# The effects of gluteus medius trigger points on hip passive range of movement and muscle strength in people with chronic non-specific low-back pain

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This thesis is submitted to AUT University in partial fulfillment of the requirements for the degree of the Master of Health Science (MHSc)

2021

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# Acknowledgements

I would like to thank my primary supervisor Associate Professor Richard Ellis for his time, commitment, and consistently positive, can-do attitude. I would also like to thank my secondary supervisor Susan Kohut for her in depth knowledge, kindness, and guidance. Thank you to Nick Garrett my biostatistician, who was invaluable in guiding me through the statistical analysis involved in my study. I would also like to thank César Fernández-de-las-Peñas; it has been very exciting having someone who is so prominent in the field of trigger point research show an interest in my study.

My partner Jono is always by my side and supportive of everything I do. Thank you for your love and enthusiasm. We welcomed our son Arlen into the world during my Masters. He has been the most wonderful baby; keeping me laughing, smiling and grounded. I would also like to mention my mother Anne, who is always my number one fan and even though is on the other side of the world; is always there for me and so encouraging. Thanks to all my family in Ireland; they are always so supportive and encouraging of my endeavors in life. I had amazing role models growing up in my grandmother Máire and my mother.

I would like to thank the community of Te Anau for it's amazing support of my research study. I was contacted by so many people enquiring about my study and have had so many caring messages regarding my progress with it. Having only lived here for a few years; I feel so supported and involved in the community.

# Attestation of Authorship

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

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29.10.21

## Abstract

Active trigger points (TrPs) are proposed to cause restricted range of movement (ROM) and muscle weakness. Simons and Travell pioneered TrP research with their publications of “The Trigger Point Manuals” (1983 & 1992); subsequently research into TrPs has informed knowledge of musculoskeletal disorders. The prevalence of TrPs in people with chronic non-specific low back pain (CNSLBP) is high; especially in the gluteus medius (GMed) muscles. CNSLBP is a complex and costly condition; for which treatment is not always successful. In this population, decreased hip range of movement (ROM) and hip muscle weakness are common findings. This study investigated if the presence of active GMed TrPs was associated with these deficits in the hips, when compared with participants with latent TrPs, and zero TrPs. Forty-two participants with CNSLBP underwent hip passive ROM and hip muscle strength testing; followed by palpation of their GMed muscles to identify the presence and types of TrPs. Analysis showed varying results regarding hip ROM and TrP status. There was an association between hip strength and TrP status. Participants with zero TrPs were the strongest and those with TrPs were weaker. In general, those with latent TrPs were the weakest. This study adds knowledge to the role that TrPs play in muscle strength and the characteristics of TrPs. This is significant for the treatment of CNSLBP and the development of more effective treatments of this multi-factorial condition.

# Chapter 1. Introduction

## 1.1 Statement of the problem

Musculoskeletal and rheumatic disorders are a major cause of disability worldwide (Vos et al., 2015). Low back pain (LBP) is the most common musculoskeletal disorder and is the leading cause of “years lived with disability” (Vos et al., 2015, p. 1551). This is also the case in New Zealand, where LBP is one of the most common musculoskeletal disorders (Tumilty, Adhia, Rhodes, & Mani, 2017). Increases in the prevalence of LBP have been observed globally, with an increase of 54% reported between 1990 and 2015 (Hartvigsen et al., 2018). Internationally, the prevalence of experiencing LBP ranges between 1.4 to 20% (Fatoye, Gebrye, & Odeyemi, 2019).

Chronic LBP (CLBP) is defined as LBP that persists or fluctuates for longer than three months (Accident Compensation Corporation, 2004; Last & Hulbert, 2009; Meucci, Fassa, & Faria, 2015; National Health Committee, 2015). The prevalence of CLBP worldwide ranges from 3.9% to 20.3% (Meucci et al., 2015). The most recent figures for the prevalence of CLBP in New Zealand is 9.1% (National Health Committee, 2015). Personal experience and psychosocial factors can contribute to the development of CLBP; with examples including distress, depressive mood, and job dissatisfaction (Raja et al., 2020). In some cases, as pain and disability continue and thoughts and beliefs about movement causing pain become solidified; long-term disability results (Last & Hulbert, 2009). CLBP related disability can present as functional, social, or psychological impairments; affecting the person’s well-being (Hartvigsen et al., 2018; Raja et al., 2020).

In New Zealand, CLBP is the biggest contributor to health loss (National Health Committee, 2015). Health loss is measured by assessing health lost from premature mortality and health lost from years lived with disability (Tobias, 2013). Disability due to CLBP leads to decreased workdays (National Health Committee, 2015). CLBP management is costly, with related costs for assessment, management, and social welfare benefit payments. For example, the estimated loss of income for people with CLBP in 2015 in New Zealand was $640 million (National Health Committee, 2015). Accident Compensation Corporation (ACC) funding is available for LBP caused by injury (Accident Compensation Corporation, 2021). ACC spends around $39.6 million per year on CLBP management. This includes physical therapies, complementary and alternative therapies, general practitioner, orthopaedic surgeons, and mental health services (National Health Committee, 2015). Non-ACC assessment, treatment, and management for CLBP is estimated to cost $180 million (National Health Committee, 2015). This

is a drastic increase from management of acute LBP (pain lasting less than three months); where the total reaches $65 million (National Health Committee, 2015).

LBP has been classified into three categories: non-specific LBP (NSLBP), suspected/ confirmed serious pathology, and radicular syndrome (Koes et al., 2010). NSLBP is the most common sub-group, representing 70% of CLBP diagnoses (Last & Hulbert, 2009; Maher, Underwood, & Buchbinder, 2016). NSLBP is defined as pain from an inconclusive source, where a diagnosis is not possible or useful (Bardin, King, & Maher, 2017). On presentation, there are several symptoms which are unable to be attributed to a single source and it is most likely the case that symptoms are a result of a variety of factors interacting with one another (Rose-Dulcina et al., 2018). The factors interacting with one another may be biomechanical, psychosocial, environmental, or physical (Maher et al., 2016; Rose-Dulcina et al., 2018). One pathology does not explain the presentation of symptoms and therefore, due to inconclusive differential diagnosis; a diagnosis on NSLBP is given (Rose-Dulcina et al., 2018)

In people who have a diagnosis of chronic NSLBP (CNSLBP), myofascial pain may be a source or contributor to the pain experienced, with the prevalence of myofascial pain being high (63.5%) in these people (C. K. Chen & Nizar, 2011). Myofascial pain along with fibromyalgia are amongst the most common musculoskeletal disorders worldwide (Evans, Behr, Gangwar, Noseworthy, & Kumbhare, 2021; Galasso et al., 2020; Shah et al., 2015). A musculoskeletal disorder is an injury to the human support system, e.g., ligament, tendon, bone, muscle, nerves, blood vessels, and joints (Hayes, Cockrell, & Smith, 2009; Hayes, Taylor, & Smith, 2012). Myofascial pain is defined as “pain of muscle origin in a painful site in muscle. This site is characterised by the myofascial trigger point” (Gerwin, 2001, p. 412). Myofascial pain is generally characterised by the presence of trigger points (TrPs); though this is not always the case (Shah et al., 2015). Fibromyalgia presents differently, it is a symmetrical pain condition with associated sleep and mood disturbances (Clauw, 2014). Myofascial pain can be felt at the site of a TrP and can be distributed regionally (Bourgaize, Newton, Kumbhare, & Srbely, 2018; Graven-Nielsen & Arendt-Nielsen, 2010; Sluka, 2016). Affected muscles can be painful to move, display subjective weakness, increased fatiguability, stiffness, and restricted range of movement (ROM) (Graven-Nielsen & Arendt-Nielsen, 2010). Decreased mood and quality life have also been linked to myofascial pain (Duarte, West, Linde, Hassan, & Kumbhare, 2021; Shah et al., 2015). Myofascial pain has physical, sensory and autonomic characteristics (Gerwin, 2001).

TrPs in the lumbo-pelvic region of the body are a common clinical finding in people with CLBP (C. K. Chen & Nizar, 2011; Iglesias-González, Muñoz-García, Rodrigues-de-Souza,

Alburquerque-Sendín, & Fernández-de-las-Peñas, 2013). On assessment of people with CNSLBP, gluteus medius (GMed) muscle weakness, and tenderness on palpation of the muscle belly reproducing the person's LBP was a common finding (Cooper et al., 2016). It is unknown if findings of muscle de-conditioning such as, weakness and increased fatigue are the cause (primary) or the result of symptomatic LBP (secondary) (Sadler, Cassidy, Peterson, Spink, & Chuter, 2019). Myofascial pain can be primary or secondary (Gerwin, 2001). In the case of primary, pain is not linked with any other conditions. An example of primary myofascial pain is an overuse injury in the upper limb, for example, lateral epicondylalgia. When myofascial pain is secondary, it is present in conjunction with other conditions, for example, chronic tension-type headaches. The person may have symptoms of nausea or light sensitivity along with active TrPs in the neck muscles (Gerwin, 2001).

The most frequently reported location for TrPs in people with CNSLBP is in the quadratus lumborum and GMed muscles (Iglesias-González et al., 2013). Decreased hip ROM (Van Dillen, Bloom, Gombatto, & Susco, 2008), along with decreased hip muscle strength (Cooper et al., 2016) are also prevalent findings in people with LBP. TrPs are proposed to prevent full lengthening of the muscle; therefore, restricting ROM (Gerwin, 2001; Simons, Travell, & Simons, 1999). TrPs are also proposed to cause weakness in the muscle they are present in (Gerwin, 2001; Simons et al., 1999). A direct link between TrPs within the muscles of the hips and deficits in movement and strength around the hips in people with CNSLBP has not been identified.

This study aimed to investigate the role of GMed TrPs in CNSLBP and their effect on hip ROM and muscle strength. CNSLBP sufferers represent a large proportion of people who experience CLBP (Last & Hulbert, 2009; Maher et al., 2016); but little is known about the role of TrPs in this patient group. It is known that TrPs are prevalent in this population (Iglesias-González et al., 2013). TrPs are linked to high pain levels, disability, and decreased quality of sleep (Iglesias-González et al., 2013); but there may be other links that have yet to be identified. Findings of restricted ROM and weakness in the hips potentially could be linked to the presence of TrPs (Simons et al., 1999); but thus far there is no research to confirm or refute this. CNSLBP sufferers are heavy consumers of the health service and due to chronicity of their condition, have high levels of disability and psychosocial issues (National Health Committee, 2015). Overall outcomes for musculoskeletal pain, in particular LBP have not improved; despite new clinical guidelines being produced and investment into ongoing research (Cook, Denninger, Lewis, Diener, & Thigpen, 2021). Further understanding about TrP mechanisms causing or contributing to CNSLBP would aid treatment and potentially make treatment more effective. Therefore,

theoretically decreasing the use of health care services by this patient population and increasing their ability to function in everyday life.

### 1.2 Research question and aims

The research question for this study was:

Is there a relationship between active gluteus medius trigger points and decreased hip passive range of movement and muscle strength in people with chronic non-specific low back pain?

The research aims were:

To establish if there was a relationship between active gluteus medius trigger points and

1) decreased hip passive range of movement

and

2) decreased hip muscle strength

in people with chronic non-specific low back pain.

### 1.3 Research hypothesis

The hypothesis of this study was:

People with chronic non-specific low back pain who have active gluteus medius trigger points will also display decreased passive range of movement and muscle strength in their hips.

### 1.4 Significance of the study

GMed TrPs are common in people with CNSLBP (Iglesias-González et al., 2013). It is plausible that myofascial pain caused by these TrPs may be a source or contributor to the person's LBP (C. K. Chen & Nizar, 2011). It is undetermined what effect the presence of GMed TrPs have on hip ROM and muscle strength. These are important measures in LBP, as deficits in these measures are common in CNSLBP and may contribute to or be a prerequisite to developing CNSLBP (Sadler et al., 2019). If a link could be established between the presence of GMed TrPs and deficits in hip ROM and muscle strength, it would enhance assessment and simplify treatment of these deficits. This would have consequences in prevention or treatment of many cases of CNSLBP. This is not a subject that has been researched; conclusions drawn from this study would influence the treatment of this condition. More information about the differing aspects contributing to the clinical presentation of CLBP would guide treatment methods in research, clinical management of LBP and inform clinical practice (Foster et al., 2018).

CNSLBP is difficult to assess and treat as the root cause of the condition is difficult to ascertain (Maher et al., 2016). Chronic pain is influenced in varying degrees from person to person by biological, psychological, and social factors (Raja et al., 2020). The multifactorial nature of CNSLBP highlights the complexity of this condition (Maher et al., 2016). Outcomes for treatment of have not improved over the past decade (Cook et al., 2021). This may reflect why CLBP patients continue to engage and heavily use the healthcare system in New Zealand. Costs to the health service and other health funded treatments are very costly ($219.6 million per year) (National Health Committee, 2015). Hence, this research demonstrates how more understanding around the myofascial component of CNSLBP, would aid assessment and treatment of this condition due to its high prevalence amongst those with CNSLBP. More effective treatment would reduce the burden and cost of this condition on the health service.

Presently, a wide range of treatment strategies are used for CNSLBP, as the mechanism for these deficits is unknown (Andersson, 1999; Last & Hulbert, 2009). GMed TrPs may be an essential focus for treatment in people with CNSLBP, enabling restoration of hip ROM and hip muscle strength, therefore, relieving pain and decreasing disability. This study may also have implications on CNSLBP prevention, as the presence of GMed TrPs and subsequent hip deficits could be prerequisites for developing CNSLBP (Almeida, de Souza, Sano, Saccol, & Cohen, 2012; Cejudo, Moreno-Alcaraz, Izzo, Santonja-Medina, & Sainz de Baranda, 2020).

In a systematic review of GMed function in people with and without LBP; alteration in GMed function were found in those with LBP (Sadler et al., 2019). Decreased GMed strength, hip abduction strength, and altered hip muscle recruitment were reported along with an increased prevalence of GMed TrPs in those with LBP when compared to those without LBP (Sadler et al., 2019). It is recommended that further research investigating the role of GMed, hip strength and TrPs would lead to more effective assessment techniques and management of LBP (Sadler et al., 2019). Treatment of CNSLBP may be greatly influenced if a link could be established between GMed TrPs and hip decreased ROM and strength. Conversely, more knowledge surrounding the role of GMed TrPs in this patient population may present new results and relationships between commonly found symptoms. Confirming or refuting the hypothesis of this study could greatly simplify diagnosis and treatment of many cases of CNSLBP.

### 1.5 Overview of the thesis

The first chapter of this thesis provides an overview of the prevalence and effects of LBP worldwide and in New Zealand. It explains the progression of acute LBP onto CLBP; and specifically, it describes and discusses the sub-group of CLBP sufferers who are categorised as

having CNSLBP. Furthermore, the role of myofascial pain, with the presence of TrPs as the source of NSLBP, is presented. The first chapter states the research question investigated in this research study. The aim and hypothesis are stated, and the significance of the study is presented.

The second chapter presents background information about defining CLBP and NSLBP. It describes the role of myofascial pain in CLBP and discusses different theories regarding TrP formation and how TrPs are a source of myofascial pain. Information on the different types of TrPs and specifically the prevalence of GMed TrPs and the role of GMed muscles is discussed in the context of CNSLBP. Current evidence is presented to link deficits in hip ROM and muscle strength in people with CNSLBP.

The third chapter explains the methodology used in this study. It provides information about the participants and recruitment process. The outcome measures used, including questionnaires, hip testing and GMed palpation protocols are explained and justified. The approach to statistical analysis that was used, and the processes used to analyse TrP data with passive range of movement (PROM) and muscle strength data, taking into consideration confounding variables are outlined and justified.

The fourth chapter presents the results of this study. This chapter begins with descriptive analysis of the TrPs identified in this study, confounding variables identified and used in this study, correlations between TrP data and PROM, and TrP data and muscle strength data.

This thesis concludes with the fifth chapter. In this chapter, the findings are discussed and interpreted. Limitations of the study are acknowledged and areas for future research are identified.

# Chapter 2. Background and literature review

## 2.1 Chronic non-specific low back pain

LBP is defined as pain experienced between the inferior rib margin and the gluteal folds (Cooper et al., 2016; Hardwick et al., 2019). LBP can be attributed to a specific source, however in most cases the exact source is inconclusive (Bardin et al., 2017; Rose-Dulcina et al., 2018). Sources of LBP can originate from spinal or musculoskeletal structures, for example, from on irritated spinal nerve root (causing radicular pain) (Engle et al., 2019), an intervertebral disc (causing discitis) (Bogduk, Aprill, & Derby, 2013; Manchikanti et al., 2001), facet joints (Manchikanti et al., 2001), or muscles (Fernández-de-las-Peñas & Dommerholt, 2018). When the source is inconclusive, LBP is diagnosed as NSLBP; as it is not linked with one specific pathology (Hardwick et al., 2019; Iglesias-González et al., 2013; Santos et al., 2013).

To help guide treatment, grouping of people with LBP is helpful (Bardin et al., 2017); despite this being a challenge, as there are diverse signs and symptoms from person to person (Wand & O'Connell, 2008). A diagnosis of NSLBP is given when classification of a spinal pathology (e.g., vertebral fracture) and radicular syndrome (e.g., nerve root impingement due to spinal stenosis) are excluded (Bardin et al., 2017). A biopsychosocial model of care is recommended to manage NSLBP. This includes addressing biological, psychological, and social contributors to symptoms (Bardin et al., 2017). The aim of treatment is to decrease symptoms and activity limitation (Bardin et al., 2017). Acknowledging the variety of factors interacting with one another optimises treatment outcomes; other factors such as environment, genetic, and cultural are also present (Rose-Dulcina et al., 2018). Studies have shown short-term pain relief following conservative manual treatment, but it is not known if this remains long-term (Tagliaferri et al., 2020). Not all contributing factors are biomechanical (Rose-Dulcina et al., 2018), therefore, treatment needs to address these accordingly (Tagliaferri et al., 2020).

CLBP is defined as LBP lasting for longer than three months (Accident Compensation Corporation, 2004; Andersson, 1999; Cooper et al., 2016; Last & Hulbert, 2009; Meucci et al., 2015; National Health Committee, 2015). Most acute LBP episodes will resolve but 30% of people continue to experience persistent symptoms, resulting in CLBP (E. Thomas et al., 1999). Factors that are considered to be strongly correlated with developing CLBP are: being female, a history of LBP, dissatisfaction with employment, widespread pain, radiating leg pain, and decreased lower back ROM as a result of the pain (E. Thomas et al., 1999).

There can be a range of physiological and psychological processes at play in a person with CNSLBP (Shemshaki, Amin Nourian, Fereidan-Esfahani, Mokhtari, & Reza Etemadifar, 2013). Sluka and George (2021) describes pain as “a personal experience” (p. 2). The experience of pain is a result of multiple factors in the peripheral and central nervous system including the brain cortex; as a result, there are different types of pain and pain experiences (Sluka & George, 2021; Tagliaferri et al., 2020). Examples of types of pain are nociceptive, nociplastic, and neuropathic pain (Chimenti, Frey-Law, & Sluka, 2018; Kosek et al., 2016). Nociceptive pain occurs when nociceptors are activated in the peripheral or central nervous system (Chimenti et al., 2018; Kosek et al., 2016). Activation of nociceptors sends signals via the spinal cord to the cortex in the ascending nociceptive pathway, signal processing occurs through all parts of the neuromatrix. Once central/cortical signals are interpreted; then acute pain can be felt, for example, an acute muscle soft tissue injury/strain in the lower back (Chimenti et al., 2018; Kosek et al., 2016). In a healthy individual, experiencing this type of acute pain; it is assumed that the somatosensory nervous system in functioning normally (Kosek et al., 2016).

Sensitisation is the underlying mechanism for nociplastic pain (Nijs et al., 2021). This type of pain is most common in chronic pain, for example, CNSLBP (Chimenti et al., 2018; Clark, Goodwin, & Yeowell, 2019). Sensitisation occurs peripherally and centrally in the nervous system (Fitzcharles et al., 2021). Centrally, hyperactivity in brain areas linked with pain is observed with decreased activity in areas linked with pain inhibition (Chimenti et al., 2018; Fitzcharles et al., 2021). There are elevated levels of substance P and glutamate concentrations in cerebrospinal fluid and reorganisation of the spinal cord (Fitzcharles et al., 2021). Peripherally, pH decreases and TrPs may form, there is an increased concentration of cytokines and chemokines. The causal factor is not always known (Fitzcharles et al., 2021). The result of this peripheral and central sensitisation causes hyperalgesia (increased pain response to painful stimulus) and allodynia (pain response to non-painful stimulus) (Fitzcharles et al., 2021). To be diagnosed with nociplastic pain, pain needs to have been present for longer than three months, it can’t be explained by nociceptive or neuropathic pain, the pain is regional, and there are clinical signs of hypersensitivity (e.g., hypersensitivity to hot/cold) (Nijs et al., 2021). Nociplastic pain can co-exist alongside nociceptive and neuropathic pain (Nijs et al., 2021). On assessment, pain appears disproportionate to the physiological processes that are occurring (Kosek et al., 2016; Nijs, Van Houdenhove, & Oostendorp, 2010).

Pre-morbid factors that predispose individuals to central sensitisation are emotional or physical trauma, decreased confidence, and lack of self-esteem (Clark et al., 2019). Psychological

processes can inhibit or facilitate the pain experience; affecting all three types of pain (Tagliaferri et al., 2020). Memory, emotion, and awareness, effect descending pain modulation pathways (Raja et al., 2020). People with CLBP display higher rates of depression and anxiety, when compared to those with acute or subacute LBP (Hüllemann et al., 2018). Pain is a subjective experience which results in differences in pain intensity, quality, and duration due to biological, psychological, and social factors (Raja et al., 2020).

Neuropathic pain results from nerve root involvement (Bardin et al., 2017). This term is a clinical description and further investigation may be warranted to give a diagnosis (Kosek et al., 2016). It can present in the form of radicular pain or radiculopathy (e.g., sensory disturbance, muscle weakness). This disruption to the nervous system can be due to a lesion, disease, or injury within the nervous system (Chimenti et al., 2018; Kosek et al., 2016). For example, spinal stenosis is a degenerative/congenital condition which can cause neuropathic pain due to narrowing of the foramen around the nerve root; causing it to become compressed (Bardin et al., 2017).

### 2.2 Myofascial pain

Myofascial pain is one of the most common musculoskeletal disorders worldwide (Evans et al., 2021). In most instances, myofascial pain is characterised by the presence of TrPs (Cao et al., 2021; Duarte et al., 2021; Evans et al., 2021; Shah et al., 2015). Though, generally the case, myofascial pain can also be present without the presence of TrPs (Shah, Phillips, Danoff, & Gerber, 2005). The association between myofascial pain and TrPs has been questioned (Fernández-de-las-Peñas & Dommerholt, 2018). Muscle and fascia are the most likely sources of pain (Shah et al., 2015); which can be felt local to an injury or within that region of the body (Bourgaize et al., 2018; Graven-Nielsen & Arendt-Nielsen, 2010; Shah et al., 2015; Sluka, 2016). Travell and Simons define a TrP as “a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band” (Simons et al., 1999, p. 5). There are different types of TrPs but active and latent are the most common (Donnelly, 2019). Travell and Simons differentiate between active and latent TrPs by attributing a clinical complaint to active TrPs; in that an active TrP can cause spontaneous pain which presents as the person’s clinical complaint or causes that pain on palpation. Latent TrPs are only painful on palpation and the pain is not familiar to the person as the pain they experience as part of their clinical complaint (Travell & Simons, 1992). Both are painful on palpation, though active TrPs are more sensitive and both can cause referred pain (Simons, 2001). Although, active TrPs can cause spontaneous pain, latent TrPs cannot (Simons, 2001).

Appropriate screening of patients with myofascial pain is pertinent, as TrPs may or may not be present; and if present, may not be part of the pain presentation (Kearns, Fernández-de-las-Peñas, Brismée, Gan, & Doidge, 2019). TrPs can be a result of some systemic autoimmune disorders, a side effect of certain medications, or due to visceral pathologies. Screening helps to decipher if the TrPs present are linked with myofascial pain or if a disease process is present (Kearns et al., 2019). There is potential for myofascial pain to become more widespread in the body (Bourgaize et al., 2018). This occur with chronicity, central and peripheral sensitisation, or accompanying conditions (Saxena, Chansoria, Tomar, & Kumar, 2015). Myofascial pain can be perpetuated by conditions such as joint osteoarthritis, ergonomic work-related activities, and medical conditions (e.g., hypothyroidism) (Saxena et al., 2015). It can also coincide with other pain disorders, for example, tendonitis, fibromyalgia, and joint pain (Cao et al., 2021; Shah et al., 2015).

The aetiology and pathophysiology of myofascial pain are not clearly understood (Bourgaize et al., 2018; Graven-Nielsen & Arendt-Nielsen, 2010). Myofascial pain is reported as being a major cause of LBP (Ramsook & Malanga, 2012) and/or contributor to LBP (C. K. Chen & Nizar, 2011); especially CNSLBP (Fernández-de-las-Peñas & Dommerholt, 2018). Several causal factors have been proposed, such as, injury, postural stress, unaccustomed eccentric contractions, and muscle overload are some examples (Graven-Nielsen & Arendt-Nielsen, 2010). Myofascial pain can occur in any region of the body (Bourgaize et al., 2018). The affected muscle can cause pain with movement, subjective weakness, increased fatiguability, stiffness, and decreased ROM (Graven-Nielsen & Arendt-Nielsen, 2010).

Myofascial pain is largely TrP related (Cao et al., 2021; Duarte et al., 2021; Evans et al., 2021; Shah et al., 2015; Stecco, Gesi, Stecco, & Stern, 2013). In other cases, pain is linked with constrictions in fascia, or disruptions to the nervous system, such as central sensitisation or increased activation of nerve receptors (Shah et al., 2015). Fascia is recognised as a potential pain source in myofascial pain; it glides over and is connected to all parts of the musculoskeletal system (Stecco et al., 2013). The fascia glides over muscle by the presence of the extracellular matrix, in particular a component called hyaluronic acid. Trauma, surgery, or over-use can cause changes to hyaluronic acid. Changes cause increased viscosity of the extracellular matrix environment and stiffening of the fascia. The fascia contains free nerve endings and stimulation of these nociceptors mechanically and chemically results in pain (Stecco et al., 2013).

In the case of TrP related myofascial pain; TrPs are proposed to be the source of localised and referred pain (Graven-Nielsen & Arendt-Nielsen, 2010). The progression from the presence

of TrPs to the experience of myofascial pain is poorly understood (Shah et al., 2005). The most widely accepted theory for TrP formation is the “Integrated theory” proposed by Simons and Travell (1981) (see section 2.3.1) (Bourgaize et al., 2018). Active TrPs cause spontaneous pain; upon palpation of a TrP, the pain felt is familiar to the person as their symptomatic pain. A latent TrP can contribute to pain but pain is only felt on palpation and is not familiar to the person as their symptomatic pain (Simons et al., 1999).

Shah et al. (2005) used a microdialysis needle to carry out in vivo biochemical analysis in human skeletal muscle; comparing the biochemical milieu present around TrPs (active and latent) and in normal muscle tissue. Acidic pH levels were lowest around active TrPs, an acidic environment is known to upregulate bradykinin, causing nociceptive activation without tissue damage being present (Shah et al., 2005). Highest concentrations of pro-inflammatory cytokines, monoamines, and inflammatory neuropeptides were found around active TrPs, followed by latent TrPs, and then normal muscle tissue (Shah et al., 2005). High concentrations of pro-inflammatory markers stimulate nociceptors which due to the axon reflex; promote the overproduction of neuropeptides such as substance P and calcitonin gene-related peptide (CGRP). These neuropeptides flow antidromically to the nociceptors; contributing to ongoing nociceptor stimulation (Shah & Gilliams, 2008). High concentrations of these neuropeptides have been found around active TrPs; contributing to peripheral sensitisation of local nociceptors (Shah et al., 2005).

Peripheral sensitisation leads to central sensitisation due to ongoing nociceptive bombardment through upregulation of the N-methyl-D-aspartate (NMDA) receptor within lamina II of the dorsal horn (Shah et al., 2005). Changes in ion channel activity leads to more receptive fields through activation of other receptive fields and the activation of ineffective synapses. Referred pain is proposed to be caused by the increase in the number of receptive fields (Shah & Gilliams, 2008). Protein kinase activation and gene induction lead to central hyperexcitability (Shah & Gilliams, 2008). The presence of peripheral and central sensitisation demonstrate the initiation of nociplastic pain processing (Nijs et al., 2021).

The presence of a TrP in a muscle can cause sensory and motor abnormalities. TrP motor-related abnormalities include muscle weakness and restricted ROM (Mense & Gerwin, 2010). These restrictions are due to muscle shortening and pain production with motion (Travell & Rinzler, 1952). There is an insufficient number of studies on muscle weakness due to TrPs, but it is purported that central motor inhibition is involved. Weakness is present without atrophy and is proposed to occur due to inhibition of muscle activity (Mense & Gerwin, 2010). Restricted ROM can occur with or without pain from a TrP as a limiting factor (Mense & Gerwin, 2010). Muscle

weakness and restricted ROM are attributed to the abnormal muscle structure (contracted sarcomeres (nodule) in a taut band) and therefore, altered muscle activation (Mense & Gerwin, 2010). Several studies have shown altered muscle recruitment and electromyographic activity in muscles that have TrPs (Florencio et al., 2017; Lucas, Polus, & Rich, 2004; Lucas, Rich, & Polus, 2010; Santos et al., 2013).

Autonomic changes that may present due to the presence of TrPs include sweating, flushing (temporary reddening of the skin), and increased pilomotor activity (Bourgaize et al., 2018). This is due to increased sympathetic nervous system activity because of increased levels of norepinephrine (NE) (Shah & Gilliams, 2008). Increased levels of NE have been found around active and latent TrPs (higher at active TrPs), when compared with normal muscle tissue (Shah et al., 2005). NE is involved in increased electrical activity at the motor endplate in the region of a TrP and decreased feedback regarding muscle length; both contribute to altered muscle function (Shah & Gilliams, 2008).

### 2.3 Historical context and theories related to trigger points

Muscle pain has been a source of discussion for centuries (Shah et al., 2015). Literature by Guillaume de Baillous (1538-1616) discussed muscle pain disorders. Nodules in muscle have been mentioned since 1816 (Balfour) and this discussion has continued in many subsequent publications, such as Froriep (1843), who noted the tight cord/band, Adler (1900) coining “muscular rheumatism”, and Gowers (1904), diagnosing fibrositis. Llewellyn and Jones (1941) were the first to write of tender nodules with radiating pain, but it was Travell and Rinzler in 1952 who first used the term myofascial trigger point (Simons et al., 1999).

Radiating pain from muscles either spontaneously or on palpation had been reported (Stockman, 1904). In the 1900’s, Kellgren injected hypertonic saline solution into tendon, muscle, and fascia, mapping out referral zones of pain. Kellgren reported diffuse muscle pain on palpation of tender spots often accompanied with referred pain (Kellgren, 1938b). He also concluded that referred pain from saline injections into muscle corresponded with spinal segmental motor nerve innervation patterns (Kellgren, 1938a). Travell and Rinzler also described referred pain from an active TrP having a particular distribution that is similar from person to person (Travell & Rinzler, 1952).

In the 1960’s, Janet Travell (1901 – 1997) collaborated with David Simons (1922 – 2010) to research the aetiology, pathophysiology, and clinical presentation of TrPs, culminating in the two volumes of Myofascial Pain and Dysfunction: The Trigger Point Manuals (Travell & Simons,

1983, 1992). It is argued that there is a correlation between TrPs and acupuncture points, as charting of both overlap (Liu, Skinner, Baxter, & McDonough, 2016). This correspondence has also been demonstrated in other publications. Dorsher (2008) reported a greater than 95% clinical correspondence between acupuncture points and TrP locations as described in the Trigger Point Manuals (Travell & Simons, 1983, 1992). Melzack, Stillwell, and Fox (1977) found a 100% anatomical correspondence and 71% clinical correspondence. In Traditional Chinese Medicine, "Ah-shi" points are painful on palpation; a similar characteristic of TrPs (Liu et al., 2016). This similarity is described in the Ling Shu (one of the original Chinese medical texts); where it describes pressing hard with a finger on a spot; if it is the right one; the patient will feel relief (Lu & Needham, 2002).

Travell and Rinzler (1952) reported a decrease in TrP pain after insertion of a needle. This practice became known as dry needling (Lewit, 1979). Travell and Simons also injected TrPs for pain relief (e.g., injections of procaine), known as wet needling (Simons et al., 1999). Dry needling emerged from this practice to become an accepted treatment for TrPs (Lewit, 1979). It involves inserting an acupuncture needle into a TrP (Kalichman & Vulfsons, 2010). This practice of inserting acupuncture needles into "Ah-shi" (painful spots) dates back to the 7$^{th}$ century AD; when Chinese physician Sun Ssu-Mo carried out this practice (Kalichman & Vulfsons, 2010; Lu & Needham, 2002). Injecting water into these points has also been described (Lu & Needham, 2002). Other types of dry needling have emerged. Intramuscular stimulation using dry needling into a taut band is proposed to result in resolution of symptoms (Gunn, 2003). Baldry (1995) pioneered superficial dry needling to stimulate subcutaneous tissue over a TrP to relieve local pain at the TrP and referred pain.

Gunn (2003) proposed the radiculopathy model of chronic pain to explain sensory, motor, and autonomic changes which present in myofascial pain. Instead of tissue injury being present, pain results from altered function of peripheral nerves from myofascial dysfunction. Peripheral nervous system changes result in central nervous system changes and pain is experienced despite no visible tissue injury being present (Gunn, 2003). Though, the generally accepted theory of TrP formation (and resulting symptoms of myofascial pain) is Simons and Travell's "Energy crisis" or "Integrated theory" (Simons & Travell, 1981). Theories about muscle recruitment and sequencing, refer to the Energy crisis as the pathophysiological process for TrP formation (Dommerholt, Bron, & Franssen, 2006). The effects of TrPs on the surrounding tissue is still debateable; biochemical, metabolic, and neurogenic theories have been purported (Shah et al., 2015).

### 2.3.1 Energy crisis and motor endplate theory (Integrated theory)

The earliest theory for TrP formation was proposed by Simons and Travell (Travell & Simons, 1983). They put forward the Energy crisis theory of TrP formation in 1981, along with the Motor endplate theory; together known as the Integrated theory. This theory developed from years of observatory clinical work alongside clinical electromyography (Simons, 1996). They postulated that when a muscle is overloaded/repetitively over-used, an excess amount of acetylcholine (ACh) is released at the motor endplate synapse. The activity at the motor endplate can be 10 to 100 times greater than normal (Simons et al., 1999). Calcium ions are released from the sarcoplasmic reticulum, causing continuous depolarisation at the post-synaptic membrane. This produces prolonged sarcomere shortening and greater cellular metabolism requirements. With ongoing sarcomere shortening, circulation and therefore, oxygen provision becomes compromised. Local sensory nerves are compressed from sustained contraction. Without sufficient oxygen and nutrients, cells cannot produce adenosine triphosphate (ATP) (Simons, 2001). Without ATP supplies, calcium reuptake to the sarcoplasmic reticulum is impaired producing an actin-myosin contraction of a sarcomere. This sequence of events continues, sustaining sarcomere shortening (Ramsook & Malanga, 2012).

