 and 69 years without any known cancer history were included.

Total scores for each of the measures (BIS, BAS, FABQ-W, TSK-11, PDI) were calculated (Table 2). Duration scores were converted into weeks, ranging from 3 to 260 weeks. Participants were grouped by the number of pain sites rather than by pain location. This decision was supported by research suggesting that pain site has little influence on prognosis (de Vos Andersen et al., 2017). Participants were categorised as belonging to one
of two groups: few pain sites (1 or 2 sites) or many pain sites (≥3 sites) (Phongamwong & Deema, 2015).

**Statistical Analysis**

All analyses were carried out using SPSS v.24. All measures were screened for normality using SPSS and standard guidelines for sphericity and kurtosis. In the case of violations of the assumption of normality equivalent, non-parametric tests were employed. There was linearity as assessed by partial regression plots and a plot of studentized residuals against the predicted values. There was independence of residuals, as assessed by a Durbin-Watson statistic of 2.078 for intensity and 1.947 for duration. There was some evidence of multicollinearity between independent variables TSK and PDI, and FABQ and PDI as assessed through a correlations table (see table 4), but correlation values did not exceed a critical threshold of .80 and Variance Inflation factor values similarly did not exceed the critical threshold of 10 (Field, 2009). Therefore, no variables were omitted in the subsequent analyses. Intensity had two studentized deleted residuals: one with a value of 3.003, and one with a value of -3.067. These cases were not removed. Duration did not have any studentized deleted residuals greater than ±3 standard deviations. Neither pain intensity nor pain duration had leverage values greater than 0.2, or values for Cook's distance above 1.

A total of 1402 adults participated in the survey; 94 participants (6.7%) completed the survey on paper at the Unitec Osteopathy Clinic, and 1308 participants (93.3%) completed the survey online. Incomplete datasets or datasets completed by participants not fulfilling the inclusion criteria for age were excluded. The final data set consisting of 709 responses were used for all analyses (see Table 1 for demographic information). Excluded cases did
not differ significantly in terms of gender or ethnicity (gender $\chi(1)=0.001, p=.973$; ethnicity $\chi(1)=4.482, p=.482$). There was a significant age difference between included and excluded cases (included $Mdn$ age=46, excluded $Mdn$ age=53, $U=19717.5$, $z=-3.353$, $p=.001$) due to the inclusion parameters (see data collection). In addition, participants that met the inclusion criteria reported a significantly greater number of pain sites ($Mdn=2$) compared to participants that were not included ($Mdn=1$) ($U=135393.5$, $z=-15.083$, $p<.001$).

Analyses were carried out in two steps. The primary analysis was conducted in order to emulate Jensen et al.(2015). For this, pain intensity (QVAS-RN) scores were trichotomized (Gelman & Park, 2009) to assess the potential of group difference between participants reporting low-intensity pain at the time of testing and those reporting high pain intensity. Secondary analyses of continuous measures included Spearman’s rho to explore correlations between dependent variables (pain intensity and pain duration) and independent variables (fear-avoidance beliefs, kinesiophobia, disability, BIS and BAS). In addition, two multiple regressions were run in order to estimate the extent to which FABQ, TSK, PDI, BIS, and BAS predict pain intensity and pain duration respectively.
**Results**

Table 1 displays participant demographics. The sample was 80% female and predominantly of New Zealand European ethnicity (17% from other ethnic backgrounds). The mean number of pain sites was 3.10, average pain duration 2.6 years, and average pain intensity 4.09 (from a maximum of 10) (see Table 2 for a summary).

**Table 1: Demographic Information of Participants**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD)</th>
<th>Number (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td>142 (20)</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td>566 (80)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>44.28 (13.77)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ/European</td>
<td></td>
<td>585 (83)</td>
</tr>
<tr>
<td>Maori</td>
<td></td>
<td>30 (4)</td>
</tr>
<tr>
<td>Pacific Island</td>
<td></td>
<td>12 (2)</td>
</tr>
<tr>
<td>Asian</td>
<td></td>
<td>7 (1)</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td>69 (10)</td>
</tr>
</tbody>
</table>

Descriptive statistics for each scale (shown in Table 2) are similar to those reported in comparable studies (Carver & White, 1994; Jorm et al., 1998; George et al., 2010; Waddell, Newton, Henderson, Somerville & Main, 1993; Tait et al., 1990; Phongamwong & Deema, 2015; Hawker et al., 2011). The number of pain sites is typically classified as 1-2 = few, 3-4 = many (Phongamwong & Deema, 2015). By this classification, the mean number of pain sites for the present participant group is “many”. The mean pain intensity of 4.09 is mild, as
0.5 - 4.4 is classified as mild pain intensity (Hawker et al., 2011). The mean duration is 2.68 years indicating chronic pain. According to Waddell, Newton, Henderson, Somerville, and Main (1993), <19 suggests low Fear-Avoidance Beliefs in relation to Work. With a mean of 15.46, these participants are in the low fear-avoidance group, but have more fear-avoidance beliefs than other chronic pain patients, as George et al. (2010) had a mean of 13.9 in participants experiencing chronic low back pain.

