

**UNIVERSITY of
STIRLING**



The role of muscle contractile mechanics in neuromuscular control and performance adaptations to resistance exercise.

By

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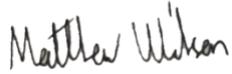
“Research is to see what everybody else has seen, and to think what nobody else has thought.”

Albert Szent-Gyorgyi

(Biochemist, Politician and Humanitarian)

Declaration

I declare that this thesis is composed by myself and that all the data were collected and analysed by myself, under the supervision of Dr Angus Hunter and Dr Lewis Macgregor. Neither the thesis, nor the original work contained herein have been submitted to this, or any other institution for a higher degree.



Matthew Thomas Wilson

Stirling, 28/09/2020

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General abstract

The implementation of strength training interventions within performance sport requires practitioners to have high levels of confidence in the training's efficacy to produce transferable increases in strength for improved sporting performance. Therefore, it is important to understand neural and morphological mechanisms of adaptation which lead to strength gain. Technological advancements now enable researchers to investigate specific sites of adaptation, however the peripheral and central regions of the neuromuscular system are typically investigated in isolation. The lack of integrated neuromuscular assessment within literature and practice represents a knowledge gap as to the inter-relationship between peripheral contractile properties and centrally governed adaptations to strength training. Whilst information regarding the specific mechanisms of adaptation can contribute to a practitioner's confidence in a training intervention, the method of inferencing applied to quantify training effects can be of equal importance. Traditional frequentist inferencing does not provide sufficient information to answer pertinent practitioner questions. Thus, an alternative method of inferencing may be more applicable within strength training contexts. As such, the overall aim of this thesis is to determine the role played by skeletal muscle contractile properties in adaptations to strength training, in relation to other peripheral and central regions of the neuromuscular system. This thesis aims to contribute to the literary gap of integrated neuromuscular assessment and outline a practically applicable method of inferencing within the context of strength training, in order to contribute to the confidence levels with which practitioners employ specific strength training interventions. Chapter 1 reviews the existing literature surrounding neuromuscular adaptations to strength training, their methods of non-invasive assessment, and inferential methods used to quantify training effects. Contractile mechanics adaptations to strength training, and their relationship with other adaptations were identified as areas requiring further investigation. Chapter 2 demonstrated the application of non-invasive contractile mechanics assessments in the context of strength training, as well as a level of construct validation for such contractile mechanics assessment; through associations between contractile properties and muscle architecture parameters. Subsequently, it was observed that contractile properties were altered prior to any other measured neuromuscular adaptation following strength training (chapter 3), and that there was no modulation effect between peripheral and central adaptations leading to strength gain. Furthermore, it was observed that firing rates of peripheral motor units did not appear to adapt following strength training, suggesting the early neural responses leading to strength gain appear to come from changes in spinal excitability. Chapter 4 confirmed the aforementioned absence of motor unit firing rate adaptations, despite being assessed in training-specific manner, using a dynamic strength test. However, this pilot study did demonstrate the applicability of motor unit

behaviour assessment during dynamic movement, providing information of high practical relevance within the context of strength training. Finally, chapter 5 demonstrated the successful application of Bayesian inferencing to quantify the certainty/uncertainty surrounding performance outcome effects following three different strength training interventions. This demonstrated analytical method provides directly interpretable information to answer the aforementioned practitioner questions and was capable of providing meaningful inferences in a situation where frequentist significance testing was unable to. This thesis demonstrates the adaptive responses of skeletal muscle contractile properties following strength training, and their relationship with other peripheral and central neuromuscular adaptations. The information provided within this thesis regarding the integrated assessment of multiple regions of neuromuscular adaptation, and the demonstrated method Bayesian inferencing, can provide practitioners with directly interpretable information upon the efficacy of employed strength training interventions, designed for improving athletic performance.

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Publications

Wilson, M.T., Ryan, A.M.F, Vallance, S.R, Dias-Dougan, A., Dugdale, J.H., Hunter, A.M., Hamilton, D.L., Macgregor, L.J. (2019).

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Presentations

Wilson, M.T., Ryan, A.M.F, Vallance, S.R, Dias-Dougan, A., Dugdale, J.H., Hunter, A.M., Hamilton, D.L., Macgregor, L.J. Tensiomyography as a tool to assess muscle specific training adaptations. *Poster session: European College of Sport Science Annual Congress, Dublin, Ireland, July 2018.*

ACSA	Anatomical cross-sectional area
AMRAP	As many repetitions as possible
aMT	Alpha motor threshold
BS	Back squat
CI	Confidence Interval
CSA	Cross-sectional area
cSP	Corticospinal silent period
CV	Coefficient of variation
dEMG	Decomposition electromyography
Dm	Radial muscle belly displacement
DL	Deadlift
E-C	Excitation contraction
EIMD	Exercise induced muscle damage
EMG	Electromyography
ES	Effect size
HDI	High density interval
HT	Hip Thrust
ICC	Intra-class correlation coefficient
M1	Motor cortex
MCMC	Markov Chain Monte Carlo
MEP	Motor evoked potential
MFR	Mean firing rate
MHC	Myosin heavy chain
M_{max}	M-wave
MU(s)	Motor unit(s)
MUAPT	Motor unit action potential train
MVC	Maximal voluntary contraction
NHST	Null-hypothesis significance testing
NUTS	No U-turn sampler
PCSA	Physiological cross-sectional area
PNS	Peripheral nerve stimulation
RIR	Repetitions in reserve
RF	Rectus femoris
RFD	Rate of force development

RPE	Rating of perceived exertion
1RM	1-maximal repetition load
5RM	5-maximal repetition load
p1RM	perceived 1-maximal repetition load
p5RM	perceived 5-maximal repetition load
PD III	Precision decomposition III algorithm
$p\eta^2$	Partial eta squared
Pre	Pre-intervention
Post	Post-intervention
SD	Standard deviation
sEMG	Surface-electromyography
Tc	Contraction time
Td	Delay time
TMS	Transcranial magnetic stimulation
Tr	Half-relaxation time
Ts	Sustain time
TE	Technical error
TMG	Tensiomyography
Vc	Contraction Velocity
VL	Vastus lateralis

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Chapter 1

Introduction and Aims

1.1 Introduction

Strength training is a staple component of training regimes in nearly all performance sports, as it is recognised as one avenue through which minor advantages over the competition can be obtained. The margins for success in performance sport are now so fine, that practitioners must understand what physiological changes are happening within their athletes during training, and what systems are involved in eliciting the desirable increases in physical strength. One such system can be termed the 'neuromuscular' system, encompassing both the central and peripheral nervous circuitries, as well as the make-up and contractile properties of skeletal muscles themselves (Enoka and Duchateau, 2019). The purpose of this chapter is to review existing literature outlining the adaptations that occur within various areas of the neuromuscular system, in the presence of a training stimulus. Specifically, within this chapter we review the effects of resistance exercise training aiming to increase strength (herein referred to as strength training) upon regions of the neuromuscular system which may undergo adaptation to increase force output, and ultimately improve athletic performance. In order to achieve optimal return from strength training interventions, neuromuscular adaptations within different areas must be understood in relation to one another (Sands et al., 2013) which in turn, will improve a coaches' confidence in strength training interventions they employ through appropriate inferencing (Flanagan, 2013). Accordingly, it is not the intent of this review to critique specific concepts of strength training for athletic performance, nor is it within the scope of this review to explore the in depth biochemical and metabolic processes associated with neuro-physiological adaptations.

As will be discussed, much of the current literature has assessed areas of neuromuscular adaptation, such as contractile mechanics, in isolation; with some assessment methods not practically applicable in the context of performance sport (Bosco et al., 2000; García-García et al., 2019). Furthermore, the 'traditional' methods of inferencing upon the efficacy of particular training interventions may also not be practically appropriate in the context of performance sport, where subtle outcome changes are often the case (Borg et al., 2018). As such, we will identify potential shortcomings of statistical inferencing within the context of intervention-derived effects and highlight alternative methods which may prove more inferentially rich to practitioners.

Thus the objectives of this chapter are threefold. Firstly, to briefly review the evidence base for including strength training within athletic training, and the inferential methods used to assess its efficacy; secondly, to outline the different areas of the neuromuscular system which are believed to contribute to increased physical strength following strength training. Specifically we will focus on

adaptations within the spinal- and supraspinal-centres, muscle motor units, muscle architecture and contractile mechanics; thirdly, in the context of non-invasive assessment methods, we will review the roles played by contractile mechanics in relation to other areas of the neuromuscular system in improving performance through increasing physical strength.

1.2 The purpose of strength training

1.2.1 Strength training for athletic performance

Strength is defined as ‘the ability to exert force on an external object’ (Siff, 2001). In sporting situations, an athlete may be required to exert large amounts of force against gravity in order to move in a certain way at a certain speed (e.g. sprinting and jumping), manipulate the body of an opponent (e.g. wrestling and rugby), or manipulate an object for projection e.g. (shot put, javelin, barbell). Within all of the aforementioned tasks, a determining factor to their success is the degree of strength (the force produced by active muscles) the athlete is able to exert. Consequently, from existent literature it appears there is no replacement for increasing strength when practitioners are looking to improve athletic performance, across both general and sport-specific skills (Suchomel et al., 2016). It has been suggested that strength training may hold the additional benefit of reducing injuries within sporting contexts (Lauersen et al., 2014), with the same authors later observing a strong dose-response relationship between increased strength and injury prevention (Lauersen et al., 2018); indicating the two go ‘hand in hand.’ Mechanistically, the reduction in injury rate following strength training may come from increases in the strength of ligaments and tendons, ligament-bone and tendon-bone junctions, and the connective sheath of muscle tissue (Fleck and Falkel, 1986). From the inclusion of strength training therefore, coaches may be able to not only elicit desired improvements in physical performance of general and sport-specific skills of their athletes but also further contribute to sporting success by reducing the risk of sport specific injuries (Shaw et al., 2016).

A number of strength associated force-time variables have been also been identified for successful sporting performance, particularly in events where time of task completion is of key importance. Considering Newton’s second law ($\text{force} = \text{mass} \times \text{acceleration}$), if the mass of an object against which force must be exerted is fixed (e.g. the athlete during a jump), how the speed of force generation becomes a limiting factor. For an athlete to be successful in a timed event, it is often deemed pertinent for them to possess a high rate of force development (RFD), and high levels of

mechanical power specific to the required task (Morrissey et al., 1995; Stone et al., 2002). RFD, defined as ‘the rate of rise in force over the change in time,’ (Aagaard et al., 2002) is believed to play particular importance owing to a large variety of sports requiring rapid movements (e.g. jumping, sprinting, throwing), where there is very little time to produce force (Andersen and Aagaard, 2006). With the previous authors showing maximal strength accounting for up to 80% of the variance in RFD when assessed in a voluntary task, it is unsurprising that increased RFD is commonly emphasised in strength training. Indeed, several studies have shown strength training to positively contribute to improvements in RFD (Häkkinen et al., 1985; Aagaard et al., 2002; Andersen et al., 2010; Folland et al., 2014).

Mechanical power (the rate at which an athlete transfers energy to complete a movement task (van der Kruk et al., 2018)) is often measured from the performance of a sport-related skill, which requires athletes to exert a maximal force over a limited time period (<300ms). Being related to a number of sporting characteristics (e.g. sprinting (Weyand et al., 2000) jumping (Cormie et al., 2010b), throwing (Marques et al., 2011) and change of direction (Nimphius et al., 2010), mechanical power may be another pivotal determinant of sporting success (Stone et al., 2002). A number of studies have revealed increases in relative or absolute lower-body power having culminated from strength training interventions (Harris et al., 2000; Cormie et al., 2010a; Speranza et al., 2016). Following a period of strength training, if greater muscular force is exerted upon an object of fixed mass, greater acceleration is produced, resulting in a greater velocity. With increased acceleration and velocity, there will be a resultant increase in mechanical power, demonstrating the importance of strength training. Indeed, previous research has shown that, for an athlete, greater mechanical power could mean the difference in being selected for a team (Young et al., 2005; Gabbett et al., 2009), or progressing to a higher level of competition (Fry and Kraemer, 1991; Hansen et al., 2011). The relationships between strength and the two aforementioned force-time variables therefore demonstrate the importance of strength training within athletic performance, and why parties involved seek to increase strength through strength training as a consequence.

1.2.2 Assessing changes in performance induced by strength training

As mentioned above, transference of muscular strength to athletic performance is a desirable outcome for coaches and athletes, with the principles of training specificity often of paramount importance. In order for practitioners to employ a particular strength training intervention or exercise, they must have confidence that it will be ‘worth their while’ and beneficial for the athlete. Such confidence usually comes from coaches’ personal experience, or from established research

protocols showing positive effects resulting from their use (Wright et al., 2012). In sport science research, null hypothesis significance testing (NHST) is commonly used to determine if a significant effect has occurred based on frequentist principles, and requires assumptions based on a large number of exact study replicates with different samples. By definition, NHST approaches posit only a null hypothesis to test against (i.e. that no change in the parameter of interest will occur) based on the returned level of significance – the *p-value*. Thus, NHST approaches do not provide as rich an inferential context as many practitioners would like, as *p-values* only inform upon the probability that an improvement is happening based on an assumption that nothing is happening (Aarts et al., 2011; Wasserstein et al., 2019). Practitioners and athletes would rather find out whether a training effect is happening, what magnitude of an effect is being seen, and how confident are we that the observed effect is a true and meaningful one.

In addressing the inferential issues of *p values*, there has been calls for moving away from ‘frequentist approaches’ to the estimation of effect sizes and the uncertainty of around those estimates. Indeed, published guidelines on statistical inferencing in sports science and moving away from binary decisions of importance to estimating sizes of effect support this (Cumming, 2008;2013). The estimation of effect sizes and uncertainty around such estimates are central to magnitude-based inferencing (MBI) (Batterham and Hopkins, 2006) which focuses on the interpretation of frequentist-based confidence intervals (CIs). Whilst it did heighten awareness of estimate-based thinking in sports science, MBI was shown to have sub-optimal properties for its proposed purpose (Barker and Schofield, 2008; Welsh and Knight, 2015). In a comprehensive review of MBI, Sainani (2018) outlines how it creates high levels of type 1 error at the expense of reducing type-2 error, at precisely the small-moderate sample sizes at which MBI was proposed optimal (Batterham and Hopkins, 2006). Similar to others, Sainani (2018) also points out the incorrect interpretation of frequentist CIs within MBI as if they were Bayesian credible intervals, leading to inappropriate conclusions being drawn (i.e. MBI interprets a 95%CI which falls entirely within the ‘trivial’ range, as meaning there is a 95% the effect is trivial).

The motivation behind an approach such as MBI is a good one, as it encourages researchers to pay more attention to CIs. Whilst the mathematical flaws in MBI have led to its controversy in being applied within sports science (Lohse et al., 2020), it does highlight the need for more inferentially rich statistical approaches, especially when hypothesised effects are expected to be small and large sample sizes are typically unavailable.

1.2.3 An alternative approach?

An alternative approach to obtain answers to practitioner questions such as; is a training effect happening? What size is the training effect being seen? How much confidence is there that the effect is a true and meaningful change? – could be based on Bayesian principles. In such an approach (Etz et al., 2018), the researcher first decides on a statistical model for the data in question, however the parameters are considered as random variables, described by a posterior probability distribution which reflects the uncertainty with which the variables are known, based upon the data at hand. This distribution is obtained by multiplying the likelihood (the probability of observing the data given specified values of the parameters) and the prior distribution(s) (the known knowledge about the effect under study before any of the current data is accounted for) (Mengersen et al., 2016). Using Bayesian theorem, the likelihood and prior distribution(s) are combined to generate the posterior distributions which are the target of Bayesian inferencing.

As previously mentioned, the movement away from NHST and movement towards estimation of effect sizes and CI's has not been without problems thus far (Lohse et al., 2020). In the case of MBI, CI interpretation was shown incorrect in its mathematical basis (Cumming et al., 2004; Cumming, 2008), resulting in misinformed conclusions to be drawn. Sainani (2018) demonstrated that the MBI misinterpretation was more akin to Bayesian credible intervals (an interval within which a population parameter value (e.g. mean) falls within a particular probability) suggesting there is perhaps already an avenue to apply Bayesian-like inferencing, centred upon effect sizes and the uncertainty around those estimates (Borg et al., 2018).

Within the sports science, the concept of inferenced based thinking poses an interesting standpoint for practitioners who wish to quantify changes in performance measures following strength training interventions. Measures of performance such as sprint time, jump height, and lower body strength are commonly used as distinguishing factors between competition level (Stone et al., 2004; Gorostiaga et al., 2005). Within such performance testing, error quantification is often critical to allow a practitioner to identify a 'true' change apart from change due to error. Such inference based thinking was recently proposed by Swinton et al. (2018). In particular, this approach estimated a 'proportion of response' following an intervention, as the proportion of posterior distribution which lies above an investigator defined threshold (Swinton et al., 2018). This threshold can be considered 'technical error' (TE) (Hopkins, 2000), which encompasses variability in test-retest measurements at baseline (e.g. the standard deviation of test-retest values). This TE is of particular

significance in sport science as multiple sources of variations often exist when carrying out performance testing (e.g., testing conditions and athlete competency). Being able to quantify TE in the context of strength training interventions, would provide more confidence in performance test outcomes which training interventions aim to improve; something that isn't possible based on frequentist p -values.

Thus, Bayesian inferencing provides directly interpretable intervals which indicate, with a level of certainty (typically 95%), the location of where the true parameter (effect) lies. In addition, the posterior distribution be used to calculate the probability of a training effect exceeding a pre-defined threshold (e.g., error or a performance test), which can be defined by the practitioner. These unique properties of Bayesian analysis may aid in helping to answer the aforementioned questions of; 'has a change in performance occurred?' 'How much of change has occurred?' and 'How much confidence is there that the observed effect is true and meaningful?'

1.3 The Neuromuscular system

To understand how strength training can lead to the previously described improvements in athletic performance, it would be pertinent to briefly review the physiological pathways involved in eliciting muscle activation and contraction. These pathways represent the locations of multiple potential adaptations (figure 1.1) which can occur due to strength training, and ultimately lead to increased strength through both neural and morphological changes.

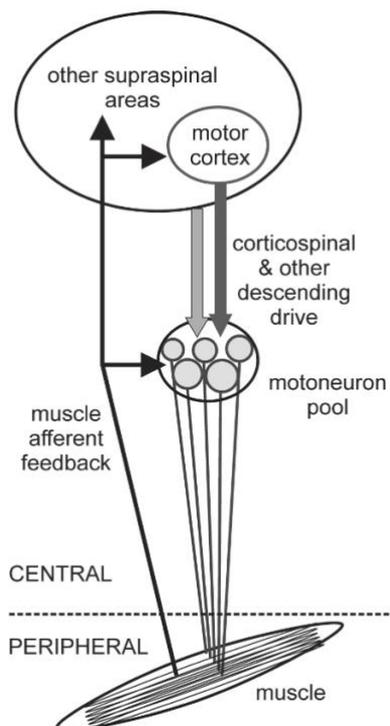


Figure 1.1 Basic schematic of the neuromuscular system outline key areas involved in the generation of muscle contractile force. Adapted from Taylor et al. (2016).

The neuromuscular system constitutes a neural network comprised of motor neurons, sensory neurons and associated skeletal muscle fibre. The base unit of the neuromuscular system can be considered as the motor unit (MU); a motor neuron and the group of muscle fibres which it innervates (figure 1.2). Motor units (MUs) represent the final pathway by which the central nervous system transmits an activation signal to a skeletal muscle (Duchateau and Enoka, 2011).

Collectively, the hundreds of MUs within a muscle are referred to as the 'motor unit pool', whose associated motor neurons gather within the spinal cord or brainstem. These motor neurons receive multiple synaptic inputs from other neurons such as sensory feedback and descending action potential volleys from cortical neurons (Heckman and Enoka, 2012), which are critical for eliciting voluntary muscle contractions. Descending volleys originate from the primary motor cortex (M1) within the brain when they receive an afferent input, signalling the requirement of a movement response (Kidgell et al., 2017). Upon stimulation, the M1 signals descending pyramidal neurons, inducing efferent signals in the spinal cord by synapsing with the motor neurons.

When an efferent input depolarises the resting membrane potential of a spinal motor neuron above its threshold, an action potential is propagated along its axon, to the axon terminals at the neuromuscular junction with the associated muscle fibres.

The neuromuscular junction, being roughly located in the middle of a muscle fibre, transmits muscle fibre action potentials in both directions to either end of the muscle fibre (Enoka and Duchateau, 2019). The sum of all MU action potentials is termed as neural drive, which establishes the amplitude of force to be elicited by the muscle. For the control of muscle force, the centrally governed efferent motor output determines how many MUs are recruited and the rates at which they discharge action potentials. To produce force, MUs are recruited according to their size (smallest/weakest to largest/strongest) - Henneman's size principle (Henneman, 1957), however the specific parameters of recruitment and discharge rate are dependent upon the type of muscle contraction required (Enoka and Duchateau, 2019). This size principle works on the mechanistic basis of smaller MUs having greater input conductance (Ohm's Law), meaning their depolarisation towards threshold is greatest upon receiving efferent signalling from supraspinal centres (Duchateau and Enoka, 2011).

The excitation of multiple muscle fibres creates contractile tension within skeletal muscle, with each muscle fibre containing thousands of myofibrils and billions of myofilaments (Frontera and Ochala, 2015). It is these myofilaments which are arranged into sarcomeres; contractile elements comprised

primarily of actin and myosin proteins. The final link in the process of contractile force generation is termed cross-bridge cycling, which is the end result of a process known as excitation-contraction (E-C) coupling (Rebeck et al., 2014). Excitation-contraction coupling is essentially the transmission of motor neuron action potentials across the neuromuscular junction, giving rise to calcium ion release from the sarcoplasmic reticulum (SR) leading to the formation of actin-myosin cross-bridges.

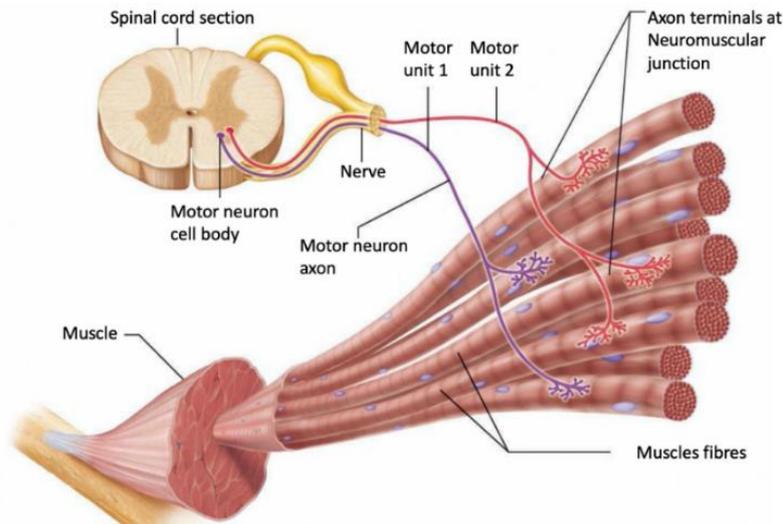


Figure 1.2 The structure of typical motor units (MUs) comprised of motor neurons and the associated muscle fibres which they innervate. Copyright 2009 Pearson Education, Inc, publishing as Benjamin Cummings.

Further discussion of the implications of cross-bridge cycling is beyond the scope of this review, however a brief summary of how contractile mechanical tension is created within muscle fibres will follow.

The formation of actin-myosin cross-bridges leads to tension being created within a muscle, and transmitted either along, or perpendicular to the sarcomere's longitudinal axis (Street, 1983; Ramaswamy et al., 2011). Longitudinal sarcomere tension is believed to account for only 20-30% of overall muscle tension created by sarcomere (Bloch and Gonzalez-Serratos, 2003) with the remaining resulting from perpendicular (lateral) tension being channelled from one sarcomere to another via the sarcolemma and extracellular matrix by costameres. For a full review into costamere structure and function see Peter et al. (2011).

The contractile mechanical tension developed during contraction is dependent upon three main factors; 1) the extent of cross-bridge formation (optimal sarcomere length produces the high number of formations and therefore the highest force (Gordon et al., 1966; Huxley and Simmons, 1971)); 2) the velocity of shortening within the muscle (faster shortening means less time for cross-bridge formation) (Hill, 1938); 3) lengthening of muscle (stretching of the S2 myosin complex as they remain attached, and then eventually detach to avoid damage however are in a state of enhanced readiness to re-attach) (Katz, 1939; Lombardi and Piazzesi, 1990; Jones et al., 2004).

1.4 Adaptations to strength training

As noted in the previous sections, training-induced increases in strength can produce desired improvements in athletic performance, both in general skills and sport-specific skills, as well as contributing to reduced injury risk. With the neuromuscular system one of the key physiological systems responsible for eliciting increases in strength, it is vitally important for practitioners and athletes to understand how the underpinning physiological and neural adaptations lead to increased muscular strength. The following discussion will provide a review of neural adaptations to strength training, both in the central and peripheral nervous systems, as well as the morphological adaptations of muscle structure and in the contractile mechanics of muscle tissue which ultimately lead to contractile force generation.

1.4.1 Mechanisms of Neural adaptations to strength training

It has long been a general consensus that initial increases in strength following strength training are a result of primarily neural adaptations (Narici et al., 1989;Enoka, 1998;Carroll et al., 2002). These early gains in strength have been associated with an increased ability to activate muscles being targeted by the respective training intervention (Hakkinen and Komi, 1983). Early studies used surface electromyography (sEMG) to determine the change in efferent motor output from cortical areas within the neuromuscular system (Davies et al., 1985;Sale, 1988;Aagaard, 2003). Indeed, the muscle fibre action potentials which are recorded by sEMG electrodes possess the same transmembrane currents as axonal potentials within the motor neuron (Enoka and Duchateau, 2019). Such sEMG signals can therefore be useful for studying neuromuscular control (Farina et al., 2016). Further discussion of the use of sEMG specifically in the context of strength training adaptations is covered below (section 1.5.1).

1.4.1.1 Cortical plasticity

Research surrounding the involvement of cortical plasticity leading to increased muscle strength has stemmed from proposed neural network similarities between skill training and strength training. For example, long-term potentiation is considered to occur at existing synapses during the early stages of skill acquisition (Rosenkranz et al., 2007), whilst the same potentiation response has been proposed to occur within the primary motor cortex (M1), underpinning early gains in muscular strength (Carroll et al., 2002;Selvanayagam et al., 2011). Through the use of Transcranial magnetic stimulation (TMS), insights have been provided into adaptive changes within the cortico-cortical

circuitry of the M1 following strength training (Kidgell and Pearce, 2010). Whilst the current section focuses on the underpinning cortical adaptations inferred from studies employing TMS, a more in-depth review of TMS application in the context of strength training can be found in section 1.5.2. To provide context to the discussion at present, TMS assessment evokes a series of descending action potentials in the corticospinal pathway, causing a muscle response referred to as a 'motor evoked potential' (MEP) (Kidgell et al., 2017). Three key variables are commonly derived from sEMG traces of a TMS assessment; motor threshold (MT) and MEP amplitude which are used to assess corticospinal excitability parameters (Kidgell et al., 2017), and corticospinal silent period (cSP) which reflects the degree of corticospinal inhibition.

Motor threshold represents the membrane excitability of corticospinal neurons, interneurons projecting within the M1, as well as the excitability of motor neurons within the spinal cord, the neuromuscular junction and the muscle itself (Rossini et al., 1994). Previously, MT has been observed to reduce following motor skill training (Pascual-Leone et al., 1995), however a meta-analysis determined strength training had no overall effect upon MT (Kidgell et al., 2017); indicating strength training has no influence upon the neuronal membrane excitability within the M1. The amplitude of a MEP represents the efficacy of the corticospinal pathway, and the excitability of the M1, and the efficiency of action potential transmission along the peripheral motor pathways (Hallett, 2000). Thus, alterations in the amplitude of MEP can reflect in the strength of corticospinal projections onto spinal motor neurons innervating skeletal muscles. Increases in corticospinal excitability have been reported following short periods (3-5 weeks) of strength training (see Table 1). Indeed, this was recently confirmed in a meta-analysis investigating the locations of early-cortical adaptations to strength training (Siddique et al., 2020). These observed increases in MEP amplitude following strength training would appear to represent enhanced excitability of M1 micro-circuitries and excitability of the spinal tract; both resulting in increased efferent drive to skeletal muscles and increased force production (Brownstein et al., 2018; Siddique et al., 2020).

Meta-analyses have also confirmed a moderate effect for strength training-induced reductions in cSP measured via TMS (Kidgell et al., 2017; Siddique et al., 2020). This cSP parameter reflects the degree of synaptic efficacy of inhibitory networks within the M1 and corticospinal pathway, referred to as 'corticospinal inhibition.' Corticospinal inhibition is mediated by the neurotransmitter gamma-aminobutyric acid (GABA) and its associated type-B receptor (GABA_B) (Werhahn et al., 1999). Studies observing a reduction in cSP following resistance training (table 1) have suggested strength training induces a reduction in the activity of inhibitory neurons, downregulating GABA-ergic inhibition

(Nuzzo et al., 2016), and leading to an increased efferent drive to produce the desired movement (Zoghi and Nordstrom, 2007). Continual sensory feedback during strength training, being transmitted via group III and IV afferents to the M1 is believed to be contributory (Gandevia and Burke, 1990).

1.4.1.2 Spinal reflexes

Outside of the degree of muscle activation that can be altered with strength training, research has looked to investigate changes in spinal reflex physiology following strength training. Changes in spinal reflex parameters provide evidence for changes in the excitatory and inhibitory networks of the spinal cord and are believed to contribute to early strength gain (Aagaard et al., 2002; Del Balso and Cafarelli, 2007; Fimland et al., 2009). Through the use of peripheral nerve stimulation and EMG recording techniques (figure 1.3) parameters such as the H-reflex (Hoffmann, 1918) can be used to assess the excitability of motor neurons (Schieppati and Crenna, 1985) and the efficacy of synaptic transmission in type Ia afferents (Nielsen and Kagamihara, 1993). Changes in the amplitude of H-reflexes are inconsistent within strength training literature (Holtermann et al., 2007; Duclay et al., 2008; Fimland et al., 2009; Ekblom, 2010). However, in the cases of increased H-reflex amplitude, an increase in the excitability of motor neurons and the efficiency of type Ia synaptic transmission is suggested as the underlying cause (Aagaard et al., 2002).

A second parameter termed 'V-wave' can be used to reflect the level of efferent neural drive from spinal motor neurons during a maximal contraction (Upton et al., 1971) and is elicited only during maximal voluntary activation. Increases in V-wave amplitude have been noted as evidence of enhanced activation of the motor neuron pool (Aagaard et al., 2002), leading to improved muscle activation and force production. When increased amplitudes of V-wave and H-reflex were observed in tandem, the increased motor neuron activation was attributed to adaptations occurring at the supra spinal level (Aagaard et al., 2002; Del Balso and Cafarelli, 2007). Like the H-reflex however, there are potential limitations to making inferences based upon V-wave amplitudes alone, particularly as they are influenced by the number and firing rate of recruited MUs, and the efficacy of synaptic transmission between type Ia afferents and motor neurons (Del Balso and Cafarelli, 2007).

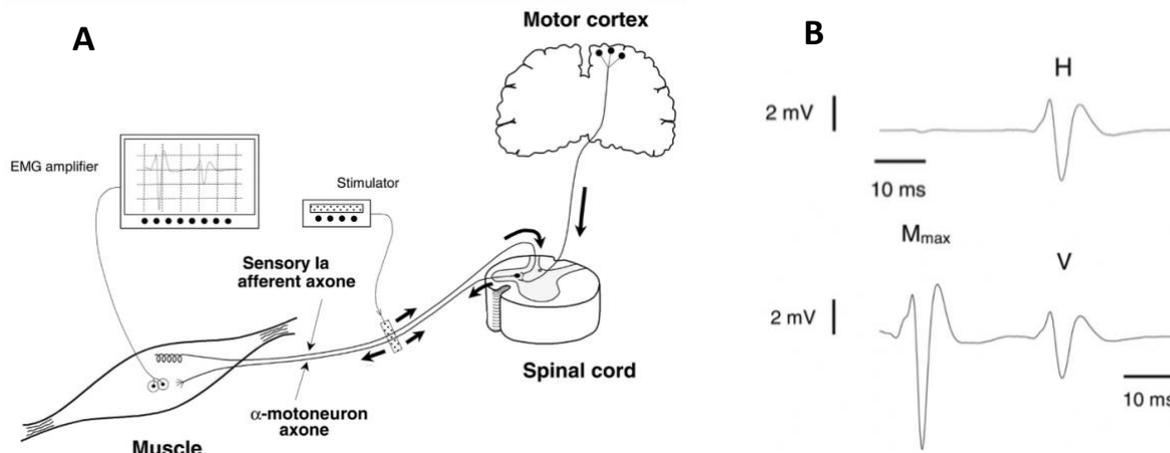


Figure 1.3; Experimental set up (A) and representative sEMG traces (B) used to record and measure spinal reflex parameters induced by peripheral nerve stimulation. H – H-reflex; V – V-wave; M_{max} – M-wave. (Adapted from Aagaard et al, 2002).

1.4.1.3 Adaptations within the motor unit

Being the final common pathway of the neuromuscular system, the MU's basic function is to transform efferent synaptic input from the motor neuron, into a mechanical force output from the muscle contractile elements (Heckman and Enoka, 2004). The mechanical force produced by a muscle has three neural dependents; the amount of MU activity (e.g. conduction velocities, synchronization, recruitment threshold) (Adrian and Bronk, 1929), the number of active MUs, and the rate at which motor neurons discharge action potentials (firing rate) (Duchateau et al., 2006). Following 12-weeks resistance training, Van Cutsem et al. (1998) observed an increase in Tibialis anterior (TA) MVC strength, and the speed of voluntary ballistic contraction. The authors postulated that this second observation was resultant of a neural adaptation as evoked TA muscle twitch-time parameters had remained unchanged. The authors found that whilst size principle of MU recruitment was maintained post-training, Tibialis anterior MUs had greater maximal firing rates during ballistic contractions and were recruited earlier post-training indicating a reduction in recruitment threshold. The aforementioned authors also observed an increase in 'doublet' firing within TA MUs during post-intervention ballistic contractions. The phenomena of 'doublet' firing refers to a double MU discharge with less than 5ms between them (inter-spike interval) and serve a functional significance of increasing the maximal rate of contractile tension developed (Van Cutsem et al., 1998).

A later study by Vila-Chã et al. (2010) investigated quadriceps MU behaviours following 6-weeks strength, or endurance training. Unlike the previous work of Van Cutsem et al. (1998), Vila-Chã and colleagues measured MU and motor output parameters (MVC and RFD) halfway-through the

intervention period (3-weeks). The authors observed an increase of ~8% in average MU discharge rate recorded at 30% MVC, after the first 3 weeks of strength training; and that this was maintained at the end of the 6-week intervention. Interestingly, the increase in discharge rate after 3 weeks training was not accompanied by similar increases in muscle fibre conduction velocity, indicating that early adaptations to strength training likely involve altered efferent drive. The aforementioned studies would suggest that altered MU properties are resultant of an increase in the net excitatory input to the motor neuron pool for the same relative contraction force. Certainly, a recent study (Del Vecchio et al., 2019) was able to observe the same previously mentioned adaptations (increased firing rate and decrease recruitment threshold) in a small number tracked MUs after 4-weeks of isometric strength training. This recent study could be considered to solidify the previously observed MU adaptations, as they were observed across tracked MUs and not only based on pooled-group averages. Despite a number of studies investigating individual MU recordings (Van Cutsem et al., 1998; Vila-Chã et al., 2010) and sEMG recordings (Aagaard et al., 2002; Balshaw et al., 2016; Del Vecchio et al., 2019), the precise nature leading to MU adaptations following strength training remains unclear. Certainly, as previous studies have, by large, not assessed MU alterations in conjunction with associated changes in cortico-spinal mechanisms or muscle contractile mechanics, information regarding the precise time-course of adaptations remains uncovered.

1.4.2 Skeletal muscle contractile property adaptations to strength training

1.4.2.1 Contraction speed

Contractile speed refers to the rate at which force is generated during muscle contraction, of which there are two measurements that can be quantified: contraction time and maximal shortening velocity.

Contraction time depends on the rate at which Ca^{2+} is released from the SR within muscle cells and represents the time to achieve peak twitch force upon activation by a single stimulus (Ørtenblad et al., 2000). When considered on a MU level, the contraction time of MUs is spread continuously and does not comprise of distinct slow- or fast-twitch MUs (Cutsem et al., 1997). The aforementioned study (Ørtenblad et al., 2000) showed that following 5 weeks training, Ca^{2+} release from the SR is enhanced due to increased SR volume and the number of ryanodine receptor responsible for Ca^{2+} release. Muscle contraction time may also be reduced via transitioning of muscle fibres towards type II 'fast twitch' fibres (Andersen and Aagaard, 2000). Further discussion on fibre type transitioning can be found below (section 1.4.3.4).

Maximal shortening velocity involves the maximal activation of a single MU and measuring the peak rate at which muscle length shortens when pulling against the lightest load possible (Schiaffino and Reggiani, 2011). The measurement indicates the rate at which thick and thin filaments slide over each other within muscle fibres and is limited by the rate of adenosine di-phosphate (ADP) release during cross-bridge cycling. Considering the force-velocity relationship (Hill, 1970), the speed of muscular contraction, and its intrinsic components as described above, can be determinants of force output during muscle contraction. Therefore, when seeking to improve force output through training, contraction speed must be considered as an influential factor.

1.4.2.2 Peak twitch force and specific tension

A second skeletal muscle contractile property is peak twitch force which depends on the number of innervated fibres by motor neurons, the average cross-sectional area (CSA) of muscle fibres, and muscle fibre specific tension – the maximal force per unit of physiological cross sectional area (Enoka and Duchateau, 2019). It is important to note that peak twitch force of a muscle does not represent its maximal force capacity, as a single stimulation is not sufficient to fully activate all MUs (Macefield et al., 1996). Changes in muscle fibre CSA following strength training interventions have been observed and therefore their alterations could result in changes of peak twitch muscle force (Narici et al., 2016). For further detail on CSA adaptations see section 1.4.3.1. Peak twitch force may also be influenced by changes in muscle fibre specific tension (Faulkner et al., 2007; Harridge and Lazarus, 2017). Indeed, increases in specific tension have been observed following strength training (Erskine et al., 2010b; Erskine et al., 2010a) and have been shown to be elevated in long term strength trained athlete's vs untrained controls (Maden-Wilkinson et al., 2019). Whilst the specific mechanisms accounting for increased specific tension have not yet been confirmed, possible explanations include an increased packing density of myofibrils within muscle fibres (Parente et al., 2008; Pansarasa et al., 2009), and an increased ability to transmit force laterally to the tendon (Jones et al, 1989). Increased lateral force transmission may occur through increased attachments between sarcomeres and the extracellular matrix via costamere proteins (Kosek and Bamman, 2008; Li et al., 2013). The consequence of this increase in specific tension would allow the 'effective' CSA of a muscle to increase, thereby by increasing its force output without additional contractile material to be required (Erskine et al., 2010b).

1.4.3 Morphological adaptations to strength training

1.4.3.1 Muscle Size

Morphological adaptations to strength training are generally considered a slower process with a delayed onset, compared to the previously discussed neural adaptations (Blazevich et al., 2007). A matter of common observation is that strength training can lead to an increase in muscle size, with numerous studies observing increased cross-sectional area (CSA) over periods of 6-12 weeks (Abe et al., 2000; Blazevich et al., 2003; Damas et al., 2015) using a variety of measurement techniques (e.g. magnetic imaging resonance (MRI), computed topography (CT), and ultrasound imaging (see section 1.5.3)).

Training-induced increases in muscle fibre size (myofibril hypertrophy) are the product of increased muscle protein synthesis over the rate of protein degradation, resulting in net contractile protein gain (Enoka and Duchateau, 2019). This increase in contractile protein is accompanied by the activity and fusion of satellite cells with muscle fibres, adding new nuclei and augmenting the number of available sites for DNA transcription in the muscle cell. The activity of satellite cells is thought to be stimulated by 'micro damage' to muscle fibres, induced by the stress and strain experienced during strength training, and is mediated by a number of intracellular signalling pathways (Snijders et al., 2015). The outcome of the increase in muscle protein synthesis is the proliferation of actin and myosin filaments within each muscle fibre, increasing the potential for cross-bridge formation and thereby the force production capacity of the muscle (Enoka and Duchateau, 2019).

Hypertrophy of muscles fibres through the above described pathways is considered primary contributor to increases in physiological cross-sectional area (PCSA) following resistance training (Franchi et al., 2014). Indeed increases in PCSA have been observed following periods of resistance training ranging from 5-14 weeks (Seynnes et al., 2007; Campbell et al., 2013; Vieira et al., 2018), supporting the consensus of hypertrophic adaptations occurring after the initial stages of resistance training. Whilst changes in PCSA are considered the most reflective measure of contractile material hypertrophy (Wisdom et al., 2015), it can be inherently difficult to quantify (Franchi et al., 2018b). Thus, there are a number of other measures reflecting increased muscle size following strength training which have been used in the literature such as; anatomical cross-sectional area (ACSA), muscle volume, muscle thickness, and fat free mass/lean mass ratios (Franchi et al., 2018b). For further details on the measurement methods used to quantify skeletal muscle hypertrophy, see section 1.5.3.

