The Effects of Dry Needling and Radial Extracorporeal Shockwave Therapy on the Sensitivity of Trigger Points in the Quadriceps and Jump Performance: a Randomised Control Trial

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List of Abbreviations

95% CI is 95% confidence intervals
95% LoA is 95% limit of agreement
α is Cronbach’s alpha reliability coefficient
A-δ-type fibres is thick myelinated afferent fibres
ALS is anatomical landmark system
ANOCVA is an analysis of covariance
ANOVA is an analysis of variance
C-type fibres is thick myelinated afferent fibres
CMJ is countermovement jump
CMJ-JH is jump height of countermovement jump
CV is coefficients of variance
ΔTrP is distance between TrPs
DJ is depth/drop jump
DJ-JH is jump height of depth jump
DN is dry needling
EMG is electromyography
ESWT is extracorporeal shockwave therapy
F-wave is the second electromyography nerve conduction velocity wave
fESWT is focused extracorporeal shockwave therapy
FP is force platforms
FU(n) is specific follow-up session
ICC(3,1) is mixed absolute intra-class coefficient
JH is jump height
JS is jump sign
ϰ is kappa coefficients
ℓn is natural logarithm
MDC is minimal detectable change
pH is power of hydrogen
PTD is photocell timing devices
PPT is pressure pain threshold
PR is pain recognition
rESWT is radial extracorporeal shockwave therapy
ρ is Pearson’s product moment correlation
RP is referred pain
RSI is reactive strength index
SD is standard deviation
SEM is standard error of the mean
t-test is paired sample t-test
TB is taut band
τf is flight time
TR is local twitch response
TrPs is myofascial trigger points
TrP-DN is trigger point dry needling
TS is tender spot
TX(n) is specific treatment session
UV is ultraviolet
VAS is visual analogue scale
VL is vastus lateralis
VL-TrP(n) is a specific trigger point location within the vastus lateralis
VM is vastus medialis
VM-TrP(n) is a specific trigger point location within the vastus medialis
WOMAC is Western Ontario McMaster Universities osteoarthritis index
X-line horizontal line of the anatomical landmark system
Y-line is vertical line of the anatomical landmark system
Abstract

Background: Trigger points (TrPs) can reduce strength. Thus, may reduce jump height. Dry needling (DN) is an effective treatment for TrPs but can cause post-needling soreness, potentially reducing jump performance. Radial extracorporeal shockwave therapy (rESWT) is an emerging treatment for TrPs that may not cause post-needling soreness. The post-needling soreness from DN may prevent an athlete training maximally. rESWT may be a possible alternative treatment for TrPs during periods of high-intensity training.

Methods: A mixed absolute intra-class coefficient (ICC(3:1)) was conducted to determine the reliability of the researcher at measuring the pressure pain threshold (PPT), anatomical landmark system (ALS) to locate TrPs. Bland-Altman plots were created to visualise the data. The ICC(3:1) of the countermovement jump (CMJ-JH), jump height of the depth jump (DJ-JH) and components of the DJ was established.

Twenty-one subjects with latent TrPs in the vastus lateralis (VL) and vastus medialis (VM) were treated with DN, rESWT or acted as a control. The outcome measures were the PPT, CMJ-JH and DJ-JH. After a Baseline session (B-line), there were three treatment sessions and two follow-up sessions (FU) at 3-4 days and seven days after the final treatment. A 2-way ANOVA was conducted to determine the effects of DN and rESWT on the PPT of TrPs, in the VL and VM. A 3-way ANOVA (factoring for PPT) was conducted to analyse the effects of DN and rESWT on CMJ-JH. A 2-way ANOVA (factoring for PPT and body mass index [BMI]) was carried out to analyse the effects of DN and rESWT on DJ-JH.

Results: The ICC(3:1) for the PPT ranged from 0.637 to 0.875. The ICC(3:3) for the ALS was 0.643 to 0.886 for the X-line and 0.603 to 0.745 for the Y-line. The ICC(3:1) for the CMJ-JH was 0.928 to 0.961. The ICC(3:1) for the DJ-JH was 0.919 to 0.956.

The mean PPT of the DN group increased from 28.25 N to 31.68 N between B-line and FU2. In the rESWT group, the mean PPT increased from 22.08 N to 24.07 N between B-line and FU2.

Results from a 2-way ANOVA reported a statistically significant interaction between group and time (p=0.003) for the PPT.

The mean CMJ-JH for the DN group increased from 27.43 cm at the Baseline to 28.07 cm between B-line and FU2; whereas, the rESWT group increased from 23.24 cm to 24.71 cm during the same period. Results from a 3-way ANOVA (factoring for the PPT) reported a statistically significant interaction between group and time for the CMJ-JH (p=0.007).

The mean DJ-JH for the DN group decreased from 22.71 cm to 20.86 cm between B-line and FU2. The rESWT group also decreased, from 21.38 cm at Baseline to 20.21 cm at Follow-up 2. A 3-way ANOVA (factoring for the PPT and BMI) reported a statistically significant interaction between group and time for the DJ-JH (p=0.001).

Conclusion: The investigator is reliable at measuring the PPT, ALS, CMJ-JH, DJ-JH. DN appears to have a positive effect on the mean PPT after one week once the post-needling soreness had subsided. rESWT shows a gradual improvement in PPT and CMJ-JH throughout the treatment phase and follow-up phase, which suggests that rESWT does not cause post-treatment soreness. Treating subjects with DN may not be the most appropriate action if they are engaging in competition or high-intensity concentric strength training in the following week. DN and rESWT can impede reactive strength for up to four days, possibly due to reducing the elastic property of the taut bands associated with TrPs. Further studies should include treating multiple TrPs in multiple muscles involved in triple extension and measuring muscle activation. Future research in this area should also consider using a longer post-treatment follow-up period.
Introduction

Myofascial pain syndrome is a condition that can cause pain at the site of the lesion, or it may refer to other structures; it may even alter movement patterns (Xiaoqiang et al., 2014). According to Bron and Dommerholt (2012), myofascial pain syndrome is believed to be caused by myofascial trigger points (TrPs). TrPs can be active where the pain is spontaneous, or latent TrPs where the TrP is only painful when palpated. The aetiology of TrPs is not entirely understood, but it is hypothesised that direct trauma (Castaldo et al., 2014) or repetitive overload (Dommerholt et al., 2011) causes a proliferation of inflammatory markers (Dommerholt et al., 2011) resulting in either peripheral sensitisation, as outlined by Simons (2004a); or central sensitisation, as noted by Dommerholt (2011). Both types of TrPs can alter joint kinematics (Shankar and Reddy, 2012), muscle activation patterns (Lucas et al., 2010; Ge et al., 2014; Sergienko and Kalichman, 2015) or reduced strength (Celik and Yeldan, 2011). The quadriceps are part of the kinetic chain which is pivotal for optimum jumping, running and changing direction at speed (Archer, 2016; Lorenz, 2016). The effects of TrPs in the quadriceps are under-investigated, but given that TrPs can reduce strength (Celik and Yeldan, 2011), it is possible that TrPs might affect an athlete’s ability to jump.

There are a plethora of treatment options for TrPs (Borg-Stein and Iaccarino, 2014; Shah et al., 2015). Dry needling (DN [p. 66]) uses filament acupuncture needles to depolarise the efferent signal associated with peripheral sensitisation and is a very effective treatment for TrPs (Fernández-de-Las-Peñas et al., 2010, 2011; Ghanbari et al., 2012; Berggreen et al., 2012; Karadaş et al., 2013; Espí-López et al., 2014a, 2014b; Taylor, 2014). However, DN is invasive and results in post-needling soreness (Martín-Pintado-Zugasti et al., 2014, 2015, 2016). Treating TrPs in the quadriceps could potentially improve jump performance in the long term. However, DN TrPs in the quadriceps may also cause post-needling soreness, and this may affect jump performance. Extracorporeal shockwave therapy (ESWT [p. 87]) uses mechanical pressure waves to deinervate the afferent signal associated with central sensitisation and is an emerging treatment for TrPs (Ji et al., 2012; Yoo et al., 2012). DN is believed to address the peripheral sensitisation of TrP formation (Dunning et al., 2014); whereas ESWT is thought to address the central sensitisation (Jeon et al., 2012). ESWT may de-innervate...
nociceptor fibres (Hausdorf et al., 2008a, 2008b). ESWT has been reported to be no less effective than acupuncture for the treatment of lateral epicondylitis (Wong et al., 2017).

A rater needs to be able to measure an outcome accurately and reliably to identify whether an intervention is effective (Kottner et al., 2011a, 2011b). The sensitivity of TrPs can be measured with the pressure pain threshold (PPT) using a device called an algometer (Jones et al., 2007). The location of TrPs can be measured using various methods such as the anatomical landmark system (Barbero et al., 2012). There are several jump tests used as measures of athletic performance (Ebben et al., 2008). According to Meylan et al. (2011) and Mizuguchi et al. (2015), the countermovement jump (CMJ [p. 47]) uses a slow stretch-shortening cycle; whereas the depth jump (DJ [p. 59]) uses a fast stretch-shortening cycle.

The aim of the project is to compare the effects of treating TrPs in the quadriceps with DN and rESWT on jump performance. This study is the first to measure the effects of treating TrPs on multiple jump performance outcomes. To the author’s knowledge, there are no other studies that investigate the effects of rESWT in jump performance. This study is also the first to investigate the effects of treating TrP to improve jump performance over a period of seven days.
2 Literature review

2.1 Myofascial pain syndrome

According to the literature (Gerwin et al., 2004; Bron and Dommerholt, 2012; Xiaoqiang et al., 2014), myofascial pain syndrome has been described as the sensory, motor, and autonomic symptoms caused by TrPs. It is suggested that anywhere from nine-million (Alvarez and Rockwell, 2016) to twenty-three-million (Ballyns et al., 2011) people in the United States suffer from myofascial pain syndrome; whereas, Henriksen et al. (2013) have stated, 20% of Europeans have reported frequent and persistent musculoskeletal pain. Myofascial pain syndrome represents the major cause of musculoskeletal pain in Europeans (Henriksen et al., 2013). The prevalence of myofascial pain syndrome among middle-aged adults (30–60 years) is reported to be 37% in men and 65% in women, respectively; and as high as 85% in mid sexagenarians (Giamberardino et al., 2011) with a greater prevalence in females has been reported in the literature (Vázquez-Delgado et al., 2009). Myofascial pain syndrome is particularly prevalent in patients with fibromyalgia (Casanueva et al., 2014). The lack of universally accepted diagnostic criteria for MPS has resulted in varied results in epidemiological studies (Giamberardino et al., 2011). Myofascial pain syndrome is caused by TrPs, and this should be considered when searching and reviewing the literature.

2.2 Trigger points

The term TrP was first described by Travell et al. (1942), the foremost authority on TrPs, but they have been reported as early as 1843 by Robert Froiep (Simons et al., 1999). TrPs are points of exquisite tenderness, or tender spot (TS), located within a series of hyper contracted sarcomeres of skeletal muscle known as a taut band (TB). They are tender when pressure is applied and can cause referred pain (RP) in distinct patterns (Bron and Dommerholt, 2012). Some other features of TrPs include jump sign (JS), a local twitch response (LTR) which may also be elicited (Ballyns et al., 2011), and autonomic phenomena such as sweating and calor (McPartland and Simons, 2011). There are two types of TrPs, active and latent; the pertinent difference between latent and active TrPs is that latent TrPs are only painful when palpated (Bron and Dommerholt, 2012). Latent TrPs can quickly be transformed into active TrPs. Both active and latent TrPs may
provoke muscle imbalance, abnormal motor recruitment, accelerated development of muscle fatigue, and weakness. Thus predisposing the tissue to be damaged further (Castaldo et al., 2014).

2.2.1 Epidemiology

The prevalence of TrPs has been reported to be 85% in patients visiting a tertiary pain clinic and as high as 95% in people with chronic pain disorders (Tough et al., 2007). TrP pain creates substantial stress to the patients and is a financial burden to society (Tough et al., 2007). TrPs were the primary source of pain in 30% to 85% of patients presenting in a primary care setting or pain clinics, yet TrPs are still underdiagnosed (Kalichman and Vulfsons, 2010). Approximately 85% of the population have had TrPs at some point during their lives (Staud, 2007).

2.2.2 Pathologies associated with trigger points

TrPs are considered a primary source of pain in patients with whiplash associated symptoms; but there is no consistent approach to the treatment of whiplash associated disorders (Tough et al., 2010). TrP are believed to be the primary causes of some orthopaedic conditions such as tension-type headaches (Fernández-de-Las-Peñas et al., 2010, 2011; Ghanbari et al., 2012; Berggreen et al., 2012; Karadaş et al., 2013; Espí-López et al., 2014a, 2014b; Taylor, 2014), temporomandibular disorder (Ariji et al., 2015; Asha et al., 2015; Christidis et al., 2015; Gonzalez-Perez et al., 2015; Quek et al., 2015; Al-Khotani et al., 2016; De Carli et al., 2016; Rai et al., 2016), non-specific lower back pain (Chen et al., 2016; Cui et al., 2016; Benjaboonyanupap et al., 2015; Koppenhaver et al., 2015; Lluch et al., 2015; Téllez-García et al., 2015; Trampas et al., 2015) and acute lower back pain (Takamoto et al., 2015). TrPs in the neck (Gerber et al., 2015; Sumen et al., 2015; Paolucci et al., 2016), thoracic region (Halski et al., 2015; Sumen et al., 2015; Wamontree et al., 2015; Buttagat et al., 2016; Öztürk et al., 2016), and shoulder muscles (Hains et al., 2010; Bron et al., 2011a, 2011b; Fernández-Lao et al., 2012; Gerber et al., 2015; Sergienko and Kalichman, 2015; Gordon et al., 2016) are reported frequently in the literature and should be considered as the primary source of pain following whiplash injuries and other widespread pain syndromes such as fibromyalgia and even central sensitisation (Casanueva et al., 2014; Castaldo et al., 2014). Other pathologies may be attributed to TrPs, but there is less evidence within the literature, these include: sub
acromial pain syndrome in the shoulder (Diercks et al., 2014), epicondyle dysfunction (Shmushkevich and Kalichman, 2013; Kheradmandi et al., 2015; Mora-Relucio et al., 2016), postoperative shoulder open reduction and internal fixation (Arias-Buría et al., 2015), osteoarthritis of the knee (Yentür et al., 2003; Henry et al., 2012; Alburquerque-García et al., 2015), patellofemoral pain syndrome (Oakes et al., 2009; Hains and Hains, 2010; Roach et al., 2013; Espí-López et al., 2017), chronic postsurgical pain following total knee replacement (Vas et al., 2004), postsurgical anterior cruciate ligament pain (Ortega-Cebrian et al., 2016), calf pain (Grieve et al., 2013a, 2013b), lowering of the medial longitudinal arch (Zuil-Escobar et al., 2015), chronic ankle instability (Salom-Moreno et al., 2015), chronic nonspecific foot pain (Hains et al., 2015), chest pain in stable coronary heart disease patients (Berg et al., 2015), post-stroke upper limb hypertonicity (Ansari et al., 2015; Mendigutia-Gómez et al., 2016), post-stroke lower limb hypertonicity (Park and Hwang, 2016), cancer (Hasuo et al., 2016; Vas et al., 2016), and post-traumatic stress syndrome (Vidaković et al., 2016).

TrPs may occur secondary to other pain generating pathologies (Gerwin, 2001; Fleckenstein et al., 2010; Borg-Stein and Iaccarino, 2014). However, there is not enough evidence at present to demonstrate a cause and effect. Addressing the pain and loss of motor function associated with TrPs could allow improved performance in asymptomatic subjects and reduced pain in symptomatic patients.

TrPs can give rise to satellite TrPs, for example, the infraspinatus referral pattern is in the anterior shoulder, which may cause latent TrP in the deltoid and biceps brachii to become active (Kuan et al., 2007).

2.2.3 Latent trigger points

Celik and Yeldan (2011) suggest that latent TrPs can reduce strength. Latent TrP can cause a muscle to remain active even during agonist contraction, thus preventing reciprocal inhibition (Ibarra et al., 2011). Potentially leading to altered activation patterns (Lucas et al., 2010). Latent TrPs are believed to affect the range of motion in the cervical region (Oliveira-Campelo et al., 2013; Bae, 2014). Latent TrPs are not spontaneously painful (Bron and Dommerholt, 2012) and a subject with latent TrPs may not be aware that they have TrPs or that the latent TrPs may be altering joint kinematics.
On review, latent TrPs may cause dysfunction as much as active TrPs, and may even be more problematic as they are not spontaneously painful and may go undiagnosed.

2.2.4 Anatomy and physiology of trigger points
In order to understand the effects of trigger points, it is important to discuss the microscopic structure of skeletal or striated muscle, the mechanism of voluntary muscle contraction in normal muscles and the chemo-physics of action potentials.

2.2.4.1 Microscopic structure of skeletal muscle
A single muscle fibre or myofibril is made up of a series of individual contractile units called sarcomeres along the length of the fibre. The myofibril is striated in appearance due to the alternating anisotropic band and isotropic bands (Huxley and Hanson, 1954). Each sarcomere is attached to the next sarcomere by the zwischenscheibe line, which is located in the middle of the isotropic band and is covered by the transverse tubule. Each sarcomere is surrounded by an extension of the transverse tubule called the sarcoplasmic reticulum. The anisotropic bands are made up of the transverse tubules which protrude from the mittelscheibe line. Each myosin head projects from the body of the thick filament at 45°. Each myosin has a large bulbous head which reverts back 90° when it is attached to the thin filament. The thin filament is comprised of the protein actin which projects from the zwischenscheibe line and interlock between the myosin in a hexagonal formation. Each actin is made up of globular strands which bind to the myosin (McArdle et al., 2009).

2.2.4.2 Action potential
An action potential is the chemo-electro mechanism of the physiological process of muscle contractions. During voluntary muscle contraction, an efferent depolarising signal is sent from the brain to the terminal axon at the neuromuscular junction. Synaptic vesicles release acetylcholine into the synaptic cleft. The acetylcholine attaches to acetylcholine receptors in the sarcomere and transmit the depolarising signal to the transverse tubules (McArdle et al., 2009). Depolarization of the transverse tubules causes a release of Calcium (Ca\(^{2+}\)) along the length of sarcomere via the lateral sacs of...
the sarcoplasmic reticulum into the sarcomere (Huxley and Taylor, 1958; Voigt et al., 2012).

2.2.4.3 The mechanism of voluntary muscle contraction

The interaction between actin and myosin to initiate a muscle contraction is called the sliding filament theory (Huxley and Niedergerke, 1954; Huxley and Hanson, 1954). The actin filaments are covered in strands of a protein called tropomyosin. Tropomyosin blocks the myosin from attaching to the actin. Tropomyosin is regulated by a protein called troponin. When troponin is activated by Ca\(^{2+}\), via the sarcoplasmic reticulum, it rotates and allows the binding site of the actin to be fully revealed. An adenosine triphosphate molecule attaches to the myosin head causing the myosin to detach from one molecule. The myosin head extends to 90° and attaches to a different actin molecule closer to the centre of the sarcomere. Pulling the Z-line closer to the mid-line of the sarcomere is known as a power stroke. The adenosine triphosphate discharges one phosphate ion, resulting in adenosine diphosphate and one free phosphate, during the myosin decoupling. Once the myosin binds to the new actin molecule, the adenosine diphosphate molecule detaches. This process will continue until there is no action potential in the form of calcium or the available adenosine triphosphate is depleted known as rigor (McArdle et al., 2009).

2.2.5 Summary of the anatomy and physiology of trigger points

The physical manifestation of TrPs is likely to be as a result of a constant contraction due to a proliferation of acetylcholine and thus a sustained action potential of a muscle fibre resulting in depletion of adenosine triphosphate and subsequent rigour.

2.2.6 Aetiology of trigger points

The aetiology of TrPs is still not fully understood (Dommerholt et al., 2011; Shah et al., 2015). Unaccustomed muscular overload results in a release of inflammatory markers such as hydrogen, potassium, bradykinin, and substance P, while this is a normal response, it can lead to sensitisation of muscle nociceptors which may result in central sensitisation (Dommerholt et al., 2011).
Direct trauma is reported in much of the literature regarding TrPs, in particular, TrPs which are associated with cervical injuries sustained during slow speed vehicular impacts (Tough et al., 2010; Castaldo et al., 2014; Fernández-Pérez et al., 2012). Abnormal electromyography (EMG) potentials and morphological changes of thin and thick muscle fibres in the vastus medialis (VM) of rats have been observed after repeated blunt force trauma (Huang et al., 2013).

There are two main schools of thought on the aetiology of TrPs; they are peripheral sensitisation and central sensitisation (Fernández-de-las-Peñas and Dommerholt, 2014; Shah et al., 2015). Physiological stress may also be a contributing factor (McNulty et al., 1994).

### 2.2.6.1 Peripheral sensitisation

There are three features in the TrP positive feedback cycle: 1) abnormal acetylcholine release, known as endplate noise; 2) increased muscle fibre tension causing taut bands; 3) the release of sensitising substances resulting in pain (Simons, 2004a). This feedback cycle is due to muscle overuse or direct trauma impacts (Itoh and Kawakita, 2002; Itoh et al., 2004b; Kawakita et al., 2008; Tough et al., 2010; Castaldo et al., 2014; Fernández-Pérez et al., 2012).

#### 2.2.6.1.1 Abnormal acetylcholine release

The dysfunction causing TrPs is hypothesised to emanate from the motor endplate, or neuromuscular junction (Simons, 2001; Simons et al., 2002). This dysfunction is also known as presynaptic dysfunction (Wang, 2005). It is due to spontaneous electrical activity across the synaptic cleft, a consequence of acetylcholine release. Excessive acetylcholine causes contractions lasting up to 60 minutes during an in vitro study (Mense et al., 2003). In an in vitro study, muscle fibres that had a 1% stretch showed a 10% increase of acetylcholine (Chen and Grinnell, 1997). As well as the presynaptic dysfunction there are two more complex issues at the motor endplate which contribute the contraction cascade, these are, intra-synaptic, and postsynaptic dysfunctions (McPartland, 2004). Intra-synaptic dysfunction is a result of a reduction in the acetylcholine deactivating enzyme acetylcholinesterase. Without acetylcholinesterase, acetylcholine is able to activate the muscle via nicotinic acetylcholine receptors. Low levels of one leg sport-specific vertical jump can be genetic or as a result of poisoning.
such as organophosphate pesticides. Postsynaptic dysfunction is as a result of hyperexcitability or a “gain of function” (McPartland, 2004) due to excessive NACHRs expression; a genetic or pathological response to choline, an ordinary serum metabolite similar to acetylcholine; or even acetylcholine channels remaining open longer than normal (Shen et al., 2005).

2.2.6.1.2 Sustained contraction

The concept of peripheral sensitisation as a cause of TrPs dates back to Henneman’s "size principle" (Henneman et al., 1964, 1965) and expanded by Hägg’s “Cinderella theory” (Hermens and Hutten, 2002). These overload theories focus on the ragged histological appearance characterised by overloaded, slow oxidative muscle fibres. During low-level muscular contractions, slow oxidative muscle fibres are recruited first, followed by fast oxidative fibres, and finally by fast glycolytic fibres. The slow oxidative muscle fibres are the first to be contracted, remain contracted, and are the last to be depolarised. The sliding filament theory utilises adenosine triphosphate to release the cross bridge between actin and myosin (Huxley and Niedergerke, 1954; Huxley and Hanson, 1954). If there is a depletion of adenosine triphosphate and there is not an adequate amount of recovery the sarcomere will go into sustained contraction or rigour (Bron and Dommerholt, 2012). Sustained contraction results in ischemia, hypoxia and insufficient adenosine triphosphate synthesis in slow oxidative muscle fibre type motor units leading to increased acidity, calcium accumulation, and subsequent sarcomere contractions (Bron and Dommerholt, 2012). Larger diameter muscle fibres require a significant action potential and are less prone to sustained contraction or rigour. However, altered morphology has been reported in thin and thick muscle fibres in rat VM after repeated blunt force trauma (Huang et al., 2013). When a muscle is in rigour, it results in an increase of pressure in the muscle and may occlude the capillaries reducing the supply of oxygen and glucose. A contraction of 10-25% maximum voluntary contraction can affect intramuscular blood circulation (Maekawa et al., 2002; Bron and Dommerholt, 2012). Eccentric loading may be more likely to cause myofascial pain syndrome and TrPs because it is associated with muscle soreness and muscle damage (Gerwin et al., 2004). Eccentric loading of the extensor digitorium to fatigue has been reported to induce transient TB (less than seven days), elicit RP and LTR, spontaneous
electrical activity and reduced pressure pain threshold (PPT a clinical measure of the sensitivity of TrPs[p.35]) (Itoh et al., 2004b; Kawakita et al., 2008). During in vitro studies of rabbits, eccentric exercise increased reflex EMG for up to seven days, and eccentric contractions under ischemia increased reflex EMG for up to 21 days (Itoh and Kawakita, 2002; Kawakita et al., 2008).

2.2.6.1.3 Sensitising

The idea of altered oxidative metabolism started with the “energy crisis hypothesis” (Simons and Travell, 1981), where the constant muscle contraction causes the surrounding tissue to become hypoxic due to the depletion of adenosine triphosphate. That was expanded to the “integrated hypnosis” (Simons, 2004c) where the hypoxia causes a release of inflammatory markers such as substance P, adenosine triphosphate, bradykinin, serotonin, prostaglandins, and potassium when muscles are damaged, which in turn stimulates the nociceptors. Active TrP sites have been reported to be more acidic and have higher levels of inflammatory chemical makers such as neuropeptides, catecholamines, and cytokines (Ballyns et al., 2011). Active TrPs have elevated levels of neuroactive mediators such as bradykinin, substance P, or serotonin (Alonso-Blanco et al., 2012). An expansion of the integrated hypothesis of TrP formation suggests callitonin-gene-related-peptide is also released during increases endplate activity. Callitonin-gene-related-peptide is responsible for the neurogenic release of inflammatory markers (Gerwin et al., 2004).

The integrated hypothesis is the most accepted model to explain TrPs. Simons (2008) suggests that two studies (Shah et al., 2005, 2008) managed to substantiate the integrated hypothesis of TrP formation. Shah et al. (2005) reported that during an in vivo study of normal subjects, subjects with latent TrPs, and patients with active TrPs, there was a lower pH (p < 0.03) and higher bradykinin, callitonin-gene-related-peptide, interleukin-6, interleukin-1β, norepinephrine, potassium, prostaglandins, serotonin, substance P, and tumor necrosis factor-α present in the active TrP group (p < 0.01). There was also an increase of callitonin-gene-related-peptide and substance P in the active TrP group when there was a LTR elicited (p < 0.02). The finding of Shah et al.’s (2008) study reported that there were higher levels of inflammatory markers present in patients with active TrPs after compressing an area with a TrP compared to an area
without TrPs, thus providing evidence for the integrated hypothesis of TrP formation. Higher levels of interleukin-6 and interleukin-8 (p < 0.01) have been reported in patients with active TrPs compared to patients with latent TrPs (Shah et al., 2008). There was a within-group difference increase for the active TrP group for bradykinin, interleukin-1β, interleukin-6, interleukin-8 norepinephrine, potassium, prostaglandins, substance P, and tumour necrosis factor-α (p < 0.05) when compared to a different site (gastrocnemius). The finding of Shahs’ (2008) study suggests that there are higher levels of inflammatory markers at the TrP sites in patients with active TrPs when compared to sites with no TrPs, thus providing supporting evidence for the integrated hypothesis of TrP formation.

### 2.2.6.2 Summary of peripheral sensitisation

The build-up of inflammatory markers is a significant contributing factor in the formation of TrPs at the motor-neuro junction. Whether this is the only factor is unclear. There is a need for further investigation into the effects of inflammation in the peripheral nervous system. The presence of these inflammatory markers may also have an effect on the afferent sensory nervous system.

### 2.2.6.3 Central sensitisation

Central sensitisation is defined as the hypersensitivity to pain which manifests in the central nervous system (Dommerholt, 2011). The presence of inflammatory markers is believed to stimulate pain sensitive axons called nociceptors (Bron and Dommerholt, 2012; Jafri, 2014). In particular the thick myelinated afferent A-δ-type or C-type fibres (Wang et al., 2010; Meng et al., 2015). There has been spontaneous electrical activity detected in TBs (Itoh et al., 2004b; Kawakita et al., 2008). It is unclear whether the spontaneous electrical activity is an A-type, efferent signal to the neuromuscular junction or it is, A-δ-type or C-type afferent axon signal to the central nervous system. There is an association between myofascial pain syndrome and central sensitisation and that TrP may perpetuate the central sensitisation or even cause it (Xu et al., 2010). Whereas C-type fibres have been purposed as a possible cause of TrP pain (Kawakita et al., 2008; Bron and Dommerholt, 2012). Sustained nociceptive mechanical stimulation of latent TrPs in the tibialis anterior has been reported to increase pain sensation on the contralateral side (Xu et al., 2010).
During an in vitro study of rats, repeated focal electrical stimulation of 1Hz was enough to stimulate the C-type fibres (Taguchi et al., 2005). Low-intensity electrical stimulation of TrPs increase the sensitivity, when measured with the PPT, and activates portions of the brain associated with the opioid system which is responsible for the perception of pain (Niddam et al., 2007). Increased PPT, for up to 15 minutes, in the infraspinatus TrP, but not the gluteus medius TrP, after treating TrPs in the supraspinatus with ultrasound (Srbely et al., 2009) and DN (Srbely et al., 2010) may suggest that anti-nociceptive effects are specific to the same spinal segment but not centrally. Whether TrP induces central sensitisation is uncertain, as other studies show a decrease in central sensitisation following deactivation of active TrPs (Niddam et al., 2007; Srbely et al., 2009; Freeman et al., 2009). The results of the study of Xu et al. (2010) suggest that 8 minutes of nociceptive mechanical stimulation significantly increases maximal pain intensity (p < 0.05) in subjects with latent TrPs. RP is believed to be a central sensitisation process mediated by peripheral sensitisation with additional sympathetic activity (Arendt-Nielsen and Svensson, 2001; Arendt-Nielsen et al., 2008). There is not enough evidence as yet to fully determine the mechanism of the development of referred pain.

2.2.6.4 Summary of central sensitisation

Inflammatory markers produced during fatigue, over load or trauma stimulate thick myelinated afferent A-δ-type or C-type fibres causing an efferent contraction as a protection mechanism. Thus contributing to the positive feedback loop.

2.2.6.5 Other theories

The fascia is highly innervated by nociceptors and is particularly sensitive to callitoningene-related-peptide and substance P (Tesarz et al., 2011) and may play a role in the development of TrPs. The fascia is strongly associated with hyaluronic acid, a high molecular weighted glycosaminoglycan polymer of the extracellular matrix, which allows lubrication of muscles within the fascia (Stecco et al., 2011). The contracted sarcomeres cause a lengthening of muscle spindle which in turn causes the muscle to contract more. The hyaluronic acid is hypothesised to become viscous in acidic conditions possibly causing more stretch and affecting the muscle spindles (Stecco et al., 2013). This theory of the influence of hyaluronic acid in relation to TrP and myofascial pain syndrome is still in its infancy. There is only a review (Stecco et al., 2013), an in vitro
histological analysis (Stecco et al., 2011), an ultrasound analysis (Stecco et al., 2011), and a mathematical model of the possible mechanics of hyaluronic acid flow in the muscle (Roman et al., 2013) to support the possible role hyaluronic acid may play in the formation of TrPs. There is not enough evidence at present to support or denounce this theory. Further research is needed to establish the influence of fascia in relation to the aetiology of TrPs (Shah et al., 2015).

2.2.7 Summary of the aetiology of trigger points
The aetiology of TrPs is multifactorial and still not fully understood. It is likely to be a positive feedback mechanism. Ischemia is a significant, if not the primary, factor in TrP aetiology. There is no definite explanation, but it is accepted to be attributed to altered motor endplate activity (Hubbard and Berkoff, 1993) due to unaccustomed overload (Itoh and Kawakita, 2002; Itoh et al., 2004b; Kawakita et al., 2008) or trauma (Tough et al., 2010; Castaldo et al., 2014; Fernández-Pérez et al., 2012). Initial peripheral pain is likely due to inflammatory markers (Gerwin et al., 2004; Shah et al., 2005, 2008; Hsieh et al., 2012) which lead to sensitising of the nociceptors, causing central sensitisation (Kawakita et al., 2008; Bron and Dommerholt, 2012), which in turn feeds back to cause sustained contraction (Wang et al., 2010; Meng et al., 2015), resulting in further release of inflammatory markers (Gerwin et al., 2004).

2.2.8 Dysfunctions caused by trigger points
TrPs are believed to cause tension-type headaches (Fernández-de-Las-Peñas et al., 2010, 2011; Ghanbari et al., 2012; Berggreen et al., 2012; Karadaş et al., 2013; Espí-López et al., 2014a, 2014b; Taylor, 2014) and neck pain (Gerber et al., 2015; Sumen et al., 2015; Paolucci et al., 2016). The pain in relation to tension-type headaches and neck pain is predominantly due to the RP (Travell and Simons, 1992; Simons et al., 1999; Dommerholt and Huijbrechts, 2011; Sanz et al., 2016) in particular from the upper trapezius (Unalan et al., 2011; Myburgh et al., 2012; Sarrafzadeh et al., 2012; Kannan, 2012; Grieve et al., 2013b; Oliveira-Campelo et al., 2013; Halski et al., 2015; Benjaboonyanupap et al., 2015; Xie et al., 2015). Other disorders associated with TrPs are temporomandibular disorder (Ariji et al., 2015; Asha et al., 2015; Christidis et al., 2015; Gonzalez-Perez et al., 2015; Quek et al., 2015; Al-Khotani et al., 2016; De Carli et al., 2016; Rai et al., 2016) and shoulder impingement (Lucas et al., 2010).
RP, there is also altered joint kinematics, possibly due to the altered muscle length caused by TBs (Simons, 2004a; Shankar and Reddy, 2012); altered muscle activation patterns caused by endplate noise (Lucas et al., 2010; Ge et al., 2014; Sergienko and Kalichman, 2015) or reduced strength as a result of a muscle’s inability to contract (Celik and Yeldan, 2011). In summary, TrPs can alter joint biomechanics and can be painful, causing dysfunction. If a muscle is unable to contract efficiently, it is unable to contribute to force production.

2.2.8.1 Trigger points in the quadriceps

The quadriceps is a four-headed muscle in the anterior compartment of the thigh comprising of the rectus femoris, vastus lateralis (VL), VM and vastus intermedius. All four heads insert into the tibial tuberosity via the patellar tendon which incorporates the patella, a sesamoid bone which functions as a pulley to increase the leverage of the quadriceps (Pasta et al., 2010). The VL, VM and vastus intermedius only extend the knee joint; whereas the rectus femoris also flexes the hip joint. The quadriceps influence stabilisation of the knee (Hains and Hains, 2010). Reduced muscle activation of the VL and VM has been reported in patients with patellofemoral pain syndrome (Cavazzuti et al., 2010; Lankhorst et al., 2013). The RP pattern for the VM due to the presence of TrPs is to the medial-anterior aspect of the knee, whereas, the RP pattern for TrPs in the VL is referred to the lateral knee and the lateral hip (Travell and Simons, 1992). The RP of TrPs in the quadriceps may confound pathologies of the knee such as patellofemoral pain syndrome (Oakes et al., 2009; Hains and Hains, 2010) and knee osteoarthritis (Yentür et al., 2003; Itoh et al., 2008b; Henry et al., 2012; Mayoral et al., 2013; Alburquerque-García et al., 2015). The majority of studies in relation to TrPs in the quadriceps are in relation to active TrPs (Oakes et al., 2009; Hains and Hains, 2010; Yentür et al., 2003; Itoh et al., 2008b; Henry et al., 2012; Mayoral et al., 2013; Alburquerque-García et al., 2015). There are, to the author’s knowledge, no studies which investigate the effects of treating latent TrPs in the VL and VM on jump performance. TrPs in the quadriceps may play a role in the pain associated with knee pathologies. Further investigation is needed to ascertain if TrPs are involved in the aetiology of knee pathologies such as patellofemoral pain syndrome or knee osteoarthritis.
2.2.8.2 The influence of trigger points on functions involving the kinetic chain

Jumping requires many muscles around the hip, knee and ankle called the kinetic chain (Decker et al., 2003). The movements involved in jumping are hip extension (gluteus maximus), knee extension (VL, VM, rectus femoris and vastus intermedius) and plantarflexion of the ankle (gastrocnemius, soleus, plantaris) often called triple extension (Lorenz, 2016). The triple extension and kinetic chain are required for optimum jumping, running and changing direction at speed (Archer, 2016; Lorenz, 2016). Latent TrPs may prevent an athlete from performing at their full potential (Ameloot et al., 2016) during jumping.

Latent TrPs in the muscles of the anterior compartment of the leg have been reported to affect the medial longitudinal arch of the foot causing dysfunction in bipedal standing and gait by impact absorption and transmission of ground reaction force (Zuil-Escobar et al., 2015). Elevated EMG activity of the VL and VM has been associated with dysfunctional foot biomechanics (Telfer et al., 2013).

2.2.8.3 The effect of trigger points on athletic performance

No study to date has analysed the effects of the presence of TrPs in a muscle on athletic performance by comparing it to a group without trigger points. Studies instead have examined if the treatment of the TrPs itself will improve performance, and thus suggest that their presence in a muscle must alter performance negatively. The number of studies that investigate the effects of the treatment of TrPs on athletes and athletic performance is limited, with to the author’s knowledge, only three peer-reviewed articles and at least four master’s theses available on the topic.

In a set of studies by Quinn et al. (2013, 2016) the author suggested that the presence of TrPs may have negative effects on a golf swing and in golf shot accuracy. In the study by Quinn et al. (2016) one group received TrP therapy and medicine ball exercises, while another group received TrP therapy, and hip flexor stretches, and another group acted as a control. However, the type and nature of the TrP therapy were not reported in this study. The study indicated that the group who received the TrP therapy and medicine ball exercise had a significant improvement ($p = 0.02$) in hip turn during the downswing phase of the golf swing at the point when the club made contact with the golf ball by
3.52° using 3D motion capture. In the earlier study by Quinn et al. (2013), there was a significant improvement ($p = 0.03$) in swing path accuracy in the group who received TrP therapy and medicine ball exercises (78.79%) compared to the control group (16.00%), but there was not a significant difference between the group that received TrP therapy and hip flexor stretches (76.47%) and the control group ($p > 0.05$). However, the type and nature of the TrP therapy were not reported in the study by Quinn et al. (2013). In both studies (Quinn et al., 2013, 2016), the post-test measure was conducted one week after the pre-test, the procedure for the TrP therapy was not specified, and there was no alteration in hip flexor length in any group.

In a novel study by Espejo-Antúnez et al. (2016), they examined the effects of compressing TrPs in the masseter muscles of the jaw while performing proprioceptive neuromuscular facilitation stretching in the hamstring to improve active knee extension. The predication of this study was based on the “fascial connection theory” (Moon and Lee, 2011). Several investigators have explored the relationship between TrPs in one region and stretching or treating a muscle in a distal location (Aparicio et al., 2009; Mansilla-Ferragut et al., 2009; Rodriguez-Blanco et al., 2015). In the study of Espejo-Antúnez et al. (2016), there was no significant difference in active knee extension ($p > 0.05$) after using proprioceptive neuromuscular facilitation stretches to the hamstring combined with ischemic compression technique to TrPs in the masseter muscles (6.67°), compared to using PNF to the hamstring alone (7.62°).

Other studies suggest that the presence of TrPs in the gastrocnemius can have an adverse effect on jump performance (O’Rourke, 2010; Devereux, 2016). In the study by O’Rourke (2010), it was reported that treating TrPs in the gastrocnemius had no effect in CMJ-JH after 24 hours but did have a positive but non-significant effect on EMG root mean$^2$ from 152.73 to 159.82 mV with an observed power of 0.07 for between day and 0.16 for between group ($p > 0.05$). Devereux (2016) reported a non-significant degradation in CMJ-JH, immediately after treating TrPs in the gastrocnemius with DN ($p > 0.05$), but did report a significant improvement between the post-treatment outcome session and the outcome session 48 hours after treatment session ($p = 0.01$).

Furthermore, some studies have reported that treating TrPs in the lower kinetic chain have reduced strength and performance (Barry, 2015; Kennedy, 2015). The study by...
Kennedy (2015) investigated the effects of TrPs in the piriformis and gluteus medialis on hip range of motion, muscle length and isometric strength. Kennedy (2015) reported a nonsignificant reduction in strength in hip abduction immediately after treating TrPs in the piriformis and gluteus medius with DN ($p > 0.05$). Furthermore, Barry (2015) explored the effects of treating TrPs in the rectus femoris and gastrocnemius on running performance. A significant decrease ($p = 0.03$) in the distance covered during a ten minutes self-paced run 24 hours after DN TrPs in the rectus femoris and gastrocnemius was reported.

### 2.2.8.4 Summary of the effect of trigger points on athletic performance

The effects of the presence of TrPs in the lower limb are under-investigated. Active and latent TrPs can potentially alter movement activation patterns, and reduce strength and power in muscles. The quadriceps are pivotal in force development in activities such as jumping and running. TrPs in the quadriceps may reduce an athlete’s ability to perform maximally. Treating TrP in a quadriceps may improve athletic performance outcome markers such as jumping. There is a substantial gap in the literature in relation to effects of TrPs on athletic performance and if treatment of TrPs can improve sports performance, especially in the lower limb. The legs and the muscles in the kinetic chain are vital in sports performance. Therefore, it is essential to determine the effects of the presence of TrPs in the lower limb on athletic performance such as jumping.

The optimum treatment strategies for TrPs are still unclear. DN has been substantially reported in the literature, and it is hard to dispute the long-term positive effects it has on symptomatic patients. The research in relation to treating TrPs to improve athletic performance is limited. Studies that have used DN to treat TrPs for athletic enhancement have reported immediate degeneration in performance, possibly as a result of post-needling soreness. Therefore it would be of particular importance to establish whether other less invasive treatments are as effective at treating TrPs as DN, such as ESWT.

There is a school of thought that suggests that health care evidence based practice relies heavily on randomised control trials and quantitative data and dismiss qualitative data sometimes to the detriment of the subjects as a whole (Holmes et al., 2006). Isolationist research such as this may fail to address or identify critical qualitative markers pertinent
to help paint a broader understanding of the patient. That being said, quantitative markers can be used to document the progression of a patient’s condition and are the ones utilised in the literature.

2.2.9 Palpating trigger points

In order to correctly ascertain a differential diagnosis in relation to TrPs, Travell and Simons (1997) suggested that a TrP must contain one or more of the following essential criteria: 1) palpable TB; 2) exquisite TS in a TB; 3) patient's pain recognition PR of current complaint when pressure is applied to the tender nodule, in the case of an active TrP; and 4) painful limit to full stretch range of motion. Other confirmatory observations may include: 1) visual or tactile identification of LTR; 2) the occurrence of a global JS induced by needle penetration of tender nodule; 3) RP or altered sensation in the pattern expected from a TrP in that muscle on compression of tender nodule; and 4) EMG demonstration of SPA characteristic of active loci in the tender nodule of a TB (Travell and Simons, 1992; Simons et al., 1999).

No gold standard exists to examine for TrPs, and their assessment relies on subjective clinical judgement (Njoo and van der Does, 1994). Palpation is still the main tool used to locate and diagnose TrPs. Experience is considered essential in diagnosing TrPs (Myburgh et al., 2011). The consensus would be that the combination of TS in a palpable TB and subject recognition of the pain, are minimum acceptable criteria (Hsieh et al., 2000; Bron et al., 2007). Njoo and van der Does (1994) suggest that JS may also be useful. Simons et al. (1999) recommend when reporting on TrPs, it should specify in the methods section specifically which TrP diagnostic criteria were used and should describe how palpating the TrP was performed (Travell and Simons, 1992; Simons et al., 1999).

2.2.9.1 The current literature of reliability of the palpation of trigger points

Four systematic reviews have been carried out to examine the reliability of identifying myofascial trigger points (Myburgh et al., 2008; Lucas et al., 2009; McEvoy and Huijbregts, 2011; Rathbone et al., 2017). There have also been thirteen published peer-reviewed reliability studies using palpation to locate and classify TrPs (Nice et al., 1992; Wolfe et al., 1992; Njoo and van der Does, 1994; Gerwin et al., 1997; Lew et al., 1997;
Hsieh et al., 2000; Al-Shenqiti and Oldham, 2005; Bron et al., 2007; Myburgh et al., 2011; Barbero et al., 2012; Zuil-Escobar et al., 2015; Mora-Relucio et al., 2016; Sanz et al., 2016). Three reliability studies have used novel methods to diagnose TrPs (Sciotti et al., 2001; Dibai-Filho et al., 2015; Skorupska et al., 2015). There are also at least five unpublished Masters theses which incorporate TrP location reliability (O’Rourke, 2010; Hynes, 2011; Barry, 2015; Kennedy, 2015; Devereux, 2016). A summary of peer reviewed literature is compiled in Table 1 (p. 21).
### Table 1: Summary of peer-reviewed literature in relation to the reliability of palpating trigger points

<table>
<thead>
<tr>
<th>Author</th>
<th>n = (male, female)</th>
<th>Study design</th>
<th>Subject characteristics</th>
<th>Muscle tested</th>
<th>Criteria used</th>
<th>Outcomes</th>
<th>Examiner experience</th>
<th>Additional training</th>
<th>Statistical analysis used</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nice et al.</td>
<td>50 (19, 31)</td>
<td>Paired inter-rater study</td>
<td>Patients with TrPs related pain to the lower back</td>
<td>Bilateral iliocostalis, lumborum, longissimus thoracis</td>
<td>PR</td>
<td>The presence of TrPs, pain reproduction</td>
<td>12 testers (6 pairs) with 3-17 years of experience (7 of which routinely treat TrPs)</td>
<td>A handout with written description of TrPs in muscles to be tested. 3 testers had specialised training in locating TrPs</td>
<td>ϰ, %-Agre, the observed proportion of positive agreement, the observed proportion of negative agreement</td>
<td>ϰ, 0.29-0.38; %-Agre, 76-79%; Observed proportion of positive agreement, 0.43-0.52; Observed proportion of negative agreement</td>
<td>Fair</td>
</tr>
<tr>
<td>Wolfe et al.</td>
<td>23 (0, 23)</td>
<td>Inter-rater study</td>
<td>FM (n=7), MFP (n=8), asymptomatic (n=8)</td>
<td>LavScap, Supra, AntScal, upper Traps, Infra, sternal portion of the Pec, sternal division of the Stcm, iliocostalis, longissimus</td>
<td>TS, TB, LTR, RP</td>
<td>Bilateral tender point rheumatology examination: occiput, lower cervical, Traps, Supra, Infra, lateral Pec, 2nd rib, lateral epicondyle, gluteal, greater trochanter and knee; Unilateral TrP examination</td>
<td>4 rheumatologists; 4 experts in myofascial pain</td>
<td>N/A</td>
<td>2 way ANOVA, χ²</td>
<td>No difference within rheumatologists in relation to TS; but there was in relation to TB, TR and RP. No difference within MFP experts in relation to TS, TB, RP; but there was in relation to TR. FM subjects reported more symptoms than MFP and asymptomatic subjects; MFP subjects reported more symptoms than asymptomatic subjects</td>
<td>Low</td>
</tr>
</tbody>
</table>

Where TrP is trigger points, PR is pain recognition, ϰ is kappa score, %-Agre is percent agreement, FM is fibromyalgia, MFP is myofascial pain, LavScap is levator scapula, Supra is supraspinatus, AntScal is anterior scalenes, Traps is trapezius, Infra is infraspinatus, Pec is pectoral major, Stcm is sternocleidomastoid, TS is tender spot, TB is taut band, LTR is local twitch response, RP is referred pain, ANOVA is analysis of variance, χ² is chi-squared test.
Table 1: Summary of peer-reviewed literature in relation to the reliability of palpating trigger points

<table>
<thead>
<tr>
<th>Author</th>
<th>n = (male, female)</th>
<th>Study design</th>
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<th>Additional training</th>
<th>Statistical analysis used</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Njoo and van der Does (1994)</td>
<td>124 (61, 53)</td>
<td>Inter-rater study</td>
<td>Symptomatic (n=61) recent lower back pain (&lt;2 months) with a previous episode of lower back pain (&gt;3 months); asymptomatic (n=63).</td>
<td>Bilateral QL, GlutMed</td>
<td>TS, TB, LTR, RP, ↓ROM, JS, PR</td>
<td>The presence of TrPs, pain reproduction</td>
<td>1 experienced general practitioner and 2 medical students</td>
<td>Practical training prior to testing; duration not specified</td>
<td>χ, χ</td>
<td>(£Q, GlutMed): TS (0.73, 0.53), RP (0.36, 0.46), TB (0.47, 0.51), LTR (0.19, -0.02), JS (0.68, 0.71), PR (0.57, 0.58). χ² control significantly different to lower back pain subjects</td>
<td>Slight to substantial</td>
</tr>
<tr>
<td>Gerwin et al. (1997)</td>
<td>Phase 1: 25 (12, 13); phase 2: 10 (3, 7)</td>
<td>2 phase paired inter-rater study</td>
<td>Asymptomatic</td>
<td>Stcm, upper and lower* Trap, AntScal*, LavScap*, Infra*, lats, TMinn*, Triceps*, ExtDig</td>
<td>TB, LTR, RP, PR</td>
<td>TrPs present, RP or PR</td>
<td>4 physicians (2 psychiatrists and 2 neurologists) experienced in diagnosing and treating TrPs</td>
<td>A review the previous night (phase 1); meeting 3 hours prior testing (phase 2)</td>
<td>χ</td>
<td>PR: 0.60-0.95; TB: 0.40-almost perfect; Stcm 0.11- almost perfect; ExtDig 0.51- almost perfect</td>
<td>Fair to almost perfect</td>
</tr>
<tr>
<td>Lew et al. (1997)</td>
<td>58 (24, 34)</td>
<td>Paired inter-rater study</td>
<td>Asymptomatic in past 3 months</td>
<td>Unilateral upper Traps</td>
<td>TS, TB, RP</td>
<td>LTrPs present, RP agreement with another therapist (Scm)</td>
<td>2 experts in TrPs</td>
<td>The agreement of LTrP location</td>
<td>Agreement of LTrP 10%; Agreement of RP LTrP 21%</td>
<td>χ</td>
<td>Agreement of LTrP 10%; Agreement of RP LTrP 21%</td>
</tr>
</tbody>
</table>

Note: *indicates muscles were not tested in the second phase.