**Table 2: Sample averages, standard deviations, range and descriptors for the measures included**

<table>
<thead>
<tr>
<th>Measures</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIS</td>
<td>19.9</td>
<td>4.0</td>
<td>7 – 28</td>
</tr>
<tr>
<td>BAS</td>
<td>36.3</td>
<td>6.1</td>
<td>14 – 51</td>
</tr>
<tr>
<td>BAS FS</td>
<td>10.7</td>
<td>2.3</td>
<td>4 - 16</td>
</tr>
<tr>
<td>BAS D</td>
<td>10.0</td>
<td>2.3</td>
<td>4 - 16</td>
</tr>
<tr>
<td>BAS RR</td>
<td>15.5</td>
<td>3.1</td>
<td>5 – 20</td>
</tr>
<tr>
<td>TSK-11</td>
<td>25.2</td>
<td>5.6</td>
<td>11 – 43</td>
</tr>
<tr>
<td>FABQ-W</td>
<td>15.5</td>
<td>10.6</td>
<td>0 – 42</td>
</tr>
<tr>
<td>PDI</td>
<td>20.0</td>
<td>14.1</td>
<td>0 – 60</td>
</tr>
<tr>
<td>Number of Pain Sites</td>
<td>3.1</td>
<td>3.1</td>
<td>1 – 20</td>
</tr>
<tr>
<td>QVAS RN</td>
<td>4.1</td>
<td>2.4</td>
<td>0 – 10</td>
</tr>
<tr>
<td>Duration</td>
<td>140.0</td>
<td>92.4</td>
<td>3 – 260 weeks</td>
</tr>
</tbody>
</table>

BIS = Behavioural Inhibition System, BAS = Behavioural Activation System, BAS Subscales FS = Fun Seeking, D = Drive, RR = Reward Responsiveness, TSK-11 = Tampa Scale for Kinesiophobia, FABQ-W = Fear-Avoidance Beliefs (Work), PDI = Pain Disability Index.
In the first step of the analysis, the dataset was trichotomized (Gelman & Park, 2009) based on pain intensity scores (QVAS RN). Mann-Whitney U tests of BIS and BAS were performed between participants reporting low pain intensity ($Mdn=2$) and participants reporting high pain intensity ($Mdn=8$). There was no significance in BIS or BAS compared to high or low pain intensity groups. There was also no significant difference between the high and low pain intensity groups and the BAS subscales of Fun Seeking, Drive Reward Responsiveness.

**Table 3: Spearman’s Rho Correlations between Dependent and Independent Variables**

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Rho Value</th>
<th>Pain intensity (QVAS RN)</th>
<th>Pain duration (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioural Inhibition (BIS)</td>
<td>-.064</td>
<td>.054</td>
<td></td>
</tr>
<tr>
<td>Behavioural Activation (BAS)</td>
<td>-.018</td>
<td>.052</td>
<td></td>
</tr>
<tr>
<td>Kinesiophobia (TSK-11)</td>
<td>.322***</td>
<td>.153**</td>
<td></td>
</tr>
<tr>
<td>Fear-Avoidance Beliefs (FABQ-W)</td>
<td>.338***</td>
<td>.106**</td>
<td></td>
</tr>
<tr>
<td>Disability (PDI)</td>
<td>.515***</td>
<td>.189***</td>
<td></td>
</tr>
</tbody>
</table>

*** $p<.001$; ** $p<.01$; * $p<.05$

The second step of analysis included production of Spearman’s rho to explore correlations between dependent and independent variables. Table 3 shows the magnitude and direction of relationships between the dependent (pain intensity; pain duration) and independent variables (BIS, BAS, TSK-11, FABQ-W, PDI). There was no significant correlation between pain intensity (QVAS RN) and measures of BIS or BAS (both $p>.05$). However, employing Hopkins (1997) magnitude of effect descriptors, there were significant and
moderate positive correlations pain intensity and kinesiophobia (TSK; rho=.322, <.001), fear-avoidance beliefs (FABQ; rho=.338, p<.001), and significant, large positive correlations between pain intensity and disability (PDI; rho=.515, p<.001). There was also a significant but small positive correlation between measures of pain intensity and pain duration (rho=.108, p<.01). For pain duration, no significant correlation was found between pain duration and measures of BIS or BAS (both p>.05). There was a significant positive relationship with kinesiophobia (rho=.153, p<.001), fear-avoidance (rho=.106, p<.01), and disability (rho=.189, p<.001).

There was a moderate correlation between BIS and BAS scores (rho=.313, p<.001). In this sample, BIS also correlated weakly with kinesiophobia (rho=.120, p<.01). There are no other significant correlations between BIS or BAS and other variables. Kinesiophobia, fear-avoidance beliefs, disability, pain duration and pain intensity all correlated significantly and positively with each other (see Table 4 for all intercorrelations).
Table 4: Spearman’s Rho Correlations Between Independent Variables

<table>
<thead>
<tr>
<th></th>
<th>Correlation coefficient (rho)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIS</td>
<td>BIS</td>
<td>.313***</td>
</tr>
<tr>
<td>TSK-11</td>
<td>.120*</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>FABQ-W</td>
<td>-.007</td>
<td>n/sig</td>
</tr>
<tr>
<td>PDI</td>
<td>-.016</td>
<td>n/sig</td>
</tr>
<tr>
<td>BAS</td>
<td>TSK-11</td>
<td>.050</td>
</tr>
<tr>
<td>FABQ-W</td>
<td>.052</td>
<td>n/sig</td>
</tr>
<tr>
<td>PDI</td>
<td>-.071</td>
<td>n/sig</td>
</tr>
<tr>
<td>TSK-11</td>
<td>FABQ-W</td>
<td>.347***</td>
</tr>
<tr>
<td>PDI</td>
<td>.506***</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>FABQ-W</td>
<td>PDI</td>
<td>.422***</td>
</tr>
</tbody>
</table>