In addition to the mechanisms of myofibril hypertrophy described above, evidence within literature points to potential for sarcoplasmic hypertrophy coinciding with myofibril hypertrophy following

strength training (Roberts et al., 2020). Whilst animal and human model studies have supported the potential for sarcoplasmic hypertrophy as contributory to increased muscle size (Meijer et al., 2015; Haun et al., 2019; Vann et al., 2020); there is equal evidence that refutes this contribution (Goldspink, 1964; Trappe et al., 2001; Roberts et al., 2018), demonstrating this is an unresolved area. Sarcoplasmic hypertrophy can be defined as “an increase in the volume of sarcoplasm accompanied by an increase in the volumes of mitochondria, SR, t-tubules or substrate content” (Haun et al., 2019). The proposed mechanism for sarcoplasmic hypertrophy following strength training is said to involve sarcoplasmic expansion accompanied by the up-regulation of sarcoplasmic proteins involved in metabolic processes (Haun et al., 2019). Further discussion surrounding sarcoplasmic hypertrophy is beyond the scope of this review, however as a possible contributor to increased muscle size following training, it must be acknowledged. For further discussion of sarcoplasmic hypertrophy see Roberts et al. (2020).

Increased muscle size through hypertrophy has been demonstrated as a consequence of strength training using multiple sample populations, muscle groups, and training methodologies. However, despite this substantial evidence, debate remains surrounding the supporting role of muscle hypertrophy contributing to strength gain following training (Loenneke et al., 2019; Taber et al., 2019). In a recent series of reviews and responses, the aforementioned authors discussed and contended the role of hypertrophy contributing to strength gain, with both sides demonstrating disparity in the literature supporting either side of the debate. Loenneke et al. argued that no clear causal relationship between increased size and increased strength exists, demonstrated by two studies (Dankel et al., 2017; Mattocks et al., 2017) showing similar increases in strength but different increases in muscle size, due to different training methodologies used over 8 weeks. In contrast, Taber et al. argue that hypertrophy may not be *necessary* or *sufficient*, but is most likely contributory to increased strength owing to the inability to measure *all* variables that could contribute to strength gain within a training study. However, all authors concluded that to increase clarity on the issue, the role of muscle hypertrophy contributing to strength gain should be investigated side-by-side to other potentially contributory mechanisms which also appear to follow the principle of specificity (Loenneke et al., 2019; Taber et al., 2019).

1.4.3.2 Structural remodelling: pennation angle

Aside from the increases in muscle size commonly observed following strength training, literature has also demonstrated various degrees of structural remodelling designed to improve the force production capacity, and the force transmission properties of skeletal muscle (Blazevich, 2006). One

key parameter of this structural remodelling is angle of fibre pennation or ‘pennation angle.’

Pennation angle can be defined as the angle at which muscle fascicles insert into the aponeurosis of a muscle (Timmins et al., 2016) (figure 1.4). Pennation angle of muscle fibres allows pennate muscle to have a physiological cross-sectional area (PCSA) that exceeds its anatomical cross-sectional area (ACSA), as PCSA equals the magnitude of muscle fibre area perpendicular to the longitudinal axis of muscle fibres multiplied by, the cosine of angle of pennation (Powell et al., 1984). Thus, the PCSA of pennate muscles increases in proportion to $\sin(\text{pennation angle})$ (Alexander and Vernon, 1975; Rutherford and Jones, 1992). The increase of pennation angle will therefore allow more contractile material to be packed into the same ACSA, increasing the number of muscle fibre attachments to the tendon (Aagaard et al., 2001). However, the amount of force that is transmitted from each fibre to the tendon, in the case of increased pennation angle following strength training, will decrease.

Therefore, the overall effect of increased pennation angle can be considered a trade-off between increased PCSA and mechanical-disadvantage (Folland and Williams, 2007). According to the relationship previously calculated by Alexander and Vernon (1975), the optimal pennation angle of muscle fibres is 45 degrees; therefore any increase in pennation angle observed as an adaptation to lower body strength training would be expected increase force production, as upper and lower leg muscles possess pennation angles of between 1-30 degrees (Ward et al., 2009). Indeed, numerous studies have observed increases in quadriceps pennation angle after lower-body strength training (Aagaard et al., 2001; Blazevich et al., 2007; Matta et al., 2014; Wells et al., 2014), demonstrating structural remodelling as a contributory adaptation to strength gain.

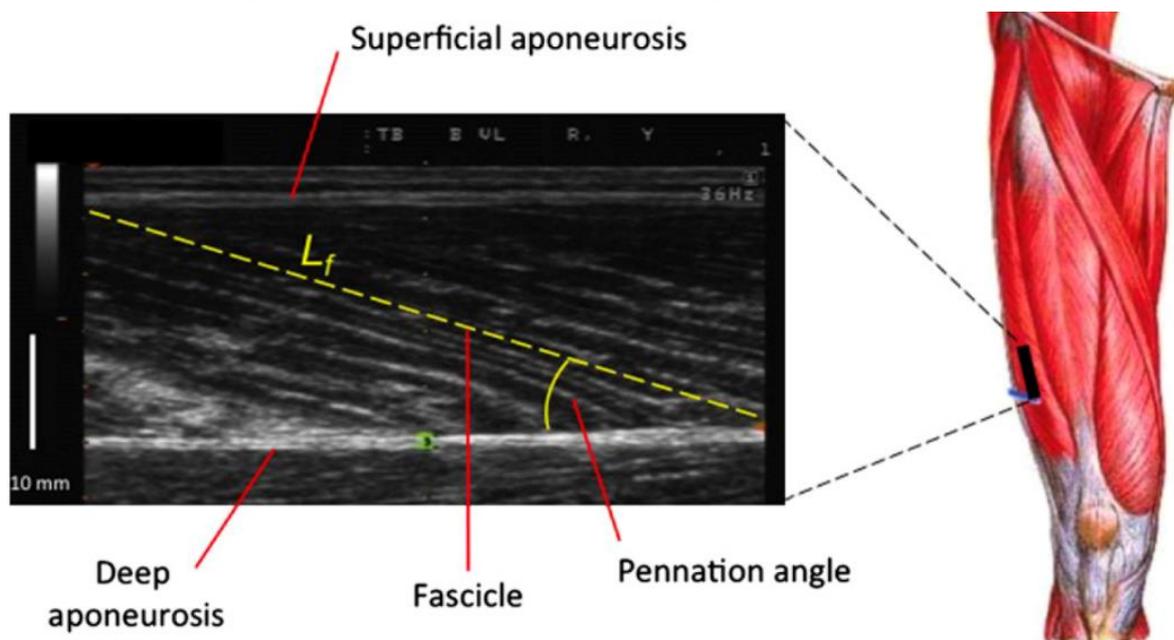


Figure 1.4; Representative longitudinal B-mode Ultrasound image of Vastus lateralis (VL) muscle architecture. Yellow dotted line denotes the length of a muscle fascicle (L_f). (Adapted from Franchi et al, 2014).

1.4.3.3 Structural remodelling; muscle fibre length

A second structural parameter that is believed to undergo adaptation following strength training is the length of muscle fascicles (fibres), which can have a profound effect upon muscle movement range, maximum shortening velocity (and subsequently muscle power), and the force-length relationship (Blazeovich et al., 2007). Muscle fibre length changes have predominantly been observed following eccentric strength training interventions (Reeves et al., 2009) supporting the preferential addition of sarcomeres in series following lengthening contractions (Franchi et al., 2014). This is further supported by a right-ward shift in the force-length relationship of a muscle being shown after periods of eccentric strength training (Franchi et al., 2017). In addition to contraction mode being a stimulus for increasing in-series sarcomere number, the use of large muscle movement ranges during training (Kumagai et al., 2000), and high movement velocity during training (Blazeovich et al., 2003) are also considered influential. The addition of sarcomeres in series (leading to increased fascicle length) appears to be mediated by similar mechano-transduction pathways as to those involved in other aspects of muscle hypertrophy (Burkholder, 2007); however in the instance of sarcomere series lengthening, the stimulus may also come from passive elements such as Titin, rather than active elements like actin and myosin (Franchi et al., 2018b). Thus, through mechano-transduction pathways, muscle protein synthesis results in the addition of new sarcomeres in series and in parallel; however the direction of muscle growth – the differential addition of sarcomeres in series or in parallel – depends on the mode of muscle contraction employed during training (Franchi et al., 2014).

1.4.3.4 Structural remodelling: Skeletal muscle Fibre Type

As previously mentioned, changes in the proportion of muscle fibre types is another possible adaptive response to strength training (Andersen and Aagaard, 2000). Specifically focusing on the myosin heavy chain (MHC) isoforms expressed within human skeletal muscle, it has been observed that strength training can induced increases in MHC IIA (fast, oxidative) and reduce the expression of MHC IIX (fastest, glycolytic) (Hather et al., 1991; Andersen and Aagaard, 2000; Folland and Williams, 2007). Interestingly, this removal of MHC IIX seems counterintuitive as this isoform has the highest power production, so in theory its removal would cause slowing and reduced muscle power output (Andersen and Aagaard, 2010). However, when considering the whole intact muscle, this inherent slowing is out-weighed by the training-induced increases in contractile strength, power and RFD. Considering training-induced increases of the aforementioned parameters alongside preferential hypertrophy of MHC-IIA and MHC-IIX fibres, a larger portion of the PCSA will be subsequently occupied by 'fast' fibres (Aagaard et al., 2001; Kosek et al., 2006). The increased proportion of fast

muscle fibre types gives rise higher muscular power and RFD following strength training and will ultimately contribute to improve performance of related sport-skills.

Throughout sections 1.3 and 1.4, the various areas of the neuromuscular system have been discussed, highlighting their importance for study within the context of increasing strength through adaptation. From looking at how cortical changes can affect the descending pathways and associated MUs, it seems intuitive that integrated assessment of such areas may provide further insight into their overall adaptations. Equally, current literature still contends the extent of contribution to increased strength, played by the neural and physiological changes highlighted above. Therefore, if an integrated approach to assessment were to be considered, it could not stop short of measuring corresponding changes in muscle physiology.

1.5 Measuring adaptations to strength training

1.5.1. Electromyography

As previously mentioned, subtle changes in the nervous system are believed to account for increases in muscular strength, as strength increases have been observed in the absence of muscle hypertrophy (Narici et al., 1989). Early evidence from Hakkinen and Komi (1983) found associations between these early-increases in muscular strength and an increased ability to activate the contributory muscles. This association was made through the use of sEMG, which provides information on the degree of muscle activation based on changes in electrical current induced by muscle fibre action potentials (Farina et al., 2016). Being considered as representative of neural drive, an increase in the amplitude of sEMG signals is interpreted as increased efferent drive following strength training (Aagaard, 2003). In addition to quantifying changes in neural drive induced by strength training, sEMG is commonly employed to build muscle activation ‘profiles’ of specific strength exercises (Clark et al., 2012; Neto et al., 2019). Such profiles can be useful for practitioners and athletes when considering the principles of training specificity for optimal strength transfer from training to athletic performance, or during rehabilitation.

In addition to sEMG providing an overview of efferent drive, intramuscular EMG (using indwelling electrodes) can be used to infer more specific information regarding firing patterns of individual MUs (Christie et al., 2009). Whilst strong relationships have been observed between the two forms of EMG (Waite et al., 2010; Allen et al., 2013), intramuscular EMG only allows inferences to be made

upon a small number of MUs. Technological advancements however now allow investigators to decrypt MU activity from large MU pools by extracting the MU action potential trains (MUAPTs) from the sEMG signal. A primary method now used in research for the decomposition of MU action potential (MUAP) trains is known as decomposition EMG (dEMG) (De Luca et al., 1982; Adam and De Luca, 2005; De Luca et al., 2006; De Luca et al., 2015a; Contessa et al., 2016).

1.5.1.1 Decomposition electromyography

Decomposition of sEMG signals to discern MUAPTs can be achieved through the use of a single, 4-channel surface sensor (De Luca et al., 1982; Adam and De Luca, 2005; De Luca et al., 2006) which uses a specific decomposition algorithm (Nawab et al., 2010); to determine an EMG signal's constituent MUAP trains (Figure 1.5). Present day algorithms are capable of identifying more than 5 MUAP trains per contraction; the number that could be previously studied with intramuscular electrode technique (De Luca et al., 2015a). The unique dEMG system provides assessments of MU firing rates and action potential amplitudes (Nawab et al., 2010) and allows evaluation of MU properties within the recruitment pool in relation to their recruitment thresholds during muscular contractions (De Luca and Contessa, 2012). Validation of dEMG against intramuscular EMG recording was recently reviewed by Enoka (2019) who concluding that decomposition methods were capable of replicating findings derived from intra-muscular EMG studies. Specific validation of the algorithm used by the single, 4-channel sensor (Delsys, Inc) has also been discussed in depth by De Luca et al. (2015b), with successful comparisons recently drawn against separate techniques (Hernandez-Sarabia et al., 2020). Whilst the aforementioned Delsys system allows for accurate decomposition of MUAPs, it would be remiss not to mention that other systems such as high-density (HD) EMG have been used in literature to improve practitioner understanding of MU physiology. Such systems may allow for a larger sampling area of MUs from a particular muscle due to their grid-based electrode design (Martinez-Valdes et al., 2016); however such electrode designs would not allow for potential application during dynamic movements, as movement from the grid can have substantial negative impact upon signal-noise ratios and overall signal quality.

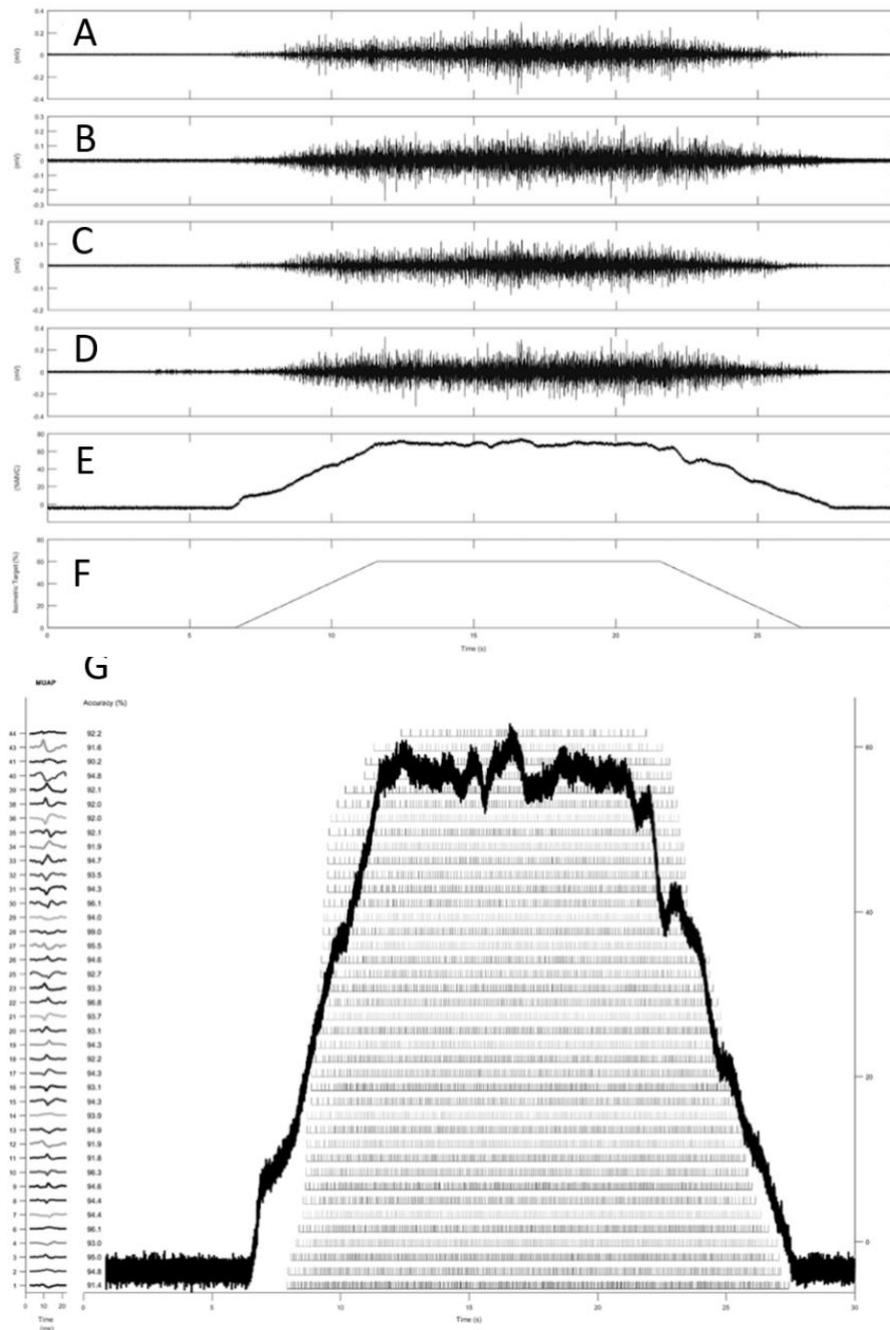


Figure 1.5 Example of raw sEMG signal collected by a 4-channel surface electrode (A-D) during an isometric trapezoid contraction for which force output (Nm) (E) and target trace (%MVC) (F) are shown. G; Example of motor unit firing rate bar plot, with each vertical bar representing a single motor unit firing. Force output of the trapezoid contraction is overlaid. Motor unit action potential shapes for each identified motor unit are plotted down the y-axis, along with the accuracy results (%) of each MU. (adapted from Macgregor et al, 2018).

Decomposition EMG has been demonstrated useful when investigating MU adaptations to different strength training strategies, providing novel insights into alterations in MU firing frequency (Kamen and Knight, 2004; Pucci et al., 2006; Vila-Chã et al., 2010; Del Vecchio et al., 2019). Additionally, the use of regression MU properties (firing rate and MUAP amplitude) against MU recruitment threshold has proven insightful into MU hypertrophy adaptations following strength training (Pope et al.,

2016;Sterczala et al., 2020), and in explaining sex-related differences in muscle CSA (Trevino et al., 2019) and muscular strength and power (Herda et al., 2020). To date, strength training studies employing dEMG assessments have employed both isometric and dynamic training interventions, yet MU properties are only assessed during isometric or low-level ballistic contractions (Del Vecchio et al., 2019;Sterczala et al., 2020); representing a potential limitation as to the task-specific nature of exercise testing.

1.5.2 Transcranial magnetic stimulation

So far, we have identified cortical and corticospinal plasticity as a key area of adaptation to strength training, with adaptations in both excitatory and inhibitory networks shown prevalent (Kidgell et al., 2017;Siddique et al., 2020). TMS is considered one of the most robust methods of assessing the function of the cortico-cortical circuitry of the M1, overcoming limitations of earlier monosynaptic spinal-reflex assessments (Siddique et al., 2020).

TMS involves the placement of a magnetic coil on the scalp generating a magnetic field impulse over the M1 (Figure 1.6A). By stimulating the M1 with a magnetic field, multiple descending volleys are elicited from corticospinal axons to activate alpha-motoneurons, causing an MEP to occur within a target muscle (figure 1.6B). MT refers to the lowest intensity of TMS stimulus required to elicit a MEP within a muscle. MT is thought to represent the membrane excitability of interneurons and corticospinal neurons within the motor cortex, as well as excitability of the spinal tract neurons, neuromuscular junction and the target muscle itself (Ziemann et al., 1996;Kobayashi and Pascual-Leone, 2003).

MEP amplitude is considered a measure of corticospinal excitability and is recorded through surface electromyography (sEMG). MEP amplitude exhibits inherent variability, both inter- and intra-subject, making interpretation of raw MEP values difficult. Thus it has become common practice to express MEP amplitude as a percentage of maximal excitability of a muscle (M-wave or M_{max}), which is assessed through peripheral nerve stimulation, and commonly used to reflect the extent of the MU pool which TMS is able to activate (Goodall et al., 2014). When MEPs are recorded during maximal voluntary activation (MVC) there is an interruption in volitional drive from the M1; this interruption, reflected as a the cSP in the sEMG signal (figure 1.6B), is considered a measure of corticospinal inhibition (Kidgell et al., 2017). The cSP is mediated by the neurotransmitter GABA and its associated type-B receptor (GABA_b).

Both excitability and inhibition parameters have demonstrated changes following short periods of resistance training (table 1), thus demonstrating the effectiveness of the technique in determining the location adaptations within the cortical and corticospinal circuitries. Indeed, two recent meta-analysis have confirmed the substantial cortical and subcortical adaptations take place following strength training in both the excitatory and inhibitory networks, ultimately functioning to increase motor neuron activation and subsequent muscular strength (Kidgell et al., 2017; Siddique et al., 2020).

The aforementioned parameters of excitability (MEP amplitude) and inhibition (cSP length) are obtained through 'single-pulse' TMS and are unable to infer upon intracortical micro-circuits of the M1 specifically (Carroll et al., 2002). 'Paired-pulse' TMS involves the use of a conditioning stimulus and a test stimulus to assess the intrinsic intracortical connections within the M1 (Kidgell et al., 2017), however does require two-stimulators. Depending on the inter-stimulus interval employed intracortical inhibitory (2-5ms) and long intracortical inhibitory (100-150ms) circuits can be examined, providing information on responses of the GABA-ergic system to strength training (Kidgell et al., 2017). In addition to delivering TMS stimulations over the M1 to elicit MEPs in the target muscle, stimulations can also be applied over the cervico-medullary junction, subcortically. As cervico-medullary stimulation does not involve the circuitries within the M1, cervico-medullary evoked potentials (CMEPs) regarded as a measure of spinal excitability (Nuzzo et al., 2017). Comparison between MEP and CMEP amplitudes allows researchers to determine whether changes in excitability occur at the cortical and/or the spinal level.

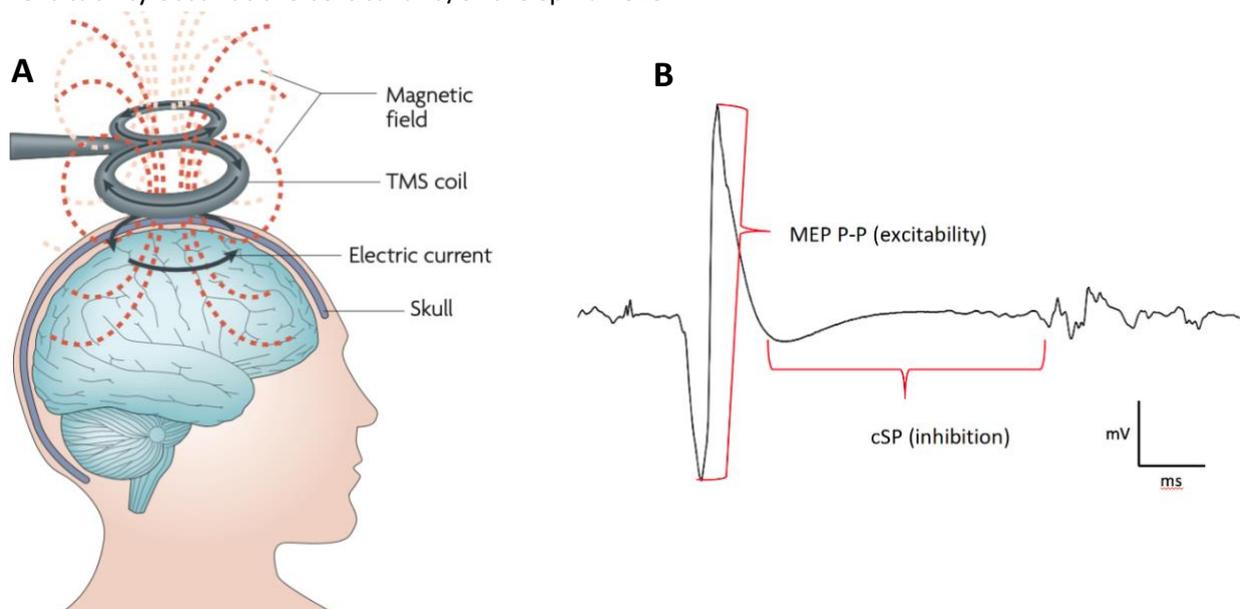


Figure 1.6 Illustration of the electromagnetic principle upon which TMS is based (A). The magnetic field generated from the TMS coil induces an electrical current which activated the underlying neuronal pool. A representative sEMG trace of a TMS motor evoked potential (MEP) in a target muscle (B). The peak-to-peak MEP amplitude represents the excitability parameter, whilst the duration of the cortical silent period (cSP) represents the level of corticomotor inhibition (adapted from Ridding and Rothwell, 2007).

Table 1.1; studies assessing cortical and cortico-spinal responses to strength training interventions. M male, f female, n sample size, MEP motor evoked potential, cSP corticospinal silent period, SICI short-interval cortical inhibition, AURC area under recruitment curve, ↑ increase, ↓ decrease, = no change.

STUDY	Sample	Training	Dependent variable(s)	Key measure(s)	Findings
(Carroll et al., 2009)	17 untrained healthy (11M & 6F, 19-35 years); Trained (n=8), control (n=9)	4-weeks isotonic (3x per week) of the wrist muscles	Strength Corticospinal excitability	Maximum strength MEP amplitude	↑ strength = MEP amplitude
(Lee et al., 2009)	23 untrained healthy (16M & 7F, 18-51 years); Trained (n=12), control (n=11)	4-weeks isotonic (3x per week) of wrist abductors	Strength Corticospinal excitability Voluntary activation	Maximum strength MEP amplitude Cortical voluntary activation	↑ strength = MEP amplitude = voluntary activation
(Kidgell and Pearce, 2010)	23 untrained healthy (11M & 12F, 18-30 years); Trained (n=13), control (n=10)	4-weeks isometric (3x per week) of biceps brachii	Strength Corticospinal excitability	Maximum strength MEP threshold & amplitude	↑ strength ↑ MEP amplitude
(Leung et al., 2017) (non-paced)	22 untrained health (12M & 10F, 20-35 years); Trained (n=11), control (n=11)	4-weeks isotonic (3x per week) of biceps brachii	Strength Corticospinal excitability Intracortical inhibition	Maximum strength Corticospinal excitability (AURC) SICI	↑ strength ↑ MEP AURC = SICI
(Leung et al., 2017) (metronome paced)	22 untrained health (12M & 10F, 20-35 years); Trained (n=11), control (n=11)	4-weeks isotonic (3x per week) of biceps brachii, paced to a metronome	Strength Corticospinal excitability Intracortical inhibition	Maximum strength Corticospinal excitability (AURC) SICI	↑ strength ↑ MEP AURC ↓ SICI
(Mason et al., 2017)	20 untrained healthy (10M & 10F, 18-35 years); Trained (n=10), control (n=10)	3-weeks isotonic (3x per week) of biceps brachii	Strength Corticospinal excitability Corticospinal inhibition	Maximum strength Corticospinal excitability (AURC) cSP duration (AURC)	↑ strength ↑ MEP AURC ↓ cSP AURC
(Christie and Kamen, 2004)	30 untrained healthy (21.9 ± 3.1 years) Trained (n=15), control (n=15)	2-weeks isometric (3x per week) of tibialis anterior muscle	Strength Corticospinal excitability Corticospinal inhibition	Maximal strength MEP amplitude cSP duration	↑ strength ↓ MEP amplitude ↓ cSP duration
(Coombs et al., 2016) (left-handed study)	15 Untrained health (7M & 8F, 18-30 years); Training (n=8, control (n=7)	3-weeks isotonic (3x per week) of wrist extensors	Strength Corticospinal excitability Corticospinal inhibition Intracortical inhibition	Maximal strength MEP amplitude cSP duration SICI duration	↑ strength = MEP amplitude ↓ cSP duration = SICI duration

(Table 1 continued)

(Coombs et al., 2016) (right-handed study)	15 untrained health (7m & 8f, 18-30 years); training (n=8, control (n=7)	3-weeks isotonic (3x per week) of wrist extensors	Strength Corticospinal excitability Corticospinal inhibition Intracortical inhibition	Maximal strength MEP amplitude cSP duration SICI duration	↑ strength = MEP amplitude ↓ cSP duration = SICI duration
(Kidgell and Pearce, 2010)	16 untrained health (13m & 3f, 18-30 years); trained (n=8), control (n=8)	4-weeks isometric (3x per week) of first dorsal interosseous	Strength Corticospinal excitability Corticospinal inhibition	Maximal strength MEP amplitude cSP duration	↑ strength ↑ MEP amplitude ↓ cSP duration
(Goodwill et al., 2012)	14 untrained healthy (7M & 7F, 18-25 years); Trained (n=7), control (n=7)	3-weeks isotonic (3x per week) of the rectus femoris	Strength Corticospinal excitability Intracortical inhibition	Maximum strength MEP amplitude SICI duration	↑ strength ↑ MEP amplitude ↓ SICI duration
(Weier et al., 2012)	12 untrained healthy (20-23 years); Trained (n=6), control (n=6)	4-weeks isotonic (3x per week) of rectus femoris	Strength Corticospinal excitability Intracortical inhibition	Maximal strength MEP amplitude SICI duration	↑ strength ↑ MEP amplitude ↓ SICI duration
(Griffen and Cafarelli, 2007)	20 untrained healthy (19M & 1F, 18-32 years); Trained (n=10), control (n=10)	4-weeks isometric (3x per week) of tibialis anterior muscle	Strength Corticospinal excitability	Maximum strength MEP amplitude	↑ strength ↑ MEP amplitude.
(Hendy and Kidgell, 2013)	20 untrained healthy (12M & 8F, 22-29 years); Trained (n=10), control (n=10)	3-weeks isotonic (3x per week) of wrist extensors	Strength Corticospinal excitability Corticospinal inhibition Intracortical inhibition	Maximal strength MEP amplitude cSP duration SICI duration	↑ strength ↓ MEP amplitude ↓ cSP duration ↓ SICI duration
(Kidgell et al., 2011)	23 untrained healthy (11M & 12F, 18-30 years); Trained (n=13), control (n=10)	4-weeks isometric (3x per week) of biceps brachii	Strength Corticospinal inhibition	Maximal strength cSP duration	↑ strength ↓ cSP duration
(Latella et al., 2012)	18 untrained healthy (14M & 4F, 18-35 years)	4-weeks isotonic (3x per week) of rectus femoris	Strength Corticospinal excitability Corticospinal inhibition	Maximal strength MEP amplitude cSP duration	↑ strength ↓ MEP amplitude ↓ cSP duration
(Leung et al., 2013a)	12 untrained healthy (3M & 12F, 21-35 years); Trained (n=6), control (n=6)	3-weeks isotonic (3x per week) of the biceps brachii	Strength Corticospinal excitability	Maximal strength MEP amplitude	↑ strength ↑ MEP amplitude
(Pearce et al., 2013)	19 untrained healthy (9M & 10F, 18-35 years); Trained (n=9), control (n=10)	4-weeks isotonic (3x per week) of the biceps brachii	Strength Corticospinal excitability	Maximal strength MEP amplitude	↑ strength ↑ MEP amplitude

1.5.3 Ultrasound imaging of muscle architecture

Quantification of muscle architecture is often obtained using two-dimensional (2D) brightness mode (B-mode) ultrasound imaging, in a wide range of health, aging, injury and activity states (Van Hooren et al., 2020). Magnetic resonance imaging (MRI) holds the advantage of high spatial resolution and the ability to measure large areas of muscle tissue simultaneously (Lieber and Ward, 2011); this being extremely useful when considering heterogeneity of architecture within some muscle (Shin et al., 2010). However, MRI is expensive and time-consuming, and requires the person being scanned to remain in a fixed position, limiting the measurement of muscle architecture of certain muscles depending on position (Narici et al., 1996; Kwah et al., 2013). Ultrasound imaging provides a much less expensive, faster, and easier to operate procedure, with innovations allowing measurement of various parameters during active contraction (Van Hooren et al., 2020).

Ultrasound imaging uses sound waves with frequencies above the range of human hearing (typically ~2-15 megahertz (MHz)), which are produced by probes/transducers which are placed on the skin. Typically, a water-soluble gel is used as a medium between the skin and transducer to improve sound wave transmission. The ultrasound waves emitted from the transducer are reflected back to it when they rebound off of boundaries between tissues in the path of the soundwaves. The speed of the ultrasound waves and time taken to echo back to the transducer are used to calculate the distance from transducer to tissue boundary, with such distances used to generate 2D images of muscle tissue (Chan and Perlas, 2011). Structures identified within scanned tissues are reflected based on their density; denser structures such as bone and connective tissue appear as brighter structures within an ultrasound image, compared with soft tissue which will show up darker. These images are then captured by appropriate software, typically for offline analysis to obtain the desired parameter(s).

In the case of muscle architecture, ultrasound images can be taken in either the longitudinal plane of a muscle or the transverse plane, both of which can yield different parameters. Transverse images (figure 1.7 A&B) can be used to measure cross-sectional parameters of skeletal muscle and subcutaneous tissue, such as muscle CSA, muscle thickness, and subcutaneous fat thickness (Blazevich et al., 2003). As previously mentioned, muscle CSA is associated with force production capacity of skeletal muscle, although is not only reflective of contractile material; but also reflects the interstitial space between muscle fibres. Thus, muscle CSA is made up of two components; ACSA (CS of a muscle perpendicular to its longitudinal axis) and PCSA (CS of a muscle perpendicular to its

muscle fibres) (Formenti et al., 2019). Muscle CSA can be measured in transverse imaging through outlining the muscle fascia (figure 1.7A). Muscle thickness is another easily identified measure of muscle as the distance between the upper and lower fascia (Figure 1.7B). Recently it was confirmed that muscle thickness is associated with muscle CSA ($r=0.69$) in the assessment of muscle architecture adaptations to strength training (Franchi et al., 2017), demonstrating its use in inferring hypertrophic adaptations using ultrasound imaging.

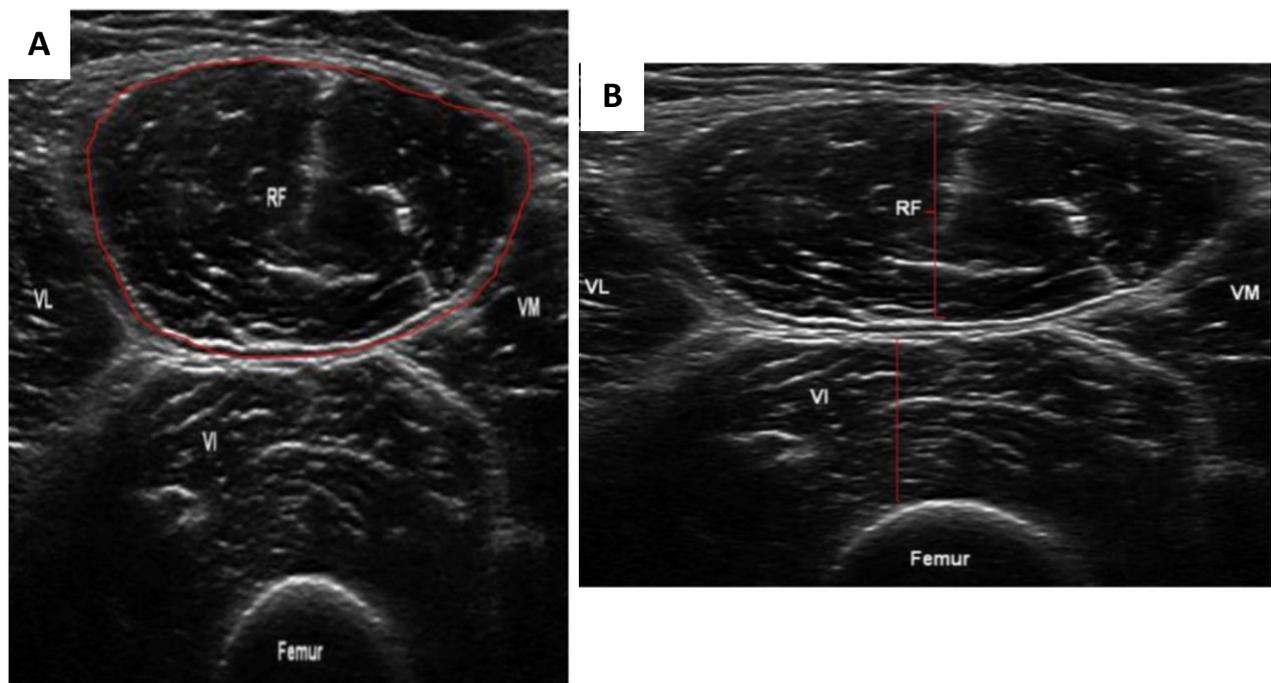


Figure 1.7; *Transverse ultrasound image showing rectus femoris RF cross-sectional area perpendicular to its longitudinal axis (red outline) (A). Muscle thickness measures (red vertical lines) of the rectus femoris and vastus intermedius are also shown (B) (adapted from Formenti et al., 2019).*

Longitudinal images are used to visualise the aponeurosis and fascicles of a muscle (Figure 1.8). From such an image, the angle of fascicle orientation to the aponeurosis can be measured and is termed the angle of fibre pennation or ‘pennation angle’ (Chan and Perlas, 2011). Based on the geometric model of pennate muscle proposed by Haxton (1944) it was later confirmed that the efficiency of force transmission from muscle to the line of pull, that is to say the tendon, was related to the angle of fibre pennation (Alexander and Vernon, 1975). Within human muscle, variations exist between different muscles as to the degree of pennation angle, and the number of angles (unipennate e.g. vastus lateralis (VL); bi-pennate e.g. rectus femoris (RF); multi-pennate e.g. deltoids) (Ema et al., 2013b). A second parameter which can be obtained from a longitudinal image is muscle fascicle

length. This can be done by measuring the path of a muscle fascicle between the upper and lower aponeurosis (figure 1.8) and is related to maximal shortening velocity and contraction speed of a muscle as it represents the number of sarcomeres in series (Bodine et al., 1982). Whilst a useful measure, it is not always possible to visualise a full fascicle within the aperture window of an ultrasound probe (Kwah et al., 2013). This has led to researchers employing trigonometry extrapolations in order to calculate fascicle length (Blazevich et al., 2006; Li et al., 2007), which may not be representative of true fascicle length as muscle fascicles are known to not run in true linear fashion (Brennan et al., 2017). However technological advancements have led to the development of extended field-of-view transducers, providing a much greater aperture window and therefore a potentially more reliable quantification of fascicle length (Adkins et al., 2017).

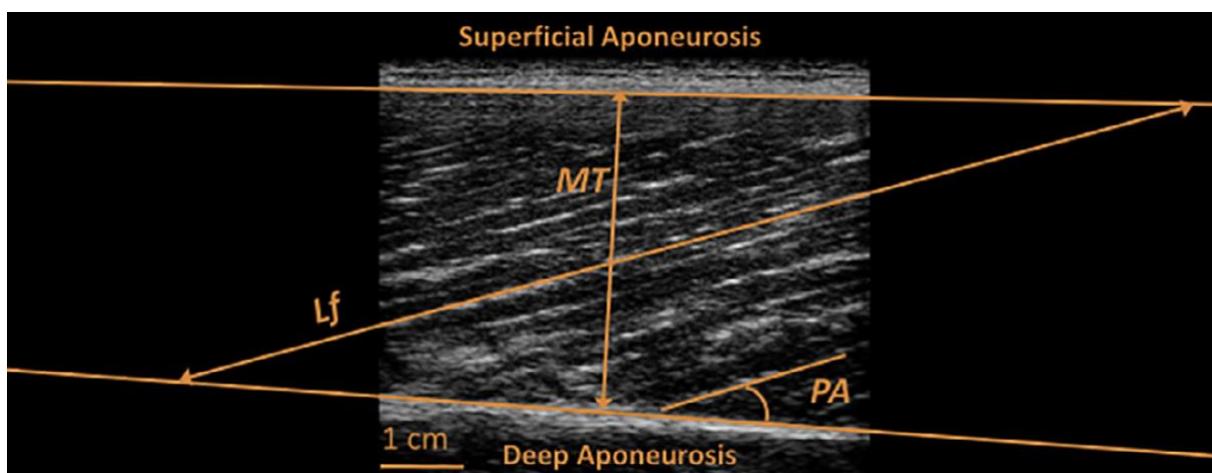


Figure 1.8; *Longitudinal ultrasound image showing vastus lateralis muscle architecture.* Horizontal orange lines denote extended upper and lower aponeurosis for fascicle length (**Lf**) extrapolation. Pennation angle (**PA**) is shown as the fascicle angle orientated with the deep aponeurosis. Muscle thickness (**MT**) is also shown for reference (adapted from Franchi et al, 2018).

1.5.3.1 Validity and reliability of Ultrasound imaging

Reliability of ultrasound imaging has received substantial focus within the literature owing to practitioner technique being the source of greatest measurement error (Kwah et al., 2013). Methodological considerations such as transducer orientation, angle of orientation to the skin-surface, pressure exerted upon the skin-surface, as well as imaging parameters such as gain and brightness must all be strictly controlled and replicated during each assessment (Kwah et al., 2013; Van Hooren et al., 2020). The reliability of muscle architecture measures obtained using B-mode ultrasound has been discussed at length previously (Kwah et al., 2013; Nijholt et al., 2017). Briefly, Kwah et al found that overall reliability for pennation angle measured in a rested state was moderate to very high (ICC 0.62-0.100, CV 0.0-8.5%), with between session reliability also moderate

to very high (ICC 0.51-1.00, CV 2.1-13.5%). The author also observed high reliability for muscles imaged in the quadriceps (ICC 0.51-1.00, CV 0.0-7.5%), indicating that pennation angle was a reliable measure obtained from ultrasound imaging. Similarly, muscle thickness measurements have also shown good-high overall reliability (ICC 0.72-1.00, CV 0.9 –6.7%), and good-high between-session reliability (0.83-0.99, CV 2.1-6.5%). Additionally, muscle thickness measures have been correlated with corresponding measures from MRI ($r=0.96-0.97$) and CT ($r=0.98$) (Dupont et al., 2001). The reliability of ultrasound-imaged muscle architecture parameters has been further analysed in healthy (Van Hooren et al., 2020), clinical (Pardo et al., 2018) and aged (Nijholt et al., 2017) populations. Thus, ultrasound imaging is a reliable, valid alternative to the gold standard of MRI imaging to assess muscle architecture. In the context of strength training, it has been shown effective at quantifying intervention adaptations, and provided detailed insight when combined with other measures of neuromuscular physiology. However, due to the potential for practitioner error to have a significant influence on measurement and analysis reliability, extreme care must be taken to ensure all assessment variables are repeated when conducting longitudinal assessments. Thus, the findings from intervention-based studies mentioned above, can be applied to our understanding of relationship between individual muscle structure and its functional capacity.