Where QL is quadratus lumborum, GlutMed is gluteus medius, TS is tender spot, TB is taut band, LTR is local twitch response, RP is referred pain, ↓ROM is reduced range of motion, JS is jump sign, PR is pain recognition, (L)TrP is (latent) trigger points, χ is kappa score, χ² is chi-squared test, Stcm is sternocleidomastoid, Traps is trapezius, AntScal is anterior scalenes, LavScap, is levator scapula, Infra is infraspinatus, Lats is latissimus dorsi, TMinn is teres minor, Triceps is triceps brachii, ExtDig is extensor digitorum.
Table 1: Summary of peer-reviewed literature in relation to the reliability of palpating trigger points

<table>
<thead>
<tr>
<th>Author</th>
<th>n = (male, female)</th>
<th>Study design</th>
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<th>Outcomes</th>
<th>Examiner experience</th>
<th>Additional training</th>
<th>Statistical analysis used</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hsieh et al. (2000)</td>
<td>52 (29, 23)</td>
<td>Inter-rater with a quasi-validity element</td>
<td>Symptomatic (n=26) non-descript lower back pain; asymptomatic (n=26)</td>
<td>Iliocostalis, lumbarorum, Rect-Fern, RectAbs, TFL, QL, GluMax, GluMin, piriformis, soleus</td>
<td>TB, LTR, RP</td>
<td>Are trained and untrained as reliable as experts at locating TrPs</td>
<td>4 licensed chiropractors (15-2 years’ experience), 4 licensed physiatrist residents (15-2 years’ experience)</td>
<td>The most experienced and 2 least experienced testers received a handout. The rest received 6 hours training</td>
<td>χ, Student’s t-test</td>
<td>χ 0.15-0.34 (trained and expert); χ 0.04-0.29 (untrained and expert); χ -0.01-0.32 (trained and untrained). Student’s t-test significant difference between the experience of testers, but poor reliability</td>
<td>Slight to fair</td>
</tr>
<tr>
<td>Sciotti et al. (2001)</td>
<td>20 (8, 12)</td>
<td>Inter-rater study</td>
<td>Asymptomatic</td>
<td>Traps</td>
<td>Must have TB, nodule and or TS, as well as JS, LTR</td>
<td>3D location of TrP and PPT</td>
<td>4 experienced clinicians with 9-12 years’ experience</td>
<td>12 hours training and practice</td>
<td>3D plotting: repeated ANOVA, G-Coef, SEM; PPT: repeated ANOVA</td>
<td>3D plotting: no significant difference between testers, G-Coef ≥0.80, SEM&lt;15mm in x, y and Z-axes; PPT: no significant difference between testers</td>
<td>High</td>
</tr>
<tr>
<td>Al-Shengiti and Oldham (2005)</td>
<td>58 (31, 27)</td>
<td>Intra-rater study</td>
<td>Diagnosed rotator cuff tendonitis for 6-78 weeks</td>
<td>Supra, Infra, TMim, Subscap</td>
<td>TS, TB, JS, LTR, PR, RP</td>
<td>TrPs present Physiotherapist (11 years’ clinical experience) and extensive training in TrP examination</td>
<td>N/A</td>
<td></td>
<td>χ 0.75-1.00 for all criteria except where there was no TrP located (Supra and Subscap)</td>
<td>Slight to almost perfect</td>
<td></td>
</tr>
</tbody>
</table>

Where Rec-Fern is rectus femoris, RectAbs is rectus abdominis, TFL is tensor fasciae latae, QL is quadratus lumborum, GluMax is gluteus maximus, GluMed is gluteus medius, GlutMin is gluteus minimus, TB is taut band, LTR is local twitch response, RP is referred pain, TrP is trigger points, χ is kappa score, Traps is trapezius, TS is tender spot, JS is jump sign, 3D is three dimensional, PPT is pressure pain threshold, ANOVA is analysis of variance, G-Coef is generalizability coefficients, SEM is standard error measurement, Supra is supraspinatus, Infra is infraspinatus, TMim is teres minor, Subscap is Subscapularis, PR is pain recognition.
### Table 1: Summary of peer-reviewed literature in relation to the reliability of palpating trigger points

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</table>
| Bron et al. (2007)      | 40 (gender not specified) | Inter-rater study | Symptomatic (n=8) unilateral or bilateral shoulder pain  
Asymptomatic (n=32) | Infra, Delt and Biceps | TB, TS, RP, LTR, JS | The ability to elicited TB, TS, RP, LTR, JS | Raters: 3 physical therapists (29, 28 and 16 years’ experience). Each has 21, 16 and 2 years specialising in TrP diagnosis in the neck shoulders and upper limb. Observers: 3 physical therapists with experience of treating MFP | 8 hours of practical and theory. A consensus on all definitions of the study | ϰ, PA% | TS in TB 45-90%-Agre (ϰ 0.16-0.75);  
RP 63-93%-Agre (ϰ -0.13-0.64);  
LTR 33-100%-Agre (ϰ -0.05-1.00);  
JS 69-93%A-gre (ϰ 0.02-0.68) | Slight to almost perfect |
| Myburgh et al. (2011)   | 81 (0, 81)        | Inter-rater study | Symptomatic (n=67) > 4 hours a day of office work and suffered from neck/shoulder pain  
Asymptomatic (n=14) | Bilateral upper Traps | TB, and either plus any two of TS, PR, RP | TrPs using global assessment | Experienced (6.0-5.5 years), inexperienced (8-9 semesters) | 5 min force curve training every day of testing | ϰ | ϰ: experienced (0.63), inexperienced (0.22) | Substantial and fair |
| Barbero et al. (2012)   | 24 (1, 23)        | Intra-rater study | Patients with neck-shoulder pain  
Bilateral upper Traps | Bilateral upper Traps | TB, TS, RP, FP | ALS, TrP_d between sessions | Physiotherapists with 10 years dealing with TrPs | Postgraduate course TrP diagnosis and treatment | ALS (X-line, Y-line): ICC, 95% CI;  
Bland-Altman Plots, LoA;  
TrP_d: t-test | X-line ICC: 0.62 (95% CI: 0.30-0.81),  
Y-line ICC: 0.81 (95% CI: 0.61-0.91), X-line Bland-Altman plots (n=23) within the 95% CI,  
Y-line Bland-Altman plots (n=22) within the 95% CI;  
TrP_d: t-test was no significant between sides | Moderate to high |

Where TB is taut band, TS is tender spot, RP is referred pain, LTR is local twitch response, JS is jump sign, MFP is myofascial pain syndrome, ϰ is kappa score, PA% is percent agreement, Traps is trapezius, PR is pain recognition, ALS is anatomical landmark system, TrP_d is distance between trigger points, ICC intra-class correlation, CI is confidence intervals, LoA is limit of agreement.
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<tbody>
<tr>
<td>Skorupska et al. (2015)</td>
<td>30 (gender not specified)</td>
<td>Intra-rater study</td>
<td>Patients with sciatic pain (n=30), asymptomatic (n=15)</td>
<td>GlutMin</td>
<td>TB, TS, RP, PR</td>
<td>Change in temperature using TTDN</td>
<td>N/A</td>
<td>N/A</td>
<td>ϰ, ICC</td>
<td>ϰ:0.56-0.90; ICC: 0.33-0.67</td>
<td>Moderate to high correlation</td>
</tr>
<tr>
<td>Dibai-Filho et al. (2015)</td>
<td>24 (1, 23)</td>
<td>Repeat intra-rater and inter-rater study</td>
<td>Cervical pain with active TrPs</td>
<td>Upper Traps</td>
<td>TB, TS, LTR, RP</td>
<td>Change in temperature using infrared thermography (point, line and area)</td>
<td>N/A</td>
<td>N/A</td>
<td>ICC, SEM, MDC</td>
<td>Intra-rater: ICC: 0.59-0.99; SEM: 0.13-1.57; MDC: 0.36-4.35 Inter-rater: ICC: 0.62-0.92; SEM: 0.43-1.22; MDC: 1.19-3.38</td>
<td>Moderate to high correlation</td>
</tr>
<tr>
<td>Zuil-Escobar et al. (2015)</td>
<td>164 (72, 92)</td>
<td>Cross-sectional interrater study</td>
<td>Patients with lowered MLA (n=82) control (n=82)</td>
<td>Gastroc, Soleus, PL, PB, EDL, FDL, Tib-Ant, VL, VM</td>
<td>TB, TS, LTR, RP, JS</td>
<td>PPT &gt;15 years'</td>
<td>N/A</td>
<td>ICC, ϰ</td>
<td>ICC: MLA group 0.84-0.92, control group 0.82-0.93; ϰ: MLA group 0.77-1.00, control group0.77-1.00</td>
<td>High correlation</td>
<td></td>
</tr>
<tr>
<td>Mora-Relucio et al. (2016)</td>
<td>52 (18, 34) each arm as separate cases</td>
<td>Observational cross-sectional study</td>
<td>Pianists: with LE (n=13), Subjects with pain in elbow (n=69), Asymptomatic (n=22)</td>
<td>Bilateral ECRB, EDC</td>
<td>TB, TS, LTR, RP, PR</td>
<td>TrP criteria (were TrP present) TrP location (TrPs within 1.50 cm). Total agreement (criteria and Agreement)</td>
<td>2 physiotherapists with 10 years' experience of TrPs; 1 physical therapist with no TrP experience</td>
<td>2X1 hour sessions to reach a consensus on the TrP criteria</td>
<td>ϰ</td>
<td>ECRB: between experts 0.55-0.62, between expert and inexperienced 0.00-0.39; EDC: between experts 0.42-0.59, between expert and inexperienced 0.12-0.30</td>
<td>Poor to moderate correlation</td>
</tr>
</tbody>
</table>

Where GlutMin is gluteus minimus, TB is taut band, TS is tender spot, RP is referred pain, PR is pain recognition, TTDN is thermo-vision technique of dry needling, ϰ is kappa score, ICC intra-class correlation, TrP is trigger points, Traps is trapezius, LRT is local twitch response, SEM is standard error measurement, MDC is minimal detectable change, MLA is medial longitudinal arch, JS is jump sign, Gastroc is gastrocnemius, PL is peroneus longus, PB is peroneus brevis, EDL is extensor digitorum longus, FDL is flexor digitorum longus, Tib-Ant is tibialis anterior, VL is vastus lateralis, VM is vastus medialis, LE is lateral epicondylalgia, ECRB is extensor carpi radialis brevis, EDC is extensor digitorum communis.
### Table 1: Summary of peer-reviewed literature in relation to the reliability of palpating trigger points

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</tr>
</thead>
<tbody>
<tr>
<td>Sanz et al. (2016)</td>
<td>40 (14, 26)</td>
<td>Interrater reliability study</td>
<td>Patients with RP to ankle (n=31), asymptomatic (n=9)</td>
<td>Tib-Ant, EDL, PB</td>
<td>TS in TB, + any 1 of: RP, LTR, JS</td>
<td>TrP critria (were TrP present)</td>
<td>Raters: 8-12 years’, Observers: 5-11 years’</td>
<td>8 hours practice. before testing</td>
<td>PA%, ϰ</td>
<td>TS in TB: Tib-Ant – 0.25-0.43, EDL – 0.25-0.60, PB - 0.24-0.47; RP: Tib-Ant – 0.26-0.51, EDL – 0.23-0.58, PB - 0.23-0.54; LTR: Tib-Ant – 0.05-0.23, EDL – 0.06-0.11, PB - 0.11-0.16; JS: Tib-Ant – 0.15-0.72, EDL – 0.25-0.58, PB - 0.19-0.41</td>
<td>Poor to moderate correlation</td>
</tr>
</tbody>
</table>

Where: RP is referred pain, Tib-Ant is tibialis anterior, EDL is extensor digitorum longus, PB is peroneus brevis, TS is tender spot, TB is taut band, RP is referred pain, LTR is local twitch response, JS is jump sign, PA% is percent agreement, ϰ is kappa score.
2.2.9.2 Reliability of the palpation of trigger points

There is a clear progression in the study design over the years which appears to have improved the reliability of TrP location. They are the class of reliability used for testing, the method of locating the TrPs, site of testing, subjects used, blinding the rater, experience of the rater, and statistics used to measure reliability.

2.2.9.2.1 Class of reliability

There are two types of reliability utilised to measure the accuracy of palpating TrPs: Inter-rater is assessing two or more testers’ ability to measure the same variable accurately; intra-rater reliability examines the reliability of the same tester to measure the same variable accurately over two or more sessions.

There appears to be a trend in the literature in relation to the reliability of locating TrPs. Older studies reported inter-rater reliability. Modern studies gravitate towards intra-rater reliability. Studies such as Nice et al. (1992) reported that the kappa coefficients (ϰ) for inter-rater reliability for confirming the presence of TrPs varied from ϰ 0.29 to 0.38; while Wolfe et al. (1992) reported significant differences (two-way ANOVA) for TS, TB, pain recognition (PR), and RP (p < 0.001). Nice et al. (1992) used subjects with long-standing lower back pain whereas Wolfe et al. (1992) compared patients with fibromyalgia or myofascial pain syndromes to asymptomatic subjects, both tested multiple sites. Some inter-rater studies incorporated asymptomatic subjects (Gerwin et al., 1997) and reported ϰ scores of 0.65 to 1.00.

Al-Shenqiti and Oldham (2005) was the first study to investigate the intra-rater reliability of TrPs and reported ϰ score of 0.75 to 1.00. Barbero et al. (2012) analysed the location of the TrPs using the anatomical landmark system (ALS) and reported moderate to high intra-class coefficient (ICC) of between 0.62 and 0.81. While O’Rourke (2010), Hynes (2011) and Barry (2015) reported ϰ scores of 0.90 to 0.93, 0.71 to 0.88, and 0.63 to 1.00, respectively. Therefore discrepancies in older studies may be due to poor study design where regional anatomy or the severity and complexity of the symptoms have not been considered. Intra-rater reliability would appear to deliver greater reliability as well as being more applicable to clinical settings. Using ratio data would mean that TrP location and severity can be measured with greater accuracy and repeatability.
2.2.9.2.2 The location of trigger points

It has been suggested that some reliability studies that locate TrPs by manual palpation lack conceptual clarity and inconsistent study design (Myburgh et al., 2008). A key component of treating TrPs, measuring the location of TrPs for subsequent treatment sessions (Sciotti et al., 2001). Many studies investigated the inter-rater reliability of experts to non-experts (Wolfe et al., 1992; Njoo and van der Does, 1994; Lew et al., 1997; Gerwin et al., 1997; Hsieh et al., 2000; Sciotti et al., 2001; Bron et al., 2007; Myburgh et al., 2011; Mora-Relucio et al., 2016) or between medical experience (Wolfe et al., 1992; Njoo and van der Does, 1994; Gerwin et al., 1997; Hsieh et al., 2000). Other studies have focused on the ability of practitioners to relocate TrPs (Sciotti et al., 2001; Al-Shenqiti and Oldham, 2005; Barbero et al., 2012), this perhaps is more practical, as it is imperative if a therapist is to treat the same TrP over multiple treatment sessions.

Lew et al. in 1997 attempted to measure the location of TrPs in the trapezius by recording the position on a 4:1 ratio body chart; while Al-Shenqiti and Oldham (2005) demonstrated a novel use of an acetate sheet to trace the position of TrPs in the rotator cuff muscles relative to two bony landmarks. Barbero et al. (2012) used the ALS to measure the location of TrPs in the upper trapezius. Where a line is drawn between two bony landmarks (C7 spinous process and acromion angle) called the ALS_d line. A perpendicular line is drawn from the TrP to the ALS_d line, called the Y-line. The shorter line (from C7 to the intersection of Y-line) is known as the X-line. Furthermore, Kennedy (2015), Devereux (2016) and Mora-Relucio et al. (2016) used ultraviolet (UV) ink and UV light to ensure observers remained blind.

More novel ways to objectively locate TrPs are beginning to emerge in the literature using infrared thermography techniques (Dibai-Filho et al., 2015; Skorupska et al., 2015) and three-dimensional measurements (Sciotti et al., 2001). Dibai-Filho et al. (2015) study reported moderate to excellent reliability with an ICC of 0.59 to 0.99. While Skorupska et al. (2015) reported poor to good reliability with an ICC of 0.44 to 0.78 whereas, Sciotti et al. (2001) stated an overall magnitude of standard error of the mean (SEM) of 3.30 to 6.60 cm between testers. These methods are expensive and are no more reliable than a trained therapist and may be better suited for use as a confirmatory tool.
The largest proportion of TrP reliability studies have been conducted in relation to the neck and shoulder (Wolfe et al., 1992; Gerwin et al., 1997; Lew et al., 1997; Sciotti et al., 2001; Al-Shenqiti and Oldham, 2005; Bron et al., 2007; Hynes, 2011; Barbero et al., 2012; Dibai-Filho et al., 2015), followed by the lower extremity (Njoo and van der Does, 1994; Hsieh et al., 2000; O’Rourke, 2010; Barry, 2015; Kennedy, 2015; Skorupska et al., 2015); with the rest assessing the lumbar region (Nice et al., 1992; Hsieh et al., 2000) and elbow (Mora-Relucio et al., 2016). Studies which investigates the reliability of locating TrPs of a plethora of muscle across multiple regions reported poor reliability (Nice et al., 1992; Wolfe et al., 1992; Gerwin et al., 1997; Hsieh et al., 2000), while studies measuring only one muscle reported better reliability (Myburgh et al., 2011; Barbero et al., 2012).

Al-Shenqiti and Oldham (2005) concurs with Gerwin et al. (1997) that reliability of locating TrPs is predominantly dependent on the muscle in which the TrP is located. The general TrP literature is slowly beginning to encompass the lower limb (Bajaj et al., 2001; Yentür et al., 2003; Itoh et al., 2008b; Mayoral et al., 2013; Roach et al., 2013; Cashman et al., 2014; Ichikawa et al., 2015; Alburquerque-García et al., 2015). This is mirrored in the increase of TrP reliability studies in relation to the lower limb. More studies should be carried out to establish the reliability of locating TrPs in the hip region (Njoo and van der Does, 1994; Hsieh et al., 2000; Kennedy, 2015; Skorupska et al., 2015), thigh and lower leg (O’Rourke, 2010; Barry, 2015). Assessment of the reliability of TrPs in these areas may help establish a greater understanding of the possible links to other pathologies such as osteoarthritis of the knee (Bajaj et al., 2001; Yentür et al., 2003; Itoh et al., 2008b; Mayoral et al., 2013) and patellofemoral pain syndrome (Roach et al., 2013; Espí-López et al., 2017).

Myburgh et al. (2008) suggest that the number of sites to be investigated should be minimised in order to prevent subject recall bias. In addition to this, testing of multiple sites may have an adverse impact on the application of the $\kappa$ score as it functions on the premise of independent observations (Vach, 2005). Therefore, future reliability studies testing for the location of TrPs in the lower extremity should consider reducing the number of sites tested to allow greater statistical clarity.
2.2.9.2.4 Subjects used
Studies which reported poor reliability used subjects with confounding pathologies. Nice et al. (1992) used subjects with long-standing lower back pain and Wolfe et al. (1992) investigated patients with fibromyalgia or myofascial pain. More reliable studies incorporated asymptomatic subjects (Gerwin et al. 1997). Therefore discrepancies in studies may have been due to regional anatomy or the severity and complexity of symptoms of other pathologies.

2.2.9.2.5 Blinding the rater in relation to the reliability of the palpation of trigger points
Blinding the rater in any study reduces the chance of bias (Myburgh et al., 2008). Of the intra-rater reliability studies that assessed TrP relocation (Al-Shenqiti and Oldham, 2005; O’Rourke, 2010; Hynes, 2011; Barbero et al., 2012; Barry, 2015; Kennedy, 2015), two studies attempted to blind the raters by comparing markings on acetate sheets between the two sessions (Al-Shenqiti and Oldham, 2005; Hynes, 2011); two compared digital images that were scaled using reference markers (O’Rourke, 2010; Barry, 2015); and one blindfolding the therapist whilst using the ALS (Barbero et al., 2012). Both Al-Shenqiti and Oldham (2005) and Barbero et al. (2012) used bony landmarks as references points for the relocation of TrPs. Having the therapist blindfolded prevented the therapist from fully assessing for the signs of TrPs such as JS and LTR (Barbero et al., 2012). Kennedy (2015) and Mora-Relucio et al. (2016) used UV ink to locate TrPs and measure the distance between TrPs (ΔTrP) under a UV light, thus still allowing the therapist to be blind to the location of TrP markings but still be able to use all the senses needed to locate a TrPs (Myburgh et al., 2008). Identifying a way to blind the therapist while allowing them to use their vision as part of the assessment is challenging but vital. Future studies should consider using UV ink and UV light in reliability testing.

2.2.9.2.6 Experience of the tester in relation to the reliability of the palpation of trigger points
There appear to be two factors which govern the reliability of practitioners: first is the experience of dealing with TrPs clinically or with additional training; the second may be the primary medical education of the tester. The studies that were found to be reliable had some form of additional training. The testers in the study completed by Gerwin et
al. (1997) reviewed TrP the night before and met 3 hours prior to the study. Bron et al. (2007) reported that the testers completed 8 hours of theory and practice as was well as reaching a consensus on all aspects of the study. Other reliable studies reported additional training in the form of 12 hours training or practice (Sciotti et al., 2001) and postgraduate course in locating TrPs (Barbero et al., 2012). The testers in the study by Myburgh et al. (2011) practised maintaining pressure for five minutes before each day of testing and found the inexperienced testers were not reliable, while some of the testers in the study conducted by Hsieh et al. (2000) were still unreliable following six hours of training. Nice et al. (1992) stated that 58.00% of the therapists in the study reported no experience in examining TrPs, but when factoring for correct technique, the $\kappa$ increased from 0.29 to 0.38 up to 0.35 to 0.46. Myburgh et al. (2011) reported a vast difference in paired agreement between experienced and non-experienced raters after 20, 40 and 60 cases, respectively; however, there was a similarly paired agreement after 81 cases. While training helps in the reliability of locating trigger points, there is no substitution for clinical experience (Hsieh et al., 2000).

The medical background of the tester may be a consideration when it comes to reliability. Wolfe et al. (1992) found that there were some discrepancies in the finding between the two examined groups. The rheumatologists identified tenderness in 42.30% of all sites whereas the myofascial pain syndromes experts identified 58.60%. A general practitioner and two medical students reported mixed results in relation to the inter-rater reliability of confirming the presence of TrP in the quadratus lumborum and gluteus medius for TB, ST, RP, JS, LTR and PR (Njoo and van der Does, 1994). Studies that were shown to have good reliability utilised testers that were physiotherapists or physical therapists (Al-Shenqiti and Oldham, 2005; Bron et al., 2007; Barbero et al., 2012). Studies that were less reliable completed testing with chiropractors (Hsieh et al., 2000) or rheumatologists (Wolfe et al., 1992). It would appear that in order to be reliable at locating and identifying TrPs, clinicians should have adequate education in musculoskeletal assessment, sufficient clinical practice specifically dealing with TrPs, as well as additional education in the location and treatment of TrPs.
2.2.9.2.7 Statistics used in relation to the reliability of the palpation of trigger points

The majority of studies examining the inter-rater reliability of identifying TrPs used nominal data, and were found to be not reliable or varied for inter-rater reliability (Nice et al., 1992; Wolfe et al., 1992; Njoo and van der Does, 1994; Lew et al., 1997; Hsieh et al., 2000; Bron et al., 2007; Myburgh et al., 2011; Mora-Relucio et al., 2016). Barbero et al. (2012) analysed the location of the TrPs with the ALS using ratio data and reported a moderate to high intra-class coefficient. The severity of a TrP can be measured using the PPT which used ratio data (Sciotti et al., 2001; Jones et al., 2007; Vanderweeën et al., 1996; Kennedy, 2015).

2.2.9.2.7.1 Statistics for reliability using nominal data

The $\kappa$ score is the most often used statistic in the literature in relation to inter-rater reliability (Nice et al., 1992; Njoo and van der Does, 1994; Hsieh et al., 2000; Bron et al., 2007) and intra-rater reliability (Al-Shenqiti and Oldham, 2005; O’Rourke, 2010; Hynes, 2011; Barry, 2015; Kennedy, 2015; Mora-Relucio et al., 2016). The $\kappa$ score is used to determine nominal or ordinal data in inter-rater and intra-rater reliability (Sim and Wright, 2005). Myburgh et al. (2008) suggest that testing multiple sites leads to patient recall bias and may not be the best application of the $\kappa$ statistical function, as the measurements are not independent observations. This could be addressed by reducing studies to one TrP site (Myburgh et al., 2011; Dibai-Filho et al., 2015). The $\kappa$ score uses arbitrary benchmarks: $\leq 0 =$ poor, 0.01-0.20 = slight, 0.21-0.40 = fair, 0.41-0.60 = moderate, 0.61-0.80 = substantial, and 0.81-1.0 = almost perfect (Sim and Wright, 2005). The literature shows a spectrum in reliability from slight (Hsieh et al., 2000) to almost perfect (Al-Shenqiti and Oldham, 2005). The differences within some studies are vast (Gerwin et al., 1997; Bron et al., 2007).

2.2.9.2.7.2 Statistics for reliability using ratio data

The recent intra-rater reliability studies used ratio data for the ALS (Barbero et al., 2012) and the $\Delta$TrPs (Barry, 2015) therefore the $\kappa$ score was not an appropriate statistical test. The ICC test is deemed a more appropriate statistical test (Chinn, 1990). Weir (2005) identifies three ICC models: one-way model for intra-class coefficient of reliability, two-way model for intra-class coefficient of reliability and mixed absolute model for intra-class coefficient of reliability.
class coefficient of reliability (ICC\(_{3,1}\)). The one-way model for intra-class coefficient of reliability has little clinical application as there is no observer variability or random error variability factored in; whereas the two-way model for intra-class coefficient of reliability does factor in observer variability which would be more applicable to reliability studies which have multiple observers. The ICC\(_{3,1}\) would be most appropriate where there is one observer, and random error variability is expected (Rankin and Stokes, 1998). While there are attempts to classify the ICC (Barbero et al., 2012; Dibai-Filho et al., 2015; Skorupska et al., 2015) these, like \(\kappa\) classification, are arbitrary (Weir, 2005). One such classification is \(\leq 0.40 = \) poor, \(0.41-0.75 = \) moderate, \(\geq 0.76 = \) excellent (Doğan and Doğan, 2015). Barbero et al. (2012) suggest that an ICC of 0.60 is clinically significant. Weir (2005) states that as well as stating the model type the SEM should be reported with any ICC, while 95% confidence intervals (95% CI) should also be reported (Atkinson and Nevill, 1998; Batterham and George, 2000).

2.2.9.2.7.3 Statistics for agreement using ratio data

Kottner et al. (2011) suggest that to improve the reporting of reliability studies agreement should also be reported. Agreement for nominal data is limited to a percent of agreement. In the literature percent agreement has been varied, 33-100% for the presence of local twitch response (Bron et al., 2007) or heterogeneous, 76-79% for the presence of TrPs (Nice et al., 1992). There are several methods to report agreement, in relation to ratio data (Atkinson and Nevill, 1998; Rankin and Stokes, 1998; Kottner et al., 2011b). These include percent of agreement, SEM (Weir, 2005), coefficients of variance (CV), limits of agreement (LoA) and Bland-Altman plots (Atkinson and Nevill, 1998). Rankin and Stokes (1998) suggest that CV is an outdated form of reporting agreement and offer the Bland-Altman plot as a feasible option to visually interpret the agreement of within-subject variances. It may be a possible disadvantage as it is more complex to interpret than the classifiable ICC. However, they also suggest that it could also be regarded as a possible advantage. The need to identify the correct statistical analysis is imperative to improve the reporting of agreement and thus allow better interpretation of the findings.
2.2.9.2 Summary of the statistics used in the reliability of the palpation of trigger points

While there are advantages and disadvantages to using certain statistical theorems, the consensus is that no one analysis is perfect and that the multiple statistical tests are preferable. Intra-rater reliability would appear to deliver greater reliability as well as being more applicable to clinical settings. The ALS and PPT may be more applicable in a clinical environment where it would be useful to measure the location and severity of TrPs in order to document patient’s progression.

2.2.9.3 Summary of the reliability of the palpation of trigger points

The literature in relation to assessing the reliability and agreement of locating TrPs goes back over 20 years mostly focusing on the neck and shoulders. Studies are beginning to shift to the lower limb especially in relation to latent TrPs. The ability to reliably locate TrPs has improved, but there is still no comprehensive method to locate TrPs. To date reliability of confirming the presence of TrPs has ranged from poor to excellent, using nominal data; whereas it ranges from good to excellent for ratio data. Studies should consider reducing the site tested for the relocation of TrPs to reduce subject recall bias. Sites should include muscles that may have a relationship to other pathologies such as the quadriceps in relation to knee osteoarthritis. Studies should focus on relocating TrP using ratio data such as the ALS and include the PPT. Blinding the tester is inherently difficult, particularly when the tester requires sight to locate TrPs, but can be achieved when using UV ink and UV lamps. Future studies should use more robust statistical analysis such as ICC, with details of the statistical model used; SEM and Bland-Altman plots.

2.3 Outcomes

Active TrPs are spontaneously painful (Bron and Dommerholt, 2012). The visual analogue scale (VAS) is the outcome most often used to measure TrPs in the lower limb (Christensen et al., 1992; Tillu et al., 2001, 2002; Williamson et al., 2007; Itoh et al., 2008a, 2008b; Suarez-Almazor et al., 2010; Mavrommatis et al., 2012; Mikashima et al., 2012; Soni et al., 2012; Mayoral et al., 2013; Ashraf et al., 2014; Pavkovich, 2015; Pecos-Martín et al., 2015; Ortega-Cebrian et al., 2016). The VAS is a reliable measure of acute pain when used in an emergency room setting to describe pain, with an ICC of 0.97 (Bijur
et al., 2001). Therefore logically, the VAS is not an appropriate measurement for latent TrPs, as the definition of latent TrPs is that they are not spontaneously painful. Thus there is a need to quantify the severity of latent TrPs when being palpated. The PPT offers this.

### 2.3.1 Pressure pain threshold

The PPT is used as a clinical measure to quantify the pressure applied by a therapist during palpation using an algometer, sometimes called algometry (Vanderweeën et al., 1996; Mutlu and Ozdincler, 2015; Wytrążek et al., 2015). The PPT can be used to measure the sensitivity of muscular (Wytrążek et al., 2015; Ziaeifar et al., 2014; Gulick, 2014; Maquet et al., 2004), tendon (Smidt et al., 2002), or articular (Mutlu and Ozdincler, 2015) based pathologies. The PPT is the steady application of increasing pressure until the subject reports a change of a feeling of pressure in a sensation of pain. TrPs are the most common pathology which uses the PPT as an outcome measure (Vanderweeën et al., 1996; Chesterton et al., 2003; Lluch et al., 2013). As there is just enough pressure applied to produce pain, there is a reduced chance of sensitisation of the TrP. An increase of the PPT would suggest that there is an improvement in the sensitivity of TrP, as it takes more pressure to induce a reaction of sensitive foci (Lee et al., 2008). The PPT allows therapists to monitor the progression of a treatment modality and assesses improvement in a pathology (Ziaeifar et al., 2014). Pain sensitivity measured by the PPT in the VL is associated with weakness during isometric knee extension \((p = 0.04)\) in healthy subjects when adjusted for gender, age, and body mass using multivariate regression analysis (Henriksen et al., 2013).

There are several version of algometers such as; analogue manual, where there are sensors attached to a spring or hydraulic gauges, digital manual, where there is a digital readout, but pressure is applied by a clinician via the pressure sensor and computerised, where there is pressure applied thru a sensor that is controlled by computer. A digital algometer with manual pressure may be the most appropriate, as it can be guided by a skilled clinician using palpation.

The reliability of the pressure pain threshold
The primary criteria for the presence of TrPs are TS in a palpable TB and PR (Hsieh et al., 2000; Bron et al., 2007). The use of algometry and the PPT in relation to TrPs date back to the late eighties (Fischer, 1988) and are still routinely being used in TrP research (Aranha et al., 2011; Cantarero-Villanueva et al., 2012). A summary of the peer-reviewed literature in relation to the PPT reliability is presented in Table 2 p.37).
Table 2: Summary of peer-reviewed studies in relation to the reliability of the pressure pain threshold of trigger points

<table>
<thead>
<tr>
<th>Author (Date)</th>
<th>n = (male, female)</th>
<th>Subjects (age)</th>
<th>Muscles tested</th>
<th>Methods</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jensen et al. (1986)</td>
<td>24 (12, 12)</td>
<td>Healthy (39 [range 24-69])</td>
<td>Temporalis</td>
<td>Part of a 4 study series. 2 sessions, 3 (range 1-6) days apart</td>
<td>CC</td>
<td>Intra-rater CC: 0.77</td>
<td>Moderate</td>
</tr>
<tr>
<td>Ohrbach and Gale (1989a)</td>
<td>45 (5, 40)</td>
<td>Patients with temporomandibular disorders (39 ± 15)</td>
<td>Posterior temporalis</td>
<td>2 session (time between sessions N/A) with 5 sec between PPT tests</td>
<td>Mixed ANOVA, ρ, ϰ</td>
<td>ANOVA: within subjects - no difference; between genders - difference. ρ : 0.83; ϰ: 0.62</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Ohrbach and Gale (1989b)</td>
<td>10 (5, 5)</td>
<td>Healthy (N/A)</td>
<td>Masseter, anterior temporalis</td>
<td>Study 1: 2 sessions, 4-5 min apart with 5 sec between PPT tests Study 2: 4 sessions, 1 week apart + 1 session 4 weeks after week 4 with 5 sec between PPT tests</td>
<td>Mixed ANOVA</td>
<td>Masseter no difference; Anterior temporalis no difference</td>
<td>No difference</td>
</tr>
<tr>
<td>Chung et al. (1991)</td>
<td>40 (19, 21)</td>
<td>Healthy (23 [range 20-27])</td>
<td>Masseter, temporalis, pterygoid, digastric, StcmCM, Sp-Cap, Traps</td>
<td>Intra-rater &amp; inter-rater: 3 sessions on consecutive days (days 1 &amp; 2 1st examiner, day 3 2nd examiner). PPT tests were at least 5 min apart</td>
<td>CC</td>
<td>Intra-rater CC (male, female): Masseter (0.82-0.83, 0.79-0.92), Temporalis (0.67-0.78, 0.7.0-0.80), Pterygoid (0.42, 0.76), Digastic (0.59, 0.80), STM (0.55-0.72, 0.70-0.81), Sp-Cap (0.78, 0.83), Traps (0.48-0.82, 0.67-0.72), Inter-rater CC (male, female): 0.28-0.85, 0.47-0.77</td>
<td>Slight to high</td>
</tr>
<tr>
<td>Delaney and McKee (1993)</td>
<td>50 (25, 25)</td>
<td>Healthy (30, [range 20-51])</td>
<td>M-Traps, L-Traps</td>
<td>Inter-class, 2 sessions (time between sessions N/A) with 5 min between PPT tests</td>
<td>Mixed ANOVA, ICC</td>
<td>Intra-rater ICC: M-Traps: 0.80-0.83; L-Traps: 0.91. Inter-rater ICC: M-Traps: 0.82-0.92; L-Traps: 0.86-0.92</td>
<td>High</td>
</tr>
</tbody>
</table>

Where CC is correlation coefficient, PPT is pressure pain threshold, ANOVA is an analysis of variance, ρ is Pearson’s rho, ϰ is kappa score, Stcm is sternocleidomastoid, Sp-Cap is splenius capitis, Traps is trapezius, M-Traps is middle trapezius, L-Traps is lower trapezius, ICC is intra-class coefficient.

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Table 2: Summary of peer-reviewed studies in relation to the reliability of the pressure pain threshold of trigger points

<table>
<thead>
<tr>
<th>Author et al. (Date)</th>
<th>n = (male, female)</th>
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<tbody>
<tr>
<td>Kosek et al. (1993)</td>
<td>12 (0, 12)</td>
<td>28 (15-33)</td>
<td>Lev-Scap, Supra, Infra, Braciorad, GlutMed</td>
<td>Intra-class: 2 sessions 1 week apart with 3-10 s between PPT tests</td>
<td>3-way ANOVA, CV</td>
<td>CV: Lev-Scap 36.1-55.8%, Supra 38.8-46.4%, Infra 59.5%, Braciorad 62.7%, GlutMed 45.7-48%</td>
<td>Moderate</td>
</tr>
<tr>
<td>Vanderweeën et al. (1996)</td>
<td>30 (15, 15)</td>
<td>Patients with pain in shoulder and arm (age not specified)</td>
<td>Pec, Supra, Infra, Traps, ECRB, Inteross (C2-T1 segmental control)</td>
<td>Intra-class: 2 sessions 5 min apart. The time between PPT tests N/A</td>
<td>1-way ANOVA, ICC Rotated factor matrix</td>
<td>ICC 0.64-0.96 (muscles not specified). Gender difference</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Isselée et al. (1997)</td>
<td>22 (11, 11)</td>
<td>Healthy [male 27 [range 21-35], female [24 [range 21-34]]</td>
<td>Masseter and temporalis</td>
<td>4 sessions 2 pairs of sessions 6-8 hours apart (1 &amp; 2, and 3 &amp; 4); 2 days between the pairs of the sessions (2 and 3)</td>
<td>Variance matrix (Subject, time, day)</td>
<td>Masseter: Subject 2.3-7.8; time/day 0.5-2.4. Temporalis: Subject 3.4-9.0; time/day 0.7-3.0</td>
<td>Moderate</td>
</tr>
<tr>
<td>Sand et al. (1997)</td>
<td>30 (7, 23)</td>
<td>Control [37 [range 23-53]], tension-type headache patients [41 [range 21-56]], Cervicogenic headache patients [38 [range 21,52]]</td>
<td>Temporal, Stm-i, Traps-i</td>
<td>Intra-rater: 3 sessions 19 days between session 1 and session 2, 33 days between session 2 and session 3. ‘a few min’ between PPT tests</td>
<td>CR, ICC</td>
<td>Between days (CR, ICC): Temporal: 13.7-24.5N, 0.70-0.73; STM: 17.7N, 0.63; Traps: 20.6N, 0.78</td>
<td>Moderate</td>
</tr>
</tbody>
</table>

Where Lev-Scap is levator scapulae, Supra is supraspinatus, Infra is infraspinatus, Braciorad is brachioradialis, GlutMed is gluteal medialis, PPT is pressure pain threshold, ANOVA is analysis of variance, CV is coefficient of variance, Pec is pectoralis major, ECRB t is extensor carpi radialis brevis, Inteross is first dorsal interosseous, C(n) is cervical level, T(n) is thoracic level, ICC is intra-class coefficient Stcm(-i) is sternocleidomastoid (insertion), Traps(-i) is trapezius (insertion), CR is the coefficient of repeatability.
Table 2: Summary of peer-reviewed studies in relation to the reliability of the pressure pain threshold of trigger points

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<th>Author (Date)</th>
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</thead>
<tbody>
<tr>
<td>Antonaci et al. (1998)</td>
<td>21 (15, 6)</td>
<td>Healthy [29 ± 12].</td>
<td>Temporal, frontal, Stcm-i, Traps-i, masseter Delts</td>
<td>Intra-rater &amp; inter-rater. 3 sessions 2-3 min apart. The time between PPT tests N/A</td>
<td>ICC, CV</td>
<td>Reliability (ICC, CV) Temporal (0.74-0.77, 17.9-19.8%) Frontal (0.76, 15.7%) SCM-i (0.74, 21.4%) Traps-i (0.70-0.91, 20.7-13.0%) Masseter (0.61, 21.2%) Delts (0.78, 18.3%)</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Nussbaum and Downes (1998)</td>
<td>35 (5, 30)</td>
<td>Healthy ([range 29 ± 15], [range 36 ± 15])</td>
<td>Biceps</td>
<td>Inter-class &amp; intra-rater, 3 sessions 24 hours apart. 10 sec between PPT tests</td>
<td>ICC</td>
<td>Single trial intra-rater ICC: 0.74-0.89; Mean of multiple trials intra-rater ICC: 0.81-0.88</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Persson et al. (2000)</td>
<td>25 (0, 25)</td>
<td>Healthy [44 (range 21-61)]</td>
<td>Traps, Delts</td>
<td>Intervention study, effects of bilateral fatigue in EMG and PPT: outcome measure at B-Line, pre-test, post-test, 15 sec post-test, 10 min after post-test. The time between B-Line and pre-test was 10 min</td>
<td>ICC, paired t-tests</td>
<td>0.81-0.94. Muscle and side not specified</td>
<td>High</td>
</tr>
<tr>
<td>Sterling et al. (2002)</td>
<td>38 (13, 25): (n=19) Asymptomatic (7, 12); (n=19) patients with chronic neck pain (32 ± 12)</td>
<td>Asymptomatic (30 ± 11), patients with chronic neck pain (32 ± 12)</td>
<td>Para-spines muscle at C2/C3, C5/C6, Tib-Ant</td>
<td>Intra-class: 2 sessions 1 week apart. The time between PPT tests N/A</td>
<td>ICC, SEM, repeat ANOVA</td>
<td>Asymptomatic ICC: C2/C3 0.80; C5/C6 0.80-0.91; Tib-Ant 0.79-0.80. Symptomatic ICC: C2/C3 0.72-0.89; C5/C6 0.88-0.92; Tib-Ant 0.81-0.92</td>
<td>Moderate to high</td>
</tr>
</tbody>
</table>

Where Stcm(-i) is sternocleidomastoid (insertion), Traps(-i) is trapezius (insertion), Delts is deltoids, PPT is pressure pain threshold, ICC is intra-class coefficient, CV is coefficient of variance, Biceps is biceps brachii, EMG is electromyography, B-Line is Baseline, Cn is cervical level, Tib-Ant is tibialis anterior, SEM is standard error of the measurements, ANOVA is an analysis of variance,
### Table 2: Summary of peer-reviewed studies in relation to the reliability of the pressure pain threshold of trigger points

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</tr>
</thead>
<tbody>
<tr>
<td>Persson et al. (2003)</td>
<td>17 (0, 17)</td>
<td>Patients with shoulder pain (47 [range 24-62])</td>
<td>Traps, Deltis, Quads</td>
<td>Intervention study, effects of unilateral fatigue (affected side) on EMG and PPT: outcome measure at B-Line, pre-test, post-test, 15 sec post-test, 10 min post-test, 15 min post-test. Time between PPT test N/A</td>
<td>ICC, paired t-tests</td>
<td>Intra class between B-Line, pre-test (affected side, unaffected side): Traps (0.83, 0.85); Deltis (0.86, 0.88); Quads (0.53, 0.72)</td>
</tr>
<tr>
<td>Persson et al. (2004)</td>
<td>24 (0, 24)</td>
<td>Healthy (42 [range 29-59])</td>
<td>Traps, Deltis</td>
<td>Intra-class: 4 sessions (days 1, 3, 28, 30). 10 min between PPT tests</td>
<td>1-way ANOVA, ICC</td>
<td>Within session reliability (R, L); Traps - day 1 (0.86, 0.86); day 3 (0.84, 0.88); day 28 (0.89, 0.90); day 30 (0.90, 0.92). Deltis - day 1 (0.85, 0.85); day 3 (0.90, 0.90); day 28 (0.91, 0.94); day 30 (0.86, 0.92)</td>
</tr>
<tr>
<td>Ylinen et al., 2007</td>
<td>20 (0, 20)</td>
<td>Patients with neck pain (47 ± 5)</td>
<td>Lev-Scap, Sp-Cap, Traps</td>
<td>Intra-class: 2 sessions 1 day apart. 30 seconds between PPT tests</td>
<td>ICC, CV</td>
<td>Reliability (ICC [R, L], CV [R, L]); Lev-Scap ([0.78, 0.91], [11, 10%]); Sp-Cap ([0.93, 0.84], [15, 20%]); Traps ([0.86, 0.85], [18, 22%])</td>
</tr>
<tr>
<td>Potter et al. (2006)</td>
<td>10 (5, 5)</td>
<td>Healthy (age not specified)</td>
<td>Bilateral Ilioc, Multif, GlutMax, Traps</td>
<td>Inter-class &amp; intra-rater, 3 sessions 5 min apart. The time between PPT tests N/A</td>
<td>ICC, SEM, SDD</td>
<td>Intra-rater within session: Ilioc: 0.80 ± 0.85; Multif: 0.90-0.92; GlutMax: 0.91-0.93; Traps: 0.84-0.96; Pooled: 0.91-0.93. Intra-rater between session: 0.87-0.96</td>
</tr>
<tr>
<td>Chesterton et al. (2007)</td>
<td>13 (12, 1)</td>
<td>Students (22 [range 20-29])</td>
<td>Inteross</td>
<td>Inter-rater: 2 sessions 10 min between sessions. 15 sec between PPT tests</td>
<td>Repeat ANOVA, ICC, SEM</td>
<td>ICC: 0.90</td>
</tr>
</tbody>
</table>

Where Traps is trapezius, Deltis is deltoids, Quads is quadriceps femoris, EMG is electromyography, PPT is pressure pain threshold, B-Line is Baseline, ICC is intra-class coefficient, ANOVA is an analysis of variance, Lev-Scap is levator scapulae, Sp-Cap is splenius capitis, CV is coefficient of variance, Ilioc is illocostal, Multif is multifidus, GlutMax is gluteus maximum, SEM is standard error of the mean, SDD is smallest detectable difference, Inteross is first dorsal interosseous.
Table 2: Summary of peer-reviewed studies in relation to the reliability of the pressure pain threshold of trigger points

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<th>Author (Date)</th>
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<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jones et al. (2007)</td>
<td>19 (0, 19)</td>
<td>Healthy (24 ± 5)</td>
<td>Biceps, Ant-Delt, Triceps, Rhomb, Post-Delt, Supra, Traps, Infra</td>
<td>Intra-rater: 4 sessions 24 hours apart. Time between PPT tests N/A</td>
<td>Repeat ANOVA, ICC</td>
<td>ICC: Biceps: 0.96-0.97; Ant-Delt: 0.96-0.98; Triceps: 0.96-0.99; Rhomb: 0.92-0.98; Post-Delt: 0.94-0.98; Supra: 0.90-0.96; Traps: 0.92-0.97; Infra: 0.93-0.97</td>
<td>High</td>
</tr>
<tr>
<td>Park et al. (2011)</td>
<td>221 (113, 108)</td>
<td>Office workers (43 ± 6)</td>
<td>Bilateral Traps, Infra, ECR, Ext-Ind</td>
<td>Intra-rater: 2 session 5 min apart. The time between PPT tests N/A</td>
<td>α</td>
<td>Traps: 0.94, 0.98; Infra: 0.93, 0.96; Ext: 0.96, 0.98; Ext-ind: 0.94, 0.97</td>
<td>High</td>
</tr>
<tr>
<td>Koo et al. (2013)</td>
<td>16 (8, 8)</td>
<td>Healthy (26 ± 4)</td>
<td>Erector spine</td>
<td>Intra-rater: 2 session 5 min apart with 45 sec between PPT tests (manual PPT Vs computerised PPT Vs computerised PDT)</td>
<td>1-way ANOVA, ICC SEM, MDC CV</td>
<td>Within day (ICC, CV). manual PPT: 0.91, 10.3 %; computerised PPT: 0.87, 15.6 %; computerised PDT: 0.86, 20.5 %</td>
<td>High</td>
</tr>
<tr>
<td>Ziaeifar et al. (2014)</td>
<td>33 (gender not specified)</td>
<td>TrP patients (26 ± 4)</td>
<td>Upper Traps</td>
<td>Intervention study: DN Vs compression therapy. Outcomes measured after treatment sessions (3 sessions per week for 1 week)</td>
<td>ICC, ANOVA</td>
<td>ICC: 0.92</td>
<td>High</td>
</tr>
</tbody>
</table>

Where Biceps is biceps brachii, Ant-Delt is anterior deltoid, Triceps is triceps brachii, Rhomb is rhomboids, Post-Delt is posterior deltoid, Supra is supraspinatus, Infra is infraspinatus, PPT is pressure pain threshold, ANOVA is analysis of variance, ICC is intra-class coefficient, ECR is extensor carpi radials, Ext-Ind is extensor indicis, α is Cronbach’s alpha reliability coefficient, PDT pressure discomfort threshold, SEM is standard error of the mean, MDC is minimal detectable change, CV is coefficient of variance, DN is dry needling.
The PPT is a reliable measure for symptomatic subjects (Ohrbach and Gale, 1989a; Vanderweeën et al., 1996; Sand et al., 1997; Ziaeifar et al., 2014) and healthy subjects (Ohrbach and Gale, 1989b; Delaney and McKee, 1993; Nussbaum and Downes, 1998; Potter et al., 2006; Chesterton et al., 2007; Jones et al., 2007; Park et al., 2011; Koo et al., 2013; Ziaeifar et al., 2014) with ICCs ranging from 0.63-0.99. The studies that investigated the reliability of the PPT have been predominantly in the upper limb (Delaney and McKee, 1993; Vanderweeën et al., 1996; Nussbaum and Downes, 1998; Potter et al., 2006; Chesterton et al., 2007; Jones et al., 2007; Park et al., 2011; Koo et al., 2013; Ziaeifar et al., 2014) and the head (Ohrbach and Gale, 1989a, 1989b; Sand et al., 1997), with only two peer-reviewed studies investigating the PPT in the gluteus maximus muscle of the lower limb (Sciotti et al., 2001; Potter et al., 2006).

One peer-reviewed study (Sciotti et al., 2001) that investigated the reliability of TrP location using three-dimensional imaging, reported that a PPT of less than 34.3 N is enough to stimulate a TrP in asymptomatic subjects; whereas Haddad et al. (2012) found that the mean PPT of the masseter and the temporal muscles in symptomatic patients was 15.10 to 16.30 N. Kennedy (2015) performed a reliability trial using PPT on the piriformis and gluteus muscles in subjects with latent TrPs and reported a PPT of 40.21 to 46.58 N in these muscles.

One of the limitations noted by Barbero et al. (2012) in their study was that they did not measure the pressure required to induce pain. This is further endorsed as a recommendation in the systematic review by Myburgh et al., (2008). There are studies that endeavour to establish the intra-rater reliability of locating TrPs (Sciotti et al., 2001; O’Rourke, 2010; Hynes, 2011; Barbero et al., 2012; Barry, 2015; Kennedy, 2015); there are also studies that aim to identify the reliability of using the PPT as a measure of severity of TrPs (Jones et al., 2007; Gulick et al., 2011; Kennedy, 2015), and use the PPT to define the presence of a TrP (Sciotti et al., 2001). However, there is no reliability study that uses the PPT as a criterion for the presence of TrPs or as an outcome measure for the severity of TrPs in conjunction with an accurate method to plot the location of TrPs. There is a clear gap in the literature in relation to the PPT in the lower limb. Future studies must consider measuring the location of the TrP together with the PPT. The PPT can be used as a tool to record the severity of TrPs especially if it is used in conjunction
with a means to measure the location of the TrPs accurately to improve the continuum of care for a patient when recording patient’s clinical notes (Weiss et al., 2009).

2.3.1.1 The pressure pain threshold protocol
The PPT is often used as a clinical outcome in studies in relation to TrP (Cantarero-Villanueva et al., 2012; Sharan et al., 2014; Chan et al., 2015) and also when DN is used as a treatment methods (Srbely et al., 2010; Ziaeifar et al., 2014; Koppenhaver et al., 2015; Abu-Taleb et al., 2016). In the literature it has been common practice to record the mean of three PPT tests with either five seconds (Ohrbach and Gale, 1989a, 1989b; Delaney and McKee, 1993), ten seconds (Nussbaum and Downes, 1998), 15 seconds (Chesterton et al., 2007), and up to 45 seconds (Koo et al., 2013) being left between each recording. There may be some treatment effect when using the PPT, especially if this outcome measure is taken in a short period of time, but is not enough to have a significant effect (Jones et al., 2007).

2.3.2 Summary of the pressure pain threshold
The PPT utilises algometry which can augment and measure the palpation of a skilled clinician. It can be highly reliable and is often used as an outcome for TrPs. There may be some treatment effect, but these are negligible even if the PPT is utilised in a short timeframe. Three PPT tests should be used. Five seconds between tests is enough to obtain a useable reading without affecting subsequent readings.

2.3.3 Jumping
There are two types of jumps used throughout the literature, (Maulder and Cronin, 2005; Moir et al., 2008; Bubanj et al., 2010; Casartelli et al., 2010; Glatthorn et al., 2011; Asadi, 2012; Byrne et al., 2014; Chelly et al., 2015). They are the CMJ and the DJ. Jump training falls within a type of exercise known as plyometrics. They can be performed at any levels of fitness. Plyometrics are designed to enhance neuromuscular performance by utilising the stretch-shortening cycle to ultimately improve jump performance (de Villarreal et al., 2010). The stretch-shortening cycle is characterised by an intense eccentric contraction followed by a rapid concentric contraction, with an interceding amortisation phase (Robinson et al., 2004). The CMJ uses a slow stretch-shortening cycle, whereas the DJ uses a faster stretch-shortening cycle (Meylan et al., 2011). There
are two proposed models of the effectiveness of the stretch-shortening cycle. They are mechanical and neurophysiological (Potach and Chu, 2008). The mechanical model hypothesis the utilisation of elastic structures which stores energy on landing. If there is an immediate muscular contraction the stored kinetic energy adds to the total force, if the contraction is not immediate then the energy dissipates in the form of heat; the neurophysiological model hypothesis propositions that the muscle spindles stretch reflex is involved. If a voluntary movement contraction and reactive contraction occur simultaneously, there should be an increase in the total force. Both models may be likely. Training which targets the stretch-shortening cycle is believed to have a positive effect on neuromuscular function and is probably due to 1) an increased neural drive to the agonist muscles, 2) changes in the muscle activation strategies and intermuscular coordination, 3) variations in the mechanical characteristics of the muscle-tendon complex, 4) changes in muscle size and or architecture, and 5) changes in single-fibre mechanics (Markovic and Mikulic, 2010). To summarise, jump training utilises the stretch-shortening cycle and is highly effective at improving performance. However, more research is needed to discern the exact mechanisms involved in the stretch-shortening cycle.

2.3.3.1 Measuring jump height

The jump height \( JH \) for the CMJ and DJ is not directly measured. \( JH \) is estimated in a number of ways depending on the device used to measure the jump; they are flight time \( t_f \), acceleration, velocity, or force. Each method uses equations based on variations of ballistic laws (Linthorne, 2001; Markovic et al., 2004).

\[
JH = t_f^{-1} \cdot g^{-1} \cdot 8^{-1}
\]

Equation 1:
Jump height derived from flight time.

where \( JH \) is jump height \( (m) \), \( t_f \) is flight time \( (s) \), \( g \) is acceleration due to gravity \( (9.81 \, m \cdot s^{-1}) \).

\[
JH = v^2 \cdot (2g)^{-1}
\]

Equation 2:
Jump height derived from velocity.
where \( JH \) is jump height (m), \( v \) is maximum vertical velocity (m\( \cdot \)s\(^2\)), \( g \) is acceleration due to gravity (9.81 m\( \cdot \)s\(^{-1}\)).

\[
JH = s_i + \left( v_i + \left( \frac{F_i}{m} - g \right) \cdot t_i \right) \cdot t_f + \left( \frac{F_i}{m} - g \right) \cdot t_i^2
\]

Equation 3: Jump height derived from initial force.

where \( JH \) is jump height (m), \( s_i \) is position of centre of gravity at \( t_i \) (m), \( v_i \) is velocity at \( t_i \) (m\( \cdot \)s\(^2\)), \( F_i \) is force at \( t_i \) (N), \( m \) is mass of jumper, \( g \) is acceleration due to gravity (9.81 m\( \cdot \)s\(^{-1}\)), \( t_i \) is 0.001 (s), \( t_f \) is flight time (s).