Eventually, sarcoplasmic reticulum function becomes altered due to depleted ATP and the contracted muscle fibre remains contracted. When there are numerous contracted muscle fibres, a palpable nodule is formed (TrP) (Simons & Travell, 1981). The motor endplate theory furthers the hypothesis that myofascial TrPs produce local spontaneous electrical activity at the motor endplate. However, this is not sufficient to cause muscle contraction, but contributes to further ACh release causing contractile activity linked to shortening (Simons, 1987; Simons & Travell, 1981). The Energy crisis is self-perpetuating; neuropeptides released at the nociceptor terminals continue to stimulate an inflammatory cascade. Sensory nerves become activated, which activate sympathetic nerve endings; the release of ACh continues to be released from the motor endplate (Simons et al., 1999) (

Figure *1: Schematic presentation of the Integrated Theory).*

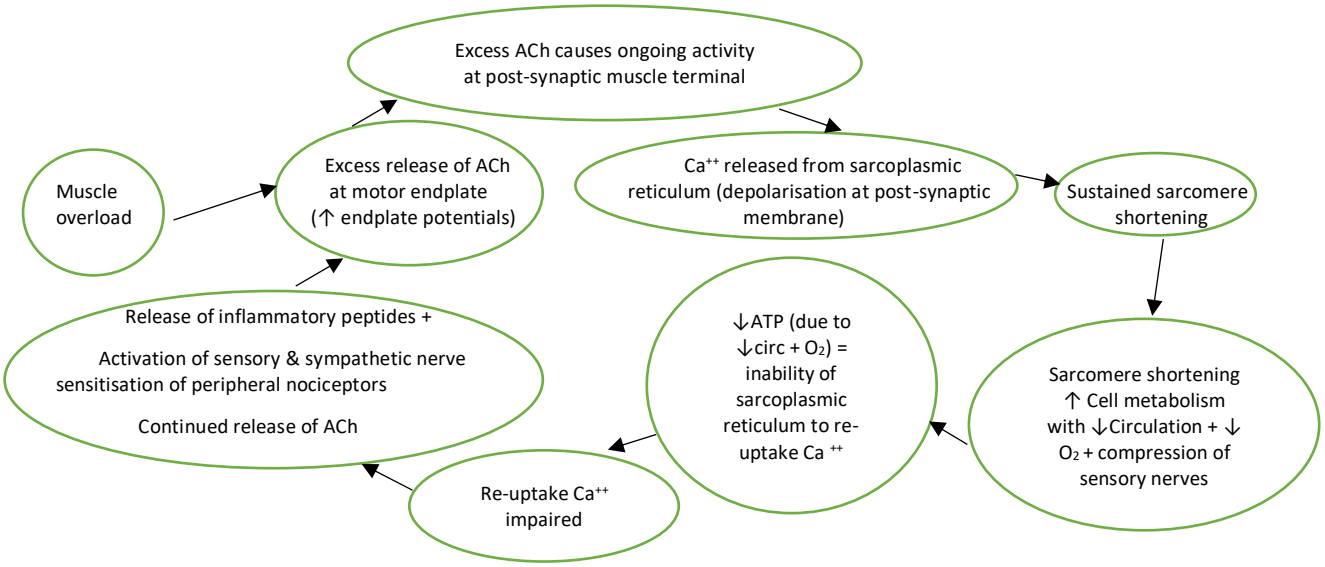



*Figure 1. Schematic representation of the Integrated Theory*

Spontaneous and increased electrical activity at the muscle endplate due to increased release of ACh has been found around TrPs in the trapezius muscle when compared to non-TrP tissue in the same muscle (Hubbard & Berkoff, 1993). This causes ongoing contractile muscle activity, which compresses local blood flow. Decreased blood flow (assessed using Doppler imaging) was observed around active and latent TrPs, although is more restricted around active TrPs in the trapezius muscles (Sikdar et al., 2009). Thus, causing an altered biochemical state, acidic, and hypoxic environment at and surrounding a TrP. Shah et al. (2005), carried out in vivo biochemical analysis in human skeletal muscle. They found pro-inflammatory peptides, cytokines, and metabolites accumulated at TrPs (Shah et al., 2005). Hence, muscle nociceptors are activated (half of muscle composition is made up of muscle nociceptors) (Willard, 2008).

The greater the number of active TrPs present, the greater the level of general sensitivity and sensitisation in the body (Palacios-Cena et al., 2017). This may be due to the higher amounts of algogenic substances and chemical mediators in the body, for example, tumour necrosis factor-α, interleukin-1β, CGRP, substance P, and bradykinin (Palacios-Cena et al., 2017; Shah et al., 2005). This is a self-perpetuating cycle with continued sensitisation and maintenance of sustained contracted sarcomeres in an acidic and hypoxic environment (Gerwin, 2005), leading to hyperalgesia around a TrP (Shah et al., 2005). Furthermore, Wall and Woolf (1984) cite

nociceptive C-fibres from muscle are more effective than those from cutaneous innervations to induce long-lasting changes in muscle behaviour.

### 2.3.2 Aetiology of trigger point formation

The Integrated theory is the generally accepted theory for TrP formation (Dommerholt et al., 2006). However, the aetiology for TrPs remains unclear. The “Cinderella Hypothesis” of muscle fibre recruitment during activity, originally observed by Hennemann in 1957 and further theorised by Hagg in 1988; suggests that Type I motor units are activated during repetitive, low force level activities and become overloaded, which creates an inflammatory response within the muscle. Biochemical markers change and TrP formation occurs due to the Energy crisis and increased motor endplate activity (Mense, 2009).

Smaller motor units are recruited first then larger ones, and the smaller ones are the last ones to switch off once the activity or movement has ceased (Hagg, 1991). Even at low activation levels, these smaller motor units are activated, for example, during sustained postural stances or very small ranges of repetitive movement (Hagg, 1991). Smaller motor units that are continually activated, become fatigued undergoing an Energy crisis, subsequently a TrP is formed (Kadefors, Forsman, Zoega, & Herberts, 1999). Based on the Integrated theory, sustained low-level contractions or repetitive contractions cause changes in the sarcoplasmic reticulum due to ischaemia and hypoxia, depleting ATP. An acidic environment is formed with an accumulation of calcium ions; resulting in TrP formation (Bron & Dommerholt, 2012).

The “Shift model” (motor rotation model) proposes a theory of TrP formation in postural muscles and tries to explain what pre-empts the Energy crisis (Minerbi & Vulfsons, 2018). At present, the “Shift model” is theoretical but does propose a physiological basis for conditions that predispose to the Energy crisis occurring (Minerbi & Vulfsons, 2018). The “Shift model” proposes that in tonic (postural) muscles (maintaining anti-gravity positions); the activation sequence is in rotation rather than in a sequenced order. Motor units are not recruited in a hierarchical pattern, for example, smaller then, larger as suggested in the “Cinderella Hypothesis”. Instead motor units are recruited in rotation; there is a sequential order of rotation (Minerbi & Vulfsons, 2018). Examples of tonic, postural muscles are the deep muscles of the neck, back, pelvis (including GMed), and calves (lower legs). A sequenced rotation of motor units contracting and relaxing allows the muscles to remain contracted for prolonged periods (Minerbi & Vulfsons, 2018). When a muscle is over-used or is involved in repetitive activities, overtime, the ratio between relaxation and contraction time becomes altered. This predisposes the motor unit to undergoing an Energy crisis (Minerbi & Vulfsons, 2018).

There are different types of TrPs; the most common are active and latent. Simons et al. (1999) specify that an active TrP “causes a clinical complaint” (p. 1). It produces spontaneous pain and pain on palpation. The pain experienced is familiar to the person as their pain complaint (Simons et al., 1999). Active TrPs are proposed to restrict full lengthening of the muscle and cause weakening. Pain can be referred and there is often accompanying autonomic changes (Simons et al., 1999). Whereas, Simons et al. (1999) describe a latent TrP as “painful only when palpated” (p. 4) and they do not produce spontaneous pain. It has all the other characteristics of an active TrP (Simons et al., 1999).

### 2.3.3 Contemporary perspectives

The majority of early TrP research was carried out by David Simons and Janet Travell in the early 80’s. More recently, research has been able to confirm the Energy crisis theory proposed by Simons and Travell (Simons, 1996). Sensory, motor, and autonomic changes occur due to the presence of TrPs (Simons et al., 1999). Sensory changes include referred pain, dysesthesias and hypesthesias. A TrP increases the tension in the muscle due to the formation of a taut band; thus, causing motor changes of stiffness, restriction of stretch ROM, and muscle weakness. Autonomic changes such as abnormal sweating and pilomotor activity can be present (Simons et al., 1999).

Decreased ROM was purported to be restricted due to pain as well as muscle stiffness (Simons et al., 1999). Pain is experienced, as muscle tension is already increased and therefore, is resistant to the stretch applied (Simons et al., 1999). Weakness is proposed to be caused by reflex motor inhibition. Muscles with active TrPs are weaker and fatigue quicker during activity; further decreasing strength (Simons et al., 1999). Both decreased ROM and muscle strength are due to altered muscle function. Surface and intramuscular electromyography show that there is disruption to normal muscle function at TrPs. This presents as muscle increased responsiveness, delayed relaxation, and premature fatiguability (Ge, Monterde, Graven-Nielsen, & Arendt-Nielsen, 2014; Lucas et al., 2004).

During isometric shoulder abduction at 90 degrees, muscle fibres around a latent TrP in the trapezius displayed impaired activation timing with delayed and incomplete muscle relaxation when compared with muscle tissue in the trapezius with no TrPs (Ge et al., 2014). Similar results were observed during arm elevation in the scapular plane when scapular muscles with latent TrPs were compared with controls. There was delayed activation and co-contraction with other muscles in other to achieve the movement in the latent TrP group, compared with stable and sequential muscle activity in the control group (Lucas et al., 2004). Differences in timing and variability were also observed in the same muscles in those with latent TrPs in another study (Lucas et al., 2010).

Florencio et al. (2017) found similar results with active TrPs in women with migraine. They displayed altered muscle activation patterns during craniocervical flexion. In chronic non-specific neck pain and CNSLBP; increased spontaneous activity at active TrPs was observed at rest with decreased activity during movement (Wytrazek, Huber, & Lisinski, 2011). Several muscles were assessed at rest and maximal contraction (including GMed); comparing TrP sites with non-TrP sites (Wytrazek et al., 2011).

Changes in muscle recruitment and activation timing when performing a task has also been observed in people with CLBP (Hemming, Sheeran, van Deursen, & Sparkes, 2019; Santos et al., 2013). During functional tasks such as sit to stand, reaching upwards, and bending over; there was high variability and no consistency in muscle activation patterns in the CNSLBP participants when compared with controls. The muscles assessed were a mixture of abdominal and spinal muscles in the lumbar area (Hemming et al., 2019). During kneeling to half kneeling, a large variability and inconsistency of muscle activation patterns were observed in women with CNSLBP in trunk and hip muscles (including GMed) (Santos et al., 2013). Surface electromyography showed increased muscle activity in the erector spinae muscles bilaterally during walking in those with CLBP, where pain lasted longer than 12 months (Manciopi, Rinaldi, & Moraes, 2017). Although, these studies did not assess the presence of TrPs, it is known that the prevalence is high in people with CNSLBP (C. K. Chen & Nizar, 2011; Iglesias-González et al., 2013).

Microanalysis using needle insertion has been carried out in the trapezius muscle at active, latent, and normal muscle tissue sites and confirms the higher concentration of biochemicals that are responsible for pain generation and inflammation at active TrPs, followed by latent, and then normal muscle tissue ($p<0.01$) (Shah et al., 2008). When a person has active TrPs, these biochemical changes are not only observed around the active TrP but also in other muscles in the body (Shah et al., 2008). Shah et al. (2008) carried out microanalysis in TrPs in the trapezius muscles and in normal (non-TrP) muscle tissue in the gastrocnemius muscles. In those with active TrPs, there were higher concentrations of analytes adjacent to active TrPs in the trapezius and remotely in gastrocnemius muscle, along with a lower pH. This may be due to central sensitisation causing a higher concentration of analytes systemically. Or, perhaps, some individuals could have a higher baseline of these analytes which predisposes them to TrP formation. Further research is required (Shah et al., 2008). Currently, we know that an Energy crisis occurs for a TrP to form but it is not clear if specific muscle fibres are more at risk or if certain muscle fibres are more susceptible via central sensitisation or the widespread increase of substances associated with

pain and inflammation. It has been postulated that some individuals may be more susceptible to TrP formation (Shah & Gilliams, 2008).

## 2.4 Clinical characteristics of trigger points

The most widely accepted contemporary definition for a TrP, based on Simons et al. (1999) work and proposed by Dommerholt and Fernández-de-las-Peñas (2013) is “a hyperirritable spot in a taut band of skeletal muscle that is painful on compression, stretch, overload or contraction of the tissue which usually responds with a referred pain that is perceived distant from the spot” (p. 3). TrPs can be located in fascia, tendons, or muscles (Ramsook & Malanga, 2012). There are several types of TrPs, the most common being active and latent (Gerwin, Dommerholt, & Shah, 2004; Simons et al., 1999).

There were four major criteria proposed for determining the presence of a TrP in a muscle. These are presence of a taut band, a hypersensitive nodule in the taut band, patient recognition of the pain produced by palpation of the nodule, and limited ROM with pain (Travell & Simons, 1992). Other findings that are used to confirm the presence of a TrP are a local twitch response (pain reaction), pain in the distribution of the TrP, and pain alleviated by stretching the muscle or injecting the TrP (Travell & Simons, 1992). The three most utilised criteria in the literature for diagnosis of a TrP are spot tenderness, referred pain, and local twitch response (Li et al., 2020). A high variability of criteria exists in TrP literature (Li et al., 2020).

### 2.4.1 Active versus latent trigger points

Both active and latent trigger points are sensitive on palpation, though active TrPs have higher sensitivity. This was shown in a study on women who experienced migraines and presented with TrPs in the neck muscles; sensitivity was highest at active TrPs (Palacios-Cena et al., 2017). In this study, participants who had active TrPs also had increased generalised sensitivity throughout their body when compared to those with latent TrPs only or no TrPs (Palacios-Cena et al., 2017). The biochemical changes that occur at a TrP are in higher concentrations at active TrPs when compared with latent TrPs (Shah et al., 2008). Latent TrPs have an altered biochemical milieu when compared with muscle tissue with no TrPs (Shah et al., 2008). This increased level of biochemicals at an active TrP may be the reason for generalised hypersensitivity, not only at the TrP but throughout the body (Palacios-Cena et al., 2017).

Both active and latent TrPs may contribute to muscle stiffness, causing restricted ROM and muscle weakness (Simons et al., 1999). Altered muscle functioning may be due to the sustained contracted position of sarcomeres; this shortened position does not allow the muscle

to function normally (Mense, 2010). Changes in muscle activation patterns may lead to muscle weakness and spasm (Lucas et al., 2010). Altered motor endplate activity is also thought to be responsible for spontaneous pain (Florencio et al., 2017). Active TrPs can cause spontaneous pain as well as pain on palpation (Gerwin et al., 2004; Simons et al., 1999). Latent TrPs do not cause spontaneous pain and are only painful on palpation (Simons et al., 1999). Studies have shown the presence of altered motor endplate activity at active and latent TrPs (Florencio et al., 2017; Ge et al., 2014). Using surface electromyography, altered activation during low-load isometric contractions was observed over active TrPs in the neck muscles in participants who experienced migraines (Florencio et al., 2017). Altered intramuscular activity has also been observed over latent TrPs in the trapezius muscles, using an intramuscular electromyographic needle. Increased activity was observed at rest and during isometric contraction of shoulder abduction at 90 degrees; in those with latent TrPs compared with those with no TrPs (Ge et al., 2014).

The significant difference between active and latent TrPs is that the pain produced by an active TrP is recognisable to the person as their symptomatic pain. This is not the case with latent TrPs (Fernández-Carnero, Fernández-de-las-Peñas, de la Llave-Rincón, Ge, & Arendt-Nielsen, 2007; Florencio et al., 2017; Simons et al., 1999). When active TrPs in the neck muscles were palpated in women who experienced migraine; palpation of active TrPs reproduced their migraine symptoms (Florencio et al., 2017). Active TrPs palpated in the forearm of those with lateral epicondylalgia reproduced the participant's symptoms (Fernández-Carnero et al., 2007). Palpation of latent TrPs also produced pain and referred pain; however, the pain was not familiar to the participants as their symptoms (Fernández-Carnero et al., 2007).

Active TrPs are more likely to be found in those who present with painful symptoms or clinical complaints (Fernández-de-las-Peñas, Alonso-Blanco, & Miangolarra, 2007; Fernández-de-las-Peñas, Cuadrado, & Pareja, 2007). Active TrPs were found in neck and shoulder muscles of elite swimmers who complained of shoulder pain when compared to swimmers who did not complain of shoulder pain. However, some non-symptomatic swimmers presented with latent TrPs on examination (Hidalgo-Lozano et al., 2013). This reflects the high prevalence of TrPs in non-symptomatic people. Lucas et al. (2004) had to review 154 non-symptomatic individuals to find 14 subjects with no TrPs in their scapular rotator muscles for their control group. Fernández-de-las-Peñas, Cuadrado, et al. (2007) compared those with episodic tension-type headache with controls who were asymptomatic, though all the control participants displayed latent TrPs in their neck muscles.

Latent TrPs can become active TrPs over time when the muscle is repetitively put under strain (Ge et al., 2014; Simons, 2001; Simons et al., 1999). It is proposed that as central sensitisation occurs and there is progressive involvement of the central nervous system in the pain experience; a latent TrP progresses into an active TrP (Mense, 2010). Pain modulating pathways in the central nervous system become more excitable and less inhibitory due to decreased threshold for activation peripherally at nociceptors (Chimenti et al., 2018). This process continues and becomes chronic due to increased concentrations of pain and inflammatory markers present (Raja et al., 2020), particularly in the muscle around the TrP (Shah et al., 2008) and sometimes throughout the body (Palacios-Cena et al., 2017). Central sensitisation is common in people with CNSLBP (Clark et al., 2019) and is thought to be the mechanism for progression of unilateral TrPs to bilateral TrPs (Fernández-Carnero, Fernández-de-las-Peñas, de la Llave-Rincón, Ge, & Arendt-Nielsen, 2008). In participants with unilateral elbow pain (lateral epicondylalgia), 88% had TrPs on the unaffected side. Those with unilateral lateral epicondylalgia also displayed decreased speed of movement during a motor task bilaterally (Bisset, Russell, Bradley, Ha, & Vicenzino, 2006). A decreased pain threshold bilaterally has been found in those with unilateral lateral epicondylalgia, suggesting a generalised mechanical hyperalgesia and decreased pain threshold due to central sensitisation (Slater, Arendt-Nielsen, Graven-Nielsen, & Wright, 2005).

Active and latent TrPs both produce referred pain (Fernández-Carnero et al., 2007; Florencio et al., 2017; Ge et al., 2014; Simons et al., 1999). Graven-Nielsen and Arendt-Nielsen (2008) define referred pain as “Pain perceived at a site adjacent to or at a distant from the site of origin” (p. 99). Referred pain can be somatic, visceral, or radicular in nature (Graven-Nielsen & Arendt-Nielsen, 2008). Nociceptive pain produces somatic pain. Nociceptors are activated due to tissue damage, producing somatic pain, for example, muscular injury (Shraim, Massé-Alarie, Hall, & Hodges, 2020). Radicular pain is a type of neuropathic pain. This pain can be referred due to a disease, lesion, or injury in the nervous system, for example, a compressed nerve due to a herniated intervertebral disc (Shraim et al., 2020). Active and latent TrPs produce somatic referred pain; peripheral and central sensitisation are suggested to be involved (Fernández-Carnero et al., 2007).

Peripheral sensitisation occurs due to the increased cascade of pain and inflammatory mediators, with concurrent inflammatory antidromic release causing neurogenic inflammation at a TrP (Shah et al., 2008). This continued noxious stimulus of nociceptors from a TrP produces a lowered threshold of nociceptive activation, with consequent overstimulation at the dorsal horn.

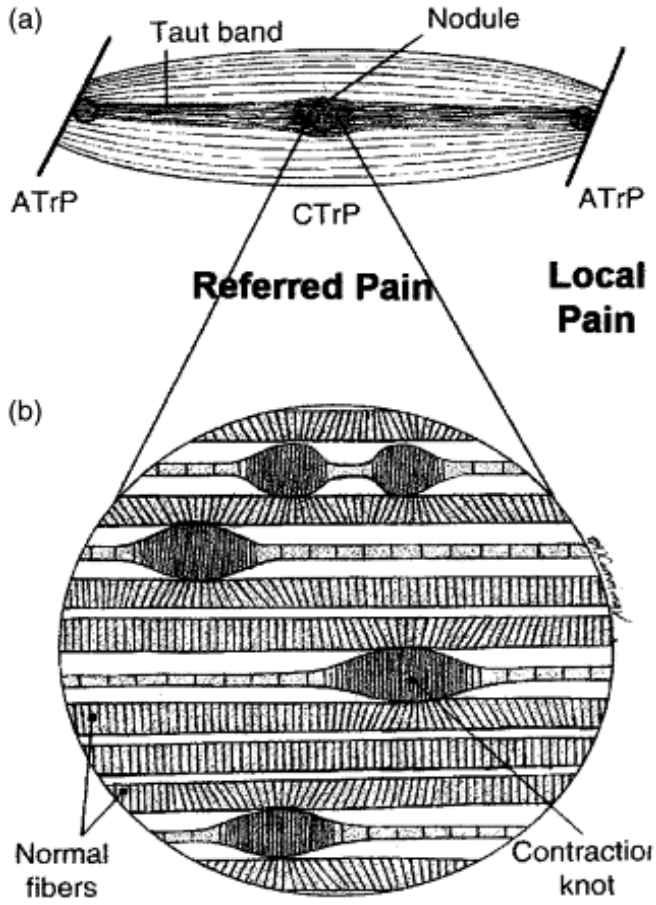
Substance P is one of these mediators and activates previously dormant NMDA spinal receptors (Mense, 2003). This creates more calcium permeable ion channels and increases efferent motor endplate activity. This change also induces apoptosis of inhibitory interneurons (Mense, 2003). Subsequently ascending neural pathways of the central nervous system are upregulated; causing alterations centrally and central sensitisation is induced throughout the neuroaxis (Graven-Nielsen & Arendt-Nielsen, 2008; Mense, 2003; Shah & Gilliams, 2008).

The referred pain caused by latent TrPs on palpation may be due to increased motor endplate activity at the TrP (Mense, 2010). This nociceptive activation sensitises the dorsal horn which in turn sensitises the central nervous system. Sensitisation of the dorsal horn activates ineffective synapses; which innervate other areas (Mense, 2010; Shah et al., 2015). This was demonstrated in rats whose inflammatory markers were increased following an injection of noxious substances. This led to activation of dormant synapses in the dorsal horn. Therefore, more neurons were involved in nociceptive transmission (Hoheisel, Koch, & Mense, 1994). The expanded connectivity at the dorsal horn led to central sensitisation and helps to explain referred pain in relation to TrPs and myofascial pain. Afferent nerve fibres have the ability to create new terminals, therefore, increasing the number of synaptic connections at the dorsal horn and increasing the receptive field for pain (Sperry & Goshgarian, 1993). TrP activity enhances activation of wide dynamic range neurons upregulating impulses via the spinothalamic tract to the brain, including the limbic system. The limbic system controls behaviour to pain with an emotional reaction. This is a major contributor to persistent chronic pain where there are emotions of fear and stress involved (Mense & Hoheisel, 2004). Therefore, the presentation of myofascial pain and TrPs is a mixture of mechanical and sensory characteristics due to neuromuscular dysfunction (Shah et al., 2015). This dysfunction leads to several other symptoms involving both the peripheral and central nervous system. These include allodynia, hyperalgesia, temporal summation, and expansion of receptive fields (Camanho, Imamura, & Arendt-Nielsen, 2011).

### 2.4.2 Trigger point identification

Palpation is the “gold standard” for identifying the presence of a hypersensitive spot in a taut band, the presence of which indicates a TrP (Barbero et al., 2012; Bron, Franssen, Wensing, & Oostendorp, 2007; Q. Chen et al., 2016; Hsieh et al., 2000; Lew, Lewis, & Story, 1997; Rathbone, Grosman-Rimon, & Kumbhare, 2017; Walsh, Kinsella, & McEvoy, 2017). The pain produced from palpation can be felt locally at the TrP or referred (Bourgaize et al., 2018). Sustained sarcomere contraction creates tension on subsequent sarcomeres in the muscle fibres, causing the formation

of a taut band. When sarcomere contraction occurs in a group of muscle fibres; a nodule can be palpated (Simons & Travell, 1981).




*(Simons, 2004b)*

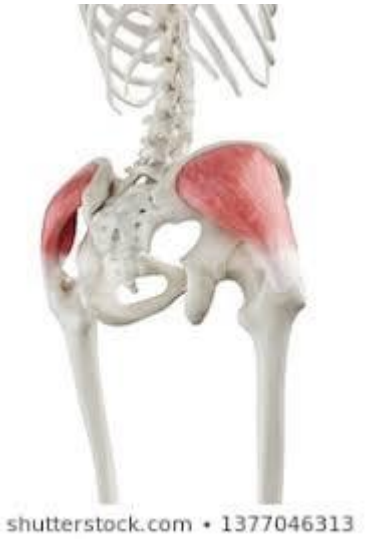
*Figure 2. Diagrammatic presentation of trigger points within a muscle*

## 2.5 Gluteus medius and related trigger points

### 2.5.1 Anatomy and function of gluteus medius

The GMed muscle is located on both sides of the pelvis. It runs between the lateral aspect of the iliac crest and the greater trochanter of the femur (Field, 2008) (Figure 3). GMed is the primary muscle for hip abduction (Arab & Nourbakhsh, 2010; Neumann, 2010). It is also a hip rotator, the anterior fibres of GMed are involved in internal rotation and the posterior fibres are involved in external rotation (Neumann, 2010). The main function of GMed is stabilisation of the pelvis during single leg stance when walking (Leetun, Ireland, Willson, Ballantyne, & Davis, 2004; Mense & Gerwin, 2010; Neumann, 2010; Travell & Simons, 1992). GMed produces compressive

forces across the hip to stabilise the pelvis over the fixed femur. It ensures the pelvis is level, particularly in single-leg stance allowing good lower limb alignment (Neumann, 2010); by preventing the pelvis from dropping on the opposite side (Field, 2008). GMed works in conjunction with the lower back muscles to provide support for the pelvis and lower back (Leetun et al., 2004; Travell & Simons, 1992). GMed and gluteus maximus work with multifidus, external obliques, and rectus abdominus to stabilise the trunk and hips (Gasibat, Simbak, Aziz, & Musa, 2017). The superior gluteal nerve which is innervated by spinal levels of L4-S1 innervates GMed (Tortora & Derrickson, 2012).

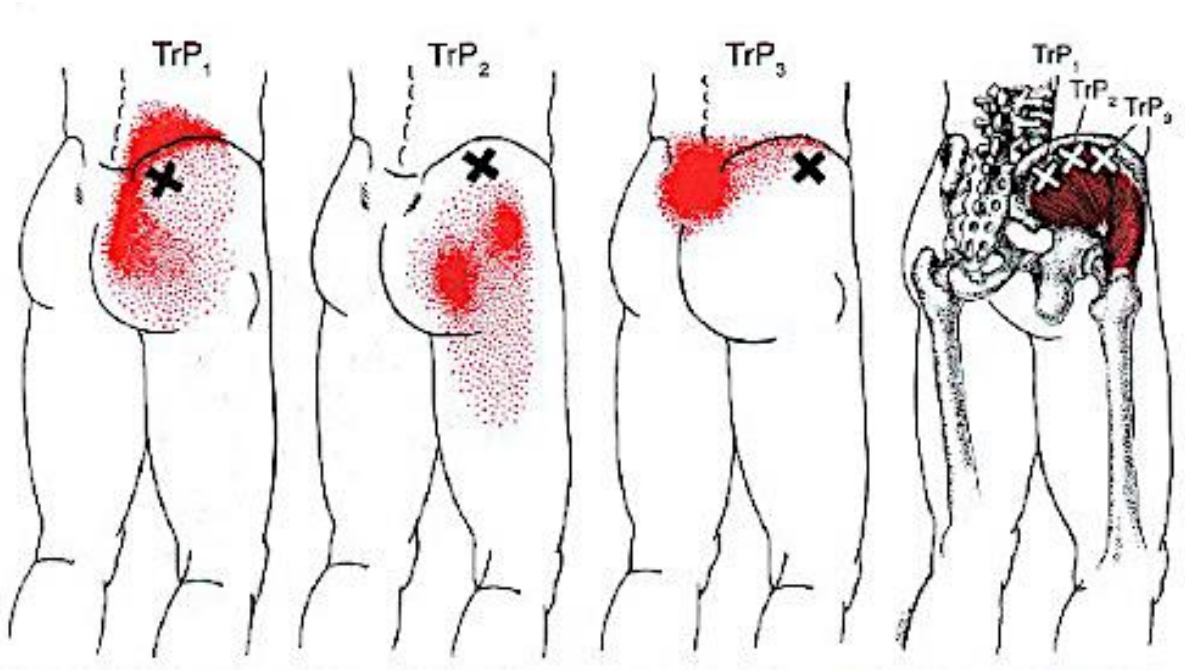



(Shutterstock, 2021)

*Figure 3. Right and left sided gluteus medius muscles*

### 2.5.2 Gluteus medius trigger points and trigger points of the lumbo-pelvic region

TrPs in the lumbo-pelvic region of the body are a common clinical finding in people with CLBP (C. K. Chen & Nizar, 2011; Iglesias-González et al., 2013). The lumbo-pelvic muscles include quadratus lumborum in the lower back and iliopsoas, piriformis, and the gluteal muscles around the hips (Dorado, López-Gordillo, Serrano-Sánchez, Calbet, & Sanchis-Moysi, 2020). The most frequent location of lumbo-pelvic TPs is reported to be within quadratus lumborum and GMed muscles (Iglesias-González et al., 2013). Active GMed TrPs are a common identified primary source of LBP; the referral pattern is across the sacrum, along the iliac crest, buttock, and upper thigh (Travell & Simons, 1992) (Figure 4).




(Travell & Simons, 1992)

*Figure 4. Referral pattern for active gluteus medius trigger points*

GMed TrPs can present bilaterally (Iglesias-González et al., 2013; S. M. Roach, Sorenson, Headley, & San Juan, 2013). From their study examining CNSLBP and looking at the presence of TrPs, Iglesias-González et al. (2013) reported that the prevalence of active GMed TrPs in people with CLBP was 35% on the more painful side and 38% on the less painful side; whilst healthy control participants had no active GMed TrPs (Iglesias-González et al., 2013). GMed TrPs also presented bilaterally in participants with patellofemoral pain (S. M. Roach, Sorenson, et al., 2013). The participants presented with unilateral patellofemoral pain but 87% of them displayed bilateral GMed TrPs (S. M. Roach, Sorenson, et al., 2013). It was recommended that assessment should include both sides (S. M. Roach, Sorenson, et al., 2013).

GMed TrPs increase pain and dysfunction in the lumbopelvic area in people with CNSLBP (Iglesias-González et al., 2013). As discussed, the presence of TrPs alters muscle function causing premature fatiguability (Hemming et al., 2019; Santos et al., 2013). For example, during kneeling to half kneeling, a large variability and inconsistency of muscle activation patterns were observed in women with CNSLBP in trunk and hip muscles (including GMed) (Santos et al., 2013). Applying this to GMed active TrPs, the GMed muscles are part of the lumbopelvic muscle support system (Leetun et al., 2004). If GMed fatigues prematurely; other muscles co-contract to compensate to carry out the task (Lucas et al., 2004). The continued Energy crisis and increased

motor endplate activity at active TrPs perpetuates the cycle of sustained sarcomere contraction (Gerwin, 2005). This can compound LBP whether GMed TrPs are the source of LBP or are a contributor to the pain (C. K. Chen & Nizar, 2011).

GMed TrPs can also be formed in response to lumbar pain originating in levels L4-S1 (their segmental innervation); for example, because of a disc prolapse causing segmental sensitisation (Samuel, Peter, & Ramanathan, 2007). It could also be the case that central sensitisation due to GMed TrPs, could increase pain perception in the segments innervated by these nerve roots (Samuel et al., 2007).

**2.6 Hip range of movement and muscle strength in people with CNSLBP**

As well as GMed TrPs being prevalent in those with CNSLBP (Iglesias-González et al., 2013); decreased hip ROM (Almeida et al., 2012; Van Dillen et al., 2008) and muscle strength (Arab & Nourbakhsh, 2010; Cooper et al., 2016) are common findings. Hip ROM is of interest to this study in relation to GMed TrPs, as GMed is a hip abductor and rotator (Neumann, 2010) and TrPs are theorised to cause decreased ROM in a muscle due to sarcomere shortening and pain (Mense & Gerwin, 2010). Sarcomeres adjacent to the TrP lengthen in order to try to maintain muscle length; overall this increases muscle tension and creates a taut band (Simons, 1987, 2004a). Pain is experienced when the shortened fibres are put under tension (Simons, 1987). The varying sarcomere length, along with the pain that is produced, limit the passive and active stretch of the muscle (Simons, 1987). Ultrasound may offer more insight into the structural properties of TrPs, currently this is mainly theoretic; hence further studies need to be carried out regarding TrPs role in ROM restriction (Srbely, Kumbhare, & Grosman-Rimon, 2016).

Asymmetry and variations in hip ROM are common findings in those with CNSLBP (Almeida et al., 2012; Cejudo et al., 2020; Vad, Gebeh, Dines, Altchek, & Norris, 2003; Van Dillen et al., 2008). Participants with CNSLBP who played racket sports, golf, and inline hockey had decreased hip rotation ROM and asymmetry between their right and left hips when compared with those without LBP (Van Dillen et al., 2008). Decreased active hip rotation ROM on the dominant side and decreased hip PROM on both sides was found in judo athletes who experienced LBP when compared with those without LBP (Almeida et al., 2012). Vad et al. (2003) found similar results when comparing tennis players with and without LBP. Those with LBP lasting longer than two weeks, had decreased hip ROM compared to those without LBP (Vad et al., 2003). In contrast, different results were found when inline hockey players were assessed by Cejudo et al. (2020); those who had experienced LBP over the previous year had increased hip external and internal rotation when compared with those players who did not experience LBP (Cejudo et al.,

2020). These studies show that participants with LBP displayed asymmetry and variations in their hip ROM when compared with those without LBP.

There are many factors that need to be considered when assessing hip ROM in relation to LBP. Gender is known to influence hip ROM; with females displaying increased total hip rotation (Hogg, Schmitz, Anh-Dung, & Shultz, 2018); therefore, both genders need to be assessed, in regards to cohorts with LBP, in order to draw more definite conclusions. For example, Vad et al. (2003), in their examination of tennis players with LBP (lasting longer than two weeks) compared with players with no experience of LBP only examined males. Additionally, only hip internal rotation was assessed; therefore, it's conclusions can only be applied to males and internal rotation. Most studies investigating the relationship between hip ROM and LBP have only assessed hip rotation ROM (Van Dillen et al., 2008). Some studies have also included hip flexion, extension, abduction, and adduction; however, the only movements that displayed significant differences between those with and without LBP were external and internal rotation (Cejudo et al., 2020). A limitation to studies exploring hip ROM in people with LBP is that the primary focus is on sports athletes and not the general public; which is the main demographic for LBP (National Health Committee, 2015).

Decreased muscle strength around the hips is a clinical finding in people with sub-acute and CLBP (Arab & Nourbakhsh, 2010; Cooper et al., 2016). GMed weakness was observed in 40-45% of CLBP sufferers (Cooper et al., 2016). People with LBP and iliotibial tightness displayed decreased hip abductor strength when compared with people without LBP (Arab & Nourbakhsh, 2010). It has been shown that muscles with TrPs display motor dysfunction e.g. weakness, inhibition, spasm, and muscle imbalance (Lucas et al., 2010). In scapular muscles with latent TrPs, there was a variability of muscle activation times in an unloaded and loaded state (Lucas et al., 2010). This was compared with a control group without TrPs who demonstrated an ordered activation of muscles when abducting the arm. The variability did not increase with load; therefore, even in an unloaded state, the presence of latent TrPs caused sub-optimal muscle activation. Therefore, even the presence of latent TrPs only, leads to muscle weakness and fatigue, spasm and muscle imbalance (Lucas et al., 2010).

### 2.7 Summary

This study investigates the relationship between active GMed TrPs and changes in hip PROM and muscle strength in people with CNSLBP. Active GMed TrPs, decreased hip ROM and weakness are all clinical findings in people with CNSLBP. The identification of a relationship between these findings could explain why these observations are found and guide future

treatment. Thus, changes induced in a muscle due to the presence of TrPs leads to the hypothesis that people with CNSLBP who have active GMed TrPs will also display decreased hip PROM and muscle strength in the directions of abduction and rotation (internal and external).

GMed TrPs are very common in people with LBP (C. K. Chen & Nizar, 2011; Iglesias-González et al., 2013) and clinically it would be useful to determine whether they are related to changes in hip PROM and muscle strength. No other study investigating the role of GMed TrPs in CNSLBP has been identified by the researcher. Other studies have investigated hip PROM, hip muscle strength, and GMed TrP prevalence in CNSLBP as separate entities. This study investigates the relationship between these common clinical findings in this patient group. The effects of TrPs on surrounding tissue is still debateable (Simons, 2004a); the factors initiating and perpetuating TrP related myofascial pain has been presented. The research question this study poses is very clinically relevant; as LBP prevalence is projected to further increase (Hartvigsen et al., 2018). TrP research is still emerging and evolving and continued exploration into this subject matter is required (Srbely et al., 2016). The requirement for more research into muscle TrPs and their effects has never been more apparent (Cao et al., 2021).