1.5.4 Tensiomyography

Measurement of an individual muscle's intrinsic contractile properties has typically used electrically evoked-twitch responses to characterise mechanical properties and, infer upon its contractile capacity. A single electrical stimulus can be applied to either the motor nerve (Regina Dias Da Silva et al., 2015) or a motor point, identified on the surface of a muscle (Orizio et al., 2016). An alternative non-invasive technique called Tensiomyography (TMG), measures time and displacement parameters of radial muscle deformation following a twitch-stimulus. TMG has seen increasing use in investigating muscle contractile properties in recent years within both clinical, and sporting settings (Martín-Rodríguez et al., 2017; Macgregor et al., 2018; García-García et al., 2019). TMG's high precision ($4\mu\text{m}$) digital displacement sensor is applied to the surface of muscle belly with a controlled pre-tension pressure ($0.015\text{N}/\text{mm}^2$) (Dahmane et al., 2001), which augments the twitch response thereby enhancing the measurement of contractile dynamics (Križaj et al., 2008). A 1-ms wide electrical stimulus is applied to the muscle via skin-surface electrodes, inducing a muscle-twitch response, which is then recorded as a radial displacement curve (figure 1.9). Given that skeletal muscle operates at a near-constant volume (Baskin and Paolini, 1967), the inward-outward deformation of the muscle surface is referred to as radial displacement; which has been related to

the orientation muscle fibres (Kaczmarek et al., 2009) and their mechanical output (Marchetti et al., 1992).

A number of parameters can be extracted from the twitch displacement-time curves (figure 1.9). Peak radial displacement of the muscle belly (D_m) which is interpreted as a measure of muscle stiffness/tension (Pišot et al., 2008). Contraction time (T_c) is the time taken to ascend between 10% and 90% of D_m , therefore reflecting the speed of twitch force generation. The length of T_c has been correlated with proportions of type-I muscle fibres (Dahmane et al., 2001; Valencic et al., 2001; Šimunic et al., 2011), with shorter T_c of the hamstring musculature being associated with faster running speeds (Završnik et al., 2017). Delay time (T_d) represents the time between delivery of stimulus and 10% D_m , providing a measure of responsiveness (García-manso et al., 2011). Half-relaxation time (T_r) is taken as the descending time between 90% -50% D_m , providing one measure of muscle fibre fatigue status in addition to Sustain time (T_s) (the time between 50% D_m on either side of the twitch-response curve) (Tous-Fajardo et al., 2010; García-manso et al., 2011). Stimulation amplitude is variable inter-individually and between different muscle groups, with typical peak twitch response recorded between 60-100mA (Macgregor et al., 2018). Thus, a progressive incremental approach of increasing stimulation intensity is recommended in order to achieve the peak D_m (Dahmane et al., 2001).

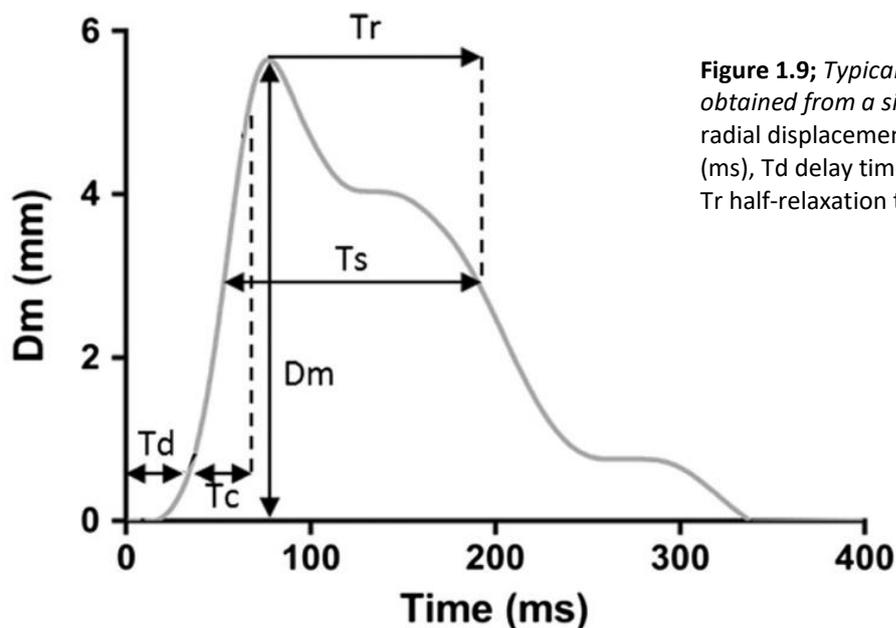


Figure 1.9; Typical displacement-time curve obtained from a single TMG evoked twitch. D_m radial displacement (mm), T_c contraction time (ms), T_d delay time (ms), T_s sustain time (ms), T_r half-relaxation time (ms).

1.5.4.1 Validity and Reliability of Tensiomyography

Validity of TMG is inherently difficult to quantify as it is an *in vivo* measure of the mechanical properties of muscle contraction. Construct validity of TMG has been determined by a number of studies by relating time TMG time parameters to the proportion of slow-twitch muscle fibres of muscles examined (Dahmane et al., 2001;Valencic et al., 2001;Dahmane et al., 2005;Šimunic et al., 2011).

The most robust of these aforementioned studies found a significant, positive correlation between the proportion of MHC-I fibres (slow, oxidative) and Tc ($r=0.88$), Td, ($r=0.61$), Tr ($r=0.67$) (Šimunic et al., 2011). Conversely, construct validation of Dm has not been determined to the same extent, again owing to the inherent difficulty of assessing muscle contractile mechanics *in vivo*. A level of construct validation for Dm can be taken from data investigating atrophy induced reductions muscle architecture parameters. The observed associations with muscle thickness and pennation angle (Pišot et al., 2008;Šimunič et al., 2019) demonstrate effective integration of measurement techniques and a mechanistic association with muscle structural remodelling. However, it should be noted the same relationships have not been established in the context of muscle hypertrophy.

Reliability of TMG has been extensively studied in a variety of conditions, both short- and long-term (table 1.2). Inter-day reliability and repeatability of TMG displacement-time curve parameters have been previously examined, with methodological parameters such as electrode size and placement (Tous-Fajardo et al., 2010). Whilst the aforementioned authors found high levels of relative, and absolute reliability, it was noted that altering the inter-electrode distance significantly impacted upon Dm; hence inter-electrode distance and transducer location must be controlled in repeated assessments (Rodríguez-Matoso et al., 2010;Wilson et al., 2018). Additionally, the interval between successive stimulations must also be controlled with <10 seconds between stimulations so as to avoid summation of action potentials (Piqueras-Sanchiz et al., 2020).

The influence of muscle status has also been the subject of reliability investigation with Dm and Tc parameters presenting the high relative and absolute reliability in lower limb muscles, when assessed in a rested state (Šimunič, 2012). However, the aforementioned condition may not be practically applicable; as such, Ditroilo et al. (2013) assessed the reliability of TMG parameters under different conditions (rested, exercised, fatigued) and their long-term (4-weeks) stability. Although reliability was observed to reduce over the 4-week interval (table 1.2), measures displayed greater reliability in the exercised or fatigue states, compared with rested state (Ditroilo et al., 2013). Thus, it has been recommended that assessments carried out using TMG should focus on Dm and Tc parameters as they appear the most stable (Benítez et al., 2013;Macgregor et al., 2018).

Table 1.2; Tensiomyography reliability. *X* – not measured, *TMG* tensiomyography, *ICC* intra-class correlation coefficient, *CV* coefficient of variation, *Dm* displacement, *Tc* contraction time, *Td* delay time, *Ts* sustain time, *Tr* half-relaxation time.

Study	Muscle	Measure	TMG parameters				
			Dm	Tc	Td	Ts	Tr
Krizaj et al, 2008	Biceps brachii	ICC (CV%)	0.98 (x)	0.97 (x)	0.94 (x)	0.89 (x)	0.86 (x)
Tous-Fajardo et al, 2010	Vastus medialis	ICC (CV%)	0.97 (4.7)	0.92 (3.4)	0.86 (2.7)	0.96 (14.2)	0.77 (2.4)
Simunic et al, 2012	Vastus lateralis	ICC (CV%)	0.99 (4.7)	0.98 (1.5)	0.89 (1.8)	0.96 (4.4)	0.89 (7.6)
	Vastus medialis		0.98 (4.7)	0.98 (2.2)	0.94 (2.8)	0.94 (4.9)	0.88 (6.4)
	Biceps femoris		0.99 (4.2)	0.98 (4.9)	0.98 (2.6)	0.95 (3.3)	0.89 (9.3)
Ditrollo et al, 2013	Gastrocnemius (medialis)	ICC (CV%)	0.86–0.95 (8.0–14.8)	0.62–0.92 (3.8–9.4)	0.56–0.62 (7.0–9.2)	0.71–0.86 (5.3–8.2)	0.67–0.79 (27.8–32.7)
De Paul Simola et al, 2016	Biceps brachii	ICC (CV%)	0.95 (10.4)	0.91 (8.7)	0.92 (2.4)	0.88 (4.9)	0.7 (20.6)
	Rectus femoris		0.92 (9.3)	0.94 (4.9)	0.87 (3.80)	0.85 (21.3)	0.86 (32.8)
	Gastrocnemius (lateralis)		0.94 (13.7)	0.93 (8.5)	0.90 (4.2)	0.87 (8.5)	0.93 (12.6)
Carrasco et al, 2011	Rectus femoris	ICC	0.92	0.83	0.89	0.90	0.88
Hunter et al, 2012	Biceps brachii	CV%	8.7 ± 3.8%	8.6 ± 3.9%	X	X	X

Even though *Tc* and *Dm* have been demonstrated as being the most stable (see previous paragraph) *Tc* should still be treated with caution as it is dependent upon *Dm*. Some researchers have sought to address this by calculating relative measures of contraction velocity (*Vc*), as *Tc* and *Dm* can alter disproportionately to each other. A number of methods to calculate *Vc* have appeared in the literature (Valenčič and Knez, 1997; García-Manso et al., 2012; de Paula Simola et al., 2015; Loturco et al., 2016; Macgregor et al., 2016); however no consensus has yet been reached as to the most appropriate method and thus further study is required to understand the most appropriate for assessing *Vc* in relation to objective markers of muscle contraction speed. As such it was not deemed appropriate to investigate such measures within this thesis.

Similar to the previously discussed ultrasound assessments of muscle architecture, the skill and experience of the practitioner also plays a significant role in the reproducibility of TMG derived contractile properties. Equally, as demonstrated by the aforementioned literature, the practical applicability of TMG means that assessment variables must be controlled in numerous environments and under many different muscle-state conditions. Therefore, in any longitudinal investigation, all reasonable steps must be taken to control the parameter of a TMG assessment.

1.5.4.2 Applications of Tensiomyography

The use of TMG within sport settings has been previously discussed (García-García et al., 2019), with particular interest surrounding demands of training and competition and how these may impact upon performance. Indeed, TMG has shown capable of detecting reduced efficiency of E-C coupling and other markers of muscular fatigue by recording prolonged Tc and reduced Dm, following local exercise-fatigue and exercise induced muscle damage protocols (Carrasco et al., 2011;García-Manso et al., 2012;Hunter et al., 2012;Macgregor et al., 2016). Thus, it appears TMG can provide useful insights into altered muscle function following local fatigue and muscle damage. TMG has also been used to assess differences in muscle mechanical properties between athletes of different training backgrounds (Loturco et al., 2015b;de Paula Simola et al., 2016;Šimunič et al., 2018); where athletes with a greater exposure to strength and power training presented shorter Tc and smaller Dm of key muscle groups, compared to athletes with greater exposure to endurance-based training.

Whilst the above-mentioned studies are cross-sectional in nature, they do present intrigue as to the potential ability of TMG to quantify changes in muscle contractile parameters following training interventions. Zubac et al. (2019) assessed TMG parameters as measures of contractile mechanics, following an 8-week training intervention using plyometric exercises in older adults. The authors observed improved counter-movement jump height and take-off velocity following the intervention, alongside shortened Tc and reduced Dm in key contributory muscles. These results echo that of a similar, earlier study conducted in younger adults (Zubac and Simunic, 2017), where the authors noted that contractile property adaptations were higher in muscles groups that experience typically lower habitual loads, demonstrating a dose-dependency. A small number of studies have employed TMG to assess within-season changes of contractile properties for specific sports (García-García et al., 2016), further demonstrating potential of the technique in a longitudinal setting. However out-with the two intervention-based studies outlined above, there is a current scarcity of data where TMG is used to quantify contractile mechanics changes following training interventions, and surroundings its integration alongside other established physiological measurement techniques.

1.6 Integrating assessments of the neuromuscular system

Outlined in the sections above, there are multiple non-invasive methodologies of quantifying neural and morphological adaptations to strength training. In isolation, these methodologies have provided great insight into the physiological pathways which they measure; however a logical step forward would be utilise them concurrently. Indeed the recent study by Sterczala et al. (2020)

concurrently measured muscle thickness via ultrasound imaging and MU properties via dEMG before and after an 8-week strength training intervention. The concurrent measurements of MU adaptations and muscle architecture allowed these authors to observe MU hypertrophy as a result of training, and to suggest this as the mechanism for increased muscular strength. A potential limitation of the aforementioned study however was that only end-point analysis was carried out; therefore, no information regarding the timeline of MU or hypertrophy adaptations within their 8-week intervention was inferred. As early strength gains (<4-weeks training (Blazevich et al., 2007;Seynnes et al., 2007;Weier et al., 2012) are believed to be predominantly neural in nature, and the contribution of muscle hypertrophy to increased muscular strength is still under debate (Loenneke et al., 2019;Taber et al., 2019). Information regarding the timeline of adaptations may provide valuable information for practitioners employing strength training interventions in athletic preparation, rehabilitation, or to combat muscle atrophy. Furthermore, similar questions remain within the timeline of neural adaptations also as concurrent measurements of spinal circuitry outputs and MU properties have not been carried out following strength training (Aagaard et al., 2020). Thus, it is not known if a modulation effect exists between the two such areas of subtle neuromuscular plasticity (Del Vecchio et al., 2019;Siddique et al., 2020).

At the other end of the neuromuscular pathway, concurrent measures are beginning to see more attention, following the recommendations of the integration of contractile mechanics assessments with other established physiological measures (Macgregor et al., 2018). As previously mentioned, TMG contractile property assessments have been combined with ultrasound imaging (Pišot et al., 2008;Šimunič et al., 2019) to infer upon longitudinal changes in muscle physiology and contractile mechanics, however such integration is yet to be applied in the context of strength training and hypertrophy. Consideration of such contractile mechanics adaptations in the case of strength gain would address a lack of intervention-based data (Macgregor et al., 2018), and may shed further light on muscle architecture associations, especially if assessed alongside other areas of the neuromuscular pathway. Therefore, concurrent assessments of both neurological and morphological adaptations may aid in answering some of the literary holes highlighted within this review. Furthermore, such data could aid practitioners in the designing of strength training interventions to complement athletic performance, rehabilitation following injury, and ultimately contribute to overall confidence in a strength training interventions' use.

1.7 Conclusion and aims

Practitioner decisions regarding the implementation of strength training interventions require high levels of confidence as to their efficacy in eliciting transferable increases in strength. In order to be sure of an intervention's efficacy, inferential methods used in determining outcomes should provide information regarding the confidence in observing meaningful increases in strength. As traditional frequentist approaches are unable to provide such inferential confidence, there is a need for an alternative approach. An inferential method capable of quantifying effect magnitude and its associated uncertainty, combined with the implementation of a user-defined threshold of meaningful response, would enable more effective interventions to be designed for improving athletic performance and in rehabilitation settings.

Whilst quantifying the outcome of a strength training intervention through a transferable performance test provides valuable information to a practitioner, information regarding the specific mechanisms of physiological adaptation leading to such an outcome, is of equal benefit. With technological advancements now enabling practitioners to assess specific regions of the neuromuscular system in isolation, we can gain a much clearer understanding of training induced adaptations. Spinal and supra-spinal regions, as well as peripheral regions of the MU and contractile elements, all play a role in muscle contraction and the degree of force production, as has been demonstrated in the literature. In particular the study of skeletal muscle contractile properties has received attention in areas such as exercise fatigue and differentiating between athletic background, however; there has been scarce application in the context of strength training. One potential reason for the aforementioned scarcity may be the lack of construct validation for contractile property assessments, and the physiological basis for differences within skeletal muscle. Additionally, there appears a scarcity of data surrounding the integration of assessing the aforementioned regions, meaning further clarity is needed upon the inter-play of adaptive regions within the neuromuscular system, and their respective adaptation timelines. By determining the adaptive responses of the aforementioned regions, in relation to each other, practitioners and athletes would be able to design training interventions of greater efficacy, and further improve confidence in the interventions employed. The non-invasive assessment techniques identified in this chapter possess the capability of providing an integrated approach of assessing multiple regions of the neuromuscular system, particularly as they allow for practical relevance to be maintained. However, the practical application within the context of sporting performance adaptations for some of the identified assessment techniques (e.g. contractile mechanics assessments) is yet to be determined. Furthermore, other identified techniques have been demonstrated effective at quantifying strength adaptations, such as

dEMG; yet discrepancies remain within the literature as to their findings. In furthering our understanding of training-induced adaptations, it is important that task/training-specificity is considered when utilising assessment techniques such as dEMG in order to accurately reflect the transference of strength gains to sporting performance. By utilising task-specific assessments, confidence in the interventions employed can be further solidified and provide clear, objective reasoning to a coaches' programming decisions for training or rehabilitation.

The overall aim of this thesis is therefore to determine the role played by skeletal muscle contractile properties in adaptations to strength training, in relation to other peripheral and central regions of the neuromuscular system. This thesis aims to contribute to the literary gap of integrated neuromuscular assessment and outline a practically applicable method of inferencing within the context of strength training, in order to contribute to the confidence levels with which practitioners employ specific strength training interventions. The use of TMG in assessing contractile mechanics adaptations, following strength training interventions, will be investigated. The concurrent assessment of morphological and neurological adaptations to strength training will be examined, in order to determine the value peripheral contractile function assessments alongside established measurement techniques. The applicability of assessing peripheral contractile function characteristics will also be evaluated. In order to accomplish this, the experimental objectives of this thesis are;

- To determine if TMG can be used to assess contractile mechanics adaptations in individual muscles following strength training interventions, by relating to measures of muscle architecture.

- To determine the time-course and relationship of contractile mechanics adaptations, in relation to established neural and morphological adaptations, following strength training.

- To determine if dEMG can be applied to a dynamic, training/task-specific performance test, to infer upon peripheral MU adaptations following a strength training intervention

- To determine if Bayesian inferencing can provide an insightful alternative method of assessing improvements in a physical performance test; and in turn, determine training exercise efficacy following different modes of strength training.

Chapter 2

Tensiomyography derived parameters reflect skeletal muscle architectural adaptations following 6-weeks of lower body strength training.

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2.1 Abstract

Introduction: Measurement of muscle specific contractile properties in response to strength training can provide practitioners valuable information regarding physiological status of individuals. Field based measurements of such contractile properties within specific muscle groups, could be beneficial when monitoring efficacy of training or rehabilitation interventions. Tensiomyography (TMG) quantifies contractile properties of individual muscles via an electrically stimulated twitch contraction and may serve as a viable option in the aforementioned applications. Thus, aims of this study were; (i) to investigate the potential use of TMG to quantify training adaptations and differences, in response to exercise specific lower limb RT; and (ii) investigate any associations between TMG parameters and accompanying muscle architectural measures. **Methods:** Non-strength trained male participants ($n=33$) were randomly assigned to 1 of 3 single-exercise intervention groups ($n=11$ per group); back squat (BS), deadlift (DL), or hip thrust (HT). Participants completed a 6-week linearized training programme (2x per week), where the assigned exercise was the sole method of lower body training. Pre- and post-intervention testing of maximal dynamic strength was assessed by one repetition maximum (1RM) of BS, DL and HT. Radial muscle belly displacement (Dm) and contraction time (Tc) were obtained via TMG from the Rectus Femoris (RF) and Vastus Lateralis (VL) pre- and post-intervention, alongside muscle architectural measures (pennation angle and muscle thickness). **Results:** All three groups displayed significant increases all 1RM strength tests ($p < 0.001$; $p\eta^2 = 0.677-0.753$). Strength increases were accompanied by significant overall increases in RF muscle thickness ($p < 0.001$, $p\eta^2 = 0.969$), and pennation angle ($p = 0.007$, $p\eta^2 = 0.220$). Additionally, an overall reduction in RF Dm ($p < 0.001$, $p\eta^2 = 0.427$) was observed. Significant negative relationships were observed between RF Dm and pennation angle ($p = 0.003$, $r = -0.36$), and with RF Dm and muscle thickness ($p < 0.001$, $r = -0.50$). **Conclusions:** These findings indicate that TMG is able to detect improved contractile properties, alongside improvements in muscle function within an untrained population. Furthermore, the observed associations between Dm and muscle architecture suggest that TMG contractile property assessments could be used to obtain information on muscle geometry.

2.2 Introduction

In the general introduction we identified methods in which contractile properties of skeletal muscles may be measured, and their adaptations which can occur following strength training; to contribute to increased force production capacity of muscle tissue. We identified TMG as potential tool for detecting such training-induced changes in skeletal muscle contractile properties, with it already having been applied within the context of performance sport (Macgregor et al., 2018). It is important however that we establish a level of construct validation and ascertain whether TMG would be suitable to detect strength training induced changes with key muscle groups for successful sporting performance.

As discussed in chapter 1, strength gains attained from appropriate strength training often transfer to improved sporting performance measures such as jump height (Fitzpatrick et al., 2019) and sprint performance (Harries et al., 2018). In order to maximise transfer of strength to performance, closed-chain strength exercises involving similar movement patterns to sporting performance are recommended (Seitz et al., 2014; Burnie et al., 2017). As such, knee extensors are a common focus of strength training owing to their involvement in athletic movement patterns (e.g., sprinting and jumping) (Jacobs et al., 1996; Simsek et al., 2016; Howard et al., 2017). Commonly, the efficacy of such strength training is commonly measured by 1 maximal repetition (1RM) tests in strength and conditioning settings (Levinger et al., 2009), providing an indication of any change observed in muscle function. Externally loaded free-weight back squats (BS) are a popular exercise held in high regard in strength training and rehabilitation. The BS is able to activate the full closed kinetic chain, requiring torque to be produced through multiple joints in the body; whilst maintaining a fixed foot position (Clark et al., 2012; Kwon et al., 2013). Performing BSs have been shown to recruit knee extensor musculature as prime movers alongside the hip, with trunk and back musculature acting in a stability/postural capacity (Caterisano et al., 2002; Schoenfeld, 2010; Marchetti et al., 2016). Conventional deadlifts (DL) are also commonly used in lower body RT. Not only does the DL elicit high activation of knee musculature, but also shares biomechanic and muscle activation similarities around the hip and knee joints with sport specific movement patterns such vertical jumping (Escamilla et al., 2002). Additionally, the hip thrust (HT) has seen increasing prevalence in strength training due to its versatility in increasing lower limb strength within different training scenarios (Collazo García et al., 2018). Electromyography profile comparisons have shown the HT able to elicit comparative knee extensor, knee flexor and hip extensor activation with the BS (Contreras et al., 2015) and the DL (Andersen et al., 2018). In particular Contreras et al (2015) showed similar levels of

VL peak activation between the HT and BS. It is on the basis of muscle activation profiles that the HT training is suggested to lead to improvements in elements of sporting performance (Contreras et al., 2017), and improved muscle function in clinical rehabilitation (Vinstrup et al., 2017). Furthermore the unique ROM and load positioning of the HT support its use as alternative to other closed-chain, lower limb strength exercises whilst still providing sufficient loading stimuli and muscle activation required to elicit adaptation (Neto et al., 2019). However, whilst training efficacy of the BS, DL and HT have been demonstrated, showing increases in knee extensor strength and force production (Thompson et al., 2015; Fitzpatrick et al., 2019), the underpinning physiological adaptations that are responsible are not fully understood. By investigating these adaptive responses within specific muscle groups such as the quadriceps, greater clarity can be provided to coaches and practitioners on the efficacy of commonly employed strength exercises for performance and rehabilitation settings.

Strength gains made through training have been observed alongside alterations within muscle morphology and architecture (Reeves et al., 2004; Alegre et al., 2006; Franchi et al., 2014; Kim et al., 2015; Nóbrega et al., 2018). Unipennate arrangement of muscles such as the VL, and bipennate arrangement of muscles such as the RF are better suited to produce higher forces than longitudinal muscle heads (Blazevich, 2006). Following periods of between 5 and 14 weeks, increases in architectural elements such as muscle thickness and angle of fibre pennation (pennation angle) are associated with improvements in force production in such aforementioned muscles (Aagaard et al., 2001; Blazevich et al., 2003). Associations between increased pennation angle and force production seen following strength training are thought to be due to increases in PCSA (Seynnes et al., 2007; Campbell et al., 2013; Vieira et al., 2018). With PCSA being the sum of the CSA of all fascicles within the muscle, increases in pennation angle (up to 45°) result in greater transmission of force to direction of pull [$(\frac{1}{1} \sin(2\theta_p))$; where θ_p is pennation angle] (Alexander and Vernon, 1975; Rutherford and Jones, 1992). Furthermore, increased pennation angle also leads to increased muscle tetanic tension, to which PCSA is directly proportional (Lieber and Fridén, 2000). Such alterations in muscle architecture following strength training could affect contractile mechanical properties of muscle tissue, which may be assessed using objective, non-invasive mechanomyography methods such as TMG.

As described in chapter 1, TMG assesses contractile properties of isolated muscle, by measuring a number of parameters in response to a twitch contraction (Valenčič and Knez, 1997). Such parameters, including Dm and Tc can be obtained quickly and with minimal input from the

participant being assessed. Tc has been previously correlated with proportions of slow twitch fibres within lower limb muscles, providing construct validation for TMG (Valencic et al., 2001; Dahmane et al., 2006; Šimunic et al., 2011). Within the literature Dm is considered to reflect muscle belly stiffness (Whitehead et al., 2001) and has been shown to alter with changes in muscle fatigue and ageing (Rusu et al., 2013; Macgregor et al., 2016). Additionally, atrophy induced changes in muscle architecture (thickness and pennation angle) are associated with increased Dm (Pišot et al., 2008; Šimunič et al., 2019); providing a level construct validation for altered Dm in the context of muscle atrophy-induced architectural changes. TMG can distinguish between muscles of different training status, with shorter Tc and smaller Dm being seen in athletes with muscle of a greater force production capacity, owing to a greater exposure to strength and power training (Loturco et al., 2015b; de Paula Simola et al., 2016; Šimunič et al., 2018). From these data, it is conceivable the association between Dm and muscle architecture could be reflected following changes in muscle hypertrophy.

With both Tc and Dm demonstrating good-excellent inter-rater and inter-day reliability (Križaj et al., 2008; Tous-Fajardo et al., 2010; Šimunič, 2012), and good long term stability under multiple conditions (Ditroilo et al., 2013); using such a twitch-torque assessment methods could be of use in monitoring rehabilitation interventions. Additionally, application of TMG in the context of training adaptations would address the previously highlighted scarcity of longitudinal TMG data, particularly surround training interventions (Macgregor et al., 2018). Furthermore, there is a requirement to integrate TMG parameters of contractile properties, namely Dm, alongside established physiological measures to strengthen its validation in longitudinal contexts.

Thus, the aims of this study were: 1) to investigate the potential use of non-invasive contractile mechanic assessments to quantify training adaptations, in response to 6 weeks of exercise specific lower limb strength training and; (2) to investigate any association between contractile parameters and accompanying muscle architectural changes within the knee extensors in order to strengthen the construct validation of Dm in the context of strength training adaptations.

We hypothesised (1) that knee extensor Dm reductions would be observed following strength training, alongside increases in muscle thickness and pennation angle. From previous literature it was hypothesised (2) that these responses would be larger following BS training, compared to the other exercises. Furthermore, we hypothesised (3) that muscle belly displacement obtained through TMG would be associated with ultrasonography measures of pennation angle and muscle thickness; thus, providing evidence to support validity of TMG's longitudinal application alongside established physiological markers.

2.3 Methods

2.3.1 Participants

Eligible participants were physically active, but non-strength trained males (recreational sports participants with >3hr/week of self-reported physical activity). To control for differing muscle strength and hypertrophy responses across different phases of the menstrual cycle (Sung et al., 2014) women were excluded from the study. All participants were required to maintain their normal exercise routines throughout the study [primarily consisted of non-contact team sports, running, cycling and swimming], to be free of musculo-skeletal injury for the previous 2 years and to complete a physical activity readiness questionnaire prior to beginning the study. An *a priori* power analysis was conducted for increases in 1RM BS strength (as a measure of dynamic muscle strength and function) ($\alpha = 0.05$; $\beta = 0.8$; Effect size: 0.4 (Zweifel et al., 2017)). It was determined a total a convenience sample of 21 participants (9 per group) was required to achieve statistical power of 0.86 for changes in 1RM BS strength (G*Power 3.1). To maximise statistical power and account for potential dropouts, a convince sample 48 voluntary participants (16 per group) was recruited for the study. Participants were randomly assigned to one of three training groups by computer generated numerical coding (Schulz et al., 2010). Of the 48 recruited participants, 10 withdrew from the study due to injuries sustained in team sports, 3 were excluded for failing to complete the training intervention (<90% attendance), and 2 were unable to complete post-intervention testing, leaving a sample of 33 participants; BS group (n=11): 179.33 \pm 5.99 cm, 79.02 \pm 17.91 kg, DL group (n=11): 180.27 \pm 6.66 cm, 78.29 \pm 6.97 kg, HT group (n=11): 182.78 \pm 5.75 cm, 81.04 \pm 11.53 kg. This study received institutional ethical approval from the University of Stirling's School of Sport Research Ethics Committee and was conducted in accordance with the Declaration of Helsinki.

2.3.2 Experimental Design

A three-group parallel, repeated measures design was implemented to investigate contractile properties and muscle architecture adaptations in response to three different strength training exercise programmes (Figure 2.1). In week 1, participants were familiarised with all testing procedures across two laboratory sessions. Session 1 included anthropomorphic measures, muscle architecture assessment and TMG contractile property assessment of the RF and VL. These data were used as the non-strength trained baseline measures, to avoid the confounding effects of the following strength assessments and were all recorded from the participants dominant limb. Pilot testing did incorporate architectural measures and TMG assessment of the biceps femoris muscle,

however due to poor ultrasound image quality and reliability, these measures were not recorded in the study.

Session 2 included familiarisation and initial performance horizontal jump testing, and of one maximal repetition (1RM) procedures for the BS, DL and HT exercises. In week 2, only the horizontal jump and 1RM session was repeated to obtain baseline performance and strength measures; baseline 1RM sessions were structured by 1RM data obtained during familiarisation, in order to control for fatigue. Participants were then informed of their allocated training groups and were familiarised with their respective exercise-specific training programme. Participants then completed a 6-week programme (2 sessions per week). Upon completion of the supervised 6-week programme, participants completed identical testing procedures to week 1.

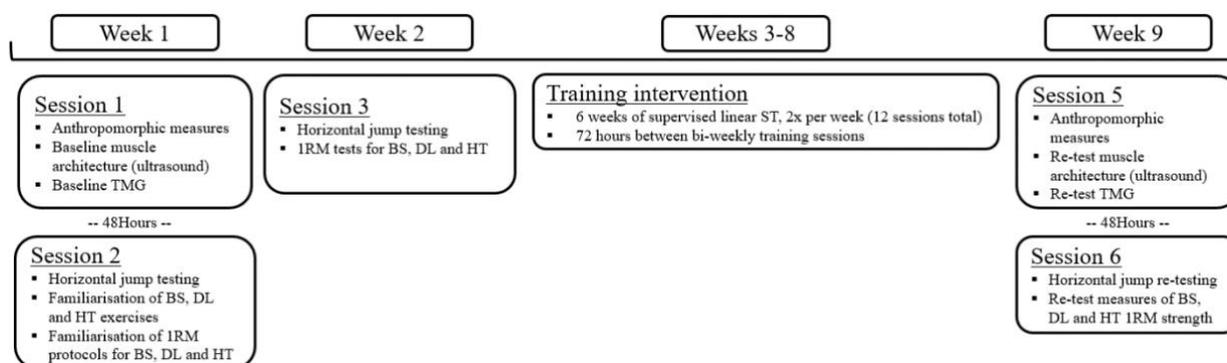


Figure 2.1; Schematic timeline of experimental design. TMG –tensiomyography; BS- back squat; DL – deadlift; HT – hip thrust; 1RM - one maximal repetition; ST – strength training.

2.3.3 Ultrasound assessment

Upon arrival participants were asked to rest quietly for 10 minutes on a bed, in a supine position to account for any redistribution of body fluids. Participants remained in the supine position for the duration of the Ultrasound examination. This position was maintained for TMG assessment (with addition of the custom angled pillow under the investigated leg). The measurement sites of the VL and RF muscles were marked and recorded according to anatomical landmarks used in the literature (Blazevich, 2006). Briefly, images of VL were taken at 36% (distal) of the distance between the superior border of the patella and the anterior superior iliac spin. Images of the RF were taken at 57% of the distance between the superior edge of the patella and the anterior superior iliac spine. The RF measurement site was chosen as the mid-point of the muscle belly, previously showing higher CSA values compared to the distal region (Matta et al., 2014). These regions of the RF and VL were also selected due to the measurement site being the same to that of TMG transducer probe; the authors considered it more appropriate to associate measures of muscle architecture from this region with TMG parameters obtained from the RF and VL muscle bellies. These sites were then replicated post-intervention. Two-dimensional brightness-mode ultrasound images were taken for

each muscle using an HDI-5000 scanner (ATL Ultrasound, Bothell, WA) with a 7- to 12-MHz linear transducer 5cm probe. A water-soluble ultrasound gel (Healthlife, Barclay-Swann Ltd., UK) was used to ensure optimal image quality whilst minimising pressure upon the participant's skin. Pennation angle was identified as the angle between a muscle fascicle and its deep aponeurosis (Blazevich, 2006). Muscle thickness was identified as the distance between the deep and superficial aponeurosis at the ends of each image (Blazevich et al., 2006) (Figure 2.2). Three images were taken at each site in a longitudinal plane, and then exported to Image J (National Institutes of health, Bethesda, MD, USA, version 1.8.0_112) for analysis. The average value from the three images were used for analysis (Aagaard et al., 2001); a methodology that has shown high absolute and relative reliability for architectural parameters being measured in this way (Ema et al., 2013b; König et al., 2014; Silva et al., 2018). All images were taken from the participants' dominant limb (defined as the leg with which they would kick a ball) by the same experienced sonographer. Repeated measurement intraclass coefficient of variation (ICC) and coefficient of variation (CV) were previously established in a pilot study for: muscle thickness (RF; CV: 2.86%, ICC: 0.97, VL; CV: 4.52%, ICC: 0.89) and pennation angle: (RF; CV: 5.38%, ICC: 0.94, VL; CV: 5.67%, ICC: 0.94).

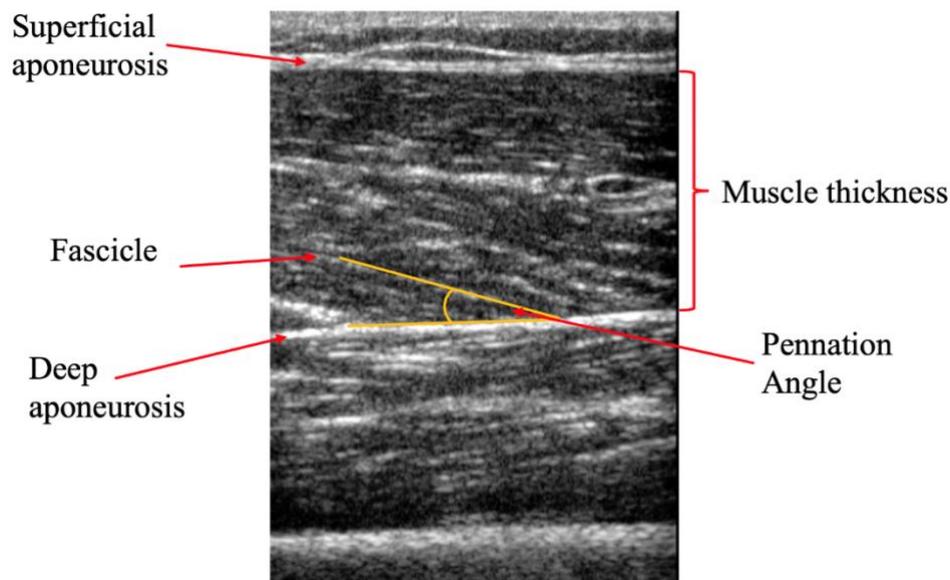


Figure 2.2; Representative longitudinal B-mode Ultrasound image of the vastus lateralis. Muscle pennation angle is represented at the angle between the fascicle and the deep aponeurosis, whilst muscle thickness is defined as the distance between the inside borders of the deep and superficial aponeurosis.

2.3.4 Tensiomyography assessment

TMG assessment of the RF and VL were carried out immediately following the ultrasound assessment. Participants remained in a supine position with a supportive pad placed underneath the knee of the dominant leg, to maintain 60° knee angle (0° = full knee extension) through the assessment. Hip angle

was maintained and controlled with angles of hip flexion ranging from 33° to 40° (0° = full hip extension) due to anatomical differences within the study's cohort. Participant positions were replicated for post-intervention testing. The measurement sites of the VL and RF were identified using manual palpation to locate the thickest part of the muscle belly (Ditroilo et al., 2013). A digital TMG sensor (GK 40, Panoptik d.o.o., Ljubljana, Slovenia) was placed perpendicular to the skin surface upon the point of maximal muscle belly displacement. Two self-adhesive surface electrodes (5cm²) (Axelgaard, USA) were placed on either side of the sensor (5cm from the midpoint of the electrode), whilst ensuring not to cross the muscle borders so as to avoid co-activation (Figure 2.3 A & B). The position of electrodes and transducer probe were marked and measured with reference to anatomical landmarks (anterior iliac crest and superior border of the patella) and recorded for replication post-intervention. A single 1ms wide stimulation was applied at the initial intensity of 20mA at a constant of 30V, with the progressive increase in amplitude of subsequent stimulations, until the maximal displacement of the muscle, measured by the linear transducer, was achieved (Figure 2.3 C). An inter-stimulation time interval of 10-15 seconds was used. The parameters of Dm (maximal radial displacement of the twitch contraction (mm)) and Tc (time between 10% and 90% of displacement (ms)) were extracted from the maximal twitch response of the muscle, by TMG software (Version 3.6.16) and used for offline analysis.

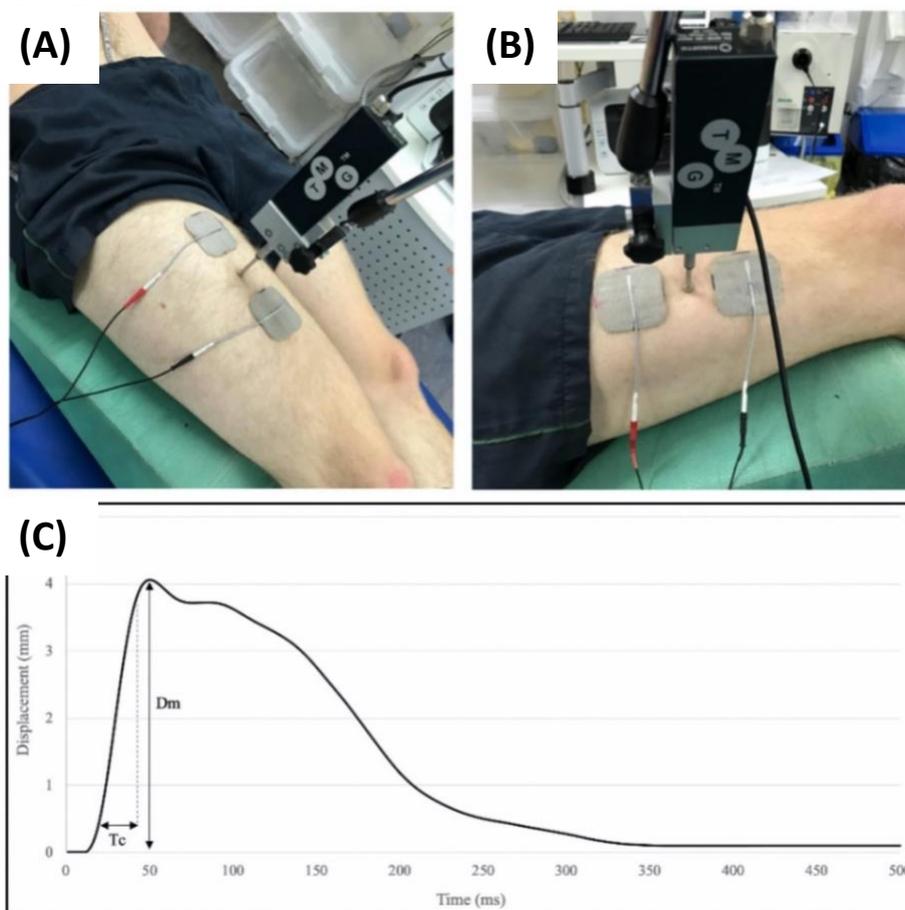


Figure 2.3;
Tensiomyography set-up for measurement of the RF (A) and the VL (B), and parameters extracted from a typical TMG trace; displacement (Dm) and contraction time (Tc) between 10% and 90% of Dm (C). Electrode and stimulator position were recorded with reference to anatomical landmarks and replicated in post-intervention measures.

