Examination of the Relationships Among Physiological Stress, Iliopsoas Tightness and Non-Specific Chronic Low Back Pain in an Adult Population

by

Arielle E. D. Nash
Bachelor of Kinesiology (Honours), McMaster University, 2000

A Thesis Submitted in Partial Fulfillment of the Requirements
for the Degree of

MASTER OF SCIENCE

In the School of Exercise Science, Physical and Health Education

Supervisory Committee

Examination of the Relationships Among Physiological Stress, Iliopsoas Tightness and Non-Specific Chronic Low Back Pain in an Adult Population

by

Arielle E. D. Nash
Bachelor of Kinesiology (Honours), McMaster University, 2000

Supervisory Committee

Dr. Lynneth Stuart-Hill, School of Exercise Science, Physical and Health Education
University of Victoria, Victoria BC Canada
Supervisor

Dr. Catherine A. Gaul, School of Exercise Science, Physical and Health Education
University of Victoria, Victoria BC Canada

Abstract

One of the most prevalent forms of pain to be linked to stress is non-specific chronic low back pain (nsCLBP). Existing studies have shown evidence of a link between stress and nsCLBP, muscle tension induced dysfunctional biomechanics and nsCLBP, and stress and muscle tension. However, little research has explored the interconnections among muscle tension, stress and nsCLBP. The purpose of this study was to investigate the relationships between these variables. Two age and sex matched subgroups (nsCLBP, and noPain) with 17 participants each (M = 8, F= 9) were created from an overall participant group (n =39, M = 18, F = 21, 23- 63 y). Iliopsoas tightness was assessed by measuring hip extension angle (HE) with a goniometer on the left (HE L) and right (HE R) side using the modified Thomas test. Heart rate variability was recorded in laying supine position for 12-minutes. A 5-minute segment extracted for analysis of low frequency power (LF; ms^2), high frequency power (HF; ms^2), LF/HF ratio and the inter-beat intervals of normal N-N sinus beats (SDNN; ms). Cortisol concentration (CORT; nmol/L) was measured using a passive drool sample taken within 2 hours of waking. Each participant was measured for all variables in one session. Significant positive correlations were found between HE and HF ($r(37)$ = .36 - .43, $p < .05$) and HE and SDNN ($r(37)$ = .27 - .41, $p \leq .05$). LF/HF and CORT were negatively correlated to HE L ($r(36)$ = -.36, p = .01). The nsCLBP group measured significantly lower ranges of motion than the noPain group for both the left and right side hip extension, with the largest difference in means of HE between groups found on the right side ($8.96^{o} \pm 3.10$, t (32)= 2.88, p =.003). There were no significant differences in CORT or HRV between the groups. The results demonstrate that iliopsoas tightness can negatively impact physiological stress and vice versa. The findings also provide evidence that individuals with nsCLBP have, on average, less range of motion in hip extension when compared to pain free

individuals. In addition, the correlation between muscle tension and stress is stronger among individuals with nsCLBP than among pain-free individuals.

Keywords: psoas, back pain, stress, HRV, cortisol, muscle tension, hip flexor, hip extension, Thomas test

Table of Contents

List of Tables

List of Figures

Acknowledgements

I would like to thank the individuals who selflessly volunteered their time and energy to be a participant in this study. Your willingness to partake in this endeavor, despite the uncertainty and challenges posed by the late stages of the COVID-19 pandemic is a testament to your generosity. Your adherence to the protocols was instrumental in ensuring the success of this research. Gratitude to Ashtanga Yoga Victoria, Inc. for providing a safe, suitable and conducive space for research testing to take place.

Thank you to the University of Victoria for accepting me as a mature student and providing a welcoming home to pursue my academic goals and grow in my profession. To the School of Exercise Science, Physical and Health Education for scholarship funding, thank you. This support has not only eased my financial burden but allowed me to focus on my studies while maintaining my responsibilities at home.

Thank you to my colleagues, friends and yoga community for the support you have bestowed upon me in this academic endeavor. To my yoga students all over the world, thank you for allowing me to be your teacher. Your experiences are the driving force behind my research. My heartfelt appreciation to Kimber, whose tireless efforts in "holding down the fort" have enabled me to take the necessary time away from work to focus on my studies. Kimber, you have always been my number one fan, and your unyielding belief in me has been a source of motivation. My sincerest thanks to Paul, who inspired me to return to school as a mature student. His trusty support, guidance, and mentorship have been critical in helping me navigate the challenges that come with pursuing higher education. Paul, your steadfast encouragement and unwavering presence in my corner have meant the world to me, and I am forever thankful to you.

Dr. Gaul, thank you for your invaluable support. Your insightful advice and clear direction have been influential in shaping my growth as a researcher and writer. With your help, I have not only improved my scientific writing ability but have also gained a deeper understanding and appreciation for research. I am incredibly grateful for your patience throughout this process. Your willingness to go above and beyond to help me achieve my academic goals has left an indelible mark on me.

Dr. Stuart-Hill, my deepest gratitude for accepting me as one of your graduate students. Your guidance and belief in my research have been invaluable to my academic growth and personal development. Your willingness to nurture my intellectual curiosity and allow me to explore my own area of interest has been pivotal in my journey as a researcher. I cannot thank you enough for the opportunity you have provided me to fuel my passion with scientific study. Thank you for being a source of encouragement and leadership throughout my graduate studies, which have played an instrumental role in shaping my professional trajectory.

To my family, I am humbled and endlessly thankful for your love and encouragement. Mom and Dad, you have always lifted me up whenever I needed it. Thank you for instilling such confidence in my abilities. Your resolute support has consistently motivated me to strive for excellence and been the foundation of my success. Dale, your unrelenting faith in me has been a guiding force in this journey. You have helped me keep perspective during the hardest moments I am lucky to have you by my side. To my remarkable children, Amakai and Jacxson, you are my heart and soul. You have taught me the true meaning of perseverance and unconditional love. I hope this work inspires you to pursue your own dreams and passions in life. Together, you are all my rocks, and for that, I am eternally grateful.

Dedication

To Amakai and Jacxson, my children and greatest teachers.

Chapter 1: Introduction

1.1 Background and Rationale

The idea that the sensorimotor response to stress is as important as the biological response stems from pioneers in somatic education and therapy (Keleman, 1985;Hanna, 1988). Psychological theories and treatments featuring muscle tension as a primary cause of psychological pathology date back to the 1890s with the work of Freud and Janet (Atarodi & Hosier, 2011). High incidence of stress, the increasing participation in mind-body practices for stress relief, and the inclusion of trauma and stressor related disorders in the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V) (American Psychiatric Association [APA], 2022), has incited the interest in the scientific community to investigate the impact of stress on physical health.

Three of four Canadians report their lives as being moderately to highly stressful (Crompton, 2011) and 25% of Canadians ages 18-64 rate their day-to-day stress as quite a bit or extremely high (Statistics Canada, 2018). That equates to nearly 6 million Canadians living with a stress-related increased allostatic load (Canada, 2019a; Seeman, 1997). The World Health Organization (WHO) reports that 30% of deaths worldwide list stress as an underlying determinant ("Cardiovascular diseases," n.d.), of which heart disease is consistently on the list of one of the top global killers (The Top 10 Causes of Death, n.d.). Gallo et al. (2014), found that 22% of individuals who experience CVD or stroke suffer from chronic stress. Stress may not be the cause of the illness however it is linked to the critical outcomes (Nunan et al., 2010).

Perhaps due to its complex nature, stress has been defined in many ways in research and investigators continue to struggle to find a consistent use of the term. A commonly used definition follows the early work of Hans Seyle where stress is seen as "an internal or external,

psychological or physical challenge that threatens, or is perceived to threaten, biological homeostasis" (Anderson et al., 2019). Even following this definition, measuring the overall load of stress on health has proven to be a complex and difficult challenge for science.

Two valid non-invasive, objective, measurable stress indicators are Heart Rate Variability (HRV) (Cysarz et al., 2015; H.-G. Kim et al., 2018; Shaffer & Ginsberg, 2017a), and salivary cortisol concentration (Shirtcliff et al., 2015; Vining et al., 1983). Although these measures do not differentiate the contribution of different types of stress - emotional, psychological or physical, they are accepted physiological biomarkers for quantifying if the body is responding to stress (Kim et al., 2018; Pulopulos et al., 2018). HRV represents the autonomic neural control of the cardiovascular system, where a reduction in parasympathetic activity and an increase in sympathetic activity reflects the system is under stress and results in a lower HRV (Shaffer & Ginsberg, 2017a). While the entire epidemiological pathway of stress to pathogenesis is not entirely clear, reduced HRV is a strong and independent predictor of adverse health prognoses, including cardiac events (Nunan et al., 2010). The sympathetic nervous system (SNS) has a variety of cardiovascular effects including increased heart rate (HR) and cardiac contractility, increased breathing capacity (bronchodilation), increased peripheral vasoconstriction and decreased venous capacitance (Vanitallie, 2002; Zhang & Anderson, 2014) . A system that is under constant stress may become dysregulated, whereby the SNS is dominating control of autonomic function. Although a higher HRV is not always indicative of better health as some pathologies can inflate HRV, optimal HRV reflects a system that is able to rapidly cope to the uncertain and constantly changing environment required to maintain homeostasis.

A higher concentration of salivary cortisol in individuals indicates a stress response state, when compared to those with lower levels (Lightman, 2008). During periods of stress, the

adrenal glands are stimulated to release the hormone cortisol, a glucocorticoid, which helps to mobilize glucose in response to higher metabolic demands (Herman, 2022). This secretion is regulated by the neuroendocrine system through a complex feedback loop, called the hypothalamo-pituitary-adrenocortical (HPA) axis (Tsigos & Chrousos, 2002).

The dysregulation of the autonomic nervous system and increased cortisol secretion brought on by stress relates it to a myriad of physical diseases, physiological ailments and mental health conditions. This includes cardiovascular disease (CVD) (Cohen et al., 2007a, 2019; Gallo et al., 2014), Type II Diabetes (Joseph & Golden, 2017; Mebazaa et al., 2013), and depression (Cohen et al., 2007a; Joseph & Golden, 2017). Stress has also been indicated as a risk factor for some mental illnesses which have been found to have a co-occurrence with Non-specific Low Back Pain (nsLBP) (Linton, 2000; Loncar et al., 2013; Stubbs et al., 2016a). The direct relationship between stress and nsLBP, however, has not been thoroughly investigated.

Cauwenbergs (2020), suggests the health of the spine has in impact on visceral function and somatic sensation, which in turn influence regulatory activity in the autonomic nervous system. This is evident in the measured correlations between stress and Low Back Pain (LBP) (Dunn et al., 2009; Loncar et al., 2013). LBP ranks among the top causes of disability globally, of which 90% is classified as non-specific (nsLBP), meaning it has no diagnostic cause (Hartvigsen et al., 2018; Hoy et al., 2010, 2012; Vos et al., 2017), and 23% of which is chronic (nsCLBP) (Balagué et al., 2012). The limited pathological understanding of nsLBP makes it difficult to treat with traditional medical interventions (Hartvigsen et al., 2018; Koes et al., 2006) and The North American Spine Society's Evidence Based Clinical Guideline for the Diagnosis and Treatment of Low Back Pain presents conflicting information on effective treatment protocols (Kreiner et al., 2020).

Muscle tension as a contributor to pain is a basic precept in pain research, and nsCLBP may be related to chronic muscle tension more than other forms of back pain (Ishihara, 2009). It is perhaps this relationship between nsCLBP and tension that directs the use of nonsteroidal anti-inflammatory drugs (NSAIDs) and exercise as treatment for nsCLBP in primary health care (Oliveira et al., 2018), and in the allied health fields, the use of alternative forms of body therapy, manipulation, acupuncture and traction (Koes et al., 2006; Scaer, 2014). Unfortunately, the evidence supporting these common treatments lack long-term clinical relevancy (Koes et al., 2006).

Ishihara (2009) suggested a causal relationship between muscle tension and LBP and postulated muscle tension as a contributing variable in the relationship between lower back imaging abnormalities and pain. Studies that support this idea have relied heavily on measurements of tension in the posterior chain of the body by using combined trunk and hip flexion range of motion (Reiman & Matheson, 2013), or other biomechanical deficits such as trunk muscle strength (Reyes-Ferrada et al., 2021). Tightness in the hamstrings and the associated decreased hip flexion range of motion is a well-documented contributor to low back pain (Fasuyi et al., 2017) however, tightness in the hip flexors and associated decreased hip extension seems to have been nearly overlooked in much of the research.

Decreased hip flexor length was reported as a predictor for nsCLBP in elite golfers by Evans et al. (2005) and limited passive hip extension was measured in subjects with nsLBP by Roach et al. (2015). Stiffness, or tension in the hip flexors limit range of motion available in hip extension movements. The iliopsoas (IP) complex has been shown to be the main source of hip flexor tensile force across all degrees of forward straight sagittal movement (Jiroumaru et al., 2014). The IP refers to the combined structure of the Iliacus (IL) muscle and Psoas Major (PM)

muscle. These two muscles merge to form a common tendon and insertion on the femur. Although the IP complex is often referred to in the literature as a single structure, IL and PM differ in their anatomy and neural innervation (Andersson et al., 2007; Sajko & Stuber, 2009a). The PM muscle has direct attachments to the lumbar spine vertebrae, which enable it to exert direct mechanical force on the lower back, the only hip flexor positioned to do so. Excessive tension, hypertonicity or stiffness in PM, can contribute to excessive anterior pelvic tilt (Evans et al., 2005), decreased range of motion in hip extension (Roach et al., 2015), and increased compressive forces on lumbar intervertebral discs (Bogduk et al., 1992), all of which have been observed in individuals with nsCLBP. In addition, individuals with nsCLBP have a higher prevalence of pain-eliciting trigger points in the IP complex and PM muscle (Andersen et al., 2012; Arbanas et al., 2013; Iglesias-González et al., 2013).

The PM muscle is referred to as the "stress muscle" in some mind-body disciplines (Koch, 2012; Morling, 2014; Staugaard-Jones, 2018). Koch (2012), highlights the PM muscle as the most significant muscle recruited during the fear cascade response. The defense, or fear cascade sequence outlines the progressive motor response sequence to a threat, from arousal to tonic immobility and, if the threat continues, eventual postural collapse (Kozlowska et al., 2015). In Koch's model, the body action response to fear or trauma involves rolling the midline of the body into a fetal curl and recruiting the IP to produce hip flexion. The constant repetition of this pattern, as would take place under repetitive fear or chronic psychological stress, interferes with the PM muscle's normal functional contribution as a spinal stabilizer and leads to chronic shortened tension in the PM muscle in the direction of hip flexion. This concept of chronic muscle tension being created as a result of psychological stress is also supported by research in

Somatic Experiencing (Payne et al., 2015; Payne & Crane-Godreau, 2015) and is the basis of somatic movement therapy (Hanna, 1988).

In a study involving refugees, Grasser et al. (2019), reported success in using body-centered treatments to decrease psychological stress symptoms. This study supports the premise that working with the body can assist with ailments that are categorized as mental health. Mind-centered or "psychological" disorders as classified by western medicine are slowly being shown to be symbiotic with the body and that moving the body acts as a method of both stress and trauma release (Grasser et al., 2019;Tekur et al., 2012). Most research, however, has been performed in those with Post Traumatic Stress Disorder (PTSD) and other clinical populations in need of therapeutic support, not as a preventative approach with the general population.

Considering the relationships outlined above between stress and muscle tension, muscle tension and pain, and pain and stress, it is possible that tension specifically in the PM muscle is an overlooked factor in the relationship between stress and nsCLBP. The current study explores the relationships among physiological markers for stress, passive hip extension angle as a measure of tightness in PM, and the presence of nsCLBP. The PM muscle is brought into focus as it is the single muscle in the human body anatomically positioned to have simultaneous effects on hip extension mobility, breathing biomechanics, and vertebral alignment (Cai et al., 2013; Neumann, 2010), in addition to the spotlight it has been given in mind-body disciplines as the stress muscle (Koch, 2012; Morling, 2014; Staugaard-Jones, 2018).

1.2 Purpose

Medical practitioners often treat mental health issues, which can be triggered by stress and musculoskeletal pain, as distinct symptoms (Oliveira et al., 2018; Stubbs et al., 2016a). Recently, mental health treatments that include therapies involving muscle and fascial tissue

have been suggested by Siccardi et al. (2021), but are not yet included in standard practice. Further understanding of common mechanisms behind the recorded co-occurrences of stress disorders and nsCLBP may help to inform the development of effective interdisciplinary protocols aimed to treat these conditions together. To the best knowledge of the author, there are no studies that have measured interrelationships of stress, PM tightness and low back pain. The aim of the current study is to begin to fill this gap in the research.

1.3 Research Questions & Hypotheses

1. What is the relationship between iliopsoas tightness (IPt) as measured by hip extension angle (HE), and physiological stress as measured by Heart Rate Variability (HRV) in an adult population?

   H_1: *There is a positive relationship between hip extension range of motion (HE) and HRV in an adult population that indicates lower HE, greater tightness in the iliopsoas with measures of HRV that indicate a higher stress response.*

2. What is the relationship between iliopsoas tightness (IPt) as measure by hip extension angle (HE), and physiological stress as measured by morning cortisol levels (CORT), in an adult population?

   H_2: *There is a significant negative relationship between iliopsoas tightness as measured by hip extension range of motion and morning cortisol, in that on average, lower CORT is correlated with greater HE.*

3. Is there a significant difference in iliopsoas tightness as measured by hip extension (HE) between adults who report incidence of Non-Specific Chronic Low Back Pain (nsCLBP) and those who do not?

H_3: *There is a significant difference in hip extension range of motion in individuals with back pain vs individuals without low back pain, in that individuals with nsCLBP measure lower hip extension angles.*

1.4 Operational Definitions

1. Heart Rate Variability (HRV):

The variation in time between heart beats, measured in the R-R interval, which reflects the autonomic nervous system control of the cardiovascular system.

2. Non-specific Chronic Lower Back Pain (nsCLBP):

Self-reported pain in the lumbar region of the back that has been persistent for longer than an 8-week period, within the previous 12 months and does not have a known cause or medical pathology.

3. Iliopsoas Tightness (IPt):

A restriction in lengthening the Psoas Major (PM) muscle or Iliopsoas (IP) complex caused by neurological tonicity, muscle tension, fascial or tendon stiffness, or short muscle fibre length, measured in degrees of passive hip extension by the Modified Thomas Test.

4. Stress:

An internal or external psychological, physical or emotional challenge that threatens or is perceived to threaten biological homeostasis, quantified by HRV and cortisol.

5. Pain:

The unpleasant sensory experience of discomfort in the body brought on by psychological or physical mechanisms.

6. Perceived Stress:

The self-rated score from an individual on current daily stress level on a scale from 1-9.

1.5 Assumptions

1. All participants followed the pre-measurement guidelines.
2. The sample is representative of the general population.
3. None of the study participants had a stress-related disorder diagnosed by a medical practitioner.
4. Study participants who reported experiencing nsCLBP, did not have a disorder that would contribute to lower back pain.

1.6 Limitations

Volunteers for the study were accepted as participants based on the inclusion criteria. Measurements were taken during a 6-week window, in the mornings in order to collect the most accurate measurement for morning cortisol levels. Availability of volunteer participants during the limited time duration and early mornings may have influenced the results to emphasize those who prefer early-morning waking.

The Modified Thomas Test is the most reliable, non-invasive, test for hip extension range of motion available to the research and in this study is being used to measure IP tightness.

Joint structure characteristics may also contribute to hip extension range of motion, therefore any limitation measured in hip extension cannot be solely due to PM or IP tightness.

There are many factors that influence stress which are not accounted for in this study. Thus, any results relating to stress cannot solely be accounted for by measures of muscle tension or the presence of nsCLBP.

1.7 Delimitations

Participants were only measured on one occasion, which may not be adequate to measure long-term relationships between variables.

As no specific physical activity level or sedentary status was included in selection criteria, results may differ with an active or athlete population.

The Modified Thomas Test does not take into consideration fascial restrictions in adjacent tissues that may also limit passive hip extension. Participants who were unable to perform the Modified Thomas Test due to severe back pain, were not able to be included in the sample. Any conclusion regarding PM or IP tension as measured by passive hip extension using the Modified Thomas Test cannot be generalized to muscle tension in other muscles or the whole body.

Stress in this study was measured at only one single point in time, which means the measure is for that point in time only and cannot infer the measure would be the same on any other day.

Chapter 2: Literature Review

Stress is a normal part of life. How the human body responds to and recovers from stress is vital to survival, optimal physiological functioning and mental well-being. Although the precise, complete mechanistic pathways remain unclear, and whether psychological, emotional or physical, stress is listed a risk factor for a myriad of physical diseases, physiological ailments, musculoskeletal pain and mental health conditions. Stress has been suggested as a significant predictor of and risk factor for non-specific chronic low back pain (nsCLBP) (Fatoye et al., 2019; Kim et al., 2006; Linton, 2000), an ailment that ranks among the top causes of disability globally (Hoy et al., 2010, 2012; Vos et al., 2017). The direct relationship between nsCLBP and stress has not been heavily investigated, however, research findings have identified pain as a stressor, (Ganesan et al., 2017; Hallman & Lyskov, 2012) suggesting nsCLBP itself may increase the stress response, in addition to the stress response exacerbating the pain, thus perpetuating a pain-stress cycle (Boersma et al., 2012; Hannibal & Bishop, 2014). In research involving temporomandibular jaw disorder (TMJD), Glaros et al. (2016), classified muscle tension as a mediator in the bidirectional relationship between distress and TMJD pain. The current study is aimed at exploring the stress-pain-tension relationship by examining nsCLBP and hip flexor tightness rather than TMJD pain and jaw muscle tension. When considering nsCLBP, the biomechanical implications of improper force distribution along the vertebral column and pelvis must also be considered. Excessive tension, hypertonicity or stiffness in one of the primary hip flexors, the psoas major (PM) muscle, contributes to excessive anterior pelvic tilt (Evans et al., 2005), decreased range of motion in hip extension (Roach et al., 2015), and increased compressive forces on lumbar intervertebral discs; all of which have been observed in individuals with nsCLBP.

The following review of literature is presented to outline the background and summarize the current scientific understandings of stress, pain and muscle tension. In addition, historical research relevant to the relationships among them is presented. The physiology applicable to the use of Heart Rate Variability (HRV) and salivary cortisol (CORT) as biomarkers of stress and the functional anatomy of the Iliopsoas (IP) complex and Psoas Major muscle (PM) and its role in hip extension range of motion (HE) are explained. The reliability of using the Modified Thomas Test (MTT), as a means of measuring tightness in the PM muscle is also discussed.

2.1 The Stress Response and Implications for Health

Maintenance of homeostasis in the human body is reliant on a complex physiological system that responds to stressors appropriately and readily recovers to baseline "no threat" functioning upon removal of the stressor. The immediate activation of the sympathetic nervous system (SNS) upon perception of a threat, begins a sequence of events preparing the body for "fight or flight". This physiological cascade, including increased heart rate, blood pressure and respiration rate, along with the secretion of adrenaline hormones and cortisol, is beneficial for survival in the short term. However, a system that is repetitively or chronically overloaded with stress or does not have the capacity to handle the stress response due to other health or lifestyle factors, may suffer negative physical or mental health consequences. Those negative consequences not only have an economic impact, but they can also sometimes be fatal.

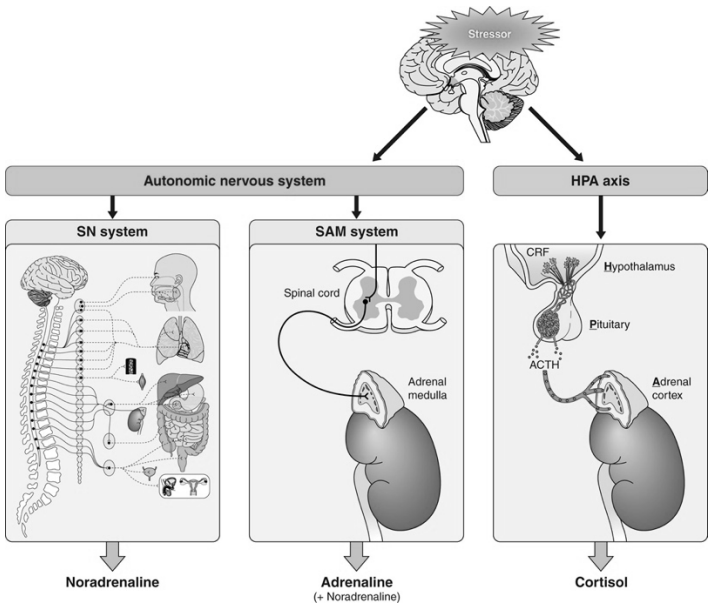
Stress is a risk factor for cardiovascular disease (CVD) (Heart and Stroke Foundation, 2022.) (Cohen et al., 2007b). In Canada, CVD constitutes 33% of deaths and an estimated $22 billion in direct annual health care costs each decade (Cardiovascular Disease - Economic Burden of Illness - Canada.ca, n.d.; Tarride et al., 2009). Stress also contributes to Type II Diabetes which has a prevalence of 8.1% in Canada (Diabetes in Canada - Canada.ca, n.d.). The

projected cost to health care in a 10-year period is over $15 billion, and climbing each year (Bilandzic & Rosella, 2017).

Stress may limit a person's ability to work, contributing to the increasing annual average of 12 lost workdays per worker per year in Canada (Statistics Canada, 2019). Although days lost specifically related to stress are not explicitly reported, an estimated 500,000 Canadians are away from work each week due to declined mental health caused by stress (Statistics Canada, 2004). In 2023 wages, that is an estimated $1.6 billion in direct costs to Canadian employers (Statistics Canada., 2021). This number does not include employee replacement costs or disability benefits, and these financial impacts of lost workdays is rising every year (Statistics Canada, 2004).

Figure 1

Effector Systems of the Stress Response



Note. From *The Corticotropin-Releasing Factor Family: Physiology of the Stress Response,* Deussing & Chen, 2018, Physiological Reviews, 98. Copyright 2018 by the American Physiological Society. Reprinted with permission.

Activated when met with a real or perceived threat, the human stress response is a complex system of neural inputs from the central nervous system (CNS) and systemic feedback, which lead to a variety of physiological responses in the body. This is achieved through interconnected autoregulatory mechanisms of the sympathetic nervous system (SNS), sympathetic-adrenomedullary (SAM) system and the hypothalamic-pituitary-adrenocortical (HPA) axis (Tsigos et al., 2020; Vanitallie, 2002). There is a consequential release of norepinephrine, epinephrine and cortisol (Deussing & Chen, 2018; Lightman, 2008; Russell & Lightman, 2019). Duessing & Chen (2018), provide a simplified diagram of this process (Figure 1).

2.1.1 Cardiovascular Effects

The activation of the SNS and SAM and the resultant release of epinephrine (adrenaline) and norepinephrine (noradrenaline) have a direct effect on the cardiovascular system. The contraction of the heart itself is moderated by both SNS and parasympathetic (PSNS) input to the sinoatrial (SA) node, the pacemaker of the heart. This dual innervation results in a variability in the time between heart beats, called heart rate variability (HRV) and represents the dynamic interaction of both the PSNS, which decreases HR and SNS which increases it (McCraty & Shaffer, 2015). Respiratory Sinus Arrhythmia (RSA) is the HRV that occurs in synchronicity with the normal respiratory cycle, where the exhale activates the PSNS and lowers HR, and the inhale activates the SNS, increasing it (Billman, 2011; Fisher et al., 2022). The measurement and proper analysis of HRV, to be discussed later in this chapter, are of clinical significance in assessing cardiac health and cardiac mortality risk (Thayer et al., 2010, Zhang & Anderson, 2018). Generally, low HRV indicates higher risk and a system that is neurologically dominated sympathetic control (McCraty and Shaffer, 2020; Shaffer & Ginsberg, 2017). In addition to an

increase in HR, SNS activation also results in increased blood pressure (BP) caused by increased cardiac contractile force and increased peripheral vasoconstriction (Cohen et al., 2007a, 2019; Fuchs & Whelton, 2020; Zhang & Anderson, 2014). In a systematic review analyzing the association between stress and high BP, Liu et al. (2017) found that individuals with chronic stress are 2.4 times more likely to suffer from hypertension (high BP). It is because of the potential damage high pressure can exert on coronary arteries and other vessels that hypertension increases the risk of CVD (Fuchs & Whelton, 2020). The increase in peripheral vasoconstriction narrows vessels that supply non-vital areas of the body in order to redistribute blood flow to vital organs, including the heart, which in turn increases the workload on it, and contributes to higher pressure.