*** p<.001; ** p<.01; * p<.05

Finally, multiple linear regression models were estimated in order to determine which of the factors included in the present study were predictive of pain intensity (QVAS RN) and pain duration. The first multiple regression model (Table 5) showed that pain intensity (QVAS RN) was significantly predicted by TSK-11, FABQ-W and PDI, F(5, 701)=61.541, p<.001, adj. $R^2=.305$. That is, TSK-11, FABQ-W and PDI were able to predict 31% of the variance in pain intensity in this dataset. In contrast, BIS and BAS were not able to significantly predict pain intensity.
Table 5. Linear regression of Model 1 (measures of Behavioural Inhibition, Behavioural Activation, Kinesiophobia, Fear Avoidance Beliefs and Perceived Disability on pain intensity in the study sample).

<table>
<thead>
<tr>
<th>DV: Pain Intensity</th>
<th>$R^2 = .305$</th>
<th>Model Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$= &lt;.001$</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Unstandardised $\beta$</th>
<th>95% CI Lower Limit</th>
<th>95% CI Upper Limit</th>
<th>SE$_B$</th>
<th>Standardised $\beta$</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIS</td>
<td>-.031</td>
<td>-.07</td>
<td>.01</td>
<td>.021</td>
<td>-.051</td>
</tr>
<tr>
<td>BAS</td>
<td>-.004</td>
<td>-.03</td>
<td>.02</td>
<td>.014</td>
<td>-.11</td>
</tr>
<tr>
<td>TSK-11</td>
<td>.038</td>
<td>.01</td>
<td>.07</td>
<td>.016</td>
<td>.086*</td>
</tr>
<tr>
<td>FABQ-W</td>
<td>.032</td>
<td>.02</td>
<td>.05</td>
<td>.008</td>
<td>.142***</td>
</tr>
<tr>
<td>PDI</td>
<td>.071</td>
<td>.06</td>
<td>.08</td>
<td>.007</td>
<td>.418***</td>
</tr>
</tbody>
</table>

**Notes:**
- DV: Dependent Variable; BIS = Behavioural Inhibition System, BAS = Behavioural Activation System, TSK-11 = Tampa Scale for Kinesiophobia, FABQ-W = Fear-Avoidance Beliefs (Work), PDI = Pain Disability Index.
- $\beta$ = unstandardised regression coefficient; SE$_B$ = standard error of the coefficient; CI = Confidence Interval
- *** $p < .001$; ** $p < .01$; * $p < .05$.

The second multiple regression model (Table 6) showed that pain duration could be significantly predicted by PDI, $F(5, 702) = 6.996, p < .001$, adj. $R^2 = .047$. Only the predictor PDI contributed to this model and accounted for 5% variance within pain disability. None of the other variables (BIS, BAS, TSK-11 or FABQ-W) added significantly to the fit of the model. Regression coefficients and standard errors can be found in Tables 5 and 6 (above).
Table 6. Linear regression of Model 2 (measures of Behavioural Inhibition, Behavioural Activation, Kinesiophobia, Fear Avoidance Beliefs and Perceived Disability on pain duration in the study sample).

<table>
<thead>
<tr>
<th></th>
<th>Unstandardized β</th>
<th>95% CI Lower Limit</th>
<th>95% CI Upper Limit</th>
<th>SEβ</th>
<th>Standardised β</th>
</tr>
</thead>
<tbody>
<tr>
<td>BIS</td>
<td>.807</td>
<td>-1.08</td>
<td>2.69</td>
<td>.962</td>
<td>.034</td>
</tr>
<tr>
<td>BAS</td>
<td>.872</td>
<td>-1.33</td>
<td>2.08</td>
<td>.615</td>
<td>.058</td>
</tr>
<tr>
<td>TSK-11</td>
<td>1.272</td>
<td>-.17</td>
<td>2.72</td>
<td>.736</td>
<td>.076</td>
</tr>
<tr>
<td>FABQ-W</td>
<td>.225</td>
<td>-.50</td>
<td>.95</td>
<td>.370</td>
<td>.026</td>
</tr>
<tr>
<td>PDI</td>
<td>.905</td>
<td>-.32</td>
<td>1.49</td>
<td>.298</td>
<td>.138**</td>
</tr>
</tbody>
</table>

DV= Dependent Variable; BIS= Behavioural Inhibition System, BAS= Behavioural Activation System, TSK-11= Tampa Scale for Kinesiophobia, FABQ-W= Fear-Avoidance Beliefs (Work), PDI= Pain Disability Index.

*** p<.001; ** p<.01; * p<.05; β=unstandardised regression coefficient; SEβ=standard error of the coefficient; CI = Confidence Interval
Discussion

The main aim of this study was to investigate the extent to which intensity and duration of musculoskeletal pain could be predicted by behavioural activation and inhibition tendencies and other known variables including fear-avoidance beliefs, kinesiophobia and disability. This was carried out to test and extend recent studies by Jensen et al. (2015, 2017), which provided the first indication of significant relationships.