2.3.5 1RM testing for the back squat, deadlift and Hip thrust

Participants performed 1RM protocols in BS, HT, and DL in each session, the order of exercises was randomised. All participants used the same barbell and weight plates (Eleiko, Sweden), and in a FT700 power rack (Fitness technologies, Australia) with safety bars and with trained spotters present. In addition, a bar pad and wrist straps (Gunsmith fitness, UK) were used for the HT and DL exercises, respectively. In week 1 participants received formal introduction and coaching on correct technique to each exercise by a qualified strength and conditioning coach (*see Appendix B*), after completing a standardized warm-up of static and dynamic stretching. National strength and conditioning Association (NSCA) guidelines for exercise technique were used to coach the BS, DL and HT (Hales, 2010; Contreras et al., 2011; Comfort et al., 2018b).

To ensure minimal fatigue, the 1RMs achieved in familiarisation were used to structure 1RM testing the following week (baseline measurement), which followed the same sets and percentage increments. Once participants felt comfortable and confident in their technique, they completed a 1RM testing protocol for each exercise (Logan et al., 2000). Briefly, this required participants to perform 5 repetitions (reps) at 50% of their individually predicted 1RM (p1RM), 3 reps at 75% p1RM, 3 further reps at 85% p1RM and 2 reps at 90% p1RM. Participants rested 2-4 minutes between each set. Participants then attempted 1 rep at 100% p1RM. If the attempt was successful a 5% increase of p1RM was added, and another attempt was performed (105%p1RM) following a rest of 2-4 minutes. In the event of a failed 1RM attempt, the participant rested 2-4 minutes and then re-attempted the same load. If the second 1RM attempt was also failed participants performed no further 1RM attempts, and the last successfully lifted load was taken as p1RM. In all 1RM testing, a reps in reserve rating of perceived exertion (RIR RPE) scale was used for participant safety as well as guiding incremental increases in the weight lifted (0 meaning no effort, and 10 meaning maximal effort/volitional fatigue) (Zourdos et al., 2016). Specifically, when a participant's RPE reached 9/10, 1RM attempts were recorded. Individual participant stance width, foot angle, and distance from feet to box (HT only), were recorded in accordance with each exercise's technique guidelines and marked each time for replication (Schoenfeld, 2010).

2.3.6 Training intervention

Participants attended two supervised training sessions per week (~72 hours apart) for 6 weeks. Each group used only their respective exercise as the method of lower body exercise, whilst the rest of the programme remained consistent for all groups (Figure 2.4). All loads for the respective exercise sets were calculated according to the participant's previously achieved 1RM value. The programme

followed a linearized progression model to ensure a sufficient intensity within each session (Fleck, 1999). A set of as many repetitions as possible (AMRAP) was included at the end of session 1 each week, to try and maximise the potential for a training response (Morton et al., 2016). All additional exercise loads were calculated according to a combination of estimated 1RM scores, and the RPE scale. Coaches present at each session ensured that participants maintained correct form for each exercise, and spotted participants when the exercise required. Following the completion of each training session (and all 1RM testing sessions), participants were provided with 40g of whey protein in a drink to aid in muscle recovery and muscle building (Macnaughton et al., 2016).

Day One	Day Two
Main Exercise (Back squat, deadlift, or hip thrust) Weeks 1 & 2: 3 x 8* - 75% 1RM Weeks 3 & 4: 4 x 6* - 80% 1RM Weeks 5 & 6: 5 x 4* - 85% 1RM * Last set is AMRAP	Main Exercise (Back squat, deadlift, or hip thrust) Weeks 1-6: 3 x 10 - 70% 1RM
Bench Press Weeks 1 & 2: 3 x 10 - 70% 1RM (RPE 7) Weeks 3 & 4: 3 x 8 - 75% 1RM (RPE 7-8) Weeks 5 & 6: 3 x 6 - 80% (RPE 8)	Dumbbell Chest supported Row Weeks 1 & 2: 3 x 10 - 70% 1RM (RPE 7) Weeks 3 & 4: 3 x 8 - 75% 1RM (RPE 7-8) Weeks 5 & 6: 3 x 6 - 80% (RPE 8)
Underhand-grip pulldown Weeks 1 & 2: 3 x 10 - 70% 1RM (RPE 7) Weeks 3 & 4: 3 x 8 - 75% 1RM (RPE 7-8) Weeks 5 & 6: 3 x 6 - 80% (RPE 8)	Incline Press Weeks 1 & 2: 3 x 10 - 70% 1RM (RPE 7) Weeks 3 & 4: 3 x 8 - 75% 1RM (RPE 7-8) Weeks 5 & 6: 3 x 6 - 80% (RPE 8)
Military Press Weeks 1 & 2: 3 x 10 - 70% 1RM (RPE 7) Weeks 3 & 4: 3 x 8 - 75% 1RM (RPE 7-8) Weeks 5 & 6: 3 x 6 - 80% (RPE 8)	Seated Row Weeks 1 & 2: 3 x 10 - 70% 1RM (RPE 7) Weeks 3 & 4: 3 x 8 - 75% 1RM (RPE 7-8) Weeks 5 & 6: 3 x 6 - 80% (RPE 8)

Figure 2.4; Linearized training protocol used for the 6-week intervention (weeks X: number of sets x number of reps). Participants had a 72-hour break between training sessions. AMRAP – as many reps as possible before failure or form was compromised. RPE scale used during sessions as a training intensity guide.

2.3.7 Statistical Analysis

All statistical analysis was carried out on Graphpad, Prism (Graphpad Software, CA). Baseline and post-intervention scores for ultrasound, TMG and 1RM assessments were assessed for normality (Shapiro-Wilk test). Two factor ANOVAs with repeated measures used to determine the main effects of the three training interventions upon measures of 1RM strength, muscle architecture and the TMG parameters of each assessed muscle [3 training groups (independent factor) x 2 time-points (repeated measure factor) individually. Pearson's product-moment correlation coefficient was used to investigate any potential relationships between muscle architecture and contractile properties within the RF and VL. Correlation coefficients of 0.1-0.3, 0.31-0.5, 0.51-0.7, and >0.71 were classified as small, moderate, large and very large correlations, respectively (Cohen, 1988). Statistical significance was set at $p < 0.05$ (*). All data are reported as mean \pm standard deviation, and with changes represented as percentage \pm upper and lower 95% confidence intervals (CI). Partial eta squared (η^2) effect sizes were calculated, with 0.01, 0.06 and 0.14 considered small, medium and large, respectively (Cohen, 1988).

2.4 Results

2.4.1 Strength

Of the 48 participants recruited, 33 completed the 6 weeks of exercise specific lower body training and all assessments, with an average training attendance of 95.7%. All 3 exercise groups displayed significant increases over time in each of the three 1RM tests (Table 2.1); BS [$F_{(1, 30)} = 91.41, p < 0.001, \rho\eta^2 = 0.753$]; DL [$F_{(1, 30)} = 63.00, p < 0.001, \rho\eta^2 = 0.677$]; HT [$F_{(1, 30)} = 78.13, p < 0.001, \rho\eta^2 = 0.723$]. However, no interaction was found between the exercise groups for any of the three exercises; BS [$F_{(2, 30)} = 3.276, p = 0.052, \rho\eta^2 = 0.179$]; DL [$F_{(2, 30)} = 1.463, p = 0.248, \rho\eta^2 = 0.089$]; HT [$F_{(2, 30)} = 0.601, p = 0.056, \rho\eta^2 = 0.039$].

Table 2.1; Pre, Post and % changes of 1 rep max strength in response to 6 weeks of exercise specific lower body training, and baseline anthropometrics. All data presented as mean \pm SD; CI, 95% confidence limits; * - significant increase over time, $P < 0.05$.

Measure	Squat group			Deadlift group			Hip Thrust group		
	Pre	Post	Δ % (CI)	Pre	Post	Δ % (CI)	Pre	Post	Δ % (CI)
Back squat 1RM (kg)	91.8 \pm 20.9	104.2* \pm 21.9	14.0 (9.3 to 18.7)	87.4 \pm 16.9	95.0* \pm 13.9	9.9 (4.1 to 15.8)	91.3 \pm 14.7	98.2* \pm 12.1	8.3 (4.2 to 12.3)
Deadlift 1RM (kg)	117.0 \pm 23.1	127.5* \pm 19.4	10.3 (3.0 to 17.5)	111.8 \pm 21.4	129.6* \pm 21.7	16.76 (9.1 to 24.4)	114.2 \pm 16.6	127.8* \pm 18.4	12.2 (7.7 to 16.7)
Hip Thrust 1RM (kg)	134.7 \pm 27.4	156.7* \pm 31.8	16.6 (10.8 to 22.5)	136.6 \pm 24.6	155.2* \pm 30.9	13.72 (6.3 to 21.1)	140.1 \pm 20.02	165.4* \pm 22.8	18.6 (11.3 to 25.9)

2.4.2 Contractile properties

For pre-post intervention changes in RF Dm, a significant time-effect was observed across the 3 groups, [$F_{(1, 30)} = 22.37, p < 0.001, \rho\eta^2 = 0.427$], however there were no between-group interaction effect observed [$F_{(2, 30)} = 2.041, p = 0.148, \rho\eta^2 = 0.114$]. Analysis of the pre-post changes in VL Dm showed no significant time [$F_{(1, 30)} = 0.141, p = 0.710, \rho\eta^2 = 0.005$], or between-group interaction effect [$F_{(2, 30)} = 0.289, p = 0.751, \rho\eta^2 = 0.019$] (Figure 2.5A & B). For pre-post intervention changes in VL Tc, no significant time [$F_{(1, 30)} = 0.7112, p = 0.406, \rho\eta^2 = 0.023$] or between-group interaction effects [$F_{(2, 30)} = 0.336, p = 0.718, \rho\eta^2 = 0.022$] were observed. Similarly, no significant time [$F_{(1, 30)} = 0.028, p = 0.869, \rho\eta^2 = 0.0009$] or between-group interaction effect [$F_{(2, 30)} = 0.651, p = 0.529, \rho\eta^2 = 0.042$] was observed for RF Tc.

2.4.3 Muscle architecture

Significant time-effects were observed for muscle thickness changes in both the RF [$F_{(1, 30)} = 30.9, p < 0.001, \rho\eta^2 = 0.969$] and the VL [$F_{(1, 30)} = 33.02, p < 0.001, \rho\eta^2 = 0.524$]. However no between-group interaction effects were observed in the RF [$F_{(2, 30)} = 1.576, p = 0.223, \rho\eta^2 = 0.612$], or the VL [$F_{(2, 30)} = 0.795, p = 0.461, \rho\eta^2 = 0.050$] (Table 2.2). For pre-post changes in RF pennation angle, a significant time effect was observed across the 3 groups, [$F_{(1, 30)} = 8.459, p = 0.007, \rho\eta^2 = 0.220$], however no significant between-group interaction effect was observed [$F_{(2, 30)} = 0.719, p = 0.50, \rho\eta^2 = 0.045$], (Figure 2.5C & D). For pre-post changes in VL pennation angle, no significant time [$F_{(2, 30)} = 2.989, p = 0.094, \rho\eta^2 = 0.091$] or between-group interaction effects [$F_{(2, 30)} = 1.126, p = 0.338, \rho\eta^2 = 0.070$] were observed.

2.4.4 Correlational analysis

As no group interaction effects were seen in measures of muscle architecture and Dm, pooled correlational analysis was performed to investigate the relationship between muscle architecture and Dm; with Pre and post measures of each variable (pennation angle, muscle thickness and Dm) being pooled together ($n=66$). There were moderate negative relationships found between Dm and pennation angle in the RF ($r = -0.36; p = 0.003$); and the VL ($r = -0.37; p = 0.002$) (Figure 2.6A & B). Furthermore, pooled correlational analysis between muscle thickness and Dm revealed a moderate negative relationship in the RF ($r = -0.50; p < 0.001$); but not in the VL ($r = -0.21; p = 0.095$) (Figure 2.6 C & D).

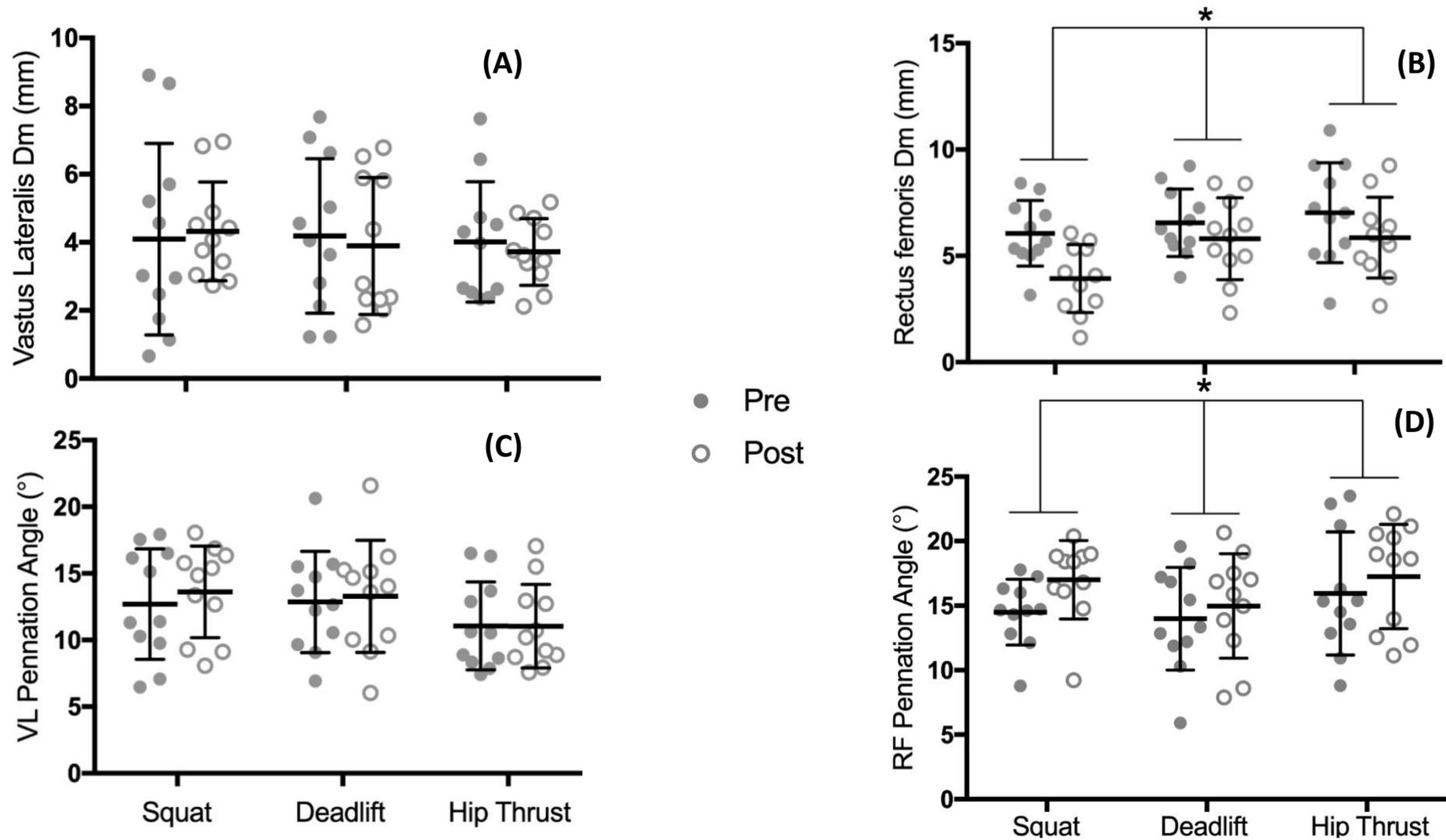


Figure 2.5; Pre and post training intervention measures of; radial muscle displacement in the Vastus lateralis (A) and Rectus femoris (B), and pennation angle of the Vastus Lateralis (C) and Rectus femoris (D), by exercise training group. Individual responses are shown with grey and white circles. Error bars display the means \pm SD. * = significant time effect observed (ANOVA, $P < 0.05$).

Table 2.2; Pre, Post and changes of muscle architecture and contractile properties of quadriceps muscles, in response to 6 weeks of exercise specific lower body training. All data presented as mean \pm SD; CI, 95% confidence limits; * - significant change over time (ANOVA), $P < 0.05$.

Muscle	Measure	Squat group			Deadlift Group			Hip Thrust Group		
		Pre	Post	Δ (CI)	Pre	Post	Δ (CI)	Pre	Post	Δ (CI)
Vastus lateralis	Pennation angle ($^{\circ}$)	12.7 \pm 4.1	13.6 \pm 3.4	0.9 (-0.2 to 2.1)	12.6 \pm 3.8	13.3 \pm 4.2	0.4 (-0.7 to 1.6)	11.1 \pm 3.31	11.0 \pm 3.14	-0.1 (-1.15 to 1.11)
	Muscle thickness (cm)	1.9 \pm 0.5	2.1* \pm 0.5	0.2 (0.1 to 0.3)	2.0 \pm 0.5	2.2* \pm 0.5	0.2 (0.1 to 0.3)	2.0 \pm 0.48	2.1* \pm 0.53	0.1 (-0.02 to 0.3)
	Dm (mm)	4.1 \pm 2.8	4.2 \pm 1.5	0.2 (-1.4 to 1.8)	4.2 \pm 2.3	3.9 \pm 2.0	-0.3 (-1.1 to 0.4)	4.0 \pm 1.8	3.7 \pm 1.0	-0.3 (-1.5 to 1.0)
	Tc (ms)	22.0 \pm 6.2	25.1 \pm 2.9	3.1 (-0.5 to 6.5)	22.00 \pm 5.9	22.7 \pm 4.0	0.7 (-2.8 to 4.2)	24.7 \pm 5.9	27.2 \pm 5.5	2.5 (-2.1 to 7.0)
Rectus femoris	Pennation angle ($^{\circ}$)	14.5 \pm 2.6	17.0* \pm 3.1	2.5 (0.1 to 4.9)	14.0 \pm 3.9	14.9* \pm 4.1	0.9 (-1.4, 3.5)	15.9 \pm 4.8	17.3* \pm 4.5	1.3 (-1.1 to 3.7)
	Muscle thickness (cm)	2.3 \pm 0.3	2.5* \pm 0.3	0.2 (0.1 to 0.4)	2.1 \pm 0.3	2.2* \pm 0.3	0.1 (0.02 to 0.2)	2.1 \pm 0.3	2.3* \pm 0.3	0.1 (0.1 to 0.2)
	Dm (mm)	6.1 \pm 1.5	3.9* \pm 1.6	-2.1 (-3.3 to -0.9)	6.6 \pm 1.6	5.8* \pm 1.9	-0.8 (-1.6 to 0.1)	7.0 \pm 2.4	5.9* \pm 1.9	-1.2 (-2.4 to 0.01)
	Tc (ms)	31.9 \pm 7.0	33.6 \pm 7.3	1.7 (-4.1 to 7.4)	30.9 \pm 5.5	30.4 \pm 3.6	-0.5 (-5.2 to 4.2)	33.6 \pm 7.9	31.8 \pm 5.9	-1.8 (-5.7 to 2.2)

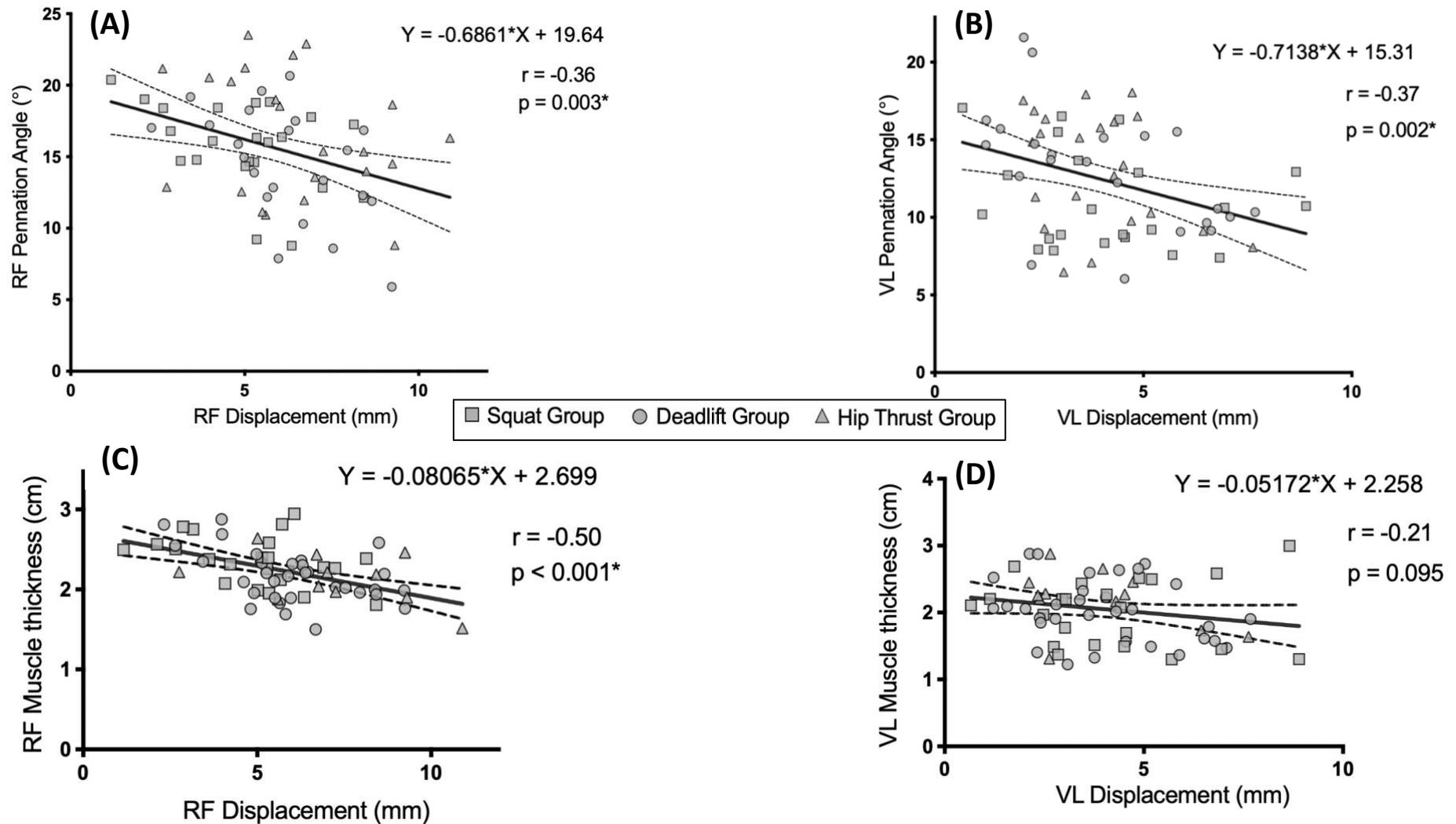


Figure 2.6; Pooled correlation analysis ($n=66$) of pennation angle vs radial muscle displacement in the rectus femoris (A), Vastus lateralis (B); and Muscle thickness vs radial muscle belly displacement in the rectus femoris (C), and Vastus lateralis (D), of all exercise groups.

2.5 Discussion

Following 3 distinct strength training interventions, RF Dm was reduced alongside an increase in pennation angle and muscle thickness. Adaptations were not uniform across the knee extensors; as no between-group differences were seen in any measure of contractile properties or muscle architecture. However, muscle contractile properties and architectural adaptations were accompanied by increases in the maximal strength of all three exercises by each training group, indicating an improvement in overall lower limb strength and quadriceps muscle function. Measures of RF and VL radial displacement were inversely associated with RF and VL pennation angle, respectively. However, only RF displacement was associated with muscle thickness, supporting the potential use of TMG to detect and measure muscle specific adaptations to RT.

To our knowledge this is the first study to employ TMG to assess muscle-specific adaptations in response to strength training aimed at increasing lower limb strength. Previously, acute changes in Dm following exercise have been investigated and been shown to track exercise-induced fatigue and decreased neuromuscular function (García-manso et al., 2011;García-Manso et al., 2012;Hunter et al., 2012;Macgregor et al., 2016). Longitudinal alterations in Dm, like those of the present study, are less understood, likely owing to the inherent difficulty in assessing muscle stiffness *in vivo* (Macgregor et al., 2018). Nevertheless TMG is capable of determining that more rapid Tc and lower Dm values are associated with greater exposure to strength and power training (Loturco et al., 2015b;de Paula Simola et al., 2016;Šimunič et al., 2018); and that the increased muscle belly stiffness was contributory to improved force transmission (García-García et al., 2013;Hughes et al., 2015). Early work by Wilson et al. (1994) supports this by showing oscillation techniques to infer relationships between musculotendinous stiffness and isometric, concentric and eccentric force production; which was corroborated by later mechanomyography studies (Yoshitake et al., 2005;Yoshitake et al., 2008). More recent support was shown by reduced lower limb muscles' Dm and Tc seen following 8 weeks of plyometric training (Zubac and Simunic, 2017) and declined RF Dm observed following 3 weeks of strength focused training (Rusu et al., 2013). Such findings support our current study as we demonstrated reductions in RF Dm were accompanied by improvements in 1RM strength in the BS, DL and HT exercises.

Whist both aforementioned studies concluded that increased muscle stiffness (or muscle tone) contributed to improved muscle function and performance, unlike Zubac and Simunic (2017), we observed no change in Tc in the present data. This disparity may be due to the different training

interventions used between studies; with Zubac and Simunic employing plyometric training where the focus is on speed of movement and explosiveness (Cormie et al., 2011), not maximal strength. Alterations in muscle thickness and pennation angle are known to occur as part of the hypertrophic response to RT and are associated with increased muscular strength (Rutherford and Jones, 1992; Alegre et al., 2006; Strasser et al., 2013; Franchi et al., 2014), which is reflected in the findings of the current study. Along with the observed reduction in Dm, the observed increases in muscle architectural measures partially confirm our initial hypothesis of seeing alterations within the knee extensors. Previously, increases in pennation angle have been seen to contribute to increase PCSA of pennate muscle, thereby increasing its maximal force production capacity (Kawakami et al., 1995; Aagaard et al., 2001). Increases in pennation angle are thought to allow more contractile material to be packed into the same ACSA, increasing PCSA, thus allowing a greater number of fascicle-tendon attachments (Blazevich, 2006). The number of fascicle-tendon attachments directly contributes to a muscle's maximal force generating capacity and therefore its overall strength. The significant increases in RF and VL muscle thickness seen in the present study, alongside increases in maximal strength are in agreement with current literature (Blazevich et al., 2003; Franchi et al., 2018a). These aforementioned mechanisms responsible for improving maximal force transmission to pull direction, may, partly explain increased 1RM strength seen in the three exercise tests by all groups (*Table 1*).

However, increased muscle thickness was not accompanied by an increase in pennation angle within the VL (*Table 2*). Potential explanations for this could be that increased VL muscle thickness was a result of an increased ACSA and not PCSA, which pennation angle has been previously associated (Aagaard et al., 2001). Additionally, architectural adaptations may not be uniform across quadriceps muscles (Mangine et al., 2018) and greater habitual usage of specific muscles may mean greater levels of hypertrophic response are required (Ema et al., 2013b). As only the mid-points of the RF and VL muscles were measured in the present study, it must be acknowledged that intra-muscular differences in architectural changes can occur (Ema et al., 2013a); owing to muscle's region-specific functional roles (Watanabe et al., 2012).

No statistically significant between-group differences were seen in architectural or contractile mechanics adaptations (*Figure 2. 5*); suggesting that training the three exercises had no differential effects upon measures in the RF or VL, which is at odds with our secondary hypothesis. Whilst the BS and DL have both shown high levels of VL and RF activation (Lee, 2015), quadriceps activation in the HT has not been as extensively investigated. Contreras et al (2015) showed no difference between peak VL activation of the BS and HT in trained women, postulating that the quadriceps play a

stability role in performing the HT movement despite the biomechanically distinct movement patterns of the respective exercises. As the RF has been shown to stabilize the pelvis during coordinated movements eliciting anterior and posterior tilting (Neumann, 2010; Lee, 2015), it may be possible that HT training could have caused sufficient stimulus for RF adaptation to occur from this stability role; particularly in the untrained population of this study. However, as RF activation profiles and pelvic tilt kinematics in the HT have not been investigated to date, these are areas that require further investigation to confirm.

Physiological development of muscle tissue can be characterised by measures of muscle architecture and more recently by changes in muscle belly stiffness using elastography (Gennisson et al., 2010; Debernard et al., 2011; Eby et al., 2015; Lima et al., 2017). Debernard et al. (2011) observed across different ages groups that increases in fascicle angle were associated with increases in shear wave velocity (from which shear modulus was calculated) as a measure of muscle stiffness, or passive tension. These authors suggested that such information regarding contractile mechanics obtained from muscle belly stiffness assessments, may be useful when assessing alterations in muscle fascicle orientation. Whilst the aforementioned literature supports the association of muscle architecture and displacement parameters, it must be acknowledged that investigations into the measurement construct of techniques such as shear wave elastography, is ongoing (Taljanovic et al., 2017). The significant negative associations found within the VL and RF of the current study are in agreement (figure 2.6), and suggests, that measurements of muscle belly displacement are reflective of increased pennation angle and muscle thickness; thus confirming our tertiary hypothesis.

Mechanistically, angle of fascicle pennation will change as result of a change in muscle tension (Erskine et al., 2010b). A change in muscle–tendon length, brought about by a change in joint angle, affects both the passive tension generated by the connective tissue (in parallel and in series) and the position of the contractile elements of the muscle. This, in turn, determines the level of tension that can be generated (Mohamed et al., 2002; Hunter et al., 2012). Pišot et al. (2008) found a similar relationship to our present study, between muscle thickness reductions and decreases in muscle belly stiffness (increased D_m). Reduced muscle belly stiffness was attributed to declined passive muscle tension, which accompanies muscle atrophy (Whitehead et al., 2001). Similarly, (Šimunič et al., 2019) recently showed that following muscle atrophy, changes in D_m were significantly associated with changes in pennation angle and muscle thickness; the latter being echoed in the present study with changes in the RF (see *appendix A*). However a potential limitation of Simunic et al.'s work, and that of the present study, is the absence of a comparable control group. In the context of muscle adaptations, such data could add strength to the observed findings of the present study.

This is an area future research should look to address. Additionally, future research should look to investigate the time course of adaptations and associations observed here; as only end-point measures were taken in the present study. The ability of Dm to reflect differing degrees of fascicle pennation may aid practitioners in monitoring, and implementing interventions alongside established physiological markers, perhaps following a strength training programme or during rehabilitation settings. TMG's assessment of skeletal muscle contractile properties provide a unique insight into the rate of excitation-contraction coupling alterations in relation to architectural adaptations. Crucially, TMG may not only provide information on muscle architecture, but could also pose a more objective and simpler method of assessment; one that involves assessing skeletal muscle in an 'active' state as opposed to the resting requirements of ultrasonography.

2.6 Conclusion

TMG detected a muscle specific training adaptation within the knee extensors, in response to 6-weeks of lower limb strength training. Furthermore, measures of muscle belly displacement were associated with corresponding measures of muscle architecture, indicating that assessment of contractile properties can also provide information regarding muscle geometry that influences contractile mechanics. Whilst not only addressing a gap in the literature (concerning the longitudinal use of TMG) the findings of current study would be of benefit to practitioners and athletes in their efforts to investigate isolated muscle function. Applications of non-invasive contractile mechanic assessments would provide objective insights into rehabilitation programmes and the impact of specific interventions upon contractile mechanics and adaptations within specific muscles. Furthermore, the objectivity and ability of TMG to assess contractile properties during an 'active' contraction, provides a unique insight into contractile mechanics. To further our understanding, relationships found in this study should be investigated in range of other muscles that play key roles in human movement. Additionally, similar strength training adaptations should be studied in ageing and female populations, as well as potential associations with other areas of the neuromuscular system.

Chapter 3

Understanding the mechanisms of strength adaptation; a time-course of neural and morphological changes throughout 6-weeks of strength training.

Matthew T Wilson, Malcolm Fairweather, Lewis J Macgregor, Angus M Hunter

3.1 Abstract

Introduction: To determine the underpinning mechanisms of adaptation to strength training, it is necessary to assess changes in relation to both the neural, and morphological pathways known to be involved in the adaptive process. The aim of this study is to investigate the time-course of contractile mechanic changes in relation to measures of muscle architecture, neuromuscular and strength adaptation across 6-weeks, lower-limb strength training. **Methods:** Forty participants completed the study; 22 intervention (10 males/12 females; 173.48 ± 5.20 cm; 74.01 ± 13.13 kg) completed 6-week strength training, and 18 control (10 males/8 females; 175.52 ± 7.64 cm; 70.92 ± 12.73 kg) performed no strength training but maintained habitual activity. Tensiomyography contractile parameters of radial muscle belly displacement (Dm) and contraction time (Tc) were obtained, alongside measures of knee extension MVC strength; corticospinal excitability and inhibition via Transcranial Magnetic stimulation; motor unit (MU) firing rate; muscle thickness and pennation angle via ultrasonography. All measures were assessed before (pre) and after 2, 4, and 6-weeks (post) of the dynamic lower limb resistance training. All physiological measurements were conducted on the quadriceps muscles of the dominant leg. Five-rep max (5RM) back squat (BS) strength was assessed pre-and post-training. **Results:** After 2-weeks training, Dm was significant reduced in both VL (25%, ES: 0.86) and RF (19%, ES: 0.53) of the intervention group ($P < 0.0001$); this was prior to any measured neural or morphological adaptation. After 4-weeks training, MVC strength was increased (15%, ES: 0.63 $P < 0.001$) and accompanied by increased corticospinal excitability (16%, ES: 0.61, $P < 0.001$); however, no change in corticospinal inhibition or motor unit firing rate was seen. After 6-weeks training (post), there was an additional increase in MVC strength (6%, ES: 0.28, $P < 0.02$) accompanied by significant increases ($P < 0.001$) in muscle thickness (VL; 16%, ES: 0.94, RF; 13%, ES: 0.88) and pennation angle (VL; 14%, ES: 1.29, RF; 13%, ES: 1.09). **Conclusion:** We demonstrated enhanced contractile properties occurred before any muscle architecture, neural and strength adaptations. Additionally, it was confirmed that early increases in muscular strength may result from altered spinal cord output rather than modifications within motor units; and that later increases in muscular strength are accounted for by architectural adaptation.

3.2 Introduction

In the previous chapter we demonstrated alterations in contractile properties and their association with altered muscle architecture, following a period of resistance training. It would be pertinent to establish a time-course of contractile mechanics adaptations in relation to other areas of the neuromuscular pathway, as muscle architecture alterations are not solely responsible for strength gain. Consensus evidence indicates that training-induced early increases in muscular strength are primarily resultant from neural adaptations (Komi et al., 1978; Moritani and deVries, 1979; Narici et al., 1989; Reeves et al., 2005); with early sEMG studies demonstrating association between increased strength and efferent output (Hakkinen and Komi, 1983; Aagaard, 2003). Owing to technological advancements, specific areas of the neuromuscular pathway experiencing early adaptations to training can be investigated in isolation, providing a greater depth in understanding. Properties of individual MUs (Van Cutsem et al., 1998; Vila-Chã et al., 2010) and parameters of the corticospinal tract and M1 (Carroll et al., 2002; Selvanayagam et al., 2011), have been used to suggest specific locations and responses underpinning adaptations in early stages of training.

Corticospinal excitability (Goodwill et al., 2012; Weier et al., 2012) and inhibition (Latella et al., 2012), measured in the RF, have been shown to increase and decrease, respectively, in response to strength training. Corticospinal excitability and inhibition are assessed through TMS (Kidgell et al., 2017), with recent meta-analyses confirming the aforementioned changes at the level of excitation and inhibition, which function to increase motoneuron output and increase strength (Kidgell et al., 2017; Siddique et al., 2020). However, these aforementioned findings represent one of several areas of potential neural adaptation to resistance training but do not explain alterations to MU behaviour. Therefore, measurements of different areas within the neuromuscular system should be assessed simultaneously.

Increased agonist muscle activation resulting from resistance training (Pucci et al., 2006; Jenkins et al., 2017) has been attributed to alterations in the behaviour of MUs (Van Cutsem et al., 1998). In particular, studies have shown the discharge rate (firing rate) of MUs to increase following periods of strength training using intramuscular EMG (Vila-Chã et al., 2010) and decomposition of sEMG (Del Vecchio et al., 2019). However, conflicting findings show maintained MU firing rates have been observed in the VL (Rich and Cafarelli, 2000; Pucci et al., 2006; Sterczala et al., 2020); demonstrating the precise nature of MU firing rate adaptations remains unclear. Whilst dEMG provides a useful, non-invasive method of assessing MU adaptations, it is unable to make direct inferences upon

adaptations within the spinal excitatory/inhibitory networks. Therefore, concurrent measurements of MU discharge properties and the associated corticospinal networks of excitation and inhibition, could provide clearer insights into the precise nature of the aforementioned early adaptations to strength training.

In the previous chapter we observed an inverse relationship between TMG derived Dm, and measures of muscle architecture (muscle thickness and pennation angle). This indicated that strength training-induced changes in muscle architecture are reflected by alterations in the contractile property Dm. However, Šimunič et al. (2019) recently demonstrated altered Dm prior to any change in muscle thickness or pennation angle, during muscle atrophy and subsequent recovery; indicating that the time-course of contractile mechanics and architectural changes are not exclusively linked. As increases in pennation angle and muscle thickness have been shown contributory to later increases in muscle strength (> 4-weeks training) (Aagaard et al., 2001; Blazevich et al., 2003), it is not known whether contractile properties would be modified prior to these hypertrophy-orientated architectural adaptations. With early increases in strength attributed to primarily neural adaptations, it may be possible that early alterations in contractile properties be influenced by altered E-C coupling (Calderón et al., 2014). Previously, TMG has been used to infer upon alterations in E-C coupling following exercise-induced muscle damage (Hunter et al., 2012); where increased Tc was associated with secondary EIMD markers, demonstrating the capability of TMG to reflect early changes in contractile mechanics following training. Additionally, some researchers hypothesize that the EIMD-associated inflammation and protein-turnover responses may contribute to early stages of hypertrophy and strength gain (Brentano and Martins Krueel, 2011; Schoenfeld, 2012) To further our understanding of strength adaptation it would be beneficial to investigate the time-course of contractile mechanic alterations in relation to other areas within the neuromuscular pathway to provide clarity to the underpinning mechanisms responsible. In particular, investigating central and peripheral areas of neuromuscular system would allow practitioners to improve the efficacy of targeted interventions for improving athletic performance, and in designing rehabilitation strategies.

Therefore, the aim of this study was to investigate time-course of contractile property changes in relation to measures of muscle architecture and neuromuscular adaptation, across a 6-week strength training programme. By assessing a time-course of changes throughout areas of the neuromuscular pathway, we aim to enhance clarity surrounding mechanisms responsible for increases in strength following training. We hypothesise that over the course of the 6-week strength

training intervention we will observe an increase in strength which will be accompanied by reductions in Tc and Dm, indicating improved contractile mechanics, and that these contractile adaptations will occur before any change muscle architecture. We also hypothesise that any increase in muscular strength in the early stages of training will be accompanied by an-increase in corticospinal excitability, reduced corticospinal inhibition, and increased firing rate of VL MUs. Furthermore, based on the available literature we hypothesise that the aforementioned early increases in strength and neural adaptations will occur prior to increases in muscle thickness and pennation angle.

3.3 Methodology

3.3.1 Participants

A priori power analysis was conducted (G*power, version 3.1.9.4, Heinrich-Heine University, Dusseldorf, Germany) using previous data (De Souza et al., 2018) and the following parameters; ANOVA: repeated measures, within-between interaction ES = 0.2, power (1- β) = 0.8 for the comparison between groups. Power analysis showed at least 18 participants per group were required to achieve an actual power of 0.81. In total, 43 eligible participants volunteered to take part in the study; 23 participants were randomly assigned to the training group and 20 participants to the control group. Based upon participant withdrawals experienced in the previous chapter, a higher number of indicated participants were recruited to account for potential dropouts. All participants participated in team sports 2 x per week and did not take part in any lower-limb resistance training for at least 6 months prior to the study. Participants were free from musculoskeletal injury or neuromuscular conditions. Prior to study commencement, participants gave informed consent, completed a physical activity readiness questionnaire, and the screening questionnaire for TMS (Appendix C) (Rossi et al., 2011). The study was approved by the University of Stirling NHS, Invasive or Clinical Research Committee, and all procedures of the study were carried out in accordance with all ethical standards outlined in the Declaration of Helsinki.

Overall, 40 participants completed the study; 22 in the training group (10 males/12 females; 173.48 \pm 5.20 cm; 74.01 \pm 13.13 kg) and 18 control participants (10 males/8 females; 175.52 \pm 7.64 cm; 70.92 \pm 12.73 kg). In total, 3 participants were withdrawn from the study; 1 from the training group for failing to complete >90% of the training sessions; 1 from the control group for sustaining an injury out-with the study; and 1 one from the control group for failing to pass the TMS screening questionnaire.