# Chapter 3. Methodology

## 3.1 Study design

This study was designed to investigate the relationship between active GMed TrPs and deficits in hip PROM and muscle strength in people with CNSLBP. The study was cross-sectional in design. It included participants with CNSLBP who had active GMed TrPs compared with participants with CNSLBP who did not have active GMed TrPs (i.e., had latent TrPs or zero TrPs). Convenience sampling was used to recruit participants. Participants attended a single assessment session which included: three questionnaires, assessment of hip PROM and muscle strength; and GMed palpation on both sides (to assess for the presence of GMed TrPs). Participant demographics were obtained (Figure 5: Study design).

## 3.2 Participants

Participants were recruited from the communities of Te Anau and Winton in Southland, New Zealand. The primary researcher (MC) was based in Te Anau; Winton is the nearest large town to Te Anau in Southland and is a one-and-a-half-hour drive away. Advertising was achieved through flyers at the medical centre, library, grocery store, and physiotherapy clinics in Te Anau and Winton. In Te Anau, a flyer was also shown at the pharmacy and an advertisement was placed in the local magazine *"Te Anau Trader".* In both Te Anau and Winton, the study was advertised on the local community Facebook pages.

The primary researcher did an in-service at the two physiotherapy clinics in Te Anau and the physiotherapy clinic in Winton. The in-service informed the staff about the study (including recruitment and methods) and provided the staff with the latest research and knowledge regarding TrP formation and the role of myofascial pain in LBP. The primary researcher also spoke to medical staff including general practitioners and nurses at the Te Anau Medical Centre to inform them about the study and gave them flyers to give to potential participants.

One hundred and fifteen people from Te Anau and six people from Winton contacted the primary researcher. Forty-six participants who met the inclusion criteria and did not meet any of the exclusion criteria were recruited between October 2019 and July 2020. All communication with participants was carried out by the primary researcher.

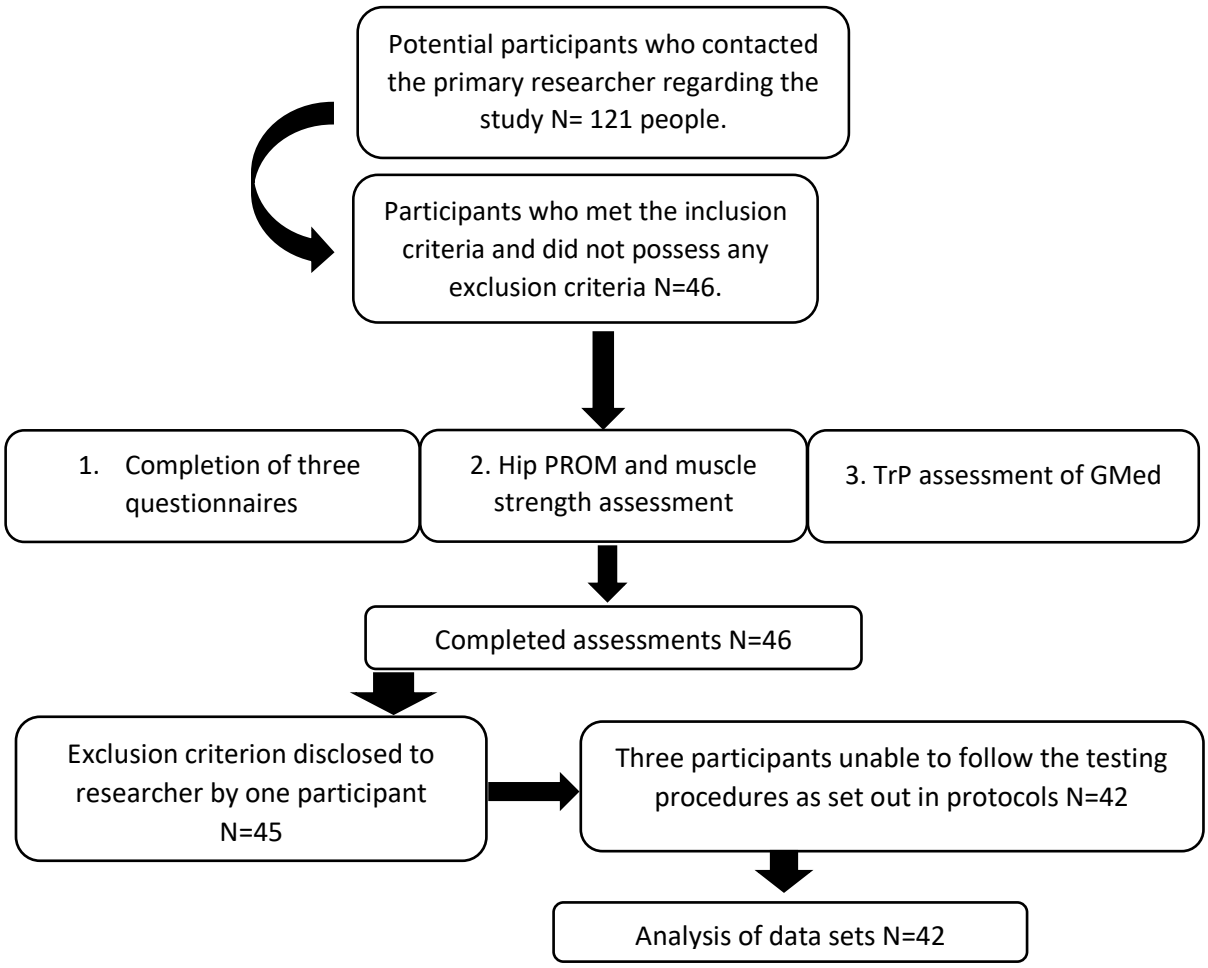



*Figure 5. Study design*

### 3.2.1 Inclusion and exclusion criteria

CLBP was defined as LBP lasting for longer than three months (Accident Compensation Corporation, 2004; Andersson, 1999; Last & Hulbert, 2009; Meucci et al., 2015; National Health Committee, 2015); between the inferior rib margin and the gluteal folds (Cooper et al., 2016). NSLBP relates to pain not linked with a specific pathology (Bardin et al., 2017; Iglesias-González et al., 2013; Santos et al., 2013). Participants were eligible to participate in the study if they had LBP lasting from three months or greater and were 18 years or older; therefore, able to consent to participate in the study.

People were excluded from partaking in the research if they met any of the following criteria: pregnant, diagnosed with a specific pathology causing their LBP, (e.g., cauda equina

syndrome, fracture, tumour, infection, herniated disc, inflammatory disorder (e.g., rheumatoid arthritis)), undergone spinal surgery, current knee pain, hip pain; a medical diagnosis of hip or knee osteoarthritis; neurological symptoms in their lower limbs; or a diagnosis of fibromyalgia.

Spinal surgery was an exclusion criterion, as having undergone spinal surgery is suggestive of a specific pathology in the spine (Cooper et al., 2016). This study was investigating people with CNSLBP; therefore, the exact cause of their LBP was unknown (Bardin et al., 2017). Knee pain was selected as an exclusion criterion as it could affect hip muscle strength (Ireland, Willson, Ballantyne, & Davis, 2003; S. M. Roach, San Juan, Suprak, Lyda, & Boydston, 2014; S. M. Roach, Sorenson, et al., 2013). For example, those with anterior knee pain (e.g., patellofemoral pain) display decreased hip strength in the directions of abduction and external rotation when compared with those without knee pain (Ireland et al., 2003; S. M. Roach et al., 2014; S. M. Roach, Sorenson, et al., 2013). People with patellofemoral pain also have a higher prevalence of bilateral GMed and quadratus lumborum TrPs when compared to those without knee pain (S. M. Roach, Sorenson, et al., 2013). The focus of this study was TrPs in relation to CNSLBP and their effect on the hips; therefore, if participants also experienced knee pain; it would be difficult to differentiate if the presence GMed TrPs and deficits in hip muscle strength were in relation to their knee pain or CNSLBP. This is also similar to hip OA, where an increased number of latent TrPs are found in the muscles around the hips (Bajaj, Bajaj, Graven-Nielsen, & Arendt-Nielsen, 2001).

Neurological symptoms (for example, pins and needles or numbness) in the lower limbs were also chosen as exclusion criteria. There can be multiple causes of lower limb neurological symptoms, such as, central sensitisation, denervation, peripheral nerve sensitisation, or nerve compression (e.g., due to an intervertebral disc) (Schäfer, Hall, & Briffa, 2009). In these instances, neurological symptoms can potentially be linked to specific pathoanatomical causes, whilst the focus of this study was NSLBP. It is important to make a distinction between fibromyalgia and myofascial pain. In fibromyalgia, there is widespread tender points of unknown cause with central nervous system involvement (Bourgaize et al., 2018). This is in comparison to myofascial pain, where an Energy crisis and increased motor endplate activity have occurred, resulting in TrP formation. However, central sensitisation may be present as myofascial pain progresses overtime (Bourgaize et al., 2018).

### 3.2.2 Sample size calculation

Calculations and statistical analysis were carried out in consultation with a biostatistician. Studies were identified that also investigated GMed muscle strength in people with CLBP in

relation to movements including hip abduction (Cooper et al., 2016) and internal and external hip rotation (Van Dillen et al., 2008) were used as references when estimating the sample size required for this study. These studies were then considered when estimating the amount of statistical power required to show significant results in this research study. An estimated sample size of 43 participants was calculated as the most optimal for both muscle strength and ROM outcomes.

For muscle strength, with 80% power and 95% significance, a difference of 0.7% in muscle strength is detectable using a standard deviation of 0.7 (as per Cooper et al. (2016)). For assessment of hip ROM in CLBP sufferers, specifically internal and external rotation ROM, with 80% power and 95% significance, a difference of -12° can be detected for external rotation; using a standard deviation of 11 (Van Dillen et al., 2008). For internal rotation, a difference of -18° can be detected using a standard deviation of 17 (Van Dillen et al., 2008).

### 3.3 Ethical and cultural considerations

Auckland University of Technology (AUT) Ethics Committee granted approval to the research study on 25th September 2019 (19/216) (Appendix A). Prior to ethics submission, there was consultation with the Mātauranga Māori Research Committee (School of Clinical Sciences) at AUT. This consultation guided the primary researcher on ways to ensure that the research content was culturally sensitive and ensure the research approach was holistic. The primary researcher met with a Māori kaumātua (respected elder) in the community of Te Anau. He provided information regarding cultural sensitivity in relation to the questionnaires and assessment techniques, and ensured they were appropriate for Māori. To ensure participants could give informed consent to participate, they were provided with a Participant Information Sheet (Appendix B) to read, prior to their assessment. Participants were advised that they could bring a support person with them to their assessment if they wished. It was made clear that participation in the research study was voluntary.

All participants were given the opportunity to ask questions prior to the commencement of their assessment. If they agreed to go ahead with the assessment, they completed a Consent Form (Appendix C). Demographic information was collected from the participants. This was the only form with the participant's personal details. On all subsequent paperwork the participant was referred to by an assigned designated number. All paperwork was stored in a locked cabinet at Fiordland Physiotherapy clinic, Te Anau which could only be accessed by the primary researcher. All paperwork was uploaded onto a hard-drive and will be stored for six years. Paperwork was shredded after it was uploaded onto the hard drive.

## 3.4 Study procedure

### 3.4.1 Study process

Advertising for the study was distributed within the Te Anau and Winton communities (see 3.2). Potential participants contacted the primary researcher by email or phone. The primary researcher spoke with each potential participant over the phone, explained the study and answered any questions they had. She also screened them for inclusion and exclusion criteria. If the potential participant met the eligibility criteria, they were given or emailed a Participant Information Sheet (Appendix B) with information about the study and an assessment day and time was arranged.

The participants attended at Fiordland Physiotherapy clinic in Te Anau or Central Physio, Winton for their assessment. They were shown the Participant Information Sheet (Appendix B) again to confirm the information contained and to answer any questions the participants had. They then completed the Consent Form (Appendix C). Subsequently, they completed a Personal Details Form (Appendix D) collecting demographics, such as age, gender, and ethnicity. The location of their LBP was also recorded by the participant marking the location of their LBP on a body chart. The participant was assigned a designated number on this form. All subsequent paperwork was assigned this number and no personal information was used. The participant then completed the three questionnaires (see 3.5.5). The primary researcher carried out PROM assessment of the hips; followed by hip muscle strength assessment on the right and left sides. The TrP examiner (see 3.5.4) carried out the palpation assessment on the right and left GMed muscles. A run sheet was used to ensure that all aspects of the assessment were completed, and all paperwork was completed. At the end of the assessment, the participant was given a $20 koha (grocery store voucher) to thank them for their time. The full assessment took approximately 65 minutes.

### 3.4.2 Blinding

Two levels of assessor blinding were conducted. First, the primary researcher conducted the hip PROM and strength measures blind to the TrP status of each participant. A separate assessor (CL) conducted the TrP assessment for each participant blind to the outcome of the hip PROM and muscle strength measures. Secondly, to avoid hip PROM results influencing the primary researcher during the three repetitions of each movement, the screen of the digital inclinometer was covered. The measurements were stored on the screen and written down after the three repetitions of the movement had been carried out.

## 3.5 Outcome measures

Demographic variables and primary outcome measures of hip PROM, hip muscle strength, and GMed trigger point status were collected.

### 3.5.1 Demographic variables

Participants completed a Personal Details Form which collected the following information: name, address, contact number, date of birth, gender, cultural background, and occupation. There was also a body chart, on which the participant marked the location of where they experienced their LBP.

### 3.5.2 Hip passive range of movement

One of the aims of this study was to explore the relationship between active GMed TrPs and hip PROM. To achieve this, PROM for abduction, external rotation, and internal rotation were assessed in both the right and left hips. These three movements were chosen as GMed is involved in all of these movements (Neumann, 2010). Three PROM measurements were recorded in each direction, holding the end position for three seconds, and the average of the measures was used in the data collection (Charlton, Mentiplay, Pua, & Clark, 2015; S. M. Roach et al., 2015; S. M. Roach et al., 2014; Van Dillen et al., 2008). The movement ceased at the point at which a firm or stiff feeling was felt by the primary researcher. This point is referred to as “end feel” (Pua, Wrigley, Cowan, & Bennell, 2008). Movement was also ceased if compensatory movement has observed or felt by the primary researcher, for example, pelvic rotation during hip rotation (Van Dillen et al., 2008).

The sequence of movements was right adduction, left external and internal rotation, right external and internal rotation, and left adduction. The sequence of movements was chosen to minimise the positional changes for the participants. Hip adduction PROM was chosen to assess for restrictions in hip abduction ROM. These movements have an agonist/antagonist relationship. As the hip is adducted, hip abduction muscle length increases; therefore, any restrictions in abduction will limit hip adduction (Welsh, Howitt, & Howarth, 2020).

To familiarise the participant to the movements of external and internal rotation; the lower limbs were passively moved once in each of these directions (Van Dillen et al., 2008). The participants could position their arms and head in any position that was comfortable for them (Van Dillen et al., 2008). An Easyangle digital inclinometer (Meloq, Sweden) was used to record the angle of PROM (**Error! Reference source not found.**). Digital inclinometry has been commonly u

sed to assess hip PROM in people with LBP (S. M. Roach et al., 2015; Takashi et al., 2015; Van Dillen et al., 2008) and without LBP (Hall & Smith, 2018; S. M. Roach et al., 2014).

When assessing ROM at different joints in the body, digital inclinometry was found to be a reliable measure (Fraeulin et al., 2020). More specifically for hip PROM, it has been shown to be efficient and reliable for assessing internal rotation (Krause, Hollman, Krych, Kalisvaart, & Levy, 2015; S. M. Roach, San Juan, Suprak, & Lyda, 2013), hip extension, and external rotation (S. M. Roach, San Juan, et al., 2013).

To ensure blinding of the primary researcher and to minimise the influence of knowing the result of each movement; the screen of the digital inclinometer was covered (Figure 6). The device beeped when zeroed, the PROM was performed to “end feel” and the button was pressed to store the number of degrees; following which a beep was heard to confirm data storage. The measurements were stored on the screen of the digital inclinometer. After the three movements were carried out, the screen was uncovered, and the three measurements were recorded on the PROM assessment sheet (Appendix E).

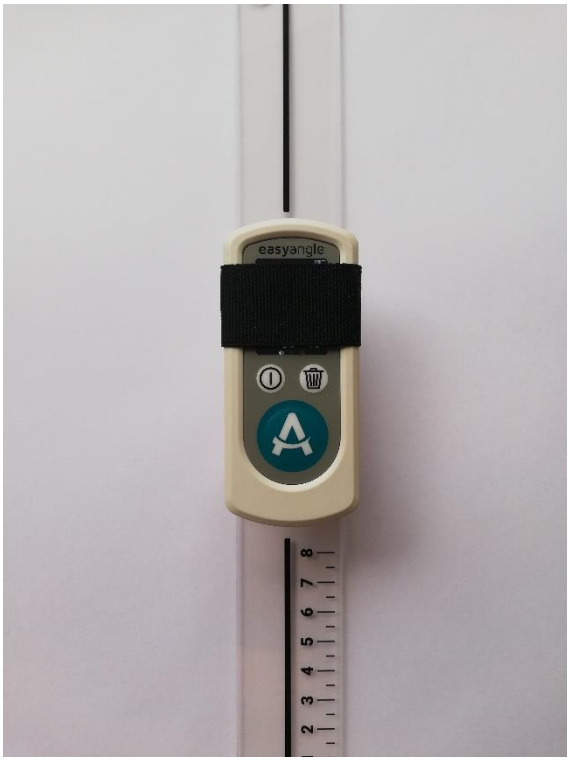



*Figure 6. Digital inclinometer with screen covered*

Hip adduction of the bottom leg was carried out in side-lying; to assess for restrictions of abduction in the bottom hip (Charlton et al., 2015; Hall & Smith, 2018). The top leg was flexed at the hip and knee to 90° (Charlton et al., 2015; Hall & Smith, 2018); resting on pillows to maintain neutral hip position. The digital inclinometer was placed mid-way on the posterior aspect of the femur; in line with the femur; knee extended (Charlton et al., 2015) (Figure 7A). The bottom leg was then adducted passively by the researcher (Charlton et al., 2015) (Appendix F: Protocol for PROM testing).

External and internal rotation PROM were carried out with the participants in prone lying on the plinth (S. M. Roach et al., 2015; S. M. Roach et al., 2014). A stabilisation belt was placed around the pelvis to prevent rotation (Van Dillen et al., 2008) (Figure 7B). The digital inclinometer was placed on the anterior aspect of the tibia along the axis of the tibial shaft; 9.5cm from the tibial tuberosity (Krause et al., 2015; Takashi et al., 2015). The inclinometer was zeroed before each movement; ensuring the tibial plateau was parallel to the plinth (Van Dillen et al., 2008).

For external rotation, the leg being tested was passively flexed to 90° at the knee (S. M. Roach et al., 2015; S. M. Roach et al., 2014; Van Dillen et al., 2008). The femur (and subsequently the hip joint) was rotated externally/outwards; until an end feel was felt (S. M. Roach et al., 2015; S. M. Roach et al., 2014; Van Dillen et al., 2008). If participants had a lot of external rotation range, the non-tested leg was abducted to provide more room (S. M. Roach et al., 2015; S. M. Roach et al., 2014). The testing was carried out on both sides (Figure 7B). For internal rotation, the knee was again flexed to 90° (S. M. Roach et al., 2015; S. M. Roach et al., 2014; Van Dillen et al., 2008). The femur (and subsequently the hip joint) was rotated internally/inwards (S. M. Roach et al., 2015; S. M. Roach et al., 2014; Van Dillen et al., 2008) (Figure 7C).

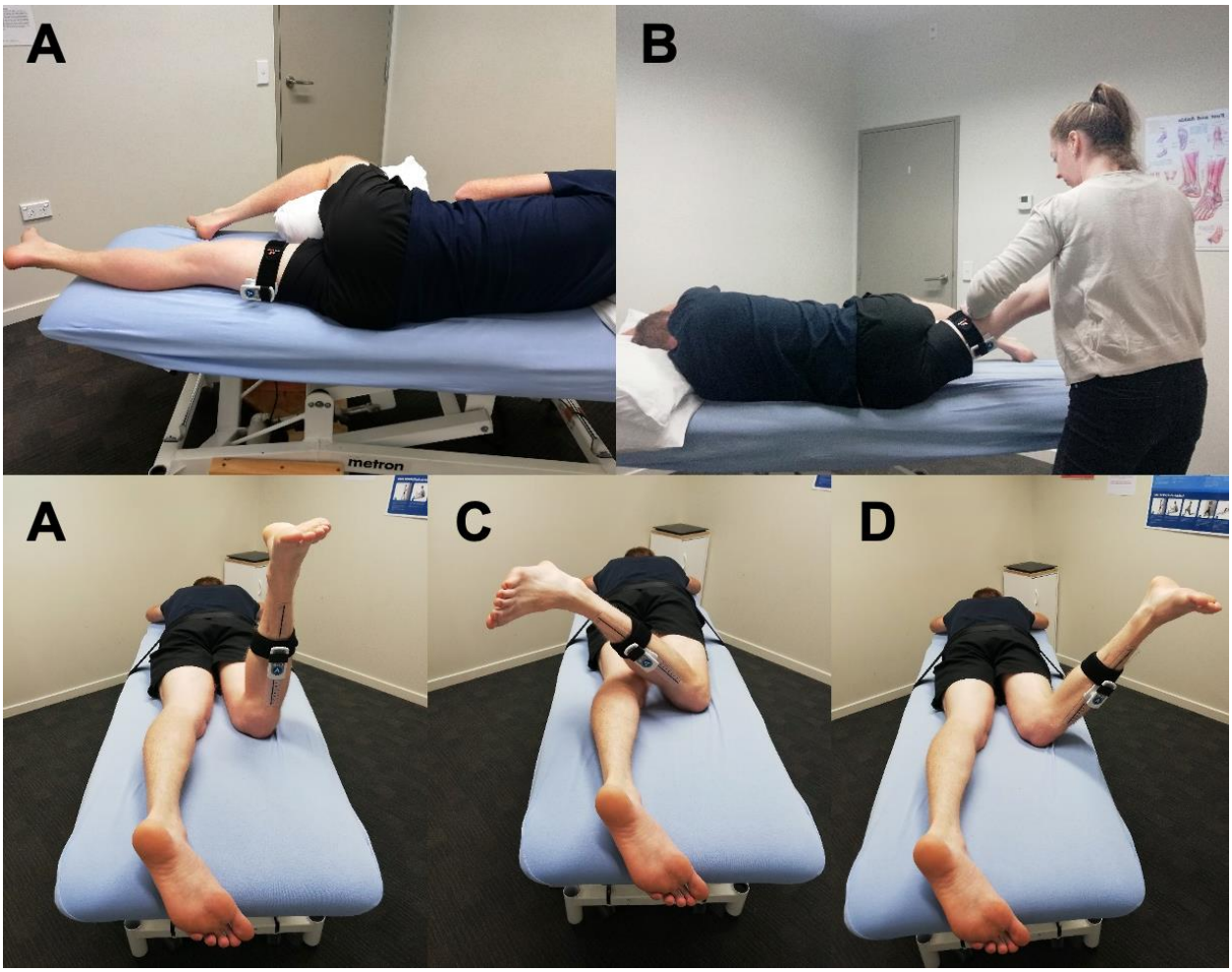



*Figure 7. PROM start positions A) and end positions for B) adduction, C) external rotation and D) internal rotation*

### 3.5.3 Hip muscle strength testing

GMed is active during abduction, external rotation, and internal rotation movements of the hip (Neumann, 2010). Therefore, strength testing (maximal isometric testing) for GMed took place in all three of these directions. Testing was carried out on the right and left sides, using a handheld dynamometer (ActivForce Digital Dynamometer System, Activbody, Inc., 2018) with stabilisation belts. Strength was measured in kilograms of force (kgs/F). The ActivForce digital dynamometer is small enough to be held in the palm of the hand and is relatively inexpensive making it clinically applicable (Figure 8).


*Figure 8. ActivForce digital dynamometer*

The use of a dynamometer with stabilisation straps has been shown to provide excellent inter- and intra-reliability when testing muscle strength during hip abduction and external rotation movements (Leao Almeida, Das Neves Rodrigues, De Freitas, & De Paula Lima, 2017). The use of stabilisation belts eliminates the influence of the examiner's own strength on testing and muscle strength output (Krause et al., 2014). The use of a stabilisation belt as an external fixation for the dynamometer makes the muscle testing procedure more valid and reliable. Without the use of a stabilisation belt, the results would be dependent on the examiner's upper limb strength and their ability to match the force being tested (Thorborg, Bandholm, Schick, Jensen, & Hölmich, 2013). The use of a stabilisation belt with the dynamometer in this study ensured that this protocol was reproducible, and results were not dependent on the examiner's strength.

The participants' weight was recorded prior to strength testing to normalise the strength measures (Appendix G: Muscle strength assessment sheet). Their weight was entered into the ActivForce phone app (ActivForce, Activbody, Inc., 2018) which automatically accounted for body mass being a confounding variable on data. The participant was asked which side was their dominant side and this was also recorded on the assessment sheet. With the dynamometer in situ, the primary researcher instructed the participant by saying: "Ready, Steady, Push". The participant pushed for five seconds, which the investigator counted down, saying stop after the allotted time was finished. There was one practice attempt (familiarisation trial), followed by the three recorded trials. There was a one-minute rest interval between each trial (Bazett-Jones,

Tylinksi, Krstic, Stromquist, & Sparks, 2017). Three strength measures were recorded in each direction and the peak value was used in the data analysis (Ireland et al., 2003; McCann, Terada, Kosik, & Gribble, 2019) (Appendix H: Protocol for hip strength testing).

For muscle strength testing during abduction, the participants were positioned in side-lying (Bazett-Jones et al., 2017; McCann et al., 2019; Piva, Goodnite, & Childs, 2005; Powers, Ghoddosi, Straub, & Khayambashi, 2017; Willson & Davis, 2009). The top leg was in placed in 10° abduction using the digital inclinometer and was maintained in position using pillows and towels (Ireland et al., 2003; McCann et al., 2019). Both legs were straight with the hips in neutral and knees extended. A stabilisation belt was used proximal to the iliac crest to stabilise the trunk and one around the dynamometer to provide resistance (the belts encircled the plinth) (Ireland et al., 2003; McCann et al., 2019; Willson & Davis, 2009). The dynamometer was placed five centimetres above the lateral femoral condyle of the top leg (Ireland et al., 2003; Leao Almeida et al., 2017; McCann et al., 2019) (Figure 9A).

For muscle strength testing during hip rotation, the participants were in prone lying on the plinth for external rotation (Bohannon, 1999; Piva et al., 2005; Willson & Davis, 2009) and internal rotation (Freke et al., 2019). A stabilisation belt was used around the pelvis to limit rotation, another belt was used around the thighs to ensure they stayed hip distance apart and neutral (Willson & Davis, 2009); and a third stabilisation belt was used encircling the dynamometer and the resistance pole (Figure 9B, 9C). A custom-made holder for the resistance pole was placed underneath the plinth to provide resistance and stability for the pole. The dynamometer was placed five centimetres proximal to the medial malleolus for external rotation (Willson & Davis, 2009) and the lateral malleolus for internal rotation.

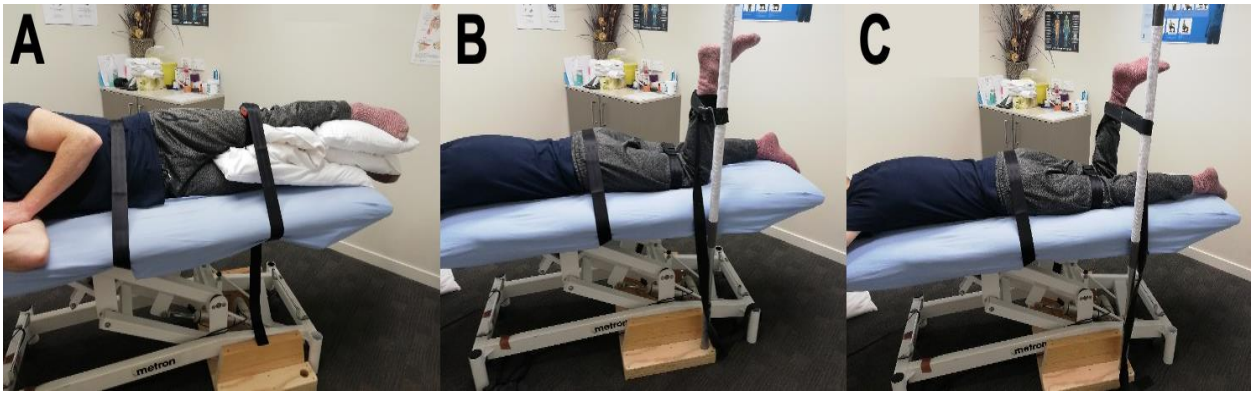



*Figure 9. Muscle strength assessment testing positions: A) abduction B) external rotation and C) internal rotation*

### 3.5.4 Gluteus medius palpation and trigger point status

#### *3.5.4.1 Trigger point protocol development*

To answer the research question: "Is there a relationship between active GMed TrPs and decreased hip PROM and muscle strength in people with CNSLBP?"; a robust TrP assessment needed to be carried out to identify the TrP status of each participant's right and left GMed muscle. Palpation using the examiner's hands is the accepted and gold standard method of assessment for the presence of TrPs (Bron et al., 2007; Rozenfeld, Finestone, Moran, Damri, & Kalichman, 2017). It displays good inter- and intra-rater reliability when identifying TrPs (Bron et al., 2007; Rozenfeld et al., 2017). This is especially the case for experienced physiotherapists (Hsieh et al., 2000), when a palpation protocol is used and there is verbal communication with the participant (Barbero et al., 2012).

Palpation was used as the method of assessment for the presence of TrPs in the GMed muscles for this study, with a palpation protocol devised to assign a TrP status of active, latent, combination of active and latent, or zero TrPs. The standardised TrP palpation protocol was devised in consultation with an expert in the field of assessment and treatment of TrPs (Prof. César Fernández-des-las-Penas, Universidad Rey Juan Carlos, Madrid, Spain) (Appendix I: Palpation protocol). Following a standardised protocol ensures a consistent method of palpation (Barbero et al., 2012; Hsieh et al., 2000; Rozenfeld et al., 2017). The TrP examiner in this study was a physiotherapist who has worked in musculoskeletal physiotherapy for over 30 years, including the assessment and management of TrPs. The TrP examiner communicated verbally with the participants' regarding what they were feeling during the palpation assessment.

A position of side-lying was chosen for palpation assessment as it was a comfortable position for the participant and optimal for the TrP examiner to easily palpate GMed. The participant was positioned in side-lying with their hips and knees bent to 45° providing the examiner full access to palpate GMed. The top buttock was exposed with towels for draping to preserve modesty and decrease risk of embarrassment. A towel was used along the posterior border of the buttock and at the front to cover the groin area. The top hip was placed in a position of 10° adduction (determined by using the digital inclinometer) and maintained in this position using pillows between the participant's knees. This position placed the GMed on slight stretch (as GMed is a hip abductor) (Arab & Nourbakhsh, 2010; Neumann, 2010). When a muscle to be palpated is under tension (stretched), taut bands within the muscle will be more easily identified (Simons et al., 1999).

To ensure the whole GMed muscle was palpated, the examiner marked out the GMed muscle on each side with a highlighter marker that was easily wiped off after the assessment. To mark out the GMed, the examiner palpated along its attachment to the iliac crest and laterally to the greater trochanter of the femur (Field, 2008). During assessment, the examiner palpated the whole GMed muscle for the presence of TrPs. Palpation was carried out in a lateral direction from midline in a systematic way using their pads of their second and third fingers. The top buttock was palpated; the same procedure was then repeated on the other side. TrPs were mapped on a body chart. Referred pain from latent TrPs was also mapped out on the same body chart.

Although Simons et al. (1999) provided a body map of the location of TrPs, it is more recently accepted that despite these designated sites for TrPs being very common, TrPs can be present anywhere in the muscle (Fernández-de-las-Peñas & Dommerholt, 2018). Studies that have examined the location of TrPs within muscles have concluded that they are located near motor endplates at the formation of the motor unit (where the neuron branch terminates with the muscle fiber) (Akamatsu et al., 2017; Wada et al., 2020). Given that there are numerous motor units within a muscle (Patton & Thibodeau, 2010); this justified the need to palpate the whole GMed muscle in order to identify the presence of TrPs.

#### *3.5.4.2 Training of the trigger point examiner*

To ensure that a correct and standardised TrP palpation technique and recording of TrP status was performed, a training session was undertaken by the primary researcher with the TrP examiner (CL). The primary researcher has worked in musculoskeletal and rehabilitation physiotherapy for nine years, including palpation and treatment of TrPs, using dry needling and

massage (digital ischaemic pressure). As stated, the secondary researcher has worked for 30 years as a physiotherapist; assessing and treating TrPs as part of her work.

The TrP examiner was trained in participant positioning, method, and direction of palpation. Following initial training, three volunteers were palpated separately by the primary researcher and the TrP examiner. This was a blinded assessment with the results compared on conclusion. For each of the three assessments, the primary researcher and the TrP examiner found the same number of GMed TrPs with uniform agreement on their status. This was undertaken to ensure the palpation protocol was reproducible and that the TrP examiner fully understood the palpation protocol and the process of determination of TrP status.

#### *3.5.4.3 Assessment of trigger point status*

The presence of active, latent, or zero TrPs was identified on the right and left GMed of each participant. Data was collected dichotomously, “yes” or “no” to the presence of an active, latent, or zero TrPs and the number of active and latent TrPs present was recorded. Active and latent TrPs have similar and differentiating characteristics. They both demonstrate localised tenderness (Njoo & Van der Does, 1994), with a palpable taut band felt within the muscle of interest (Hsieh et al., 2000). A non-recognisable pain indicated a latent TrP; whereas, pain, recognisable to the participant as their LBP (for example) indicated an active TrP (C. K. Chen & Nizar, 2011; Ge et al., 2014; Njoo & Van der Does, 1994). If no pain was elicited on palpation of GMed, then zero TrPs were present. A palpable band was required within the muscle to determine a TrP (Basford et al., 2014; Q. Chen et al., 2016). If pain was reported by the participant but no palpable band was present; zero TrPs were recorded. Referred pain patterns were also recorded on a body chart on the palpation assessment sheet (Appendix J). If referred pain was produced this was marked on a body chart and confirmed if the pain produced on palpation was familiar and recognisable to the participant as their LBP.

### 3.5.5 Secondary outcome measures

When researching CLBP, physiological and non-physiological factors should be considered, as both entities will influence performance testing (Huijnen, Verbunt, Wittink, & Smeets, 2013). Therefore, alongside hip PROM and muscle strength testing; the participant’s completed questionnaires to gauge their pain, function, and fear avoidance beliefs in relation to their CLBP. Details of the questionnaires follows in the next subsections. The Numeric Pain Rating Scale (NPRS) (Appendix K) was used to assess the participants’ pain levels due to their LBP. Collecting pain scores allowed analysis of associations between LBP and TrP status. Also, pain intensity affects physical performance (Huijnen et al., 2013); therefore, the participant’s pain

levels were included in statistical analysis for association with hip PROM and muscle strength measures.

The Oswestry Low Back Pain Disability Questionnaire (ODQ) (Appendix L) was used to gather information on the participant's function/disability. Disability is a component of back pain (Fairbank & Pynsent, 2000); and therefore, should be included in analysis of outcome measures (Fairbank & Pynsent, 2000). High levels of psychological distress alongside high levels of pain have been shown to increase inhibition of muscle activity (Verbunt et al., 2005). Additionally, CLBP sufferers who display high pain related fear have been shown to display decreased peak muscle activation (J. S. Thomas, France, Sha, & Wiele, 2008). The Tampa Scale-11 (TSK-11) (Appendix M) explored the participants' thoughts about fear of movement and re-injury. As this study assessed muscle strength, it was imperative to establish if there was a relationship between hip muscle strength and fear avoidance beliefs (TSK-11) scores. It would be expected that participant's with high pain related fears would display decreased muscle strength scores (J. S. Thomas et al., 2008).