Comparing the current average scores to those reported in similar studies shows low disability (20.06) in the present sample compared to other studies (32.5-34.5) (Tait et al., 1990) and marginally low Reward Responsiveness (15.54, as assessed by the BAS-RR subscale of the Behavioural Activation Scale) compared to other studies (15.0 – 17.6)(Carver & White, 1994a; Jorm et al., 1998). All other average scores reported here match those reported in other literature. BIS average was 19.90 here compared to 19.99 (Carver & White, 1994a), and 19.3 – 22.0 depending on age range and gender (A. Jorm et al., 1998). The total BAS score is 36.30, which fits within an average range from 33.6 - 40.2 (A. Jorm et al., 1998). For BAS FS the average was 10.75, otherwise a range of means has been reported from 9.5 – 12.3, and for BAS D the average was 10.02 while others report a range of 8.2 – 10.9 (A. Jorm et al., 1998). Kinesiophobia also scored similarly to other reports, with the current study averaging 25.16 compared to 24.5 – 44.2 in 3085 participants with a range of pain complaints (Roelofs et al., 2011), and 22.6 in 53 chronic low back pain patients (George et al., 2010).

BIS and BAS

In contrast to the results of Jensen et al. (2015), this study did not identify a significant relationship between pain intensity or pain duration and BIS or BAS, and neither BIS nor
BAS were predictors for pain intensity or duration. These findings are in contrast to the results of Jensen et al. (2015) but in partial agreement with the results of Jensen et al. (2017).

It has been theorised that once pain reaches a moderate intensity (a score of more than four out of a possible ten on a visual rating scale (Chris Eccleston & Crombez, 1999)), it can have an interruptive, protective function, which Jensen et al. (2015) suggested could relate to behavioural tendencies. The average pain intensity within this participant group was low, so may not have interrupted behaviour, potentially explaining why we did not find any significant relationships between BIS, BAS and pain intensity. However, Jensen et al. (2017) reported a moderate pain intensity, yet their results did not support a relationship between behavioural inhibition and pain intensity. In light of these contrasting findings, the nature of the relationship between behavioural inhibition and pain intensity would benefit from further exploration.

The present data showed a significant positive relationship between BIS and kinesiophobia. This is supported by Jensen et al.’s (2016) BIS/BAS Model of pain, which suggests that people who are high in BIS at baseline may be more prone to developing kinesiophobia in the presence of pain (Jensen et al., 2016) and may also be more likely to be affected by depression (Jensen et al. (2017)). A relationship between behavioural inhibition and kinesiophobia may suggest that avoiding potentially painful movements could relate to the development and persistence of chronic pain conditions, as described the Fear-Avoidance model (Vlaeyen & Linton, 2000; Wertli, Rasmussen-Barr, Held, et al., 2014). Indeed, work by Vlaeyen et al. (1995) found that kinesiophobia did relate to pain intensity coping, higher scores for kinesiophobia displayed more avoidance in movement tasks.
Results of the current study also showed a significant positive relationship between BIS and BAS. This supports some of the suggestions made by Jensen et al. (2016) that BIS and BAS are not mutually exclusive, but rather function together. Behavioural inhibition and activation are postulated to be separate systems in different brain areas that respond to different environmental cues, but that overlap in some areas (for example the presence of a threat may cause a momentary increase in inhibitory behaviours and a simultaneous decrease in activation behaviours).

Reward responsiveness can be altered in patients with chronic pain (Becker, Gandhi, & Schweinhardt, 2012). A study by Elvemo, Landrø, Borchgrevink, and Haberg (2015) found reward responsiveness to be significantly reduced in chronic pain patients \((p=.005)\) compared to matched healthy controls. As the pain duration for the present participant group is in the chronic range (average 2.7 years) the findings by Elvemo et al. (2015) could explain why this population scored lower than average in reward responsiveness, as compared to studies by Carver and White (1994a) and Jorm et al. (1998) which were not assessing participants based on pain, but rather personality traits.

**Intensity Regression (Independent Variables that Predict Pain Intensity)**

The extent to which scores for BIS, BAS, kinesiophobia, fear-avoidance beliefs and disability could predict pain intensity was examined. Pain intensity was predicted by variables kinesiophobia, fear-avoidance beliefs and disability, accounting for 31% of the variance in this dataset. There are a number of studies examining the predictive ability of these variables for pain intensity. Parr et al. (2013) showed fear of pain to be the strongest predictor of pain intensity from baseline to follow up (96 hours) after inducing
experimental pain. They also found catastrophic thinking to be a strong predictor of pain intensity, and kinesiophobia to be a strong predictor of disability. These findings are also reported in other populations, discussed above.