3.3.2 Experimental design

The study ran for 9 weeks in total (figure 3.1). Participants attended 2 laboratory sessions in week 1. The first of these laboratory sessions (L1) entailed; ultrasound imaging of muscle architecture, TMG assessment of contractile mechanics, isometric MVC, TMS, M_{max} , and dEMG during submaximal isometric contractions. After 48-72hrs, the second lab session (L2) consisting of a 5-rep max (5RM) back squat (BS) test was carried out. Lab sessions carried out in week 1 served as familiarisation sessions, with the same schedule carried out a week later to obtain baseline measurements (Pre). Following this the intervention group began 6-weeks of lower limb resistance training (2x per week). After the 2nd (Wk 2) and 4th (Wk 4) weeks of training, all participants from both groups underwent session L1 to obtain time-point measures of any neuromuscular changes. Measurements taken at WK2 and WK4 were conducted at least 72 hours after the previous training session to account for any intra-muscular swelling and post-training fatigue in the intervention group. Upon completion of the 6-weeks resistance training, all participants underwent sessions L1 and L2 (~4 days after the last training session) to obtain post-intervention measures (Post).

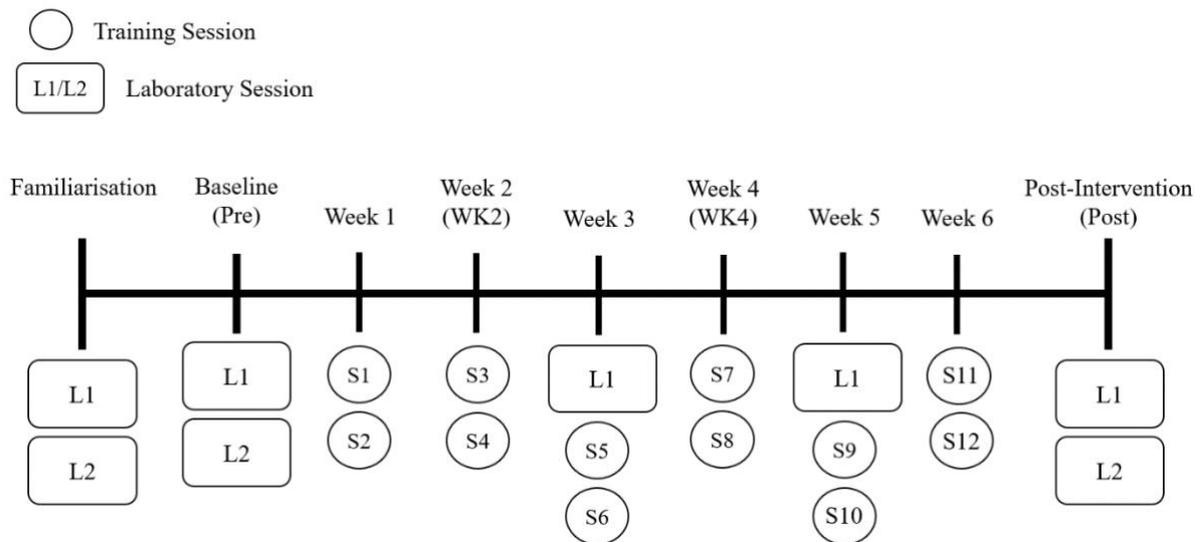


Figure 3.1; Schematic overview of the study design; L1 - laboratory session 1; L2 – laboratory session 2; S1-12 – training sessions.

3.3.3 Skeletal muscle architecture and adipose tissue thickness assessment

Ultrasound images of VL and RF muscle architecture were obtained using a Philips Lumify linear transducer (12-4 MHz, 34.5mm) (Philips, USA). For further details on measurement procedures see section 2.3.3

In addition to muscle thickness and pennation angle, adipose tissue thickness was measured as the straight-line distance between the top of the muscle fascia and bottom of the cutaneous layer (cm) (Sterczala et al., 2020). Adipose tissue was measured to strengthen the validity of any training-

induced changes in EMG parameters throughout the study; adipose tissue is thought to act as a filter upon EMG amplitude (Sterczala et al., 2020), and thus could inadvertently change EMG parameters between time-points.

3.3.4 Tensiomyography

TMG assessment of contractile mechanics was carried out upon the VL and RF muscles of the participant's dominant leg. For details see section 2.3.4.

From the maximal twitch-torque curve the following parameters were recorded and taken forward for statistical analysis; maximal radial muscle displacement (Dm), contraction time between 10-90% of Dm (Tc).

3.3.5 Isometric MVC

Participants were seated in an isokinetic dynamometer with their dominant leg secured to a calibrated load cell (Biodex System 3, Medical Systems, New York, USA). Knee angle was set to 60° knee flexion (0° = full knee extension) with the arm of the dynamometer being set such that the axis of rotation was in line with the participant's lateral femoral condyle. Participants remained in this position for all subsequent testing procedures involving isometric contractions (TMS, M_{max} , dEMG). Participants were required to perform 3 MVCs of 5s in duration, in response to a randomly timed audio signal. Participants were instructed to kick as hard and as fast as possible for the duration of the 5s contraction. Participants received strong verbal encouragement throughout. There was a minimum of 60 seconds rest between MVC attempts. The maximal torque produced from the best of the 3 attempts was used in analysis and for prescription in the subsequent testing procedures.

3.3.6 Transcranial magnetic stimulation (TMS)

Single pulse TMS was used to elicit MEPs in the RF which were assessed using sEMG recordings (see below). Single stimuli of 1ms in duration were applied over the contralateral M1 using a magnetic stimulator (Magstim 2002, The Magstim Company Ltd. Whitland, UK) and a 110mm double cone coil attachment. Optimal coil location for MEP generation was determined by placing the coil over the M1, laterally to the vertex; the coil location at which the largest MEPs were elicited was identified and marked with ink (Goodall et al., 2009). This position was recorded for replication in subsequent visits. The quadriceps femoris' active motor threshold (aMT) was determined by increasing the stimulator output from 10% in 5% increments until discernible MEPs were visible whilst the participant held a 20%MVC (Wilson et al., 1995). Once aMT was established, all subsequent stimulations were delivered at 130% aMT.

To assess corticomotor excitability participants were required to hold 20% MVC contraction whilst 20 single pulse stimulations were delivered over M1, with a 6 second window separating each stimulation. Corticomotor excitability was determined as the average MEP peak-to-peak amplitude normalised to the maximal response elicited by motor nerve stimulation (%Mmax, see below).

To assess corticomotor inhibition participants were required to perform a MVC of 5s in duration whilst a single stimulation was elicited over the M1. This process was repeated 3 times with at least 60 seconds rest between contractions, with the average cSP taken forward for analysis.

Corticomotor inhibition was quantified as the cSP duration, taken from the stimulation artefact to the resumption of discernible, uninterrupted EMG activity in the RF. Corticospinal excitability and inhibition were assessed using the RF as opposed to the VL due to clearer EMG signal and Mmax being detected during pilot testing. Additionally, a study by Temesi et al. (2014) found no differences in between the VL, VM and RF for active or resting MT stimulus-response curves; suggesting that one muscle could be used as representative of all three.

3.3.7 Femoral nerve stimulation to assess M_{max}

Stimulation of the peripheral femoral nerve was conducted using an electrical stimulator (Biopac Systems, Inc). Stimulation site was determined by locating the femoral artery and placing a self-adhesive surface electrode (cathode) lateral to it, high over the femoral triangle. The anode electrode was placed on the gluteus maximus. Single stimuli were delivered to the muscle while participants maintained a rested position, and the intensity of stimulation was incrementally increased until a plateau in twitch amplitude and RF M-wave (M_{max}) were observed. Participants then received 3 further single stimulations whilst maintaining 20%MVC to ensure M_{max} had been obtained. Again, RF was used based upon due to the reasons outlined in section 3.3.6.

3.3.8 Surface EMG

Throughout TMS and femoral nerve stimulation protocols, sEMG recordings were taken from the RF muscle on the dominant leg, recorded using a wireless system (Biopac Systems, Inc, Goleta, CA, USA). Data was sampled at a rate of 2kHz and filtered using 500Hz low and 1.0Hz high band filters. All signals were analysed off-line (Acqknowledge, v 3.9.1.6, Biopac Systems, Inc. Goleta, CA, USA). EMG activity was collected using Ag/AgCl surface electrodes (Ambu Ltd., UK) with an intra-electrode distance of 2cm. The areas of interest were shaved and abraded prior to electrode application, as per surface electromyography for the non-invasive assessment of muscles (SENIEM) guidelines; which were also used to determine electrode location on the RF (50% distance on the line between anterior spina iliaca superior to the superior part of the patella). A reference electrode was

positioned on the patella of the dominant limb also. The positions of each electrode were recorded in reference to anatomical landmarks for accurate replication in all subsequent lab sessions.

3.3.9 Submaximal Isometric Trapezoid

Following the completion of the TMS and PNS (peripheral nerve stimulation) protocols, surface electrodes on the RF were removed and the skin area cleaned. A separate electrode was fixed to the VL muscle to obtain EMG data for decomposition (see below). Participants were then asked to perform an isometric knee extension trapezoid contraction at 60% Pre-MVC. Participants were provided with a visual feedback trace through the task where they linearly increased torque output at 10%MVC/second, held a sustained 60%MVC contraction for 10 seconds, and then linearly decreased torque output at a rate of 10%MVC/second. If the participant failed to adhere to the visual feedback trace, they were allowed a second attempt after 2 minutes rest. Force equating to 60% Pre-MVC was used in all subsequent testing sessions.

3.3.10 Decomposition EMG

A Delsys Trigno Lab wireless system was used to obtain surface EMG data for decomposition. A 4-pin surface electrode (Trigno-Galileo, Delsys, Natick) was fixed to the VL muscle belly (same location used for Ultrasound imaging and TMG) and secured with micropore tape. The reference end of the electrode was fixed to the patella as per manufactures guidelines. The VL was selected for measurement due to a typically lower adipose tissue thickness compared to the RF during pilot testing (reducing the spatial filtering influence adipose tissue on EMG signals). All four channels of EMG data were visually inspected, prior to data recording, to ensure excessive background noise and artefact were not present: baseline noise not exceeding 10 μ V according to manufactures guidelines. Signal to noise ratio is calculated within the acquisition software (EMGworks4, Delsys, Natick) according to the following formula: $20\log(S_{RMS}/N_{RMS})$ [where S = EMG signal and N = baseline noise]. All EMG signals were low pass (fourth-order Butterworth, 24dB/octave slope, 1750-HZ cut-off) and high pas (second-order Butterworth 12dB/octave slope, 20-HZ cut-off) filtered prior to sampling at a rate of 20,000 Hz.

The four separate filtered EMG signals from the Galileo array were entered were into the Precision Decomposition III (PD III) algorithm and decomposed into constituent MU action potential trains (Neuromap, Delsys Inc, Boston, USA). The PD III technique was originally described by Adam and De Luca (2005), and then subsequently refined by Nawab et al. (2010). The PD III uses artificial

intelligence to identify action potential shapes and assign them to individual MUs; with validity and reliability being previously shown at various contraction intensities up to 100% MVC (Hu et al., 2013).

The accuracy of the dEMG process for each trapezoid contraction was assessed by reconstruct and test analysis (Nawab et al., 2010; De Luca and Contessa, 2011; De Luca et al., 2015b). This analysis assesses the level of firing rate accuracy of each identified MU and the number of errors per second, across the entire trapezoid contraction ($\text{Accuracy} = 1 - N_{\text{error}}/N_{\text{truth}}$; where N_{error} is the total number of unmatched events, and N_{truth} is total number of true events). Only MUs which displayed >90% accuracy were included in analysis. Mean firing rate (MFR) curves were smoothed using a 600ms Hanning window, as recommended by the software manufacturer.

From analysis of individual action potential trains, mean firing rate (MFR) during the steady state plateau was calculated. MFR was calculated as the inverse of the averaging interspike interval during the distal 3 seconds of the steady-state torque plateau. The 3 second portion towards the distal end of the plateau phase had been previously deemed to be appropriate to use for analysis, displaying the greatest reliability (Balshaw et al., 2017).

3.3.11 5-rep max back squat strength

In the second lab session (L2), participants completed a 5RM BS test. 5RM exercise tests have been previously shown reliable and valid in recreational athletes in both trained and untrained backgrounds (Sascha and Künzell, 2014). Following a standardised warm-up, participants completed a structured 5RM protocol. Participants were given up to three attempts to obtain their 5RM. Throughout the session participants provided RPEs for each set completed based upon the repetitions in reserved (RIR) scale (Zourdos et al., 2016). These RPE scores were used to guide incremental increases in load lifted for each exercise set. In week 1 (Familiarisation), participants were assessed on correct and proper back-squat technique by qualified exercise professionals according to published exercise technical guidelines (Comfort et al., 2018a). The 5RM scores achieved in familiarisation were used to structure the incremental loads for pre-intervention testing. This ensured that 5RM was achieved within 3 attempts and helped mitigate the learning effect before pre-intervention measures were taken. Foot angle and stance width were recorded during baseline testing and replicated in post-intervention testing with the aid of marker tape (Schoenfeld et al., 2016). Participants did not have to complete all 3 attempts if they achieved their 5RM after 1 or 2 attempts. Participants were given 2-minute rest intervals between sets 1-4, and then 4 minutes between 5RM attempts. The achieved pre-intervention 5RM load was incorporated into the post-

intervention testing as the final warm-up set before the 1st post-intervention 5RM attempt (see chapter 4, table 4.1).

3.3.12 Training intervention

The intervention group completed a 6-week resistance training programme (2 sessions per week, separated by ~72 hours), consisting of compound lower limb exercises following a linear periodisation model (Table 3.2). All training sessions were supervised by qualified instructors to ensure safe technique and correct performance of each exercise. The baseline 5RM BS score was used to prescribe the loads and increments for each participant's BS sets, and all other accessory exercise loads were guided by RIR RPEs to ensure a sufficient training stimulus (Zourdos et al., 2016). All participants were provided with drink containing 20g serving whey protein isolate to aid recovery drink throughout the 6-week training period (West et al., 2017). Drinks were provided at the end of each training session to the intervention group, and at the same corresponding time points for the control group. To help ensure compliance, each drink was provided directly from the investigatory team for both groups.

Session One	Session Two
1) Back squat 1 & 2: 5 x 5 - 80% 5RM 3 & 4: 5 x 5 - 82.5% 5RM 5 & 6: 5 x 5 - 85% 5RM	1) Back squat 1-6: 3 x 10 - 70% 5RM
2) Dumbbell walking lunges 3) Dumbbell step-ups 4) Goblet squats 1: 3 x 8 (RPE 6-8) 2: 3 x 10 (RPE 6-8) 3: 3 x 12 (RPE 6-8) 4: 3 x 8 (RPE 7-9) 5: 3 x 10 (RPE 7-9) 6: 3 x 12 (RPE 7-9)	2) Bulgarian unilateral split squats 3) Barbell Hip thrusts 4) Box squats 1: 3 x 8 (RPE 6-8) 2: 3 x 10 (RPE 6-8) 3: 3 x 12 (RPE 6-8) 4: 3 x 8 (RPE 7-9) 5: 3 x 10 (RPE 7-9) 6: 3 x 12 (RPE 7-9)

Table 3.1; *Template of training programme used for the 6-week training intervention (week (1-6): number of sets x number of repetitions); 5RM – 5-rep max; RPE – rate of perceived exertion.*

3.3.13 Statistical analysis

All statistical analysis was carried out using Prism 8 (GraphPad software, CA, USA). Baseline, WK2, WK4 and Post-interventions data sets were checked for normality (Shapiro-Wilk test). For dEMG data, MU firing rate was analysed for each participant on a contraction-by-contraction basis; meaning data were not pooled from multiple contractions, nor multiple participants within a group. Two factor ANOVAs with repeated measures were used to determine the main effects of the training

intervention upon measures of muscle architecture; contractile mechanics, MVC strength; corticospinal excitability and inhibition; dEMG derived MU MFR [2 groups (independent factor) x 4 time-points (repeated measures factor)], individually. Where significance was detected Tukey *post hoc* tests were used to identify where any significant difference occurred. Cohen's *d* effect sizes (ES) were also calculated by; $d = (\text{Mean}_1 - \text{Mean}_2) / \text{SD}_{\text{pooled}}$, where $\text{SD}_{\text{pooled}} = \sqrt{[(\text{SD}_1^2 + \text{SD}_2^2) / 2]}$. ES are interpreted as follows: ≤ 0.5 = trivial, 0.5-1.25 = small, 1.25-1.9 = medium, ≥ 2.0 = large, for untrained participants (Rhea, 2004). All data are reported at mean \pm standard deviation (SD), with changes represented as percentage % \pm upper and lower 95% confidence intervals (CI). Alpha was set at $P < 0.05$.

3.4 Results

3.4.1 Strength:

Analysis of 5RM strength revealed a significant time effect [$F_{(1,38)} = 104.1$, $P < 0.0001$] across both groups, however a significant [$F_{(1,38)} = 75.72$, $P < 0.0001$] interaction effect showed that only the intervention group significantly increased their 5RM BS strength ($P < 0.0001$, ES = 0.9, 95%CI: 16.34 to 22.80), with no significant change in the control group ($P = 0.53$, ES = 0.13, 95%CI: -2.019 to 5.13). Post-intervention 5RM BS strength was significantly greater in the intervention group compared to control ($P < 0.0001$, ES = 1.67, 95%CI: 17.27 to 43.59) (table 3.2).

Analysis of knee extension MVC strength revealed a significant time effect [$F_{(2.54, 96.42)} = 15.01$, $P < 0.0001$] across both groups, however a significant interaction effect [$F_{(3,114)} = 9.297$, $P < 0.0001$] showed only the intervention group to significantly increase MVC torque by week 4 ($P < 0.001$, ES = 0.63, 95% CI: -42.41 to -12.06) and then a further increase week 4 – post ($P = 0.017$, ES = 0.28, 95%CI: -25.30 to -2.086).

Knee extension MVC torque was not significantly different between the intervention and control groups at week 4 ($P = 0.92$, ES = 0.23, 95%CI: -25 to 45.62) or at post ($P = 0.46$, ES = 0.47, 95%CI: -17.0 to 62.0).

Table 3.2; Time course of % changes in strength measures across 6-weeks of lower limb resistance training. Participant anthropomorphic data are also shown. All data presented as mean \pm SD; CI, 95% confidence limits; * - significant change from Pre; # significant change from Week 4, $P < 0.05$.

		Knee Extension MVC (Nm)				5RM Back Squat (kg)		Height (cm)	Body Mass (kg)
		Pre	Week 2	Week 4	Post	Pre	Post		
Intervention	Mean \pm SD	193.95 \pm 42.67	207.66 \pm 47.48	221.19 \pm 44.15*	234.88 \pm 53.50*#	60.52 \pm 20.71	84.24 \pm 22.74*	173.48 \pm 5.20	70.49 \pm 13.03
	Δ % from Pre (CI)		7.75% (-29.95 to 2.54)	15.31% (-42.41 to -12.06)	21.81% (-58.43 to -23.43)	30.56% (-22.80 to -16.34)			
Control	Mean \pm SD	195.92 \pm 41.77	200.5 \pm 42.98	200.13 \pm 41.83	201.20 \pm 42.04	53.13 \pm 12.40	54.61 \pm 12.28	175.52 \pm 7.64	66.91 \pm 12.66
	Δ % from Pre (CI)		2.70% (-16.04 to 6.37)	2.64% (-16.72 to 7.83)	3.11% (-17.80 to 6.65)	3.03% (-5.13 to 2.02)			

3.4.2 Contractile properties:

Analysis of VL Dm showed a significant time effect [$F_{(2.68, 101.7)} = 6.17, P < 0.01$] across both groups however, a significant interaction effect [$F_{(3, 114)} = 3.811, P = 0.01$] revealed only the intervention group to significantly reduce VL Dm by week 2 ($P < 0.001, ES = 0.86, 95\% CI: 0.62$ to 2.02). This reduction in VL Dm was also maintained at week 4 ($P = 0.02, ES = 0.82, 95\% CI: 0.11$ to 2.06), and at post ($P = 0.01, ES = 0.73, 95\% CI: 0.26$ to 2.01) (figure 3.2A).

Similarly for RF Dm, a significant time effect [$F_{(2.43, 92.16)} = 3.10, P = 0.04$] was observed across both groups, however a significant [$F_{(3, 114)} = 3.08, P = 0.03$] interaction effect showing only the intervention group to significantly reduced RF Dm by week 2 ($P = 0.003, ES = 0.53, 95\% CI: 0.47$ to 2.55). This reduction was maintained at post-intervention ($P = 0.004, ES = 0.67, 95\% CI: 0.54$ to 3.26), but not at week 4 ($P = 0.17, ES = 0.46, 95\% CI: -0.39$ to 3.03) (figure 3.2B).

Neither group significantly changed VL Tc over time [$F_{(2.37, 90.01)} = 2.17, P = 0.11$], nor was any significant interaction effect found [$F_{(3, 114)} = 2.48, P = 0.07$] (figure 3.2C). Similarly, neither group significantly changed RF Tc over time [$F_{(2.71, 102.9)} = 1.42, P = 0.24$], nor was any significant interaction effect found [$F_{(3, 114)} = 2.17, P = 0.10$] (figure 3.2D).

3.4.3 Muscle architecture:

Muscle architecture data is presented in table 3.3. For VL muscle thickness, a significant time effect [$F_{(2.87, 109.2)} = 27.49, P < 0.0001$] was observed across both groups, however a significant interaction effect [$F_{(3, 114)} = 14.62, P < 0.0001$] revealed only the intervention group to significantly increase VL muscle thickness by week 4 ($P = 0.002, ES = 0.39, 95\% CI: -0.24$ to -0.05), and then a further increase week 4-post ($P < 0.0001, ES = 0.52, 95\% CI: -0.29$ to -0.13). Similarly, for RF muscle thickness, a main time effect [$F_{(2.70, 102.60)} = 15.31, P < 0.0001$] was observed across both groups. However a significant interaction effect [$F_{(1, 114)} = 7.44, P < 0.001$] revealed only the intervention group to significantly increase RF muscle thickness pre-post ($P < 0.0001, ES = 0.88, 95\% CI: -0.41$ to -0.20).

Whilst a time effect [$F_{(2.42, 91.82)} = 29.04, P < 0.0001$] was observed across both groups for VL pennation angle, a significant interaction effect [$F_{(3, 114)} = 24.37, P < 0.0001$] revealed only the intervention group to increase VL pennation angle pre-post ($P < 0.0001, ES = 1.29, 95\% CI: -3.46$ to -1.76). Similarly, for RF pennation angle, a time effect [$F_{(1.78, 67.44)} = 11.14, P < 0.001$] was observed across both groups. However a significant [$F_{(3, 114)} = 13.86, P < 0.0001$] interaction effect revealed only the intervention group to significantly increase RF pennation angle pre-post ($P < 0.001, ES = 1.09, 95\% CI: -3.35$ to -0.99).

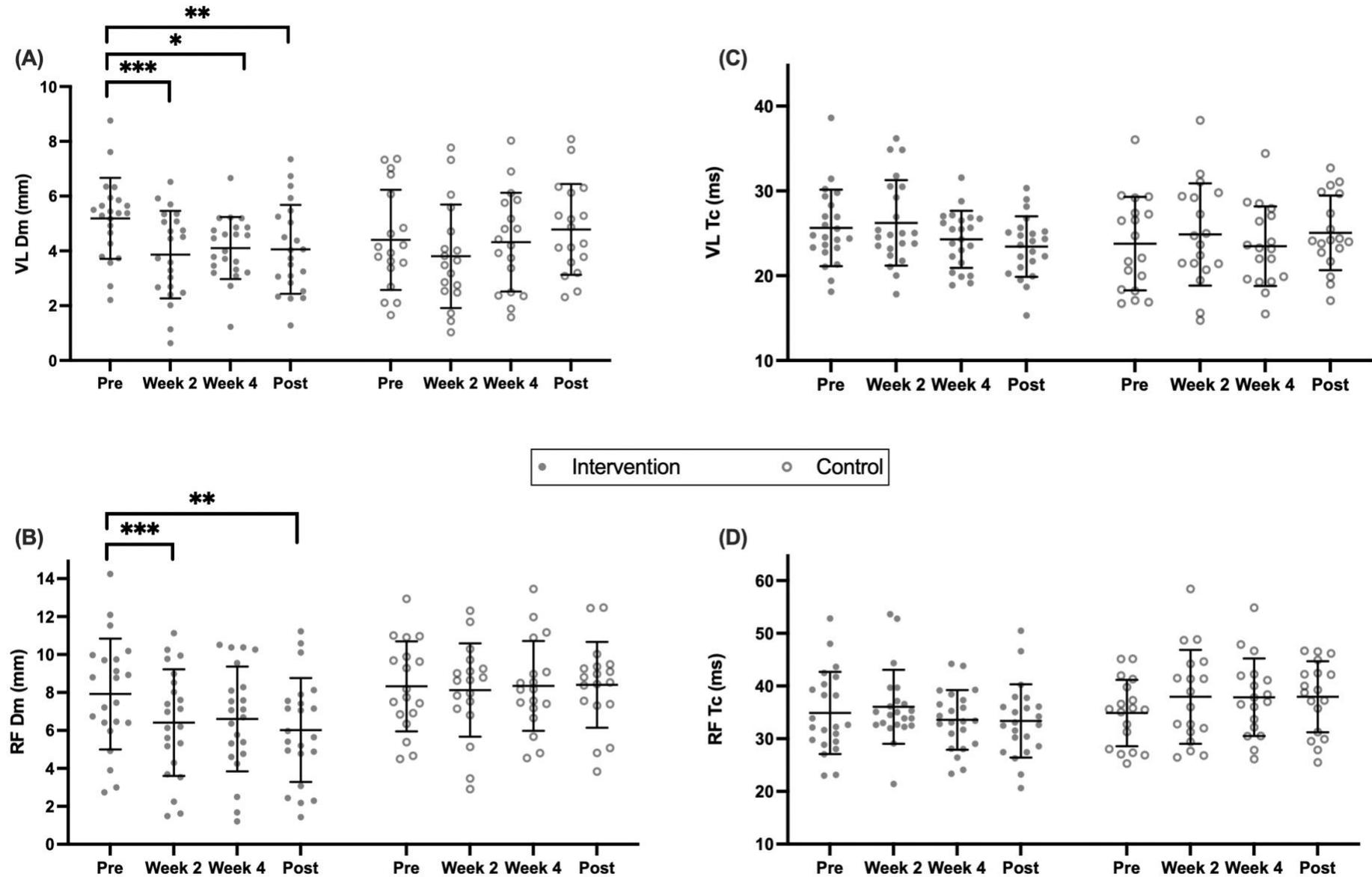


Figure 3.2; Time-course measures of contractile properties including; Dm (radial muscle displacement) in the vastus lateralis (VL) (A) and in the rectus femoris (RF) (B); Tc (contraction time) in the VL (C) and RF (D), by group. Individual responses are shown in filled and outline circles, bars display mean \pm SD; * - significant decrease from pre, $P < 0.05$; ** - significant decrease from pre, $P < 0.01$; *** - significant decrease from pre, $P < 0.001$.

Table 3.3; *Muscle architecture measures* of the vastus lateralis (VL) and rectus femoris (RF) assessed over 6-week training programme; values are mean \pm SD; * - significant increase from baseline; # - significant increase from week 4, $P < 0.05$.

	Intervention Group						Control Group					
	Pennation Angle (°)		Muscle Thickness (cm)		Adiposity thickness (cm)		Pennation Angle (°)		Muscle Thickness (cm)		Adiposity thickness (cm)	
	VL	RF	VL	RF	VL	RF	VL	RF	VL	RF	VL	RF
Pre	19.04 \pm 2.08	16.54 \pm 1.91	2.29 \pm 0.34	2.27 \pm 0.37	0.65 \pm 0.26	1.10 \pm 0.46	18.74 \pm 2.55	16.02 \pm 2.97	2.18 \pm 0.41	2.16 \pm 0.49	0.62 \pm 0.30	0.99 \pm 0.47
Week 2	19.01 \pm 2.14	16.68 \pm 1.80	2.34 \pm 0.34	2.32 \pm 0.37	0.61 \pm 0.25	1.14 \pm 0.47	18.97 \pm 2.59	16.16 \pm 2.69	2.23 \pm 0.39	2.27 \pm 0.46	0.62 \pm 0.29	1.00 \pm 0.41
Week 4	19.69 \pm 2.15	16.99 \pm 1.59	2.43 \pm 0.39*	2.36 \pm 0.36	0.64 \pm 0.26	1.05 \pm 0.44	18.93 \pm 2.61	16.03 \pm 2.42	2.27 \pm 0.38	2.24 \pm 0.29	0.60 \pm 0.29	1.03 \pm 0.42
Post	21.65 \pm 1.98*	18.72 \pm 2.07*	2.64 \pm 0.41*#	2.58 \pm 0.32*	0.65 \pm 0.26	1.07 \pm 0.49	18.97 \pm 2.55	15.96 \pm 2.47	2.25 \pm 0.38	2.26 \pm 0.41	0.64 \pm 0.31	1.01 \pm 0.48

3.4.4 TMS:

Although a time effect [$F_{(2.51, 95.55)} = 8,12$, $P < 0.001$] was observed across both groups for corticospinal excitability (MEP amplitude), a significant interaction effect [$F_{(3, 114)} = 3.93$, $P = 0.01$] revealed only the intervention group to significantly increase corticospinal excitability by 16% at week 4 ($P = 0.027$, $ES = 0.61$, 95% CI: -20.66 to -0.95); with this increase maintained post-intervention (21%) ($P < 0.001$, $ES = 0.87$, 95% CI: -22.21 to -6.53) (figure 3.3A).

Unlike corticospinal excitability, corticospinal inhibition (cSP duration) did not significantly [$F_{(2.63, 99.93)} = 0.49$, $P = 0.67$] change over time, with no significant [$F_{(3, 114)} = 0.69$, $P = 0.56$] interaction effect found (figure 3.3B).

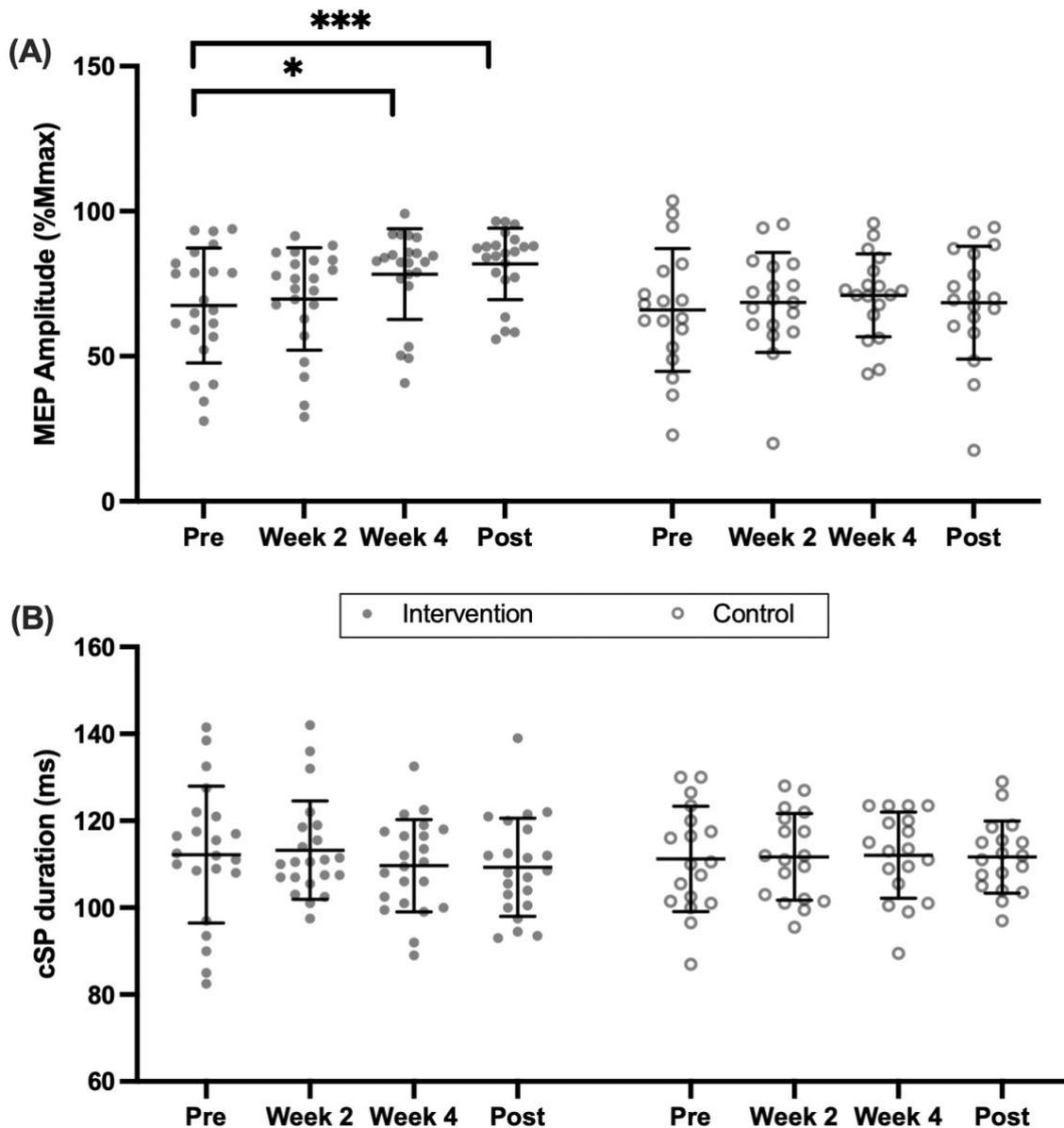


Figure 3.3; Time-course measures of corticospinal excitability (A) and corticospinal inhibition (B), assessed by Transcranial magnetic stimulation, by group. Individual responses are shown in filled and outline circles, bars display mean \pm SD; * - significant increase from pre, $P < 0.05$; ** - significant increase from pre, $P < 0.01$.

3.4.5 Motor unit firing rate

Despite the number of MUs identified at each time point being similar between groups (intervention - pre, 15 ± 6 ; week 2, 16 ± 5 ; week 4, 15 ± 5 ; post, 16 ± 4 ; control - pre, 16 ± 5 ; week 2, 15 ± 6 ; week 4, 16 ± 5 ; post, 16 ± 6), neither group showed a significant change in MU MFR over time [$F_{(2.49, 94.78)} = 2.70$, $P = 0.06$], and no significant interaction effect [$F_{(3, 114)} = 1.89$, $P = 0.14$] was found (table 3.4).

Table 3.4; Mean firing rates (MFR) for vastus lateralis motor units assessed during 60% MVC isometric trapezoid contraction, across 6-week training programme; pps – pulses per second; values are mean \pm SD.

	Mean firing rate (pps)			
	Pre	Week 2	Week 4	Post
Intervention	12.62	14.21	13.72	14.19
n	± 2.48	± 2.85	± 1.88	± 2.00
Control	12.83	12.78	13.53	13.19
	± 1.93	± 2.71	± 2.08	± 2.01

3.5 Discussion

Over the course of 6-weeks dynamic strength training, participants demonstrated increased 5RM BS strength. A decrease in VL and RF Dm was observed after 2-weeks of training and prior to any increase in isometric strength, neural adaptation or change in muscle architecture. After 4-weeks of training, an increase in maximal quadriceps strength was observed, and accompanied by, an increase in corticospinal excitability; however, no changes in MU MFR within the quadriceps were seen. Furthermore, an additional increase in maximal quadriceps strength was observed from week 4 to post-training, alongside increases in muscle thickness and pennation angle within quadriceps muscles.

The present study demonstrated increases in 5-rep max BS strength of the training group following the 6-week intervention. Whilst increases in 5-rep max BS strength may be subject to a technique-learning effect; increases in MVC torque observed at week 4 and then again week 4-post-intervention (table 3.1), represent increased strength of the quadriceps.

We observed increased quadriceps muscle tone and viscoelasticity of the muscle-tendon complex (Evetovich et al., 1997), as shown by a decrease in VL and RF Dm (figure 3.2A & 3.2B). Decreased quadriceps Dm before altered muscle architecture suggests a separate mechanistic change may be responsible for increased muscle tone. A recent study (Šimunič et al., 2019) demonstrated similar

decreases in Dm prior to changes in muscle architecture during a recovery intervention following atrophy, also supporting the notion of mechanisms other than architectural adaptation being responsible for the altered contractile properties. Previously, impaired excitation-contraction (E-C) coupling has been represented by reduced Dm and increased Tc accompanying increased limb girth following EIMD (Hunter et al., 2012). Unlike the aforementioned study however, no accompanying change in force output (table 3.2) or Tc (figure 3.2C & 3.2D) was observed in the present data, indicating E-C coupling was not impaired. The aforementioned may be explained by the present reduction in Dm being ~10% less than that of Hunter et al, indicating the less severe form of EIMD; liken to which commonly occurs in early stages of resistance training (Damas et al., 2015). Thus a plausible explanation for reduced Dm is the alteration of intra-muscular tissue fluid content (Kasuga, 2015) known to occur with EIMD in the early stages of resistance training (Chen et al., 2012). Such a non-invasive marker of contractile function could be useful for practitioners to gain objective insight into the efficacy of training interventions in their early stages. Future work should look to directly measure intra-muscular fluid content changes following strength training concurrently with changes in contractile properties, to provide precise mechanistic understanding.

Initial increases in muscular strength after 4-weeks of training were accompanied by neural adaptation in the form of increased MEP amplitude (figure 3.3A), partially confirming our secondary hypothesis. Increased MEP amplitude represents an increase in corticospinal excitability which includes the excitability of the M1 and the efficiency of descending volley transmission through the spinal cord and into the muscles (Di Lazzaro et al, 2004). Similar results have been previously shown using dynamic and isometric training interventions, whereby increased corticospinal excitability has accounted for early strength gain (Griffin and Cafarelli, 2007; Kidgell and Pearce, 2010; Leung et al., 2013b; Mason et al., 2017). Along with the findings of a recent meta-analysis (Siddique et al., 2020), the present data indicates that early-increases in muscular strength can be attributed to improved efficacy of neural transmission along the descending corticospinal tract. Interestingly, whilst corticospinal excitability remained increased (compared to baseline) at post-intervention assessment, there was no further increase from week 4 to post (figure 3.3A). There is limited data upon adaptation in corticospinal parameters after the initial increases presently observed as, by large, previous studies have employed 3-5 week training interventions (Kidgell et al., 2017; Siddique et al., 2020). However, present data would suggest corticospinal excitability may not increase further after the initial observed change but rather, remain elevated after 4 weeks of training. Future work investigating the time-course of corticospinal responses could look to precisely determine the nature of this 'ceiling effect' in corticospinal excitability and the influences of new training stimuli.

At odds with recent meta-analyses (Kidgell et al., 2017; Siddique et al., 2020) and our own secondary hypothesis was a lack in change of corticospinal inhibition (figure 3.3B). Recently, Ansdell et al. (2020) also demonstrated no change in cSP duration when assessing short-term training adaptations; however these authors also saw no change in MEP amplitude unlike the present data. Therefore, it is possible the presently observed results are not due to alterations in the M1 but rather somewhere else along the corticospinal tract. However, as TMS MEPs are unable to precisely differentiate between intra-cortical mechanisms (Brownstein et al., 2018) this cannot be confirmed. It should also be acknowledged that whilst shown to be a reliable method of analysis (Damron et al., 2008), cSP measurement does involve practitioner discretion as to when discernible EMG signal recommences following the silent period; holding a potential for variance. This Future work should explore techniques which allow such differentiation to be made within the corticospinal tract, such as stimulation of the cervico-medullary junction, to determine efficacy of corticospinal-motoneuronal synapses (Nuzzo et al., 2016).

In order to gain comprehensive and concurrent insight into early-resistance training adaptations we employed sEMG decomposition to infer upon MU discharge property adaptations (Rich and Cafarelli, 2000; Kamen and Knight, 2004). No change in MU MFR measured during 60%MVC isometric contraction was observed at any time point in either group (table 3.4). The present MFR data is at odds with a recent study by Del Vecchio et al. (2019) who observed increased MFR in the tibialis anterior after 4-weeks isometric training. Disparity between the present observations and that of the aforementioned study may be due to methodological differences such as the training-specific tests in which MFR was assessed and the specific equipment used to collect MFR data. In the present study we employed Delsys 4-pin Trigno electrode to collect EMG data for decomposition, whereas Del Vecchio et al used a high-density EMG grid (64-pin) which also employs a different algorithm for decomposition. A direct comparison between the two systems has yet to be conducted, with both having their own unique advantages; the 64-pin HD grid can sample a larger MU population and the Delsys 4-pin electrode employs artificial intelligence in its decomposition algorithm (De Luca et al., 2015b). Thus, it is still unclear on which system would be superior for MU decomposition in the context of training interventions.

Another methodological difference was that Del Vecchio et al pooled MFR data from multiple contractions whereas the present study did not. Pooling MFR data from multiple contractions has the potential to alter numbers of low-threshold (faster firing) MUs (Van Cutsem et al., 1998) and inadvertently influence MFR. Indeed, MU MFR analysed on a per-contraction basis (like the present study) suggests MFR is not altered post-training (Beck et al., 2011; Stock and Thompson,

2014;Sterczala et al., 2020). Furthermore, changes in adipose tissue thickness may arbitrarily alter MU properties derived from sEMG due to spatial filtering (Petrofsky, 2008). Thus, as a further measure of control, adipose tissue thickness at the VL and RF electrode site remained unchanged over the course of the present study (table 3.3). It is possible that resistance training presently altered the recruitment thresholds of MUs, or the degree of MU hypertrophy (Sterczala et al., 2020); however this cannot be confirmed as the relationships between MU property-recruitment thresholds were not measured. Therefore, in line with a previous suggestion (Contessa et al., 2016), future studies should look to these aforementioned relationships when investigating training induced changes in MU behaviour to obtain greater clarity along the recruitment threshold spectrum.