#### *3.5.5.1 Numeric Pain Rating Scale*

Participants were asked to indicate the intensity of their current, best, and worst pain over the past 24 hours on NPRS. They chose a number to represent this pain from a scale of zero to 10. Zero indicated no pain and 10 indicated the worst pain imaginable. NPRS is a commonly used tool to assess pain in research, especially research into CNSLBP (Akodu & Odunfa, 2020; Fagundes Loss et al., 2020; Lalkate, Agrawal, & Agashe, 2020). It has demonstrated good validity when assessing pain in people with LBP (Childs et al., 2005; Jensen, Turner, & Romano, 1994). Other studies investigating pain in relation to TrPs have used NPRS to assess the participants' pain levels (Alburquerque-García, Rodrigues-de-Souza, Fernández-de-las-Peñas, & Alburquerque-Sendín, 2015; Iglesias-González et al., 2013).

#### *3.5.5.2 Oswestry Low Back Pain Disability Questionnaire*

The ODQ records a person's pain-related disability in relation to acute, sub-acute, and CLBP (Fairbank & Pynsent, 2000). There are 10 questions, one related to pain and nine related to activities of daily living for example, walking, standing, and personal care. Each question is scored from zero to six and the total score indicates the degree of disability the person has reported. A score of zero indicates no disability and a score of 100 indicates maximum disability (Fairbank & Pynsent, 2000).

The ODQ has been shown to have high internal consistency (Fairbank & Pynsent, 2000). It has also demonstrated good test-retest reliability (Davidson & Keating, 2002). It has adequate content validity and covers activities that people with LBP carry out in their everyday lives. The ODQ is a suitable outcome measure to use in research (Smeets, Köke, Lin, Ferreira, & Demoulin, 2011); due to its good validity, reliability and responsiveness when used with people with LBP (Chapman et al., 2011).

#### *3.5.5.3 Tampa Scale-11*

The questions on the TSK-11 questionnaire related to two subscales: avoidance of movement and harm because of movement (Celletti et al., 2021). The participants circled either: strongly disagree, somewhat disagree, somewhat agree, or strongly agree in relation to 11 statements. Examples of two of the statements are: “I’m afraid I might injure myself if I exercise” and “Pain always means I have injured my body” (Woby, Urmston, Roach, & Watson, 2005).

The TSK-11 questionnaire is a commonly used questionnaire to explore musculoskeletal pain related fear of movement; specifically, CNSLBP (Celletti et al., 2021; de Oliveira Meirelles, de Oliveira Muniz Cunha, & da Silva, 2020). Although with other conditions for example, anterior cruciate ligament injury, it has demonstrated low validity and responsiveness but adequate reliability (Hui et al., 2019). When exploring pain-related fear of movement in chronic pain, specifically LBP, TSK-11 has been shown to have adequate validity and reliability (Hapidou et al., 2012).

### 3.6 Confounding variables

Potential confounding variables that may influence the data to be collected were considered and reviewed. Discussion was had within the research team (MC and supervisors) in conjunction with reference to the literature concerning variables that may influence hip PROM and muscle strength. The confounding variables that were highlighted and brought forwards to be included in statistical analysis were gender, age, age groups, NPRS (-current, -best, and -worst), ODQ, TSK-11, ethnicity, weight, weight groups, and site of LBP. Even though, these variables were not part of the causal relationship between TrP status and deficits in hip PROM and muscle strength; they needed to be considered for their influence on these outcome variables (Hoffmann, 2021).

From the existing literature, gender and ODQ have been shown to influence hip PROM and muscle strength (Hogg et al., 2018; Matsumura et al., 2015; Steultjens, Dekker, van Baar, Oostendorp, & Bijlsma, 2000); therefore, these were kept in consideration when reviewing the

results of the initial analysis. Physiological reasons were also considered, for example, weight and site of LBP and how these could potentially affect hip PROM and muscle strength assessment and resulting measures.

### 3.7 Statistical analysis

Statistical analysis aimed to answer the following research question and either confirm the hypothesis or null hypothesis:

Research question: Is there a relationship between active GMed trigger TrPs and deficits in hip passive range of movement and muscle strength in people with CNSLBP?

Hypothesis: CNSLBP sufferers who have active gluteus medius trigger points will also have decreased passive range of movement and muscle strength in their hips due to the presence of the active gluteus medius trigger points.

Null hypothesis: There will be no difference in hip passive range of movement and muscle strength regardless of trigger point status in gluteus medius.

SPSS Statistics Data Editor Version 26 (IBM Corporation, New York, USA) was used for all statistical analysis in this research study.

#### 3.7.1 Descriptive analysis

Initial descriptive analysis of the demographic information was carried out presenting participant demographic and clinical characteristics. This data included the number of participants, by gender, age groups, weight groups, ethnic backgrounds, and occupations. Descriptive analysis was also used to present data regarding TrP status, number of TrPs, distribution of TrP status for right and left sides, and sub-groups of the number of TrPs in each status.

#### 3.7.2 Analysis of measures and confounding variables

Normality tests were carried out on the two dependent variables, namely hip PROM, and muscle strength. Data was normally distributed and as a result, parametric tests were used to further analyse TrP data with the dependent variables of hip PROM and muscle strength. Confounding variables were also considered for their effect on hip PROM and muscle strength. General linear modelling (using univariate analysis) was used to analyse hip PROM and muscle strength and included in the analysis using univariate analysis as part of general linear modelling. for possible confounding variables. This type of analysis was also used to identify significant

associations between hip PROM and TrP data, and muscle strength and TrP data, adjusted for confounding factors.

### 3.8 Funding

An application was submitted to the Southland Medical Foundation. This is a trust that provides funding to health professionals who are carrying out research or professional development that will benefit people in the Southland region of New Zealand. A grant of $1000 was received from the Southland Medical Foundation for the research study. Any excess costs were covered by the primary researcher (MC).

# Chapter 4. Results

## 4.1 Descriptive analysis

Forty-six participants volunteered and consented to participate in this study, which took place from October 2019 to July 2020 (Figure 5: Study design). Following pre-screening by telephone call or in person conversation, participants who met the inclusion criteria and did not possess exclusion criteria continued onto assessment. However, one participant disclosed to the primary researcher, at the conclusion of her assessment, that she did experience occasional pins and needles and numbness in her saddle region but had been too embarrassed to disclose this information earlier. This participant's assessment had been completed but her data was excluded from analysis. A further three participants were unable to follow the testing procedures. For one participant, significant cramping was experienced during PROM testing. He needed to get up and walk around and take breaks between each movement. The order of PROM movements was changed to try ease his pain and decrease his need to change position. After the PROM testing, he was generally more uncomfortable as he continued the muscle strength testing portion of the assessment. The other two participants experienced increased LBP after the first PROM movement assessment and pain persisted at that higher level for the entirety of PROM testing. The order of testing was changed for their muscle strength testing to decrease their need to change position. As a result of increased pain on testing, these three participants were unable to follow the research protocol and hence, their data was excluded for analysis. This resulted in data from 42 participants being taken forwards for analysis (Figure 5).

To participate in this research study, all participants had experienced pain for a minimum of three months; however, the precise length of time with symptoms was not quantified. Demographic information was collected from all participants (Table 1). There were 35 females and seven males. Thirty participants identified New Zealand/ European as their ethnic background, nine identified themselves as European, one identified Māori as their first ethnicity and three identified as Māori as part of their ethnicity, and three were other ethnicities (Israeli, Tongan, and Peruvian). One participant did not want to specify his ethnicity.

There was a large weight range in this study, from 48.9 kgs to 120.6kgs. The New Zealand national average weight for a NZ European male is 87kgs and a Māori male is 95kgs (Ministry of Health NZ, 2020). The New Zealand national average weight for a NZ European female is 73.9kgs and a Māori female is 83.6kgs (Ministry of Health NZ, 2020). In this current study, the average weight for males was 91.8kgs and the average weight for females was 79.9kgs. There was a range of ages. Females ranged from ages 29 years to 67 years and males ranged from 30 years

to 75 years. The mean overall age was 44.2 years. Most participants were in their 30's (16 participants) and 40's (11 participants); with others in their 50's (8 participants), and 60's (5 participants) (Table 1).

*Table 1. Demographic information of the participants*

| Weight | Females | Males | Ethnicities | Age (years) |
|---|---|---|---|---|
| 40-49kgs | 1 | 0 | European | 32 |
| 50-59kgs | 2 | 0 | European, Israeli | 29, 30 |
| 60-69kgs | 8 | 0 | NZ European, European | 30, 33, 44, 46, 51, 57, 67 |
| 70-79kgs | 9 | 1 | NZ European, European/ Māori, NZ/ Māori, | 34, 39, 40, 41, 48, 52, 54, 61, 64, |
| 80-89kgs | 5 | 3 | NZ European, European | 30, 36, 37, 53, 54, 67, 75 |
| 90-99kgs | 5 | 1 | NZ European, European | 35, 39, 47, 48, 64 |
| 100+kgs | 5 | 2 | NZ European, Māori, Tongan, Peruvian, did not specify | 31, 37, 42, 45, 49, 53 |

Occupations varied over many sectors: manual jobs = four (e.g., factory worker, mechanic, metal fabricator), farming related jobs = seven, tourism = four (e.g., bus driver, boat crew), hospitality = three, health sector = three (e.g., health coach, social worker, massage therapist), administrative roles = four, accountant = one, retail = six, teacher = three, childcare = one, student = one, mother = four, retired/semi-retired = two, and unemployed = one.

### 4.2 Trigger point status, number, and distribution

A TrP status of either: active, latent, combination of active and latent, and zero TrPs was assigned to the participant's right and left side GMed. Of the 42 participants, 39 presented with GMed TrPs on assessment. TrPs were on either one side or bilaterally. Latent TrPs were the most common type of TrP found. Thirty-one participants had bilateral latent TrPs. Seventeen participants had latent TrPs on the right side only and 16 participants had latent TrPs on the left side only. The second most common TrP status was a combination of active and latent; followed by zero TrPs, and active (Figure 10).

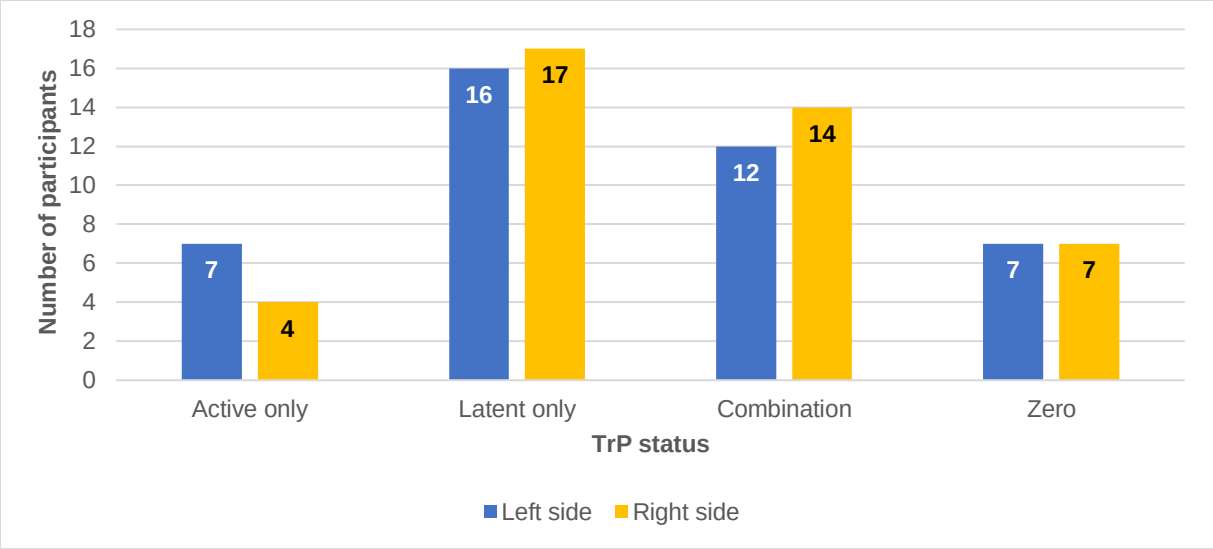



*Figure 10. Right and left side trigger point distribution*

During the assessment, the participants were not asked to specify if their LBP was right sided, left sided, central, or bilateral. They had previously marked where there LBP was located on a body chart. The location of LBP was extrapolated from the body charts. From the locations marked on the body charts, five participants marked right-sided LBP, five marked left-sided LBP, five marked central LBP, and 27 marked bilateral LBP. The TrP distribution with site of LBP is shown in (Table 2). Site of LBP was considered a possible confounding variable; though on analysis, no association was identified between site of LBP and TrP status.

*Table 2. Location of LBP with trigger point distribution (% of participants)*

| | **Location of Gluteus Medius trigger points** | | | |
|---|---|---|---|---|
| **Location of low back pain** | **Right side only** | **Left side only** | **Bilateral** | **Zero trigger points bilaterally** |
| **Right sided** | 20% | 20% | 60% | 0% |
| **Left sided** | 0% | 20% | 60% | 20% |
| **Central** | 20% | 20% | 60% | 0% |
| **Bilateral** | 7.5% | 4% | 81% | 7.5% |

TrP information was further analysed to compare the number of TrPs that the participants displayed. The number of TrPs present per status was similar for the right and left sides. However, the total number of TrPs was higher on the participants' right side when

compared with the left, which was representative of a higher number of latent TrPs on this side. The number of active TrPs was similar for the right and left sides. There was a large range of TrPs present for each status (Table 3).

*Table 3. Number of trigger points per side and per status*

| | Both sides combined | | | Right side | | | Left side | | |
|---|---|---|---|---|---|---|---|---|---|
| | **N** | **Mean** | **Range** | **N** | **Mean** | **Range** | **N** | **Mean** | **Range** |
| **Total number of TrPs** | 413 | 5 | 0-18 | 225 | 5.4 | 0-18 | 188 | 4.5 | 0.17 |
| **Active TrPs only** | 71 | 7 | 0-13 | 35 | 8.8 | 0-13 | 36 | 5.1 | 0-11 |
| **Latent TrPs only** | 143 | 4.4 | 0-18 | 81 | 4.8 | 0-18 | 62 | 3.9 | 0-17 |
| **Combination active and latent TrPs** | 199 | 7.7 | 0-16 | 109 | 7.8 | 0-16 | 90 | 7.5 | 0-14 |

The number of TrPs was further analysed into sub-groups of number of TrPs per status. Sub-groups were divided into 0, 1-2, 3-4, and 5+ TrPs. The highest numbers are the number of participants with no active TrPs, as active TrP status was the least prevalent TrP type. The distribution of active TrPs on the right and left sides was similar. The most common number of latent TrPs was 1-2 on the right and 0 on the left. The distribution between right and left was also very similar (Table 4).

*Table 4. Sub-groups of number of trigger points per participant*

| **Number of TrPs** | **Right Active** | **Left Active** | **Right Latent** | **Left Latent** |
|---|---|---|---|---|
| **0** | 24 | 23 | 11 | 14 |
| **1-2** | 6 | 6 | 14 | 10 |
| **3-4** | 5 | 6 | 8 | 10 |
| **5+** | 7 | 7 | 9 | 8 |

### 4.3 Confounding variables

Results of the univariate analyses and the known confounding factors, plus consistency of confounders for left and right sides were considered before arriving at the final set of covariates for each measure of interest for hip PROM and muscle strength. The confounding variables under consideration for analysis were gender, age, age groups, NPRS (-current, -best, and -worst), ODQ, TSK-11, ethnicity, weight, weight groups, and site of LBP. Age and weight were analysed as continuous measures and within age and weight categories. Age groups were: 28-35, 36-45, 46-55, and 56-75 years. Weight groups were: 48.9-69kg, 70-84kg, 85-99kg, and 100+kg.

Ethnicity was categorised into two groups, the first group represented New Zealand European and European, and the second group represented all other ethnicities. This sub-grouping was carried out to simplify statistical analysis. Site of LBP (as determined from the body chart representation) was categorised into four groups: right-sided LBP, left-sided LBP, bilateral LBP, and central LBP. To reach significance, a p-value of <0.05 was used. Variables greater but close to the p-value (<0.2) were also kept for consideration (Table 5).

*Table 5. Initial univariate analysis of effects (with p-values) of confounding variables for PROM*

| | |
|---|---|
| **Right adduction** | Significant: Weight group (0.003) and ODQ (0.047)<br>Close: None identified |
| **Left adduction** | Significant: Weight group (0.045) and weight (0.003)<br>Close: ODQ (0.141) and TSK-11 (0.153) |
| **Right internal rotation** | Significant: None identified<br>Close: TSK-11 (0.137) |
| **Left internal rotation** | Significant: TSK-11 (0.05)<br>Close: Gender (0.219) and age group (0.296) |
| **Right external rotation** | Significant: NPRS-worst (0.048)<br>Close: ODQ (0.094) and ethnicity (0.072) |
| **Left external rotation** | Significant: Gender (0.04), NPRS-worst (0.047), ODQ (0.030) and site of LBP (0.036)<br>Close: ODQ (0.120), NPRS-current (0.141), age group (0.133), ethnicity (0.108) |

From the existing literature, gender and ODQ have been shown to influence hip PROM and muscle strength (Hogg et al., 2018; Matsumura et al., 2015; Steultjens et al., 2000); therefore, these were kept in consideration when reviewing the results of the initial analysis. Physiological reasons were also considered, for example, weight and site of LBP and how these could potentially affect hip PROM and muscle strength assessment and resulting measures. These initial findings were brought forward into further univariate general linear modelling analysis to identify the strongest correlated variables. This was carried out for both PROM and muscle strength on PROM. The same was process was carried out for muscle strength (Table 6).

*Table 6. Initial univariate analysis of effects (with p-values) of confounding variables for muscle strength*

| | |
|---|---|
| **Right abduction** | Significant: Gender (0.002)<br>Close: Ethnicity (0.132) and ODQ (0.157) |
| **Left abduction** | Significant: Gender (0.001)<br>Close: Site of LBP (0.164) and ODQ (0.064) |
| **Right internal rotation** | Significant: Gender (0.000), ODQ (0.010), and site of LBP (0.009)<br>Close: Age group (0.109), NPRS-current (0.072), and weight (0.125) |
| **Left internal rotation** | Significant: Gender (0.151)<br>Close: Age group (0.151), site of LBP (0.112), ODQ (0.204) and NPRS-current (0.146) |
| **Right external rotation** | Significant: Gender (0,000) and ODQ (0.057)<br>Close: Age group (0.068), site of LBP (0.090), and weight (0.098) |
| **Left external rotation** | Significant: Gender (0.000) and ODQ (0.040)<br>Close: Age group (0.122), weight group (0.148), weight (0.084), and site of LBP (0.107) |

After further analysis (stepwise univariate general linear modelling); final confounding variables were established. These included the final variables identified through analysis and known variables of gender and ODQ (

Table 7). These final confounding variables were carried forward into analysis of the influence of TrP status on hip PROM and muscle strength.

*Table 7. Final confounding variables*

| | **PROM** |
|---|---|
| **Adduction** | ODQ and weight group |
| **Internal rotation** | Gender |
| **External rotation** | Gender and ODQ |
| | **Muscle strength** |
| **Abduction** | Gender and ethnicity |
| **Internal rotation** | Gender, site of LBP, age group, and ODQ |
| **External rotation** | Gender, site of LBP, age group, and ODQ |

### 4.4 The influence of trigger point status on hip PROM

The influence of TrPs on hip PROM measures were analysed on the right and left sides. Crossover effects were also analysed for example, if the presence of TrPs on the right influenced left side PROM and vice versa. The final confounding variables identified were included in the analysis (using univariate general linear modelling). The ranges and mean of each hip PROM are shown (Table 8).

*Table 8. PROM directions: Summary of range and means*

| **PROM** | **Mean (degrees)** | **Range (degrees)** |
|---|---|---|
| **Right adduction** | 28.5 | 9.3 – 41.3 |
| **Left adduction** | 28.9 | 18.6 – 45 |
| **Right internal rotation** | 42.4 | 23.6 – 55.3 |

| | | |
|---|---|---|
| **Left internal rotation** | 42.4 | 26.6 – 55.6 |
| **Right external rotation** | 39.7 | 23 – 58 |
| **Left external rotation** | 41.9 | 24.6 – 58 |

### 4.4.1 Trigger point influences on PROM adduction

The analysis showed a significant association between the number of active TrPs on the right side and right sided hip adduction (p=0.003). Estimated marginal means showed that the highest measures of right sided hip adduction were in the participants who had active TrPs only (active: 30.8°, latent: 27.9°, zero TrPs: 26.2°, and combination: 24.6°) (Figure 11). No significant associations were identified between TrPs and the left side for hip adduction. Active TrPs were divided into sub-groups by number: 0, 1-2, 3-5, and 6+. Analysis was carried out, but no significant associations were identified between a particular number of active TrPs and its influence on hip adduction. No crossover effects were identified between TrP status and hip adduction.

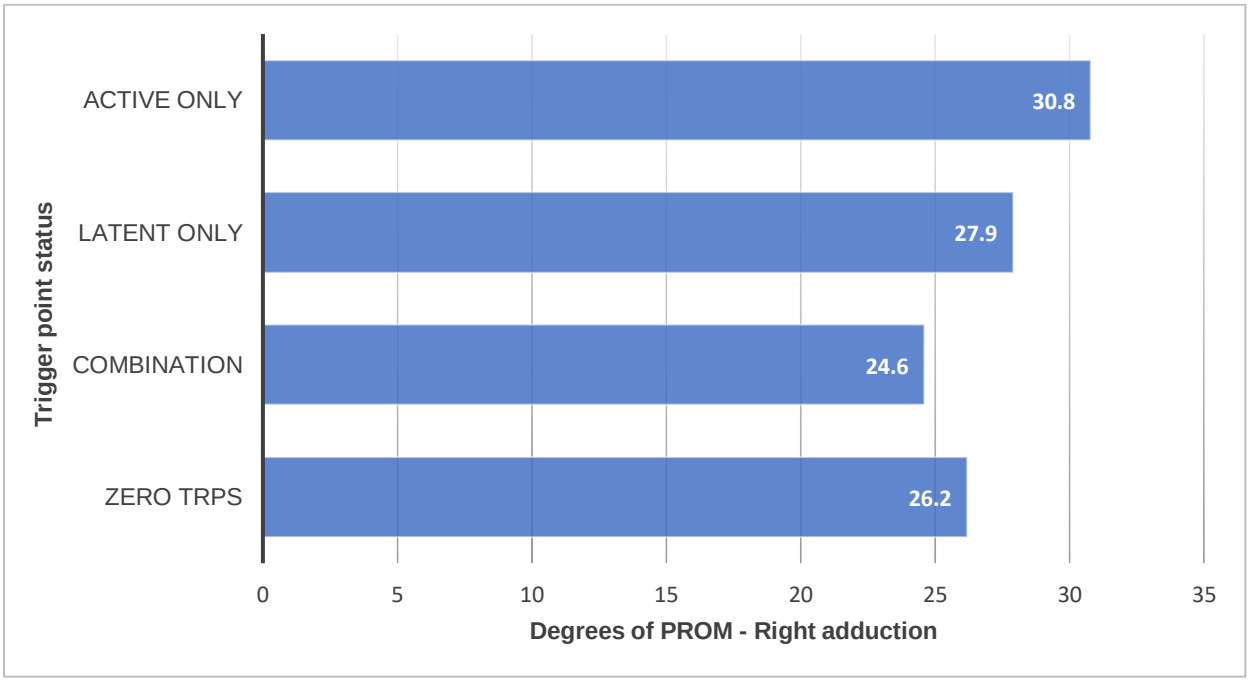



*Figure 11. The effect of trigger point status on PROM - right adduction*

### 4.4.2 Trigger point influences on PROM internal rotation

A significant association was identified between the number of active TrPs on the right side and hip internal rotation measures on the right side (p=0.042) (Figure 12). Estimated marginal means showed that the highest ROM for hip internal rotation were achieved by participants who had active TrPs only (active: 46.3°, combination: 44.1°, latent: 42.7°, and zero TrPs: 39.1°). No significant associations were identified between TrP status and left hip internal rotation. The closest correlations were observed between active TrP status and the number of active TrPs on the left side with highest ROM for hip internal rotation on the left side (p=0.110). No crossover effects were identified. When the number of active TrPs were divided into sub-groups: 0, 1-2, 3-5, and 6+. The number of active TrPs was not associated with the degrees of hip internal rotation.

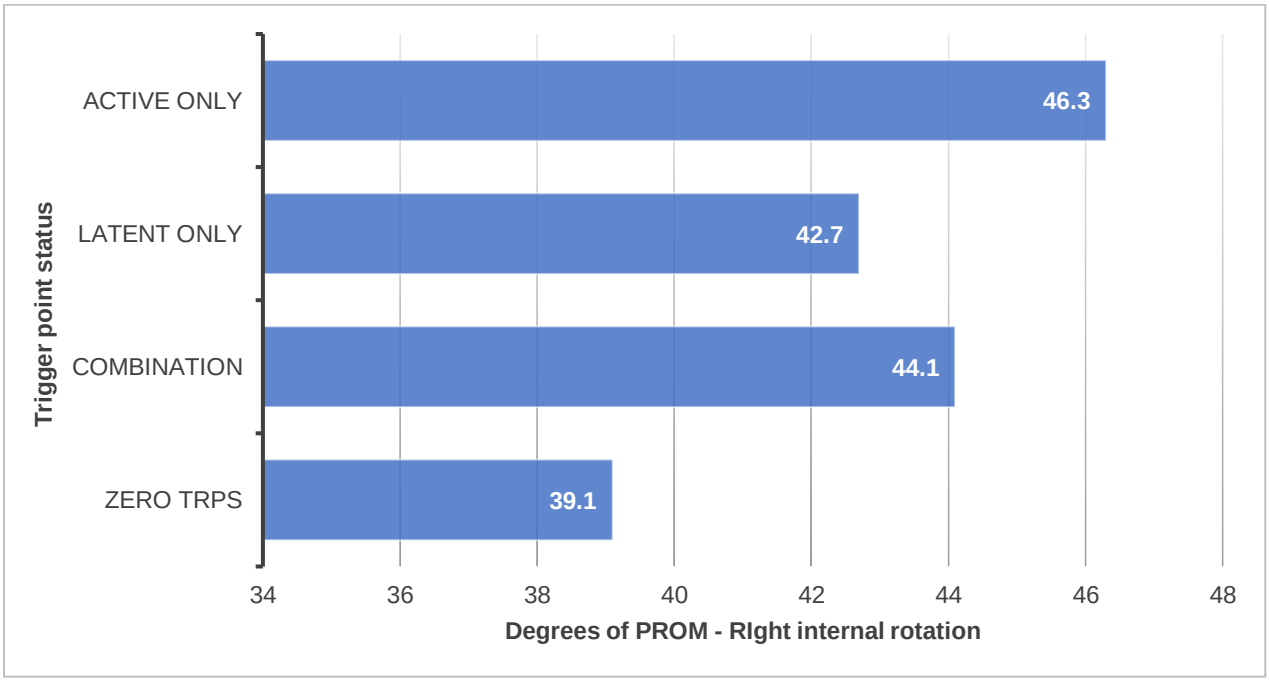



*Figure 12. The effect of trigger point status on PROM - right internal rotation*

### 4.4.3 Trigger point influences on PROM external rotation

There were no significant associations between right external rotation and TrP status on the right side. On the left side, there was a significant association. The presence of active TrPs were associated with left external rotation (p=0.018). Participants with active TrPs (i.e., a status of active TrPs or combination of active and latent TrPs) displayed the lowest ROM for left hip external rotation (*Figure 13*). No crossover effects were identified between TrP status and hip

external rotation. The highest ROM was observed in the latent TrP group, followed by zero TrPs, active, and combination of active and latent TrPs.

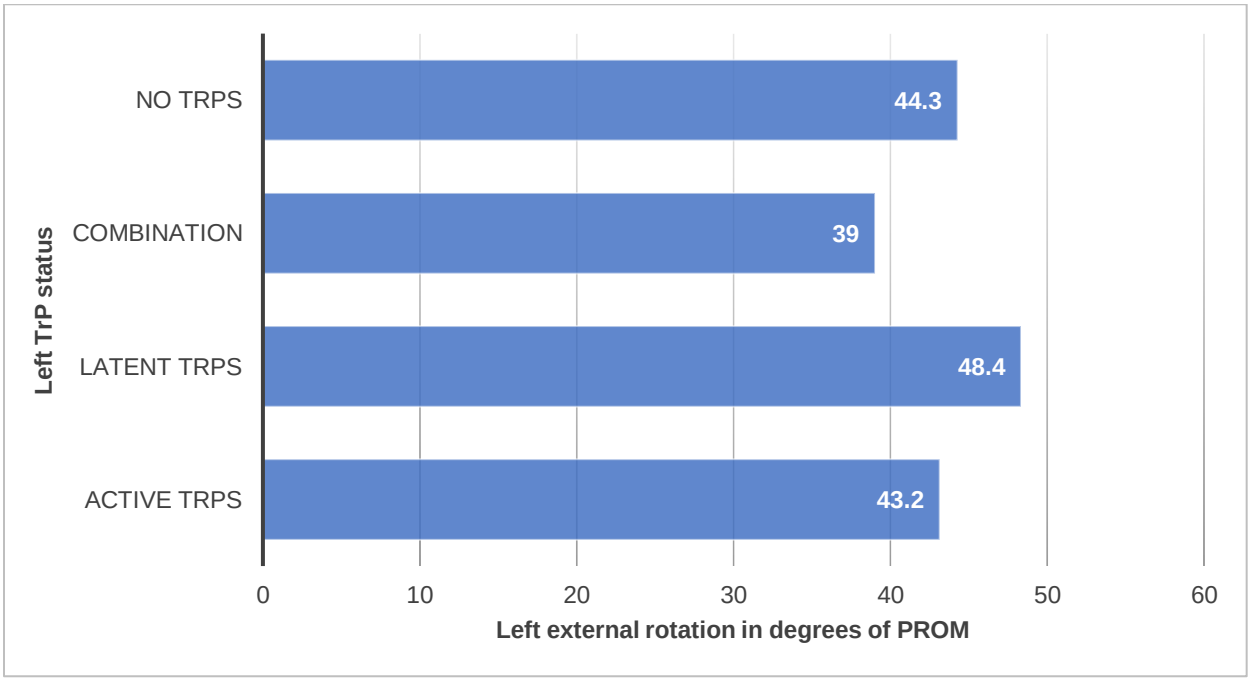



*Figure 13. The effect of trigger point status on PROM - left external rotation*

### 4.4.4 Summary of PROM results

In summary, the presence of active TrPs appeared to have the largest effect on hip PROM. For adduction and internal rotation on the right side, the presence of active TrPs resulted in the largest PROM. For both movements, zero TrPs displayed the lowest PROM. External rotation was different to the other two movements. No associations were observed for the right side, but active TrPs on the left side resulted in the lowest PROMs. Active only and combination groups displayed the lowest ROM, with latent TrPs status group displaying the largest PROMs. A summary of findings for the influence of TrP status on hip PROM is shown below (Table 9).

*Table 9. Summary of the influence of trigger point status on hip PROM*

| PROM | Association with TrP status |
|---|---|
| **Right adduction** | Number of active TrPs on the right<br>Highest ROM in active TrPs only group |
| **Left adduction** | No associations with TrP data |
| **Right internal rotation** | Number of active TrPs on the right<br>Highest ROM in active TrPs only group |
| **Left internal rotation** | No associations with TrP data |
| **Right external rotation** | No associations with TrP data |
| **Left external rotation** | TrP status on the left side and presence of active TrPs on the left<br>Lowest ROM in active only and combination of active and latent TrP groups |

### 4.5 The influence of trigger point status on muscle strength

The influence of TrPs on hip muscle strength was analysed. Crossover effects were also analysed for example, if the presence of TrPs on the right influenced left side muscle strength and vice versa. The final confounding variables identified were included in the analysis (using univariate general linear modelling).

#### 4.5.1 Trigger point influences on hip abduction strength

No significant associations were identified between TrP status and hip abduction on the right or left sides. Although, not significant, a trend was observed on the left side between those with a TrP status of zero TrPs and the highest hip abduction strength measures ($p=0.099$). A crossover effect was identified. A significant association was identified between a TrP status of zero TrPs on the left and right abduction strength ($p=0.026$). Participants with zero TrPs on their left side, displayed the highest strength scores for hip abduction on the right side (Figure 14).

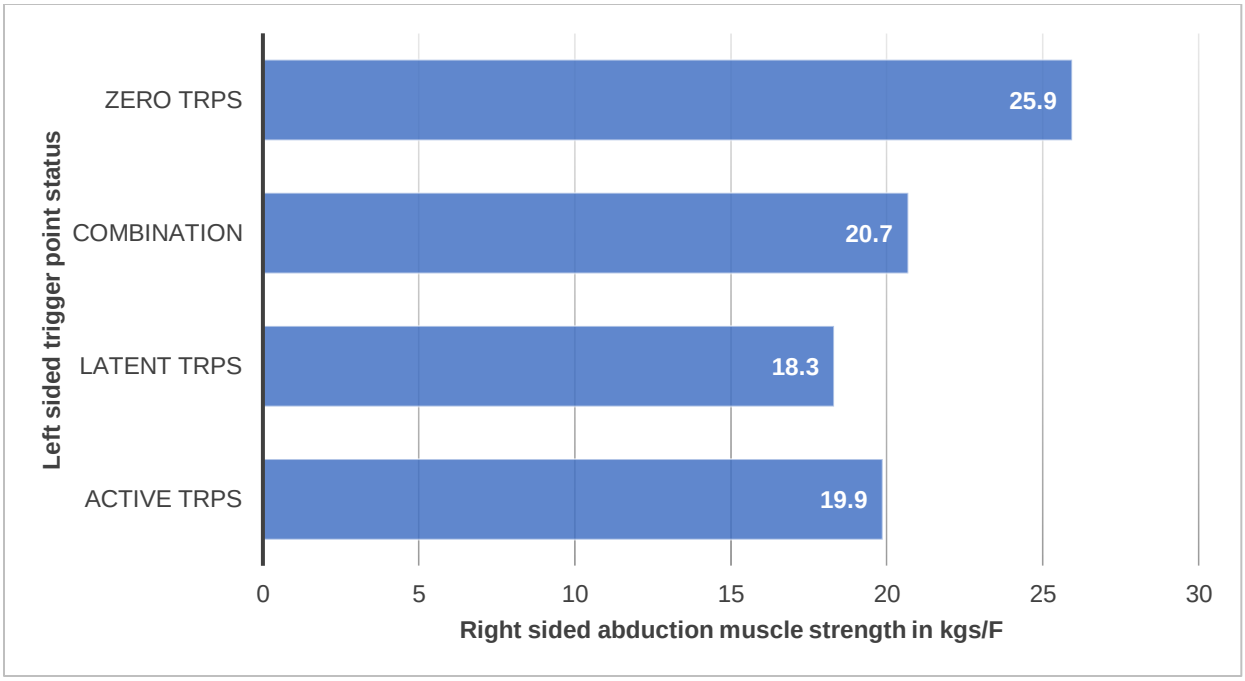



*Figure 14. The influence of left sided trigger point status on right abduction*

### 4.5.2 Trigger point influences on hip internal rotation strength

No significant associations were identified on the right side between right sided TrP status for right internal rotation strength. On the left side, a significant association was identified between a latent TrP status and left internal rotation strength (p=0.047). Participants with a status of latent TrPs on their left side displayed the lowest internal rotation strength (Figure 15).

Akin to, right abduction strength; a crossover effect was identified. There was a significant association identified between left sided TrP status of latent TrPs (p=0.004) and zero TrPs (p=0.035) with right sided internal rotation strength. Participants with a TrP status of latent TrPs on their left side, displayed the lowest right internal rotation strength. Participants with a TrP status of zero TrPs on their left side, displayed the highest internal rotation strength on their right side.