**Duration Regression (Disability as a Predictor of Pain Duration)**

In addition to examining potential predictors of pain intensity, predictors of pain duration were also examined. Disability was the only variable to predict pain duration and accounted for 5% of the variance within the pain duration dataset. There was no statistically significant predictive value found of kinesiophobia or fear-avoidance beliefs on pain duration within this dataset. A review by Wertli, Rasmussen-Barr, Weiser, et al. (2014) states that the reported pain durations differed widely between the articles they assessed, making it difficult to draw any conclusions about how other variables, such as fear-avoidance beliefs, may relate to specific durations of pain. Instead, when pain duration is directly examined, it is more often in a controlled environment of experimentally induced pain which is typical of short-term duration and therefore not easily generalised to a wider population (Parr et al., 2013). It has been theorised as part of the Fear-Avoidance Model of pain that disability could increase as pain duration increases through physical deconditioning secondary to avoiding movements or activities that may cause, or historically have caused, pain (Wideman et al., 2013). The lack of a predictive association between pain duration and fear avoidance beliefs in this study is interesting as it would suggest that there is no clear predictive value of fear-avoidance beliefs on the duration of patient's pain in a large sample of chronic low-intensity musculoskeletal pain sufferers.
Correlations

Results of this study showed a strong positive relationship between pain intensity and disability, and a moderate positive relationship between pain intensity and fear-avoidance beliefs and kinesiophobia. These relationships have previously been reported, often in conjunction with other psychological factors. For example, a study by Arnstein, Caudill, Mandle, Norris, and Beasley (1999) found that high pain intensity related with higher disability, and with lower self-efficacy. A second example by Denison, Åsenlöf, and Lindberg (2004) found that pain intensity explained a significant proportion of the variance in disability scores in a large sample of musculoskeletal pain patients. Thirdly, Vranceanu et al. (2014) found that psychological variables including catastrophic thinking, pain anxiety and depression were strongly associated with pain intensity and disability. Altogether, this suggests that the present dataset represents what is currently known about interrelationships between psychological variables.

There were weak positive relationships between pain duration and fear-avoidance beliefs, kinesiophobia and disability. Pain duration has not been noted to relate to disability, fear-avoidance beliefs or kinesiophobia in similar studies of chronic pain patients (Crombez, Vlaeyen, Heuts, & Lysens, 1999; Denison et al., 2004), and subacute stage patients (Wideman & Sullivan, 2011). A review by Wertli, Rasmussen-Barr, Weiser, Bachmann, and Brunner (2014) found that in patients who had had pain for four weeks to six months, high fear-avoidance beliefs were prognostic of delayed return to work/higher number of pain-related sick days. They also noted that in very acute (<3 weeks) and chronic (>3 months) pain populations, there was no relationship between fear-avoidance beliefs and pain duration or disability. This suggests that fear-avoidance beliefs may have more impact
on pain duration within 3 – 12 weeks post-injury. So, although there is some evidence to suggest there is a relationship between fear-avoidance beliefs and pain duration, as the current data set has shown, these variables are not widely found to significantly relate to pain duration.

Consistent with Denison et al. (2004), there were significant positive relationships between kinesiophobia, fear-avoidance beliefs and disability. Similarly, Vlaeyen et al. (1995) showed that kinesiophobia was closely related to catastrophising, depression, and also to pain intensity. Both Denison et al. (2004) and Vlaeyen et al. (1995) examined participants experiencing musculoskeletal pain in a primary healthcare setting, similarly to the current participant group. In summary, while there is mild variability between the present study and previous findings detailing interrelationships between pain intensity, pain duration, kinesiophobia, fear-avoidance beliefs and disability, the current results are broadly in line with the existing literature.

**Comparison of current study with previous literature**

The general framework of the current study and Jensen et al. (2015) was similar, in both used a survey to collect data on BIS and BAS levels using the BIS/BAS Scale (Carver & White, 1994b) while also asking about pain in various body locations and the intensity of that pain. However, there were several methodological differences between our study and those of Jensen et al. (2015, 2017), these include sample characteristics (size, age and ethnicity), the measures used (including how the BIS/BAS Scale was utilised, pain intensity measures, and the use of pain duration rather than frequency), and statistical analysis.
The current study involved a larger sample population (n=709 compared to n=563, and n=88 respectively). Regarding sample population, the chances of discovering trends in the data may be increased by having a more homogeneous sample population, as in Jensen’s studies. However, having a larger, more heterogeneous sample population may mean results are more representative of the general population, and therefore more generalizable and clinically applicable.

The current sample population had much more variance in age (Mean (SD) = 44.28 (13.77)) compared to Jensen et al. (2015) (Mean (SD) = 19.78 (1.29)), and slightly more than Jensen et al. (2017) (Mean (SD) = 52.90 (11.35)). There does not appear to be any difference in pain or pain reporting within younger or older adult populations (Herr & Garand, 2001; von Baeyer et al., 2011) so it is unlikely that different mean ages have affected the differences in results.

Regarding participant's cultural and ethnic backgrounds, most of Jensen et al. (2015) participants were Chinese, and Jensen et al. (2017) were Canadian, whereas the current population sample was made up predominantly of New Zealand Europeans. Pain experience (pain intensity, impact on life and disability) between New Zealand Europeans and Chinese immigrants living in New Zealand is suggested to be similar (Ho & Johnson, 2013). However, the response to pain between these two groups was different, with more New Zealand Europeans seeking medical help and social support than Chinese immigrants. The authors reported difficulty in comparing these results to studies of Chinese people in China, as many of those studies are condition-specific. They also noted that many of their Chinese participants also demonstrated signs of western acculturation regarding health management approaches, therefore a comparison of New Zealanders and Chinese in China
could yield different results. Ethnicity has been shown to relate to differences in pain reporting among other groups. For example, Day and Thorn (2010) found ethnic background to be a unique predictor of pain reporting between two ethnic groups in the USA, with African-Americans reporting significantly higher pain intensities than White Americans. Additionally, Hobara (2005) found a significant effect of both sex and culture on the acceptability of pain behaviours across Japanese and Euro-American adults. These findings suggest that, although different ethnic and cultural backgrounds may account for some difference in pain reporting, no specific conclusions can be drawn on how ethnicity may have affected the results of this study compared with Jensen et al. (2015) and Jensen et al. (2017).