The later increase in MVC strength (week 4 to post) was accompanied by architectural adaptations in both the VL and RF muscle; namely increased pennation angle and muscle thickness (table 3.2). Previously, increases in muscle thickness and pennation angle have been demonstrated following similar timeframes of resistance training (Blazevich et al., 2003;DeFreitas et al., 2011;Damas et al., 2015), and have been shown contributory to increased capacity for maximal force production (Aagaard et al., 2001;Campbell et al., 2013). Presently, it was seen that muscle thickness increased in the VL prior to that of the RF (table 2); this inter-muscle difference possibly resulting from differing stimuli during training (Floyd, 2014). Previously, differences in hypertrophic response have been observed between VL and RF (Mangine et al., 2018), with differences in joint articulation involved in the exercises used in the present intervention suggested as explanatory; despite both muscles being controlled by the same innervation point (Page et al., 2019). Despite this inter-muscle difference in adaptation, the present data supports our initial hypothesis of muscle architecture enhancements accounting for strength gain in the latter stage of training, as VL muscle thickness also increased from week 4 to post-intervention. Interestingly, VL muscle thickness increased concurrently with corticospinal excitability after 4-weeks training, suggesting that neural and architectural adaptations may not be mutually exclusive. Additionally, corticospinal excitability remained elevated in the presence of further quadriceps architectural adaptation (week 4-post), supporting the cumulative effect of neural and architectural adaptations accounting for strength gain.

3.6 Conclusion

We have demonstrated that increased quadriceps muscle tone was observed in the early stages of training prior to strength gain and architectural adaptation. However precise mechanistic

understanding of this change in skeletal muscle contractile mechanics remains to be determined. Alongside increases in isometric and dynamic strength, we observed increases in corticospinal excitability but not in inhibition, nor in motor unit firing rate. The integrated assessment approach employed in the present study supports the consensus that early strength is attributed to changes in the neural physiology, however future work is needed to precisely determine the location of adaptation. The cumulative effects of neural and physiological adaptations observed here, provide practitioners with greater clarity as to the time-course of training-induced adaptations to improve the efficacy of training and rehabilitation monitoring and planning.

Chapter 4

Decomposition electromyography during a dynamic strength assessment: A pilot study

Matthew T Wilson, Lewis J Macgregor, Angus M Hunter

4.1 Abstract

Introduction: Investigations using decomposition electromyography (dEMG) to infer upon motor unit (MU) firing rate adaptations following strength training typically use isometric tasks in efforts to maintain a low-signal complexity of the EMG data. However, such isometric assessment is not typically task-specific to the training intervention employed, and therefore may not accurately reflect the respective neural adaptations believed to contribute to strength gain. In this report we outline a methodology to apply dEMG within a dynamic strength test typically used to assess training intervention outcomes. Furthermore, we describe the absolute and relative inter-day reliability of MU firing rate assessed during a dynamic task, as well as an application to infer upon potential MU firing rate adaptations following lower-limb strength training. **Methods:** The present data were collected as part of the study described in chapter 3. A randomly selected sub-sample of 17 completed all assessment protocols; 9 intervention (4 males/5 females, height; 172 ± 6.24 cm, weight; 74.8 ± 9.25 kg) completed 6-weeks lower-limb strength training, and 8 control (5males/3 females, height; 176.78 ± 7.61 cm, weight 71.99 ± 6.87 kg) performed no strength training but maintained habitual activity. Five rep max (5RM) back squat (BS) strength was assessed pre- and post-strength training. Average MU firing rate of vastus lateralis (VL) MUs were assessed across all 5 repetitions during the 5RM BS test ($MFR_{average}$), with pre- and post-training measures obtained during the same absolute load. Absolute (coefficient of variation (CV%) and relative (intra class correlation coefficient (ICCs) inter-day reliability of $MFR_{average}$ was also assessed. **Results:** dEMG assessment during the 5RM BS test revealed comparable MU yields (18 ± 8) and identification accuracies ($91.83 \pm 2.43\%$) with that of isometric assessments of the previous chapter, and within the literature. $MFR_{average}$ demonstrated excellent relative ($ICC = 0.87$ (0.34 to 0.97)), and moderate absolute ($CV = 13\%$) inter-day reliability, when assessed 6-weeks apart. However, whilst the intervention group increased their 5RM BS strength (26.71%, ES: 0.72, $p < 0.001$), there was no observed change in VL $MFR_{average}$ in either group ($p = 0.66$). **Conclusion:** We have demonstrated a methodology of applying dEMG within a dynamic, strength test commonly used to assess training intervention outcomes owing to its biomechanical similarities with elements of sporting performance. The observed MU yields and identification accuracies were comparable with that of isometric assessment, demonstrating the capability of dEMG to infer upon MU firing rates within dynamic movement. Additionally, strength training did not influence MU firing rate and therefore do not appear to contribute to the strength gains following training.

4.2 Introduction

In the previous chapter we assessed the potential for modification in behaviour of VL MUs across a period of 6-weeks lower-limb, dynamic strength training. Whilst modifications in corticospinal output were observed, no changes were observed in MU firing rate, suggesting the neural response to training arose from within the spinal circuitries and had no influence upon MU discharge rate. As previously discussed in chapter 1 (section 1.4.1.3) and chapter 3, the amount of force produced by a muscle is dependent on the rate of action potential discharge (firing rate) within a MU (Duchateau et al., 2006). Previously observed increases in MU firing rate following training (Van Cutsem et al., 1998; Vila-Chã et al., 2010), have been suggested to result from a net increase in excitatory input to the MU pool, and demonstrate the contribution MU firing rate makes towards training adaptations to increase strength.

As previously discussed, a potential limitation in chapter 3 was that the task used to assess MU firing rate via dEMG, was not specific to the dynamic training intervention employed, but rather involved an isometric contraction task. Neural adaptations can be considered a form of motor learning, as an individual learns to produce specific muscle recruitment strategies which are associated with optimal performance of the trained task (Carroll et al., 2001). This notion of task-specificity in neurophysiological testing has been highlighted, particularly when assessing corticospinal adaptations to strength training; with neural adaptations only evident when assessed in a task-specific manner (Beck et al., 2007; Schubert et al., 2008; Taube et al., 2020). Whilst the question of task-specific assessment of MU behaviour has received less attention, studies have used end-point isometric assessments (ranging from 20%-100% MVC (Hu et al., 2013) of MU firing rate, despite employing dynamic training interventions (Kamen and Knight, 2004; Stock and Thompson, 2014; Sterczala et al., 2020). Whilst the aforementioned studies proposed other MU behaviour changes to account for increases in strength (e.g. altered recruitment thresholds, MU hypertrophy), a recent study (Del Vecchio et al., 2019) found significant increases in MU firing rate to accompany strength gain, when assessed in a training-specific manner (isometrically). Considering the aforementioned findings, training-testing specificity may play a role in revealing alterations in MU firing rates in post-training assessments, therefore such transference should be considered when designing training studies investigating MU adaptations to strength training.

Notably, the current accepted method for assessing MU behaviours through dEMG is through the use of isometric, trapezoidal contractions which provide a steady-state force plateau from which MU

populations can be sampled (De Luca and Contessa, 2012). The steady state force plateau ensures the sEMG signal complexity remains relatively low as muscle fibres are kept near to, or at a constant length (De Luca et al., 2015a), increasing the likelihood of identifying MU through the decomposition process. However, muscles responsible for locomotion and sporting movements mostly contract anisometrically. In order to accurately reflect MU behavioural adaptations in such muscles, they should be assessed through dynamic contractions representative of the performance task for which they are recruited, thus considering the principles of training specificity. Owing to technological advancements in decomposition of sEMG signal, De Luca et al. (2015a) demonstrated the application of dEMG for characterising MU properties assessed during cyclic activity (repeated eccentric/concentric contractions), with levels of MU yield (~16-28) and identification accuracy (~90%) comparable to those obtained from isometric contractions. Whilst data surrounding the application of dEMG to dynamic movements are limited, the aforementioned authors analysed muscles of the upper (biceps brachii) and the lower (tibialis anterior & VL) body, observing similar MU yields and accuracies in all respective tasks. It was noted, however, that MU identification accuracy in particular, was increased during tasks of multiple repetitions of cyclic movement (walking, repeated bicep curls), owing to the pattern recognition elements of the decomposition algorithm (De Luca et al., 2015a). However, as no repeated measures were taken, absolute and relative inter-day reliability were not reported; for dynamic application of dEMG to be applied in intervention settings, this must be addressed.

The aforementioned study (De Luca et al., 2015a) also demonstrated the activation and recruitment of MUs during cyclic contractions was similar to that during isometric contractions, highlighting the potential of such application to infer upon adaptations in MU behaviours during dynamic contractions. In particular, the demonstrated effectiveness of dEMG during cyclic movements (De Luca et al., 2015a), suggests similar MU yields and identification accuracies may be obtained during a strength exercise of multiple repetitions. In the context of strength training, applying such assessment would allow practitioners to gain valuable insight into MU behaviours during the performance of strength training exercises and sub-maximal strength assessments. Such application could provide more in-depth knowledge of MU behavioural adaptations, than available from isometric end-point assessments currently investigated within the literature. Therefore, a logical step forward would be to explore the application of dEMG during a performance test commonly used to assess training-induced strength. Maximal repetition (rep max) strength tests are the most common form of dynamic strength assessment (Levinger et al., 2009), with submaximal rep max tests commonly employed (Gail and Künzell, 2014); owing to their reduced injury risk and to them

being less fatigue-inducing (Morales and Sobonya, 1996). In particular, 5-rep max (5RM) strength testing has been shown valid and reliable in both trained and untrained populations (Gail and Künzell, 2014).

Applying dEMG to such an exercise test could provide novel insight into MU behaviours during dynamic strength exercises, which are commonly employed due to their biomechanical similarities with elements of sporting performance. Aside from an application during strength training, dEMG application in other repetitive physical activity tests e.g., walking gait, hold significant potential to advance rehabilitation strategies and practitioner knowledge of MU activity during movement.

Therefore, the aim of this pilot study is to explore the application of dEMG with a dynamic performance test, the 5RM BS test which is commonly used to assess changes whole body strength, but with particular focus on the lower limbs, following a strength training programme. We will report on a dynamic dEMG method used to obtain MU firing rates at the start and end of a 6-week, dynamic training programme designed to increase lower limb strength, using a training-specific strength test. We will also assess the inter-day reliability of dynamic dEMG, as well as the MU yield and accuracy of decomposed, identified MUs from the sEMG signal.

4.3 Methods

4.3.1 Experimental design

The data from this chapter were collected as part of the study described in Chapter 3, and therefore the overall experimental design follows the details outlined in section 3.3.2. Pertaining to this chapter, participants underwent familiarisation of 5RM BS procedure during which participants were also familiarised with dEMG electrode placement and data collection. One week after familiarisation, participants repeated the same testing session where baseline measures (pre) of 5RM BS strength and dEMG were recorded. Following the completion of the 6-week lower body resistance training programme (training group only), participants repeated the 5RM testing session to obtain post-intervention measurements (post) of 5RM BS strength and dEMG variables. During post-intervention testing, dEMG data was collected under the same 5RM BS load achieved pre-intervention; this load was incorporated into the warm-up sets during post-intervention testing to minimise fatigue. During each 5RM testing session, sEMG was recorded during each 5RM attempt from the dominant quadriceps and saved for later offline analysis (see below). Before post-intervention testing, at least 72-hours rest was given from the final training session.

4.3.2 Participants

A sub-sample of 18 participants was randomly selected from the sample used in Chapter 3. Randomised number generation was used to obtain the sub-sample. Nine participants were selected from the training and control groups, respectively, however one participant from the control group withdrew due to injury sustained out-with the study. This left an overall sub-sample of 17 participants; 9 intervention (4 males/5 females, height; $172 \pm 6.24\text{cm}$, weight; $74.8 \pm 9.25\text{kg}$), and 8 control (5males/3 females, height; $176.78 \pm 7.61\text{cm}$, weight $71.99 \pm 6.87\text{kg}$). All participants met the same criteria as outlined in section 3.3.1 and were instructed to maintain their habitual activity levels over the course of the study.

4.3.3 Back squat 5-rep max test

Participants of both groups underwent the same 5RM BS testing procedure as outline in section 3.3.11. To enable measurement of any potential adaptations in MU properties post-intervention, the achieved pre-intervention 5RM load was prescribed into the warm-up protocol for post-intervention testing; enabling an absolute load comparison (table 4.1). The same RIR RPE scale was used as described in chapter 3. The pre-intervention 5RM load was the last warm up set (set 4) before the first attempt at a post-intervention 5RM load. sEMG data from the successful pre-intervention attempt, and post-intervention set 4 were taken forward for MFR analysis.

Table 4.1; *Protocol for 5-repetition max (5RM) back squat (BS) test.* Participants completed each BS warm-up set with a load prescribed based on a combination of perceived 5RM (familiarisation only) and rate of perceived exertion (RPE) from the previous set. In post-intervention testing, set 4 was prescribed as the participants pre-intervention 5RM (5RM_{pre}) load, in order to obtain post-intervention MFR data.

Set	Reps	Load
1	10	Bar (20kg)
2	8	50% of pMax (RPE 5)
3	6	70% of pMax (RPE 7)
4 (5RM_{pre})	5	90% of pMax (RPE 9)
5 (1 st 5RM attempt)	5	100% of pMax (RPE 10)
6 (2 nd 5RM attempt)	5	100% of pMax (RPE 10)
7 (3 rd 5RM attempt)	5	105% of pMax (RPE 10)

4.3.4 Decomposition EMG:

A Delsys Trigno Lab wireless system was used to obtain sEMG data for decomposition. A 4-pin surface electrode (Trigno-Galileo, Delsys, Natick) was fixed to the VL muscle belly (same location used for sEMG collection during the submaximal isometric trapezoid contraction, as described in section 3.3.10). Due to the movements of the knee joint during squatting, the reference end of the

electrode was fixed to the medial side of the quadriceps on manufacturer recommendations. The mono-articular VL was selected for investigation due to typically lower levels of subcutaneous tissue observed during pilot testing. This selection would also allow some comparisons to be drawn against isometric data collected in chapter 3. The VL muscle site was identified, shaved, lightly abraded and sanitised with alcohol wipes as per SENIEM guidelines (Hermens et al., 2000). The same process was conducted for the reference site. The EMG sensor location was recorded in reference to anatomical landmarks for replication in all subsequent testing sessions. The EMG sensor was secured to the skin using Tegaderm film (3M, UK) and micropore tape.

All dEMG data were collected under the same settings as described in section 3.3.10. Briefly, the same parameters were employed for calculating signal-to-noise ratio, low and high pass filtering, and the reconstruct-test analysis to determine firing rate accuracy (Nawab et al., 2010; De Luca and Contessa, 2011; De Luca et al., 2015b). Data were analysed on a participant-by-participant basis in order to limit the potential of reporting changes in MFR by altered numbers of low-threshold (faster firing) MUs measured post-training, rather than any true change in MU discharge properties (Sterczala et al., 2020).

From analysis of the action potential trains (MUAPTs) of each contraction, mean firing rate (MFR) in pulses per second (pps) were calculated for each participant across all 5 reps (MFR_{average}). MFR was calculated as the inverse of the average interspike interval across all 5 repetitions (figure 4.1). This is similar to the previous work of De Luca et al. (2015a) who analysed MFR during a walking task, quantifying MFR across an average of 8 strides. Due to the dynamic nature of the 5RM back squat test, it was not possible to measure recruitment threshold in a controlled manner as previously described for isometric testing, within the literature.

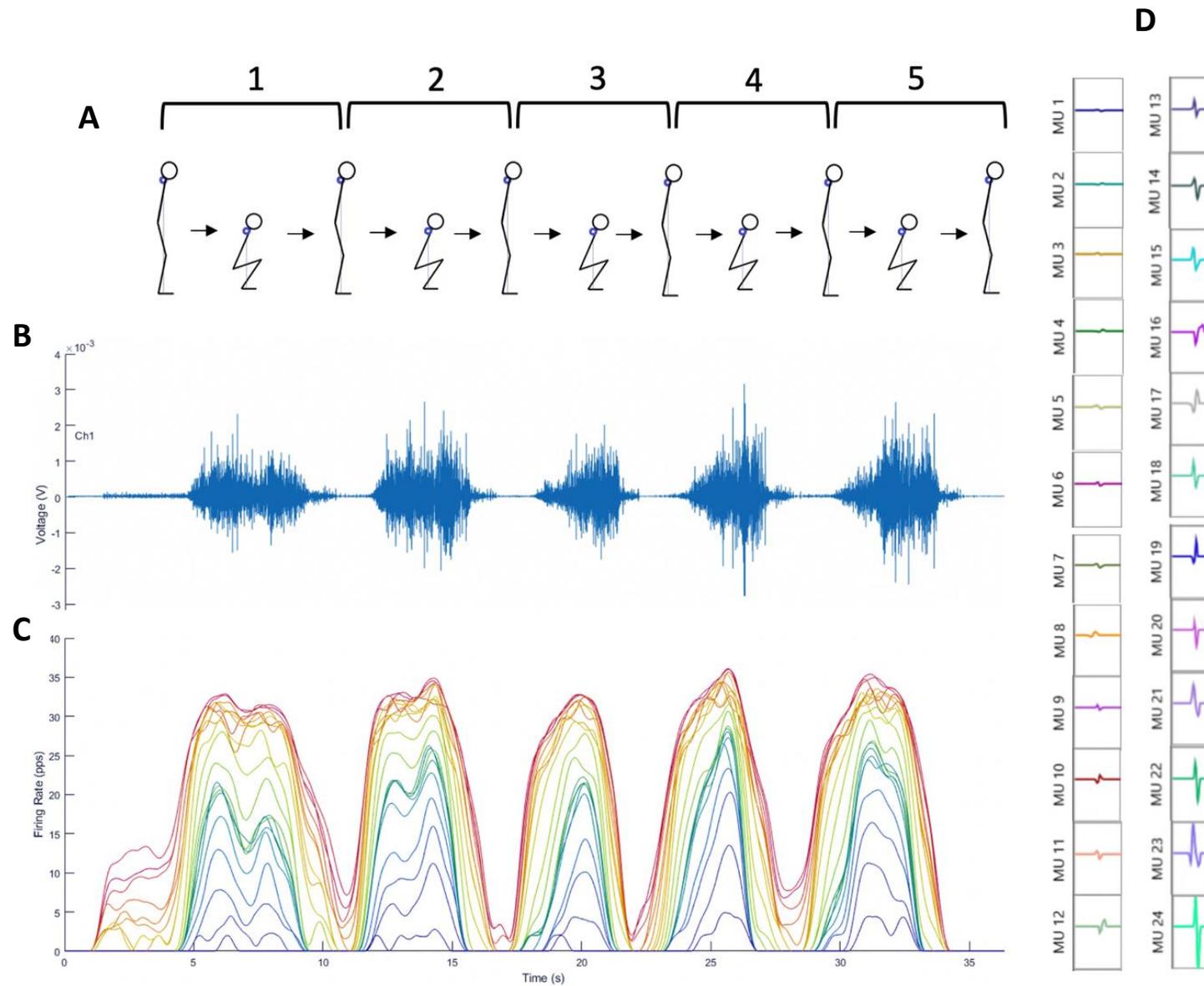


Figure 4.1; Decomposition of sEMG recorded from the VL from 1 participant during 5RM back squat test. **(A)** Schematic representation of cyclic nature of 5RM BS test, divided by single repetitions. **(B)** Raw sEMG signal collected from VL to undergo decomposition by the PDIII algorithm. **(C)** Mean firing rates of individually identified motor units (MUs) and **(D)** their MU action potential waveforms.

4.3.5 Training intervention

The same 6-week lower limb resistance training intervention was used as described in section 3.3.12, under the same supervisory conditions, and again using the RIR RPEs to inform upon exercise prescription loads (Zourdos et al., 2016) (Table 3.2). The achieved 5RM_{pre} was used to prescribe the BS loads in the training programme also.

4.3.6 Statistical analysis:

All statistical analysis was carried out using GraphPad Prism 8 (Graphpad software, CA, US). The distributions of baseline data sets (5RM load, MFR_{average}) were assessed for Gaussian distributions using Shapiro-Wilk normality test. To assess for pre-post changes in 5RM BS and MFR_{average}, separate two-way repeated measure ANOVAs were conducted (time; *Pre vs Post*, x group; *Intervention vs control*). Reliability measures of Intraclass coefficient of correlation (ICC) and coefficient of variation (CV) were calculated from control group data sets to determine test-retest reliability of MFR_{average}. ICC values were interpreted as follows: ≤ 0.39 = poor; 0.4-0.59 = fair; 0.6-0.74 = moderate; 0.75-1.0 = excellent (Cicchetti, 1994), with associated 95% confidence intervals (CI). Cohen's *d* effect sizes (ES) for changes in 5RM load were also calculated by; $d = (\text{Mean}_1 - \text{Mean}_2) / \text{SD}_{\text{pooled}}$, where $\text{SD}_{\text{pooled}} = \sqrt{[(\text{SD}_1^2 + \text{SD}_2^2) / 2]}$. ES are interpreted as follows: ≤ 0.5 = trivial, 0.5-1.25 = small, 1.25-1.9 = medium, ≥ 2.0 = large, for untrained participants (Rhea, 2004). All data are reported at mean \pm standard deviation (SD), with changes represented as percentage $\% \pm$ upper and lower 95% confidence intervals (CI). Alpha was set at $P < 0.05$.

4.4 Results

4.4.1 Comparisons Pre – Post intervention:

Whilst a main time effect [$F_{(1,15)} = 91.70, p < 0.0001$] was observed for 5RM BS load following training, a significant interaction effect [$F_{(1,15)} = 45.08, p < 0.0001$] showed that only the intervention group significantly increased their 5RM BS strength ($p < 0.0001$, ES = 0.72, 95%CI: 20.22 to 13.22); with no change in the control group ($p = 0.1314$, ES = 0.23, 95%CI: -6.65 to 0.77) (table 4.2). Post-intervention 5RM BS strength was also significantly higher in the intervention group, compared to control ($p = 0.02$, ES = 1.28, 95%CI: 2.93 to 46.49).

Table 4.2; Changes in 5 rep max back squat (BS) strength after 6-weeks lower-limb strength training. All data presented as mean \pm SD; CI, 95% confidence limits; * - significant increase, $p < 0.05$.

5RM Back Squat (kg)			
	Pre	Post	% change (CI)
Intervention	66.11 \pm 22.19	82.82 \pm 24.18	26.71% * (-20.22 to - 13.22)
Control	55.19 \pm 12.82	58.13 \pm 12.59	5.67% (-6.65 to 0.77)

For MFR_{average} changes pre-post intervention, no main effects for time [$F_{(1,15)} = 0.20$, $p = 0.66$] or between-group interaction [$F_{(1,15)} = 0.92$, $p = 0.35$] were found, indicating no change in MFR_{average} occurred in either group (figure 4.2): intervention group pre – 7.46 ± 2.92 pps; post – 8.59 ± 2.15 pps, ES = 0.44; control group pre – 9.58 ± 3.16 pps; post – 9.17 ± 2.95 pps, ES = 0.13).

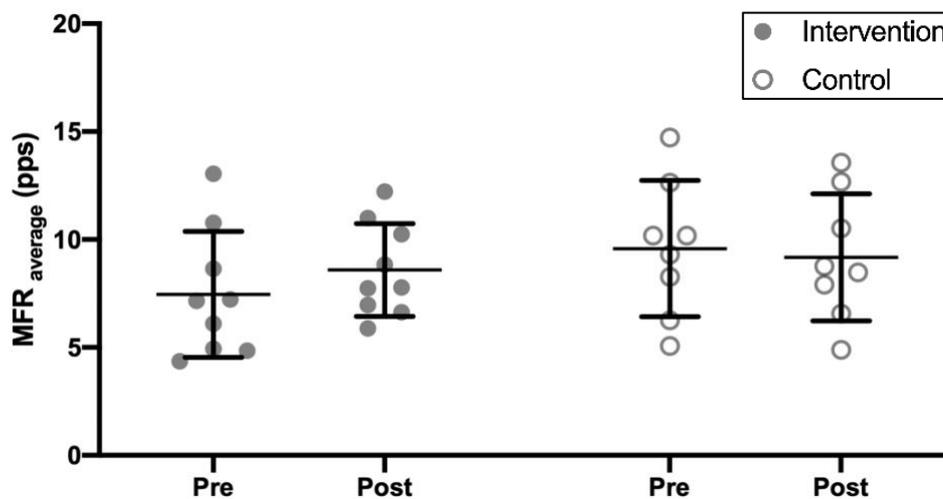


Figure 4.2; Mean firing rate (MFR_{average}) of vastus lateralis motor units before and after 6-weeks resistance training, separated by group. Individual data points represented as filled and outlined circles, bars display mean \pm SD.

4.4.2 Characteristics of dEMG procedure

For the 5-rep max BS test (figure 4.1), the average MU yield per participant ($n=17$) was 18 ± 8 with accuracies of $91.83 \pm 2.43\%$ following the DSDC protocol.

When separated up by training group, the number of identified MUs did not differ between groups [$F_{(1,15)} = 0.61$, $p = 0.45$] or over time [$F_{(1,15)} = 0.29$, $p = 0.60$] (figure 4.3A): Intervention pre – 16.44 ± 9.68 ; post – 19.22 ± 9.07 ; control pre – 20.38 ± 6.72 ; post – 19.88 ± 9.25 . Similarly, the accuracy of

identified MUs in each group did not differ between groups [$F_{(1,15)} = 2.43, p=0.14$] or over time [$F_{(1,15)} = 0.02, p=0.90$] (figure 4.3B): Intervention pre – $91.39 \pm 2.92\%$; post – $92.46 \pm 2.99\%$; Control pre – $92.33 \pm 1.79\%$; post – $91.06 \pm 0.73\%$.

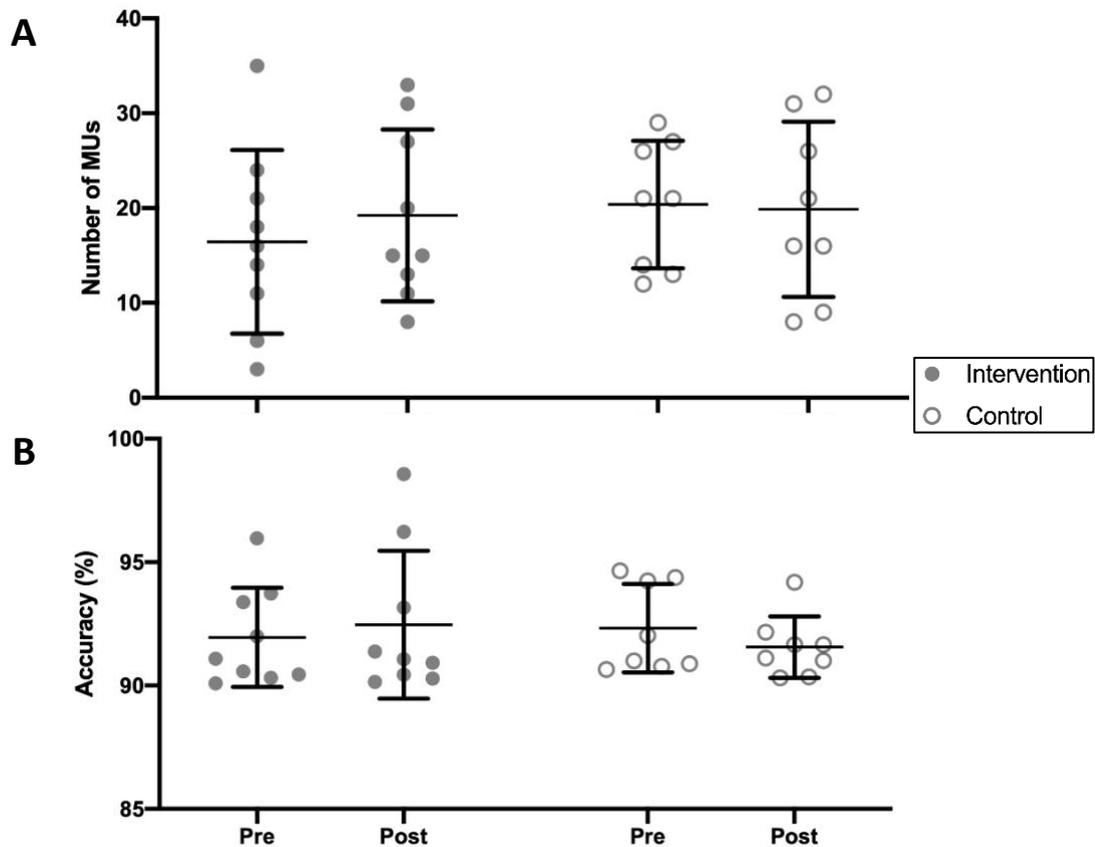


Figure 4.3; Characteristics of decomposition EMG data obtained during 5-rep max back squat (BS) test; (A) number of identified motor units (MUs) and (B) Accuracy of identified MUs by the PDIII algorithm. Individual data points represented as filled and outlined circles, bars display mean \pm SD.

4.4.3 Long-term stability:

Test-retest reliability of VL $MFR_{average}$ was assessed using data from the control group pre and post intervention ($n=9$). Using a Two-way mixed model with absolute agreement, $MFR_{average}$ showed excellent long-term stability; ICC = 0.87 (0.34 to 0.97), and a moderate CV of 13%.

4.5 Discussion

We have demonstrated the potential application of dEMG to infer upon MU behaviours during a dynamic, cyclic performance test, designed to assess lower-limb strength; by showing that dEMG is able to identify high numbers of MUs based upon MUAPT identification, with a sufficient level of accuracy ($> 90\%$). Furthermore, we also demonstrated a high level of relative inter-day

reliability for dEMG application to a 5RM BS test, however absolute reliability was moderate. Whilst participants of the training group displayed significant increases in 5RM BS strength following the training intervention, there was no accompanying change in MFR_{average} of VL MUs when assessed in the dynamic task; this being reflective of the findings in chapter 3 when isometric assessment was employed.

The application of dEMG was demonstrated by assessing MU behaviours during dynamic movement, whereupon we measured MFR_{average} before and after a 6-week strength training intervention, using a commonly employed dynamic strength test. Whilst participants of the intervention group displayed increased 5RM BS strength, there was no accompanying increase in VL MFR_{average} measured during the training-specific test. Potential explanations for the observed increases in strength are discussed in the previous chapter (section 3.4); however, the present data suggests that MU firing rate changes are not contributory to increased muscular strength even when assessed in a training-specific task. Previous studies employing similar dynamic training interventions also observed no increase in MU MFR but did see alterations in the MUAP amplitude-recruitment threshold relationships of contributory muscles (Pope et al., 2016; Sterczala et al., 2020). Both of the aforementioned studies observed increases in slope coefficients of the aforementioned relationship, indicating an increase in MU hypertrophy and specifically within larger MUs at the upper end of the recruitment threshold spectrum (Sterczala et al., 2020). Whilst it was not possible to measure recruitment threshold of VL MUs within the present study, the force required by a muscle to maintain a fixed load during an anisometric contraction is related to joint angle (De Luca et al., 2015a). Indeed, the aforementioned authors found the same inverse linear relationship when regressing MU firing rate against recruitment threshold as has been found in isometric tasks (Harmon et al., 2019); and changed following strength training, allowing bigger MUs to be recruited earlier on the recruitment threshold spectrum (Patten and Kamen, 2000; Del Vecchio et al., 2019). As De Luca et al. (2015a) observed no difference in MU recruitment order during concentric and eccentric muscle contractions, integration of knee angle measurements may allow further in-depth analysis into strength training-induced changes in MU behaviour within dynamic movements. Thus, a future solution could be to assess the angle of MU recruitment via motion capture or goniometry assessment knee angle during dynamic strength tests.

We observed an average MU yield of 18 ± 8 per participant over the course of the study, with no difference between intervention and control groups, or between pre- and post-intervention testing. The presently observed MU yield is comparable to that of the isometric assessment of the previous chapter (16 ± 6), and previous studies using dEMG to infer upon VL MU behaviours training

intervention settings (16-28 MUs) (Stock and Thompson, 2014;Pope et al., 2016;Sterczala et al., 2020); however it should be noted these aforementioned studies employed isometric testing when assessing MU behaviours. Such isometric testing will likely present slightly higher MU yield and MUAPT accuracies due to the lower sEMG signal complexity comparative to that of dynamic contractions (De Luca et al., 2015a). To date, the only other study to apply dEMG to a dynamic exercise task, involving lower limbs muscles, was De Luca et al. (2015a). These authors recorded a comparable MU yield (18 ± 8) and accuracy level ($92.2 \pm 3.3\%$) in the tibialis anterior muscle during a walking gait task with 8 contraction cycles (40 steps/second frequency). Whilst De Luca et al. also measured VL MU activity with dEMG during the same task, the MU yield (13 ± 9) and degree of accuracy ($86.4 \pm 3.1\%$) was slightly lower in comparison to the present data. Potential reasons for this disparity in VL MU data could be due to differences in decomposition algorithms (the presently used algorithm being updated for specific dynamic application), or differences in frequency of movement; with higher movement frequency believed to negatively impact upon MU yield and MUAPT accuracy. Indeed, when De Luca et al. (2015a) conducted the same gait task at a higher frequency (60 steps/second), MU yield and MUAPT accuracy declined in both the tibialis anterior (11 ± 5 ; $90.5 \pm 3.2\%$) and VL (10 ± 7 ; $85.1 \pm 3.8\%$) muscles. Therefore, it would appear that movement frequency is a key consideration when applying dEMG to dynamic tasks. In the context of rep max strength testing such as that used here, measurement of bar-path velocity could be a solution to ensuring a specified movement frequency is maintained/not exceeded.

The process of using dEMG during the 5RM BS test was shown to have excellent inter-day reliability (ICC = 0.87) and moderate absolute reliability (CV= 13%), when assessed 6-weeks apart in the control group. To date, no study has reported any comparable long-term reliability for dEMG application within dynamic movements, however the presently observed reliability data is comparable to previously studies using isometric tasks (Martinez-Valdes et al., 2016;Colquhoun et al., 2018). It should be noted at this stage; no measurement of movement velocity was collected during this study. The replication of electrode placement, and variables of the 5RM BS test (foot placement, squat depth etc.) were tightly controlled and during each testing session, however identification of MUAPTs can be influenced by the frequency of movement (De Luca et al., 2015a), thereby potentially influencing reliability. Whilst we observed 'excellent' relative, and 'moderate' absolute inter-day reliability, future investigations should consider the implementation of a movement velocity control (e.g. metronome-pacing) to further ensure acceptable test-re-test reliability for dEMG in dynamic movement.

As mentioned, the addition of movement velocity control and knee angle measures during a strength test such as the 5RM BS, could improve the reliability of, and the information gained from dEMG application. The inclusion of such measures would also allow MU behaviours to be assessed between-repetitions, and within specific eccentric and concentric phases of those repetitions. Within BS research in particular, there has been a particular emphasis on sEMG analysis into the activity of contributory muscles during these respective phases of movement (Ebben and Jensen, 2002; McBride et al., 2010), allowing practitioners to improve their programming and rehabilitation strategies by improving exercise specificity and transfer. As we have presently demonstrated the effectiveness of inferring MU behaviour during a dynamic strength test involving the BS, future investigations could apply dEMG in such settings to further improve our understanding of MU behaviours within strength exercise movements. In addition, future studies should look to analysis both mono- and bi-articular muscles. Such understanding could be complemented by the inclusion of movement velocity, and joint-angle measurements to provide new insight into the interaction between MU behaviours and kinematic variables of such strength training exercises.

4.6 Conclusion

Here we have demonstrated the application of dEMG within a dynamic strength assessment test to infer upon MU behaviour within lower limb musculature. We have demonstrated this process of dEMG application in a dynamic exercise test provides data of comparable MU yield and accuracy with that of the previous chapter, and with previous studies employing both dynamic movement, and isometric tasks.

Furthermore we have shown high levels of long-term inter-day reliability for MU firing rate assessed during the 5RM BS task; this being comparable with previously reported data for dEMG in isometric assessments. We have also demonstrated the practical application of such a methodology in a simple strength training study, and made recommendations to guide future research focusing on the integration of dEMG with established measurement variables, in dynamic performance tests.

Chapter 5

Bayesian approach in quantifying the efficacy of three different training interventions upon physical performance.

Matthew T Wilson, Lewis J MacGregor, Angus M Hunter, David L Hamilton, Iain J Gallagher

5.1 Abstract

Introduction: In the settings of performance sport, practitioners require a high degree of confidence that the strength training interventions they employ will elicit the desired improvements in performance outcomes, within their athletes. In contrast to null-hypothesis significance testing (NHST), Bayesian inferencing would allow practitioners to answer the pertinent questions of; whether an athlete has improved their performance in a test; how big the improvement in performance is; and with what degree of confidence can we be sure the improvement is a meaningful one. Thus, the aim of this chapter is to demonstrate an application of Bayesian inferencing using a contextual example training study, comparing the effects of three different training exercises upon horizontal jump performance. **Methods:** The contextual example data were collected as part of the study described in chapter 2. Non-strength trained male participants (n=33) were randomly assigned to 1 of 3 single-exercise intervention groups (n=11 per group); back squat (BS), deadlift (DL), or hip thrust (HT). Participants completed a 6-week linearized training programme (2x per week), where the assigned exercise was the sole method of lower body training. Pre- and Post-training, horizontal jump distance was assessed in all participants. We applied Bayesian regression modelling to analyse the difference in horizontal jump performance between the three training groups, combining the likelihood of the data with established prior distributions. We incorporated a threshold of technical error (TE) within the posterior distributions of horizontal jump performance to provide a 'cut-off' of technical measurement variation; with meaningful changes in horizontal jump performance exceeding this TE threshold. **Results:** Following their respective training interventions, participants improved their horizontal jump performance in each group; BS – $0.08\text{m} \pm 0.01\text{m}$, HT – $0.08\text{m} \pm 0.05\text{m}$, DL – $0.015\text{m} \pm 0.09\text{m}$. The proportion of mean values exceeding the TE threshold for each group were; BS – 77.9%, HT – 78.8%, DL – 19.8%. **Conclusions:** The results indicated that BS and HT training were much more likely to improve horizontal jump performance above the level of TE, when compared to DL. The present application of Bayesian methodology demonstrates its inferential capacity in small-scale training studies where large effects are not expected. Furthermore, the outline analysis method provides interpretable statements for practitioners and coaches which can provide direct confidence as to their training programme decision.

5.2 Introduction

In the general introduction we reviewed the problem investigators have in sports science of estimating small effects from small samples that provide unreliable point estimates (Pereira et al., 2012; IntHout et al., 2015). In the context of performance sport, the use of NHST does not provide answers to practitioners' questions such as; whether an athlete has improved their physical performance in a test, or how big that improvement may be. *P*-values only inform on the probability that an improvement in performance is happening based on an assumption that nothing is happening. Thus, NHST does not offer as rich an inferential context as many scientist and practitioners would like (Aarts et al., 2011; Wasserstein et al., 2019) and results are frequently misinterpreted (Hoekstra et al., 2014; Greenland et al., 2016; Pernet, 2017; Amrhein et al., 2019).

As described in section 1.2, there has been movement away from NHST, and towards the estimation of effect sizes and the uncertainty around those estimates. Chapter one discusses how this manifested in the formation of MBI (reference) advocacy within sport science; however, was then later criticized for its sub-optimal mathematical basis and common mis-interpretation (reference).

As outlined in section 1.2.4.1, Bayesian inferencing may be an alternative approach to effect size inferencing which is more inferentially rich than traditional NHST (Borg et al., 2018) (i.e. it would tell us if a change in a performance variable has occurred following a strength training intervention, and with what magnitude). Furthermore, in combination with the use of an investigator defined threshold, as recently demonstrated by (Swinton et al., 2018); Bayesian inferencing can provide a level of certainty as to the effectiveness of a particular strength training intervention.

Thus, the aim of this chapter is to demonstrate an application of Bayesian inferencing in a simple strength training setting. The following sections will use a data set taken from Chapter 2 to demonstrate how Bayesian inferencing may be used to assess efficacy of strength training interventions aiming to improve a parameter of athletic performance. After introducing the example research question, this chapter will explain how Bayesian modelling was applied to analyse our data. The results pertaining to the example research question will then be discussed with reference to Bayesian inferences. Finally, the application of Bayesian modelling will be discussed and compared to NHST, along with future considerations for the use of Bayesian inferencing in similar contexts.

5.2.1 Contextual example: Strength training exercises for improving horizontal jump performance.

Efficacy of strength training interventions are often assessed using physical performance tests to quantify changes in strength or power (Paul and Nassis, 2015;Lockie et al., 2016;Sperlich et al., 2016). Jump testing is commonly used to assess lower limb strength and power capabilities (i.e., vertical and/or horizontal), with jump performance being considered a key indicator of athletic performance (Kuzmits and Adams, 2008;Schuster and Jones, 2016). Horizontal jump performance is primarily dependent upon forces produced by lower limb muscles at the instance of take-off (Krishnan et al., 2017). The distance achieved during horizontal jumping is dependent on the athlete's ability to transfer linear momentum of this force from the ground to peak horizontal acceleration of the body's centre of mass, which is also critical to attain high velocities over short distances (Brechue et al., 2010;Hudgins et al., 2013;Loturco et al., 2015a).

