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Hip flexor length as a predictor of hip extensor weakness in recreational runners

By

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A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, in partial fulfilment of the requirements for the degree of Master of Science (Medicine) in Sport Science

Johannesburg

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DECLARATION:

I, Gerard Jose Correia, declare that this research report is my own work. It is being submitted for the degree of Master of Science (Medicine) in Sports Science at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other University.

_____

(Gerard Jose Correia)

____day of October, 2013.

ABSTRACT:

Introduction: Janda described lower crossed syndrome (LCS) as tightness of the thoracolumbar extensors along with tightness of the iliopsoas and rectus femoris muscles. This is crossed with weakness of the deep abdominal muscles and weakness of the gluteus maximus and gluteus medius muscles. Janda identified the LCS as common postural changes involving the hip flexor extensor mechanism. Janda's LCS provides the framework that can account for the postural observations and changes noted around hip musculature. Although this framework has been routinely applied and cited in an attempt to understand faulty posture better, the complexities of his theory still remain conceptual. Limited evidence seems to be available that has specifically shown how an antagonistic muscle group (hip flexor and hip extensor) can directly affect each other sufficiently to disrupt normal muscle functioning.

Methodology: This study's aim was to determine whether the length of the hip flexor can be used to predict hip extensor weakness. Thirty two recreational runners were recruited to participate in this study. The modified Thomas test was performed on each participant to measure hip flexion length along with an isokinetic hip extension test that measured hip extensor strength. The correlational study was undertaken to compare the hip flexor length to ipsilateral hip extensor strength.

Results: The results did uncover variability in male versus female hip function in the population of recreational runners. In addition, a strong negative relationship between two-joint hip flexor length and hip extensor strength was found in the female group.

Conclusion: Two-joint hip flexor length may be a predictor of hip extensor strength in female recreational runners.

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LIST OF ABBREVIATIONS

LBP	LOWER BACK PAIN
1 HF	ONE JOINT HIP FLEXION RANGE OF MOVEMENT
SD	STANDARD DEVIATION
2 HF	TWO JOINT HIP FLEXION RANGE OF MOVEMENT
P.T.	PEAK TORQUE
PT/BW	PEAK TORQUE/BODY WEIGHT OR RELATIVE PEAK TORQUE
PFPS	PATELLOFEMORAL PAIN SYNDROME
MRI	MAGNETIC RESONANCE IMAGING
AV.PT	AVERAGE PEAK TORQUE
MBS	MOVEMENT BALANCE SYSTEM
CP	CEREBRAL PALSY
CNS	CENTRAL NERVOUS SYSTEM
PCSA	PHYSIOLOGICAL CROSS-SECTIONAL AREA
WORK	TOTAL WORK
WRK/BW	TOTAL WORK/ BODY WEIGHT OR RELATIVE TOTAL WORK
HF Tor	HIP FLEXOR PEAK TORQUE

HF Tor/BW	HIP FLEXOR PEAK TORQUE/BODY WEIGHT OR RELATIVE HIP FLEXOR TOQUE
Ant/Agon	ANTAGONIST/AGONIST RATIO
LCS	LOWER CROSSED SYNDROME
ROM	RANGE OF MOVEMENT
EMG	ELECTROMYOGRAPHY
SIJ	SACROILIAC JOINT
N-M	NEWTON METRES
R	CORRELATION COEFFICIENT
%	PERCENTAGE
^{0}	DEGREES

Chapter One: Introduction

Low back pain (LBP) is an affliction that is believed to affect most people some time in their lives. There is also a direct effect on work productivity, causing more sick leave and disability than that is caused by any other medical condition. Overactive and tight hip flexors can contribute to lower back pain by causing an anterior pelvis shift, and an increased lumbar lordosis, placing excessive pressure on the posterior portion of the lumbar disc and facet joints. Although relatively inactive office workers present to medical professionals with complaints of lower backache, similar findings are common among those individuals who participate in regular exercise[1].

This highlights a possible common problem, not with exercise or activity levels, but rather with biomechanical abnormalities affecting posture, particularly during the running stride. Runners with poor running technique usually have an increased swing phase created by short and tight hip flexors along with a shortened stance phase, coupled with an increased lumbar lordosis and decreased facilitation of hip extensors. Additionally, weakened abdominal muscles may lead to a forward lean as the runner is left hunching over, which actually reinforces tighter and shorter hip flexors, exacerbating this faulty running pattern[2].

In 1979, a theory concerning muscle imbalance, as a cause for posture irregularities, known as Lower Crossed Syndrome (LCS), was first identified by Vladamir Janda[3]. Janda described LCS as tightness of the thoracolumbar extensors with tightness of the iliopsoas and rectus femoris muscles. Along with this finding, there is weakness of the deep abdominal muscles and weakness of the gluteus maximus and gluteus

medius muscles. Janda found that some muscles may be stimulated, inhibited, or changed in tone, based on a response to pain or changes in proprioceptive input[3-4]. There are studies subsequent to Janda's postural theory which have reinforced his theory[5-6]. Janda identified LCS as common postural changes involving the hip flexor extensor mechanism. These subsystems or muscular functional sets (hip flexor and hip extensor) are interdependent and develop their own adaptations and compensations to sustain the movement pattern[4]. However, the research available attempting to retest and validate these hypotheses is limited. In particular there is limited evidence validating Janda's LCS theory that tight overactive hip flexors can inhibit and create weak hip extensors.

Janda's LCS theory provides the framework that can account for the postural observations and changes noted around the hip musculature. Although this framework has been routinely applied and cited in an attempt to understand faulty posture better, the complexities of his theory still remain conceptual. There is still a question around the validation and reproducibility of his theory. Limited evidence seems to be available that has specifically shown how an antagonistic muscle group (hip flexor and hip extensor) can directly affect each other sufficiently to disrupt a normal functional pattern. Therefore, this study aimed to assess the relationship between the hip flexor length and hip extensor strength.

1.1 Aim of the Study

The aim of this study was to determine whether there is a relationship between (one-joint and two-joint) hip flexor length and hip extensor muscle strength.

1.2 Objective of the Study

The objectives of this study are:

1. to test the one-joint and two-joint hip flexor length of recreational runners;
2. to test the hip extensor isokinetic strength of recreational runners;
3. to determine if there is a correlation between the one-joint hip flexor length and hip extensor strength of recreational runners; and
4. to determine if there is a correlation between the two-joint hip flexor length and hip extensor strength of recreational runners.

1.3 Hypotheses

The hypotheses of this study are set out below.

Null hypothesis: There is no relationship between (one-joint and two-joint) hip flexor length and hip extensor strength.

Alternative hypothesis: There is a relationship between (one-joint and two-joint) hip flexor length and hip extensor strength.

Chapter Two: Literature Review

Professor Vladimir Janda described predictable patterns of muscle imbalance due to posture, pain or pathology[3]. These muscle imbalances lead to altered movement patterns in particularly the hip extensors. By altering the length of one muscle, the strength of its antagonist will be reflexively improved. Although Janda described most of these muscle imbalances within a static position or posture, very little research has been done to assess whether this pattern/relationship exists in a more dynamic setting. In order to understand and describe Janda's theory from a dynamic perspective, it is imperative to understand the functional anatomy of the muscles involved and how these influence a running gait. The following section describes the functional anatomy of the hip flexors and extensors, and their relationship during running, and will also provide the anatomical basis for the research theory.

2.1 Hip Flexors

The anatomy and function of the hip muscles in question provide the background and foundation for this review. The origin and insertion of the hip flexors establishes normal muscle function as a comparison for dysfunctional muscle activation and action. The primary movers are the iliopsoas and the rectus femoris muscles. The iliopsoas is made up of the psoas major and the iliacus. Although the psoas minor assists the psoas major in flexing the pelvis and the lumbar vertebra, it is only present in 50% to 60% of people. The sartorius, pectineus, gracilis and the tensor fascia lata are secondary or synergistic movers assisting in hip flexion. (Appendix A)[7].

2.1.1 The functional anatomy of the iliopsoas

The iliopsoas is a major hip flexing muscle. The force produced by the iliopsoas acts across the hip, the sacroiliac joint, the lumbosacral junction, and the lumbar spine due to its proximal attachments to the lumbar spine[7](Appendix A).

The iliopsoas is involved in both the flexing of the hip as well as the flexing of the trunk. As a stabiliser it provides vertical stability when the hip is pushed into extension[8]. The conjoined distal tendon begins where the iliacus and the psoas major cross to create a singular force[7]. This conjoined tendon crosses the superior pubic ramus of the pubis sufficiently to create a greater mechanical advantage for hip flexion. When the hip is at 90 degrees, this advantage is even greater[8].

2.1.2 The functional anatomy of the rectus femoris

Although the rectus femoris is also considered a primary hip flexor it functions both as a hip flexor as well as a knee extensor. The proximal rectus femoris also has two origins. There is a straight head arising from the anterior–inferior iliac spine and then there is the reflected head, arising from the superior acetabular ridge and hip joint capsule. The two heads form a conjoined tendon below their origins[8]. The rectus femoris narrows sufficiently to become a tendon which inserts into the superior pole of the patella[9].

The origin, insertion and innervation of the secondary hip flexor movers including the pectineus, the sartorius, the gracilis, the adductor longus and the tensor fascia latae are shown in Appendix A[7].

In running, the rectus femoris, psoas major, and iliacus contribute to hip flexion activation during the early and middle swing phases of the running gait cycle[10]. The iliopsoas and the rectus femoris working as hip flexors will contract eccentrically to slow hip extension into the terminal swing phase just prior to initial heel contact[10].

2.1.3 Psoas Major: a stabiliser

A case study on the psoas major, which included a review of its anatomy, biomechanics, and clinical implications, found a large body of evidence[11]. This evidence is based on magnetic resonance imaging (MRI) of the psoas major during a lumbar extension movement test that was sufficient to induce pain. The study reported a reduction in psoas major activity with lower back pain. Furthermore, in patients with chronic low back pain the multifidis was inhibited along with the loss of cross-sectional area of the psoas major[11]. Other studies have also shown the psoas major to play a significant role in ensuring uninterrupted lumbar stability. It is believed that while the iliacus would torque the pelvis into an anterior pelvic tilt, the psoas major continually works against these forces. The continuous co-activation of both muscles that make up the iliopsoas increases shearing stiffness in the lumbar segments[12]. Contrary to the gluteus maximus, which stabilises the spine during running, the psoas major functions as a trunk stabiliser while a person is standing. This stability is achieved through a bilateral contraction of both left and right psoas major muscles. It appears to flex the lower lumbar vertebrae and the lumbar spine is consequently compressed into an S shape[13].

2.1.4 Hip flexor pathomechanics

An overactive hip flexor can give rise to pathomechanical changes. Brolinson et al., hypothesised that hip capsule laxity can contribute to atraumatic hip instability. If the hip capsule is injured or fails to provide adequate static hip stability, the psoas major contracts to provide hip stability. Over time, an overactive and shortened iliopsoas can lead to the internal snapping hip syndrome[13]. This syndrome is caused by a snapping or frictioning of the iliopsoas tendon over the iliopectineal eminence[14]. According to Ilizaliturri et al., this occurs when the hip is extended from a flexed position and a snap is caused in the anterior part of the groin. Internal snapping hip syndrome may also occur during any activity resulting in repeated hip flexion or external rotation of the femur[15]. Furthemore, Laible et al., reported that dancers are typically vulnerable to this hip pathology. The pathomehanical changes are determined by the dancer trying to achieve a greater hip external rotation (turnout) during active extension. The dancer assumes a hyperlordotic pelvic posture causing the femoral head to be pushed into an increasingly anterior position resulting in the iliopsoas tendon snapping over the iliopectineal emminence[16].

Of importance is that altered hip biomechanics through the tight iliopsoas, tensor fascia lata, or rectus femoris can potentially limit hip extension and inhibit gluteus maximus activation. The gluteus maximus is unable to produce its paradoxical effect on the hip joint by eccentrically managing hip flexion range[17]. An overactive hip flexor, along with an inhibited gluteus maximus, allows for an increased anterior pelvic tilt. This changing pelvic posture increases lumbar lordosis creating unequal stress on the lumbar region.

Furthermore, this tight hip flexor mechanism contributes to an overactive knee mechanism. Piva et al., also confirmed that limited quadriceps flexibility has been found to be associated with patients suffering from patella femoral pain[18]. Waryasz et al., proposed that the quadriceps tightness may be the cause of these high patellofemoral stresses[19]. In addition, a systematic review found strong evidence that female runners with PFPS were associated with a decrease in hip extension, external rotation and abduction strength[20].

Souza et al., found that increased hip internal rotation was accompanied by a significant decrease in hip extension strength, particularly in the gluteus maximus, in participants with PFPS[21]. Indeed, Powers et al., found that the primary contributor to a lateral patella tilt and displacement was internal rotation of the femur. Powers stated that the gluteus maximus provides triplanar stability of the hip, which means it acts as a hip extensor, a hip abductor and is the most powerful hip external rotator, and therefore would resist excessive hip flexion, adduction, and internal rotation. These maladaptive patterns of muscle activation are reinforced and magnified, especially when the hip is stressed dynamically[22].

According Noehren et al., runners with PFPS were found to have greater hip internal rotation and hip adduction strength. The changes in hip muscle strength increase the lateralisation of the patella, reducing the patellofemoral contact area and concomitantly increasing retropatellar pressure, thereby facilitating the onset of knee pain. However, increasing the flexibility of the hip flexors and tensor fascia lata would allow the pelvis to rotate posteriorly. This would allow relatively unobstructed femoral

external rotation to align the patella in the trochlear groove of the femur[23]. Interestingly, Chumanov et al., found that runners with a higher step rate exhibited increased gluteus maximus and medius activity. A higher step rate increased the late swing phase of the running gait cycle, a phase where gluteus maximus and medius activity is increased. Running with an increased step rate was also shown to have a reduced peak hip adduction angle during the stance phase. This observation suggested that this running strategy may benefit those runners diagnosed with anterior knee pain[24].

2.2 Hip Extensors

The origin and insertion of the hip extensors, detailed in Appendix B, provide a better understanding of the direction of force acting on the hip and pelvis. The major hip extensors are the gluteus maximus, the gluteus medius, the hamstring muscles and the adductor magnus.[7]

2.2.1 Functional anatomy of the hip extensors

The extension action on the hip and pelvis generates sufficient force to produce the desired movement and a lasting normalised motor pattern. Whether or not it is influenced by its antagonistic muscle group, the hip flexors, the force generated by hip extensors may not be sufficient to create the expected action its muscle length and size suggest.

Muscles act on joints to generate force, which is dependent on the moment arm. A moment arm can increase or decrease the torque-generating capacity of a muscle. For example, the maximal force of the gluteus maximus is undermined by its muscle length. It requires a longer, more sustained contraction to maintain that same force

producing capacity suggested by its size and various areas of attachment[25]. Indeed, muscles with large physiological cross-sectional areas (PCSA) and short fibre lengths generate large forces over small length changes, while muscles with small PCSAs and long fibres generate small forces over large length changes. The gluteus maximus and adductor magnus have large PCSAs and long fibres, suggesting that they are expected to generate large forces over a wide range of lengths. On the other hand, short external rotators, like the pirifomis muscle, have large PCSAs and very short fibres, suggesting that they are designed to stabilise the hip and pelvis[26].

Although the gluteus maximus may be the largest hip extensor in terms of PCSA, its mechanical advantage and torque producing qualities vary depending on body position. For example, when the hip is flexed, the adductor magnus has a larger hip extension force than does the gluteus maximus. With the added extension force created by the hamstring muscle, this adds up to nearly twice as much force as that produced by the gluteus maximus[25]. In standing (the anatomical position) the posterior head of the adductor magnus has the greatest moment arm for extension. The semitendinosus creates the second largest moment arm followed by the gluteus maximus. The force created by these extensor muscles increases as the hip is flexed to 60° [26]. For example, the hip extensor force is at its greatest mechanical advantage when generating torque from a position of hip flexion such as a running or sprint start from the blocks or the knee extension component of a full squat[27].

However, in reaching a hip flexion greater than 60° there seems to begin a reduction in the gluteus maximus production of hip external rotation. In fact, at 90° the anterior fibres concerned with external rotation actually become internal rotators. Even the

deeper external rotators, like the piriformis and the superior and inferior gemellus, also become internal rotators at 90° of hip flexion[25].

This illustrates the fact that as the hip flexor moves into its inner range, the gluteus maximus loses its ability to maintain its neuromuscular control over the hip. There is also an obvious loss of the triplanar motion of the gluteus maximus as its capacity to adequately extend, abduct and externally rotate the hip is significantly reduced in favour of a sustained hip flexion with a larger hip internal rotation and adduction moment. The body position of the hip alters the tension produced by the gluteus maximus due to its proximal attachment to multiple areas. The influence of the hip flexors, adductors and internal rotators over the triplanar motion potentiated by the gluteus maximus creates a scenario where certain sustained postures such as long periods of sitting may prevent the appropriate force tension generation[28]. Therefore, it is assumed that shortened hip flexors in a sitting posture may consistently have a mechanical advantage over the gluteus maximus.

2.2.2 The hip extensor muscles involved in running

The gluteus maximus is primarily involved in hip extension and external rotation. The upper fibres also assist in abduction of the hip while the lower fibres adduct the thigh. The gluteus maximus also attaches to the iliotibial tract which helps to stabilise the knee while locked in extension. The proximal attachment to the sacrotuberous ligament of the gluteus maximus provides rigidity to the ligament thereby increasing sacroiliac joint stability and reducing mobility[28]. As the hip and knee move into extension the gluteus maximus both acts as the primary mobiliser and also provides stability to the pelvis. The hamstring muscle group and adductor magnus also assist in

hip extension. In fact, the adductor magnus has been referred to as a fourth hamstring, which also acts as a powerful hip extensor[29].