2.1.2 Hormonal Effects

The SAM's role in stress response is not as complex. The SNS has a direct innervation to the adrenal medulla, which it stimulates to release epinephrine and norepinephrine in preparation for fight or flight. Epinephrine acts on multiple organs and tissues to prepare the body for action, including increasing HR and BP, bronchodilation, and the breakdown of glycogen from the liver and muscles. Norepinephrine (NE) released into the bloodstream directly by the SNS contributes to the cardiovascular changes already discussed. NE released by the adrenal medulla, through direct SNS innervation can stimulate the adrenal cortex to secrete the hormone cortisol (Vanitallie, 2002). Cortisol is also released by the adrenal cortex under the regulatory commands of the neuroendocrine feedback loop of the HPA axis. Cortisol has multiple functions in the body. Of relevance in this study are its role in promoting vasoconstriction which contributes to higher blood pressure and mobilization of glucose for fuel, together with modulating inflammation. Under normal conditions, cortisol fluctuates throughout the day, with an initial 30

– 40 minute rapid rise to a peak after waking (cortisol awakening response – CAR), then a gradual decrease throughout the day, reaching its lowest at night and during sleep (Adam & Kumari, 2009; Sveinsdottir et al., 2016). Approximately 15-minutes after the onset of stress, cortisol levels rise in the system and remain elevated for several hours before returning to normal levels (Hannibal & Bishop, 2014). The acute increase in cortisol secretion serves to reduce inflammation, increase available energy, increase awareness and improve immunity (Adam & Kumari, 2009). Repetitive stress exposures prolong or excessively activate the HPA axis and may result in higher than normal levels of cortisol. This elevated cortisol level increases blood sugar levels which can lead to increased fat storage, insulin resistance and the development of Type II diabetes (Geer et al., 2014).

Prolonged heightened levels may exhaust the HPA-Axis and lead to cortisol resistance or dysfunction and ultimately dampen secretion (Fries et al., 2005; Hannibal & Bishop, 2014). Chronically low cortisol levels, including decreased morning levels and blunted cortisol responses to acute stress have been measured in individuals suffering from stress-related disorders and individuals exposed to high daily work stress (Heim et al., 2000). Termed hypocortisolism, this phenomenon may be caused by multiple complex mechanisms that exhaust the HPA-Axis (Heim, et al., 2000; Fries et al., 2005) and lead to the overall reduction in basal cortisol.

It is evident that sustained or repetitive stress has a cumulative impact on physiological outcomes, which increases risk for CVD and Type II Diabetes. Stress also has a strong association with pain.

2.2 The Stress – Pain Relationship

Pain can manifest in various forms and with different attributes, encompassing both emotional and physical aspects. In the current study, pain is classified as the unpleasant sensory experience of discomfort in the body, brought on by psychological or physical mechanisms. According to the International Association for the Study of Pain and International Classification of Diseases (ICD) 11, acute pain is a symptom of an underlying cause and chronic pain is that which extends beyond three months, irrespective of the cause and is considered a health condition on its own (Treede et al., 2019). The neurobiological mechanisms of pain are extremely complex, and not yet fully understood (Boersma et al., 2012). Stated simply, sensory and/ or cognitive information that has the potential to damage or injure is transmitted to the CNS, processed and then perceived as the sensation of pain (De Ridder et al., 2021).

2.2.1 How Pain Works

Nociceptors are specialized sensory receptors that communicate with polysynaptic interneurons in the spinal cord, which transmit information up the spinothalamic tract to the brain. Nociceptor activation serves to initiate pain sensation in the affected region as a form of protection until the specified region can heal (De Ridder et al., 2021). Nociceptors are activated by possibly harmful chemical, temperature, mechanical or electrical stimuli (De Ridder et al., 2021). After processing in the brain, once at the spinal cord, there are several possible efferent pathways the signal may travel. The effects can include, local muscle hypertonicity, increase in sympathetic nerve activation, increase of neuroendocrine stress response and changes in systemic visceral activity (Dubin & Patapoutian, 2010).

The pain-stress relationship is bi-directional as pain itself is recognized in the scientific community as a stressor (Ganesan et al., 2017; Hallman & Lyskov, 2012). When pain signals reach the brain, they activate the hypothalamus, a key regulator of the HPA-axis and autonomic

nervous system, resulting in stimulation of the physiological stress response. Chronic pain can develop from multiple different mechanisms, two of which are: persistent activation of nociceptors and stress-induced shifts in the HPA-axis and SNS (De Ridder et al., 2021; Salberg et al., 2020). This pain-stress cycle is perpetuated by the increase in pain sensitivity which occurs as a protective response to stress and is mediated by the release of cortisol and increase in SNS activation. One such type of pain is musculoskeletal pain, which can be both instigated by and exacerbated by stress (McBeth et al., 2007).

2.2.2 Stress and Musculoskeletal Pain

Research linking psychological stress and musculoskeletal pain has primarily focussed on chronic widespread pain as an experienced symptom of fibromyalgia, chronic fatigue syndrome, rheumatism, temporomandibular joint dysfunction (TMJD) or post-traumatic stress syndrome (PTSD) (McFarlane, 2007). In a longitudinal study, McBeth et al., (2007) provided evidence of psychological stress as a source of chronic musculoskeletal pain. The study was designed to examine the potential modulation effect of abnormalities in the HPA-axis, measured by cortisol levels, in the onset of pain. From a cohort of 463 randomly selected participants who, based on psychological profile were flagged for future risk of chronic musculoskeletal pain, 267 pain-free participants qualified for the study. Salivary cortisol was analyzed from 9:00 am and 10:00 pm samples. Follow up samples were taken 15-months later, when participants reported on whether they were experiencing new pain symptoms. After controlling for depression, sleep, sex, trauma and other psychosocial factors, the results revealed that failure to suppress the HPA axis, lower morning and higher evening cortisol levels were independent predictors of onset of chronic pain. Individuals with morning salivary cortisol levels below 3 nmol/L were found to be 1.5 times more likely to develop pain and those with evening cortisol higher than 1nmol/L were 2.5 times

more likely to develop symptoms. The pain reported by participants in the McBeth et al. study, was classified to be widespread - the type of pain developed in those who suffer from fibromyalgia, and not isolated to any specific body region, a limitation common to research linking pain and stress.

Hallman & Lyskov (2012), found a significant decrease in parasympathetic contribution to HRV measurements in subjects who did not suffer from fibromyalgia, but reportedly suffered from unexplained chronic isolated shoulder, neck and upper back pain. In the study, analysis of 24-hour monitoring of HRV showed significantly lower high frequency power and time variation in normal heart beats in the pain group when compared to asymptomatic controls. They did not observe a difference between the groups in self-reported perceived stress or normalized low frequency power values. In a laboratory study involving rats, Alvarez et al. (2013), reported an increased susceptibility to chronic muscle pain in adulthood after experience of early-life psychological stress.

Reduced blood flow, inflammation, muscle tension and poor posture are all mechanisms that may contribute to the muscular pain-stress relationship. The increased vasoconstriction of the periphery during stress causes a reduction of blood flow to muscles. Lack of blood flow can result in lack of oxygen and nutrients reaching the tissues (Cohen et al., 2007b, 2019; Fuchs & Whelton, 2020; Zhang & Anderson, 2014). Muscles that do not receive the metabolites needed for function can become tight, fatigued and painful (Mense, 2008, 2010, 2013b, 2013a). As previously explained, the release of cortisol during stress triggers an increase in inflammatory molecules in the body. The inflammatory accumulation of fluid in muscles can create a pressure stimulus on nociceptors, resulting in pain sensation in the muscle (Mense, 2008, 2010, 2013b, 2013a). When stressed, the body's natural response is to tense muscles, which can result in

muscles becoming tight and strained (Shahidi et al., 2013). Over time, this tension can lead to the development of trigger points, which are areas of the muscle that are tender to touch, and can cause pain (Celik & Mutlu, 2013). The observation of postural shifts during stress and stress-related psychological disorders can put additional strain on muscles and cause disruption in biomechanics leading to musculoskeletal pain (Shahidi et al., 2013). Stress-pain research that includes the contribution of postural and/ or muscle tension mechanisms in the relationship is not heavily reported.

As previously noted, there is evidence of a positive correlation between back pain and mental health. Stubbs et al. (2016) reported that individuals who suffer with back pain are associated with a two-fold increase in the odds of suffering from anxiety. Although studies are unable to claim any bidirectionality to these relationships, evidence does support the bidirectional relationship connection between stress and musculoskeletal pain. The suggestion for mental health practitioners to screen for back pain and for a musculoskeletal therapist to screen for mental health is given supported by Pinar et al., (2015) who reported a significant decrease in stress markers in subjects who received back massage to release muscle tension when compared to those who did not. In addition to reduced blood flow and inflammation, muscle tension and poor posture have also been identified as mechanisms in the stress-muscular pain relationship.

2.2.3 Stress and Muscle Tension

The exercise pressor reflex (EPR) is a reflexive mechanism that is activated by the contraction of muscle as a result of stimulation of mechanically and metabolically sensitive afferent nerve endings within the muscle (Kaufman & Hayes, 2002). It is a crucial component of the automimic control of vascular tone during exercise (Lydakis & Sinoway, 2007; J. H. Mitchell

et al., 2003). The EPR has two main components: the muscle mechanoreflex and the metaboreflex.

The muscle mechanoreflex is initiated by the stimulation of mechanoreceptors that respond to mechanical stress elicited on muscle during contraction (Kaufman & Hayes, 2002). Such stimulation transmits signals to the CNS to increase activity in the SNS. This results in a reflexive increase in heart rate, contractility and peripheral resistance, all which help to maintain blood flow to working muscles (Kaufman & Hayes, 2002). The metaboreflex, on the other hand, is activated by metabolic stress in the muscles, such as oxygen debt (Kaufman & Hayes, 2002). Again, signals are sent to the CNS, which further increase SNS activity.

Although the complete neural mechanisms underlying this effect are unclear, a chronically overactive muscle could be the result of a hyperactive EPR (Murayama et al., 2012; Willmann et al., 2012). The EPR provides a mechanistic explanation as to how chronic muscle tension, which is created by persistent muscle contraction, can create cardiovascular changes indicative of a physiological stress response via activation of the SNS (Lydakis & Sinoway, 2007). An overactive EPR has been consistently recognized as the cause of the abnormal cardiovascular response to exercise in patients with hypertension (Smith et al., 2015) and may contribute to cardiac mortality in patients with heart failure (Lydakis & Sinoway, 2007).

In response to stress, involuntary contraction of skeletal muscles occurs in the initial arousal stage, which is the start of the defense cascade. Beginning in the brain, when a stimulus is perceived as a threat, the hypothalamus pathway is activated resulting in increased tone in the SNS and somatic nervous system. Following the initial arousal, if the threat continues, or a misperception of the stimulus as a threat continues, the hypothalamus continues to activate the SNS and increase muscular tone to prepare the body for action. This is the fight or flight stage.

Remaining in a state of fight or flight, which is hyperarousal, for a prolonged period, results in increased muscle tension and musculoskeletal pain (Nyboe et al., 2017). Hazlett et al., (1994) used electromyography (EMG) to record muscle activity in the gastrocnemius and frontalis muscles in eighteen individuals with general anxiety disorder and compared them to EMG in non-anxious individuals. During a laboratory cognitive stress-inducing activity, EMG results showed increased muscle activity in both groups. The groups did not show a difference in the frequency or duration of muscle contraction however the general anxiety disorder group did have significantly stronger contractions.

Individuals who experience pain and muscle tension associated with temporomandibular jaw disorder (TMJD), typically display higher levels of emotional distress than those without TMJD-associated pain (Glaros et al., 2016). Glaros et al., (2016) aimed to understand the directionality of the relationships among TMJD pain, distress and muscle tension. In their study, data were collected from a single group of 171 participants who tracked their level of distress, muscle tension and TMJD pain symptoms for a 1-week period. Overall, the analyses showed increases in distress had stronger influence on tension than increases in tension had on distress (distress ⇄ tension). However, the results revealed significant bidirectional relationships for all variables; TMJD pain ↔ distress, muscle tension ↔ TMJD pain, and TMJD pain ↔ distress. These results were coherent with the 2015 cross-sectional study by Tosato et al. (2015). The Tosato et al. study reported a significant negative correlation between TMJD muscular activation triggered by stress and salivary cortisol levels. One aspect of exclusion criteria for the Tasato et al. study, which was not reported by Hazlett et al., (1994) was active participation in physiotherapy to alleviate musculoskeletal pain or manage biomechanical deficiencies. The EMG measured tension in the masseter muscles by Tosato et al., was analyzed against severity

of TMJD pain and cortisol level. Moderate to strong significant positive correlations were found between EMG activity and TMJD pain for all levels of severity, and a negative correlation between salivary cortisol and EMG activity which was only significant in those who experienced severe pain. This indicates that severity of pain influences the relationship between muscle tension and cortisol.

Body treatment therapies that aim to reduce muscle tension, have shown efficacy in decreasing stress response (Delaney et al., 2002; Morling, 2009; Pinar et al., 2015). Delaney et al. (2002) used analysis of HRV to reveal an increase in parasympathetic (PSNS) activity following 20 minutes of trigger-point massage of the head, neck and shoulder area. They reported an associated 39% decrease in muscle tension in the treatment group, where the control group showed no change. One limitation of the study was that although it significantly related muscle tension and stress, the measurement of muscle tension was subjective. This is consistent throughout much of the literature on the muscle tension- stress relationship. The RCT by Pinar et al., (2015) examined the effect of back massage on self-reported anxiety, cortisol and blood pressure in a group of cancer care givers. The results showed a significant decrease in self-reported anxiety, measured cortisol levels, and blood pressure after one week of daily 15-minute massage. Massage has been shown to decrease muscular tension (Gasibat & Suwehli, 2017) however, Pinar et al., did not use an objective quantitative measure of muscle tension, and thus it's not possible to conclude the results they measured were because muscle tension was decreased by the massage.

The exact mechanisms underlying the relationship between stress, muscle tension and sympathetic activity are complex and not fully understood. However, the literature does support that the sympathetic nervous system plays a key role in regulating muscle tension in response to

stress via the EPR, somatic defense cascade and secretion of cortisol. The relationship may be bidirectional, as supported by Glaros et al., (2005) with stress causing muscle tension and muscle tension further stimulating sympathetic activity. An additional area that is important to consider, is the contribution of biomechanical deficiencies and fascial health in the muscle tension-pain relationship.

2.2.4 Muscle Tension, Stiffness and Pain

Chronic muscular tension has been identified as a source of chronic pain. Physiologically, muscle tension is the result of the contraction of muscle fibres, which are controlled by signals from the somatic nervous system (Derrickson, 2017). When the muscle receives a signal to contract, the muscle fibres shorten and produce tension which can be felt as a sense of hardness or tightness. The degree of tension varies based on the rate at which muscle fibres are stimulated, the length of the muscle fibres prior to the contraction and the diameter of the fibres. Overall, whole muscle tension is dependent on the number of muscle fibres that are simultaneously recruited and the number of motor units (combination of individual motor neuron and the fibres it innervates) that are activated (Derrickson, 2017). Consistent, repetitive activation of the somatic nervous system, as may occur in chronic stress, results in short, tight muscles due to continuous patterns of tension.

Muscle tension can cause pain through a variety of mechanisms. Sustained muscle tension reduces blood flow to the muscle which can cause muscle fibres to be deprived of oxygen. This creates an accumulation of metabolic waste products, which can activate nociceptors, leading to pain (Mense, 2013). Tense muscles can also cause compression on nerves or vessels which evokes a mechanical stimulus for pain (Campbell & Meyer, 2006; Rempel et al., 1999; Tonkin, 2010). In addition, shifts in posture due to spending prolonged time

in the same body position, for example computer sitting, or from repetitive activities may cause compensatory muscle tension in other areas of the body, or tension in from overuse, exacerbating the muscle tension-pain relationship (Tonkin, 2010).

Not included in the definition of muscle tension, but an important factor in examining possible mechanisms of musculoskeletal pain, is in the fascia. Fascia is multi-layered tissue primarily comprised of a collagen matrix and hyaluronan. The fascial system refers to the three-dimensional continuum of soft, collagen containing, loose and dense fibrous connective tissue that permeates the body and enables all body systems to operate in an integrated manner (Schleip et al., 2019). It includes neurovascular sheaths, ligaments, tendons, intramuscular and intermuscular connective tissues (Schleip et al., 2019). Fascia is rich in mechanoreceptors, including nociceptors (Bordoni & Varacallo, 2018; Bordoni & Zanier, 2013; Schleip, 2017). The stimulation of these nociceptors elicits activity in the sympathetic nervous system and in surrounding viscera (Schleip, 2017).

The mysofascia, a subcategory of the fascial system, is a part of the intermuscular connective tissue and is composed of the endomysium, perimysium and epimysium. The myofascial system transmits mechanical force, which can either assist or limit joint movement. This mechanical force is distributed along tissues aligned within the same directional arrangement, and in presence of adhesions or inflammation, to adjacent tissues. As a result of injury, consistent muscle contraction, inactivity, repetitive movement or habitual poor posture, myofascia can start to deform. These forms of acute and chronic loading of fascial tissues stimulate collagen remodeling in order to continue to support the structural integrity of the body (Zügel et al., 2018). Continuous collagen remodeling at the same site increases fascial tissue density and adhesions which leads to decreased muscle extensibility and elasticity (Maganaris et

al., 2017). When a muscle loses its extensibility, range of motion at the joints the muscle acts on will be decreased (Maganaris et al., 2017; Schleip et al., 2010). In addition, adhesions in fascia can increase over time, causing the myofascia to compress the muscle it surrounds, stimulating the muscle mechanoreflex, contributing to ischemia induced muscle tension and painful trigger points (Driscoll, 2017).

Along with being a source of musculoskeletal pain, muscle tension and tissue stiffness, rigidity and decreased elasticity all contribute to limited range of motion within joints. It is because of the interrelationship between muscle tension and fascial stiffness on the extensibility of muscle, the term "tension" in this study is taken to operationally mean the combined tensile forces exhibited by muscle contraction and the muscle's associated fascial tissue.

2.2.5 Stress and Low Back Pain

Low back pain (LBP) is the leading cause of disability around the world, the majority of which can be classified as non-specific (nsLBP), meaning without clinical diagnosis (Hartvigsen et al., 2018). However, non-specific pain that lasts a period of three months or longer, whether consistent or intermittent is considered chronic and is represented as non-specific chronic low back pain (nsCLBP) (Balagué et al., 2012; Deyo, 1986). Although psychological stress is indicated as a risk factor for depression and anxiety, both which have a co-occurrence with both nsCLBP and acute nsLBP (Linton, 2000; Loncar et al., 2013; Stubbs et al., 2016b), and stress has been identified as an important risk factor for low back pain (Fatoye et al., 2019; Puschmann et al., 2020), research investing the relationship between stress and nsLBP or nsCLBP has varied results. In its relationship to depression, anxiety, PTSD (Atarodi & Hosier, 2011; Hartvigsen et al., 2018; Loncar et al., 2013; Scaer, 2014) and overall mental health (Stubbs et al., 2016a), nsCLBP has been referred to as a pain that originates from psychological factors rather than

physical ones, known as psychosomatic or psychogenic pain. There are no specific statistics outlining the percentage of individuals who relate their back pain to stress, and as previously explained, the relationship between stress and pain is complex and multifactorial. Some research does however suggest that stress can be a significant contributing factor to back pain in some people and the overriding tenet in many studies is that psychological and emotional factors trigger some type of physical change resulting in back pain. Overall, however, the empirical evidence is inconclusive in identifying the extent or validity of this relationship.

Using data from the World Health Survey, Stubbs et al. (2016) categorized any pain in the back experienced within the 30 days prior to the survey as "back pain", and "chronic back pain" as pain experienced on all 30 days. From the cross-sectional community-based study, spanning seventy countries, 190,593 individuals from 43 countries made up the final sample. Along with depression, psychosis, sleep problems and anxiety, stress sensitivity was analyzed as an exposure variable for back pain or chronic back pain outcomes. They found a linear increase in the prevalence of both types of back pain with higher severity of stress sensitivity. A one-unit increase in stress sensitivity (measured as a range of 2-10), was associated with 1.13 times higher odds for any back pain. As one of the largest studies to link back pain and psychological stress to date, it provided evidence in support of including mental health screening in assessing cases of back pain and screening for back pain in those who suffer from mental illness. The study, however, did not use any validated biomarkers to measure stress objectively, which does not conclude a definitive link between physiological stress response and back pain.

Ganesan et al., (2017) reported survey data from 1355 adults aged 18-35 including sociodemographic and lifestyle factors that are considered risk factors for LBP, and experience

of lower back pain. Stress was reported by 24.2% of participants and was concluded to be a significant predictor of back pain. The study, however, did not clearly define or quantify stress.

In their recent systematic review, Bandeira et al., (2021) identified only two RCTs that used ECG to measure baseline resting HRV in individuals 18-65 years of age with nsCLBP. They reported on two very different studies. In one of the studies, Shankar et al., (2011) aimed to observe the effects of electro acupuncture therapy in the management of nsCLBP. Along with blood pressure, HRV was measured for 1 minute with a controlled 6 breath per minute breath rate. A low (during exhale) to high (during inhale) R-R interval ratio (E:I) between groups was analyzed. They found significantly higher BP and lower E:I ratio in the nsCLBP at baseline, indicating decreased parasympathetic and increased sympathetic tone compared to the controls. The second study reported by Bandeira et al. was by Kalezic et al., (2010) who aimed to investigate autonomic regulation in response to functional health testing. They measured baseline HRV during paced 5 breaths per minute, controlled breath rate in 93 nsCLBP patients ages 20-50, and compared to 32 healthy controls. A significantly lower HRV was found in the patients with nsCLBP, specifically higher LF and lower HF values, which represent sympathetic hyperactivity and parasympathetic withdrawal. SDNN measures, however, did not show a significant difference. This is supported by the work of Pontes-Silva et al. (2022) who found dysregulation in the PSNS as measured by lower resting HF in individuals with both neck and back pain when compared to controls.

Salivary cortisol has been sparsely used as a measurement of stress in back pain research. Sveinsdottir et al., (2016) compared Cortisol Awakening Response (CAR), daytime cortisol slope and evening cortisol levels analyzed from salivary cortisol samples in 305 nsCLBP patients. When compared to normative values, evening levels and daytime slope were similar,

however CAR values were found to be significantly higher (7.63 vs 4.23nml/L) in the patients. The study showed a weak, but significant negative correlation between CAR and pain in the hips and back. The shared variance between CAR and all body pain was found to be 5.1%, but was not significant for back pain alone. Earlier work by Sudhaus et al. (2007) differed in its findings of significantly lower overall cortisol profiles in patients with acute or chronic low back pain patients compared to healthy controls. Sudhaus et al. did not find a significant increase in cortisol secretion in either group upon eliciting an acute thermal pain stimulus, a result later replicated by Muhtz et al. (2013).

In investigating the relationships among cortisol, mental health and low back pain, Muhtz et al. (2013) induced pain in three separate groups - individuals with nsCLBP and not depression (n=20), with depression and not nsCLBP (n=22) and healthy controls (n=33). The nsCLBP group measured lower cortisol when compared to depressed patients, which were not different from controls. There was no significant increase in cortisol at any time point when acute thermal pain was elicited, and no significant relationship was found between the duration or the intensity of the acute pain and cortisol. The research of Sudhaus et al. (2007) and Muhtz et al. support the idea that constant nociceptor stimulation in the presence of chronic pain and may lead to an exhaustion of the HPA-axis (Fries et al., 2005; Heim et al., 2000) but is not congruent with the other well-established science that acute pain stimulates the HPA axis, increasing cortisol (Hannibal & Bishop, 2014).

It is important to note that not all individuals who experience back pain will attribute it to stress, or report experiencing increased stress as a result of back pain. Many other factors such as physical activity, posture and medical conditions can also contribute to back pain.

Additionally, stress may not be the primary cause of back pain, but rather a contributing factor that exacerbates existing pain or makes it more difficult to manage.

2.3 Anatomy and Function of the Iliopsoas and Psoas Major

Located deep within the anterior hip joint and lower spine lies the PM. The PM is a large muscle, unlike any other in the abdominal area, arising from the lower thoracic and lumbar spine and terminating distally on the proximal femur. Although commonly referred to as a single continuous hip flexing muscle, dissection has revealed that the muscle is a series of overlapping segmental fascicles, with each fascicle arising from a distinct point on the lumbar spine (Bogduk et al., 1992). The attachment points on the lumbar spine are either on the posterior 7/8ths of the lateral surface of each intervertebral disc between and including T12-L1 to L4-L5 and or the transverse process from T12-L5 (Gray, 2009). These bundles pass caudally, laterally and anteriorly to join with the iliacus (IL), forming the common iliopsoas tendon, inferior to the pelvic brim (Gray, 2009; Myers, 1996). The PM and the IL together form the iliopsoas (IP) complex. Although the IP complex is often referred in literature as a single structure, PM and IL differ in their anatomy and neural innervation (Andersson et al., 2007; Sajko & Stuber, 2009).

The PM is innervated directly from the anterior rami of L1-L4 of the lumbar plexus (Siccardi et al., 2021; Singh & Al Khalili, 2022), while the IL is innervated by the femoral nerve (L2, L3), which is a nerve that forms after exiting the lumbar plexus (Martini et al., 2012). The IL is a fan-shaped muscle that occupies the internal iliac fossa, originating on its upper two-thirds. The muscle bundles move toward the lateral side of the PM tendon, on which they insert (Siccardi et al., 2021).

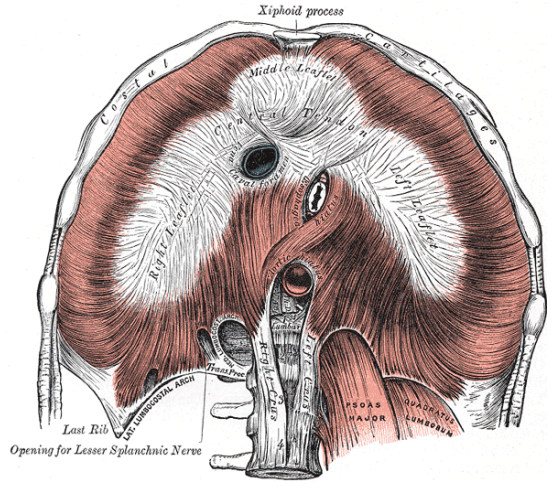
The classically cited insertion of the PM as a part of the IL at the lesser trochanter of the femur (Tortora & Grabowski, 1996) in most textbooks is an oversimplification which limited the

biomechanical classification of PM to a hip flexor for several decades. The posterior fascicles of the psoas attach directly to the pelvic brim and it's inferomedial fascia stemming from the lower lumbar vertebrae is continuous with the fascia of the pelvic floor (Gray, 2009; Gray's Anatomy E-Book Loc 7391 of 8322 "Iliac Region"; Myers & Garcia, 2021) This forms a connection with the musculature of the pelvic floor, transversus abdominus, and the internal obliques (Myers, 1996; Sajko & Stuber, 2009b).

As shown in Figure 2, the most proximal attachment of the PM connects to the diaphragm via the medial arcuate ligament (Cai et al., 2013; Gray et al., 1974 p. 352). The diaphragm becomes tendonous on either side of the aorta transitioning to the right and left crus, the attachment points on the spine at L1through L3, which merge to become the anterior longitudinal ligament moving caudally to the pelvic basin. (Gray, 1974 p.353).

Figure 2

The Diaphragm Under surface (Gray, 1974)



The importance of the attachment point of the PM and diaphragm at L2 on respiratory function is currently under investigation by Vats et al., (2021). Their study tested the efficacy of releasing

tension in the psoas muscle to improve respiratory efficiency in patients with nsCLBP, however results have not yet been published. Because the direction of force production in the PM is in the same plane but opposite direction as the posterior inferior fibres of the diaphragm, tension in the PM can limit the pliability of the diaphragm during an inhalation (Bordoni & Zanier, 2013; Vats et al., 2021). The fascial continuity of the PM, crus of the diaphragm, anterior longitudinal ligament and pelvic basin position the PM to exert influence on the balance of the dynamics of respiratory function and the functional relationship between the diaphragm and pelvic floor (Siccardi et al., 2021).

Singh and Al Khalili (2022) document the lumbar plexus as a web of somatic peripheral nerves exiting the first three lumbar vertebrae, innervating primarily the lower limbs, in addition to the PM, quadratus lumborum, internal obliques and transversus abdominis muscles and lies within the PM, beginning at the 12th thoracic vertebrae. According to Gray (2009), the lumbar sympathetic nervous filaments pass beneath tendonous arches between the vertebrae and the PM in order to protect the nerve fibres from pressure during the contraction of the muscle. However, the dissection of 54 cadavers by Feigl et al. (2013) recorded the lumbar sympathetic trunk's mean distance to the PM was 0.3mm at L2/L3 and at its origin was observed directly within the fibres of PM, which was also reported in earlier medical dissection research (Rocco et al., 1995).