Secondly, the current study and both Jensen et al. (2015) and Jensen et al. (2017) all utilised Carver and White’s (1994) BIS/BAS Scale to assess levels of BIS and BAS. However, Jensen et al. (2015) only used five BIS items rather than all seven – they removed the reverse-scored items to increase the internal validity of the BIS scale. Jensen et al. (2017) only used the BIS subscale, and not the BAS subscales. Either of these alterations to the BIS/BAS Scale could account for some variation in BIS scores and results. Additionally, pain intensity scores were captured with the use of different measures. For pain intensity, the current study employed a quadruple Visual Analogue Scale (Q-VAS) score to assess participants' average pain intensity per pain site, whereas Jensen et al. (2015) and Jensen et al. (2017) used a Numerical Rating Scale for their pain intensity at bothersome areas over the past week. Clinically, the Numerical Rating Scale for pain intensity over the past week would be quick to use and provide a sufficient baseline. However, in research, particularly when considering cases of chronic pain, it may be more valid to use a measure such as the Q-VAS, which provides an average score that is
representative of all the pain intensities the participant feels in relation to their chronic pain. The difference in pain intensity measures may also have contributed to discrepancies in results between the two studies and thereby influencing the relationship between pain intensity and the BIS/BAS measures.

Further exploring the differences in measures between the current study and Jensen et al. (2015), while they drew a focus to headache frequency, this study did not analyse a specific pain area but instead assessed the number of pain sites for participants. Headaches are often one of the more common complaints in young adults, which may explain why Jensen and colleagues chose to analyse them (Dunn, Jordan, Mancl, Drangsholt, & Le Resche, 2011). However, there is such variability in causes for headaches, such as stress and medication overuse, that do not necessarily represent musculoskeletal pain (Aaseth et al., 2008; Stark et al., 2013). Therefore headaches were not specifically assessed for this study.

Another key difference in the assessment of pain between the current study and Jensen et al. (2015) was frequency versus duration. Frequency of pain has been used in child and adolescent populations, presumably where persistent or chronic pain is less prevalent (Dunn et al., 2011; Rathleff, Roos, Olesen, & Rasmussen, 2013). However, frequency of pain does not seem to be the most clinically useful way of considering pain when compared to the acute or chronic definitions that are most commonly used in other literature (Das, 2015; Hoy, Brooks, Blyth, & Buchbinder, 2010). Therefore the present study assessed pain duration instead of pain frequency. We also included additional measures for fear-avoidance beliefs, kinesiophobia and disability, as well as asking about participants’ duration of pain. These established variables were included to further the BIS/BAS Model of chronic pain by assessing how they relate to the behavioural systems.
Finally, the methods of statistical analysis are different between this study and the two by Jensen and colleagues (Jensen et al., 2015; Jensen et al., 2017). The current study utilised non-parametric correlations and regression analyses, whereas Jensen et al. (2015) have used a series of ANOVAs. Although it is highly likely that Jensen's dataset was normally distributed since it was reasonably homogeneous with regard to demographics, the authors did not report data screening procedures, and it is therefore not possible to assess whether the use of parametric testing was justified. On the one hand, by using nonparametric testing we may not have picked up on trends emerging that may have come to light within Jensen's results. However as we did have a skewed dataset, this should not have been the case, as non-parametric tests are only less powerful when there are no violations of normality (Field, 2009). On the other hand, using repeated 1-way ANOVAs does increase the risk of over-reporting emerging trends, whereas regression approaches are more robust to this because all variables are added to the analysis at once. The study by Jensen et al. (2017) was more similar in analysis: they reported a normally distributed dataset and employed a hierarchical linear regression. Though there were differences in analyses between the three studies, assuming each dataset was tested based on its assumptions of normality, the results should be reasonably robust in all.

The theory of behavioural inhibition and activation playing a role in how pain is perceived is still plausible despite the lack of findings in this study. While the theory of a two-factor neurophysiological system has been tested in other areas, such as personality and motivations, more examination around their relation to pain is indicated here. Considering pain, particularly chronic pain, from a perspective of behavioural activation and inhibition offers a unique, yet plausible take on patient presentation and provides more focused
treatment options (Jensen et al., 2016). However, a limitation of this theory, and indeed of this study, seems to be the measure of BIS and BAS.

**Limitations**

This study relied on self-report or pain symptomology rather than any clinical analysis. This, coupled with the recruitment strategy of advertising on Facebook, could have allowed for participants over- or under-reporting of symptoms. There was also no control in place to screen for confounding factors such as mental health status or the use of medications that may alter pain experience, neurobiology and emotion regulation. These could all have impacted on the validity of the results. Furthermore, while the BIS/BAS Scale is the most widely used tool for assessing inhibition and activation systems (Demianczyk et al., 2014), it was designed as an assessment tool for Gray’s reinforcement sensitivity theory of approach and avoidance behaviours in the psychology field (Carver & White, 1994a). It does not have any items to account for pain experience or pain-related complaints.