In running, the gluteus maximus, the adductor magnus and hamstring muscle groups contract concentrically in the early stance phase, which creates a hip extensor moment that drives the body over the foot[30]. The gluteus maximus facilitated hip extension becomes particularly important in this phase. A prolonged hip flexion action has been observed during the stance phase when gluteus maximus activation was removed. An inactive gluteus maximus cannot prevent increased hamstring activation which results in an exaggerated knee flexion action during the stance phase. The hip flexors and hamstrings overact in the absence of gluteus maximus[31].

Weimann et al., found that the gluteus maximus will externally rotate an already extended hip. There is also an abduction force created by the gluteus maximus that inhibits the sagittal plane forces created by hip extension action. This hinders the hip flexion of the unsupported leg during the swing phase in the running gait. The adductor magnus becomes a powerful hip extensor when the femur falls below the level of the hip joint during the swing phase of the running gait. However, during the stance phase the adductor magnus works with the adductor group of muscles to balance the strong abduction and external rotation forces created by the gluteus maximus. As soon as the support leg moves into full foot contact and is approaching mid-stance, the gluteus maximus no longer needs an antagonist to compensate for the generation of its horizontal and frontal plane forces (abduction and external rotation force respectively). This adduction function is now taken over by gravity, pulling the pelvis medially onto the unsupported leg still in the swing phase of the

running gait. The gluteus maximus activity slows down at this time and has completely stopped as the hip moves into the mid-stance phase of the running gait. Further hip extension is now taken over by the hamstring group of muscles[32].

2.2.3 Injuries associated with running gait

The hamstring muscles are at their most active during late the stance phase as well as during the late swing phase of running. Peak hamstring length occurs just prior to initial foot contact[33-34]. There is also evidence that this hamstring group is further lengthened by increased hip flexion during the late swing phase. In this lengthened state, the hamstring muscle would, therefore, only need a transient increase in loading to induce injury. There is a simultaneous contraction or co-contraction, where the hip flexors will contract concentrically to slow hip extension, while the hamstrings would contract eccentrically to control hip flexion[34].

In fact, it is estimated that with every degree of decreased hip flexor flexibility increases the risk of hamstring injury by 15%[35]. Athletes exhibiting a deficit in active hip flexion could be adjusting their running gait to avoid overloading their hamstrings based on their experiences of previous injury. As the hip flexors work to stabilise the pelvic complex it also reduces its mobility during the running cycle. Hip flexor length and concentric strength now become important components for peak hip extension during a running gait cycle. A reduction in hip flexion length could result in the restriction of the required hip extension to complete a normal running gait cycle[36]. Therefore, an overactive and shortened hip flexor, restricting the maximum length of an eccentrically loaded hamstring, now forces lumbar extensor muscles to act as secondary hip extensors in the presence of a weak and inhibited gluteus maximus. Although LCS is the description of a static posture, it is the propensity for this posture

that is magnified through distinctive LCS changes, identified in a running gait, that now predisposes a hamstring injury.

2.3 Lower Crossed Syndrome

Lower crossed syndrome (LCS) refers to a pattern of muscle activation that has habituated into postural dysfunction. Janda observed this posture that manifests itself in patterns of muscle tightness and weakness across the pelvis. The tight and short tonic hip flexors, the iliopsoas and the rectus femoris muscle, crosses with the overactive and short thoracolumbar extensors, the erectae spinae. The weak and inhibited gluteus maximus is crossed with the weakened and inhibited abdominal muscles[3].

This pattern of muscle imbalance creates joint dysfunction, especially at both the lower lumbar joint levels and it also impacts the sacroiliac and hip joint. The postural changes associated with LCS include anterior pelvic tilt, increased lumbar lordosis, lateral lumbar shift and distally tibial external rotation and knee hyperextension. More specifically, Janda found that if the lumbar lordosis is restricted to the lumbar area, then the imbalance is predominantly in the pelvic muscles[3]. According to Kendall et al., lordotic postures are observed with shortened hip flexors and lumbar erector spinae in a shortened potion, as well as hip extensors and abdominals in a lengthened position[37].

2.3.1 Tonic and phasic muscle systems

Janda identified two groups of muscles that have evolved into their function. Functionally, muscles can be classified as "tonic" or "phasic". The tonic system consists of the "flexors". The phasic system consists of the "extensors" [3]. The tonic system muscles are prone to tightness or shortness, and the phasic system muscles are prone to weakness or inhibition[4]. Tonic muscles also tend to have increased tone in their resting length and are less pliable. Phasic muscles, however, are inclined to be flaccid and easily fatigued. Janda identified the hip flexors as tonic muscles, and the gluteus maximus, the hip extensor as a phasic muscle[5]. Therefore, using this theory, the hip flexors are likely to be tight while the gluteus maximus is likely to present as being weak or inhibited.

2.3.2 Muscle imbalance

Muscle balance is defined as the equivalent muscle length, strength and overall muscle activity between an agonist and an antagonist. However a similar definition of muscle balance can also include the strength comparisons between the ipsilateral versus contralateral or left and right muscle groups[3]. Patterns of muscle imbalance would be elicited by reduced muscle activity, an on-going repetitive movement, development of inflexibility or pain and stiffness associated with movement[5].

Muscle imbalance, as the result of an ongoing adaptation, is the tendency of an antagonistic group of muscles to act continuously and unequally in a reciprocal fashion. The tight tonic muscles remain short and overactive while the antagonists

remain weak and inhibited. Postural antigravity muscles are tonically tight, where phasic fast twitch muscles weaken[3].

On the contrary, Key et al., uncovered a predisposition for the antigravity muscles to have both a lower tone and also to be inhibited[38]. However, this was specific to a population of spinal pain sufferers. Muscles that are part of the intervening and compensatory strategy were found to be shortened, overactive and tight[39]. Inhibited phasic muscles will strengthen spontaneously if the antagonistic muscle activity is normalised[3]. For example, Ohtsuki et al., observed immediate changes in chronic lower back pain after introducing a stretching intervention of the tensor fascia latae, hamstrings and adductor magnus. They attributed a shortened tensor fascia latae to an anteriorly tilted pelvis[40.] Janda has categorised all three of these muscles as tonic muscles prone to shortening and over activity' and the LCS includes tight and shortened hamstrings along with overactive hip flexors[38].

When muscle imbalance is a result of dysfunction, this is referred to as pathological muscle imbalance. Pathological muscle imbalance impairs normal movement and function. The increased joint stress results in dysfunction and may predispose the onset of pain[3]. Janda stated that chronic musculoskeletal pain, along with muscle imbalance, is considered a functional pathology that is largely controlled and managed by the central nervous system (CNS). This implicates neurological involvement as a significant component of muscle weakness. This is based on observations of patients with chronic back pain who exhibited the same patterns of muscle tightness and reciprocal muscle weakness as those patients with upper motor neuron injuries, such as cerebral palsy (CP) [3]. The pattern of muscle activation found in those afflicted with

CP is created without CNS inhibition in response to afferent information based on the external environmental forces acting on joints and musculature[5]. Therefore, the firing patterns of unbalanced muscles are also a neurological response rather than simply a structural one[1-2].

These changes produce incorrect information that is processed in the CNS and over time, an altered motor pattern is generated. This reinforces the cycle of pain and dysfunction through a now well established imbalance in muscle firing pattern[3]. Furthermore, the pain stimulus is capable of changing the sensitivity to the perception of pain by the CNS in order to alter the efferent response to negative afferent information[5]. Janda found that an altered movement pattern can be identified through key movement tests. For example, Table 2.1 describes the presentation of a positive hip extension movement.

Table 2.1: Signs of a Positive Hip Extension Movement Test[3]

1. Decreased gluteal muscle bulk
2. Increased hamstring bulk
3. Anterior pelvic tilt
4. Obvious horizontal creases at the level of the lumbar spine
5. Increased paraspinal muscle bulk
6. Decreased trailing limb posture at terminal stance phase of walking gait

These muscular system changes, as seen in Table 2.1, are a response to pain that directly influences the changes in muscle pattern activation. Using a condition such as CP, an injury to the developing motor neural system, to illustrate this concept, the

tonic flexor muscles tend to be spastic and the phasic extensor muscles tend to be flaccid, creating a typical spastic, diplegic posture and gait pattern[3]. This implicates CNS in the patterns of muscle imbalance rather than structural changes, and reinforces the influence of chronic pain on the CNS.

However pain does not have to precede muscle inhibition. Patients with lower back pain have significantly weaker lumbar extensor muscles. Therefore changes in the motor system occur before the onset of pain and increase the risk of onset of pain[3]. In addition to the inhibitory effect of the painful stimuli, the cycle of weakened muscle patterns is reinforced[5]. Janda's pain adaptation model predicts that in the presence of pain there would be a decrease in electromyography (EMG) activity of agonists and an increase in antagonists [3].

Janda found that there was an imbalance based on the inherent characteristics of the tonic and phasic muscle fibres. Tight tonic muscles are a third stronger than phasic muscles. EMG analysis has found that there are delays in the muscle activation of phasic muscles along with a decrease in peak amplitude and muscle fibre recruitment[4].

The reciprocal nature of functional muscle groups becomes dysfunctional as the deeper stabilising muscles are inhibited, forcing the more superficial larger mobilising muscles to act earlier. The perpetuation of this dysfunction leads to new a habit forming movement strategy. A new strategy now becomes part of the movement patterns produced by the central nervous system[38].

2.3.3 Force closure

Force closure may be another mechanism that further reinforces the LCS theory. Force closure is understood as a compressive force created between two surfaces of a joint that provides enough friction to augment stability. The force closure applied to the sacroiliac joint (SIJ) is created by muscles and fascia around the hip and pelvis, acting across the joint to provide compression and optimal congruency to the SIJ[41-42]. However, if there is increased compressive force acting on the pelvis, the delayed onset of the gluteus maximus and semitendinosus may be improved. Takasaki et al., found that producing a compressive force via an artificial external source, reduced the delay in gluteus maximus activation[41]. The transversus abdominus has been shown to be the key muscle in the functional stability of the lumbosacral pelvic area and hip joint[43]. Furthermore, this is seen in low back pain patients, where inhibition of transversus abdominus delays the onset of the gluteus maximus activation[41]. Therefore the application of this mechanism includes increasing the force closure across the pelvis anteriorly in order to reduce the onset delay of the gluteus maximus. This reinforces the LCS theory that, for hip flexors and the thoracolumbar muscle and fascia to be short and overactive, there have to be inhibited deep abdominal muscles along with weak and inhibited gluteus maximus.

2.4 Anterior tilted pelvis

Limited hip extension ROM and tight hip flexors, especially iliopsoas muscles, predisposes an anterior pelvic tilt. The pelvis is more physically stable than the femur. If the pelvis is inadequately stabilised by other muscles, a sufficiently strong force from the iliopsoas or rectus femoris (or any other hip flexor muscle) could rotate or tilt the

pelvis anteriorly. Weakened abdominal muscles combined with a strong hip flexion contraction can create excessive anterior tilting of the pelvis[14]. Kendall et al., have also identified that this combination of weak abdominals, shortened hip flexors and an anterior pelvic tilt exists within a typical lordotic posture[37]. The abdominal muscles must generate a posterior pelvic tilt of sufficient force to neutralise the strong anterior pelvic tilt potential created by the hip flexor muscles. This synergistic activation of the abdominal muscles and the extent to which it can assist in preventing an anterior pelvic tilt is dependent on the action the hip is forced to perform. The rapid flexion of the hip should generate enough abdominal muscle fibre recruitment just before the body initiates the activation of the hip flexor muscles. Without sufficient stabilisation of the pelvis by the abdominal muscles, a strong contraction of the hip flexor muscles may inadvertently tilt the pelvis anteriorly[41]. An excessive anterior tilt of the pelvis typically accentuates the lumbar lordosis.

LCS describes a posture that includes an exaggerated lumbar lordosis as well as a shortened and overactive hamstring muscle. Janda proposed that there is a normal pattern of muscle activation during the execution of the prone hip extension exercise. This sequence begins with the activation of the hamstring muscle group. This is followed by the activation of the gluteus maximus, the activation of the contralateral erectae spinae and finally the ipsilateral erectae spinae. A faulty movement pattern would involve the over activation of the hamstrings and the erectae spine along with the delayed activation of the gluteus maximus. Janda found this pattern is observed as an anterior pelvic tilt with a hyperlordosis at the level of the lumbar spine and is associated with a typical postural analysis of hypertrophy of the thoracolumbar extensors and hamstrings and atrophy of the gluteus maximus[3]. Therefore, on further

analysis of this posture, it would appear that a tight hip flexor (causing lumbar lordosis) would inadvertently cause a weak and inhibited gluteus maximus.

A prone hip extension exercise performed with the addition of an abdominal drawing in manoeuvre resulted in the reduction of an anterior pelvic tilt. The abdominal activation was executed in combination with gluteus maximus activation as the hip in prone moved into extension[41]. Therefore the co-activation of the abdominal musculature and gluteus maximus highlights a relationship existing between these two muscles, especially where management and control of an anterior tilted pelvis are concerned.

This is consistent with Janda's theory about LCS that found weak abdominals and a weak gluteus maximus failed to provide sufficient torque to prevent an anteriorly tilted pelvis. Chance-Larsen et al., observed that during pre-intervention testing the gluteus maximus was consistently recruited after the biceps femoris. The intervention exercise involved the use of a pressure biofeedback unit (PBU) placed under the lower abdomen, and inflated to 70mm Hg. The participants were given instructions to hollow their lower abdomen whilst breathing to keep the pressure in the PBU unchanged. Then the participants were asked to contract their buttocks and to lift their knee off the bed. Post-intervention testing revealed that there was no change in firing patterns of the muscles but there was a significant reduction in the onset delay of the gluteus maximus firing relative to the biceps femoris. Chance-Larsen et al., also inferred that these changes may be able to be maintained and transferred to functional movements such as running[44].

An early onset of activation of the hip extensor muscles could become more involved in controlling hip extension concentrically, and hip flexion eccentrically, thereby

lowering the demands on the hamstring muscle group[42]. Janda also saw clinical application of the prone hip extension test. The correct sequence of muscle activation can be sufficiently taught using this test protocol that maintains the body in a neutral hip position[3]. The biceps femoris serves as the primary extensor of the standing/balancing limb if the gluteus maximus is weakened. Muscle balance is restored by a compensatory activation of the synergistic muscles or deactivation of the antagonistic muscles[45]. This again reinforces Janda's observation of correcting muscle firing patterns to improve defective postures.

2.4.1 Sherrington's law of reciprocal inhibition

Contraction of the abdominal muscles can help hip extensor muscles in stretching a tight hip capsule or facilitating length in a shortened hip flexor muscle. Co-activating the abdominal and gluteal muscles, while simultaneously performing a passive-stretch manoeuvre of the hip flexor muscles, may provide an additional stretch to these muscles. This is consistent with Sherrington's law of reciprocal inhibition which states that there is a reflex inhibition of the muscle in response to the activation of the antagonist[1]. For example, activating the hip extensors in a shortened range to likely to inhibit the hip flexors from contracting, allowing them to relax and lengthen[37].

Sahrmann's movement balance system (MBS) approach states that active stretching is supposed to increase the flexibility of the tight muscles while simultaneously improving the activation and muscle fibre recruitment of the antagonistic muscles. Active stretching may be seen as an effective method for increasing the flexibility of tight hip flexor muscles[46]. The MBS approach may be of clinical value, and is routinely used in practice, however, it is not evidenced-based and needs further support.

Therefore, from this approach it can be hypothesised that stretching the hip flexors may lead to improvement in the strength of the hip extensors.

This can be further seen by the relationship between hip joint hypomobility and the weakness of the gluteus maximus. Addressing hip mobility to restore normal joint range had a positive effect on the strength of the gluteus maximus. The proposed reason for this is attributed to reciprocal inhibition: a tight and immobile anterior hip capsule would facilitate the iliopsoas muscle while inhibiting the gluteus maximus[47]. Therefore, mobilising joints in the hip and spine, while strength testing subjects, actually creates an increase in muscular strength.

2.4.2 Reduced hip extension and anterior pelvic tilt

Limited hip extension mobility has also been found as a causal factor for an increased anterior pelvic tilt in both running and walking gait. The gait strategy highlighted here found over activity of the hip flexors, with compensatory anterior pelvic tilt, contributed to the reduction in hip extension. In the elderly, both a reduced hip extension and increased anterior pelvic tilt were observed during the walking gait assessment. The findings were observed in the gait assessment of the elderly group but did not find the same observations with a static posture assessment [47].

During running, peak anterior pelvic tilt was strongly correlated with peak hip extension [48]. This finding was validated by Franz et al., who also found a strong correlation between these two variables[49]. Further developing this relationship also

underlines the inter-dependence Janda uncovered through the identification of structures involved in LCS.