The kidneys are located superior to each side of PM and the posterior layer of the renal fascia extends medialward behind the kidney and blends with the fascia on the quadratus lumborum (QL) and the PM (Raptopoulos et al., 1986; Sim & Webb, 2004). On the medial aspect of PM are the lumbar arteries, the sympathetic ganglia and its communicating branches with the spinal nerves, the vena cava on the right and the aorta on the left (Feigl et al., 2013; Kanemura et al., 2017).

The anatomical research is clear - there are several fascial connections between the PM and adjacent structures, including visceral organs that play a role in physiological stress response (Kanemura et al., 2017).

The PM muscle does not work alone to produce movement in or stabilize the lumbar spine. Also involved are: posteriorly the quadratus lumborum, multifidi and erector spinae; anteriolaterally the internal and external oblique abdominal muscles and the transversus abdominis; anteriorly the rectus abdominis. Researchers continue to disagree on the PM and subsequently the IP's involvement in lumbar spine flexion, extension and lateral flexion, as well as hip rotation, adduction and abduction (Bogduk et al., 1992; Gray, 2009; Koch, 2012; Santaguida & McGill, 1995; Yoshio et al., 2002). However, most researchers agree in its involvement in flexion of the hip joint and lumbar spine stabilization (Santaguida & McGill, 1995; Yoshio et al., 2002), which led to the development of the Thomas Test in assessing tightness in IP (Kendall et al., 1993).

2.3.1 Lower Back Pain and The Psoas Major Muscle

The PM is connected to the spine and as such, elicits force on the spine in a variety of vectors (Santaguida & McGill, 1995), making it an area of interest when assessing biomechanical mechanisms of LBP. Asymmetry between the right and left PM can create a tensional force on the spine or unilaterally on the pelvis to disrupt lumbopelvic alignment, resulting in a chronic muscle tension created by compensatory muscle activity. Bilateral tension can apply force on the femur to decrease available hip extension range of motion, in addition to creating excessive compressive forces on the lumbar spine leading to pain (Cholewicki & McGill, 1996). One area of study that presents noteworthy findings is that of the examination of the cross-sectional area (CSA) of the PM and implications for nsCLBP.

Dangaria and Naesh (1998) used MRI to determine the CSA of PM in patients with unilateral sciatica caused by lumbar disc herniation and compared to asymptomatic volunteers. They found ipsilateral (same side) decreased CSA in PM in the patients but no difference in PM CSA on left and ride side in the volunteers. Although this study implied that the herniation causes the ipsilateral PM atrophy, the authors also suggested that it may not be atrophy at all, but compensatory hypertrophy of the opposite side.

Barker et al. (2004) also used MRI to measure CSA symmetry of the PM. However, their study focused on patients with nsCLBP instead of herniations and they also included measures of CSA of the multifidus. examined the relationship between atrophy in the PM in addition to the multifidus with symptoms of unilateral nsCLBP. In the 48 patients with unilateral nsCLBP, an imbalance in cross sectional area at different levels of the PM attachment to the spine was present. The decrease in PM and multifidus CSA at each L1-L5 spinal level coincided with pain at that level of the spine on the same side. That is, when a significant decrease in PM and the multifidus CSA at L1 was measured, pain had been reported at the L1 spinal level on the same side. They concluded that there is selective ipsilateral atrophy of PM and multifidus in the presence of unilateral nsCLBP. A flaw in their study is they only measured the CSA on the side of pain, so although there was a decrease in PM at the level of pain, it is possible that the non-symptomatic side may have shown hypertrophy at those spinal levels. The later work by Seyedhoseinpoor et al. (2022)measured CSA using MRI in PM, multifidus and erector spinae on both sides of the spine at L2-L3 and L5-S1. Their results found that only asymmetry in PM was a significant factor in patients with nsCLBP.

Atrophy can happen for number of different reasons, and although these studies postulate that the pain is causing the atrophy, it is also possible that the imbalance in size and subsequent

force that can be elicited on the spine by a larger muscle (i.e., larger CSA), may be a contributor to the pain. The 2013 study by Arbanas et al. showed evidence of this. They compared analyses of MRI measurements of PM CSA at different spinal levels of 42 nsCLBP patients to 49 controls. Patients measured larger PM CSA at L3-L4 and L4-L5 when compared to the controls. In contrast to the previously mentioned studies on unilateral pain, Arbanas et al. did not measure a significant difference in the CSA between sides in nsCLBP patients. Instead, they suggested an increased activity of the PM in the presence of nsCLBP. None of the studies measuring PM CSA in the presence of back pain noted the duration of pain or intensity of pain in subjects, which could account for the contrasting results. Arbanas et al. also did not report on whether the pain present in patients was unilateral or not. The current study can draw insights from these studies as the cross-sectional area (CSA) of a muscle is proportional to its overall size, and thus determines its ability to generate force (Derrickson, 2017; p.370). A muscle with a larger CSA has a greater potential to produce and retain tension.

Another important area of literature relevant in examining the impact of tightness of the PM on nsCLBP is the exploration of myofascial trigger points. Felt as knots, or areas of muscle hardness, myofascial trigger points are hypersensitive points located in skeletal muscle or fascia which cause local or referred pain, and autonomic changes when compressed (Celik & Mutlu, 2013). Active trigger points produce constant local or referred pain, where latent trigger points are only painful when they are compressed (Celik & Multu, 2013). Latent trigger points, however, can become active in the presence of stress (Iglesias-González et al., 2013). In their cross-sectional study involving 42 patients with nsCLBP, Iglesias-González et al. did not find a significant difference in active trigger points in the PM in patients with LBP when compared to pain-free individuals. However latent trigger points, which can lead to restricted range of motion

and alternating muscle activation patterns (Celik & Mutlu, 2013) were the highest in PM out of any other spinal or hip muscle tested and were significantly higher in LBP group compared to pain-free. Both active and latent trigger points have been shown to have increased metabolites in the surrounding tissue, Shah and Gilliams (2008) which makes it plausible to say that myofascial trigger points can activate the metaboreflex, triggering an increase in sympathetic activity. Consequently, the increased number of latent trigger points in PM in individuals with LBP (Iglesias-González et al., 2013) increases the opportunity for activation of the sympathetic nervous system.

An earlier study by Ingber (1989) reported on seven case studies of patients with nsCLBP All seven had tried multiple different treatments including chiropractic, physiotherapy, bed rest and anti-inflammatories to ease their pain. After bilateral trigger point therapy with dry needling to the IP motor area, all seven reported a significant increase in functionality, decrease in tenderness on palpation and decrease in back pain. In addition, there was an immediate change in hip extension ability after the therapy. The study did not however quantify hip extension angle or the longevity of results.

When a tight IP tendon impinges on the femoral head and anterior hip capsule, it causes pain called IP tendinopathy (Zhu et al., 2020). Individuals who exhibit IP tendinopathy often have accompanied LBP. In a study involving 45 patients with IP, 41 of whom also had LBP, Zhu et al. (2020) injected anesthetic and corticosteroids into the fascial trigger point of the IP tendon. Ten minutes following the treatment, reported pain fell from 7.7 to 2.6 on the Numerical Rating Scale (1-10) for back and hip pain. After one year, pain was down to 0.75 one year later, and 38 of the 41 patients reported absolute pain relief. Using the Harris Hip Score, a validated measure of hip pain and function, patients had a 50% improvement in both hip pain and function

after one year. The study, however, did not report on any treatments that may have been received in the year, which could have influenced results, nor was a control or placebo group used.

In clinical practice, evidence connecting nsCLBP and unfavorable biomechanics (i.e., increased anterior pelvic tilt, increased lumbar lordosis and limitations in hip extension) that can be linked to increased PM tension can be seen in the success of fascial release and other somatic interventions that focus on releasing tension from PM and improving biomechanics.

Avrahami and Potvin (2014) used manual fascial-muscular lengthening therapy of the IP on two groups of male varsity athletes: athletes with tight hip flexors (THF) and nsCLBP and athletes with tight hip flexors (THF), but no pain. Hip flexor tightness was defined as an angle greater than zero degrees (equivalent to a negative angle in the current study) in the Modified Thomas Test, measured with a goniometer. Maximum voluntary trunk flexion and extension and passive hip extension range of motion were measured pre and post treatment protocol. Manual fascial muscular lengthening (also known as Active Release Therapy®) was applied to the IP in five sessions over two weeks. Both groups showed a significant increase in hip extension (13.1 degrees) and the nsCLBP-THF group showed a significant decrease in pain (as measured by 10-cm Visual Analogue Scale) following the protocol. The increase in hip extension in the nsCLBP-THF group was significantly greater than that of the other group. A limitation of this study was the short duration of only two weeks and lack of control group.

Research has identified tightness in the anterior hip as a mechanism of nsCLBP (Roach et al., 2015). In their examination of a variety of biomechanical variables in patients with nsCLBP, Youdas et al. (2000) concluded that it is more likely tightness in the hip flexors and not lack of abdominal strength that creates anterior pelvic tilting seen in patients with nsCLBP. Kitamura et

al. (2019) studied swimmers with lower back pain and found that when in a prone swimming position, their hip extension was limited and elastic modulus, or stiffness (Ateş et al., 2015) in the PM was higher than in those without nsCLBP. Although there is a body of evidence that reports chronic hip flexor tightness, is a symptom of nsCLBP, studies have not specifically reported on or controlled for psychological stress variables.

When considering the role of PM in nsCLBP, it's important to consider not only its impact on creating dysfunctional biomechanics, but also its potential physiological effects. Included in the effects of nociceptor activation are: local muscle hypertonicity, increase in sympathetic nerve activation, increase of neuroendocrine stress response and changes in systemic visceral activity (Dubin & Patapoutian, 2010). An increased cross-sectional area of the PM, as seen in patients with nsCLBP (Arbanas et al., 2013) increases the potential for mechanical pressure to be elicited on adjacent organs and tissues and stimulate a nociceptor response. Latent and active trigger points in skeletal muscle have been shown to have an increase of biochemicals in the surrounding tissue (Shah & Gilliams, 2008) which are stimuli for c-fibre nociceptor depolarization. The anatomy of the PM previously described explains the unique position of this muscle in its relationship with the diaphragm, vertebral column, kidneys and nerves of the lumbar spine and its connection to hip extension and back pain. The literature in this section provides evidence of the involvement PM in nsCLBP, which will be explored in the current study in the use of the Modified Thomas Test in measuring hip extension range of motion, which is limited by tension in the PM, in individuals with nsCLBP compared to those without pain.

2.3.2 Measuring Iliopsoas Tightness

Due to the controversy in the actions of the PM and the complexity of its anatomy, designing a reliable protocol for assessing tension across the structure has been challenging in

biomechanics. Surface and intramuscular electromyography (EMG) are reliable tools for measuring electrical impulses of a muscle's motor units, and thus muscle load and its associated tension (Roman-Liu et al., 2013). However, intramuscular EMG is invasive and due to the depth of the PM at the proximal end, accuracy in needle placement is problematic, and a posterior entrance in the lumbar area creates great discomfort (Nachemson, 1966), therefore, it was not realistic within the realm of the current study. Although one study, (Mcgill et al., 1996) concluded that accurately placed surface EMG can detect deep tissue activation in a muscle like PM, the results have yet to be accepted as a mode of measuring PM tension in research. The research by McGill et al. (1966) only indicated validity in comparing electrical activity with load placed on muscle due to body position, which is not applicable in the current study.

The manual assessment of muscle length through specific range of motion tests is a foundational practice in physical medicine. Specific orthopaedic tests have been designed to determine whether a certain muscle is too short or has decreased extensibility or too long with excessive extensibility (Kendall et al., 1993; p. 5). Extensibility of the muscle is inversely proportional to its tension which is a function of muscular contraction, myofascial and tendon density, muscle fibre length and neural activity, this has also been called tissue flexibility (Ferber et al., 2010), as previously discussed. In clinical practice, a simple positive or negative score, without quantitative measure is typically used in the assessment of biomechanics. However, to be more specific, muscle length tests provide objective data using measurement tools, such as a goniometer, tape measure or inclinometer (Kendall et al., 1993). The test for hip flexor length, or tight hip flexor muscles is the Modified Thomas Test. Specifically, within clinical protocol, Kendall et al. (1993) describe the use of the Thomas Test for IP length, where a smaller hip extension angle refers to increased tension or tightness in the IP.

Adductor longus and brevis, pectineus, iliacus (IL), psoas major (PM), rectus femoris (RF), tensor fascia latae (TFL), sartorius, gracilis and the anterior fibres of gluteus minimus are all part of the hip flexor group of muscles (Martini et al., 2012; Neumann, 2010). The primary hip flexors acting predominantly in the sagittal plane are IL and PM, together the iliopsoas (IP), and RF. All other hip flexors also to act pull the femur into adduction, abduction or rotational movements while flexing the hip (Martini et al., 2012; Neumann, 2010). The RF is a two-joint hip flexor that also extends the knee when contracted. As explained in section 2.3, the IL is a single-joint hip flexor which can work with or independently from the PM.

Although currently the most reliable manual test for measuring extensibility in hip flexors, some doubt remains in the Modified Thomas Test's ability to isolate the PM or IP. Overall, the literature shows varied results on the test's reliability, however intra-rater reliability is very strong when key criteria during measurement are met. Peeler and Anderson (2007) measured 54 individuals to test the inter and intra rater reliability of the Thomas Test in measuring IP flexibility. From their study, which used three different experimenters, Peeler and Anderson concluded that the test should be administered by the same tester to ensure consistency in landmarking. In their study, they used the fibular head instead of the lateral condyle of the femur for the distal landmark in an attempt to improve consistency between testers. The standard, and the protocol used in all other known studies, however, is to mark the lateral condyle of the femur, which shows high intra-rater reliability provided the landmarking is all done by the same person and that the person is experienced in palpation (Ferber et al., 2010). Although their study did show high reliability between trigonometric calculations and the goniometer, Peeler & Anderson identified challenges with using the goniometer to measure hip angle during the Thomas Test, including the lack of identifiable landmark above the hip joint for

the stationary arm, difficulty in aligning the axis of rotation of the tool with the landmark for the hip joint and difficulty in maintaining alignment of the tool with the distal long lever landmark. Peeler & Anderson did not allow the leg being tested to pass the plane of the table, and instead had the individual fully supported by the table. Their methodology created difficulty in placement of the goniometer axis at the greater trochanter mark (hip joint), however is easily overcome when the test is administered with the thigh being able to pass the plane of the table (Ferber et al., 2010; Vigotsky et al., 2016). Although there is no standard landmark superior to the hip for the stationary arm of the goniometer, an experienced tester can easily palpate the iliac crest and identify the lateral line of the pelvis if pelvic tilt is controlled, something not done by Peeler & Anderson (Vigotsky et al., 2016). Aligning the distal arm accurately can be performed by using an additional marker on the leg following the straight line between the greater trochanter and the lateral condyle of the femur, or by adding length to the arm of the goniometer or another means to ensure a straight line is followed.

Evidence of the reliability of using a goniometer has been provided by studies that have compared goniometry with other digital measurements. The clinical observational study by Clapis et al. (2008) measured the reliability of the Thomas Test using a goniometer and inclinometer. This was the first study of its kind to determine a high correlation between measurements using the two instruments, in addition to showing high inter-rater reliability. Although only pain-free subjects were used in this study, the study did an excellent job at explaining the standardized protocol for the Thomas Test and the use of each measurement tool, in order to limit tester error (Clapis et al., 2008). Roach et al. (2013), however did find a significant difference in using the goniometer and inclinometer in all measures of hip range of motion and concluded the two tools should not be used interchangeably. Fraeulin et al. (2020)

aimed to test inter and intra rater reliability using motion capture, tape measure and inclinometer. Their study showed very high, almost perfect reproducibility of measurements when the pelvis was checked for a zero (sacrum on table) angle between measurement tools and testers. They did caution using an inclinometer placed on the anterior surface of the thigh as the angle could be changed by the amount of subcutaneous fat. This was not, however a limitation of their study as all participants had low BMI values. The conflicting results in the Clapis et al. and the Roach et al. studies could be due to this difference in methodology. Clapis et al. used a straight line to connect the landmarks on the femur and placed the inclinometer in alignment with it, whereas Roach et al. placed the inclinometer on the anterior mid-thigh. Roach et al. also reported doubting their success of stabilizing the pelvis during the test, which has been provided as a necessary part of the protocol to enhance the test's reliability (Roach et al., 2013; Vigotsky et al., 2016) .

A 2016 study by Vigotsky et al. which aimed to measure criterion reference validity identified that proper measurement of hip extension, or angle of flexion with the Thomas Test required the measurement to be taken in relationship to the pelvis and not the table. The results revealed that when anterior pelvic tilt and lumbar lordosis were corrected, there was high criterion reference validity. They also concluded that the stabilization of the pelvis in a zero position was necessary for the measurement to be valid. Because the IL does not attach to the lumbar vertebrae and is therefore unable to elicit direct force on the spine, Vigostky et al. (2016) added that for this reason, goniometry using the lateral line of the pelvis as the stationary arm landmark is more likely to isolate the PM rather than include tension in the IP complex, as it accounts for the tension elicited on the pelvis by the lumbar spine. Preventing the thigh from rotating, abducting or adducting, will further isolate the PM by removing tensional contribution

from the TFL, gluteus minimus and adductors. In addition, maintaining a fixed flexed position of the knee lengthens the RF, limiting its ability to produce force at the hip and bring the hip into flexion, maximizing the isolation of IP in limiting hip extension during the Modified Thomas Test.

Regardless of some studies showing reporting high inter-rater reliability (Ferber et al., 2010), it remains important for the validity in the Thomas Test to be high, the assessment is performed by the same practitioner, and that the practitioner is experienced. (Vigotsky et al, 2016; Ferber et al. 2010).

With an aim of setting normative values in the Thomas Test, Ferber et al. (2010) measured hip extension in the Thomas Test in 300 recreational athletes using an inclinometer. They deemed a critical value for inflexibility of IP at -9.69 to -6.66 degrees and noted that the leg being measured must have the thigh free from the examination table to allow for full extension of the thigh. Moreside and McGill (2011) further attempted to set normative values for young males (18-35 years), using both 3D (motion capture) and 2D (goniometry) during the Modified Thomas Test. In addition to finding a high significant positive correlation between the two measurement computations ($r^2 = .99$, $p < .05$), and deeming goniometry a reliable measuring method, they recorded -8^{o} as the 50th percentile, but did not report mean scores. Although there seems to be agreement between the studies on the range of normal and between measurement tools, no clinically accepted normal ranges have been published.

2.4 Measuring Stress

2.4.1 Heart Rate Variability (HRV)

Based on the relationship between the autonomic nervous system and the cardiovascular system, measurement of HRV is a non-invasive method to analyze physiological and

psychological well-being (Kemp & Quintana, 2013; Kim et al., 2018). HRV is a measurement of the fluctuation of time intervals between adjacent heartbeats (Shaffer & Ginsberg, 2017), and more specifically the R-R interval (Task for of the European Society and the North American Society of Pacing and Electrophysiology, 1996; Thayer et al., 2010). Optimal variability, seen in a healthy heart, shows oscillations that are reflective of the body's ability to respond and adapt to a changing environment, or the individual's self-regulation capacity and resilience (Shaffer & Ginsberg, 2017).

HRV is controlled by the autonomic nervous system (ANS) and indicates the dynamic relationship between its parasympathetic (PSNS) and sympathetic (SNS) branches (Cysarz et al., 2015; Kim et al., 2018; Shaffer & Ginsberg, 2017). The sinoatrial (SA) node takes inputs from both the SNS and PSNS to produce a typical resting heart rate between 60-80bpm. PSNS input to the SA node via the Vagus X cranial nerve decreases heart rate by decreasing the action potential in the cells. The SNS makes SA node cells more excitable by the release of norepinephrine, resulting in faster repolarization and increased heart rate. With only SNS input, heart rate would be set at approximately 100bpm, however it is brought down by the PSNS and this adjustment by both inputs creates the inter-beat variability. At rest, the PSNS dominates, however it is withdrawn when HR needs to increase suddenly, as in an increased workload during exercise or increased arousal (Perini & Veicsteinas, 2003).

During normal stress response in a resilient individual, the SNS is stimulated and then PSNS rebound restores balance once the stressor is removed. If the ANS is imbalanced, and the SNS is hyperactive (sympathetic dominance) and PNS underactive, an individual's entire system is out of balance, making them more susceptible to disease, illness and premature aging (Thayer et al., 2010). This is of clinical importance in assessing cardiac health and cardiac related

mortality risk and can be seen with the proper analysis of HRV (Malik et al., 1996). As previously noted, an increased HRV is associated with several markers for psychological health and a decreased HRV has been shown to be associated with a hypoactive PSNS (Kemp & Quintana, 2013). The standard of methods, definition of physiological and pathophysiological correlates, and identification of clinical use was developed by a multidisciplinary task force assigned by the European Society of Cardiology and The North American Society of Pacing and Electrophysiology (1996).

2.4.1.1 Common Domains for Analyzing HRV

HRV is displayed in two domains: time (ms) and frequency (ms^2), with measurements recommended to be taken in short-term intervals of 5-minutes or 24-hour long-term intervals (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996; Munoz et al., 2015; Shaffer & Ginsberg, 2017). The time domain is typically used in longer-term analysis and the frequency domain used for short-term and is more common in research due to the ease of data collection under time constraints (Malik et al., 1996). The short-term measurements of 5-minute intervals focus on the relationship between SNS and PSNS and regulatory the mechanisms that control HR via respirator driven speeding and slowing of the heart via the vagus nerve (respiratory sinus arrhythmia - RSA), baroreceptor reflex and rhythmic changes in vascular tone (Shaffer & Ginsberg, 2017). Although long-term or 24-hour intervals are recommended for the use of the time domain, Munoz et al. (2015) found a high correlation between multiple very short measures, beginning at 10s and the standard 240-300s, indicating that the time domain has validity over shorter periods of time. Of dominant importance, as stated by the Task Force may not be the duration of the interval, but the

uniformity of the intervals for all samples, and the understanding that longer time periods will show higher accuracy and thus more power in clinical predictive strength (Malik et al., 1996).

The Frequency Domain

The frequency domain measures how much of each heartbeat lies within a frequency distribution of relative power on the ECG and can be separated into four bands: ultra-low frequency (ULF), very low frequency (VLF), low frequency (LF; absolute power of low frequency band 0.04-0.15 Hz) and high frequency (HF; absolute power of high frequency band 0.15-0.4 Hz) (Shaffer & Ginsberg, 2017). ULF and VLF are rarely used in research, however the body of literature on their utility is beginning to grow, relating to thermal stress and chronic inflammation and slow biological processes (Carney et al., 2007; Shaffer & Ginsberg, 2017).

VLF is recorded in minimum blocks 5 minutes, where LF is 2 or more minutes and HF is 1 minute or longer. As previously mentioned, the most heavily analyzed are LF and HF and the ratio of LF/HF, which provide a quantitative analysis of the SNS-PSNS balance (Shaffer & Ginsberg, 2017). Related efferent vagally mediated influences are present in LF when respiration rate is below 8.5bpm or 7s, and it is modulated by both the PSNS and SNS (Kim et al., 2018; Malik et al., 1996; Sayers, 1973). HF reflects PSNS activity and corresponds to HR variations caused by the RSA. Lower HF band power is correlated with higher stress, and therefore typically higher levels are recorded during the day, and lower levels at night (Shaffer & Ginsberg, 2017). When considering the LF/HF ratio, exactly what is being measures relies heavily on the measurement conditions (Shaffer & Ginsberg, 2017). LF/HF varies between 1.5-2.0 in a healthy adult, where a measure below 1.5 indicates and imbalance in favour of the PSNS and above 2.0 is indicative of perturbations favouring SNS activity (Perini & Veicsteinas, 2003; Shaffer & Ginsberg, 2017).

The Time Domain

The time domain is considered the gold standard measure due to its clinical predictive power when measured in 24-hour intervals (Shaffer & Ginsberg, 2017), however the frequency domain provides a more accurate overview of physiological functioning (Malik et al., 1996). Frequency domain analysis does, however, contribute to the understanding of autonomic background of R-R Interval fluctuations in the HR. Simple time domain variables can be calculated including the mean N-N interval (time between normal heart beats or each QRS complex; R-R interval), the mean HR, and the longest or the shortest heartbeat. Statistical time-domain measures can also be used for recordings over long periods to see fluctuations in circadian rhythms and other activities throughout the day, however they must be used with caution for short-term analysis due to decreased accuracy (Malik et al., 1996; Shaffer & Ginsberg, 2017). The standards of measurement are as follows:

- uniform measurement for a minimum of 5-minutes while stationary for the frequency domain and 18 hours with at least one overnight for time domain;
- consistent environment for each subject when doing comparing measurements between subjects;
- normative values must be taken as a function of age and sex, as they are primary variables to influence HR;
- standardized equipment should be used (Malik et al., 1996).

HRV decreases with age and is typically lower in females.

The Standard Deviation of the inter-beat intervals of normal sinus beats N-N interval (SDNN) is the statistical measurement of the variance between normal heart beats (Shaffer & Ginsberg, 2017). Both SNS and PSNS activity contribute to SDNN and it is highly correlated

with total power. Short-term resting variation is primarily sourced by the RSA, which is PSNS-mediated. SDNN is an accurate predictor of cardiac health when measured over a 24-hour period, where a value below 50ms is classified as unhealthy, 50-100ms as compromised health and above 100ms as healthy (Shaffer & Ginsberg, 2017). SDNN is the commonly used measure of HRV in many electronic wearable devices and has become considered a global measure of HRV, which is why it was included in the current study (Rodrigues et al., 2022) .

2.4.1.2 Equipment for Measuring HRV

In 1982, Polar released the world's first wireless HR monitor ("Polar's 40 Years of Incredible Firsts | Polar Blog," n.d.). Since that time, there has consistent advances in technology allowing individuals to measure various components of their biological function. To date, most lack peer-reviewed validation research comparing to the gold-standard ECG (Reeder & David, 2016), thus leaving their validity and accuracy questionable for use in scientific research. Despite this controversy, wearable heart monitors and smart watches are highly utilized in studies due to ease of use and cost-effectiveness.

The wearable Equivital EQ02 LifeMonitor is a two-lead chest strap with shoulder holster to hold the device in place for increased measurement reliability. This monitor uses ECG tracings to produce reliable and simple HR and HRV data. Different from mobile devices which may lack accuracy in targeting the cardiac cycle or produce data that may be highly dirtied with artifacts, the Equivital EQ02 LifeMonitor has been validated in research (Akintola et al., 2016; Liu et al., 2013).

When compared to the gold-standard ECG device, the Holter, the Equivital EQ02 LifeMonitor had correlations of 0.724, 0.955 and 0.997 for datasets containing all data and data with <50% or <20% artifacts respectively. Perhaps due to the ability of the Equivital EQ02

LifeMonitor to record respiration in addition to HR, it is able to mitigate artifacts potentially created by respiratory disturbance (Akintola et al., 2016). When compared with several other standard measuring devices, Liu et al. (2013) found there was no significant differences in HR and R-R scores recorded by the Equivital EQ02 LifeMonitor, deeming it clinically valid.

2.4.1.3 HRV and Health

As previously noted, HRV is an easy and non-invasive way to identify imbalances in the ANS. A high HRV (R-R interval) typically indicates better cardiovascular health and resilience to stress. Several studies have shown clinical relevance of using HRV to measure risk of disease and associated low HRV with a myriad of health conditions, many of which list stress as a risk factor. In psychophysiology, low HRV has been linked to mood disorders (Kemp & Quintana, 2013), depression (Carney et al., 2007), PTSD (Schuman & Killian, 2019; Tan et al., 2011) and anxiety (Chalmers et al., 2014).

The Framingham Heart Study, a longitudinal study spanning over 70 years discovered that health behaviours, which are often linked to stress, such as alcohol consumption, smoking and inactivity were related to lower HRV in cardiac patients. In the same study, low HRV related to all-cause mortality (Tsuji et al., 1994). HRV is consistently correlated with CVD and used as a mortality predictor in a clinical setting (Greiser et al., 2009; Hillebrand et al., 2013). Thayer et al. (2010) found that low HRV increases the risk of a cardiovascular event by 32-45% in persons without CVD history.

Lower HRV has been measured in patients with chronic pain, including during sleep which indicates reduced PSNS activation and increased SNS, and thus pointing to stress as an element of chronic muscle pain (Hallman & Lyskov, 2012). This reduced HRV has been

specified as a reduced RSA in patients with nsCLBP (Prim et al., 2019), indicating a decreased cognitive control over psychological events.

2.4.2 Cortisol

The evaluation of biological compounds in saliva, which are produced as a result of the physiological stress response, is an effective non-invasive method of collecting data on the state of an individual's system. Specifically, the concentration of cortisol in the saliva reflects activity of the hypothalamus-pituitary-adrenal axis (HPA-axis) (Raff et al. ,2002; Vining et al., 1983). On direction from the central nervous system (CNS), the hypothalamus detects stress and signals the pituitary gland to release adrenocorticotropic hormone (ACTH) to the adrenal cortex which release cortisol (Lightman, 2008). Upon waking, cortisol levels are at their highest, for approximately 30 min (cortisol awakening response – CAR), and cycle on a diurnal pattern throughout the day (Kumari et al., 2011). After the peak at waking, cortisol levels gradually decrease throughout the day (Kumari et al., 2011; Norman et al., 2014; Ryan et al., 2016). Salivary levels of cortisol are directly proportional to blood serum levels and may even be a better measure (Vining et al., 1983). Collection by passive drool is a simple and non-invasive collection measure and is best taken prior to 11am if comparing to normative values.