Further limitations of the BIS/BAS Scale itself are the varied number of items per subscale, where the BAS subscale includes 13 items compared to the 7 BIS subscale items. Additionally, the BAS scale has been split up to allow for individual assessment of BAS-related traits or Fun Seeking, Reward Responsiveness and Drive, whereas the BIS scale has no items relating to specific traits. Hence the scale is not weighted to assess BIS and BAS equally.
Future Research

Although there have been attempts to revise the BIS/BAS Scale to improve psychometric properties (Demianczyk et al., 2014), it has not been altered or tested for use in pain reporting (Jensen et al., 2016). To reiterate a point made by Jensen et al. (2016), the next logical step in pursuing this theory is, the development of a measure for BIS/BAS in the context of pain. Such an instrument appears to be in development stages (Jensen, Ward, Thorn, Ehde, & Day, 2017), but nothing is yet ready for research or clinical use.

Conclusions

The primary aim of this study was to add to the data published by Jensen et al. (2015) by testing whether average intensity and duration of persistent musculoskeletal pain could be predicted by behavioural activation and inhibition tendencies, as well as other known variables including fear-avoidance beliefs, kinesiophobia and disability.

While this study’s results did not reproduce the purported relationship between BIS, BAS and pain intensity, there was a trend toward a significant relationship between high and low pain intensity groups and BIS and BAS, such as was found in Jensen et al. (2015). The relationship found here between BIS and kinesiophobia was similarly reported in more recent literature (Jensen et al., 2017). Furthermore, relationships between pain intensity and duration, and kinesiophobia, fear-avoidance beliefs and disability were found consistent with the wider literature.

We conclude that the theory of behavioural inhibition and activation systems relating to pain may still be valid, regardless of the lack of statistically significant findings in this
study. We propose that a revised measure of behavioural inhibition and activation systems including items specific to pain experience may reveal more about the relationship and have important clinical utility. If a more specific measure were to become available for use in managing musculoskeletal pain complaints it could allow for tailoring treatment and rehabilitation programmes to better address and avoid chronic pain states. This, in turn, could decrease the financial and societal burden caused by pain-related complaints.
References


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*Personality and Individual Differences*, **40**(8), 1649–1658. 


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Fredin, K., & Lorås, H. (2017). Manual therapy, exercise therapy or combined treatment in
the management of adult neck pain – A systematic review and meta-analysis. 


Pain


Rathleff, M. S., Roos, E. M., Olesen, J. L., & Rasmussen, S. (2013). High prevalence of


Supplementary

The Survey

Nina's Master of Osteopathy project

Dear participant,
Thank you for deciding to help me out with my research project. On the following pages, you will find the questionnaires relating to your pain. Please do not take too long thinking about your responses, but select the first thing that comes to mind. For some of the questions, there are more specific instructions at the beginning. Please read these carefully before answering.

Filling in this questionnaire should take about 10 minutes.

Please indicate the number(s) of the areas in which you experience pain. If you have selected more than one painful area, indicate the number of the area that is the most concerning to you at present.

How long have you been experiencing the above pain for? For multiple sites, please give an indication for each separately.

<table>
<thead>
<tr>
<th></th>
<th>3 weeks</th>
<th>3 – 12 weeks</th>
<th>12 weeks – 6 months</th>
<th>6 months – 1 year</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 weeks</td>
<td>3 – 12 weeks</td>
<td>12 weeks – 6 months</td>
<td>6 months – 1 year</td>
<td></td>
</tr>
<tr>
<td>1 – 2 years</td>
<td>2 – 5 years</td>
<td>5+ years</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. Respond to each item as if it were the only item. That is, don’t worry about being “consistent” in your responses. Choose from the following four response options:

<table>
<thead>
<tr>
<th></th>
<th>Very false for me</th>
<th>Somewhat false for me</th>
<th>Somewhat true for me</th>
<th>Very true for me</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A person's family is the most important thing in life.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2. Even if something bad is about to happen to me, I rarely experience fear or nervousness.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3. I go out of my way to get things I want.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4. When I'm doing well at something I love to keep at it.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5. I'm always willing to try something new if I think it will be fun.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6. How I dress is important to me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7. When I get something I want, I feel excited and energized.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8. Criticism or scolding hurts me quite a bit.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9. When I want something I usually go all-out to get it.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10. I will often do things for no other reason than that they might be fun.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11. It's hard for me to find the time to do things such as get a haircut.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>12. If I see a chance to get something I want I move on it right away.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>13. I feel pretty worried or upset when I think or know somebody is angry at me.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>14. When I see an opportunity for something I like I get excited right away.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>15. I often act on the spur of the moment.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>16. If I think something unpleasant is going to happen I usually get pretty &quot;worked up.&quot;</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>17. I often wonder why people act the way they do.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>18. When good things happen to me, it affects me strongly.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>19. I feel worried when I think I have done poorly at something important.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>20. I crave excitement and new sensations.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>21. When I go after something I use a &quot;no holds barred&quot; approach.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>22. I have very few fears compared to my friends.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>23. It would excite me to win a contest.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>24. I worry about making mistakes.</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank.