Tight hip flexor musculature or a tight anterior hip capsule was hypothesised to be a possible cause of increased anterior tilt of the pelvis during running. However, the results reported by the author did not yield any significant correlation for hip extension flexibility with either anterior pelvic tilt or hip extension ROM during running[50]. Janda's theory of LCS is based on a snapshot of posture: a static observation of a tighter and a weaker functional muscle system [3]. LCS identifies postural changes that include anterior pelvic tilt, and increased lumbar lordosis. If the hip loses its ability to extend in the terminal stance phase of the gait cycle, there needs to be a compensatory increase in both anterior pelvic tilt and lumbar lordosis. The same study, however found that anterior pelvic tilt is related to peak hip extension ROM during running. Increasing anterior pelvic tilt angle was found in participants with reduced absolute peak hip extension ROM. These results provide evidence that anterior pelvic tilt tended to be increased in runners who displayed reduced hip extension range during terminal stance. Terminal stance is defined as the last stage in the stance phase of the running gait and is also where peak hip extension takes place [50]. The evidence highlights a strong association with increased hip flexion and increased anterior pelvic tilt, along with a weak and inhibited hip extension running strategy.

2.5 Hip Strength

Hip strength in runners has been assessed using various methods, ranging from hand-held dynamometers to isokinetic devices[51]. Isokinetic dynamometers use a lever arm to move at a chosen and fixed speed. The resistance changes and increases according to the effort produced by the participant[52]. This accommodating resistance, according to Baltzopoulos et al., means that the isokinetic strength evaluation is not limited to the weakest point in the range[53]. Isokinetics is a popular method of assessing hip muscle strength, as it provides objective information and optimal and efficient loading of muscles and joints throughout the range[54].

Furthermore, isokinetic strength testing has been shown to be valid and reliable. Burnett et al., found the isokinetic hip extension strength testing has the highest intraclass correlation, followed by isokinetic hip flexion testing[55]. The Biodex System 3 Isokinetic Dynamometer has been quantitatively assessed to be reliable and valid in both a clinical and research setting[56,57]. Drouin et al., found the Biodex System 3 isokinetic dynamometer to be reliable and valid measurement of angular position [56]. In addition to validity and reliability, the reproducibility of the Biodex 3 system Isokinetic Dynamometer's is considered to be excellent [57].

2.5.1 Interpretation of results

Muscle performance can be identified as normal or abnormal based on the outcome of the work, power and torque produced during an isokinetic test[52]. Left side to right side, or injured to uninjured side, data can be compared to identify deficits in strength and endurance[57]. Information on agonists/antagonists ratios would also be available due to

the use of reciprocal contractions. It has been suggested that the hamstring to quadriceps ratio is more important than the maximum torque in the assessment of muscle function[58]. These ratios provide information on any lingering muscle imbalances. These values could also be compared to the established normative values to create a more focused rehabilitation and conditioning programme[52].

2.5.2 Factors related to participants

However, when performing isokinetic testing, and interpreting results, there are factors that need to be considered. Age, height, weight and gender have the potential to influence the generation of peak torque and total work. For example, the torque generation tends to decrease with increasing age**.** Predictably, male participants would generally produce far higher forces than females based on similar bodyweight**[52].** This has been further confirmed in hip strength testing, where Cahalan et al., found that older women had the lowest hip torque production, whilst younger men had the highest hip strength[54]. In addition to age and gender, the individual's body weight will also affect torque production. Therefore, interpretation of results needs to be normalised to the participant's body weight [58].

2.5.3 Standardised Testing Protocol

A standardised testing protocol needs to be consistent in order to produce a repeatable test irrespective of the participant or patient. This kind of testing approach improves the control over variables encountered by the tester that could influence the results. These variables can directly affect the outcome of the test and therefore need to be identified, addressed and managed.

Participant understanding of the test procedure helps to reduce anxiety and improve familiarisation. The education provided to the participant needs to be given both verbally and physically by allowing the participant to have a few practice sessions. Improving familiarity increases the possibility of producing the most accurate results and ensures that a maximum effort isokinetic test is created[52].

A standardised warm up and familiarisation protocol is important for consistency and reproducibility of the test. The participant would typically perform a cardiovascular warm up of about 10 minutes before beginning the test. The participant is then strapped into the Biodex isokinetic dynamometer to stabilise the tested joint to avoid any accessory movement. The tester provides both verbal understanding of the testing procedure as well as verbal encouragement[59]. Prescribed verbal commands are used to ensure consistency in the results both in practice and during actual testing. Campenella et al., did not uncover significant changes to peak torque after introducing verbal encouragement to an isokinetic test. However, it was reported in the same study that at least two other studies had revealed significant peak torque changes when verbal encouragement was utilised[60].

Furthermore, sufficient rest time is needed between strength testing sets. This is to allow adequate recovery time to guarantee consistency in the results[52]. Uniformity in the resting intervals is required to ensure reproducibility of the results. According to Bottaro et al., resting intervals of at least 30 seconds are essential in reproducing similar peak torque results[59].

Visual feedback may also alter the outcomes of the test results. Studies have shown that visual feedback can both improve and compromise results. For example Campenella et al., found that, regardless of gender, all participants produced significantly greater quadriceps and hamstrings peak torque values when provided with visual feedback[60].This feedback, however, could also lead to earlier muscle fatigue during the testing procedure as the participant tries to beat the previous test by attempting to improve the maximal effort[52]. Therefore it is important to be consistent with the amount and the use of visual feedback.

2.6. Flexibility

Kendall et al., defines flexibility as the ability to continuously adapt to changes in position or alignment. Flexibility can be classified as normal, or limited or excessive. According to Kendall et al., inability of the hip to extend to its neutral position constitutes a limited ROM of the one-joint hip flexors (i.e., the iliopsoas, pectineus, and the adductors longus and brevis), whereas an inability of the knee to flex beyond 80^0 constitutes a two-joint hip flexor limited ROM (i.e., the rectus femoris, tensor fasciae latae, and the sartorius)[37]. The Modified Thomas test is commonly used to assess the flexibility of both the one-joint hip flexors and the two-joint hip flexors [61,62,63,64,65,]. The Modified Thomas test has been adapted from the original Thomas test which was designed to identify hip flexion contractures[66]. The Modified Thomas test is similar to the Thomas test with the exception that the involved limb hangs off the edge of the table, allowing the examiner to observe both one-joint hip flexion ROM at the hip and two-joint hip flexion ROM at the knee[67].

Various studies have assessed the reliability of the modified Thomas test. Clapis et al., noted that a goniometer is a reliable instrument when used to measure one and two-joint hip flexion ROM using the Modified Thomas test[68]. Winters et al., and Harvey et al., also reported high reliability for both one and two joint hip flexor tightness using the Modified Thomas test[46, 67]. A study by Peeler et al., reported on a moderate reliability for the Modified Thomas test to assess two-joint hip flexion ROM [63]. The possible difference between these studies, when compared to the work of Peeler et al., was the hip placement on the plinth. Clapis et al., [68] placed the hip on the edge of the plinth where Peeler et al., [69] placed the knee at the edge of the plinth. Ferber et al., in creating normative values for one-joint hip flexion ROM, had comparable values to that reported by Clapis et al.,[68] who also cited the results of Peeler et al., [69]as questionable based on the testing procedure used[70].

2.6.1 One and two-joint hip flexion ROM normal values.

According to Kendall et al., normal one-joint hip flexor ROM is 10^0, whereas normal two-joint hip flexor ROM is 80^0 when measured using the Modified Thomas test[37]. Ferber et al., provided normative one-joint hip flexion values comparable to those of Kendall et al[70]. However, for the two-joint hip flexors, six authors reported a range of mean values from 50.8^0 to 54.4^0 of a combined population of 325 participants[61,62,63,64,65,66]. The disparity in values is based on the different testing procedures used to generate these two-joint hip flexion ROM values. All six authors used the testing procedure as set out by Harvey et al., where the hip is placed at the edge of the examination table. The testing procedure carried out by Kendall et al., has the participant with the knee at the edge of the examination table[37]. Schache et al[64]., and Van Heerden et al [61]., measured one and two joint Hip flexion ROM respectively,

using a population of runners. They reported very similar results to the values of Harvey et al[67]. Using the testing procedure set out by Harvey et al, Schache et al[64]., and Van Heerden[61]., reported one and two-joint Hip flexion ROM values that were not comparable to those of Kendall et al[37].

2.7 Conclusion

The literature review has described the muscles involved in the hip flexor/extensor mechanism. This included the various interactions and influences as well as how changes in length and strength can produce altered movement patterns.

The hypotheses and the study objectives set out to find whether a relationship exists between two opposing sets of muscles groups. Faulty movement patterns as observed by Janda become essential in uncovering the influences the hip flexors and extensors have over each other. It is in this state that it becomes easier to observe tighter, shorter muscle groups and the reciprocal underactive and inhibited muscles that fail to act appropriately. Sherrington's law of reciprocal inhibition provides a better understanding of how the hip flexors and hip extensors influence each other. This also reveals that if one is to apply this law clinically then by default the hip flexor extensor mechanism functions dependently. Hip extensors may be inhibited and poorly activated as the hip moves dynamically, leading to overactive lumber spine musculature and a tendency to lean on the hip flexors in order to complete the desired movement. The purpose of identifying these movement patterns is to compare these to the balanced hip biomechanics devoid of muscle lengths and strengths beyond the

normative ranges. LCS theory describes the hip flexor/extensor mechanism as a function of a flawed posture. LCS theory provides an answer to less than perfect postures through clinical observations whose theoretical underpinnings have not been tested sufficiently. While Janda explains a typical static posture through predictable muscle activation on both sides of the hip, it is dynamically that these prescribed activations are also observed. The running gait changes highlight typical muscle pattern activation that is in line with Janda's theory of LCS. These changes increase the risk of injury to distal joints. The relative tightness to the anterior hip created by the LCS posture forces the distal joint to compensate by increasing joint range and muscle activation. However, there is still limited quantifiable evidence confirming whether the changes to the length, strength and the onset of activation of hip flexor and hip extensor muscles can directly affect posture. Hip flexibility and hip strength need to be measured beyond just anecdotal evidence of observations. One and two-joint hip flexion ROM measured using a Modified Thomas test provides quantifiable evidence on the shortness of one and two-joint hip flexors. Isokinetic strength testing could provide the relevant data of hip extensors through their full active ROM, with patients observed with a LCS posture.

Chapter Three: Methodology

3.1 Subjects

The purpose of this observational study was to determine the relationship between hip flexor length and hip extensor strength in recreational runners. In order to fulfil the objectives of the study, a convenience sample of recreational level runners from the running club, Varsity Kudus, based at the University of Witwatersrand in Johannesburg, was invited to participate. The invitation was then extended to surrounding running clubs in Johannesburg. An information sheet (Appendix C) was provided to each participant to explain the details of the study.

3.1.1 Inclusion criteria

Potential participants needed to be recreational runners with an age greater than 18 years old, running more than 15 kilometres per week, with no current musculoskeletal injury.

3.1.2 Exclusion criteria

In order to remove any extraneous variables created through the influence of injuries from other sports, participants who also play sports like aerobics, dancing, basketball, volleyball, and racquet sports more than five times per week were excluded. Other reasons for exclusion included pregnant females and any current hip or knee injuries or past injuries (within the previous 12 months). Participants with conditions such as osteoarthritis, and total hip replacement, that might compromise the normal running gait or strength of the participants, were also excluded. Participants on medication that

directly affected skeletal muscle, by decreasing skeletal muscle tone, were also excluded.

3.2 The Testing Evaluation

3.2.1 Information Sheet, calibration and measuring of height, weight

A participant data sheet (Appendix C) was completed by all potential participants on accepting the invitation to participate in the research report. The data sheet (Appendix C) also included all relevant medical history and injury history relevant to the participant's running experience. A standardised explanation of the details of the study was conveyed verbally to the participants. The participants were also given an information sheet that conveyed a written explanation on the details of the study, (Appendix E). When the participant had agreed to participate, written consent was obtained (Appendix F). The height and weight of the participants were measured after the information sheet was completed. All participants were required to take off their shoes as both anthropometric measurements were performed barefoot. The weight scale and the Biodex System 3 isokinetic dynamometer were calibrated once at the beginning of the testing procedure. The same scale had been used to weigh all participants. The same goniometer was used to measure one and two-joint hip flexion ROM on all participants. All testing procedures took place on the same day at the Centre for Exercise Science and Sports Medicine (CESSM). All goniometric and isokinetic measurements were written into the testing data sheet (Appendix D).

3.2.2 Description of Modified Thomas test

One and two-joint hip flexion ROM was measured using the Modified Thomas Test. For this test, each participant sat on the end of a plinth. Each participant rolled backwards on to the plinth while holding both knees to the chest. This position ensured that the pelvis was posteriorly tilted (flat on the plinth) and the lumbar spine remained flexed on the plinth. The participant then held only one limb close to his/her chest, (this ensured a stable pelvic position), while dropping the other limb to be measured. The un-held limb hung over the plinth towards the floor with the knee, unsupported, in a relaxed position (see Figure 3.1 below). The head and shoulders of each participant remained in a flat position on the plinth throughout the test. The experimenter used a goniometer to measure the angle formed between a horizontal reference line (the lateral midline of the pelvis) and a line connecting the greater trochanter to the lateral femoral epicondyle[67]. Three readings were taken on both the left and right side. The average measurement based on the three readings was used in analysing the data. Measurements were taken to the closest degree.



Figure 3.1: the Modified Thomas Test[67]

One-joint hip flexion ROM (test 1) was determined by measuring the angle formed between a horizontal reference line (the lateral midline of the pelvis) and a line connecting the greater trochanter to the lateral femoral epicondyle. See Figure 3.2 below[67].



Figure 3.2: Measuring One joint hip flexion ROM [60]

The two-joint hip flexion ROM (test 2) was determined by measuring the angle formed between the lines connecting the shaft od the femur and the lateral femoral condyle with the shaft of the fibula. See Figure 3.3 below[67].



Figure 3.3: Measuring two joint hip flexion ROM [67]

3.2.3 Hip extensor strength testing: Biodex System 3

The Biodex System 3 isokinetic dynamometer (Biodex Medical, Shirley, NY) was used to test the ipsilateral hip extensor strength. This machine has been found to be both valid and reliable[56]. The participants were positioned over the Biodex Isokinetic dynamometer with their trunk flexed at 90° and their arms wrapped around the base of the Biodex chair (see Figure 3.4 below). The axis of rotation for the dynamometer was aligned with the greater trochanter of the femur of the testing leg. The lever arm was attached to the posterior thigh just superior to the level of the knee joint. The non-tested leg supported the body of the participant[77]. A strap was also placed across the lower back of the participant to minimise any movement on the part of the participant.



Figure 3.4: the Setup of the Testing Protocol on the Biodex Systems 3 dynamometer[77]

The participants were told to exert maximal effort against the dynamometer in both hip extension and hip flexion. The testing knee of each participant remained flexed at 90° throughout the testing procedure.

The verbal Instructions given are set out below:

1. Keep your knee flexed so that your calf muscle stays in contact with the bottom of the lever arm pad at all times.
2. Kick up to the ceiling.
3. Then pull your hip towards yourself at hard as you can.
4. Keep your head down at all times.

Participants used eight practice repetitions to familiarise themselves with the testing procedure without effort. Effort was gradually increased during the practice, for example, the first repetition was done at 50 % of maximal testing effort, the second repetition at 75 % of testing effort, and the third repetition at 90% of testing effort[69]. Thereafter, the participant then performed the next five continuous maximum effort repetitions at 60°/s as part of the first testing set. The right hip was always tested first followed by the left hip. Both hips were tested first before the second test of five repetitions began. After a two-minute rest, the same set was repeated as part of the second testing set. Positive reinforcement was provided throughout the five repetition protocol. Consistent encouragement was given during both sets.

When the isokinetic testing had been completed all participants were given an exercise form (Appendix G) with hip flexor stretches for shortened overactive hip flexor muscles and hip extensor strengthening exercises for weak and inhibited hip extensor muscles.

3.3 Ethics

Before beginning the study, the appropriate and relevant consent forms were issued to all participants (Appendix F). The Human Ethics Committee of the University of the Witwatersrand approved the undertaking of this study with clearance certificate number M120132 (Appendix H). Each participant was invited to participate in a counselling session before participating in the study. Medical history (appendix C), relevant to the study, and consent from each participant was sought before taking part in the study. All information provided by each participant was completely confidential, only the tester and supervisors had access to the completed information sheets. An anonymous study number was used to identify each participant. These study numbers ensured anonymity for all participants. There was no form of compensation for participating in the study.

3.4 Data Analysis

Descriptive statistics including the mean, median and standard deviation of the one and two-joint hip flexion ROM and the ipsilateral hip extensor muscle torque and total work were generated for each participant. Independent t-tests were done to determine the differences between the male and female population. Dependant t-tests were done to identify the differences between left and right limb. In order to observe the relationship between one and two-joint hip flexion ROM and hip extensor torque, correlations between one and two-joint hip flexion ROM and hip extensor torque and total work were performed using Pearson's correlations. The STATISTICA (version 10) package was used to generate the mean and standard deviation as well as

Pearson’s correlations using the study’s continuous data. Statistical significance was set at $p<0.05$.

Chapter Four: Results

4.1 Introduction

The aim of this research report was to determine whether there is a relationship between the hip flexor and hip extensor of the same side. The researcher sought to determine if it is indeed possible to describe an ipsilateral hip in terms of its hip flexor/extensor mechanism. This mechanism, if it does exist, would provide an explanation of how the relative length of one group of muscles is dependent on the action and activation of another ipsilateral group of muscles. Therefore, from the 32 participants who participated in the study (N=32), 64 hips were tested ipsilaterally. There are numerous studies that have used recreational runners to create a better understanding around hip strength and running gait cycle, that also have used sample sizes in the region of 32 participants [51,71,72,73,74,75].