2.4.2.1 Cortisol and Health

Many recent studies show that patients with chronic back pain have higher cortisol levels than control subjects (Vachon-Presseau et al., 2013). Cortisol has successfully been used a marker of stress in several intervention studies using body therapies as a form of stress management (Pascoe et al., 2017; Pinar et al., 2015). One study by Sveinsdottir et al. (2016) using salivary cortisol collection, showed a higher CAR and slower daytime decline in patients with CLBP, indicating that CLBP and stress are related. Consistent increased levels of free

cortisol have been recorded in patients with CVD and diabetes (Joseph & Golden, 2017; Whitworth et al., 2005), further illustrating stress as a risk factor for these conditions.

2.5 Summary

The above review of literature provides evidence of the stress-pain cycle, the identification of muscle tension or tightness as both a source and cause of pain sensation, and stress as an instigator and symptom of muscle tension. Although tightness in the hip flexors has been identified as a potential contributor to nsCLBP, no other study has compared the relationship between tightness in the hip flexors and stress in individuals who report nsCLBP and those who do not. The current study aims to investigate the relationship between stress and muscle tension by focussing on the IP, which has a unique anatomical location, as a potential primary contributor to back pain.

Chapter 3: Methods

Approval for this study was obtained from the University of Victoria Human Research Ethics Board and thus meets the ethical standards required by Canadian universities and National regulatory bodies for in-person research involving human participants (Appendix A).

3.1 Study Design

This is a single test, cross-sectional observational design.

3.2 Participants

This study used 40 volunteer participants overall. The group included 19 individuals who reported experience of nsCLBP for a period of at least 3 months in the previous 12 months, and 21 individuals who reported no nsCLBP in the previous 12 months. For between-group statistical comparisons, two subgroups (nsCLBP and noPain) were created from the overall participant group with 17 individuals each. The sections below fully describe how volunteers were recruited, selected and grouped for the study (Figure 3).

3.2.1 Recruitment

Volunteers were obtained through snowball recruitment by means of email to the researcher's personal and professional contacts, yoga studios in Victoria BC, and the University of Victoria current graduate student database (Appendix B). In addition, recruitment posters (Appendix B) were placed at chiropractors and yoga studios in Victoria BC, and within the McKinnon Building at the University of Victoria. Social media posts on Instagram and Facebook were made by the researcher on their personal and professional pages (Appendix C). Eighty-nine individuals (F=60, M=29; nsCLBP=30, noPain=59), ranging in age from 18-72 years old, completed the pre-screening questionnaire (Appendix D).

3.2.2 Sample

Only adults aged 18-65 were eligible to participate. Due to the influence some disorders have on HRV and cortisol measures (see section 2.1), potential participants were excluded if they reported a prior or current diagnosis of an anxiety or stress-disorder, diabetes, CVD, or if they were pregnant or breast feeding. In addition, those with musculoskeletal back or hip conditions were also excluded. Any individuals who indicated they would not be comfortable laying on their back for a period of 15 minutes without pain were also excluded. Of the 89 participants who completed the pre-screening, 15 were deemed ineligible.

The process of participant selection (Figure 3) was intended to increase homogeneity between the final nsCLBP group and noPain group, and to limit the influence of age and sex in between group comparative analyses of heart rate variability, passive hip extension and cortisol concentration. The 74 eligible participants were categorized as either eligible for the noPain (n=50) or nsCLBP (n=24) group as indicated in their pre-screening questionnaire. Each of these preliminary groups were then sorted by biological sex. All eligible participants in the nsCLBP category were invited to participate first. They were sent the Letter of Information for Implied Consent (Appendix E) explaining the protocols, a copy of the pre-test protocol (Appendix F) and sent a link to self-select an available 45-minute time slot through an online appointment calendar. These volunteers were given 10 days to book an appointment in an available 4-week time period. In addition, they needed to confirm they had received the letter and could adhere to the pre-test guidelines. Nineteen volunteers (M = 10, F = 9) booked appointments through this process and made up the participants in the overall group who reported experience of nsCLBP group. These individuals were sorted by biological sex, then each sex group was organized by age. The remaining 50 volunteers (noPain category) were also sorted by biological sex and age.

A preliminary matching by sex and closeness in age (± 6 yrs) was made between the nsCLBP participants and the remaining volunteers. Any duplicate age-sex matches were paired by random assignment. Of the 50 remaining volunteers, 19 were matched with the nsCLBP participants and contacted to participate. Six of these volunteers were removed as unresponsive or voluntarily withdrew from the study. Remaining noPain volunteers were contacted to participate following the same protocol, with closest matching until the group was full. Forty participants formed the overall sample group (n = 40; M = 18, F = 22), twenty-one of those participants (M = 8, F =13) were categorized as "noPain".

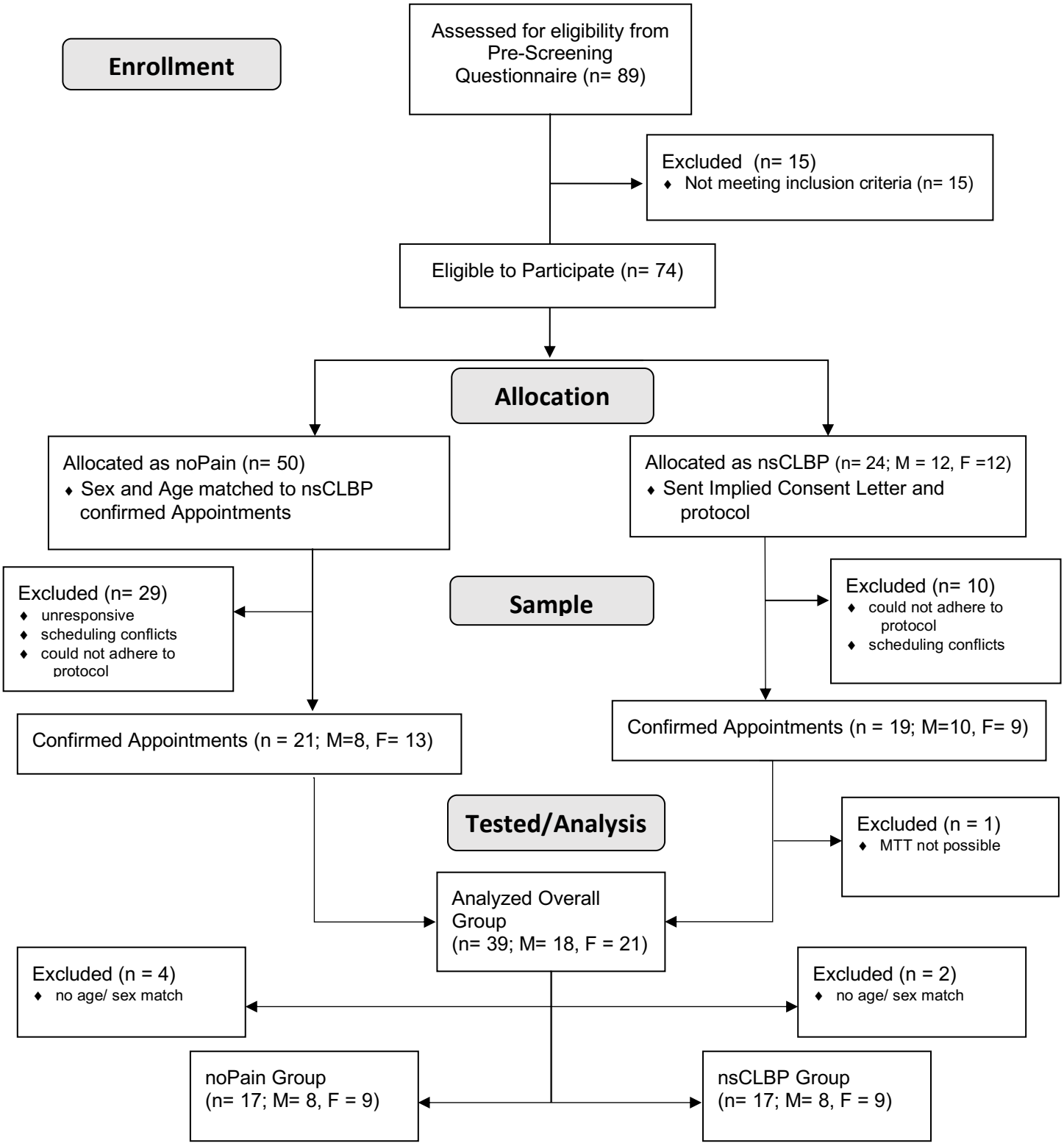
From the overall sample group, further refined nsCLBP and noPain final subgroups were formed to create the largest groups possible with an equal number of males and females who age-matched. Each subgroup had 17 individuals (M = 8, F = 9). An independent t-test, including Levene's test for homogeneity of variance, was performed to determine age homogeneity between the nsCLBP and noPain groups.

To ensure physical activity levels had limited influence on between-group comparisons, questionnaires were reviewed to confirm there was no large disparity in activity level between the groups. Participants reported participation in an activity if they participated in it a minimum of twice per week on a consistent basis. Each activity selected was given a score of one, then all activity scores were summed to create a cumulative activity score. Disparity was considered a score of 0. No participants were eliminated due to physical activity disparity. A two-tailed independent samples t-test was performed to compare participation in each physical activity between the groups. A Fisher's exact test was performed to test association between participation in each activity and nsCLBP. Prior to arriving to the testing location, all participants performed

a self-check for symptoms of COVID-19. The question-based health check was performed again verbally by the lead researcher upon arrival for testing.

Figure 3

Flowchart of Participants



3.3 Data Collection Procedures

Participants self-registered for one 45-minute time slot that fell within 2 hours of normal waking time and was between 6 am and 10am. Each participant had all variables measured in a single visit, at the same location (Ashtanga Yoga Victoria). The testing room was private, temperature-controlled at 21^{o} Celsius, quiet and without interruption with just the participant and lead researcher present. All participants were measured within one 6-week period. In the pre-test guidelines, participants were asked to refrain from consuming alcohol or using recreational drugs for 24 hours prior to their appointment, to refrain from any form of physical activity that was more than 2 blocks of walking to the appointment, and to avoid consumption of caffeine or other stimulants the morning of their appointment. In addition, they were asked to avoid eating or brushing their teeth within 30 minutes of their scheduled time. Upon arrival for testing, all participants confirmed to following the pre-test requirements. Before beginning to take measurements, the researcher confirmed that answers provided in the participant's pre-screening questionnaire were still valid and answered truthfully, in addition to ensuring waking time was less than 2 hours prior.

3.3.1 Testing Protocol

1. Participants were asked to rate their current sense of life stress level (Rate of Perceived Stress; RP) on an unvalidated 9-point Likert scale. Participants in the nsCLBP group were asked to rate their current pain level on an unvalidated 9-point Likert scale. Ratings were recorded on the data sheet as a number from 1-9.
2. Participants were fitted with an Equivital EQ02 Life Monitor for HRV measurements. The order of the data collection procedures was explained and is detailed below.

3. **Saliva Sample Collection:** A 1.8mL saliva sample was collected for analysis of baseline morning cortisol levels via passive drool according to the Salimetrics Immunoassay protocols for cortisol (Salimetrics, 2021b). The sample collection tube was pre-labelled with the participant's unique alphanumeric identifier, which was also noted on the data collection sheet. The individual was instructed to fill the 1.8mL collection tube with their saliva. The individual capped the sample tube and placed it in a Ziploc bag, which was also pre-labelled with the participant's identifier and placed that bag into a cooler with ice blocks. The samples were held on ice for a period of no longer than 4 hours before being transferred to a freezer where they were stored at -20^{o}C for no longer than 10 weeks while awaiting subsequent analysis (Garde & Hansen, 2009). Assays were performed on the saliva at a Biosafety Level 2 certified laboratory at the University of Victoria following the Salimetrics expanded range high sensitivity enzyme immunoassay protocol (Salimetrics, 2021b).
4. Prior to the Modified Thomas Test, the Equivital EQ02 Sensor Electronics Module (SEM) was removed from the participant's Equivital EQ02 Life Monitor harness, and its contents read through the computer software to ensure the device was functioning and recording properly. These data were not saved or used in any statistical analysis. The SEM was then returned into the harness. Any adjustments required due to lack of recording were made and the device retested.

Modified Thomas Test: The participant was familiarized with the procedures for the Modified Thomas Test. Manual palpation of the relevant landmarks was performed by the lead researcher, who has 20 years of landmark palpation experience, to identify the location of the greater trochanter and lateral femoral condyle. To

landmark the femur, adhesive markers were placed on the greater trochanter and lateral femoral condyle of the participant's right and left legs. The participant lay supine on a firm treatment table, with both knees bent, the hip joint at approximately 135-degrees and feet supported by a step. The participant was instructed to feel the back of their pelvis and lower back on the table, but not their tailbone. This positioning was monitored by the researcher by visually checking the greater trochanter marker, which lined up just past the end of the table, and the gluteal fold being at the edge of the table. This ensured the table did not impede available extension of the femur at the hip during the test. The researcher placed their hands under the participant's lumbar spine, and asked the participant to press into their hand, bringing the pelvis to neutral while the feet remained supported by the step. This was to ensure the participant possessed enough strength in the abdomen to help prevent anterior tilting of the pelvis and increasing lumbar lordotic curve.

The test was performed twice on each side and measured each time with a goniometer. To measure passive hip extension angle, the participant was instructed to pull the opposite knee to their chest, holding it with their hands and lift their shoulders away from the bench, as shown in Figure 4. The researcher then removed the step from under the primary leg and placed the fulcrum of the goniometer at the center of the greater trochanter adhesive marker. The stationary arm was lined up with the lateral midline of the pelvis (0^{o}) and the movement arm moved to line up with the lateral midline of the femur as indicated by the lateral epicondyle marker. The participant was in the position less than 10 seconds for each test on each side.

Goniometer measurements less than 180º were recorded as negative angles. For example, a measurement of 160º on the movement arm was recorded at -20º.

Figure 4

Modified Thomas Test

Note. This image was taken during pre-study reliability testing using an inclinometer. It is shown here to demonstrate the positioning of the individual during the modified Thomas test.

This procedure was repeated for the second leg, then again for a second time on each leg. For all participants, the left side was measured first. The participant's body position was checked between each test. During the test, the researcher visually monitored for adduction or abduction of the thigh and knee extension, verbally instructing the participant to make necessary corrections to remain in the sagittal plane with the quadriceps muscles relaxed. These control procedures were aimed to capture a hip extension measurement that was limited by the iliopsoas and not the tensor fasciae latae, the hip adductor muscles, or the rectus femoris.

5. **Resting HRV**: Immediately following the Modified Thomas Test, participants were positioned supine on a mat on the floor for a single 12- minutes duration to collect HRV data via the SEM. Blankets were available to ensure comfort, the lights were dimmed, mobile devices silenced and there were no interruptions for any participants. Participants were instructed to lay with their arms on the floor and to remain as still as possible for the full 12 minutes. This time frame allowed for a 5-minute wash-out period and the recommended 5-minute segment, from 00:05:00 – 00:10:00, for analyses (Shaffer & Ginsberg, 2017a). The data were computed through Kubios software.
6. At 12 minutes, participants were informed the protocol had been completed.
7. In the time period between participants, approximately 15 minutes, the data were extracted from the SEM and saved to an excel file labeled with the participant's alphanumeric identifier code for later analysis.

Table 1 includes the instruments used in the study procedures. Validity and reliability of these instruments has been documented and discussed in Chapter 2.

3.4 Statistical Analyses

All statistical analyses were completed using IBM SPSS Statistics Version 28.0.0.0. Initial descriptive statistical analyses were performed for each variable. T-tests were performed to determine if any significant differences in age between the sexes existed for each group. This was done to ensure age or sex differences had limited effect on HRV, HE and CORT data.

For clarity, the data analyses procedures are separated below by each research question. In addition, some post-hoc supplemental analyses were performed to support thorough discussion of the results and are presented in Appendix G. Missing data were handled on a case-by-case basis, dependant on the appropriate method for the type of data (Chapter 4). Outliers were removed from the analysis for that variable. All statistical testing was conducted with a significance level set at $\alpha = 0.05$, therefore when $p \leq 0.05$, the research hypotheses as stated in Chapter 1 are accepted.

Table 1

Study Instruments and Measures

Variable	Instrument & Test Name	Measurement
Salivary Stress Substance	Salimetrics ELISA Immunoassay	cortisol concentration in mg/dL converted to nmol/L (CORT)
Heart Rate Variability (HRV)	Equivital EQ02 Life Monitor	Low Frequency power in ms^2 (LF)
	Kubios HRV software for data conversion	High Frequency power in ms^2 (HF)
		LF/HF Ratio (LF/HF)
		Standard Deviation of normal-to-normal R-R-Intervals in ms (SDNN)
Iliopsoas Tightness (IPt)	Modified Thomas Test	Passive Hip Extension Left, Right (HE R, HE L)
Passive Hip Extension (HE)	Medical 12” goniometer	Degrees of femur extension at the hip, relative to lateral line of pelvis
Non-specific Low Back Pain	Pre-Screening Questionnaire	Presence of low back pain (nsCLBP or noPain)
Perceived Stress (Stress RP)	Verbal Self-Report	Likert Scale 1-9

3.4.1 Research Question 1

What is the relationship between iliopsoas tightness (IPt) as measured by hip extension angle (HE), and physiological stress as measured by Heart Rate Variability (HRV) in an adult population?

Because each measure for HE was taken twice, the mean value for each leg and was used in the relevant analysis. For example: $\text{HE L} = \frac{\text{HE L}_1 + \text{HE L}_2}{2}$

To determine the relationship between HE and HRV a Pearson bivariate correlation analysis was performed with data from the overall group (n = 39). Table 2 displays the variables that were measured and used for the correlation analysis. Next, to further explore the functionality of any measured relationship, and again using the data from the overall group (n = 39), a simple linear regression analysis was performed to determine how HE predicts changes in HRV. Age and sex were added as independent variables in a multivariate regression model to test significance of any confounding effects of age and sex.

Table 2

Variables used in Statistical Analyses for HRV – HE Relationship Determination

HRV	Hip Extension
LF	HE L
HF	HE R
LF/HF	
SDNN	

3.4.2 Research Question 2

What is the relationship between iliopsoas tightness (IPt) as measure by hip extension angle (HE), and physiological stress as measured by morning cortisol levels (CORT), in an adult population?

As with research question 1, a Pearson correlation coefficient was computed to assess the linear relationship between CORT and HE for the overall group (n = 39, Table 3). The remaining analyses addressing this research question and testing the hypothesis, followed the same steps as the analysis for question 1, however, the HRV measures were removed as variables and replaced by CORT. Regression was run in the direction of HE as a predictor for changes in CORT. Group-wise CORT-HE and HRV-HE associations were examined using Pearson bivariate correlation analysis for each subgroup (nsCLBP and noPain). A Fisher's transformation was performed to test the difference in correlations between the nsCLBP group and noPain group. One-tailed paired t-tests were performed to test between group differences in stress measures.

Table 3

Variables used in Statistical Analyses for CORT – HE Relationship Determination

Salivary Cortisol	Hip Extension
CORT	HE L
	HE R

3.4.3 Research Question 3

Is there a significant difference in iliopsoas tightness as measured by hip extension (HE) between adults who report incidence of Non-Specific Chronic Low Back Pain (nsCLBP) and those who do not?

A one-tailed independent t-test was used to compare the difference in HE between the nsCLBP group (n = 17) and noPain group (n=17). In addition, a post-hoc one-tailed paired t-test was

performed to test for significant difference between the right and left side measures for each group.

3.4.4 Supplementary Analyses

To assess the effect of age on the stress-HE relationship, Pearson correlation analysis was performed to determine if a significant relationship existed between age and HRV, age and CORT and age and HE. Sex was added to the regression models as a predictor variable to test significance of any confounding effects of sex on the prediction accuracy of changes in HRV and CORT from changes in HE.

Post-hoc analyses were performed to provide supplementary information useful to interpreting the results. This included correlation comparisons using *z* transformation for age-controlled HE-Stress correlations (Diedenhofen & Musch, 2015; Hittner et al., 2010), partial correlation analyses controlling for age in HE-Stress correlations, the addition of Rate of Perceived Stress (Stress RP) as a variable in hip extension correlation analyses and t-tests for sex differences between the nsCLBP group and noPain group.

Chapter 4: Results

The results for this study are explained below first by presenting overall descriptive statistics for whole the sample, groups and key variables. Following that, each research question and corresponding hypothesis from Chapter 3 are restated and the results presented. Additional supplementary results are included in Appendix G.

There were two measurements with missing data. One participant (F, 64y) was unable to perform the Modified Thomas Test due to their inability to control the pelvis in a neutral position, in addition to experiencing extreme discomfort during the required movement. The individual was removed from the analysis as the hip extension measures are hypothesized to be related to other variables, therefore the overall group had 39 participants. The second piece of missing data was HRV from the Equivital SEM for one individual (F, 63y, noPain group). Because all other data were available for this participant, and the data were considered missing completely at random, the mean of the overall group's measurements for HRV (SDNN, LF, HF, LF/HF) replaced the participant's missing data and the individual was included in the analyses (Grace-Martin, n.d.; Kang, 2013).

4.1 Sample Description

Table 4 shows the age and sex of the overall, nsCLBP and noPain groups. More females (n=21) than males participated (n=18) in the study, with no significant difference in age between the sexes ($t(37) = 0.21$, $p = .84$). Both the nsCLBP group and noPain group had seventeen participants (9 female) with no significant difference in age between groups ($t(32) = -.37$, $p = .71$; two-tailed). The nsCLBP group had a slightly larger age range than the noPain group, with no significant age difference between males and females for any group.

Table 4

Mean Age in Years by Sex and Group

Group	Sex	n	Mean (years)	*SD*	Min	Max
Overall	F	21	44.7	12.4	24	63
	M	18	45.3	10.2	23	59
	Total	39	45.1	11.3	23	63
noPain	F	9	45.2	13.6	24	63
	M	8	43.6	10.9	29	59
	Total	17	45.1	12.1	24	63
nsCLBP	F	9	47.4	14.4	26	63
	M	8	44.4	10.9	23	57
	Total	17	46.1	12.5	23	63

Of the 17 participants in the nsCLBP group, 15 (78.95%) reported the presence of some level of pain on the morning of their testing. When rating their current pain level (1-9), most individuals with pain present reported a level of 2 out of 9 or less (66.67%), and all reported a pain level less than 6.

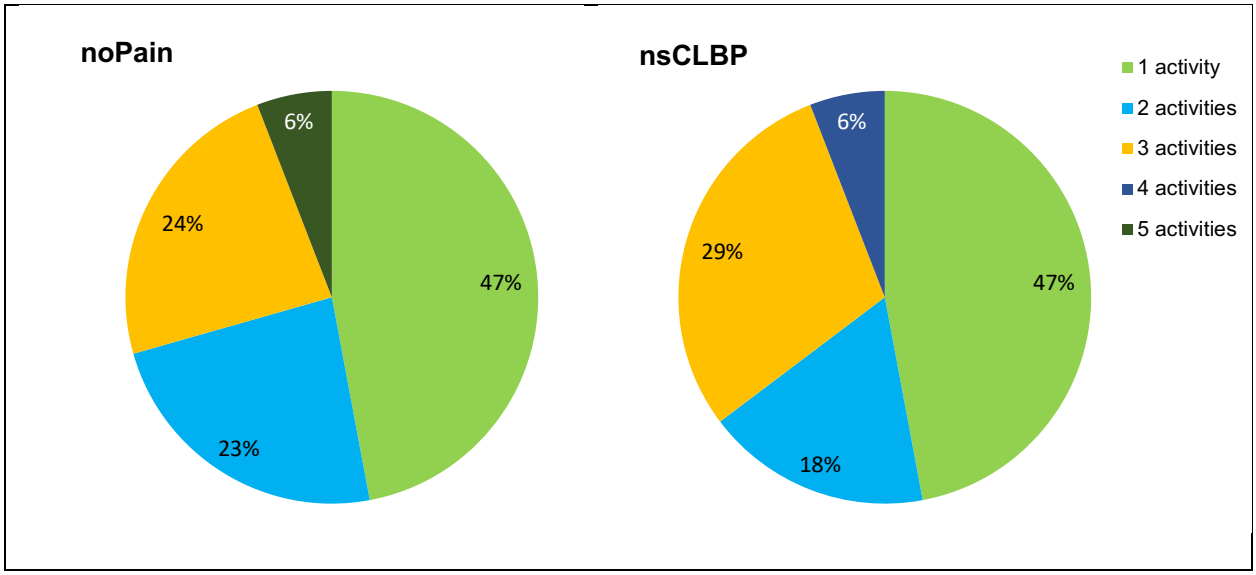
Although not a requirement for participation in the study, all participants reported being physically active in at least one activity twice per week. The t-test (two-tailed) did not show a significant difference in cumulative physical activity scores between the nsCLBP group and noPain group ($t(32) = -0.16$, $p = .88$); the distribution of activity frequency for each group is seen in Figure 5. More detailed results for participant's physical activity can be found in Appendix G.

All participants reported that they followed the pre-test guidelines provided to them: they did not participate in any exercise, including stretching, consume caffeine or other stimulants, eat or brush their teeth the morning of the measurements. All reported having refrained from

alcohol and recreational drugs for the 24hours prior to the testing. All variables for all groups passed Shapiro-Wilks test for normality.

Figure 5

Number of Physical Activities Reported by Participants by Group (n = 17)



Note. 0% of individuals in the nsCLBP group participated in five activities; 0% of individuals in the noPain participated in four activities.

4.2 Findings by Research Question

4.2.1 Research Question 1

What is the relationship between iliopsoas tightness (IPt) as measured by hip extension angle (HE), and physiological stress as measured by Heart Rate Variability (HRV) in an adult population?

Significant correlations were found between stress and hip extension angles in the overall group (n=39). HRV measures of SDNN and HF showed significant positive correlation with both the left and right side measures of HE (Table 5), with the highest correlation being between the right side measures and HF ($r(37) = .43$, $p = .004$). LF/HF showed significant negative

correlation (Table 5), with the left side only. LF did not show significant correlation with HE in the overall group. The post-hoc analysis of difference in age-controlled correlations can be found in Appendix G, Table G3.

Table 5

Correlations for Physiological Stress Measures and Hip Extension Measures in degrees (n = 39)

Variable	SDNN (ms)	LF (ms^2)	HF (ms^2)	LF/HF	CORT (nmol/L)
HE L	.29*	.02	.36*	-.36*	-.26*
p	.04	.45	.01	.01	.05
HE R	.41**	.20	.43**	-.21	-.24
p	.005	.12	.004	.10	.06

Note. One-tailed Pearson correlation. Prob > |*r*| under *HO: Rho = 0.*

LF, HF, LF/HF n = 38 due to the removal of the outlier.

** p* ≤ .05*; ** p* ≤ .005

Simple linear regression analyses were used to test if HE significantly predicted each stress variable. Using the significant HE-Stress correlations from Table 5 (Thomas et al., 2015), R^2 values showed that between 13.0% (HE L ⟶ HF or LF/HF) and 19.0% (HE R ⟶ HF) of the variance in stress measures can be accounted for by changes in hip extension angle (Table 6). The regressions predicted that SDNN (β = .41, *p* = .01), and HF (β = .43, *p* = .01), have the greatest increases when right side hip extension is increased by one degree, whereas LF/HF was significantly decreased when left side hip extension increased. Regression accurately predicted HF from both right side and left side measures of HE (Table 6). SDNN was only accurately predicted from HE R. A summary of the hip extension angle for stress measures predictions are shown on Table 6.

Table 6

Regression Summary for HE as predictor of Physiological Stress (n=39)

Effect	R^2	Coefficient	*SE*	95% CI		*p*	*Standardized Coefficient β*
				LL	*UL*		
	HE L						
SDNN	.09	0.63	0.34	-0.06	1.332	.07	.29
HF†	.13*	25.04	10.88	2.98	47.11	.03	.36
LF/HF†	.13*	-0.21	0.09	-0.39	-0.03	.03	-.36
CORT	.07	-0.14	0.08	-0.30	0.03	.10	-.26
	HE R						
SDNN	.17*	0.97	0.35	0.26	1.69	.01	.41
HF†	.19*	32.72	11.46	9.48	55.96	.01	.43

Note. †Outlier removed n = 38; * $p \leq .05$

Group-wise HRV-HE Association

nsCLBP Group: In the nsCLBP group, SDNN showed moderate to high significant positive correlations with HE on both sides. HF also showed significant positive correlations for both HE R and HE L. LF showed significant positive correlation with the right side, but not the left side. The LF/HF ratio was not significantly correlated with hip extension in the nsCLBP group. Correlations by group and associated *p* values are in Table 7.

noPain Group: By contrast, in the noPain group, no significant correlations were found for SDNN, HF or LF with either right or left side HE. LF/HF showed a moderate significant negative correlation with the left side HE in the noPain group (Table 7).