<table>
<thead>
<tr>
<th></th>
<th>I'm afraid that I might injure myself if I exercise</th>
<th>Strongly disagree</th>
<th>Disagree</th>
<th>Agree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>If I were to try to overcome it, my pain would increase</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>My body is telling me I have something dangerously wrong</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>People aren't taking my medical condition seriously enough</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>My accident has put my body at risk for the rest of my life</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>Pain always means I have injured my body</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>8</td>
<td>I wouldn't have this much pain if there weren't something potentially dangerous going on in my body</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>Pain lets me know when to stop exercising so that I don't injure myself</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>I can't do all the things normal people do because it's too easy for me to get injured</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>11</td>
<td>No one should have to exercise when he/she is in pain</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Here are some of the things which other people have told us about their pain. For each statement please circle any number from 0 to 6 to say how much physical activities such as bending, lifting, walking or driving affect or would affect your pain.

The following statements are about how your normal work affects or would affect your pain.

<table>
<thead>
<tr>
<th>Statement</th>
<th>Completely disagree</th>
<th>Unsure</th>
<th>Completely agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. My pain was caused by my work or by an accident at work</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>2. My work aggravated my pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>3. I have a claim for compensation for my pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>4. My work is too heavy for me</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>5. My work makes or would make my pain worse</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>6. My work might harm my injured area</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7. I should not do my normal work with my present pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>8. I cannot do my normal work with my present pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9. I cannot do my normal work until my pain is treated</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>10. I do not think that I will be back to my normal work within 3 months</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>11. I do not think that I will ever be able to go back to that work</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

Please circle the number that best describes the question being asked.

Note: If you have more than one complaint, please answer each question for each individual complaint and indicate the score for each complaint. Please indicate your pain level right now, average pain and pain at its best and worst.

<table>
<thead>
<tr>
<th>Example:</th>
<th>No pain</th>
<th>Elbow</th>
<th>Wrist</th>
<th>Shoulder</th>
<th>Worst possible pain</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4 5 6 7 8 9 10</td>
</tr>
</tbody>
</table>

1. What is your pain RIGHT NOW? Worst possible pain

2. What is your TYPICAL or AVERAGE pain?

3. What is your pain level AT ITS BEST? (How close to “0” does your pain get at its best?) Worst possible pain

4. What is your pain level AT ITS WORST? (How close to “10” does your pain get at its worst?) Worst possible pain
For each of the 7 categories of life activity listed, please circle the number on the scale that describes the level of disability you typically experience. A score of 0 means no disability at all, and a score of 10 signifies that all of the activities in which you would normally be involved have been totally disrupted or prevented by your pain.

**Family/home responsibilities:** This category refers to activities of the home or family. It includes chores or duties performed around the house (e.g., yard work) and errands or favors for other family members (e.g., driving the children to school).

No disability 0 1 2 3 4 5 6 7 8 9 10 Worst disability

**Recreation:** This category includes hobbies, sports, and other similar leisure time activities.

No disability 0 1 2 3 4 5 6 7 8 9 10

**Social activity:** This category refers to activities that involve participation with friends and acquaintances other than family members. It includes parties, theater, concerts, dining out, and other social functions.

No disability 0 1 2 3 4 5 6 7 8 9 10

**Occupation:** This category refers to activities that are a part of or directly related to one’s job. This includes nonpaying jobs as well, such as that of a housewife or volunteer worker.

No disability 0 1 2 3 4 5 6 7 8 9 10

**Sexual behavior:** This category refers to the frequency and quality of one’s sex life.

No disability 0 1 2 3 4 5 6 7 8 9 10

**Life-support activity:** This category refers to basic life-supporting behaviors such as eating, sleeping, and breathing.

No disability 0 1 2 3 4 5 6 7 8 9 10

**Demographical Information**

**Gender**

Male

Female

**Date of birth**

**Ethnicity**

NZ European

Maori

Pacific Island

Asian

Other:

**Do you currently live in New Zealand?**

Yes

No
Ethical Approval Letter

Nina Sanson
1080a Peak Road
Helensville 0675

21.9.16

Kia ora Nina,

Your file number for this application: 2016-1069
Title: Reinforcement sensitivity theory and musculoskeletal pain.

Your application for ethics approval has been reviewed by the Unitec Research Ethics Committee (UREC) and has been approved for the following period:

Start date: 11.10.16
Finish date: 11.10.17

Please note that:

1. The above dates must be referred to on the information AND consent forms given to all participants.

2. You must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.

Yours sincerely,

Nigel Adams,
Deputy Chair, UREC

cc: Jesse Masen
Sylvia Hach
Cynthia Almeida
Declaration

Name of candidate: Nina Sanson

This Thesis/Dissertation/Research Project entitled: Behavioural activation and inhibition systems in relation to pain intensity and duration in a large chronic musculoskeletal pain sample is submitted in partial fulfillment for the requirements for the Unitec degree of Master of Osteopathy

Principal Supervisor: Sylvia Hach

Associate Supervisor/s: Jesse Mason

CANDIDATE'S DECLARATION

I confirm that:

- This Thesis/Dissertation/Research Project represents my own work;
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: UREC 2016-1069

Candidate Signature: [Signature]  Date: 10/7/18

Student number: 1417392
Full name of author: Nina Sanson

ORCID number (Optional): ..............................................

Full title of thesis/dissertation/research project ('the work'):
Behavioural activation and inhibition systems in relation to pain intensity and duration in a large chronic musculoskeletal pain sample

Practice Pathway: HEALTHCARE

Degree: Master of Osteopathy

Year of presentation: 2018

Principal Supervisor: Sylvia Hach

Associate Supervisor: Jesse Mason

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Date: 1.8.18