Both the left and right hips of the participants were tested creating data based not only on the 32 who participated but also on the 64 hips tested in the study. Therefore the correlation from the data collected of the one-joint and two-joint hip flexor length with hip extensor isokinetic torque is actually based on 64 individually tested hips from a population of 32 participants. These measurements have been captured and reported as total population then separated according to side as left and right hips and according to gender as male and female.

The results section will discuss, firstly, the length of the hip flexors for this population and secondly, the hip extensor strength in the group. Lastly, the relationship between the hip flexors and extensors will be discussed.

Table 4.1 below shows the descriptive statistics for the anthropometric data of the sample. The average weight of the all the participants tested was 70.37kg with a standard deviation (SD) of ±15.2kg. The average age of the 32 participants was 33.28 years ±8.5 years old and the average height was 164.11cm ±43.82 cm.

Table 4.1:The anthropometric measurements of the sample (n=32)

	Mean (±SD)	**Median (Range)**
Age(Years)	33.28 (±8.52)	32(19-57)
Height (cm)	164.11 (±43.82)	176(151-201)
Weight (kg)	70.37 (±15.21)	73.05 (45-120)

Table 4.2 below shows the comparison of the male and female anthropometric measurements. The 32 participants were made up of 11 females and 21 males. Both left and right hips were tested providing a total of 64 limbs. Therefore the female population contributed 22 testable hips and the males 42 testable hips. The male participants were slightly older than their female counterparts, and weighed over 20kg more. The males were also on average seven centimetres taller than the female participants

Table 4.2: Comparison of the height, weight and age of the male and female population

	Males (n=21)		Females (n=11)	
	Mean (±SD)	**Median (range)**	**Mean (±SD)**	**Median (range)**
Age(Years)	34.14 (±8.45)	33 (21-57)	31.64 (±8.6)	30 (19-46)
Height (cm)	171.63 (±39.66)	178 (171-201)	164.51 (±48.59)	161.5 (151-181)
Weight (kg)	78.61 (±11.52)	76 (57.9-120)	54.65 (±6.33)	57(45-65)

4.2 Hip Flexor Length

4.2.1 Hip flexor length for all participants

Table 4.3 below shows the descriptive statistics including the mean, median and standard deviation of the one and two-joint hip flexion ROM. The mean one-joint hip flexor ROM was 12.12° (±8.6°). The mean two-joint hip flexor ROM was 48.88° (±13.07°).

Table 4.3: Hip flexion range of movement (N=64)

	Mean (±SD)	**Median (range)**
1 HF (°)	12.12 (±8.66)	11.17 (-6-35)
2 HF (°)	49.59 (±14.76)	46.50 (21-81)

1 HF: One joint hip flexor; 2 HF: Two joint hip flexor

Table 4.4 below shows the mean value of the one joint hip flexion ROM of the right hips was 0.17^0 higher than the mean value of the left hips. However this difference was not statistically significant. The mean value of the two-joint hip flexion ROM of the right hips was 1.30° higher than the mean value of the left hips. However this difference was not statistically significant.

Table 4.4: Comparison of the right and left hip flexors (n=32)

	Right side (n=32)	**Left side (n=32)**	**P-value**
	Mean (±SD)	**Mean (±SD)**	
1 HF (°)	12.04 (±8.19)	12.21 (±9.25)	0.316
2 HF (°)	49.52 (±16.34)	48.22 (±13.23)	0.699

1 HF: One joint hip flexor; 2 HF: Two joint hip flexor; *indicates statistical significance

Table 4.5 below shows the classification of the one and two-joint hip flexor ROM in terms of tight, normal, and increased ROM[37]. The majority of the total population, 75% (n=48) had normal one-joint hip flexion ROM and 56.3% (n=36) had normal two-joint hip flexion ROM.

Table 4.5: Classification of hip flexor ROM[37]

	One joint hip flexor		**Two joint hip flexor**	
Range	**ROM (^{0})**	**N (%)**	**ROM (^{0})**	**N (%)**
Tight	-20-0	6 (9.4)	15-45	23 (35.9)
Normal ROM	0-<20	48 (75)	45-75	36 (56.3)
Increased ROM	20-40	10 (15.6)	75-105	5 (7.8)

Table 4.6 below shows the comparison of the one and two-joint hip flexion ROM in the male and female population. The mean value of one-joint hip flexion ROM in the male population was 12.83° (±7.76°) while the mean value in the female population was 10.77° (±10.23°). Therefore the mean value of the male population was 2.06° higher than that of the mean value of the female population.

The mean value of the two-joint hip flexion ROM in the male population was 46.99° (±11.44°) while the mean value of the two-joint hip flexion ROM in the female population was 54.53° (±14.77°). Therefore the mean in the female population was 7.53^0 higher than the mean value of the male population. The males were significantly (p=0.026) tighter in their two-joint hip flexors than were the females.

Table 4.6: Comparison of the hip flexor ROM in males and females

	Males (n=42)		**Females (n=22)**		
	Mean (±SD)	**Median (range)**	**Mean (±SD)**	**Median (range)**	**p-value**
1 HF (°)	12.83 (±7.76)	11.83 (-6.00-30.00)	10.77 (±10.23)	10.33 (-3.66-35.00)	0.37
2 HF (°)	46.99 (±11.44)	46.33 (21.00-81.00)	54.53* (±14.77)	46.67 (31.66-80.66)	0.026

1 HF: One joint hip flexor; 2 HF: Two joint hip flexor; *indicates statistical significance

4.3 Hip isokinetic strength

4.3.1 Hip isokinetic Strength for all participants

Table 4.7 below shows the descriptive statistics of hip flexor and extensor isokinetic values of the total population (N=64). The mean absolute hip extensor peak torque was 184Nm (±66.79). The mean relative hip extensor peak torque was 257.38Nm (±59.04). The mean average hip extensor peak torque was 168.14Nm (±59.67). Total work had a mean of 764.73 Joules (±283.72) and relative total work was 252.33% (±122.93). The mean hip flexor torque was 154Nm (±52.98). The mean relative hip flexor torque was 225.64% (±49.84). The antagonist/agonist ratio was 85% (±16.33).

Table 4.7: Isokinetic hip strength values for all hips (N=64)

	Mean (±SD)	**Median (range)**
Extensor PT (N-m)	184.00 (±59.04)	171.20 (80.7-328.9)
Extensor PT/BW (%)	257.38 (±59.67)	247.95 (144-392.3)
Extensor AV PT (N-m)	168.14 (±283.72)	150.30 (72.1-281.6)
Extensor Work (Joules)	764.73 (±122.99	689.15 (228.3-140.2)
Extensor WRK/BW (%)	233.52 (±55.38)	234.25 (112.5-357)
Flexor PT (N-m)	149.68(±52.98)	155.60 (58.3 -247.1)
Flexor PT/BW (%)	208.28 (±49.74)	202.55 (122.2-316.4)
Ant/Agon (%)	82.18(±16.33)	80.20 (50 -142.7)

PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; Ant/Agon: Antagonist agonist ratio

4.3.2 Comparison of the of the isokinetic values in right and left hips

Table 4.8 below shows all mean left hip flexor and extensor isokinetic values were higher than those of the right hip in the total population.

Left hip extensor peak torque was 1.09 Nm larger than the right hip. Left relative Peak torque was also 2.08 % higher than the right hip.

These results were unexpected, as the majority of the participants were found to be right dominant. This may be explained by the fact that the testing procedure called for all right hips to be tested first. There might have been a possible learning effect from the first tested hip that benefited the testing procedure of the left hip.

Total work of the left hip was 5.74J higher than the right hip, while the relative total work of the left hip was 20.01% higher than the right hip.

The hip flexor peak torque of the left hip was on 6.16 Nm higher than the right hip with the antagonist/agonist ratio of the left hip also 2.43% higher than the right hip.

Table 4.8: Comparison of isokinetic strength between the left and right sides

	Right (n=32) Mean (±SD)	Left (n=32) Mean (±SD)
Extensor PT (N-m)	183.46 (±67.68)	184.55 (±66.65)
Extensor PT/BW (%)	256.32 (±58.63)	258.44(±60.37)
Extensor AV.PT (N-m)	167.96 (±59.04)	182.89 (±94.17)
Extensor Work (Joules)	761.85 (±276.17)	767.59 (±295.59)
Extensor WRK/BW (%)	235.04 (±58.37)	255.05 (±58.63)
Flexor PT (N-m)	146.60(±57.01)	152.76(±53.22)
Flexor PT/BW (%)	204.10(±42.72)	212.56(±46.21)

PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; Ant/Agon: Antagonist agonist ratio

4.3.3 Comparison of the isokinetic values in male and female population

Table 4.9 below shows the comparison of hip extensor and flexor isokinetic values in the male and female population.

The peak hip extensor torque of the male population was on average 91.88Nm higher, which was significantly higher than that of the females (p=0.000). Interestingly, even when normalising for body weight, the male populations strength was still significantly (p=0.03) higher (44.31%) than that of the females. Similarly, the total work in the male population was significantly (p=0.000) higher, at 341.82J higher than the females.

However, when this was normalised for body weight, the 42.84% difference between the two was not significant.

The peak hip flexor torque in the male population was on average 69.81 Nm higher than the females (p=0.000). Even when normalised for body weight the males were significantly (p=0.000) stronger (48.11%) than the females.

The antagonist/agonist ratio was 12.60% higher in the male population, but this was not statistically significant (p=0.243).

Table 4.9: Comparison of isokinetic strength between the males and females

	Males (n=42)		**Females (n=22)**		
	Mean (±SD)	**Median (range)**	**Mean (±SD)**	**Median (Range)**	**p value**
Extensor PT (N-m)	215.59* (±60.62)	207.95 (83.00- 328.9)	123.71 (±20.03)	130.85 (80.70-154.20)	p<0.05
Extensor PT/BW (%)	272.61* (±62.72)	252.05 (144.00-392.30)	228.30 (±37.77)	240.75 (149.80-287.50)	p=0.03
Extensor AV.PT (N-m	196.21* (±54.01)	187.9 (80.80- 281.60)	114.56 (±20.10)	123.65 (72.10-148.30)	p<0.05
Extensor Work (Joules)	887.70* (±268.94)	832.35 (383.10-1402.00)	545.88 (±88.3)	574.2 (336.10-687.50)	p<0.05

Extensor WRK/BW (%)	261.58 (±138.23)	238.55 (114.30- 357)	218.74 (±40.92)	229.7 (148.00-293.100)	p=0.161
Flexor PT (N-m)	173.67* (±44.56)	175.65 (60.9-247.1)	104.48(±39.75)	97.57 (225.3-66)	P=0.000
Flexor PT/BW (%)	225.2* (±46.352)	234.07 (135.33-315.58)	177.09 (±40.79)	165.390 (274.76-123.81)	P=0.000
Ant/Agon (%)	86.49 (±17.077)	84.45 (59.5-142.7)	73.89 (±12.57)	74.4 (97-53.5)	P=0.243

PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; Ant/Agon: Antagonist agonist ratio

* reflects statistical significance

4.4 The correlational results of hip flexor length and hip isokinetic strength

4.4.1 The correlational results of hip flexor length and hip isokinetic strength in the total population

Table 4.10 below shows the correlation results with respect to the measured variables of all the participants tested in this study.

A very small, insignificant (p>0.05) effect size was seen for the one-joint hip flexor ROM and strength. There was a correlation of 0.01 with absolute peak and 0.18 with relative torque. The one-joint hip flexor ROM was found to have a correlation of -0.13 with total work and 0.11 with relative total work.

The two-joint hip flexor ROM had a slightly larger negative correlation than the one-joint hip flexors. There was a statistically significant (p=0.03) correlation of -0.27 with

peak torque and -0.30 (p=0.02) with relative peak torque. Although the results were statistically significant the correlational coefficient was still not large enough to reflect a strong negative correlation.

The two-joint hip flexor ROM had weak negative, and non-significant correlation with work. A correlation of -0.19 with found for total work, and - 0.02 for relative total work, respectively.

The one-joint hip flexor ROM had a correlation of -0.18 with the two-joint hip flexor ROM.

Table 4.10: Correlations between the hip flexion ROM and isokinetic strength (N=64)

N=64 p<0.05	PT (N-m) *r* (p-value)	PT/BW (%) *r* (p-value)	AV.PT (N-m) *r* (p-value)	Total Work (Joules) *r* (p-value)	WRK/BW (%) *r* (p-value)	1 HF (°) *r* (p-value)
1 HF (°)	0.001	0.18	-0.005	-0.01	0.11	
	(p= 0.99)	(p=0.15)	(p=0.97)	(p=0.92)	(p=0.37)	
2 HF (°)	-0.27*	-0.30*	-0.28*	-0.19	-0.08	-0.18
	(p=0.03)	(p=0.02)	(p=0.03)	(p=0.13)	(p=0.52)	(p=0.15)

PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; 1 HF: One-joint hip flexor; 2 HF: Two-joint hip flexor; *reflects statistical significance

4.4.2 The correlational results of the hip flexor length and hip extensor strength of the male population

Table 4.11 below shows the correlation results with respect to the measured variables of the male participants tested in this study.

Small, non-significant ($p>0.05$) correlations were found with the one-joint hip flexion ROM and strength. For example, a correlation of 0.10 was found with peak torque/body weight and a -0.18 correlation with peak torque, -0.21 with average peak torque and -0.20 with total work. There was also a 0.08 correlation with relative total work.

The same trend was found for the two-joint hip flexion ROM, which had a correlation of -0.05 with peak torque, -0.05 peak torque/body weight and 0.04 with average peak torque. A 0.08 correlation was found with total work and 0.08 with total work/body weight.

The one-joint hip flexion ROM had a 0.26 correlation with the two-joint hip flexion ROM. However, none of the correlational results reported for the male population were found to be statistically significant.

Table 4.11: Correlations of hip flexion ROM and the hip extensor isokinetic values in the males

N=42 p<0.05	PT (N-m) r (p-value)	PT/BW (%) R (p-value)	AV.PT (N-m) r (p-value)	Total Work (Joules) r (p-value)	WRK/BW (%) R (p-value)	1 HF (°) R (p-value)
1 HF (°)	-0.18 (p=0.27)	0.10 (p=0.53)	-0.21 (p=0.19)	-0.20 (p=0.21)	0.08 (p=0.61)	
2 HF (°)	-0.05 (p=0.7)	-0.05 (p=0.78)	-0.04 (p=0.78)	0.08 (p=0.6)	0.08 (p=0.61)	0.26 (p=0.09)

PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; 1 HF: One-joint hip flexor; 2 HF: Two-joint hip flexor; *reflects statistical significance

4.4.3 The correlational results of the hip flexor length and hip extensor strength of the female population

Table 4.12 below shows the correlation results with respect to the measured variables of the female participants tested in this study.

The study found only small, non-significant (p>0.05) correlations when looking at the one-joint hip flexion ROM and strength. For example, a 0.17 correlation was found for peak torque/body weight and a 0.31 for peak torque and 0.23 for average peak torque. Furthermore, when looking at one-joint hip flexion ROM and work, a 0.26 correlation was found for total work, and 0.27 for relative total work, respectively.

However, when assessing the relationship between the two-joint hip flexion ROM and strength, a much larger, statistically significant relationship emerged. For example, a medium, statistically significant, correlation was found for the two-joint hip flexor ROM and peak torque (r=-0.56, p=0.000). Furthermore, an even stronger relationship

(r=-0.72) was found between the two joint ROM and relative strength (p=0.000).

This relationship extended to two-joint hip flexor ROM and work, with a significant medium relationship with total work (r=-0.62; p=0.002) and relative work (r=-0.66; p=0.001).

The one-joint hip flexion ROM had a medium negative, but significant correlation with the average length of hip flexor length measured at the knee (r=-0.64; p=0.001).