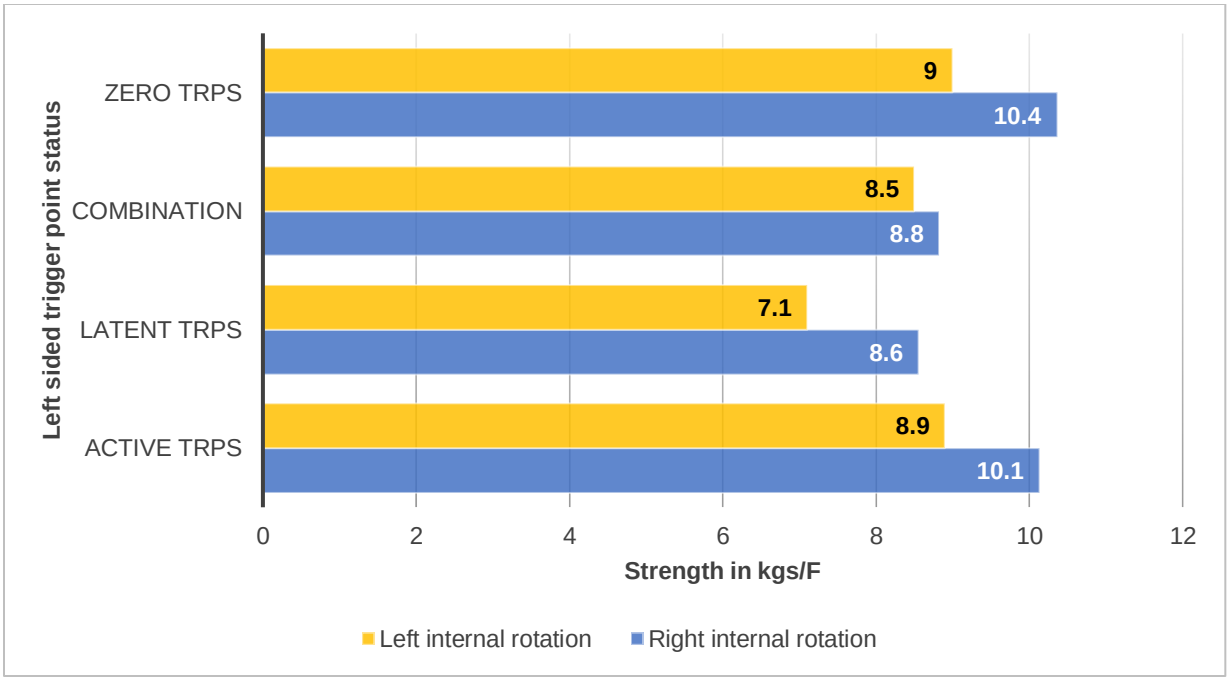



*Figure 15. The influence of left sided trigger point status on right and left internal rotation strength*

### 4.5.3 Trigger point influences on hip external rotation strength

No significant associations were identified between TrP status and right external rotation strength. On the left side, latent TrPs (p=0.015), number of latent TrPs (p=0.040) and total number of TrPs (p=0.026) were associated with external rotation strength. Participants who had latent TrPs, either with a status of latent TrPs or a combination of active and latent TrPs, achieved the lowest external rotation strength (Figure 16). No crossover effects were identified. Although, no significant associations were found between right external rotation and TrP status, strength results show that those with zero TrPs were the strongest and those with active TrPs were the weakest.

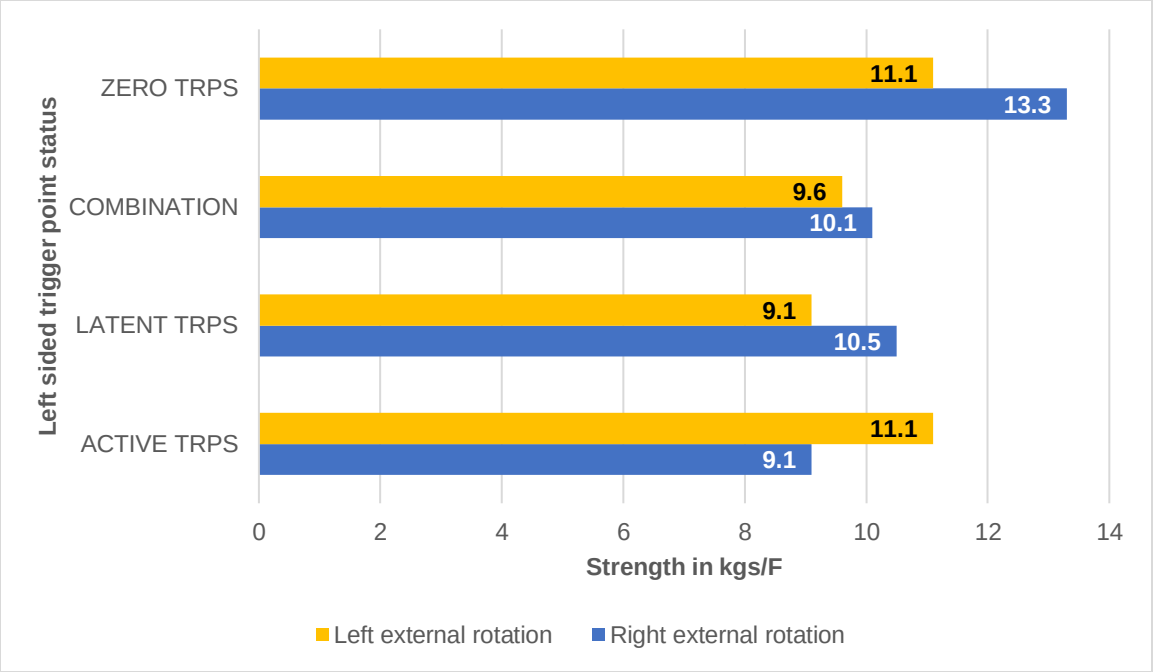



*Figure 16. The influence of left sided trigger point status on right and left external rotation strength*

Left external rotation strength was significantly associated with the number of latent TrPs on the left side. The number of latent TrPs on the left side ranged from 0 to 17 and was split into four sub-groups: 0, 1-2, 3-5, and 6+. Fourteen participants had 0 latent TrPs on the left side, ten had 1-2, eleven had 3-5, and seven had 6+ left sided latent TrPs. No significant association was seen with the groupings (p=0.075). The lowest muscle strength scores were seen in the 1-2 latent TrP group (8.8 kgs/F) and the highest scores in the group with 0 TrPs (11 kgs/F) (Figure 17). The total number of TrPs on the left side was 188. This was split into the same sub-groups as the latent TrPs. Analysis did not show an association between left external rotation strength and any of the sub-groups of total number of TrPs on the left side (p=0.111).

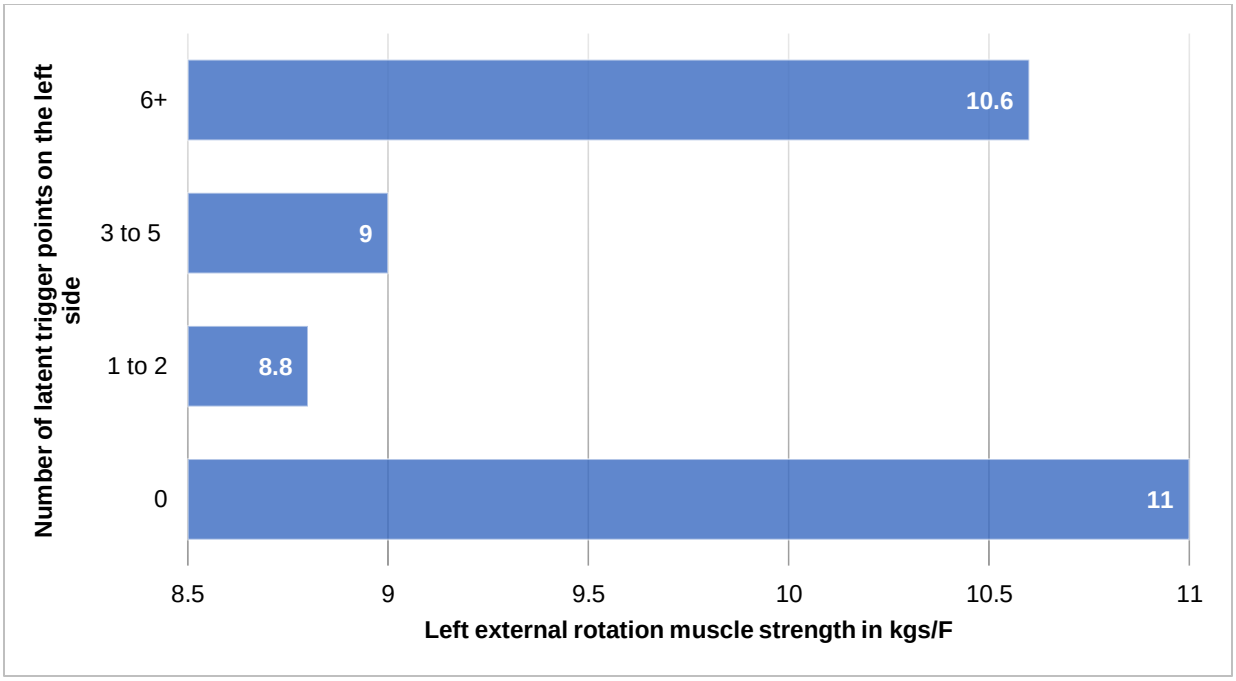



*Figure 17. The influence of left sided latent trigger points on left external rotation strength*

### 4.5.4 Summary of muscle strength results

In summary, participants with zero TrPs on the side being tested, displayed the highest muscle strength scores on that side. Participants with latent TrPs only consistently displayed the lowest scores. A summary of muscle strength results is shown in Table 10.

*Table 10. Associations between muscle strength and trigger point data*

| Strength | Association with TrP status |
|---|---|
| **Right abduction** | Left sided TrP status especially zero TrPs<br>Participants with zero TrPs on the left displayed the highest scores |
| **Left abduction** | No associations identified |
| **Right internal rotation** | Left sided TrP status especially latent TrPs and zero TrPs<br>Participants with zero TrPs on the left displayed the highest scores and those with left sided latent TrPs displayed the lowest scores |
| **Left internal rotation** | Left sided TrP status especially latent TrPs<br>Participants with left sided latent TrPs displayed the lowest scores |
| **Right external rotation** | No associations identified |
| **Left external rotation** | Left sided latent TrPs, number of latent TrPs, and total number of TrPs<br>Participants with left sided TrPs displayed the lowest scores |

There was a significant crossover effect with the highest right sided abduction (p=0.026) and internal rotation (p=0.035) strength scores in participants with zero TrPs on their left side (Table 11).

*Table 11. Crossover associations between right and left muscle strength and trigger point data*

| | Right sided associations | Left sided associations |
|---|---|---|
| **Right abduction** | No | Yes |
| **Left abduction** | No | No |
| **Right internal rotation** | No | Yes |
| **Left internal rotation** | No | No |
| **Right external rotation** | No | No |
| **Left external rotation** | No | No |

### 4.6 Other findings

From analysis of secondary outcome measures (the three questionnaires); NPRS was the only one with significant associations with TrP status. NPRS-current was significantly associated with the presence of active TrPs (p=0.006). The highest pain scores were reported by participants who displayed active TrPs on assessment (Figure 18 and Figure 19). NPRS-best was significantly associated with the presence of active TrPs on the right (p=0.019), although the same trend was not found on the left. When location of LBP was analysed in these participants, bilateral LBP was most prevalent. NPRS-worst was significantly associated with the presence of active TrPs on the left (p=0.041); this was not found on the right. The highest scores were again most prevalent in those with bilateral LBP.

For the right side, the number of active TrPs present was also significant for NPRS-current (p=0.050) and NPRS-best (p=0.004) but not for NPRS-worst. For NPRS-current and NPRS -best, the greater the number of active TrPs the higher the pain scores. On the left side, the number of active TrPs present was significantly associated for NPRS-worst (p=0.045), but not for NPRS-current and NPRS-best. For NPRS-best, there was also a significant association with the presence of right sided latent TrPs (p=0.026).

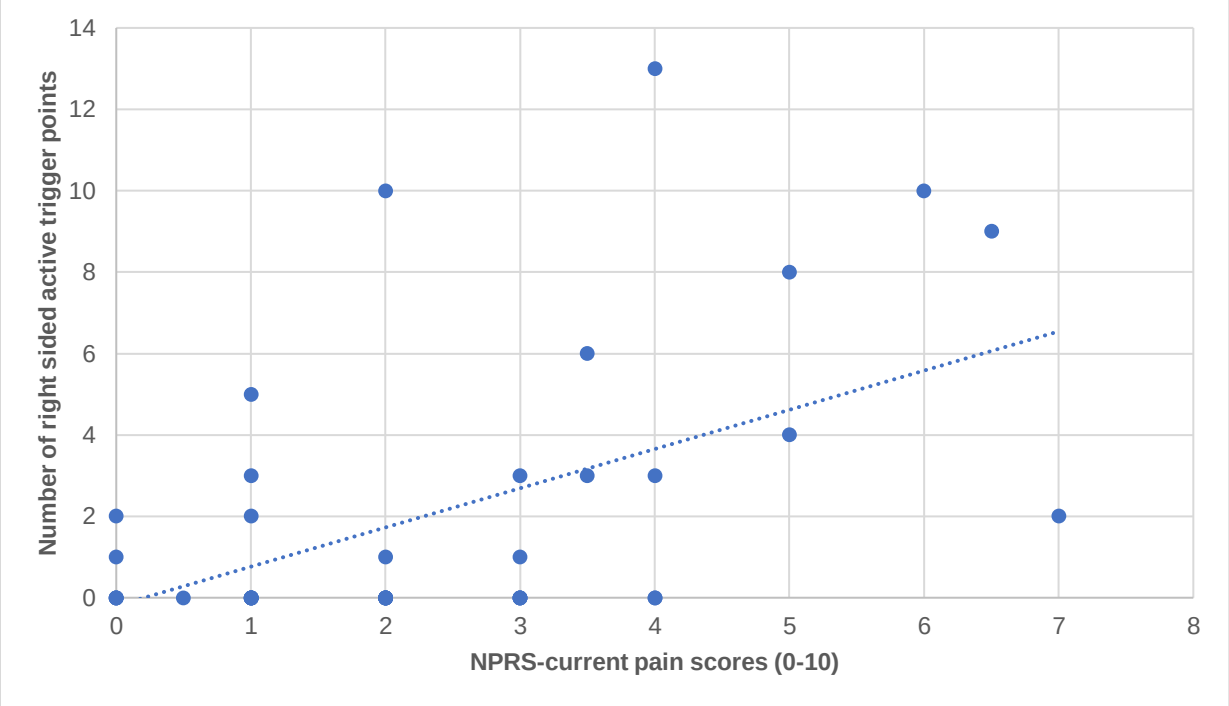



*Figure 18. Association between NPRS-current and the number of right sided active trigger points*

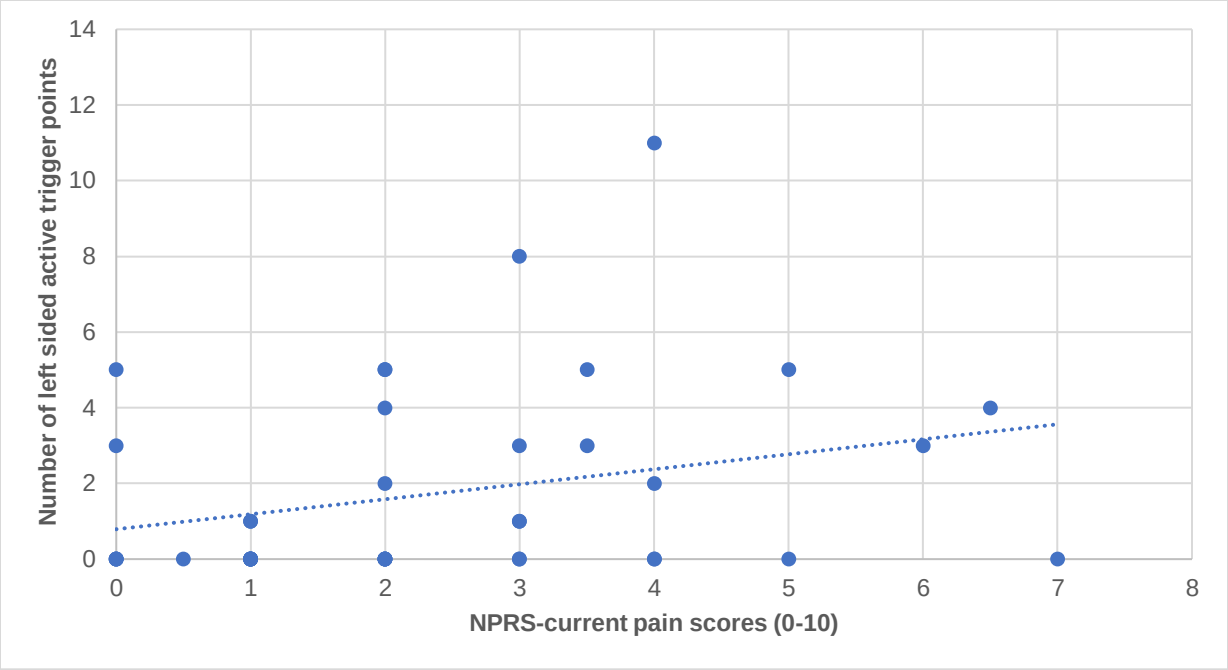
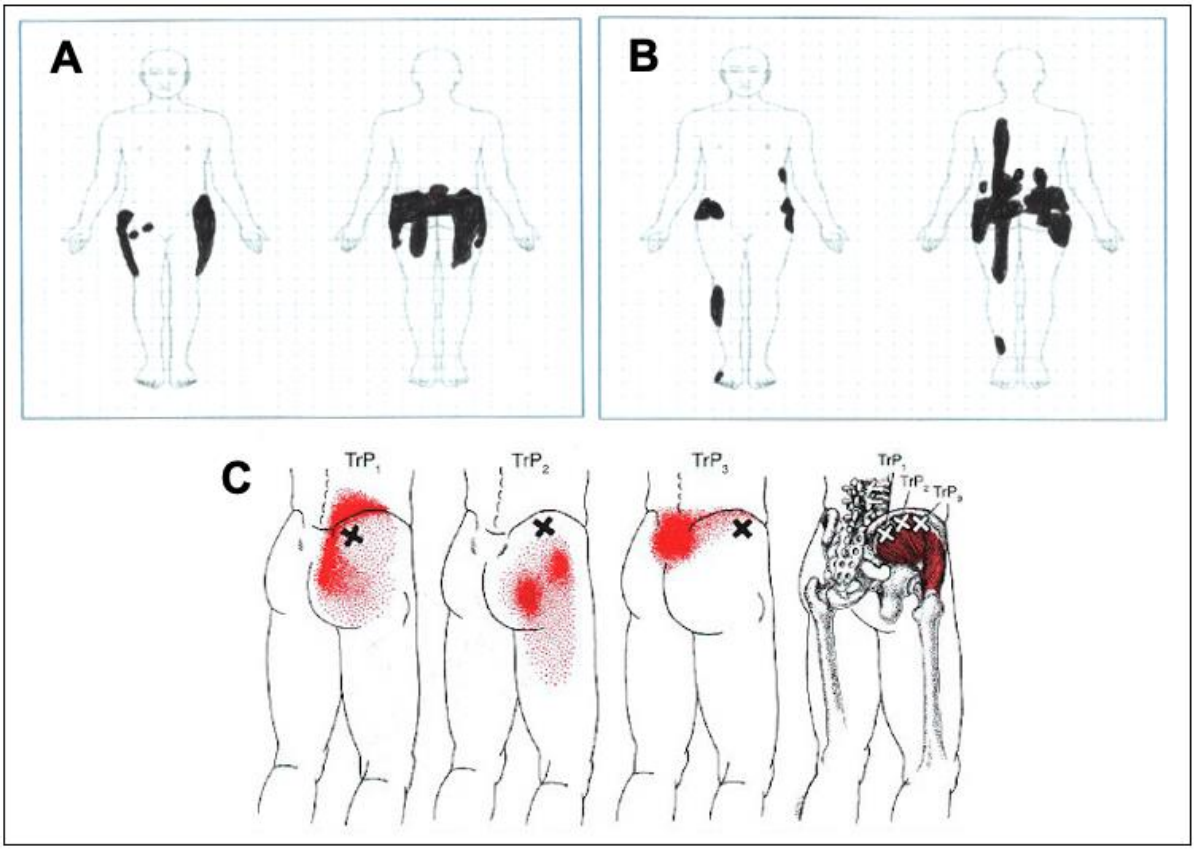



*Figure 19. Association between NPRS-current and the number of left sided active trigger points*

Referred pain was recorded on a body chart by the TrP examiner during assessment of TrPs by palpation (Figure 20). In the case of active TrPs, this referred pain was familiar to the participant, in other words it represented their LBP. Referred pain was produced on palpation of some latent TrPs. This pain was not familiar to the participants as their own pain (for example, it felt different and/or was felt in a different location). There was a wide variation in active and latent referral patterns on palpation of TrPs. No significant association was found between the presence of referred pain from a latent TrP and its influence on hip PROM or muscle strength. Out of the 31 participants, that had latent TrPs, 14 of them reported referred pain on palpation of those TrPs.




(Travell & Simons, 1992)

*Figure 20. Referral patterns for (A) active and (B) latent trigger points from included participants compared to (C) published referral patterns for active gluteus medius trigger points*

# Chapter 5. Discussion

## 5.1 Overview

The requirement for more research into muscle TrPs and their effects has never been more apparent; pain-related fear needs to be eliminated by patient education and comprehensive treatment (Cao et al., 2021). Musculoskeletal-related pain is responsible for the most prevalent conditions associated with disability and persistent pain in all ages and geographies worldwide (e.g., LBP and neck pain) (Vos et al., 2015). The current study is the first to investigate the effects of active GMed TrPs on hip PROM and muscle strength in people with CNSLBP. The hypothesis of this study was that people with CNSLBP who have active GMed TrPs will also display decreased PROM and muscle strength in their hips. This hypothesis was partly proven. People with CNSLBP who had active GMed TrPs did not display decreased PROM in their hips; but did display decreased muscle strength. This implies that people who have CNSLBP and active GMed TrPs will demonstrate weakness around their hips, but not necessarily restricted hip ROM. Combined with targeted interventions for the active TrPs themselves, treatment for people with CNSLBP should include exercises aimed at increasing hip strength.

## 5.2 Gluteus medius trigger points

There is a high prevalence of TrPs amongst people with CNSLBP (C. K. Chen & Nizar, 2011). This is especially true for the GMed muscles (Iglesias-González et al., 2013). This study found that 39 out of the 42 participants, all of whom had CNSLBP, displayed GMed TrPs either unilaterally or bilaterally. The prevalence of active GMed TrPs in this study was 52% (22 out of 42 participants). This is higher than in other studies. C. K. Chen and Nizar (2011) reported a 12% prevalence of active GMed TrPs in their cohort of participants with CLBP. Iglesias-González et al. (2013); reported that just under 50% of their participants with CNSLBP had active GMed TrPs and less had latent TrPs. In this current study, latent TrPs represented the largest TrP status group. Having one to two latent GMed TrPs was the most common presentation.

To be included in the current study, participants experienced LBP for longer than three months. This study was not case-controlled and the length of time with LBP was not specified. With increasing chronicity of LBP, it is possible that more active TrPs may have been present in participants included in this study, as was seen in the study by Iglesias-González et al. (2013). Their participants had LBP lasting longer than three years and active TrPs represented the largest TrP status when compared with the amount of latent TrPs; the opposite was found in the current study.

The most prevalent musculoskeletal-related pain worldwide are LBP and neck pain (Vos et al., 2015). Most research that has examined TrPs has been largely carried out with clinical cohorts of people suffering from headache (Fernández-de-las-Peñas, Cuadrado, et al., 2007); neck pain (Fernández-de-las-Peñas, Alonso-Blanco, et al., 2007); upper limb pain, for example, shoulder pain (Calvo-Lobo et al., 2018) and elbow pain (Fernández-Carnero et al., 2008); and patellofemoral pain (S. M. Roach, Sorenson, et al., 2013). More studies examining TrPs in people with LBP are required considering the high prevalence of TrPs in this clinical cohort.

When analysing if the presence of active GMed TrPs were associated with the symptomatic side of LBP; no significant association was identified in the current study. There was a trend towards the symptomatic side possessing more active TrPs but it did not reach significance. Iglesias-González et al. (2013) found similar results to this current study, in that there no significant association between symptomatic side of LBP and TrP status. In fact they reported more TrPs on the less painful side for active and latent TrPs. In the current study, right or left side dominance did not reach a significant association with TrP data. This correlates with the findings of Iglesias-González et al. (2013), where no significant association between GMed TrPs and dominance was reported.

The current study found that the presence of active TrPs was associated with higher pain scores when compared with latent, combination, or zero TrPs. This has been shown in other studies. In people who experienced episodic migraines; those with active TrPs displayed higher widespread pain sensitivity in the head, neck, and shoulder muscles (Palacios-Cena et al., 2017). This was also found in elderly women with bilateral knee osteoarthritis; those with active TrPs reported higher pain levels (Alburquerque-García et al., 2015). In participants with shoulder impingement, those with active TrPs in their shoulder and neck muscles had increased pain sensitivity when compared with those without (Hidalgo-Lozano et al., 2010). Biochemical changes induce higher concentrations of algogenic substances and chemical mediators at active TrPs, when compared to latent TrPs and muscle tissue without TrPs (Shah & Gilliams, 2008; Shah et al., 2005). These substances result in increased sensitivity to pain and cause nociceptors to become sensitised (Shah & Gilliams, 2008; Shah et al., 2005). Higher numbers of TrPs have also been associated with higher pain levels in musculoskeletal disorders around the head and neck (Fernández-de-las-Peñas, Simons, Cuadrado, & Pareja, 2007). This may suggest a spatial summation of nociceptor activity from sensitised peripheral nociceptors, resulting in decreased tolerance to stimulus peripherally and sensitisation of the central nervous system (Fernández-de-las-Peñas, Simons, et al., 2007).

TrPs are linked to excitability of the central nervous system due to peripheral sensitisation producing ongoing afferent input, resulting in central sensitisation (Fernández-de-las-Peñas & Dommerholt, 2014). Peripheral and central sensitisation are likely in people with CNSLBP (Chimenti et al., 2018; Clark et al., 2019). Sensitisation of GMed may occur in people with CNSLBP causing segmental sensitisation of the innervation levels of GMed (L4-S1) (Samuel et al., 2007). Multi-segmental sensitisation can occur as was shown by Hidalgo-Lozano et al. (2010); where tibialis anterior displayed a decreased threshold to pain in people with shoulder impingement who had active TrPs in the shoulder and neck muscles. Higher concentrations of algogenic substances and chemical mediators have been found in normal tissue in the gastrocnemius muscle in people with active TrPs in their trapezius muscles; suggesting widespread sensitisation (Shah et al., 2008). This may explain the widespread pain patterns recorded for active and latent TrPs in the current study.

It has been proposed that latent TrPs can progress to become active TrPs over time (Mense & Gerwin, 2010). This transition from active to latent may be linked to chronicity, repetitive movements, or altered biomechanics (Mense, 2010). The degree of sensitisation peripherally and centrally is partially responsible for the transition (Dommerholt & Fernández-de-las-Peñas, 2013). The presence of latent TrPs result in motor dysfunction because of variability and inconsistency of muscle activation timing (movement efficiency) and motor recruitment (Lucas et al., 2004; Lucas et al., 2010; Santos et al., 2013). Motor dysfunction, in this regard, presents as premature muscle fatigue, altered biomechanics (e.g., compensatory movements) and co-contraction with other muscles in other to complete an activity (Lucas et al., 2004). Changes in muscle function are due to altered motor endplate activity; which has been observed by surface electromyography for both active and latent TrPs (Florencio et al., 2017; Ge et al., 2014). Increased activity over motor units near latent TrPs is proposed to be because of compensation for weakness and premature fatigue in the muscle (Ge, Arendt-Nielsen, & Madeleine, 2012). The presence of either active or latent TrPs are implicated in sensory-motor disturbances (Florencio et al., 2017; Ge et al., 2012).

Referred pain patterns from latent GMed TrPs were collected in the current study. No significant association was identified in the current study regarding the presence of latent TrP referral and the effect on hip PROM or muscle strength. The patterns for referred pain from GMed TrPs were more extensive, diverse and widespread compared to referral patterns originally suggested by Simons (1987). It is acknowledged that the referral patterns suggested by Simons (1987) represent the most common referral patterns; but variations do exist (Fernández-de-las-

Peñas & Dommerholt, 2018). Referred pain can be described as numerous different sensations, such as, tingling or burning pain and can be poorly localised (Fernández-de-las-Peñas & Dommerholt, 2018). Referred sensations may be a better descriptor rather than referred pain and, as suggested by the current study, no specific referred sensation area should be expected (Fernández-de-las-Peñas & Dommerholt, 2018).

**5.3 The influence of gluteus medius trigger points on hip passive range of movement**

Of note was the decision to use hip PROM rather than active ROM for one of the dependent variables in the current study. The choice to use PROM was based on other studies that have also assessed hip ROM in people with CLBP (Van Dillen et al., 2008). Also, differences between active and PROM are considered to be negligible (Murray, Birley, Twycross-Lewis, & Morrissey, 2009). When active ROM and PROM were both tested for internal and external rotation of the hips in people with LBP versus a control group, there were no significant differences detected between PROM and active ROM in either the LBP or control groups (Murray et al., 2009). Furthermore, active ROM can be dependent on the motivation of the participant to achieve end of range movement, whereas, when using PROM; the examiner is performing the movement to achieve the maximum ROM available (Gerhardt, Cocchiarella, & Lea, 2002).

In trying to establish whether there was a relationship between GMed TrPs and hip PROM people with CNSLBP, the hypothesis of the current study stated that the presence of active GMed TrPs would lead to decreased hip PROM. Theoretically, the presence of an active TrP would be expected to result in decreased ROM (Simons, 1987). This was proposed to be due to sarcomere shortening at the TrP (actin and myosin filaments contracting) (Mense & Gerwin, 2010; Simons, 2004a). Fernández-de-las-Peñas, Cuadrado, et al. (2007) reported decreased total neck ROM in those with episodic tension-type headaches (participants with active TrPs in their neck muscles) when compared with controls (participants who had latent TrPs only).

The findings from the current study, however, were mixed in relation to the effect of active GMed TrPs on hip PROM, with the previously stated hypothesis being confirmed in one instance, but not confirmed in another. For hip PROM into external rotation, the presence of active TrPs (either alone or combined with latent TrPs) resulted in the lowest recorded ROM; however, this was only significantly associated on the left side. Therefore, for PROM left sided external rotation measures, the hypothesis was confirmed. This finding would be expected when based on TrP theories proposed by the existing literature (Gerwin et al., 2004; Mense & Gerwin, 2010). Restricted ROM would be expected due to sarcomere contraction in a group of muscle fibres forming a nodule and creatin tension resulting in the formation of a taut band (Simons & Travell,

1981). This increased tension and stiffness would be expected to cause restricted ROM (Simons et al., 1999).

However, for left external rotation, those with 'latent TrPs only' displayed the highest recorded PROM scores; this is contrary to current knowledge. Using sonoelastography, Calvo-Lobo et al. (2017) reported increased stiffness and mechanosensitivity of the relevant erector spinae muscles over active and latent TrPs when compared with non-TrP muscle areas in participants with lumbopelvic pain. There was no significant difference between the two types of TrPs regarding mechanosensitivity; active TrPs displayed increased stiffness when compared with latent TrPs (Calvo-Lobo et al., 2017). However, in the current study, participants with active GMed TrPs achieved the highest PROM ranges for adduction and internal rotation (statistically associated on the right side only). This finding is contrary to the hypothesis that those with active GMed TrPs would have the most restricted hip PROM.

Findings from the current study indicated that the presence of TrPs influenced the amount of PROM of the hips. Depending on the movement, the results differed. The direction of stretch or pull in the muscle may have influenced the amount of PROM present. GMed is generally divided into three sections: anterior, middle, and posterior fibres (Flack, Nicholson, & Woodley, 2012). The GMed is fan-shaped and fibre orientation differs across the muscle belly (Flack et al., 2012). The anterior fibres are involved in internal rotation and the posterior fibres are involved in external rotation (Flack et al., 2012). The findings of the current study suggest it may be worth recording the exact location of the GMed on assessment. Depending on whether the TrP is in the anterior, middle or posterior fibres of GMed; this could influence which movement of GMed may be affected. Functionally, when GMed contracts during weight bearing, the whole muscle pulls and depresses the pelvis towards the thigh on that side (McGuinness, 2010). Considering this function of preventing pelvic drop; a TrP in any part of the muscle belly could alter this mechanism. The presence of TrPs in muscle have been shown to alter muscle activation and induce early muscle fatigue in neck muscles (Florencio et al., 2017); scapular muscles (Lucas et al., 2004; Lucas et al., 2010); and trunk and hip muscles, including GMed (Santos et al., 2013).

The current study highlights the complexity of CNSLBP. For example, it is extremely difficult to determine what came first: GMed weakness or increased/decreased hip ROM, a latent/active TrP, or LBP. Increased or decreased hip ROM could be a pre-cursor to the development of LBP, due to altered biomechanics in the lumbopelvic region (Almeida et al., 2012; Cejudo et al., 2020). Judo athletes with a history of LBP displayed decreased internal rotation and total rotation of the hip for active and PROM (Almeida et al., 2012). Whereas, hip ROM was

greater for inline hockey players with a history of LBP when compared with players without LBP (Cejudo et al., 2020). Changes in hip ROM may be a compensatory mechanism to alleviate LBP (Almeida et al., 2012; Cejudo et al., 2020). In contrast, decreased hip ROM and asymmetry between right and left hips has been observed in people with CLBP (Van Dillen et al., 2008).

There are very few studies published presenting normative ranges for hip ROM (Larkin, van Holsbeeck, Koueiter, & Zaltz, 2015). K. E. Roach and Miles (1991) reviewed literature regarding hip ROM and normal ranges. The most reported "normal" range for internal and external rotation was 0-45°. However, more recent publications report normative values for adduction between 0-30°, internal rotation (tested in prone) 0-50° and external rotation (tested in prone) 0-40° (Zhang, 2021). The current study found PROM ranges for hip adduction 0-46°, internal rotation 0-55.6° and external rotation 0-58°. Therefore, the results of PROM testing in this current study show that the participants with CNSLBP displayed slightly greater ROM in their hips compared to normative values. Low quality evidence is available to support or refute whether people with CNSLBP display decreased or increased hip ROM, or if there is an association between hip ROM and CNSLBP (Avman, Osmotherly, Snodgrass, & Rivett, 2019).

Finally, there appears to be clinical uncertainty as to whether restricted ROM is an essential characteristic of a TrP. When using diagnostic criteria set by Travell and Simons (1992), the presence of restricted ROM due to pain is a major criterion. They proposed four criteria for the diagnosis of a TrP: presence of a taut band, which is hypersensitive to touch, reproduction of the person's pain (if active), and restricted ROM (Travell & Simons, 1992). In 2018 a Delphi study was carried out to try to determine the essential criteria for TrP diagnosis, as it was acknowledged that criteria varies substantially (Fernández-de-las-Peñas & Dommerholt, 2018). Of sixty experts involved, 7% of them considered restricted ROM as an essential criterion and 94% considered it a confirmatory criterion (Fernández-de-las-Peñas & Dommerholt, 2018). In 2020, a systematic review of TrP studies found that out of 198 studies, only 61.5% of studies specified how a TrP was diagnosed (Li et al., 2020). Limited ROM was included in the six most common criteria for diagnosing a TrP (Li et al., 2020).

### 5.4 The influence of gluteus medius trigger points on muscle strength

The second hypothesis of the current study stated that the presence of active GMed TrPs would lead to decreased strength in hip abduction, internal, and external rotation. This hypothesis was confirmed, though, the presence of either active or latent TrPs resulted in decreased muscle strength, whilst strength scores were highest in participants who had zero TrPs. Therefore, participants without TrPs were the strongest. Participants with active TrPs or a combination of

active and latent TrPs were the next strongest groups, with participants with latent TrPs only being the weakest.

The presence of TrPs caused weakness in GMed, however latent TrPs induced the greater weakness, rather than active TrPs. It is known that the presence of latent TrPs alters the activation of the muscle they are located in (Lucas et al., 2010). As already discussed in section 2.3.3; altered muscle activation patterns have been observed using surface electromyography over latent and active TrPs (Florencio et al., 2017; Lucas et al., 2004; Lucas et al., 2010; Santos et al., 2013). Muscle activation had high variability and did not follow an expected sequence over active TrPs (Florencio et al., 2017) and over latent TrPs when compared with non-TrP muscle tissue (Lucas et al., 2010). In healthy subjects with latent TrPs in the scapular and shoulder muscles, decreased strength in shoulder flexion and scaption was observed (Celik & Yeldan, 2011). It is difficult to explain why participants in the current study with latent TrPs were the weakest. The presence of active TrPs did result in muscle weakness, but weakness was not exacerbated by active TrPs when compared with latent TrPs. It may be expected that muscles with active TrPs may be weaker than those with latent TrPs due to the ongoing, progressive cycle of biochemical changes occurring at TrPs (Shah et al., 2005). Left external rotation weakness was most strongly associated with the presence of latent TrPs; in particular, the presence of one to two latent TrPs. Therefore, even one or two latent TrPs in a muscle was sufficient for muscle weakness to be present.