The Fisher's *r* to z transformation showed that the difference in correlations between the groups was significant for SDNN for HE L and HE R measures (Table 7). The differences in HRV measures between the groups were not significant (Appendix G, Table G1).

4.2.2 Research Question 2

What is the relationship between iliopsoas tightness (IPt) as measure by hip extension angle (HE), and physiological stress as measured by morning cortisol levels (CORT), in an adult population?

As shown above, in Table 5, there was a negative correlation between CORT and HE, which was significant for HE L, in the overall group (n=39). Simple linear regression showed non-significant variance measured in CORT can be accounted for by HE (Table 6). CORT was not accurately predicted by HE R or HE L.

Group-wise CORT-HE Association

There was no group-wise significant correlation for CORT. The Fisher's *r* to z transformation did not show any significant difference in CORT correlations between the nsCLBP and noPain group (Table 7) nor did the t-test (one-tailed) show a significant difference in CORT between the nsCLBP group and noPain group (t (32) = .29, p = .39; Appendix G, Table G1).

Table 7

Correlations for Physiological Stress Measures and Hip Extension Measures in Degrees By Subgroup (n=34)

Group	SDNN (ms)	LF (ms^2)	HF (ms^2)	LF/HF	CORT (nmol/L)
			HE L		
noPain	-.12	-.15	.22	-.52*	-.34
p	.32	.28	.20	.02	.09
nsCLBP	.68**	.40	.49*	-.26	-.22
p	.001	.06	.03	.17	.20
Fisher's *z*	2.47*	1.49	.81	.81	.34
p	.01	.07	.21	.21	.37
			HE R		
noPain	.19	0.13	.27	-0.35	-.30
p	.23	0.32	.15	0.08	.13
nsCLBP	.735**	.61*	.59*	0.16	-.28
p	<.001	0.01	.01	0.27	.14
Fisher's *z*	1.88*	1.53	.46	1.40	-.20
p	0.02	.06	.32	.08	.42

Note. * p ≤ .05; ** p ≤ .005; one-tailed Pearson *r*. Prob > |*r*| under HO: *Rho* = 0.

nsCLBP n = 16 for LF, HF, LF/HF correlations due to the removal of a single outlier.

Fisher's *z* shows *r* to *z* transformation for difference in correlation coefficient between groups and associated *p* values.

4.2.3 Research Question 3

Is there a significant difference in iliopsoas tightness as measured by hip extension (HE) between adults who report incidence of Non-Specific Chronic Low Back Pain (nsCLBP) and those who do not?

Shown in Table 8, the nsCLBP group measured significantly lower ranges of motion than the noPain group for both the left and right side hip extension. The largest difference in means of

HE between groups was on the right side, 8.96º ± 3.10 (*t* (32)= 2.88, *p* =.003). The large effect sizes, ranging from *d* = 7.89 – 9.32 for HE measures demonstrate the level of practical significance.

Table 8

Mean Hip Extension in Degrees by Group (n=34)

Measure	nsCLBP (n=17)		noPain (n=17)					
	Mean (º)	*SD*	Mean (º)	*SD*	Difference (º)	*SE Diff.*	*t*	*p*
HE L	-10.98	12.86	-2.71	6.85	8.23*	3.53	2.34	.01
HE R	-12.82	10.70	-3.86	7.05	8.96**	3.10	2.88	<.005

Note. * $p \leq .05$, ** $p < .005$, one-tailed.

A paired sample t-test (one-tailed) found no significant difference in hip extension between sides for either group (nsCLBP *t(16)* = .80, *p* =.44; noPain *t(16)* = .84, *p* = .41).

Chapter 5: Discussion

This study examined possible relationships between measures of physiological stress and tightness in the hip flexors in adults with and without nsCLBP. Numerous other studies have focused on various aspects of the stress-pain-tension relationship, however none of them have investigated this specific research concept. The primary findings show a significant positive correlation between stress and hip flexor tightness, with stronger relationships among individuals who report experience of nsCLBP. Specifically, lower hip extension (HE) angles were found in the nsCLBP group, in addition, lower HE angles were correlated with lower HRV. Significant correlations found between different measures of HE and cortisol provide further evidence of the stress-tension relationship.

5.1 Stress-Muscle Tension Relationship

Question 1: What is the relationship between iliopsoas tightness (IPt) as measured by hip extension angle (HE), and physiological stress as measured by Heart Rate Variability (HRV) in an adult population?

H_1: There is a positive relationship between hip extension range of motion (HE) and HRV in an adult population that indicates lower HE, greater tightness in the iliopsoas with measures of HRV that indicate a higher stress response.

The significant positive correlations found between HE and HRV measures of SDNN and HF support the H_1 research hypothesis. These results suggest that less tightness in the IP as measured by higher HE is associated with better autonomic regulation. Likewise, a significant negative correlation was established between LF/HF and HE.

5.1.1 HE-HRV Time Domain

While HRV time domain measures, such as SDNN, express a more comprehensive assessment of HRV over a 24-hour period, the use of a consistent 5-minute measure for all participants in this study validates the comparison of SDNN values with other variables and individuals within the same study (Malik et al., 1996). The variability in these short-term measures is primarily due to the parasympathetic influence on respiratory sinus arrhythmia (RSA) (Shaffer & Ginsberg, 2017). Therefore, the significant positive correlation found between SDNN and HE in this study represents the relationship between IP tightness and the parasympathetic contribution to resting daytime HRV.

For comparison, the most relatable research comes primarily from intervention studies in massage or stretching techniques. A study by Delaney et al.(2002) focused on using tension releasing massage in the back and shoulders and reported measures following the treatment that displayed a decreased stress response. They measured a 17% decrease in LF/HF, 28% increase in SDNN, and 90% increase in HF. Farinatti et al. (2011) also measured an increase in SDNN after trunk and hamstring stretching in low flexibility individuals when compared to pre-stretching baseline. The increase in SDNN is indicative of enhanced vagal modulation of heart rate or increase in parasympathetic activity. By contrast, a 2021 study by Lee and Lee (2021) did not find a significant difference in any HRV measures after myofascial treatment. They did, however report significant improvement in trunk (sit and reach) and shoulder (back scratch test) range of motion. Lee and Lee used one bout of 1-minute foam rolling self-myofascial release (SMR) on thoracic, lumbar, quadriceps, hamstrings, gluteal and calf target areas. Although they measured significant improvement in the two flexibility tests, they did not find a significant correlation between range of motion and HRV. Despite evidence of SMR improving pain

perception in athletes (Nakamura et al., 2019), for deconditioned individuals SMR may be painful (Nakamura et al., 2021). This pain activates nociceptors, which in turn decreases HRV due to increased SNS activity. Any HRV effect potentially created by the increase in range of motion in the Lee and Lee study could be dampened by the amount of SNS activity from the painful stimulus. There was no indication in the methods that participants were screened for chronic pain symptoms, which could be exacerbated by the treatment and stimulate a prolonged nociceptor response (Ferrer-Montiel et al., 2016). SMR has been shown to increase pain threshold in the specified area, however the single bout used in the Lee and Lee study may not meet the required dose for such an outcome (Nakamura et al., 2019). The results from the current study support the previous work by both Farinatti et al. and Delaney et al. by also identifying a positive correlation between decreased muscle tension and increased SDNN. This study also augments previous findings by using the objective Thomas test measure for muscle tightness.

5.1.2 HE-HRV Frequency Domain

The significant negative correlations found between the LF/HF ratio and HE L in the current study further support H_1. A simplified explanation of the LF/HF ratio is that its value displays the level of autonomic balance (Shaffer & Ginsberg, 2017a), and a more precise explanation is that an LF/HF ratio above 2.0 indicates a dominance in the contribution of the SNS to HRV (Nunan et al., 2010). In the present study, LF/HF means for all groups were greater than 2.0. This indicates that on average the participants in the study showed signs of sympathetically dominant autonomic control on testing day. Sympathetic dominance indicates a system is under stress. Although the source of 100% of the stress cannot be identified by the current study, what can be identified is that individuals with more tightness in the iliopsoas were

exhibiting LF/HF measures indicative of individuals who may be under high amounts of stress. The main limitation of hip extension range of motion in the Modified Thomas Test has been primarily identified as tightness in the iliopsoas (IP), caused by muscle tension or tissue stiffness. One potential explanation of these results is that tension in the IP elicits pressure on the dense network of mechanoreceptors in the lumbar and visceral areas which results in the increase in SNS activity (Kaufman & Hayes, 2002; Lydakis & Sinoway, 2007; Murayama et al., 2012). The more tension, the greater stimulation of mechanoreceptors and the higher the SNS activity. It is also possible that an increase in psychological stress has created a physiological stress response which increases muscle tension, making the relationship bi-directional (Nyboe et al., 2017).

When considering LF and HF as individual metrics, outside of the ratio, LF is often taken to be associated with activity in the SNS when breathing is not controlled, where higher HF accompanies higher overall HRV and higher PSNS tone (Shaffer & Ginsberg, 2017). LF, however, is mediated by both sympathetic and parasympathetic activities (Malik et al., 1996). In the current study, there was no significant relationship between HE and LF in the overall group, which is consistent with other studies that have explored the muscle tension-stress relationship (Farinatti et al., 2011; Wong & Figueroa, 2021).

HF was the HRV measure revealed to be the most influenced by changes in IP tightness (Table 3). These findings support the hypothesis by relating less IP tightness to increased PSNS tone. Previous studies using static stretching protocols for the trunk, hamstrings, and calf muscles have shown an increase in PSNS activity measured by increased HF after stretching (Wong & Figueroa, 2021). One outlier is a study by Silva et al. (2016) that did not report an increase in HRV after active static stretching. The difference in the findings can be attributed to the low volume of stretching used by Silva et al. which may not have been enough to produce

significant changes in autonomic function. Silva et al. also only measured young (mean age of 22), trained men and used a small sample size. Since HRV is significantly correlated with age, and a higher volume of stretching may be required to elicit changes in HRV in young, fit men compared to older, untrained individuals, the outcomes of Silva et al. may be questionable due to methodological issues (Farinatti et al., 2011; Wong & Figueroa, 2021).

The strong bidirectional relationship between muscle tension in temporomandibular jaw disorder (TMJD) and stress reported by (Glaros et al., 2016)is also supported by the current study. Glaros et al., however, did not use a range of motion test, but used a subjective scale for rating the amount of muscle tension in TMJD in relationship to stress.

A consistent limitation of the relevant research in this area is the lack of objective measure of muscle tension or tightness or range of motion, apart from the Lee and Lee (2021) study. The current study stands apart in its examination of the muscle tension-stress relationship with the use of defined objective measures of both muscle tension and stress. In addition, no other studies targeted the IP or hip extension specifically. An important part of the rationale for the current study is the focus on the IP, which, due to its biomechanical contributions to the lumbar spine and hip joints, proximity to the lumbar plexus, visceral organs and connection to the diaphragm has unique potential for tension in response to stress or initiation of SNS activity when tense or tight. The present findings extend previous research to include a positive correlation between higher parasympathetic tone as measured by higher HF and increased hip extension angle, indicating less tension in IP.

5.1.3 HE-Cortisol

Question 2: What is the relationship between iliopsoas tightness (IPt) as measure by hip extension angle (HE), and physiological stress as measured by morning cortisol levels (CORT), in an adult population?

H_2: There is a significant negative relationship between iliopsoas tightness as measured by hip extension range of motion and morning cortisol, in that on average, lower CORT is correlated with greater HE.

The significant negative correlation between CORT and left side HE suggests that less tension in IP is related to lower levels of daytime cortisol, and thus supports H_2. Although CORT displays a diurnal cycle throughout the day including increased secretion in periods of stress, in general, a lower daytime CORT indicates a well-regulated neuroendocrine system (Adam et al., 2017; Hannibal & Bishop, 2014; Joseph & Golden, 2017). A well-regulated neuroendocrine system is present in individuals who either are not under increased psychological stress or have healthy physiologic adaptability to stress (Adam & Kumari, 2009; Cohen et al., 2007b; Vanitallie, 2002).

Cortisol's role in modulating inflammation in the body is an important consideration in understanding the relationship between CORT and HE. Although acute release of cortisol serves to decrease inflammation (Adam & Kumari, 2009), chronic, prolonged cortisol release can lead to cortisol dysfunction and increased stiffness in the collagen network present in connective tissues, including tendons ("Devastating Impact of Stress on Collagen", 2023). The fascial matrix of the myofascial system may become dense and stiff, limiting the muscle's ability to shorten and lengthen (Maganaris et al., 2017; Schleip et al., 2010). The result is a decreased available range of motion around the joint the muscle acts on. Another possible mechanism is

the role of cortisol in the mobilization of amino acids in response to increasing energy demands or needed protein synthesis during stress (Deussing & Chen, 2018). Amino acids may be mobilized from muscle tissue which decreases the number of muscle fibres, and can decrease the rate of collagen synthesis which is needed to allow shortening and lengthening of the muscle, again restricting joint range of motion (Deussing & Chen, 2018; Maganaris et al., 2017; Schleip et al., 2010).

There is limited research investigating the relationship between salivary cortisol and muscle tension, tissue stiffness or range of motion. The current study is the first of its kind to specifically measure the relationship between CORT and hip extension range of motion. However, drawing on research in the areas of massage, stretching and yoga, we can gain more clarity. Pinar et al. (2015) measured a significant decrease in CORT following one week of daily back massage when compared with controls. Yoga postures (Kuen, 2019) and static stretching (Behm et al., 2021; Corey et al., 2014) have also been shown to increase range of motion. Eda et al. (2020) measured a decrease in salivary cortisol after a stretching based yoga protocol. Although the study confirmed the relaxation effects of yoga, no non-yoga simple stretching group was used for comparison, nor was range of motion or muscle tension measured to rule out any decrease in muscle tension as a possible contributing mechanism to the measured decrease in cortisol. Although it is plausible that the psychological relaxation effects of each yoga and stretching were responsible for the lower cortisol, an earlier study by Corey et al. (2014) reported a significant decrease in salivary cortisol in a stretching group over a relaxation-based yoga group, indicating the stretching activity component of yoga itself is an important factor to consider in the relationship. By contrast, Fuller & Stewart (2010) did not find a significant difference in cortisol following a short bout of static stretching. Fuller & Stewart however,

measured cortisol after only one session of partner-assisted stretching in 13 young subjects, where Corey et al. followed a 6-month protocol in 136 subjects. The methods of Corey et al. were specifically designed to isolate the effects of the relaxation response in the yoga group where the stretching protocol in the study was design by a physiotherapist with the goal of "mobilizing soft tissue to improve muscle length and range of motion" (Corey et al., 2014). In addition, Corey et al. recorded a decrease in self-reported stress ratings in the stretching group, and not the yoga group. The results by Corey et al, are highly relevant to the current study as the aim of the protocol which resulted in the decrease in cortisol was explicitly designed to increase range of motion. While Corey et al. attributed the cortisol change in the stretching group to psychosocial factors, the study did not objectively measure muscle tissue length or range of motion or include discussion that the increase in range of motion could be a potential contributing factor in their results, as shown in the present study.

In the current study, the fact that the left side HE only showed significant negative correlation with CORT, but the right side did not, may be meaningful. It is possible that the relationship between CORT and HE is not linear. HE L was, on average, larger than HE R, which can be interpreted as less muscle tightness on the left side. Although the difference was not statistically significant, it was enough to impact the significance of the correlation with CORT. Since lower HE values did not significantly relate to higher CORT, it cannot be concluded from this study that increased muscle tension in the PM elicited a neuroendocrine stress response or that CORT caused a detrimental change in the tissue. However, the results do support the previous literature by indicating that a decrease in tissue stiffness, or muscle tension may influence neuroendocrine function, and vice versa.

5.2 Stress-Muscle Tension Relationship in Presence of nsCLBP

Although there were no significant differences in stress measures between the nsCLBP and noPain groups (Appendix G, Table G2), the correlations between HRV and HE when examined by group reveal some interesting findings. The strength of the relationship between HE and parasympathetic influence on HRV is shown to be greater when back pain is present. This is evident in the strong significant correlations between HE and SDNN and HE and HF in the nsCLBP group, that are not present in the noPain group. Even though the lower SDNN and HF measures in the nsCLBP group did not show a significant difference from the noPain group (Appendix G, Table G2), the difference is very relevant when considering how changes in muscle tension or pain are included, and therefore has a level of practical significance. The current data do not provide a precise explanation for the mechanism behind this shift, but it does imply that there exists a complicated, interrelated dynamic between IP tightness, autonomic activity and pain. This supports previous research that links muscle tension, pain and stress (Glaros et al., 2016).

It cannot be determined from the current study if hip flexor tension is solely responsible for the higher measures of stress in individuals who display less range of motion in hip extension. The regression was conducted to use HE as a predictor of stress to provide a unique perspective on the relationship, since stress has previously been shown to contribute to muscle tension via SNS arousal. Despite this methodological decision, it is likely that the relationship between stress and IP tightness is bi-directional. In addition, stress can be physical or psychological, current or anticipatory, and changes in CORT and decreases in HRV can occur for many different reasons. Asking participants to rate current stress levels attempted to rule out perceived or anticipatory stress as responsible for any increase in stress measures, in addition to

controlling the environment by setting a comfortable room temperature, dim lighting and consistent time windows for measures. However, sleep duration and quality, respiration rate, fitness level, life factors, historical substance use, or regular hormone cycles were not controlled for and can also influence these measures (Cohen et al., 2019). Likewise, passive range of motion in hip extension can be related to genetics, joint structure, mechanical and neural factors, inflammation, muscle fibre distribution, hormone circulation, connective tissue density, joint pathologies, delayed onset muscle soreness, abdominal strength, training history, psychological factors and lifestyle factors (Mitchell et al., 2022; Mizuno, 2017; Neumann, 2010).

Overall, the current study's findings are consistent with previous intervention research that show physiological stress measurements indicative of higher parasympathetic tone and lower cortisol levels are related to less muscle tightness. Moreover, the present study enhances our understanding of this relationship by demonstrating a noteworthy correlation between larger hip extension range of motion – which is in part attributable to less tension in the IP – and increased HRV as well as reduced daytime cortisol levels.

5.3 Muscle Tension-Pain Relationship

Question 3: Is there a significant difference in iliopsoas tightness as measured by hip extension (HE) between adults who report incidence of Non-Specific Chronic Low Back Pain (nsCLBP) and those who do not?

H_3: There is a significant difference in hip extension range of motion in individuals with back pain vs individuals without low back pain, in that individuals with nsCLBP measure lower hip extension angles.

Participants with nsCLBP had significantly lower HE when compared to those without nsCLBP (Table 8). These results add to previous research which has identified significant

correlations between tightness in the hip flexor muscles using the modified Thomas test (MTT) and nsCLBP (Kim & Shin, 2020; Shin, 2020). Using a similar protocol to this current study, Roach et al. (2015) found a 10-degree difference in the MTT measured hip extension between those with nsCLBP and those without. The results of the present study show a similar difference between the groups (9-degrees; Table 8). The systematic review of the relationship between hip mobility and nsLBP by Avman et al. (2019) identified four studies in addition to the work of Roach et al., sharing similar results using the MTT and goniometer. Overall, the review reported inconclusive results for decreased hip extension range of motion in individuals with nsLBP as the evidence was reportedly low quality due to inconsistency in findings, small sample sizes, subjects not being recruited from similar populations, variability in range of motion measurement methods and lack of adjustments for confounders (Avman et al., 2019).

Kitamura et al., (2019) found that athletes with nsCLBP display greater anterior tilt due to stiffness in the PM muscle as measured with shear wave elastography. These results supported an earlier study by Youdas et al. (2000) which concluded hip flexor tightness was a more likely cause of anterior pelvic tilt leading to back pain than lack of abdominal strength. They did not however, rule out all contribution of abdominal strength. Hamstring tightness (Fasuyi et al., 2017), limited hip internal rotation (Avman et al., 2019; Shin, 2020), and tensor fasciae latae muscle tightness (Abe et al., 2014) have also been reported as significantly higher in individuals with nsCLBP. In the current study, the femurs were closely monitored for rotation and abduction, limiting the movement tested in the MTT to a measure of extension and decreasing the possibility of other tissue tightness contributing to any lack of movement.

Kim and Shin (2020) provided evidence that asymmetry in hip extension range of motion is a contributing factor to back pain. The degree of asymmetry showed a strong correlation with

the degree of pain experienced (Kim & Shin, 2020). Although the nsCLBP group in the current study showed the most asymmetry, neither group showed a significant difference between sides (Table 8), indicating that asymmetry is not a significant contributing factor to back pain in this sample. Although this appears inconsistent with previous findings, the degree of pain in relationship to HE asymmetry was not analyzed in this study.

Despite the large body of evidence identifying increased stress in the presence of nsCLBP pain (Ganesan et al., 2017; Hallman & Lyskov, 2012; Hannibal & Bishop, 2014; Linton, 2000; Sveinsdottir et al., 2016), the current study did not find a significant difference in physiological stress measures or perceived stress level between the groups. It is possible that the duration and intensity of pain are important variables in the contribution of pain to stress or contribution of physiological stress to pain, rather than simply the presence of the pain experience. This is a limitation of the current study as pain intensity was not measured. Comparisons of the measures between groups for stratified levels of physiological stress measurements would be required to determine if pain is more likely at a specific given level of HRV or cortisol. A possible explanation of the current findings is that the decreased hip extension range in the nsCLBP is a biomechanical contributor to the pain, and/or that the pain experience of the nsCLBP group creates hip flexor tightness as a bi-directional relationship, and that regardless of muscle tension, the stress-pain relationship is highly individual.

5.4 Limitations

Even though the time of day for cortisol collection was within the first 2 hours of waking, there was no minimum window for waking time prior to collection. It is possible that some individuals in the study woke within 30-40 minutes of the sample collection which meant their CORT measures may have been taken during the Cortisol Awakening Response (CAR), which

could be 50-60% above normal resting levels (Adam et al., 2017). This could cause some limitations when drawing conclusions in this study as the measured cortisol may not be a true daytime resting level. Although the mean CORT of the sample population (6.64nmol/L) was within normal range by medical standards (Salimetrics, 2021a), it is on the higher end of what would be expected for average daytime resting levels. Females do exhibit higher cortisol levels on average than males (Adam & Kumari, 2009), however because there was no significant difference in CORT between males and females in this study, the higher CORT measures cannot be attributed to having more females in the sample.

Measurements in this study were taken at a single time point. Short term measures for SDNN in this study cannot be compared to normative values or used to estimate cardiac risk level, therefore HE cannot be used to predict any cardiac outcome risks based on the regression equation with SDNN.

The physical activity summary of participants (Figure 3) indicated that all participants in the study (both nsCLBP and noPain groups) were consistent yoga practitioners. As such, the results may not be transferrable to those who do not regularly participate in yoga.

The age range for the sample was quite large, and although both age and HRV measures showed a normal distribution, there was large variance in both variables. Results may vary for samples that contain different, smaller age groups.

5.5 Practical Implications and Future Directions

Treatment of nsCLBP and treatment of stress-based symptoms are highly segmented in the medical world, The current management of nsCLBP is commonly placed in the hands of medical practitioners and allied health practitioners who primarily focus on the physical body by targeting specific body pain symptoms. Such treatments are musculoskeletal and biomechanical-

corrective in origin, such as physiotherapy, chiropractic medicine, massage therapy and osteopathic manual therapy. Likewise, symptoms indicative of chronic or increased psychological stress, such as anxiety, irritability, 'scattered mindedness' and insomnia are treated through mind, brain or behaviour-centered professionals through psychological methods including cognitive behavioural therapy, counselling and behavior modification training or life coaching. Perhaps one of the most relevant and impactful practical applications of the results of the current study is the evidence it provides to support a shift in this paradigm. Such a shift would incorporate a multidisciplinary approach to the treatment of back pain. Or, conceivably, a model whereby the psychotherapist is trained in teaching simple body-based movements or physical positions or stretches designed to alleviate the pain while also contributing to decrease a decrease in stress. Likewise, the physical body practitioner could use the same protocol, increasing their pain management impact beyond the physical body structure, especially where research has shown a potential for psychogenic causes of pain. Such protocols may already exist in the realm of somatic movement, yoga or other mind-body disciplines. At a minimum, provided herein is empirical data validating the efficacy of the inclusion of physical pain screening by behavior-centered psychological practitioners and a screening for psychological contributors to pain be a part of the thorough training of a physical body practitioner. At least if such screening were to take place, a referral to an additional practitioner with a different area of expertise may improve the long-term effectiveness of treatment.

Just as a physiotherapist performs a flexibility test for the hamstrings in their assessment of possible causes for low back pain, it would not be cumbersome or unrealistic to include the assessment of both hip range of motion and stress in the development of a thorough treatment plan for nsCLBP. Further intervention-based protocols are needed to identify the efficacy of

specific multi-disciplinary treatment plans or exercises and their effects on the stress-pain-mobility relationship.

Left side hip extension predicted LF/HF accurately and though standardized values of HE are shown to statistically increase HF more, the impact of HE on LF/HF ratio may be more practical. When examining the regression, increasing HE by a mere 1 degree is predicted to decrease the LF/HF ratio by 0.21. Healthy individuals have LF/HF ratios between 1.5-2.0, which means a hip mobility protocol that increases range of motion in hip extension by 3 degrees may be highly impactful to health. This may be particularly important for individuals who suffer with nsCLBP, as the relationship between LF/HF and HE is stronger in the presence of nsCLBP than without pain. Intervention studies to examine the required dose of mobility needed for this outcome, in addition to measuring associated changes in HRV could provide the insight needed to develop mobility-based protocols that increase autonomic balance and simultaneously support the treatment of nsCLBP.

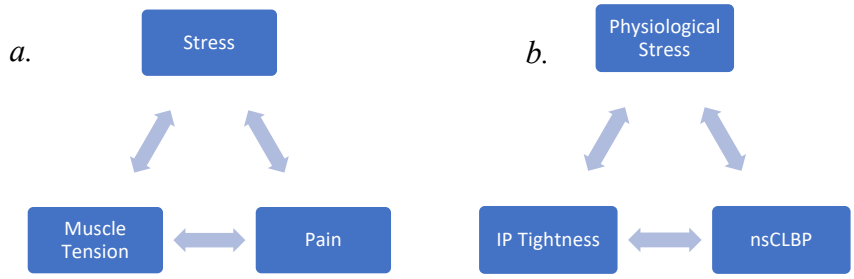
To further validate the results of the current study, intervention studies aimed to release tension or tightness in IP and measuring HRV outcomes are needed.

5.6 Conclusion

The results of this research demonstrate that tightness in IP can negatively impact physiological stress, or vice versa, and supports previous literature which identified the bidirectional relationship between muscle tension and stress. The findings also provide evidence that individuals with nsCLBP have, on average, less range of motion in hip extension when compared to pain free individuals. This may have important implications for developing effective treatment plans for nsCLBP and highlighting the importance of hip range of motion in prevention of low back pain. Further, the findings suggest that the relationship between muscle tension and stress is stronger in individuals who have nsCLBP when compared to those who do not, adding support to the current understanding of the pain-stress cycle. This may provide important insight into why and how mind body practices and psychosomatic therapies have been shown to support improvement in pain symptoms and decrease psychological stress. An overall conclusion is displayed in Figure 6 below, indicating that not only do the bi-directional relationships of stress ↔ pain, pain ↔ muscle tension and muscle tension ↔ stress exist, but the novel idea that there exists a cyclical or interrelationship among stress, muscle tension and pain.

Figure 6

Schematic of the Interrelationship Among Variables



Note. *a.* indicates the general relationship *b.* represents the specific relationship examined in this study.