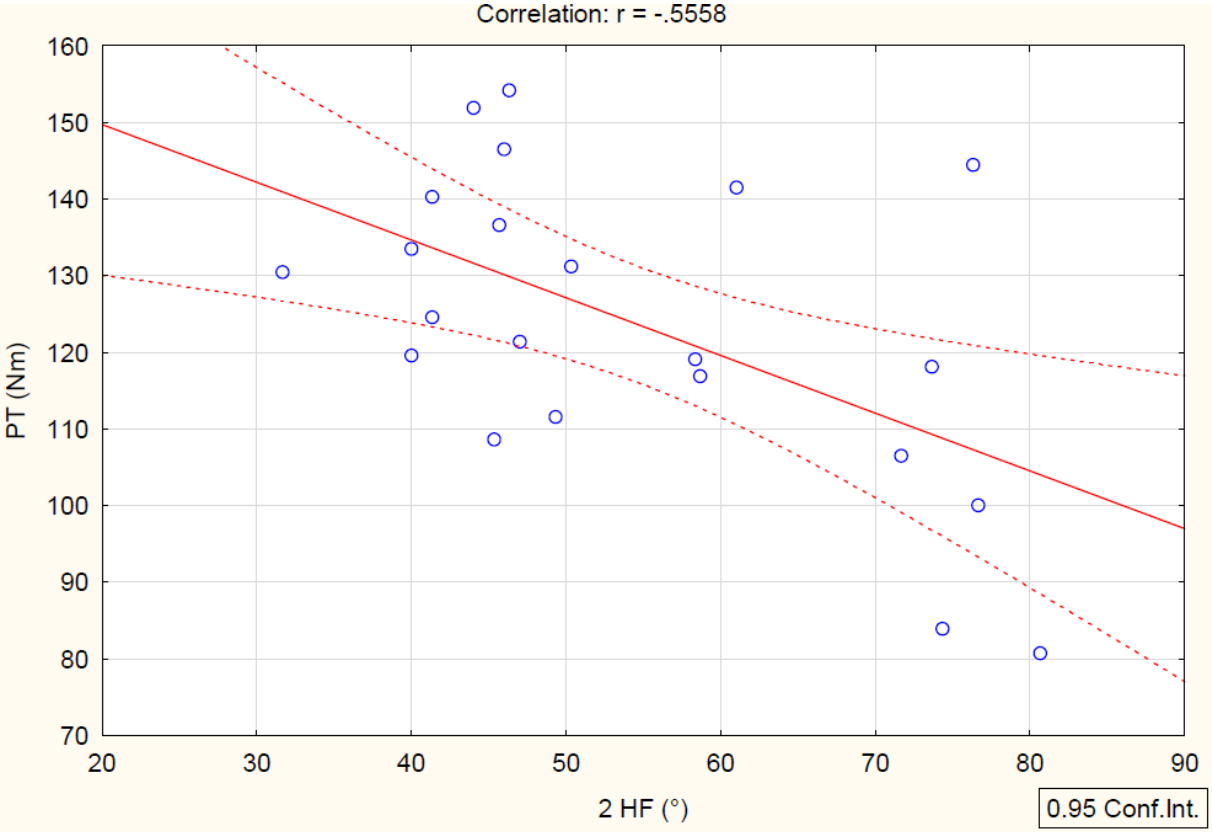
Table 4.12: Correlations of hip flexion ROM and the isokinetic values in the females

N=22 p<0.05	PT (N-m) r (p-value)	PT/BW (%) R (p-value)	AV.PT (N-m) r (p-value)	Total Work (Joules) r (p-value)	WRK/BW (%) r (p-value)	1 HF (°) R (p-value)
1 HF (°)	0.17 (p=0.17)	0.31 (p=0.16)	0.23 (p=0.31)	0.26 (p=0.25)	0.27 (p=0.23)	
2 HF (°)	-0.56* (p=0.000)	-0.72* (p<0.05)	-0.58* (p=0.005)	-0.62* (p=0.002)	-0.66* (p=0.001)	-0.64* (p=0.001)

PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; 1 HF: One-joint hip flexor; 2 HF: Two-joint hip flexor

*reflects statistical significance

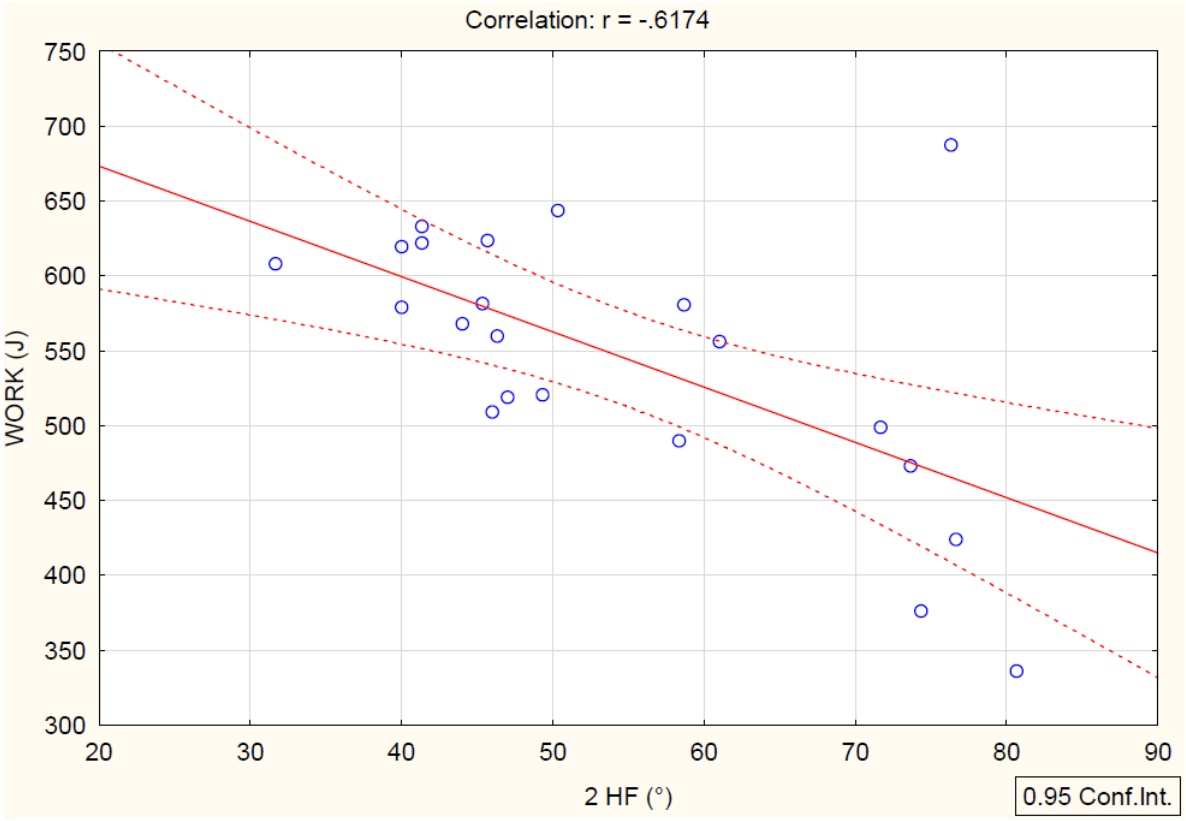
The Figure 4.1 below shows a scatterplot of the correlation between the two-joint hip flexion ROM and total work. This distribution graphically represents the strong negative correlation between these two variables. Therefore it appears that as the two-joint hip flexion ROM increases the hip extension strength decreases.



2 HF: Two-joint hip flexor; PT: peak torque

Figure 4.1: A scatterplot of the correlation between two-joint hip flexion ROM and peak torque.

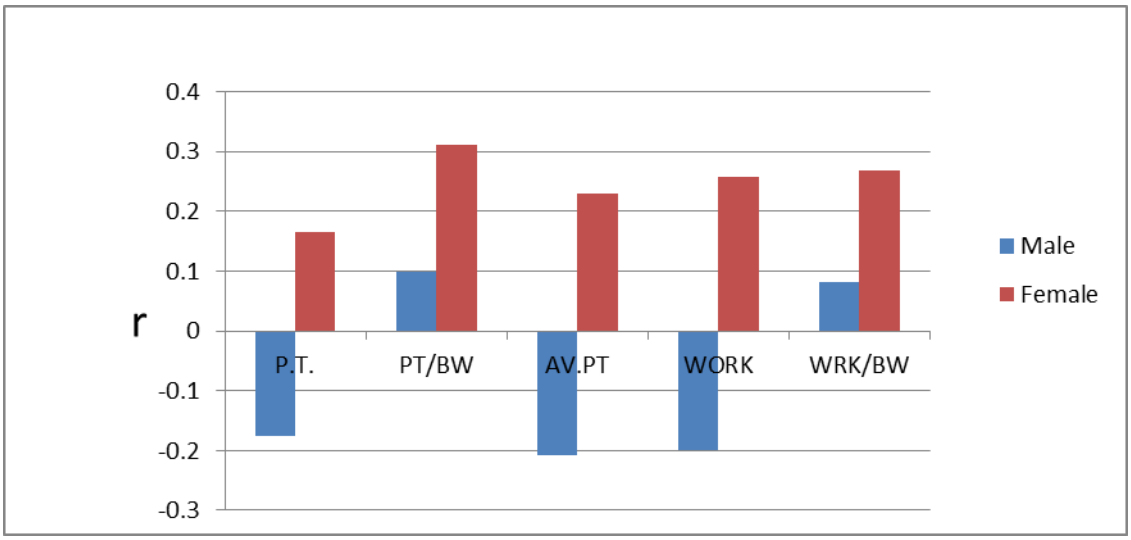
The figure 4.2 below shows the scatterplot of the correlation between the two-joint hip flexion ROM and Total work. This distribution graphically represents the strong negative correlation between these two variables. Therefore in additional to strength it appears that as the two-joint hip flexion ROM increases the overall muscle performance of the hip extensors also decreases.



2 HF: Two-joint hip flexor; Work: Total Work

Figure 4.2: A scatterplot of the correlation between two-joint hip flexion ROM and total work.

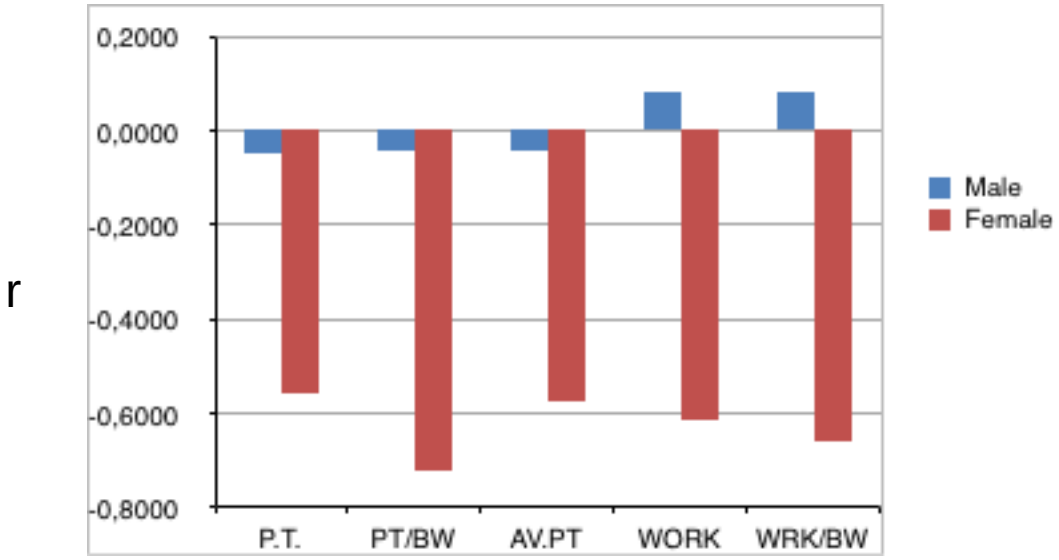
Figure 4.3 below shows the comparison of the correlation of the hip extensor isokinetic dynamometer values, and the one-joint hip flexion ROM of the male and female population. The figure displays the female population with a positive correlation while the male population demonstrates a negative correlation in four of the five measured variables when correlated with one-joint hip flexion ROM. However none of these results exhibited a strong enough correlation and they were also found not to be statistically significant.



X axis: PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; 1 HF: One-joint hip flexor; 2 HF: Two-joint hip flexor. **Y** axis: r= correlation coefficient

Figure 4.3: A comparison between the males and females for correlational results of the one-joint hip flexor ROM

Figure 4.4 below shows the comparison of the correlation of the hip extensor isokinetic dynamometer values, and the two-joint hip flexion ROM of the male and female population. The figure displays the female population with a strong negative correlation, while the male population demonstrates no correlation in all five measured hip extensor isokinetic values. All the correlational results of the male population were not found to be statistically significant, while all correlational results of the female population were found to be statistically significant.



X axis: PT: Peak torque; PT/BW: Relative peak torque; AV. PT: Average peak torque; WRK/BW: Relative work; 1 HF: One-joint hip flexor; 2 HF: Two-joint hip flexor. **Y** axis: r= correlation coefficient

Figure 4.4: A comparison between males and females of correlational results for the two-joint hip flexor ROM

Table 4.13 below shows the correlation of the one-joint hip flexion Rom reclassified into tight, normal and increased ROM with hip extension peak torque. There are two strong negative correlations, tight classification of -0.73 in total population and -0.72 in the males. However none of the correlations were found to be statistically significant.

Table 4.13: Frequency table and correlations for 1 HF [37]

	Total population (N=64)		Females (n=22)		Males (n=42)	
Classification	N (%)	PT (r-value)	N (%)	PT (r-value)	N (%)	PT (r-value)
Tight (<0°)	6 (9)	-0.73	3 (14)	0.14	3 (7)	-0.72
Normal (0°-19°)	48 (75)	0.21	15 (69)	0.13	33 (79)	0.01
Increased ROM (20°-40°)	10 (16)	0.49	4 (18)	0.32	6 (14)	-0.52

PT: Peak torque

* reflects statistical significance

Table 4.14 below shows the correlation of the two-joint hip flexion ROM reclassified into tight, normal and increased ROM with hip extension peak torque. Only the strong negative correlation of (-0.58) of the normal two-joint hip flexion ROM was found to be statistically significant.

Table 4.14 Frequency table and correlations for 2 HF [37]

	Total population (N=64)		Females (n=22)		Males (n=42)	
Classification	N (%)	PT (r-value)	N (%)	PT (r-value)	N (%)	PT (r-value)
Tight (15°-45°)	23 (36)	0.36	6(28)	0.39	17(41)	0.46
Normal (45°-75°)	36 (56)	-0.22	13(59)	-0.58*	23(55)	-0.23
Increased ROM (75°-105°)	5 (8)	-0.15	3 (14)	-0.78	2(5)	

PT: Peak torque

* reflects statistical significance

CHAPTER FIVE: DISCUSSION

The purpose of this research report was to investigate the relationship between hip flexor length and hip extensor strength in recreational runners. To achieve this, 32 participants, including 21 males and 11 females, were recruited to participate in the study.

The one-joint and two-joint hip flexor ROM were measured along with the hip extensor peak torque and total work in order to determine if a relationship exists. This chapter will provide a discussion of the findings of the current study in comparison to current literature.

5.1 Hip flexion ROM

5.1.1 One-joint hip flexion ROM

The first objective of the study was to determine the hip flexor length in recreational runners. The current study found the mean value for one-joint hip flexors in this group was 12.12°. This is similar to Harvey et al., who tested 117 athletes using the Modified Thomas test, and reported a mean value of 11.91°[67]. Furthermore, these values are just above the normal range as described by Kendall et al., of 10°[37]. This normal range was further supported by Ferber et al., who reported one-joint hip flexion ROM of 10.60^0 was based on 300 recreational athletes[62].

The examination procedure plays an important role in expected hip length values. For instance, Corkery et al., reported one-joint hip flexion ROM values in the range of 2.1^0

to 2.4$^{0.}$ However this ROM was measured with the knee on the examination bed and thus did not allow full hip extension[62]. The similarity of the current research report's results and four other studies reporting results of 11.14^{o}, based on a combined population of 356[62,65,70,77], can be largely explained by the use of the same testing procedure.

However when this research report's mean value of 12.12^{o} was compared to runner specific populations it was found to be slightly lower. The running population has been found to have a slightly tighter and shorter one-joint hip flexion ROM. For example, Harvey et al., reported a mean value of 14.15^{o} for a running population[67]. Furthermore, Schache et al., reported a 17.4^{0} one-joint hip flexor length based on 14 track athletes. However, when this study population was reclassified into tight, normal and increased ROM of one-joint hip flexion ROM, only 9.4% were found to be tight. Unexpectedly, the current runners were far less tight than anticipated. According to Dillman et al., as running speed increases, there is an increase in hip flexion duration and a decrease in hip extension time[66]. It was, therefore, hypothesised that this increased hip flexion time would produce tighter hip flexors when compared to the normal expected length[64]. The recreational runners in the current study, however, exhibited hip flexor length within the accepted range.

This may be explained by the fact that dynamic movements of the hip and pelvis during running do not always reflect in static measurements. For example, Schache et al., found that static hip flexibility, using the Modified Thomas test, was not reflective of dynamic running movements. The author suggested that flexibility measured statically may not directly impact anterior pelvic tilt or peak hip extension ROM in a running

population. A hypothetical set of influences was suggested including dynamic neuro-motor pattern changes, pelvic positioning and even other external and extraneous forces that may act directly on the hip joint rather than just static flexibility[50].

Furthermore, there may be differences in one-joint hip flexion ROM across the various sporting codes. For example, Harvey et al., found that rowers and tennis players had less iliopsoas flexibility than runners. Runners are able to reach up to 20° of hip extension within their running gait cycle. Rowers and tennis players, however, are in a position of hip flexion for most of their activity. Therefore, running may actually provide more active ROM than other sports, and this may be a potential explanation for the normal range experienced in the population of the current study.

5.1.2 Two joint hip flexion ROM

Kendall et al., define two-joint hip flexor tightness as the inability of the knee to achieve 80^0 when in a Thomas test position[37]. This research report had a lower mean value of 49.59°, which is approximately 30° below the normal value stated by Kendall et al. The reason for the lower mean value may be due to two factors, namely the testing procedure and the specific population.

Firstly, the testing procedure may account for the variation in values. The Modified Thomas test has been described as a more functional position for testing runners. Gabbe et al., described the test position as the hip extended and the knee flexed with gravity. This position mimics the terminal stance and pre-swing position of the leg while running or sprinting[63]. In a study using the same testing procedure, Harvey et

al., found the mean value for the two-joint hip flexors were 52.51°, which supports the values found in the current study[67].

Secondly, despite a lower mean value from normal, the results of the study was consistent with other studies that assessed runners specifically. Harvey et al., found a mean value of 50.59° for the two-joint hip flexors for runners. This was in line with the research of Van Heerden et al., who reported a two-joint hip flexion ROM of 50.8°, based on 20 middle distance runners[61].