The findings from the current study regarding muscle strength may illustrate the unique role that GMed plays in pelvic and lower limb stability and weight transference. A cross-over effect was identified between TrP status on the left side and muscle strength for abduction and internal rotation on the right side. The weakest internal rotation scores on the right side were associated with latent TrP status on the left and the strongest internal rotation scores on the right side were associated with a status of zero TrPs on the left side. The strongest right abduction strength scores were associated with a status of zero TrPs on the left side. GMed's functional role works both ipsilaterally and contralaterally. The crossover functional role of GMed prevents the pelvis dropping on the opposite side to the limb that is weight bearing (McGuinness, 2010). Forty one out of the 42 participants were right side dominant, yet the TrP status on the left side was significantly associated with decreased muscle strength for right side abduction and internal rotation. GMed activates on the contralateral side to the leg that is moving when a person is transitioning from double to single leg stance in order to prevent pelvic drop (Kim, Unger, Lanovaz, & Oates, 2016). The crossover effect found in the results of the current study may highlight the

unique action of GMed in its role in contralateral pelvic and lower limb support (DeJong, Mangum, Resch, & Saliba, 2019). The crossover effect of TrP status on one side affecting hip strength on the other side as not been highlighted in any other TrP studies.

Muscle weakness was associated with the presence of TrPs and therefore, may need to be incorporated into a more robust criterion list for TrP diagnosis. In a Delphi study carried out by Fernández-de-las-Peñas and Dommerholt (2018), 60 experts had to choose criteria from a list which included muscle weakness. These criteria would be essential when diagnosing the presence of a TrP. None of the experts chose muscle weakness. There was also no mention of muscle weakness in the criteria for diagnosis of a TrP in a more recent systematic review of TrP studies by Li et al. (2020).

In summary, hip weakness was observed in participants with CNSLBP who displayed TrPs, with the lowest scores in those who had latent TrPs only. When attempting to unravel the role that TrPs play in GMed weakness, the reason for the TrP being present needs to be pondered. Considering the Integrative theory of TrP formation proposed by Simons and Travell (1981); the energy crisis occurs in GMed due to an insult to the muscle. This could be in the form of an injury, repetitive movement, or over-use of the muscle. A latent TrP is formed as a result. Due to the presence of the TrP, muscle weakness ensues. In this scenario, the latent TrP pre-empts the muscle weakness (Travell & Simons, 1992). However, when considering the “Shift model” of TrP formation (Minerbi & Vulfsons, 2018); it would be proposed that a weakened GMed muscle is present first. It has been weakened due to the relaxation/contraction ratio being altered to a certain threshold which causes the energy crisis to occur. As a result, a latent TrP is formed (Minerbi & Vulfsons, 2018). In this proposed mechanism, the GMed muscle is already weakened before the TrP is present. GMed TrPs and muscle weakness could be a causative factor in developing CNSLBP (Cooper et al., 2016). This current study has identified an association between the presence of TrPs and muscle weakness; more research into this association may add to knowledge about this clinical presentation and how best to address these findings; whether that be treatment of the TrP, or exercises aimed at increasing hip muscle strength.

### 5.5 The influence of confounding variables

The effect of confounding variables was considered during analysis and in reference to the literature. For PROM ‘weight group’ was taken forwards as a covariate that may influence adduction PROM, because of the results of analysis and on consideration of the possible effects of testing positions. The participants were in a side-lying position for PROM adduction testing; their bottom leg was passively adducted and measured. Therefore, the bottom lateral hip

(including GMed) of the tested leg was compressed because of the side-lying position. In the current study, the presence of hip pain was an exclusion criterion. In side-lying the attachment site for GMed was compressed and this could have impacted adduction PROM. Participant comfort needs to be considered in future research, regarding positioning for passive hip adduction testing.

The confounding variable of gender was chosen for internal and external rotation PROM as it is known from the literature that total rotation of the hip, in particular internal rotation, is increased in females when compared with males in those with and without hip pain (Freke et al., 2019; Hogg et al., 2018). The ODQ showed an association with decreased hip external rotation ROM. Higher ODQ scores resulted in decreased ROM. This has also been confirmed in the literature (Steultjens et al., 2000). In those who reported higher disability due to their hip or knee pain (because of osteoarthritis), decreased ROM was recorded (Steultjens et al., 2000).

On analysis of possible confounding variables affecting muscle strength in the current study, gender was statistically associated for all movements and sides. Males were found to display increased strength when compared with females. Evidence suggests healthy young males are stronger than young females during hip abduction testing (Matsumura et al., 2015). In addition, age groups, and ODQ were also statistically associated with both internal and external rotation. Increased age and ODQ scores were associated with decreased strength scores. From the literature, older females (aged 69-82 years) display decreased overall hip strength when compared with younger females (21-28 years) (both groups experienced no hip pain) (Dean, Kuo, & Alexander, 2004).

**5.6 Clinical implications**

The current study aimed to identify if there was a relationship between active GMed TrPs and decreased hip PROM and muscle strength in people with CNSLBP. Firstly, regarding hip PROM, the presence of active TrPs had different affects depending on which hip movement was being tested. The presence of active TrPs resulted in the lowest hip PROM for left external rotation but the highest PROM for right adduction and internal rotation. Clinically, it may be of note to record the location of the TrP within GMed as this may give more information as to which hip movement may be restricted. Depending on the location of the TrP and the direction of pull of the muscle fibres it is located in; only one movement of GMed may be affected (Flack et al., 2012).

Secondly, regarding active GMed TrPs and decreased muscle strength in people with CNSLBP; the presence of active GMed TrPs did result in decreased muscle strength as was also

seen for the presence of latent TrPs. The participants with zero TrPs were the strongest, those with latent TrPs were the weakest, though the presence of active GMed TrPs did result in decreased hip muscle strength. Clinically, if active GMed TrPs are present on assessment; it is likely that hip strength will be decreased. Furthermore, the presence of latent TrPs should also be considered as their effect may also be clinically influential upon hip muscle strength. All the participants had CNSLBP, and their hip strength did differ depending on their TrP status.

Clinically, it is evident that the presence of GMed TrPs, whether they are active or latent, results in decreased hip strength. When assessing people with CNSLBP, the presence of whether TrPs are present in GMed needs to be identified. Treating TrPs and prescribing exercises aimed at increasing hip strength may be beneficial. When assessing the potential clinical contribution that active GMed TrPs could have in people with CNSLBP, both sides need to be assessed (Cooper et al., 2016). In the current study, 25 out of the 39 participants that displayed GMed TrPs, had TrPs bilaterally.

### 5.7 Limitations and areas for future research

There were some limitations associated with the current study. During the assessment, the participants were not asked to specify if their LBP was right sided, left sided, central, or bilateral. This information was extrapolated from where the participant had marked their LBP on the body chart given to them at the assessment. Asking the participants directly about the location of their LBP may have given clearer descriptions regarding the location of their LBP. No associations were found between the site of LBP and TrP data in this study, but this could be further analysed more specifically in future research.

In the current study, it would have been helpful to specifically ask the participants about their lower limb dominance as hip strength was being assessed. Participants were asked to specify if they were right or left side dominant, but this question was not specific to lower limb dominance. It was not considered that some people may have a different upper limb and lower limb dominance, and this should be clarified in future research investigating lower limb muscle strength.

It would have been helpful to record the length of time that participants had been experiencing their CNSLBP. There could have been potential discussion points around the effects of TrPs on sensory-motor dysfunction and central sensitisation with chronicity of LBP. Even though these measures are incredibly difficult to assess, it would have been of interest to compare chronicity of CNSLBP with TrP status and number of TrPs present. A control group would have

added strength to the conclusions being drawn from the results. A control group would have given good comparison for the prevalence of TrPs and TrP status, and the presence of decreased hip PROM and muscle strength in the general population and in those without CNSLBP.

The accuracy of diagnosing TrPs using criteria is different for each muscle being assessed. Location of the muscle is one such factor; GMed is located deep to gluteus maximus and as a result may have been affected by TrPs in this muscle (Fernández-de-las-Peñas & Dommerholt, 2018). There is moderate evidence for the reproducibility of referred sensations from GMed (Myburgh, Larsen, & Hartvigsen, 2008). In future research, all factors that may affect diagnosis of TrP in the specific muscle being assessed need to be considered.

To keep the statistical analyses within the scope of Masters level research and under that assumption that left and right sides acted independently, analysis examined right and left sides separately. Left/right crossover effects were examined and confirmed in this current study which may inform future research. There is a lot of scope for future research into TrPs. As an association was established between hip active GMed TrPs and decreased hip muscle strength; future research could investigate if treating the TrP restores hip strength. Following treatment of a TrP, muscle strength could be reassessed. It is an advantage of this study that the techniques used for muscle strength testing that can be reproduced by clinicians in practice. Most research studies assessing muscle strength use expensive and large pieces of equipment e.g., Biodex; which is inaccessible and too costly for a clinician to use. Regarding the presence of TrPs and their effect on hip PROM; other factors appeared to be present. Further research into the possibility of the location of the TrP being of importance as to which hip PROM movement is affected would shed more light on these results.

GMed works as part of the lumbopelvic support unit (Dorado et al., 2020). Considering GMed with other muscles in this unit, for example quadratus lumborum may be helpful for the treatment of CNSLBP and other clinical conditions. In women with patellofemoral pain, treatment of GMed and quadratus lumborum TrPs with dry needling alongside exercise therapy resulted in better pain and function scores when compared with exercise alone (Zarei, Bervis, Piroozi, & Motealleh, 2020).

Further analysis could be carried out comparing the roles of active and latent TrPs and the significance (if any) of the number of TrPs present. More information would simplify assessment and treatment of these clinical presentations. More ultrasound studies are required

to further expand the knowledge around the physiological changes at and around a TrP; as currently, the knowledge is mainly theoretical (Srbely et al., 2016).

### 5.8 Conclusions

Thirty-nine out of 42 participants in this study displayed GMed TrPs. The presence of latent TrPs only was the most common TrP status. The presence of GMed TrPs produced varying results regarding hip PROM. Some movements with active or latent only TrPs produced the highest ranges and some movements with zero or combination of TrPs produced the most restricted ranges. For muscle strength, participants with zero TrPs displayed the strongest muscle strength scores. Those with latent TrPs were the weakest in all directions.

GMed TrPs are highly prevalent in people with CNSLBP and should be included in a general assessment of this patient population. Bilateral GMed assessment is recommended on assessing of the presence of GMed TrPs. It is not yet clear what role if any, they play regarding their effect on PROM. Their role in decreased muscle strength and causation of pain appears to be significant. When diagnosing the presence of a TrP, restricted PROM may not be a significant criterion. This study suggests that muscle weakness may be a more helpful finding when determining the presence of a TrP and clinically reasoning its effects on the muscle.

# References


Accident Compensation Corporation. (2004). *New Zealand acute low back pain guide*. Retrieved from https://www.acc.co.nz/assets/provider/acc1038-lower-back-pain-guide.pdf. https://doi.org/https://www.acc.co.nz/assets/provider/acc1038-lower-back-pain-guide.pdf

Accident Compensation Corporation. (2021). *Injuries we cover*. Retrieved from www.acc.co.nz

Akamatsu, F. E., Yendo, T. M., Rhode, C., Marialtezerote, A., Hojaij, F., Andrade, M., . . . Jacomo, A. L. (2017). Anatomical basis of the myofascial trigger points of the gluteus maximus muscle. *BioMed Research International*. https://doi.org/10.1155/2017/4821968

Akodu, A. K., & Odunfa, K. S. (2020). Effects of core stabilization and McKenzie back extension exercises on pain, disability and insomnia in patients with non-specific chronic low back pain. *Sports Medicine Journal / Medicina Sportivâ, 16*(1), 3190-3196. Retrieved from http://www.medicinasportiva.pl

Alburquerque-García, A., Rodrigues-de-Souza, D. P., Fernández-de-las-Peñas, C., & Alburquerque-Sendín, F. (2015). Association between muscle trigger points, ongoing pain, function, and sleep quality in elderly women with bilateral painful knee osteoarthritis. *Journal of Manipulative and Physiological Therapeutics, 38*(4), 262-268. https://doi.org/10.1016/j.jmpt.2014.10.018

Almeida, G. P. L., de Souza, V. L., Sano, S. S., Saccol, M. F., & Cohen, M. (2012). Comparison of hip rotation range of motion in judo athletes with and without history of low back pain. *Manual Therapy, 17*(3), 231-235. https://doi.org/10.1016/j.math.2012.01.004

Andersson, G. B. J. (1999). Epidemiological features of chronic low-back pain. *Lancet, 354*(9178), 581-585. https://doi.org/10.1016/S0140-6736(99)01312-4

Arab, A. M., & Nourbakhsh, M. R. (2010). The relationship between hip abductor muscle strength and iliotibial band tightness in individuals with low back pain. *Chiropractic & Osteopathy, 18*(1), 1-5. https://doi.org/10.1186/1746-1340-18-1

Avman, M. A., Osmotherly, P. G., Snodgrass, S., & Rivett, D. A. (2019). Is there an association between hip range of motion and nonspecific low back pain? A systematic review. *Musculoskeletal Science and Practice, 42*, 38-51. https://doi.org/10.1016/j.msksp.2019.03.002

Bajaj, P., Bajaj, P., Graven-Nielsen, T., & Arendt-Nielsen, L. (2001). Trigger points in patients with lower limb osteoarthritis. *Journal of Musculoskeletal Pain, 9*(3), 17. Retrieved from https://www-tandfonline-com.ezproxy.aut.ac.nz/toc/imup20/current

Baldry, P. (1995). Superficial dry needling at myofascial trigger point sites. *Journal of Musculoskeletal Pain, 3*(3), 117-126. https://doi.org/10.1300/J094v03n03_10

Barbero, M., Bertoli, P., Cescon, C., Macmillan, F., Coutts, F., & Gatti, R. (2012). Intra-rater reliability of an experienced physiotherapist in locating myofascial trigger points in upper trapezius muscle. *Journal of Manual and Manipulative Therapy, 20*(4), 171-177. https://doi.org/10.1179/2042618612Y.0000000010

Bardin, L. D., King, P., & Maher, C. G. (2017). Diagnostic triage for low back pain: A practical approach for primary care. *Medical Journal of Australia, 206*(6), 268-273. https://doi.org/10.5694/mja16.00828

Basford, J., Chen, Q., Wang, J. J., Gay, R., Thompson, J., Manduca, A., & An, K. N. (2014). Rater reliability in the quantification of myofascial taut bands. *Annals of Physical and Rehabilitation Medicine, 57*, e250-e250. https://doi.org/10.1016/j.rehab.2014.03.909

Bazett-Jones, D. M., Tylinksi, T., Krstic, J., Stromquist, A., & Sparks, J. (2017). Peak hip muscle torque measurements are influenced by sagittal plane hip position. *International Journal of Sports Physical Therapy, 12*(4), 535-542. Retrieved from https://ifspt.org/

Bisset, L. M., Russell, T., Bradley, S., Ha, B., & Vicenzino, B. T. (2006). Bilateral sensorimotor abnormalities in unilateral lateral epicondylalgia. *Archives of Physical Medicine and Rehabilitation, 87*(4), 490-495. https://doi.org/10.1016/j.apmr.2005.11.029

Bogduk, N., Aprill, C., & Derby, R. (2013). Lumbar discogenic pain: State-of-the-art review. *Pain Medicine, 14*(6), 813-836. Retrieved from https://academic.oup.com/painmedicine

Bohannon, R. W. (1999). Intertester reliability of hand-held dynamometry: A concise summary of published research. *Perceptual and Motor Skills, 88*(3), 899-902. Retrieved from https://journals.sagepub.com

Bourgaize, S., Newton, G., Kumbhare, D., & Srbely, J. Z. (2018). A comparison of the clinical manifestation and pathophysiology of myofascial pain syndrome and fibromyalgia: implications for differential diagnosis and management. *Journal of the Canadian Chiropractic Association, 62*(1), 26-41. Retrieved from https://www.chiropractic.ca/jcca-online/

Bron, C., & Dommerholt, J. D. (2012). Etiology of myofascial trigger points. *Current Pain and Headache Reports, 16*(5), 439-444. https://doi.org/10.1007/s11916-012-0289-4

Bron, C., Franssen, J., Wensing, M., & Oostendorp, R. A. B. (2007). Interrater reliability of palpation of myofascial trigger points in three shoulder muscles. *Journal of Manual & Manipulative Therapy, 15*(4), 203-215. Retrieved from https://www-tandfonline-com.ezproxy.aut.ac.nz/loi/yjmt20

Calvo-Lobo, C., Diez-Vega, I., Garrosa-Martín, G., Martínez-Pascual, B., Fernández-Martínez, S., De La Cueva-Reguera, M., & Rodríguez-Sanz, D. (2017). Tensiomyography, sonoelastography, and mechanosensitivity differences between active, latent, and control low back myofascial trigger points. *Medicine (United States), 96*(10). https://doi.org/10.1097/MD.0000000000006287

Calvo-Lobo, C., Pacheco-da-Costa, S., Martinez-Martinez, J., Rodriguez-Sanz, D., Cuesta-Alvaro, P., & Lopez-Lopez, D. (2018). Dry needling on the infraspinatus latent and active myofascial trigger points in older adults with nonspecific shoulder pain: A randomized clinical trial. *Journal of Geriatric Physical Therapy, 41*(1), 1-13. https://doi.org/10.1519/JPT.0000000000000079

Camanho, G. L., Imamura, M., & Arendt-Nielsen, L. (2011). Genesis of pain in arthrosis. *Revista Brasileira de Ortopedia, 46*(1), 14-17. https://doi.org/10.1590/S0102-36162011000100002

Cao, Q.-W., Peng, B.-G., Wang, L., Huang, Y.-Q., Jia, D.-L., Jiang, H., . . . Huang, D. (2021). Expert Consencus on the diagnosis and treatment of myofascial pain syndrome. *World Journal of Clinical Cases, 9*(9), 2077-2089. https://doi.org/10.12998/wjcc.v9.i9.2077

Cejudo, A., Moreno-Alcaraz, V. J., Izzo, R., Santonja-Medina, F., & Sainz de Baranda, P. (2020). External and total hip rotation ranges of motion predispose to low back pain in elite spanish inline hockey players. *International Journal of Environmental Research and Public Health, 17*(4858), 4858-4858. https://doi.org/10.3390/ijerph17134858

Celik, D., & Yeldan, İ. (2011). The relationship between latent trigger point and muscle strength in healthy subjects: A double-blind study. *Journal of Back and Musculoskeletal Rehabilitation, 24*(4), 251-256. https://doi.org/10.3233/BMR-2011-0302

Celletti, C., Paolucci, T., Maggi, L., Volpi, G., Billi, M., Mollica, R., & Camerota, F. (2021). Pain management through neurocognitive therapeutic exercises in hypermobile Ehlers–Danlos Syndrome Patients with chronic low back pain. *BioMed Research International*, 1-7. https://doi.org/10.1155/2021/6664864

Chapman, J. R., Norvell, D. C., Hermsmeyer, J. T., Bransford, R. J., DeVine, J., McGirt, M. J., & Lee, M. J. (2011). Evaluating common outcomes for measuring treatment success for chronic low back pain. *Spine, 36*(21), 54-68. https://doi.org/10.1097/BRS.0b013e31822ef74d

Charlton, P. C., Mentiplay, B. F., Pua, Y.-H., & Clark, R. A. (2015). Reliability and concurrent validity of a Smartphone, bubble inclinometer and motion analysis system for

measurement of hip joint range of motion. *Journal of Science and Medicine in Sport, 18*, 262-267. https://doi.org/10.1016/j.jsams.2014.04.008

Chen, C. K., & Nizar, A. J. (2011). Myofascial pain syndrome in chronic back pain patients. *Korean Journal of Pain, 24*(2), 100-104. https://doi.org/10.3344/kjp.2011.24.2.100

Chen, Q., Wang, H., Gay, R. E., Thompson, J. M., Manduca, A., An, K., . . . Basford, J. R. (2016). Quantification of myofascial taut bands. *Archives of Physical Medicine and Rehabilitation, 97*(1), 67-73. https://doi.org/10.1016/j.apmr.2015.09.019

Childs, J. D., Piva, S. R., Fritz, J. M., Childs, J. D., Piva, S. R., & Fritz, J. M. (2005). Responsiveness of the numeric pain rating scale in patients with low back pain. *Spine, 30*(11), 1331-1334. https://doi.org/10.1097/01.brs.0000164099.92112.29

Chimenti, R. L., Frey-Law, L. A., & Sluka, K. A. (2018). A mechanism-based approach to physical therapist management of pain. *Physical Therapy, 98*(5), 302-314. https://doi.org/10.1093/ptj/pzy030

Clark, J. R., Goodwin, P. C., & Yeowell, G. (2019). Exploring the pre-morbid contexts in which central sensitisation developed in individuals with non-specific chronic low back pain. A qualitative study. *Brazilian Journal of Physical Therapy / Revista Brasileira de Fisioterapia, 23*(6), 516-526. Retrieved from https://www.journals.elsevier.com/brazilian-journal-of-physical-therapy

Clauw, D. J. (2014). Fibromyalgia. A clinical review. *Journal of the American Medical Association, 311*(15). https://doi.org/10.1001/jama.2014.3266

Cook, C. E., Denninger, T., Lewis, J., Diener, I., & Thigpen, C. (2021). Providing value-based care as a physiotherapist. *Archives of Physiotherapy, 11*(1), 1-5. https://doi.org/10.1186/s40945-021-00107-0

Cooper, N. A., Scavo, K. M., Strickland, K. J., Tipayamongkol, N., Nicholson, J. D., Bewyer, D. C., & Sluka, K. A. (2016). Prevalence of gluteus medius weakness in people with chronic low back pain compared to healthy controls. *European Spine Journal, 25*(4), 1258-1265. https://doi.org/10.1007/s00586-015-4027-6

Davidson, M., & Keating, J. L. (2002). A comparison of five low back disability questionnaires: reliability and responsiveness. *Physical Therapy, 82*(1), 8-24. https://doi.org/10.1093/ptj/82.1.8

de Oliveira Meirelles, F., de Oliveira Muniz Cunha, J. C., & da Silva, E. B. (2020). Osteopathic manipulation treatment versus therapeutic exercises in patients with chronic nonspecific low back pain: A randomized, controlled and double-blind study. *Journal of Back and Musculoskeletal Rehabilitation, 33*(3), 367-377. https://doi.org/10.3233/BMR-181355

Dean, J. C., Kuo, A. D., & Alexander, N. B. (2004). Age-related changes in maximal hip strength and movement speed. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences, 59*(3), 286-292. https://doi.org/10.1093/gerona/59.3.m286

DeJong, A. F., Mangum, L. C., Resch, J. E., & Saliba, S. A. (2019). Detection of gluteal changes using ultrasound imaging during phases of gait in individuals with medial knee displacement. *Journal of Sport Rehabilitation, 28*, 494-504. https://doi.org/10.1125/jsr.2017-0336

Dommerholt, J., Bron, C., & Franssen, J. (2006). Myofascial trigger points: An evidence-informed review. *Journal of Manual & Manipulative Therapy, 14*(4), 203-221. Retrieved from https://www.tandfonline.com/toc/yjmt20/current

Dommerholt, J., & Fernández-de-las-Peñas, C. (2013). *Trigger point dry needling E-book: An evidence and clinical-based approach*. https://doi.org/10.1016/B978-0-7020-4601-8.00001-3

Donnelly, J. M. (2019). *Travell, Simons & Simons' myofascial pain and dysfunction : the trigger point manual* (Third ed.). Retrieved from http://ebookcentral.proquest.com/lib/AUT/detail.action?docID=5944634

Dorado, C., López-Gordillo, A., Serrano-Sánchez, J. A., Calbet, J. A. L., & Sanchis-Moysi, J. (2020). Hypertrophy of lumbopelvic muscles in inactive women: A 36-week pilates study. *Sports health, 12*(6), 547-551. https://doi.org/10.1177/1941738120918381

Dorsher, P. T. (2008). Can classical acupuncture points and trigger points be compared in the treatment of pain disorders? Birch's analysis revisited. *The Journal of Alternative and Complementary Medicine, 14*(4), 353-359. https://doi.org/10.1089/acm.2007.0810

Duarte, F. C. K., West, D. W. D., Linde, L. D., Hassan, S., & Kumbhare, D. A. (2021). Re-examining myofascial pain syndrome: Toward biomarker development and mechanism-based diagnostic criteria. *Current Rheumatology Reports, 23*(8), 69. https://doi.org/10.1007/s11926-021-01024-8

Engle, A. M., Chen, Y., Marascalchi, B., Wilkinson, I., Abrams, W. B., He, C., . . . Cohen, S. P. (2019). Lumbosacral radiculopathy: Inciting events and their association with epidural steroid injection outcomes. *Pain Medicine, 20*(12), 2360-2370. https://doi.org/10.1093/pm/pnz097

Evans, V., Behr, M., Gangwar, A., Noseworthy, M. D., & Kumbhare, D. (2021). Potential role of MRI imaging for myofascial pain: A scoping review for the clinicians and theoretical considerations. *Journal of Pain Research, 14*, 1505-1514. https://doi.org/10.2147/JPR.S302683

Fagundes Loss, J., de Souza da Silva, L., Iã Ferreira, M., Groisman, S., Santiago Wagner Neto, E. S., C., & Tarragô Candotti, C. (2020). Immediate effects of a lumbar spine manipulation on pain sensitivity and postural control in individuals with nonspecific low back pain: a randomized controlled trial. *Chiropractic & Manual Therapies, 28*(1), 1-10. https://doi.org/10.1186/s12998-020-00316-7

Fairbank, J. C. T., & Pynsent, P. B. (2000). The oswestry disability index. *Spine, 25*(22), 2940-2953. https://doi.org/10.1097/00007632-200011150-00017

Fatoye, F., Gebrye, T., & Odeyemi, I. (2019). Real-world incidence and prevalence of low back pain using routinely collected data. *Rheumatology International, 39*, 619-626. https://doi.org/10.1007/s00296-019-04273-0

Fernández-Carnero, J., Fernández-de-las-Peñas, C., de la Llave-Rincón, A. I., Ge, H. Y., & Arendt-Nielsen, L. (2007). Prevalence of and referred pain from myofascial trigger points in the forearm muscles in patients with lateral epicondylalgia. *Clinical Journal of Pain, 23*(4), 353-360. https://doi.org/10.1097/AJP.0b013e31803b3785

Fernández-Carnero, J., Fernández-de-las-Peñas, C., de la Llave-Rincón, A. I., Ge, H. Y., & Arendt-Nielsen, L. (2008). Bilateral myofascial trigger points in the forearm muscles in patients with chronic unilateral lateral epicondylalgia: A blinded, controlled study. *Clinical Journal of Pain, 24*(9), 802-807. https://doi.org/10.1097/AJP.0b013e31817bcb79

Fernández-de-las-Peñas, C., Alonso-Blanco, C., & Miangolarra, J. C. (2007). Myofascial trigger points in subjects presenting with mechanical neck pain: A blinded, controlled study. *Manual Therapy, 12*(1), 29-33. https://doi.org/10.1016/j.math.2006.02.002

Fernández-de-las-Peñas, C., Cuadrado, M. L., & Pareja, J. A. (2007). Myofascial trigger points, neck mobility, and forward head posture in episodic tension-type headache. *Headache: The Journal of Head & Face Pain, 47*(5), 662-672. https://doi.org/10.1111/j.1526-44610.2006.00632.x

Fernández-de-las-Peñas, C., & Dommerholt, J. (2014). Myofascial trigger points: Peripheral or central phenomenon? *Current Rheumatology Reports, 16*(1). https://doi.org/10.1007/s11926-013-0395-2

Fernández-de-las-Peñas, C., & Dommerholt, J. (2018). International consensus on diagnostic criteria and clinical considerations of myofascial trigger points: A delphi study. *Pain Medicine, 19*(1), 142-150. https://doi.org/10.1093/pm/pnx207

Fernández-de-las-Peñas, C., Simons, D., Cuadrado, M. L., & Pareja, J. (2007). The role of myofascial trigger points in musculoskeletal pain syndromes of the head and neck.

*Current pain and headache reports, 11*(5), 365-372. https://doi.org/10.1007/s11916-007-0219-z

Field, D. (2008). *Field's lower limb anatomy, palpation, and surface markings*. Retrieved from https://ezproxy.aut.ac.nz/login?url=https://www.sciencedirect.com/science/book/9780702030185

Fitzcharles, M.-A., Cohen, S. P., Clauw, D. J., Littlejohn, G., Usui, C., & Häuser, W. (2021). Nociplastic pain: towards an understanding of prevalent pain conditions. *Lancet, 397*(10289), 2098-2110. https://doi.org/10.1016/S0140-6736(21)00392-5

Flack, N. A. M. S., Nicholson, H. D., & Woodley, S. J. (2012). A review of the anatomy of the hip abductor muscles, gluteus medius, gluteus minimus, and tensor fascia lata. *Clinical Anatomy, 25*(6), 697-708. https://doi.org/10.1002/ca.22004

Florencio, L. L., Grossi, D. B., Ferracini, G. N., Chaves, T. C., Speciali, J. G., Palacios-Ceña, M., . . . Falla, D. (2017). Active trigger points in the cervical musculature determine the altered activation of superficial neck and extensor muscles in women with migraine. *Clinical Journal of Pain, 33*(3), 238-245. https://doi.org/10.1097/AJP.0000000000000390

Foster, N. E., Anema, J. R., Cherkin, D., Chou, R., Cohen, S. P., Gross, D. P., . . . Maher, C. G. (2018). Prevention and treatment of low back pain: evidence, challenges, and promising directions. *Lancet, 391*(10137), 2368-2383. https://doi.org/10.1016/S0140-6736(18)30489-6

Fraeulin, L., Holzgreve, F., Brinkbaeumer, M., Dziuba, A., Friebe, D., Klemz, S., . . . Ohlendorf, D. (2020). Intra- and inter-rater reliability of joint range of motion tests using tape measure, digital inclinometer and inertial motion capturing. *PloS One, 15*(12). https://doi.org/10.1371/journal.pone.0243646

Freke, M., Kemp, J., Crossley, K., Sims, K., Russell, T., & Semciw, A. (2019). Strength and range of movement deficits are associated with symptom severity in people scheduled for hip arthroscopy. *European Journal of Pain (London, England), 23*(6), 1083-1090. https://doi.org/10.1002/ejp.1371

Galasso, A., Urits, I., An, D., Nguyen, D., Borchart, M., Yazdi, C., . . . Viswanath, O. (2020). A comprehensive review of the treatment and management of myofascial pain syndrome. *Current pain and headache reports, 24*(8), 43. https://doi.org/10.1007/s11916-020-00877-5

Gasibat, Q., Simbak, N., Aziz, A. A., & Musa, R. M. (2017). Effect of the stabilization training programme in the improvement of trunk and hip muscles activations among healthy

females subject: An implication for the rehabilitation experts. *Journal of Physical Education & Sport, 17*, 948-954. Retrieved from https://efsupit.ro/

Ge, H.-Y., Arendt-Nielsen, L., & Madeleine, P. (2012). Accelerated muscle fatigability of latent myofascial trigger points in humans. *Pain Medicine, 13*(7), 957-964. Retrieved from https://painmed.org/pain-medicine-journal/

Ge, H.-Y., Monterde, S., Graven-Nielsen, T., & Arendt-Nielsen, L. (2014). Latent myofascial trigger points are associated with an increased intramuscular electromyographic activity during synergistic muscle activation. *Journal of Pain, 15*(2), 181-187. https://doi.org/10.1016/j.jpain.2013.10.009

Gerhardt, J., Cocchiarella, L., & Lea, R. (2002). *The practical guide to range of movement assessment*: American Medical Association: Chicago IL.

Gerwin, R. D. (2001). Classification, epidemiology, and natural history of myofascial pain syndrome. *Current Pain and Headache Reports, 5*(5), 412-420. https://doi.org/10.1007/s11916-001-0052-8

Gerwin, R. D. (2005). A review of myofascial pain and fibromyalgia- factors that promote their persistence. *Acupuncture in Medicine, 23*(3), 121-134. https://doi.org/10.1136/aim.23.3.121

Gerwin, R. D., Dommerholt, J., & Shah, J. P. (2004). An expansion of Simons' integrated hypothesis of trigger point formation. *Current Pain and Headache Reports, 8*(6), 468-475. https://doi.org/10.1007/s11916-004-0069-x

Graven-Nielsen, T., & Arendt-Nielsen, L. (2008). Sensory and motor manifestations of muscle pain. *Journal of Musculoskeletal Pain, 16*(1/2), 93-105. https://doi.org/10.1080/10582450801960420

Graven-Nielsen, T., & Arendt-Nielsen, L. (2010). Assessment of mechanisms in localized and widespread musculoskeletal pain. *Nature Reviews Rheumatology, 6*(10), 599-606. https://doi.org/10.1038/nrrheum.2010.107

Gunn, C. C. (2003). Intramuscular stimulation (IMS): the technique. *Pain*. Retrieved from http://www.istop.org

Hagg, G. M. (1991). Static work loads and occupational myalgia- A new explanation model. *Electromyographical Kinesiology, Elsevier Science Publishers (Biomedical Division)*, 141-144. Retrieved from www.researchgate.net

Hall, M., & Smith, J. C. (2018). The effects of an acute bout of foam rolling on hip range of motion on different tissues. *International Journal of Sports Physical Therapy, 13*(4), 652-660. https://doi.org/10.26603/ijspt20180652

Hapidou, E. G., O'Brien, M. A., Pierrynowski, M. R., de las Heras, E., Patel, M., & Patla, T. (2012). Fear and avoidance of movement in people with chronic pain: Psychometric properties of the 11-Item Tampa Scale for Kinesiophobia (TSK-11). *Physiotherapy Canada, 64*(3), 235-241. https://doi.org/10.3138/ptc.2011-10

Hardwick, D., Becker, M., Essick, K., Lalime, N., Staniulyte, R., & Haladay, D. (2019). Definition variability of chronic non-specific low back pain in physical therapy: A scoping review of the literature. *Orthopaedic Physical Therapy Practice, 31*(4), 199-206. Retrieved from https://health.ebsco.com/

Hartvigsen, J., Kongsted, A., Hancock, M. J., Louw, Q., Ferreira, M. L., Genevay, S., . . . Woolf, A. (2018). What low back pain is and why we need to pay attention. *The Lancet, 391*(10137), 2356-2367. https://doi.org/10.1016/S0140-6736(18)30480-X

Hayes, M., Cockrell, D., & Smith, D. (2009). A systematic review of musculoskeletal disorders among dental professionals. *International Journal of Dental Hygiene, 7*(3), 159-165. https://doi.org/10.1111/j.1601-5037.2009.00395.x

Hayes, M., Taylor, J., & Smith. (2012). Predictors of work-related musculoskeletal disorders among dental hygienists. *International Journal of Dental Hygiene, 10*(4), 265-269. https://doi.org/10.1111/j.1601-5037.2011.00536.x

Hemming, R., Sheeran, L., van Deursen, R., & Sparkes, V. (2019). Investigating differences in trunk muscle activity in non-specific chronic low back pain subgroups and no-low back pain controls during functional tasks: a case-control study. *BMC Musculoskeletal Disorders, 20*(1), 1-10. https://doi.org/10.1186/s12891-019-2843-2

Hidalgo-Lozano, A., Arroyo-Morales, M., Fernández-de-las-Peñas, C., Ge, H.-Y., Arendt-Nielsen, L., & Alonso-Blanco, C. (2010). Muscle trigger points and pressure pain hyperalgesia in the shoulder muscles in patients with unilateral shoulder impingement: A blinded, controlled study. *Experimental Brain Research, 202*(4), 915-925. https://doi.org/10.1007/s00221-010-2196-4

Hidalgo-Lozano, A., Fernández-de-las-Peñas, C., Calderón-Soto, C., Domingo-Camara, A., Madeleine, P., & Arroyo-Morales, M. (2013). Elite swimmers with and without unilateral shoulder pain: mechanical hyperalgesia and active/latent muscle trigger points in neck-shoulder muscles. *Scandinavian Journal of Medicine and Science in Sports, 23*(1), 66-73.