The height of the CMJ and DJ are measured using a variety of devices such as jump mats, photocell timing devices, force platforms, and accelerometers. All of which are highly reliable.

2.3.3.2 Jump mats
Jump mats use pressure switches built into a light weight mat to measure the time that there is no contact time, as an approximation of \( t_f \) (Arteaga-Ortiz et al., 2000; Markovic et al., 2004; Moir et al., 2004; Maulder and Cronin, 2005; Leard et al., 2007; Moir et al., 2008; Slinde et al., 2008; Acero et al., 2011; Nuzzo et al., 2011; Farias et al., 2013). Jump mats are portable and are ideal for use in field studies. Jump mats cannot measure the force developed when jumping or landing and therefore cannot offer variables such as force or power.

2.3.3.3 Photocell timing devices
Photocell timing devices use beams of infrared light between two banks of photosensitive cells to measure the non-contact time (Enoksen et al., 2009; Sassi et al., 2009; Glatthorn et al., 2011; Castagna et al., 2013). Photocell timing devices are lightweight and portable but need to be set up on a flat surface such as an indoor court. As with the jump mat, force and power cannot be measured with photocell timing devices.

2.3.3.4 Force platforms
Force platforms, or plates, use highly sensitive force sensors and measure the \( t_f \) using force equations, such as Equation 3 (p 45). Any deviation from vertical can be noted using ground reaction force data as well as peak force, peak power, and velocity.
(Maulder and Cronin, 2005; Cormack et al., 2008; Slinde et al., 2008; Enoksen et al., 2009; Hori et al., 2009; Ditroilo et al., 2011; Glatthorn et al., 2011; Castagna et al., 2013; Mizuguchi et al., 2015). FPs are considered the gold standard for measuring jump performance (Leard et al., 2007; Enoksen et al., 2009; Bubanj et al., 2010; Casartelli et al., 2010; Glatthorn et al., 2011; Castagna et al., 2013). They are however expensive and limited to laboratory settings.

2.3.3.5 Wireless accelerometry

Wireless accelerometers use miniature accelerometers and gyroscopes similar to those found in smart mobile telephones. They can measure acceleration and velocity (Bubanj et al., 2010; Casartelli et al., 2010; Nuzzo et al., 2011; Castagna et al., 2013). Wireless accelerometers are extremely portable and can be used to capture power variables during a dynamic task such as running (Gindre et al., 2015).

2.3.4 Summary of devices used to measure height

There are several methods of measuring the performance of the CMJ and DJ, utilising a myriad of calculations to determine the $JH$. All of these have been shown to be reliable. The choice of apparatus would depend on the location which the testing is to take place.

2.3.5 Inter-device validity

With the variety of devices used to measure jump performance, directly and indirectly, there has been a need to determine the validity the reliability between the different devices. Maulder and Cronin (2005) reported that there was a near perfect reliability between the jump mat and the FP for measuring $JH$ (ICC: 0.95) and $t_f$ (ICC: 0.99); whereas Enoksen et al. (2009) found that there was a marked bias (2.8 cm) towards the FP compared to the PTD. The bias was accredited to knee flexion during landing or assumptions made into relation to ballistic calculations. There was no difference between the FP compared to the PTD in the study by Glatthorn et al. (2011), who reported a concurrent validity of $> 0.99$ (ICC). While Nuzzo et al. (2011) also reported high inter-device reliability between jump mat, wireless accelerometer, or analogue jump vanes (ICC: 0.887-0.95).
2.3.6 Summary of measures of jump height
The gold standard for measuring jump height is a FP. Some devices are unable to process data in real-time. Therefore using a timing device simultaneously with the FP such as PTD would allow verification of a portion of a jump, such as the contact phase of the depth jump. All of the devices used to measure jump performance are reliable. However, there may be some discrepancies between devices. Therefore, caution is needed in interoperating the results and using the data between devices interchangeably should be avoided.

2.3.7 Countermovement jump
The CMJ is often used synonymously with the vertical jump and is used to measure strength, power, and performance (Maulder and Cronin, 2005; Moir et al., 2008; Bubanj et al., 2010; Casartelli et al., 2010; Glatthorn et al., 2011). The vertical jump is a vital physiological component of many sports (Castagna et al., 2013) such as diving and volleyball (Leard et al., 2007), Australian rules football (Cormack et al., 2008), basketball (Casartelli et al., 2010; Rodriguez-Rosell et al., 2016), football (Casartelli et al., 2010), and rugby (Castagna et al., 2013; Roe et al., 2016). The CMJ is also used as a performance and functional outcome in non-elite subjects (Slind et al., 2008; McMahon et al., 2016; Stanton et al., 2017), school children (Acero et al., 2011), and the elderly (Ditroilo et al., 2011; Farias et al., 2013). The CMJ has also been used to measure neuromuscular fatigue, a limiting factor in performance (Cormack et al., 2013). The CMJ has a higher proportion of concentric contraction than the DJ (Meylan et al., 2011; Mizuguchi et al., 2015).

2.3.7.1 The reliability of the countermovement jump
The CMJ is a reliable means to measure the jump height. The CMJ is a reliable measurement for elite athletes (Cormack et al., 2008; Slind et al., 2008; Enoksen et al., 2009; Casartelli et al., 2010), the elderly (Ditroilo et al., 2011; Farias et al., 2013), and school children (Acero et al., 2011) with ICCs ranging from 0.86-0.99, as outlined in Table 3 (p.49).

The CMJ is often measured in conjunction with other jumps such as the squat jump (Arteaga-Ortiz et al., 2000; Markovic et al., 2004; Moir et al., 2004; Casartelli et al., 2010;
Acero et al., 2011; Glatthorn et al., 2011) and the DJ (Arteaga-Ortiz et al., 2000; Ebben et al., 2008). The CMJ can be used as a reliable universal tool to measure the jump ability of a variety of demographics.
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

<table>
<thead>
<tr>
<th>Author (Date)</th>
<th>n= (Male, Female)</th>
<th>Subjects (Age)</th>
<th>Study Protocol</th>
<th>CMJ Action</th>
<th>Data Acquisition</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arteaga-Ortiz et al. (2000)</td>
<td>17 (8, 9)</td>
<td>Students (24 ± 2)</td>
<td>2 session 20-15 days apart. SJ, CMJ, DJ, HT. 6 X1 sets of each jump. Rest between jumps N/A</td>
<td>Hands on hips. Knees flexed to c. 90°</td>
<td>CGD, Power, work done, inter jump indexes, derived from ( t_f ) (resistive platform)</td>
<td>CV</td>
<td>Reliability: SJ: 5.4%, CMJ: 6.3%, DJ:6.2%, HT: 31.9%</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Markovic et al. (2004)</td>
<td>93 (93, 0)</td>
<td>Students (20 ± 2)</td>
<td>3 sessions 2-3 days apart. Session 1: SarJ, SLJ; Session 2: SJ STJ; Session 3: CMJ, Abl-A; Session 4: Abl-NA 48-72 hours apart. 3X1 (15 min rest)</td>
<td>Hands on hips ( JH ) derived from ( t_c ) contact time (resistive platform)</td>
<td>AVR, ICC, ( \alpha ), CV</td>
<td>Reliability (AVR, ICC, ( \alpha ), CV): SarJ: 0.90, 0.96, 0.96, 3.0%; SLJ: 0.86, 0.95, 0.95,2.4%; SJ: 0.91, 0.97, 0.97, 3.3%; STJ: 0.83, 0.93, 0.93, 2.9%; CMJ: 0.94, 0.98, 0.98, 2.8%; Abl-A: 0.81, 0.93, 0.93, 4.6%; Abl-NA: 0.85, 0.94, 0.94, 4.1%</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>Moir et al. (2004)</td>
<td>10 (10, 0)</td>
<td>Physically active students (25 ± 7)</td>
<td>5 sessions over 3 weeks. 3X1 SJ, SJ+10, CMJ, CMJ+10kg, 20m sprint (0-10m split, 10-20m split). Rest between jumps N/A</td>
<td>Hands on hips, jump as high as possible ( JH ) derived from ( t_f ) (jump mat)</td>
<td>ICC, CV, 2-way ANOVA</td>
<td>Reliability (ICC, CV) SJ: 0.91, 2.4%; SJ+10: 0.89, 2.6%; CMJ: 0.93, 2.4% CMJ+10: 0.87, 2.1; 0-10m: 0.93, 2.0%; 10-20m: 0.91, 1.9%</td>
<td>High</td>
<td></td>
</tr>
</tbody>
</table>

Where (H)SJ(+10) is (horizontal) squat Jump (plus 10kg), (H)CMJ(+10) is (horizontal) countermovement Jump (plus 10kg), DJ is depth/drop jump, HT is hopping test, CGD is centre of gravity displacement, CV is coefficient of variation, SarJ is Sargent’s jump, SLJ is standing long jump, STJ is standing triple jump, Abl-(N)A is Abalakow’s vertical jump (without) with arm swing, \( JH \) is jump height, \( t_f \) is flight time, AVR is average intertrial correlation coefficients, ICC is intra-class correlation coefficients, \( \alpha \) is Cronbach’s alpha reliability coefficient.
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

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<tbody>
<tr>
<td>Maulder and Cronin, (2005) 18 (18, 0)</td>
<td>Physically active (25 ± 4)</td>
<td>1 session. 3X1 each leg SJ, CMJ, RI(3), HSI, HCMJ, HRU(3), 20m sprint. 3 min rest between jumps</td>
<td>Hands on hips, knee flexed to c. 120°</td>
<td>JH derived from t_f, contact time (contact mat Vs FP)</td>
<td>ICC, CV</td>
<td>Reliability (ICC [Dom, non-Dom]; CV [Dom, non-Dom]); SJ:[0.86, 0.82], [3.3%, 4.4%]; CMJ: [0.86, 0.95], [3.3%, 4.1%]; RI: [0.71, 0.81], [5.5%, 8.8%]; HSI: [0.90, 0.89], [1.1%, 1.9%]; HCMJ: [0.95, 0.80], [1.9%, 2.0%]; HRU: [0.97, 0.95], [1.9%, 1.8%], contact mat Fp – flight time ICC 0.95, contact time; ICC 0.99</td>
<td>Moderate to High</td>
<td></td>
</tr>
<tr>
<td>Leard et al. (2007) 39 (14, 25)</td>
<td>Students (21)</td>
<td>1 session. 2X1 CMJ</td>
<td>Semi-squat with arm swing</td>
<td>JH based on t_f measured with 3Cam Vs Jump mat Vs jump and reach</td>
<td>ρ</td>
<td>3Cam Vs Jump mat: ρ =0.97; 3Cam Vs jump and reach: ρ =0.91</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td>Cormack et al. (2008) Study 1 15 (15, 0); Study 2 15 (15, 0)</td>
<td>Study 1 Pro-AFL (23 ± 4); Study 2 Pro-AFL (23 ± 3)</td>
<td>Study 1 2 sessions 2 days apart. 1X1 CMJ Study 2 2 sessions 7 days apart. 1X1 CMJ</td>
<td>Hand on hips at a self-selected depth</td>
<td>JH, t_f, PP, rPP, MP, rMP, PF, rPF, MF, rMF, ET, CT, E:C, EF, t_f:ET, t_f:CT or t_f:CT(5) captured on a FP</td>
<td>CV</td>
<td>Reliable [CMJ, CMJ(5)]: t_f: 2.9, 2.0% PP: 3.5, 4.4% rPP: 3.6, 3.8% PF: 3.5, 3.3% rPF: 3.4, 2.8% MF: 1.1, 2.4% rMF: 6.1, 1.5%</td>
<td>High</td>
<td></td>
</tr>
</tbody>
</table>

Where [H]SJ is (horizontal) squat Jump, [H]CMJ is (horizontal) countermovement Jump, [H]RI(3) is (horizontal) 3 repeat jumps, JH is jump height, t_f is flight time, FP is force platform, ICC is intra-class correlation coefficients, CV is coefficient of variation, Dom is dominant leg. 3Cam is three camera motion capture system, ρ is Pearson’s product moment correlation, Pro-AFL is professional Australian Football League players, CMJ(5) is 5 repetitive countermovement Jumps, PP is peak power, rPP is relative peak power, MP is mean power, rMP is relative mean power, PF is peak force, rPF is relative peak force, MF is mean force, rMF is relative mean force, ET is eccentric time, CT is concentric time, E:C is eccentric ratio, EF is end of eccentric force, t_f:ET is flight time: eccentric time ratio, t_f:CT(5) is flight time: concentric time ratio for the repeated countermovement jump.

Walsh, R. (2017)
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

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</thead>
<tbody>
<tr>
<td>Ebben et al.</td>
<td>24 (11, 13)</td>
<td>Healthy (23 ± 3)</td>
<td>2 sessions, duration between sessions not specified, 1X1 Cone hop, 0.61m box jump, Tuck jump, VJ, SJ 30%, Ankle hops, Pike jump, 1 leg VJ, DJ 0.30m, DJ 0.61m. 1 min rest between jumps</td>
<td>N/A</td>
<td>Quadriceps, hamstring, gastrocnemius activity (EMG)</td>
<td>ICC</td>
<td>Reliability: ICC 0.75 to 0.93 specific ICC for individual jumps N/A</td>
<td>Moderate to High</td>
</tr>
<tr>
<td>Moir et al.</td>
<td>69 (35, 34)</td>
<td>Healthy (22 ± 2 [males], 21 ± 2 [females])</td>
<td>4 sessions 1 week apart. 3X1 CMJ 3 min rest between jumps</td>
<td>N/A</td>
<td>JH derived from t&lt;sub&gt;f&lt;/sub&gt;, t&lt;sub&gt;f&lt;/sub&gt; (contact mat)</td>
<td>ICC, CV</td>
<td>Reliability (ICC, CV): Males 0.89-0.94, 3.3-5.2%; Females 0.90-0.95, 4.1-6.0%</td>
<td>High</td>
</tr>
<tr>
<td>Slinde et al.</td>
<td>30 (13, 17)</td>
<td>Non-elite athletes (23 ± 1 [males], 22 ± 2 [females])</td>
<td>2 session 4-7 days apart. 3X1 CMJ, CMJ+A, Abl-A. Rest not specified</td>
<td>CMJ: Hands on hips. Fast knees flexion to c. 90°, jump as high as possible. CMJ+A: Knees flexed to c. 90°, use arm swing Jump as high as possible.</td>
<td>JH derived from t&lt;sub&gt;f&lt;/sub&gt; (contact mat)</td>
<td>ICC, Bland-Altman plots, α</td>
<td>Reliability (ICC, α): CMJ: 0.93, 0.96; CMJ+A: 0.93, 0.96; Abl-A: 0.87, 0.93</td>
<td>High</td>
</tr>
</tbody>
</table>

Where VJ is vertical jump, SJ30% is squat jump with 30% of front squat one repetition maximum, DJ is depth jump, EMG is electromyography, ICC is intra-class correlation coefficients, CMJ(+A) is countermovement Jump (plus arm swing), JH is jump height, t<sub>f</sub> is flight time, CV is coefficient of variation, Abl-(N)A is Abalakow’s vertical jump (without) with arm swing, α is Cronbach’s alpha reliability coefficient.
## Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

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</tr>
</thead>
<tbody>
<tr>
<td>Enoksen et al. (2009)</td>
<td>20 (20, 0)</td>
<td>Pro soccer (19 ± 4)</td>
<td>2 sessions, duration between sessions not specified. 3X1 CMJ, SJ, 0-20m, 20-40m sprint. 3 min between attempts</td>
<td>Hands on hips. Fast downward movement, knees flexed to c. 90°, jump as high as possible</td>
<td>JH measured using contact mat with high-density infrared beam</td>
<td>95% LoA, paired t-test, ρ</td>
<td>LoA (contact mat, FP): CMJ:0.01m, 0.01m; SJ: 0.01m, 0.01m; 0-20m:1.0sec, 1.02sec; 20-40m: [0.02sec, 1.01sec. Validity - ρ; CMJ: -0.08; SJ: 0.07; 0-20m: 0.23; 20-40m: -0.28</td>
<td>High</td>
</tr>
<tr>
<td>Hori et al. (2009)</td>
<td>24 (24, 0)</td>
<td>Physically active (25 ± 4)</td>
<td>1 session 2X2 CMJ. Rest between jumps N/A</td>
<td>Hands on hips. Fast downward movement, knees flexed to c. 90°, jump as high as possible</td>
<td>GRF, PP, MP, PF, Pν, Min-v, PRFD, TPF MRPD, TPP captured on a FP at 500, 400, 250, 200, 100, 50, 25Hz</td>
<td>ICC, CV</td>
<td>Reliability (ICC range, CV range): PP:0.96-0.98, 2.3-2.6%; MP: 0.71-0.85, 7.4-12.3%; PF: 0.92-0.93, 3.9-4.1%; Mf: 0.84-0.94, 3.7-6.3%; Pν: 0.95-0.98, 1.3-1.7%; Min v: 0.75-0.78, 9.8-10.0%. PRFD: 0.66-0.75, 20.7-24.0%; TPF: 0.74-0.78, 10.8-13.4%; MRPD: 0.87-0.95, 7.9-14.9%; TPP: 0.57-0.85, 7.0-14.4%</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Sassi et al. (2009)</td>
<td>86 (34, 52)</td>
<td>Students (23 ± 4)</td>
<td>2 session on the same day. 3X1 CMJ, A-T-test, m-A-T-test, 10m sprint. 3 min between attempts</td>
<td>Knees flexed to c. 90°, use arm swing. Jump as high as possible</td>
<td>PνH derived from t (PTD)</td>
<td>ICC, CV</td>
<td>CMJ reliability: Males ICC 0.97; Females ICC 0.93; A-T-test: ICC 0.90; Females ICC 0.97; m-A-T-test, [ male ICC 0.95, CV 2.7%], Females ICC 0.92, CV 2.6%</td>
<td>Moderate to high</td>
</tr>
</tbody>
</table>

Where CMJ is countermovement Jump, SJ is squat jump, (P)νH is (peak)jump height, PTD is photocell timing device, FP is force platform, LoA is limit of agreement, ρ is Pearson’s product moment correlation, GRF is ground reaction force, PP is peak power MP is mean power, PF is peak force, MF is mean force, Pν is peak velocity, Min-v is minimum velocity, PRFD is peak rate of force development, TPF is time to peak force, MRPD is mean rate of power production, TPP is time to peak power, ICC is intra-class correlation, CV is coefficient of variation, A-T-test is agility T-test, mA-T-test is modified agility T-test.
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

<table>
<thead>
<tr>
<th>Author</th>
<th>n= (Male, Female)</th>
<th>Subjects (Age)</th>
<th>Study Protocol</th>
<th>CMJ Action</th>
<th>Data Acquisition</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bubanj et al. (2010)</td>
<td>10 (10, 0)</td>
<td>Students (24 ± 2)</td>
<td>2 sessions, 2 weeks apart. SX1 CMJ</td>
<td>Hands on hips. knees flexed to c. 90°</td>
<td>JH, power, force, velocity (wireless accelerometer).</td>
<td>α.</td>
<td>Reliability: α: 0.86</td>
<td>High</td>
</tr>
<tr>
<td>Casartelli et al. (2010)</td>
<td>44 (44, 0)</td>
<td>Bkt-ball players (15 ± 4)</td>
<td>2 sessions, 2-15 days apart. CMJ, SJ, R15s, 3 min rest between jumps</td>
<td>Hands on hips. knees flexed to c. 90°</td>
<td>JH derived from τ, JH based on tAcc Vs vAcc Vs PTD</td>
<td>ICC, LoA, CV, 1-way ANOVA</td>
<td>Reliability: TAcc (ICC, CV), VAcc (ICC, CV), PTD (ICC, CV) SJ: (0.92, 4.9%), (0.83, 11.3%), (0.95, 5.8%); CMJ: (0.93, 3.6%), (0.89, 6.4%), (0.97, 3.7%); R15s: (0.92, 5.1%), (0.56, 13.2%), (0.94, 5.7%) JH with TAcc &amp; VAcc is significantly higher than PTD</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Acero et al. (2011)</td>
<td>56 (26, 30)</td>
<td>School children (7 ± 1)</td>
<td>2 sessions, 7 days apart. 4X1 CMJ, SJ, 1 min rest between jumps</td>
<td>Hands on hips. knees flexed to c. 90°</td>
<td>JH derived from τ (contact platform)</td>
<td>ICC, ME, repeated ANOVA</td>
<td>Reliability - Within session ICC (1, 2), between session ICC: SJ: (0.83, 0.99), 0.70; CMJ: (0.95, 0.99), 0.86 Within session CV (1, 2), between session ME: SJ: (11.0, 9.2%) 15.1%; CMJ: (8.7, 8.5%), 9.9%</td>
<td>High</td>
</tr>
</tbody>
</table>

Where CMJ is countermovement Jump, JH is jump height, α is Cronbach’s alpha reliability coefficient, Bkt-ball is basketball, SJ is squat jump, R15s is repeated jumps for 15 repetitions, JH is jump height, τ is flight time, tAcc is time measured with accelerometer, vAcc is velocity measured with accelerometer, PTD is photocell timing device, LoA is limits of agreement, ANOVA is analysis of variance, ME is methodical error.
<table>
<thead>
<tr>
<th>Author</th>
<th>Study</th>
<th>Subjects</th>
<th>Study Protocol</th>
<th>CMJ Action</th>
<th>Data Acquisition</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ditroilo et al. (2011)</td>
<td>82 (35, 47)</td>
<td>Healthy ([male 55-65 years' group 60 ± 3], [male 66-75 years' group 70 ± 3], [female 55-65 years' group 60 ± 3], [female 66-75 years' group 70 ± 3])</td>
<td>2 session, 4 weeks apart. 3X1 CMJ. 30 sec rest between jumps</td>
<td>Hands on hips, knees flexed to self-selected angle</td>
<td>JH based on t_f; JH derived from force PP; PP:BM; PF; PF:BM (FP)</td>
<td>ICC, CV, SEM, MDC</td>
<td>Reliability (intra-session ICC range based on age and gender, inter-session ICC range based on age and gender): JH: 0.79-0.92, 0.48-0.88; FH: 0.84-0.94, 0.84-0.93; PP: 0.89-0.97, 0.84-0.97; PP:BM: 0.85-0.94, 0.62-0.94; PF: 0.90-0.94, 0.59-0.95; PF:BM: 0.75-0.91, 0.42-0.83; Reliability (intra-session CV [1,2] range based on age and gender, inter-session MDC range based on age and gender): JH: [5.3-10.8%, 5.6-9.8%], 0.57-0.76m; FH: [5.4-9.6%, 6.9-9.2%], 0.24-0.57m; PP: [2.9-4.2%, 3.3-5.7%], 197.7-444.7W; PP:BM: [2.9-4.3%, 3.1-5.7%], 3.2-5.7W∙kg⁻¹; PF: [4.1-5.5%, 5.1-8.4%], 235.2-447.0N; PF:BM: [4.1-4.6%, 5.0-7.8%], 2.8-5.6N∙kg⁻¹</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Glatthorn et al. (2011)</td>
<td>Study 1: 20 (18, 2); Study 2: 20 (10, 10)</td>
<td>Healthy (study 1 [22 ± 6], study 2 [30 ± 6])</td>
<td>Study 1 2 sessions, 1 week apart. 3X1 SJ, CMJ, CMJ+A. 30 sec rest between jumps</td>
<td>CMJ: Hands on hips. Fast knees flexed to c. 90°. Jump as high as possible. CMJ+A: Knees flexed to c. 90°. Use arm swing, jump as high as possible</td>
<td>JH derived from t_f performed simultaneously on a FP and a PTD</td>
<td>ICC, CV, Bland-Altman</td>
<td>Tests-retest reliability (ICC, CV): SJ: 0.98, 3.1%; CMJ: 0.99, 2.2%; CMJ+A: 0.98, 2.8% Concurrent validity (ICC): SJ: &gt;0.99; CMJ: &gt;0.99; CMJ+A: &gt;0.99</td>
<td>High</td>
</tr>
</tbody>
</table>

Where CMJ(+A) is countermovement Jump (plus arm swing), JH is jump height, t_f is flight time, PP is peak power, PP:BM is peak power to body weight ratio, PF is peak force, PF:BW is peak force to body weight ratio, FP is force platform ICC is intra-class correlation, CV is coefficient of variation, SEM is standard error of the measurement, MDC is minimal detectable change, SJ is squat Jump, PTD is photocell timing device.

Walsh, R. (2017)
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

<table>
<thead>
<tr>
<th>Author</th>
<th>n= (Male, Female)</th>
<th>Subjects (Age)</th>
<th>Study Protocol</th>
<th>CMI Action</th>
<th>Data Acquisition</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nuzzo et al. (2011)</td>
<td>79 (40, 39)</td>
<td>Students (male [20 ± 2], female [20 ± 2])</td>
<td>2 sessions, 48 hours apart. 3X1 CMJ. 1 min rest between jumps</td>
<td>Use arm swing, Jump as high as possible</td>
<td>JH derived from t_f performed simultaneously on a jump mat vs wireless accelerometer vs analogue jump vanes</td>
<td>ICC, CV, SEM, 1-way ANOVA, Bland-Altman plots, p</td>
<td>Intra-session reliability grouped by gender (ICC range, CV range): Jump mat: 0.90-0.93, 4.2-5.2%; Accelerometer: 0.91-0.95, 3.9-5.5%; Analogue jump vanes: 0.87-0.94, 4.6-7.6%. No difference for the 3 measuring devices</td>
<td>High</td>
</tr>
<tr>
<td>Castagna et al. (2013)</td>
<td>20 (20, 0)</td>
<td>Regional level rugby players (16± 1)</td>
<td>2 session, 24 hours apart. 3-5X1 CMJ. 30 sec rest between jumps</td>
<td>Hands on hips. Fast knees flexed to c. 90°, jump as high as possible</td>
<td>JH and v derived from acceleration, performed simultaneously on a FP sampling at 1000Hz vs PTD vs wireless accelerometer</td>
<td>ICC, CV, 1-way ANOVA, Bland-Altman plots</td>
<td>Inter-measure ICC: FP vs PTD 0.99; FP vs wireless accelerometer 0.88</td>
<td>High</td>
</tr>
<tr>
<td>Farias et al. (2013)</td>
<td>31 (0, 31)</td>
<td>Elderly (70 ± 6)</td>
<td>2 sessions, 5-7 days apart. 4X1 VJ 40 sec rest between jumps</td>
<td>Hands on hips and jump as high as possible</td>
<td>JH derived from t_f (jump mat)</td>
<td>ICC Repeat ANOVA, SEM, CV</td>
<td>Reliability (within session [1,2], between sessions): ICC: [0.96, 0.95], 0.91; SEM: [≤0.01&lt;0.01], N/S; CV: [10.1, 9.1%], 23.2%</td>
<td>High</td>
</tr>
<tr>
<td>Markwick et al. (2015)</td>
<td>13 (13, 0)</td>
<td>Pro-Bkt-ball (26 ± 4)</td>
<td>1 session. 3X1 DJ, CMJ 1 min rest between jumps and 3 min rest between drop heights</td>
<td>Holding a nearly weightless pipe across the back of the shoulders to prevent arm action. Drop to self-selected depth. Jump as high as possible</td>
<td>JH derived from t_f(jump mat)</td>
<td>ICC, CV</td>
<td>CMJ intra-day reliability: ICC: 0.96; CV: 3.3%</td>
<td>High</td>
</tr>
</tbody>
</table>

Where CMJ is countermovement Jump, JH is jump height, t_f is flight time, ICC is intra-class correlation coefficients, CV is coefficient of variation, SEM is standard error of measurement, ANOVA is analysis of variance, p is Pearson product moment correlations, v_i is velocity at take off, FP is force platform, PTD is photocell timing device, VJ is vertical Jump, Pro-Bkt-ball is professional basketball players, DJ is depth jump.

Walsh, R. (2017)
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

<table>
<thead>
<tr>
<th>Author (Date)</th>
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<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mizuguchi et al. (2015)</td>
<td>Part 1 (12 [6, 6]). part 2 (20 [15, 5])</td>
<td>Students (part 1 [22 ± 3], part 2 [23 ± 5])</td>
<td>2 sessions, 48 hours apart. 2X1 CMJ (within 5% more attempt were permitted). 1 min rest between jumps</td>
<td>Holding a nearly weightless pipe across the back of the shoulders to prevent arm action</td>
<td>CMJ action: JH derived from ( t_f ) (FP); C-net, A-net, net-H, net-W, RFD, SF, net-P</td>
<td>Part 1: ICC, CV. part 2: ( \rho )</td>
<td>Reliability (ICC, CV): JH: 1.00, 2.3%; Cnet: 1.00, 1.2%; Inet: 1.00, 1.2%; net-H: 0.98, 4.6%; net-W: 0.91, 5.1%; RFD: 0.78, 22.3%; SF: 0.89, 3.3%; net-P: 0.96, 1.5%</td>
<td>High</td>
</tr>
<tr>
<td>Gallardo-Fuentes et al. (2016)</td>
<td>21 (14, 7)</td>
<td>InterN &amp; national track &amp; field athletes (21 ± 4)</td>
<td>2 sessions 48 hours apart. 5X1 SJ, 5X1 CMJ, 5X1DJ (0.40 m). 2 min rest between jumps</td>
<td>Hands on hips elbows out</td>
<td>( JH ) derived from ( t_f ) (FP vs. Jump-App)</td>
<td>( \alpha ), CV, ( \rho )</td>
<td>FP: ( \alpha ): 0.99; CV: 4.63–5.46%; ( \rho ): 0.95; Jump-App. ( \alpha ): 0.99; CV: 4.65–4.74%; ( \rho ): 0.93</td>
<td>High</td>
</tr>
<tr>
<td>Harper et al. (2016)</td>
<td>10 (10, 0)</td>
<td>Collegiate level soccer players (22 ± 3)</td>
<td>2 sessions 7 days apart. 3X1 CMJ. 10 sec rest between jumps; 3X1 20 m sprints. 25 sec rest between sprints. Tested were conducted at 0 min, 60 min (end of 1st half), 61 min (start of 2nd half), 90 min, 120 min of a 120 min simulated soccer exercise with a 15 min half time</td>
<td>N/A</td>
<td>N/A</td>
<td>CV, TE, ( \rho )</td>
<td>Reliability at (0 min, 60 min, 61 min, 90 min, 120 min) CV: 3.8%, 3.1%, 4.0%, 3.5%, 4.9%; TE: 0.17 m, 0.12 m, 0.14 m, 0.13 m, 0.18 m; ( \rho ): 0.85, 0.92, 0.88, 0.88, 0.86</td>
<td>High</td>
</tr>
</tbody>
</table>

Where CMJ is countermovement Jump, \( JH \) is jump height, \( t_f \) is flight time, FP is force platform, C-net is criterion net impulse, A-net is alternative net impulse, net-H is impulse height, net-W is net impulse weight, RFD is rate of force development, SF is shape factor, net-P is net impulse proportion, ICC is intra-class correlation coefficients, CV is coefficient of variation, \( \rho \) is Pearson’s moment coefficient, SJ is squat jump, DJ is depth jump, InterN is international, Jump-App is mobile telephone jump application, \( \alpha \) is Cronbach’s alpha reliability coefficients, TE is typical error.
Table 3: Summary of peer-reviewed studies that investigated the reliability of the countermovement jump

<table>
<thead>
<tr>
<th>Author et al. (Date)</th>
<th>n= (Male, Female)</th>
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<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jarvis et al. (2016)</td>
<td>7 (7, 0)</td>
<td>Rec or collegiate sports (22 ± 1)</td>
<td>2 sessions, time between sessions not specified. 3X1 CMJ, 3X1 RbJ, 3X1 DJ (0.30, 0.40 m), 3X1 SL-CMJ, 3X1 SL-RbJ, 3X1 SL-DJ. 2 min rest between jumps</td>
<td>N/A</td>
<td>EMG (VL, BF): %MVIC (Ecc phase, Con phase), FP: rPGRF, REccP, RImp</td>
<td>SEM, ICC, SDD</td>
<td>Reliability (CMJ [SEM, ICC, SDD]): VL Ecc: 5.60%MVIC,0.97; 15.90%MVIC; VL Con: 17.10%MVIC,0.99; 47.50%MVIC; BF Ecc: 1.40%MVIC,0.98; 3.80%MVIC; BF Con: 4.70%MVIC,0.86; 13.00%MVIC; rPGRF: 0.10N∙kgbw⁻¹, 0.96, 0.20N∙kgbw⁻¹; REccP: 0.10N∙kgbw⁻¹, 0.96, 0.30N∙kgbw⁻¹; RImp: 0.01N∙kgbw⁻¹, 0.96, 0.01N∙kgbw⁻¹</td>
<td>High</td>
</tr>
<tr>
<td>McMahon et al. (2016)</td>
<td>18 (18, 0)</td>
<td>Collegiate level athletes (23 ± 5)</td>
<td>1 session. 6X1 CMJ. 2 min rest between jumps</td>
<td>Hand on hips</td>
<td>JH derived from tₐ (jump mat vs FP).</td>
<td>ICC, CV, ρ</td>
<td>Reliability ICC: Jump mat 0.96, FP 0.96; CV: Jump mat 3.7%, FP 4.7%; p: 1.00</td>
<td>High</td>
</tr>
<tr>
<td>Rodriguez-Rosell et al. (2016)</td>
<td>186 (186, 0)</td>
<td>Under-15 (14 ± 1) soccer, under-18 (17 ± &lt;1) soccer, adult (22 ± 5) soccer, under-15 (15 ± 1) Bkt-ball, under-18 (16 ± &lt;1), Bkt-ball, adult (23 ± 4) Bkt-ball players</td>
<td>1 sessions. 3X1 CMJ, ABI-NA, 1-LSSJ, 2-LSSJ. 3 min rest between jumps</td>
<td>Hand on hips</td>
<td>JH derived from tₐ (PTD)</td>
<td>ICC, SEM, CV, MD</td>
<td>CMJ Reliability (ICC, SEM, CV MD): Under-15 soccer: 0.990, 0.82, 2.60%, 7.11%; under-18 soccer: 0.989, 0.84, 2.32%, 6.34%; adult soccer: 0.995, 0.61, 1.61%, 4.40%; under-15 Bkt-ball: 0.992, 0.77, 2.36%, 6.67%; under-18 Bkt-ball: 0.992, 0.91, 2.70%, 7.62%; adult Bkt-ball: 0.995, 0.74, 2.15%, 5.87%</td>
<td>High</td>
</tr>
</tbody>
</table>

Where Rec is recreational, (SL-)CMJ is (single leg) countermovement Jump, (SL-)DJ is (single leg) depth Jump, (SL-)RbJ is (single leg) rebound Jump, EMG is electromyography, VL is vastus lateralis, BF is bicep femoris, %MVIC is percent of maximum voluntary isometric concentric, Ecc is eccentric, Con is concentric, rPGRF is relative peak ground reaction force, REccP is relative peak eccentric power, RImp is relative impulse, SEM is standard error of measure, ICC is intra-class correlation coefficients, SDD is smallest detectable difference, JH is jump height, tₐ is flight time, FP is force platform, CV is coefficient of variation, p is Pearson’s moment coefficient, Bkt-ball is basketball, ABI-NA is Abalakow’s vertical jump without arm swing, 1-LSSJ is one leg sport-specific vertical jump, 2-LSSJ is two legs sport-specific vertical jump, PTD is photocell timing device, MD is minimal difference.
<table>
<thead>
<tr>
<th>Author (Date)</th>
<th>n= (Male, Female)</th>
<th>Subjects (Age)</th>
<th>Study Protocol</th>
<th>CMI Action</th>
<th>Data Acquisition</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roe et al. (2016)</td>
<td>25 (25, 0)</td>
<td>Youth rugby union players (18 ± &lt;1)</td>
<td>2 sessions 5 days apart. 3XCMJ, plyometric push-ups. 1 min rest between jumps</td>
<td>Hand on hips</td>
<td>JH, t, PP, MP, PF, FT:C (FP)</td>
<td>SWC, CV</td>
<td>Reliability (SWC, CV)</td>
<td>Poor to good</td>
</tr>
<tr>
<td>Stanton et al. (2017)</td>
<td>29 (10, 19)</td>
<td>Rec athletes (26 ± 5)</td>
<td>2 Session 7 days apart. 2X1 CMJ, DJ. 2 min rest between jumps</td>
<td>Hand on hips</td>
<td>JH derived from t (Jump-App vs FP)</td>
<td>ICC, Bland-Altman plots</td>
<td>CMJ-ICC</td>
<td>High</td>
</tr>
</tbody>
</table>

Where CMJ is countermovement Jump, DJ is depth jump, JH is jump height, t is flight time, PP is peak power, MP is mean power, PF is peak force, MF is mean force, FT:C is ratio of flight time to contraction, FP is force platform, SWC is smallest worthwhile change, CV is coefficient of variation, Rec is recreational, Jump-App is mobile telephone jump application, ICC is intra-class correlation coefficients.
2.3.7.2 Countermovement jump protocol

The number of jumps performed during testing should be enough to get accurate data but as few as possible to prevent fatigue. The number of jumps used in studies in relation to CMJ reliability ranged from one jump (Arteaga-Ortiz et al., 2000; Cormack et al., 2008; Ebben et al., 2008) to five jumps (Bubanj et al., 2010; Castagna et al., 2013). The predominant number of jumps is three (Markovic et al., 2004; Moir et al., 2004; Maulder and Cronin, 2005; Moir et al., 2008; Slinde et al., 2008; Enoksen et al., 2009; Sassi et al., 2009; Ditroilo et al., 2011; Glatthorn et al., 2011; Nuzzo et al., 2011). The rest between each jump should be long enough to allow recovery and prevent fatigue. Thirty seconds (Ditroilo et al., 2011; Glatthorn et al., 2011; Castagna et al., 2013) and three minutes rest are common (Moir et al., 2008; Enoksen et al., 2009; Sassi et al., 2009) with the most common rest time reported in the literature between jumps is one minute (Ebben et al., 2008; Acero et al., 2011; Nuzzo et al., 2011; Mizuguchi et al., 2015). A small number of studies allowed free arm swing (Sassi et al., 2009; Nuzzo et al., 2011). However, the majority of reliability studies in relation to the CMJ instructed subjects to ‘place hands on hips and drop down until knees are about 90° and jump as high as possible’ (Arteaga-Ortiz et al., 2000; Markovic et al., 2004; Moir et al., 2004; Maulder and Cronin, 2005; Cormack et al., 2008; Slinde et al., 2008; Enoksen et al., 2009; Hori et al., 2009; Bubanj et al., 2010; Casartelli et al., 2010; Acero et al., 2011; Ditroilo et al., 2011; Glatthorn et al., 2011; Castagna et al., 2013; Farias et al., 2013). One study used a polyvinyl pipe held across the shoulders to prevent arm swing (Mizuguchi et al., 2015). Using an arm action can increase $JH$ by 20%. Keeping the hands on the hips allows more for accurate measures of the lower limb power (Slinde et al., 2008). A protocol involving CMJ should afford enough jumps and adequate recovery between jumps to the subject to allow appropriate data collection without causing fatigue. A protocol of three jumps with one minute’s recovery is favoured in the literature. Preventing arm swing will measure just the power of the lower limb.

2.3.8 Depth jump

DJs typify plyometric training with their quick eccentric and concentric phases, often called reactive strength (Byrne et al., 2010). DJs may potentiate performance in sports in which running and jumping are fundamental components (Asadi, 2012; Byrne et al.,
DJs are performed by stepping off a platform and initiating a maximum vertical jump as fast as possible (Potach and Chu, 2008). The height of the platform can vary from 0.20 m to 0.90 m (Arteaga-Ortiz et al., 2000; Byrne et al., 2010; Markwick et al., 2015; Matic et al., 2015; Byrne et al., 2017). There are two versions of DJs: the counter movement version, which loads the musculature; and the bounce movement which loads the tendons (Byrne et al., 2010, 2014; Matic et al., 2015; Byrne et al., 2017). It has been suggested that contact time ($t_c$) of less than 0.25 seconds is needed in order to ensure the stretch-shortening cycle is utilised, as there is less movement in the lower limb (Byrne et al., 2010, 2014, 2017). It has been suggested that 0.40 seconds is also acceptable (Matic et al., 2015). Therefore, the bounce version of the DJ may better employ the stretch-shortening cycle (Byrne et al., 2010, 2014, 2017; Matic et al., 2015; Byrne et al., 2017). The reactive strength is often measured by the reactive strength index ($RSI$) which factors in the time to reverse the downward force of the landing into upward force of jump power or $t_c$ and height of the $JH$ as illustrated in Equation 4.

$$RSI = JH \cdot t_c^{-1}$$

Equation 4: Reactive strength index.

Where $RSI$ is reactive strength index, $JH$ is jump height (m), $t_c$ is contact time (s).

### 2.3.8.1 Depth jump reliability

The $RSI$, $JH$, and $t_c$ during DJs are methods of measuring reactive strength (Table 4 [p.61]) and have been found to be reliable (Beattie and Flanagan, 2015; Markwick et al., 2015; Schuster and Jones, 2016; Byrne et al., 2017; Stanton et al., 2017). The DJ is often tested with other jumps such as the squat jump and CMJ (Arteaga-Ortiz et al., 2000; Ebben et al., 2008; Markwick et al., 2015). The $RSI$ and $JH$ are reliable when tested with elite athletes (Flanagan et al., 2008; Beattie and Flanagan, 2015; Markwick et al., 2015; Byrne et al., 2017) and non-elite subjects (Arteaga-Ortiz et al., 2000; Ebben et al., 2008).
Table 4: Summary of peer-reviewed studies that investigated the reliability of the depth jump

<table>
<thead>
<tr>
<th>Author (Date)</th>
<th>n= (Male, Female)</th>
<th>Subjects (Age)</th>
<th>Study Protocol</th>
<th>DJ Action</th>
<th>Data Acquisition</th>
<th>Statistics</th>
<th>Results</th>
<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arteaga-Ortiz et al. (2000)</td>
<td>17 (8, 9) Students (24 ± 2)</td>
<td>2 session, 20-15 days apart. DJ (0.20, 0.30, 0.40, 0.50, 0.60, 0.70, 0.80, 0.90m), SJ, CMJ, HT. 6 sessions 1X1 of each jump. Rest between jumps not specified</td>
<td>Minimise knee flexion. Jump up as fast as possible</td>
<td>ODH - where there was a plateau in JH derived from t₇ (jump mat)</td>
<td>CV</td>
<td>Reliability: SJ: 5.4%, CMJ: 6.3%, DJ:6.2%, HT: 31.9%</td>
<td>Moderate to high</td>
<td></td>
</tr>
<tr>
<td>Ebben et al. (2008)</td>
<td>24 (11, 13) Healthy (23 ± 3)</td>
<td>2 sessions, duration between sessions not specified. 1X1 Cone hop, 0.61m box jump, Tuck jump, VJ, SJ 30%, Ankle hops, Pike jump, 1 leg vertical jump, DJ (0.30m, 0.61m), 1 min rest between jumps</td>
<td>Action not specified</td>
<td>Quadriceps, hamstring, gastrocnemius activity (EMG)</td>
<td>ICC</td>
<td>Reliability: ICC 0.75 to 0.93. Specific ICC for individual jump N/A</td>
<td>Moderate to high</td>
<td></td>
</tr>
<tr>
<td>Flanagan et al. (2008)</td>
<td>22 (22, 0) Collegiate athletes (20 ± 2)</td>
<td>2 sessions. The time between sessions was not specified. 3X1 DJ (0.30m). 1min rest between jumps</td>
<td>Step off box jump as fast and as high as possible think of a “hot plate”. Arms ab lib</td>
<td>JH, t₇, RSI, TTS derived from t₇ (jump mat)</td>
<td>α</td>
<td>Intra-trial reliability: JH: 0.99, CT: 0.98, RSI: 0.99, TTS: 0.68</td>
<td>Moderate to high</td>
<td></td>
</tr>
</tbody>
</table>

Where DJ is depth/drop jump, SJ is squat Jump, CMJ is countermovement Jump, HT is hopping test, ODH is optimum drop height, JH is jump height, t₇ is flight time, CV is coefficient of variation, VJ is vertical jump, EMG is electromyography, ICC is intra-class correlation coefficients, TTS is time to stabilisation, α is Cronbach’s alpha reliability coefficient.
Table 4: Summary of peer-reviewed studies that investigated the reliability of the depth jump

<table>
<thead>
<tr>
<th>Author (Date)</th>
<th>n= (Male, Female)</th>
<th>Subjects (Age)</th>
<th>Study Protocol</th>
<th>DJ Action</th>
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<th>Reliability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beattie and Flanagan (2015)</td>
<td>15 (15, 0)</td>
<td>Junior InterN rugby players (19 ± 0)</td>
<td>2 sessions, 15 days apart 96 hours after a match during competition 3X1 DJ (0.40m), 1min rest between jumps</td>
<td>Hands on hips. Step off box jump as fast and as high as possible</td>
<td>JH, tc, RSI derived from tf (jump mat)</td>
<td>ICC, CV, SWC</td>
<td>Trial-to-trial (ICC, CV, SWC): JH: 0.70, 5.7%, 3.2%; tc: 0.85, 8.4%, 5.6%; RSI: 0.90, 5.3%, 2.8% Inter-day reliability (ICC, CV, SWC): JH: 0.84, 9.0%, 3.1%; tc: 0.89, 8.9%, 5.6%; RSI: 0.93, 8.5%, 3.3%</td>
<td>Moderate to high</td>
</tr>
<tr>
<td>Markwick et al. (2015)</td>
<td>13 (13, 0)</td>
<td>Pro Bkt-ball (26 ± 4)</td>
<td>1 session. 3X1 DJ (0.20, 0.30, 0.40, 0.50m). CMJ 1 min rest between jumps and 3 min rest between drop heights</td>
<td>Holding a nearly weightless pipe across the back of the shoulders to prevent arm action. Step out. Jump as high and as fast as possible</td>
<td>JH, tc, RSI derived from tf (jump mat)</td>
<td>ICC, CV</td>
<td>RSI intra-day reliability (ICC, CV): 0.20m: 0.96, 3.1%; 0.30m: 0.89, 4.2%; 0.40m: 0.95, 3.0%; 0.50m: 0.99, 2.1% JH intra-day reliability (ICC, CV): 0.20m: c.0.98, c.3.3%; 0.30m: c.0.97, c.3.4%; 0.40m: 0.98, 2.8%; 0.50m: c.0.97, 3.5%</td>
<td>High</td>
</tr>
<tr>
<td>Gallardo-Fuentes et al. (2016)</td>
<td>21 (14, 7)</td>
<td>InterN &amp; national track &amp; field athletes (21 ± 4)</td>
<td>2 sessions 48 hours apart. 5X1 SJ, 5X1 CMJ, 5X1DJ (0.40m). 2 min rest between jumps</td>
<td>Hands on hips elbows out</td>
<td>JH - derived from tf (FP vs Jump-App)</td>
<td>α, CV</td>
<td>FP Intra-session (α: 0.98, CV: 4.63–5.46); Inter-session (ρ: 0.98); Jump-App. Intra-session (α: 0.99, CV: 6.06–6.13); Inter-session (ρ: 0.97)</td>
<td>High</td>
</tr>
</tbody>
</table>

Where InterN is international, DJ is depth/drop jump, JH is jump height, tc is contact time, tf is flight time, ICC is intra-class correlation coefficient, CV is coefficient of variation, SWC is smallest worthwhile change, RSI is reactive strength index, Pro is professional, Bkt is basketball, CMJ is countermovement jump, SJ is squat jump, Jump-App is mobile telephone jump application, α is Cronbach’s alpha reliability coefficients, ρ is Pearson’s moment coefficient.

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Table 4: Summary of peer-reviewed studies that investigated the reliability of the depth jump

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Read et al. (2016)</td>
<td>26 (26, 0)</td>
<td>Pro soccer youth academy (pre-growth spur [12 ± 0], post-growth spur [17 ± 1])</td>
<td>2 sessions 7 days apart. 1X1 DJ (0.30 m), 75% Hop, SL-CMJ</td>
<td>Hands free to replicate a natural jump-landing position</td>
<td>pVGRF, time to pVGRF (bilateral FP)</td>
<td>ICC, CV, 95% CI</td>
<td>pVGRF: ICC: 0.50 CV (95% CI): 20.5% (17.1-25.8%) time to pVGRF: ICC: 0.54 CV (95% CI): 49.7% (40.6-64.4%)</td>
<td>Fair</td>
</tr>
<tr>
<td>Jarvis et al. (2016)</td>
<td>7 (7, 0)</td>
<td>Rec or collegiate sports (22 ± 1)</td>
<td>2 sessions, time between sessions not specified. 3X1 CMJ, 3X1 RbJ, 3X1 DJ (0.30, 0.40 m), 3X1 SL-CMJ, 3X1 SL-RbJ, 3X1 SL-DJ. 2 min rest between jumps</td>
<td>Hand position not specified</td>
<td>EMG (VL, BF): %MVIC (Ecc phase, Con phase), FP: rPGRF, REccP, Rimp</td>
<td>SEM, ICC, SDD</td>
<td>Reliability (SEM, ICC, SDD): VL Ecc: 14.50-15.80%MVIC, 0.74-0.89, 40.30-43.90%MVIC; VL Con: 8.60-9.60%MVIC, 0.86-0.94, 13.30-13.80%MVIC; BF Ecc: 5.00-4.80%MVIC, 0.96-0.97, 13.30-13.80%MVIC; BF Con: 5.60-5.70%MVIC, 0.92-0.94, 15.60-15.80%MVIC; rPGRF: 0.20-0.30N kgbw⁻¹, 0.85-0.97, 0.60-0.90N kgbw⁻¹; REccP: 0.40-0.60N kgbw⁻¹, 0.91-0.96, 1.20-1.70N kgbw⁻¹; Rimp: &lt;0.00-0.01N kgbw⁻¹, 0.97-0.99, 0.01N kgbw⁻¹</td>
<td>Moderate to High</td>
</tr>
<tr>
<td>Schuster and Jones (2016).</td>
<td>19 (19, 0)</td>
<td>Collegiate level athletes (23 ± 3)</td>
<td>1 session. 3X1 SL-DJ (0.20 m), SL-HDj, 20 m sprint. 45 s rest between jumps.</td>
<td>Hands on hips</td>
<td>RSI, τc, JH, all derived from τf (FP)</td>
<td>ICC, SEM SDD</td>
<td>Between leg reliability (ICC, SEM SDD) RSI: 0.987, 0.03, 8.08%; τc: 0.992, 0.01, 7.14%; JH: 0.957, 0.01, 10.53%</td>
<td>High</td>
</tr>
</tbody>
</table>

Where Pro is professional, (SL-DJ) is (single leg) depth/drop jump, 75%Hop is 75% of maximum horizontal hop and stick, (SL-CMJ) is (single leg) countermovement jump, rPGRF is relative peak ground reaction force, FP is force platform, ICC is intra-class correlation coefficient, CV is coefficient of variation, 95% CI is 95% confidence intervals, Rec is Recreational, (SL-RbJ) is (single leg) rebound jump, EMG is electromyography, VL is vastus lateralis, BF is bicep femoris, %MVIC is percent of maximum voluntary isometric concentric, Ecc is eccentric, Con is concentric, REccP is relative peak eccentric power, Rimp is relative impulse, SEM is standard error of measure, SDD is smallest detectable difference, RSI is reactive strength index, τc is contact time, JH is jump height, τf is flight time.

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Table 4: Summary of peer-reviewed studies that investigated the reliability of the depth jump

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</tr>
</thead>
<tbody>
<tr>
<td>Byrne et al. (2017)</td>
<td>19 (0, 31)</td>
<td>Hurlers (23 ± 3)</td>
<td>2 session 48 hours apart. 2X1 DJ (0.30, 0.40, 0.50, 0.60m). 30sec rest between jumps, 1 min between jump heights</td>
<td>Hands on hips. Jump as fast as possible</td>
<td>JH - derived from ( t_f ) PTD; ( t_c ); ODH - where there was a plateau in JH; Peak RSI</td>
<td>ICC, CV</td>
<td>Inter-day reliability of ODH: ICC: 0.81, CV: 3.0%. Inter-day reliability of Peak RSI: ICC:0.87, CV: 4.2%</td>
<td>High</td>
</tr>
<tr>
<td>Stanton et al. (2017)</td>
<td>29 (10, 19)</td>
<td>Rec athletes (26 ± 5)</td>
<td>2 Session 7 days apart. 2X1 CMJ, DJ (0.30 m). 2 min rest between jumps</td>
<td>Hand on hips</td>
<td>JH derived from ( t_f ) (Jump-App vs FP)</td>
<td>ICC, Bland-Altman plots</td>
<td>DJ-ICC Between device: 0.99; Between days: 0.99</td>
<td>High</td>
</tr>
</tbody>
</table>

Where DJ is depth/drop jump, JH is jump height, \( t_f \) is flight time, PTD is photocell timing device, \( t_c \) is contact time, ODH is optimum drop height, RSI is reactive strength index, ICC is intra-class correlation coefficient, CV is coefficient of variation, Rec is Recreational, CMJ is countermovement jump, Jump-App is mobile telephone jump application, FP is force platform.
2.3.8.2 Depth jump protocol

The number of jumps performed during testing should be enough to generate accurate data but without causing fatigue (Ebben et al., 2008). The number of jumps used in reliability studies in relation to the DJ range from one (Arteaga-Ortiz et al., 2000) up to three (Flanagan et al., 2008; Beattie and Flanagan, 2015; Markwick et al., 2015). The predominant number of jumps is three (Flanagan et al., 2008; Beattie and Flanagan, 2015; Markwick et al., 2015). The rest between DJs should be long enough to allow recovery and prevent fatigue (Byrne et al., 2014). 30 seconds (Byrne et al., 2017) and three minutes rest period have been reported between jump height adjustment (Markwick et al., 2015). The most common rest time reported in the literature is one minute (Beattie and Flanagan, 2015; Markwick et al., 2015). The arm swing has been allowed in some studies (Arteaga-Ortiz et al., 2000; Flanagan et al., 2008). The majority of reliability studies instructed subjects to do a variation of ‘place hands on hips, step off, jump as high as possible when landing as if landing on a hot plate’ (Beattie and Flanagan, 2015; Byrne et al., 2017). One study used a polyvinyl pipe held across the shoulders to prevent arm swing (Markwick et al., 2015). The effects of arm action on DJ performance is under investigated. However, there a positive effect of arm movement in the CMJ (Slinde et al., 2008). Keeping the hands on the hips allows a more accurate measure of lower limb power. A protocol involving DJs should have enough jumps and recovery between jumps to allow appropriate data without causing fatigue. A protocol of three jumps with one-minute recovery is favoured in the literature. Preventing arm swing allows just the power of the lower limb to be measured.