More specifically, horizontal jumping ability has been positively correlated with both 10 m sprint velocity (Maćkała et al., 2015), and 100 m sprint times (Loturco et al., 2015c), making it a meaningful parameter for coaches and practitioners to assess when looking to quantify lower-limb power production.

When designing a training programme with the aim to improve an element of athletic performance e.g. lower limb power, the specificity principle must be considered (Suchomel et al., 2018). This principle predicts that the closer the training intervention employed is to the requirements of the desired outcome (e.g. horizontal jump performance), the better the outcome will be due for efficient transfer strength gains (Hawley, 2008). The barbell BS and the barbell DL are commonly utilised in strength training to improve lower limb power; owing to their unique abilities to activate the lower limb musculature (Delgado et al., 2019). BS training has increased vertical and horizontal jumping scores with associated improvements in hip and knee extensor strength (Chelly et al., 2009), as well as contributory increases in core strength (Clark et al., 2016); whilst DL training induced increases in knee flexor rate of torque production have been positively associated with increased jumping height and distance (Thompson et al., 2015). Considering biomechanical similarities between the aforementioned exercises; the axial force vectors relative to the athlete within which the BS and DL are performed (vertical), are thought to enhance the positive translation of training gains across to comparable elements of athletic performance (Young, 2006;Randell et al., 2010). Thus, both exercises have been shown to improve jumping performance following strength training, however a direct comparison of their respective training adaptations upon horizontal jump performance has not yet been made.

Recent literature has proposed that such transference as described above, is maximised when the training exercise and desired athletic movement are of the same force vector (Loturco et al., 2018). Such discussions have increased the prevalence of barbell HT and it being classified as a ‘horizontally orientated’ exercise (Zweifel, 2017). The unique loading mechanics (Brazil et al., 2021) and hip extensor activation patterns (Contreras et al., 2015; Neto et al., 2019) during the HT have been suggested advantageous when training for horizontally orientated tasks; and as more reflective of horizontal force production in relation to the global frame (Zweifel et al., 2017). Therefore, presenting unique higher degrees of hip extensor activation compared to the BS and DL when at full hip extension (Andersen et al., 2018; Neto et al., 2019), and a unique orientation of force expression, training the HT exercise could elicit superior improvements in tasks such as horizontal jump distance.

Therefore, the aim of this study design is to compare effects of training vertical and horizontally ‘orientated’ exercises upon horizontal jump performance. Six-weeks of single exercise lower limb resistance training were performed in one of three modes – DL only, HT only or BS only. Specifically, we were interested in estimating the magnitude and associated uncertainty of horizontal jump performance changes following the three interventions. Based on the current literature, we hypothesised that increases in horizontal jump performance would occur from training all three exercises, but with the largest improvement resulting from HT training.

5.3 Methods

The data used in the present study were collected as part of chapter 2. For details on the experimental design, see section 2.3.2.

5.3.1 Participants

Forty-eight male participants volunteered to participate in the study (16 per training group from random assignment). Of the original 48 participants who volunteered, 33 participants completed all aspects of the study, this being 11 participants in each of the three training groups. For further participant details see section 2.3.1.

5.3.2 Horizontal jump testing

Horizontal jump displacement was measured using a fixed tape measure and straight ruler. Participants were positioned on the start line, lining the front of their shoes with the 0cm mark.

Participants were instructed to jump horizontally as far as possible. Participants were allowed to swing their arms during the jump. Participants had to land on both feet, with no secondary motions of correction, for the jump to be counted successful. A failed jump was determined if; the participant moved their feet forward after the initial landing and/or if participants lost their balance upon take-off or landing. The horizontal jump distance was taken as the distance from the 0cm mark to the back of the participant's heel closest to that start position. Participants completed a standardised warm-up protocol of cycling, dynamic stretches, and vertical jumps. Each participant was given three practice jumps, with 1 minute of rest in between attempts. Following this, participants completed 3 maximal effort jumps, with the best score subsequently recorded. In the case of any failed jump attempt, participants were allowed one additional attempt, in efforts to ensure participants completed 3 valid jumps.

5.3.3 Training intervention:

The resistance training intervention followed a linear periodization model to ensure a sufficient training stimulus was maintained across the 6 week duration (Fleck, 1999). Participants completed two training sessions per week, with each session encompassing only the allocated group's respective exercise. For further details on the training intervention, see section 2.3.7.

5.3.4 Bayesian statistical analysis:

We used Bayesian regression modelling with Stan (Carpenter et al., 2017) via the brms package (Bürkner, 2017) in R (R Development Core Team, 2010) to analyse the difference in horizontal jump performance between training programmes. Stan is a probabilistic programming language which uses Hamiltonian Markov Chain Monte Carlo (MCMC) with the No U-Turn sampler (NUTS) algorithm to generate the posterior distribution (Hoffman and Gelman, 2011).

5.3.4.1 Defining the Model and Priors:

Invoking maximum entropy (Lyon, 2013;McElreath, 2018) we assumed that the difference in horizontal jump pre-to-post training intervention is (approximately) normally distributed:

$$diff_{ij} \sim N(\mu_i, \sigma) \quad (1)$$

Where $diff_{ij}$ is the pre-to-post difference for individual j in training group i . The model shown in (1) is the likelihood function for our data. The likelihood function describes the generative process for the

data. In this case we believe that the differences in horizontal jump are normally distributed with some unknown mean per group (i) and a common standard deviation.

We then model the means for each group using a linear model:

$$\mu_i = \beta_0 + \beta_{1i} + \varepsilon_{ij} \quad (2)$$

Here μ_i is the group specific mean β_0 is the intercept term (here the mean for the reference group), β_{1i} represents the difference in means between group i and the reference group and ε_{ij} is the residual for each individual. Based on previous data showing statistically similar changes in horizontal jump distance following BS and HT training (Zweifel et al., 2017; Fitzpatrick et al., 2019) the DL group was set as the reference group. The location (mean, μ_i) of the distribution in equation (1) for each group is $\beta_0 + \beta_{1i}$ from equation (2). The residuals are assumed to be normally distributed with a mean of zero and an unknown standard deviation (σ) to be estimated from the data. This is a standard assumption in linear modelling. The Bayesian approach requires setting prior probability distributions over each parameter to be estimated. In the model above we are estimating three parameters - β_0 , β_1 and σ . The prior distributions set over these parameters should at least partly reflect our knowledge about the expected change in horizontal jump.

The intercept term (β_0), the mean of the reference group, was given a normally distributed prior with a mean set at the mean value of the pooled differences data and a standard deviation equal to the difference standard deviation multiplied by 3. We chose the value of 3 reflecting the recommendation from Rhea (2004) that a large effect size in untrained individuals over a strength training intervention was >2 . The 95% probability interval over this prior encompasses a change of approximately 0.5m in each direction. In Bayesian analysis this might be considered a weakly informative prior i.e. there is some information about what we expect for the value before we carry out analysis or collect data. In this context we can be rather confident, based on both physiological constraints and prior data that the difference across the training intervention will not be as large as the prior suggests. However the prior on β_0 allows the data to inform the posterior distribution, allows a physiologically credible probability space for parameter estimation and is wide enough to limit overfitting to the data at hand.

The β_1 term, representing the offsets for the other two groups from the DL mean, was given a normally distributed prior with a mean of zero and a standard deviation as above (i.e. difference

standard deviation multiplied by 3). We did not expect large differences from the reference (DL) group and wanted to allow for both increments and decrements in performance. This is also a weakly informative prior and in the current context this prior suggests that the other two groups will not be different from the DL group but does allow a considerable difference from the DL group should the data suggest this.

The residuals were assumed to be normally distributed with a mean of zero and an unknown standard deviation to be estimated from the data. The unknown standard deviation term, σ was given a half-t prior with shape parameter (degrees of freedom) of 4, a mean of 0 and standard deviation equal to the standard deviation of the differences, times 10. This large standard deviation (relative to the data) reflects our uncertainty about the value of the standard deviation of the differences. The half-t distribution constrains the values of σ to be positive as standard deviation cannot be negative. Thus, the model and priors used were Figure 5.1 & Figure 5.3A):

$$diff_{ij} \sim N(\beta_0 + \beta_{1i}, \sigma) \quad (3)$$

$$\beta_0 \sim N(\mu_{diff}, \sigma_{diff} \times 3) \quad (4)$$

$$\beta_{1i} \sim N(0, \sigma_{diff} \times 3) \quad (5)$$

$$\sigma \sim t_{half}(4, 0, \sigma_{diff} \times 10) \quad (6)$$

In summary these priors reflect our supposition that there would not be large effects between the training groups, nor effects in a particular direction i.e., any group could experience an increase or decrease in horizontal jump performance following the interventions. They also allow probability over physiologically plausible values but are wide enough to prevent overfitting or overly constraining the model.

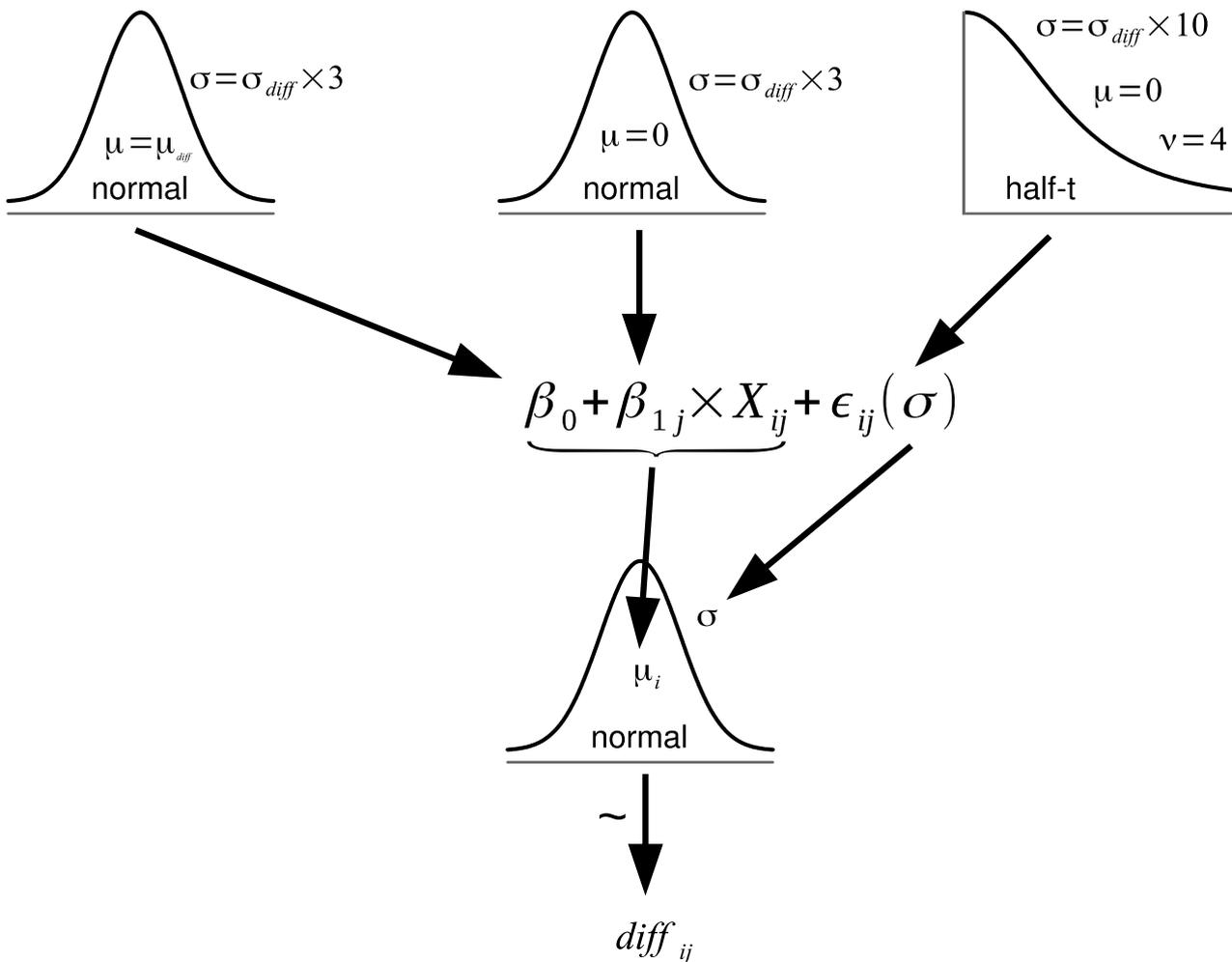


Figure 5.1: Graphical representation of the Bayesian model used in our analysis. The differences are modelled by a Normal distribution with a mean for each group composed of the intercept and slope coefficient from a linear model ($\beta_0 + \beta_1$). Each of these parameters have prior Normal distributions parameterized as in the figure. The prior for β_0 is wide on the scale of the data. The prior for β_1 allows for increments or decrements in performance and small effects. The residuals of the linear model are assumed normally distributed with a mean of zero and standard deviation (σ) to be estimated from the data. The σ term is modelled by a half-t distribution parameterised as suggested by Gelman et al (2013). Note these distributions are not to scale. The representation follows the style of Kruschke (Kruschke, 2015) and were made available by R. Bååth (<http://www.sumsar.net/blog/2013/10/diy-kruschke-style-diagrams/>).

5.3.4.2 *Generating posterior distributions:*

Bayesian modelling requires combining the prior distributions with the likelihood of the data to generate a posterior distribution. Analytical solutions are often unavailable and instead the posterior distribution is approximated by a sampling procedure. MCMC algorithms allow sampling from the posterior distribution even when we do not have a mathematical form for this distribution. Briefly, these algorithms begin with a random point on the posterior distribution and move to a second random point based on the probability of this second point. Repeating this process generates a Markov Chain - a directed random walk through the posterior distribution that leads to a chain of values from that distribution. This Markov Chain contains posterior values in proportion to their probability in the underlying distribution. Using Markov Chain to draw a large number of samples from the posterior allows us to use Monte Carlo integration to examine properties of the sample and characterise the posterior distribution. Characterising a distribution in practice usually involves more than one Markov Chain and discarding some number of initial samples (termed burn-in or warmup) as they may not be representative of the distribution. There are various MCMC algorithms and accessible introductions to MCMC are found in Kruschke (2015), McElreath (2018), and Lambert (2018)

In this study we used the R package brms (Bürkner, 2017) to carry out a Bayesian analysis of response to three different resistance training programmes. This package allows us to write models using the usual R syntax (Wilkinson and Rogers, 1973; Bates et al., 2014). The brms package provides an interface to the Stan probabilistic programming language (Carpenter et al., 2017) which uses Hamiltonian MCMC with the NUTS (Hoffman and Gelman, 2011) to generate the MCMC chains used to characterise the posterior distribution. After defining the model and priors above, the other values set in the call to brms were 4 MCMC chains with 10000 iterations each and a burn-in period of 500 iterations. We also set the thinning parameter to 5 to account for any autocorrelation in the MCMC chains. Following the creation of posterior distributions, we carried out post-hoc model checking using our generated posterior distributions to create new datasets to check the model fit; this is termed posterior predictive checking.

In summary, we used a Bayesian approach to estimate the mean change in horizontal jump performance for each training group and a common standard deviation. Bayesian methods require prior probability distributions over parameters of interest. We used prior distributions over the mean for each group and a common standard deviation (see section 5.2.4, Figure 5.1 & Figure 5.3A) with the aim of having a pragmatic trade-off between physiologically plausible values and a liberal

parameter space to explore. The posterior distributions were then characterised using Hamiltonian MCMC and the NUTS algorithm (Hoffman and Gelman, 2011). The advantage of the Bayesian approach is that it derives probability distributions rather than point estimates. Thus, we can directly characterise uncertainty around point estimates.

5.4 Results

Data for horizontal jump performance before and after the three resistance training programmes are displayed in Figure 5.2. Prior to the training programme, the horizontal jump mean (\pm SD) values for each group were $1.97\text{m} \pm 0.25\text{m}$, $1.92\text{m} \pm 0.17\text{m}$, $1.98\text{m} \pm 0.2\text{m}$ for the DL, HT, and BS groups respectively. After completing the training programme, horizontal jump the mean (\pm SD) distance jumped was $1.98\text{m} \pm 0.24\text{m}$, $2\text{m} \pm 0.19\text{m}$ and $2.06\text{m} \pm 0.2\text{m}$ for the DL, HT, and BS groups respectively (Figure 5.2A). The mean (\pm SD) changes in horizontal jump performance for each training group were; DL – $0.015\text{m} \pm 0.09\text{m}$ (6/11 participants improved) HT – $0.08\text{m} \pm 0.05\text{m}$ (11/11 participants improved); BS – $0.08\text{m} \pm 0.01\text{m}$ (10/11 participants improved) (Figure 5.2B).

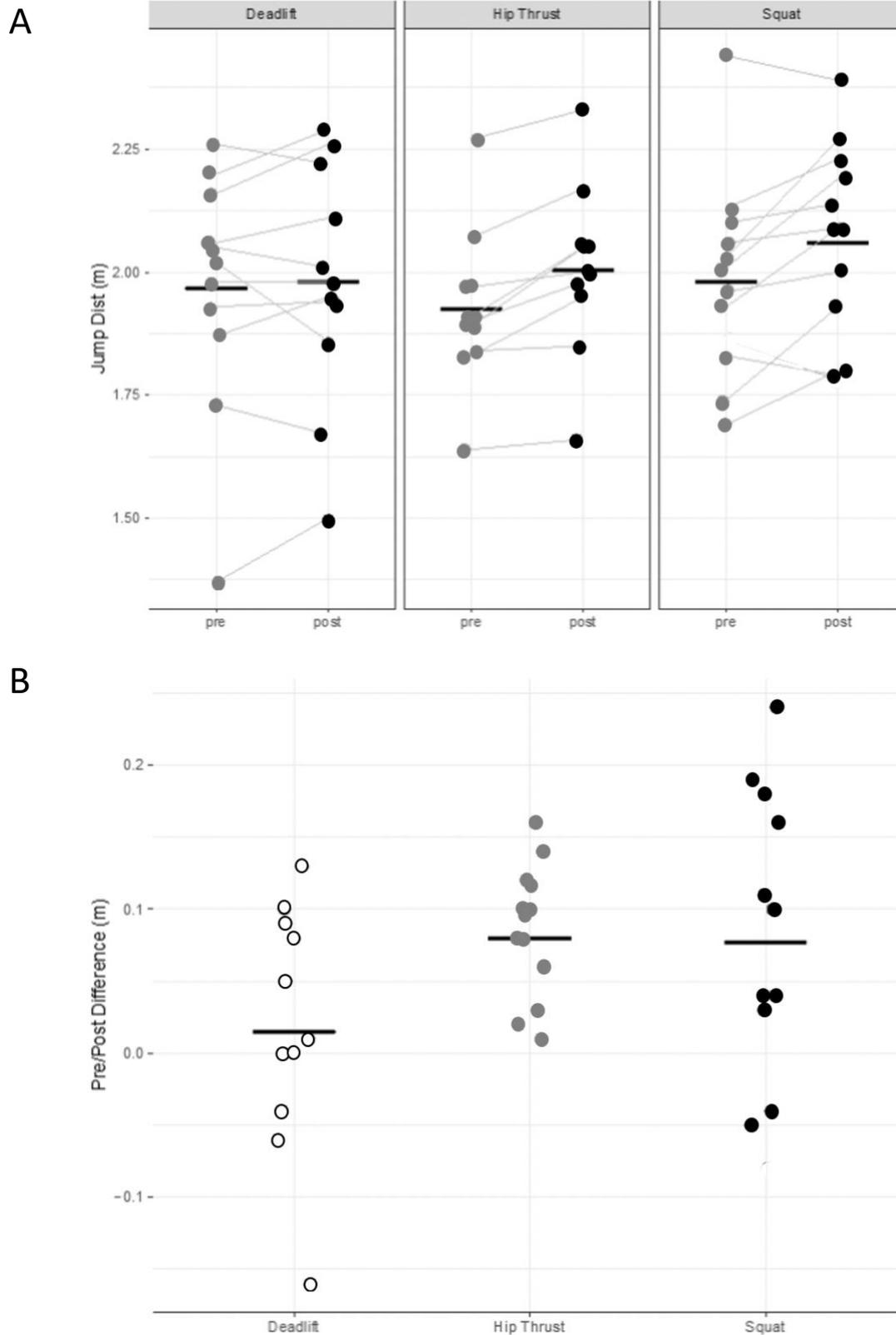


Figure 5.2: (A) Broad jump distance (m) before (grey) and after (black) a 6-week resistance training programme. The horizontal bars are the means of each data set and the grey lines show changes in each participant. (B) Differences in horizontal jump distance for the Deadlift (white), Hip Thrust (grey) and Squat (black) groups over the 6-week resistance training programme. The horizontal bars show the mean for each data set.

5.4.1 Bayesian analysis:

The Bayesian estimates for the means and common standard deviation are shown in Table 5.1 and estimates from the standard linear modelling procedure in 'R' are shown in Table 5.2. The estimated parameters are essentially identical in both approaches. The intercept term (the mean improvement of the DL group in meters, Table 5.1) is 0.016m, the HT group improved by 0.063m over the DL group, whilst the BS group improved by 0.06m over the DL group. The estimate for the common standard deviation was 0.09m. Using standard NHST to assess whether the HT and BS differences are statistically significant (compared to DL) results in a failure to reject the null hypothesis at the conventional $p \leq 0.05$ level (Table 5.2).

Table 5.1: *Estimates of effects for each training modality from the linear model with 95% highest density estimates (HDIs).* For the Deadlift group the estimate is the mean change in horizontal jump (m) over the training intervention. For the other two groups, the estimate represents the change (m) above the change for Deadlift. HDI 95% (Highest Density Estimate - 95%) is the interval encompassing the highest 95% of the posterior probability.

<i>Predictors</i>	Pre-post Difference (m)	
	<i>Estimates</i>	<i>HDI (95%)</i>
Intercept (Deadlift)	0.016	-0.03 to 0.06
Hip Thrust	0.063	-0.003 to 0.13
Squat	0.06	-0.002 to 0.12
Sigma	0.09	0.07 to 0.11

Table 5.2: *Estimates of effects for each training modality using the usual ordinary least squares, maximum likelihood linear model with 95% confidence intervals.* For the Deadlift group the estimate is the mean change in broad jump in meters over the training intervention. For the other two groups the estimate represents the change in meters above the change for Deadlift.

<i>Predictors</i>	Pre-post Difference (m)		
	<i>Estimates</i>	<i>95%CI</i>	<i>p</i>
Intercept (Deadlift)	0.015	-0.04 to 0.07	0.580
Hip Thrust	0.065	-0.01 to 0.14	0.102
Squat	0.062	-0.01 to 0.13	0.095
Sigma	0.09	NA	NA

To illustrate how Bayesian procedures reassign probability after combining prior probability and data the prior and posterior distributions are shown in Figure 5.3. Comparing the x-axis the posterior distributions (Figure 5.3B) are notably narrower than the prior distributions (Figure 5.3A) demonstrating the influence of the data on the estimates of the differences in each group and the standard deviation of the differences. Referring to Table 5.1 the Intercept (DL) estimate of 0.016 is the median of the Intercept (DL) posterior distribution in Figure 5.3B; the HT posterior estimate of 0.063 is the median of the HT posterior distribution in Figure 5.3B; the BS posterior estimate of 0.06 is the median of the BS posterior distribution in Figure 5.3B and the Sigma estimate of 0.09 is the median of the Sigma posterior distribution in Figure 5.3B.

These analyses directly address all three of our questions. The graphical examination of the data (Figure 5.2A & B) together with the Bayesian estimates of sizes (Table 5.1, Figure 5.3B) suggest that something is indeed happening; the Bayesian estimates of horizontal jump changes give us a sense of how large the effect is; we can also see directly from comparing the posterior distributions (Figure 5.3B) how confident we should be that there are differences between the groups. Notably, a conventional NHST and p -value approach (Table 5.2) would lead to a failure to reject the null hypothesis, which tells us no change has resulted from the training programmes; thus demonstrating NHST's inability to detect potentially meaningful within/between group differences.

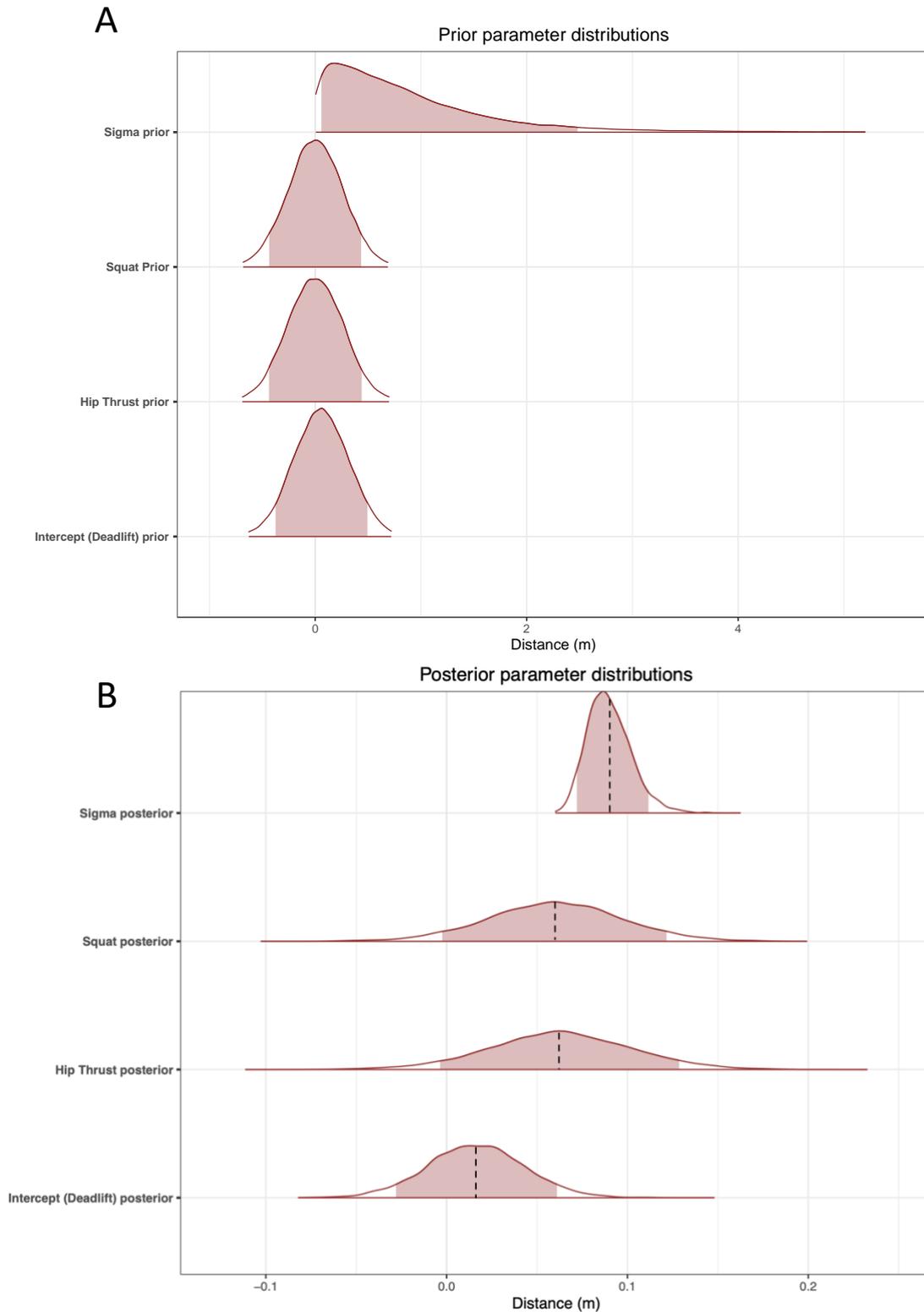


Figure 5.3: *Prior (A) and posterior (B) predictive distributions for each of the parameters estimated by the model.* The region spanning 95% of the probability is shaded. Dashed black lines represent median values (see table 5.1). The prior distributions could be considered wide on the scale of expected changes (see main text); e.g., the prior probability for each of the β coefficients has a 95% probability range of $\sim \pm 0.5$ m. After incorporating information from the data via Bayes theorem the posterior distributions for the β coefficients have 95% probability ranges of $\sim \pm 0.05$ - 0.01 m. The probability around the residual SD parameter (σ) also shrinks considerably.

5.4.2 Typical error:

The posterior distribution for each of the training groups allows us to directly estimate the probability of a response above a threshold (Swinton et al., 2018). We used the typical error (TE) (Hopkins, 2000) of the horizontal jump to operationalise a cut-off. A coefficient of variation (CV) of 2.4% has been previously established in untrained males (n=93) by Markovic et al. (2004). Due to the aforementioned, test-retest assessment of horizontal jump displacement was not necessary for our analysis. Future studies with less well-established CV parameters should consider the inclusion of test-retest assessments in order to accurately calculate TE. The equation used to calculate TE is provided by Swinton et al. (2018);

$$\widehat{TE} = (\bar{x} \times CV) \div 100$$

Where \widehat{TE} is the estimated typical error and \bar{x} is the baseline sample mean in our data. Using our baseline mean value of 1.96m and the CV value of 2.4% (Markovic et al., 2004) we calculated a horizontal jump \widehat{TE} value of 0.047m. We set our threshold to this \widehat{TE} , in essence defining the threshold at the estimated technical measurement variation. The posterior distributions for the total estimated horizontal jump distance after training for each group are shown in Figure 5.4A, with the accompanying 95% HDIs in Table 5.3. The probability of a change above the threshold is indicated by the proportion of each probability distribution to the right of the dashed line (representing the \widehat{TE} value of 0.047cm). The probability of a change above the threshold for the DL group was ~10%. For the HT and BS groups, the probability of a change above the threshold was ~86%. Thus, analysis of the posterior distribution directly informs us about how sure we should be that we are seeing an effect above TE in this study.

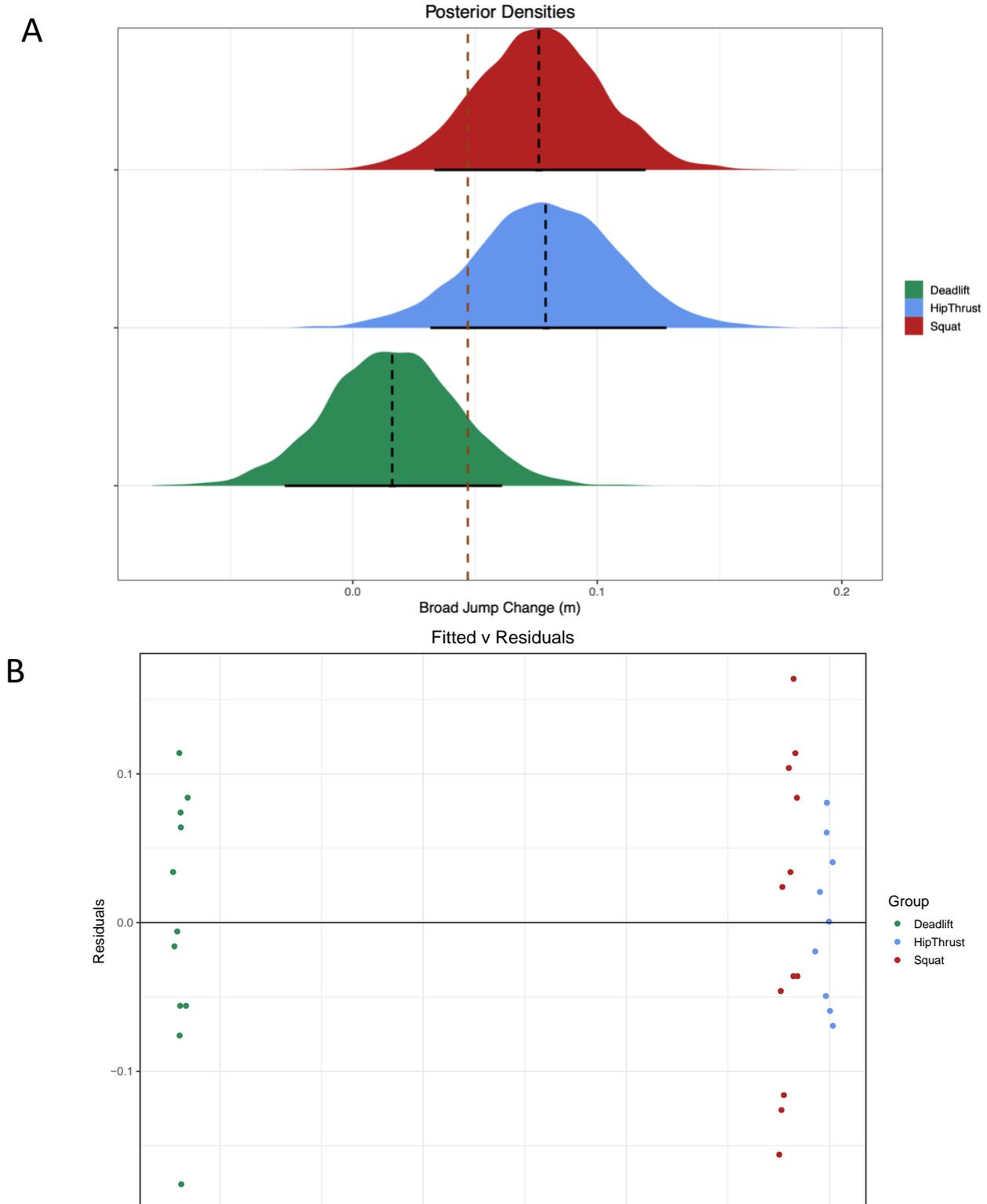


Figure 5.4: (A) Posterior densities for change in broad jump over the training intervention. Each posterior density is shown with the median value (dashed black line) and a 90% highest density interval (HDI) covering 90% of the probability in that density (black lines extending from median). The horizontal dashed lines show the TE above a value of 0. Note that for the Hip Thrust and Squat groups these plots show the intercept + effect i.e., the estimated total horizontal jump distance after training. (B) Fitted versus residual values for the model demonstrating that the model meets the assumption of normally distributed residuals around zero. The variance across the residuals is also similar for each group meeting the homogeneity assumption.

Table 5.3: Total SBJ difference (m) above zero for each group after 6 weeks resistance training and 95% highest density estimates (HDIs). The 95% HDIs show the range of values encompassing 95% of the probability in the density plots of Figure 5.4A. The intervals in the distance column are the sum of the mean for deadlift and the effect of training modality estimated from the model and seen in the Estimates column of Table 5.1.

Total Estimated Differences (m)		
Group	Estimated distance (m)	95% HDI (m)
Deadlift	0.015	-0.03 to 0.06
Hip Thrust	0.079	0.03 to 0.13
Squat	0.076	0.03 to 0.12

The standard assumptions for residuals in linear modelling apply to our data analysis. In Figure 5.4B we show that residuals are approximately normally distributed around zero and that residual variance for each group is approximately the same. There are also diagnostic checks for the Bayesian procedures we used. Appendices D & E illustrates that the MCMC chains used to sample from the posterior for each estimated parameter all converged (Appendix D); the Gelman-Rubin statistic \hat{R} for all parameters was also 1 (not shown) as recommended (Gelman and Shalizi, 2013). Autocorrelation amongst samples from the posterior distribution was minimal (Appendix E) suggesting that the MCMC samples we used to characterise each posterior distribution can be considered independent.

In summary, although standard NHST failed to reject the null hypothesis (Table 5.2) of a difference from the DL for HT or BS group a Bayesian approach allows a richer interpretation. There is in fact substantial probability HT and BS training improve horizontal jump performance above the TE of the horizontal jump, and we can also be reasonably sure that DL does not lead to a change in horizontal jump distance.

5.4.3 Model checking:

Model checking is an important part of analysis. The gold standard check for any model is to compare model performance to a new, independent dataset but we do not have such a dataset available. The posterior distribution can be used to generate new datasets and these simulations can be used to check model fit. Notably the posterior distribution incorporates both uncertainty around parameter estimates and sampling uncertainty. We sample values from the posterior distribution (i.e., mean changes and standard deviation in horizontal jump according to our model), simulate new study replicates from these samples and examine how well these simulated datasets match the actual realised dataset we observed. This is termed posterior predictive checking. If the model is a

reasonable reflection of the data generating process then the distribution of calculated statistics (called $T(y^{rep})$ by Gelman and Carlin (2014) from the replicated data should be centred around the data derived calculated statistics ($T(y)$). In particular the tail probabilities calculated from simulated samples, termed posterior predictive (or Bayesian) p -values, should be close to 50% (Gelman and Shalizi, 2013). Specifically we use the posterior predictive check as a goodness of fit measure for the model (Gelman and Shalizi, 2013). We generated 5000 replicated datasets (y^{rep}) from our posterior distribution and for each of these datasets we calculated mean in each training group as a test statistic, $T(y^{rep})$. As seen in Figure 5.5 the $T(y^{rep})$ values distribute evenly around the actual values from the study (black vertical lines in Figure 5.5) for each group. The proportion of the distribution above the black vertical lines in Figure 5.5 is 51%, 48.1% and 48.3% for DL, HT and BS respectively. These values suggest a model that reflects the data well.

It would be useful to have some idea of the results of a future study and an indication of the uncertainty around effect size estimates in future studies. The posterior predictive distributions can also be used to estimate the proportion of means we would see above the chosen threshold in repeat studies. Our \widehat{TE} value of 0.047m is shown in Figure 5.5 by the dashed vertical line on each histogram. The proportion of mean values for each training group exceeding this threshold were 19.8%, 78.8% and 77.9% for DL, HT and BS respectively. Again, these can be interpreted directly as probabilities meaning that in replications of this study, we would expect to see the mean of the DL group exceed the TE threshold ~20% of the time whilst for the HT and BS groups the mean would exceed the TE threshold ~80% of the time. Thus, we can be reasonably confident that future studies would likewise suggest improvements in horizontal jump performance exceeding the TE for both the HT and BS, but not for the DL.

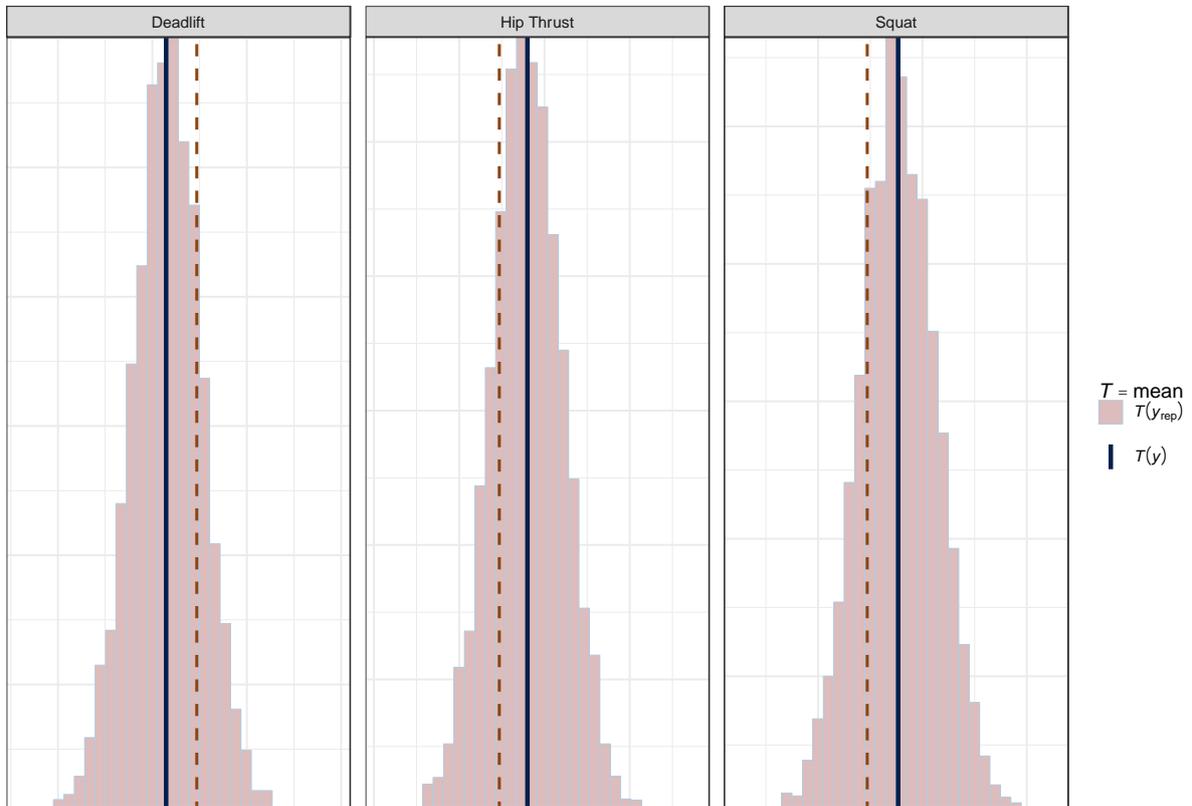


Figure 5.5: Posterior predictive data for horizontal jump change under three resistance training programmes. The black vertical line shows the mean jump difference across the intervention in each group from the actual data. The dashed line shows the TE (see main text). We simulated 5000 datasets from our posterior distribution and calculated the group means ($T(y_{rep})$) for each dataset. Each histogram shows the distribution of the 5000 simulated means from each group. The proportion of each histogram that lies above the black vertical line is the Bayesian p-value. In each case the mean from the actual data ($T(y)$) is approximately in the middle of the distribution as it should be if the model fits the actual data well. The proportion of each histogram above the dashed line represents the expected proportion of future datasets that would return a mean greater than the TE.