References

Abe, A., Sato, K., Mitsueda, S., Kimura, M., Hirooka, E., Tsuchiya, A., & Kanisawa, I. (2014). The influence of muscle tightness of lower extremities on low back pain in young female figure skaters. *British Journal of Sports Medicine*, *48*(7), 560.1-560. https://doi.org/10.1136/bjsports-2014-093494.1

Adam, E. K., & Kumari, M. (2009). Assessing salivary cortisol in large-scale, epidemiological research. *Psychoneuroendocrinology*, *34*(10), 1423–1436. https://doi.org/10.1016/j.psyneuen.2009.06.011

Adam, E. K., Quinn, M. E., Tavernier, R., McQuillan, M. T., Dahlke, K. A., & Gilbert, K. E. (2017). Diurnal cortisol slopes and mental and physical health outcomes: A systematic review and meta-analysis. *Psychoneuroendocrinology*, *83*, 25–41. https://doi.org/10.1016/j.psyneuen.2017.05.018

Akintola, A. A., van de Pol, V., Bimmel, D., Maan, A. C., & van Heemst, D. (2016). Comparative analysis of the equivital eq02 lifemonitor with holster ambulatory ecg device for continuous measurement of ecg, heart rate, and heart rate variability: A validation study for precision and accuracy. *Frontiers in Physiology*, *7*(SEP). https://doi.org/10.3389/fphys.2016.00391

Alvarez, P., Green, P. G., & Levine, J. D. (2013). Stress in the adult rat exacerbates muscle pain induced by early-life stress. *Biological Psychiatry*, *74*(9), 688–695. https://doi.org/10.1016/j.biopsych.2013.04.006

American Psychiatric Association. (2022). Trauma and stressor related disorders. *Diagnostic and Statistical Manual of Mental Disorders*. Psychiatry Online. American Psychiatric Association Publishing.

https://doi.org/doi:10.1176/appi.books.9780890425787.x07_Trauma_and_Stressor_Related_Disorders

Andersen, T. E., Andersen, P. G., Vakkala, M. A., & Elklit, A. (2012). the traumatised chronic pain patient—Prevalence of posttraumatic stress disorder - PTSD and pain sensitisation in two scandinavian samples referred for pain rehabilitation. *Scandinavian Journal of Pain*, *3*(1), 39–43. https://doi.org/10.1016/j.sjpain.2011.10.001

Anderson, G. S., Di Nota, P. M., Metz, G. A. S., & Andersen, J. P. (2019). The impact of acute stress physiology on skilled motor performance: Implications for policing. *Frontiers in Psychology*, *10*. https://doi.org/10.3389/fpsyg.2019.02501

Andersson, E., Oddsson, L., Grundström, H., & Thorstensson, A. (2007). The role of the psoas and iliacus muscles for stability and movement of the lumbar spine, pelvis and hip. *Scandinavian Journal of Medicine & Science in Sports*, *5*(1), 10–16. https://doi.org/10.1111/j.1600-0838.1995.tb00004.x

Arbanas, J., Pavlovic, I., Marijancic, V., Vlahovic, H., Starcevic-Klasan, G., Peharec, S., Bajek, S., Miletic, D., & Malnar, D. (2013). MRI features of the psoas major muscle in patients with low back pain. *European Spine Journal*, *22*(9), 1965–1971. https://doi.org/10.1007/s00586-013-2749-x

Atarodi, S., & Hosier, S. (2011). Trauma in the mind and pain in the body: mind-body interactions in psychogenic pain. *Human Architecture: Journal of the Sociology of Self-Knowledge*, *9*(1), 10. https://www.researchgate.net/profile/Steven-Hosier/publication/254695219_Trauma_in_the_Mind_and_Pain_in_the_Body_Mind-Body_Interactions_in_Psychogenic_Pain/links/53f61a750cf2888a74929d7a/Trauma-in-the-Mind-and-Pain-in-the-Body-Mind-Body-Interactions-in-Psychogenic-Pain.pdf

Ateş, F., Hug, F., Bouillard, K., Jubeau, M., Frappart, T., Couade, M., Bercoff, J., & Nordez, A. (2015). Muscle shear elastic modulus is linearly related to muscle torque over the entire range of isometric contraction intensity. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, *25*(4), 703–708. https://doi.org/10.1016/j.jelekin.2015.02.005

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 & Practice*, *42*, 38–51. https://doi.org/10.1016/j.msksp.2019.03.002

Avrahami, D., & Potvin, J. R. (2014). The clinical and biomechanical effects of fascial-muscular lengthening therapy on tight hip flexor patients with and without low back pain. *The Journal of the Canadian Chiropractic Association*, *58*(4), 444. http://www.ncbi.nlm.nih.gov/pubmed/25550670

Balagué, F., Mannion, A. F., Pellisé, F., & Cedraschi, C. (2012). Non-specific low back pain. *The Lancet*, *379*(9814), 482–491. https://www.spinedragon.com/student_material/reading/2018_nslbp_lancet_1.pdf

Bandeira, P. M., Reis, F. J. J., Sequeira, V. C. C., Chaves, A. C. S., Fernandes, O., & Arruda-Sanchez, T. (2021). Heart rate variability in patients with low back pain: A systematic review. *Scandinavian Journal of Pain*, *21*(3), 426–433. https://doi.org/10.1515/SJPAIN-2021-0006

Barker, K. L., Shamley, D. R., & Jackson, D. (2004). Changes in the cross-sectional area of multifidus and psoas in patients with unilateral back pain: the relationship to pain and disability. *Spine*, *29*(22), E515–E519. https://doi.org/10.1097/01.brs.0000144405.11661.eb

Behm, D. G., Alizadeh, S., Anvar, S. H., Drury, B., Granacher, U., & Moran, J. (2021). Non-local acute passive stretching effects on range of motion in healthy adults: a systematic review with meta-analysis. *Sports Medicine 2021 51:5*, *51*(5), 945–959. https://doi.org/10.1007/S40279-020-01422-5

Bilandzic, A., & Rosella, L. (2017). The cost of diabetes in Canada over 10 years: Applying attributable health care costs to a diabetes incidence prediction model. *Health Promotion and Chronic Disease Prevention in Canada*, *37*(2), 49–53. https://doi.org/10.24095/hpcdp.37.2.03

Billman, G. E. (2011). Heart rate variability - A historical perspective. *Frontiers in Physiology*, *2 NOV*, 86. https://doi.org/10.3389/fphys.2011.00086/bibtex

Boersma, K., Macdonald, S., & Linton, S. J. (2012). Longitudinal relationships between pain and stress problems in the general population: predicting trajectories from cognitive behavioral variables. *Journal of Applied Biobehavioral Research*, *17*(4), 229–248. https://doi.org/10.1111/jabr.12000

Bogduk, N., Pearcy, M., & Hadfield, G. (1992). Anatomy and biomechanics of psoas major. *Clinical Biomechanics*, *7*(2), 109–119. https://doi.org/10.1016/0268-0033(92)90024-X

Bordoni, B., & Varacallo, M. (2018). Anatomy, fascia. *StatPearls*. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/pubmed/29630284

Bordoni, B., & Zanier. (2013). Anatomic connections of the diaphragm influence of respiration on the body system. *Journal of Multidisciplinary Healthcare*, *6*, 281. https://doi.org/10.2147/JMDH.S45443

Cai, W., Li, H. Z., Zhang, X., Song, Y., Ma, X., Dong, J., Chen, W., Chen, G. F., Xu, Y., Lu, J. S., Wang, B. J., & Shi, T. P. (2013). Medial arcuate ligament: A new anatomic landmark

facilitates the location of the renal artery in retroperitoneal laparoscopic renal surgery. *Journal of Endourology*, *27*(1), 64–67. https://doi.org/10.1089/END.2012.0152

Campbell, J. N., & Meyer, R. A. (2006). Mechanisms of neuropathic pain. *Neuron*, *52*(1), 77. https://doi.org/10.1016/J.NEURON.2006.09.021

Cardiovascular disease - Economic burden of illness - Canada.ca. (n.d.). Retrieved April 10, 2020, from https://www.canada.ca/en/public-health/services/chronic-diseases/cardiovascular-disease/cardiovascular-disease-economic-burden-illness.html

Cardiovascular diseases (CVDs). (n.d.). Retrieved April 11, 2020, from https://www.who.int/en/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)

Carney, R. M., Freedland, K. E., Stein, P. K., Miller, G. E., Steinmeyer, B., Rich, M. W., & Duntley, S. P. (2007). Heart rate variability and markers of inflammation and coagulation in depressed patients with coronary heart disease. *Journal of Psychosomatic Research*, *62*(4), 463–467. https://doi.org/10.1016/j.jpsychores.2006.12.004

Cauwenbergs, P. (2020). Vertebral subluxation and the anatomic relationships of the autonomic nervous system. https://musculoskeletalkey.com/vertebral-subluxation-and-the-anatomic-relationships-of-the-autonomic-nervous-system/

Celik, D., & Mutlu, E. K. (2013). Clinical implication of latent myofascial trigger point. *Current Pain and Headache Reports 2013 17:8*, *17*(8), 1–7. https://doi.org/10.1007/S11916-013-0353-8

Chalmers, J. A., Quintana, D. S., Abbott, M. J. A., & Kemp, A. H. (2014). Anxiety disorders are associated with reduced heart rate variability: A meta-analysis. *Frontiers in Psychiatry*, *5*(JUL). https://doi.org/10.3389/fpsyt.2014.00080

Cholewicki, J., & McGill, S. M. (1996). Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. *Clinical Biomechanics*, *11*(1), 1–15. https://doi.org/10.1016/0268-0033(95)00035-6

Clapis, P. A., Davis, S. M., & Davis, R. O. (2008). Reliability of inclinometer and goniometric measurements of hip extension flexibility using the modified Thomas test. *Physiotherapy Theory and Practice*, *24*(2), 135–141. https://doi.org/10.1080/09593980701378256

Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007a). Psychological stress and disease. *JAMA*, *298*(14), 1685. https://doi.org/10.1001/jama.298.14.1685

Cohen, S., Murphy, M. L. M., & Prather, A. A. (2019). Ten surprising facts about stressful life events and disease risk. *Annual Review of Psychology*, *70*(1), 577–597. https://doi.org/10.1146/annurev-psych-010418-102857

Corey, S. M., Epel, E., Schembri, M., Pawlowsky, S. B., Cole, R. J., Araneta, M. R. G., Barrett-Connor, E., & Kanaya, A. M. (2014). Effect of restorative yoga vs. stretching on diurnal cortisol dynamics and psychosocial outcomes in individuals with the metabolic syndrome: The PRYSMS randomized controlled trial. *Psychoneuroendocrinology*, *49*(1), 260–271. https://doi.org/10.1016/j.psyneuen.2014.07.012

Costa Silva, G., Conceição, R., Di Masi, F., Domingos, T., Herdy, C., & Silveira, A. (2016). Low intensity static stretching does not modulate heart rate variability in trained men. *MedicalExpress*, *3*(3), M160304. https://doi.org/10.5935/medicalexpress.2016.03.04

Crompton, S. (2011). *What's stressing the stressed? Main sources of stress among workers*. Statistics Canada.

Cysarz, D., van Leeuwen, P., Edelhäuser, F., Montano, N., Somers, V. K., & Porta, A. (2015). Symbolic transformations of heart rate variability preserve information about cardiac autonomic control. *Physiological Measurement*, *36*(4), 643.

Dangaria, T., & Naesh, O. (1998). Changes in cross-sectional area of psoas major muscle in unilateral sciatica caused by disc herniation. *Spine*, *23*(8), 928–931. http://ovidsp.ovid.com/ovidweb.cgi?T=JS&PAGE=reference&D=ovftc&NEWS=N&AN=00007632-199804150-00016.

De Ridder, D., Adhia, D., & Vanneste, S. (2021). The anatomy of pain and suffering in the brain and its clinical implications. *Neuroscience and Biobehavioral Reviews*, *130*, 125–146. https://doi.org/10.1016/J.NEUBIOREV.2021.08.013

Delaney, J. P. A., Leong, K. S., Watkins, A., & Brodie, D. (2002). The short-term effects of myofascial trigger point massage therapy on cardiac autonomic tone in healthy subjects. *Journal of Advanced Nursing*, *37*(4), 364–371. https://doi.org/10.1046/j.1365-2648.2002.02103.x

Derrickson, B. H. (2017). *Human physiology*. Wiley.

Deussing, J. M., & Chen, A. (2018). The corticotropin-releasing factor family: physiology of the stress response. *Physiological Reviews*, *98*(4), 2225–2286. https://doi.org/10.1152/physrev.00042.2017

Deyo, R. A. (1986). Early diagnostic evaluation of low back pain. *Journal of General Internal Medicine*, *1*(5), 328–338. https://doi.org/10.1007/BF02596214

Diabetes in Canada - Canada.ca. (n.d.). Retrieved March 10, 2020, from https://www.canada.ca/en/public-health/services/publications/diseases-conditions/diabetes-canada-highlights-chronic-disease-surveillance-system.html

Diedenhofen, B., & Musch, J. (2015). cocor: A comprehensive solution for the statistical comparison of correlations. *PLOS ONE*, *10*(4), e0121945. https://doi.org/10.1371/JOURNAL.PONE.0121945

Driscoll, M. (2017). Fascia – The unsung hero of spine biomechanics. *Journal of Bodywork and Movement Therapies*, *22*. https://doi.org/10.1016/j.jbmt.2017.10.014

Dubin, A. E., & Patapoutian, A. (2010). Nociceptors: the sensors of the pain pathway. *The Journal of Clinical Investigation*, *120*(11), 3760. https://doi.org/10.1172/JCI42843

Dunn, A. S., Passmore, S. R., Burke, J., & Chicoine, D. (2009). A cross-sectional analysis of clinical outcomes following chiropractic care in veterans with and without post-traumatic stress disorder. *Military Medicine*, *174*(6), 578–583. https://doi.org/10.7205/milmed-d-02-3508

Eda, N., Ito, H., & Akama, T. (2020). Beneficial effects of yoga stretching on salivary stress hormones and parasympathetic nerve activity. *Journal of Sports Science and Medicine*, *19*, 695–702. https://pubmed.ncbi.nlm.nih.gov/33239943/

Evans, K., Refshauge, K. M., Adams, R., & Aliprandi, L. (2005). Predictors of low back pain in young elite golfers: A preliminary study. *Physical Therapy in Sport*, *6*(3), 122–130. https://doi.org/10.1016/j.ptsp.2005.05.003

Farinatti, P. T. V., Brandão, C., Soares, P. P. S., & Duarte, A. F. A. (2011). Acute effects of stretching exercise on the heart rate variability in subjects with low flexibility levels. *Journal of Strength and Conditioning Research*, *25*(6), 1579–1585. https://doi.org/10.1519/JSC.0b013e3181e06ce1

Fasuyi, F. O., Fabunmi, A. A., & Adegoke, B. O. A. (2017). Hamstring muscle length and pelvic tilt range among individuals with and without low back pain. *Journal of Bodywork and Movement Therapies*, *21*(2), 246–250. https://doi.org/10.1016/J.JBMT.2016.06.002

Fatoye, F., Gebrye, T., & Odeyemi, I. (2019). Real-world incidence and prevalence of low back pain using routinely collected data. *Rheumatology International*, *39*(4), 619–626. https://doi.org/10.1007/s00296-019-04273-0

Feigl, G. C., Kastner, M., Ulz, H., Breschan, C., Pixner, T., Dreu, M., Umschaden, H. W., & Likar, R. (2013). The lumbar sympathetic trunk: its visibility and distance to two anatomical landmarks. *Surgical and Radiologic Anatomy*, *35*(2), 99–106. https://doi.org/10.1007/s00276-012-1015-y

Ferber, R., Kendall, K. D., & McElroy, L. (2010). Normative and critical criteria for iliotibial band and iliopsoas muscle flexibility. *Journal of Athletic Training*, *45*(4), 344–348. https://doi.org/10.4085/1062-6050-45.4.344

Ferrer-Montiel, A., Haberberger, R. V., Macefield, V. G., Burton, A. R., & Fazalbhoy, A. (2016). Sympathetic responses to noxious stimulation of muscle and skin. *Frontiers in Neurology, 7*, 109. https://doi.org/10.3389/fneur.2016.00109

Fisher, J. P., Zera, T., & Paton, J. F. R. (2022). Respiratory–cardiovascular interactions. *Handbook of Clinical Neurology*, *188*, 279–308. https://doi.org/10.1016/B978-0-323-91534-2.00006-0

Fraeulin, L., Holzgreve, F., Brinkbäumer, M., Dziuba, A., Friebe, D., Klemz, S., Schmitt, M., Anna-Lena Theis, A., Tenberg, S., van Mark, A., Maurer-Grubinger, C., & 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), e0243646. https://doi.org/10.1371/journal.pone.0243646

Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, *30*(10), 1010–1016. https://doi.org/10.1016/J.PSYNEUEN.2005.04.006

Fuchs, F. D., & Whelton, P. K. (2020). High blood pressure and cardiovascular disease. *Hypertension*, 285–292. https://doi.org/10.1161/HYPERTENSIONAHA.119.14240

Gallo, L. C., Roesch, S. C., Fortmann, A. L., Carnethon, M. R., Penedo, F. J., Perreira, K., Birnbaum-Weitzman, O., Wassertheil-Smoller, S., Castañeda, S. F., Talavera, G. A., Sotres-Alvarez, D., Daviglus, M. L., Schneiderman, N., & Isasi, C. R. (2014). Associations of chronic stress burden, perceived stress, and traumatic stress with cardiovascular disease prevalence and risk factors in the Hispanic community health study/study of Latinos sociocultural ancillary study. *Psychosomatic Medicine*, *76*(6), 468–475. https://doi.org/10.1097/psy.0000000000000069

Ganesan, S., Acharya, A. S., Chauhan, R., & Acharya, S. (2017). Prevalence and risk factors for low back pain in 1,355 young adults: A cross-sectional study. *Asian Spine Journal*, *11*(4), 610–617. https://doi.org/10.4184/asj.2017.11.4.610

Garde, A. H., & Hansen, Å. M. (2009). Long-term stability of salivary cortisol. *Scandinavian Journal of Clinical and Laboratory Investigation*, *65*(5), 433–436. https://doi.org/10.1080/00365510510025773

Gasibat, Q., & Suwehli, W. (2017). Determining the benefits of massage mechanisms: A review of literature. *Article in Journal of Rehabilitation Sciences*, *2*(3), 58–67. https://doi.org/10.11648/j.rs.20170203.12

Geer, E. B., Islam, J., & Buettner, C. (2014). Mechanisms of glucocorticoid-induced insulin resistance: focus on adipose tissue function and lipid metabolism. *Endocrinology and Metabolism Clinics of North America*, *43*(1), 75. https://doi.org/10.1016/j.ecl.2013.10.005

Glaros, A. G., Marszalek, J. M., & Williams, K. B. (2016). Longitudinal multilevel modeling of facial pain, muscle tension, and stress. *Journal of Dental Research*, *95*(4), 416–422. https://doi.org/10.1177/0022034515625216

Grace-Martin, K. (n.d.). *Missing Data: Two big problems with mean imputation - The analysis factor*. Retrieved April 3, 2023, from https://www.theanalysisfactor.com/mean-imputation/

Grasser, L., Al-Saghir, H., Wanna, C., Spinnei, J., & Javanbakht, A. (2019). Moving through the trauma: dance/movement therapy as a somatic-based intervention for addressing trauma and stress among syrian refugee children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *58*(11), 24–26. https://doi.org/10.1016/j.jaac.2019.07.007

Gray, H. (2009). *Gray's anatomy: with original illustrations by Henry Carter*. Arcturus Publishing.

Gray, H., Pick, T. P., & Howden, R. (1974). *Anatomy, descriptive and surgical*. Running Press.

Gray391 - *File:Gray391.png - Wikimedia Commons*. (n.d.). Retrieved October 22, 2022, from https://commons.wikimedia.org/wiki/File:Gray391.png#/media/File:Gray391.png

Greiser, K. H., Kluttig, A., Schumann, B., Swenne, C. A., Kors, J. A., Kuss, O., Haerting, J., Schmidt, H., Thiery, J., & Werdan, K. (2009). Cardiovascular diseases, risk factors and short-term heart rate variability in an elderly general population: the CARLA study 2002-2006. *European Journal of Epidemiology*, *24*(3), 123–142. https://doi.org/10.1007/s10654-009-9317-z

Hallman, D. M., & Lyskov, E. (2012a). Autonomic regulation, physical activity and perceived stress in subjects with musculoskeletal pain: 24-hour ambulatory monitoring. *International Journal of Psychophysiology*, *86*(3), 276–282. https://doi.org/10.1016/j.ijpsycho.2012.09.017

Hallman, D. M., & Lyskov, E. (2012b). Autonomic regulation, physical activity and perceived stress in subjects with musculoskeletal pain: 24-hour ambulatory monitoring. *International Journal of Psychophysiology*, *86*(3), 276–282. https://doi.org/10.1016/j.ijpsycho.2012.09.017

Hanna, T. (1988). *Somatics: Reawakening the mind's control of movement, flexibility, and health*. Addison-Wesley Reading, MA.

Hannibal, K. E., & Bishop, M. D. (2014). Chronic stress, cortisol dysfunction, and pain: A psychoneuroendocrine rationale for stress management in pain rehabilitation. *Physical Therapy*, *94*(12), 1816–1825. https://doi.org/10.2522/ptj.20130597

Hartvigsen, J., Hancock, M. J., Kongsted, A., Louw, Q., Ferreira, M. L., Genevay, S., Hoy, D., Karppinen, J., Pransky, G., Sieper, J., Smeets, R. J., Underwood, M., Buchbinder, R., Hartvigsen, J., Cherkin, D., Foster, N. E., Maher, C. G., Underwood, M., van Tulder, M., … 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

HAZLETT, R. L., McLEOD, D. R., & HOEHN-SARIC, R. (1994). Muscle tension in generalized anxiety disorder: Elevated muscle tonus or agitated movement? *Psychophysiology*, *31*(2), 189–195. https://doi.org/10.1111/J.1469-8986.1994.TB01039.X

Heart and Stroke Foundation. *HeartrRisk & prevention* (n.d.). Retrieved March 27, 2023, from https://www.heartandstroke.ca/heart-disease/risk-and-prevention

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology*, *25*(1), 1–35. https://doi.org/10.1016/S0306-4530(99)00035-9

Herman, J. P. (2022). The neuroendocrinology of stress: Glucocorticoid signaling mechanisms. *Psychoneuroendocrinology*, *137*, 105641. https://doi.org/10.1016/j.psyneuen.2021.105641

Hillebrand, S., Gast, K. B., de Mutsert, R., Swenne, C. A., Jukema, J. W., Middeldorp, S., Rosendaal, F. R., & Dekkers, O. M. (2013). Heart rate variability and first cardiovascular event in populations without known cardiovascular disease: Meta-analysis and dose–response meta-regression. *Europace*, *15*(5), 742–749.

Hittner, J. B., May, K., & Silver, N. C. (2010). A monte carlo evaluation of tests for comparing dependent correlations. *Journal of General Psychology, 130(*2), 149-168. https://doi.org/10.1080/00221300309601282

Hoy, D., Bain, C., Williams, G., March, L., Brooks, P., Blyth, F., Woolf, A., Vos, T., & Buchbinder, R. (2012). A systematic review of the global prevalence of low back pain. *Arthritis & Rheumatism*, *64*(6), 2028–2037. https://doi.org/10.1002/art.34347

Hoy, D., March, L., Brooks, P., Woolf, A., Blyth, F., Vos, T., & Buchbinder, R. (2010). Measuring the global burden of low back pain. *Best Practice & Research Clinical Rheumatology*, *24*(2), 155–165. https://doi.org/10.1016/j.berh.2009.11.002

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*, *14*(12), 1964–1970. https://doi.org/10.1111/pme.12224

Ingber, R. S. (1989). Iliopsoas myofascial dysfunction: A treatable cause of "failed" low back syndrome. *Archives of Physical Medicine and Rehabilitation*, *70*(5), 382–386. https://pubmed.ncbi.nlm.nih.gov/2524183/

Ishihara, K. (2009). Spinal imaging abnormality, low back and leg pain, and muscle tension—A five-phase hypothesis considering generative sequence and causal relationship. *Medical Hypotheses*, *73*(5), 698–702. https://doi.org/10.1016/j.mehy.2009.05.006

Jiroumaru, T., Kurihara, T., & Isaka, T. (2014). Measurement of muscle length-related electromyography activity of the hip flexor muscles to determine individual muscle contributions to the hip flexion torque. *SpringerPlus*, *3*(1). https://doi.org/10.1186/2193-1801-3-624

Joseph, J. J., & Golden, S. H. (2017). Cortisol dysregulation: The bidirectional link between stress, depression, and type 2 diabetes mellitus. *Annals of the New York Academy of Sciences*, *1391*(1), 20–34. https://doi.org/10.1111/nyas.13217

Kalezic, N., Åsell, M., Kerschbaumer, H., & Lyskov, E. (2010). Physiological reactivity to functional tests in patients with chronic low back pain. *Journal of Musculoskeletal Pain, 15*(1), 29-40.https://doi.org/10.1300/j094v15n01_05

Kanemura, T., Satake, K., Nakashima, H., Segi, N., Ouchida, J., Yamaguchi, H., & Imagama, S. (2017). Understanding retroperitoneal anatomy for lateral approach spine surgery. *Spine Surgery and Related Research*, *1*(3), 107–120. https://doi.org/10.22603/ssrr.1.2017-0008

Kang, H. (2013). The prevention and handling of the missing data. *Korean Journal of Anesthesiology*, *64*(5), 402. https://doi.org/10.4097/KJAE.2013.64.5.402

Kaufman, M. P., & Hayes, S. G. (2002). The exercise pressor reflex. *Clinical Autonomic Research*, *12*(6), 429–439. https://doi.org/10.1007/s10286-002-0059-1/metrics

Keleman, S. (1985). *Emotional anatomy: The structure of experience*. Center Press (Berkeley, CA).

Kemp, A. H., & Quintana, D. S. (2013). The relationship between mental and physical health: Insights from the study of heart rate variability. In *International Journal of Psychophysiology* (Vol. 89, Issue 3, pp. 288–296). Elsevier. https://doi.org/10.1016/j.ijpsycho.2013.06.018

Kendall, F., McCreary, E., & Provance, P. (1993). Manual Muscle Testing. In J. Butler (Ed.), *Muscles Testing and Function* (Fourth Ed., pp. 4–5). Lippincott Williams and Wilkins.

Kim, H.-G., Cheon, E.-J., Bai, D.-S., Lee, Y. H., & Koo, B.-H. (2018). Stress and heart rate variability: A meta-analysis and review of the literature. *Psychiatry Investigation*, *15*(3), 235–245. https://doi.org/10.30773/pi.2017.08.17

Kim, T.-S., Pae, C.-U., Hong, C.-K., Kim, J.-J., Lee, C.-U., Lee, S.-J., Paik, I.-H., & Lee, C. (2006). Interrelationships among pain, disability, and psychological factors in young Korean conscripts with lumbar disc herniation. *Military Medicine*, *171*(11), 1113–1116. https://doi.org/10.7205/milmed.171.11.1113

Kim, W. D., & Shin, D. C. (2020). Correlations between hip extension range of motion, hip extension asymmetry, and compensatory lumbar movement in patients with nonspecific chronic low back pain. *Medical Science Monitor: International Medical Journal of Experimental and Clinical Research*, *26*. https://doi.org/10.12659/msm.925080

Kitamura, G., Tateuchi, H., & Ichihashi, N. (2019). Greater lumbar extension during dolphin kick and psoas major tightness in swimmers with low back pain. *Journal of Sport Rehabilitation*, *29*(6), 716–722. https://doi.org/10.1123/JSR.2018-0262

Koch, L. (2012). *The psoas book*. Guinea Pig publications.

Koes, B. W., van Tulder, Mw., & Thomas, S. (2006). Diagnosis and treatment of low back pain. *British Medical Journal*, *332*(7555), 1430–1434. https://www.jstor.org/stable/25689624

Kozlowska, K., Walker, P., McLean, L., & Carrive, P. (2015). Fear and the defense cascade. *Harvard Review of Psychiatry*, *23*(4), 263–287. https://doi.org/10.1097/hrp.0000000000000065

Kreiner, D. S., Matz, P., Bono, C. M., Cho, C. H., Easa, J. E., Ghiselli, G., Ghogawala, Z., Reitman, C. A., Resnick, D. K., Watters, W. C., Annaswamy, T. M., Baisden, J., Bartynski, W. S., Bess, S., Brewer, R. P., Cassidy, R. C., Cheng, D. S., Christie, S. D., Chutkan, N. B., … Yahiro, A. M. (2020). Guideline summary review: An evidence-based clinical guideline for the diagnosis and treatment of low back pain. *The Spine Journal*, *20*(7), 998–1024. https://doi.org/10.1016/j.spinee.2020.04.006

Kumari, M., Shipley, M., Stafford, M., & Kivimaki, M. (2011). Association of diurnal patterns in salivary cortisol with all-cause and cardiovascular mortality: Findings from the Whitehall II study. *Journal of Clinical Endocrinology and Metabolism*, *96*(5), 1478–1485. https://doi.org/10.1210/jc.2010-2137

Lee, C., & Lee, S. (2021). Acute effects of foam rolling exercises on arterial stiffness, flexibility and autonomic nervous system function in young and middle-aged women. *Exercise Science*, *30*(4), 491–500. https://doi.org/10.15857/ksep.2021.00465

Lightman, S. L. (2008). The neuroendocrinology of stress: A never ending story. *Journal of Neuroendocrinology*, *20*(6), 880–884. https://doi.org/10.1111/j.1365-2826.2008.01711.x

Linton, S. J. (2000). A review of psychological risk factors in back and neck pain. *Spine*, *25*(9), 1148–1156. https://oce-ovid-com.ezproxy.library.uvic.ca/article/00007632-200005010-00017/html

Liu, M. Y., Li, N., Li, W. A., & Khan, H. (2017). Association between psychosocial stress and hypertension: A systematic review and meta-analysis. *Neurological Research; Journal of Progress in Neurosurgery, Neurology and Neurosciences*, *39*(6), 573–580. https://doi.org/10.1080/01616412.2017.1317904

Liu, Y., Zhu, S. H., Wang, G. H., Ye, F., & Li, P. Z. (2013). Validity and reliability of multiparameter physiological measurements recorded by the equivital lifemonitor during activities of various intensities. *Journal of Occupational and Environmental Hygiene*, *10*(2), 78–85. https://doi.org/10.1080/15459624.2012.747404

Loncar, Z., Curić, G., Mestrović, A. H., Mićković, V., & Bilić, M. (2013). Do IL-1B and IL-1RN modulate chronic low back pain in patients with post-traumatic stress disorder? *Collegium Antropologicum*, *37*(4), 1237–1244. http://www.ncbi.nlm.nih.gov/pubmed/24611340

Lydakis, Ch., & Sinoway, L. (2007). Sympathetic nervous system and muscle: A two way interaction in health and disease. *Αρτηριακή Υπέρταση* , *16*(1), 11–20.