2.3.9 Summary of jumping as an outcome measure

The CMJ is a measure of strength and power where the development of force is comprised predominantly of muscular components, in particular, the gluteal region and the quadriceps. The DJ is a measure of reactive strength where tendons are the main generator of propulsion. The TBs associated with TrPs may be as a result of sarcomeres being in a state of rigour due to the depletion of adenosine triphosphate. If treating TrPs releases the actin and myosin cross-bridge bond, then the released sarcomeres should be able to contribute to the force development required for jumping. Therefore, when
TrPs are treated it could be theorised that there should be an improvement in jump performance particularly in jumps dependent on muscular strength such as the CMJ.

2.4 Treatments

2.4.1 Dry needling
The concept of TrP and DN therapies began from injecting anaesthetic into a muscle to treat painful musculoskeletal conditions (Legge, 2014). The first real substantial research in the West was conducted by John H. Kellgren (1938) and his mentor Sir Thomas Lewis (1938) who noted referred pain after injecting a site with saline. The baton was passed to Jane Travell (Travell et al., 1942), who coined the term ‘Trigger points’, and her lifelong collaborator David G Simons (Travell and Simons, 1992; Simons et al., 1999). Both dedicated their careers to mapping the location of the main TrPs and the aetiology of TrPs (Simons, 2004a, 2004c). Brav and Sigmond (1941) conducted a study to determine whether it was the substance in a hypodermic needle or the needle itself which had the effect on TrPs. It transpired that the needle might be the positive factor. It was not until Lewit (1979) used acupuncture needles that DN, as we now use it, was used for myofascial pain. There have been three models of needling reported in the literature they are 1) Gunn’s radiculopathy model, 2) spinal segmental sensitisation model, and 3) TrP-DN model. TrP-DN is the most widely accepted model due to positive empirical results (Dommerholt et al., 2006) and is synonymous with the term DN. DN utilities intramuscular stimulation of contracted sarcomeres. Other terminology includes intramuscular manual therapy, deep DN, TrP acupuncture, paraspinal needling, intramuscular and nerve root needling, needle release, acupuncture needling and needling therapy (Dunning et al., 2014). Electrical impulses similar to transcutaneous electrical neural stimulation can be passed via the inserted needles, sometimes called electrical intramuscular stimulation or electroacupuncture which may intensify the Melzack and Wall (1965) pain gate theory (Zhang et al., 2014a). Superficial needling can also be used in compromising structures such as the scalenes (Dunning et al., 2014).

2.4.1.1 Theory of the principles of dry needling
Although the pathophysiology of TrPs remains unclear, muscle overload associated with repetitive and prolonged activities as well as low-level muscle contractions may produce
changes in the fibre structure, localised tissue stiffness, and the blood flow properties appear to be a factor, which may cause acidosis, elevated levels of inflammatory mediators, neuropeptides, and proinflammatory cytokines, which are typically associated with persistent pain and tenderness (Tough et al., 2010). DN targets neural, muscular, and connective tissue (Dunning et al., 2014). The mechanism behind DN is believed to be due to the rapid depolarization of the involved muscle fibres, which results in a LTR (Chen et al., 2001). The insertion of the needle may have a positive effect on central sensitisation (Hocking, 2010, 2013; Fernández-de-las-Peñas and Dommerholt, 2014). A meta-analysis of studies that mapped functional magnetic resonance imaging during acupuncture found that certain parts of the brain become activated during needle activity (association cortices, anterior cingulate cortex, thalamus, and insula); whereas other regions of the brain that became deactivated (amygdala, prefrontal cortex, posterior cingulate cortex, and caudate). Chen et al. (2001) suggest that needling of the bicep femoris of a rabbit reduced spontaneous electrical activity. Electroacupuncture across the joint line of the knee may have a pain gait effect similar to transcutaneous electrical neural stimulation (Melzack and Wall, 1965; Ng et al., 2003) or as a peripheral opioid mediator (Zhang et al., 2014a), especially when combined with nonsteroidal anti-inflammatory drugs such as Diclofenac (Sangdee et al., 2002).

### 2.4.1.2 Safety of dry needling

DN is considered to be safe and effective (Vulfsons et al., 2012). In a recent survey by Brady et al. (2014) of 7,629 DN treatments only a small proportion of adverse reactions were reported, they were: bleeding (7.55%), bruising (6.65%), pain during needling (3.01%), pain after needling (2.19%), and aggravation of symptoms (0.88%). This is similar to the findings of McDowell and Johnson (2014), who reported 184 cases of adverse reactions in all types of needling over a 15 year period. DN and sustained needling resulted in 26 (14.80%) of those cases. Major conditions were fainting (n = 11) and retained needles (n = 9). Minor adverse reactions were the most common of 31,822 treatments and include bleeding (3.10%), needling pain (1.10%), and aggravation of symptoms (9.60%). 70% of which reported subsequent improvement (White et al., 2001). Post-needling tenderness is a major consideration with DN (Ga et al., 2007; Martín-Pintado-Zugasti et al., 2015, 2016). It has been reported that the soreness after
DN can last up and up to 72 hours in latent TrPs (Martín-Pintado-Zugasti et al., 2016) and up to five days in patients with active TrPs (Hong, 1994). Post-needling sensitivity can affect lifestyle, work and social activates including sport (Martín-Pintado-Zugasti et al., 2016).

### 2.4.1.3 Dry needling as a treatment

DN as a treatment strategy is growing in popularity throughout Canada, Europe, South Africa, and the South Americas; but fails to gain traction in the USA and Australia, (Dommerholt et al., 2006). At present physical therapy state boards within the USA have a limited scope of the practice of DN (Dunning et al., 2014). In a meta-analysis, DN has been reported to be better than control or sham DN but not as effective as acupuncture or other forms of needling techniques in the short term in relation to DN in the neck and trapezius (Kietrys et al., 2013; Liu et al., 2015). Whereas in a meta-analysis of DN versus wet needling in the neck and trapezius, by Ong and Claydon (2014), it was reported that that initially wet needling was more effective, but better long-term effects were seen with DN. The instant improvement seen with wet needling may be a result of the medication used; while the manipulation of the needle in DN may cause the lasting effect. These mirror the findings of Tough and White (2011) who reported a better weighted mean difference for DN versus sham acupuncture or conservative treatment. Huang et al. (2011) suggest that DN has great potential for treating TrPs, especially when there are poor predictors of TrPs present, such as prolonged pain duration, high intensity of pain, poor quality of sleep, and or repetitive work is present. DN has a more prominent effect on portions of the brain associated with central sensitisation when compared to tactile pressure (Chae et al., 2013).

In a recent study into the effects of DN for the treatment of TrPs in the upper trapezius muscle, a 40.9-60.9% improvements in the VAS, 50.5% for the neck pain questionnaire, and 54.9%-57.4% in the PPT (p < 0.001) were reported up to one month after treatment (Pecos-Martín et al., 2015). DN has also be found to improve shoulder pain, and disability index score (p < 0.001), as well as the range of motion (p < 0.001), compared to conventional physiotherapy in adhesive capsulitis of the shoulder. However, that study only measured outcomes after one week (Sukumar and Lawrence, 2014).
Advice and exercise in conjunction with any needling technique can be 24.85-27.01% less expensive than advice and exercise rehabilitation alone (Whitehurst et al., 2011). Williamson et al. (2007) suggest the potential removal of patients from total knee arthroplasty operations would be substantial. The number of DN studies is too small to draw a definitive conclusion, and there is a need for high-quality randomised control trial with adequate follow-up (Dunning et al., 2014). Cotchett et al. (2010) recommend parallel-group randomised controlled trials, as well as using the Standards for Reporting Interventions in Controlled Trials of Acupuncture criteria.

2.4.1.4 The crossover of acupuncture, dry needling and wet needling

Although TrP therapy uses an acupuncture needle, the therapy is based on the theories of Western medicine (Kalichman and Vulfsons, 2010). The terms acupuncture literally translates to ‘needle penetration’ (‘acu’ = needle; ‘puncture’ = penetration) and has been used synonymously to describe DN clinically and within the literature. While the terminology, theoretical constructs, and philosophies are different, the actual procedure of inserting thin monofilament needles is similar to TrP-DN and acupuncture (Dunning et al., 2014). There is a strong correlation between the extraordinary acupuncture (Ah Shi) points and TrPs (Hong, 2000; Dommerholt et al., 2006); while Gazi et al. (2011) reported that acupuncture had been shown to reduce the number of TrP and pain intensity (p < 0.0001) in the neck and shoulder and is no different to TrP injection (wet needling) combined with cyclobenzaprine chloralhydrate and sodium dipypron (p > 0.05). There is moderate-quality evidence that acupuncture reduces pain compared with a control (Jamtvedt et al., 2008). Western acupuncture practitioners have conducted large-scale randomised controlled trials and have used western medical diagnoses such as plantar fasciitis, knee osteoarthritis, and carpal tunnel syndrome (Manyanga et al., 2014). However, it is suggested that the benefits of acupuncture as a treatment for osteoarthritis are uncertain (Fernandes et al., 2013; McAlindon et al., 2014; Bennell et al., 2014). Some authors advise against using acupuncture (Brown, 2013), whereas Hochberg et al. (2012) suggest, acupuncture may be an option for patients waiting for knee arthroplasty.
2.4.1.5 Dry needling in the lower limb

A systematic review of TrPs in the lower quarter (Morihisa et al., 2016) reported that there have only been four peer-reviewed RCT which investigated the effects of DN, they were in relation to posterior thigh pain (Huguenin et al., 2005), osteoarthritis and post total knee replacement surgery (Itoh et al., 2008b; Mayoral et al., 2013) and plantar heel pain (Cotchett et al., 2014), as well as two RCT of DN in relation to back pain (Macdonald et al., 1983; Edwards and Knowles, 2003). There have also been at least two master theses (Barry, 2015; Kennedy, 2015) which investigated the effects of TrP-DN on strength, muscle length, and performance in hip muscles.

2.4.1.6 Needling in the quadriceps

There is a substantial amount of research in relation to the effects of acupuncture in the quadriceps, in particular, for knee osteoarthritis (Corbett et al., 2013; Manyanga et al., 2014). In comparison, there is little research in relation to DN (Morihisa et al., 2016) or wet needling in the quadriceps (Yentür et al., 2003). A review of all peer-reviewed studies that report an insertion of a needle into the VM or VL is presented in Table 5 (p. 71).
<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Study type (Tx)</th>
<th>Number (male, female)</th>
<th>Subjects</th>
<th>Muscles / Acu Points</th>
<th>Outcome measures</th>
<th>Outcome time frame</th>
<th>sessions frequency (n)</th>
<th>Protocol (needle depth)</th>
<th>Control</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Christensen et al. (1992)</td>
<td>Phase 1: RCT (Acu), 16 weeks; Phase 2: RCT (Acu) of subjects from phase 1 with objective ↑, 34 additional weeks</td>
<td>Phase 1: 29 (9, 20); Phase 2: 17 (gender not specified)</td>
<td>Patients awaiting TKR</td>
<td>Local: Spl10 (VM), ST34 (VL), ST35, ST36, Xi yan; Distant: LI4</td>
<td>Subjective: Pain evaluation, VAS; Objective: HSS, walk 50, climb 20</td>
<td>Phase 1: subjective B-line, Weeks: 4, 6, 7, 8, 12, 14, 15, 16; objective: B-line, weeks 4, 8, 16; Phase 2: subjective: month 3 &amp; 6; Objective: week before each scheduled treatment &amp; every week of last month</td>
<td>Phase 1: subjective Acu group 3-4/7 @ week 2; Delayed Acu group 3-4/7 @ week 10; Phase 2 1/12 if needed</td>
<td>20 min with manual needle manipulation (10-15mm deep)</td>
<td>Delayed Acu (@ week 10)</td>
<td>Phase 1: between-group - Acu group ↑ objective measures 2-4 weeks (p&lt;0.01); Phase 2: within-group at 3 months - both groups ↑ objective measures compared to B-line (p&lt;0.01); within-group at 6 months - both groups ↑ objective measure compared to B-line (p&lt;0.05)</td>
</tr>
<tr>
<td>Takeda and Wessel (1994)</td>
<td>PRT (Acu)</td>
<td>40 (20, 20)</td>
<td>Knee OA patients</td>
<td>GB34, Spl9, ST35, Heding, Xiyan</td>
<td>McGill pain scale, PPT at knee joint line, WOMAC</td>
<td>B-line, weeks 3, 7</td>
<td>3/7 (9)</td>
<td>30 min manual manipulation every 5 min (&lt;30mm, deep enough to elicit de qi)</td>
<td>Non-Acu points VM, Med joint line, Lat joint line, Med and Lat area of the tibia</td>
<td>Within-group at 3 and 7 weeks - both groups ↑ McGill pain scale, WOMAC compared to B-line (p&lt;0.05). Within-group at 3 weeks - both groups ↑ PPT compared to B-line (p&lt;0.05). Between groups ↔ (p&gt;0.05)</td>
</tr>
</tbody>
</table>

Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee, - full list of acupuncture site are in Appendix A (p. Apx. ii). *De qi or de chi is a dull aching pain associated with acupuncture.

Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, ↑ is improvement, TKR is total knee replacement, Spl is spleen meridian, VM is vastus medialis, ST is stomach meridian, VL is vastus lateralis, Li is large intestines meridian, VAS is visual analogue scale, HSS is Hospital for Special Surgery knee score, Walk 50 is time to walk 50m, climb 20 is time to climb 20 steps, B-line is Baseline, PRT is prospective randomised trial, GB is gall bladder meridian, OA is osteoarthritis trial, PPT is pressure pain threshold, WOMAC is Western Ontario McMaster Universities osteoarthritis index, Med is medial, Lat is Lateral, ↔ is no difference.
### Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Study type (Tx)</th>
<th>Number (male, female)</th>
<th>Subjects</th>
<th>Muscles / Acu Points</th>
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<th>Outcome time frame</th>
<th>sessions frequency (n)</th>
<th>Protocol (needle depth)</th>
<th>Control</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Tillu et al. (2001)</td>
<td>PRT (Acu)</td>
<td>44 (9, 35)</td>
<td>Knee OA patients</td>
<td>Unilateral SpI9, SpI10 (VM), ST34 (VL), ST36</td>
<td>HSS, walk 50, climb 20, VAS</td>
<td>B-line, 2 months, 6 months</td>
<td>1/7 (6)</td>
<td>15 min with manual needle manipulation 4 times (deep)</td>
<td>Bilateral SpI9, SpI10 (VM), ST34 (VL), ST36</td>
<td>Both groups ↑ 0-2 months (p&lt;0.05) Both groups ↑ 2-6 months (p&gt;0.05) for all outcomes No between-group difference (p&gt;0.05)</td>
</tr>
<tr>
<td>Tillu et al. (2002)</td>
<td>Non-RCT (Acu)</td>
<td>60 (26, 34)</td>
<td>Knee OA patients</td>
<td>Local: SpI9, SpI10 (VM), ST34 (VL), ST36; Distant: LI4</td>
<td>HSS, walk 50, climb 20, VAS</td>
<td>B-line, 2 months</td>
<td>1/7 (6)</td>
<td>15 min with manual needle manipulation 4 times (deep)</td>
<td>No Acu</td>
<td>Acu group ↑ 0-2 months (p&lt;0.05) No Acu group ↓ 0-2 months (p&lt;0.05) Between-group difference, in favour of Acu (p&gt;0.05)</td>
</tr>
<tr>
<td>Yentür et al., (2003)</td>
<td>RCT (wet needling – Lid to TrPs.)</td>
<td>33 (0, 33)</td>
<td>Knee OA patients</td>
<td>Rec-Fem, VL, VM, sartorius, AddL, TFL, gracilis, pectineus, ilioopsoas, BF, Semitend, Semimem, AddM, Gastroc, soleus</td>
<td>ROM, Pain during: squatting, sitting down, ascending / descending, walking, taking off socks, getting in / out of the car</td>
<td>B-line, 3 weeks</td>
<td>1/7 (2)</td>
<td>0.5% Lid to any TrPs (deep enough to elicit LTR)</td>
<td>3X 2ml IA of Na-HA to knee joint &amp; Lid to TrPs</td>
<td>Within-group: Lid - all outcomes ↑ (P&lt;0.001), IA - squatting, walking (P&lt;0.05) Between-groups: Lid - all outcomes ↑ (P&lt;0.001)</td>
</tr>
</tbody>
</table>

Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee - full list of acupuncture site are in Appendix A (p. Apx. ii). De qi or de chi is a dull acheing pain associated with acupuncture.

Where Tx is treatment type, Acu is acupuncture, PRT partial randomised control trial, OA is osteoarthritis, SpI is spleen meridian, VM is vastus medialis, ST is stomach meridian, VL is vastus lateralis, HSS is Hospital for Special Surgery knee score, Walk 50 is time to walk 50m, climb 20 is time to climb 20 steps, VAS is visual analogue scale, B-line is Baseline, ↑ is improvement, RCT is randomised control trial, LI is large intestine meridian, ↓ is deterioration, Lid is 0.5% lidocaine, TrPs is Trigger points, Rec-Fem is rectus femoris, AddL is adductor longus, TFL is tensor fasciae latae, BF is biceps femoris, Semitend is semitendinosus, Semimen is semimembranosus, AddM is adductor magnus, Gastroc is gastrocnemius, ROM is range of motion, LTR is local twitch response, IA is intra-articular injection, Na-HA is Na-Hyaluronate.
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<tr>
<th>Author (year)</th>
<th>Study type</th>
<th>Number (male, female)</th>
<th>Subjects</th>
<th>Muscles / Acu Points</th>
<th>Outcome measures</th>
<th>Outcome time frame</th>
<th>sessions frequency (n)</th>
<th>Protocol (needle depth)</th>
<th>Control</th>
<th>Results</th>
</tr>
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<tbody>
<tr>
<td>Witt et al. (2005)</td>
<td>Stratified ratio 2:1:1 (Acu: Min Acu; Waiting list) RCT (Acu)</td>
<td>294 (99, 195)</td>
<td>Knee OA patients</td>
<td>Local: BL40, GB33 (VL), GB34, K10, L18, Spi9, Spi10(VM), ST34 (VL), ST35, ST36, Heding, Xiyan; Distant: BL20, BL57, BL58, BL60, K3, Spi4, Spi5, Spi6, ST6</td>
<td>WOMAC, SF-36</td>
<td>B-line, 8 weeks, 26 weeks, 52 weeks</td>
<td>weeks 1-4 - 2/7, weeks 5-8, 1/7 (12)</td>
<td>30 min with manual needle manipulation at least once (superficial)</td>
<td>Min Acu; Waiting list - no needling until week 8</td>
<td>At 8 weeks: (Acu Vs Min Acu) Acu ↑ WOMAC (p&lt;0.01); (Acu Vs waiting list) Acu ↑ WOMAC &amp; SF-36 (p&lt;0.05). At 26 weeks: (Acu Vs Min Acu) Acu ↑ WOMAC pain &amp; disability (p&lt;0.05); (Acu Vs waiting list) Acu ↑ WOMAC stiffness (p&lt;0.05)</td>
</tr>
<tr>
<td>Scharf et al. (2006)</td>
<td>RCT (Acu + Physio)</td>
<td>1,037 (344, 693)</td>
<td>Long-term knee OA patients</td>
<td>GB34, Spi9, Spi10(VM), ST34 (VL), ST36, Xiyan, + Any four Ah Shi points around the knee (not specified)</td>
<td>WOMAC, SF-12, Pat Sat</td>
<td>B-line, 13 weeks, 26 weeks</td>
<td>1-2/7 (10), +5 optional sessions at patient’s digression. All groups participated in 6 Physio sessions</td>
<td>20-30 min with manual needle manipulation twice (0.5-3.5mm)</td>
<td>Sham + physio, Meds + Physio</td>
<td>Acu + Physio Vs Meds + Physio: Acu + Physio ↑ (p&lt;0.01). Sham + Physio Vs Meds + Physio: Sham + Physio ↑ (p&lt;0.01). Acu + Physio Vs Sham + Physio: ↔ (p&gt;0.05)</td>
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Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee, Ah-shi points are acupuncture’s equivalent to trigger points - full list of acupuncture site are in Appendix A (p. Apx. ii). *De qi or de chi is a dull aching pain associated with acupuncture. Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, OA is osteoarthritis, BL is bladder meridian, GB is gall bladder meridian, VL is vastus lateralis, K is kidney meridian, LI is large intestine meridian, Spi is spleen meridian, ST is stomach meridian, VM is vastus medialis, WOMAC is Western Ontario McMaster Universities osteoarthritis index, SF-36 is modified version of the German Society for the Study of Pain survey, B-line is Baseline, Min Acu is minimal acupuncture, ↑ is improvement, Physio is physiotherapy, Meds is medication, ↔ is no difference.
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<tr>
<td>Williamson et al. (2007).</td>
<td>RCT (Acu)</td>
<td>181 (84, 97)</td>
<td>Patients awaiting TKR</td>
<td>Local: GB34, Sp19, Sp10 (VM), ST35, ST36, Xiyan; Distal: LV3; + 3 in any TrP or Acu point</td>
<td>OKS, WOMAC, VAS, HAD, Walk 50</td>
<td>B-line†, Week 1†, Week 12†, 3 months</td>
<td>1/7 (6)</td>
<td>60 min with needle in situ (deep enough to achieve <em>de chi</em>); Control (exercise and advice); Physio</td>
<td>Acu ↑ than Control and Physio for OKS after 7 weeks (p&lt;0.05)</td>
<td></td>
</tr>
<tr>
<td>Foster et al. (2007)</td>
<td>RCT (Acu + Physio)</td>
<td>339 (123, 216)</td>
<td>Knee OA patients</td>
<td>Local: GB34, Sp19, Sp10 (VM), ST34 (VL), ST35, ST36, Xiyan, any TrPs; or distal: LV4, TH5, SP16, LI3, ST44, KI3, BL60, GB41</td>
<td>WOMAC, Pat Sat</td>
<td>B-line, 2 weeks (phone call), 6 weeks, 6 months, 12 months</td>
<td>2/7 (6)</td>
<td>25-35 min with manual needle manipulation as appropriate (depth not specified); Physio not specified</td>
<td>Sham (blunt tip needle), Control (exercise and advice)</td>
<td>Control Vs Acu + Physio: ↔ (p&gt;0.05); Control Vs sham: pain &amp; function week 6 control ↑ (p&lt;0.05). Control Vs sham: pain &amp; function 6 months &amp; 12 months Control ↔ (p&gt;0.05)</td>
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Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee - full list of acupuncture site are in Appendix A (p. Apx. ii). *De qi or de chi is a dull aching pain associated with acupuncture †pre TKR.

Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, TKR is total knee replacement, GB is gall bladder meridian, Sp1 is spleen meridian, VM is vastus medialis, ST is stomach meridian, LV is liver meridian, TrP is Trigger point, OKS is Oxford knee score, WOMAC is Western Ontario McMaster Universities osteoarthritis index, VAS is visual analogue scale, HAD is hospital anxiety and depression score, Walk 50 is time to walk 50m, B-line is Baseline, Physio is physiotherapy, ↑ is improvement, OA is osteoarthritis, VL is vastus lateralis, BL is bladder meridian, LI is large intestine meridian, K is kidney meridian, Pat Sat is patient satisfaction.
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<th>Control</th>
<th>Protocol (needle depth)(^b)</th>
<th>Results</th>
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<tr>
<td>Itoh et al. (2008a)</td>
<td>RCT (Acu)</td>
<td>32 (11, 21) – data taken at B-line, gender of withdrawn patients not specified (n=8)</td>
<td>Knee OA patients</td>
<td>GB34, Sp19, Spl10 (VM), ST34 (VL), ST35, ST36</td>
<td>VAS, WOMAC</td>
<td>VAS: B-line, weeks 1-5, week 10; WOMAC: B-line, 5 weeks, 10 weeks</td>
<td>Control (analgesic deep heating liniment), TENS (settings: 122 Hz), Acu &amp; TENS</td>
<td>15 min as 5 min ‘sparrow pecking’ until de qi was felt, 10 min in situ</td>
<td>Between-groups Acu + TENS ↑ than all others for VAS (p&lt;0.001) &amp; WOMAC (p&lt;0.05) at week 5</td>
</tr>
<tr>
<td>Itoh et al. (2008b)</td>
<td>RCT (DN)</td>
<td>30 (3, 27)</td>
<td>Knee OA patients</td>
<td>Quads, iliopsoas, adductors, hamstrings, sartorius, Popliteus, GlutMin, others (not specified)</td>
<td>VAS, WOMAC</td>
<td>VAS: B-line, weeks 1-5, week 10; WOMAC: B-line, 5 weeks, 10 weeks</td>
<td>Sham DN as DN but with blunt needles and no penetration</td>
<td>'Sparrow pecking' until TR was elicited (10-30mm)</td>
<td>Within-group: VAS - ↑ TrP 1-5, 10 weeks (p&lt;0.001), ↑ Acu 2-4 weeks (p&lt;0.05), week 5 (p&lt;0.001); WOMAC - ↑ TrP 10 weeks (p&lt;0.05). Between-groups TrP ↑ than sham for VAS &amp; WOMAC (p&lt;0.05)</td>
</tr>
</tbody>
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Note: \(^a\)Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee - full list of acupuncture site are in Appendix A (p. Apx. ii). \(^b\)De qi or de chi is a dull aching pain associated with acupuncture.

Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, OA is osteoarthritis, GB is gall bladder meridian, Spl is spleen meridian, VM is vastus medialis, ST is stomach meridian, LV is liver meridian, VAS is visual analogue scale, WOMAC is Western Ontario McMaster Universities osteoarthritis index, B-line is Baseline, TENS is transcutaneous neuromuscular electrical stimulation, DN is dry needling, Quads is Quadriceps, GlutMin is gluteus minimus, LTR is local twitch response,
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<th>Protocol (needle depth)^b</th>
<th>Control</th>
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<tr>
<td>Suarez-Almazor et al. (2010)</td>
<td>Stratified RCT (E-Acu)</td>
<td>455 (117, 338)</td>
<td>Knee OA patients</td>
<td>True local: GB34, Sp9, Heding, Xiyan; true distal: Sp16, ear knee; Sham local: VL, Tib-Ant, Sham distal: FA-Ext</td>
<td>J-MAP, WOMAC, SF-12, VAS, ROM, TUG SKIP</td>
<td>B-line, Week 4, Month 3</td>
<td>Treatment duration 6 weeks (frequency and total session not specified).</td>
<td>20 min Max Tol E-Acu - settings: 15-50Hz; 20 Cycle/min; 5-60V (4-30mm)</td>
<td>Stratified Hi-Exp Vs Neut-Exp. Sham 0V after minimum V felt by the patient. Waiting list</td>
<td>By group: E-Acu Vs sham ↔ (p&gt;0.05), within-group: waiting ↑ J-MAP, WOMAC, VAS (p&lt;0.001). By Exp: Hi-Exp Vs Neut-Exp - Hi-Exp ↑ J-MAP, SKIP (p&lt;0.001).</td>
</tr>
<tr>
<td>Miller et al. (2011)</td>
<td>RCT (Acu + NSAIDs)</td>
<td>55 (16, 38)</td>
<td>Knee OA patients</td>
<td>Local: ST35, Heading, Xi Yan any As-Shi points; Distal: GB43 Contra, Sp15, Li11; Shu-stream: K3 or ST43; Local (depending on Shu-stream used) K10, or ST34 (VL)</td>
<td>KSS</td>
<td>B-line, Week 8, Week 12</td>
<td>2/7 (16)</td>
<td>20 min with manual needle manipulation every 5 min (&lt;30mm)</td>
<td>Sham Acu + NSAIDs</td>
<td>Within group: KSS - ↑ Acu + NSAIDs &amp; Sham Acu + NSAIDs (p&lt;0.05). By group: Acu + NSAIDs Vs Sham Acu + NSAIDs ↔ (p&gt;0.05)</td>
</tr>
</tbody>
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Note: ^Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee, Shu-stream points are points within meridians that are associated with joints, As-Shi points are acupuncture’s equivalent to trigger points - full list of acupuncture site are in Appendix A (p. Apx. ii). ^De qi or de chi is a dull aching pain associated with acupuncture. Where Tx is treatment type, (E-)Acu is (electro-)acupuncture, RCT is randomised control trial, OA is osteoarthritis, GB is gall bladder meridian, Sp is spleen meridian, VL is vastus lateralis, Tib-Ant is tibialis anterior, FA-Ext is forearm extensors, J-MAP is joint-specific multidimensional assessment of pain, WOMAC is Western Ontario McMaster Universities osteoarthritis index, SF-12 short form health survey, VAS is visual analogue scale, ROM is range of motion, TUG is timed up and go test, SKIP is satisfaction with knee procedure, B-line is Baseline, Max Tol is maximal tolerance achieved by patient, Hi-Exp is high expectation therapist phrases, Neut-Exp is neutral expectation therapist phrases, ↔ is no difference, ↑ is improvement, NSAIDs are nonsteroidal anti-inflammatory drugs, ST is stomach meridian, LI is large intestine meridian, K is kidney meridian, KSS is Knee Society score.
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<tr>
<td>Mavrommatis et al. (2012)</td>
<td>RCT (Acu/ E-Acu + Meds)</td>
<td>120 (29, 91)</td>
<td>Knee OA patients</td>
<td>Local: GB34, Sp9, Sp10 (VM), ST36; Heding, Xiyian; Distal: Li4, K3, Sp16, ST40</td>
<td>WOMAC, SF-36, VAS, PPT</td>
<td>B-line, Week 4, Week 8, Week 12</td>
<td>2/7 - E-Acu was used after 3 sessions (18)</td>
<td>20 min E-Acu - settings after week 3: 2-6 Hz 150ms (&lt;30mm, deep enough to elicit de qi)</td>
<td>Sham Acu + Meds (sham E-Acu was used after 3 sessions Meds)</td>
<td>Week 4 &amp; 8: Acu/ E-Acu + Meds ↑ than sham + Meds &amp; Meds for WOMAC, SF-36, VAS (p&lt;0.05). Week 12: Acu/ E-Acu + Meds ↑ than sham + Meds &amp; Meds for WOMAC, SF-36, VAS, PPT (p&lt;0.05)</td>
</tr>
<tr>
<td>Mikashima et al. (2012)</td>
<td>RCT (Acu + Meds)</td>
<td>80 (22, 58)</td>
<td>Post TKR patients</td>
<td>BL23, BL25, BL37, BL60, GB31 (VL), GB39, GB40, GB41, GB42, K13, Sp9, ST31, ST32 (VL), ST38</td>
<td>Ratio of VAS, Ratio knee swelling, Time to preoperative ROM</td>
<td>B-line, day 6, week 2, week 3</td>
<td>3/7 – day 7 to day 21 post-TKR</td>
<td>20 min in situ (10-15mm)</td>
<td>Meds</td>
<td>Ratio of VAS, Acu ↑ at weeks 2 &amp; 3 (p&lt;0.01) Ratio knee swelling weeks 2 &amp; 3 (p&lt;0.01) Time to preoperative ROM: Acu 15.2/7, control 20.9/7 (p&lt;0.01)</td>
</tr>
<tr>
<td>Soni et al. (2012)</td>
<td>RCT (Acu + physio)</td>
<td>56 (28, 28)</td>
<td>Patients awaiting TKR</td>
<td>LV3, LV8, GB34, Sp9, Sp10 (VM), ST34 (VL), up to 3 TrPs at therapist’s discretion</td>
<td>VAS, HAD, OKS</td>
<td>B-line, Week 6; month 3</td>
<td>1/7(4), 2/52 (2), 1/12 (~16')</td>
<td>Physio: isometric, concentric, &amp; functional quad exercises; calf stretches. Acu: 20 min in situ (&lt;25mm, deep enough to elicit de qi)</td>
<td>Exercise and advice leaflet</td>
<td>3 months post-TKR ↔ VAS, HAD, OKS. (p&gt;0.05)</td>
</tr>
</tbody>
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Note: *Heding or Heading is superior patella border, Xiyian or Xi Yan is both sides of patellar tendon of the knee - full list of acupuncture site are in Appendix A (p. Apx. ii). 1De qi or de chi is a dull aching pain associated with acupuncture pre TKR. 2Depending on TKR time frame. Where Tx is treatment type, (E-)Acu is (electro-)acupuncture, RCT is randomised control trial, Meds is medication, B-line is Baseline, OA is osteoarthritis, GB is gall bladder meridian, Sp is spleen meridian, VM is vastus medialis, LI is large intestine meridian, K is kidney meridian, ST is stomach meridian, WOMAC is Western Ontario McMaster Universities osteoarthritis index, , SF-36 is modified version of the German Society for the Study of Pain survey, VAS is visual analogue scale, PPT is pressure pain threshold, ↑ is improvement, TKR is total knee replacement, BL is bladder meridian, VL is vastus lateralis, ROM is range of motion, HAD is hospital anxiety and depression score, OKS is Oxford knee score, ↔ is no difference.
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<tr>
<td>Mayoral et al. (2013)</td>
<td>RCT (DN)</td>
<td>40 (11, 19)</td>
<td>Patients awaiting TKR</td>
<td>TFL, hip adductors, hamstrings, quadriceps, Gastroc, popliteus</td>
<td>VAS, WOMAC, ROM, strength</td>
<td>B-line, 1 month, 3 months, 6 months</td>
<td>Once, pre TKR (under local anaesthetic)</td>
<td>20 X fast-in-fast-out (deep enough to elicit LTR)</td>
<td>Sham under local anaesthetic &amp; blinded</td>
<td>1 month: DN ↑ VAS (p&lt;0.05); 3 &amp; 6 months: ↔ VAS (p&gt;0.05); ↔ WOMAC, ROM, strength (p&gt;0.05)</td>
</tr>
<tr>
<td>Ashraf et al. (2014)</td>
<td>Crossover trial (Acu)</td>
<td>40 (11, 29)</td>
<td>Knee OA patients</td>
<td>Local: ST35, ST36, Heding, Xiyang, Distal: BL60, GB34, GV20, ST44</td>
<td>VAS, WOMAC, Knee joint cartilage thickness</td>
<td>B-line, 6 weeks (4 weeks wash out)</td>
<td>3/7 (10)</td>
<td>30 min in situ (&lt;25mm)</td>
<td>Lateral wedge insole (2-5 mm for 1 month)</td>
<td>Between-groups ↔ is no difference (p&gt;0.05)</td>
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Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee, ear-knee is an auricular acupuncture point - full list of acupuncture site are in Appendix A (p. Apx. ii). *De qi or de chi is a dull aching pain associated with acupuncture.

Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, DN is dry needling, TKR is total knee replacement, TFL is tensor fasciae latae, Quads is quadriceps, Gastroc is gastrocnemius, VAS is visual analogue scale, WOMAC is Western Ontario McMaster Universities osteoarthritis index, ROM is range of motion, B-line is Baseline, LTR is local twitch response, ↑ is improvement, ↔ is no difference,
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<tr>
<td>Hinman et al. (2014)</td>
<td>Zelen-design RCT (Acu)</td>
<td>282 (143, 139)</td>
<td>Chronic knee pain (&gt;3 months)</td>
<td>4 Local: BL39, BL40, BL57, GB34, GB35, GB36, LV7, LV8, LV9 (VM), Spī9, Spī10 (VM), ST34 (VL), ST35, ST36, TrP in hamstrings; 2 Distal: BL60, GB41, LV3, Spī6, ST40; or Segmental: BL21, BL22, BL23, GB30, GB31; or General: BL11, GV14, GV20, LI11, ear-knee</td>
<td>NRS, WOMAC, AQoL6D, SF-12</td>
<td>B-line, 12 weeks; 1 year</td>
<td>1-2/7 (8-12)</td>
<td>20 min in situ (&lt;40mm)</td>
<td>LASER Acu (settings: 10mW, 0.2J/point), sham LASER Acu, control</td>
<td>Acu ↑ than control for NRS, WOMAC at 12 weeks (p&lt;0.05). LASER Acu ↑ than control at 12 weeks (p&lt;0.05)</td>
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Note: ⁿHeding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee, ear-knee is an auricular acupuncture point - full list of acupuncture site are in Appendix A (p. Apx. ii). ⁱDe qi or de chi is a dull aching pain associated with acupuncture.

Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, OA is osteoarthritis, ST is stomach meridian, BL is bladder meridian, GB is gall bladder meridian, GV is governing vessel meridian, VAS is visual analogue scale, WOMAC is Western Ontario McMaster Universities osteoarthritis index, B-line is Baseline, ↔ is no difference, LV is liver meridian, Spī is spleen meridian, VM is vastus medialis, VL is vastus lateralis, TrP is Trigger point, Li is large intestine meridian, NRS is pain numeric rating scale, AQoL6D is assessment of Quality of life instrument version 2, SF-12 short form health survey, LASER is light amplification by stimulated emission of radiation.
### Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Study type (Tx)</th>
<th>Number (male, female)</th>
<th>Subjects</th>
<th>Muscles / Acu Points</th>
<th>Outcome measures</th>
<th>Outcome time frame</th>
<th>sessions frequency (n)</th>
<th>Protocol (needle depth)</th>
<th>Control</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pavkovich (2015)</td>
<td>Case series (E-DN &amp; CPT)</td>
<td>4 (Gender not specified)</td>
<td>Chronic lateral hip and thigh pain of duration &gt;90 days</td>
<td>VL &amp; ITB region</td>
<td>VAS, LEFS</td>
<td>B-line, after Tx, 12.25 months post-Tx</td>
<td>1-2/7 (4-8)</td>
<td>6 needles placed 4 finger breadth apart left in situ 15 min (depth not specified)</td>
<td>N/A</td>
<td>↑ VAS, ↑ LEFS</td>
</tr>
<tr>
<td>Vas et al. (2014)</td>
<td>Case (PRF, DN, CPT, Meds)</td>
<td>2 (1, 1)</td>
<td>Chronic post-TKR pain</td>
<td>Hip adductors, hamstrings, Quads, sartorius, gracilis, Gastroc, popliteus</td>
<td>ROM, OKS, PHQ-9, S-LANSS, NRS</td>
<td>B-line, 15 days, 1 month, 3 months, 6 months</td>
<td>2/7 month 1, 1/7 months 2-3 (16)</td>
<td>Left in situ 30 min (&gt;2–3 mm)</td>
<td>N/A</td>
<td>↑ ROM, ↑ OKS, ↑ PHQ-9, ↑ S-LANSS, ↑ NRS</td>
</tr>
</tbody>
</table>

Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee - full list of acupuncture sites are in Appendix A (p. Apx. ii). **De qi or de chi is a dull aching pain associated with acupuncture.

Where Tx is treatment type, Acu is acupuncture, E-DN is electrotherapy muscle stimulation via dry needle, CPT is conventional physical therapy, VL is vastus lateralis, ITB is iliotibial band, VAS is visual analogue scale, LEFS is Lower Extremity Functional Scale, B-line is Baseline, N/A not applicable, ↑ is an improvement, PRF is ultrasonography-guided pulsed radiofrequency, Meds. is medication, TKR is total knee replacement surgery, Quads is quadriceps, Gastroc is gastrocnemius, ROM is range of motion, OKS is Oxford knee score, PHQ-9 is Patient Health Questionnaire-9, S-LANSS is Self-Administered Leeds Assessment of Neuropathic Symptoms and Signs, NRS is Numeric Rating Scale.
Table 5: Summary of peer-reviewed studies of the effects of needling in the vastus lateralis and or vastus medialis

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<th>Control</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ortega-Cebrian et al. (2016)</td>
<td>Single intervention. (DN)</td>
<td>20 (20, 0)</td>
<td>Advanced phase ACL rehab</td>
<td>VL, VM, Rec-Fem</td>
<td>Stiffness, decrement, resistance, &amp; creep (Myoton-Pro™); Passive &amp; Sub-Max. muscle activation (sEMG); Flx &amp; Ext ROM; VAS</td>
<td>Immediately after DN</td>
<td>Once</td>
<td>&lt; 60mm, until there were no more LTRs</td>
<td>N/A</td>
<td>↔ stiffness, ↔ decrement, ↔ resistance, ↔ creep, ↔ passive muscle activation, ↔ Sub-Max muscle activation, ↑ Flx ROM, ↔ Ext ROM, ↑ VAS</td>
</tr>
</tbody>
</table>

Espí-López et al. (2017) | RCT (MT+EX+DN vs. MT+EX) | 60 (31, 29) | Subjects with PFPS | VL, VM | KOOS, KSS, IKDC | B-line, 1 week, 3 months | 3/7 (3) | Fast-in-fast-out (deep enough to elicit RP to knee) Most painful TrPs in VL and VM | N/A | Within group: MT+EX+DN @1 Week & 3 months ↑ KOOS, KSS, IKDC (p<0.05); MT+EX @1 Week & 3 months ↑ KOOS, KSS, IKDC (p<0.05); Between groups: 1 week: ↔ KOOS, KSS, IKDC (p<0.05); 3 months: ↔ KOOS, KSS, IKDC (p<0.05) |

Note: *Heding or Heading is superior patella border, Xiyan or Xi Yan is both sides of patellar tendon of the knee - full list of acupuncture sites are in Appendix A (p. Apx. ii). **De qi or de chi is a dull aching pain associated with acupuncture. Where Tx is treatment type, Acu is acupuncture, RCT is randomised control trial, DN is dry needling, ACL Rehab is anterior cruciate ligament rehabilitation, VL is vastus lateralis, VM is vastus medialis, Rec-Fem is rectus femoris, Sub-Max is sub maximal, sEMG is surface electromyography, Flx is flexion, Ext is extension, LTR is local twitch response, ↔ is no difference, ↑ is improvement, MT is manual therapy, EX is exercise, PFPS is patellofemoral pain syndrome, KOOS is Knee Injury and Osteoarthritis Outcome Score, KSS is Knee Society Score, IKDC is International Knee Documentation Committee, B-line is Baseline, TrPs is Trigger points, RP is referred pain.
Itoh et al. (2008b) investigated the effects of acupuncture and DN on pain relief in elderly patients with knee osteoarthritis. The DN group had significant improvements compared with the sham group for both VAS (p < 0.001) and WOMAC (p < 0.001). The benefits of DN lasted longer (ten weeks) compared to acupuncture (four weeks). These improvements in VAS were similar to the findings of Mayoral et al. (2013) who used DN on patients, while under anaesthetic, who were about to undergo total knee arthroplasty (p = 0.04). However, the results were short-lived, and there was not a significant improvement in the WOMAC, the range of motion or strength (p > 0.05). This may be due to there being only one treatment session. Whereas, DN of the gluteus of 52 male athletes with pain in the posterior thigh did not improve straight leg raise, internal rotation of the hip, nor VAS (Huguenin et al., 2005). The results of that study were partly attributed to not using multiple treatments modalities and not utilising multiple treatments (Huguenin et al., 2005). DN of the calf and foot muscle resulted in a reduction in plantar heel pain at the first step (Cotchett et al., 2014) but was not within the minimum important difference needed to be considered clinically relevant. Yentür et al. (2003) investigated the effects of intra-articular injection of Na-hyaluronate into the knee of patients with knee osteoarthritis and 0.5% lidocaine injection to TrPs (wet needling) of muscles associated with the knee compared to intra-articular injection only. The wet needling and intra-articular injection were effective in improving range of motion, pain during squatting, walking, ascending and descending, as well as taking off socks (p < 0.001). While intra-articular injections during that study on their own, improved pain during squatting and walking (p < 0.05), thus suggesting that TrPs play a significant role in the pain perceived by patients with knee osteoarthritis.

Acupuncture points which are in the VL include the stomach meridian points 32-34 and gall bladder meridian points 31 and 32; while in the VM, the acupuncture points are the liver meridian point-9 and spleen meridian points-10 and 11 (Urbanski, 2007). Acupuncture has been reported to be an effective treatment method for osteoarthritis compared to control (Christensen et al., 1992; Williamson et al., 2007; Itoh et al., 2008a), and minimal acupuncture (Witt et al., 2005); whereas it has been found that there was no significant difference in acupuncture and physiotherapy (Foster et al., 2007) or acupuncture and physiotherapy versus exercise and advice in the form of an information
leaflet (Soni et al., 2012). Acupuncture in conjunction with other therapies such as physiotherapy (Scharf et al., 2006) or nonsteroidal anti-inflammatory drugs (Miller et al., 2011) appears to be no better than a sham treatment in combination with the other therapies, respectively. The positive effects of DN on the VAS and WOMAC scores of knee osteoarthritis patients may manifest sooner and last longer than those of acupuncture (Itoh et al., 2008b).

In summary, there is a substantial void in the literature in relation to DN, especially in the lower limb. Therefore more high-quality randomised control trials are needed. It is imperative to treat all muscle that can refer to a given area. Multiple treatment sessions to replicate clinical situations should be considered, thus making studies generalizable. Needling may be an effective pain reliever for osteoarthritis patients awaiting total knee arthroplasty, but there are vast discrepancies in treatment sites, treatment protocol, and the number of treatments reported in the literature.

### 2.4.1.7 Needling protocol

There have only been three studies (Itoh et al., 2008b; Mayoral et al., 2013; Espí-López et al., 2017) and at least two master’s theses (Barry, 2015; Devereux, 2016) that investigated the effects of DN to the quadriceps on which to base needling protocol. The variables, especially in the VL and VM, which should be considered are the size of needle, needling technique, duration of needling, number of sites to be treated, number of sessions and time between sessions.

#### 2.4.1.7.1 Needle size

Due to the depth of the quadriceps, the needle should be long enough to penetrate the deepest muscle fibres. Both Itoh et al. (2008b) and Mayoral et al. (2013) used needles 50mm in length. However, the subjects were predominantly females with knee osteoarthritis with a mean age ranging from 70.5 to 74.2 years. Therefore, may have atrophy associated with osteoarthritis (Wyatt et al., 2001). The needles used should be long enough to reach all of the muscle fibres.

#### 2.4.1.7.2 Needling technique

Acupuncture consists of placing the needle in specific points and manipulating the needle until there is a dull aching sensation called de-qi. The needle is then left in situ.
for up to five minutes and is then manipulated again until *de-*qi is achieved again. The “fast-in-fast-out” technique of DN may be a faster version of this process thus explaining improvements in a shorter time frame (Tsai et al., 2010; Gerber et al., 2015). The fast-in-fast-out technique is used in a fan or cone shape and is the most effective technique to ensure that a TrP is penetrated. It also elicits as many LTRs as possible (Hong and Hsueh, 1996; Hong, 2000; Hsieh et al., 2012; Dunning et al., 2014; Salom-Moreno et al., 2014; Pecos-Martín et al., 2015).

### 2.4.1.7.3 Needling timeframe

The number of fast-in-fast-out penetrations has been standardised to 8-10 times (Pecos-Martín et al., 2015) and up to 20 times (Mayoral et al., 2013) in some DN randomised control trials to prevent any treatment variability, whereas in another study (Hsieh et al., 2012) the sensitive loci within the TrP were stimulated until the LTR was exhausted. Other studies stimulate the TrP for 25-30 seconds (Salom-Moreno et al., 2014) or as long as 2 minutes (Barry, 2015); whereas the typical acupuncture session can last 20 minutes (Tukmachi et al., 2004) or up to 2 hours (Berman et al., 2004). In order to stimulate all sensitive loci, a time frame standardisation should be used. This should be done until the LTR has ceased, whereupon subsequent loci within the TB should be stimulated. Future studies should try to use a standardised treatment of two minutes for each TrP.

### 2.4.1.7.4 The number of needling sites

Painful muscles and connective tissue appeared to co-manifest with osteoarthritis (Galletti et al., 1990). While there is not enough evidence to suggest that TrPs causes knee osteoarthritis the use of needling does appear to improve the pain felt by osteoarthritis patients. There are several acupuncture studies in relation to osteoarthritis in the knee that did not insert a needle into the VM or VL (Berman et al., 1999, 2004; Ng et al., 2003; Tukmachi et al., 2004; Vas et al., 2004). Witt et al. (2006) reported that there are at least 30 acupuncture sites used for knee osteoarthritis as reported in 15 randomised control trials, 53.3% of which use stomach meridian point-34 (VL) and 60.0% of which used spleen meridian point-10 (VM).

Mayoral et al. (2013) investigated the effects of TrPs in the tensor fasciae latae, hip adductors, hamstrings, quadiceps, gastrocnemius and popliteus in patients about to complete total knee replacement. Whereas Itoh et al. (2008b) also treated the iliopsoas,
sartorius, and gluteus minimus. Mayoral et al. (2013) found that one month after surgery VAS was lower in the DN group (p = 0.003). But there was no significant difference in VAS at the 3 or 6 months follow-up. Itoh et al. (2008b) on the other hand, reported lower WOMAC score for the TrP group after 10 weeks (p < 0.001). All needling studies in relation to the lower limb treat multiple sites, whereas acupuncture treats multiple sites locally and distally. It cannot be determined which TrP is specifically responsible for a pain of return of function if multiple TrPs are treated. It is unclear whether the effects reported in relation to acupuncture are as a result of manipulating the local channel points, the distal points, *Ah shi* points, or a combination of all the points being stimulated. Studies that inserted needles into the VL or VM reported positive effects in relation to knee pain and function. Which specific TrP is responsible is unclear, but multiple sites would be optimal.

It has been reported that unilateral acupuncture is as effective as bilateral acupuncture (Tillu et al., 2001) for the Hospital for Special Surgery knee score, time to walk 50m, time to climb 20 steps, and VAS after 2 months. Audette et al. (2004) suggest that mirrored LTR during unilateral TrP-DN is a result of a central component in the epidemiology of TrPs. Therefore bilateral treatments should be used.

### 2.4.1.7.5 Number of sessions

The treatment course of acupuncture is prolonged, sometimes up to 26 weeks (Berman et al., 2004). The number of treatments for TrP-DN is as few as one session (Mayoral et al., 2013) and up to five meetings (Itoh et al., 2012). Session frequency can be from three times a week (DiLorenzo et al., 2004; Ilbuldu et al., 2004; Itoh et al., 2004a, 2006, 2007) or as infrequent as once a month (Soni et al., 2012). Shorter treatment courses may prevent poor appointment adherence (Berman et al., 2004). Three times a week is optimal for effective treatment and to replicate clinical practice (ISCP, 2012).

### 2.4.1.7.6 Time between session

The biggest concern in relation to treating TrPs to improve athletic performance is the effects of post-needling soreness. Post-needling soreness can last up to 72 hours (Martín-Pintado-Zugasti et al., 2014, 2015, 2016). There have at least three masters studies that have investigated the short-term effects of DN on athletic performance, and have reported reduced jump (Kennedy, 2015; Devereux, 2016) and running
performance (Barry, 2015), and has been attributed to post-needling soreness. The mechanism behind post-needling soreness is not understood but may be due to the release of intercellular fluid such as creatine kinase from the needle penetrating the muscle cell wall which would have similar effects as delay onset of muscle soreness, which incidentally take 72 hours to subside (Cheung et al., 2003). If post-needling soreness is akin to delayed onset of muscle soreness you would expect to see significant reductions in strength and power parameters during delayed onset of muscle soreness especially in the legs (Eston et al., 1996), and altered recruitment patterns (Edgerton et al., 1996; Cheung et al., 2003). Therefore, 72 hours between treatment sessions should be observed. Also, there should be more than 72 hours between the final treatment session and the final follow-up session.

2.4.1.8 Summary of needling protocol
There are substantial differences between the treatment protocols for DN and acupuncture. The needle has to be long enough to stimulate the deepest muscle fibre. The fast-in-fast-out technique is the most effective unless it causes the patient too much pain and discomfort, in which case, leaving the needle in situ is best. The ideal length of time for treating a single TrP is until the LTR is exacerbated. However, in experimental conditions, a specified period should be implemented in order to reduce variability. Some acupuncture studies in relation to osteoarthritis of the knee did not place needles in the thigh. Any study that investigates the effect of needling on the function and pain associated with the knee should include muscle that stabilises the knee joint and govern patella tracking such as the VL and VM. Three sessions replicate clinical practice and offer maximum exposure to a treatment modality.