Hoffmann, J. P. (2021). *Linear Regression Models : Applications in R*. Retrieved from http://ebookcentral.proquest.com/lib/AUT/detail.action?docID=6682569

Hogg, J. A., Schmitz, R. J., Anh-Dung, N., & Shultz, S. J. (2018). Passive hip range-of-motion values across sex and sport. *Journal of Athletic Training (Allen Press), 53*(6), 560-567. https://doi.org/10.4085/1062-6050-426-16

Hoheisel, U., Koch, K., & Mense, S. (1994). Functional reorganization in the rat dorsal horn during an experimental myositis. *Pain, 59*(1), 111-118. https://doi.org/10.1016/0304-3959(94)90054-X

Hsieh, C.-Y., J. , Hong, C.-Z., Adams, A. H., Platt, K. J., Danielson, C. D., Hoehler, F. K., & Tobis, J. S. (2000). Interexaminer reliability of the palpation of trigger points in the trunk and lower limb muscles. *Archives of Physical Medicine and Rehabilitation, 81*(3), 258-264. https://doi.org/10.1053/apmr.2000.0810258

Hubbard, D. R., & Berkoff, G. M. (1993). Myofascial trigger points show spontaneous needle emg activity. *Spine, 18*(13), 1803-1807. https://doi.org/10.1097/00007632-199310000-00015

Hui, H., Masashi, N., Hitoshi, A., Jun, S., Hirofumi, N., Yohei, K., . . . Kazuo, K. (2019). Reproducibility, responsiveness and validation of the Tampa Scale for Kinesiophobia in patients with ACL injuries. *Health and Quality of Life Outcomes, 17*(1), 1-8. https://doi.org/10.1186/s12955-019-1217-7

Huijnen, I. P. J., Verbunt, J. A., Wittink, H. M., & Smeets, R. J. E. M. (2013). Physical performance measurement in chronic low back pain: measuring physical capacity or pain-related behaviour? *European Journal of Physiotherapy, 15*(3), 103-110. https://doi.org/10.3109/21679169.2013.830643

Hüllemann, P., Keller, T., Kabelitz, M., Gierthmühlen, J., Freynhagen, R., Tölle, T., . . . Baron, R. (2018). Clinical manifestation of acute, subacute, and chronic low back pain in different age groups: Low back pain in 35,446 patients. *Pain Practice, 18*(8), 1011-1023. https://doi.org/10.1111/papr.12704

Iglesias-González, J. J., Muñoz-García, M. T., Rodrigues-de-Souza, D. P., Alburquerque-Sendín, F., & Fernández-de-las-Peñas, C. (2013). Myofascial trigger points, pain, disability, and sleep quality in patients with chronic nonspecific low back pain. *Pain Medicine (United States), 14*(12), 1964-1970. https://doi.org/10.1111/pme.12224

Ireland, M. L., Willson, J. D., Ballantyne, B. T., & Davis, I. M. (2003). Hip strength in females with and without patellofemoral pain. *Journal of Orthopaedic and Sports Physical Therapy, 33*(11), 671-676. Retrieved from https://www-jospt-org.ezproxy.aut.ac.nz/

Jensen, M. P., Turner, J. A., & Romano, J. M. (1994). What is the maximum number of levels needed in pain intensity measurement? *Pain, 58*(3), 387-392. https://doi.org/10.1016/0304-3959(94)90133-3

Kadefors, R., Forsman, M., Zoega, B., & Herberts, P. (1999). Recruitment of low threshold motor-units in the trapezius muscle in different static arm positions. *42*(2), 359-375. Retrieved from https://www-tandfonline-com

Kalichman, L., & Vulfsons, S. (2010). Dry needling in the management of musculoskeletal pain. *Journal of American Board of Family Medicine, 23*(10), 640-646. https://doi.org/10.3122/jabfm.2010.05.090296

Kearns, G., Fernández-de-las-Peñas, C., Brismée, J. M., Gan, J., & Doidge, J. (2019). New perspectives on dry needling following a medical model: are we screening our patients sufficiently? *Journal of Manual and Manipulative Therapy, 27*(3), 172-179. https://doi.org/10.1080/10669817.2019.1567011

Kellgren, J. H. (1938a). Observations on referred pain arising from muscles. *Clinical Science, 3*(3), 175-190. Retrieved from http://www.biochemistry.org

Kellgren, J. H. (1938b). A preliminary account of referred pains arising from muscle. *British Medical Journal, 1*, 325-327. Retrieved from http://group.bmj.com

Kim, D., Unger, J., Lanovaz, J. L., & Oates, A. R. (2016). The relationship of anticipatory gluteus medius activity to pelvic and knee stability in the transition to single-leg stance. *American Academy of Physical Medicine and Rehabilitation, 8*(2), 138-144. https://doi.org/10.1016/j.pmrj.2015.06.005

Koes, B. W., van Tulder, M., Lin, C. W., Macedo, L. G., McAuley, J., Maher, C., . . . Maher, C. (2010). An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. *European Spine Journal, 19*(12), 2075-2094. https://doi.org/10.1007/s00586-010-1502-y

Kosek, E., Cohen, M., Baron, R., Gebhart, G. F., Mico, J.-A., Rice, A. S. C., . . . Sluka, A. K. (2016). Do we need a third mechanistic descriptor for chronic pain states? *Pain, 157*(7), 1382-1386. https://doi.org/10.1097/j.pain.0000000000000507

Krause, D. A., Hollman, J. H., Krych, A. J., Kalisvaart, M. M., & Levy, B. A. (2015). Reliability of hip internal rotation range of motion measurement using a digital inclinometer. *Knee Surgery, Sports Traumatology, Arthroscopy, 23*(9), 2562-2567. https://doi.org/10.1007/s00167-014-3096-0

Krause, D. A., Neuger, M. D., Lambert, K. A., Johnson, A. E., DeVinny, H. A., & Hollman, J. H. (2014). Effects of examiner strength on reliability of hip-Strength testing using a

handheld dynamometer. *Journal of Sport Rehabilitation, 23*(1), 56-64. https://doi.org/10.11223/JSR.2012-0070

Lalkate, S., Agrawal, R., & Agashe, G. K. (2020). A comparative study effectiveness of conventional physiotherapy versus yoga yherapy on pain, core muscle endurance, lumbar flexion range of motion and functional disability in patients with chronic mechanical low back pain. *Indian Journal of Physiotherapy & Occupational Therapy, 14*(4), 177-182. https://doi.org/10.37506/ijpot.v14i4.11322

Larkin, B., van Holsbeeck, M., Koueiter, D., & Zaltz, I. (2015). What is the impingement-free range of motion of the asymptomatic hip in young adult males? *Clinical Orthopaedics and Related Research, 473*(4), 1284-1288. https://doi.org/10.1007/s11999-014-4072-1

Last, A. R., & Hulbert, K. (2009). Chronic low back pain: Evaluation and management. *American Academy of Family Physicians, 79*(12), 1067-1074. Retrieved from https://www.aafp.org/journals.html

Leao Almeida, G. P., Das Neves Rodrigues, H. L., De Freitas, B. W., & De Paula Lima, P. O. (2017). Reliability and validity of the hip stability isometric test (HipSIT): A new method to assess hip posterolateral muscle strength. *Journal of Orthopaedic and Sports Physical Therapy, 47*(12), 906-913. https://doi.org/10.2519/jospt.2017.7274

Leetun, D. T., Ireland, M. L., Willson, J. D., Ballantyne, B. T., & Davis, I. M. (2004). Core Stability Measures as Risk Factors for Lower Extremity Injury in Athletes. *Medicine and Science in Sports and Exercise, 36*(6), 926-934. https://doi.org/10.1249/01.MSS.0000128145.75199.C3

Lew, P. C., Lewis, J., & Story, I. (1997). Inter-therapist reliability in locating latent myofascial trigger points using palpation. *Manual Therapy, 2*(2), 87-90. Retrieved from https://doi.org/10.1054/math.1997.0289

Lewit, K. (1979). The needle effect in the relief of myofascial pain. *Pain, 6*, 83-90. Retrieved from http://www.spinalmanipulation.org

Li, L., Stoop, R., Clijsen, R., Hohenauer, E., Fernández-de-las-Peñas, C., Huang, Q., & Barbero, M. (2020). Criteria used for the diagnosis of myofascial trigger points in clinical trials on physical therapy updated systematic review. *Clinical Journal of Pain, 36*(12), 955-967. https://doi.org/10.1097/AJP.0000000000000875

Liu, L., Skinner, M. A., Baxter, G. D., & McDonough, S. M. (2016). Traditional Chinese Medicine acupuncture and myofascial trigger needling: The same stimulation points? *Complementary Therapies in Medicine, 26*, 28-32. https://doi.org/10.1016/j.ctim.2016.02.013

Lu, G.-D., & Needham, J. (2002). *Celestial lancets : a history and rationale of acupuncture and moxa*. Retrieved from http://ebookcentral.proquest.com/lib/AUT/detail.action?docID=1075218

Lucas, K. R., Polus, B. I., & Rich, P. A. (2004). Latent myofascial trigger points: their effects on muscle activation and movement efficiency. *Journal of Bodywork and Movement Therapies, 8*(3), 160-166.

Lucas, K. R., Rich, P. A., & Polus, B. I. (2010). Muscle activation patterns in the scapular positioning muscles during loaded scapular plane elevation: The effects of latent myofascial trigger points. *Clinical Biomechanics, 25*(8), 765-770. https://doi.org/10.1016/j.clinbiomech.2010.05.006

Maher, C. G., Underwood, M., & Buchbinder, R. (2016). Non-specific low back pain. *The Lancet, 10*. https://doi.org/10.1016/S0140-673(16)30970-9

Manchikanti, L., Singh, V., Pampati, V., Damron, K. S., Barnhill, R. C., Beyer, C., & Cash, K. A. (2001). Evaluation of the relative contributions of various structures in chronic low back pain. *Pain Physician, 4*(4), 308-316. Retrieved from https://www.painphysicianjournal.com/

Manciopi, P. A. R., Rinaldi, N. M., & Moraes, R. (2017). Prehension combined with gait in individuals with chronic low back pain. *Motor Control, 21*(1), 90-111. https://doi.org/10.1123/mc.2014-0044

Matsumura, M., Ogawa, D., Ichikawa, K., Hata, M., Usa, H., Takei, H., . . . Furuya, H. (2015). Age and gender differences in lower extremity range of motion and muscle strength: A cross-sectional study of males and females, ages 20–70. *Rigakuryoho Kagaku, 30*(2), 239-246. https://doi.org/10.1589/rika.30.239

McCann, R. S., Terada, M., Kosik, K. B., & Gribble, P. A. (2019). Landing Kinematics and Isometric Hip Strength of Individuals With Chronic Ankle Instability. *Foot and Ankle International, 40*(8), 969-977. https://doi.org/10.1177/107119846085

McGuinness, H. (2010). *Anatomy & physiology : therapy basics* (Fourth edition. ed.). Retrieved from http://ebookcentral.proquest.com/lib/AUT/detail.action?docID=660509

Melzack, R., Stillwell, D. M., & Fox, E. J. (1977). Trigger points and acupuncture points for pain: Correlations and implications. *Pain, 3*(1), 3-23. https://doi.org/10.1016/0304-3959(77)90032-x

Mense, S. (2003). The pathogenesis of muscle pain. *Current Pain and Headache Reports, 7*(6), 419-425. https://doi.org/10.1007/s11916-003-0057-6

Mense, S. (2009). Algesic agents exciting muscle nociceptors. *Experimental Brain Research, 196*(1), 89-100. https://doi.org/10.1007/s00221-008-1674-4

Mense, S. (2010). How do muscle lesions such as latent and active trigger points influence central nociceptive neurons? *Journal of Musculoskeletal Pain, 18*, 348-353. https://doi.org/10.3109/10582452.2010.502621

Mense, S., & Gerwin, R. D. (2010). *Muscle pain: Diagnosis and treatment*. Retrieved from https://link-springer-com.ezproxy.aut.ac.nz/

Mense, S., & Hoheisel, U. (2004). Central Nervous Sequelae of Local Muscle Pain. *Journal of Musculoskeletal Pain, 12*(3/4), 101. https://doi.org/10.1300/J094v12n03_15

Meucci, R. D., Fassa, A. G., & Faria, N. M. X. (2015). Prevalence of chronic low back pain: systematic review. *Revista de Saúde Publica, 49*, 1-10. https://doi.org/10.1590/S0034-8910.2015049005874

Minerbi, A., & Vulfsons, S. (2018). Challenging the cinderella hypothesis: A new model for the role of the motor unit recruitment pattern in the pathogenesis of myofascial pain syndrome in postural muscles. *Rambam Maimonides Medical Journal*(3), e0021. https://doi.org/10.5041/RMMJ.10336

Ministry of Health NZ. (2020). *New Zealand Health Survey. Annual data explorer*. Retrieved 4/10/21, from www.health.govt.nz

Murray, E., Birley, E., Twycross-Lewis, R., & Morrissey, D. (2009). The relationship between hip rotation range of movement and low back pain prevalence in amateur golfers: An observational study. *Physical Therapy in Sport 10*(4), 131-135. https://doi.org/10.1016/j.ptsp.2009.08.002

Myburgh, C., Larsen, A. H., & Hartvigsen, J. (2008). A systematic, critical review of manual palpation for identifying myofascial trigger points: Evidence and clinical significance. *Archives of Physical Medicine and Rehabilitation, 89*(6), 1169-1176. https://doi.org/10.1016/j.apmr.2007.12.033

National Health Committee. (2015). Low back pain: A pathway to prioritisation. Retrieved from https://www.health.govt.nz/system/files/documents/publications/nhc-lbp-pathway-to-prioritisation.pdf

Neumann, D. A. (2010). Kinesiology of the hip: a focus on muscular actions. *Journal of Orthopaedic and Sports Physical Therapy, 40*(2), 82-94. https://doi.org/10.2519/jospt.2010.3025

Nijs, J., Lahousse, A., Kapreli, E., Bilika, P., Saraçoğlu, İ., Malfliet, A., . . . Huysmans, E. (2021). Nociplastic pain criteria or recognition of central sensitization? Pain phenotyping in the

past, present and future. *Journal of Clinical Medicine, 10*(15), 3203-3203. https://doi.org/10.3390/jcm10153203

Nijs, J., Van Houdenhove, B., & Oostendorp, R. A. B. (2010). Recognition of central sensitization in patients with musculoskeletal pain: Application of pain neurophysiology in manual therapy practice. *Manual Therapy, 15*(2), 135-141. https://doi.org/10.1016/j.math.2009.12.001

Njoo, K. H., & Van der Does, E. (1994). The occurence and inter-rater reliability of myofascial trigger points in the quadratus lumborum and gluteus medius: a prospective study in non-specific low back pain patients and controls in general practice. *Pain, 58*(3), 317-323. https://doi.org/10.1016/0304-3959(94)90125-2

Palacios-Cena, M., Ferracini, G. N., Florencio, L. L., Ruiz, M., Guerrero, A. L., Arendt-Nielsen, L., & Fernández-de-las-Peñas, C. (2017). The number of active but not latent trigger points associated with widespread pressure pain hypersensitivity in women with episodic migraines. *Pain Medicine, 18*(12), 2485-2491. https://doi.org/10.1093/pm/pnx130

Patton, K. T., & Thibodeau, G. A. (2010). *Anatomy and Physiology* (7th ed.). St Louis Missouri: Mosby Elsevier.

Piva, S. R., Goodnite, E. A., & Childs, J. D. (2005). Strength around the hip and flexibility of soft tissues in individuals with and without patellofemoral pain syndrome. *Journal of Orthopaedic and Sports Physical Therapy, 35*(12), 793-801. https://doi.org/10.2519/jospt.2005.35.12.793

Powers, C. M., Ghoddosi, N., Straub, R. K., & Khayambashi, K. (2017). Hip strength as a predictor of ankle sprains in male soccer players: A prospective study. *Journal of Athletic Training (Allen Press), 52*(11), 1048-1055. https://doi.org/10.4085/1062-6050-52.11.18

Pua, Y. H., Wrigley, T. W., Cowan, S. M., & Bennell, K. L. (2008). Intrarater test-retest reliability of hip range of motion and hip muscle strength measurements in persons with hip osteoarthritis. *Archives of Physical Medicine and Rehabilitation, 89*(6), 1146-1154. Retrieved from https://www.archives-pmr.org

Raja, S. N., Carr, D. B., Cohen, M., Finnerup, N. B., Flor, H., Gibson, S., . . . Vader, K. (2020). The revised International Association for the Study of Pain definition of pain: concepts, challenges, and compromises. *Pain, 161*(9), 1976-1982. https://doi.org/10.1097/j.pain.0000000000001939

Ramsook, R. R., & Malanga, G. A. (2012). Myofascial low back pain. *Current Pain and Headache Reports, 16*(5), 423-432. https://doi.org/10.1007/s11916-012-0290-y

Rathbone, A. T. L., Grosman-Rimon, L., & Kumbhare, D. A. (2017). Interrater agreement of manual palpation for identification of myofascial trigger points: A systematic review and meta-analysis. *Clinical Journal of Pain, 33*(8), 715. Retrieved from http://journals.lwww.com

Roach, K. E., & Miles, T. P. (1991). Normal hip and knee active range of motion: The relationship to age. *Physical Therapy, 71*(9), 656-665. https://doi.org/10.1093/ptj/71.9.656

Roach, S. M., San Juan, J. G., Suprak, D. N., & Lyda, M. (2013). Concurrent validity of digital inclinometer and universal goniometer in assessing passive hip mobility in healthy subjects. *International Journal of Sports Physical Therapy, 8*(5), 680-688. Retrieved from https://ifspt.org/

Roach, S. M., San Juan, J. G., Suprak, D. N., Lyda, M., Bies, A. J., & Boydston, C. R. (2015). Passive hip range of motion is reduced in active subjects with chronic low back pain compared to controls. *International Journal of Sports Physical Therapy, 10*(1), 13-20. Retrieved from https://ifspt.org

Roach, S. M., San Juan, J. G., Suprak, D. N., Lyda, M., & Boydston, C. (2014). Patellofemoral pain subjects exhibit decreased passive hip range of motion compared to controls. *International Journal of Sports Physical Therapy, 9*(4), 468-475. Retrieved from https://ifspt.org

Roach, S. M., Sorenson, E., Headley, B., & San Juan, J. G. (2013). Prevalence of myofascial trigger points in the hip in patellofemoral pain. *Archives of Physical Medicine and Rehabilitation, 94*(3), 522-526. https://doi.org/10.1016/j.apmr.2012.10.022

Rose-Dulcina, K., Vuillerme, N., Tabard-Fougère, A., Dayer, R., Dominguez, D. E., Armand, S., & Genevay, S. (2018). Identifying subgroups of patients with chronic nonspecific low back pain based on a multifactorial approach: Protocol for a prospective study. *Journal of Medical Internet Research, 7*(4), e104. https://doi.org/10.2196/resprot.9224

Rozenfeld, E., Finestone, A. S., Moran, U., Damri, E., & Kalichman, L. (2017). Test-retest reliability of myofascial trigger point detection in hip and thigh areas. *Journal of Bodywork and Movement Therapies, 21*(4), 914-919. https://doi.org/10.1016/j.jbmt.2017.03.023

Sadler, S., Cassidy, S., Peterson, B., Spink, M., & Chuter, V. (2019). Gluteus medius muscle function in people with and without low back pain: a systematic review. *BioMed Central Musculoskeletal Disorders, 20*(1-17), 1. https://doi.org/10.1186/s12891-019-2833-4

Samuel, A. S., Peter, A. A., & Ramanathan, K. (2007). The association of active trigger points with lumbar disc lesions. *Journal of Musculoskeletal Pain, 15*(2), 11-18. https://doi.org/10.1300/J094v15n02_04

Santos, F. G., Carmo, C. M., Fracini, A. C., Pereira, R. R. P., Takara, K. S., & Tanaka, C. (2013). Chronic low back pain in women: Muscle activation during task performance. *Journal of Physical Therapy Science, 25*(12), 1569-1573. Retrieved from https://www.jstage.jst.go.jp/

Saxena, A., Chansoria, M., Tomar, G., & Kumar, A. (2015). Myofascial pain syndrome: An overview. *Journal of Pain & Palliative Care Pharmacotherapy, 29*(1), 16-21. https://doi.org/10.3109/15360288.2014.997853

Schäfer, A., Hall, T., & Briffa, K. (2009). Classification of low back-related leg pain-A proposed patho-mechanism-based approach. *Manual Therapy, 14*(2), 222-230. https://doi.org/10.1016/j.math.2007.10.003

Shah, J. P., Danoff, J. V., Desai, M. J., Parikh, S., Nakamura, L. Y., Phillips, T. M., & Gerber, L. H. (2008). Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. *Archives of Physical Medicine and Rehabilitation, 89*(1), 16-23. https://doi.org/10.1016/j.apmr.2007.10.1018

Shah, J. P., & Gilliams, E. A. (2008). Uncovering the biochemical milieu of myofascial trigger points using in vivo microdialysis: An application of muscle pain concepts to myofascial pain syndrome. *Journal of Bodywork and Movement Therapies, 12*(4), 371-384. https://doi.org/10.1016/j.jbmt.2008.06.006

Shah, J. P., Phillips, T. M., Danoff, J. V., & Gerber, L. H. (2005). An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *Journal of Applied Physiology, 99*, 1977-1984. https://doi.org/10.1152/japplphysio.00419.2005

Shah, J. P., Thaker, N., Heimur, J., Aredo, J. V., Sikdar, S., & Gerber, L. H. (2015). Myofascial trigger points then and now: A historical and scientific perspective. *Journal of Injury, Function & Rehabilitation, 7*(7), 746-761. https://doi.org/10.1016/j.pmrj.2015.01.024

Shemshaki, H., Amin Nourian, S.-M., Fereidan-Esfahani, M., Mokhtari, M., & Reza Etemadifar, M. (2013). What is the source of low back pain? *Journal of Craniovertebral Junction and Spine, 4*(5), 21-24. https://doi.org/10.4103/0974-8237.121620

Shraim, M. A., Massé-Alarie, H., Hall, L. M., & Hodges, P. W. (2020). Systematic review and synthesis of mechanism-based classification systems for pain experienced in the musculoskeletal system. *The Clinical journal of pain, 36*(10), 793-812. https://doi.org/10.1097/AJP.0000000000000860

Shutterstock. (2021). Retrieved from http://www.shutterstock.com

Sikdar, S., Gerber, L. H., Shah, J. P., Gebreab, T., Yen, R. H., Gilliams, E., & Danoff, J. (2009). Novel applications of ultrasound technology to visualize and characterise myofascial trigger points and surrounding soft tissue. *Archives of Physical Medicine and Rehabilitation, 90*(11), 1829-1838. https://doi.org/10.1016/j.apmr.2009.04.015

Simons, D. G. (1987). Myofascial pain syndrome due to trigger points. *International Rehabilitation Medicine*. Retrieved from http://www.tandfonline.com

Simons, D. G. (1996). Clinical and etiological update of myofascial pain from trigger points. *Journal of Musculoskeletal Pain, 4*(1-2), 93-122. https://doi.org/10.1300/J094v04n01_07

Simons, D. G. (2001). Myofascial Pain Syndromes- Trigger Points. *Journal of Musculoskeletal Pain, 9*(3), 85. Retrieved from https://www.tandfonline.com/

Simons, D. G. (2004a). New aspects of myofascial trigger points etiological and clinical. *Journal of Musculoskeletal Pain, 12*(3/4), 15. Retrieved from https://www.tandfonline.com/

Simons, D. G. (2004b). Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. *Journal of Electromyography and Kinesiology, 14*(1), 95-107. https://doi.org/10.1016/j.jelekin.2003.09.018

Simons, D. G., & Travell, J. G. (1981). Myofascial trigger points, a possible explanation. *Pain, 10*(1), 106-109. Retrieved from https://www-sciencedirect-com

Simons, D. G., Travell, J. G., & Simons, L. S. (1999). *Myofascial pain and dysfunction: The trigger point manual. Volume 1. Upper half of body* (Second ed.). Retrieved from http://ezproxy.aut.ac.nz/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cat05020a&AN=aut.b10551669&site=eds-live

Slater, H., Arendt-Nielsen, L., Graven-Nielsen, T., & Wright, A. (2005). Sensory and motor effects of experimental muscle pain in patients with lateral epicondylalgia and controls with delayed onset muscle soreness. *Pain, 114*(1-2), 118-130. https://doi.org/10.1016/j.pain.2004.12.003

Sluka, K. A. (2016). *Mechanisms and management of pain for the physical therapist* (Second edition. ed.). Retrieved from http://ebookcentral.proquest.com/lib/AUT/detail.action?docID=4850655

Sluka, K. A., & George, S. Z. (2021). A new definition of pain: Update and implications for physical therapist practice and rehabilitation science. *Physical Therapy, 101*(4), 1-3. https://doi.org/10.1093/ptj/pzab019

Smeets, R., Köke, A., Lin, C. W., Ferreira, M., & Demoulin, C. (2011). Measures of function in low back pain/disorders: Low Back Pain Rating Scale (LBPRS), Oswestry Disability

Index (ODI), Progressive Isoinertial Lifting Evaluation (PILE), Quebec Back Pain Disability Scale (QBPDS), and Roland-Morris Disability Questionnaire (RDQ). *Arthritis Care and Research, 63*(11), 158-173. https://doi.org/10.1002/acr.20542

Sperry, M. A., & Goshgarian, H. G. (1993). Ultrastructural Changes in the Rat Phrenic Nucleus Developing within 2 h after Cervical Spinal Cord Hemisection. *Experimental Neurology, 120*(2), 233-244. https://doi.org/10.1006/exnr.1993.1058

Srbely, J. Z., Kumbhare, D., & Grosman-Rimon, L. (2016). A narrative review of new trends in the diagnosis of myofascial trigger points: diagnostic ultrasound imaging and biomarkers. *Journal of the Canadian Chiropractic Association, 60*(3), 220-225. Retrieved from https://www.chiropractic.ca/jcca-online/

Stecco, A., Gesi, M., Stecco, C., & Stern, R. (2013). Fascial components of the myofascial pain syndrome topical collection on myofascial pain. *Current Pain and Headache Reports, 17*(8). https://doi.org/10.1007/s11916-013-0352-9

Steultjens, M. P. M., Dekker, J., van Baar, M. E., Oostendorp, R. A. B., & Bijlsma, J. W. J. (2000). Range of joint motion and disability in patients with osteoarthritis of the knee or hip. *Rheumatology, 39*, 955-961. https://doi.org/10.1093/rheumatology/39.9.955

Stockman, R. (1904). The cause, pathology, and treatment of chronic rheumatism. *15*, 107-116. Retrieved from http://www.ncbi.nlm.nih.gov

Tagliaferri, S. D., Miller, C. T., Owen, P. J., Mitchell, U. H., Brisby, H., Fitzgibbon, B., . . . Belavy, D. L. (2020). Domains of chronic low back pain and assessing treatment effectiveness: A clinical perspective. *Pain Practice, 20*(2), 211-225. https://doi.org/10.1111/papr.12846

Takashi, N., Abt, J. P., Sell, T. C., Keenan, K. A., Clark, N. C., Smalley, B. W., . . . Lephart, S. M. (2015). Lumbar spine and hip flexibility and trunk strength in helicopter pilots with and without low back pain history. *Work, 52*(3), 715-722. https://doi.org/10.3233/WOR-152192

Thomas, E., Silman Alan, J., Croft Peter, R., Papageorgiou Ann, C., Jayson Malcolm, I. V., & MacFarlane Gary, J. (1999). Predicting who develops chronic low back pain in primary care: A prospective study. *British Medical Journal, 318*(7199), 1662-1667. Retrieved from https://www.jstor.org/stable/25184988

Thomas, J. S., France, C. R., Sha, D., & Wiele, N. V. (2008). The influence of pain-related fear on peak muscle activity and force generation during maximal isometric trunk exertions. *Spine, 33*(11), E342-E348. https://doi.org/10.1097/BRS.0b013e3181719264

Thorborg, K., Bandholm, T., Schick, M., Jensen, J., & Hölmich, P. (2013). Hip strength assessment using handheld dynamometry is subject to intertester bias when testers are

of different sex and strength. *Scandinavian Journal of Medicine and Science in Sports, 23*(4), 487-493. https://doi.org/10.1111/j.1600-0838.2011.01405.x

Tobias, M. (2013). Health loss in New Zealand : a report from the New Zealand burden of diseases, injuries and risk factors study, 2006-2016. Retrieved from https://www.health.govt.nz/

Tortora, G. J., & Derrickson, B. (2012). *Introduction to the human body : the essentials of anatomy and physiology* (Ninth ed.) [Non-fiction]: John Wiley & Sons.

Travell, J. G., & Rinzler, S. H. (1952). The myofascial genesis of pain. *Postgraduate Medicine, 11*(5), 425-434. https://doi.org/10.1080/00325481.1952.11694280

Travell, J. G., & Simons, D. G. (1983). *Myofascial pain and dysfuntion. The trigger point manual. Volume 1. Upper half of body* Williams and Wilkins: Maryland.

Travell, J. G., & Simons, D. G. (1992). *Myofascial pain and dysfunction. The trigger point manual. Volume 2. The lower extremities*: Baltimore, Md. : Williams & Wilkins, 1992.

Tumilty, S., Adhia, D. B., Rhodes, R., & Mani, R. (2017). Physiotherapists' treatment techniques in New Zealand for management of acute nonspecific low back pain and its relationships with treatment outcomes: a pilot study. *Physical Therapy Reviews, 22*(1-2), 95-100. https://doi.org/10.1080/10833196.2017.1282073

Vad, V. B., Gebeh, A., Dines, D., Altchek, D., & Norris, B. (2003). Hip and shoulder internal rotation range of motion deficits in professional tennis players. *Journal of Science and Medicine in Sport, 6*(1), 71-75. Retrieved from https://www.jsams.org/

Van Dillen, L. R., Bloom, N. J., Gombatto, S. P., & Susco, T. M. (2008). Hip rotation range of motion in people with and without low back pain who participate in rotation-related sports. *Physical Therapy in Sport 9*(2), 72-81. https://doi.org/10.1016/j.ptsp.2008.01.002

Verbunt, J. A., Seelen, H. A., Vlaeyen, J. W., Bousema, E. J., van der Heijden, G., Heuts, P. H., & Knottnerus, J. A. (2005). Pain-related factors contributing to muscle inhibition in patients with chronic low back pain - An experimental investigation based on superimposed electrical stimulation. *The Clinical Journal of Pain, 21*(3), 232-240. Retrieved from http://journals.lwww.com

Vos, T., Allen, C., Arora, M., Barber, R. M., Bhutta, Z. A., Brown, A., . . . Dandona, L. (2015). Global, regional, and national incidence, prevalence, and years lived with disability for 310 acute and chronic diseases and injuries, 1990-2015: A systematic analysis for the global burden of disease study 2015. https://doi.org/10.1016/S0140-6736(16)31678-6

Wada, J. T., Akamatsu, F. E., Hojaij, F., Itezerote, A., Scarpa, J. C., Andrade, M., & Jacomo, A. L. (2020). An anatomical basis for the myofascial trigger points of the abductor hallucis muscle. *BioMed Research International, 2020*. https://doi.org/10.1155/2020/9240581

Wall, P. D., & Woolf, C. J. (1984). Muscle but not cutaneous C-afferent input produces prolonged increases in the excitability of the flexion reflex in the rat. *Journal of Physiology, 356*, 443-458. https://doi.org/10.1113/jphysiol.1984.sp015475

Walsh, R., Kinsella, S., & McEvoy, J. (2017). The intra-rater reliability of locating and measuring the severity of latent trigger points in the quadriceps. *Journal of Bodywork and Movement Therapies, 21*(4), 926-932. https://doi.org/10.1016/j.jbmt.2016.11.015

Wand, B. M., & O'Connell, N. E. (2008). Chronic non-specific low back pain - sub-groups or a single mechanism? *BMC Musculoskeletal Disorders, 9*, 11. https://doi.org/10.1186/1471-2474-9-11

Welsh, P., Howitt, S., & Howarth, S. J. (2020). The influence of hip joint angle on the ratio between adduction and abduction torque in experienced, recreational male ice hockey players. *International Journal of Sports Physical Therapy, 15*(1), 22-33. https://doi.org/10.26603/ijspt20200022

Willard, F. (2008). *Basic mechanisms of pain*: Humana Press.

Willson, J. D., & Davis, I. S. (2009). Lower extremity strength and mechanics during jumping in women with patellofemoral pain. *Journal of Sport Rehabilitation, 18*(1), 76-90. Retrieved from https://journals-humankinetics-com.ezproxy.aut.ac.nz/journal/jsr

Woby, S. R., Urmston, M., Roach, N. K., & Watson, P. J. (2005). Psychometric properties of the TSK-11: A shortened version of the Tampa Scale for Kinesiophobia. *Pain, 117*(1-2), 137-144. https://doi.org/10.1016/j.pain.2005.05.029

Wytrazek, M., Huber, J., & Lisinski, P. (2011). Changes in muscle activity determine progression of clinical symptoms in patients with chronic spine-related muscle pain. A complex clinical and neurophysiological approach. *Functional Neurology, 26*(3), 141-149. Retrieved from http://www.mondino.it/functional-neurology/

Zarei, H., Bervis, S., Piroozi, S., & Motealleh, A. (2020). Added value of gluteus medius and quadratus lumborum dry needling in improving knee pain and function in female athletes with patellofemoral pain syndrome: A randomized clinical trial. *Archives of Physical Medicine and Rehabilitation, 101*(2), 265-274. https://doi.org/10.1016/j.apmr.2019.07.009

Zhang, C. (2021). *Hip surgery : a practical guide*. Retrieved from https://ezproxy.aut.ac.nz/login?url=https://link.springer.com/10.1007/978-981-15-9331-4

# Appendices

Appendix A. Ethics approval letter from AUTEC

**Auckland University of Technology Ethics Committee (AUTEC)**

Auckland University of Technology
D-88, Private Bag 92006, Auckland 1142, NZ
T: +64 9 921 9999 ext. 8316
E: ethics@aut.ac.nz
www.aut.ac.nz/researchethics

25 September 2019

Richard Ellis
Faculty of Health and Environmental Sciences

Dear Richard

Re Ethics Application: **19/216 The effects of gluteus medius trigger points on hip passive range of movement and muscle strength in people with chronic non-specific low-back pain**

Thank you for providing evidence as requested, which satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC).

Your ethics application has been approved for three years until 24 September 2022.

**Non-Standard Conditions of Approval**

1. Include the AUT masthead on the Information Sheet.
2. Data and Consent Forms must be stored by the application on AUT premises. Advise participant of this in the Information Sheet.

Non-standard conditions must be completed before commencing your study. Non-standard conditions do not need to be submitted to or reviewed by AUTEC before commencing your study.

**Standard Conditions of Approval**

1. The research is to be undertaken in accordance with the Auckland University of Technology Code of Conduct for Research and as approved by AUTEC in this application.
2. A progress report is due annually on the anniversary of the approval date, using the EA2 form.
3. A final report is due at the expiration of the approval period, or, upon completion of project, using the EA3 form.
4. Any amendments to the project must be approved by AUTEC prior to being implemented. Amendments can be requested using the EA2 form.
5. Any serious or unexpected adverse events must be reported to AUTEC Secretariat as a matter of priority.
6. Any unforeseen events that might affect continued ethical acceptability of the project should also be reported to the AUTEC Secretariat as a matter of priority.
7. It is your responsibility to ensure that the spelling and grammar of documents being provided to participants or external organisations is of a high standard.

AUTEC grants ethical approval only. You are responsible for obtaining management approval for access for your research from any institution or organisation at which your research is being conducted. When the research is undertaken outside New Zealand, you need to meet all ethical, legal, and locality obligations or requirements for those jurisdictions.

Please quote the application number and title on all future correspondence related to this project.

For any enquiries please contact ethics@aut.ac.nz. The forms mentioned above are available online through http://www.aut.ac.nz/research/researchethics

Yours sincerely,

Kate O'Connor
Executive Manager
**Auckland University of Technology Ethics Committee**

Cc: mariannecarroll.w@gmail.com; Susan Kohut; nick.garrett@aut.ac.nz

Appendix B. Participant information sheet

AUT
TE WĀNANGA ARONUI O TĀMAKI MAKAU RAU

# Participant Information Sheet

This information sheet is for potential participants in the below mentioned research study.