5.5 Discussion

We used a Bayesian approach to estimate the effectiveness of 3 different strength training exercises for improving horizontal jump performance and the uncertainty around the estimate, as well as the probability of the training effect exceeding the 'noise' in the measure (i.e., the TE). Our analysis demonstrates that given our model and the data we have to hand, improvements in horizontal jump performance were 78.8% and 77.9% likely to exceed the TE from HT and BS training, respectively, yet only 19.8% likely to exceed the TE from DL training. Changes in performance after DL training fall rather evenly around zero, whereas changes in performance after HT and BS are mostly greater than zero. These aforementioned findings address whether we are seeing an effect, our uncertainty around the effect, and give us a directly interpretable probability that we are really seeing an effect. Additionally, using posterior predictive checking, we have examined how well our model reflects the data generating process and have estimated that future studies would be very likely to produce similarly sized effects in each group. Notably the conclusions we would have drawn from a standard NHST based analysis would have been quite different using the conventional statistical cut-off of $p \leq 0.05$, where no training group would have displayed any significant improvement post-training.

5.5.1 *The data in context*

Contrary to our initial hypothesis, only the BS and HT training groups showed likely improvements in horizontal jump distance beyond the TE, and that training the DL alone did not (Figure 5.4A); suggesting an effective positive transfer of strength gains made from BS and HT training, across to horizontal jump performance.

Previously, increases in horizontal jump distance have been seen following BS (Zweifel et al., 2017) and HT training (Contreras et al., 2017; Fitzpatrick et al., 2019), supporting observations in the present study. Reasoning as to why BS training causes increased horizontal jump distance is centred around the BS's ability to elicit high concentric knee and hip extensor activation during training (Contreras et al., 2015; Delgado et al., 2019). Recently, Yokozawa et al. (2019) demonstrated how knee extensor force production during the propulsive phase of a horizontal jump, was positively correlated with jump distance; this relationship suggests increased knee extensor force production, and adaptation seen following BS training (Aagaard et al., 2002; Chelly et al., 2009), will lead to increased horizontal jump distance. Furthermore, an activity such as the BS can be considered biomechanically similar to activities such as horizontal jumping according to the theory of dynamic correspondence (DC), based on the direction of the ground reaction forces relative to the athlete being similar (Siff, 2001; Goodwin and Cleather, 2016; Fitzpatrick et al., 2019). Such biomechanical

similarities provide further reasoning for the task-specific transference of strength gains made through BS training.

The principles of DC can also provide explanation as to why HT training was observed effective at increasing horizontal jump distance. According to the DC theory, an exercise can be considered more mechanically specific to a task, if increased force production occurs within similar ranges of movement to the task (Goodwin and Cleather, 2016). In contrast to the BS, the HT has been shown to elicit higher degrees of hip extensor activation and force production as the hip approaches full extension (Contreras et al., 2013; Contreras et al., 2015; Neto et al., 2019); thus, providing a rationale for including a range of appropriate exercises when seeking performance adaptation. Indeed, similar improvements in horizontal jump distance have been observed following HT and front squat training (Contreras et al., 2017), further supporting their inclusion within strength training programmes. More recently, Fitzpatrick et al. (2019) demonstrated similar improvements in both vertical and horizontal jumping performance following HT training, also citing the principles of the DC theory as the potential explanation for HT improving jumping performance. Our observations in the present data, of similar improvements in horizontal jump distance in both the BS and HT groups, would appear to support the further discussion of Fitzpatrick et al. (2019). The aforementioned authors mentioned the impracticalities of characterising performance tasks and training exercises based on their expression of force relative to the global, fixed axis as seen in previous literature (Contreras et al., 2017; Zweifel, 2017; Loturco et al., 2018).

Contrary to our initial hypothesis, DL training did not present equal likelihoods of eliciting increases in horizontal jump distance, when compared to training the BS or HT. There is limited available data on the effects of longitudinal DL training upon horizontal jump performance, however the principles of DC may provide some explanation as to our findings. The DL has been shown to elicit lower levels of knee extensor activation during its concentric phase, compared to the BS (Ebben et al., 2009; Garceau et al., 2010); this being the range of movement within which knee extensor force production is required during horizontal jumping (Yokozawa et al., 2019). Additionally, the degree of hip extensor activation during the DL has been shown to be comparatively lower than the HT and BS (Andersen et al., 2018; McCurdy et al., 2018), and to reduce throughout the concentric phase of the DL i.e. when approaching full hip extension (Nijem et al., 2016). Whilst the DL does incorporate coordinated extension between the hip and knee joints during its concentric phase (Nijem et al., 2016), the progressive reduction in hip extensor activation may be a limiting factor as to the exercises ability to transfer strength gains over to horizontal jump performance. It should be acknowledged

however, that in order to confirm this explanation, a kinematic and muscle activation profile comparison between horizontal jumping and the DL should be investigated.

Thus, these present data suggest that training the BS and HT are capable of stimulating greater improvements in horizontal jump performance, compared to training the DL. Potential reasons for the disparity in differences of performance adaptation following the three training interventions, are centred around task-specific similarities in muscle activation, and the regions these muscles are required to produce force. These present data provide justification for including the BS and HT exercises when designing strength training programmes with the aim of improving lower limb power production, horizontal jumping distance.

5.5.2 Process of Analysis:

We conceptualised the distribution of differences in standing horizontal jump performance as normally distributed. Our aim was to estimate the group means and a common standard deviation for this normal distribution. We used a linear modelling approach where the mean of each group was represented by a combination of β coefficients. Each of these coefficients and for the common standard deviation required prior distribution. We chose normal distributions for the β coefficients and a half- t distribution for the common standard deviation (Figure 5.1), allowing for both increments and decrements in jump performance, in addition to small effects. Choosing values to parameterise the priors may seem difficult and subjective but some knowledge of the phenomena under study and in this case some sense of physiological constraints leads to sensible values. We chose to centre our prior for the reference group (β_0) on the mean of the data we observed and at zero for the offsets (β_1) for the other two groups. This reflects our best estimate of an average change across training and the fact that we considered group differences could be positive or negative. We used data from Rhea (2004) to inform the width (i.e. the standard deviation) of the β coefficient prior distributions. In particular Rhea suggested that a large effect in a strength training intervention would be twice the standard deviation of the data. We used our standard deviation multiplied by 3 to deliberately exceed Rhea's estimate of a large effect size. These choices may be considered subjective, but they seem reasonable, and they make use of explicit knowledge we already have about the expected response (Figure 5.3A).

We chose to model the common standard deviation (σ) as half- t distributed partly based on advice from the Stan development team (Carpenter et al., 2017). The half- t was parameterised over the positive real line (because mathematically a negative standard deviation makes no sense) with a

shape (degrees of freedom) parameter of 4, a mean of zero and a standard deviation as the standard deviation of the differences data multiplied by 10. This prior standard deviation reflected our relative ignorance about what the common standard deviation would be. Inspection of the prior is a useful step in a Bayesian approach to make sure that the choices made are sensible. In a simple model, like the present study (linear regression), we do not expect to see any untoward behaviour, but more complex models benefit from assessment of the prior distribution(s) (Muth et al., 2018). Examination of the probability over the possible effects defined by the prior (Figure 5.3A) suggests that our choices led to substantial probability for changes beyond those we would expect to see given physiological constraints. Horizontal jump decrements or improvements on the order of 0.5m are physiologically very unlikely. However, a liberal prior means we lowered the risk of overfitting (i.e. allowing the data to dominate the posterior distribution).

Once the prior has been defined the posterior distribution is generated by multiplying together the likelihood of the data (Etz et al., 2018) and the prior distribution which describes the credibility we give different parameter values. The posterior distribution then directly characterises parameter values that are consistent with our data (means and a standard deviation). The outcome of this process can be seen by comparing the x-axis in Figures 5.3A&B. The posterior distributions are considerably narrower than the liberal priors we set and the coefficients for the HT and BS groups have moved away from the prior value of zero. The most dramatic change is in the distribution for the sigma term which shrinks over two scales of magnitude from meters to centimetres.

5.5.3 Comparison with NHST:

As with many studies in sport and performance science we had a small sample size (11 per group). Small samples sizes lead to unreliable point estimates and are notoriously hard to reproduce (Pereira et al., 2012; IntHout et al., 2015). We have mitigated this to some extent by using a Bayesian approach to data analysis that allows us to directly visualise and estimate uncertainty relating to parameter estimates and the sampling procedure. We suggest this is a much richer inferential approach than NHST and frequentist-based intervals in this context. Specifically, the Bayesian approach allows us to directly address whether some phenomena is happening, how big the effect might be and how sure we should be that we are seeing that phenomena without recourse to an assumed large number of exact study replicates.

Several authors have advocated a move from inference based on 'bright line' statistics such as p -values towards estimation of effects sizes and credibility of observed effect sizes (Cumming,

2013;Swinton et al., 2018). Bayesian methods are naturally suited to the purpose of estimation (Kruschke and Liddell, 2018) and several authors have illustrated or suggested Bayesian approaches to small sample parameter estimation in sport science (Welsh and Knight, 2015;Mengersen et al., 2016;Borg et al., 2018). Whilst we have, from our data, a concrete mean and standard deviation we know we only have a sample and therefore entertain uncertainty about these data derived values. As we illustrate, the posterior distribution can be used to generate probability intervals for parameters of interest. These Bayesian probability intervals are directly interpretable; for example, in this study the 95% highest density interval (HDI) for the mean of the change in horizontal jump in the HT group ran from approximately zero to 0.13m (Table 5.1). We can directly interpret this as meaning we'd expect, with 95% probability that the average change in horizontal jump in the HT group falls in this interval. Notably, frequentist 95% confidence intervals are often misinterpreted in this manner (Greenland et al., 2016); but actually reflect, with 95% confidence, the interval within which we'd expect to find the true (unknown) population parameter, based on hypothesized repeats of the experiment (Hespanhol et al., 2019). The posterior distribution can also be summarised using point estimates (e.g., mean or median) but this loses the richer inference available in the Bayesian approach and detracts from the idea of uncertainty around estimates.

The change in horizontal jump after resistance training is not expected to be large (and in our study the effect sizes are not large) and we have small group sizes. Using the conventional p -value cut off of $p \leq 0.05$ would lead us to fail to reject the null hypothesis when looking for differences between the groups. This is easily, but incorrectly (Pernet, 2017), interpreted as 'no effect' – however absence of evidence is not evidence of absence. Using a Bayesian approach gives us a full posterior distribution rather than simple point estimates. Examining the posterior distribution paints a very different picture from the conclusions drawn from NHST. We can see that although there is some overlap between the three groups, the HT and BS groups were much more likely to exceed our chosen cut off for a meaningful effect (change in jump above estimated TE) than the DL group (Figures 5.3B and 5.4A). This is valuable information when examining the effectiveness of an intervention and is central to the idea of quantifying uncertainty around an effect. As the magnitudes of change seen in the present study (as well as sample size) are not dissimilar to that of other performance measures, the current methodology demonstrates a wide range of applicability across sport science analysis and not just limited to quantifying changes from resistance training interventions.

We can also use the posterior distribution to examine the consequences of the model using posterior predictive sampling. Simulating from the posterior predictive distribution combines uncertainty about the parameters with uncertainty about the sampling procedure (McElreath, 2018). If the model reflects the data generating process well, we should see predicted values scattered around the values from our data. In Figure 5.5 we show that the means of simulated datasets are equally distributed around the group specific mean values from our data. Furthermore, we can also use these posterior predictive distributions to estimate the probability we would see a group mean above the estimated TE in a replicated study. This is shown in Figure 5.5 by the area of the histogram above the dashed vertical line. Using the posterior predictive distribution in this way allows us to directly incorporate parameter and sampling uncertainty into predictions about future values.

5.5.4 Limitations:

One longstanding criticism of Bayesian approaches is that they incorporate subjective knowledge. We certainly used subjective knowledge to define our prior distributions. However, we would defend this on two fronts. Firstly, no scientific study is carried out without subjective decisions (Jahn and Dunne, 2007; Kochan, 2013). The Bayesian approach simply makes some of these decisions explicit rather than implicit. Secondly, we have some degree of expert knowledge to bring to the definition of the prior. Where we lack that knowledge or are very uncertain, we can use estimates derived from others as we did with the incorporation of strength intervention effect sizes from (Rhea, 2004) to inform our prior estimate for the common standard deviation. Alternatively, we can invoke e.g., physiological constraints and be liberal, however remain grounded in terms of what is a physiologically possible change in performance. By using expert knowledge or previously published data to inform prior distributions, Bayesian analysis also incorporates an element of protection against ‘statistical hacking,’ meaning there is further objectivity involved, (i.e., if less informative priors are used without physiological grounding, the posterior distributions will lose a portion of their inferential value.)

Although not a limitation *per se*, both the Bayesian and frequentist-based effect size estimate approaches may require a shift in paradigm on the part of the investigator. For Bayesian analysis the definition of the prior distribution can be a stumbling block. We hope that the process we followed in this study helps to provide clarity to this process. Also, moving away from considering probability in traditional frequentist terms, to considering it as a more subjective property may be difficult. However, evidence indicates that many people misinterpret frequentist estimates as if they were

Bayesian (Dienes, 2011) and so changing outlook on probability is less likely to be a problem. The Bayesian process explicitly gives many people what they thought they were getting anyway. For effect estimation, investigators should be aware that they will have to loosen their preconceptions around the p -value as an arbiter and instead look to correctly interpret interval estimates (frequentist or Bayesian) and effect directions to inform their conclusions.

5.6 Conclusions

We have demonstrated that Bayesian statistics can be effectively applied in small-scaled studies and provide meaningful inferences upon data in a sport science setting. The methodological approach outlined can yield directly interpretable outcomes in a setting where NHST was unable to and, is better suited to estimating the size of outcome effects. With the additional quantification of TE, the approach described here produces inferentially rich intervals which allow us to quantify; if an effect is happening; how big that effect may be; and the level of certainty with which we are seeing a true effect. The findings of the example data used here could be used to inform a practitioners' considerations for lower limb resistance exercise selection, and their planning, when seeking optimal transference across to sporting performance. Practitioners should also consider the approach we have outline here when looking to make practical decisions on their data. As the Bayesian approach provides richer inferences than traditional frequentist 'yes/no' point estimates, it has the potential to ultimately provide coaches with more confidence in their planning and decision-making processes.

Chapter 6

Summary and Conclusions

The overall aim of this thesis was to explore the nature of non-invasive assessment techniques used to characterise adaptations to strength training, with an emphasis upon the role of contractile mechanics and how they adapt in relation to other areas of the neuromuscular system. As such, strength training adaptations designed to benefit athletic performance, were the subject of this research; with the underlying mechanisms responsible, and methods of their quantification, being a primary focus. Increased strength is associated with improved athletic performance (Suchomel et al., 2016) and reduced injury risk (Lauersen et al., 2018), with strength also being a common predictor of sporting success (Cormie et al., 2010b). Increased strength is resultant of effective planning and execution of strength training interventions by coaches and practitioners, and thus requires detailed understanding of various areas of the neuromuscular system undergoing adaptation on both large and small scales, to subsequently increase force output.

We identified that contractile mechanics of skeletal muscle can positively contribute to strength gain through neuromuscular adaptation; however there remains a scarcity of data, particularly surrounding their adaptation to strength training. Whilst the potential use of a non-invasive, objective assessment method of contractile mechanics, such as TMG, was highlighted; the establishment of further construct validation of its Dm measurement in the context of training interventions was warranted. The aforementioned being, in part, due to the inherent difficulty in quantifying muscle belly stiffness *in vivo*. Following three different strength training interventions we observed increased RF muscle belly stiffness alongside increased strength. Whilst there is a notable literature gap surrounding TMG application in strength training, our findings are in line with previously published cross-sectional data (de Paula Simola et al., 2016; Šimunič et al., 2018), indicating increased muscle belly stiffness in individuals with great exposure to strength and power training. Further support for our aforementioned finding can be taken from observed gender-associated differences in muscle stiffness using free-oscillation techniques; where stronger males displayed increased levels of VL muscle stiffness compared to weaker females (Wang et al., 2015). Furthermore, we observed a negative relationship between muscle belly stiffness and parameters of muscle architecture, indicating a mechanistic association between them. Whilst similar relationships have been observed in muscle atrophy literature (Pišot et al., 2008; Šimunič et al., 2019), increases in in muscle belly stiffness have been shown to accompany increased force production (Rusu et al., 2013; Zubac and Simunic, 2017). The aforementioned increase in stiffness and associated degree of force production centres upon alterations in the active force element of the length-tension relationship (Gordon et al., 1966) leading to an altered mechanical loading stimulus for muscle hypertrophy (Goldspink, 1999). This is the first time the relationship between contractile mechanics

and muscle architecture has been observed in the context of strength training and muscle hypertrophy, positively contributing to the construct validation for the longitudinal use of TMG to assess contractile mechanics adaptations.

A potential limitation in chapter 2 was the absence of a comparable control group. Whilst similar findings to that of chapter 2 have been demonstrated previously (Pišot et al., 2008; Šimunič et al., 2019), such data would add strength to the observed changes in contractile mechanics and muscle architecture parameters. At the time of data collection, participant recruitment and withdrawal rates imposed significant challenges meaning we were unable to incorporate a control group sample into the study design. The inclusion of a control group was also prevented due to equipment access and funding. Additionally, in chapter 2 we employed only end-point assessments, providing 'snap shots' of contractile mechanics. In order to get the most out of a training intervention, practitioners often utilise continual assessment at regular intervals to track the intervention's efficacy, allowing continued modification. As such, we examined the time-course of contractile mechanics adaptations throughout a strength training intervention, in relation to other areas of the neuromuscular system. As reviewed in chapter 1, exploring multiple areas of the neuromuscular system simultaneously would allow a greater understanding into adaptations to strength training, and potentially reveal further relationships between such areas. An approach which combines the measurement of training adaptations within skeletal muscle contractile mechanics, individual MUs, and corticospinal networks, could provide extremely detailed insight into the neuromuscular system's responses to strength training.

In chapter 3 we observed altered contractile mechanics prior to architectural adaptation in the early stages of strength training, as previously shown in muscle atrophy literature (Šimunič et al., 2019); however, we were unable to precisely determine the underlying mechanism for this. Whilst we postulated such a change in VL and RF contractile mechanics may be due to altered E-C coupling, we observed no change in corresponding Tc or force output measures at the corresponding time point (after 2-weeks strength training). Thus, it may be that such altered contractile mechanics are due to altered intramuscular fluid levels, typically seen in the early stages of strength training (Damas et al., 2015). However, the aforementioned suggestion cannot be confirmed by the present data and requires further investigation.

In relation to other areas of the neuromuscular system, we observed adaptations in VL and RF contractile mechanics prior to that of corticospinal excitability which appears to account for early strength gains observed at the same time-point. The observed increase in excitability indicates

improved efficacy of neural transmission within the spinal cord, considering that no change in corticospinal inhibition was observed from M1 stimulation. To determine the location of excitability adaptation, future work should compare changes in MEP amplitude with MEPs evoked from cervico-medullary stimulation (Nuzzo et al., 2017).

Whilst we observed increased corticospinal excitability in the RF to accompany increased force output after 4-weeks of strength training, there was no change in MU firing rate; suggesting altered spinal outputs have no influence upon MU firing rates, and that increased MU firing rate may not be a contributory adaptation for increased force output. It is possible that MUs experienced other behavioural adaptations not measured in this thesis, such as MU hypertrophy (Pope et al., 2016) or alterations in their recruitment threshold (Sterczala et al., 2020). Additionally, the practical application of MU behaviour assessment during isometric tasks should also be considered, as this was not conducted in a training-specific manner within chapter 3. This notion of training/task-specificity in neurophysiological testing has been previously highlighted (Beck et al., 2007; Taube et al., 2020) and so, we explored the potential to conduct dEMG assessments in a more training-specific manner in efforts to increase the practical relevance of potential findings. Firstly, we determined that applying dEMG during a dynamic strength assessment of cyclic nature was plausible, as we reported 'excellent' relative inter-day reliability for obtained MU firing rates, and comparable MU yields and identification accuracies to those found during isometric testing in chapter 3. These observations demonstrate dEMG as capable of successfully identifying MUs during dynamic strength exercises, which are commonly employed in strength training owing to their biomechanical similarities with elements of sporting performance. Whilst we reported high levels of relatively reliability in chapter 4, absolute reliability was diminished in comparison. With previous authors (De Luca et al., 2015a) noting that movement frequency may influence the decomposition of the sEMG signal, the implementation of a measure of movement velocity control should be explored in future work as this may aid in improving inter-day reliability further.

The successful application of dEMG during a dynamic, training-specific task, allowed us to further explore the task-specific nature of neuromuscular adaptations in chapter 4. We compared a subsample of MU firing rates assessed during the 5RM BS strength test, before and after the 6-week training intervention employed in chapter 3. Similar to our findings during isometric assessment, we observed no change in MU firing rates, indicating this not to be an adaptive response contributory to the observed strength gain. This also supports previous research following dynamic strength training (Stock and Thompson, 2014; Sterczala et al., 2020), although it is again possible that adaptations in recruitment threshold-firing rate relationships may be contributory to strength gain. However, the

measurement of recruitment threshold during the dynamic task was not possible. This is due to the inherent difficulty of determining the point of force generation for a single muscle during a compound, closed chain movement. A future consideration of incorporating measurements of joint angle during such dynamic movements, would allow similar relationships to that of recruitment threshold to be investigated by practitioners. Unfortunately, due to equipment constraints, it was not possible to incorporate such measure as goniometry or motion capture within chapter 4's study design. Outside of strength training adaptations, exploring MU recruitment and decruitment angles during training exercises such as the BS could give practitioners unique insight into MU behaviours during biomechanically comparable elements of sporting performance, such as sprinting and jumping. By inferring upon MU behaviours within specific muscle groups, practitioners may be able to choose more specific interventions, giving a greater prospect of strength increase or recovery from injury. Going beyond strength training, dEMG has potential to provide in-depth information on MU activity in areas such as rehabilitation and at-risk populations. By being able to infer upon patterns of MU activity, practitioners may be able to better design intervention and recovery strategies.

Thus far we have alluded to the mechanisms by which the human neuromuscular system adapts in various areas to increase strength. With the ultimate goal of strength training to improve aspects of athletic performance via the aforementioned mechanisms, practitioners should be able to have confidence in the efficacy of the training interventions they employ, and the observed effects upon task performance following them. This requires practitioners to be sure that improvements in performance are occurring from the employed training; to be sure of the magnitude of that change; and that the change observed is a meaningful one that is going to benefit performance. We have demonstrated and described an application of Bayesian analysis which addresses the above-mentioned practitioner concerns. The Bayesian approach allowed us to directly visualise and estimate the certainty/uncertainty surrounding the effects of three training interventions, upon the physical performance parameter of horizontal jump distance. This approach determined that BS and HT training were 77.9% and 78.8% likely to result in improved horizontal jump distance, above the associated TE of the performance test, in comparison to only 19.8% following DL training. Additionally, we also demonstrated how Bayesian HDIs are potentially more informative than the often mis-interpreted 95% CI of frequentist approaches. The HDI described in chapter 5 represents the range within which we'd expect, with 95% probability, the average change in a test parameter to fall following the intervention; thus, providing a directly interpretable interval for practitioners. The use of this interval is further strengthened by the implementation of a user defined threshold

allowing the investigator to define what a meaningful change in the test parameter may be. Notably, in our example data this took the form of TE associated with the test, directly addressing the previously noted requirement for practitioners to be sure the observed change following training is a meaningful one. Conversely, NHST would have led us to reject the presence of an effect following strength training, suggesting that neither of the three interventions were effective at improving an element of physical performance. However, the described Bayesian approach points to the inclusion of BS and HT exercises in strength training for improved jump performance, over inclusion of the DL. Despite the primary purpose of the data used in chapter 5 to be demonstrative, the aforementioned findings do provoke some interesting thought surrounding task-specific similarities in muscle activation, and the biomechanical regions within which contributory muscles are required to produce force during such tasks. It would appear that similarities between strength training exercises and performance tasks, in terms of biomechanical regions of muscle activation, may be more important than similarities in force expression relative to the global frame. With training specificity being a key consideration in the planning and development of training and rehabilitation interventions, future research could look to employ EMG assessments to provide further clarity upon the aforementioned potential similarities.

It must be acknowledged that the adoption of Bayesian approaches to estimate the size of training intervention effects may require a shift in paradigm on behalf of the researcher, requiring them to consider estimations of effects and their associated uncertainty, rather than point estimates (*p values*). Additionally, the procedures of Bayesian do require knowledge of statistical coding software packages and script writing. Whilst many researchers perform statistical analysis using such software packages, further work is required to improve accessibility. Recent literature has helped taken steps to mitigate this issue by exploring the use of common data processing software (e.g., Microsoft Excel) to build templates for performing Bayesian analysis (Swinton et al., 2018)

However, Bayesian approaches such as the one described in this thesis represent a wide range of applicability within performance sport; where sample sizes are typically small, study replicates are scarce, and effect sizes are not expected to be large. Importantly, the Bayesian approach allows us directly address practitioner concerns of, is an effect happening? How big is the effect and is the effect a meaningful one?; thereby facilitating a practitioner's confidence with which they employ a particular intervention.

In conclusion, within the context of physiological adaptations to strength training, practitioners require detailed understanding as to the mechanisms responsible for increased strength following

training or injury rehabilitation. Such requirement necessitates the objective means to assess the adaptive roles played by different areas of the neuromuscular system simultaneously, and a means of analysis capable of providing confidence to practitioner decisions. This thesis has demonstrated specific involvement of contractile mechanics in adaptations for strength gain within quadricep muscles, as well as directly applicable applications of their assessment within the setting of strength training for athletic performance. The use of techniques such as TMG may offer an easily accessible method of assessing strength training adaptations, in situations where traditional strength assessments may be disruptive or not feasible. The application of a non-invasive, contractile mechanics measurement technique has demonstrated valuable insight into the mechanisms of strength adaptations; and provided detailed information when investigated alongside other areas adaptive responses throughout the neuromuscular system. Assessment techniques described and utilised in this thesis may provide new insight when used to explore specific strength training programmes, through detailed information regarding their elicited adaptations. In the interest of further applicability to performance sport, we have also proposed an inferentially rich method to analyse outcome measures following strength training interventions. The described method is capable of addressing key concerns and questions from practitioners surrounding the efficacy and utilisation of specific strength training interventions.

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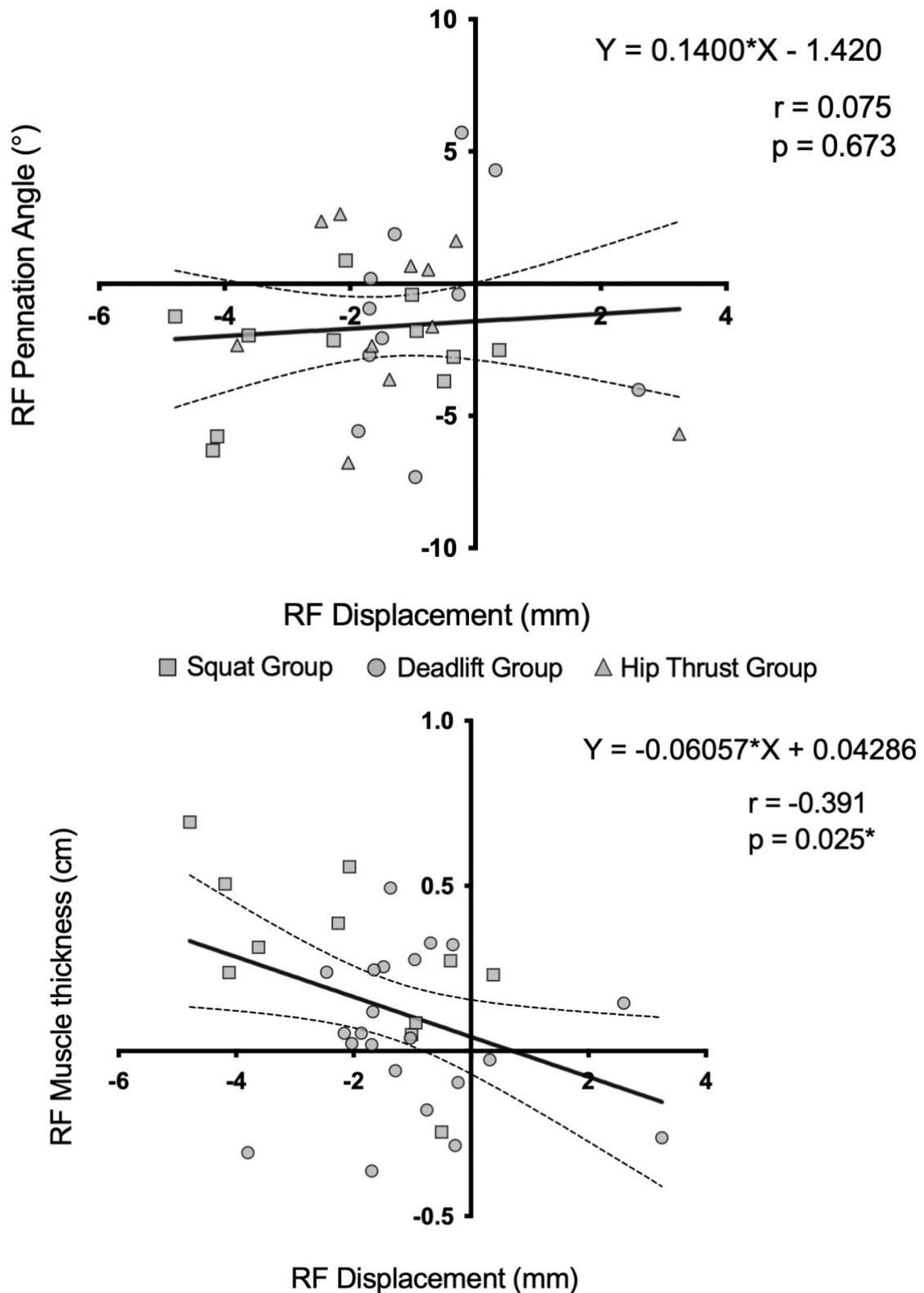
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Appendices

Appendix A: Correlation analysis ($n=33$) of pennation angle vs radial muscle displacement in the rectus femoris (A), and muscle thickness vs radial muscle belly displacement in the rectus femoris (B), of all exercise groups.



Appendix B: Images of exercise technique standards to which participants were coached to (referred to in section 2.3.5).

Back squat Technique:

Start/Finish position



Bottom position



Deadlift Technique:

Start/Finish position



Top Position



Hip Thrust Technique:



Start/Finish position



Top Position

TMS screening questionnaire referred to in section 3.3.1.

Physiology, Exercise and Nutrition Research Group TMS screening questionnaire v1.0



Brain Stimulation Safety Screening Form (Confidential)

If you agree to take part in this study, please answer the following questions. The information you provide is for screening purposes only and will be kept completely confidential

1. Do you have epilepsy, or have you ever had a convulsion or a seizure (fit)? YES NO
 If YES please state your relationship to the affected family member _____
2. Has anyone in your wider family suffered from seizures? YES NO
 If YES please state your relationship to the affected family member _____
3. Have you ever had a fainting spell or syncope? YES NO
 If YES please describe on which occasion(s) _____
4. Have you ever had a head trauma that was diagnosed as a concussion or was associated with loss of consciousness? YES NO
5. Do you have any hearing problems or ringing in your ears? YES NO
6. Do you have cochlear implants? YES NO
7. Are you pregnant, or is there any chance that you might be? YES NO
8. Do you have metal in the brain, skull or elsewhere in your body (e.g., splinters, fragments, clips, etc.)? YES NO
 If YES, specify the type of metal and where it is located _____
9. Do you have an implanted neurostimulator (e.g. DBS, epidural/subdural, VNS)? YES NO
10. Do you have a cardiac pacemaker or intracardiac lines? YES NO
11. Do you have a medication infusion device? YES NO
12. Are you taking any prescribed or unprescribed medications (or herbal remedies)? YES NO
 If YES, please list _____
13. Did you ever undergo TCS or TMS in the past? YES NO
 If YES, please state if there were any problems and describe them _____

 When was your last TMS/TCS session? _____
 How many TMS/TCS sessions have you had in the past month? _____
 How many TMS/TCS sessions have you had in the past 12 months? _____
14. Did you ever undergo MRI in the past? YES NO
 If YES, please state if there were any problems and describe them _____

(Based on Screening13-item Questionnaire for rTMS Candidates recommended by Rossi, Hallett, Rossini and Pascual-Leone 2011; updated 15 Dec 2015)

Physiology, Exercise and Nutrition Research Group TMS screening questionnaire v1.0



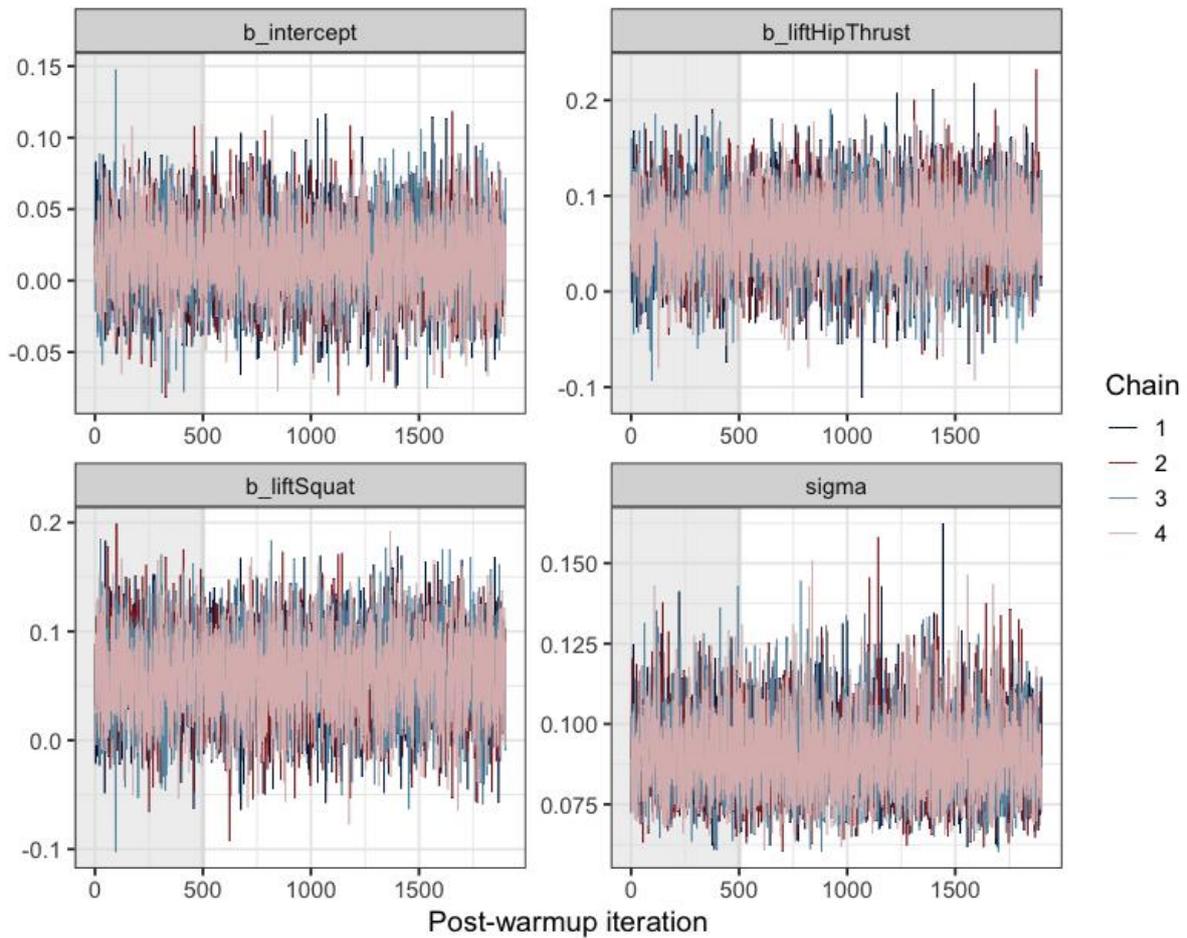
15. Have you ever undergone a neurosurgical procedure (including eye surgery)? YES NO
 If YES, please give details _____
16. Are you currently undergoing anti-malarial treatment? YES NO
17. Have you drunk more than 3 units of alcohol in the last 24 hours? YES NO
18. Have you drunk alcohol already today? YES NO
19. Have you had more than one cup of coffee, or other sources of caffeine, in the last hour? YES NO
20. How much liquid in total have you drunk already today? _____ ml
21. When was your last meal? _____ hours ago
22. Have you used recreational drugs in the last 24 hours? YES NO
23. How many hours sleep did you have last night? _____

I (please give full name in CAPITALS) _____ confirm
 that I have personally completed the above questionnaire.

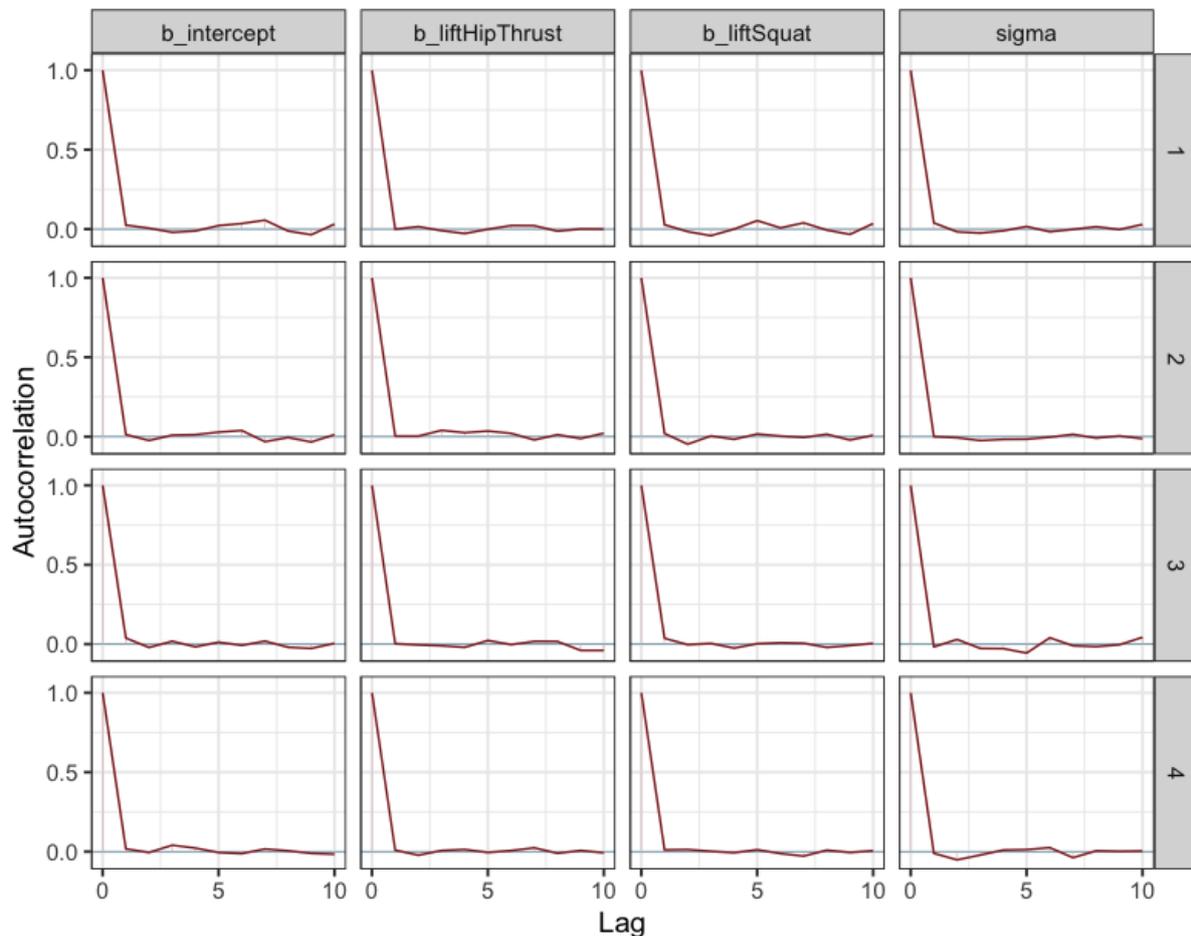
Signature _____ Date _____

Please note: All data arising from this study will be held and used in accordance with the Data Protection Act (1984).
 The results of the study will not be made available in a way that could reveal the identity of individuals.

Diagnostic checks for the Bayesian analysis approach referred to in section 5.3.3.



Appendix D: *Mixing of the 4 MCMC chains estimating each of the parameters of the linear model.* The first 500 warmup samples are shown with a shaded background. There is no evidence of divergence in these chains indicating good estimation of the relevant model parameter. The x-axis does not extend to 10,000 (the total number of samples in each MCMC chain) because we thinned the chains with lag 5.



Appendix E: *Autocorrelation across the 4 MCMC chains for each parameter.* Autocorrelation is essentially gone at lag 1 indicating that most of the MCMC draws for estimating each parameter can be considered independent.

Supporting information:

Learning the Bayesian approach to data analysis can be a challenge and at present there is little choice but to learn a programming language as well. Excellent software frameworks now exist in R (Burkner, 2017, Goodrich, 2018) and other programming languages (see <https://mc-stan.org/users/interfaces/> for a list of interfaces) to ease the burden of using the underlying Stan probabilistic modelling language. Other choices are also available (e.g. see http://www.inferencelab.com/mcmc_software/). We used the `brms` package (Burkner, 2017) because it allows us to write our model using the R language modelling syntax which is familiar to us. Two opensource programmes with point and click interfaces that implement Bayesian approaches are `jasp` (www.jasp-stats.org) and `jamovi` (www.jamovi.org).