2.4.2 Summary of dry needling
DN is different to acupuncture but may have a significant overlap. The standing hypothesis in relation to DN is that the insertion of the needle into the neuro-muscular junction stops the excessive calcium resulting from one leg sport-specific vertical jump proliferation. All forms of needling, when appropriate safety considerations are followed, are safe and have few serious adverse reactions. DN is a favoured and effective treatment of TrPs in patients with pain in the neck and shoulders. However, the number of quality long-term randomised control trials, especially in the lower limb is limited.
TrPs may affect physical performance. There are no peer-reviewed studies on the effects of DN on performance, Masters theses (Barry, 2015; Kennedy, 2015; Devereux, 2016) have investigated the short-term effects of DN latent TrPs vis à vis jump and running performance, and have found that DN can have a negative effect on jump performance up to 72 hours (Devereux, 2016), possibly due to post-needling soreness. The use of DN as a treatment of TrPs in the quadriceps should consider the appropriate needle length, stimulating technique, duration of therapy time, muscles to be treated, the number of treatment sessions, the transient effect on performance, as well as treating subjects bilaterally.

2.4.3 Shockwave

Shockwaves are Infrasound, three-dimensional pressure pulses lasting microseconds in duration, with peak pressures of 35-120 MPa (Sheveleva et al., 2010; Wang, 2012; Speed, 2013). Extracorporeal shockwave therapy (ESWT) was first used for lithotripsy of kidney and bladder stones in the 1940s (Schmitz et al., 2015). During in vitro studies, it was noted that the ESWT had a positive cellular effect on orthopaedic structures and conditions such as disturbances in bone healing, tendinopathy, spasticity, chronic skin ulcers, bone vascular diseases, myocardial ischaemia and dental conditions (Romeo et al., 2013). Excellent clinical results have been reported for plantar fasciitis, Achilles tendinopathy, non-calcific supraspinatus tendinopathy and calcifying tendonitis of the shoulder (Speed, 2013).

2.4.3.1 Types of shockwave therapy

The more traditional form of medical ESWT involves focused-ESWT (fESWT). The shockwaves are concentrated into small focal areas of 2–8mm and deliver an energy flux density (mJ·mm$^{-2}$) to a focused site within the target tissue (Wang, 2012; Speed, 2013; Schmitz et al., 2015). There is another form of shockwave called radial extracorporeal shockwave therapy (rESWT), however, ‘radial pulse therapy’ is a more descriptive term (Speed, 2013). The pressure waves dissipate the deeper the wave pass through tissue (Schmitz et al., 2015).

rESWT typically has a peak pressure of 0.4-0.5 MPa compared to 50 MPa for fESWT. Both have a uni-phasic waveform; whereas ultrasound is bi-phasic and has a peak pressure of
0.05 MPa (Wang, 2012). Other measures are bar and mJ-mm\(^{-2}\) (Equation 5). The distinction between radial ESWT as ‘low-energy ESWT’ and focused ESWT as ‘high-energy ESWT’ is not correct and should be abandoned, and there is no scientific evidence in favour of either rESWT or fESWT with respect to treatment outcome (Schmitz et al., 2015). Speed (2013) suggest that fESWT and rESWT should be considered as different treatment modalities.

\[
0.05 \, mJ \cdot mm^{-2} = 1 \, bar = 0.1 \, MPa
\]

Equation 5: Energy flux density conversion.

There is some debate as to whether rESWT is as effective as fESWT to treat TrPs. Using rESWT may be severely limited as the rapid decay of the pressure wave away from the emitter (Simons 2004). This does not seem to be the case as a positive result has been reported with the use of rESWT (Damian and Zalpour, 2011; Cho et al., 2012; Gür et al., 2014). This may be due to the superficial nature of the TrPs in the trapezius. The mechanism of shockwave therapy is not entirely understood but is believed to microscopically cause interstitial and extracellular responses leading to tissue regeneration (Shevelova et al., 2010; Wang, 2012; Romeo et al., 2013; Speed, 2013; Ramon et al., 2015; Schmitz et al., 2015).

2.4.3.2 The theory of the physiological effect of shockwave therapy

The mechanism behind the effects of ESWT in relation to TrPs (Jeon et al., 2012; Wong et al., 2017) and muscles with spasticity (Manganotti and Amelio, 2005; Moon et al., 2013) is unclear. The proposed effect of fESWT is at a molecular-biological and cellular level (Müller-Ehrenberg and Licht, 2005; Jeon et al., 2012). It is believed to microscopically cause interstitial and extracellular responses leading to tissue regeneration, such as neovascularization and upregulation of angiogenetic growth factors which lead to the improved blood supply (Wang, 2012).

2.4.3.2.1 Molecular

ESWT is believed to suppress substance P and other inflammatory markers (Müller-Ehrenberg and Licht, 2005). During in vitro studies of ESWT to the distal femurs of rabbits, with internal control, the presence of substance P and prostaglandin-E2 was assessed at 6 hours, 24 hours and 6 weeks. After 6 successive eluations, there were
higher concentrations of substance P in the treated limb at 6 and 24 hours but less after six weeks; whereas there was no change in relation to prostaglandin-E2 (Maier et al., 2003). Substance P is found in unmyelinated C-fibres and lightly myelinated A-δ-fibres (Schelling et al., 1994). A reduction in substance P may prevent a stimulation of the C- and A-δ-nociceptor fibres. Possibly preventing a positive feedback loop which is believed to cause central sensitisation (Jeon et al., 2012).

2.4.3.2.2 Vascular

Another effect of ESWT is thought to be a result of neovascularization or cell membrane permeability (Sheveleva et al., 2010; Gür et al., 2014). fESWT is believed to alter pain signal, by promoting angiogenesis and increase perfusion of ischemic tissues (Ji et al., 2012; Ramon et al., 2015). Improved blood perfusion has been reported after ESWT (Kisch et al., 2016). ESWT may improve angiogenic factors in stem cells and protect cells from apoptosis (Zhang et al., 2014b). In an in vivo study (Kisch et al., 2016), single high energy dose (10 J) and three repetitive doses of high energy dose (30 J) fESWT on blood flow of muscular was investigated. There was an increase in blood flow in both groups (p < 0.05). There was an accumulation effect in the repetitive dose group. Improvement lasted up to 5 minutes after the second dose and up to 10 minutes after the third dose.

ESWT may cause a thixotropy effect of stimulating enzymatic and nonenzymatic nitric oxide synthesis, which may cause vasodilatation and result in muscular proliferation (Amelio and Manganotti, 2010; Moon et al., 2013; Santamato and Micello, 2014). 0.03mJ/mm² is a sufficient energy flux to produce nonenzymatic nitric oxide in human umbilical vein endothelial cells and rat glioma C6 cells which have an anti-inflammatory effect (Mariotto et al., 2005; Ciampa et al., 2005). 1000 pulses may be an optimum number of pulse to provoke nonenzymatic nitric oxide synthesis (Maier et al., 2003). ESWT may cause reformation of blood vessels via nonenzymatic nitric oxide or other means, which facilitate connective tissue recovery, resulting in the stimulation of adenosine triphosphate production and remove sensitising inflammatory markers (Cho et al., 2012).

2.4.3.2.3 Neural

It is suggested that ESWT affects the neuromotor junction, during in vitro investigations at least (Kenmoku et al., 2012). While the number of one leg sport-specific vertical jump
receptors are reduced, there is evidence that ESWT does not affect EMG F-waves, which are antidromically reactivations of the effort of neuro-pathways of muscles with spasticity in patients with cerebral palsy or stroke (Manganotti and Amelio, 2005; Sohn et al., 2011). This might explain the recent data, that suggests ESWT might slow down muscle atrophy after an acute nerve injury activating the damaged nerve (Lee and Cho, 2013). In a pair of studies, Hausdorf et al., (2008a, 2008b) investigated the molecular and cellular mechanisms behind ESWT. It is purposed that the analgesic effect is a result of ESWT de-innervating unmyelinated nerve endings due to cavitation (Schelling et al., 1994). The effects of fESWT (0.09mJ/mm²) on the femoral nerve and sciatic nerve, when targeted at the anterior femur of rabbits, was a reduction in the density (57%) and number (59%) of unmyelinated nerve fibres in the femoral nerve (p < 0.001), but there was not a reduction of the estimated number of neurons at the L5-L7 dorsal root ganglia. These studies suggest that there was a significant difference in the estimated number of neurons immunoreactive for substance P (p < 0.001). These studies indicate that ESWT may de-innervate the A-δ-type and C-type nerve that transmit pain signal via the neurotransmitter substance P.

ESWT has been shown to effects A-δ-type and C-type nerve fibres in vitro. However, there is little evidence at present to confirm or refute this hypothesis in human subjects. The effects of ESWT can last up to four weeks, how this effect might transfer to the TrP is still uncertain.

2.4.3.2.4 Other theories

Another proposed theory is that the mechanism of the ESWT breaks up the actin-myosin links (Gleitz and Hornig, 2012; Ramon et al., 2015). There are no studies, to the author’s knowledge, that have investigated this hypothesis and it is purely theoretical at present and is difficult to accept the hypothesis considering that the actin-myosin decoupling is moderated by the availability of adenosine triphosphate (McArdle et al., 2009).

2.4.3.3 Summary of the theory of physiological effects of shockwave therapy

It is unclear if ESWT directly affects the cellular structure or whether it promotes synthesis of molecules which modulate cell proliferation and metabolism. ESWT may appear to prevent the afferent pain single to the brain by de-innervating neurones.

Walsh, R. (2017)
associated with the neurotransmitter substance P, while not affecting the efferent motor nerve fibres. If this theory holds true then treating TrPs with ESWT breaks the positive feedback loop before the possible centralisation of the pain associated with TrP can manifest. Other treatments for TrPs rely on the depolarisation of the motor-neuron junction to suppress the positive feedback loop which may be difficult if there is central sensitisation. ESWT may offer benefits to patients with fatigue-induced muscle disorders, delayed-onset muscle soreness, spine-related neuromuscular muscle disorders, muscle-related neuromuscular muscle disorders, and moderate-to-subtotal muscle tears due to the release of inflammatory markers, in particular, substance P. The proliferation theorised in the vascular model may also improve recovery in athletes due to the increase in blood flow bringing with it oxygen and thus an increase of adenosine triphosphate.

### 2.4.3.4 Shockwave therapy for musculoskeletal pathologies

ESWT has become a favoured conservative treatment option for an array of orthopaedic conditions. The majority of the research involves plantar fasciitis (Wang et al., 2002; Weil et al., 2002; Ogden et al., 2002; Malay et al., 2006; Wang et al., 2006; Tornese et al., 2008; Yucel et al., 2010; Ilieva, 2013; Kim et al., 2015), Achilles tendinopathy (Furia, 2008; Fridman et al., 2008; Rasmussen et al., 2008; Vulpiani et al., 2009; Magnussen et al., 2009; Yoo et al., 2012), and lateral epicondylitis (Rompe et al., 2001; Jih-Yang et al., 2001; Speed et al., 2002; Rompe, 2004; Chung et al., 2005; Ilieva et al., 2012).

### 2.4.3.5 Shockwave therapy in relation to trigger points

There is some literature beginning to emerge into the effects of ESWT on TrPs (Cho et al., 2012; Jeon et al., 2012; Ji et al., 2012; Müller-Ehrenberg and Licht, 2005; Bauermeister, 2007; Damian and Zalpour, 2011; Zhao et al., 2013; Gür et al., 2014; Moghtaderi et al., 2014). There are seven peer-reviewed articles in relation to the use of ESWT for the treatment of TrPs Simons (2004) reports of two abstracts using ESWT as a tool for the diagnosis of TrPs. Suggesting that ESWT could be used to confirm the symptom reproduction of TrPs. It is unclear whether this would be better than palpation techniques to confirm the presence of TrPs, as there have not been any reliability studies conducted to collaborate or confirm those suggestions. The majority of the relevant literature is in relation to TrPs in the neck and shoulder (Damian and Zalpour, 2011; Cho
et al., 2012; Gleitz and Hornig, 2012; Jeon et al., 2012; Ji et al., 2012). With more recent reports in relation to the lower limb (Zhao et al., 2013; Moghtaderi et al., 2014). A full review of the peer-reviewed literature is presented in Table 6 (p. 93).
#### Table 6: Summary of peer-reviewed studies of the effects of extracorporeal shockwave therapy on trigger points

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Study type</th>
<th>Number (male, female)</th>
<th>Subjects</th>
<th>Muscles</th>
<th>Outcome measures</th>
<th>Outcome time frame</th>
<th>Mean sessions (range)</th>
<th>Pules</th>
<th>ESWT type (energy density)</th>
<th>Stats</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Müller-Ehrenberg and Licht (2005)</td>
<td>Case</td>
<td>30 (21, 9)</td>
<td>Patients with acute, sub-acute, chronic TrP</td>
<td>All the TrP that refer to spine, elbow, heel.</td>
<td>VAS at rest, VAS with activity, PR, RP</td>
<td>B-Line, 3 months</td>
<td>7.3 (2-16)</td>
<td>800-1000</td>
<td>fESWT (0.04-0.26 mJ/mm², 6Hz)</td>
<td>t-test</td>
<td>All outcomes - ↑ (p&lt;0.001)</td>
</tr>
<tr>
<td>Damian and Zalpour (2011)</td>
<td>RCT: ESWT + massage + Stretching Vs massage + Stretching</td>
<td>26 (10, 16)</td>
<td>Musicians with shoulder-neck pain &gt;6 months</td>
<td>Temporalis, masseter, Traps, sternocleidomastoid, rhomboids</td>
<td>VAS, neck ROM, CC°, PPT, SPADI, NPD IQ</td>
<td>B-Line, Pre and Post Tx @ Weeks 1, 2, 3, 4, 5*</td>
<td>1/7 5-6 weeks*</td>
<td>N/A</td>
<td>rESWT (N/A)</td>
<td>t-test, MW test</td>
<td>VAS - ↑ (p&lt;0.001), neck ROM - ↔, CC° - ↔, PPT - ↔, SPADI - ↑ (p&lt;0.01), NPD IQ - ↑ (p&lt;0.01)</td>
</tr>
<tr>
<td>Cho et al. (2012)</td>
<td>RCT: SSE+ESWT Vs ESWT Vs SSE</td>
<td>36 (N/A)</td>
<td>TrPs in shoulder-neck</td>
<td>Upper trapezius</td>
<td>VAS, PPT, NDI, CMS</td>
<td>B-Line, 4 weeks</td>
<td>1 Tx</td>
<td>1000</td>
<td>rESWT (0.12 mJ/mm²)</td>
<td>1-way ANOVA</td>
<td>VAS: SSE+ESWT, ESWT, SSE ↑ (p&lt;0.05); PPT: ESWT, ↑ (p&lt;0.05); NDI: SSE+ESWT, ESWT, SSE ↑ (p&lt;0.05); CMS: SSE+ESWT, ESWT, SSE ↑ (p&lt;0.05)</td>
</tr>
</tbody>
</table>

Note: *if a subject missed an appointment treatment was extended by 1 week.
Where TrP is trigger points, VAS is visual analogue scale, PR is pain recognition, RP is referred pain, B-Line is Baseline, (f- is focused)(r- is radial)ESWT is extracorporeal shockwave therapy, t-test is paired sample t-test, ↑ is improvement, RCT is randomised control study, Traps is trapezius, CC° is cranio-cervical range of motion, ROM is range of motion, PPT is pressure pain threshold, SPADI is shoulder pain and disability index, NPD IQ is neck pain disability index questionnaire, Tx is treatment, MW- test is Mann-Whitney test, SSE is shoulder stabilisation exercises, NDI is neck disability index, CMS is constant Murley scale, Tx is treatment, ANOVA is analysis of variance.
## Table 6: Summary of peer-reviewed studies of the effects of extracorporeal shockwave therapy on trigger points

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Study type</th>
<th>Number (male, female)</th>
<th>Subjects</th>
<th>Muscles</th>
<th>Outcome measures</th>
<th>Outcome time frame</th>
<th>Mean sessions (range)</th>
<th>Pules</th>
<th>ESWT type (energy density)</th>
<th>Stats</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jeon et al. (2012)</td>
<td>RCT: ESWT Vs TrPI + TENS</td>
<td>30 (22, 8)</td>
<td>MPS in Traps</td>
<td>Traps</td>
<td>McGill-PQ, VAS, PPT, neck ROM</td>
<td>B-Line, week 1, week 4</td>
<td>1/7 3 weeks</td>
<td>1500</td>
<td>fESWT (0.10 mJ·mm⁻²)</td>
<td>rANOVA</td>
<td>Between-group: VAS - TrPI↑ (p&lt;0.05); PPT - TrPI↑ (p&lt;0.05). Within-group: McGill-PQ, VAS, Neck ROM ↑ (p&lt;0.05)</td>
</tr>
<tr>
<td>Ji et al. (2012)</td>
<td>RCT: ESWT Vs Placebo</td>
<td>20 (3, 17)</td>
<td>MPS in Traps</td>
<td>Traps</td>
<td>VAS, PPT</td>
<td>B-Line, Immediately after the final session</td>
<td>2/7 2 weeks</td>
<td>700 @ TB, 300 @ Surrounding area.</td>
<td>fESWT (0.06 mJ·mm⁻²)</td>
<td>MW, WSR</td>
<td>Between-group: VAS and PPT - ESWT↑ (p&lt;0.05); Within-group: VAS, PPT - ESWT↑ (p&lt;0.05)</td>
</tr>
<tr>
<td>Zhao et al. (2013)</td>
<td>RCT: ESWT Vs Placebo</td>
<td>70 (25, 45)</td>
<td>Symptomatic knee OA</td>
<td>TrPs around the knee</td>
<td>VAS, WOMAC, Lequesne index</td>
<td>B-Line, day 1, after the final session, week 12</td>
<td>1/7 4 weeks</td>
<td>1000</td>
<td>rESWT (0.25mJ·mm⁻²)</td>
<td>1-way ANOVA</td>
<td>Between-group and within-group: VAS, WOMAC, Lequesne index ESWT↑ (p&lt;0.05)</td>
</tr>
</tbody>
</table>

Where RCT is randomised control study, (f- is focused)(r- is radial)ESWT is extracorporeal shockwave therapy, TENS is transcutaneous neuromuscular stimulation, MPS is myofascial pain syndrome, Traps is Trapezius, McGill-PQ is McGill pain questionnaire, VAS is visual analogue scale, PPT is pressure pain threshold, ROM is range of motion, B-Line is Baseline, (r) ANOVA is (repeated) measure analysis of variance, TB is taut band, MW- test is Mann-Whitney test, WSR is Wilcoxon signed test, OA is osteoarthritis, TrP is trigger points, WOMAC is Western Ontario and McMaster University Osteoarthritis Index.
Table 6: Summary of peer-reviewed studies of the effects of extracorporeal shockwave therapy on trigger points

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<tr>
<th>Author (year)</th>
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<th>Pules</th>
<th>ESWT type (energy density)</th>
<th>Stats</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gür et al. (2014)</td>
<td>RCT: 1 session Vs 3 sessions</td>
<td>60 (12, 48)</td>
<td>MPS patients with confirmed TrPs</td>
<td>Any muscle with TrPs associated with MPS</td>
<td>VAS, n of TrPs, PGA, MDGA, NPDS, NHP, HAM-A</td>
<td>B-Line, week 3, week 12</td>
<td>1/7 3 weeks</td>
<td>1000</td>
<td>fESWT (0.25 mJ∙mm$^{-2}$)</td>
<td>rANOVA</td>
<td>Between Groups: ↔ (p&lt;0.05). Within group: Baseline-week 3, Baseline-week 12 all outcomes ↑ (p&lt;0.001)</td>
</tr>
<tr>
<td>Moghtaderi et al. (2014)</td>
<td>RCT: ESWT to heel and calf (Combo) Vs ESWT to heel (control)</td>
<td>40 (13, 27)</td>
<td>Patients with plantar fasciitis</td>
<td>Gastroc, soleus</td>
<td>VAS, MRM</td>
<td>B-Line, Week 9</td>
<td>3/7 1 week</td>
<td>3000 to heel (both groups) + 400 to each TrP (combo)</td>
<td>fESWT (0.20 mJ∙mm$^{-2}$)</td>
<td>rANOVA</td>
<td>Between Groups Combo ↑ VAS, MRM (p&lt;0.05). Within group: Combo ↑ (p&lt;0.001); control ↑ (p&lt;0.02)</td>
</tr>
</tbody>
</table>

Where RCT is randomised control study, MPS myofascial pain syndrome, (n is number of) TrP is trigger points, VAS is visual analogue scale, PGA is patient global assessment, MDGA is physician global assessment, NPDS is neck pain and disability scale, NHP is Nottingham health profile, HAM-A is Hamilton anxiety rating scale, fESWT is extracorporeal shockwave therapy rANOVA is repeated measure analysis of variance, ↔ is no improvement, ↑ is improvement, Combo is combined, Gastroc is gastrocnemius, MRM is modified Roles and Maudsley score.
There appear to be a significant statistical improvements for all VAS and pain rated scales after ESWT (Müller-Ehrenberg and Licht, 2005; Damian and Zalpou, 2011; Cho et al., 2012; Jeon et al., 2012; Ji et al., 2012; Zhao et al., 2013; Gür et al., 2014; Moghtaderi et al., 2014). The PPT also improved statistically after ESWT (Müller-Ehrenberg and Licht, 2005; Damian and Zalpou, 2011; Cho et al., 2012; Jeon et al., 2012; Ji et al., 2012; Zhao et al., 2013; Gür et al., 2014; Moghtaderi et al., 2014). One study (Damian and Zalpou, 2011), reported that they were not confident to state that the improvement in the PPT was statistically significant due to the high standard deviation. In the study by Ji et al. (2012), the baseline PPT was very high for the treatment group (40.36 N) and placebo group (43.67 N). The PPT increased to 61.20 N which was substantially greater than the PPT reported in previous studies (Vanderweeën et al., 1996; Sciotti et al., 2001; Chesterton et al., 2003; Jones et al., 2007). It is interesting that the PPT increased immediately after the first treatment. It is intuitive that the treated TrPs would have a transient tenderness (Damian and Zalpou, 2011). The increased PPT may be a result of the analgesic effect of the ESWT. Therefore, future studies should examine the effects of ESWT on muscle activation to determine the mechanism behind improvements in the PPT after ESWT. The effects of ESWT on cervical range of motion after treating TrP in the muscles in the neck and shoulder is varied with no significant improvements noted (Damian and Zalpou, 2011); whereas there was an improvement ($p < 0.001$) reported in all cervical movements except extension four weeks after one treatment of TrP in the muscles in the neck and shoulder with ESWT (Jeon et al., 2012).

The literature in relation to the use of ESWT as a treatment for TrPs is limited. ESWT appears to have a positive effect of VAS and PPT. There are limited findings vis-à-vis the effects ESWT on the range of motion of the neck, although there may be a time factor for the effects of ESWT to manifest in neck range of motion. Future research should consider the effect of ESWT in isolation from other treatment options to assess its impact on TrPs. It would appear that fESWT and rESWT are safe and effective treatment options for TrPs. In order to evaluate the influence of ESWT on the signs of TrPs such as TB, it would be wise to consider
the effect of ESWT on other pathologies that cause prolonged contractions of sarcomeres such as spasticity.

2.4.3.6 The effects of shockwave therapy on muscles with spasticity

There has also been some limited research conducted into the effects of rESWT on muscle spasticity in cerebral palsy (Amelio and Manganotti, 2010; Vidal et al., 2011) and stroke patients (Manganotti and Amelio, 2005; Sohn et al., 2011; Moon et al., 2013; Daliri et al., 2015). While the aetiology of spasticity of cerebral palsy and stroke patients are different to TrPs, it is in part a result of increase in motor neuron excitability (Daliri et al., 2015) primarily due to lesion in the immature brain (Amelio and Manganotti, 2010; Vidal et al., 2011) or spinal hyperexcitability (Manganotti and Amelio, 2005). The net result of severe protracted spasticity, if not controlled, can lead to pain, postural abnormality, and disability (Daliri et al., 2015). Therefore the findings and theories behind the mechanisms of ESWT on muscles with spasticity should be considered in relation to better understanding the effects of ESWT on muscle with TrPs.

ESWT seems to have positive effects on muscle spasticity. Improvements in Ashworth scale, or variations of same, have been reported throughout the literature, in particular, the ankle and wrist flexors (Sohn et al., 2011; Vidal et al., 2011; Moon et al., 2013; Santamato and Micello, 2014; Daliri et al., 2015). Short term improvements in range of motion have also been reported (Manganotti and Amelio, 2005; Amelio and Manganotti, 2010; Sohn et al., 2011; Vidal et al., 2011; Santamato and Micello, 2014). Improvements in subtle muscle control, such as the ability to maintain an erect standing position without compensatory swaying or movement, as measured using instrumental pedobarographic measures, increasing from 20.50 to 90.06 kPa (p < 0.001) after one week, have been reported after ESWT of gastrocnemius and soleus muscles of children with cerebral palsy (Amelio and Manganotti, 2010). Improvements in peak eccentric torque have been noted up to four weeks after ESWT of the gastrocnemius of post-stroke patients, 8.62 to 6.87 Nm at 180°$\cdot$s$^{-1}$ (p < 0.001); while enhancements in torque threshold angles, were only observed up to one week after ESWT, 9.33 to 12.92° at 180°$\cdot$s$^{-1}$ (p < 0.02), on the gastrocnemius of post-stroke patients (Moon et al., 2013).
2.4.3.6.1 Time frame of the effects of shockwave therapy on spasticity

The improvements reported with ESWT were recorded four weeks after treating the gastrocnemius and soleus muscles of children with cerebral palsy, but these improvements were not seen after 12 weeks (Amelio and Manganotti, 2010). The temporary benefits were also seen up to five weeks modified Ashworth score in the wrist flexors, 2.30 out of 4.00 to 1.22 out of 4.00 (p < 0.05) of patients with post-stroke spasticity (Daliri et al., 2015). Improvements in the Ashworth scale for wrist flexors and finger range of motion did not last longer than 12 weeks; but the Ashworth scale for finger flexors was still significantly improved, compared to Baseline, up to 12 weeks (Manganotti and Amelio, 2005). Post-stroke patients with plantar-flexor muscles spasticity grades I to III, as determined by the Heckmatt scale, reported improvements in modified Ashworth scale and passive ankle dorsiflexion up to 30 days, whereas patients with grades IV on the Heckmatt scale, only reported improvements in modified Ashworth scale and passive ankle dorsiflexion more than 1 day (Santamato and Micello, 2014). There was a net negative effect, compared to placebo, three months after three weekly sessions of ESWT on the agonist as well as the agonist and antagonist combined, in subjects with cerebral palsy (Vidal et al., 2011).

The time frame of the efficacy of ESWT is unclear. The effects seem to last between eight and twelve weeks in muscle with spasticity, how this transfers to the treatment of TrP, where there is a positive feedback mechanism is present, is uncertain. As well as how treatment over a prolonged time frame would affect muscle with spasticity or TrPs.

The Hoffmann reflex is an EMG method of measuring evoked potential in the A-type afferent nerve fibres (Kamen and Gabriel, 2010). The F-wave is similar to the Hoffmann reflex as in it measures evoked potential, but rather of the motor nerve (McLeod and Wray, 1966). If the hypothesis that ESWT has an effect on A-δ and C-type nerve fibres (Hausdorf et al., 2008b, 2008a) and substance P production (Maier et al., 2003; Jeon et al., 2012), then there would be no effects noted in Hoffmann reflex nor F-wave. The lack of change in EMG outcomes (motor nerve conduction, compound motor action potential Latencies, and amplitudes; as well as F-wave, mean latencies and amplitudes) may suggest that there is no muscular denervation and that the effect may be solely on the passive structures.
(Manganotti and Amelio, 2005). Sohn et al. (2011) also reported no changes in F-wave responses or an H-M ratio of Hoffmann reflex of the tibial nerve after fESWT (p > 0.05). ESWT appears not to have an effect on motor nerve fibres (Sohn et al., 2011). How ESWT effects spastic muscle is unclear, it may be due to the vascular effect or breaking the actin-myosin filament cross bridge.

2.4.3.6.2 Summary of the effect of shockwave therapy on muscles with spasticity

ESWT might be able to break-up the active-myosin links (Ramon et al., 2015). This may explain the improvement of the Ashworth scales but no alteration in efferent neural activity measured in the Hoffmann reflex or F-waves in muscle with spasticity (Manganotti and Amelio, 2005; Sohn et al., 2011; Santamato and Micello, 2014; Daliri et al., 2015). There is a caveat in relation to the efficacy of ESWT as a treatment for muscles with spasticity, and possibly TrPs; the effects may only be short-lived.

2.4.3.7 Shockwave therapy protocol

There is still no specific protocol for ESWT in relation to spasticity (Moon et al., 2013); therefore the same should be considered for TrPs. Present recommendations are based on clinical experience (Gleitz and Hornig, 2012; Ramon et al., 2015; Schmitz et al., 2015). Application of insufficient energy adversely affects the outcome of ESWT. Therefore, the highest energy flux density that can be tolerated by the patient should be applied (Schmitz et al., 2015). Before there was an understanding of the effects of ESWT on muscles, it was suggested that using rESWT may be severely limited as there is a rapid decay of the pressure wave away from the emitter (Simons, 2004a). However, one distinct benefit of r-ESWT is that the surrounding fibres (in the orientation of the muscle distal to proximal) can also be treated with 1000-4000 pulses at 1.2-1.8 bar 10-20Hz (Gleitz and Hornig, 2012). Application of local anaesthesia adversely affects the outcome of ESWT (Ramon et al., 2015; Schmitz et al., 2015). Follow-up sessions should be conducted 6 weeks, 3, 6, 12 months, respectively after treatment (Ramon et al., 2015). fESWT recommended setting are 300-600 up to 1000-2000 shockwaves at an energy level of 0.1 to 0.35 mJ/mm² and a frequency of 4 Hz depending on muscle size; and rESWT: locally 500-1000 pulses with pressure of 1.0-1.5 up
to 2.0-2.6 bar for small muscles, and 1.5-2.0 up to 3.0-4.0 bar for large muscles with a frequency of 4-20 Hz; Both radial and focused ESWT can be performed 1-2 sessions per week for 1-5 up to 6-10 sessions depending on the severity of the conditions (Gleitz and Hornig, 2012; Ramon et al., 2015).

2.4.4 Summary of shockwave therapy

ESWT is a safe and effective treatment modality that imparts more energy than another electrotherapy such as ultrasound by a factor of 100. ESWT has been extensively used to treat tendon and fascial pathologies. There is an emergence of research into the effects of ESWT on muscles. However, this is in relation to muscles with spasticity, which may have similar effects on TrPs. The mechanism of ESWT in orthopaedic condition is still unclear but is believed to have a molecular, vascular or neural effect. Of which, the regulation of the molecular neurotransmitter substance P, muscular angiogenesis due to the synthesis of nonenzymatic nitric oxide and deinnervation of unmyelinated C-fibres and lightly myelinated A-δ-fibres. The application of ESWT is not dependent on whether the type of ESWT is focused or radial; as long as sufficient accumulative energy flux density is applied.

2.5 Summary of the literature in relation to trigger points

TrPs are the sensory, motor, and autonomic symptoms myofascial pain syndrome. TrPs are TBs of skeletal muscle with a tender nodule at their epicentre. There can be a LTR and or a JS. Each TrP can have a particular pattern of RP. TrPs occur secondarily as a result of a sustained contraction due to repeated overload or trauma. The resulting hypoxia cause dysfunction at the neuromotor junction compounding the sustained contraction. There is also a local build-up of inflammatory markers which participate towards central sensitisation. The both of these result in a positive feedback loop. There are two types of TrPs; active TrPs are spontaneously painful, and latent TrP are only painful when palpated. Both types can alter movement patterns. There are a plethora of treatment option for TrPs. DN is the more effective. It is hypothesised that DN stops the afferent dysfunction at the neuromuscular junction. ESWT may be an alternative to DN as there is less post-treatment soreness and is believed to prevent the positive feedback loop at the efferent nerve endings.
preventing the central sensitisation. The PPT is a reliable method to measure the severity of TrPs. The CMJ can be used reliably to measure the strength and power in the lower limb. The DJ can be used to measure the reactive strength of the lower limb.

2.6 Gap analysis

There is a dearth of research in the following: The reliability of the sensitivity and location of TrPs in the VL and VM, the treatment of TrP to help improve athletic performance, the comparison of DN and ESWT as a treatment for TrPs; and the effects of post-needling soreness on athletic performance.

2.7 Summary

TrPs can affect muscular strength. Jumping is a measure of athletic performance which uses muscular force, particularly from the quadriceps. DN and ESWT are effective at treating TrPs. DN may cause post-needling soreness which may negatively affect jump performance. ESWT may improve jump performance without any transient deficits to sports performance.

2.8 Aims and outcomes

The aim of the present study is to measure the short-term effects of DN and rESWT on the sensitivity of TrPs and athletic performance.

The outcomes of the current project are to:

1. Assess the reliability of measuring the location of TrPs using the anatomical landmark system.
2. Determine the reliability of measuring the severity of TrPs using the pressure pain threshold.
3. Identify the reliability of measuring athletic performance using the countermovement jump and depth jump.
4. Examine the difference between genders in relation to TrPs and jump performance.
5. Determine the short-term and medium-term effects of post-treatment soreness following DN and rESWT.
2.9 Hypotheses

The null hypotheses are

1. The rater will not be reliable at measuring the location of TrPs in the VL and VM using the ALS.
2. The severity of TrPs in the VL and VM using the PPT will not be reliably measured by the rater.
3. Gender will not have an effect on the number of features of TrPs
4. Gender will not have an effect on the sensitivity of TrPs.
5. The rater will not be reliable at measuring the CMJ-JH, DJ-JH, DJ-tc and DJ-RSI.
6. There will be no correlation between physiological measures and jump outcomes.
7. Using DN or rESWT to treat TrPs in the VL and VM will not affect the PPT.
8. Treating TrPs in the VL and VM with DN or rESWT will not affect CMJ jump performance.
9. Gender will not have an effect on jump performance.
10. Treating TrPs in the VL and VM with DN or rESWT will not affect the jump performance of the DJ.
3 Locating trigger points reliability

3.1 Introduction

In order to offer a continuation of care to a patient, it is important that a therapist can reliably relocate and measure the severity of TrPs to document a patient’s progression. To the author’s knowledge, no study to date has assessed the reliability of measuring TrPs in the VL or VM. The ALS allows the location of a TrP to be measured once palpated and has been previously demonstrated to be reliable (Barbero et al., 2012). No study to date has assessed the reliability of locating TrPs in conjunction with measuring the severity of the TrPs using PPT. The aim of the present study is to establish the intra-rater reliability of measuring the location and severity of latent TrPs in the VM and VL of asymptomatic subjects.

3.1.1 Null hypotheses

1. The rater will not be reliable at measuring the location of TrPs in the VL and VM using the ALS.

2. The severity of TrPs in the VL and VM using the PPT will not be reliably measured by the rater.

3. Gender will not have an effect on the number of features of TrPs.

4. Gender will not have an effect on the sensitivity of TrPs.
3.2 Methods

3.2.1 Experimental approach

Experimental sessions were conducted in the physiology laboratory in Institute of Technology Carlow. The study was a randomised repeat design to test the intra-rater reliability of locating Latent TrPs in the VL and VM as illustrated in Figure 1. Variables that were examined were: PPT, using a Commander™ algometer (JTech Medical, Midvale, Utar, USA); TrP location using the ALS (Barbero et al., 2012); as well as the ΔTrP from the TrP from Session 1 and TrP from Session 2. Subjects that met the inclusion criteria were between 18 and 35 years of age. Subjects were excluded from the study if they have any substantial difference in muscle size or asymmetry, systemic diseases of the muscular or nervous system, congenital or childhood hip disease (n = 2), history of hip or knee trauma surgery in the lower extremity in the past 12 months, inflammatory joint disease, tumours, lower limb and or lower back injury and treatment of respective muscles in the past 6 months (n = 4). Thirty-six were initially tested, and seven were excluded for not meeting the inclusion criteria: not having TrPs (n = 6) or withdrawing from testing (n = 1).

Figure 1: Trigger point location reliability study flow chart. Where TrPs is trigger points.
3.2.2 Testing procedure

Testing was approved by the Institute of Technology Carlow’s Ethics Board (Appendix ii [p. Apx.v]). A full safety statement was conducted (Appendix iii [p. Apx.xiii]). Prior to testing subjects completed a screening and informed consent form (Appendix iii [p. Apx.xv]). Subjects were informed to refrain from vigorous exercise 24 hours prior to testing. Subjects presented to the Institute of Technology Carlow’s Physiology Laboratory where physiological measurements (height, mass, and body mass index [BMI]) and baseline testing was recorded.

The order of muscle and region for both sessions was randomly assigned (Office Excel® 2013, Microsoft Corporation, Redmond, Washington, USA) on Windows® 7 (Microsoft Corporation, Redmond, Washington, USA). To ensure the rater was blind to the location of TrP and ALS measures of Session 1 during Session 2 a Sure Code® ultra-violet (UV) marker (Security, Aldershot, UK) was used for Session 1, while illuminated with a Safescan® UV lamp (Safescan BV, Zoetermeer, the Netherlands). Whereas, the ALSs and TrPs in Session 2 were marked with a red Lumocolor® whiteboard marker (Staedtler, Nuremberg, Germany). The location of the most painful TrP in each region was marked with a “+”, The ALS for each respective TrPs was measured, The PPT was also measured.

Jones et al. (2007) reported that testing PPT on consecutive days does not affect. Session 2 was conducted no more than 24 hours after Session 1. The ALS and PPT were re-recorded. Once all measures for Session 2 were recorded, and the difference in distance from the Session 1, and Session 2 were also measured (ΔTrP) under UV light.
### 3.2.3  Locating the trigger points

Testing was completed on the non-dominant leg (the standing leg during kicking). The flat palpation method was used to locate the TrPs. To classify that a TrP was located the following criteria needed to be met: first, there must be a TB within the muscle that is parallel to the orientation of the muscle; second, there must be a TS that produces exquisite pain locally or following specific referral pattern for each TrP; third any of the following criteria may be present, a JS where the limb or subject involuntarily recoils from pressure to TrP, a LTR uncontrolled twitching in the muscle that may be palpated by the therapist or felt by the subject, RP where pain may radiate in specific patterns if the TrP is active, or locally if the TrP is latent (Dommerholt, 2011).

#### 3.2.3.1  Vastus lateralis

The subject (wearing shorts) was exposed to the hip and placed in side-lying on a plinth with the knee was placed in 30.00° of flexion and supported with a pillow to avoid the hip being placed into adduction (Travell and Simons, 1992). The VL was split into five regions (Travell and Simons, 1992). The first region (VL-TrP₁) is located towards the insertion onto the patella; The second region (VL-TrP₂) is located posteriorly to VL-TrP₁; The third region (VL-TrP₃) is located in the posterior muscle belly; The fourth region (VL-TrP₄) is located anteriorly to the VL-TrP₃. The fifth region (VL-TrP₅) is located towards the origin of the VL. The referral pattern radiates pain to the VL and the lateral aspect of the knee joint and the postero-lateral aspect of the thigh (Figure 2a [p. 107]).

#### 3.2.3.2  Vastus medialis

The VM was split into regions: One (VM-TrP₁) is located towards the insertion at the patella; the second (VM-TrP₂) is located in the muscle belly. TrPs in the VM refers towards the anteromedial aspect of the knee and the distomedial aspect of the thigh (Figure 2b [p. 107]).
Figure 2: Trigger points of the quadriceps muscles, a vastus lateralis, b vastus medialis. Where TrP is trigger point. Adapted from Travell and Simons (1992).
3.2.4 Anatomical Landmark System

The TrPs were marked with a “+” using a UV marker. A modification to Barbero et al.’s (2012) ALS was used to measure the distance of the ALS ($ALS_d$) of the VL, and VM. A line was drawn and measured using a UV marker from the anterior superior iliac spine to the head of the fibula (VL) and the apex of the patella (VM), respectively. A perpendicular line (X-line) from the TrP to the $ALS_d$ line was drawn with the UV marker and measured. The distance from the ASIS and the intersection of the $ALS_d$ and X-line was measured (Y-line) as illustrated in Figure 3. The subjects were instructed to refrain from vigorously scrubbing the thigh until after the retest session. The process was repeated for Session 2 but with a whiteboard marker.

![ALS schematic for VM. Where ALS is anatomical landmark system, ALS_d is anatomical landmark system distance, TrP is trigger point.](image)

Figure 3: ALS schematic for VM. Where ALS is anatomical landmark system, ALS_d is anatomical landmark system distance, TrP is trigger point.
3.2.5 The pressure pain threshold

The PPT was measured using a Commander™ algometer and was described to the subject “as the point where the sensation of pressure turned to a perception of pain” (Ohrbach and Gale, 1989b). To prevent bruising a 1cm² head was used (Takahashi et al., 2005). The rater was blinded to the pressure placed through the algometer through the maximum pressure recall feature of the algometer. The rater increased pressure via the algometer by 1N∙s⁻¹ until the subject reported the change of the pressure to pain (Nussbaum and Downes, 1998).

3.2.6 Statistical analysis

The intra-class coefficient with two-way mixed absolute model [ICC(3,1)] was calculated for the X-line, the Y-lines of the ALS and the PPT. The criteria for interpreting the ICC(3,1) were: ≤0.40 = poor, 0.41-0.75 = moderate, ≥0.76 = excellent (Doğan and Doğan, 2015). Clinical significance for ICC(3,1) was set at 0.60 (Barbero et al., 2012). The SEM was also reported (Equation 6 [p. 110]). Bland-Altman plots (Bland and Altman, 1999) were provided to give a visual representation of data for the X-line, Y-line and PPT, respectively. One-sample t-tests were conducted for the ΔTrP, with the test value set at 1.50cm (Barbero et al., 2012). Cases were split according to gender. A one-way analysis of variance (ANOVA) was conducted to analyse the difference in gender of the severity of TrPs. and Independent t-tests were calculated for between gender differences for the PPT. The SD of all the Y-lines were proportionally high compared to the mean of the respective Y-lines. Therefore the data for ALS and PPT for both sites were log transformed using the base of natural logarithm (ln) to account for heteroscedasticity (Chinn, 1990). Proportional bias was determined using linear regression (β < 0.05) according to Ludbrook (2002). Normal distribution was tested for using the Shapiro-Wilk test. All data were analysed using SPSS® 22 (IBM Incorporated, Armonk, New York, USA). Significance was set at p < 0.05.
\[ SEM = \frac{SD}{\sqrt{SS}} \]

Equation 6: Standard error of mean.

Where \( SEM \) is standard error of mean, \( SD \) is standard deviation, \( SS \) is sample size.

### 3.3 Results

Twenty-nine were suitable for retesting and analysis. The fifteen males and fourteen female subjects had a mean age of 22.4 ± 3.9 (SD) years, a mean mass of 70.77 ± 15.27 kg and a mean stature of 172.48 ± 9.36 cm (Table 7).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (% female)</td>
<td>48.27</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>22.41 ± 3.91</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>70.77 ± 15.27</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.48 ± 9.36</td>
</tr>
<tr>
<td>Hours training (hr)</td>
<td>6.93 ± 3.35</td>
</tr>
<tr>
<td>Standing leg (% right)</td>
<td>24.14</td>
</tr>
<tr>
<td>VL-ALS (_d) (cm)</td>
<td>55.86 ± 2.7</td>
</tr>
<tr>
<td>VM-ALS (_d) (cm)</td>
<td>53.35 ± 2.8</td>
</tr>
</tbody>
</table>

Table 7: Mean and standard deviation (SD) of subject demographics and characteristics

VL-ALS \(_d\) is distance of anatomical landmark system of the is vastus lateralis (anterior superior iliac spine to head of fibula); VM-ALS \(_d\) is distance of anatomical landmark system of the vastus medialis (anterior superior iliac spine to tibial tuberosity) (n = 29).

All measurements were recorded by the same observer with two years of experience of treating TrPs. The ICC\([3,1]\) values for all sites using the ALS ranged from 0.643 (95% CI: 0.246 to 0.832) to 0.886 (95% CI: 0.744 to 0.944) for the X-line and from 0.603 (95% CI: 0.133 to
0.797) to 0.745 (95% CI: 0.465 to 0.883) for the Y-line (Table 8); whereas the ICC \((3,1)\) for the PPT was 0.637 (95% CI: 0.103 to 0.800) to 0.848 (95% CI: 0.689 to 0.931) as illustrated in Table 9 (p. 112). SEM for all measurements were also calculated. The mean ΔTrP ranged from 0.97 ± 0.72 cm and 1.52 ± 1.02 cm.

Table 8: Reliability, ICC \((3,1)\), and agreement measure, SEM, of between session mean distance in relation to the ALS

<table>
<thead>
<tr>
<th>Site</th>
<th>(X_1 \pm SD) (cm)</th>
<th>(X_2 \pm SD) (cm)</th>
<th>(X) ICC ((3,1)) (95% CI)</th>
<th>(X) SEM</th>
<th>(Y_1 \pm SD) (cm)</th>
<th>(Y_2 \pm SD) (cm)</th>
<th>(Y) ICC ((3,1)) (95% CI)</th>
<th>(Y) SEM</th>
<th>ΔTrP (\pm SD) (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL-TrP1</td>
<td>44.68 ± 3.12</td>
<td>45.14 ± 2.66</td>
<td>0.870* (0.720-0.937)</td>
<td>0.22</td>
<td>3.80 ± 0.82</td>
<td>3.76 ± 0.87</td>
<td>0.745* (0.465-0.883)</td>
<td>0.38</td>
<td>1.29 ± 0.85†</td>
</tr>
<tr>
<td>VL-TrP2</td>
<td>44.87 ± 3.01</td>
<td>45.34 ± 2.63</td>
<td>0.789* (0.553-0.90)</td>
<td>1.08</td>
<td>3.23 ± 1.24</td>
<td>2.96 ± 1.13</td>
<td>0.667* (0.292-0.840)</td>
<td>0.68</td>
<td>1.52 ± 1.02†</td>
</tr>
<tr>
<td>VL-TrP3</td>
<td>33.62 ± 3.26</td>
<td>34.26 ± 3.08</td>
<td>0.886* (0.744-0.944)</td>
<td>0.68</td>
<td>3.07 ± 0.90</td>
<td>2.91 ± 0.96</td>
<td>0.683* (0.332-0.852)</td>
<td>0.51</td>
<td>1.01 ± 0.90‡</td>
</tr>
<tr>
<td>VL-TrP4</td>
<td>33.95 ± 3.21</td>
<td>33.77 ± 2.02</td>
<td>0.850* (0.687-0.931)</td>
<td>0.62</td>
<td>4.16 ± 0.87</td>
<td>3.76 ± 1.01</td>
<td>0.603* (0.133-0.797)</td>
<td>0.64</td>
<td>1.07 ± 0.79‡</td>
</tr>
<tr>
<td>VL-TrP5</td>
<td>22.61 ± 2.52</td>
<td>23.05 ± 2.34</td>
<td>0.643* (0.246-0.832)</td>
<td>1.49</td>
<td>3.59 ± 1.31</td>
<td>3.61 ± 1.27</td>
<td>0.689* (0.346-0.859)</td>
<td>0.70</td>
<td>1.37 ± 1.06†</td>
</tr>
<tr>
<td>VM-TrP1</td>
<td>43.57 ± 2.70</td>
<td>43.69 ± 2.59</td>
<td>0.881* (0.752-0.945)</td>
<td>0.60</td>
<td>6.57 ± 0.93</td>
<td>6.15 ± 1.02</td>
<td>0.722 (0.335-0.854)</td>
<td>0.48</td>
<td>0.97 ± 0.72‡</td>
</tr>
<tr>
<td>VM-TrP2</td>
<td>35.33 ± 3.09</td>
<td>35.09 ± 2.34</td>
<td>0.748* (0.471-0.884)</td>
<td>1.25</td>
<td>6.07 ± 1.20</td>
<td>5.91 ± 1.21</td>
<td>0.647* (0.255-0.836)</td>
<td>0.75</td>
<td>1.02 ± 0.81†</td>
</tr>
</tbody>
</table>

TrP is trigger point, ICC \((3,1)\) is intra-class coefficient (two-way mixed, absolute model), SEM is standard error of measurement, 95% CI is 95 percent confidence interval, ALS is the anatomical landmark system, X(n) is X-line (session number), Y is Y-line (session number), ΔTrP is measured distance between location of TrP1 and location of TrP2, VL is vastus lateralis, VM is vastus medialis (n = 29). * ICC \((3,1)\) statistical significance between test and re-test (p < 0.05), † No statistical difference to ΔTrP set at 1.50 cm using a one-sample t-test, ‡ statistically less than ΔTrP set at 1.50 cm using one-sample t-test.
Table 9: Reliability, ICC\(^{(3,1)}\), and agreement measure, SEM, of between session mean pressure in relation to the PPT

<table>
<thead>
<tr>
<th>Site</th>
<th>PPT1 ± SD (N)</th>
<th>PPT2 ± SD (N)</th>
<th>PPT ICC(^{(3,1)}) (95% CI)</th>
<th>PPT SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL-TrP(_1)</td>
<td>23.68 ± 5.02</td>
<td>22.96 ± 4.92</td>
<td>0.807* (0.592-0.909)</td>
<td>1.76</td>
</tr>
<tr>
<td>VL-TrP(_2)</td>
<td>25.63 ± 4.39</td>
<td>22.55 ± 5.82</td>
<td>0.637 (0.103-0.800)</td>
<td>3.21</td>
</tr>
<tr>
<td>VL-TrP(_3)</td>
<td>24.32 ± 5.13</td>
<td>23.98 ± 5.79</td>
<td>0.848* (0.684-0.931)</td>
<td>1.55</td>
</tr>
<tr>
<td>VL-TrP(_4)</td>
<td>25.15 ± 5.24</td>
<td>24.28 ± 6.26</td>
<td>0.682* (0.329-0.851)</td>
<td>3.20</td>
</tr>
<tr>
<td>VL-TrP(_5)</td>
<td>27.10 ± 5.99</td>
<td>25.58 ± 5.59</td>
<td>0.875 (0.697-0.936)</td>
<td>0.07</td>
</tr>
<tr>
<td>VM-TrP(_1)</td>
<td>22.66 ± 4.77</td>
<td>20.80 ± 3.92</td>
<td>0.736* (0.359-0.861)</td>
<td>2.05</td>
</tr>
<tr>
<td>VM-TrP(_2)</td>
<td>22.45 ± 4.86</td>
<td>21.15 ± 3.59</td>
<td>0.744* (0.434-0.872)</td>
<td>1.95</td>
</tr>
</tbody>
</table>

TrP is trigger point, ICC\(^{(3,1)}\) is intra-class coefficient (mixed absolute model), SEM is standard error of measurement, 95% CI is 95 percent confidence interval, PPT (session number) is pain pressure threshold, VL is vastus lateralis, VM is vastus medialis (n = 29). * ICC\(^{(3,1)}\) statistical significance between test and re-test (p < 0.05).
TB and TS were present in the VL and VM for all subjects that were analysed. RP was present in 3-8 subjects for the VL and 0-1 subjects for the VM. JS was present in 4-7 subjects in the VL and 3-8 subjects for the VM. LTR was noted in 4-8 subjects for the VL and 3-8 subjects for the VM. The between session $\kappa$ scores were 0.47-1.00 in the VL and 0.65-1.00 in the VM (Table 10).