Maganaris, C. N., Chatzistergos, P., Reeves, N. D., & Narici, M. V. (2017). Quantification of internal stress-strain fields in human tendon: Unraveling the mechanisms that underlie regional tendon adaptations and mal-adaptations to mechanical loading and the effectiveness of therapeutic eccentric exercise. *Frontiers in Physiology*, *8*. https://doi.org/10.3389/fphys.2017.00091

Malik, M., Bigger, J. T., Camm, A. J., Kleiger, R., Malliani, A., Moss, A., & Schwartz, P. (1996). Heart rate variability Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal*, *17*, 354–381. https://academic.oup.com/eurheartj/article-abstract/17/3/354/485572

Martini, F. H., Timmons, M. J., & Tallitsch, R. (2012). *Human Anatomy* (L. Berriman, Ed.; 7th ed.). Benjamin Cummings.

McBeth, J., Silman, A. J., Gupta, A., Chiu, Y. H., Ray, D., Morriss, R., Dickens, C., King, Y., & Macfarlane, G. J. (2007). Moderation of psychosocial risk factors through dysfunction of the hypothalamic–pituitary–adrenal stress axis in the onset of chronic widespread musculoskeletal pain: Findings of a population-based prospective cohort study. *Arthritis & Rheumatism*, *56*(1), 360–371. https://doi.org/10.1002/art.22336

McCraty, R., & Shaffer, F. (2015). Heart rate variability: New perspectives on physiological mechanisms, assessment of self-regulatory capacity, and health risk. *Global Advances in Health and Medicine*, *4*(1), 46. https://doi.org/10.7453/gahmj.2014.073

McFarlane, A. C. (2007). Stress-related musculoskeletal pain. *Best Practice & Research. Clinical Rheumatology*, *21*(3), 549–565. https://doi.org/10.1016/j.berh.2007.03.008

Mcgill, S., Jukerts, D., & Kropftii, P. (1996). Appropriately placed surface EMG electrodes reflect deep muscle activity (psoas, quadratus lumborum, abdominal wall) in the lumbar spine. *Journal of Biomechanics*, *29*(11), 1503–1507.

Mebazaa, A., Gayat, E., Lassus, J., Meas, T., Mueller, C., Maggioni, A., Peacock, F., Spinar, J., Harjola, V. P., van Kimmenade, R., Pathak, A., Mueller, T., Tavazzi, L., Disomma, S., Metra, M., Pascual-Figal, D., Laribi, S., Logeart, D., Nouira, S., … Januzzi, J. L. (2013). Association between elevated blood glucose and outcome in acute heart failure: Results from an international observational cohort. *Journal of the American College of Cardiology*, *61*(8), 820–829. https://doi.org/10.1016/j.jacc.2012.11.054

Mense, S. (2008). Muscle pain: Mechanisms and clinical significance. *Deutsches Ärzteblatt International*, *105*(12), 214. https://doi.org/10.3238/artzebl.2008.0214

Mense, S. (2010). Functional anatomy of muscle: Muscle, nociceptors and afferent fibers. In R. D. Gerwin & S. Mense (Eds.), *Muscle Pain: Understanding the mechanisms* (pp. 17–48). Springer. https://doi.org/10.1007/978-3-540-85021-2_2

Mense, S. (2013a). Muscle nociceptors, neurochemistry. In G. F. Gebhart & R. F. Schmidt (Eds.), *Encyclopedia of pain* (pp. 1944–1950). Springer. https://doi.org/10.1007/978-3-642-28753-4_2529

Mense, S. (2013b). muscle pain model, ischemia-induced and hypertonic saline-induced BT. In G. F. Gebhart & R. F. Schmidt (Eds), *Encyclopedia of pain* (pp. 1957–1963). Springer. https://doi.org/10.1007/978-3-642-28753-4_2535

Mitchell, J. H., Kaufman, M. P., & Iwamoto, G. A. (2003). The exercise pressor reflex: Its cardiovascular effects, afferent mechanisms, and central pathways. *Annual Review of Physiology, 45*, 229–242. https://doi.org/10.1146/annurev.ph.45.030183.001305

Mitchell, U. H., Owen, P. J., Rantalainen, T., & Belavý, D. L. (2022). Increased joint mobility is associated with impaired transversus abdominis contraction. *Journal of Strength and Conditioning Research*, *36*(9), 2472–2478. https://doi.org/10.1519/jsc.0000000000003752

Mizuno, T. (2017). Changes in joint range of motion and muscle–tendon unit stiffness after varying amounts of dynamic stretching. *Journal of Sports Sciences*, *35*(21), 2157–2163. https://doi.org/10.1080/02640414.2016.1260149

Moreside, J. M., & McGill, S. M. (2011). Quantifying normal 3D hip ROM in healthy young adult males with clinical and laboratory tools: Hip mobility restrictions appear to be plane-specific. *Clinical Biomechanics*, *26*(8), 824–829. https://doi.org/10.1016/j.clinbiomech.2011.03.015

Morling, G. (2009). Understanding iliopsoas: clinical implications for the massage therapist. *Journal of the Australian Traditional-Medicine Society*, *15*(1), 7.

Morling, G. (2014). The massage paradox: When touch causes fear (with reference to the iliopsoas), *Journal of the Australian Traditional-Medicine Society*, *20*(1), 28–30.

Muhtz, C., Rodriguez-Raecke, R., Hinkelmann, K., Moeller-Bertram, T., Kiefer, F., Wiedemann, K., May, A., & Otte, C. (2013). Cortisol response to experimental pain in patients with chronic low back pain and patients with major depression. *Pain Medicine, 14*(4), 498–503. https://doi.org/10.1111/J.1526-4637.2012.01514.x

Munoz, M. L., Van Roon, A., Riese, H., Thio, C., Oostenbroek, E., Westrik, I., De Geus, E. J. C., Gansevoort, R., Lefrandt, J., Nolte, I. M., & Snieder, H. (2015). Validity of (ultra-)short recordings for heart rate variability measurements. *PLoS ONE*, *10*(9). https://doi.org/10.1371/journal.pone.0138921

Murayama, M., Watanabe, K., Kato, R., Uchiyama, T., & Yoneda, T. (2012). Association of muscle hardness with muscle tension dynamics: a physiological property. *European Journal of Applied Physiology*, *112*(1), 105–112. https://doi.org/10.1007/s00421-011-1959-3

Myers, T., & Garcia, T. (2021). *Lumbopelvic Stability Live Dissection*.

Myers, T. W. (1996). The 'anatomy trains'. *Journal of Bodywork and Movement Therapies*, *1*(2), 91–101. https://doi.org/10.1016/S1360-8592(97)80010-1

Nachemson, A. (1966). Electromyographic studies on the vertebral portion of the psoas muscle. *Acta Orthopaedica Scandinavica*, *37*(2), 177–190. https://doi.org/10.3109/17453676608993277

Nakamura, M., Konrad, A., Kiyono, R., Sato, S., Yahata, K., Yoshida, R., Yasaka, K., Murakami, Y., Sanuki, F., & Wilke, J. (2021). Local and non-local effects of foam rolling

on passive soft tissue properties and spinal excitability. *Frontiers in Physiology*, *12*, 859. https://doi.org/10.3389/fphys.2021.702042/bibtex

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

NG, S. K. (2019). The effects of 12-week yoga practice on flexibility and dynamic balance of female college students. *Medicine & Science in Sports & Exercise*, *51*(6S), 961–962. https://doi.org/10.1249/01.mss.0000563387.96188.3e

Norman, G. J., Berntson, G. G., & Cacioppo, J. T. (2014). Emotion, somatovisceral afference, and autonomic regulation. *Emotion Review*, *6*(2), 113–123. https://doi.org/10.1177/1754073913512006

Nunan, D., Sandercock, G. R. H., & Brodie, D. A. (2010). A quantitative systematic review of normal values for short-term heart rate variability in healthy adults. *Pacing and Clinical Electrophysiology*, *33*(11), 1407–1417. https://doi.org/10.1111/J.1540-8159.2010.02841.x

Nyboe, L., Bentholm, A., & Gyllensten, A. L. (2017). Bodily symptoms in patients with post traumatic stress disorder: A comparative study of traumatized refugees, Danish war veterans, and healthy controls. *Journal of Bodywork and Movement Therapies*, *21*(3), 523–527. https://doi.org/10.1016/j.jbmt.2016.08.003

Oliveira, C. B., Maher, C. G., Pinto, R. Z., Traeger, A. C., Lin, C. W. C., Chenot, J. F., van Tulder, M., & Koes, B. W. (2018). Clinical practice guidelines for the management of non-specific low back pain in primary care: an updated overview. *European Spine Journal*, *27*(11), 2791–2803. https://doi.org/10.1007/S00586-018-5673-2/tables/6

Pascoe, M. C., Thompson, D. R., & Ski, C. F. (2017). Yoga, mindfulness-based stress reduction and stress-related physiological measures: A meta-analysis. *Journal of Psychoneuroendocrinology,86,* 152-168. https://doi.org/10.1016/j.psyneuen.2017.08.008

Payne, P., & Crane-Godreau, M. A. (2015). The preparatory set: A novel approach to understanding stress, trauma, and the bodymind therapies. *Frontiers in Human Neuroscience*, *9.* https://doi.org/10.3389/fnhum.2015.00178

Payne, P., Levine, P. A., & Crane-Godreau, M. A. (2015). Somatic experiencing: Using interoception and proprioception as core elements of trauma therapy. *Frontiers in Psychology*, *6*. https://doi.org/10.3389/fpsyg.2015.00093

Peeler, J., & Anderson, J. E. (2007). Reliability of the Thomas test for assessing range of motion about the hip. *Physical Therapy in Sport*, *8*(1), 14–21. https://doi.org/10.1016/j.ptsp.2006.09.023

Perini, R., & Veicsteinas, A. (2003). Heart rate variability and autonomic activity at rest and during exercise in various physiological conditions. *European Journal of Applied Physiology*, *90*, 317–325. https://doi.org/10.1007/s00421-003-0953-9

Pinar, R., Afsar, F., & H. (2015). Back massage to decrease state anxiety, cortisol level, blood pressure, heart rate and increase sleep quality in family caregives of patients with cancer: a randomized controlled trial. *Asian Pacific Journal of Cancer Prevention*, *16*(18), 8127–8133. https://doi.org/10.7314/apjcp.2015.16.18.8127

Polar's 40 years of incredible firsts | Polar Blog. (n.d.). Retrieved April 17, 2020, from https://www.polar.com/blog/40-years-of-incredible-firsts-polar-history/

Pontes-Silva, A., Bassi-Dibai, D., Fidelis-De-Paula-Gomes, C. A., da Silva Souza, C., de Oliveira Pires, F., Mostarda, C. T., & Dibai-Filho, A. V. (2022). Comparison of the

autonomic nervous system dysfunction between different chronic spine disorders: Neck pain versus low back pain. *Revista Da Associação Médica Brasileira*, *68*(9), 1288–1296. https://doi.org/10.1590/1806-9282.20220406

Prim, J. H., Ahn, S., Davila, M. I., Alexander, M. L., McCulloch, K. L., & Fröhlich, F. (2019). Targeting the autonomic nervous system balance in patients with chronic low back pain using transcranial alternating current stimulation: a randomized, crossover, double-blind, placebo-controlled pilot study. *Journal of Pain Research*, *12*, 3265–3277. https://doi.org/10.2147/JPR.S208030

Pulopulos, M. M., Vanderhasselt, M. A., & De Raedt, R. (2018). Association between changes in heart rate variability during the anticipation of a stressful situation and the stress-induced cortisol response. *Psychoneuroendocrinology*, *94*, 63. https://doi.org/10.1016/J.psyneuen.2018.05.004

Puschmann, A. K., Drießlein, D., Beck, H., Arampatzis, A., Catalá, M. M., Schiltenwolf, M., Mayer, F., & Wippert, P. M. (2020). Stress and self-efficacy as long-term predictors for chronic low back pain: A prospective longitudinal study. *Journal of Pain Research*, *13*, 613–621. https://doi.org/10.2147/JPR.S223893

Raff, H., Homar, P. J., & Burns, E. A. (2002). Comparison of two methods for measuring salivary cortisol. *Clinical Chemistry*, *48*(1), 207–208. https://doi.org/10.1093/clinchem/48.1.207

Raptopoulos, V., Kleinman, P. K., Marks Jr, S., Snyder, M., & Silverman, P. M. (1986). Renal fascial pathway: Posterior extension of pancreatic effusions within the anterior pararenal space. *Radiology*, *158*(2), 367–374.

Reeder, B., & David, A. (2016). Health at hand: A systematic review of smart watch uses for health and wellness. *Journal of Biomedical Informatics*, *63*, 269–276. https://doi.org/10.1016/j.jbi.2016.09.001

Reiman, M. P., & Matheson, J. W. (2013). Restricted hip mobility: Clinical suggestions for self-mobilization and muscle re-education. *International Journal of Sports Physical Therapy*, *8*(5), 729-740. https://pubmed.ncbi.nlm.nih.gov/24175151/

Rempel, D., Dahlin, L., & Lundborg, G. (1999). *Biological response of peripheral nerves to loading: pathophysiology of nerve compression syndromes and vibration induced neuropathy*. National Academies Press. https://www.ncbi.nlm.nih.gov/books/nbk230871/

Reyes-Ferrada, W., Chirosa-Rios, L., Rodriguez-Perea, A., Jerez-Mayorga, D., & Chirosa-Rios, I. (2021). Isokinetic trunk strength in acute low back pain patients compared to healthy subjects: A systematic review. *International Journal of Environmental Research and Public Health*, *18*(5), 1–13. https://doi.org/10.3390/ijerph18052576

Roach, S. M., Juan, J. G. S., 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. https://pubmed.ncbi.nlm.nih.gov/25709858/

Roach, S., 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*, *5*, 680–688. https://pubmed.ncbi.nlm.nih.gov/24175147/

Rocco, A. G., Palombi, D., & Raeke, D. (1995). Anatomy of the lumbar sympathetic chain. *Regional Anesthesia*, *20*(1), 13–19.

Rodrigues, E., Lima, D., Barbosa, P., Gonzaga, K., Guerra, R. O., Pimentel, M., Barbosa, H., & Maciel, Á. (2022). HRV monitoring using commercial wearable devices as a health indicator for older persons during the pandemic. *Sensors, 22*(5), 2001. https://doi.org/10.3390/s22052001

Roman-Liu, D., Grabarek, I., Bartuzi, P., & Choromański, W. (2013). The influence of mental load on muscle tension. *Ergonomics*, *56*(7), 1125–1133. https://doi.org/10.1080/00140139.2013.798429

Russell, G., & Lightman, S. (2019). The human stress response. *Nature Reviews Endocrinology*, *15*(9), 525–534. https://doi.org/10.1038/s41574-019-0228-0

Ryan, R., Booth, S., Spathis, A., Mollart, S., & Clow, A. (2016). Use of salivary diurnal cortisol as an outcome measure in randomised controlled trials: A systematic review. *Annals of Behavioral Medicine*, *50*(2), 210–236. https://doi.org/10.1007/s12160-015-9753-9

Sajko, S., & Stuber, K. (2009). Psoas major: A case report and review of its anatomy, biomechanics, and clinical implications. *The Journal of the Canadian Chiropractic Association*, *53*(4), 311–318. http://www.ncbi.nlm.nih.gov/pubmed/20037696

Sakada, S. (1974). Mechanoreceptors in fascia, periosteum and periodontal ligament. *The Bulletin of Tokyo Medical and Dental University*, *21*, 11–13.

Salberg, S., Sgro, M., Brady, R. D., Noel, M., & Mychasiuk, R. (2020). The development of adolescent chronic pain following traumatic brain injury and surgery: The role of diet and early life stress. *Developmental Neuroscience*, *42*(1), 2–11. https://doi.org/10.1159/000508663

Salimetrics. (2021a). *Expanded range high sensitivity salivary cortisol enzyme immunoassay kit* (pp. 1–21). https://salimetrics.com/wp-content/uploads/2018/03/salivary-cortisol-elisa-kit.pdf

Salimetrics. (2021b). *Collection methods: Passive drool using the saliva collection aid*. https://salimetrics.com/wp-content/uploads/2018/02/passive-drool-saliva-collection-instructions.pdf

Santaguida, P. L., & McGill, S. M. (1995). The psoas major muscle: A three-dimensional geometric study. *Journal of Biomechanics*, *28*(3), 339–345. https://doi.org/10.1016/0021-9290(94)00064-B

Sayers, B. (1973). Analysis of heart rate variability. *Ergonomics*, *16*(1), 17–32. https://doi.org/10.1080/00140137308924479

Scaer, R. (2014). *The body bears the burden: Trauma, dissociation, and disease* (3rd ed.). Routledge. https://doi-org.ezproxy.library.uvic.ca/10.4324/9780203081822

Schleip, R. (2017, June 1). Fascia as a sensory organ: Clinical applications. *Terra Rosa E-Mag*, *20*, 2–7.

Schleip, R., Hedley, G., & Yucesoy, C. A. (2019). Fascial nomenclature: Update on related consensus process. *Clinical Anatomy, 32*(7), 929. https://doi.org/10.1002/ca.23423

Schleip, R., Zorn, A., & Klingler, W. (2010). Biomechanical properties of fascial tissues and their role as pain generators. *Journal of Musculoskeletal Pain*, *18*(4), 393–395. https://doi.org/10.3109/10582452.2010.502628

Schuman, D. L., & Killian, M. O. (2019). Pilot study of a single session heart rate variability biofeedback intervention on veterans' posttraumatic stress symptoms. *Applied Psychophysiology Biofeedback*, *44*(1), 9–20. https://doi.org/10.1007/s10484-018-9415-3

Seeman, T. E. (1997). Price of adaptation—Allostatic load and its health consequences. *Archives of Internal Medicine*, *157*(19), 2259. https://doi.org/10.1001/archinte.1997.00440400111013

Seyedhoseinpoor, T., Taghipour, M., Dadgoo, M., Ebrahimi Takamjani, I., Sanjari, M. A., Kazemnejad, A., M. Elliott, J., & Hides, J. (2022). Relationship between the morphology and composition of the lumbar paraspinal and psoas muscles and lumbar intervertebral motion in chronic low-back pain: An exploratory study. *Clinical Anatomy*, *35*(6), 762–772. https://doi.org/10.1002/CA.23893

Shaffer, F., & Ginsberg, J. P. (2017). An overview of heart rate variability metrics and norms. *Frontiers in Public Health*, *5*. https://doi.org/10.3389/fpubh.2017.00258

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

Shahidi, B., Haight, A., & Maluf, K. (2013). Differential effects of mental concentration and acute psychosocial stress on cervical muscle activity and posture. *Journal of Electromyography and Kinesiology*, *23*(5), 1082–1089. https://doi.org/10.1016/j.jelekin.2013.05.009

Shankar, N., Thakur, M., Tandon, O. P., Saxena, A. K., Arora, S., & Bhattacharya, N. (2011). Autonomic status and pain profile in patients of chronic low back pain and following electro acupuncture therapy: a randomized control trial. *Indian Journal of Physiology and Pharmacology*, *55*(1), 25–36. https://europepmc.org/article/med/22315807

Shin, D. (2020). Correlation between non-specific chronic low back pain and physical factors of lumbar and hip joint in office workers. *Medical Hypotheses*, *144*, 110304. https://doi.org/10.1016/j.mehy.2020.110304

Shirtcliff, E. A., Buck, R. L., Laughlin, M. J., Hart, T., Cole, C. R., & Slowey, P. D. (2015). Salivary cortisol results obtainable within minutes of sample collection correspond with traditional immunoassays. *Clinical Therapeutics*, *37*(3), 505–514. https://doi.org/10.1016/j.clinthera.2015.02.014

Siccardi, M. A., Tariq, M. A., & Valle, C. (2021). Anatomy, bony pelvis and lower limb, psoas major. *StatPearls*. https://www.ncbi.nlm.nih.gov/books/nbk535418/

Sim, I. W., & Webb, T. (2004). Anatomy and anaesthesia of the lumbar somatic plexus. *Anaesthesia and Intensive Care*, *32*(2), 178–187. https://doi.org/10.1177/0310057x0403200204

Singh, O., & Al Khalili, Y. (2022). Anatomy, back, lumbar plexus. In *National Library of Medicine*. StatPearls Publishing. http://www.ncbi.nlm.nih.gov/pubmed/31424721

Smith, S. A., Leal, A. K., Murphy, M. N., Downey, R. M., & Mizuno, M. (2015). Muscle mechanoreflex overactivity in hypertension: a role for centrally-derived nitric oxide. *Autonomic Neuroscience: Basic & Clinical*, *188*, 58. https://doi.org/10.1016/j.autneu.2014.12.004

Statistics Canada. (2004a). *Fact-sheet on work absences*. https://www150.statcan.gc.ca/n1/en/pub/75-001-x/10304/5990-eng.pdf?st=SfKb1Eaq

Statistics Canada. (2004b). *Perspective on Labour and Industry: Fact-sheet on work absences*. https://www150.statcan.gc.ca/n1/en/pub/75-001-x/10304/5990-eng.pdf?st=SfKb1Eaq

Statistics Canada. (2018). *Perceived life stress, by age group*. https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1310009604

Statistics Canada (2019a). *Population estimates on July 1st, by age and sex*. https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1710000501

Statistic Canada. (2019b). *Work absence of full-time employees by public and private sector, annual*. Table 14-10-. https://doi.org/https://doi.org/10.25318/1410019601-eng

Statistics Canada. (n.d.). *Table 14-10-0134-01. Average weekly earnings, average hourly wage rate and average usual weekly hours by union status, annual*. https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1410013401

Staugaard-Jones, J. A. (2018). *The vital psoas muscle: Connecting physical, emotional, and spiritual well-being*. North Atlantic Books.

Stubbs, B., Koyanagi, A., Thompson, T., Veronese, N., Carvalho, A. F., Solomi, M., Mugisha, J., Schofield, P., Cosco, T., Wilson, N., & Vancampfort, D. (2016a). The epidemiology of back pain and its relationship with depression, psychosis, anxiety, sleep disturbances, and stress sensitivity: Data from 43 low- and middle-income countries. *General Hospital Psychiatry*, *43*, 63–70. https://doi.org/10.1016/j.genhosppsych.2016.09.008

Stubbs, B., Koyanagi, A., Thompson, T., Veronese, N., Carvalho, A. F., Solomi, M., Mugisha, J., Schofield, P., Cosco, T., Wilson, N., & Vancampfort, D. (2016b). The epidemiology of back pain and its relationship with depression, psychosis, anxiety, sleep disturbances, and stress sensitivity: Data from 43 low-and middle-income countries. *General Hospital Psychiatry*, *43*, 63–70. https://doi.org/10.1016/j.genhosppsych.2016.09.008

Sudhaus, S., Fricke, B., Schneider, S., Stachon, A., Klein, H., Von Düring, M., & Hasenbring, M. (2007). The cortisol awakening response in patients with acute and chronic low back

pain. Relations with psychological risk factors of pain chronicity. *Der Schmerz, 21*(3), 202–211. https://doi.org/10.1007/S00482-006-0521-4

Sullivan, M., Carberry, A., Evans, E. S., Hall, E. E., & Nepocatych, S. (2019). The effects of power and stretch yoga on affect and salivary cortisol in women. *Journal of Health Psychology*, *24*(12), 1658–1667. https://doi.org/10.1177/1359105317694487

Sveinsdottir, V., Eriksen, H. R., Ursin, H., Hansen, Å. M., & Harris, A. (2016). Cortisol, health, and coping in patients with nonspecific low back pain. *Applied Psychophysiology and Biofeedback*, *41*(1), 9–16. https://doi.org/10.1007/s10484-015-9300-2

Tan, G., Dao, T. K., Farmer, L., Sutherland, R. J., & Gevirtz, R. (2011). Heart rate variability (HRV) and post-traumatic stress disorder (PTSD): A pilot study. *Applied Psychophysiology and Biofeedback*, *36*(1), 27–35. https://doi.org/10.1007/s10484-010-9141-y

Tarride, J. E., Lim, M., DesMeules, M., Luo, W., Burke, N., O'Reilly, D., Bowen, J., & Goeree, R. (2009). A review of the cost of cardiovascular disease. *Canadian Journal of Cardiology*, *25*(6), 195. https://doi.org/10.1016/S0828-282X(09)70098-4

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. *European Heart Journal*, *17*(3), 354–381.

Tekur, P., Nagarathna, R., Chametcha, S., Hankey, A., & Nagendra, H. R. (2012). A comprehensive yoga programs improves pain, anxiety and depression in chronic low back pain patients more than exercise: An RCT. *Complementary Therapies in Medicine*, *20*, 107–118. https://doi.org/10.1016/j.ctim.2011.12.009

Thayer, J. F., Yamamoto, S. S., & Brosschot, J. F. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology*, *141*(2), 122–131. https://doi.org/10.1016/j.ijcard.2009.09.543

The top 10 causes of death. (n.d.). Retrieved September 18, 2022, from https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death

Thomas, J. R., Nelson, J., & Silverman, S. J. (2015). *Research methods in physical activity*. Human Kinetics.

Tonkin, M. (2010). Nerve compression syndromes. In P. Sambrook, L. Schrieber, T. Taylor, & E. Ellis (Eds.), *Musculoskeletal System* (2nd Ed., pp. 33–45). Churchill Livingstone. https://doi.org/10.1016/b978-0-7020-3377-3.00003-2

Tortora, G., & Grabowski, S. (1996). *Principles of anatomy and physiology, 8th Editon*. John Wiley and Sons Inc.