**Date Information Sheet Produced:**

25th September 2019

**Project Title**

The effects of gluteus medius trigger points on hip passive range of movement and muscle strength in people with chronic non-specific low-back pain

An Invitation

I am Marianne Carroll, a physiotherapist in Te Anau. I am inviting you to participate in my research study which I am completing for my Masters degree. No conflicts of interest have been identified in relation to your participation in this research study.

**What is the purpose of this research?**

The incidence of experiencing back pain in New Zealand during a lifetime is high. Chronic low back pain (pain lasting longer than three months) of a non-specific cause is a significant contributor to pain and disability globally. Muscle "knots/ sensitive spots" (known as trigger points) in the gluteus medius muscles (one of the buttock muscles) are a common clinical finding in people with chronic non-specific low back pain. Other clinical findings are decreased hip movement and muscle strength. Trigger points can be a source or contributor to low back pain; trigger points cause pain and altered movement patterns.

This research aims to investigate the role of gluteus medius trigger points in people with chronic non-specific low back pain and establish if there is a relationship between them and decreased hip passive range of movement and muscle strength. Clinically, if a relationship is established, treatment of the trigger point may be an effective treatment for deficits in muscle strength and range of movement in this patient population. The findings of this research may be used for academic publications and presentations.

**How was I identified and why am I being invited to participate in this research?**

People who are experiencing low back pain for longer than three months are invited to participate in this research study. You are receiving this information sheet to learn more about the study for you to be able to give informed consent should you wish to partake.

Potential participants will be unable to partake in this research study if they possess any of the following exclusion criteria: pregnant, have a diagnosed specific pathology causing their low back pain (for example: cauda equina syndrome, fracture, tumour, infection, herniated disc, inflammatory disorder); have undergone spinal surgery; have current knee pain; hip pain; a medical diagnosis of hip or knee osteoarthritis; have neurological symptoms (pins and needles or numbness) in their lower limbs; or a diagnosis of fibromyalgia.

**How do I agree to participate in this research?**

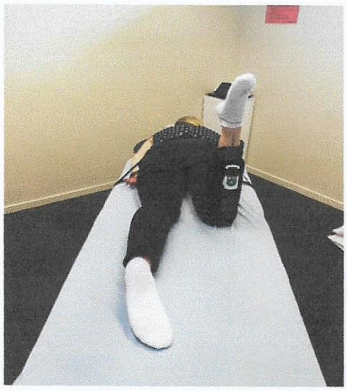
To take part in this research, you should contact the Primary Researcher: Marianne Carroll, mariannecarroll.w@gmail.com, 021 08717823. You will then be given a consent form to complete prior to participation in the study.

Your participation in this research is voluntary (it is your choice) and whether or not you choose to participate neither advantages nor disadvantages you. You are able to withdraw from the study at any time. If you choose to withdraw from the study, then you will be offered the choice between having any data that is identifiable as belonging to you removed or allowing it to continue to be used. However, once the findings have been produced, removal of your data may not be possible. You can also choose to bring along a support person or whanau member should you wish.

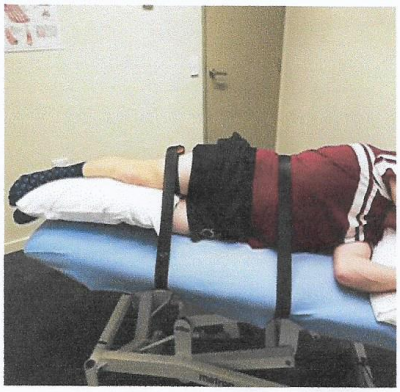
**What will happen in this research?**

As a participant in this research study, you will attend a one-off assessment session. At this assessment session, you will complete three questionnaires. These questionnaires will ask you about your pain, level of function, and your thoughts about movement in relation to your low-back pain. You will then have an assessment of your hip movement and muscle strength; then palpation of your gluteus medius muscles which are along the top of the

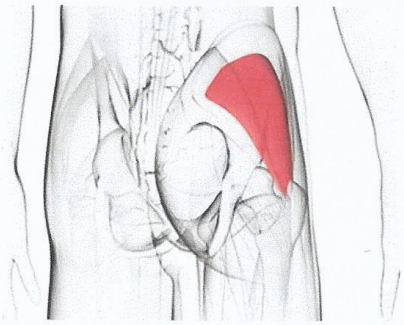
buttocks. Hip movement and muscle strength testing will be carried out in lying, on a physiotherapy treatment bed. Three movements of the hip will be tested. Separate pieces of equipment will be used to take movement and strength measures. The final part of the assessment will be carried out by a second assessor who is also a physiotherapist. They will feel/ palpate the gluteus medius muscle while you are in side lying, they will palpate right and left sides to find any trigger points (tight knots) you may have through exposed skin. All information that is collected will be used solely for this research study. You will receive a $20 groceries voucher to thank you for your time.



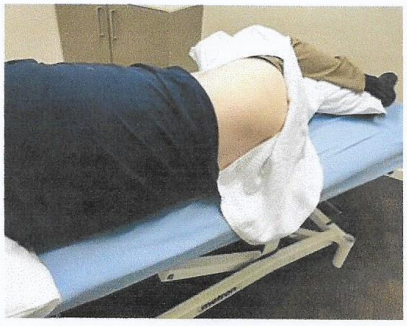
Example of position for range of movement testing



Example of positioning for muscle strength testing



Anatomical location of gluteus medius muscle (Verywellhealth.com, 2019)



Example of positioning for palpation of gluteus medius and identification of area to be palpated

**What are the discomforts and risks?**

There is a very low risk that you may experience discomfort or an increase in your low back pain on hip testing and palpation of your gluteus medius muscle.

Appropriate draping will be used during palpation of gluteus medius (see photo above) to ensure modesty and decrease risk of embarrassment.

**How will these discomforts and risks be alleviated?**

A handout is available should you experience any discomfort or pain following the assessment session. This handout will give you information on how to manage this discomfort/ pain.

**What are the benefits?**

I am completing this research study in order to obtain a Masters Degree from Auckland University of Technology (AUT). The results of this research study will contribute to the existing literature regarding assessment and treatment of chronic non-specific low back pain. This is especially important, as the occurrence of low-backpain is projected to increase, especially in low- and middle-income societies.

You will also receive a voucher (koha) as a thanks for your time. This will be a $20 voucher for Freshchoice grocery store. You will also be able to receive a copy of your hip movement, muscle strength, and trigger point status should you wish.

Participation in this research will help you, the locality and the New Zealand people. This research will contribute to low-back pain assessment and treatment.

**How will my privacy be protected?**

All data collected will be solely used for the purpose of the study. Information will be stored in a locked cabinet at Fiordland Physiotherapy clinic which will only be accessible to the primary researcher. Besides the initial collection of personal details relating to: name, age, cultural background, and occupation; all participants will be assigned a participant number which will be used on all paperwork; therefore, data collection sheets will be anonymised.

**What are the costs of participating in this research?**

There is no financial cost to the participant in relation to participation in the research study. You will be required to attend one assessment session which will take 30- 40 minutes.

**What opportunity do I have to consider this invitation?**

From initial contact with the primary researcher and receipt of the Information Sheet, you have one week to consider if you want to partake in the research study. If the primary researcher has not heard from you within one week, she will carry out a follow-up phone call with you.

**Will I receive feedback on the results of this research?**

A summary of the findings of the research study can be supplied to you by email or post on completion of the study.

**What do I do if I have concerns about this research?.**

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, Richard Ellis, richard.ellis@aut.ac.nz, +64 9 921 9999 ext 7612.

Concerns regarding the conduct of the research should be notified to the Executive Secretary of AUTEC, Kate O'Connor, *ethics@aut.ac.nz*, 921 9999 ext 6038.

**Funding disclosure**

Some funding has been received from The Southland Medical Trust, funding will also be received from AUT. Any outstanding costs will be covered by the primary researcher Marianne Carroll.

**Whom do I contact for further information about this research?**

Please keep this Information Sheet and a copy of the Consent Form for your future reference. You are also able to contact the research team as follows:

***Researcher Contact Details:***

Marianne Carroll. Email: mariannecarroll.w@gmail.com. Contact number: 021 08717823.

***Project Supervisor Contact Details:***

Richard Ellis. Email: Richard.ellis@aut.ac.nz. Contact number: 09 921 9999 ext 7612.

**Approved by the Auckland University of Technology Ethics Committee on *25th September 2019*, AUTEC Reference number *19/216*.**

Appendix C. Consent form

AUT
TE WĀNANGA ARONUI O TĀMAKI MAKAU RAU

# Consent Form

*Project title:* ***The effects of gluteus medius trigger points on hip passive range of movement and muscle strength in people with chronic non-specific low-back pain***

*Project Supervisor:* ***Richard Ellis Senior Lecturer and Associate Head of Research, AUT***

*Researcher:* ***Marianne Carroll***

- I have read and understood the information provided about this research project in the Information Sheet dated 25th September 2019.
- I have had an opportunity to ask questions and to have them answered.
- I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time without being disadvantaged in any way.
- I understand that if I withdraw from the study then I will be offered the choice between having any data that is identifiable as belonging to me removed or allowing it to continue to be used. However, once the findings have been produced, removal of my data may not be possible.
- I am not pregnant, have a diagnosed specific pathology causing my low back pain, have not undergone spinal surgery; I don't have current knee or hip pain; or a medical diagnosis of hip or knee osteoarthritis; I don't have neurological symptoms in my lower limbs and I don't have a diagnosis of fibromyalgia.
- I agree to attend an assessment at which my hip movement, muscles strength will be tested, followed by palpation of my gluteus medius muscles.
- I agree to take part in this research.
- I wish to receive a summary of the research findings (please tick one): YesO NoO

Participant's signature: .....

Participant's name: .....

Participant's Contact Details (if appropriate):

.....

.....

.....

.....

Date:

***Approved by the Auckland University of Technology Ethics Committee on 25th September 2019 AUTEC Reference number 19/216***

Appendix D. Personal details form

AUT UNIVERSITY NEW ZEALAND

**Personal Details Form**

**Name:**

**Home address:**

**Contact phone number:**

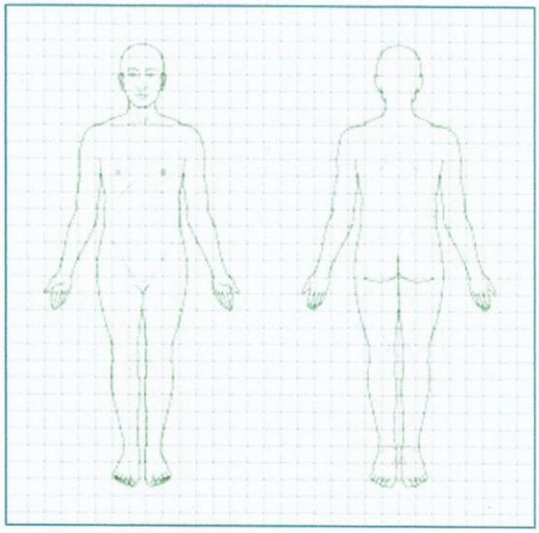
**Date of birth:**

**Sex:**

**Ethnic background:**

**Occupation:**

**Where is your low-back pain?**



Appendix E. Passive range of movement assessment recording sheet

**Passive range of movement assessment**

**Adduction range**

Right: _____ _____ _____ Average: _____

**External rotation**

Left: _____ _____ _____ Average: _____

**Internal rotation**

Left: _____ _____ _____ Average: _____

**External rotation**

Right: _____ _____ _____ Average: _____

**Internal rotation**

Right: _____ _____ _____ Average: _____

**Adduction range**

Left: _____ _____ _____ Average: _____

**Comments:**

Appendix F. Hip passive range of movement assessment protocol

**Hip Passive Range of Movement Assessment Protocol**

All movements are held for three seconds at end feel, completed three times and the average is used in the data collection. To familiarize the participant with the movements of rotation and ensure they are pain-free, the movements are completed once in each direction on each side.

Sequence of testing: Right adduction, left ER and IR, right ER and IR; then left adduction. To ensure blinding of the investigator and the influence this would have on subsequent measurements; the screen of the digital inclinometer is covered. The device beeps when zeroed, the button is pressed again to store a measurement; it also beeps when this is done. The measurements are stored on the screen of the digital inclinometer. After the three movements have been carried out, the screen is uncovered, and the three measurements are recorded on the assessment sheet.

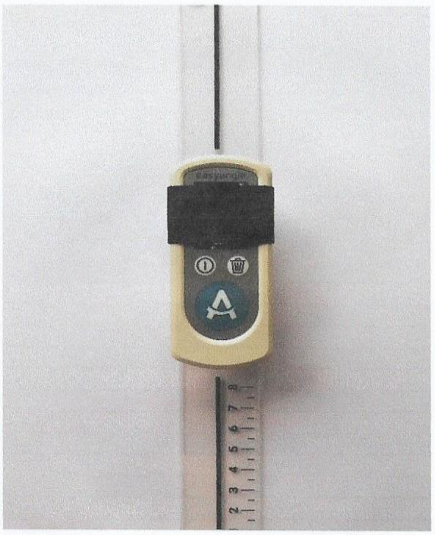


*Figure 1. Digital Inclinometer with screen covered*

**Adduction**

Participant is in side-lying, top leg is flexed to 90 degrees at the hip and knee. The top knee is supported on pillows to maintain neutral position and ensure no adduction of the top hip. The digital inclinometer is placed on the posterior aspect of the thigh, midway; in line with the femur. The bottom leg is then adducted until end range is felt.

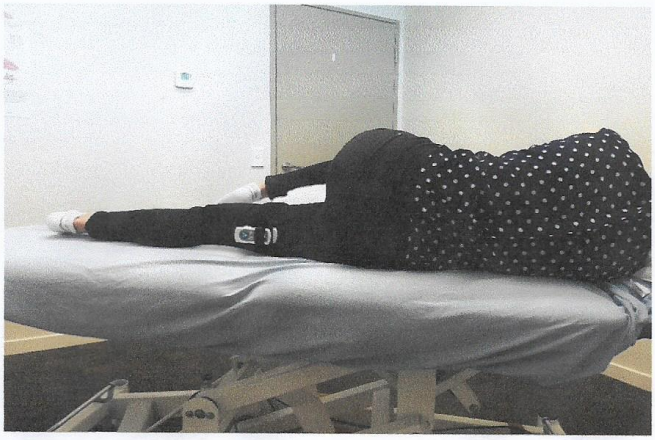


*Figure 2. Adduction PROM testing*

**External rotation**

Participant is in prone lying, stabilisation belt around the pelvis. The digital inclinometer is placed on the anterior aspect of the tibia, 9.5cm from the tibial tuberosity. The knee is passively flexed to 90 degrees (Fig 3.). The femur (and subsequently the hip joint) is rotated externally/outwards (Fig. 4). If the participant has a lot of external rotation range, the non-tested leg is abducted to provide more room. This is repeated on the other side.

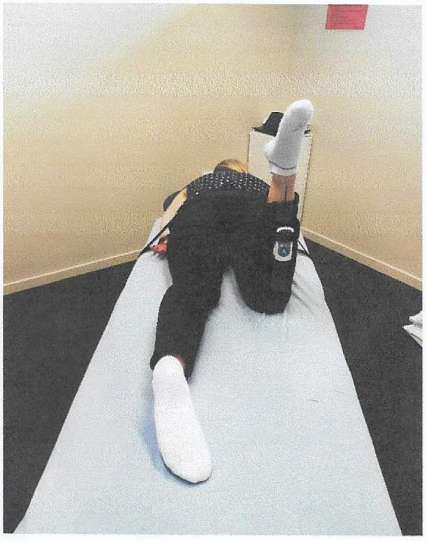


*Figure 3. Starting position for external and internal rotation*

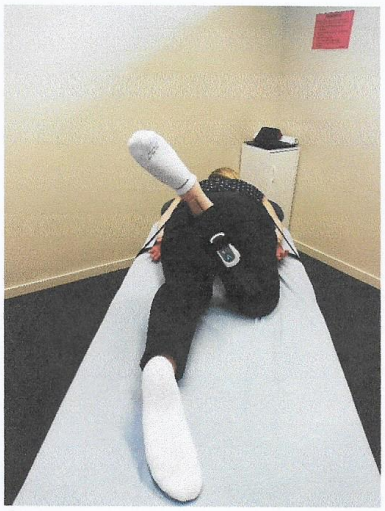


*Figure 4. External rotation PROM testing*

**Internal rotation**

Same participant and digital inclinometer positioning as for external rotation. The knee is again passively flexed to 90 degrees. The femur (and subsequently the hip joint) is rotated internally/inwards (Fig. 5). This is repeated on the other side.

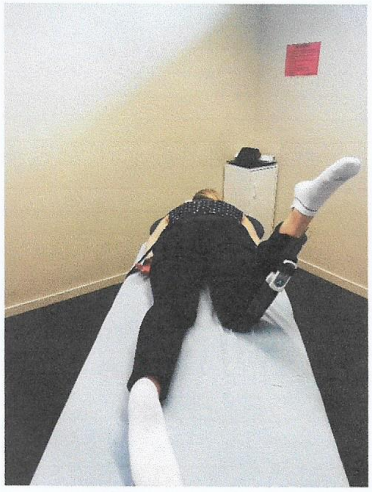


*Figure 5. Internal rotation PROM testing*

Appendix G. Hip muscle strength testing recording sheet

**Hip Muscle Strength Testing Recording Sheet**

Weight: kg

Dominant side: **R** **L** (please circle)

Hip Muscle Strength

Right abduction: _____ _____ _____ Peak value:

Left internal rotation: _____ _____ _____ Peak value:

Right external rotation: _____ _____ _____ Peak value:

Left external rotation: _____ _____ _____ Peak value:

Right internal rotation: _____ _____ _____ Peak value:

Left abduction: _____ _____ _____ Peak value:

Comments:

Appendix H. Hip muscle strength testing protocol

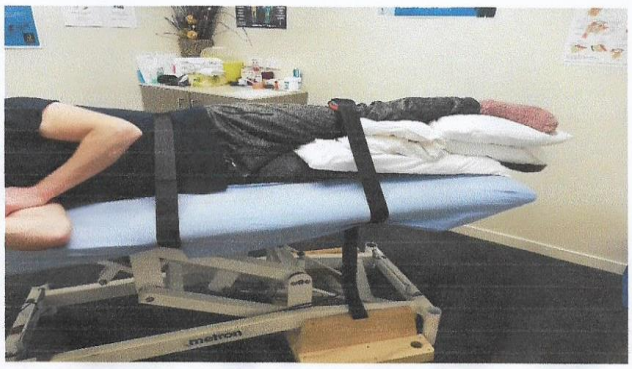
**Hip Muscles Strength Testing Protocol**

Abduction

The participant is in side-lying, the top leg being tested. The top hip is in 10 degrees abduction with the position being maintained by pillows/ towels. Both legs are straight and a stabilisation belt is used proximal to the iliac crest to stabilise the trunk and one around the dynamometer to provide resistance (the belts encircle the plinth). The dynamometer is placed five centimetres above the lateral femoral condyle of the top leg.

**Figure 1**

Hip abduction testing position



External and Internal Rotation

The participants are in prone lying on the plinth. A stabilisation belt is used around the pelvis and around the thighs (to maintain hips in neutral). A third stabilisation belt is used around the dynamometer and pole. The custom-made pole holder is placed underneath the plinth and moved so the pole is in line with the lower leg when the knee is flexed. The dynamometer is placed five centimetres proximal to the medial malleolus for external rotation and lateral malleolus for internal rotation.

**Figure 2**

Hip external rotation position

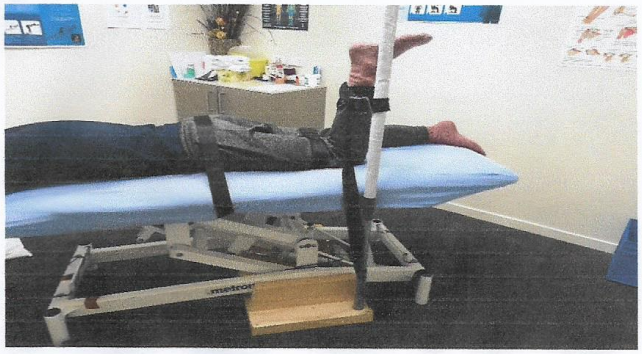
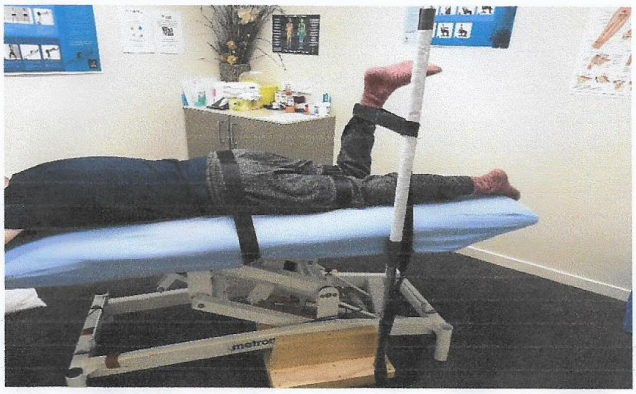


**Figure 3**

Hip internal rotation position



Instructions

The investigator instructs the participant to push by saying: Ready, steady, push. The participant pushes for five seconds, which the investigator counts down, saying stop after the allotted time. There is one practice attempt, followed the three recorded attempts. There is a one-minute rest interval between each effort. The participants' weight is recorded in order to normalise strength measures. The dominant side is also noted. The peak value of muscle strength for each side is the value used for data collection.

**Figure 4**

Custom made pole holder, moveable along the bottom of the plinth

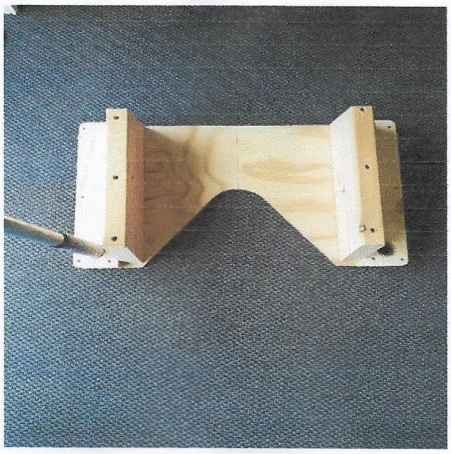


**Figure 5**

Resistance pole which the stabilisation encircles during rotation testing

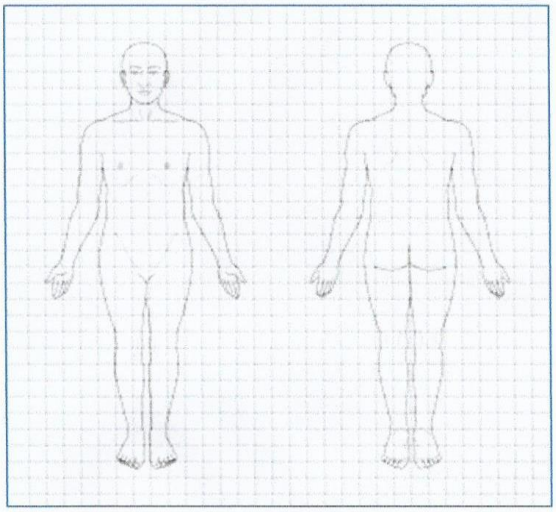
Appendix I. Palpation protocol

**Palpation Protocol**

The aim of palpation in this research study is to identify active trigger points in the gluteus medius muscles. Palpation will be used to identity:

- Localised tenderness
- Palpable taut band

The presence of both criteria indicate the presence of a trigger point. Next, it needs to be determined whether there is referral of pain on palpation of the trigger point. If there is, the assessor will mark on body chart where the person is describing. At this stage, the trigger point could still be active or latent.

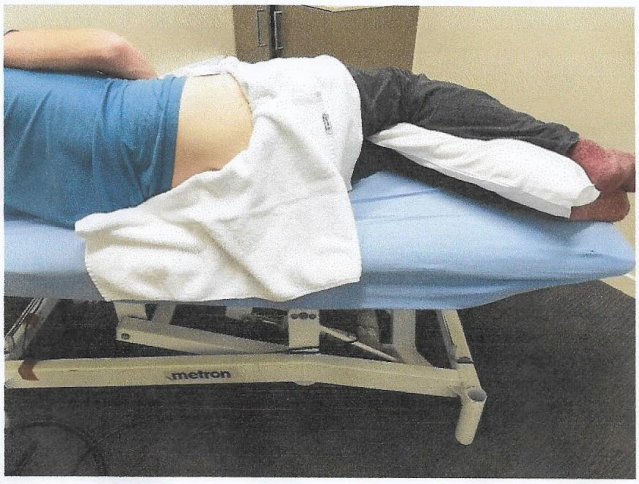


It needs to be confirmed whether the pain that is referred is recognizable/ familiar to the participant as their low back pain. The participant will have marked their low back pain on a body chart on their Personal Details Form at the beginning of their assessment session. The assessor needs to confirm whether this referred pain is the same as their low back pain. If the pain produced is their low back pain, the trigger point is active, if not it is deemed latent.

Positioning:

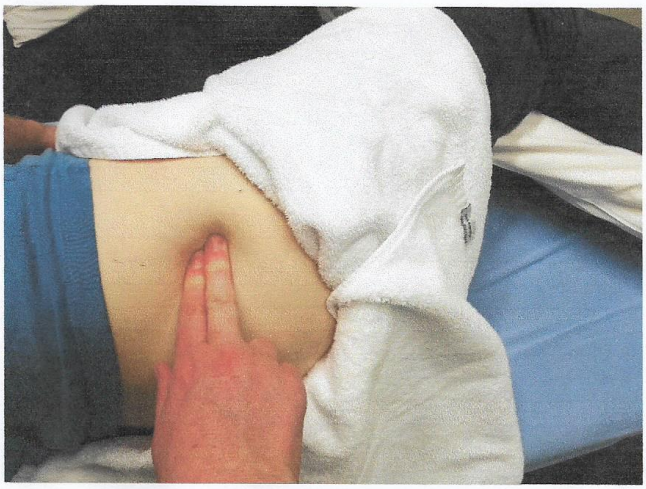
The participant will be in side lying, hips and knees bent to 45 degrees. The top hip will be in 10 degrees of adduction. This will put gluteus medius on slight stretch and aid palpation of trigger points. Pillows can be used to maintain this position, or knee resting on knee depending on the person; in order to maintain 10 degrees adduction.

The top buttock will be palpated and then this is repeated on the other side. The top buttock is exposed with towels for draping to preserve modesty and decrease risk of embarrassment. A towel will be used along the posterior border of the buttock and at the front to cover the groin area.




Method of palpation:

Using 2nd and 3rd fingers, fibre to fibre will be palpated; from a posterior to anterior direction. The assessor may mark out the borders of the gluteus medius muscle if this is helpful, especially with the first few participants.



Appendix J. Palpation assessment sheet

**Palpation Assessment Sheet**

*Right side*

1. Is there an area of localised tenderness and a palpable taut band?

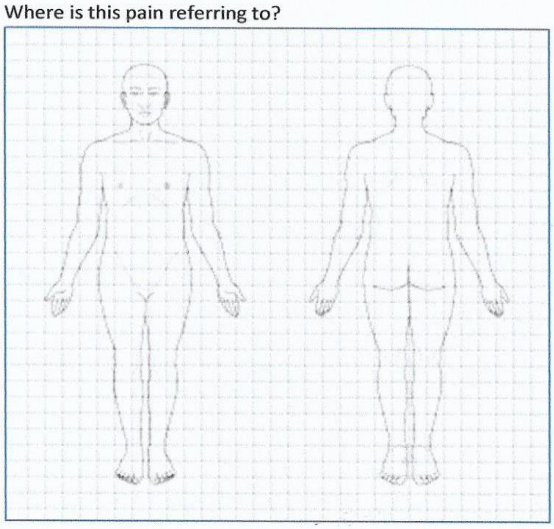
☐ Yes ☐ No

If no, there are no trigger points in gluteus medius.

2. Is the pain referring/ spreading to other areas?

☐ Yes ☐ No

Where is this pain referring to?



3. Is the pain produced on palpation of the trigger point recognizable/ familiar to the participant as THEIR LOW BACK PAIN?

☐ Yes ☐ No

If Yes = active TrP

If No = latent TrP

Number of active TrPs=_____ Number of latent TrPs=_____

Comments=_____

*Left side*

1. Is there an area of localised tenderness and a palpable taut band?

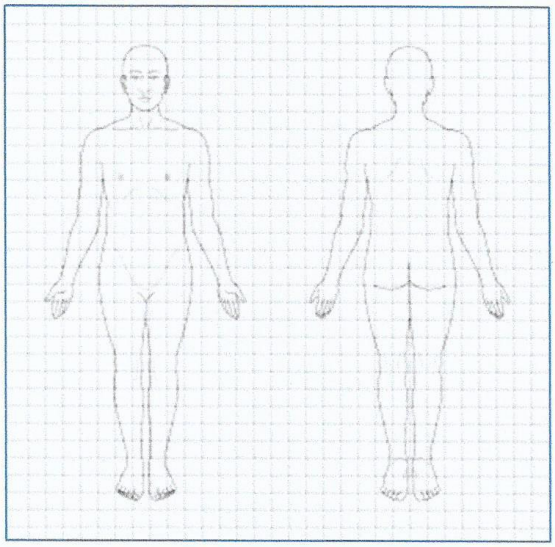
☐ Yes ☐ No

If no, there are no trigger points in gluteus medius.

2. Is the pain referred/ spreading to other areas other than where I am pressing?

☐ Yes ☐ No

Where is the pain referring to?



3. Is the pain produced on palpation of the trigger point recognizable/ familiar to the participant as THEIR LOW BACK PAIN?

☐ Yes ☐ No

If Yes = active TrP

If No = latent TrP

Number of active TrPs=_____ Number of latent TrPs=_____

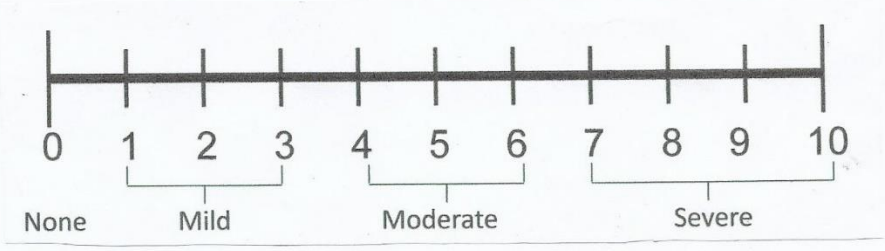
Comments=_____

Appendix K. Numeric pain rating scale

**The Numeric Pain Rating Scale**

Participant Instructions

"Please indicate the intensity of current, best, and worst pain levels over the past 24 hours on a scale of 0 (no pain) to 10 (worst pain imaginable)"




Intensity of your current pain:

Intensity of your best pain over the past 24 hours:

Intensity of your worst pain over the past 24 hours:

Reference: McCaffery, M., Beebe, A., et al. (1989). Pain: Clinical manual for nursing practice, Mosby St. Louis, MO.

Appendix L. Oswestry low back pain disability questionnaire



# Oswestry Low Back Pain Disability Questionnaire

## Instructions

This questionnaire has been designed to give us information as to how your back or leg pain is affecting your ability to manage in everyday life. Please answer by checking ONE box in each section for the statement which best applies to you. We realise you may consider that two or more statements in any one section apply but please just shade out the spot that indicates the statement which most clearly describes your problem.

**Section 1 – Pain intensity**

- ☐ I have no pain at the moment
- ☐ The pain is very mild at the moment
- ☐ The pain is moderate at the moment
- ☐ The pain is fairly severe at the moment
- ☐ The pain is very severe at the moment
- ☐ The pain is the worst imaginable at the moment

**Section 2 – Personal care (washing, dressing etc)**

- ☐ I can look after myself normally without causing extra pain
- ☐ I can look after myself normally but it causes extra pain
- ☐ It is painful to look after myself and I am slow and careful
- ☐ I need some help but manage most of my personal care
- ☐ I need help every day in most aspects of self-care
- ☐ I do not get dressed, I wash with difficulty and stay in bed

**Section 3 – Lifting**

- ☐ I can lift heavy weights without extra pain
- ☐ I can lift heavy weights but it gives extra pain
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently placed eg. on a table
- ☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned
- ☐ I can lift very light weights
- ☐ I cannot lift or carry anything at all

**Section 4 – Walking***

- ☐ Pain does not prevent me walking any distance
- ☐ Pain prevents me from walking more than 2 kilometres
- ☐ Pain prevents me from walking more than 1 kilometre
- ☐ Pain prevents me from walking more than 500 metres
- ☐ I can only walk using a stick or crutches
- ☐ I am in bed most of the time

**Section 5 – Sitting**

- ☐ I can sit in any chair as long as I like
- ☐ I can only sit in my favourite chair as long as I like
- ☐ Pain prevents me sitting more than one hour
- ☐ Pain prevents me from sitting more than 30 minutes
- ☐ Pain prevents me from sitting more than 10 minutes
- ☐ Pain prevents me from sitting at all

**Section 6 – Standing**

- ☐ I can stand as long as I want without extra pain
- ☐ I can stand as long as I want but it gives me extra pain
- ☐ Pain prevents me from standing for more than 1 hour
- ☐ Pain prevents me from standing for more than 3 minutes
- ☐ Pain prevents me from standing for more than 10 minutes
- ☐ Pain prevents me from standing at all

**Section 7 – Sleeping**

- ☐ My sleep is never disturbed by pain
- ☐ My sleep is occasionally disturbed by pain
- ☐ Because of pain I have less than 6 hours sleep
- ☐ Because of pain I have less than 4 hours sleep
- ☐ Because of pain I have less than 2 hours sleep
- ☐ Pain prevents me from sleeping at all

**Section 8 – Sex life (if applicable)**

- ☐ My sex life is normal and causes no extra pain
- ☐ My sex life is normal but causes some extra pain
- ☐ My sex life is nearly normal but is very painful
- ☐ My sex life is severely restricted by pain
- ☐ My sex life is nearly absent because of pain
- ☐ Pain prevents any sex life at all

**Section 9 – Social life**

- ☐ My social life is normal and gives me no extra pain
- ☐ My social life is normal but increases the degree of pain
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests eg, sport
- ☐ Pain has restricted my social life and I do not go out as often
- ☐ Pain has restricted my social life to my home
- ☐ I have no social life because of pain

**Section 10 – Travelling**

- ☐ I can travel anywhere without pain
- ☐ I can travel anywhere but it gives me extra pain
- ☐ Pain is bad but I manage journeys over two hours
- ☐ Pain restricts me to journeys of less than one hour
- ☐ Pain restricts me to short necessary journeys under 30 minutes
- ☐ Pain prevents me from travelling except to receive treatment

*Note: Distances of 1 mile, ½ mile and 100 yards have been replaced by metric distances in the Walking section

Appendix M. Tampa scale-11

**Tampa Scale-11 (TSK-11)** **Name:** **Date:**

*This is a list of phrases which other patients have used to express how the view their condition. Please circle the number that best describes how you feel about each statement.*

| | Strongly Disagree | Somewhat Disagree | Somewhat Agree | Strongly Agree |
|---|---|---|---|---|
| 1. I'm afraid I might injure myself if I exercise. | 1 | 2 | 3 | 4 |
| 2. If I were to try to overcome it, my pain would increase. | 1 | 2 | 3 | 4 |
| 3. My body is telling me I have something dangerously wrong. | 1 | 2 | 3 | 4 |
| 4. People aren't taking my medical condition serious enough. | 1 | 2 | 3 | 4 |
| 5. My accident/problem has put my body at risk for the rest of my life. | 1 | 2 | 3 | 4 |
| 6. Pain always means I have injured my body. | 1 | 2 | 3 | 4 |
| 7. Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening. | 1 | 2 | 3 | 4 |
| 8. I wouldn't have this much pain if there wasn't something potentially dangerous going on in my body. | 1 | 2 | 3 | 4 |
| 9. Pain lets me know when to stop exercising so that I don't injure myself. | 1 | 2 | 3 | 4 |
| 10. I can't do all the things normal people do because it's too easy for me to get injured. | 1 | 2 | 3 | 4 |
| 11. No one should have to exercise when he/she is in pain. | 1 | 2 | 3 | 4 |

Source: Woby et al. (2005), Psychometric properties of the TSK-11: A shortened version of the Tampa Scale for Kinesiophobia. Pain, 117, 137-144.