Table 10: Number and $\kappa$ score for features of TrPs in the vastus lateralis and vastus medialis

<table>
<thead>
<tr>
<th>Site</th>
<th>TB</th>
<th>TS</th>
<th>RP</th>
<th>JS</th>
<th>LTR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vl-TrP1</td>
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<td></td>
<td></td>
<td></td>
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</tr>
<tr>
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<td>29</td>
<td></td>
</tr>
<tr>
<td>Test 2</td>
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<td>29</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Vl-TrP2</td>
<td></td>
<td></td>
<td></td>
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<td>Test 1</td>
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<td>29</td>
<td></td>
</tr>
<tr>
<td>Test 2</td>
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<td>29</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Vl-TrP3</td>
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<td>Test 2</td>
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<td>29</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Vl-TrP4</td>
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</tr>
<tr>
<td>Vl-TrP5</td>
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</tr>
<tr>
<td>Test 2</td>
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</tr>
<tr>
<td>Vm-TrP1</td>
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</tr>
<tr>
<td>Test 2</td>
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</tr>
<tr>
<td>Vm-TrP2</td>
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<td></td>
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</tr>
<tr>
<td>Test 1</td>
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<tr>
<td>Test 2</td>
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<td>29</td>
<td>29</td>
<td>29</td>
<td></td>
</tr>
</tbody>
</table>

Note: $^a$Insufficient split in data distribution, kappa coefficient is not applicable. TrP is trigger point, $\kappa$ is Kappa coefficient, % Agre. is percent agreement, TB is taught band, TS is tender spot, RP is referred pain, JS is jump sign, LTR is twitch response, Pres is present, Vl-TrP$_{n}$ is site of trigger point within the vastus lateralis, Vm-TrP$_{n}$ is site of trigger point within the vastus medialis (n = 29). * Kappa statistical significance between test re-test (p < 0.05).
The data for the ALS and PPT was tested for normal distribution using the Shapiro-Wilk test and was found to be (p > 0.05) normally distributed, except for the X-line at VL-TrP. The SD of all the Y-lines were proportionally high compared to the mean of the respective Y-lines. Therefore the data for ALS and PPT of all sites were log transformed using the base of the natural logarithm (\(\mathbb{e}n\)). Bland-Altman plots for differences in \(\mathbb{e}n(X\text{-line})\), \(\mathbb{e}n(Y\text{-line})\) and \(\mathbb{e}n(PPT)\) and means of X-line, Y-line and PPT, respectively, are illustrated in Figure 4 (p. 115) to Figure 10 (p. 121). The limits of agreement (LoA) for \(\mathbb{e}n(X\text{-line})\), \(\mathbb{e}n(Y\text{-line})\) and \(\mathbb{e}n(PPT)\) were also calculated.
Difference between $\ln(1)$ and $\ln(2)$ at VL-TrP

Mean $1$ and $2$ at VL-TrP (cm)

1.96 SD 
-0.08

-1.96 SD 
-0.10

Bias 
0.03

Mean  
0.01

Difference between $\ln(PPT1)$ and $\ln(PPT2)$ at VL-TrP (N)

Mean PPT1 and PPT2 at VL-TrP (N)

1.96 SD 
0.37

-1.96 SD 
-0.30

Bias 
-0.01

Mean  
0.03

Figure 4: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the VL-TrP. (a) X-line (ALS), (b) Y-line (ALS) and (c) PPT variables. Difference in $\ln$ mean measures ($X$, $Y$, $PPT$) of each subject during both sessions are plotted against mean of mean measures ($X$, $Y$, $PPT$) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where TrP is trigger point, VL-TrP is site of TrP in vastus laterus, $\ln$ is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical land mark system (n=29).
Figure 5: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the vastus lateralis TrP site 2. (a) X-line, (b) Y-line (ALS) and (c) PPT variables. Difference in ln mean measures (X, Y, PPT) of each subject during both sessions are plotted against mean of mean measures (X, Y, PPT) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where VL is vastus laterus, TrP is trigger point, ln is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical land mark system (n=29).
Figure 6: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the vastus lateralis TrP site 3. (a) X-line, (b) Y-line (ALS) and (c) PPT variables. Difference in ln mean measures (X, Y, PPT) of each subject during both sessions are plotted against mean of mean measures (X, Y, PPT) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where VL is vastus laterus, TrP is trigger point, ln is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical landmark system (n=29).
Figure 7: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the vastus lateralis TrP site 4. (a) X-line, (b) Y-line (ALS) and (c) PPT variables. Difference in \( \log \) mean measures (X, Y, PPT) of each subject during both sessions are plotted against mean of mean measures (X, Y, PPT) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where VL is vastus lateralis, TrP is trigger point, \( \log \) is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical landmark system (n=29).
Figure 8: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the vastus lateralis TrP site 5. (a) X-line, (b) Y-line (ALS) and (c) PPT variables. Difference in $\ln$ mean measures (X, Y, PPT) of each subject during both sessions are plotted against mean of mean measures (X, Y, PPT) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where VL is vastus laterus, TrP is trigger point, $\ln$ is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical landmark system (n=29).
Figure 9: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the vastus medialis TrP site 1. (a) X-line, (b) Y-line (ALS) and (c) PPT variables. Difference in $\ln$ mean measures (X, Y, PPT) of each subject during both sessions are plotted against mean of mean measures (X, Y, PPT) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where VM is vastus medialis, $\ln$ is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical landmark system (n=29).
Figure 10: Bland-Altman plots showing the intra-rater agreement of relocating TrPs in the vastus medialis TrP site 2. (a) X-line, (b) Y-line (ALS) and (c) PPT variables. Difference in ln mean measures (X, Y, PPT) of each subject during both sessions are plotted against mean of mean measures (X, Y, PPT) of each subject. The solid line is the mean of difference. The dotted line is the proportional bias line. The two dashed lines are the upper and lower 95% limits, respectively. Where VM is vastus medialis, ln is base of natural logarithm, PPT is pain pressure threshold, ALS is anatomical land mark system (n=29).
3.3.1 Distal-anterior vastus lateralis

The VL-TrP₁ had a mean X-line of 44.68 (95% CI: 43.49 to 45.86) cm and 45.86 (95% CI: 44.13 to 46.15) cm for the two sessions, respectively; and 3.80 (95% CI: 3.49 to 4.11) cm and 3.76 (95% CI: 3.43-4.09) cm along the Y-line for both sessions. The X-line had an ICC₃,₁ of 0.870 and a SEM of 0.22, and was not significantly different between the test and retest (p = 0.22); whereas the Y-line had an ICC₃,₁ of 0.745 and a SEM of 0.38, and was found not to be significantly different between test and retest (p = 0.79). The distance between the TrPs was measured (ΔTrP₁). The mean ΔTrP for VL-TrP₁ was 1.29 (95% CI: 0.97 to 1.61) cm. A one sample t-test was conducted and was found to be not significantly different (p = 0.19) to ΔTrP₁ set at 1.50 cm. The VL-TrP₁ had a mean PPT of 23.68 (95% CI: 21.77 to 25.59) N for the first session and a mean PPT of 22.96 (95% CI: 21.09 to 24.83) N for the second session. ICC₃,₁ and SEM for the PPT at VL-TrP₁ were 0.807 and 1.76, respectively. The test and retest data for PPT was not significantly different (p = 0.34). LoA for ℓn(X-line) was -0.10 to 0.08cm, while ℓn(Y-line) was -0.73 to 0.40cm and ℓn(PPT) was -0.30 to 0.37N (Figure 4 [p. 115]).

Gender differences in the features of TrP in the VL-TrP₁ were: 0.00% of males and 6.67% of females for RP; 14.29% of males and 13.33% of females for JS; 14.29% of males and 13.33% of females for LTR.

3.3.2 Distal-posterior vastus lateralis

The mean X-line for VL-TrP₂ was 44.87 (95% CI: 43.72 to 46.01) cm and 45.34 (95% CI: 44.34 to 46.34) cm for the two sessions respectively; whereas the mean Y-line was 3.23 (95% CI: 2.76 to 3.71) cm and 2.96 (95% CI: 1.24 to 2.96) cm for both sessions. The X-line had an ICC₃,₁ of 0.789 and a SEM of 1.08, while the ICC₃,₁ and SEM for the Y-line was 0.667 and 0.68, respectively. Both the X-line and the Y-line were not significantly different between test and retest, respectively, (p = 0.29) and (p = 0.22). The mean ΔTrP for VL-TrP₂ was 1.52 (95% CI: 1.13 to 1.90). A one sample t-test was conducted and was found to be not significantly different (p = 0.91) to the ΔTrP set at 1.50 cm. The mean PPT for VL-TrP₂ was 25.63 (95% CI: 23.96 to 27.30) N for session one and 22.55 (95% CI: 20.33 to 24.76) N for session two. The ICC₃,₁ and SEM for the PPT at VL-TrP₂ was 0.637 (p < 0.01) and 3.21, respectively. LoA for ℓn(X-line), ℓn(Y-line) and ℓn(PPT) were -0.11 to 0.09cm, -0.78 to...
0.98 cm and -0.29 to 0.58 N, respectively (Figure 5[p. 116]). Gender differences in the signs of TrPs in the VL-TrP₂ were: 0.00% of males and 13.33% of females for RP; 14.29% of males and 33.33% of females for JS; 21.43% of males and 33.33% of females for LTR.

### 3.3.3 Middle-posterior vastus lateralis

At VL-TrP₃ the mean X-line for session one was 33.62 (95% CI: 32.38 to 34.86) cm and 34.26 (95% CI: 33.09 to 35.43) cm for session two. The mean Y-line was 3.07 (95% CI: 2.72 to 3.41) cm and 2.91 (95% CI: 2.55 to 3.28) cm for both sessions. The ICC(3,1) and SEM for the X-line was 0.886 and 0.68 (p = 0.10), respectively. While the Y-line had an ICC(3,1) of 0.683 and a SEM of 0.51 (p = 0.38). The mean ΔTrP for VL-TrP₃ was 1.00 (95% CI: 0.67-1.35). A one sample t-tests was conducted and was found to be significantly less than (p < 0.01) the mean ΔTrP at 1.50 cm. The mean PPT for VL-TrP₃ was between 24.32 (95% CI: 22.37 to 26.28) N and 23.98 (95% CI: 21.78 to 26.19) N for the two sessions. The ICC(3,1) and SEM for the PPT at VL-TrP₃ was 0.848 (p = 0.65) and 1.55, respectively. LoA for ℓn(X-line) was -0.14 to 0.10 cm, whereas ℓn(Y-line) was -0.59 to 0.71 cm and ℓn(PPT) was -0.31 to 0.35 N (Figure 6 [p. 117]). Differences in gender for the features of TrP in the VL-TrP₃ were: 0.00% of males and 20.00% of females for RP; 7.14% of males and 46.67% of females for JS; 7.14% of males, 26.67% of females for LTR. There was a significant difference between genders for the mean PPT for the re-test (male, 26.26; female, 21.86; p = 0.039).

### 3.3.4 Middle-anterior vastus lateralis

At VL-TrP₄ the mean X-line for session one was 33.95 (95% CI: 33.09 to 34.87) cm and 33.77 (95% CI: 33.00 to 34.54) cm for session two. The mean Y-line was 4.16 (95% CI: 3.82 to 4.49) cm and 3.76 (95% CI: 3.37 to 4.14) cm for both sessions. The ICC(3,1) for the X-line was 0.850 and was found not to be significantly different between test and retest (p = 0.55). The SEM for the X-line was 0.62. While the Y-line had an ICC(3,1) of 0.683 (p = 0.04) and a SEM of 0.64. The mean ΔTrP for VL-TrP₄ was 1.07 (95% CI: 0.77 to 1.37). A one sample t-test was conducted and was found to be significantly less than (p < 0.01) the mean ΔTrP set at 1.50 cm. The mean PPT for VL-TrP₄ was 25.15 (95% CI: 23.16 to 26.66) N and 24.28 (95% CI: 21.90 to 26.66) N for the two sessions. The ICC(3,1) and SEM for the PPT at VL-TrP₄ was 0.682 (p = 0.42) and 3.20. The LoA for ℓn(X-line), ℓn(Y-line) and ℓn(PPT) were -0.09 to 0.10 cm, -0.42 cm and -0.31 to 0.35 N (Figure 6 [p. 117]). Differences in gender for the features of TrP in the VL-TrP₄ were: 0.00% of males and 20.00% of females for RP; 7.14% of males and 46.67% of females for JS; 7.14% of males, 26.67% of females for LTR. There was a significant difference between genders for the mean PPT for the re-test (male, 26.26; female, 21.86; p = 0.039).
to 0.66 cm and -0.39 to 0.49 N, respectively (Figure 7 [p. 118]). Differences in gender for the signs of TrP in the VL-TrP4 were: 14.29% of males and 6.67% of females for RP; 14.29% of males and 26.67% of females for JS; 7.14% of males and 33.33% of females LTR. There was a significant difference between genders for the mean PPT for the test (male, 27.14; female, 23.29; p = 0.045) and re-test (male, 26.80; female, 21.93; p = 0.034).

### 3.3.5 Proximal vastus lateralis

At VL-TrP5 the mean X-line for the first session was 22.61 (95% CI: 21.65 to 23.57) cm and 23.05 (95% CI: 22.16 to 23.94) cm for the second session. The mean Y-line was 3.59 (95% CI: 3.09 to 4.09) cm and 3.61 (95% CI: 3.12 to 4.09) cm for both sessions. The ICC(3,1) was 0.643 (p = 0.35) and 0.689 (p = 0.94) for the X-line and Y-line, respectively. The SEM was of 1.49 and 0.70 for the X-line and Y-line, respectively. The mean ∆TrP for VL-TrP5 was 1.37 (95% CI: 0.96-1.77). A one sample t-test was conducted and was found to not be significantly different (p = 0.51) to the mean ∆TrP set at 1.50 cm. The mean PPT for VL-TrP5 was 27.10 (95% CI: 24.83 to 29.38) N and 25.58 (95% CI: 23.45 to 27.71) N for both sessions. The ICC(3,1) and SEM for the PPT at VL-TrP5 was 0.875 (p = 0.04) and 1.36, respectively. LoA for ℓn(X-line) was -0.23 to 0.19 cm, while ℓn(Y-line) was -0.72 to 0.69 cm and ℓn (PPT) was -0.39 to 0.49 N (Figure 8 [p. 119]). Gender differences in the features of TrP in the VL-TrP5 were: 7.14% of males, 43.33% of females for RP; 7.14% of males and 20.00% of females for JS; 0.00% of males, 33.33% of females for LTR.

### 3.3.6 Distal vastus medialis

The mean X-line for VM-TrP1 was 43.54 (95% CI: 42.54 to 44.52) cm and 43.69 (95% CI: 42.71 to 44.68) cm for both sessions. The X-line ICC(3,1) was 0.881, while the SEM was 0.60 and was not significantly different between tests (p = 0.65). The mean Y-line at VM-TrP1 was 6.57 (95% CI: 6.21 to 6.92) cm for the first session and 6.15 (95% CI: 5.76 to 6.54) cm for the second session. The ICC(3,1) was 0.722 (p = 0.02) and the SEM was 0.48. The mean ∆TrP for VM-TrP1 was 0.97 (95% CI: 0.69 to 1.24). A one sample t-tests was conducted and was found to be significantly less than (p < 0.01) the mean ∆TrP set at 1.50 cm. The mean PPT for VM-TrP1 was 22.66 (95% CI: 20.85 to 24.47) N for session one and 20.80 (95% CI: 19.31 to 22.30) N for session two. The PPT at VM-TrP1 had an ICC(3:1) and SEM of 0.736 (p = 0.02) and 2.05,
respectively. LoA for ℓn(X-line), ℓn(Y-line) and ℓn(PPT) were -0.08 to 0.07 cm, -0.23 to 0.38 cm and -0.31 to 0.47 N, respectively (Figure 9 [p. 120]). Differences in gender for the signs of TrP in the VM-TrP were: 7.14% of males and 6.67% of females for RP; 0.00% of males and 0.00% of females for JS; 14.29% of males and 26.67% of females for LTR. There was a significant difference between genders for the mean PPT for the test (male, 24.46; female, 20.97; p = 0.047) and re-test (male, 22.29; female, 19.41; p = 0.046).

3.3.7 Middle vastus medialis

The VM-TrP had a mean X-line of 35.33 (95% CI: 34.15 to 36.50) cm for the first session and 35.09 (95% CI: 34.19 to 35.98) cm for the second session. The ICC(3,1) was 0.748 (p < 0.01) and the SEM was 1.25. The mean Y-line at VM-TrP was 6.07 (95% CI: 5.61 to 6.53) cm for the first session and 5.91 (95% CI: 5.45 to 6.38) cm for the second session. The ICC(3,1) and SEM was 0.674 (p < 0.01) and 0.75, respectively. Both the X-line and the Y-line were not significantly different between test and retest with p-values of 0.61 and 0.50, respectively. The mean ΔTrP for VM-TrP was 1.02 (95% CI: 0.71 to 1.32). A one sample t-test was conducted and was found to be significantly less than (p < 0.01) to the mean ΔTrP set at 1.50 cm. The mean PPT for VM-TrP was 22.45 (95% CI: 20.60 to 24.30) N and 21.15 (95% CI: 19.79 to 22.52) N for sessions one and two, respectively. The PPT at VM-TrP had an ICC(3,1) of 0.744 (p < 0.01) and a SEM of 1.95. LoA for ℓn(X-line) were -0.13 to 0.14 cm, ℓn(Y-line) -0.37 to 0.42 cm and ℓn(PPT) was -0.30 to 0.40 N (Figure 10 [p. 121]). Gender differences in the features of TrP in the VM-TrP were: 0.00% of males and 6.67% of females for RP; 0.00% of males and 0.00% of females for JS; 7.14% of males and 13.33% of females for LTR. Males had a significantly higher mean PPT for the test (male, 24.29; female, 20.27; p = 0.046) and re-test (male, 22.66; female, 19.74; p = 0.025).

3.3.8 Gender difference in the pressure pain threshold

The mean PP for VLTrP was 26.26 (95% CI: 22.85 to 29.67) for the males and 21.86 (95% CI: 19.11 to 24.61) for the females. The mean PP for VMTrP was 24.46 (95% CI: 22.18 to 26.75) for the males and 20.97 (95% CI: 18.23 to 23.72) for the males. The mean PP for VMTrP was 22.66 (95% CI: 21.08 to 24.25) for the males and 19.74 (95% CI: 17.65 to 21.83). The differences between gender are illustrated in Figure 11 (p. 126).
Figure 11: The difference between genders for the PPT of TrPs in the (a) middle anterior VL, (b) distal VM, (c) middle VM. Where PPT is pressure pain threshold TrPs is trigger points VL is vastus lateralis, VM is vastus medialis.
3.4 Discussion

Null hypothesis 1 states that “The rater will not be reliable at measuring the location of TrPs in the VL and VM using the ALS”. Twenty-nine asymptomatic subjects were assessed in the first phase of the reliability experiment by the same clinician for the features, location and severity of TrPs in the VL and VM and was found to be reliable in relation to ICC\(_{3,1}\) for the X-line (0.643 to 0.886) and the Y-line (0.603 to 0.745). Therefore null hypothesis 1 can be rejected.

The results were similar to the findings of Barbero et al. (2012), who reported an ICC\(_{1,1}\) for the X-line (0.62) and the Y-line (0.81) in the trapezius muscle using an ALS. Barbero et al. (2012) however used a one-way model for intra-class coefficient of reliability, which does not take into consideration random or observer variability and may not be considered clinically relevant (Rankin and Stokes, 1998). The results are also similar to those of Dibai-Filho et al. (2015) who reported an inter-rater ICC\(_{2,1}\) of 0.62 to 0.92 and an intra-rater ICC\(_{2,1}\) of 0.59 to 0.99 when using infrared thermographic imaging of the upper trapezius muscle in subjects with cervical pain and active TrPs.

The discrepancy in results of a previous study between the X-line and Y-line using the ALS were attributed to the orientation of the muscle relative to the ALS\(_d\) line (Barbero et al., 2012). They speculated that the Y-line in the trapezius muscle is easier to locate, thus more reliable, as the fibres of the trapezius are perpendicular to the ALS\(_d\) line and snap when palpated, relying on the objective signs of TB. The X-line in the trapezius depends on the subjective symptoms of TS and RP to ascertain the most tender spot. This could be as a result of the vague sensory feedback of C-type afferent nerve fibres which are associated with TrPs (Kawakita et al., 2008). Barbero et al.’s (2012) comments regarding the discrepancy in results between the X and Y lines and fibre orientation may also be applicable in the present study. The greater ICC\(_{3,1}\) of the X-line observed at VL-TrP\(_3\), VL-TrP\(_4\) and VM-TrP\(_1\) would suggest that the X-line in the VL and VM are easier to locate as their oblique orientation are perpendicular to the muscle fibres at the medial VL and distal VM (Blazevich et al., 2007). The pennate structure of the distal VM may also account for the high
proportion of TrP features detected in VM-TrP₁, as the VM has short muscle fibres (Wickiewicz et al., 1983) and thus prone to longitudinal shortening and fatigue (Armstrong, 2011). TrPs cause the muscle fibre to contract at the site of the TrP (Mense et al., 2003). During in vitro studies in mice and rats, it has been reported, that if a muscle is maintained in a shortened position, there can be a reduction in the numbers of series sarcomeres (Williams and Goldspink, 1978) and muscle fibre length (Spector et al., 1982).

Null hypothesis 2 was that “The severity of TrPs in the VL and VM using the PPT will not be reliably measured by the rater”. The results for the PPT of the reliability study in the twenty-nine asymptomatic subjects for the severity of TrPs in the VL and VM and was found to be reliable in relation to ICC(3,1) 0.643 to 0.886. Given the current results, it may be concluded that null hypothesis 2 can be rejected.

Previous studies have examined the reliability of assessing the PPT in TrPs of various muscles (Sand et al., 1997; Jones et al., 2007; Kennedy, 2015). ICC values can improve with practice (Sand et al., 1997). Kennedy (2015) used a non-specified model ICC to determine the PPT in the piriformis and gluteus medius muscles, which varied from 0.85-0.89. While Jones et al. (2007) used a non-specified model ICC to report on the reliability for PPT in the upper back, shoulder and upper arm muscles which he reported varied from 0.90-0.98. The ICC(3,1) in the present study did not fall below 0.60 and therefore can be considered clinically useful (Barbero et al., 2012). The ICC(3,1) for the PPT in the present study was 0.637-0.875 and can be interpreted as moderate to excellent (Doğan and Doğan, 2015). Supplementary testing should be conducted on TrPs regions with ICC(3,1) below 0.750 while the patient is in the position that they would be treated. The additional practice may result in improved reliability as was noted by Myburgh et al. (2011).

where \( x \) statistic was applicable, the \( x \) scores for TB and TS were 1.00; while RP, JS and LTR had \( x \) scores of 0.47-1.00. The \( x \) scores in a previous intra-rater study (Al-Shenqiti and Oldham, 2005) were 1.00 for TB, TS, JS and familiar pain; while RP and LTR had \( x \) statistics ranging from 0.75 to 1.00, where \( x \) statistic was applicable. The higher \( x \) score reported by Al-Shenqiti and Oldham (2005) may be due to the fact that subjects had rotator cuff injuries.
for at least six weeks and may not have developed central sensitisation (Xu et al., 2010) or irritated satellite TrPs (Niddam et al., 2007). Skorupska et al. (2015) reported intra-rater $\kappa$ scores of 0.56 to 0.90 when using infrared thermographic imaging of the gluteus minimus in subjects with sciatic pain as well as asymptomatic subjects. Non-peer-reviewed studies reported $\kappa$ scores of 1.00 for TB and TS; 0.88-1.00 for JS; 0.64-0.90 for LTR; and 0.4-1.00 for RP in asymptomatic gastrocnemius (O’Rourke, 2010), rotator cuffs (Hynes, 2011) and rectus femoris (Barry, 2015). The present study could be interpreted as fair ($\kappa$ 0.47-0.60), moderate ($\kappa$ 0.60-0.80) and almost perfect ($\kappa$ > 0.81) depending on site and feature (Sim and Wright, 2005). The present study reported superior reliability ($\kappa$ = 0.65-0.10) compared to many inter-rater reliability studies: $\kappa$ 0.02-1.00 (Bron et al., 2007); $\kappa$ 0.22 (Myburgh et al., 2011); $\kappa$ 0.00-0.39 (Mora-Relucio et al., 2016). Reliability seems to be dependent on the muscle investigated (Al-Shenqiti and Oldham, 2005; Kennedy, 2015) and even the orientation of the muscle fibres (Barbero et al., 2012). The lower $\kappa$ scores seem to be predominantly reported in relation to the subjective features such as RP ($\kappa$ = 0.63 to 0.65) which are similar to for the infraspinatus ($\kappa$ = 0.75) reported by Al-Shenqiti and Oldham (2005); as well as LTR ($\kappa$ = 0.67) which is similar to for the rectus femoris ($\kappa$ = 0.63) reported by Barry (2015).

Data for Bland-Altman plots (Bland and Altman, 1999), and LoA (Atkinson and Nevill, 1998) were transformed using $\ln$ due to heteroscedasticity (Chinn, 1990) of the $Y$-line. The $SD$ of the $Y$-line (VL - 0.82 to 1.31 cm; VM - 0.93 to 1.21 cm, respectively) was high relative to the mean of the $Y$-line (2.91 to 4.16 cm for the VL; 5.91 to 6.57 cm for the VM). This large deviation may be due to the girth of the thigh especially between genders (Beck et al., 2000) and possibly quadriceps strength (Slemenda et al., 1997).

There is no classification for the Bland-Altman plot, and it relies on the visual interpretation of the agreement of the within-subject variances. The closer the difference between test mean variable to the mean variable, the greater the agreement (Atkinson and Nevill, 1998; Rankin and Stokes, 1998). All between test mean variable were within the 1.96 $SD$ LoA of their respective Bland-Altman plots with one (VL-TrP$\_1$ PPT, VL-TrP$\_2$ PPT, VL-TrP$\_3$ X-line, VL-TrP$\_3$ Y-line, VL-TrP$\_4$ PPT, VL-TrP$\_5$ X-line, VL-TrP$\_5$ Y-line, VL-TrP$\_5$ PPT, VM-TrP$\_2$ X-line) or two
(VL-TrP$_1$ Y-line, VL-TrP$_2$ X-line, VL-TrP$_2$ Y-line, VL-TrP$_3$ PPT, VL-TrP$_4$ X-line, VM-TrP$_1$ PPT, VM-TrP$_2$ PPT) close outliers, except for the VL-TrP$_4$ Y-line which had three close outliers. These are similar to the findings of previous studies who had 1-2 outliers outside of the 1.96 $SD$ LoA (Nussbaum and Downes, 1998; Bijur et al., 2001; Ylinen et al., 2007; Barbero et al., 2012). Proportional bias ($\beta > 0.05$) was only observed in 2 cases (VL-TrP$_3$ Y-line and VM-TrP$_1$ PPT) suggesting that there is strong agreement (Ludbrook, 2002).

The $SEM$ for the $X$-line ranged from 0.22 to 1.49 cm. The $SEM$ for the $Y$-line ranged from 0.38 to 0.75 cm. The $SEM$ for the PPT was between 0.07 and 3.21 N. An intra-rater reliability for the change in temperature in TrPs in the upper trapezius reported a $SEM$ of 0.13-1.57° C (Dibai-Filho et al., 2015). Whereas, Koo et al. (2013) reported a $SEM$s of 1.79 to 2.58 N for measuring the PPT of TrPs in the erector spinae. A $SEM$ closer to 0.00 suggests that the measurement of a variable is accurate. The $SEM$ will be used to ascertain whether an intervention in a randomised control trial has clinical significance.

Null hypothesis 3 states “Gender will not have an effect on the number of features of TrPs.” There was a high incidence of the features of TrP in the VL and VM reported by females, this may account for the high proportion of females in previous TrP reliability studies (Nice et al., 1992; Wolfe et al., 1992; Lew et al., 1997; Scioti et al., 2001; Myburgh et al., 2008; Dibai-Filho et al., 2015; Mora-Relucio et al., 2016) and recent general TrP studies (Bakar et al., 2014; Nie-Asher et al., 2014; Yeganeh Lari et al., 2016). Therefore, Null hypothesis 3 can be rejected.

Null hypothesis 4 states “Gender will not have an effect on the sensitivity of TrPs.” The PPT was significantly lower in females ($p < 0.05$) at VL-TrP$_3$, VL-TrP$_4$, VM-TrP$_1$ and VM-TrP$_2$. The difference in gender in relation to PPT has been previously reported (Ohrbach and Gale, 1989b; Vanderweeën et al., 1996; Chesterton et al., 2003). The locations that reported the difference in gender are also the sites where the muscles are pennated in structure (Blazevich et al., 2007). The pennate structure at the middle VL and distal VM may also account for the high proportion of TrP features detected at these sites, perhaps due to short muscle fibres (Wickiewicz et al., 1983) and are susceptible to longitudinal shortening and
fatigue (Armstrong, 2011). TrPs cause the muscle fibre to contract at the site of the TrP (Mense et al., 2003) and stretch the remainder of the muscle fibre (Chen and Grinnell, 1997). It has been reported during animal in vitro studies (Williams and Goldspink, 1978; Spector et al., 1982) that if a muscle is maintained in a shortened position, there can be a reduction in numbers of series sarcomeres and muscle fibre length. It would appear that TrPs are associated with patellofemoral pain syndrome (Roach et al., 2013; Espí-López et al., 2017) and knee osteoarthritis (Bajaj et al., 2001; Yentür et al., 2003; Itoh et al., 2008b; Mayoral et al., 2013; Ichikawa et al., 2015; Alburquerque-García et al., 2015) especially in females. Therefore, Null hypothesis 4 can be rejected.

This was the first study to assess the reliability of locating TrPs in the VL and VM. Previous intra-rater reliability studies have focused on the trapezius muscle (Barbero et al., 2012; Dibai-Filho et al., 2015) and the gluteus minimus (Skorupska et al., 2015). It has been recommended that reliability should be completed on every muscle as the reliability of TrP location is highly dependent on the muscle, and the rater’s expertise and training (Myburgh et al., 2008; Lucas et al., 2009; McEvoy and Huijbregts, 2011; Barbero et al., 2012). The present study was limited to the two muscles in order to reduce patient recall bias (Myburgh et al., 2008).

Previous studies have been concerned with replicating the features of TrPs (Nice et al., 1992; Wolfe et al., 1992; Lew et al., 1997; Gerwin et al., 1997; Al-Shenqiti and Oldham, 2005; Bron et al., 2007; O’Rourke, 2010; Hynes, 2011; Myburgh et al., 2011; Kennedy, 2015; Mora-Relucio et al., 2016). There have been attempts to determine the reliability of clinicians to locate TrPs (Sciotti et al., 2001; Barbero et al., 2012; Skorupska et al., 2015; Dibai-Filho et al., 2015; Barry, 2015). Some studies have attempted to measure the severity of TrPs using the PPT (Jones et al., 2007; Gulick et al., 2011; Kennedy, 2015). This is the first study to attempt to measure the location and measure the severity of TrP. The present study was reliable in measuring the location and severity of TrP.

Being able to blind subjects and therapists allows for more robust testing (Myburgh et al., 2008). It is inherently difficult when the confirmation of TrPs rely on the patient’s feedback.
in relation to TS, RP and LTR as well as the therapist depending on multiple scenes to confirm TB, JS and LTR. The use of UV markers and UV light (Kennedy, 2015; Mora-Relucio et al., 2016) allows the therapist to use all of their senses while locating TrPs. A previous study that measured the location of TrPs blindfolded the rater (Barbero et al., 2012). Therefore the rater may not have been able to use all of their senses to determine the location of the TrP. The present study combined these methods to use reliably measure the location of the TrPs while remaining blind to the location of the previous test.

It has been suggested that reliability and agreement studies should use multiple statistical methods to avoid errors in interpreting statistical findings (Chinn, 1990; Atkinson and Nevill, 1998; Rankin and Stokes, 1998; Bland and Altman, 1999; Batterham and George, 2000; Ludbrook, 2002; Weir, 2005; Kottner et al., 2011b, 2011a). The present study utilised the $\kappa$ score (Sim and Wright, 2005) for reliability and percent agreement for agreement in nominal data (Kottner et al., 2011b, 2011a) such as the features of TrPs. The reliability of ratio data was analysed using ICC $\left(3:1\right)$ to account for one observer and where random error variability is expected (Atkinson and Nevill, 1998; Rankin and Stokes, 1998; Batterham and George, 2000). The agreement was analysed using SEM (Weir, 2005), LoA and Bland-Altman plots (Atkinson and Nevill, 1998; Bland and Altman, 1999) with proportional bias accounted for (Ludbrook, 2002).

### 3.4.1 Clinical relevance

The ALS can be considered a clinically reliable tool to measure the location of TrPs in the VL and VM. This is important when the same therapist is treating a patient over multiple treatment sessions. The ALS could also be used when there are multiple therapists who use clinical notes to provide a consistent level of care for patients with TrPs. The PPT can be used to measure the severity of TrPs (Gulick, 2014). The SEM could be utilized as a clinical significance in an intervention trial. If the PPT changed by more than SEM, then it could be suggested that there is a clinical improvement.

The present study established that there was gender difference for the PPT and features of TrPs at different sites. The gender difference in relation to the PPT corroborates the finding
of previous studies (Ohrbach and Gale, 1989b; Vanderweeën et al., 1996; Chesterton et al., 2003). The high rates of the features of TrPs in the VL and VM in females suggest the there may be a connection between TrPs in the musculature that govern movement and support the knee and pathologies related to the knee commonly associated with females such as patellofemoral pain syndrome (Roach et al., 2013; Espí-López et al., 2017) and knee osteoarthritis (Bajaj et al., 2001; Yentür et al., 2003; Itoh et al., 2008b; Mayoral et al., 2013; Ichikawa et al., 2015; Alburquerque-García et al., 2015).

### 3.4.2 Limitations

The range of the PPT in the present study may be due to gender perceptions of the PPT (Chesterton et al., 2003) or due to muscle architecture (Blazevich et al., 2007) at the different sites. Another reason for the variance of the PPT could have been a treatment effect in some subjects as there was a reduction in RP at VL-TrP₂ and VM-TrP₂; a decrease in JS at VL-TrP₁, VL-TrP₂, VL-TrP₃, and VM-TrP₂; a reduction in LTR at VM-TrP₁ on the second day of testing (Sand et al., 1997). The pressure needed to reach the PPT is maintained for less than 3 sec for the algometer to record the peak pressure. Algometers have been used as a treatment option for TrPs in the trapezius, but the pressure was maintained for three sets of one minute (Abu-Taleb et al., 2016). However, the limited duration of the placement of the algometer could have a sensitisation effect in other subjects as there was an increase in LTR at VL-TrP₁, VL-TrP₃, VL-TrP₄ and VL-TrP₅ (Nussbaum and Downes, 1998; Jones et al., 2007).

The 95% CI for the ICC(3,1) for the ALS and PPT were in the order of 0.246 to 0.900 for the X-line, 0.133 to 0.883 for the Y-line, and 0.103 to 0.936 for the PPT. Thus, suggesting that larger sample size would be needed to for future reliability studies with respect to ALS and PPT.

The time needed for any positive or adverse effects of the PPT to return to pre-test levels has not been fully established and requires further investigation as it may affect the reliability of using the PPT to measure the sensitivity of TrPs (Sand et al., 1997; Jones et al., 2007; Nussbaum and Downes, 1998).
Were the present study to be repeated, a longer period between test sessions should be used to prevent any treatment or sensitising effect from the PPT (Sand et al., 1997; Nussbaum and Downes, 1998; Jones et al., 2007). The duration of any sensitising or treating effects caused by the use of the algometer has not been fully established and need further investigation (Sand et al., 1997; Jones et al., 2007; Nussbaum and Downes, 1998).

The present study used the non-dominant leg (standing leg) to determine whether latent TrPs were present in the VL and VM of asymptomatic subjects. It has been reported that the standing leg is not as strong as the kicking leg (Ross et al., 2004). If the standing leg is used more in weight bearing positions, it may be susceptible to fatigue and therefore TrPs (Castaldo et al., 2014). This may explain the increased pain and stiffness in the quadriceps of the nondominant leg after the induced delayed onset of muscle damage reported by Hody et al. (2013), even though the results of that study were not significant (p > 0.05).

The present study, unlike previous studies, did not determine the inter-rater reliability nor compared experts to non-experts (Nice et al., 1992; Njoo and van der Does, 1994; Myburgh et al., 2011). There are no studies that investigate the inter-rater reliability of relocating TrP using ratio data such as the ALS (Barbero et al., 2012) in conjunction with PPT (Vanderweeën et al., 1996; Jones et al., 2007; Abu-Taleb et al., 2016) and should be considered in future studies.

TrPs develop with excessive load (Dommerholt et al., 2011) or from trauma (Huang et al., 2013), inevitably in weight bearing positions such as running (Barry, 2015) or jumping (O’Rourke, 2010; Devereux, 2016). The palpation of TrPs is conducted in a non-weight bearing position with the muscle in a relaxed state (Travell and Simons, 1992). TrPs are characterised by endplate noise from abnormal acetylcholine release which may alter EMG activity (Simons, 2004b). However, many of the studies that have examined the EMG activity of TrPs had the subject in a non-weight bearing position as the study used inter-muscle EMG (Simons, 2001; Simons et al., 2002). Studies that have accessed EMG activity in a functional test have been in the upper body (Donohuge et al., 2016). Therefore, future
studies examining TrPs in the leg should consider monitoring EMG activity in TrP in functional movements such as jumping.

3.4.3 Conclusion

TrP in the quadriceps femoris have been associated with knee pathologies and may affect athletic performance. The ICC of the ALS to measure the location of latent TrPs and the PPT to measure the severity of latent TrP in the VL and VM was assessed. The finding of the present study suggests that the ALS in combination with the PPT are reliable methods to locate and determine the severity of latent TrP in the VL and VM. There appears to be a difference between sites in the VL and VM as well as between genders in relation to the presence and severity of TrP in the VL and VM. Future studies should establish the effects of TrP on functional strength, athletic performance of the lower limb and pathologies such as patellofemoral pain syndrome and knee osteoarthritis.
3.4.4 Supplementary reliability

Some of the ICC(3:1) data from the TrP location reliability study were below the 0.750 value set as excellent in the methods (Doğan and Doğan, 2015). This possibly may have been due to the position of the leg when measuring the ALS for the VL. In the TrP location reliability study, the ALS of VL was measured with the subject in supine; this was to an attempt to keep the hip in a neutral position. When treating the VL, the subject should be in side-lying to allow maximum exposure to the whole muscle (Travell and Simons, 1992). Therefore, side-lying may be a better option to measure the VL. Additional practice of between 40 and 81 cases has also been shown to improve the reliability of inexperienced raters (Myburgh et al., 2011).

3.4.4.1 Supplementary reliability methods

The observed power for the repeated measure of the X-line of the VL-TrP1 was 0.07 (β). A priori sample size calculation (one tail, ρ 0.5, α 0.05, 1-β) was performed using G*Power: Statistical Power Analyses™ 3 (Universität Kiel, Germany) for Windows® and was determined that the minimum sample size needed to be six. Seven subjects were retested to determine the ICC(3,1) of the ALS sites that had an ICC(3,1) of less than 0.750 in the ALS reliability study. The seven subjects who were recruited had the highest number of features of TrPs (RP, JS, or LTR) in the ALS reliability study.

The subjects were placed in side-lying with the top knee resting on a pillow to prevent adduction to measure the VL. The knee was placed into 30.00° of flexion measured with a True-Angle® goniometer (Gaiam-Pro, Boulder, Colorado, USA). One ALS was drawn, with a Sharpie® permanent marker (Newell Brands, Downers Grove, Illinois, USA) for the VM and VL, respectively. The ALS was from the greater trochanter to the medial angle of the patella. The ALS was marked with visible ink to allow the skin to move free of the fascia between sessions (Benjamin, 2009). The order of muscle and subsequent sites were randomised (Excel random function). The TrP, X-line and Y-line were marked with UV ink and measured with a tape measure, to account for the curvature of the thigh, while under UV illumination.
### 3.4.4.2 Supplementary reliability results

The ICC\(_{(3,1)}\) for the \(X\)-line ranged from 0.764 (CI 95\%: -0.443 to 0.965) to 0.992 (CI 95\%: 0.919 to 0.998) and the \(SEM\) from 0.22 to 1.21; whereas the \(Y\)-line had an ICC\(_{(3,1)}\) from 0.854 (CI 95\%: 0.204 to 0.978) to 0.960 (CI 95\%: 0.798 to 0.993) and a \(SEM\) of 0.17 to 0.93 (Table 11).

**Table 11: Reliability, ICC\(_{(3,1)}\), and agreement measure, \(SEM\), of between session mean distance in relation to the ALS (supplementary reliability)**

<table>
<thead>
<tr>
<th>Site</th>
<th>(X1 \pm SD) (cm)</th>
<th>(X2 \pm SD) (cm)</th>
<th>(X ICC_{(3,1)}) (95% CI)</th>
<th>(X SEM)</th>
<th>(Y1 \pm SD) (cm)</th>
<th>(Y2 \pm SD) (cm)</th>
<th>(Y ICC_{(3,1)}) (95% CI)</th>
<th>(Y SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VL-TrP(_2)</td>
<td>29.66 ± 2.01</td>
<td>29.93 ± 2.12</td>
<td>0.992* (0.919-0.998)</td>
<td>0.22 ± 1.02</td>
<td>2.20 ± 0.90</td>
<td>0.960* (0.798-0.993)</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>VL-TrP(_3)</td>
<td>32.10 ± 3.82</td>
<td>35.89 ± 3.20</td>
<td>0.899* (0.299-0.977)</td>
<td>1.21 ± 0.84</td>
<td>2.86 ± 1.05</td>
<td>0.854* (0.204-0.978)</td>
<td>0.34</td>
<td></td>
</tr>
<tr>
<td>VL-TrP(_4)</td>
<td>33.61 ± 1.69</td>
<td>33.33 ± 2.72</td>
<td>0.917* (0.574-0.987)</td>
<td>0.49 ± 1.02</td>
<td>4.26 ± 0.87</td>
<td>0.876* (0.155-0.971)</td>
<td>0.36</td>
<td></td>
</tr>
<tr>
<td>VL-TrP(_5)</td>
<td>9.03 ± 2.07</td>
<td>9.79 ± 2.48</td>
<td>0.952* (0.544-0.989)</td>
<td>0.55 ± 0.64</td>
<td>2.41 ± 0.54</td>
<td>0.981* (0.907-0.997)</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>VM- TrP(_1)</td>
<td>38.11 ± 3.01</td>
<td>38.34 ± 2.66</td>
<td>0.981* (0.906-0.997)</td>
<td>0.24 ± 1.57</td>
<td>3.40 ± 1.28</td>
<td>0.937* (0.673-0.991)</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>VM- TrP(_2)</td>
<td>34.06 ± 1.81</td>
<td>34.24 ± 1.96</td>
<td>0.764* (-0.443-0.965)</td>
<td>0.52 ± 1.84</td>
<td>6.50 ± 1.30</td>
<td>0.862* (0.245-0.979)</td>
<td>0.93</td>
<td></td>
</tr>
</tbody>
</table>

Note: TrP is trigger point, \(SD\) is standard deviation, ICC\(_{(3,1)}\) is intra-class coefficient (mixed, absolute model), \(SEM\) is standard error of measurement, 95\% CI is 95 percent confidence interval, ALS is the anatomical landmark system (Barbero et al., 2012), \(X(n)\) is \(X\)-line (session number), \(Y\) is \(Y\)-line (session number), VL is vastus lateralis, VM is vastus medialis (\(n = 7\)). * ICC\(_{(3,1)}\) statistical significance between test and re-test (\(p < 0.05\)).
3.4.4.3 Supplementary trigger point location reliability testing

Seven subjects were re-tested for any ALS variable that reported ICC(3,1) of less than 0.75. The ICC(3,1) for the second phase was 0.764 (CI 95%: 0.443 to 0.965) to 0.992 (CI 95%: 0.919 to 0.998) for the X-line: and 0.8554 (CI 95%: 0.204 to 0.978) to 0.960 (CI 95%: 0.798 to 0.993) for the Y-line. The improved results may be due to the additional practice of locating TrP during phase two which mirror the results of Myburgh et al. (2011) who reported improved inter-rater reliability, between non-experienced and experienced clinicians, after eighty-one cases. Or perhaps that the ALS used bony landmarks on the femur (greater trochanter) and the patella thus reducing the effect of hip and knee movement and are closer to the origins and insertions of the VL (lateral intertrochanteric line, margin of greater trochanter, lateral margin of gluteal tuberosity and lateral lip of linea aspera to quadriceps femoris tendon and patella) and VM (medial intertrochanteric line, pectineal line, medial lip of linea aspera to quadriceps femoris tendon and patella).

The work conducted in the present study means that the rater will be able to accurately measure the location and severity of TrPs in the VL and VM in a randomised control trial on the effects of DN and rESWT on jump performance.
The effects of dry needling and shockwave on trigger points: a randomised control study

4.1 Introduction

TrPs can cause pain locally (Gerber et al., 2015; Sumen et al., 2015; Paolucci et al., 2016) or refer pain (Fernández-de-Las-Peñas et al., 2010, 2011; Ghanbari et al., 2012; Berggreen et al., 2012; Karadaş et al., 2013; Espí-López et al., 2014a, 2014b; Taylor, 2014). Latent TrPs are not spontaneously painful (Bron and Dommerholt, 2012). Latent TrPs can reduce strength (Celik and Yeldan, 2011) and can cause a muscle to remain active even during an agonist contraction, thus preventing reciprocal inhibition (Ibarra et al., 2011), potentially leading to altered activation patterns (Lucas et al., 2010). A subject with latent TrPs may not be aware that they have TrPs or that the latent TrPs may be altering joint kinematics (Jafri, 2014).

Jumping involves many muscles around the hip, knee and ankle with the quadriceps extending the knee (Lorenz, 2016). Latent TrPs may prevent an athlete from performing at full potential (Ameloot et al., 2016). The CMJ and DJ are often used as outcome measures for athletic performance (Markwick et al., 2015; Gallardo-Fuentes et al., 2016; Jarvis et al., 2016), and PPT has been used to measure the sensitivity of TrPs (Sciotti et al., 2001).

DN is a treatment for TrPs and uses filament acupuncture needles to stimulate the muscle directly (Mendigutia-Gómez et al., 2016; Ortega-Cebrian et al., 2016; Vas et al., 2016). DN has had much success as a treatment option for TrPs (Fernández-de-Las-Peñas et al., 2010, 2011; Ghanbari et al., 2012; Berggreen et al., 2012; Karadaş et al., 2013; Espí-López et al., 2014a, 2014b; Taylor, 2014). DN causes post-needling soreness lasting up to 72 hours (Martín-Pintado-Zugasti et al., 2015, 2016) and has been shown to negatively affect athletic performance for 24 hours (Barry, 2015; Kennedy, 2015; Devereux, 2016). rESWT has recently been proposed as a treatment of TrPs (Jeon et al., 2012; Ji et al., 2012; Ramon et al., 2015). rESWT offers a non-invasive treatment strategy for TrPs and may be a treatment that may not cause post-treatment soreness.
Previous studies that have investigated TrPs in the VL and VM have focused on patients with pathologies. There is a substantial gap in the literature in relation to effects of TrPs on athletic performance, especially in the lower limb. Therefore, the aim of the present study is to determine the effects of DN and rESWT on the sensitivity of latent TrPs in the VL and VM as well as their effects on athletic performance during jumping.

### 4.1.1 Null hypotheses

1. The rater will not be reliable at measuring the CMJ-JH, DJ-JH, DJ-tc and DJ-RSI.
2. There will be no correlation between physiological measures and jump outcomes.
3. Using DN or rESWT to treat TrPs in the VL and VM will not affect the PPT.
4. Treating TrPs in the VL and VM with DN or rESWT will not affect CMJ jump performance.
5. Gender will not have an effect on jump performance.
6. Treating TrPs in the VL and VM with DN or rESWT will not affect the jump performance of the DJ.

### 4.2 Methods

This investigation was conducted following the Consolidated Standards of Reporting Trials (CONSORT) 2010 guidelines for randomised control trial (see Appendix E [p. Apx. xviii])

#### 4.2.1 Procedure

The present study was a parallel with equal allocation ratio and was approved by the Institute of Technology Carlow’s Ethics Board (see Appendix F [p. xviii]). A full safety statement was carried out before data collection started (see Appendix G [p. xxxix]). Subjects were screened for physiological measures including age, height, body mass, and BMI. Subjects were then tested for the presence of TrPs using the flat palpation method to meet the inclusion criterion having at least two TrPs in both the VL and VM. If TrPs were present, the subject was selected for testing. If more than two TrPs were present, then the two most tender TrPs in each of the VL and VM were treated.
The order of locating the TrPs was randomised according to the leg and muscle using the random function in Microsoft Excel™ 2013 for Windows™ 7 (Redmond, Washington, USA). The four muscles were right VM, right VL, left VM and left VL. For the palpating of TrPs in the VM, subjects were placed in a supine position. For the palpation of the VL, subjects were positioned in side-lying with their hips and knees at 30° of flexion and a pillow between their knees. Subjects were draped with a towel for modesty. The thigh to be treated was exposed as needed for all interventions. The location of the TrPs at the Baseline testing was recorded using the ALS as described by Barbero et al. (2012). The location of the TrPs for subsequent testing sessions was determined using the ALS.

Subjects performed five minutes of self-paced light jogging over a 20 m distance and five minutes of dynamic stretches targeting the gluteus, hamstrings, adductors, quadriceps, and gastrocnemius over a 20 m distance before every session (Turki et al., 2011). Subjects were ranked according to their performance of a DJ performed on an Opto-Jump Next™ PTD from Microgate (Bolzano Italy) to stratify group allocation. The PTD is a reliable measure of JH derived from with an ICC of 0.95 (Glatthorn et al., 2011). The outcomes were measured before each treatment, with PPT being conducted first to prevent any effects to JH caused by possible TrP sensitisation. The three treatment sessions were conducted over one week (DiLorenzo et al., 2004; Ilbuldu et al., 2004; Itoh et al., 2004a, 2006, 2007; Ziaeifar et al., 2014). Follow-up outcome sessions were conducted 2-4 days after, depending on the availability of the laboratory such a weekend (Ziaeifar et al., 2014) and seven days after the last treatment (Williamson et al., 2007; Itoh et al., 2008a, 2008b; Pecos-Martín et al., 2015). The order the TrPs were palpated was randomised (Excel™) for every session.

4.2.2 Subjects

Twenty-seven recreational athletes who participate in more than four hours a week of a sport that involve jumping such as athletics, basketball, dancing, Gaelic football, hurling (or camogie), rugby, soccer, tennis, or volleyball were sampled using convenience sampling from the Health Science and Sports Departments in Institute of Technology, Carlow, between September 2016 and February 2017. Before testing, subjects completed a
screening and informed consent form (see Appendix H [p. Apx.xviii]). Subjects were precluded from the study if they were not between 18 and 35 years of age; having or have had a systemic diseases of the muscular or nervous system, congenital or acquired hip disease; hip or knee trauma and or surgery in the lower extremity in the past 12 months; inflammatory joint disease, tumours, lower limb or lower back injury requiring treatment in the previous six months. One subject (n = 1) had a lower limb injury in the previous six months and was precluded. Subjects also had to have met the inclusion criterion, of having at least two latent TrPs in both the VL and VM. One subject (n = 1) failed to meet the inclusion criterion. Two subjects (n = 2) declined to participate in the study. Twenty-three subjects began testing. Two subjects (n = 2) were not analysed as they withdrew from the study, one for personal reasons and one because of an ankle injury. Subjects were asked to refrain from vigorous exercise 24 hours before testing.

### 4.2.3 Interventions

Subjects were placed in the same position as they were for the palpation of the TrPs. Group allocation was randomised using block design. Block allocation was stratified according to the rank of DJ performance (highest ranking eight, middle ranking seven and bottom ranking eight). Subjects in each block were randomly assigned to one of the treatment groups or the control group using the random function (Excel™ 2013): DN group (n = 8), rESWT group (n = 8) or control group (n = 7). Subjects were treated in the same order as they were palpated to measure the PPT. Enrollment, randomisation and assignment were conducted by the Masters candidate.

#### 4.2.3.1 Needling

For the DN group a 0.30 mmX60 mm L-type™ disposable sterile stainless steel acupuncture needle (Seirin Corporation, Shizuoka, Japan) was inserted into the two most painful TrPs in the VL and VM, bilaterally (n = 8), using a cone shape technique (Hong and Hsueh, 1996; Hsieh et al., 2000). Once a LTR was elicited, the sensitive loci within the TrPs were stimulated dynamically using the fast-in-fast-out technique for 30 seconds (Salom-Moreno et al., 2014). If the fast-in-fast-out stimulation was too painful to be tolerated by the subject,
then the needle was stimulated statically by twisting the needle. If there was still a LTR present, then a subsequent 30 seconds of treatment was applied. TrP stimulation continued in 30 seconds increments until LTR was exhausted (Hsieh et al., 2012) or for a maximum of two minutes (Barry, 2015).

### 4.2.3.2 Extracorporeal shockwave therapy

In the rESWT group, rESWT was applied bilaterally to the two most painful TrP in the VL and VM \( (n = 8) \) with an Intellect™ radial pressure wave shockwave therapy device (Chattanooga™, DJO Global, Guildford, Surrey, UK). The maximum tolerable pressure setting was used for each subject (Schmitz et al., 2015). Each TrP was treated using the 15mm Deep Impact™ (DI15) transducer head with 1000 pulses at 20Hz (Schmitz et al., 2015) with the transducer head left in situ. The surrounding tissue was treated using the 35 mm D-ACTOT™ (D35) transducer head with 2000 pulses at 20Hz (Gleitz and Hornig, 2012) with the transducer head travelling in alignment with the fibres of the muscle.

### 4.2.3.3 Control

The control group did not receive any treatment with the aim being to determine the training effect, if any, of the jump tests, as jump testing requires maximal effort which is similar to jump training (Chelly et al., 2015). The control group rested for 7 minutes in each of the four positions the TrPs were palpated in.