Tosato, J. D. P., Caria, P. H. F., Gomes, C. A. F. D. P., Berzin, F., Politti, F., Gonzalez, T. D. O., & Biasotto-Gonzalez, D. A. (2015). Correlation of stress and muscle activity of patients with different degrees of temporomandibular disorder. *Journal of Physical Therapy Science*, *27*(4), 1227–1231. https://doi.org/10.1589/jpts.27.1227

Treede, R. D., Rief, W., Barke, A., Aziz, Q., Bennett, M. I., Benoliel, R., Cohen, M., Evers, S., Finnerup, N. B., First, M. B., Giamberardino, M. A., Kaasa, S., Korwisi, B., Kosek, E., Lavand'Homme, P., Nicholas, M., Perrot, S., Scholz, J., Schug, S., … Wang, S. J. (2019). Chronic pain as a symptom or a disease: The IASP classification of chronic pain for the international classification of diseases (ICD-11). *Pain*, *160*(1), 19–27. https://doi.org/10.1097/j.pain.0000000000001384

Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research*, *53*(4), 865–871. https://doi.org/10.1016/S0022-3999(02)00429-4

Tsigos, C., Kyrou, I., Kassi, E., & Chrousos, G. P. (2020). Stress: Endocrine physiology and pathophysiology. *Endotext*. https://www.ncbi.nlm.nih.gov/books/nbk278995/

Tsuji, H., Venditti, F. J., Manders, E. S., Evans, J. C., Larson, M. G., Feldman, C. L., & Levy, D. (1994). Reduced heart rate variability and mortality risk in an elderly cohort: The Framingham heart study. *Circulation*, *90*(2), 878–883. https://doi.org/10.1161/01.cir.90.2.878

Vachon-Presseau, E., Roy, M., Martel, M.-O., Caron, E., Marin, M.-F., Chen, J., Albouy, G., Plante, I., Sullivan, M. J., & Lupien, S. J. (2013). The stress model of chronic pain: Evidence from basal cortisol and hippocampal structure and function in humans. *Brain*, *136*(3), 815–827. https://doi.org/10.1093/brain/aws371

Vanitallie, T. (2002). Stress: A risk factor for serious illness. *Metabolism*, *51*(6), 40–45. https://doi.org/10.1053/meta.2002.33182

Vats, S., Goyal, M., & Kothiyal, S. (2021). Efficacy of iliopsoas muscle release on respiratory parameters in patients with chronic low back pain: A single blinded, two-groups, pre-test/post-test randomized controlled trial protocol. *Journal of Physiotherapy Research*, *11*(2), 411–419. https://doi.org/10.17267/2238-2704RPF.V11I2.3591

Vigotsky, A. D., Lehman, G. J., Beardsley, C., Contreras, B., Chung, B., & Feser, E. H. (2016). The modified Thomas test is not a valid measure of hip extension unless pelvic tilt is controlled. *PeerJ*, *2016*(8). https://doi.org/10.7717/peerj.2325/fig-3

Vining, R. F., Mcginley, R. A., Maksvytis, J. J., & Ho, K. Y. (1983). Salivary cortisol: a better measure of adrenal cortical function than serum cortisol. *Annals of Clinical Biochemistry*, *20*, 329–335. https://doi.org/https://journals.sagepub.com/doi/pdf/10.1177/000456328302000601

Vos, T., Abajobir, A. A., Abbafati, C., Abbas, K. M., Abate, K. H., Abd-Allah, F., Abdulle, A. M., Abebo, T. A., Abera, S. F., Aboyans, V., Abu-Raddad, L. J., Ackerman, I. N., Adamu, A. A., Adetokunboh, O., Afarideh, M., Afshin, A., Agarwal, S. K., Aggarwal, R., Agrawal, A., … Murray, C. J. L. (2017). Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990–2016: a systematic analysis for the global burden of disease study 2016. *The Lancet*, *390*(10100), 1211–1259. https://doi.org/10.1016/S0140-6736(17)32154-2

Whitworth, J. A., Williamson, P. M., Mangos, G., & Kelly, J. J. (2005). Cardiovascular consequences of cortisol excess. *Vascular Health and Risk Management*, *1*(4), 291. https://doi.org/10.2147/vhrm.2005.1.4.291

Willmann, M., Langlet, C., Hainaut, J.-P., & Bolmont, B. (2012). The time course of autonomic parameters and muscle tension during recovery following a moderate cognitive stressor: Dependency on trait anxiety level. *International Journal of Psychophysiology*, *84*(1), 51–58. https://doi.org/10.1016/j.ijpsycho.2012.01.009

Wong, A., & Figueroa, A. (2021). Effects of acute stretching exercise and training on heart rate variability: A review. *Journal of Strength and Conditioning Research*, *35*(5), 1459–1466. https://doi.org/10.1519/jsc.0000000000003084

Yoshio, M., Murakami, G., Sato, T., Sato, S., & Noriyasu, S. (2002). The function of the psoas major muscle: Passive kinetics and morphological studies using donated cadavers. *Journal of Orthopaedic Science*, *7*(2), 199–207. https://doi.org/10.1007/s007760200034

Youdas, J. W., Garrett, T. R., Egan, K. S., & Therneau, T. M. (2000). Lumbar lordosis and pelvic inclination in adults with chronic low back pain. *Physical Therapy*, *80*(3), 261–275. https://doi.org/10.1093/ptj/80.3.261

Yuzo Nakamura, F., George Behm, D., Wiewelhove, T., Döweling, A., Schneider, C., Hottenrott, L., Meyer, T., Kellmann, M., Pfeiffer, M., & Ferrauti, A. (2019). A meta-analysis of the effects of foam rolling on performance and recovery. *Frontiers in Physiology, 1*, 376. https://doi.org/10.3389/fphys.2019.00376

Zhang, D. Y., & Anderson, A. S. (2014). The sympathetic nervous system and heart failure. *Cardiology Clinics*, *32*(1), 33. https://doi.org/10.1016/J.ccl.2013.09.010

Zhu, Z., Zhang, J., Sheng, J., Zhang, C., & Xie, Z. (2020). Low back pain caused by iliopsoas tendinopathy treated with ultrasound-guided local injection of anesthetic and steroid: A retrospective study. *Journal of Pain Research*, *13*, 3023. https://doi.org/10.2147/jpr.s281880

Zügel, M., Maganaris, C. N., Wilke, J., Jurkat-Rott, K., Klingler, W., Wearing, S. C., Findley, T., Barbe, M. F., Steinacker, J. M., Vleeming, A., Bloch, W., Schleip, R., & Hodges, P. W. (2018). Fascial tissue research in sports medicine: from molecules to tissue adaptation, injury and diagnostics: Consensus statement. *British Journal of Sports Medicine*, *52*(23), 1497 LP – 1497. https://doi.org/10.1136/bjsports-2018-099308

Appendix

Appendix A: Certificate of Ethics Approval from the University of Victoria

University of Victoria
Office of Research Services I Human Research Ethics Board
Michael Williams Building Rm B202 PO Box 1700 STN CSC Victoria BC V8W 2Y2 Canada
T 250-472-4545 I F 250-721-8960 I uvic.ca/research I ethics@uvic.ca

Certificate of Approval - Annual Renewal

PRINCIPAL INVESTIGATOR:	**Lynneth Stuart-Hill** (Supervisor)	**ETHICS PROTOCOL NUMBER** Expedited review - delegated	**20-0053**
PRINCIPAL APPLICANT:	**Arielle Nash** **Master's student**	ORIGINAL APPROVAL DATE:	23-Jul-2021
		APPROVED ON:	02-Aug-2022
UVIC DEPARTMENT:	**Exercise Science, Physical and Health Education EPHE**	APPROVAL EXPIRY DATE:	22-Jul-2023

PROJECT TITLE: **Examining the relationships between stress, lower back pain and hip range of motion: An observational study**

RESEARCH TEAM MEMBERS:
Thomas Service - Research Assistant - Cortisol Analysis, PhD Candidate EPHE
Greg Mulligan - Research Assistant, Sr. Laboratory Instructor & McKinnon 171 Co-ordinator

DECLARED PROJECT FUNDING: **None**

DOCUMENTS INCLUDED IN THIS APPROVAL:
tcps2_core_certificate.pdf - 20-Oct-2020
Snowball Recruitment Email.pdf - 04-Nov-2020
Recruitment Email.pdf - 04-Nov-2020
Social Media Posts.pdf - 09-Nov-2020
Recruitment Poster 1.pdf - 09-Nov-2020
GONIOMETER.pdf - 09-Nov-2020
Research Team Daily Health Check.pdf - 13-Nov-2020
Participant Health Check.pdf - 13-Nov-2020
equivital images.docx - 17-Nov-2020
EPHE_SWP_Vera20200923 (002).pdf - 17-Nov-2020
Verification_of_Registration 2021.pdf - 28-Jan-2021
Phase 2 CAMTEC FBS SWP Bob Wright B214_ Reviewed May 22 (2).docx - 29-Jan-2021
ServiceBioSafety (1).pdf - 01-Feb-2021
Study Specific COVID Assessment V3.pdf - 10-May-2021
ANASH uvic-request-to-conduct-field-research-form1_SRH.pdf - 30-Jun-2021
Communicable Disease Prevention Plan.pdf - 06-Jul-2021
inclinometer.pdf - 09-Sep-2021
letter for implied consent V7.pdf - 09-Sep-2021
Pre-Screening Questionnaire V4.pdf - 09-Sep-2021

Conditions of approval

This Certificate of Approval is valid for the above term provided there is no change in the protocol.

Amendments
To make changes to the approved research procedure in your study, please submit "Amendments" or "Annual renewal with amendments" form. You must receive research ethics approval before proceeding with your amended protocol.

Renewals
Your ethics approval must be current for the period during which you are recruiting participants or collecting data. To renew your protocol, please submit a "Request for Renewal" form before the expiry date on your certificate. You will be sent an emailed reminder prompting you to renew your protocol about six weeks before your expiry date.

Project Closures
When you have completed all data collection activities and will have no further contact with participants, please notify the Human Research Ethics Board by submitting a "Notice of Project Completion" form.

Certification

This certifies that the UVic Human Research Ethics Board has examined this research protocol and concluded that, in all respects, the proposed research meets the appropriate standards of ethics as outlined by the University of Victoria's policies for research involving human participants.

Dr. Sandra Gibbons
Chair, Human Research Ethics Board

Dr. Matthew Murphy
Vice-chair, Human Research Ethics Board

Certificate Issued On: 02-Aug-2022

Appendix B: Snowball Recruitment Email Script

Dear (First Name/ Company Name),

I am writing to request your assistance in the recruitment of a research study taking place at the University of Victoria. The study "Examining the relationships between psoas muscle tension, stress and lower back pain", is being performed as a part of a Master of Science thesis in the school of Exercise Science, Physical and Health Education.

We are seeking 80 adults aged 18-65 who have or have not experienced back pain in the last 12 months. It is a study that may be of interest to your clients. If they have agreed to be contacted, could I send you a recruitment email to send onto your database?

Thank you.

Arielle Nash

Appendix B: Recruitment Poster 1

University of Victoria

School of Exercise Science, Physical and Health Education

LOW BACK PAIN?
RESEARCH PARTICIPANTS NEEDED:
EXAMINING THE RELATIONSHIPS AMONG HIP MOBILITY, STRESS & LOWER BACK PAIN

Who: Otherwise healthy adults aged 18-65; with back pain.

Description:

This research project aims to explore the validity of psychosomatic theories linking hip extension range of motion, stress and pain. Research evidence may be used to inform multidisciplinary approaches to pain relief, stress symptom management and stress relief.

Participation in the study requires approximately 1 hour of your time in a single visit to the satellite lab set up downtown Victoria. During the one hour, our research team will measure the state of your autonomic nervous system and hip range of motion. All measurements are non-invasive and low risk.

If you are interested in participating, you will be fully informed of all procedures and sent a consent form. To show your interest, please contact Arielle with the email below or scan the QR code to fill out the screening questionnaire.

Masters Student Researcher:
Arielle Nash

Lead Investigator:
Dr. Lynneth Stuart-Hill

Appendix B: Recruitment Poster 2

University of Victoria

School of Exercise Science, Physical and Health Education

RESEARCH PARTICIPANTS NEEDED:
EXAMINING THE RELATIONSHIPS AMONG HIP MOBILITY, STRESS & LOWER BACK PAIN

Who: Healthy Adults aged 18-65; with or without back pain.

Description:

This research project aims to explore the validity of psychosomatic theories linking hip extension range of motion, stress and pain. Research evidence may be used to inform multidisciplinary approaches to pain relief, stress symptom management and stress relief.

Participation in the study requires approximately 1 hour of your time in a single visit to the satellite lab set up downtown Victoria. During the one hour, our research team will measure the state of your autonomic nervous system and hip range of motion. All measurements are non-invasive and low risk.

If you are interested in participating, you will be fully informed of all procedures and sent a consent form. To show your interest, please contact Arielle with the email below or scan the QR code to fill out the screening questionnaire.

Masters Student Researcher:
Arielle Nash

Lead Investigator:
Dr. Lynneth Stuart-Hill

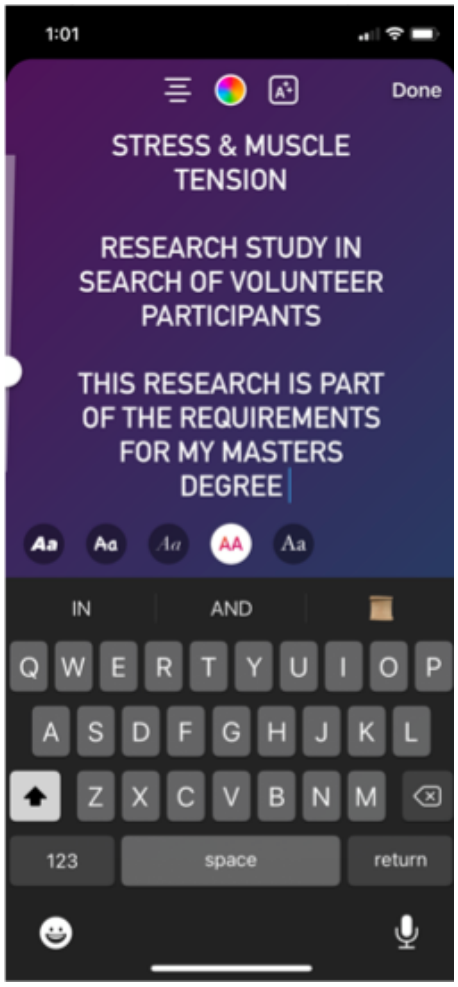
Appendix C: Social Media Posts

Instagram and Facebook post read “I am looking for volunteers for my Master’s Research.

Please PM me if you’re interested in participating. You need to live in Victoria, BC.

Figure C1

Instagram and Facebook Story Screenshot



Appendix D: Pre-Screening Questionnaire

Stress, Low Back Pain and Muscle Tension - Participant Pre-Screening

All information provided is protected by the privacy and confidentiality protocol for this study as approved by the Human Research Ethics Board at the University of Victoria.

* Required

1. Participant's name *

2. Email address *

3. Phone Number *

4. Participant's Age *

5. Biological Sex *

- ◯ Male
- ◯ Female
- ◯ Other

6. Are you pregnant or breast feeding? *

- ◯ Yes
- ◯ No
- ◯ Unsure

7. Select any current diagnosis (as diagnosed by a licensed healthcare practitioner). *

- ☐ Diabetes (Type I or II)
- ☐ Cardiovascular Disease
- ☐ Stress or Anxiety Disorder
- ☐ None of the above

8. Have you experienced lower back pain in the past 12 months for a period of 3 months or longer? *

- ◯ Yes
- ◯ No

9. Is/ was your back pain caused by a specific acute injury? *

- ◯ Yes
- ◯ No
- ◯ Unsure

10. Do you currently experience back pain or has it been resolved? *

- ◯ My pain is current
- ◯ My pain has been resolved

11. Does your back pain limit you from laying on your back for a period of time? *

- ◯ Yes
- ◯ No
- ◯ Unsure

12. Please select any activities your participate in regularly (minimum twice per week). *

- [ ] Running/ Jogging
- [ ] Cycling/ Spinning
- [ ] Yoga
- [ ] Weight Training/ Strength Training
- [ ] Calisthenics
- [ ] HIIT
- [ ] Swimming
- [ ] Sport (organized, not otherwise listed)
- [ ] Dance
- [ ] None
- [ ] Other

13. Are you considered at high-risk of serious illness from COVID-19? Note: The CDC states that high risk for serious illness has been shown to exist in adults with health conditions such as cancer, kidney disease, chronic obstructive pulmonary disease, down syndrome, any heart condition, weakened immunity due to organ transplant, obesity, pregnancy, sickle cell disease, smoking diabetes, heart disease, lung disease or any respiratory dysfunction. Additional information on moderate risk can be found here https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/index.html (https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/index.html) *

- ( ) Yes
- ( ) No

Appendix E: Letter of Information and Implied Consent

University of Victoria

Letter of Information for

Implied Consent

Stress, Muscle Tension and Back Pain Project

You are invited to participate in a study entitled "An Examination of the relationships among psoas muscle tension, stress and lower back pain." that is being conducted by Dr. Lynneth Stuart-Hill and Arielle Nash.

Arielle Nash is a graduate in the department of Exercise Science, Physical and Health Education at the University of Victoria and you may contact her if you have further questions.

As a graduate student, I am required to conduct research as part of the requirements for a degree in Master of Science. It is being conducted under the supervision of Dr. Lynneth Stuart-Hill. You may contact my supervisor.

Purpose and Objectives
The purpose of this research study is to explore the validity of psychosomatic theories which link pain with muscle tension and stress. Specifically, measured tension in the iliopsoas muscle (a hip flexor complex and spinal stabilizer), human physiological stress markers and lower back pain.

Importance of this Research
Research of this type is important because stress is a risk factor for many ailments which carry a high morbidity. Stress has previously been implicated as a cause for low back pain, however, the relationship has not been heavily investigated. Research evidence from this study may assist in understanding mechanisms of stress and back pain and potentially be used to inform multidisciplinary approaches to pain relief, stress symptom management and stress relief.

Participants Selection
You are being asked to participate in this study because you volunteered to participant and are a generally health adult.

What is involved
If you consent to voluntarily participate in this research, your participation will include a single session of data collection at Ashtanga Yoga Victoria. This testing will be performed in a dedicated room to ensure your privacy. This testing will be one morning session lasting approximately one-hour scheduled a maximum 2 hours past your normal waking time. Arielle Nash, one of the researchers is also the owner and operator of Ashtanga Yoga Victoria. The following protocol will be completed:

1. Researcher will record participant's age, current perceived stress level and verify previously submitted information.
2. Provide saliva sample for analysis of resting cortisol levels.
3. While lying supine, the Modified Thomas Test will be administered. This involves you lifting one leg off of the ground, flexing your hip, and the researcher taking a measurement for hip extension on the opposite side. This will be measured twice on both sides.
4. Lie still, face up (supine) position on a treatment bed for 12 minutes for heart rate variability measurement.

During testing, you will wear a device on your upper body, including a chest strap to measure your heart rate and the variability of your heart rate. The device is called Equivital EQ02 Life Monitor. The equipment is non-invasive and is completely safe. There is no risk of physical harm to the test subjects.

You have already completed the pre-screening questionnaire to qualify for this study. There will be no further health questionnaires needed.

Inconvenience
Participation in this study may cause some inconvenience to you, including the need to travel to the testing facility, and an uninterrupted block of time for testing. Also, the time of day for measurement may cause disruption in normal weekday. You will be asked to avoid alcohol, recreational substance use and any physical activity (including stretching) or rehabilitation therapy 24 hours prior to the test. In addition to no caffeine intake the morning of measurement, which may cause temporary headaches and discomfort for some. If one of the research team does not pass the self-check health screening, timeslots may be rescheduled at the last minute. You are also required to travel to Ashtanga Yoga Victoria, downtown Victoria, in Market Square without walking or cycling more than 4 blocks.

Risks
There are some potential risks to you by participating in this research and they include minimal risk of contracting communicable diseases, including COVID-19, due to close human contact. The laboratory and those conducting this research minimize this risk by following a Safe Work Plan for research as required by the University of Victoria for off-campus, in-person research. Participants are asked to wear face masks when entering the building, and while in the testing site. The research team will be wearing masks, disinfecting all equipment between participants, observing physical distancing where possible, and limiting the number of people in the space. All participants will be required to complete a COVID-19 Health Screening questionnaire prior to entering the testing room.

It is possible that some subjects with back pain may experience some temporary discomfort by lying supine for the extended period of time, or during the Modified Thomas test. Should you need to change your position due to pain, you may inform the investigator during the testing.

Benefits
The potential benefits of your participation in this research include being a part of a study that may contribute to the knowledge of possible mechanisms and/or contributors and treatments to back pain and physiological stress. You will receive immediate verbal general rating of your hip flexor range of motion.

Voluntary Participation
Your participation in this research must be completely voluntary. If you do decide to participate, you may withdraw at any time without any consequences or any explanation. If you do withdraw from the study after data collection, we must receive your permission to use your collected data in the analysis. If you wish to withdraw, contact Arielle Nash by email.

Anonymity
You will be given an alphanumeric code to be used instead of your full name. The coding will be done by and only known to Arielle Nash. The coding sheet will be created and stored separately from the data collection files.
Although participants are scheduled far enough apart and outside of Ashtanga Yoga Victoria's regular class operating hours, that cross-over should not occur, it is possible that you may be scheduled in a back-to-back time frame with someone you know previously. Should this happen, it may compromise your anonymity, however in no way will affect your ability to proceed in the study. In the case of a COVID-19 exposure for an individual associated with this project, anonymity may be compromised to comply with contact tracing requirements of public health authorities.

Confidentiality
Your confidentiality and the confidentiality of the data will be protected by not using your real name and all data will be kept on an encrypted password protected computer in a locked room. All identifiable personal data will be removed and subject numbers assigned.

Dissemination of Results
It is anticipated that the results of this study will be shared with others in the following ways:
- Master's Thesis
- Scholarly Conferences/ Presentations
- Published Articles
- Directly to participants

Disposal of Data
Data from this study will be stored for a period of 5 years, at which time it will be erased.

Contacts
OMITTED for publication

In addition, you may verify the ethical approval of this study, or raise any concerns you might have, by contacting the Human Research Ethics Office at the University of Victoria.

By booking an appointment, having completed and submitting the pre-screening questionnaire, **YOUR FREE AND INFORMED CONSENT IS IMPLIED** for your participation in this study, including the use of your questionnaire and your participation in the aforementioned testing at Ashtanga Yoga Victoria. It also indicates that you understand the above conditions of participation in this study and that you have had the opportunity to have your questions answered by the researchers.

Please retain a copy of this letter for your reference.

Appendix F: Participant Pre-test Protocol

Day Before:
Refrain from alcohol or other recreational substances for a 24-hour period before your scheduled appointment.

Testing Day:
Refrain from physical activity in any form (including stretching), caffeine or other stimulants prior to appointment. No food or teeth brushing within 30 minutes of your scheduled time. To assist with saliva sample collection, drink plenty of water up to 30 minutes before your appointment.

Perform the participant Health Check. Wear clothing that is easy to move in, however tight fitting. For example, cycling shorts or leggings.

Travelling to the testing location should involve a MAXIMUM activity of 2 blocks walking plus the stairs to get into the building. Remember to bring a mask.

Please arrive no more than 5 minutes before your appointment.

Upon Arrival at Testing Location:

Please note that masks are mandatory inside the testing building.
Ring the bell at the entrance and you will be admitted to the building.
Use the designated waiting area until the researcher calls on you to come into the testing room.

Testing Location Information:
Inside Market Square (560 Johnson St), Victoria BC
Suite 203 – Auxiliary Space

Entering Market Square through the main Johnson Street gate, use the exterior staircase (up to 2nd Floor) to your left. Ring the bell at the door.

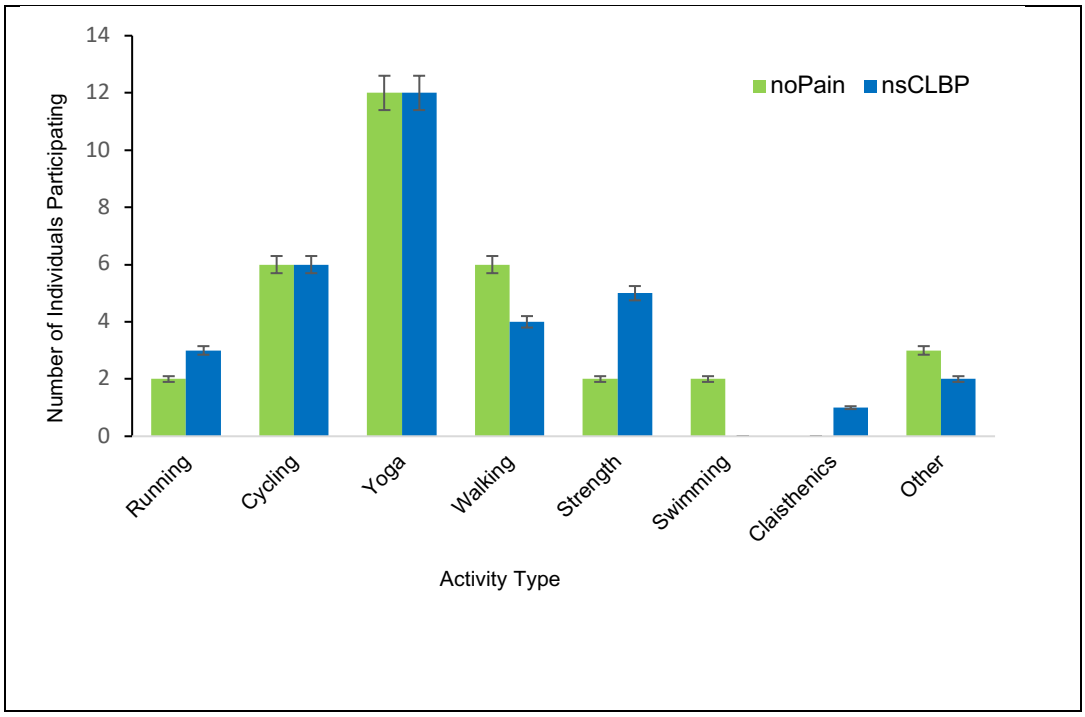
Appendix G: Supplementary Results

G. 1 Participant Physical Activity

Participants in both groups reported being physically active a minimum of twice per week for no specific duration, in activities such as walking, hiking, running, cycling, swimming, strength / body weight training, organized sport, fitness classes and yoga (Figure G1). Yoga was the activity reported to have the most participation, with 65.0% of the overall group participating and no significant difference between the noPain (70.6%) and nsCLBP group (64.7%). Cycling (32.5%) and walking (35.0%) were the next most popular activities for the overall group. Fisher's exact test did not show significant association between noPain or nsCLBP and any physical activity (Figure G1).

Figure G1

Specific Activity Participation Reported by Individuals in Each Subgroup (n = 17; CI = 95%)



Note. Participation in an activity as minimum twice per week.

G. 2 Supplementary Results for Hip Extension

Mean HE in the overall group (n=39) was -8.52° ± 9.78° on the left and -7.45° ± 9.54° on the right (Table G1). The difference between left and right side was not significant ($t(37) = 1.20$, $p = .239$) nor was there any significant sex difference for any measure of HE.

Table G1

Data Summary for Hip Extension for Overall Group (n=39)

Measure	Mean (°)	Min.	Max.	*SD*
HE L	-7.45	-36.00	9.00	9.78
HE R	-8.52	-28.50	13.00	9.54

G. 3 Supplementary Results for Stress Measures

Morning cortisol levels (CORT) had a mean of 0.24mcg/dL (*SD*=0.20mcg/dL), or 6.69nmol/L (*SD*= 5.44nmol/L), ranging from 2.13nmol/L as the lowest measurement to 24.00nmol/L as the highest. The means and variation for all stress measures in displayed in Table G2. The t-tests (one-tailed) did not show a significant difference in any stress measure between the nsCLBP group and the nsCLBP group (Table G2).

The overall average level of perceived stress was 4.9/9 (SD=2.25) for all 39 participants. There was no significant difference between the sexes (females: $M = 5.07$, $SD = 1.94$, males: $M = 4.69$, $SD = 2.63$, $p = .304$). The most frequent response among all participants was 7 out of 9, making up 17.5% of the sample. Just over half (52.5%) of the participants responded with a self-perceived current stress rating of 5.0 or higher. There was no significant difference in self-reported perceived stress between the noPain and nsCLBP groups, $t(32) = 0.27$, $p = .388$.

Overall, there was no significant correlation identified between perceived stress and any other measure in the study for any group.

Table G2

Summary of Stress Measures for all Groups

Stress Measure	Overall Group (n=39)		noPain Group (n=17)		nsCLBP Group (n=17)		*t*	*p*
	Mean	*SD*	*Mean*	*SD*	*Mean*	*SD*		
STRESS RP	4.90	2.25	5.00	2.13	5.24	2.63	-.29	.39
SDNN (ms)	46.96	22.71	46.62	20.39	42.52	21.88	.57	.29
LF (ms^2)	1490.95	1536.85	1404.27	1420.76	1267.73†	1357.05	.28	.39
HF (ms^2)	688.88	694.49	846.41	759.91	544.21†	673.48	1.21	.12
LF/HF (ms^2)	4.96	5.71	3.52	4.46	6.23†	6.91	-1.37	.09
CORT (nmol/L)	6.64	5.44	6.89	5.93	5.97	4.18	.52	.30

Note. † outlier removed, nsCLBP group n = 16 for variables LF, HF and LF/HF. One-tailed independent t-test did not show significant difference in stress measures between the noPain group and nsCLBP group.

G. 4 Supplementary and Post-hoc Relationship Findings

In assessing age as a confounding variable in the HE-Stress relationships, age was found to be significantly correlated to HE L ($r(38) = -.28$, $p = .04$), SDNN ($r(38) = -.57$, $p < .001$), LF ($r(37) = -.38$, $p = .01$) and HF ($r(37) = -.55$, $p = <.001$). When age was controlled for in post-hoc partial correlation analysis, the correlation of SDNN with HE L was no longer significant. All other correlations remained significant (Table G3).

Table G3

Age-Controlled Correlations for Physiological Stress Measures and Hip Extension Measures in degrees (n = 39)

Variable	SDNN (ms)	LF (ms^2)	HF (ms^2)	LF/HF	CORT (nmol/L)
HE L	.17[a]	-.09	.27*	-.33*	-.26*
p	.16	.31	.05	.02	.05
HE R	.33*	-.12	.37*	-.17	-.24
p	.02	.24	.01	.16	.06

Note. One-tailed Partial Pearson correlation. Control variable = age. Prob > |*r*| under *HO: Rho = 0.* LF, HF, LF/HF n = 38 due to the removal of the outlier. * $p \leq .05$; ** $p \leq .005$. [a] significant when not controlling for age as shown in Table 2.