According to a study by Gabbe et al., the Modified Thomas test has been identified as the test of choice when attempting to predict time to hamstring injury in amateur athletes. The risk of incurring a hamstring injury increases with a rectus femoris length, as measured using the Modified Thomas test, of anything less than 51° [63]. Therefore, with a mean value of 49.59°, the participants of the current study indicate that these runners may be at risk of developing hamstring injuries.

Furthermore, the current study highlights a clinically important gender difference in the two-joint hip flexor ROM. This research found significantly lower mean values for males when compared to females. This is similar to findings by Corkery et al., who found males to have 2.5° less ROM when compared to their female counterparts[62]. Therefore, within a clinical setting, it is important for clinicians to be aware of the gender differences when measuring two-joint hip flexion ROM using the Modified Thomas test.

The results of the current study demonstrate that the runners had normal one-joint hip flexion ROM. Although this was unexpected for a group of runners, the values appeared to relate to the general athletic population. Furthermore, the current study found a marked decrease in two-joint hip flexion ROM when compared to the normative values indicated by Kendall et al.[37] This may indicate patterns of tightness in runners, and should be assessed relative to injury risk. In addition, clinicians should be aware of gender differences when assessing hip flexibility in runners.

5.2 Hip Strength

Differences have been found in both positioning on the isokinetic dynamometer and isokinetic speed when testing hip strength. In isokinetic testing of the knee, peak torque values change when the testing position and speed are altered [78, 79]. Isokinetic hip extension peak torque has been reported with participants tested in supine, prone and standing positions, making results inconsistent and thus impossible to compare. Thorpe et al., tested relative hip extension strength in supine at 60°/sec with reported results of 139%. This research report, testing in the prone position, also at 60°/sec, reported much larger relative strength (257%) [80].

However when the hip extensor isokinetic strength is tested in the standing position it appears to produce even higher values than in prone and supine. For example, Tis et al., using a standing position, reported mean hip extensor peak torque of 297.8Nm at 20°/sec81. At 60°/sec, Sugiura et al., reported a mean hip extensor peak torque value of 250.25Nm. Therefore, the positioning of the participants, and the variable ROM, as well as the chosen isokinetic speed, all affect the potential to generate peak torque[82].

The current study used a unique testing protocol in the prone position, which, to the authors' knowledge, has only been described only once before. The mean hip extensor isokinetic values in this research report are far higher (184 Nm) than the previous literature using the same protocol (136.47 Nm). The possible reason for the difference in strength was that Boling et al., used a single fixed ROM whereas this research used ROM that was participant-specific[76].

It is well known that, in addition to testing position and speed, gender also affects isokinetic torque production. Worrel et al., found males recorded higher absolute and relative peak toque value than the female participants. Similarly, the current study found that men produced significantly more than the women, however this gap expectedly decreased when normalised for body weight[78]. Although absolute strength values are important to report, hip muscle strength has been found to be closely associated with body mass[83].

The current study also found decreased hip flexion strength, when compared to testing in standing. However, when comparing for males only, Robinson et al., found an average peak torque of 171.4 Nm, which is comparable to the current study's male peak torque 173.67Nm, despite the difference in position[84].

Accordingly, the current study provides useful isokinetic data for the running population in a unique testing position. Hip extension strength was better in this population when compared to other studies using the same protocol.

5.3 The correlation of one-joint hip flexion ROM and isokinetic hip extension strength

Janda observed that shortened and overactive hip flexors and hamstrings provide sufficient torque to inhibit both the gluteal and abdominal muscles[3]. This research report attempted to reproduce the principles of the LCS by utilising quantifiable parameters to fulfil the objectives of the study. Overall the 32 participants tested yielded 64 tested hips. This research report used one-joint and two-joint hip flexion ROM to measure hip flexor length, and isokinetic hip extensor peak torque, and total work to measure hip extensor strength.

However no, or very little, correlation was found between the one-joint hip flexor ROM and the absolute isokinetic hip extensor peak toque. The one-joint hip flexor ROM reflected only a very small, insignificant, positive correlation with relative hip extensor peak torque, and did not reflect the desired relationship between tight hip flexors and weak hip extensors set out in the initial hypothesis. There is no previous literature testing these correlations, yet, Sole et al., found no change in the onset of gluteus maximus activation with increased hamstring activation post hamstring injury[35]. Therefore, Janda's observations suggest that gluteus maximus is inhibited and delayed in the onset of activation with increased hamstring activation. These results serve only to weaken Janda's LCS theory that increased hamstring activation would also result in increased hip flexor activation and conversely hip extensor inhibition and weakness.

Although it has been reported that tight hip flexors perform differently when exposed to hip extensor strength exercises, this does not appear to affect performance outcome. Therefore, there seems to be enough of an adaptation to the shorter, tighter iliopsoas to prevent a slump in performance unlike that described by Janda's LCS. Similarly to this study, Gage et al., also attempted to demonstrate how tight hip flexors would cause the pelvis to tilt anteriorly, but failed to confirm this relationship. Instead they proposed other factors, such as neuromuscular control of the abdominal muscles, which may contribute to pelvic tilt more than hip flexor tightness[85].

Likewise, Nourbakhsh et al., attempted to validate Janda's LCS in patients with LBP by measuring the strength of the abdominal and gluteal muscles along with the flexibility of the iliopsoas and back extensor muscles. However, this study failed to find a direct relationship between increased lumbar lordosis and lower back pain. Although weaker abdominal and gluteal muscles, shorter back extensor and hamstrings muscles were reported, hip flexor length remained unchanged. Therefore, the lower back pain participants did not present with a typical LCS posture as observed by Janda. The author considers the hamstring muscle shortness in patients with lower back pain to be a compensatory mechanism to reduce pelvic instability due to gluteal muscle weakness, and not something which is due to LCS posture[86].

Even when the current research population was redefined in terms of gender, the male population had no correlation with relative peak torque or total work load. The female population had a small correlation with relative peak torque and a small correlation with relative total work load. Although neither gender demonstrated a strong correlation or significant relationship there are obvious differences found in

these results when separated by gender. Female hip flexor/extensor mechanism is seen to have a closer relationship than that of their male counterpart within this study. Willson et al., found greater gluteal muscle activation in female runners as compared to the studied male participants studied during research conducted on running kinesiology. In fact, there was a 40 % increase in gluteus maximus and gluteus medius peak activation in females. Research conducted by Willson et al., may provide answers for the gender differences found in this study. It also provides evidence of gender-specific strategies employed in hip muscle activation during the running gait [87].

5.4 The correlation of two-joint hip flexion ROM and isokinetic hip extension peak torque and total work

In contrast to the one-joint hip flexors, the two-joint hip flexor ROM showed a small and yet significant negative relationship with relative and absolute peak torque. When separated by gender, the males showed no relationship between two-joint hip flexor ROM and isokinetic strength.

However, the female population reflected very different results. The female's two-joint hip flexor ROM showed a significant and strong negative correlation across all the five isokinetic hip extensor variables. This infers that the smaller the degree of knee flexion (the shorter the rectus femoris) the larger the hip extensor strength. Conversely this can also be interpreted as the larger the degree of knee flexion (the longer the rectus femoris) the weaker the hip extensors become. This result is unexpected, as the rectus femoris is a hip flexor and knee extensor, and was anticipated to be longer and more inhibited as the hip extensor strength increased.

Furthermore, Willson et al., found that females have greater gluteal activity during running over their male counterparts. They also suggested that early gluteal fatigue during running predisposes females to patellofemoral joint pain syndrome[87]. In addition, Prins and van der Wurff stated in a systematic review that females with patellofemoral joint pain syndrome have decreased hip muscle strength[20]. Souza et al., found that females runners diagnosed with patellofemoral joint pain syndrome demonstrated increased hip internal rotation along with decreased hip extensor strength. These runners exhibit as much as 15% decrease in hip extension and hip abduction strength. Gluteus maximus strength deficit decreases not only extensor strength but also both the external rotation and abduction forces necessary to inhibit a typical valgus knee joint pattern associated with patellofemoral joint pain syndrome[21]. Of importance is that this dysfunctional knee mechanism commonly includes overactive quadriceps, specifically rectus femoris. Waryasz et al., not only stated that overactive quadriceps were found in all five studies on patellofemoral joint syndrome but that a specifically tight rectus femoris was also found to be associated with patellofemoral joint pain syndrome[19]. However Chumanov et al., found that by increasing a running gait's step rate there was an increase in rectus femoris activity during the early swing phase of the cycle. The same study also found a concomitant increase in gluteus maximus and gluteus medius activity during the late swing phase of the same cycle[24].

The current study also found that in the females, the tighter and shorter the rectus femoris is, the longer and more inhibited the iliopsoas becomes. Sarhman provides anecdotal observations where tighter a rectus femoris can exist alongside a weaker

and inhibited iliopsoas. According to Sahrmann, relative flexibility exists, where increased stiffness or over activity of one muscle may result in the compensatory under activity of adjacent muscles. Furthermore, Sahrmann's movement impairment syndrome (MIS) states that an underactive iliopsoas muscle will contribute directly to lower back pain in conjunction with an overactive and often strained rectus femoris. Therefore when a muscle is strained, tight or overactive, this is accompanied by a weak and underactive synergistic muscle. The iliopsoas is the only hip flexor group capable of pulling the hip above 90 degrees. However, in the presence of a weak iliopsoas, the femur is still able to move above the level of the hip. This is facilitated through the momentum created primarily by its synergist the rectus femoris. Sahrmann's MIS does provide an interpretation of these reported results[88]. However Sahrmann's interpretation of the functional anatomy around the hip is based on postural observations and clinical interpretations, and like Janda's theories, also lacks quantifiable evidence.

5.5 Limitations

The limitations of this study may lie in the methodology. The protocols used to measure hip flexor length and hip extensor strength may be critical in the outcome of the results. In testing the hip extensor strength, a protocol was selected that would isolate the hip extensors. Janda's description of the LCS was essentially a snapshot of posture, where an observed tightness anteriorly plays a significant role posteriorly on hip function and activation. Isolating the hip extensor muscle may alter the muscle activation observed in postures diagnosed as LCS.

During the running stance phase, if an anterior pelvic tilt is increased, it will occur concurrently with a reduced hip extension[50]. This occurs as the thigh extends back behind the body during the push-off phase of the stride. Therefore it seems that the protocol chosen to test hip extensor strength, simulating the running stride, would measure hip strength correctly based on a population of runners. However, this protocol tests in an open kinetic chain movement. The stance phase in the running gait takes place in a closed kinetic chain and the gluteus maximus is only concentrically active from the initial contact to the mid-stance of the stance phase during running gait. The gluteus maximus does function in an open kinetic chain during the running gait. This activation occurs during the swing phase of the running gait. However this activation only occurs eccentrically as the hip extensors move into their maximum length required to decelerate the hip as the swing phase ends in preparation for initial heel contact. This study only measured hip extension strength concentrically and in an open chain position. There may be a mis-match in the methodology between the chosen hip extension strength protocol and the manner in which the body uses hip extensor muscles during the running gait. The relationship between hip flexor length and hip extensor strength failed to meet the hypothetical expectations set out in the objectives. Restructuring the methodology so that hip extensor strength is also eccentrically tested may provide useful information for functional performance.

Further research should also test hip extensor strength in both open kinetic chain and closed kinetic chain protocols. This would ensure the most comprehensive undertaking available to uncover whether different positions constitute better strength involvement or simply different muscle recruitment. Closed chain exercises have been

thought to recruit muscle in a more functional and familiar pattern and therefore differently to open chain exercises and movements[89]. The hip flexor length's true influence antagonistically may have to be tested in the context in which they function.

The data analysis undertaken by this study used Pearson's correlation for parametric data. Although correlational statistics demonstrate relationship strengths between two variables they however, cannot prove one variable causes a change in another[90]. This study is unable to show causation but only that a relationship exists.

The methodology could be modified to use a digital inclinometer instead of a manual goniometer. The goniometer has been validated specifically for the Modified Thomas test, however a digital inclinometer may offer more accurate results. A study concluded also that a static one joint hip flexion ROM measurement, measured using the Modified Thomas test, could not be directly correlated to the dynamic hip movement found in running[50].

The sample size might not have been large enough to have created an accurate reflection of the running population. The skewed male to female data, 21 males to 11 females, may under represent the females' runners in this study. The inclusion criteria only called for a minimum of running kilometres per week. Without limiting the maximum running kilometres per week within the inclusion criteria, the current training variation practised by this study's population, may be large. Building in categories of running experience and training goals could have resulted in better understanding of the profiles of those participating in this study.

Furthermore, reclassifying the inclusion criteria to include only runners with tight one- and two-joint hip flexors would better assess whether a deficit in hip flexor length facilitates hip extensor weakness. Even a large sample from each flexibility classification would assist in assessing the relationship between hip flexor length and hip extensor strength.

5.6 Recommendations for further research

The current study should be extended to include a larger population in order to include non-running participants. This could be compared to the current data created by this study. Generalising the population, not limiting the inclusion criteria to runners, may also create more relevant data in support of, or against Janda's habitual muscle activation pattern theories.

The limited citations found using the isokinetic hip extension protocol in this study also necessitate the validation of this protocol with a larger population. This protocol has been suggested to be more functional than the conventional isokinetic protocol as it simulates hip flexion/extension closely to walking or running[76].

Modifying the methodology to include both eccentric and concentric isokinetic results in order to understand the relative actions of hip extension during running gait also needs revisiting. The inclusion of both open and closed kinetic chain testing scenarios would cover all the necessary permutations of hip muscle activation found in the running gait.

Furthering research on female runners in light of the results reflected here and the new evidence presented by Willson et al., may provide new insights into gender differences in hip kinematics of the running gait[87].

Future research could include using an EMG to test muscle activation for the variables of this study as well as the inclusion of other muscles like the hamstrings and transversus abdominus as part of a new correlation based on muscle activation and recruitment rather than absolute and relative strength.

5.7 Clinical Applications

This study does not support Janda's theory of LCS when considering the methodology used and the limited sample size based on specific inclusion criteria. However the female results have uncovered a unique scenario that may add to growing literature dedicated to understanding the distinctive hip strategy employed by females in their running gait. This is an observable approach to a running gait that might enhance the clinician's ability to interpret the relative changes in a female's running gait.

The hip extensor strength protocol set out by Boling et al[76]., was reproduced by this study. However taking into account the limitations reported by the previous author, this study used a larger, more natural range of motion. This method of hip extension testing should be employed routinely as the protocol of choice for isokinetic hip extensor strength testing. Clinicians should be satisfied with the results obtained in this study as it reproduced data based on the original protocol successfully[76].

The results set out by this study also provide clinicians with an insight into the gender differences when assessing running injuries. For example the potential for a tighter rectus femoris relationship with long and inhibited iliopsoas in female runners, may prove to be diagnostically valid.

5.8 Conclusion

The purpose of this study was to validate one component of a postural rule of thumb as observed by Janda: hip flexor length as a predictor of hip extensor strength. This research report failed to demonstrate a strong positive relationship between one joint hip flexor ROM and hip extensor strength. This relationship is fundamental to the postural changes that have been observed and described for at least three decades. This influence of tightness and over activity coupled with antagonistic weakness and inhibition may underpin Janda's LCS. Janda's tight 'tonic' muscles included the hip flexors such as the iliopsoas and the rectus femoris, while his weak and inhibited 'phasic' muscles included the hip extensors, the gluteus maximus and the gluteus medius. However isolating a portion of the LCS and testing its validity based only on strength and length changes may omit less obvious variations. These include altered neuromuscular control through a protracted adaptation to environmental demands that may not produce the desired linear progression in strength and length changes that this research report hypothesised.

This study, having generated data which does not support the LCS as observed by Janda, has uncovered variability in male versus female hip function in their running gait. In addition to this it is also revealed a strong negative relationship between two-

joint hip flexion ROM and hip extensor strength. This significant correlation was only found in the female population. No correlation, or only as much as a small correlation at best, was reported on for their male counterparts. The lack of correlation also extended to the total population as a whole.

This study is limited by its testing protocol. Muscle activation and isokinetic strength testing needs to be tested and applied to the appropriate phase of the running gait. Future research should comprise both concentric as well as eccentric isokinetic strength testing. A closed chain form of muscle strength testing should also be contained as part of a comprehensive form of data collecting specific to runners' actioning hips.

Although this research report failed to establish a relationship between hip flexor length and hip extensor strength, it has added much value in hip mechanics of runners. Hip flexor length values were provided, using a protocol by Harvey et al., and in addition, hip strength profiles were produced for a uniquely functional isokinetic testing position. This data is useful for further research as well as clinically important.

Reference List

1) Torre D. The effectiveness of chiropractic manipulation in conjunction with a rehabilitation exercise program in the management of chronic lower back pain in patients exhibiting pelvic crossed syndrome [dissertation]. Johannesburg: University of Johannesburg; 2003.

2) Gray J. Are measurements of hip extension and anterior pelvic tilt taken from static photographs during a constrained forward lunge test valid and reliable in healthy adult runners? [dissertation]. Nova Scotia: Dalhousie University; 2011.

3) Page P, Frank C, Lardner R. Assessment and treatment of muscle imbalance: The Janda Approach United States: Human kinetics; 2010.

4) Bullock-Saxton J, Murphy D, Norris C, Richardson C, Tunnell P. The muscle designation debate: the experts respond; Journal of Bodywork and Movement Therapies. 2000; 4(4): 225-227.

5) Bullock-Saxton JE, Janda V, Bullock MI. Reflex activation of gluteal muscles in walking. Spine 1993; 18(6):704–708.