### 4.2.4 Outcomes

Outcome measures were used to assess the severity of the TrPs and the athletic performance. The outcomes measures used were the PPT, CMJ-JH, DJ-JH, DJ-\( t_c \) and DJ-RSI. The PPT was conducted before maximum jump tests to avoid any sensitisation due to fatigue or hypoxia (Shah et al., 2008). Subjects were allowed to perform two practice jumps before testing at all outcome measuring sessions using PTD with \( JH \) derived by \( t_r \). If there was more than 5% difference in \( JH \) (≈1 cm), the subject was allowed to complete two more jump tests. Three minutes recovery was allowed before trial jumps (Moir et al., 2008). Subjects performed three of the respective jumps (Markovic et al., 2004; Moir et al., 2004; Maulder and Cronin, 2005; Moir et al., 2008; Enoksen et al., 2009; Sassi et al., 2009; Ditroilo

Walsh, R. (2017)
et al., 2011; Staehli et al., 2010; Nuzzo et al., 2011). A 9286BA™ portable FP from Kister (Belgrade, Serbia) operated by SmartPerformance™ software (BTS Bioengineering SpA: Milan, Italy) sampling at 1,000 Hz was used (Hori et al., 2009) simultaneously with the PTD. The PTD straddled the FP to allow the photocell beams to be broken when the subjects landed on the FP. The JH was determined from $t_f$ (see Equation 1 [p. 44]). The mean of three jumps was analysed. Subjects rested for 60 seconds between all jumps (Ebben et al., 2008; Acero et al., 2011; Nuzzo et al., 2011; Mizuguchi et al., 2015). The order of the jump tests was randomised (Excel™ 2013).

4.2.4.1 The pressure pain threshold
The PPT was used to measure the severity of TrPs and has been shown to be reliable (Castagna et al., 2013). The PPT was measured using a Commander™ algometer (JTECH medical, Midvale, Utah, USA) and was described to the subject as “The point where the sensation of pressure turned to a perception of pain” (Ohrbach and Gale, 1989b). To prevent bruising a 1cm² head was used (Takahashi et al., 2005). The rater was blinded to the pressure placed through the algometer by using the maximum pressure recall feature of the algometer. The rater increased pressure via the algometer by 1N·s⁻¹ (Nussbaum and Downes, 1998) until the subject reported the change of the pressure to pain.

4.2.4.2 Countermovement jump
The CMJ was used to measure lower limb slow SCC and has been shown to be reliable. The subjects started the jump from an upright position. During the CMJ, the subjects were instructed to “keep their hands on their hips and squat to a self-selected depth of approximately 90° knee flexion, and jumped immediately as high as possible without pausing” (Castagna et al., 2013; Farias et al., 2013).

4.2.4.3 Depth-jump
The bounce DJ was used to measure the lower limb fast stretch-shortening cycle. A standardised jump height was set at 0.30 m to allow a low enough height to maintain a contact time quicker than 0.25 seconds and to prevent a possible training effect (Chelly et al., 2015). The subjects were instructed to “step off the box and land on the force platform
and jump as high and as fast as possible” (Young et al., 1995). The subjects kept their hands on their hips.

4.2.5 Sample size

The sample size was calculated using Equation 6 (Gissane, 2015), where the standard deviation was determined from the retest of the PPT data of the distal posterolateral aspect of the VL (22.55 ± 5.82 N) in the PPT reliability study conducted previously in this thesis (see Results from Chapter 3 [p.110]). This data was chosen as it was the lowest ICC\(_{(3,1)}\) (0.644 [95% CI:0.246-0.832]) of all the sites in relation to the PPT, thus, ensuring that the power was significant enough to prevent a type II error in the results. The minimum detectable change (MDC), as illustrated in Equation 7 (Koo et al., 2013) was calculated, that in order to have a power of 0.80, with an α level of 0.05 with a level of confidence of 1.96. It was determined that a minimum of seven (n = 7) subjects were needed for each group. The JH and reliability data of the DJ, from Beattie and Flanagan’s (2015) study of junior international rugby players, suggest that seven is also enough subjects to demonstrate appropriate statistical power in relation to jump performance when the same equation was used.

\[
n = 16 \cdot \frac{SD^2}{MDC^2}
\]

Equation 7: Sample size generation.

Where \(n\) is group size, \(SD\) is standard deviation, \(MDC\) is minimal detectable change.

\[
MDC = (SD \sqrt{(1 - ICC)}) \cdot 1.96 \cdot \sqrt{2}
\]

Equation 8: Minimum detectable change.

Where \(MSD\) is minimum detectable change, \(SD\) is standard deviation, \(ICC\) is inter-class correlation.

4.2.6 Statistical analysis

The mean PPT, CMJ-JH, DJ-JH, DJ-\(t_c\) and DJ-RSI were assessed for normal distribution using the one sample Kolmorogov-Smirnov test. To ascertain if there are any covariance for jump
performance correlations were performed between the physiological measures, PPT and jump measures using a Spearman's correlation coefficient $\rho (r_s)$. > 0.30 and < 0.70 was moderate correlation, > 0.70 was set as good correlation (Bron et al., 2011a). The reliability of jump variables was analysed using $ICC_{(3,1)}$, CV, and $SEM$ as outlined by Rankin and Stokes (1998). A principal components analysis (PCA) was conducted to derive a body composition factor and a PPT factor. A two-way analysis of variance (ANOVA) was performed for the PPT. Variances were session time (Baseline (B-line), Treatment 1 [Tx$_1$] [1-4 days after the B-line]; Treatment 2 [Tx$_2$] [2-3 days after Tx$_1$], Treatment 3 [Tx$_3$] [2-3 days after Tx$_2$], Follow-up 1 [FU$_1$] [2-4 days after Tx$_3$], Follow-up 2 [FU$_2$] [7 days after Tx$_3$]) and intervention group (DN, rESWT, control). A Three-way analysis of covariance (ANOCVA) was conducted to determine the statistically significant difference between 1) session time (B-line, Tx$_1$, Tx$_2$, Tx$_3$, FU$_1$, FU$_2$); 2) intervention group (DN, rESWT, control); 3) gender (male, female), controlling for the PPT of the most tender and second most tender TrPs of the VL and VM of both legs for the $JH$ of the CMJ. A Two-way analysis of covariance (ANOCVA) was conducted to determine the statistically significant difference between session time (B-line, Tx$_1$, Tx$_2$, Tx$_3$, FU$_1$, FU$_2$) and intervention group (DN, rESWT, control) controlling for the PPT and BMI for the $JH$ of DJ as well as the $t_c$ and $RSI$ of the DJ. A statistical significance of $p < 0.05$ was set. All statistical analyses were conducted in SPSS™ version 22 software (SPSS Incorporated, Chicago, Illinois, USA).
4.3 Results

The study schematic is illustrated in Figure 12.

![Study Schematic](image)

Figure 12: The effects of DN and rESWT study flow chart. Where DJ is depth jump, TrP is trigger points, VL is vastus lateralis, VM is vastus medialis, DN is dry needling, rESWT is radial extracorporeal shockwave therapy Tx is treatment.

4.3.1 Normality

Twenty-one subjects completed the testing and were assessed. Data for age, height, mass, VM-ALS\_d (Left and right), and VL-ALS\_d (Left and right) were tested for normality using the one sample Kolmorogov-Smirnov test and were found to be normally distributed (p > 0.05).
4.3.2 Descriptive

The mean age of the female subjects (n = 12) was 22.42 (95% CI: 20.74 to 24.10) years; compared to the mean age of the males (n = 9), which was 23.44 (95% CI: 21.13 to 25.75) years. The mean mass of the male subjects was 78.16 (95% CI: 68.71 to 87.61) kg; while the mean mass of the females was 63.65 (95% CI: 54.49 to 72.91) kg. The mean stature of the males was 1.78 (95% CI: 1.73 to 1.84) m; whereas, the females had a mean height of 1.65 (95% CI: 1.60 to 1.70) m. The mean BMI of the male subjects was 24.38 (95% CI: 23.86 to 24.90) kg∙m$^{-2}$; while the mean BMI of the females was 23.22 (95% CI: 22.47 to 23.97) kg∙m$^{-2}$.

Physiological measures are presented in Table 12.

Table 12: Physiological measure

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Sample group (n=22) (95% CI)</th>
<th>DN (n=7) (95% CI)</th>
<th>rESWT (n=7) (95% CI)</th>
<th>Control (n=7) (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (% female)</td>
<td>57.1% (95% CI)</td>
<td>57.1% (95% CI)</td>
<td>62.5% (95% CI)</td>
<td>42.9% (95% CI)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>22.86 (21.59 - 24.12)</td>
<td>22.28 (19.86 – 24.71)</td>
<td>23.00 (20.14 – 25.86)</td>
<td>22.71 (20.23 – 25.20)</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>69.87 (62.95 - 76.79)</td>
<td>70.56 (55.72 – 85.40)</td>
<td>66.72 (52.56 – 80.88)</td>
<td>72.76 (61.24 – 84.29)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.71 (1.66 - 1.76)</td>
<td>1.74.21 (1.63 – 1.84)</td>
<td>1.70 (1.66 – 1.75)</td>
<td>1.69 (1.58 – 1.82)</td>
</tr>
<tr>
<td>BMI (kg∙m$^{-2}$)</td>
<td>23.72 (21.90 - 25.54)</td>
<td>23.03 (20.14 – 25.91)</td>
<td>22.95 (18.51 – 27.38)</td>
<td>25.17 (22.69 – 27.63)</td>
</tr>
<tr>
<td>Hours of physical activity per week (hours)</td>
<td>6.71 (5.45 - 7.98)</td>
<td>6.36 (3.13 – 9.58)</td>
<td>5.62 (3.33 – 7.91)</td>
<td>6.79 (2.92 – 10.65)</td>
</tr>
<tr>
<td>Standing leg (% right)</td>
<td>28.6%</td>
<td>28.6 %</td>
<td>25.0%</td>
<td>28.6%</td>
</tr>
</tbody>
</table>

*Where VM is vastus medialis, VL is vastus lateralis, ALS$\_d$ is estimated length of muscle using the anatomical landmark system (n=21).*
4.3.3 Jump reliability

The mean CMJ-JHs were 27.03 (95%CI: 23.87 to 30.19) cm and 28.16 (95% CI: 24.82 to 31.52) cm measured with the PTD and 26.06 (95% CI: 23.87 to 30.19) cm to 26.89 (95% CI: 23.55 to 30.24) cm when measured with the FP. The ICC(3:1) was 0.961 for the PTD and 0.928 for the FP.

The mean DJ-JHs were 22.63 (95% CI: 19.90 to 25.36) cm and 22.26 (95% CI: 19.13 to 25.39) cm measured with the PTD and 23.17 (95% CI: 20.65 to 25.69) cm and 26.89 (23.55 to 30.24) cm when measured with the FP. The ICC(3:1) was 0.956 for the PTD and 0.919 for the FP. All ICC(3:1), CV and SEM are illustrated in Error! Reference source not found.

Table 13: Intrarater reliability of the performance outcomes

<table>
<thead>
<tr>
<th></th>
<th>PTD</th>
<th></th>
<th></th>
<th></th>
<th>FP</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B-Line</td>
<td>Tx1 (SD)</td>
<td>ICC(3:1)</td>
<td>95% CI</td>
<td>SEM</td>
<td>B-Line</td>
<td>Tx1 (SD)</td>
<td>ICC(3:1)</td>
</tr>
<tr>
<td>CMJ-JH (cm)</td>
<td>27.03 (6.36)</td>
<td>28.16 (6.74)</td>
<td>0.961</td>
<td>0.822-0.903</td>
<td>4.54 1.26</td>
<td>26.06 (6.21)</td>
<td>26.89 (6.72)</td>
<td>0.928</td>
</tr>
<tr>
<td>DJ-JH (cm)</td>
<td>22.63 (5.49)</td>
<td>22.26 (6.29)</td>
<td>0.956</td>
<td>0.891-0.983</td>
<td>5.99 1.15</td>
<td>23.17 (5.07)</td>
<td>23.08 (6.58)</td>
<td>0.919</td>
</tr>
<tr>
<td>(t_c) (s)</td>
<td>0.20 (0.02)</td>
<td>0.19 (0.02)</td>
<td>0.818</td>
<td>0.536-0.930</td>
<td>4.38 0.01</td>
<td>0.19 (0.01)</td>
<td>0.19 (0.01)</td>
<td>0.838</td>
</tr>
<tr>
<td>RSI (m/s)</td>
<td>1.15 (0.33)</td>
<td>1.20 (0.49)</td>
<td>0.902</td>
<td>0.750-0.962</td>
<td>7.59 0.10</td>
<td>1.21 (0.29)</td>
<td>1.21 (0.41)</td>
<td>0.861</td>
</tr>
</tbody>
</table>

Where PTD is photocell timing device, FP is force platform, CMJ is countermovement jump, JH is jump height, SD is standard deviation, ICC(3:1) is a mixed absolute model intrarater correlation, 95% CI is 95% confidence intervals, CV is coefficient of variation, SEM is standard error of measurement, DJ is depth jump, \(t_c\) is contact time, RSI is reactive strength index (n=23).
4.3.4 Correlation of pressure pain threshold and jump height

Moderate negative $r_s$ were found between the PPTs in the VM and CMJ-JH, DJ-JH, and DJ-RSI when measured with the PTD, but only between the PPT and the DJ-JH, when measured with the PF. Moderate positive $r_s$ was only reported between the PPT in the VL of the standing leg and DJ-$t_c$. $r_s$ between PPT of all sites and jump measure are presented in Table 14.

Table 14: The Spearman's correlation coefficient rho between pressure pain threshold and jumps measures

<table>
<thead>
<tr>
<th>(r$_s$)</th>
<th>PTD</th>
<th></th>
<th>FP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CMJ-JH (cm)</td>
<td>DJ-JH (cm)</td>
<td>DJ-$t_c$ (s)</td>
</tr>
<tr>
<td>S-VL PPT (N)</td>
<td>-0.079</td>
<td>-0.198</td>
<td>0.347</td>
</tr>
<tr>
<td>nS-VL PPT (N)</td>
<td>-0.096</td>
<td>-0.183</td>
<td>0.266</td>
</tr>
<tr>
<td>S-VM PPT (N)</td>
<td>-0.262</td>
<td>-0.260</td>
<td>0.272</td>
</tr>
<tr>
<td>nS-VM PPT (N)</td>
<td>-0.431</td>
<td>-0.495*</td>
<td>0.221</td>
</tr>
</tbody>
</table>

Where PPT is pressure pain threshold, PTD is photocell timing device, FP is force platform, $r_s$ is Spearman's correlation coefficient rho, CMJ-JH is jump height of countermovement jump, DJ-JH is jump height of depth jump, DJ-$t_c$ is contact time of depth jump, DJ-RSI is reactive strength index of depth jump, S- is standing leg, nS- is non-standing leg, VL is vastus lateralis (site of TrP in muscle), VM is vastus medialis (n=21). *Correlation is significant at the 0.05 level (2-tailed).
4.3.5 Correlations between physiological measures and jumps measures

The $r_s$ between the CMJ-JH and body mass was assessed and was found to be 0.192 and 0.232 when measured with the PTD and FP respectively (Table 15). As a group, there was a poor positive correlation between the $t_c$ of the DJ-JH and body mass for the PTDF (0.086) and FP (0.250).

There was a poor negative correlation between the BMI and $RSI$ (-0.276 to -0.288) when measured with the PTD and FP, respectively. When the group was split according to gender, the males had a moderate positive relationship with regards the $t_c$ of the DJ-JH and body mass. The correlation was 0.555 when measured with the PTD and 0.608 when measured with the FP; whereas the females demonstrated a poor positive correlation (0.037-0.083) when measured with the PTD and FP, respectively. For the BMI and $RSI$, there was no correlation in the males, but there was a moderate negative correlation between BMI and $RSI$ with regards to females: PTD (-0.770), FP (-0.547).

Table 15: The Spearman’s correlation coefficient rho between physiological measures and jumps measures

<table>
<thead>
<tr>
<th>(r_s)</th>
<th>PTD</th>
<th>FP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CMJ-JH (cm)</td>
<td>DJ-JH (cm)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>0.550**</td>
<td>0.605**</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>0.192</td>
<td>0.107</td>
</tr>
<tr>
<td>BMI (kg-m⁻²)</td>
<td>-0.190</td>
<td>-0.347</td>
</tr>
</tbody>
</table>

PTD is photocell timing device, FP is force platform, BMI is body mass index, $r_s$ is Spearman’s correlation coefficient rho, CMJ-JH is jump height of countermovement jump, DJ is jump height of depth jump, DJ- $t_c$ is contact time of depth jump, $RSI$ is reactive strength index of depth jump (n=23). *Correlation is significant at the 0.05 level (2-tailed), ** Correlation is significant at the 0.01 level (2-tailed).
4.3.6 The effects of dry needling and shockwave on the pressure pain threshold

The mean PPT for the most and second most painful TrPs in the VL and VM of both legs were collated. The PPT of the DN group was 28.25 (95% CI: 26.61 to 29.90) N at the B-line and increased to 31.68 (95% CI: 29.73 to 33.62) N at FU2. The rESWT group also demonstrated an increase in the PPT over the same period, from 22.08 (95% CI: 20.48 to 23.67) N at the B-line to 24.07 (95% CI: 22.64 to 25.00) N at FU2. The PPT for the control group went from 31.55 (95% CI: 29.86 to 33.24) N at the B-line to 32.09 (95% CI: 30.52 to 33.65) N at FU2.

A two-way ANOVA was performed for the PPT. Variables entered into the ANOVA were session time and intervention group. There was a statistical significant interaction between group versus session time (F[10, 987] = 2.715, p = 2.71∙10^{-3}, ηp² = 0.027) with an observed power of 0.969. A Tukey post hoc test for time revealed that the DN group was statistically significantly different to the rESWT group (p = 5.09∙10^{-9}) and the control group (p = 0.002) and the rESWT group was significantly different to the control group for time (p = 5.09∙10^{-9}). A Tukey post hoc test revealed that the B-line measurement was statistically significantly different to Tx1 (p = 7.46∙10^{-4}) and FU2 (p = 4.47∙10^{-4}). Tx1 was statistically different to FU1 (p = 3.28∙10^{-8}) and FU2 (p = 6.59∙10^{-14}). Tx2 was statistically significantly different to FU1 (p = 6.25∙10^{-4}) and FU2 (p = 1.34∙10^{-8}). Tx3 was statistically significantly different to FU1 (p = 0.016) and FU2 when accounting for group (p = 2.32∙10^{-6}). FU1 was not significantly different to FU2 for when accounting for group (p = 0.297). The PPT for the DN group significantly increased from B-line to FU2 by 12.92%; compared to the rESWT group which significantly increased by 13.26%; and the control group which significantly increased by 4.83%. The PPT between the B-line and Tx3 decreased by 6.49% for the DN group; whereas, the PPT increased in the rESWT group by 2.82%, and by 0.18% in the control group. Between Tx3 and FU1 PPT in the DN group significantly increased by 20.73%, the rESWT group significantly increased by 6.86%, and the control group significantly increased by 0.70%. The PPT for the DN group significantly increased from Tx3 to FU2 by 29.58%; compared to the rESWT group which significantly increased by 10.16%; and the control group which
significantly increased by 4.20%. Percent changes are presented in Table 16. The mean PPTs of the most painful TrPs in each muscle are illustrated in Figure 13 (p. 154) and Figure 14 (p. 155).

Table 16: Pressure pain threshold percent change for key times

<table>
<thead>
<tr>
<th></th>
<th>DN (n=7)</th>
<th>rESWT (n=7)</th>
<th>Control (n=7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>B-line - FU₂</td>
<td>12.92%*</td>
<td>13.27%*</td>
<td>4.38%</td>
</tr>
<tr>
<td>B-line - TX₃</td>
<td>-6.46%</td>
<td>2.82%</td>
<td>0.18%</td>
</tr>
<tr>
<td>TX₃ - FU₁</td>
<td>20.73%*</td>
<td>6.86%</td>
<td>0.70%</td>
</tr>
<tr>
<td>TX₃ - FU₂</td>
<td>29.58%*</td>
<td>10.16%*</td>
<td>4.20%</td>
</tr>
</tbody>
</table>

Note: *is a significantly different to control for time in the analysis of variance (P < 0.01). Where B-line is Baseline, TXₙ is Treatment, FUₙ is Follow-up.
Figure 13: Mean PPT of the most painful TrP in the vastus lateralis of the (a) standing leg (b) non-standing leg. Where PPT is pain pressure threshold DN is dry needling, rESWT is radial extracorporeal shockwave therapy, B-line is Baseline, Tx[n] is Treatment, FU[n] is Follow-up, error bars are standard error, * is a difference between sessions (p<0.05) for both legs, ** is a difference between sessions (p<0.01) for both legs, † is a difference between DN and control (p<0.05), †† is a difference between DN and rESWT (p<0.01), ‡ is a difference between rESWT and control (p<0.01).
Figure 14: Mean PPT of the most painful TrP in the vastus medialis of the (a) standing leg (b) non-standing leg. Where PPT is pain pressure threshold DN is dry needling, rESWT is radial extracorporeal shockwave therapy, B-line is Baseline, Tx is Treatment, FU is Follow-up, error bars are standard error, * is a difference between sessions (p<0.05) for both legs, ** is a difference between sessions (p<0.01) for both legs, † is a difference between DN and control (p<0.05), †† is a difference between DN and rESWT (p<0.01), ‡ is a difference between rESWT and control (p<0.01).
4.3.7 The effects of dry needling and shockwave on countermovement jump performance

The CMJ measures using the FP and PTD were collated (n = 42). The mean CMJ-JH for the DN group increased from 27.43 (95% CI: 23.24 to 31.61) cm at the B-line to 28.07 (95% CI: 23.24 to 32.90) cm at FU; whereas the rESWT group increased from 23.55 (95% CI: 20.72 to 26.37) cm at the B-line to 24.71 (95% CI: 21.11 to 28.25) cm at FU. For the DN group, the mean JH of the DJ at the B-line was 22.09 (95% CI: 19.87 to 24.31) cm and decreased to 20.86 (95% CI: 17.68 to 23.89) cm at FU; Whereas the rESWT group slightly increased vis à vis mean DJ-JH: 20.18 (95% CI: 17.57 to 22.80) cm at the B-line and 20.21 (95% CI: 16.78 to 23.65) cm at FU. The control group went from 26.86 (95% CI: 22.46 to 31.25) cm at the B-line to 26.79 (95% CI: 22.01 to 31.56) cm at FU. Mean CMJ-JH are presented in Table 17 (p. 157).
Table 17: The effects of DN, rESWT, and control on mean CMJ-JH ± SD (cm)

<table>
<thead>
<tr>
<th>Group</th>
<th>Sample (n=21)</th>
<th>B-line</th>
<th>Tx1 (%) change</th>
<th>Tx2 (%) change</th>
<th>Tx3 (%) change</th>
<th>FU1 (%) change</th>
<th>FU2 (%) change</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN</td>
<td></td>
<td>27.43 ± 7.25</td>
<td>28.54 ± 7.79 (+4.05)</td>
<td>26.38 ± 6.94 (-3.83)</td>
<td>27.23 ± 8.20 (-0.73)</td>
<td>26.79 ± 7.58 (-2.33)</td>
<td>28.07 ± 8.36 (+2.33)</td>
</tr>
<tr>
<td>rESWT</td>
<td></td>
<td>24.38 ± 4.37</td>
<td>24.43 ± 4.93 (+0.21)</td>
<td>24.57 ± 5.40 (+0.78)</td>
<td>25.58 ± 5.49 (+4.92)</td>
<td>24.64 ± 4.68 (+1.07)</td>
<td>24.71 ± 6.12 (+1.35)</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>26.86 ± 7.61</td>
<td>27.92 ± 7.46 (+3.95)</td>
<td>27.93 ± 7.33 (+3.98)</td>
<td>27.79 ± 7.07 (+3.46)</td>
<td>27.64 ± 7.14 (+2.90)</td>
<td>26.79 ± 8.28 (-0.26)</td>
</tr>
</tbody>
</table>

| Group       | Males (n=10)  | 34.67 ± 2.06 | 36.60 ± 5.59 (+5.57) | 33.40 ± 4.39 (-3.66) | 35.80 ± 2.86 (+3.26) | 34.33 ± 2.58 (-0.98) | 36.00 ± 5.66 (+3.84) |
| rESWT       |               | 25.50 ± 1.00  | 27.50 ± 3.67 (+7.84) | 27.25 ± 2.75 (+6.86) | 28.50 ± 1.91 (+11.76) | 27.50 ± 2.38 (+7.84) | 27.75 ± 1.71 (+8.82) |
| Control     |               | 32.25 ± 3.69  | 32.38 ± 4.44 (+0.40) | 33.13 ± 3.44 (+2.73) | 32.88 ± 2.30 (+1.97) | 32.50 ± 3.34 (+0.78) | 31.75 ± 5.80 (-1.55) |

| Group       | Female (n=11) | 22.00 ± 4.00  | 23.50 ± 3.25 (+6.82) | 22.00 ± 3.78 (0.00) | 21.88 ± 5.30 (-0.55) | 21.13 ± 4.05 (-3.95) | 22.13 ± 3.56 (+0.59) |
| rESWT       |               | 23.89 ± 5.23  | 23.20 ± 4.96 (-2.89) | 23.50 ± 5.90 (-1.63) | 24.13 ± 6.12 (+1.00) | 23.50 ± 4.97 (-1.63) | 23.50 ± 6.88 (-1.63) |
| Control     |               | 19.67 ± 4.80  | 20.80 ± 5.40 (+5.74) | 21.00 ± 4.73 (+6.76) | 21.00 ± 5.10 (+6.76) | 21.17 ± 5.38 (+7.63) | 20.17 ± 6.24 (+2.54) |

Note: percent change from B-line. Where DN is dry needling, rESWT is radial extracorporeal shockwave, CMJ-JH is countermovement jump height, SD is standard deviation, B-line is Baseline, Tx(n) is Treatment, FU(n) is Follow-up.
A three-way ANOCVA was conducted to determine the significant statistical difference between 1) session time, 2) group and gender controlling for the PPT (PPT-PCA = 9.71·10^{-3}) on mean CMJ-JH. There was a significant statistical interaction between the groups and treatment session for the CMJ controlling for PPT (F[12, 185] = 2.382, p = 0.007, η_p^2 = 0.134) with an observed power of 0.960 as illustrated in Figure 15. A pairwise comparison analysis was conducted between groups; the DN group was significantly different to the rESWT group (p = 7.84·10^{-6}), the rESWT group was significantly different to the control group (p = 1.02·10^{-6}). A pairwise comparison analysis was conducted between sessions; FU_1 significantly increased to FU_2 (p = 0.030).

Figure 15: The effects of DN and rESWT on the adjusted mean of the CMJ-JH when controlling for the PPT (PPT-PCA= 9.71·10^{-3}), error bars are standard error. Where DN is dry needling and rESWT is radial corporeal shockwave therapy, CMJ-JH is jump height of the countermovement jump, B-line is Baseline, Tx(n) is Treatment, FU(n) is Follow-up, PPT is pressure pain threshold, PPT-PCA is pressure pain threshold principal components analysis. * is a difference between sessions (p<0.05), † is DN difference to control (p < 0.05), ‡ is rESWT is difference to DN (p < 0.05), § is control is different to rESWT (p < 0.05).
4.3.8 The effects of dry needling and shockwave on depth jump performance

The DJ-JH measured with the FP and PTD were collated (n=42). The mean DJ-JH for the DN group decreased from 22.71 (95% CI: 20.57 to 24.86) cm at the B-line to 20.86 (95% CI: 17.83 to 23.86) cm at FU2; The mean DJ-JH for the rESWT group also decreased, from 21.38 (95% CI: 18.60 to 24.17) cm at the B-line to 20.21 (95% CI: 16.78 to 28.65) cm at FU2. The control group went from 23.14 (95%CI: 19.13 to 27.15) cm at the B-line to 20.57 (95% CI: 17.51 to 23.68) cm at FU2. Mean DJ-JH are presented in Table 18.

Table 18: The effects of DN, rESWT, and control on mean DJ-JH ± SD (cm)

<table>
<thead>
<tr>
<th></th>
<th>B-line</th>
<th>TX1 (% change*)</th>
<th>TX2 (% change*)</th>
<th>TX3 (% change*)</th>
<th>FU1 (% change*)</th>
<th>FU2 (% change*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN</td>
<td>22.71 ± 3.71</td>
<td>22.77 ± 4.85 (+0.26)</td>
<td>20.23 ± 4.55 (-10.92)</td>
<td>19.14 ± 3.48 (-15.72)</td>
<td>20.14 ± 4.26 (-11.32)</td>
<td>20.86 ± 5.25 (-8.15)</td>
</tr>
<tr>
<td>rESWT</td>
<td>21.38 ± 4.61</td>
<td>19.64 ± 8.07 (-8.14)</td>
<td>20.50 ± 6.53 (-4.12)</td>
<td>18.75 ± 8.35 (-12.30)</td>
<td>18.92 ± 5.91 (-11.51)</td>
<td>20.21 ± 5.91 (-5.47)</td>
</tr>
<tr>
<td>Control</td>
<td>23.14 ± 6.95</td>
<td>22.92 ± 7.90 (-0.94)</td>
<td>21.43 ± 6.69 (-7.39)</td>
<td>21.21 ± 6.18 (-8.34)</td>
<td>20.86 ± 5.33 (-9.87)</td>
<td>20.57 ± 5.30 (-11.11)</td>
</tr>
</tbody>
</table>

Note* percent change from B-line. Where DN is dry needling, rESWT is radial extracorporeal shockwave, DJ-JH is depth jump height, SD is standard deviation, B-line is Baseline, TX(n) is Treatment, FU(n) is Follow-up.
A two-way ANOVA was conducted to determine the significant statistical difference between session time X group controlling for the PPT (PPT-PCA = 8.15·10⁻²) and BMI (BMI PCA = 70.05) on DJ-JH. There was a significant statistical difference between the groups and treatment session for the CMJ controlling for PPT (F[17, 245] = 4.46, p = 1.08·10⁻³, η² = 0.305) with an observed power of 1.00 as illustrated in Figure 16. A pairwise comparison analysis was conducted between groups; the DN group was significantly different to the rESWT group (p = 0.009), the rESWT group was significantly different to the control group (p = 6.36·10⁻⁴). A pairwise comparison analysis was conducted between sessions; Tx₃ was significantly decreased from B-line (p = 0.019).

Figure 16: The effects of DN and rESWT on the adjusted mean of DJ-JH when controlling for the PPT (PPT-PCA = 8.15·10⁻³) and BMI (BMI-PCA = 70.05), error bars are standard error. Where DN is dry needling and rESWT is radial corporeal shockwave therapy, DJ-JH is jump height of the depth jump, B-line is Baseline, Txᵢ is Treatment, FUᵢ is Follow-up, PPT is pressure pain threshold PPT-PCA is pressure pain threshold principal components analysis, BMI is body mass index, BMI-PCA is body mass index principal components analysis. * is a difference between sessions (p < 0.05), † is DN difference to control (p < 0.05), ‡ is rESWT is difference to DN (p < 0.05), § is control is different to rESWT (p < 0.05).
4.3.9 The effects of dry needling and shockwave on depth jump contact time

The DJ-\( t_c \) measured with the FP and PTD were collated (n=42). The mean DJ-\( t_c \) for the DN group improved from 0.206 (95% CI: 0.196 to 0.216) seconds at the B-line to 0.195 (95% CI: 0.182 to 0.209) seconds at FU\(_2\); whereas the rESWT group regressed from 0.196 (95% CI: 0.193 to 0.210) seconds at the B-line to 0.198 (95% CI: 0.183 to 0.213) seconds at FU\(_2\). The control group went from 0.205 (95%CI: 0.193 to 0.217) seconds at the B-line to 0.204 (95% CI: 0.193 to 0.213) seconds at FU\(_2\). All mean \( t_c \) are presented in Table 19.

<table>
<thead>
<tr>
<th></th>
<th>B-line</th>
<th>Tx(_1)</th>
<th>Tx(_2)</th>
<th>Tx(_3)</th>
<th>FU(_1)</th>
<th>FU(_2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN</td>
<td>0.206± 0.016</td>
<td>0.202± 0.023</td>
<td>0.203± 0.015</td>
<td>0.204± 0.020</td>
<td>0.194± 0.019</td>
<td>0.195± 0.023</td>
</tr>
<tr>
<td>rESWT</td>
<td>0.196± 0.020</td>
<td>0.199± 0.022</td>
<td>0.187± 0.015</td>
<td>0.186± 0.023</td>
<td>0.194± 0.019</td>
<td>0.198± 0.026</td>
</tr>
<tr>
<td>Control</td>
<td>0.205± 0.021</td>
<td>0.198± 0.027</td>
<td>0.201± 0.031</td>
<td>0.194± 0.023</td>
<td>0.199± 0.024</td>
<td>0.204± 0.019</td>
</tr>
</tbody>
</table>

Where is dry needling, rESWT is radial extracorporeal shockwave, DJ-\( t_c \) is depth jump contact time, B-line is Baseline, Tx(n) is Treatment, FU(n) is Follow-up.

A two-way ANOVA was conducted to determine if there was a significant statistical difference between session time X group controlling for the PPT (PPT-PCA = 2.92·10\(^{-2}\)) and BMI (BMI PCA = 69.59) on DJ-\( t_c \). There was no significant statistical difference between the groups and treatment session for the DJ-\( t_c \) controlling for PPT and BMI (F[10, 240] = 1.10, \( p = 0.360, \eta_{p}^2 = 0.048 \) with an observed power of 0.573.
4.3.10 The effects of dry needling and shockwave on depth jump reactive strength index

The DJ-RSI measured with the FP and PTD were collated (n=42). The mean DJ-RSI for the DN group increased from 1.07 (95% CI: 0.935 to 1.20) m·s$^{-1}$ at the B-line to 1.08 (95% CI: 0.934 to 1.23) m·s$^{-1}$ at the FU$_2$; whereas the rESWT group decreased from 1.03 (95% CI: 0.854 to 1.202) m·s$^{-1}$ at the B-line to 0.939 (95% CI: 0.720 to 1.16) m·s$^{-1}$ at the FU$_2$. The control group went from 1.14 (95%CI: 0.911 to 1.373) m·s$^{-1}$ at the B-line to 1.03 (95% CI: 0.835 to 1.23) m·s$^{-1}$ at the FU$_2$. All DJ-RSI data is presented in Table 20.

<table>
<thead>
<tr>
<th></th>
<th>B-line</th>
<th>Tx$_1$</th>
<th>Tx$_2$</th>
<th>Tx$_3$</th>
<th>FU$_1$</th>
<th>FU$_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN</td>
<td>1.07 ±</td>
<td>1.15 ±</td>
<td>1.00 ±</td>
<td>0.946 ±</td>
<td>1.06 ±</td>
<td>1.08 ±</td>
</tr>
<tr>
<td></td>
<td>0.200</td>
<td>0.299</td>
<td>0.198</td>
<td>0.153</td>
<td>0.233</td>
<td>0.256</td>
</tr>
<tr>
<td>rESWT</td>
<td>1.03 ±</td>
<td>1.12 ±</td>
<td>1.14 ±</td>
<td>1.06 ±</td>
<td>0.988 ±</td>
<td>0.939 ±</td>
</tr>
<tr>
<td></td>
<td>0.259</td>
<td>2.285</td>
<td>0.328</td>
<td>0.350</td>
<td>0.298</td>
<td>0.378</td>
</tr>
<tr>
<td>Control</td>
<td>1.14 ±</td>
<td>1.24 ±</td>
<td>1.07 ±</td>
<td>1.11 ±</td>
<td>1.07 ±</td>
<td>1.03 ±</td>
</tr>
<tr>
<td></td>
<td>0.400</td>
<td>0.587</td>
<td>0.353</td>
<td>0.345</td>
<td>0.332</td>
<td>0.343</td>
</tr>
</tbody>
</table>

Where DN is dry needling, rESWT is radial extracorporeal shockwave, DJ-RSI is depth jump reactive strength index, Baseline, Tx$_{(n)}$ is Treatment, FU$_{(n)}$ is Follow-up.

A two-way ANOCVA was conducted to determine if there was a significant statistical difference between session time X group controlling for the PPT (PPT-PCA = 4.14·10$^{-2}$) and BMI (BMI PCA = 69.63) on DJ-RSI. There was no significant statistical difference between the groups and treatment session for the DJ-RSI controlling for PPT and BMI (F[10, 236] = 1.02, $p = 0.429$, $\eta_p^2 = 0.043$) with an observed power of 0.530.
4.4 Discussion

4.4.1 Jump reliability

Null hypothesis 1 states “The rater will not be reliable at measuring the CMJ-JH, DJ-JH, DJ-\(t_c\) and DJ-\(RSI\).” All jump metrics demonstrated high reliability when measured with the \(ICC_{(3:1)}\) and \(SEM\). These findings are consistent with those of Mizuguchi et al. (2015) who reported reliability when measuring the CMJ of students who participated in regular physical activity three to seven days a week and had no injuries or medical conditions. These findings are in line with other studies investigating the DJ-JH and its constituent parts (Beattie and Flanagan, 2015; Markwick et al., 2015; Byrne et al., 2017). Therefore, Null hypothesis 1 can be rejected.

4.4.2 Correlation

Null hypothesis 2 states “There will be no correlation between physiological measures and jump outcomes.” There were some moderate \(r\)s between PPT and jump performance. While this does not suggest a causation effect, it should be considered a factor in measuring jump performance. The \(r\)s for height and CMJ-JH were none to moderate. Likewise for height and components of the DJ (DJ-JH and DJ-\(RSI\)). There were a moderate negative \(r\)s for BMI and DJ-JH. Furthermore, there were a moderate negative \(r\)s for BMI and DJ-\(RSI\). When the group was split according to gender, there was also a moderate to strong correlation between body mass and DJ-\(t_c\) in the males; and a moderate to strong correlation between BMI and DJ-\(RSI\) in the females. The discrepancies between \(RSI\) and BMI vis à vis gender may be due to lower lean body mass in females relative to BMI (Janssen et al., 2014). A jumper’s ground reaction force and vertical displacement and therefore \(JH\) are related to the mass of the jumper (Linthorne, 2001). An increase in BMI for a female may not equate to an increase in lean body mass; whereas a male can readily increase lean body mass more than a female due to higher levels of testosterone (Shariat et al., 2015). An increase in lean body mass could improve \(JH\) and \(RSI\) as it contributes to power output and performance (Olds, 2001), but may skew the BMI. The adverse effect of poor body composition could affect an
athlete’s ability to translate ground reaction force into upward propulsion, rendering the benefits of SCC redundant. Therefore, Null hypothesis 2 can be rejected.

### 4.4.3 Outcome for pressure pain threshold

Null hypothesis 3 was “Using DN or rESWT to treat TrPs in the VL and VM will not affect the PPT”. There was a group X time interaction for PPT ($p = 0.003$). Thus, suggesting that changes in the session are as a result of the interventions rather than chance. The DN group improved TrP sensitivity by 12.92% between B-line and FU$_2$; compared to the rESWT group who reported an improvement of 13.26%.

There was a significant improvement ($p = 0.002$) in the PPT for the DN group compared to the control group, from 27.60 N at the B-line to 31.17 N at FU$_2$, equating to 12.92%, which was greater than the highest SEM (3.21 N) reported in the PPT reliability study (p. 110). The findings of the present study are similar to the results of Ziaeifar et al. (2014) who reported an improvement in the PPT of active TrPs in the upper trapezius muscle after one session of DN.

The rESWT group also demonstrated a significant improvement ($p = 5.09 \times 10^{-9}$) in the PPT compared to the control group, from B-line (21.08 N) to FU$_2$ (23.88 N) by 13.26% which was greater than the mean SEM (1.97 N) reported in the PPT reliability study. The improvements of the present study, in relation to rESWT, are similar to the findings of Cho et al. (2012) who reported improvements in the PPT of active TrPs in the upper trapezius muscle when treating TrPs with rESWT. The findings, however, of the present study are not as substantial as a previous study that reported an increase of 25.42 N with one treatment session of 1,000 pulses of 0.12 mJ·mm$^{-2}$ of rESWT (Jeon et al., 2012). This less substantial improvement in PPT may be due to the fact that the present study was treating latent TrPs.

The difference in PPT of the most painful TrP in VM of the standing leg and the VM of non-standing leg was lowered by 1.08 N, suggesting that the non-standing limb may respond better to treatment as it is not subjected to a repetitive load, compared to the standing limb, which is a predisposing factor for the formation of TrPs (Dommerholt, 2011).
There was also a significant decrease in the PPT ($p = 7.46 \cdot 10^{-4}$) between B-line and before Tx$_1$ of 2.29 N. The reduction in the pre-test and post-test PPT is similar to the findings of the PPT in the VM and VL reported in the reliability study. However, they are in contrast to the pre-test and post-test PPT of bilateral erector spinae muscles at the spinous processes of lumbar levels 1, 3 and 5, respectively (Koo et al., 2013). It should be noted that in the study by Koo et al. (2013) the PPT was measured at the spinous processes, not at TrPs. The findings of the present study are comparable to the results of Jones et al. (2007) who reported reducing PPT in the muscle of the upper limb when measured over three consecutive days. Thus suggesting that using an algometer may have a sensitising effect in subjects with latent TrPs.

Between Tx$_2$ and FU$_1$, there was a statistically significant ($p = 6.25 \cdot 10^{-4}$) increase of sensitivity in the DN group with a reduction in the PPT of 4.89 N. Suggesting that there was some post-needling soreness. These PPTs are similar to the finding of Martín-Pintado-Zugasti et al. (2014, 2015, 2016) who treated latent TrPs in the trapezius. The DN group did demonstrate a greater level of improvement from Tx$_3$ to FU$_2$ (7.53 N) compared to the rESWT group.

There was a steady, gradual decrease in the sensitivity of the PPT in subjects in the rESWT group (1.99 N). This gradual increase of pressure would suggest that the rESWT does not produce post-treatment soreness and may be used on patients who have high levels of sensitivity in TrPs. Post-needling soreness is an area of research that is still being investigated, but the literature would suggest that it can last up to 72 hours in healthy subjects and longer in patients with active TrPs (Martín-Pintado-Zugasti et al., 2014, 2015, 2016). It is unknown if the post-needling soreness affects function and further investigation is needed in this area. Therefore, Null hypothesis 3 can be rejected.

4.4.4 Outcomes for countermovement jump

Null Hypothesis 4 stated, “Treating TrPs in the VL and VM with DN or rESWT will not affect CMJ jump performance”. There was a significant statistical improvement ($p = 0.007$) in the adjusted mean CMJ-JH when factoring for the PPT at FU$_2$ for the DN group (1.40 cm) and
the rESWT group (0.95 cm) in comparison to the B-line measures. There was a 2.63% improvement in the actual mean of the CMJ-JH one week after treatment for the DN group compared to the 2.05% improvement in the rESWT group.

These results are analogous to the findings of Tsoukos et al. (2016), who reported a postactivation potentiation effects on CMJ performance by 3.8%, 12 minutes after completing 3 sets of 3 seconds maximum voluntary isometric contractions with a one minute rest between each set squat with knees flexed at 140°. Ong et al. (2016) also reported an increase in CMJ-JH after eccentric contractions. However, increase power production derived from postactivation potentiation is short lived, in the order of three to seven minutes, with a reduction in jump performance seen with rest periods of more than ten minutes (Wilson et al., 2013).

The improvement in the CMJ-JH may be due to the increase in muscle length (Babault et al., 2003) from the possible dissipation of the taut bands. The improvement may also be as a result of post-activation potentiation and its inverse relationship with fatigue (Tillin and Bishop, 2009). The depletion of adenosine triphosphate associated with TrPs leads to increased acidity and calcium accumulation (Bron and Dommerholt, 2012), and are often used as markers for fatigue (Siegler et al., 2015). The short-term nature of the improvements seen in the present study suggests that treating TrPs does have some post-activation potentiation element as the time frame is too short for any hypertrophic change which can take nine weeks to manifest (Talpey et al., 2016). Given that there was no improvement in the control group it could be assumed that there was no training effect incurred. Therefore, Null hypothesis 4 can be rejected as there was a group X time interaction for CMJ-JH when factoring for PPT.

4.4.4.1 The effect of gender on countermovement jump height

Null hypothesis 5 stats “Gender will have an effect on jump performance.” The reduction in the CMJ-JH for the females in the rESWT group may be due to the higher proportion of females in the rESWT group (62.5%) compared to the DN group (57.1%). Other possible explanations are that treating TrPS in the VL and VM may improve muscle function but could
also negatively affect hip and knee stability in females (Hewett et al., 2005; McCurdy et al., 2014). This lack of neuromuscular control has been attributed to the valgus angle of the knee extenuated by the Q angle and quadriceps and hamstring activation patterns in females (Malfait et al., 2016). These physiological and neurological deficits may be predisposing factors in knee pathologies predominantly in female such as patellofemoral pain syndrome (Oakes et al., 2009; Hains and Hains, 2010; Roach et al., 2013; Espí-López et al., 2017), knee osteoarthritis (Yentür et al., 2003; Itoh et al., 2008b; Henry et al., 2012; Mayoral et al., 2013; Alburquerque-García et al., 2015), and postsurgical anterior cruciate ligament pain (Ortega-Cebrian et al., 2016). Therefore, future studies should consider assessing joint kinematics, especially in the hip and knee, with the use of motion capture. Therefore, Null hypothesis 5 can be rejected.

### 4.4.5 Outcomes for depth jump

Hypothesis 6 stated, “Treating TrPs in the VL and VM with DN or rESWT will not affect the jump performance of the DJ”. There was a statistically significant ($p = 1.08 \cdot 10^{-3}$) decrease for the adjusted mean DJ-JH from the B-line to T$\chi_3$ when factoring for the PPT and BMI in the DN group (4.10 cm) and the rESWT group (3.00 cm). There was a net decrease in the real DJ-JH in the DN group (15.72%) and rESWT group (12.30%). The control group demonstrated a steady, gradual decline of DJ-JH by 2.57 cm (11.11%) over all of the sessions, possibly due to poor motivation because of the repetitive nature of the six sessions (Brewin and Bradley, 1989).

The degradation in the DJ may be as a result of a loss of muscle stiffness in the quadriceps. The transfer of load during running and jumping may result in muscle stiffness further up the kinetic chain (Butler et al., 2003). Increased muscle stiffness may improve the stretch-shortening cycle (Pruyn et al., 2014; Wang et al., 2015) as well as knee biomechanics (Wang et al., 2015). However, excess stiffness is believed to contribute to injury (Butler et al., 2003; Wang et al., 2015, 2017). Muscle stiffness has an intrinsic and reflexive component. The essential element is potentially due to the deformation of the actin-myosin filament cross-bridges, and the reflexive element is integrated into the central nervous system (Butler et
The increased muscle stiffness in males is possibly due to increased muscle mass; whereas the poor biomechanics in the knees, especially in females, may attributable to reduced muscle stiffness (Wang et al., 2015). Recent studies into the muscle stiffness in the VL suggest that females had lower relaxed and contracted muscle stiffness (270 N·m\(^{-1}\) and 332 N·m\(^{-1}\), respectively) in the VL than their male counterparts (364.4 N·m\(^{-1}\) and 495 N·m\(^{-1}\), respectively). Females also have lower musculoarticular stiffness across the knee in isometric contraction in extension supporting 30% of maximum voluntary isometric contractions (Wang et al., 2015). Wang et al. (2016) also reported that after fatigue-inducing exercise there was a reduction in normalised peak torque by 8.66% in males (2.77 to 2.53 N·m·kg\(^{-2}\)) and 6.22% females (2.41 to 2.26 N·m·kg\(^{-2}\)); as well as musculoarticular stiffness, as outlined in Wang et al. (2015), by 7.19% (1450 to 1345 N·m\(^{-1}\)) for the males and by 7.32% (1027 to 952 N·m\(^{-1}\)) for the females. Furthermore, there was an improvement in relaxed and contracted muscle stiffness by 22.59% (364.43 to 446.75 N·m\(^{-1}\)) and 10.36% (495.07 to 546.37 N·m\(^{-1}\)) in males, respectively and 13.37% (270.27 to 307.39 N·m\(^{-1}\)) and 5.08% (332.34 to 349.21 N·m\(^{-1}\)) in females, respectively.

The depth jump is predominantly propelled by the plantar-flexor muscles in the triceps surae complex which may be further enhanced with increased muscle stiffness. It should be noted that while there are no links to muscle stiffness and the formation of TrPs, at present, however, there are some similarities; extended actin-myosin coupling, the involvement of the central nervous system, an intrinsic relationship to fatigue, and force development especially in females (Wang et al., 2015). TrP formation may be due to a pathological maladaptation to reduced muscle stiffness. The possible relationship of muscle stiffness and the formation of TrPs should be investigated in future studies. The quadriceps also play a role in the bounce DJ (Byrne et al., 2014). During the DJ, rectus femoris activation, measured using surface EMG, is four time that of the biceps femoris during the landing phase of the DJ and twice as much as the biceps femoris during the takeoff phase (Peng et al., 2011). While the activation of the rectus femoris significantly increased when dropping from 20 cm and 30 cm compared to dropping from 60 cm (p < 0.05). Whereas, Häkkinen et al. (1986) report no change in VL and VM surface EMG in elite weight lifters performing DJ from 20
cm through to 100 cm in 20 cm increments. Furthermore, Aboodarda et al. (2014) reported increased activation of the rectus femoris, VL, VM of the quadriceps and the soleus, measured using surface EMG, prior to landing in subjects performing a loaded DJ utilising elastic bands. Therefore, Null hypothesis 6 can be rejected as there was a group X time interaction for DJ-JH when factoring for PPT and BMI. However, the effect of DN and rESWT on DJ-JH was not positive.

4.4.6 Limitations

The major limitations of the present study are that the only muscles treated were the VL and VM and that treatment was restricted to just the two most painful TrPs in each of the respective muscles. Jumping involves many muscles around the hip, knee and ankle called the kinetic chain (Decker et al., 2003). Extension of the hip (gluteus maximus), extension of the knee (VL, VM, rectus femoris and vastus intermediates) and plantar flexion of the ankle (gastrocnemius, soleus, plantarius) are critical for optimum jumping, running and change of direction (Archer, 2016; Lorenz, 2016).

Devereux (2016) reported delayed improvement when treating the rectus femoris and gastrocnemius compared to just the rectus femoris or gastrocnemius. It should stand that treating all of the sensitive loci within a muscle should improve muscle function (Tsai et al., 2010). Conditions such as tension-type headaches (Fernández-de-Las-Peñas et al., 2006), myofascial pain syndrome (Tekin et al., 2013) and knee osteoarthritis (Itoh et al., 2008b) have required the treatment of multiple TrPs in multiple muscles. Treating multiple TrPs may also benefit subjects who may have satellite TrPs (Hsieh et al., 2007). The propulsion of the DJ is primarily generated in the triceps surae complex (Byrne et al., 2014). The biomechanics of the triceps surae complex is an efficient mode of propulsion during jumping (Prilutsky and Zatsiorsky, 1994; Biewener, 2016; Hirayama et al., 2017). The biomechanics of the muscle-tendon unit during DJs appears to improve with plyometric training (Hirayama et al., 2017). Therefore, it would be prudent to assess the effects of DN and rESWT to TrPs in the whole kinetic chain.
4.4.6.1 Effects of dry needling on muscle activation

Latent TrPs are believed to alter activation patterns in the shoulder complex (Lucas et al., 2010; Ge et al., 2014; Sergienko and Kalichman, 2015). The quadriceps contribute towards the stabilisation of the knee (Hains and Hains, 2010). There is an emerging body of evidence suggesting that the RP from TrPs in the VL, VM and other surrounding muscles are at least a secondary source of the pain associated with patellofemoral pain syndrome (Oakes et al., 2009; Hains and Hains, 2010; Roach et al., 2013; Espí-López et al., 2017), knee osteoarthritis (Yentür et al., 2003; Itoh et al., 2008b; Henry et al., 2012; Mayoral et al., 2013; Alburquerque-García et al., 2015), and postsurgical anterior cruciate ligament pain (Ortega-Cebrian et al., 2016). Pathologies such as patellofemoral pain syndrome and knee osteoarthritis and post anterior cruciate ligament reconstruction pain are predominantly due to poor biomechanics (Crossley, 2014; Culvenor et al., 2014) especially in unilateral activities (Herrington, 2014). Addressing the soft tissue may improve knee kinematics (Barton et al., 2015). The timing of the VL and VM has been associated with patellofemoral pain syndrome (Lankhorst et al., 2013). The delayed activation of the VM in patellofemoral pain syndrome patients during sit-to-stand tasks may cause fatigue in the VL (Cavazzuti et al., 2010). McClinton et al. (2007) reported on the activation magnitude ratio for the VM and VL in subjects with patellofemoral pain syndrome and asymptomatic subjects. The VL being more active, during step-up tasks were 85-96% less than healthy subjects, but the duration ratio of the VM and VL was 8-12% greater than healthy subjects with the VM being active for longer. Extended activation in the VM and VL may cause fatigue and perhaps TrPs. Given that the referral pattern of the VL and VM are to the knee (Travell and Simons, 1992) links between TrPs and patellofemoral pain syndrome, knee osteoarthritis and post anterior cruciate ligament reconstruction pain is possible. There is a need to measure the activation of the VL and VM with EMG in more functional lower limb tasks such as the depth jump (Tsai et al., 2016) to understand the effects of treating TrPs in the VL and VM.