6) Bullock-Saxton JE, Janda V, Bullock MI. The influence of ankle sprain injury on muscle activation during hip extension. International Journal of Sports Medicine. 1994; 15(6):330–334.

7) Moore KL. Clinically oriented anatomy. 3rd ed. Williams and Wilkins; 1992.

8) Gyftopoulos S, Rosenburg Z, Schweitzer M, Bordalo-Rodrigues M. Normal Anatomy and strains of the deep musculotendinous junction of the proximal rectus femoris: MRI features. American Journal of Roentgenology. 2008; 190:182-186.

9) Andrikoula S, Tokis A, Vasiliadis H, Georgoulis A. The extensor mechanism of the knee joint: an anatomical study. Knee Surgery, Sports Traumatology, Arthroscopy. 2006; 14(3): 214-220.

10) Franz J, Paylo K, Dicharry J, Riley P, Kerrigan D. Changes in the coordination of hip and pelvis kinematics with mode of locomotion; Gait and Posture. 2009; 29: 494-498.

11) Sajko S, Stuber K. Psoas Major: a case report and review of its anatomy, biomechanics, and clinical implications. Journal of Canadian Chiropractic Association. 2009; 53(4):311-318.

12) Bogduk N, Pearcy M, Hadfield G. Anatomy and biomechanics of psoas major. Clinical Biomechanics. 1992; 7:109–119.

13) Brolinson P, Rogers M. Functional and kinetic evaluation of the hip and pelvis. In: Seidenberg P, Bowen J, editors. The Hip and pelvis in Sports Medicine and Primary Care. Springer Science. 2010; p. 37-70.

14) Novacheck T, Trost J, Schwartz M. Intramuscular psoas lengthening improves dynamic hip function in children with cerebral palsy. Journal of Pediatric Orthopaedics. 2002; 22 (2): 158-174.

15) Ilizaliturri Jr VM, Villalobos Jr FE, Chaidez PA, Valero FS, Aguilera JM. Internal snapping hip syndrome: treatment by endoscopic release of the iliopsoas tendon. Arthroscopy: The Journal of Arthroscopic and Related Surgery. 2005; 21(11): 1375-1380.

16) Laible C, Swanson D, Garofolo G, Rose D. Iliopsoas Syndrome in Dancers. Orthopaedic Journal of Sports Medicine. 2013; 1(3).

17) Lewis, C. Extra-articular snapping hip: a literature review. Sports Health. 2010; *2* (3*): 186-190.*

18) Piva SR, Goodnite EA, Childs JD. Strength around the hip and flexibility of soft tissues in individuals with and without patellofemoral pain syndrome. Journal of Orthopaedic Sports Physical Therapy. 2005; 35(12): 793-801.

19) Waryasz G, McDermott A. Patellofemoral pain syndrome (PFPS): a systematic review of anatomy and potential risk factors. Dynamic medicine. 2008; 7(1): 9.

20) Prins M, Van Der Wurff P. Females with patellofemoral pain syndrome have weak hip muscles: a systematic review. Australian Journal of Physiotherapy. 2009; 55: 9-15.

21) Souza R. Powers C. Predictors of hip internal rotation during running: An Evaluation of hip strength and femoral structure in women with and without Patellofemoral pain. The American Journal of Sports Medicine. 2009; 37(3): 579-587.

22) Powers, CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. Journal of Orthopaedic and Sports Physical Therapy. 2003; 33: 639-646.

23) Noehren B, Pohl M, Sanchez Z, Cunningham T, Lattermann C. Proximal and distal kinematics in female runners with patellofemoral pain. Clinical Biomechanics. 2012; 27(4): 366-371.

24) Chumanov E, Wille C, Michalski M, Heiderscheit B. Changes in muscle activation patterns when running step rate is increased. Gait and Posture. 2012; 36(2): 231-235.

25) Ward S, Winters T, Blemker S. The architectural design of the gluteal muscle group: implications for movement and rehabilitation. The Journal of Orthopaedic and Sports Physical Therapy. 2010; 40(2): 95-102.

26) Neumann, D. Kinesiology of the hip: a focus on muscular action. The Journal of Orthopaedic and Sports Physical Therapy. (2010); 40(2): 82-94.

27) Wagner T, Behnia N, Ancheta W Lau, Shen R, Farrokhi S, Powers C. Strengthening and neuromuscular re-education of the gluteus maximus in a triathlete with exercise-associated cramping of the hamstrings. The Journal of Orthopaedic and Sports Physical Therapy 2010; 40(2): 112-119.

28) Wilson J, Ferris E, Heckler, A, Maitland L, Taylor C A. Structured review of the role of gluteus maximus in rehabilitation. New Zealand Journal of Physiotherapy. 2005; 33(3): 95-100.

29) Vohra S, Arnold G, Doshi S, Marcantonio D. Normal MR imaging Anatomy of the thigh and Leg. Magnetic Resonance Imaging Clinics North America. 2011; 19: 621-636.

30) Novachek T. The biomechanics of running. Gait and Posture. 1998; 7: 77-95.

31) Flack N, Nicholson H. Woodley S: A review of the anatomy of the hip abductor muscles, gluteus medius, gluteus minimus, and tensor fascia lata. Clinical Anatomy. 2012; 25(6): 697–708.

32) Wiemann K, Tidow G. Relative activity of hip and knee extensors in sprinting-implications for training. New Studies in Athletics. 1995; 10: 23-29.

33) Thelen DG, Chumanov ES, Hoerth DM, Best TM, Swanson SC, Young M, Heiderscheit BC. Hamstring muscle kinematics during treadmill sprinting. Medicine and Science in Sports and Exercise. 2005; 3(1):108-114.

34) Yu B, Queen RM, Abbey AN, Liu Y, Moorman CT, Garrett WE. Hamstring muscle kinematics and activation during overground sprinting. Journal of Biomechanics. 2008; 41(15):3121-3126.

35) Sole G, Milosavljevic S, Nicholson H, Sullivan SJ. Altered muscle activation following hamstring injuries. British Journal of Sports Medicine. 2012; 46(2):118-23.

36) Schache A, Blanch P, Murphy T. A relation of anterior pelvic tilt during running to clinical and kinematic measures of hip extension. British Journal of Sports Medicine. 2000; 34:279-283.

37) Kendall FP, McCreary EK, Provance PG. Muscles: testing and function with posture and pain. 5th ed. Baltimore, MD 21201, Lippincott Williams & Wilkins; 2005.

38) Key, J. The Pelvic Crossed Syndromes: A reflection of imbalanced function in the myofascial envelope: A further exploration of Janda's work. Journal of Bodywork and Movement Therapies. 2010; 14(3): 299-301.

39) Morris C, Greenman P, Bullock M, Basmajian J, Kobesova A. Vladimir Janda, MD, DSc tribute to a master of rehabilitation. Spine. 2006; 31:1060–1064.

40) Ohtsuki K, Suzuki TA. Comparison of the immediate changes in subjects with chronic lower back pain effected by lower back pain exercises and direct stretching of the tensor fascia latae, the hamstring and the adductor magnus. Journal of Physical Therapy Science. 2012; 24: 97-100.

41) Takasaki H, Lizawa T, Hall T, Nakamura T, Kaneko S. The influence of increasing sacroiliac joint force closure on hip and lumbar spine extensor muscle firing pattern. Manual Therapy. 2009; 14: 484-489.

42) Wingerden J, Vleeming A, Buyruk H, Raissadat K. Stabilisation of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. European Spine Journal. 2004; 13(3): 199-205.

43) Richardson C, Snijders C, Hides J, Damen L, Pas Martijn Strom J. The relation between the transversus abdominus muscles, sacroiliac joint mechanics, and low back pain. Spine. 2002; 27(4): 399-405.

44) Chance-Larsen K, Littlewood CG. A prone hip extension with lower abdominal hollowing improves the relative timing of gluteus maximus activation in relation to biceps femoris. Manual Therapy. 2010; 15(1): 61-65.

45) Sneijders C, Ribbers M, Bakker H, Stoeckart R, Stam H. EMG recordings of abdominal and back muscles in various standing postures: validation of a biomechanical model for sacroiliac joint stability. Journal of Electromyography and Kinesiology. 1998; 8 (4): 205-214.

46) Winters M, Blake C, Trost J, Marcelle-Brinker T, Lowe L, Garber M, Wainner R. Passive versus active stretching of the hip flexor muscles in subjects with limited hip extension: a randomised control trial. Physical Therapy. 2004; 84: 800-807.

47) Lee L, Zavarei K, Evans J, Lelas J, Riley P, Kerrigan C. Reduced hip extension in the elderly: dynamic or postural? Archives of Physical Medicine and Rehabilitation. 2005; 86: 1851-1854.

48) Lee L, Kerrigan D, Casey D, Croce U Dynamic. Implications of hip flexion contractures; American journal of physical Medicine and Rehabilitation 1997; 76(6):502-508.

49) Franz J, Paylo K, Dicharry J, Riley P, Kerrigan D. Changes in the coordination of hip and pelvis kinematics with mode of locomotion. Gait and Posture. 2009; 29: 494-498.

50) Schache A, Blanch P, Murphy T. A relation of anterior pelvic tilt during running to clinical and kinematic measures of hip extension. British Journal of Sports Medicine. 2000; 34:279-283.

51) Niemuth P, Johnson R, Myers M, Thieman T. Hip muscle weakness and overuse injuries in recreational runners. Clinical Journal of Sport Medicine. 2005; 15(1):14-21.

52) Brown L. Isokinetics in human performance 3rd ed. Human Kinetics; 2000.

53) Baltzopoulos V, Brodie D. A. Isokinetic dynamometry. Sports Medicine. 1989; 8(2): 101-116.

54) Cahalan TD, Johnson ME, Liu S, Chao EYS. Quantitative measurements of hip strength in different age groups. Clinical Orthopaedics and Related Research. 1989; 246: 136-145.

55) Burnett CN, Betts EF, King WM. Reliability of isokinetic measurements of hip muscle torque in young boys. Physical Therapy. 1990; 70(4): 244-249.

56) Drouin J, Tamara A, Valovich-mcLeod C, Shultz S, Gansneder B, Perrin D. Reliability and validity of the Biodex system 3 pro isokinetic dynamometer velocity, torque and position measurements. European Journal of Applied Physiology. 2004; 91: 22–29.

57) Almosnino S, Stevenson J, Bardana D, Diaconescu, Dvir Z. Reproducibility of isokinetic knee eccentric and concentric strength indices in asymptomatic young adults. Physical Therapy in Sport. 2011; 1-7.

58) Emery CA, Maitland ME, Meeuwisse WH. Test-retest reliability of isokinetic hip adductor and flexor muscle strength. Clinical Journal of Sport Medicine. 1999; 9(2): 79-85.

59) Bottaro M, Russo A, Oliveira RJ. The effects of rest interval on quadriceps torque during an isokinetic testing protocol in the elderly. Journal of Sports Science Medicine. 2005; 4(3): 285-90.

60) Campenella B, Mattacola CG, Kimura IF. Effect of visual feedback and verbal encouragement on concentric quadriceps and hamstrings peak torque of males and females. Isokinetics and Exercise Science. 2000; 8(1):1-6.

61) Van Heerden, Z. An assessment of performance testing in middle distance running. [Dissertation]; Johannesburg: University Of The Witwatersrand; 2005.

62) Corkery M, Briscoe H, Ciccone N, Foglia G, Johnson P, Kinsman S, Legere L Lum, Canavan B. Establishing normal values for lower extremity muscle length in college-age students. Physical Therapy in Sport. 2007; 8(2): 66-74.

63) Gabbe BJ, Bennell KL, Wajswelner H, Finch CF. Reliability of common lower extremity musculoskeletal screening tests. Physical Therapy in Sport. 2004; 5(2): 90-97.

64) Schache AG, Blanch PD, Rath DA, Wrigley TV, Bennell KL. Are anthropometric and kinematic parameters of the lumbo-pelvic-hip complex related to running injuries? Research in Sports Medicine. 2005; 13(2): 127-147.

65) Young W, Clothier P, Otago L, Bruce L, Liddell D. Acute effects of static stretching on hip flexor and quadriceps flexibility, range of motion and foot speed in kicking a football. Journal of Science and Medicine in Sport. 2004; 7(1): 23-31.

66) Seidenberg PH, McFadden DP, Physical examination of the hip and Pelvis, In: Seidenberg P, Bowen J, editors. The Hip and pelvis in Sports Medicine and Primary Care. Springer Science. 2010; p. 9-36.

67) Harvey D. Assessment of the flexibility of elite athletes using the Modified Thomas test. British Journal of Sports Medicine. 1998; 32:68-70.

68) Clapis P, Davis M, Davis R. Reliability of inclinometer and goniometer measurements of hip extension flexibility using the Modified Thomas test. Physiotherapy Theory and Practice. 2008; 24(2): 135-141.

69) Peeler, J, Anderson, J. Reliability limits of the modified Thomas test for assessing rectus femoris muscle flexibility about the knee joint. Journal of Athletic Training. 2008; 43(5):470-476.

70) Ferber R, Kendall K, McElroy L. Normative and critical criteria for iliotibial band and iliopsoas muscle flexibility. Journal of Athletic Training. 2010; 45(4): 344-348.

71) Finnoff J, Hall M, Kyle K, Krause D, Lai J, Smith J. Hip strength and knee pain on high school runners: a prospective study. The American Academy of Physical Medicine and Rehabilitation. 2011; 3(9):792-801.

72) Mikkola J, Vesterinen V, Taipale R, Capostagno B, Hakkinen K, Nummela A. Effect of resistance training regimens on treadmill running and neuromuscular performance in recreational runners. Journal of Sports Science. 2011; 29(13): 1359-1371.

73) Burnett D, Smith K, Smeltzer C, Young K, Burns S. Perceived muscle soreness in recreational female runners. International Journal of Exercise Science. 2010; 3(3):108-116.

74) Dierks T, Davis I, Hamill J. The effects of running in an exerted state on lower extremity kinematics and joint timing. Journal of Biomechanics. 2010; 43: 2993-2998.

75) Morin J, Samozino P, Zameziati K, Belli A. Effects of altered stride frequency and contact time on leg spring behaviour in human running. Journal of Biomechanics. 2007; 40: 3341-3348

76) Boling M, Padua D, Creighton R. Concentric and eccentric torque of the hip musculature in individuals with and without patellofemoral pain. Journal of Athletic Training. 2009; 4(1):7-13.

77) Reeves J, Hume P, Wenham M. Reliability of passive lower limb flexibility in rugby sevens players measured with a new hand held dynamometer: a pilot study. In Isbs-Conference Proceedings Archive. 2012; 1(1)

78) Barr A, Duncan, P. Influence of position on knee flexor peak torque. The Journal of Orthopaedic and Sports Physical Therapy. 1988; 9(8): 279-284.

79) Worrell TW, Perrin DH, Denegar CR. The influence of hip position on quadriceps and hamstring peak torque and reciprocal muscle group ratio values. Journal of Orthopaedic Sports Physical Therapy. 1989; 11(3): 104-107.

80) Thorpe J, Ebersole T. Unilateral balance performance in female collegiate soccer athletes. The Journal of Strength and Conditioning Research. 2008; 22(5): 1429-1433.

81) Tis L, Perrin D, Snead D, Weltman A. Isokinetic strength of the trunk and hip in female runners. Isokinetics and Exercise Science. 1991; 1: 22-25.

82) Sugiura Y, Saito T, Sakuraba K, Sakuma K, Suzuki E. Strength deficits identified with concentric action of the hip extensors and eccentric action of the hamstrings predispose to hamstring injury in elite sprinters. The Journal of Orthopaedic and Sports Physical Therapy. 2008; 38(8): 457-464.

83) Bazett-Jones D, Cobb S, Joshi MN, Cahin SE, Earl JE. Normalising muscle hip strength: establishing body-size independent measurements. Archive of Physical Medicine and Rehabilitation. 2011; 92:76-82.

84) Robinson RL, Nee RJ. Analysis of hip strength in females seeking physical therapy treatment for unilateral patellofemoral pain syndrome. Journal of Orthopaedic Sports Physical Therapy. 2007; 37:232-238.

85) Gage J. The acute effects of proprioceptive neuromuscular facilitation (PNF) stretching on selected performance parameters [dissertation]. Chapel Hill: University of North Carolina; 2011.

86) Nourbakhsh MR, Arabloo AM, Salavati M. The relationship between pelvic cross syndrome and chronic low back pain. Journal of Back and Musculoskeletal Rehabilitation. 2006; 19(4): 119-128.

87) Willson J, Petrowitz I, Butler R, Kernozek T. Male and female gluteal muscle activity and lower extremity kinematics during running. Clinical Biomechanics. 2012; article in press.

88) Sahrmann SA. Diagnosis and treatment of movement impairment syndromes. St. Louis: Mosby; 2002.

89) Karandikar N, Vargas O. Kinetic chains: a review of the concept and its clinical applications. Physical Medicine and Rehabilitation. 2011; 3(8): 739-746.

90) Zou KH, Yuncali K, Silverman S. Correlation and simple linear regression. Radiology. 2003; 227(3):617-622.

91) Wilson JM, Lyndsey MH, Jeong-Su K, Ugrinowitsch C, Sang-Rok L, Zourdos M, Sommer B, Panton L. Effects of static stretching on energy cost and running endurance performance. The Journal of Strength & Conditioning Research. 2010; 24 (9): 2274-2279.

92) Distefano LJ, Blackburn JT, Marshall SW, Padua, DA. Gluteal muscle activation during common therapeutic exercises. The Journal of Orthopaedic and Sports Physical Therapy. 2009; 39(7): 532-540.