4.4.6.2 Time frame of jump performance

In the present study, there was an upward trend showing in the CMJ-JH and DJ-JH at the two follow-up sessions; this was also replicated in the PPT of the most painful TrP in both
the VL and VM of the non-standing leg and the VM of the standing leg. A previous study in the effects of DN multiple muscles in the lower limb, including the quadriceps, in patients with knee osteoarthritis, reported an improvement in WOMAC functional scale in patients with knee osteoarthritis ten weeks after four once a week treatment sessions. There was a marked improvement in strength one week after patients with postoperative shoulder pain received physiotherapy and one session of DN compared to patients who just received physiotherapy (Arias-Buría et al., 2015). The present investigation is the first study to assess the effects of rESWT in TrPs vis à vis strength or performance. A longer follow-up period may identify the time course of the effects of DN and rESWT on TrPs with regards performance and strength.

4.4.6.3 The effects of a lack of motivation

There was a reduction in the DJ-JH in the control group possibly due to poor motivation because of the repetitive nature of the six sessions (Brewin and Bradley, 1989). Subjects in the control group were not blinded and therefore may have had poor motivation because of the repetitive character of the trial. Using a placebo, such as a placebo rESWT should keep all groups blinded and prevent poor motivation over multiple session of a randomised control trial (Gupta and Verma, 2013). Placebo or sham non-penetrating needles have been used in some investigations using DN (Tough et al., 2009) including the gastrocnemius and soleus (Cotchett et al., 2014). However, Chae et al. (2013) reported that even the tactile stimulation of a blunt non-penetrating needle could affect the same areas of the brain, such as the thalamus and insula, as an inserted needle, but to a lesser extent.

Placebo rESWT had been used in studies where rESWT has been used to treat the gastrocnemius and soleus muscles of children with cerebral palsy (Amelio and Manganotti, 2010) and adults with cerebral palsy (Vidal et al., 2011). The placebo rESWT in the study by Amelio and Manganotti (2010) seemed to serve its purpose, as there was no improvement seen in the passive range of motion, pedobarometric evaluation and modified Ashworth scale in the gastrocnemius and soleus. However, no study to date has used placebo rESWT in a randomised control trial to investigate the effects of rESWT on TrPs. Future studies should consider using a placebo device to negate any effects of low motivation from
multiple trials. Placebo rESWT appears not to have a positive effect compared to placebo DN. Placebo rESWT, therefore, may be a superior option.

A final limitation of the present study is that the lean body mass was not measured. The BMI is a popular method of measuring body composition (Mei et al., 2002) but does not account for lean body mass, therefore, the inclusion of methods to measure lean body mass such as magnetic resonance imaging (Janssen et al., 2014) or dual-energy X-ray absorptiometry (Glickman et al., 2004) should be included as a physiological measure in studies where subjects are performing tasks that are directly affected by gravity. The correlation of lean body mass and jump performance should be investigated to determine the effects of power to weight ratios in vertical jump performance.

4.4.7 Clinical application

In the present study, DN has been shown to improve in PPT in TrPs in the VL and VM as well as the CMJ-JH. In the present study, there is a temporary adverse effect on the PPT in TrPs in the VL and VM as well as the DJ-JH which can last up to three days. This negative effect may be as a result of post-needling soreness (Martín-Pintado-Zugasti et al., 2015, 2016). rESWT does not appear to have such a transient adverse effect and may be an alternative treatment for TrPs in athletes who cannot afford to reduce training load or who are preparing for a major competition.

In the present study, DN and rESWT may have a negative effect on the fast stretch-shortening cycle for up to seven days, possibly due to altering the muscle stiffness in the kinetic chain (Butler et al., 2003; Pruyn et al., 2014; Wang et al., 2015). Therefore, treating TrPs in athletes where the fast stretch-shortening cycle is a vital component of athletic performance, such as sprinting, should consider the current training load, season periodization and upcoming competition.

4.4.8 Conclusion

An investigation was conducted into the effects of DN and rESWT to latent TrPs in the VL and VM in CMJ and DJ performance. DN appears to have a positive effect on the PPT of the
VL and VM once the post-needling soreness had subsided. The rESWT group demonstrated a less substantial improvement at the final follow-up session but does not show any post-treatment soreness. Therefore the null hypotheses “using DN or rESWT to treat TrPs in the VL and VM will not affect the PPT, CMJ or DJ” can be rejected. rESWT may even have a post-activation potentiation element. Future studies should include treating multiple TrPs in multiple muscles involved in triple extension. Also, outcomes measuring muscle activation should be included. Additionally, there is a relationship between females, TrP sensitivity and jump performance which warrants further investigation. Appreciation for these relationships is need when interoperating any result where there are females involved. Furthermore, longer follow-up periods should be used to determine the time course of DN and rESWT. Therapists should consider the current training load, season periodization and upcoming competition to deliver the optimum treatment strategy for athletes with latent TrPs.
5 Conclusion

This project is the first study to examine the reliability of measuring both the location and the severity of TrPs in the leg using the ALS system and PPT. The rater was found to be reliable at measuring both the location and the severity of TrPs in the VL and VM. The implication of these finding is that the rater can use the ALS system to locate TrPs in the VL and VM accurately and measure the severity of TrPs in the VL and VM using the PPT.

The present investigation was the first to attempt to establish the correlation between physiological measures, such as height body mass and BMI; and jump performance. There was a moderate correlation between physiological measures and jump performance, especially when the group was split according to gender. Therefore, physiological measures should be considered when using jump performance as an outcome measure. The lean body mass might be the most appropriate physiological measure with respects to jump performance.

The present project is the first clinical trial that explored the effects of DN and rESWT, on TrPs in the lower limb. Once post-needling soreness subsides, DN improved jump performance; whereas rESWT more gradually improve jump performance. From the finding of the present study it can be suggested that DN and rESWT both have a positive effect on TrPs in the legs, but do so by different mechanisms. This investigation is the first study that examined the relationship between post-needling soreness and jump performance. DN reduced PPT, CMJ-JH and DJ-JH during the treatment phase and up to three days after the final treatment. There would appear to be a moderate correlation between TrP sensitivity and jump performance which would suggest that post-needling soreness in TrPs in the VL and VM effects jump performance. The present study is the first investigation to compare the effects of treating TrPs with DN or rESWT to improve jump performance over a one week period. Post-needling soreness can effect jump performance up to three days, but then jump performance improves when the post-needling soreness has alleviated, rESWT, on the other hand, may not cause as much of an improvement but shows no detrimental transient post-treatment effects. Treating TrPs in the VL and VM with DN or rESWT
improves jump performance up to one week after a three-session treatment period. The DJ-JH, factoring for PPT and BMI, decreased for the DN group and rESWT group possibly due to reduced muscle stiffness. It should be noted that the control group also showed a reduction in DJ-JH perhaps as a result of boredom or lack of motivation as the group was not blinded to group allocation.

Further investigation is needed to measure and identify the role of lean body mass in jump performance (in particular the DJ), to investigate the medium-term effects of DN and rESWT on the PPT and jump performance, to integrate a placebo treatment into clinical trial involving rESWT to improve the experimental design and to measure muscle activation patterns using EMG. Future experiments should also treat multiple muscles in the kinetic chain and treat all of the TrPs within a specific muscle.

There is a relationship between females, a higher number of TrP features in the quadriceps, higher sensitivity of TrP in the quadriceps and lower jump performance. Possibly due to biomechanics or body composition differences. These relationships should be further investigated. Appreciation for these relationships is need when interoperating any result where there are females involved. As well as if training females in a sport which are dependent on explosive athletic movements.

The use of DN to treat latent TrPs in muscles involved in jumping may improve performance, provided that there be sufficient time to allow post-needling soreness to subside before a period of high-intensity training or a competition. In that interim time, rESWT may be used as an alternative option to maintain muscle activation and prevent TrP sensitisation.
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7 Appendices
A. Acupuncture points around the knee

List of name and anatomical location of acupuncture point in relation to knee pain (Urbanski, 2007)

A.i Local points:

A.i.i Bladder meridian:
- BL37 (mid hamstring),
- BL39 (lateral popliteal fossa),
- BL40 (popliteal fossa),
- BL57 (muscular-tendon junction of medial head of gastrocnemius and Achilles’ tendon);

A.i.ii Gallbladder meridian:
- GB30, (piriformis),
- GB31 (VL),
- GB33 (VL),
- GB34 (proximal tubular fibular joint),
- GB35 (above the tip of the lateral malleolus on the posterior border of the fibula),
- GB36 (above the tip of the lateral malleolus on the anterior border of the fibula);

A.i.iii Kidney meridian:
- K10 (distal thigh between sartorius and semitendinosus);

A.i.iv Large intestines:
- LI8 (medial joint line of knee);

A.i.v Liver meridian:
- LV7 (posterior and inferior to the medial condyle of the tibia in the upper portion of the medial head of the gastrocnemius muscle),
- LV8 (above the medial end of the transverse popliteal crease, posterior to the medial epicondyle of the tibia),
- LV9 (above the medial epicondyle of the femur, between vastus medialis and sartorius muscles);

A.i.vi Spleen meridian:
- Spl9 (medial tibia),
- Spl10 (VM);

A.i.vii Stomach meridian:
- ST 31 (rectus femurs),
- ST32 (rectus femurs),
- ST34 (VL),
- ST35 (anterolateral joint line of knee),
- ST36 (lateral tibia),
- ST38 (mid portion of tibialis anterior);
A.ii  Distant points:

A.ii.i  Bladder meridian:

- BL11 (slightly lateral and to spinous process of T1),
- BL20 (transverse process of T11),
- BL21 (slightly lateral and to spinous process of T12),
- BL22 (slightly lateral and to spinous process of L1),
- BL23 slightly lateral and to spinous process of L2),
- BL25 (transverse process of L4),
- BL57 (muscular-tendon junction of medial head of gastrocnemius and Achilles’ tendon),
- BL58 (muscular-tendon junction of pennate junction of gastrocnemius and Achilles’ tendon),
- BL60 (posterior lateral malleolus),
- BL62 (inferior lateral malleolus);

A.ii.ii  Ear knee (upper crura of antihelix of the ear);

A.ii.iii  Gallbladder meridian:

- GB39 (above the lateral malleolus between the posterior border of the fibula and the tendons of peroneal longus and brevis muscles),
- GB40 (Anterior and inferior to the lateral malleolus on the lateral side of the extensor digitorum longus tendon),
- GB41 (4th intermetatarsal space),
- GB42 (Posterior to the 4th metatarsophalangeal joint between the fourth and fifth metatarsals, medial to the tendon of extensor digiti minimi);

A.ii.iv  Gouverneur vessel meridian:

- GV14 (C7),
- GV20 (anterior to crown of head);

A.ii.v  Kidney meridian:

- K3 (medial Achilles’ tendon);

A.ii.vi  Large intestines meridian:

- LI11 (with a bent elbow, the lateral end of the transverse cubital crease),
- LI3 (1st intermetacarpal space),
- LI4 (1st metacarpal web space);

A.ii.vii  Liver meridian:

- LV3 (proximal metacarpal-phalangeal joint of 2nd digit),
- LV4 (medial malleolus),
- LV11 (proximal adductor longus);

A.ii.viii  Spleen meridian:

- Spl4 (arch of foot),
- Spl5 (anterior medial malleolus),
- Spl6 (medial muscular-tendon junction of gastrocnemius/soleus and Achilles’ tendon);
A.ii.ix  Stomach meridian:
    ST6 (mastator),
    ST40 (superior and lateral to the tip of the lateral malleolus),
    ST43 (1st intermetatarsal space),
    ST44 (2nd intermetatarsal space);

A.ii.x  Triple heater meridian:
    TH5 (mid forearm extensors);

A.iii  Extra points:

    Heding/Heading (superior patella border),
    Xian/Xi Yan (both sides of patella tendon of knee),
    Shu-stream points - points on a meridian that are associated with joint pain (K3, ST43),
    Ah-shi points tender points strongly associated with trigger points (Hong, 2000).
B. Pressure pain threshold reliability ethics form

Application to the IT Carlow Research Ethics Committee for Ethical Approval of a Research Project Involving Human Participants (Individual Participation or donation of human derived material)

Please append any relevant interview schedules, consent forms, detailed research proposals etc. that are available.

Name of student submitting research proposal: Richie Walsh

Masters advisor(s): Sharon Kinsella

Medical Consultant:

Project Title: “Pilot study to determine the reliability of detecting and measuring latent trigger points in the quadriceps femoris.”

Describe the basic purposes of the research proposed.

To determine the reliability of the tester in detecting latent trigger points in the quadratus femoris muscles in healthy subjects.

Outline the design and methodology of the project.
Subjects will be recruited from the student population. Each subject will read and sign a screening and consent form in the presence of the tester.

Subjects will be included in the study based on the following requirements:

The presence of latent trigger points in the rectus femoris, vastus lateralis, vastus medialis, or vastus intermedius portions of the quadriceps femoris.

Subjects will be excluded based on the following:

Any variance in muscle size or asymmetry
Systemic diseases of the muscular or nervous system
Congenital or childhood hip disease
History of hip trauma
Recent surgery
Inflammatory joint disease
Tumors
Lower limb/ lower back injury and treatment of respective muscles in the past 6 months.

Apparatus: Plinth, non-toxic ultraviolet (UV) marker, ultraviolet light, digital camera, non-toxic semi-permanent marker, algometer, tape measure and acetate sheets

Design: An intra-class correlation coefficient design

Subjects will be required to attend two sessions.

**Session 1:** Prior to testing, once consent forms have been completed, the subject’s height, mass and age will be recorded. Subjects will be placed in supine position on the plinth and palpated for myofascial trigger points based on the criteria outlined by Travell & Simons (1999), spot tenderness, taut band, jump sign, local twitch response and referral of pain. Pain pressure of the trigger point will be recorded using an algometer. The location of the trigger point marked with a UV marker. The trigger point will be measured using the anatomical landmark system (ALS), modified from Babero et al. (2012). An intersecting line will be drawn
between two bony landmarks with a UV marker. The landmarks are the anterior superior iliac spine (ASIS), and the apex of the patella for the rectus femoris, vastus intermediate and vastus medialis, the landmarks for the vastus lateralis are the greater trochanters and the head of the lateral process of the patella. The distance along the ALS_d line from the proximal landmark will be measured. The perpendicular distance, relative to the ALS_d line from the trigger point to the ALS will be marked with a UV marker. A camera, positioned superiorly, with a UV light will be used to photograph the subject in the supine position. The photograph will be of the torso down, in order to prevent any identifiable features be photographed. The subject will be instructed to refrain from vigorous scrubbing of the thigh. Until the second session.

**Session 2:** The subject will return three days following the first session. The trigger points will be located and marked using a semi-permanent marker. The location will be measured using the ALS. A second photograph, with a UV light, will be taken. The pain pressure and magnitude of pressure will be recorded.

Acetate sheets will be placed over the photographs from session one and two respectively and the location of the bony landmarks and trigger point marked. Both acetate sheet will be superimposed and the distance between trigger points measured. An intra-class correlation coefficient (ICC) for pressure pain threshold and ALS will be conducted. Bland-Altman plots will be conducted for the ALS data.

**Describe the research procedures as they affect the research subject and any other parties involved.**

Subjects will be recruited through email and verbal invitation. Subjects will consist of 30 healthy individuals, male and female, between the ages of 18-35 years old who are willing to participate in the study. The requirements of the study will be made clear to the subject and participation will require subjects fulfilling the inclusion and exclusion criteria as outlined above in the previous section. Each subject will read and sign screening and consent in the presence of the tester. Subjects will be included in the study based upon satisfying the following criteria:

The presence of latent trigger points in the vastus medialis, and vastus lateralis of the dominant limb.

Subjects will be required to be physically active (minimum of three 1 hour exercise sessions per week). Subjects will be excluded from the study based upon the criteria stated in the previous section.
Subjects will be required to attend two sessions in accordance with the timeframes outlined above.

Session 1: The subject will be made aware of all the requirements of the study prior to the commencement of testing as outlined in the consent form. They will be fully aware they could leave the study at any time. Physiological measurements will be recorded. Subjects will be placed in supine position on the plinth with the dominant leg (the leg used to kick with) exposed from the hip to the ankle. The subject will be palpated for myofascial trigger points based on the criteria outlined by Travell and Simons (1999), these are spot tenderness, taut bad, jump sign, local twitch response and referral of pain. Pain pressure of the trigger point will be recorded using an algometer. Its location will be measured using the anatomical landmark system using a UV marker.

Session 2: The subject will return on the third day for a retesting session. The session will include measuring the pressure pain threshold with the algometer, the location of the trigger point using the ALS with a semi-permanent marker, and a photograph was taken under UV light.

What in your opinion are the ethical considerations involved in this proposal?

The subject may experience minor discomfort or pain on palpation of the myofascial trigger points. This discomfort or pain will persist throughout the duration of palpation and pain may be felt at a location away from the area of palpation consistent with pain referral. This pain or discomfort will be minimal. The subject’s leg will be exposed from the hip to the ankle for the purpose of the study. Proper screening will be used to protect the patient’s privacy and correct draping techniques will also be used.

Outline the reasons, which lead you to be satisfied that the possible benefits to be gained from the project justify any risks or discomforts involved.

It has been well established that myofascial trigger points are a ‘common cause of pain in clinical practice’ (Gerwin et al., 1997). There are many studies examining the effect of myofascial trigger points treatments. However, the effects of trigger point and performance outcomes are relatively limited to date. The aim of this study is to establish the reliability of the tester to identify trigger point in the hope to be able to treat trigger points in further studies involving dry needling and shockwave therapy.

The presence of latent myofascial trigger point may affect the efficient transfer of force from muscle to muscle, therefore affecting performance.
The information gathered from the study will be valuable to the rehabilitative sciences area and clinicians working with athletes aiming to achieve full performance potential. The risks are justified as the potential gains will far outweigh the risks.

Who are the investigators (including assistants) who will conduct the research and what are their qualifications and experience?

I, Richie Walsh, will conduct the research. I graduated from the Institute of Technology Carlow with an honours degree in Sports Rehabilitation and Athletic Therapy in 2015. I have gained two years’ experience working in a clinical environment, both in I.T Carlow and outside settings. I have worked as a Sports Rehabilitator with numerous teams in various sports. I am a member of the Athletic Rehabilitation Therapy Ireland (ARTI) and British Association of Sports Rehabilitators and Trainers (BASRaT) through which I am an insured practitioner. I will have completed a post graduate certificate in Trigger point therapy and dry needling.

Are arrangements for the provision of clinical facilities to handle emergencies necessary? If so, briefly describe the arrangements made.

I, the tester, am a trained emergency first responder and cardiac first responder. In the event of an emergency, I will immediately inform the laboratory technician and act accordingly to the seriousness of the emergency. I am aware that there is a defibrillator on site at main reception and in the Human Performance Laboratory should I need it. I have access to a telephone and the contact details for the doctor, nurse and relevant lecturing staff on campus.

Specify whether subjects will include students or others in a dependent relationship.

The population tested will be students from Institute of Technology Carlow.

Specify whether the research will include children or those with mental illness, disability or handicap. If so, please explain the necessity of using these subjects.

The research will include only adult subjects who are healthy and understand the requirements of the study fully.
Will payment be made to any research subject?

No payment will be made to the subject for the involvement in the research project.

Describe the procedures to be used in obtaining a valid consent from the subject. Please supply a copy of the information sheet provided to the individual subject.

A consent form will be signed in the presence of the tester. Any questions or queries that the subject may have will be explained and understood.

Comment on any cultural, social or gender-based characteristics of the subject which have affected the design of the project or which may affect its conduct.

It is not expected that any cultural, social or gender-based characteristics will affect the design of the study.

Give details of the measures, which will be adopted to maintain the confidentiality of the research subject.

Participants will be assigned an ID number against which all data and photographic images will be stored. Details linking the ID number and name of the participant will not be held together. Physical results will be held by the project supervisors under lock and key in an undisclosed location at Carlow I.T. A digital copy will also be held by the project supervisors protected by a password on the computer system at Carlow I.T. The information will be kept for a minimum of three years or indefinitely pending publication. Information will be used anonymously in the preparation of the project dissertation and other scientific reports for dissemination at scientific congress or in refereed publications. The research team will adhere to the 1997 Freedom of Information Act, (www.itcarlow.ie/resources/freedom-of-information.htm) and the data protection policy (www.itcarlow.ie/resources/data-protection.htm) and will inform participants of same.

Will the information gained be anonymised? If not, please justify.
The information will be anonymous as each subject will be allocated a case number and the results will detail this number only.

Will the intended group of research subjects, to your knowledge, be involved in other research? If so, please justify.

The intended group of research subjects will be allowed to participate in other research at their own discretion.

Date on which the project will begin
October 2015.

Please state location(s) where the project will be carried out.
The project will be carried out in the Human Performance Laboratory C149, in the Institute of Technology, Carlow.

Signed: __________________________ Date: ____________________

Project supervisor or Principal Investigator

Signed: __________________________ Date: ____________________

(Supervisor of student)

COMMENT FROM HEAD OF DEPARTMENT/GROUP/INSTITUTE/CENTRE
Signed: ________________________  Date ______________________
(Head of Department/Group/Institute/Centre)
### C. Pressure pain threshold reliability risk assessment

*Please complete this form, adding extra lines where necessary. Risk Rating is calculated according to the scoring guide at the end of this document. Submit one copy to your supervisor, retaining a copy for yourself. The form may be amended during the course of your project work. A copy should be included with your final project document.*

<table>
<thead>
<tr>
<th>Activity</th>
<th>Hazard</th>
<th>Risk Rating (Severity x Probability)</th>
<th>Control Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Severity</td>
<td>Probability</td>
</tr>
<tr>
<td>1. Using algometer</td>
<td>Physical none</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Chemical none</td>
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<td>1</td>
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<tr>
<td></td>
<td>Biological none</td>
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<td>1</td>
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<tr>
<td></td>
<td>Human/Health</td>
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<td>2</td>
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<tr>
<td></td>
<td>Bruising from</td>
<td></td>
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<tr>
<td></td>
<td>using an algometer</td>
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Risk Assessment

Risk assessment is based on estimating the likelihood of an adverse event occurring combined with the severity of the outcome of the event in terms of death or injury. The matrix below acts as a simple guide to selecting the level of risk.

<table>
<thead>
<tr>
<th>Probability</th>
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<th>2</th>
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<tr>
<td>Almost certain</td>
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<td>5</td>
<td>10</td>
<td>15</td>
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<tr>
<td>likely</td>
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<td>4</td>
<td>8</td>
<td>12</td>
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<td>unlikely</td>
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<td>4</td>
<td>6</td>
<td>8</td>
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<tr>
<td>rare</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

16-20 = high risk  12-15 = moderate risk  5-10 = low risk  1-4 = very low risk
D. Pressure pain threshold reliability informed consent

CONSENT FORM: Human Participants

RESEARCH - INFORMED CONSENT FORM

Project Title:

“Pilot study to determine the reliability of detecting and measuring latent trigger points in the quadriceps femoris.”

Introduction to the study:

Trigger points are discrete, focal, hyperirritable spots located in a taut band of skeletal muscle. A latent trigger point does not cause pain but may cause restricted movement and weakness of the muscle containing the trigger point.

I am being asked to take part in this research study.

The purpose of the study is to determine if the tester can accurately locate latent myofascial trigger points in the Quadratus Femoris muscles.

This research study will take place at I.T. Carlow and will require me to attend the Institute for two 30-40 minute sessions over a 3 day period.

This is what will happen during the research day.

The subject’s height, mass and age will be measured. The subject will be in a supine position on the plinth with the dominant leg exposed. The leg will be palpated from the front and outside of the thigh looking for the presence of trigger points. The pressure needed to elicit the referral pattern and the levels of discomfort will be recorded using an algometer. Once the trigger points have been identified and marked with a UV
marker, the locations will be measured from two lines drawn on the thigh with a UV marker. The subject will be then free to leave. The subject will need to refrain from vigorous scrubbing of the thigh during the three days between testing. The pressure pain threshold will be measured as the first session. The location of the trigger point will be marked with a semi-permanent marker and measure as the previous session using the ALS.

Sometimes there are problems associated with this type of study. These are:

1. The subject may experience minor pain or discomfort in the area, or other locations due to referral patterns, where the palpation will be applied. I understand that this pain or discomfort is commonly associated with myofascial trigger points. This pain or discomfort will last for a short period of time.

2. The tester will be marking the location of bony landmarks, an intersecting line between the landmarks, as well as the trigger points with a marker pen which may last on the skin for several days and may mark articles of clothing.

If there are any adverse effects during my visit, the subject will be monitored until the effects pass.

There may be benefits for me from this treatment. These are that the results of this research project may improve power output for people involved in sports.

My confidentiality will be maintained. IT Carlow will make reasonable efforts to protect the information about me and my part in this study, and no identifying data or images will be published. This will be achieved by assigning me an ID number against which all data will be stored. Details linking my ID number and name will not be stored with the data. The results of the study may be published and used in further studies.

If you have any questions about the study, I am free to call Richie Walsh on 0857855088. Taking part in this study is my decision. If I do agree to participate, I may withdraw at any point including during the exercise test. There will be no penalty if I withdraw before I have completed all stages of the study.
I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project entitled “Pilot study to determine the reliability of detecting and measuring latent trigger points in the quadriceps femoris.”

Signature: __________________________  Date: __________________________

Witness: __________________________  Witness: __________________________

Signature  Printed name
### E. CONSORT checklist

<table>
<thead>
<tr>
<th>Section/Topic</th>
<th>Item No</th>
<th>Checklist Item</th>
<th>Reported on page No</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Title and abstract</strong></td>
<td>1a</td>
<td>Identification as a randomised trial in the title</td>
<td>p. 139</td>
</tr>
<tr>
<td></td>
<td>1b</td>
<td>Structured summary of trial design, methods, results, and conclusions (for specific guidance see CONSORT for abstracts)</td>
<td>p. 1</td>
</tr>
<tr>
<td><strong>Introduction</strong></td>
<td>2a</td>
<td>Scientific background and explanation of rationale</td>
<td>p. 139</td>
</tr>
<tr>
<td>Background and objectives</td>
<td>2b</td>
<td>Specific objectives or hypotheses</td>
<td>p. 140</td>
</tr>
<tr>
<td><strong>Methods</strong></td>
<td>3a</td>
<td>Description of trial design (such as parallel, factorial) including allocation ratio</td>
<td>p. 140</td>
</tr>
<tr>
<td>Trial design</td>
<td>3b</td>
<td>Important changes to methods after trial commencement (such as eligibility criteria), with reasons</td>
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</tr>
<tr>
<td>Participants</td>
<td>4a</td>
<td>Eligibility criteria for participants</td>
<td>p. 141</td>
</tr>
<tr>
<td>4b</td>
<td>Settings and locations where the data were collected</td>
<td>p. 140</td>
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<tr>
<td>Interventions</td>
<td>5</td>
<td>The interventions for each group with sufficient details to allow replication, including how and when they were actually administered</td>
<td>p. 142</td>
</tr>
<tr>
<td>Outcomes</td>
<td>6a</td>
<td>Completely defined pre-specified primary and secondary outcome measures, including how and when they were assessed</td>
<td>p. 143</td>
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<tr>
<td>6b</td>
<td>Any changes to trial outcomes after the trial commenced, with reasons</td>
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<tr>
<td>Sample size</td>
<td>7a</td>
<td>How sample size was determined</td>
<td>p. 145</td>
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<td>Randomisation:</td>
<td>7b</td>
<td>When applicable, explanation of any interim analyses and stopping guidelines</td>
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<tr>
<td>Sequence generation</td>
<td>8a</td>
<td>Method used to generate the random allocation sequence</td>
<td>p. 143</td>
</tr>
<tr>
<td>Allocation concealment</td>
<td>8b</td>
<td>Type of randomisation; details of any restriction (such as blocking and block size)</td>
<td>p. 143</td>
</tr>
<tr>
<td>mechanism</td>
<td>9</td>
<td>Mechanism used to implement the random allocation sequence (such as sequentially numbered containers), describing any steps taken to conceal the sequence until interventions were assigned</td>
<td>p. 143</td>
</tr>
<tr>
<td>Implementation</td>
<td>10</td>
<td>Who generated the random allocation sequence, who enrolled participants, and who assigned participants to interventions</td>
<td></td>
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<td>---------------</td>
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<td>-------------------------------------------------------------------------------------------------------------</td>
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<tr>
<td>Blinding</td>
<td>11a</td>
<td>If done, who was blinded after assignment to interventions (for example, participants, care providers, those assessing outcomes) and how</td>
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<tr>
<td></td>
<td>11b</td>
<td>If relevant, description of the similarity of interventions</td>
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<tr>
<td>Statistical methods</td>
<td>12a</td>
<td>Statistical methods used to compare groups for primary and secondary outcomes</td>
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<tr>
<td></td>
<td>12b</td>
<td>Methods for additional analyses, such as subgroup analyses and adjusted analyses</td>
<td></td>
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<tr>
<td>Results</td>
<td>13a</td>
<td>For each group, the numbers of participants who were randomly assigned, received intended treatment, and were analysed for the primary outcome</td>
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<tr>
<td></td>
<td>13b</td>
<td>For each group, losses and exclusions after randomisation, together with reasons</td>
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</tr>
<tr>
<td>Recruitment</td>
<td>14a</td>
<td>Dates defining the periods of recruitment and follow-up</td>
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<tr>
<td></td>
<td>14b</td>
<td>Why the trial ended or was stopped</td>
<td></td>
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<tr>
<td>Baseline data</td>
<td>15</td>
<td>A table showing baseline demographic and clinical characteristics for each group</td>
<td></td>
</tr>
<tr>
<td>Numbers analysed</td>
<td>16</td>
<td>For each group, number of participants (denominator) included in each analysis and whether the analysis was by original assigned groups</td>
<td></td>
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<tr>
<td>Outcomes and estimation</td>
<td>17a</td>
<td>For each primary and secondary outcome, results for each group, and the estimated effect size and its precision (such as 95% confidence interval)</td>
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<tr>
<td></td>
<td>17b</td>
<td>For binary outcomes, presentation of both absolute and relative effect sizes is recommended</td>
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<tr>
<td>Ancillary analyses</td>
<td>18</td>
<td>Results of any other analyses performed, including subgroup analyses and adjusted analyses, distinguishing pre-specified from exploratory</td>
<td></td>
</tr>
<tr>
<td>Harms</td>
<td>19</td>
<td>All important harms or unintended effects in each group (for specific guidance see CONSORT for harms)</td>
<td></td>
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<tr>
<td>Discussion</td>
<td>20</td>
<td>Trial limitations, addressing sources of potential bias, imprecision, and, if relevant, multiplicity of analyses</td>
<td></td>
</tr>
<tr>
<td>Generalisability</td>
<td>21</td>
<td>Generalisability (external validity, applicability) of the trial findings</td>
<td></td>
</tr>
</tbody>
</table>

Walsh, R (2017)  
Apx. xix
**Interpretation**
22  Interpretation consistent with results, balancing benefits and harms, and considering other relevant evidence  

**Other information**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Registration</td>
<td>23</td>
</tr>
<tr>
<td>Protocol</td>
<td>24</td>
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<tr>
<td>Funding</td>
<td>25</td>
</tr>
</tbody>
</table>

*We strongly recommend reading this statement in conjunction with the CONSORT 2010 Explanation and Elaboration for important clarifications on all the items. If relevant, we also recommend reading CONSORT extensions for cluster randomised trials, non-inferiority and equivalence trials, non-pharmacological treatments, herbal interventions, and pragmatic trials. Additional extensions are forthcoming: for those and for up to date references relevant to this checklist, see www.consort-statement.org.*
F. The effects of dry needling and shockwave on trigger point ethics form

Application to the IT Carlow Research Ethics Committee for
Ethical Approval of a Research Project Involving Human Participants or Samples
Donated by Human Participants (e.g. tissue or blood samples)
(FORM REC2-L9(R)/ L10)

Applicants are advised to submit any supporting documentation they may feel relevant to their research proposal (e.g. sample interview schedules, consent forms, third party licenses or ethical approvals).

A. Applicant Details
A.1 Researcher Details:
Name: Richie Walsh
Email: richie.walsh@itcarlow.ie
Telephone: 085 7855088

A.2 Principal Investigator / Research Supervisor(s):
Name: Sharon Kinsella
Email: sharon.kinsella@itcarlow.ie
Telephone: 059 9175515

A.3 Additional Expertise (if applicable)
Name: 
Email: 
Telephone: 
A.4 Does this research form part of a programme of study?  

☐ Yes  ☐ No  

☑

If yes – please give details

| The study is the essential part of research masters being undertaken in the HealthCore group |

A.5 I confirm that I have read and understood the following IT Carlow Policies:

- Ethics Policy  

  ☐ Yes  ☐ No

- Ethics Procedures and Guidance notes

  ☑ Yes  ☐ No

- On completing this form

  ☑

- Data Protection Policy

  ☐ Yes  ☐ No

- Anti-Plagiarism Policy

  ☑ Yes  ☐ No
B. Research Proposal

B.1 Title of the proposed research project

The effects of Shockwave Therapy and Dry Needling Therapy of Trigger Points on Muscle Function and Performance in the Quadriceps Femoris

B.2 To what extent has this topic already been researched and written about (e.g. is there a significant body of existing published work)?

There are very few studies that describe the use of electrotherapy as a treatment modality for trigger points. There is only a hand full of recent studies that focus on trigger points in the lower extremity. The literature would suggest that the focus on trigger points, especially latent trigger points, is moving toward the effects of trigger points on muscle function.

B.3 From that, describe how this proposed research is contributing to what is known about the topic

Being able to compare the treatments options for trigger points may help to understand better the pain mechanics associated with trigger points. As well as the effects of trigger points on functional movement and performance.

B.4 Provide a brief description of research (not more than 200 words in any section)

a) The aims and objectives
The aim of the present study is to establish if electrotherapy is as effective as dry needling in the treatment of trigger points in the quadratus femoris in relation to pain pressure, function and performance.

b) The research design

(Note: This section can include an overview of methodology research design proposals regarding for example, evaluation and data gathering. In describing the research design, applicants are required to explain the reasoning behind their choice of method)

Design: The study is a randomised controlled parallel group design.

Subjects will be required to attend a familiarisation session, three treatment sessions and two follow-up measurement sessions.

Familiarisation session: After screening and completing an informed consent, subjects will attend a familiarisation session. Baseline pain pressure threshold (PPT) measurements in the thigh will be recorded. After a generic warm-up, subjects will randomly perform two types jumps (countermovement jump and depth jump). Subjects will be randomly assigned to dry needling group, electrotherapy group or control group.

The treatment sessions: Subjects will return no more than 3 days later for testing. PPT and performance measures for each exercise will be recorded. The respective treatment will be carried out. Re-tests of performance measures will be randomly completed. Two more treatment sessions will be conducted 48 hours and 120 hours after the first treatment session, respectively.

Follow up sessions: The subjects will return three and five days after the final treatment. PPT and performance measures for each exercise will be recorded, randomly.

c) The size and composition of sample
Sixty subjects will be recruited from the student population. Each subject will read and sign a screening and consent form in the presence of the tester.

Subjects will be included in the study based on the following requirements:

Being healthy and free from pathology in the lower limb.

d) The method of how participants are expected to be selected, approached and recruited in conducting this proposed research?

(Note: The process of participant selection is required to be outlined clearly. If for example, participants are being contacted through an organisation, e.g. school, an initial step would be to seek permission from the organisation to approach the participants. Any inclusion or exclusion criteria must also be specified.

Potential subjects will be recruited from the student population of the Health Science and Sports courses at Institute of Technology, Carlow (Carlow campus). Permission will be sort from Course Coordinators and Heads of Department, respectively, prior to contacting students. Subjects will be recruited through e-mail, information talk before class and verbal invitation. Subjects will consist of 60 healthy individuals, male and female, between the ages of 18-35 years old who are willing to participate in the study. The requirements of the study will be made clear to the subject and participation will require subjects fulfilling the inclusion and exclusion criteria as outlined below. Each subject will read and sign screening and consent forms in the presence of the tester.

Subjects will be included in the study based upon satisfying the following criteria:

- Being familiar with strength, power and plyometric training,
- Having trigger points in the quadriceps femoris.

Subjects will be excluded based on the following:

- Any noticeable variance in muscle size or asymmetry;
- Systemic diseases of the muscular or nervous system;
- Congenital or childhood hip disease;
- History of hip trauma;
- Recent surgery;
- Inflammatory joint disease;
- Tumors;
- Lower limb/ lower back injury and treatment of respective muscles in the past 6 months;
- Meeting any contraindication for dry needling (ISCP, 2012).
  - Suffer from any bleeding disorders
  - Taking immunosuppression therapy
  - Mental incapacity
  - Lack of sensory feedback
  - Fever, inflammation or general malaise
  - Pathological skin lesions or infection
  - Pregnancy
  - Thrombosis
- Meeting any contraindication for electrotherapy (DJO Global, 2012)
  - Suffer from any bleeding disorders
  - Taking immunosuppression therapy
  - Mental incapacity
  - Lack of sensory feedback
  - Pregnancy
  - Prosthesis
  - Thrombosis

(e) Describe the procedures that will be adopted to maintain the confidentiality of research subject(s).

Participants will be assigned an ID number against which all data linked. Details linking the ID number and name of the participant will not be held together. Physical results will be held by the project supervisors under lock and key in an undisclosed location at Carlow I.T. A digital copy will also be held by the project supervisors protected by a password on the computer system at Carlow I.T. The information will be kept for a minimum of three years or indefinitely pending publication. Information will be used anonymously in the preparation of the project dissertation and other scientific reports for dissemination at scientific congress or in refereed publications. The research team will adhere to the 1997 Freedom of Information Act, (www.itcarlow.ie/resources/freedom-of-information.htm) and the data protection policy (www.itcarlow.ie/resources/data-protection.htm) and will inform participants of same.
f) Will any member of the intended group of research subjects, to your knowledge, be involved in other research projects or activities? If so, please give details and explain the nature of the engagement with other projects.  
The intended group of research subjects will be allowed to participate in other research at their own discretion.

g) Describe how the information is gathered, stored, handled and anonymised.  
The information will be anonymous as each subject will be allocated a case number and the results will detail this number only. Any written data collected will only use case numbers. Physical results will be held by the project supervisors under lock and key in an undisclosed location at I.T. Carlow. A digital copy will also be held by the project supervisors protected by a password on the computer system at I.T. Carlow. The information will be kept for a minimum of three years. An application to seek permission to publish will be submitted to the Ethics Committee if required.

h) Please state the location(s) the proposed research is to be conducted

The project will be carried out in the Human Performance Laboratory (C149), in the Institute of Technology, Carlow.

i) The proposed starting date of research/study
January 2016.

B.5 Has this research proposal received ethical approval from any other body? – if so please provide details.

There are no other bodies involved in the present research.

B.6 Does this proposed research require licensing approval? – if so please provide

details of licenses obtained.
B.7 Describe the research procedures as they affect the research subject and any other parties involved.

Subjects will be required to attend six sessions.

Session 1 (familiarization):

The subject will be made aware of all the requirements of the study prior to the commencement of testing as outlined in the consent form. They will be fully aware they can leave the study at any time. Physiological measurements will be recorded. Pain pressure threshold (PPT) will be recorded. Subjects perform a generic warm up. Surface electromyography electrodes will be placed on the thigh of one leg (this may require shaving the electrode placement site if necessary. When the participants are shaved a single use razor will be used, and the razor will be disposed of in the yellow sharps bin. The electrodes for the EMG machine which attach to the skin come in single use, sterile electrode packs. The subjects will randomly perform 3 attempts the countermovement jump and depth jump. A second familiarisation session may be needed if the four attempts vary more than 5% in the performance measures of exercises.

Session 2, 3 and 4 (treatments):

The subjects will return no more than 3 days after familiarisation for performance measures of strength, power and plyometric muscle activity and PPT. The subjects will be randomly assigned into three groups (dry needling, electrotherapy or control) and treated. Post-treatment measure will consist of performance measures.

Dry needling involves inserting an acupuncture needle 50-90mm into each identified trigger point in the quadriceps for approximately 90 seconds each.
Electrotherapy involves stimulating the tissue at a cellular level to improve metabolism and is non-invasive.

Session 5 and 6 (follow-up assessments):

The subjects will return after 5 and 7 days to randomly measure performance outcomes and PPT.

B.8 Describe (a) the ethical considerations of this proposal and (b) the steps to be taken to address these.

The subject may feel twitching, minor discomfort or pain at the site of trigger points or at a different location during palpation, measuring pain pressure threshold (PPT) and dry needling of the trigger points. This is a characteristic associated with trigger points and is a criterion for the location, measuring PPT and treatment of trigger points.

The subject’s thigh area will be exposed to the assessment and treatment of trigger points. Correct draping techniques and adequate screening will be in place to protect the subject’s privacy. The subject’s Skin will be disinfected with antiseptic wipes before and during each testing session, as required. The guide tube for dry needling is single use and will be disposed of in the bin after use. The subject may experience minor discomfort in relation to the performance measures in the following days due to delayed onset of muscle soreness. This is a normal phenomenon associated with exercise.

There are minor adverse effects associated with dry needling and electrotherapy, such as bruising, skin irritation and infection. The investigator has undergone additional training in both modalities. The thigh to be treated will be disinfected. Sterile single use solid filament needles with a guide tube will be used. They will only be opened immediately prior to insertion and disposed of in biohazards receptacles.

All subjects are free to withdraw from the study at any stage.
B.9 Please list the investigators (including assistants) who will conduct the research. Please provide details of their qualifications and experience

I, Richie Walsh, will conduct the research. I graduated from the Institute of Technology Carlow with an honours degree in Sports Rehabilitation and Athletic Therapy in 2015. I have gained two years’ experience working in a clinical environment, both in I.T Carlow and outside settings. I have worked as a Sports Rehabilitator with numerous teams in various sports. I have completed a continual professional development course in dry needling (David G Simons Academy™: Winterthur, Switzerland) the premier dry needling research and education centre in the world; and electrotherapy (DJO Education Training Course: Guilford, UK). I am a member of the Athletic Rehabilitation Therapy Ireland (ARTI). The scope of practice with ARTI covers therapeutic interventions including soft tissue techniques and therapeutic modalities including energy modalities. I also hold my own indemnity insurance (€6,500,000) as well as liability insurance (€150,000) covering dry needling and electrotherapy (Balens: Warwickshire, UK). As an insured and accredited practising professional athletic therapist, it is standard practice to diagnose and treat neuromuscular conditions without GP approval, particularly if subjects do not fit the contraindication outlined in the exclusion criteria.

B.10 Are arrangements for the provision of clinical facilities to handle emergencies necessary? If so, briefly describe the arrangements made.

I, the tester, am a trained emergency first responder and cardiac first responder. In the event of an emergency, I will immediately inform the laboratory technician and act accordingly to the seriousness of the emergency. I am aware that there is a defibrillator on site at main reception and in the Human Performance Laboratory should I need it. I have access to a telephone and the contact details for the doctor, nurse and relevant lecturing staff on campus. Clinical facilities required for study are the safe disposal of sharps and biohazard waste removal.
These facilities are already in use in the Human Performance Laboratory where the testing will be completed. Infection controls that are standard practices in relation to dry needling. Universal precautions will be used to keep the area that will be dry needled, including sterilising the area, and handwashing will be used prior to the application of latex gloves, single use needles with single used guide tubes, disposable razors and correct disposal of sharps and biohazard waste removal.

Participants will be allowed to be accompanied by a chaperone if they so wish of their choosing or a chaperone can be made available to them.

B.11 Specify whether research subjects include students or others in a dependent relationship.

Subjects will be recruited from the student population. Each subject will read and sign a screening and consent form in the presence of the tester.

B.12 Specify whether the research will include primary respondents such as children, individuals with mental health issues, individuals deemed to be of diminished responsibility, individuals with a physical or intellectual disability. If so, please explain the rationale for accessing these subjects for the proposed research. Please indicate alternative measures investigated to avoid the necessity for direct access to these primary respondents.

The research will only include adult subjects who are healthy and understand the requirements of the study fully.

B.13 Please confirm that no payment will be made to any research subject

No payment will be made to the subject for the involvement in the research project.
B.14 Describe the procedures to be used in obtaining a valid consent from the subject. Please supply a copy of the information sheet provided to the individual subject(s).

A consent form will be signed in the presence of the tester. Any questions or queries that the subject may have will be explained and understood.

B.15 Please indicate if there are any cultural, social, gender-based characteristics or sexual orientation, practices or behaviour of the subject(s) which have affected the design of the project or which may affect its outcomes.

The research will include only adult subjects who are healthy and understand the requirements of the study fully.

Signed: ____________________________ Date: __22/01/2016___
Researcher

Signed: ____________________________ Date ______________
Principal Investigator
(Supervisor)

REVIEWER COMMENT IF APPLICABLE FROM HEAD OF DEPARTMENT/GROUP/ INSTITUTE/CENTRE

Signed: ____________________________ Date ______________
(Head of Department/Group/CORE/Institute/Centre)
I do not believe that the participants’ GP needs to give written approval to take part in this study. Previous MSc projects on dry needling at I.T. Carlow have not had this as a requirement from the ethics committee (Barry 2015, Kennedy, 2015) and papers that have been published using dry needling on latent myofascial trigger points similar to the present ethics application have not reported this as a requirement (Lari et al., 2016, Martin-Pintado-Zugasti et al., 2014, 2015). It is not standard practice for physiotherapists to request the client get GP permission to be dry needled and is not a requirement under the ISCP guidelines for dry needling practice (ISCP guidelines for dry needling practice, ISCP, August 2012).

References


G. The effects of dry needling and shockwave on trigger point risk assessment

Please complete this form, adding extra lines where necessary. Risk Rating is calculated according to the scoring guide at the end of this document. Submit one copy to your supervisor, retaining a copy for yourself. The form may be amended during the course of your project work. A copy should be included with your final project document.

<table>
<thead>
<tr>
<th>Student: Richie Walsh</th>
<th>Project title: The effects of dry needling and shockwave on trigger point</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activity</td>
<td>Hazard</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Using algometer</td>
<td>Physical none</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chemical none</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Biological none</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Human/Health Bruising from using an algometer</td>
</tr>
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<td>3. Jumping</td>
<td>Physical none</td>
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<td>Category</td>
<td>Subcategory</td>
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<tr>
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<td>-------------------------------------------------------</td>
</tr>
<tr>
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</tr>
<tr>
<td>Biological</td>
<td>none</td>
</tr>
<tr>
<td>Human/Health</td>
<td>Musculoskeletal injury from jumping/ landing</td>
</tr>
<tr>
<td></td>
<td>Doing a warm-up prior to each session,</td>
</tr>
<tr>
<td></td>
<td>Instructing subject how to jump/land correctly.</td>
</tr>
<tr>
<td>4. Dry needling</td>
<td>Physical</td>
</tr>
<tr>
<td></td>
<td>none</td>
</tr>
<tr>
<td></td>
<td>Chemical</td>
</tr>
<tr>
<td></td>
<td>none</td>
</tr>
<tr>
<td></td>
<td>Biological</td>
</tr>
<tr>
<td></td>
<td>Blood contaminated sharps</td>
</tr>
<tr>
<td></td>
<td>Contaminated sharps will be disposed of and destroyed in the appropriate manor.</td>
</tr>
</tbody>
</table>
Human/Health
Post-needling soreness | 2 | 3 | 6 | Pressure will be applied post-needling to negate post-needling soreness.

5. Shockwave therapy

| Physical | none | 1 | 1 | 1 |
| Chemical | none | 1 | 1 | 1 |
| Biological | none | 1 | 1 | 1 |

Human/Health
Bruising from treatment | 2 | 2 | 4 | Only the maximum shockwave that the subject can tolerate will be used per session. Subjects can in for the therapist to reduce shockwave if desired.
**Risk Assessment**

Risk assessment is based on estimating the likelihood of an adverse event occurring combined with the severity of the outcome of the event in terms of death or injury. The matrix below acts as a simple guide to selecting the level of risk.

<table>
<thead>
<tr>
<th>PROBABILITY</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Almost certain</td>
<td>5</td>
<td>5</td>
<td>10</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>likely</td>
<td>4</td>
<td>4</td>
<td>8</td>
<td>12</td>
<td>16</td>
</tr>
<tr>
<td>possible</td>
<td>3</td>
<td>3</td>
<td>6</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>unlikely</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>rare</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

16-20 = high risk  12-15 = moderate risk  5-10 = low risk  1-4 = very low risk
H. The effects of dry needling and shockwave on trigger point informed consent

HUMAN PARTICIPANTS INFORMATION SHEET and

INFORMED CONSENT FORM

Project Title:

“The effects of electrotherapy and dry needling therapy of trigger points on muscle function in the quadriceps femoris and performance: a Randomised control Trial.”

Introduction to the study:

Being able to treat trigger points may improve muscle activity as well as strength and power performance markers.

I am being asked to take part in this research study.

The purpose of the study is to identify whether electrotherapy is as effective as dry needling in treating trigger points in the quadriceps femoris in order to improve muscle function as well as power and strength related performance.

This research study will take place at I.T. Carlow and will require me to attend the Institute for six 40-90 minute sessions over a 13 day period.

This is what will happen during the research days.

Day one:

My height, mass and age will be measured. I will perform 3 attempts of three exercises (isometric mid-thigh pull, counter movement jump and the depth jump). As well as pressure pain threshold (PPT) on trigger points in my thigh. I will be then free to leave.

Day two three and four:

I will complete the three exercise and PPT. I will be allocated to a treatment group, and will be treated accordingly. Performance exercises will be recorded again.
Day five and six:
I will perform a single bout of performance exercises and PPT.

Sometimes there are problems associated with this type of study. These are:
1. I may experience some dermatological irritation from the preparation and placement of the electromyography electrodes.
2. I may experience minor discomfort in the following days due to delayed onset of muscle soreness.
   1. The treatments may have adverse effect but are rare, these are:
      ▪ Dizziness / vertigo;
      ▪ Fainting;
      ▪ Excessive sweating;
      ▪ Injuries to nerves veins arteries;
      ▪ Infection;
      ▪ Bleeding;
      ▪ Bruising;
      ▪ Broken needles;
      ▪ Pneumothorax.

If there are any adverse effects during the visit I will be monitored until the effects pass.

There may be benefits to me from this treatment. These are may include improved power output in relation to my sport.

My confidentiality will be maintained. I.T. Carlow will make reasonable efforts to protect the information about me and my part in this study and no identifying data will be published. This will be achieved by assigning me an ID number against which all data will be stored. Details linking my ID number and name will not be stored with the data. The results of the study maybe published and used in further studies.

I am free to withdraw from this study at any stage I see fit for any reason. If I choose to do so it will not have any negative effects towards me now or in the future.

I will not be allowed to participate in this study if I have any of the following:
   ▪ Any noticeable variance in muscle size or asymmetry;
   ▪ Systemic diseases of the muscular or nervous system;
   ▪ Congenital or childhood hip disease;
   ▪ History of hip trauma;
   ▪ Recent surgery;
   ▪ Inflammatory joint disease;
   ▪ Tumors;
Lower limb/ lower back injury and treatment of respective muscles in the past 6 months;
- Bleeding disorders
- Immunosuppression
- Mental incapacity
- Lack of sensory feedback
- Fever, inflammation or general malaise
- Pathological skin lesions or infection
- Pregnancy
- Thrombosis
- Prosthesis

If I have any questions about the study, I am free to call Richie Walsh on 0857855088. Taking part in this study is my decision. If I do agree to take part, I may withdraw at any point including during the exercise test. There will be no penalty if I withdraw before I have completed all stages of the study.

Signature of participant: ____________________________________________

I have read and understood the information in this form. My questions and concerns have been answered by the researchers, and I have a copy of this consent form. Therefore, I consent to take part in this research project entitled “The effects of electrotherapy and dry needling therapy of trigger points on muscle function in the quadriceps femoris and performance: a Randomised Control Trial.”

Signature of participant: __________________ Date: __________________

Signature Witness: __________________________

Witness printed name: __________________________
Subject Screening Form

Department of Science and Health

School of Science

Institute of Technology Carlow

Carlow

Name:……………………………...…… Case number:……………………………………

D.O.B:………………………………….. Age:…………………………………………

The information obtained from this screening form is confidential and will not be disclosed to anyone without your permission.

1. Do you suffer from any lower back or lower limb injury(ies) which is currently preventing you from participating in your sport? Yes / No

2. Do you suffer from any neurological signs/symptoms (altered sensation, pins and needles, weakness) in the back, buttock, legs or feet? Yes / No

3. Do you suffer from any rheumatoid/systemic arthritis? Yes / No

4. Do you, to your knowledge, have any congenital or acquired hip deformities? Yes / No

5. Have you ever had pelvic or lowe r back surgery? Yes / No

6. Have you been treated for any lower back or lower limb injury(ies) in the past 6 months? Yes / No

7. Do you train, if so, how often.........(hours per week)? Yes / No

8. Do you participate in any other sports or physical activities, if so, how often.........hours per week. Yes / No

Signature of participant: _____________________ Date: ____________________

Signature Witness: _______________________

Witness printed name: _________________________
I. Research output generated from this project

I.i Presentations


I.ii Posters


I.iii Accepted


I.iv Under submission


extracorporeal shockwave therapy on countermovement jump and depth jump performance Journal Manual and Manipulative Therapy.