Appendices

Appendix A

The proximal attachment, distal attachment and the innervation of the iliopsoas[7]

Proximal attachment of Psoas Major	The anterior and lower borders of the transverse processes of T12-L5 and the intervertebral discs between them.
Proximal attachment of the Iliacus	The iliac fossa, iliac crest, ala of sacrum and anterior sacroiliac ligaments
Distal attachment	The anterior aspect of the femur onto the lesser trochanter of the femur via a conjoined tendon
Innervation	Psoas major: lumber plexus (L1,L2 andL3) Iliacus: femoral nerve (L2 and L3)

The proximal attachment, distal attachment and the innervation of the psoas minor[7]

Proximal attachment	Laterally to the T12 and L1 vertebrae and intervening intervertebral disc.
Distal attachment	Iliopubic eminence on the pelvic brim.
Innervation	Ventral ramus of L1 nerve.

The proximal attachment, distal attachment and the innervation of the Rectus Femoris[7]

Proximal attachment	Anterior inferior iliac spine and groove superior to the acetabulum.
Distal attachment	Via quadriceps tendon into the patella and via patella tendon into the tibial tuberosity.
Innervation	Femoral nerve (L2,L3 and L4)

The proximal attachment, distal attachment and the innervation of the Pectineus[7]

Proximal attachment	Pectineal line of the pubis
Distal attachment	Pectineal line of the femur
Innervation	Femoral nerve (L2 and L3), may also receive a branch from the obturator nerve.

The proximal attachment, distal attachment and the innervation of the sartorius[7]

Proximal attachment	The anterior-superior iliac spine.
Distal attachment	The superior paret of the medial surface of the tibia.
Innervation	Femoral nerve (L2 and L3).

The proximal attachment, distal attachment and the innervation of the gracilis[7]

Proximal attachment	The body and inferior ramus of pubis
Distal attachment	The superior part of the medial surface of the tibia.
Innervation	Anterior branch of obturator nerve (L2 and L3)

The proximal attachment, distal attachment and the innervation of the adductor longus[7]

Proximal attachment	Body of Pubis, inferior to pubic crest
Distal attachment	Middle third of linea aspera of femur.
Innervation	Anterior branch of Obturator nerve (L2,L3 and L4)

The proximal attachment, distal attachment and the innervation of the tensor fascia lata[7]

Proximal attachment	The anterior superior iliac spine and the anterior part of the external lip of the iliac crest.
Distal attachment	The iliotibial tract that attaches to lateral condyle of the tibia.
Innervation	Superior gluteal nerve (L4 and L5).

Appendix B

The origin and the insertion of gluteus maximus[7]

Origin attachment	Posterior gluteal ilial line, the sacrospinalis tendon, the posterior surface of the sacrum and coccyx and the sacrotuberous ligament, the superficial laminae of the posterior thoracolumbar fascia and the fascia covering the gluteus medius. In the pelvis the gluteus maximus blends with the ipsilateral multifidus through the raphe of the thoracolumbar fascia and the contralateral latissimus dorsi through the superficial laminae of the thoracolumbar fascia
Distal attachment	Gluteal tuberosity of the femur, and the iliotibial tract level of the fascia lata which inserts into the lateral condyle of tibia.
Innervation	Inferior gluteal nerve (L5,S1 and S2)

The proximal attachment, distal attachment and the innervation of the gluteus medius[7]

Proximal attachment	External surface of the ilium between the anterior and posterior gluteal lines.
Distal attachment	The lateral surface of the greater trochanter of the femur
Innervation	Superior gluteal nerve (L5, S1)

The origin and insertion of the hamstring group of muscles[7]

Origin attachment	Semitendonosus, Semimembranosus and long head of biceps femoris all attach via common tendon to the posterior part of the ischial tuberosity. Short head of biceps femoris attaches to the lateral lip of the linea aspera and the lateral supracondylar line of the shaft of the femur.
Distal attachment	The semitendinonsus attaches to the posteromedial aspect of the medial condyle of the tibia. The medial aponeurosis of the semimembranosus attaches to the posteromedial surface of the medial condyle of the tibia just below the joint capsule. Laterally the long head of biceps femoris attaches, along with the short head, to the lateral aspect of the head of the fibula.
Innervation	Semimembranosus: tibial division of the sciatic nerve (L5,S1 and S2). Semitendinosus: tibial division of the sciatic nerve (L5,S1 and S2). Long head of Biceps Femoris: tibial division of the sciatic nerve (L5,S1 and S2). Short head of Biceps Femoris: common fibular division of the sciatic nerve (L5,S1 and S2).

The proximal attachment, distal attachment and the innervation of the adductor magnus[7]

Proximal attachment	Adductor part: inferior ramus of pubis, ramus of ischium. Hamstring part: the ischial tuberosity anteroinferiorly.
Distal attachment	Adductor part: along the medial length of the linea aspera, the gluteal tuberosity, and medial supracondylar line of the shaft of the femur. Hamstring part: adductor tubercle of the femur.
Innervation	Adductor part: posterior branch of obturator nerve (L2, L3 and L4). Hamstring part: tibial portion of the sciatic nerve (L4)

Appendix C

Data Sheet

Study No: _____

Date:_____

Age (years): _____

Sex: M / F Running Club:_____

Running duration (hours of running per week):_____

Participation in any other sports:_____

Frequency and duration of participation in sports stated above (weekly):_____

Current Medication history: _____

_____

Weight (kg):	
Height (m):	
Dominant Leg	

Past injuries Yes / No Date of injury: _____

Please specify_____

_____

_____

Current injuries Yes / No

Please specify_____

_____

_____

Injuries specific to hip Yes / No

Please specify_____
_____
_____

Injuries specific to knee Yes / No

Please specify_____
_____
_____

Appendix D

<u>Testing Data Sheet</u>:

Isokinetic Testing Protocol and the Modified Thomas Test

Biodex System 3	
TEST Protocol	Max Voluntary Contraction
Contraction Cycle	con/con
Speed/s	60^0/second
Trial Repetitions	8 with no effort
	3 (50%, 75%, 90% effort)
Test Repetitions	5 (maximum effort)
Sets	2
Rest between sets	2 min
GET	Yes

Biodex Dynamometer Reading:	Hip Extension Left Hip	Hip Extension Right Hip
Peak Torque (Nm)		
Average Peak Torque (Nm)		
Total Work (Joules)		

Modified Thomas Test Goniometer Readings						
	Reading 1 (Degrees)		Reading 2 (Degrees)		Reading 3 (Degrees)	
	Left	Right	Left	Right	Left	Right
One-joint hip flexion ROM						
Two-joint hip flexion ROM						

Appendix E

Research participants Information Sheet

Research topic: Hip Flexor length as a predictor of Hip Extensor weakness in recreational runners

Introduction

Hello my name is Gerard Correia, I am physiotherapist working in private practice.

You are invited to participate in a research study as part of a master's research report in the field of exercise and sport science. The purpose of this study is to evaluate the relationship between hip flexor muscle (hip bending muscles) and hip extensor (hip straightening muscles) muscles. This study aims to determine whether tightness of your hip flexor muscles can predict the weakness of the hip extensor muscles. Determining this relationship can better improve our understanding of optimal running efficiency.

What procedures are involved?

As an invited participant you will be asked to perform two tests. The first test is the Thomas test which measures the length of your hip flexor muscle located at the top of your thigh muscle. Both hip flexors will be measured. As part of this test your height and weight will also be measured. The second test, measures hip extensor strength, which will be conducted using a Biodex 3 system isokinetic dynamometer. The machine will measure your hip extensor strength, these are the muscles located around your buttocks and back of your thigh area.

How long will the testing take

The testing procedure will be completed in no more than thirty minutes; this includes the Thomas test for both hip flexors and testing both hip extensors using the Biodex 3 system isokinetic dynamometer.

Why should I participate?

Your participation will help further research in the field of exercise and sport science.

Do I have to participate?

Your participation is completely voluntary. We require your consent to participate and you may withdraw from the study at any time.

Are there any possible risks involved?

The muscle strength and length testing protocols are safe procedure and will not leave the participants with any lasting effects or cause any muscle or joint damage. Short term effects may include muscle fatigue, or muscle stiffness.

Are there any possible benefits from participating?

This study will provide personal information regarding the hip length of your hip flexors and the strength of your hip extensor muscles. There will be recommendations for participants for corrective changes if tests reveal tight and overactive hip flexor muscles or weak and inhibited hip extensor muscles. It will also provide you with the opportunity to contribute to research in the field of exercise and sport science.

What about confidentiality

All information provided by yourself will remain confidential. Access of this information is limited to the tester and supervisor.

Who is doing the research?

I, Gerard Correia, enrolled in Masters of sport Science programme at the University of the Witwatersrand in Johannesburg, will be administering all aspects of the testing procedure.

Appendix F

INFORMED CONSENT

Part A - Your details

Name:_____

Part B - Explanation of the study

The purpose of the study is to assess both the hip flexor length as well as the hip extensor strength of the hip on the same side. All participants will receive an adequate explanation on the method and outcomes of the study before the testing procedure begin. The Assessment will only involve measuring hip flexor length and the hip extensor strength of the same side, both hips will be assessed. Assessment tools used are both valid and reliable and relevant to the study undertaken.

The qualified allied health professional with sufficient experience in operating the assessment tools will be used to gather data.

The muscle strength and length testing protocols are safe procedure and will not leave the participants with any lasting effects or cause any muscle or joint damage. Short term effects may include muscle fatigue, or muscle stiffness.

Your personal information will remain confidential and anonymous. It will not be reported on, on an individual basis, but rather form part of a bigger sample group.

- **Enquiries**

  Questions regarding the procedures used in this study are encouraged, and can be addressed to Gerard Correia, by email at gerardcorreia@gmail.com.

- **Freedom of consent**

Your permission to participate in this study is strictly voluntary. You are free to deny consent, or withdraw from the study at any time.

I, _____ have been fully informed by Gerard Correia on the testing procedures involved and the outcomes of the trial. I agree to participate in this trial having fully understood the merits and what is required of me as an invited participant of the trial.

This trial has been approved by the University of Witwatersrand Human Research Ethics Committee (HREC). If you have any questions or queries on please contact the HREC chairmen.

Contact Details of the HREC Chair's:

Prof Cleaton Jones 011 717 2301
(Monday-Wednesday-Friday mornings)
Prof Feldman 011 488 3840
Prof Woodiwiss 011 717 2153
Prof Dhai 011 717 2718

Signed: | |

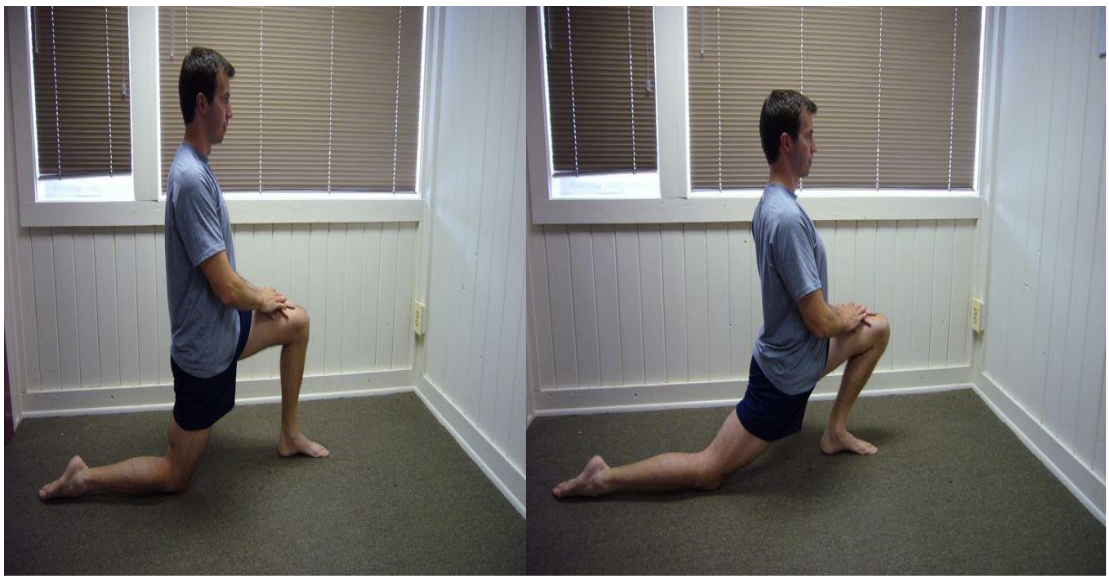
Date: | |

Appendix G

Hip Flexor Stretches and Hip Extensor Strengthening Exercises

Hip Flexor Stretches[91]

1. Start from a kneeling lunge
2. Tighten your gluteal muscles as you bend your front knee and stretch the hip flexors in your back leg
3. Make sure to keep your torso upright (don't lean forward!)
4. You should feel a stretch in the front of your hip, often this stretch continues into your abdomen .
5. Hold the stretch for 30 seconds and repeat 3 times with each leg .



Quadriceps[91]

1. Start from a kneeling lunge.
2. Bend back knee and grasp foot with the same-side hand.

3. Bend one knee fully and grasp the ankle with your free hand

4. Contracting the hamstrings and glutes, pull your leg back until you feel a good stretch in the front of your thigh.

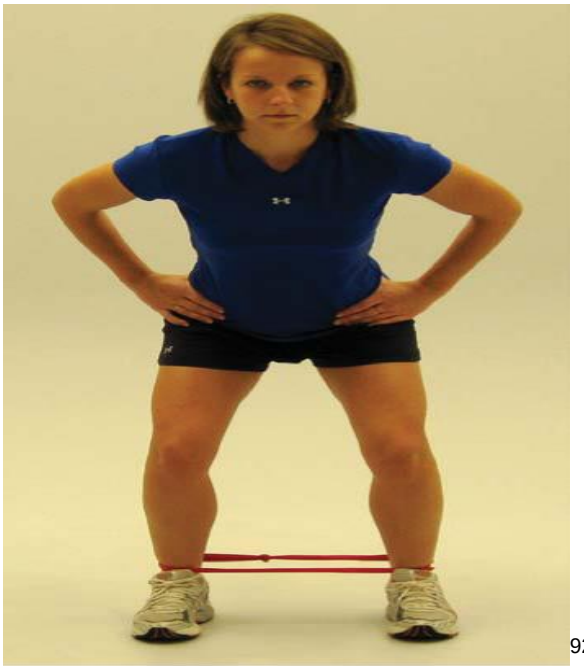
5. Be sure not to arch your back. Hold the stretch for30 seconds before releasing the stretch and repeat 3 times with each leg.


91

Hip Extensor/Abductor Exercises

Lateral Band Walks [92]

1. An elastic theraband is tied around your ankles.
2. Maintain your knees and hips in 30° of flexion.
3. Hands on your hips and began with your feet together.
4. Next, sidestep, leading with your dominant foot for a distance of just more than your shoulder width then let your non dominant foot slowly move back towards your dominant foot back to the starting position.
5. Keep their toes pointed straight ahead and Keep knees inline and behind your third toe.
6. Repeat 5 times in one direction then start the exercise in the opposite direction for 5 repetitions.
7. Have a 1 minute break and repeat the whole sequence 2 more times



92

Single-Limb Squat[92]

1. Balance on one leg with knee and hip flexed approximately 30° and hands on hips.
2. Slowly lower yourself toward the ground, using their ankle, knee, and hip joints, get your opposite middle finger to the outside of your foot without reaching with your shoulder.
3. Keep knees inline and behind your third toe.
4. Return to the starting position.
5. Repeat 8 times, have a 1 minute break and repeat the whole sequence 2 more times.


92

Single-Limb Deadlift [92]

1. Balance on one leg with knee and hip flexed approximately 30° and hands on hips.
2. Slowly flexed their hip and trunk and lower yourself toward the ground, using your ankle, knee, and hip joints, get your opposite middle finger to the outside of your foot without reaching with your shoulder.
3. Keep knees flexed at 30° while your hip and torso flexes forward.
4. Keep knees inline and behind your third toe throughout the exercise.
5. Return to the starting position.
6. Repeat 8 times, have a 1 minute break and repeat the whole sequence 2 more times.



92

Appendix H:

Ethics Clearance

UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG
Division of the Deputy Registrar (Research)

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)
R14/49 Mr Gerard Jose Correia

CLEARANCE CERTIFICATE	**M120132**
PROJECT	Hip Flexor Length as a Predictor of Hip Extensor Weakness in Recreational Runners
INVESTIGATORS	Mr Gerard Jose Correia.
DEPARTMENT	Centre for Exercise Science & Sport Medicine
DATE CONSIDERED	27/01/2012
DECISION OF THE COMMITTEE*	Approved unconditionally

Unless otherwise specified this ethical clearance is valid for 5 years and may be renewed upon application.

DATE 20/02/2012 **CHAIRPERSON** .....
(Professor PE Cleaton-Jones)

*Guidelines for written 'informed consent' attached where applicable
cc: Supervisor : Estelle Watson

DECLARATION OF INVESTIGATOR(S)

To be completed in duplicate and **ONE COPY** returned to the Secretary at Room 10004, 10th Floor, Senate House, University.
I/We fully understand the conditions under which I am/we are authorized to carry out the abovementioned research and I/we guarantee to ensure compliance with these conditions. Should any departure to be contemplated from the research procedure as approved I/we undertake to resubmit the protocol to the Committee. **I agree to a completion of a yearly progress report.**

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES...