The influence of training and athletic performance on the neural and mechanical determinants of muscular rate of force development

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ABSTRACT

Neuromuscular explosive strength (defined as rate of force development; RFD) is considered important during explosive functional human movements; however this association has been poorly documented. It is also unclear how different variants of strength training may influence RFD and its neuromuscular determinants. Furthermore, RFD has typically been measured in isometric situations, but how it is influenced by the types of contraction (isometric, concentric, eccentric) is unknown. This thesis compared neuromuscular function in explosive power athletes (athletes) and untrained controls, and assessed the relationship between RFD in isometric squats with sprint and jump performance. The athletes achieved a greater RFD normalised to maximum strength (+74%) during the initial phase of explosive contractions, due to greater agonist activation (+71%) in this time. Furthermore, there were strong correlations ($r^2 = 0.39$) between normalised RFD in the initial phase of explosive squats and sprint performance, and between later phase absolute explosive force and jump height ($r^2 = 0.37$), confirming an association between explosive athletic performance and RFD. This thesis also assessed the differential effects of short-term (4 weeks) training for maximum vs. explosive strength, and whilst the former increased maximum strength (+20%) it had no effect on RFD. In contrast explosive strength training improved explosive force production over short (first 50 ms; +70%) and long (>50 ms; +15%) time periods, due to improved agonist activation (+65%) and maximum strength (+11%), respectively. Explosive strength training therefore appears to have greater functional benefits than maximum strength training. Finally, the influence of contraction type on RFD was assessed, and the results provided unique evidence that explosive concentric contractions are 60% more effective at utilising the available force capacity of the muscle, that was explained by superior agonist activation. This work provides a comprehensive analysis of the association between athletic performance and RFD, the differential effects of maximum vs. explosive strength training, and the influence of contraction type on the capacity for RFD.

Keywords: Rate of force development, explosive strength, strength training, contractile properties, neural activation, contraction type, muscle length, muscle-tendon stiffness


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CHAPTER 1

Introduction
The capacity of the neuromuscular system for explosive force production, often defined as rate of force development (RFD), is considered important during functional human movements where time to develop force is limited (Newton, Kraemer 1994, Aagaard et al. 2002a, de Ruiter et al. 2004, Hakkinen, Komi & Alen 1985, Suett et al. 2004). This includes explosive sports activities (such as sprinting and jumping; (Haff et al. 1997, Weyand et al. 2000, Luhtanen, Komi 1979, Da pena, Chung 1988)) or injury avoidance mechanisms (such as re-stabilising a joint following mechanical perturbation; (Gruber et al. 2007, Krosshaug et al. 2007, Domire, Boros & Hashemi 2010)). Despite the proposed relationship between RFD and explosive functional performance, there is limited evidence to support this association. Strength training is a widely recommended physical activity, which likely also contributes to the distinct neuromuscular characteristics of high performing athletes, yet how strength training and its many variants influences explosive strength is equivocal. Finally, RFD is typically assessed in isometric contractions, despite the dynamic nature of functional human movement. Therefore, little is known about RFD during different types of muscle contractions.

One approach that may improve our understanding of the association between athletic performance, RFD, and the neural and mechanical determinants of RFD, is to compare explosive neuromuscular performance in explosive power athletes, a group with demonstrated ability for explosive performance, and untrained individuals. Studies have reported greater absolute RFD in explosive power athletes than endurance athletes or untrained individuals (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989); however, these studies did not provide a comprehensive analysis of the neural and mechanical explanations for these differences. This could be achieved by recording agonist activation during explosive voluntary contractions, and comparing the RFD produced in this situation with that measured during electrically evoked involuntary contractions. Chapter 3 of this thesis employed these techniques in order to compare RFD and its determinants in explosive power athletes and untrained individuals.

A more direct approach to understanding the relationship between athletic performance and explosive force production is to quantitatively relate these parameters for a range of individuals. Two studies have investigated the relationship between knee extensor explosive force and vertical jump performance, and whilst one reported a significant
correlation between these variables (de Ruiter et al. 2006) the other did not (de Ruiter et al. 2007). This discrepancy may, in part, be due to the studies only measuring knee extensor RFD, whilst vertical jumping (as well as most other explosive athletic activities) relies on the contribution of multiple muscle groups (Pandy, Zajac 1991). Therefore assessing RFD during multiple joint actions may facilitate a better understanding of its association with athletic performance. Two studies have compared CMJ performance to RFD during multiple joint situations (isometric squats and mid-thigh clean pulls), but reported a surprisingly poor relationship between these variables (Nuzzo et al. 2008, Kawamori et al. 2006). However, these studies considered explosive force production at only one time point during the rising force-time curve, and therefore may not have assessed the relevant force-time characteristics for jumping. Chapter 4 of this thesis investigated the association between explosive athletic performance (specifically sprinting and CMJ performance) and RFD over different time periods during a multiple joint (isometric squat) situation.

Traditional strength training has typically involved high load (>70% of maximal voluntary force or one repetition maximum) sustained (>3-s) contractions with the aim of increasing maximum strength (‘training for maximum strength’). Training for maximum strength has proved very effective at enhancing this component of strength (Aagaard et al. 2002a, Suetta et al. 2004, Andersen et al. 2010, Blazevich et al. 2009, Kubo et al. 2001, Kubo et al. 2007b, Rich, Cafarelli 2000), with long-term gains thought to be predominantly due to peripheral adaptations (Narici et al. 1996). In contrast, short-term maximum strength gains are thought to be due to neural adaptations (specifically increased agonist activation and/or decreased antagonist activation; (Del Balso, Cafarelli 2007, Narici et al. 1989, Hakkinen, Komi 1983, Hakkinen et al. 1998, Kubo et al. 2006)), although evidence of this is equivocal (Kubo et al. 2001, Rich, Cafarelli 2000, Narici et al. 1996, Garfinkel, Cafarelli 1992, Pucci, Griffin & Cafarelli 2006, Cannon et al. 2007). This may be primarily due to methodological issues associated with the two techniques typically used to assess voluntary activation; electromyography (EMG) and the interpolated twitch technique (Folland, Williams 2007a, Folland, Williams 2007b). Chapter 5 of this thesis aimed to address these methodological issues in order to better understand the neural and/or peripheral adaptations responsible for short term maximum strength gains.
The effects of training for maximum strength on RFD are equivocal (Aagaard et al. 2002a, Suett et al. 2004, Andersen et al. 2010, Blazevich et al. 2009, Kubo et al. 2001, Rich, Cafarelli 2000), and evidence suggests that this type of training may only influence RFD over long periods (>200 ms) of force production (Andersen et al. 2010); however, the physiological explanation for this response to training is unclear. It is possible that the neural adaptations (e.g., increased agonist activation) to maximum strength training are specific to only high force levels, and do not translate to the early phase of the rising force-time curve, but this has not been investigated. Therefore a secondary aim of Chapter 5 was to assess the effects of training for maximum strength on RFD and its determinants.

In contrast to traditional strength training, ‘training for explosive strength’ may be better served by performing short (~1-s) high RFD contractions that provide the specific stimuli required to improve this component of strength. However, the effects of training for explosive strength on RFD and its neural and mechanical determinants are not widely documented. Three studies have observed concomitant improvements in initial RFD and agonist activation during the initial phase of explosive contractions (Gruber et al. 2007, Barry, Warman & Carson 2005, Van Cutsem, Duchateau & Hainaut 1998). These studies however, did not provide a comprehensive analysis of the potential peripheral adaptations to explosive strength training, and therefore the relative contribution of any neural and mechanical adaptations to changes in RFD remains unclear. This issue may be addressed by comparing changes in voluntary RFD to changes in RFD recorded during a supramaximal octet contractions at 300 Hz (which provides a measure of the maximal capacity of the MTU for RFD; (de Ruiter et al. 2004, de Ruiter et al. 1999)); but this has not been done in the context of strength training. Chapter 6 of this thesis aimed to provide a comprehensive analysis of the effects of explosive strength training on RFD and its determinants.

The capacity for maximal force production is known to be strongly affected by the type of contraction being performed, as depicted by joint torque-angular velocity relationship (Yeadon, King & Wilson 2006). However, the influence of the type of contraction on explosive torque production has not been investigated. This is likely to be because of methodological issues with measuring rate of torque development (RTD) in concentric and eccentric contractions. Specifically, the mechanics of the system
interact with torque in a non-linear manner, confounding RTD measurements, unless the concentric or eccentric efforts are performed at either a constant velocity or constant acceleration. Furthermore, joint angle changes (which also influence force production; discussed below) occur in opposite directions during concentric and eccentric contractions, so it is only possible to match torque in the different types of contractions at a single time point (joint angle) along the torque-time curve. This issue may be addressed by normalising explosive torque produced at any time point during the different types of contractions to the maximum strength available at that specific joint angle and angular velocity. This will also enable the investigator to assess whether explosive torque production changes in proportion to maximum strength. Chapter 7 aims to investigate the influence of contraction type on RTD, and the ability to utilise the available torque capacity of the muscle in an explosive effort.

The capacity for maximal torque production is also influenced by muscle length/joint angle, as depicted by the torque-angle relationship (Rassier, MacIntosh & Herzog 1999). It is therefore conceivable that joint angle will also influence the capacity for explosive torque production. Evidence shows that explosive torque production in humans during the initial phase of explosive contractions changes with joint angle, but only in proportion to maximum strength (de Ruiter et al. 2004). In contrast in vitro animal studies have reported a steeper force-time curve normalised to peak force at late, but not early, phases of the contraction (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980). This suggests that muscle length (and therefore joint angle) may influence the capacity for explosive torque production in humans during the later phase of explosive contractions, but this has not been investigated. Therefore a secondary aim of Chapter 7 was to investigate the affects of joint angle on the rising force-time curve.

The purpose of this work was to investigate the influence of athletic performance and strength training on explosive force production and its neural and mechanical determinants. This was done by (i) comparing explosive neuromuscular function in groups of different athletic/training backgrounds and considering how explosive strength was related to athletic performance (specifically jumping and sprinting); (ii) assessing the differential effects of training for maximum and explosive strength on RFD and its determinants; and (iii) investigating the influence of type of contraction, and joint angle on the capacity for explosive force production.
CHAPTER 2

Literature review
2.1 Introduction

For voluntary human movement to occur the skeletal muscles must generate forces by converting chemical energy (obtained from the hydrolysis of ATP) to mechanical work (via myosin-actin cross-bridge cycles), and transmit these forces via connective tissues (aponeurosis and tendon) to the bones. The size of these forces and the length of time over which they are produced determines the change in velocity of the body’s mass, and thus the success of human movements and athletic performance. The capacity of the muscle, or group of muscles, to produce force in a given situation is often referred to as strength (Knuttgen, Komi 1992). Strength can be subdivided into maximum strength (the maximum force capacity of the muscle in a given situation) and explosive strength (the ability to rapidly exert muscular force in a given situation) (Logan et al. 2000).

Maximum strength is defined by the greatest peak of the force-time curve (Figure 2.1A) irrespective of when it occurs, whilst explosive strength is defined by the slope of the force-time curve (Figure 2.1B), and is therefore often referred to as rate of force development (RFD). In explosive sports activities, where performance is largely determined by a high velocity at take-off (such as in sprinting and jumping activities), release (such as in throwing activities), or impact (such as in punching or tackling activities), the muscles must produce as much force over as much time as possible (Newton, Kraemer 1994). However, in explosive sports activities the time to develop force is limited (typically 50-250 ms; (Haff et al. 1997, Weyand et al. 2000, Luhtanen, Komi 1979, Dapena, Chung 1988)), and considered insufficient to achieve maximal force (Aagaard et al. 2002a, Thorstensson et al. 1976). Likewise, there is also limited time for maximal force development when attempting to decelerate/re-stabilise body mass and avoid injury following a loss of balance/stability (Gruber et al. 2007, Krosshaug et al. 2007, Domire, Boros & Hashemi 2010). Consequently, the ability of skeletal muscles to produce force as quickly as possible (explosive strength) is considered an important attribute to the performance and/or success of explosive sports activities and injury avoidance mechanisms (Newton, Kraemer 1994, Aagaard et al. 2002a, de Ruiter et al. 2004, Hakkinen, Komi & Alen 1985, Suett et al. 2004).
Figure 2.1. A force-time curve (A) and the slope (first derivative) of the force-time curve (B) recorded during an isometric maximal voluntary contraction of the knee extensors, whilst a participant is sat in an isometric strength testing chair. The rate of force development can be calculated as the slope of the force time curve over any given time period, and is a measure of explosive strength. Peak slope represents peak rate of force development. The greatest peak force recorded during a maximal voluntary contraction is defined as maximal voluntary force and is a measure of maximum strength.

Theoretically, if all other factors are equal (e.g., level of activation, contractile speed, stiffness of the system) the capacity for explosive force production will be scaled to the capacity for maximal force production. However, in voluntary contractions in humans there are many other neural and mechanical characteristics of the neuromuscular system that will influence explosive strength. These include the level of agonist activation (de Ruiter et al. 2004, de Ruiter et al. 2006, de Ruiter et al. 2007), the intrinsic contractile properties of the muscle (Andersen, Aagaard 2006), and stiffness of the muscle-tendon unit (MTU) (Bojsen-Moller et al. 2005, Wilson, Murphy & Pryor 1994). Furthermore, given that maximal force production is limited by muscle length/joint angle (Rassier, MacIntosh & Herzog 1999) and the type of contraction (Hill 1938); it is conceivable
that these parameters will also influence explosive force production. The contribution of
the above neural and mechanical factors to explosive force production should be
assessed in order to understand the limitations of RFD. It is also important to
understand the association between athletic performance and explosive strength, as well
as the influence of training on RFD and its determinants. The next section will explain
how maximum and explosive strength are measured. Section 2.3 will then discuss the
central and peripheral limitations of voluntary force production, whilst section 2.4 will
discuss the cross-sectional evidence for the neural and mechanical determinants of
explosive strength. Finally, sections 2.5 and 2.6 will discuss the association between
athletic performance and RFD, and the influence of training on the determinants of
RFD.

2.2 Measuring Maximum and Explosive Strength

2.2.1 Maximum Strength
Maximum strength is specific to a muscle, or group of muscles, and the mechanical
situation in which it is measured (Knuttgen, Komi 1992). During an activity that
involves an isoinertial contraction, such as a free-weight exercise, maximum strength is
typically defined as the maximum weight that can be lifted for one (or multiple)
repetition of that activity (Logan et al. 2000). During a maximal isometric or isovelocity
contraction (which can be performed in an isometric strength testing rig or isovelocity
dynamometer, respectively) maximum strength is typically defined as the greatest peak
force or torque (Figure 2.1A) recorded during that situation (Wigley, Strauss 2000). If
the isometric or isovelocity contraction is a maximum voluntary effort (maximal
voluntary contraction; MVC) then the greatest peak force/torque is referred to as
maximum voluntary force/torque (MVF or MVT) (Folland, Williams 2007b).

2.2.2 Explosive Strength
RFD (or rate of torque development; RTD) is most commonly assessed during
explosive (‘fast and hard’) isometric contractions, that are measured with an isovelocity
dynamometer (Aagaard et al. 2002a, Blazevich et al. 2009), isometric strength testing
or on a force plate (Nuzzo et al. 2008, Kawamori et al. 2006). Whilst some studies have assessed RFD during the acceleration phase of isoinertial (dynamic) contractions (Haff et al. 1997, Wilson, Murphy & Pryor 1994, Adamson et al. 2008), this situation is experimentally problematic, as the mechanics of the system (displacement, velocity, and acceleration) interact with force in a non-linear fashion, thus confounding RFD measurement. For example, an increase in force will cause a change in velocity (according to Newton’s second law of motion), which in turn will affect the amount of subsequent force that can be produced (according to the force-velocity relationship; see section 2.3.1). No previous study has attempted to control the mechanics of the system in order to gain a valid measure of RFD during dynamic contractions. In contrast, isometric contractions provide a situation in which muscle length/joint angle remains constant, and therefore a valid measure of RFD can be obtained and the neural and mechanical determinants investigated. The remainder of this literature review will only refer to those studies that have measured RFD during isometric contractions.

Whilst functional human movement involves the coordination of multiple joints, explosive force production has been typically assessed in isolated muscle groups such as the knee extensors (Aagaard et al. 2002a, de Ruiter et al. 2004, Andersen, Aagaard 2006), ankle plantarflexors (Gruber et al. 2007, Del Balso, Cafarelli 2007), and elbow flexors (Barry, Warman & Carson 2005). This minimises the number of potentially confounding variables influencing RFD measurement, and provides a controlled situation in which to isolate the neural and mechanical determinants of explosive force production. A few studies have measured RFD in multiple joint activities such as isometric squats (Nuzzo et al. 2008), leg extensions (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989) and mid-thigh clean pulls (Nuzzo et al. 2008, Kawamori et al. 2006); however, these investigations did not assess the neural and mechanical determinants of explosive force production. Therefore, unless otherwise stated, the remainder of this review will only refer to those studies that have investigated explosive force production in single joint actions by isolated muscle groups. Nevertheless, further research assessing RFD in multiple joint activities is warranted in order to better understand its association with explosive performance in functional human movement.
Many studies have quantified RFD by measuring the peak slope of the force-time curve (peak RFD; Figure 2.1B) (de Ruiter et al. 2004, Gruber et al. 2007, Del Balso, Cafarelli 2007, Adamson et al. 2008). However, this only occurs at a single point on the force-time curve, and thus provides limited information on an individual’s ability for explosive force production. In contrast, other studies have measured explosive force production over different time periods from force onset (e.g., 0-10, 0-20… 0-250 ms; (Aagaard et al. 2002a, Andersen et al. 2010, Blazevich et al. 2009, Barry, Warman & Carson 2005, Andersen, Aagaard 2006, Blazevich et al. 2008). This provides a more detailed assessment of the force-time curve that facilitates the investigation of the underlying determinants of RFD.

Several methodological issues should be considered when measuring RFD. The level of pre-tension in the muscle prior to performing an explosive contraction has been shown to affect force production over the initial phase of the contraction (de Ruiter et al. 2006), and should therefore remain consistent across trials and participants. This can be controlled by performing explosive contractions from a relaxed state (i.e., no active pre-tension), which also facilitates measurement of the very initial phase of the force-time curve. The investigator should also ensure that there is no countermovement (a drop in baseline force) immediately prior to the explosive contraction, as this has been shown to influence subsequent explosive force measurements (Grabiner 1994, Kamimura et al. 2009). Finally, consideration should be given to the method used to identify force onset. The majority of past studies have used automated methods to identify force onset such as absolute force thresholds (Aagaard et al. 2002a, Andersen, Aagaard 2006) or mathematical algorithms (e.g., 3 standard deviations above the mean baseline force; (de Ruiter et al. 2004)). However, manual identification (via visual inspection) is considered the gold standard method (Allison 2003, Hodges, Bui 1996, Moretti et al. 2003, Staude 2001, Van Boxtel et al. 1993, Pain, Hibbs 2007), detecting signal onsets up to 60 ms earlier than automated methods (Allison 2003, Pain, Hibbs 2007, Pulkovski et al. 2008). This supports the use of manual identification for detecting force onsets, particularly if the investigator is interested in the initial phase of the force-time curve.
2.3 Limitations of Voluntary Force Production

The physiological and biomechanical parameters that influence voluntary force production can be separated into central and peripheral factors. Central factors are associated with the level of neural drive to the muscle. Peripheral factors include muscle size, architecture, composition, length (and/or joint angle), the type of contraction being performed (and/or the contractile velocity), contractile properties of the muscle, and MTU-stiffness.

2.3.1 Central Factors

2.3.1.1 The Process of Producing Voluntary Force

The force output of a muscle during a voluntary contraction is largely dependent on the level of neural activation. Neural activation of a muscle commences at the cerebral cortex of the brain, where the intent of an activity is defined, and a plan of activation developed. Command signals (central command) are then sent to the lower neural centres (brainstem and spinal cord). At the brainstem the central command is integrated with sensory information from the vestibular apparatus, receptors in the neck region, and the cerebellum, thus modulating the command. The command is then transmitted along the spinal cord to the relevant motoneurons. Further modulation of the command occurs at the spinal cord, whereby sensory feedback, sent via afferent neural fibres from mechanoreceptors at relevant muscles and soft tissue, inhibits or potentiates the activation of particular motoneurons (Enoka 2008).

A muscle is innervated by a number of motoneurons and each motoneuron innervates a specific group of muscle fibres. Activation of a single motoneuron excites all of the muscle fibres it innervates, and thus a single motoneuron and the muscle fibres it innervates are considered as a single functional entity termed a motor unit (MacIntosh, Gardiner & McComas 2006). Motor units can be arranged into groups depending on their contractile properties; type I, which are slow contracting; and type II, which are fast contracting (Enoka 2008). A more detailed explanation of the contractile properties of motor units is provided in section 2.3.2.6.
If a motoneuron is activated, a single electrical impulse (action potential) will be transmitted along the axon of the motoneuron to the neuromuscular junctions of the muscle fibres. The electrical impulse will be transferred across the neuromuscular junction where it will cause a wave of depolarisation across the sarcolemma. This wave of depolarisation is propagated into the sarcoplasm of the muscle fibres via the transverse tubules. This in turn will initiate the excitation-contraction coupling process, which commences when Ca^{2+} ions are released from the sarcoplasmic reticulum, diffuse across the muscle fibre, and attach to troponin molecules on the thin (actin) filaments of the contractile element (sarcomere). This results in a positional change in the troponin, which in turn lifts tropomyosin molecules away from the actin filaments to reveal active binding sites. If a myosin head carrying one ATP molecule is within range of an available binding site, it will attach to form a cross-bridge. During the cross-bridge formation the ATP molecule is hydrolysed, catalysed by myosin ATPase, producing ADP and one phosphate (P_i) molecule. The P_i molecule is then released from the cross-bridge, causing a positional change in the myosin head, which generates a forceful conformational change between the actin and myosin that results in the two filaments sliding across each other. This is known as the powerstroke. During this process the ADP molecule is released from the cross-bridge. If there is another ATP molecule available in the sarcoplasm, the myosin head will detach from the actin binding site, engage with the ATP molecule and the cross-bridge cycle will begin again. If the initial electrical impulse received at the sarcolemma is not followed by another electrical impulse, the sarcolemma will begin to repolarise. During this process Ca^{2+} ions detach from the troponin molecules and are pumped back into the sarcoplasmic reticulum. Once a troponin molecule has released a Ca^{2+} ion it reverts back to its initial position, holding tropomyosin in place over the actin binding sites, and preventing further cross bridge cycles from occurring. The excitation-contraction coupling process is discussed in much greater detail by (Enoka 2008).

### 2.3.1.2 Central Regulation of Force Output

To activate a particular motoneuron the current generated at the thousands of synapses within the spinal cord (rheobase current), must be great enough to generate a voltage change that will exceed the activation threshold of that motoneuron, and produce an action potential. According to Ohms law the rheobase current (I) is determined by the
voltage change (V) required to produce an action potential and the input resistance (R) of the motoneuron (I = V / R). Assuming that the membrane behaves linearly between the resting potential and the voltage at which an action potential is generated, and considering that resting potential does not differ between motoneurons, it appears that input resistance is the major determinant of the current required to activate the motoneuron (Gardiner 2001). Input resistance of a motoneuron is inversely proportional to its size (MacIntosh, Gardiner & McComas 2006). Therefore, referring back to Ohms law, smaller motoneurons have a lower activation threshold because a smaller rheobase current is required to generate an action potential. Motoneuron size is typically consistent with the type of motor unit it belongs to. Type I motor units have a smaller motoneuron than type II units. Therefore, motoneurons belonging to type I motor units are more excitable than type II motor units. As a result, it is widely accepted that during voluntary activation a ‘size principal’ exists (Henneman, Somjen & Carpenter 1965), whereby type I motor units are recruited first, followed type II as the force demand increases (Monster, Chan 1977).

Once a motor unit is recruited i.e., its activation threshold has been achieved, further increases in the applied rheobase current will result in proportional increases in firing frequency of that motor unit (Heckman, Binder 1991). Considering type I motor units have a lower activation threshold, this would assume that they also respond with a higher firing frequency for the same rheobase current in excess of the activation threshold. However, firing frequency of a motor unit is also determined by its refractory period (or hyperpolarisation period) following motoneuron recruitment (MacIntosh, Gardiner & McComas 2006). Hyperpolarisation is typically shorter in motoneurons belonging to type II motor units, so type II motor units are likely to have a greater maximum firing frequency than type I units.

Increasing the number of recruited motor units will increase the number of muscle fibres contributing to force production, and in turn increase force output (MacIntosh, Gardiner & McComas 2006). Force output of a muscle will also depend on the firing frequency of its individual motor units. The effect of firing frequency on force output can be understood by considering the contractile response of a muscle to one vs. multiple electrical impulses. If the muscle is stimulated with a single electrical impulse the muscle will contract and relax; this is known as a twitch contraction (Enoka 2008).
If a second electrical impulse is elicited before the muscle has completely relaxed from the first (typically within 125-200 ms of the first impulse; 5-8 Hz; (Thomas, Johansson & Bigland-Ritchie 1991, Fuglevand, Macefield & Bigland-Ritchie 1999)), the force output will be a summation of the two impulses. This summation of force, which will continue as long as consecutive impulses are elicited at a frequency that doesn’t allow the muscle to relax, will eventually plateau. This contractile response is known as a tetanic contraction. The amplitude of the tetanus will depend on the frequency at which impulses are elicited (firing frequency), with higher firing frequencies inducing greater tetanic peak forces. The force-frequency relationship is described by a sigmoid curve (Figure 2.2; (Fuglevand, Macefield & Bigland-Ritchie 1999, Macefield, Fuglevand & Bigland-Ritchie 1996)); the greatest change in peak force occurs between frequencies of 8-20 Hz, as at higher frequencies peak force begins to plateau before reaching a maximum between 80-100 Hz (Enoka 2008, Thomas, Johansson & Bigland-Ritchie 1991, Fuglevand, Macefield & Bigland-Ritchie 1999, Macefield, Fuglevand & Bigland-Ritchie 1996).

![Figure 2.2](chart.png)

**Figure 2.2.** The relationship between motor unit firing frequency and peak tetanic force in a human foot muscle. Maximum tetanic force occurs between 80-100 Hz. Adapted from the data of Macefield, Fuglevand & Bigland-Ritchie (1996).

Firing frequency of a motor unit will also influence its peak RFD. The relationship between motor unit stimulation frequency and peak RFD has been documented by de Ruiter et al. (1999) for the adductor pollicis (Figure 2.3). Peak RFD increased with firing frequency, and the rate of this increase was greatest at the lower frequencies (2-50 Hz). Beyond 50 Hz peak RFD continued to increase, but at a more gradual rate, with
the greatest peak RFD being recorded at 300 Hz. In human whole-muscle mean motor unit firing frequencies in the initial phase (e.g., first 4 impulses) of explosive voluntary contractions (up to 189 Hz; (Van Cutsem, Duchateau & Hainaut 1998, Desmedt, Godaux 1977)) are typically higher than those required for maximum force output (80-100 Hz), but decrease immediately within this initial phase (Van Cutsem, Duchateau & Hainaut 1998). It therefore appears unlikely that humans will be able to achieve their greatest evoked peak RFD. An interesting characteristic of motor unit activation that also appears to benefit RFD is the catch property (Rothwell 1987, Van Cutsem, Duchateau 2005). This is where a single additional electrical impulse is inserted into a low frequency tetanic contraction, within 5 ms of a previous impulse (Van Cutsem, Duchateau & Hainaut 1998), resulting in a rapid rise in force that plateaus and remains at a level greater than that observed prior to the inserted impulse (Rothwell 1987).

Figure 2.3. The relationship between peak rate of force development (RFD) and stimulation frequency during electrically evoked contractions of human adductor pollicis muscle. Maximum peak RFD occurred at 300 Hz. Adapted from the data of de Ruiter et al. (1999).

2.3.1.3 Measuring Neural Activation

Several methods have been employed to assess neural activation (and/or the excitability of motor neurons) and include electromyography (EMG), the interpolated twitch technique (ITT), V-waves, H-waves, mechanomyograms, trans-cranial magnetic stimulation, and magnetic resonance imaging. This section will review the two methods that have been most commonly used to quantify neural activation at MVF, and during explosive contractions; EMG and ITT.
Electromyography measures the voltage potential generated across the sarcolemma of muscle fibres, in response to neural activation (MacIntosh, Gardiner & McComas 2006, De Luca 1997). There are two methods of EMG analysis; intramuscular and surface. Intramuscular EMG can be used to measure the activation timing and firing rate of individual motor units. Data is collected via fine wire or needle electrodes which are positioned within the belly of the muscle (Turker 1993). Intramuscular EMG however, is better suited for analysing small peripheral muscles, or those located deep within the body, rather than large muscle groups (Turker 1993). Additionally, the impulse patterns become too dense during strong muscular contractions to accurately determine the activation patterns of individual motor units (MacIntosh, Gardiner & McComas 2006). Consequently, intramuscular EMG is not ideal for assessing neural activation during maximal and/or explosive contractions.

Surface EMG is better suited for analysing larger muscle groups, and involves the placement of two or more electrodes on the surface of the skin, over the muscle belly. The surface EMG signal is a summation of all detected voltage potentials at the skin surface, and is affected by the amplitude, duration and shape of these motor unit action potentials, the number of activated motor units, the firing frequency of motor units, and the conduction velocity of an action potential across the activated muscle fibres (De Luca 1997). Specifically, amplitude of the EMG signal increases with increased motor unit recruitment and firing frequency, suggesting that it might be a useful measure of neural activation (De Luca 1997). The characteristics of the EMG signal can be further assessed by converting the signal into its frequency-power spectrum using a Fast Fourier Transformation. This can be used as a fatigue index (Lindstrom, Kadefors & Petersen 1977), and may provide information on the muscle fibre action potential conduction velocity (van Boxtel, Schomaker 1984, Farina 2006).

There are however, many complex methodological and physiological factors unrelated to the level of neural activation that can influence the surface EMG signal. These factors include electrode placement, signal crosstalk from other muscles, subcutaneous tissue, blood flow, fibre diameter, muscle biochemistry (De Luca 1997), equipment noise (Turker 1993), signal nonstationarity (change in signal properties with respect to time), and electrode shift (movement relative to the contracting muscle fibres; (Farina...
Furthermore, one study has compared EMG amplitudes in two simulated signals that were created from the sum of separate motor unit action potentials; one before and one after rectifying the action potentials (Keenan et al. 2005). They identified reduced EMG amplitude of up to 85% in the signal that was not pre rectified (which represents the response of surface EMG in vivo), and suggested that this was due to signal cancelation when summing negative with positive portions of the separate action potentials. These confounding factors have led some investigators to question the use of surface EMG for quantifying neural activation (Keenan et al. 2005). Nevertheless, the positive curvilinear relationship between EMG amplitude and force output (Kooistra, de Ruiter & de Haan 2007, Disselhorst-Klug, Schmitz-Rode & Rau 2009, Alkner, Tesch & Berg 2000) supports its use as a global indicator of the level of neural activation.

Due to the many factors that can affect EMG amplitude a normalisation procedure is widely recommended (Burden, Bartlett 1999) if the purpose of a study is to compare EMG amplitude between individuals, groups or over repeated trials. One common method of EMG normalisation is to report EMG relative to peak EMG during an isometric MVC (Burden, Bartlett 1999); however, this method becomes problematic when it is necessary to compare peak EMG during an MVC across participants and trials. An alternative method of normalising EMG is to report it as a ratio of a maximal M-wave (Mmax) (Gandevia 2001). The M-wave is a compound muscle action potential recorded in response to electrical stimulation of the motoneurons, and Mmax is achieved when all motoneurons in a motor pool are activated maximally (Maffiuletti et al. 2001). Therefore, Mmax coincides with the maximum force response for a single electrical impulse, and is thus likely to be a more reliable variable for normalisation of EMG amplitude during a voluntary contraction (Folland, Williams 2007a, Gandevia 2001).

Another method that has been employed to assess the level of voluntary activation is the ITT. The ITT involves comparing the amplitude of an electrically evoked maximal twitch contraction elicited while at rest, with that of one superimposed upon a MVC (Gandevia, Herbert & Leeper 1998). The rationale for the ITT is that the superimposed twitch will recruit those motor units not already active and/or increase the firing frequency of active motor units that are firing at submaximal frequencies (Taylor 2009). Therefore, the superimposed twitch response as a proportion of the control twitch is
considered to represent the inactive component of the muscle, with any twitch response during an MVC thought to demonstrate sub-maximal activation. Quantitatively the ratio of the superimposed to control twitch, subtracted from 1 and multiplied by 100, has been widely used to quantify the level maximal voluntary activation (Del Balso, Cafarelli 2007, Pucci, Griffin & Cafarelli 2006, Taylor 2009, Jones, Rutherford 1987, Reeves, Narici & Maganaris 2004).

Several investigators have questioned the use of the ratio of superimposed twitch to control twitch as a reliable method for measuring maximal activation (de Haan, Gerrits & de Ruiter 2009). This method assumes that the superimposed twitch occurs at MVF, and that the relationship between the superimposed twitch force and voluntary force is linear (Folland, Williams 2007b). However, it is unlikely that the investigator will be able to elicit the superimposed twitch at precisely the same instant as MVF occurs. Furthermore, the relationship between the superimposed twitch force and voluntary force tends to be more of a concave curvilinear shape, whereby there are very small changes in superimposed twitch force at high levels (>80%) of MVF (Kooistra, de Ruiter & de Haan 2007). Considering these issues Folland, Williams (2007b) suggested that a more valid measure of maximal voluntary activation may be obtained by calculating the difference between MVF and theoretical maximum force (TMF), where TMF has been extrapolated from an appropriate curvilinear model of the superimposed twitch force-voluntary force relationship. However, few studies have adopted this variation of the ITT method for quantifying maximal voluntary activation.

Regardless of the ITT method used to quantify maximal voluntary activation, there are several methodological issues that should be considered to improve the reliability and/or validity of the measurements. The superimposed twitch appears to be potentiated, and should therefore be compared with a potentiated control twitch elicited immediately after the MVC, rather than an unpotentiated control twitch elicited before the MVC (Folland 2009). Furthermore, the MVC should be performed in a low compliant isometric rig, and at low compliance muscle-tendon lengths, as these factors contribute to non-linearities in the superimposed twitch-voluntary force relationship (Taylor 2009, Loring, Hershenson 1992, Allen, McKenzie & Gandevia 1998, Arampatzis et al. 2007b). Finally, some authors have suggested that a superimposed doublet (two electrical impulses, rather than one) elicited at 100 Hz, may provide a
more valid measure of activation, as the second impulse is likely to depolarise those motor units that were not firing maximally, but were in a refractory period at the time of the first impulse (Duchateau 2009). However, evidence suggests that activation determined via multiple superimposed impulses is similar to that of a single twitch (Folland, Williams 2007b, Allen, McKenzie & Gandevia 1998, Behm, St-Pierre & Perez 1996, Scaglioni, Martin 2009). Furthermore, the increased discomfort experienced during evoked contractions of multiple impulses, has been shown to cause inhibition and thus lower voluntary forces during the MVC (Button, Behm 2008). This supports the use of single superimposed twitch contractions when quantifying maximal voluntary activation via the ITT.

A limitation of the ITT is that the superimposed twitch must be elicited during a stable part of the force-time curve to ensure: one, accurate interpolation of the twitch force; and two, that the superimposed twitch occurs at a stable level of voluntary activation. Therefore, whilst the ITT method has been used to assess maximal voluntary activation at the plateau of a MVC, it cannot be used to assess explosive voluntary activation during the ascending force-time curve, when neither the slope of the curve or the level of activation is linear. Therefore, surface EMG normalised to $M_{max}$ would appear to be a more useful method of quantifying neural activation during an explosive contraction. Alternatively, one study has compared RFD recorded during an explosive voluntary contraction, to that measured during an electrically evoked supramaximal octet (8 impulses at 300 Hz) contraction (de Ruiter et al. 2004). The supramaximal octet was thought to elicit the maximal RFD from the muscle, and thus provide a reliable reference for comparison of effectiveness of voluntary RFD by the nervous system. Therefore, the ratio of voluntary to octet RFD provides a measure of the ability to utilise the maximal capacity of the MTU for explosive force production, during a voluntary contraction.

### 2.3.2 Peripheral Factors

#### 2.3.2.1 Muscle Size

Whole muscle consists of cylindrical muscle fibres grouped into bundles known as muscle fascicles. Each muscle fibre consists of a bundle of myofibrils arranged in
parallel, and each myofibril is a series of contractile elements, known as sarcomeres. Sarcomeres consist of thick (myosin) and thin (actin) filaments (contractile proteins) that form cross-bridges under certain biochemical conditions, causing the muscle to contract and thus produce force (the process of muscular contraction is discussed in more detail in section 2.3.1). Theoretically an increased number of contractile proteins (and thus sarcomeres) arranged in parallel, will increase the number of cross-bridges at any one time, which in turn will increase the amount of force produced per cross sectional area. A greater number of sarcomeres in parallel is likely to increase the size of muscle, and therefore it is no surprise that studies have observed a strong relationship (0.58<r<0.86) between indices’ of muscle size (such as anatomical and physiological cross-sectional area, and muscle volume) and maximum strength (Hakkinen, Keskinen 1989, Maughan, Watson & Weir 1983, Schantz et al. 1983, Bamman et al. 2000, Hakkinen, Hakkinen 1991).

2.3.2.2 Muscle Architecture

Skeletal muscle can be categorised depending on the alignment of its fibres relative to the tendon and aponeurosis; muscle fibres of parallel and fusiform muscles are arranged parallel with the tendon and aponeurosis; whilst muscle fibres of pennate muscles are at an oblique angle to the tendon and aponeurosis (Figure 2.4; (MacIntosh, Gardiner & McComas 2006)). In pennate muscle the angle (θ) of pennation will influence maximum strength, as the sum of forces being transmitted to the aponeurosis by the individual fibres is reduced by a factor of cosθ (Fukunaga et al. 1997). However, a larger angle of pennation allows a greater number of muscle fibres to be arranged in parallel, resulting in a trade-off (Folland, Williams 2007a, MacIntosh, Gardiner & McComas 2006), where isometric force per anatomical cross sectional area increases with increased angle of pennation until 45º (Alexander, Vernon 1975); beyond which the reduced transfer of force to the tendon (caused by the angle of pennation) puts the muscle at a mechanical disadvantage.
2.3.2.3 Muscle Composition

Whole muscle contains different types of muscle fibres that are catagorised as type I (slow twitch) and type II (fast twitch) fibres (the latter can be further catagorised into type IIA and Type IIX), depending on their contractile properties. The contractile properties of muscle and their relevance to force production are discussed in greater detail in section 2.3.2.6. There is evidence that maximum strength is positively related to a greater distribution of fast twitch muscle fibres (Maughan, Watson & Weir 1983, Thorstensson, Grimby & Karlsson 1976, Aagaard, Andersen 1998). This may be partly due to the higher specific tension (force per cross sectional area) that has been observed in fast twitch fibres (Bottinelli et al. 1999, Stienen et al. 1996), although some investigators have questioned this phenomenon (Fitts, McDonald & Schluter 1991).
2.3.2.4 Muscle Length / Joint Angle

The influence of muscle (sarcomere) length on the capacity for maximal force production has been widely documented \textit{in vitro} via the force-length relationship (Rassier, MacIntosh & Herzog 1999, Gordon, Huxley & Julian 1966, Close 1972, Zuurbier et al. 1995) (Figure 2.5); where force output is proportional to the amount of overlap between the myosin and actin filaments, and between adjacent actin filaments of the same sarcomere. Along the descending limb of the force-length curve (long sarcomere lengths) there is minimal overlap between myosin and actin filaments, minimising the number of potential myosin-actin cross-bridges and thus minimising the capacity for force production. At the peak of the force-length relationship there is optimal overlap between the myosin and actin filaments maximising the number of potential myosin-actin cross bridges, and thus maximising the capacity for force production. Along the ascending limb of the force-length relationship (short sarcomere lengths) there is overlap of the adjacent actin filaments which interferes with the myosin-actin cross-bridge mechanism, and minimises the capacity for force production. A similar force-length relationship is observed with whole muscle fibres, although the curve is rounded due to non-uniform sarcomere lengths along the fibres (Gordon, Huxley & Julian 1966).
Figure 2.5. A schematic of a single sarcomere at different lengths (A, B, C, D, E, and F), and a graph illustrating how different sarcomere lengths influence force production (force-length relationship). Along the descending limb of the force-length relationship (A-B) there is minimal overlap of the actin and myosin filaments, minimising the number of potential myosin-actin cross-bridges, and therefore minimising force production. At the plateau of the force-length relationship there is optimal overlap of the myosin and actin filaments (B-C), maximising the number of potential myosin-actin cross-bridges and therefore maximising force production. Along the ascending limb of the force-length relationship (D-F) adjacent actin filaments overlap, interfering with the myosin-actin cross-bridge mechanism, and thus reducing the potential for force production. At very short sarcomere lengths (E-F) the myosin filament collides with the z-discs causing further disruption to the myosin-actin cross-bridge mechanism. Adapted from Gordon, Huxley & Julian (1966) and Rothwell (1987).

In humans *in vivo* the MTU consists of four components; a contractile component, which consists of the contractile proteins; a series elastic component (SEC), which consists of the connective tissue in series with the contractile component (tendon and aponeurosis); a parallel elastic component, which consists of the connective tissue in parallel with the contractile component; and a viscous component, which works as a mechanical damper. Whilst the contractile component of *in vivo* whole muscle displays the same force-length relationship as that observed *in vitro*, the parallel and series
elastic components provide a passive tension, which increases with muscle length, adding to total force production (Rassier, MacIntosh & Herzog 1999, Rack, Westbury 1969). Furthermore in vivo muscles cross joints, and therefore generate moments of force (force multiplied by internal moment arm; often measured as torque). Changes in joint angle results in concomitant changes in MTU length and internal moment arm (Pigeon, Yahia & Feldman 1996), which both influence maximal torque production (Figure 2.6). The in vivo voluntary torque-angle relationship of joints in humans is usually described by a parabola (King, Wilson & Yeadon 2006) or normal distribution curve (Forrester et al. 2010; In Press).

**Figure 2.6.** The torque-angle relationship (A) and the influence of joint angle on internal moment arm (single line) and muscle length (dashed line) of a hypothetical joint in the human body (B). Joint torque is the product of muscle force production (which is limited by muscle length) and the internal moment arm of the muscle (which changes with joint angle). The torque-angle relationship is described by a parabola (King, Wilson & Yeadon 2006).
2.3.2.5 Type of Contraction

There are three types of muscular contraction; concentric, the muscle shortens under tension; eccentric, the muscle lengthens under tension; and isometric, the muscle is under tension but does not change length. The influence of the type of contraction on the capacity for maximal force production is depicted via the *in vitro* force-velocity relationship (Figure 2.7A); where concentric force is lower than isometric force, and decreases in a hyperbolic curve with increased shortening velocity (Hill 1938); and eccentric force increases initially with lengthening velocity (as depicted by a rectangular hyperbola), but plateaus at $\geq 140\%$ of isometric force (Hill 1938, Edman, Elzinga & Noble 1978, Harry et al. 1990).

An explanation for the *in vitro* force-velocity relationship was provided by Huxley (1957), who suggested that the rate of myosin-actin cross-bridge detachment during isometric contractions was slower than the rate of attachment, increasing the number of active cross-bridges at any one time, and thus increasing the capacity for force production. In contrast, the rate of detachment increases with increased shortening velocity during concentric contractions, reducing the number of active cross-bridges at any one time, and thus decreasing the capacity for force production. Huxley (1957) also predicted that the myosin-actin cross-bridge displays elastic properties over a relatively wide range of distances (~10 nm). As a result, the cross-bridge will be stretched during an eccentric contraction increasing the amount of force developed between the actin and myosin filaments. However, during concentric contractions the myosin head may attach at a relatively close binding site, which would reduce the elastic tension, and thus force produced, between the actin and myosin filaments. Furthermore, as concentric velocity increases it is likely that more myosin-actin cross-bridges will form beyond the point of zero force, and therefore contribute a negative force to net force output. At maximum shortening velocity net force is zero because those cross-bridges in a state of positive force production are equal to those in a state of negative force production (Rothwell 1987).

In humans *in vivo* during MVCs the force-velocity relationship differs to that measured *in vitro*, whereby eccentric maximum force is typically only 90-110% of isometric maximum force (Pain, Forrester 2009, Webber, Kriellaars 1997, Dudley et al. 1990).
(Figure 2.7B). This discrepancy is thought to be due to a neural inhibition mechanism during the lengthening contraction that protects the MTU from potentially harmful loads. Studies utilising EMG to assess neural drive have measured lower agonist muscle EMG amplitudes during eccentric than concentric contractions (Pain, Forrester 2009, Aagaard et al. 2000, Seger, Thorstensson 1994, Westing, Cresswell & Thorstensson 1991, Babault et al. 2001).

**Figure 2.7.** A schematic of the *in vitro* force-velocity relationship (A) and the *in vivo*, in humans voluntary force-velocity relationship (B). The dashed line represents an isometric condition (zero velocity). *In vitro* maximum eccentric force production is up to $\geq 140\%$ of maximum isometric force production; however, maximum eccentric force production during maximal voluntary contractions in humans is similar to that of isometric maximum voluntary force. The capacity for force production decreases with increased shortening (concentric) velocity in both situations.
2.3.2.6 Intrinsic Contractile Properties

The contractile properties of a skeletal muscle describe its force response to a known electrically or magnetically evoked input stimulus (e.g., supramaximal single impulse that elicits a twitch contraction or a train of impulses to evoke a tetanic contraction). The contractile properties can be measured for a single motor unit, a skinned muscle fibre, or for a whole muscle group in vivo. For analysis of single motor unit contractile properties, a needle electrode is inserted into a nerve bundle to elicit electrical impulses along individual motoneurons (Thomas, Johansson & Bigland-Ritchie 1991, Fuglevand, Macefield & Bigland-Ritchie 1999). The contractile properties of a skinned muscle fibre are assessed by attaching the fibre to a rig and passing it in and out of two chambers containing activation and relaxation fluid (Harridge et al. 1996). For a whole muscle group in vivo, the contractile properties are assessed by eliciting impulses via percutaneous electrical stimulation (or magnetic stimulation) directly over the muscle belly (Harridge et al. 1996, Maffiuletti 2010, Bergquist, Clair & Collins 2010), or over the nerve bundle that innervates the muscle of interest (de Ruiter et al. 2004, Bergquist, Clair & Collins 2010).

Whilst the contractile properties may explain many aspects of muscular contraction (such as the force-frequency relationship, explained in section 2.3.1.2; and fatigability), this section of the review will focus on the contractile properties measured to quantify the force response to a single twitch or tetanic contraction. In an isometric situation these typically include peak force, time to peak force (often referred to as contraction time), RFD (peak RFD or RFD over a given time period), and half relaxation time (time for the descending force-time curve to reach half peak force; Figure 2.8; (Enoka 2008, MacIntosh, Gardiner & McComas 2006)). It is also common to measure maximum unloaded shortening velocity during dynamic conditions in vitro (Harridge et al. 1996).
Figure 2.8. A schematic of the isometric force-time response of a single muscle fibre to a single electrical impulse. The characteristics of this response can be assessed by measuring the intrinsic contractile properties which include peak force, time to peak force, rate of force development on the ascending slope, and half relaxation on the descending slope.

Skinned muscle fibres and/or single motor units can be categorised into type I (fast twitch) or type II (slow twitch) muscle fibres/motor units depending on their contractile properties (Burke et al. 1973). Typically, fast twitch muscle fibres/motor units display higher peak forces, RFD, and maximum unloaded shortening velocity, as well as a shorter time to peak force and half relaxation time, than slow twitch fibres/motor units (Bottinelli et al. 1999, Harridge et al. 1996, Burke et al. 1973, Garnett et al. 1979). This discrepancy between fast and slow twitch fibres/motor units is thought to be due to the histochemical properties and the myosin heavy chain (MHC) isoform expression of the associated muscle fibres. These factors will influence the excitation-contraction coupling process, and therefore the contractile response to a certain stimulus. For example, studies have shown that fast and slow twitch fibres express different types of myosin ATPase (Harridge et al. 1996, Burke et al. 1973, Garnett et al. 1979, Edman et al. 1988), and it is assumed that the type of ATPase in fast twitch fibres favours a faster rate of ATP hydrolysis, and thus rate of cross-bridge cycle. Furthermore, fast twitch fibres have been shown to contain a greater concentration of anaerobic enzymes (such as phosphorylase; (Burke et al. 1973)), which would increase the rate of ATP resynthesis and thus ATP availability during the excitation-contraction coupling process. Finally, fast twitch fibres are composed of type IIA or IIX MHC compared to slow twitch fibres which are composed of type I MHC (Bottinelli et al. 1999, Harridge et al.
1996, Danieli-Betto, Betto & Midrio 1990, Larsson, Moss 1993). The type of MHC is thought to affect the Ca$^{2+}$ sensitivity and the myosin-actin cross-bridge interaction (Bottinelli et al. 1999, Danieli-Betto, Betto & Midrio 1990, Larsson, Moss 1993) in a way that favours a greater rate of cross-bridge cycling in fast twitch fibres.

Whilst the relationship between the contractile properties of the muscle and its histochemical and/or molecular characteristics is well documented for skinned muscle fibres and single motor units, there is limited evidence of an association between these parameters in whole muscle \textit{in vivo}. Harridge et al. (1996) analysed the contractile properties and the percentage distribution of type II (A and X) MHC of three muscles (soleus, vastus lateralis, and triceps brachii) in a small cohort (n = 7) of human participants. The greatest percentage distribution of type II MHC, shortest time to twitch peak force, and shortest time to 50% of peak tetanic (50 Hz) force was recorded in the triceps brachii, followed by the vastus lateralis and the soleus. Whilst these results suggest an association between contractile properties and the distribution of MHC in whole muscle \textit{in vivo}; the contractile properties of the three separate muscles in this situation are also likely to be influenced by muscle architecture, muscle length, moment arm, and MTU stiffness. Surprisingly, when Harridge et al. (1996) considered each muscle separately there was no relationship between the contractile properties of that muscle and the percentage distribution of MHC II. Whilst this may partly be due to the small sample size (n = 7), further work is clearly required to understand the association between the contractile, histochemical and molecular properties of whole muscle \textit{in vivo}.

\subsection*{2.3.2.7 Muscle-Tendon Unit Stiffness}

When a force is applied to a material it will result in deformation of that material. Stiffness is a mechanical property that explains the resistance of a material to this deformation. In a material with one degree of freedom stiffness can be defined as the slope of the force-deformation curve; where a steeper slope (i.e., less deformation for a given force) corresponds to a stiffer material, whilst a shallower slope (i.e., more deformation for a given force) corresponds to a more compliant material (e.g., compliance = 1 / stiffness).
When muscles contract *in vivo* the forces they generate must be transmitted to the bones via the parallel elastic component and SEC. As force production increases from rest there is a relative shortening of the contractile component and lengthening of the SEC (Figure 2.9). This displacement can be seen when an ultrasound probe, positioned over the muscle belly (Stafilidis et al. 2005, Kubo, Kanehisa & Fukunaga 2005, Arampatzis et al. 2007a) or muscle-tendon junction (Bojsen-Moller et al. 2005, Stafilidis et al. 2005, Kubo, Kanehisa & Fukunaga 2005, Kubo et al. 2007a), is used to record video images of the MTU during a contraction. Displacement can be quantified by digitising the position of cross points (Bojsen-Moller et al. 2003), either between the muscle fascicles and the aponeurosis (if the probe was placed over the muscle belly) or between the muscle and tendon (if the probe was placed over the muscle-tendon junction), in video images recorded during a progressive increase in force (Figure 2.9). Displacement can then be plotted against internal muscle force recorded at the same point in time during the contraction (Figure 2.10), and the slope of the force-displacement curve used to define MTU stiffness (if displacement was recorded at the aponeurosis; (Stafilidis et al. 2005, Kubo, Kanehisa & Fukunaga 2005)), and tendon stiffness (if displacement was recorded at the muscle-tendon junction; (Stafilidis et al. 2005, Kubo, Kanehisa & Fukunaga 2005)). In exercise science it is typical to record MTU (or tendon) stiffness during a ramped MVC and define stiffness as the slope of the curve from 50-90% (Bojsen-Moller et al. 2005, Bojsen-Moller et al. 2003) or 50-100% (Kubo et al. 2007a) of MVF, as this appears to be the most linear portion of the force-displacement curve (Figure 2.10).
Figure 2.9. A schematic of a muscle-tendon unit at rest (A) and during a maximal voluntary contraction (B). During the contraction, force produced by the muscle causes the muscle fascicles to shorten. This pulls on the aponeurosis and tendon (series elastic components), causing them to stretch. By tracking the displacement of cross points between the muscle fascicles and the aponeurosis (CP 1) or between the aponeurosis and tendon (CP 2), and plotting this displacement against force recorded during the same contraction it is possible to calculate muscle-tendon unit stiffness (slope of the force-displacement curve).

Figure 2.10. The relationship between force and displacement of the aponeurosis recorded at the vastus lateralis muscle during an isometric maximal voluntary contraction. The slope of this curve between 50-90% of maximal voluntary force (denoted by the thick dashed line) is a measure of muscle-tendon unit stiffness.
Studies have also measured MTU stiffness indirectly using a method known as the free-oscillation technique (Wilson, Murphy & Pryor 1994, Walshe, Wilson & Murphy 1996, Watsford et al. 2010, Wilson, Wood & Elliott 1991, Ditroilo, Watsford & De Vito 2010). During this method the participant is positioned in an isometric rig, set up to isolate the joint of interest, and maintains an isometric contraction at a pre-determined percentage of their MVF. A perturbation is then applied to the loaded system, causing oscillations in joint acceleration that are quickly damped due to the viscoelastic properties of the MTU (Ditroilo, Watsford & De Vito 2010). Analysis of the acceleration time curve produced by these oscillations (which can be recorded with an accelerometer), are used to determine the damped natural stiffness and the damping coefficient, which in turn can be used to determine MTU stiffness. The free-oscillation technique is described in detail by Walshe, Wilson & Murphy (1996). Whilst it is considered to be a reliable method (Walshe, Wilson & Murphy 1996, Ditroilo, Watsford & De Vito 2010), it is important to note that MTU stiffness measured using the free-oscillation technique will also be influenced by stiffness of the skin, ligaments, and articular surfaces (Ditroilo, Watsford & De Vito 2010), as well as the level of co-contraction of the agonist and antagonist muscles, and is therefore sometimes defined as musculo-articular stiffness (Ditroilo, Watsford & De Vito 2010). Considering that this measure is used to infer MTU stiffness, this is the term that will be employed throughout this thesis to avoid confusion.

MTU stiffness is likely to influence force output in several ways: one, a more compliant MTU will result in greater shortening of the contractile element resulting in a change in position on the force-length relationship (Maganaris, Baltzopoulos & Sargeant 2006); two, the greater muscle shortening associated with a more compliant MTU, will increase the contractile velocity of the muscle, causing a shift in its position on the force-velocity curve (Bojsen-Moller et al. 2005, Wilson, Murphy & Pryor 1994, Wilkie 1949); and three, a stiffer tendon is thought to provide a more efficient transfer of force to the bone (Wilson, Murphy & Pryor 1994). These effects are discussed in more detail with relation to RFD in section 2.4.4.
2.4 Evidence for the Determinants of Explosive Strength

2.4.1 Maximum Strength

Theoretically, a muscle with a greater maximal force capacity will have a greater RFD, should all the other determinants of explosive force production be equal. However, in humans in vivo maximum strength is only one contributing factor to RFD. When considering the inter-individual differences in RFD the relative contribution of maximum strength to RFD appears to depend on the proportion of the force-time curve being considered. Andersen, Aagaard (2006) measured the bivariate relationship between knee extensor RFD over different time periods form force onset (e.g., 0-10, 0-20, 0-30…0-200 ms) and MVF. Results showed that MVF accounted for only 18-25% of the variance in initial voluntary RFD (0-50 ms); however, RFD became increasingly more dependent on MVF as the time from force onset increased, so that MVF accounted for 52-81% of the variance in RFD over time periods ≥ 90 ms (Figure 2.11). Therefore, maximum strength appears to be a good determinant of explosive strength during the late, but not early phases of an explosive contraction. This may be expected considering that force during an explosive contraction is increasing towards a fixed asymptote at 100% MVF. Given the influence of maximum strength on explosive strength, studies should normalise explosive force to MVF in order to compare the relative capacity for RFD across participants and sessions. Furthermore, normalising RFD to MVF will enable a clearer investigation of the other neural and mechanical determinants of RFD.
Figure 2.11. The bivariate relationship (Pearson’s R) between maximal voluntary force and rate of force development recorded over different time periods from force onset (0-10, 0-20…0-250 ms) during isometric explosive voluntary contractions of the knee extensors. The relationship was significant (P<0.05) at all time periods. Adapted from the results of Andersen, Aagaard (2006).

2.4.2 Neural Activation

Theoretically, a greater neural drive to the muscles during an explosive voluntary contraction (resulting in an increased number and firing frequency of recruited motor units) will benefit explosive force production. However, the influence of neural activation on voluntary RFD, particularly when considered relative to the influence of peripheral factors (e.g., maximum strength, contractile properties, and tendon stiffness), has not been widely investigated. Studies have reported strong relationships \( r = 0.80-0.91 \) in small cohorts \( n = 7-11 \) between agonist EMG (normalised to EMG at MVF or \( M_{\text{max}} \)) and explosive torque production (normalised to either MVT, or explosive torque during a 300 Hz octet) recorded during the first 40 ms of explosive voluntary contractions of the knee extensors (de Ruiter et al. 2004, de Ruiter et al. 2006, de Ruiter et al. 2007). These results provide good evidence that the level of neural activation has a strong influence on explosive force production during the initial phase (first 40 ms), of an explosive voluntary contraction. Unfortunately, these studies did not analyse RFD and EMG during later phases of the contraction, and so the contribution of neural activation to RFD during longer time phases is unclear.
2.4.3 Contractile Properties

A greater RFD of whole muscle *in vivo* during electrically evoked involuntary contractions represents a greater capacity of the MTU for explosive force production, which should theoretically benefit RFD during an explosive voluntary contraction. However, there is limited evidence to support an association between measures of involuntary and voluntary explosive force production. Andersen, Aagaard (2006) observed only a moderate relationship (0.45<r<0.60) between twitch peak RFD and voluntary RFD of the knee extensors during the initial phase (0-50 ms) of an explosive contraction, and this relationship became weaker when voluntary RFD was analysed over longer time periods from contraction onset (>50 ms; P>0.05). It is important to note that RFD (voluntary and involuntary) in this study was not normalised to MVF, and therefore the observed association between initial voluntary and peak involuntary RFD may have been partly due to a common influence of maximum strength on both variables.

Twitch peak RFD is only 25-30% of the maximal RFD (de Ruiter et al. 1999), and therefore a single twitch contraction may not be a good indicator of the explosive capacity of the MTU. A stronger association between voluntary and involuntary RFD may be observed if a tetanic contraction eliciting the maximal RFD is employed instead of a twitch contraction. Two studies have measured maximal explosive torque production during the initial 40 ms of electrically evoked octet contractions (8 electrical impulses at 300 Hz), but surprisingly found that it was unrelated to explosive voluntary torque production in the same time period (de Ruiter et al. 2004, de Ruiter et al. 2007). However, the sample sizes in these investigations were small (n = 7-11), and explosive voluntary force production beyond the initial 40 ms (<20% MVF) was not assessed, so it is unclear how involuntary RFD may relate to voluntary RFD at later time points in the contraction.

Whilst there is limited evidence of the association between evoked contractile properties of the muscle and voluntary RFD, an early study has reported low-moderate correlations (0.34<r<0.48) between fibre composition (determined via histochemical analysis) of the vastus lateralis, and time taken to achieve different proportions of MVF (5% increments) during explosive voluntary isometric leg press (Viitasalo, Komi 1978).
Specifically, participants with a greater distribution of fast twitch fibres recorded greater RFD normalised to MVF. Whilst the histochemical characteristics of the muscle (which are thought to influence the contractile properties) only explained 12-23% of the variance in normalised explosive voluntary force production, this is one of the few studies to provide good evidence of an association between these parameters in humans in vivo. The sample size in this study was relatively large (n = 39), and consisted of a broad spectrum of individuals with different athletic/training backgrounds, and with a wide range of percentage slow twitch fibres (~35-85%). It is therefore possible that these conditions are required to identify a commonality between fibre type/contractile properties and voluntary explosive performance. Clearly, further work is required to understand the influence of the intrinsic contractile properties and/or fibre type distribution of the muscle on voluntary RFD.

### 2.4.4 Muscle-Tendon Unit Stiffness

The influence of system compliance on muscular RFD was first identified by Wilkie (1949). This early investigation measured a reduced RFD of the wrist flexors when a compliant spring was introduced between the participant and the force transducer, suggesting that a stiffer system is more conducive to explosive force production. Whilst the independent variable in this study was compliance of the testing equipment (and not the MTU), MTU compliance will reduce overall stiffness of the system and is therefore also likely to influence RFD negatively. Evidence for this theory has been provided by more recent studies that have reported a positive linear relationship between MTU stiffness (measured using the free-oscillation technique) and RFD in single (Ditroilo, Watsford & De Vito 2010) and multiple joint actions (Wilson, Murphy & Pryor 1994, Walshe, Wilson & Murphy 1996). Likewise, Bojsen-Moller et al. (2005) measured positive correlations between MTU stiffness of the vastus lateralis (measured using ultrasonography) and absolute RFD over the initial 100 ms (r = 0.65) and 200 ms (r = 0.68) of explosive isometric knee extensions. There was also a relationship between these parameters, though it was not as strong, when both MTU stiffness and RFD were scaled to body mass (0.54<r<0.56).

The observed association between MTU stiffness and RFD may be explained by the trade-off between SEC lengthening and muscle shortening. A more compliant MTU
will result in greater lengthening of the SEC at the expense of muscle shortening, which will in turn result in an increase in contractile velocity (Bojsen-Moller et al. 2005, Wilson, Murphy & Pryor 1994, Wilkie 1949), and thus a reduced capacity for force production (as explained by the force-velocity relationship discussed in section 2.3.2.5). Furthermore, if the muscle typically operates and/or is tested on the ascending limb of its force-length relationship (discussed in section 2.3.2.4), the shortening of muscle fibres in more compliant systems will cause a shift down the ascending limb towards lower force outputs (Maganaris, Baltzopoulos & Sargeant 2006). Of course, this explanation does not account for situations where muscles are tested on the descending limb of their force-length relationship, where MTU compliance may cause a shift up the descending limb towards greater force outputs (Maganaris, Baltzopoulos & Sargeant 2006). An additional explanation for the proposed association between RFD and MTU stiffness is that a stiffer MTU is thought to augment the efficiency of force transmission to the bone (Wilson, Murphy & Pryor 1994), although evidence for this explanation is anecdotal.

Whilst there does appear to be strong evidence and a physiological rationale for a positive influence of MTU stiffness on RFD, it is important to note that the observed correlations do not denote cause and effect. As discussed in section 2.4.1, RFD is influenced by maximum strength particularly at later phases in the explosive contraction (Andersen, Aagaard 2006). Interestingly, MTU stiffness also appears to be positively related to maximum strength (Wilson, Murphy & Pryor 1994, Arampatzis et al. 2007a), which may be partly associated with the concomitant increase in these variables with strength training (Kubo et al. 2001, Kubo et al. 2007b, Seynnes et al. 2009, Kubo et al. 2010, Reeves, Narici & Maganaris 2003). Consequently, it is possible that the measured association between MTU stiffness and RFD may (at least in part) be due to a common influence of maximum strength. This possibility can be assessed by normalising RFD to MVF; however, the aforementioned studies that investigated the relationship between RFD and MTU stiffness (Bojsen-Moller et al. 2005, Wilson, Murphy & Pryor 1994, Walshe, Wilson & Murphy 1996, Ditroilo, Watsford & De Vito 2010) did not do this. Surprisingly, in the study of Bojsen-Moller et al. (2005) MTU stiffness shared a similar relationship with RFD during the first 100 and 200 ms, despite these variables being measured over different force levels (MTU stiffness, 50-90% of MVF; RFD over 100 ms, ~0-50% of MVF; and RFD over 200 ms, 0-78% of MVF).
Theoretically, we may expect MTU stiffness to be more strongly related to RFD during the first 200 ms, as these variables shared a greater similarity in the force levels over which they were measured, than MTU stiffness and RFD during the first 100 ms. Considering that the influence of maximum strength on RFD appears to be similar over both 100 and 200 ms (Andersen, Aagaard 2006), the results of Bojsen-Moller et al. (2005) may provide indirect evidence of a common influence of maximum strength on RFD and MTU stiffness. Further work, normalising RFD to MVF and assessing MTU stiffness over force levels relevant to the measured RFD time window, is required to improve our understanding of the association between MTU stiffness and RFD.

2.4.5 Muscle Length / Joint Angle

Whilst the influence of muscle length (and joint angle) on the capacity for maximal force production is well documented it is unclear how this will influence the capacity for explosive force production. It has been shown in rats that peak RFD changes with muscle length, but only in proportion to peak force (Haan, Huijing & Vliet 2003). A similar result has been reported in humans where peak RTD, and explosive torque production in the first 40 ms of explosive voluntary isometric knee extensions, changed with knee angle in proportion with MVT (de Ruiter et al. 2004). These results suggest that explosive strength is affected by muscle length in a similar way to maximum strength. However, peak RTD typically occurs during the early phase (0-67 ms) of explosive contractions (de Ruiter et al. 2004, de Ruiter et al. 2007), and de Ruiter et al. (2004) did not measure explosive torque production beyond the initial 40 ms. In contrast, animal studies have reported a faster time to peak force at shorter muscle lengths (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980, Rack, Westbury 1969). This is thought to be due to less efficient excitation-contraction coupling (specifically, lower Ca\(^{2+}\) release or a reduced affinity of troponin C for Ca\(^{2+}\) (Rassier, MacIntosh 2002)), and/or the interference of the cross-bridge formation caused by overlapping of the actin filaments (Gordon, Huxley & Julian 1966). Interestingly, the faster time to peak force with decreasing muscle length appears to be associated with an increase in the slope of the normalised force-time curve (normalised to peak force), at late (but not early) phases of the contraction (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980). Thus muscle length may affect late, but not initial, explosive strength
when normalised to maximum strength; however, this possibility has not yet been investigated in humans.

2.4.6 Type of Contraction

Although the capacity for maximal force production as a function of the type of contraction is well documented, the influence of the type of contraction on the capacity for explosive force production has not been investigated, and is therefore unknown. If this issue is to be addressed several methodological issues should be considered. First, explosive concentric and eccentric contractions should be performed at a steady state velocity or acceleration, to avoid non linear changes in velocity or acceleration confounding force measurements. Second, concentric and eccentric contractions are dynamic and therefore result in changes in joint angle, which will effect force production. Furthermore, this joint angle change is in opposite directions for concentric and eccentric contractions, whilst there is no joint angle change in isometric contractions. Consequently, it is not possible to match joint angle throughout the different types of contractions, apart from at a single time point/angle. This issue may be addressed by normalising explosive voluntary torque, recorded at any time point during the different types of contraction, to MVT at that specific joint angle and angular-velocity. This would require first establishing a dynamic MVT function (Yeadon, King & Wilson 2006, Forrester et al. 2010; In Press, King, Yeadon 2002). Another approach to address the issue of joint angle changes is to normalise the voluntary explosive torque recorded during the different types of contractions to explosive torque production during an evoked tetanic contraction in the same contractile condition. Both of these normalisation procedures (normalising to a joint angle/angular velocity specific MVT or to evoked tetanic torque in the same conditions) will also enable the investigator to assess the ability to explosively utilise the available torque producing capacity of the muscle. These methodological issues should be considered in order to understand the influence of the type of contraction on explosive force production.
2.4.7 Summary of Determinates of Explosive Strength

There are thought to be several neural and mechanical determinants of explosive force production. The influence of maximum strength on RFD appears to increase with time from force onset, whilst RFD in the initial phase of explosive contractions appears to be primarily determined by agonist activation. However, the influence of agonist activation on RFD beyond the initial 40 ms has not been investigated. Theoretically, the contractile properties of the muscle and MTU stiffness will influence voluntary explosive force production, but evidence supporting these associations is unclear, and may be primarily due to a common influence of maximum strength on all these variables. It is also unclear whether the capacity for explosive force production will change in proportion to MVF, as a function of muscle length/joint angle and/or type of contraction. Clearly, further work is required to understand the relative contribution of the different neural and mechanical determinants of RFD. Furthermore, the influence of these determinants on explosive performance is likely to be affected by athletic background and/or training. This will be discussed in the following sections.

2.5 The Influence of Athletic Performance

Although explosive force production is thought to be associated with explosive athletic ability, there is limited evidence for this. Two studies have assessed the bivariate relationship between explosive force production of the knee extensors and vertical jump performance (in squat and counter movement jumps; CMJ) in small cohorts (n ≤ 11) of trained volley ball players (de Ruiter et al. 2007) and untrained individuals (de Ruiter et al. 2006). These studies reported linear correlations (0.75<r<0.86) between knee extensor explosive force and vertical jump performance in the untrained individuals (de Ruiter et al. 2006), but not the trained volley ball players (de Ruiter et al. 2007). Whilst this may suggest that RFD is associated with vertical jump performance in untrained, but not trained participants, the conflicting results may also be due to RFD only being measured in the knee extensors. Vertical jumping relies on the simultaneous contribution of several muscle groups (ankle plantar flexors, knee extensors and hip extensors; (Pandy, Zajac 1991)). Consequently, there may have been discrepancies in the jump technique adopted by the different groups of participants in the
aforementioned studies (de Ruiter et al. 2006, de Ruiter et al. 2007), resulting in one population loading the knee extensors to a greater extent.

Explosive athletic activities involve multiple joint actions, and therefore a better understanding of the association between athletic performance and RFD may be gained by measuring explosive force in multiple joint isometric actions (as discussed in section 2.2.2). There appears to be only two studies that have assessed the relationship between explosive athletic performance (specifically CMJ height) and RFD in multiple joint activities (specifically isometric squats (Nuzzo et al. 2008) and mid-thigh clean pulls (Kawamori et al. 2006)), but both reported poor correlations ($r \leq 0.12$) between these parameters. However, these studies did not provide a detailed analysis of the rising force-time curve, analysing RFD at just a single time point. It is therefore possible that the force-time characteristics relevant to jumping performance were not assessed during the isometric explosive efforts. Furthermore, it wasn’t clear how these studies addressed some of the methodological issues discussed in section 2.2.2, such as pre-tension and/or countermovement prior to performing the explosive efforts, or the method used to identify force onset. Future work should control for these factors and investigate the relationship between performance in different explosive athletic activities (such as sprinting and jumping) and multiple joint RFD over different time periods from force onset.

The association between athletic performance and RFD may also be assessed indirectly by comparing explosive power athletes, a group with a demonstrated ability for explosive athletic performance, with other populations. This methodological approach will also allow studies to investigate the neural and mechanical factors underpinning any discrepancies in RFD between different populations. Earlier studies have reported greater absolute leg press RFD in explosive power athletes compared to groups of other athletic/training backgrounds (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989). However, when two of these studies normalised RFD to MVF, to control for strength discrepancies, their results were mixed, with one reporting greater normalised RFD in explosive power athletes (Hakkinen, Keskinen 1989), and the other observing similar results for the groups (Kyrolainen, Komi 1994). These investigations did not address the methodological issues discussed in the previous
paragraph, which may have influenced their results. Therefore, the effect of athletic/training background on RFD remains unclear.

None of the studies investigating differences in RFD between populations of different athletic/training backgrounds assessed the neural and mechanical factors underpinning any differences in RFD. On the other hand, the determinants of RFD have been investigated separately as a function of athletic/training background. There is good evidence that explosive power athletes are stronger than endurance athletes (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989, Arampatzis et al. 2007a, Paasuke, Ereline & Gapeyeva 1999b) and untrained individuals (Arampatzis et al. 2007a, Paasuke, Ereline & Gapeyeva 1999b, Dowson et al. 1998, Lattier et al. 2003, Mcbride et al. 1999), but not individuals that train for purely maximum strength (Hakkinen, Keskinen 1989, Mcbride et al. 1999). There is also evidence that neural activation during MVCs is greater in explosive power athletes than untrained individuals (Lattier et al. 2003, Amiridis et al. 1996, Ahtiainen, Hakkinen 2009), although differences in neural activation during explosive contractions has not been assessed. The effects of athletic/training background on the intrinsic contractile properties of the muscle are equivocal, with some studies reporting differences in twitch characteristics between explosive power athletes and endurance athletes or untrained individuals (Lattier et al. 2003, Paasuke, Ereline & Gapeyeva 1999a), and others not (Garrandes et al. 2007, Carrington, Fisher & White 1999). Likewise, there is also conflicting evidence for the influence of athletic/training background on MTU stiffness; some studies have reported greater MTU stiffness in explosive power athletes than endurance athletes or untrained individuals (Arampatzis et al. 2007a, Rabita, Couturier & Lambertz 2008), whilst another study reported a negative relationship between MTU stiffness and sprint performance (Kubo et al. 2000). Clearly, further work is required to understand the association between athletic performance and the neuromuscular determinants of RFD.
2.6 The Influence of Strength Training

2.6.1 Training for Maximum Strength

Training for maximum strength via sustained (>3 s) high load (>70% MVF or 1 repetition maximum) contractions has proved successful at increasing this component of strength (Aagaard et al. 2002a, Suetta et al. 2004, Andersen et al. 2010, Blazevich et al. 2009, Kubo et al. 2001, Kubo et al. 2007b, Rich, Cafarelli 2000). The long term gains in maximal force production are thought to be primarily due to peripheral adaptations, as shown by the parallel increase in muscle size and strength after the first 2 months of training for maximum strength (Narici et al. 1996). However, many studies have observed increases in maximum strength within the first 2-8 weeks of maximum strength training without equivalent increases in muscle size (Narici et al. 1996, Narici et al. 1989, Adamson et al. 2008, Abe et al. 2000). These early improvements in maximum strength are thought to be primarily due to central adaptations. It is important that the central and peripheral adaptations to maximum strength training are understood in order to inform best practice.

2.6.1.1 Central Adaptations

Specific central adaptations likely to contribute to the observed improvements in maximum strength include increased neural activation of the agonist muscles and/or reduced neural activation of the antagonist muscles during a MVC. However, evidence to support these responses to training remains equivocal. Some studies have reported increased agonist EMG amplitude at MVF over the first 12 weeks of maximum strength training (Del Balso, Cafarelli 2007, Narici et al. 1989, Hakkinen, Komi 1983, Hakkinen et al. 1998, Kubo et al. 2006), whilst other studies have not (Kubo et al. 2001, Narici et al. 1996, Garfinkel, Cafarelli 1992). These studies did not normalise agonist EMG to a M_{max}, so their results may have been confounded by those issues discussed in section 2.3.1.3. Two more recent studies have normalised agonist EMG to M_{max} and reported no change within the first 3-8 weeks of training for maximum strength (Rich, Cafarelli 2000, Pucci, Griffin & Cafarelli 2006), despite considerable improvements in MVF in this time. In contrast one study reported that whilst EMG normalised to M_{max} was unchanged after the first 9 sessions (3 weeks) of training for maximum strength (Cannon et al. 2007), it did significantly increase after a further 9 training sessions (18
sessions or 6 weeks in total). This latter finding suggests that whilst neural adaptations may occur during the early stages of maximum strength training, a sufficient training volume may be required to detect them.

A better understanding of the neural contributions to maximum strength gains may be obtained by assessing the agonist EMG-force relationship (Sale 1988, Moritani, deVries 1979). A rightward shift in this relationship (i.e., greater force for the same level of agonist EMG) suggests that peripheral adaptations are the main cause of improved maximum strength. On the other hand, a consistent relationship despite increased MVF suggests that neural adaptations are the main cause of improved maximum strength. It is no surprise that studies have reported a rightward shift in the absolute EMG-force relationship after the first 2-6 months of training for maximum strength (denoting peripheral adaptations; (Narici et al. 1996, Hakkinen, Komi 1983, Garfinkel, Cafarelli 1992, Moritani, deVries 1979)). However, the effect of training for maximum strength on the EMG-force relationship during the first 2 months of training (when neural adaptations are thought to contribute to maximum strength gains) has not been documented.

The ITT has also provided conflicting evidence of improved agonist activation with maximum strength training. Some studies have reported an increase in agonist maximal voluntary activation (determined via the ITT; (Pucci, Griffin & Cafarelli 2006, Reeves, Narici & Maganaris 2004, Scaglioni et al. 2002, Shima et al. 2002)), whilst others have not (Cannon et al. 2007, Jones, Rutherford 1987, Brown, McCartney & Sale 1990). However, these studies used the ratio of superimposed to control twitch amplitude to determine maximal voluntary activation, which may partly explain their conflicting results. As discussed in section 2.3.1.3, the ratio of superimposed to control twitch method assumes that the superimposed twitch is elicited at MVF, but this is unlikely to occur. Consequently, in those studies that did report increased maximal voluntary activation using the ITT, it is possible that they were actually observing an increased ability to maintain a steady force-time curve during a MVC (Keen, Yue & Enoka 1994), which might result in an increased likelihood of the superimposed twitch being elicited at a force close to MVF. The ratio of superimposed to control twitch amplitude also assumes that the voluntary-superimposed force relationship is linear, but this relationship appears to be curvilinear, with only very small changes in superimposed
twitch amplitude above 80% of MVF (Kooistra, de Ruiter & de Haan 2007). This low sensitivity of the ITT at high force levels may also have contributed to the conflicting results of past studies. The difference between TMF and MVF, where TMF is extrapolated from an appropriate relationship of the voluntary-superimposed force relationship, may provide a more reliable estimation of maximal voluntary force (Folland 2009), but no strength training studies appear to have adopted this method.

Activation of the antagonist muscles during an MVC will reduce net force output of a joint, and will thus negatively influence maximum strength. Consequently, observed reductions in antagonist activation with maximum strength training (Hakkinen et al. 1998, Carolan, Cafarelli 1992), are thought to contribute to maximum strength gains. However, other studies have reported no change (Hakkinen et al. 1998, Pucci, Griffin & Cafarelli 2006) or even an increase (Simoneau et al. 2006, de Boer et al. 2007) in antagonist activation following maximum strength training. These conflicting results may be due to a possible trade-off between reduced antagonist activation to enhance net force output, and increased antagonist activation to counteract any increases in agonist activation that may compromise joint integrity (Cochrane et al. 2006). Therefore, investigators should quantify the level of co-activation to better understand this trade-off and its influence on maximum strength gains with training. A detailed analysis of the agonist-antagonist activation relationship through the force levels may provide a comprehensive analysis of changes in co-activation, but this method has not been employed by previous studies.

2.6.1.2 Peripheral Adaptations

It is widely documented that indices of muscle size (such as anatomical cross sectional area and muscle volume) increase within the first 8-12 weeks of training for maximum strength (Narici et al. 1996, Garfinkel, Cafarelli 1992, Abe et al. 2000, Tracy et al. 1999), although a recent study has detected this change as early as 3 weeks from the start of a training programme (Seynnes, de Boer & Narici 2007). Increased muscle size is thought to be due to the hypertrophy of muscle fibres (increased muscle fibre cross sectional area), facilitating the increase in contractile proteins arranged in parallel (Folland, Williams 2007a). Muscle fibre hypertrophy tends to occur earlier and to a greater extent, in type II than type I muscle fibres (Folland, Williams 2007a, Staron et
Training for maximum strength may also lead to hyperplasia (an increased number of muscle fibres), which has been shown in rats (Gollnick et al. 1981). However, evidence of hyperplasia in humans is largely anecdotal due to the ethical and methodological issues of assessing the number of fibres in human muscle (Folland, Williams 2007a). The packing of contractile proteins in parallel with muscle fibre hypertrophy (and potential hyperplasia) is thought to cause the increase in angle of pennation of pennate muscles (Folland, Williams 2007a) following training for maximum strength (Reeves, Narici & Maganaris 2004, Aagaard et al. 2001, Blazevich et al. 2007). These hypertrophic adaptations to training for maximum strength will result in a greater number of myosin-actin cross-bridges, and therefore a greater force production, at any one time during a muscular contraction. Moreover, given the greater specific tension of type II muscle fibres, the preferential hypertrophy of these fibres may contribute to increases in muscle specific tension also shown to occur when training for maximum strength (Erskine et al. 2011, Erskine et al. 2010b, Erskine et al. 2010a), although evidence to support this theory is equivocal (Erskine et al. 2011).

Another peripheral adaptation that has been observed with maximum strength training is a shift in MHC isoform expression from type IIX (the type most conducive to fast contractions) to type IIA (Baumann et al. 1987, Jurimae et al. 1996, Pette, Staron 1997, Staron et al. 1994). This adaptation typically occurs within the first 2-3 months of training for maximum strength (Folland, Williams 2007a), with some studies reporting subtle shifts in MHC isoform expression as early as 4 weeks from the start of a training programme (Jurimae et al. 1996, Staron et al. 1994). Although the shift in MHC from fast-to-slow isoform expression may be expected to negatively affect the intrinsic contractile properties of the muscle, several studies have reported increases in twitch peak force and RFD following training (Rich, Cafarelli 2000, Del Balso, Cafarelli 2007, Barry, Warman & Carson 2005, Duchateau, Hainaut 1984). However, these studies reported twitch parameters in absolute terms, rather than normalising to maximum strength, which was also seen to improve. Therefore, the observed changes in twitch parameters may have been due to an improved capacity for maximal force production, rather than adaptations in the intrinsic contractile properties of the muscle. On the other hand, other studies have reported no change in twitch parameters despite improved MVF, following maximum strength training (Pucci, Griffin & Cafarelli 2006, Scaglioni
et al. 2002, Lee, Gandevia & Carroll 2009). Therefore, whilst there is a general consensus over the influence of training for maximum strength on MHC isoform expression, its effects on the intrinsic contractile properties of the muscle are unclear.

Increased MTU stiffness has also been observed with training for maximum strength (Kubo et al. 2001, Kubo et al. 2007b, Kubo et al. 2006, Reeves, Narici & Maganaris 2004), although the earliest observation of this adaptation is 9-10 weeks (Seynnes et al. 2009, Kubo et al. 2010). A stiffer response to loading following training for maximum strength is thought to help protect soft tissue from the increased risk of injury, caused by the improved capacity of the MTU for maximal force production (Buchanan, Marsh 2002, Garrett 1990). The mechanisms of increased MTU stiffness are unclear, but are thought to be associated with an increased number and/or density and/or a change in alignment of collagen fibres (the primary constituent of connective tissue) (Buchanan, Marsh 2002).

Counterintuitive to the increase in MTU stiffness, recent studies have reported an increase in fascicle length with maximum strength training (Blazevich et al. 2007, Reeves et al. 2009). This response predominantly occurs within the first 5 weeks of training for maximum strength, with minimal changes after this time (Blazevich et al. 2007). Furthermore, greater increases in fascicle length are likely to be measured when training with eccentric contractions (Reeves et al. 2009). The longer fascicles are thought to be due to an increased number of sarcomeres in series, and cause a rightward shift in the force-length relationship of muscle towards longer lengths (Rassier, MacIntosh & Herzog 1999). Increased fascicle length may also decrease MTU stiffness, due to greater shortening of the contractile component during the initial phase of a contraction. Blazevich et al. (2009) provided dubious evidence of this when they observed an association between a rightward shift in the force-length relationship of muscle and reduced RFD following training for maximum strength. The authors proposed that the shift in the force-length relationship was due to increased fascicle length, whilst the reduced RFD characterised an increase in MTU compliance; however, they did not measure MTU stiffness and thus were unable to validate their hypothesis.
In summary, short and long term maximum strength gains are thought to be primarily due to central and peripheral adaptations, respectively. However, evidence for central adaptations, specifically increased agonist activation and decreased agonist-antagonist co-activation, is equivocal. This may be due to the methods used to assess agonist and antagonist activation.

2.6.2 Training for Explosive Strength

2.6.2.1 Maximum Strength Training

Whilst training for maximum strength is clearly effective at improving this component of strength, evidence of its effects on the capacity for explosive force production are equivocal (Aagaard et al. 2002a, Suetta et al. 2004, Andersen et al. 2010, Blazevich et al. 2009, Kubo et al. 2001, Rich, Cafarelli 2000). These conflicting results may be due to competing influences of different peripheral adaptations. For example, whilst increased MTU stiffness in response to maximum strength training (discussed in section 2.6.1.1) is thought to benefit RFD, Blazevich et al. (2009) reported an association between a rightward shift in the force-length relationship and decreased RFD, following 5 weeks of maximum strength training. There may be another conflict of influence between increased maximal force production and a shift in MHC isoform expression from type IIX to IIA. After 14 weeks of maximum strength training Andersen et al. (2010) reported that RFD recorded over large time windows (>200 ms) increased in proportion to MVF. In contrast, the authors also measured a decrease in RFD normalised to MVF during the initial phase (0-100 ms) of the contraction, and suggested that this was due to an observed shift in MHC from type IIX to IIA. However, this conclusion seems dubious considering that absolute RFD in the initial phase of the contraction was unchanged. It appears more likely that those participants that responded to the training with a greater increase in MVF, are also likely to display a greater decrease in normalised RFD during the initial phase of the contraction (as absolute RFD was unchanged in this period), and potentially a larger shift in MHC isoform expression.

The Andersen et al. (2010) study did not measure agonist activation pre and post training for maximum strength; however, it is possible that the increased RFD over the
first 200 ms, despite unchanged RFD during the first 100 ms was due to neural adaptations specific to the training stimulus. Specifically, agonist activation at high force levels may have increased (and thus benefited RFD over longer periods of force production), but agonist activation during the rising force-time curve may have remained unchanged. Evidence of the effects of training for maximum strength on agonist activation (measured via EMG amplitude and/or rate of rise in EMG) during the initial phase (first 100-200 ms) of explosive contractions is equivocal, with two studies reporting an increase (Aagaard et al. 2002a, Del Balso, Cafarelli 2007) and one study reporting no change (Rich, Cafarelli 2000). Furthermore, Blazevich et al. (2009) separated their participants into those that responded to 5 weeks of training for maximum strength with increased RFD and those that didn’t, and only the former recorded increased agonist activation in the initial phase of explosive contractions. The aforementioned studies however, did not normalise agonist EMG to $M_{\text{max}}$ and therefore their results may be confounded by the issues discussed in section 2.3.1.3. Further work is required to understand the influence of maximum strength training on explosive force production and its determinants.

### 2.6.2.2 Explosive Strength Training

Training with short (~1 second) high RFD contractions, which place a substantial emphasis on producing explosive rather than maximal force (training for explosive strength), may prove more effective at improving this aspect of force production than training for maximum strength. Despite the small loading volume associated with explosive strength training, studies have measured improvements in RFD within just 4 weeks of this type of training (Gruber et al. 2007, Barry, Warman & Carson 2005). This would suggest that important functional improvements can be made without the level of discomfort and fatigue typically associated with maximum strength training. However, the central and peripheral adaptations to explosive strength training, and their effects on RFD, have not been widely investigated.

Early evidence has linked an increase in RFD after 14 weeks of training for explosive strength to increased motor unit firing frequency and incidence of the catch property (doublet discharges) during the initial phase of explosive contractions (Van Cutsem, Duchateau & Hainaut 1998). More recent studies have observed concomitant increases
in initial (0-100 ms) RFD and agonist EMG during explosive contractions, following 4 weeks of training for explosive strength (Gruber et al. 2007, Barry, Warman & Carson 2005); however, these studies did not normalise agonist EMG to $M_{\text{max}}$. Furthermore, the aforementioned (Gruber et al. 2007, Barry, Warman & Carson 2005, Van Cutsem, Duchateau & Hainaut 1998) studies did not provide a comprehensive assessment of the possible peripheral adaptations that may have occurred with the training, making it difficult to assess the contribution of any neural changes to improved explosive force production. This issue may be addressed by comparing changes in voluntary RFD with changes in RFD during supramaximal octet contractions at 300 Hz (which provide a measure of the maximal RFD capacity of the MTU; as discussed in section 2.3.2); however this has not been done in the context of strength training.

Given the small loading volume of training for explosive strength it appears unlikely that it will result in the same peripheral adaptations as those expected from training for maximum strength (such as changes in muscle size, contractile properties, and MTU stiffness). On the other hand, explosive contractions are likely to provide a greater mechanical strain rate than contractions typically performed when training for maximum strength. Due to the viscous component of the MTU, a greater strain rate predisposes the system to greater mechanical stress and strain energy for the same mechanical strain (McElhaney 1966). This may in turn provide sufficient stimulus for peripheral adaptations with training for explosive strength; however, this possibility is yet to be investigated.

2.7 Summary and Main Aims

Explosive force production is thought to be functionally important for explosive sports activities and injury avoidance mechanisms where time to develop force is limited; however, there is limited evidence, from both group comparison and correlation studies, to support this association. Therefore the first main aim of this thesis was to investigate the association between athletic performance and explosive force production by: (i) comparing neuromuscular function in explosive power athletes and untrained individuals; and (ii) measuring the relationship between explosive force production in a multiple joint activity and performance of explosive athletic activities (specifically
sprinting and jumping). It was hypothesised that there would be an association between explosive force production and athletic performance, and that this would be reflected by greater RFD in explosive power athletes, and significant correlations between explosive force production and athletic performance.

Whilst there are likely to be discrepancies in explosive strength between groups of distinct athletic/training backgrounds, the neural and/or mechanical factors underpinning any differences have not been investigated. For example, there is equivocal evidence that maximum strength explains the discrepancies in explosive strength between explosive power athletes and untrained individuals. However, it is unclear whether neural factors and the intrinsic contractile properties of the muscle may also contribute to potential differences in explosive strength between the aforementioned groups. Therefore the second main aim of this thesis was to investigate the contribution of factors such as maximum strength, neural activation, and the intrinsic contractile properties of the muscle to potential discrepancies in explosive strength between explosive power athletes and untrained individuals. It was hypothesised that maximum strength, neural activation and the intrinsic contractile properties of the muscle would all contribute to potential differences in explosive strength between the two groups.

Previous research suggests that short term gains in maximal force production when training for this component of strength are primarily due to central, rather than peripheral, adaptations; specifically, increased agonist activation and/or decreased agonist-antagonist co-activation. However, evidence for these effects is equivocal, which may be due to several methodological issues with measuring agonist activation and agonist-antagonist co-activation (discussed in detail above). Therefore the third main aim of this thesis was to address these methodological issues and investigate the neural adaptations to short term training for maximum strength. It was hypothesised that any short term increases in maximum strength would be due to increased agonist activation and decreased agonist-antagonist co-activation.

Early evidence suggests that training with short high RFD contractions (training for explosive strength) may prove more effective at improving this aspect of force production than training for maximum strength. However, the neuromuscular
adaptations to explosive strength training, and their contribution to any changes in explosive force production, particularly during short-term training when only neural adaptations are thought to occur, are largely unknown. Therefore, the fourth main aim of this thesis was to investigate the neural, mechanical, and architectural adaptations to short term training for explosive strength, and assess the contribution of these parameters to any changes in RFD. It was hypothesised that the training for explosive strength would improve this component of force production as a result of neural adaptations, but that the training would not provide a sufficient stimulus for mechanical and/or architectural adaptations.

It is widely documented that maximal torque production is affected by the type of contraction being performed. However, the influence of the type of contraction on explosive torque production has not been investigated, most likely because of methodological issues associated with measuring RFD in dynamic situations (discussed above). Therefore the fifth main aim of this thesis was to address these methodological issues and investigate the influence of contraction type on the ability to produce explosive torque. Given the association between maximal and explosive torque production it is possible that explosive torque will change in proportion to maximal torque as a function of contraction type. On the other hand, the other neural and mechanical determinants of explosive torque production may be differentially affected by contraction type, thus influencing the ability to utilise the available torque capacity in an explosive situation. Therefore it was hypothesised that the ability to utilise the available torque capacity of the muscle would be influenced by the type of contraction.

Early animal studies have shown that muscle length influences the slope of the force-time curve, relative to maximal force, at high but not low levels of force production. However, the influence of muscle length/joint angle on relative explosive torque production in humans *in vivo* has not been investigated. Therefore, the sixth main aim of this thesis was to investigate the influence of joint angle on explosive force production. It was hypothesised that explosive torque production would be influenced by joint angle, in a similar manner to that observed in animal studies.
Chapter 3: Explosive Athletes vs. Untrained

CHAPTER 3

Neuromuscular Performance of Explosive Power Athletes vs. Untrained Individuals
3.1 Introduction

Explosive muscular contractions are fundamental to sports activities such as sprinting, jumping, and punching, and are important for preventing injury following mechanical perturbation (Aagaard et al. 2002a, Minshull et al. 2007). During explosive contractions the time for the muscles to develop force is often limited to 50-250 ms (Aagaard et al. 2002a, Haff et al. 1997). Consequently, the electromechanical delay (EMD; the time delay between the onset of electrical activity at the muscle and the generation of force (Winter, Brookes 1991)) and the rate of force development (RFD; the slope of the force-time curve) are considered important descriptors of performance in explosive contractions (Newton, Kraemer 1994, Aagaard et al. 2002a, de Ruiter et al. 2004, Hakkinen, Komi & Alen 1985, Bojsen-Moller et al. 2005, Minshull et al. 2007, Gourgoulis et al. 2003). Explosive power athletes have a clear ability for explosive contractions, but their capabilities in relation to untrained individuals have been poorly documented, with few attempts made to investigate the physiological mechanisms for any differences. Such a comparison may enhance our understanding of explosive contractions and provide implications for enhancing athletic performance and reducing injury risk in all populations.

Whilst a shorter EMD would theoretically enhance explosive performance by decreasing the neuromuscular response time to a particular stimulus, it is not known whether EMD is shorter in explosive power athletes. Fibre type distribution and muscle-tendon unit (MTU) stiffness are known to affect EMD (Kubo et al. 2001, Taylor et al. 1997), and previous studies have reported differences in these factors when comparing explosive power athletes to untrained individuals (Kubo et al. 2000, Mero et al. 1981), suggesting that EMD discrepancies may also exist. Measurement of involuntary EMD, from electrically evoked twitch contractions, may reveal if aspects of muscle-tendon morphology and mechanics contribute to any differences in voluntary EMD. During explosive voluntary contractions EMD is ~100% (16-25 ms) longer than during involuntary contractions (Minshull et al. 2007, Zhou et al. 1995, Zhou 1996), indicating that neural activation may also play an important role in EMD. The difference between the EMD of voluntary and involuntary contractions may be an effective method of quantifying the voluntary neurological aspect of the delay.
Explosive power athletes would also be expected to have a greater RFD than other populations. A greater absolute RFD has been found in explosive power athletes when compared to endurance athletes (Kyrolainen, Komi 1994). However, when RFD is normalised to maximal voluntary force (MVF) the difference between explosive power athletes and endurance athletes or untrained individuals is equivocal (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989), perhaps because these studies made only a single observation of RFD at variable time points from force onset. Furthermore these reports did not investigate the specific causes for any differences in RFD.

Muscle morphology and mechanics are expected to affect the intrinsic contractile properties of the muscle, determined via electrically stimulated involuntary contractions (Harridge et al. 1996, Almeida-Silveira et al. 1994, Oda et al. 2007). Assessment of involuntary contractions may therefore help to explain any enhanced RFD of explosive power athletes. However, the relationship between the intrinsic contractile properties of the muscle and voluntary RFD is equivocal (de Ruiter et al. 2004, de Ruiter et al. 2007, Andersen, Aagaard 2006). Alternatively, the level of neural activation during the initial phase of a contraction could also be responsible for differences in RFD. Recent cross-sectional studies have confirmed a relationship ($r^2 = 0.75-0.83$) between agonist muscle activation, assessed with EMG amplitude, and the torque-time integral in the first 40 ms of an explosive contraction (de Ruiter et al. 2004, de Ruiter et al. 2006, de Ruiter et al. 2007). In addition to the level of neural activation, the degree of synchrony in the activation onset of all agonists/synergist muscles contributing to net force production is also likely to affect RFD, but this issue has not been investigated.

The aim of this study was to compare the neuromuscular function, of explosive power athletes and untrained individuals (controls) during voluntary and involuntary explosive isometric contractions of the knee extensors, with particular attention to EMD and the RFD throughout the ascending force-time curve. The comparison of voluntary and involuntary contractions, and the assessment of neural activation with EMG, may help to delineate the importance of neural factors vs. the intrinsic contractile properties of the MTU in explosive muscular performance. It was hypothesised that explosive power athletes would display greater voluntary explosive force production and shorter EMD.
than the controls, and that neural activation and the intrinsic contractile properties of the MTU would contribute to these differences.

### 3.2 Methods

#### 3.2.1 Participants

Nineteen male participants were recruited to form two groups; explosive power athletes (n = 9; age, 21 ± 3 yrs; height, 181 ± 7 cm; and mass 80 ± 8 kg) and controls (n = 10; age, 22 ± 4 yrs; height, 179 ± 5 cm; and mass 81 ± 13 kg). The athletes were sprinters or jumpers competing at a national/international level, and performing regular strength/power training (>3 x per week) for ≥ 2 years. The controls consisted of light to moderately active individuals (≤ 3 x aerobic activity a week), who were not involved in any strength or power training. All the participants were healthy, injury free and provided written informed consent prior to their involvement in this study, which was approved by the Loughborough University ethical advisory committee.

#### 3.2.2 Overview

Participants visited the lab for 60-75 min at a consistent time of day on three occasions, to complete a familiarisation trial and two main trials in which measurements were recorded. Trials were separated by one week and consisted of the same protocol. Participants sat in an isometric strength testing chair (Bojsen-Moller et al. 2005, Parker et al. 1990) and completed a series of isometric voluntary and involuntary contractions of the knee extensors of their dominant leg. Specifically, twitch and tetanic electrically stimulated contractions, explosive voluntary and maximal voluntary contractions (MVC) were performed. In addition, knee flexor isometric MVCs were also completed at the end of each trial. Knee extension/flexion force and surface electromyography (EMG) of the superficial knee extensors and the biceps femoris were recorded throughout these contractions.
3.2.3 Force Measurements

Participants were firmly secured to the strength testing chair with a waist belt and shoulder straps. The hip and knee angle was fixed at 100° and 85°, respectively (180° was full-extension). An ankle strap was placed 2 cm proximal to the medial malleolus and was in series with a calibrated U-shaped aluminium strain gauge (linear response up to 1000 N; (Jones, Parker 1989)). The strain gauge was positioned perpendicular to tibial movement during knee extension/flexion. The force signal was amplified (x500), interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). The force signal was notch filtered at 100 and 200 Hz in both directions with an infinite impulse response digital filter (q-factor of 100), to remove any harmonics of the mains frequency. Real-time biofeedback of the force response was provided on a computer monitor.

3.2.4 Electromyography Measurements

Surface EMG was recorded from the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and the long head of the bicep femoris (BF), using a Delsys Bagnoli-4 EMG system (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), a single differential surface electrode configuration (1 cm inter-sensor distance, model DE-2.1, Delsys, Boston, USA) was attached over the belly of each muscle parallel to the presumed orientation of the muscle fibres, using adhesive interfaces. The reference electrode was placed on the patella of the same limb. The same investigator placed the electrodes in the same relative position on all participants, and marks left on the skin by the electrodes in the first main trial were used to relocate the electrodes in the second main trial. Each EMG signal was amplified (x100, differential amplifier 20-450 Hz), synchronised with the force signal using the same analogue to digital converter and PC software, sampled at 2000 Hz, and band-passed filtered in both directions between 6-500 Hz using a 2nd order Butterworth digital filter.
3.2.5 Electrical Stimulation

The femoral nerve was electrically stimulated with square wave pulses (0.1 ms duration) to elicit single pulse twitch contractions, and facilitate compound muscle action potentials (M-waves). The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom adapted stimulation probe (1 cm diameter, Electro Medical Supplies, Wantage, UK) protruding 2 cm perpendicular from the centre of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve in the femoral triangle. The precise location of the cathode was determined as the position that evoked the greatest twitch response for a particular submaximal electrical current.

One second tetanic contractions (100 Hz) were evoked with a train of electrical impulses (square wave pulses, 0.1 ms duration), delivered via two carbon rubber electrodes (14 x 10 cm; Electro-Medical Supplies, Greenham, UK) taped securely to the anterior surface of the thigh (the cathode 8 cm proximal from the patellar, and the anode 10 cm proximal from the cathode). The electrical impulses for all involuntary contractions were delivered with a constant current, variable voltage stimulator (DS7AH, Digitimer Ltd., UK), and triggered by the CED micro 1401. The stimulator output was also recorded by the analogue to digital converter and PC software.

3.2.6 Protocol

Measurements were completed in the following order.

3.2.6.1 Maximal M-wave and evoked Twitches

A series of twitch contractions at incremental electrical currents were elicited until a simultaneous plateau in M-wave and twitch tension response was observed. Thereafter, the electrical current was increased by 20% and three supramaximal pulses were elicited at 12 second intervals. The average M-wave response (peak-peak amplitude of the EMG signal) to these three supramaximal impulses was defined as the maximal M-wave (M_max) and used for EMG normalisation (see below). Peak tension, time to peak tension, and RFD in the first 50 ms from force onset (change in force divided by 0.05 s) was also averaged across the three supramaximal twitch contractions. Twitch RFD was
measured in absolute terms and relative to peak twitch tension. In each twitch contraction the time difference between M-wave onset and force onset was determined for the superficial knee extensors, and the largest value of the three muscles was defined as involuntary EMD\textsubscript{max} (Zhou et al. 1995, Zhou 1996). M-wave onset was identified manually (see below) and defined as a positive or negative deflection away from the baseline following the initial stimulation artefact. Involuntary EMD\textsubscript{max} was averaged across the three supramaximal twitches.

3.2.6.2 Explosive Contractions

Participants completed a two minute warm-up of the knee extensors of their dominant leg with a series of sub-maximal contractions. They then performed 10 voluntary explosive isometric contractions, separated by a 20-s rest (pilot testing confirmed this recovery time was sufficient). For each contraction participants were instructed to relax, take a deep breath, and following an auditory signal, attempted to extend their knee as ‘fast and hard’ as possible for 1-1.5 s, with an emphasis on ‘fast’. Participants were asked to avoid any countermovement prior to force onset. To provide biofeedback on whether a countermovement had occurred, the resting force level was displayed on a sensitive scale. The slope of the force-time curve (1 ms time constant) was also displayed throughout these contractions and the peak slope from each contraction used as biofeedback.

The three contractions with the largest peak slope and no discernible countermovement or pre tension (change of baseline force of < 0.5 N during the 100 ms prior to contraction onset) were used for analysis. Analysis consisted of measurement of force-time and EMG-time curves in three, 50 ms time windows (0-50, 50-100, and 100-150 ms) after their respective onsets (Figure 3.1). RFD for each time window (change in force divided by 0.05 s) was measured in absolute terms and normalised to MVF (detailed below) i.e., RFD/MVF.
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Figure 3.1. Vastus medialis filtered electromyograph (A) and knee extensor force (B) signals recorded during an explosive isometric contraction. Both signals were analysed in 3 consecutive 50 ms time windows from their respective onsets (0-50, 50-100, and 100-150 ms). Electromechanical delay (EMD) was defined as the time difference between the onset of each signal.

The root mean square (RMS) of the EMG signal during each time window was calculated for each muscle (VM, VL, RF, BF). Antagonist (BF) EMG was normalised to peak BF activation during the knee flexor MVCs (detailed below). EMG amplitude for the agonists (VM, VL, RF) was normalised to $M_{max}$ (i.e. EMG RMS/$M_{max}$). Normalised agonist EMG for each time window was also averaged across the knee extensors to give a mean quadriceps value. The rate of change in normalised EMG ($\Delta$EMG/$\Delta$time) between rest and the first 50 ms time window (denoted by 0-25 ms, with 25 ms representing the midpoint of the first 50 ms window), the first and second time windows (25-75 ms), and the second and third time windows (75-125 ms), was also determined for each knee extensor and averaged across the three muscles. Median Frequency (MF) of each agonist EMG signal was calculated for the first 150 ms from
onset, with a frequency resolution of 6.7 Hz, and was averaged across the three knee extensors. To determine synchrony in the activation onset of the three agonist muscles, the time difference between the EMG onset of the first and last muscle to be activated was calculated for each contraction. This time difference and all RFD and EMG variables were averaged across the three explosive contractions.

In each explosive contraction, the time difference between EMG and force onset (EMD) was determined for each of the superficial knee extensors and the largest EMD of the three muscles (true EMD; Zhou et al. 1995, Zhou et al. 1996)) was defined as EMD_{max}. The three contractions with the shortest EMD_{max} were averaged to give voluntary EMD_{max}, which is also reported as a percentage of involuntary EMD_{max}.

### 3.2.6.3 Maximal Voluntary Contractions (MVCs)

Participants completed four knee extensor isometric MVCs (separated by ≥ 30 s). In response to an auditory signal they were instructed to push as hard as possible for 3 seconds. Biofeedback and verbal encouragement were provided during and between each MVC. Knee extensor MVF was the greatest voluntary force achieved by a participant, in any of the knee extensor MVCs or explosive contractions during that laboratory visit.

### 3.2.6.4 Tetanic Contractions

A series of tetanic contractions at incremental electrical currents were elicited until 50% of MVF was evoked. Thereafter, three tetanic contractions of 50% MVF were evoked (20 s apart) and recorded. Absolute and normalised RFD variables, determined via the same methods as the explosive contractions, were averaged across the three tetanic contractions.

### 3.2.6.5 Knee Flexor MVCs

Following a 2-min warm-up of submaximal contractions participants performed three isometric MVCs of the knee flexors (separated by 30 s), at a knee joint angle of 105°. This knee joint angle was required as participants were not comfortable performing the
knee flexor MVCs at an 85° knee joint angle. The highest RMS of the BF EMG signal was determined using a 100 ms time constant for each MVC, and was averaged to give peak BF activation.

3.2.7 Data Analysis and Statistics

For all voluntary and involuntary contractions identification of force and EMG onsets were made manually (visually). Manual identification of signal onsets is the ‘gold standard’ method (Allison 2003, Hodges, Bui 1996, Moretti et al. 2003, Staude 2001, Van Boxtel et al. 1993, Pain, Hibbs 2007). Whilst it is considered more subjective than the mathematical algorithms (automated methods) typically used in exercise science (e.g., ≥ 2 SD of the baseline), manual identification is more sensitive and accurate (Allison 2003, Hodges, Bui 1996, Staude, Wolf 1999), detecting onsets up to 60 ms earlier than automated methods (Allison 2003, Pain, Hibbs 2007, Pulkovski et al. 2008). This level of accuracy was particularly important in the current study given that the quantification of signal onsets and rapid changes during the initial phase of a contraction (50 ms) was integral to its aims. The same investigator analysed all signal onsets. Initially, signal recordings were viewed with a constant y-axis scale of ~1 N and 10 mV, for force and EMG respectively, and an x-axis scale of 500 ms. These scales provided a good resolution from which the pattern of the noise could be established and the signal onset (last peak/trough before the signal deflected away from baseline noise) interpolated. A vertical cursor was placed on signal onset and the signals were then viewed with a higher resolution (y-axis scale of ~0.5 N and 6 mV, for force and EMG respectively, and an x-axis scale of 25 ms), to verify that the vertical cursor was on the apex of the peak/trough (Figure 3.2). The instant of force onset was confirmed by also displaying the first derivative of the force-time trace. To determine the reliability of manually identifying force and EMG onsets, 19 explosive contractions (one from each participant) were chosen at random and re-analysed a week after the original analysis. From this repeat analysis the typical error of the measurement was calculated for force onset, EMG onset, and voluntary EMD_{max}. 

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Figure 3.2. Typical rectus femoris electromyograph (A) and force (B) signals, prior to the onset of an explosive voluntary isometric contraction of the knee extensors. The graphs are displayed on the scale by which the signal onset was manually detected, and the electromyograph has been converted to pre-amplification values. The inserts are magnifications of the signal within the dashed-line box, and illustrate the scale by which the investigator confirmed that signal onset (dashed line) had been placed on the apex of the last peak/trough, before the signal deflected from the baseline noise.

For each dependent variable between trials coefficient of variation (CV) was calculated (across the two main trials) for each participant and averaged across all participants.
Dependent variables for each participant were averaged across the main two trials before group values (mean ± SD) were generated. For variables measured at three time points (force, RFD, EMG and the rate of change in EMG during the explosive contractions; and RFD during the tetanic contractions) the influence of time and group was analysed with a two-way repeated measures ANOVA (2 groups x 3 repeated measures). A stepwise Bonferroni corrected Paired t-tests was then used to determine differences between groups at specific time points. All other dependent variables (median frequency, MVF and twitch parameters) were assessed using an independent samples t-test. Statistical analysis was completed using SPSS version 14, and the significance level was set at P<0.05.

### 3.3 Results

#### 3.3.1 Electromechanical Delay

Involuntary EMD\(_{\text{max}}\) during the supramaximal twitches was similar for both groups (P = 0.28; Table 3.1). During the explosive contractions there was no consistent order of activation of the three knee extensors, even within an individual. Athletes and controls had similar voluntary EMD\(_{\text{max}}\) whether expressed in absolute terms or as a percentage of involuntary EMD (0.14<P<0.19; Table 3.1). The typical error of measurement for force and EMG (all three knee extensors) onset identification was 0.9 and 0.7-1.3 ms, respectively. The typical error of measurement of voluntary EMD\(_{\text{max}}\) was 1.2 ms. Between trials CV for voluntary and involuntary EMD\(_{\text{max}}\) were 12.6 and 10.0%, respectively.

<table>
<thead>
<tr>
<th></th>
<th>Athletes</th>
<th>Controls</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Involuntary EMD(_{\text{max}}) (ms)</td>
<td>7.3 ± 0.9</td>
<td>6.7 ± 1.1</td>
<td>0.282</td>
</tr>
<tr>
<td>Voluntary EMD(_{\text{max}}) (ms)</td>
<td>13.5 ± 4.0</td>
<td>15.9 ± 3.7</td>
<td>0.188</td>
</tr>
<tr>
<td>Voluntary EMD(_{\text{max}}) (%)</td>
<td>188 ± 63</td>
<td>243 ± 82</td>
<td>0.141</td>
</tr>
</tbody>
</table>
3.3.2 Voluntary Force

The athletes produced a greater MVF than the controls (742.6 ± 73.4 vs. 579.4 ± 131.6 N; P = 0.004). During the explosive contractions the athletes achieved a greater force at 50, 100 and 150 ms (P<0.001; Figure 3.3A), but their absolute RFD was only greater during the 0-50 ms time window (3542 ± 887 vs. 1580 ± 605 N.s⁻¹; P<0.001; Figure 3.4A).

When RFD of the explosive contractions was normalised to MVF, the athletes had a greater normalised RFD in the 0-50 ms time window (4.9 ± 1.5 vs. 2.8 ± 1.2 MVF.s⁻¹; P = 0.004; Figure. 3.4B), and consequently, achieved a greater percentage of MVF after 50 ms (P = 0.004; Figure 3.3B). In contrast, the controls had a greater normalised RFD during the 50-100 ms time window (athletes, 6.7 ± 0.9 vs. controls, 7.9 ± 1.1 MVF.s⁻¹; P = 0.02; Figure 3.4B), and this resulted in no difference in the percentage of MVF achieved after 100 ms (P = 0.31; Figure 3.3B). There was also no difference between the groups in normalised RFD during the 100-150 ms time window. Between trials CV for MVF was 2.3%, and for RFD was 12.8, 5.7, and 12.5% during the 0-50, 50-100, and 100-150 ms time windows, respectively.
Figure 3.3. Absolute (A) and normalised (B) force of explosive power athletes (filled squares; n = 9) and controls (clear circles; n = 10) during explosive isometric contractions of the knee extensors. Data are mean ± SD. ** P<0.01, or *** P<0.001.
3.3.3 Neural Activation during Explosive Contractions

The order of onset of the three agonist muscles was not consistent within or between participants of either group. Nevertheless, the time difference between the EMG onset of the first and last agonist muscle to be activated was shorter in the athletes (7.9 ± 2.4 ms) than the controls (14.4 ± 7.9 ms; P = 0.031), suggesting a greater synchronisation of agonist activation onset in the former.

Figure 3.4. Absolute (A) and normalised (B) rate of force development of explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during explosive isometric contractions of the knee extensors. Data are mean ± SD. * P<0.05, ** P<0.01, or *** P<0.001.
Athletes had greater normalised EMG during the 0-50 ms time window for each of the three knee extensors (P<0.05) and the mean quadriceps (0.26 ± 0.07 vs. 0.15 ± 0.06 \( M_{\text{max}} \); \( P = 0.003 \); Figure 3.5). The mean quadriceps rate of change in EMG was greater in athletes over 0-25 ms (\( P = 0.003 \); Figure 3.6), but was greater in the controls over 25-75 ms (\( P = 0.003 \); Figure 3.6). There was no difference between the groups in normalised antagonist (BF) EMG during any of the 50 ms time windows (collapsed across the three time windows; athletes, 0.09 ± 0.07 vs. controls, 0.17 ± 0.23 peak RMS; \( P>0.33 \)).

**Figure 3.5.** Neural activation of the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and mean of the three muscles (Mean Quadriceps) of explosive power athletes (dark bars; \( n = 9 \)) and controls (light bars; \( n = 10 \)), during explosive isometric contractions of the knee extensors. Neural activation was measured with the root mean square of the EMG signal normalised to maximal M-wave (EMG RMS/\( M_{\text{max}} \)) for each muscle and analysed in three, 50 ms time windows; 0-50 (1), 50-100 (2), 100-150 (3). Data are mean ± SD. * \( P<0.05 \), or ** \( P<0.01 \).
Figure 3.6. The rate of change in normalised EMG for explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during explosive isometric contractions of the knee extensors. The rate of change in EMG was calculated over three time windows (0-25, 25-75, and 75-125 ms) for each muscle (VM, VL and RF) and averaged for the superficial knee extensors. Data are mean ± SD. ** P<0.01.
Athletes had a greater median frequency of the VL EMG signal (P<0.001), but not the RF or VM, during the first 150 ms of the explosive contractions (Figure 3.7). Average median frequency of the three superficial knee extensors (mean quadriceps) was also greater in the athletes (P = 0.02; Figure 3.7). The between trial CV for mean quadriceps EMG data and median frequency was 12.2% and 9.9%, respectively.

![Figure 3.7. Median frequency (MF) of the EMG signal of the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and the mean of these three muscles, for explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during the first 150 ms of explosive isometric contractions of the knee extensors. Data are means ± SD. * P<0.05, or *** P<0.001.](image)

### 3.3.4 Intrinsic Contractile Properties

There was no difference between the groups in peak twitch tension (athletes, 152 ± 22 vs. controls, 131 ± 32 N), time to peak twitch tension (athletes, 90 ± 10 vs. controls, 84 ± 8 ms), absolute RFD (athletes, 2373 ± 285 vs. controls, 2115 ± 572 N.s⁻¹) or relative RFD (athletes, 15.7 ± 0.9 vs. controls, 16.1 ± 1.0 peak tension.s⁻¹) in the first 50 ms of the twitch. Tetanic peak tension was greater in the athletes (388 ± 37 vs. 288 ± 63 N; P<0.001), but was similar once normalised to MVF (athletes, 51.0 ± 2.6 vs. controls, 50.0 ± 3.4% MVF; P = 0.5). Athletes had a greater absolute RFD in the 0-50 and 100-150 ms periods (P<0.01, Figure 3.8A), however, there was no difference between the groups in normalised RFD for any of the time windows (0.27<P<0.50; Figure 3.8B). Between trials CV for twitch and tetanic force variables ranged from 2-7%. 
Figure 3.8. Absolute (A) and normalised (B) rate of force development of explosive power athletes (dark bars; n = 9) and controls (light bars; n = 10) during tetanic contractions of the knee extensors. Data are mean ± SD. ** P<0.01, or *** P<0.001.

3.4 Discussion

The present study compared EMD, RFD, neural activation and the intrinsic contractile properties of the knee extensors in explosive power athletes and controls. There were no differences in EMD between the groups during voluntary explosive contractions; however, normalised RFD was greater in the athletes from 0-50 ms and higher in controls from 50-100 ms (Figure 3.4B). Whilst normalised involuntary RFD was similar for the two groups, the athletes had a greater EMG amplitude in the first 50 ms, and greater synchrony in the activation onset of the agonist muscles. In contrast, the
controls had a greater change in EMG amplitude from the first to the second 50 ms time windows. This is the first study to show that discrepancies in voluntary RFD between explosive power athletes and controls appear to be due to differences in neural activation, and not the intrinsic contractile properties of the muscle.

The EMD values recorded in this study (6-15 ms) were relatively low compared to previous studies (14-90 ms, (Kubo et al. 2001, Minshull et al. 2007, Zhou et al. 1995, Zhou 1996, Muraoka et al. 2004, Yeung, Au & Chow 1999, Vos, Harlaar & van Ingen Schenau 1991)). The long muscle-tendon length, low inertia of the shank, and limited mechanical compliance of our isometric model may explain these short EMD values. Additionally, manual identification of force/EMG onsets has been found more accurate, and detects onsets up to 60 ms earlier than automatic threshold methods (Allison 2003, Pain, Hibbs 2007, Pulkovski et al. 2008).

Electromechanical delay is suggested to be an important factor in the performance of explosive sports activities (Gourgoulis et al. 2003). Explosive power athletes have a clear ability for explosive contractions, but no previous studies have assessed whether they have a shorter EMD than normal individuals. The present study did not observe any differences between the groups for voluntary (absolute and normalised) or involuntary EMD\textsubscript{max} (Table 1). EMD is thought to be determined by multifactorial aspects of muscle-tendon morphology (e.g., fibre types (Taylor et al. 1997)) and mechanics (e.g., MTU slack (Muraoka et al. 2004, Vint, McLean & Harron 2001) and compliance (Kubo et al. 2001)). Type II muscle fibres and MTU compliance may exert opposing effects on EMD, yet both are reported to be greater in the knee extensors of explosive power athletes (Kubo et al. 2000, Mero et al. 1981), and could therefore explain the similar group values we have found. The employment of methods such as ultrasonography (Chen et al. 2009) and muscle biopsies in future studies may help to determine the contribution of morphological and mechanical properties to EMD. Neural activation may also influence voluntary EMD, but similar normalised voluntary EMD\textsubscript{max} suggests that any differences in neural activation between the two groups did not affect the volitional aspect of the delay. Finally, the relatively high CV for voluntary (12.6%) and involuntary EMD\textsubscript{max} (10.0%) appear to be an artefact of short EMD values, and may have reduced the likelihood of finding a small, but distinct difference between the groups.
Explosive power athletes had a greater absolute force at 50, 100, and 150 ms (Figure 3.3A), due to differences in RFD during the 0-50 ms time window (Figure 3.4A). The absolute RFD is known to be related to strength (Andersen, Aagaard 2006), and as athletes in the present study had a greater MVF, differences in absolute RFD could have been entirely due to this discrepancy in strength. However, this was not the case, and normalised RFD was greater in the athletes during the first 50 ms of contraction (Figure 3.4B). Other studies have also reported a greater normalised RFD in explosive power athletes when compared to endurance athletes (Hakkinen, Keskinen 1989) or untrained individuals (Viitasalo, Komi 1978), although, these studies did not investigate the mechanism of any differences. Theoretically the discrepancies in normalised RFD that we observed could have been due to neural activation, and/or the intrinsic muscle-tendon properties.

The magnitude, RFD and time-course of the maximal twitch response were similar for the two groups, and there was no difference between the groups in any of the tetanic RFD variables once they were normalised to MVF (Figure 3.8B). This may seem surprising, but whilst some studies have reported differences in maximal twitch characteristics between explosive power athletes and untrained individuals or endurance athletes (Paasuke, Ereline & Gapeyeva 1999b, Lattier et al. 2003), others have not (Garrandes et al. 2007, Carrington, Fisher & White 1999). We are not aware of any previous studies that have compared the tetanic RFD between explosive power athletes and controls. The similar responses of the groups to evoked twitch and tetanic contractions, suggest that the intrinsic contractile properties of the knee extensors did not explain the observed differences in voluntary normalised RFD.

In contrast, the athletes had a greater mean quadriceps (agonist) EMG amplitude during the 0-50 ms time window (Figure 3.5) and a greater rate of change in EMG from 0-25 ms (Figure 3.6). Therefore, whilst there was no difference in antagonist activation, greater agonist activation appears to account for the higher normalised RFD of athletes. In support of this neurological explanation, a number of cross-sectional studies have strongly correlated ($r^2$ 0.75-0.83) agonist activation, measured by preceding surface EMG, with initial torque output (de Ruiter et al. 2004, de Ruiter et al. 2006, de Ruiter et al. 2007). There is good evidence that explosive ballistic training (presumably similar in
nature to the training performed by the athletes in the present study) increases single motor unit discharge frequency and incidence of double discharges in the early phase of an explosive contraction (Van Cutsem, Duchateau & Hainaut 1998), and could well explain the enhanced neural activation we have found. Other mechanisms of greater neural activation might include increased motor unit recruitment (De Luca 1997) and/or synchronisation (Yao, Fuglevand & Enoka 2000).

In addition to the level of neural activation, the synchrony in activation onset of the agonist muscles may also have influenced initial RFD. The athletes activated all three superficial quadriceps in half the time of the controls, and therefore had more muscles contributing to knee extension force earlier in the contraction. To our knowledge, this is the first study to document this effect, which could be the result of training or innate differences between the groups.

The median frequency of the mean quadriceps EMG signal was greater in the athletes (Figure 3.7) during the first 150 ms of the contraction. This was despite use of a relatively broad frequency resolution (6.7 Hz); that was limited by the need to analyse the power spectrum in a short time window (150 ms). These results could be interpreted as greater recruitment of larger fibres and/or type II fibres in the athletes, as these factors are thought to increase conduction velocity and shift the frequency-power spectrum to higher frequencies (De Luca 1997, Wakeling et al. 2002, Solomonow et al. 1990). However, the frequency power spectrum is also influenced by the location of active motor units (relative to the EMG electrodes), and may be only weak-moderately related to conduction velocity during the ascending limb of the force-time curve (Farina, Fosci & Merletti 2002). Nevertheless, the median frequency results of this study provide additional evidence for contrasting agonist innervation of athletes and controls (Hagg 1992).

Neural activation also appears to explain the surprisingly greater normalised RFD in the controls from 50-100 ms. The athletes achieved a near maximum level of voluntary activation in the first 50 ms, leaving less scope for further increases in agonist activation. In contrast the controls had a greater change in EMG from 25-75 ms (Figure 3.6), and this appears to explain the greater relative change in force output, during the 50-100 ms time window (Figure 3.3B). Therefore, despite the athletes achieving a
greater percentage of their MVF at 50 ms (P<0.01; Figure 3.3B), there was no difference in the percentage of MVF achieved at 100 ms (Figure 3.3B). Past cross sectional studies have only investigated EMG amplitude and RFD over different time phases commencing from EMG/force onset (e.g., 0-30, 0-40, 0-50…0-200 ms; (Aagaard et al. 2002a, de Ruiter et al. 2004, de Ruiter et al. 2006, de Ruiter et al. 2007, Andersen, Aagaard 2006, Bojsen-Moller et al. 2005)). Results of the present study suggest that analysing RFD and EMG over short, consecutive time windows may provide a better understanding of the factors that affect RFD.

The findings of the present study have practical implications for enhancing sports performance and reducing the risk of injury in all populations. The greater force produced by the athletes within 50 ms of contraction onset (absolute RFD, 2.3 fold) was primarily due to their ability to achieve a higher percentage of MVF (1.73 fold) than controls, and this difference appeared to be due to neural activation. Therefore training for tasks requiring force production over this time period (e.g., sprinting or punching), including joint stabilisation for injury prevention, should focus on developing neural activation. On the other hand, the similarity of normalised RFD and neural activation over time periods greater than 100 ms suggests that tasks requiring force production over this time period should focus on developing strength to attain a greater absolute force.

In conclusion, whilst EMD was similar for both groups, the greater synchrony of activation onset and level of activation of the athletes’ agonist muscles during the first 50 ms of explosive voluntary contractions explained their higher normalised RFD. In contrast the controls had a greater increase in activation from 25-75 ms after EMG onset, and higher normalised RFD during the second 50 ms. Coupled with their similar contractile response to involuntary twitch and tetanic contractions, these results suggest that neural activation was responsible for the different pattern of RFD in athletes and controls. Future studies should attempt to establish whether the same discrepancies exist under dynamic conditions.
CHAPTER 4

The Association between Explosive Athletic Performance and Rate of Force Development in Isometric Squats
4.1 Introduction

Chapter 3 provides good evidence of an association between athletic performance and explosive force production (typically defined as the isometric rate of force development; RFD) that had previously only ever been assumed (Newton, Kraemer 1994, Aagaard et al. 2002a, de Ruiter et al. 2004). However, the relationship between RFD and the performance of different explosive athletic activities (e.g., sprinting and jumping) has not been quantified. Furthermore, explosive force production has generally been measured during isolated single joint actions, whilst, functional sports activities such as sprinting and jumping are whole-body multiple joint actions that rely on the simultaneous contribution of several muscle groups to generate force. It is therefore important to determine the association between multiple joint isometric RFD and explosive athletic performance, specifically sprinting and jumping.

Chapter 3 found that explosive force production of the knee extensors was influenced by athletic performance, as a group of explosive power athletes achieved >2 fold greater absolute force, and a higher proportion of their maximal voluntary force (MVF) within the initial phase (first 50 ms) of explosive contractions compared to untrained individuals. Nevertheless, it is unclear whether athletic performance/training influences the force-time curve of isometric multiple joint activities in the same way to that observed in a single joint situation. Earlier studies have observed differences in multiple joint isometric leg press explosive force production between groups of distinct training backgrounds (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989); however, when force was normalised to MVF, to control for strength discrepancies, their results were mixed (Kyrolainen, Komi 1994, Hakkinen, Keskinen 1989). Furthermore, these studies did not appear to address the methodological issues known to influence explosive force measurements (discussed below).

A more direct approach to understanding the relationship between athletic performance and explosive force production is to quantitatively relate these parameters for a range of individuals. Two studies have assessed the relationship between explosive force production of the knee extensors and countermovement jump (CMJ) performance in small cohorts (n ≤ 11), and whilst one reported a linear correlation between these parameters (de Ruiter et al. 2006) the other did not (de Ruiter et al. 2007).
discrepancy in these results may be because there are several muscle groups in addition to the knee extensors that contribute to CMJ performance. Other studies have investigated explosive force production in multiple joint actions, specifically isometric squats (Nuzzo et al. 2008) and mid-thigh clean pulls (Kawamori et al. 2006), but reported a surprisingly poor relationship between CMJ height and measures of RFD in both activities. However, these studies considered explosive force production at only one time point during the rise of the force-time curve, and therefore may not have assessed the relevant force-time characteristics for jumping. To our knowledge, the relationship between sprint performance and explosive strength has not been examined. Clearly, further investigation is required to understand the relationship between athletic performance and explosive isometric force production.

RFD has most often been measured in isolated muscle groups during single joint actions such as knee extensions (Aagaard et al. 2002a, de Ruiter et al. 2004, Andersen et al. 2010), ankle plantarflexions (Gruber et al. 2007, Del Balso, Cafarelli 2007), and elbow flexions (Barry, Warman & Carson 2005). These single joint measurements provide an experimentally controlled situation for investigation of the neuromuscular determinants of explosive force production. As discussed above, a few studies have measured RFD during multiple joint situations (Kyrolainen, Komi 1994, Viitasalo, Komi 1978, Hakkinen, Keskinen 1989, Nuzzo et al. 2008, Kawamori et al. 2006), but none of these investigations have reported the reliability of their measurements. Furthermore, these studies did not appear to consider a number of methodological issues that may have influenced their measurements of explosive force production. Explosive isometric contractions should be performed from a steady baseline force to ensure that the level of pre-tension in the muscle is consistent and no countermovement occurs, as these factors can confound subsequent force measurements (de Ruiter et al. 2006, Grabiner 1994, Kamimura et al. 2009). A steady baseline force will also facilitate accurate identification of explosive force onset (as discussed in Chapter 3). Finally, steps should be taken to minimise joint angle changes caused by soft tissue or measurement system compliance (Bojsen-Moller et al. 2003, Tsaopoulos et al. 2007), which are likely to confound force measurements. Clearly there is a need to address these methodological issues and establish the reliability of measuring explosive force production during isometric multiple joint situations.
The purpose of the current study was to investigate: (i) the between trials reliability of explosive isometric force production at different time points along the force-time curve during a multiple joint activity - specifically a squat; and (ii) the association between athletic performance and absolute and normalised force-time curves during explosive isometric squats (thus progressing the work of Chapter 3). The latter was assessed by comparing explosive force production in two groups with distinct athletic performance/training backgrounds, and by examining the relationship between explosive force production and sprinting and jumping performance. We chose to investigate explosive isometric performance in squats because this exercise is widely used for improving maximal and explosive strength of the lower body, and is kinematically similar to many functional sports activities (Schoenfeld 2010), including sprinting and jumping. It was hypothesised that explosive force production in isometric squats would be associated with sprinting and jumping performance.

4.2 Methods

4.2.1 Participants

Eighteen rugby union players (athletes; age, 20 ± 1 yrs; height, 183 ± 7 cm; and mass, 92 ± 8 kg) and eight untrained individuals (controls; age, 22 ± 1 yrs; height, 186 ± 6 cm; and mass, 83 ± 7 kg) volunteered to participate in this study. The athletes were elite varsity players competing in the English National League 2 level or higher, and were current (2010) champions of the British Universities and Sport Rugby Union trophy. The athletes were involved in regular strength and power training of the lower body (>3 times a week) in addition to regular matches (1 per week) and specific rugby training (≥ 3 times a week). The controls were low to moderately active (≤ 4 x aerobic activity a week), and were not involved in any strength and/or power training. All the participants were healthy, injury free and provided written informed consent prior to their involvement in this study, which was approved by the Loughborough University ethical advisory committee.
4.2.2 Overview

Participants were instructed to refrain from any strenuous physical activity for 36 hrs and from alcohol consumption for 24 hrs, prior to visiting the laboratory. All measurements, which took place between 8:00 and 11:00 am, were preceded by a warm-up of light aerobic exercise and dynamic stretches. The athletes visited the laboratory on a single occasion and completed a series of maximal and explosive lower body activities (taking ~1 hr) including short (20 m) sprints, CMJs, and maximal and explosive isometric squats (>5 min separated each activity). The athletes were familiar with good squat technique and performed sprinting and jumping activities on a regular basis in training. The controls visited the laboratory at a consistent time of day on 3 separate occasions (each separated by a week), and completed a series of maximal and explosive isometric squats during each visit. The first visit was used to familiarise the controls with good squat technique, whilst repeated measures recorded on the second and third visits (trial 1 and 2) were used to assess between trials reliability. The effect of group on isometric squat performance was also investigated by comparing the athletes to the controls (trial 1 measurement).

4.2.3 Measurements

4.2.3.1 Sprints

A photocell timing system (Fusion Sport Smartspeed Queensland, Australia) was used to measure sprint times. Following 3 sub-maximal sprints (at 50, 75 and 90% of maximum perceived effort, separate by 1 min), participants completed 3 maximal sprints (each separated by 3 min) in which they were instructed to run 26 m as quickly as possible. The sprint start was standardised as follows: participants assumed a split stance crouch position (Cronin et al. 2007) with the toes of their preferred leg behind the start line. Once in position participants were instructed to lean back and hold their body weight over their back leg, where they were then given a ‘3-2-1-go’ countdown. The first timing gate was positioned 1 meter from the start line (Figure 4.1), whilst the second and third timing gates were positioned at 5 and 20 m, respectively, from the first timing gate. The finish line was 5 m after the third timing gate to ensure that the participants did not slow down prematurely. The time taken for participants to run between the first and second (5 m) and first and third (20 m) timing gates was measured.
to the nearest millisecond via a hand held wireless module, and the best 5 and 20 m sprint time of the three sprints was recorded. Verbal encouragement and feedback on performance was given throughout.

Figure 4.1. A schematic of the sprint course completed by the athletes. Participants were instructed to run as fast as possible from the start to the finish line. The time taken to travel between the first and second timing gates (5 m) and between the first and third timing gates (20 m) was recorded.

4.2.3.2 Countermovement Jumps

CMJs were completed on a 920 x 920 mm portable force plate (Kistler Quattro Jump, Winterthur, Switzerland) that sampled vertical ground reaction force at 500 Hz. Following 2 practice attempts, participants completed 3 maximal CMJs (each separated by ≥ 30 s). At the start of each CMJ participants were instructed to stand up straight and still in the centre of the force plate with their shoulders pulled back, to ensure that their centre of mass was in a standardised position and at zero velocity. When ready, the participants were instructed to jump as high as possible utilising a countermovement to a self-selected depth and arm swing to optimise their performance. A CMJ was repeated if the participant did not land successfully in the centre of the force plate, or if they felt that their attempt was not maximal. CMJ height (difference between the position of the centre of mass prior to commencing the jump and peak vertical displacement) was determined automatically using the double integration method by the force plate computer software (Quattro Jump, Type 2822A1-1, Version 1.0.9.2). Instantaneous power was determined as the product of force and velocity at any given time point. The greatest CMJ height, peak power, and peak power relative to body mass (i.e., W.kg⁻¹) of the three attempts was recorded.
4.2.3.3 Isometric Squats

Isometric squats were completed on a custom designed, low compliance squat rig (Figure 2), which consisted of a horizontal bar positioned above a force plate (Kistler 92868A, Winterthur, Switzerland). The bar height could be adjusted and fixed in 2.5 cm increments. Ground reaction forces recorded by the force plate were interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK), and low pass filtered at 160 Hz with a 4th order zero lag Butterworth filter. Zero force was defined as the participant’s body weight. A computer monitor placed in front of the squat rig provided the participants with biofeedback. Participants stood (bare foot) on the force plate in a typical back-squat position with the horizontal bar touching their posterior deltoids and middle trapezius. The bar height was set at 75% of the participant’s stature which, when the participant loaded the bar, gave ankle, knee, and hip angles of 83 ± 4, 118 ± 5, and 131 ± 8º, respectively. These joint angles were established by manually digitising (using Silicon Coach Software, Student version 6, New Zealand) sagittal plane photographs taken with a camera positioned 2.5 m from the participant (Figure 4.2). Following a series of sub-maximal isometric squats, the participants completed 3 maximal squats (each separated by ~1 min) where they were instructed to push against the bar as hard as possible for 3-5 s. Throughout these squats (and the explosive squats; see below) the participants were allowed to place their fingers below the horizontal bar, but were instructed not to grip the bar. This ensured that the weight of the arms was not removed from the force plate during the effort. The greatest peak vertical ground reaction force of the 3 maximal squats was defined as maximum voluntary force (MVF), which was reported in absolute terms and relative to body mass by allometrically scaling with an appropriate power value (i.e., N.kg⁻⁰.⁶⁶; (Folland, Mc Cauley & Williams 2008)).
Figure 4.2. A schematic of the frontal view (A) and a photograph of the side view (B) of the isometric squat rig used to assess maximal and explosive leg strength. The participant positioned themselves on the force plate, in a back-squat position, with the horizontal bar touching the posterior deltoids and middle trapezius. The bar height was fixed at 75% of the participant’s stature, which gave ankle, knee and hip angles of 83 ± 4, 118 ± 5, and 131 ± 8°, respectively, when the bar was loaded. A computer monitor in front of the participant provided biofeedback on performance.

The participants were given a 5 min recovery prior to performing the explosive squats. At the start of each explosive squat the participants assumed the squat position (as for the maximal squats) on the force plate and were instructed to apply a light steady baseline force (between 20 and 70 N) to the bar. To ensure that this was the case a computer monitor displayed baseline force in front of the participant, with the 20 - 70 N range highlighted. This baseline force, which subsequent analysis revealed was on average 39 ± 6 N, ensured a good contact between the participant and bar, and removed the initial compliance caused by soft tissue compression. Once the baseline was steady the same investigator gave an auditory signal, upon which the participants were required to push against the bar as ‘fast and hard’ as possible for 1 s. Following one explosive squat the participants were instructed to reproduce a steady baseline force (which typically took 5-10 s) before attempting a second repetition. After 3-4
repetitions the participants were asked to step off the force plate and were given 30-60 s recovery before attempting another set of 3-4 explosive squats. Each participant completed 4-5 sets of explosive squats (>10 repetitions in total). The slope of the force-time curve (determined via a 1 ms epoch) was also displayed on the computer monitor in front of the participants to provide biofeedback on performance, and this was brought to the attention of participants after each explosive squat.

During off-line analysis explosive contractions were discarded if baseline force changed by more than 10 N in the 200 ms prior to explosive force onset, to ensure that the explosive contractions were performed from a steady baseline. Contractions were also discarded if baseline force was not between 1-3% of MVF. This ensured that explosive force onset occurred at a similar point on the normalised force-time curve (normalised to MVF; see below) in all trials and participants. Explosive force onset was defined as the last time that the slope of the force-time curve crossed zero. Of those explosive squats that met the criteria, the three with the greatest peak slope were analysed further. Specifically, force was recorded at 50 ms intervals from explosive force onset up to 250 ms, and normalised to absolute MVF (i.e., expressed as a percentage of MVF). Absolute and normalised force values were averaged across the three explosive squats analysed.

4.2.4 Statistical Analysis

Between trials reliability of the maximal and explosive isometric squat measurements were assessed by calculating the intra-class correlation coefficient (ICC) and the coefficient of variation (CV). An ICC was considered significant at P<0.05. The influence of group (athletes vs. controls) on MVF and explosive squat force (absolute and normalised) at each time point from force onset was analysed with independent t-tests. Pearson’s product moment correlations were used to assess the strength of bivariate relationships between different dependent variables in the athletes. For group effects and bivariate relationships the significance level was set at P<0.05. Statistical analysis was completed using SPSS version 17 and group data are presented as mean ± standard deviation (SD).
4.3 Results

Measurements of explosive and maximal isometric squat strength had good between trials reliability, demonstrated by the significant ICCs (Table 4.1). MVF and explosive force after 100 ms had the highest ICC values (0.96), with explosive force after 50 ms having the lowest ICC (0.74). During the explosive contractions between trials CV was largest at 50 ms from force onset (14.6%), but improved substantially at later time points in the contraction (5.5-7.4%). The lowest CV was for MVF (4.0%; Table 4.1).

Table 4.1. The intra-class correlation coefficients (ICC) and average between trials coefficient of variation (CV) for repeated measures of maximal voluntary force (MVF) and explosive force recorded at 50 ms intervals from force onset during explosive isometric squats, in untrained individuals. A significant ICC is denoted by * (P<0.05), ** (P<0.01), or *** (P<0.001).

<table>
<thead>
<tr>
<th></th>
<th>CV</th>
<th>ICC</th>
</tr>
</thead>
<tbody>
<tr>
<td>MVF</td>
<td>4.0</td>
<td>0.96***</td>
</tr>
<tr>
<td>Explosive force at 50</td>
<td>14.6</td>
<td>0.74*</td>
</tr>
<tr>
<td>Explosive force at 100</td>
<td>7.4</td>
<td>0.96***</td>
</tr>
<tr>
<td>Explosive force at 150</td>
<td>5.3</td>
<td>0.88**</td>
</tr>
<tr>
<td>Explosive force at 200</td>
<td>7.9</td>
<td>0.78*</td>
</tr>
<tr>
<td>Explosive force at 250</td>
<td>6.3</td>
<td>0.88*</td>
</tr>
</tbody>
</table>

The athletes achieved a greater MVF than the controls (athletes, 2934 ± 339 N; controls, 2142 ± 431 N; P<0.001). Furthermore, although the athletes were heavier (athletes, 92.2 ± 8.5 vs. controls, 82.7 ± 6.8 kg; P = 0.01) they also had a greater MVF scaled to body mass (athletes, 149 ± 19 vs. controls, 117 ± 24 N.kg^{0.66}; P = 0.001). Despite similar absolute force during the initial 100 ms of the explosive squats, athletes achieved a greater absolute explosive force at 200 (+29%; P = 0.043; Figure 4.3A), and 250 ms (+28%; P = 0.024; Figure 4.3A), and there was a tendency for group difference at 150 ms (+24; P = 0.086) from force onset. However, there were no differences between athletes and controls in explosive force normalised to MVF at any of the measured time points from force onset (Figure 4.3B).
Figure 4.3. Absolute (A) and normalised (B) force during explosive isometric squats in elite varsity rugby union players (open circles; n = 18) and untrained individuals (black squares; n = 8). Absolute force was vertical ground reaction force after body weight was subtracted, whilst normalised force was expressed as a percentage of maximal voluntary force (MVF). Data are group means ± SD. A group effect is denoted by * (P<0.05).

The mean and range of sprint times (5 and 20 m), and CMJ performance parameters (height, peak power, and peak power relative to body mass) for the athletes are reported in Table 4.2. CMJ height was negatively related to sprint time over 20 m (r = -0.62; P = 0.006; Figure 4.4), but not 5 m (r = -0.19; P = 0.44). CMJ height was correlated with absolute MVF (r = 0.48; P = 0.046), but only tended to be related to MVF scaled to body mass (r = 0.42; P = 0.081). There was no relationship between MVF (absolute or scaled to body mass) and sprint performance (5 or 20 m; -0.04<r<0.25). Bivariate relationships between explosive isometric squat force (absolute and normalised to MVF) at each measured time point and functional explosive performance (sprint and
CMJ) are reported in Table 4.3. Sprint performance (5 and 20 m) was negatively correlated with absolute explosive force at only one time point (100 ms: $r = -0.5; 0.034<P<0.037$). However, 5 m sprint time was negatively correlated with normalised force at 100 ms ($r = -0.63; P = 0.005$; Figure 4.4), and tended to be negatively related to normalised force at 50 ($r = -0.42; P = 0.081$), and 150 ms ($r = -0.46; P = 0.057$). Sprint time over 20 m was negatively correlated with normalised force at 100 ms ($r = -0.54; P = 0.02$), and tended to be negatively correlated with normalised force at 150 ms ($r = -0.44; P = 0.066$). CMJ height was significantly correlated with absolute force at 100 ($r = 0.51; P = 0.014$) 150 ($r = 0.61; P = 0.006$; Figure 4.5), 200 ($r = 0.57; P = 0.02$) and 250 ms ($r = 0.51; P = 0.035$), and tended to be related to normalised force at 100, 150, and 200 ms ($0.33<r<0.43; 0.052<P<0.089$).

**Table 4.2.** Mean ± SD and the range of scores of functional explosive performance parameters recorded in elite varsity rugby union players (n = 18). Performance parameters included 5 and 20 m sprint time; and countermovement jump (CMJ) height, peak power, and peak power per kg of body mass.

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sprint time</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 m (s)</td>
<td>1.003 ± 0.035</td>
<td>0.943 – 1.051</td>
</tr>
<tr>
<td>20 m (s)</td>
<td>2.994 ± 0.073</td>
<td>2.908 – 3.149</td>
</tr>
<tr>
<td><strong>CMJ performance</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>59.7 ± 7.4</td>
<td>44.2 – 75.6</td>
</tr>
<tr>
<td>Peak power (W)</td>
<td>6134 ± 891</td>
<td>4464 – 8241</td>
</tr>
<tr>
<td>Peak power (W.kg⁻¹)</td>
<td>66.6 ± 8.0</td>
<td>49.8 – 84.7</td>
</tr>
</tbody>
</table>
Table 4.3. Correlation coefficients of bivariate relationships between force (absolute and normalised) measured during explosive isometric squats in elite varsity rugby union players (n = 18) and: 5 m sprint time, 20 m sprint time, and counter movement jump (CMJ) height. Explosive force, which was measured at 50 ms intervals from explosive force onset, was normalised to absolute MVF. A significant correlation is denoted by * (P<0.05) or ** (P<0.01).

<table>
<thead>
<tr>
<th>Squat force at 50 ms intervals from onset</th>
<th>Sprint time</th>
<th>CMJ height</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absolute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>-0.32</td>
<td>-0.33</td>
</tr>
<tr>
<td>100</td>
<td>-0.50*</td>
<td>-0.50*</td>
</tr>
<tr>
<td>150</td>
<td>-0.30</td>
<td>-0.38</td>
</tr>
<tr>
<td>200</td>
<td>-0.14</td>
<td>-0.18</td>
</tr>
<tr>
<td>250</td>
<td>-0.03</td>
<td>-0.07</td>
</tr>
<tr>
<td>Normalised</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>-0.42</td>
<td>-0.36</td>
</tr>
<tr>
<td>100</td>
<td>-0.63**</td>
<td>-0.54*</td>
</tr>
<tr>
<td>150</td>
<td>-0.46</td>
<td>-0.44</td>
</tr>
<tr>
<td>200</td>
<td>-0.36</td>
<td>-0.36</td>
</tr>
<tr>
<td>250</td>
<td>-0.16</td>
<td>-0.15</td>
</tr>
</tbody>
</table>
Figure 4.4. The relationship between countermovement jump (CMJ) height and 20 m sprint time in elite varsity rugby union players (n = 18). The correlation coefficient was significant (P = 0.006).

\[ y = -0.006x + 3.36 \]
\[ R^2 = 0.39 \]

Figure 4.5. The relationship between normalised force at 100 ms from force onset during explosive isometric squats and 5-m sprint time, in elite varsity rugby union players (n = 18). Normalised force is vertical ground reaction force after body weight is subtracted expressed as a percentage of maximal voluntary force (after body weight is subtracted; MVF). The correlation coefficient was significant (P = 0.005).
Figure 4.6. The relationship between force at 150 ms from force onset during explosive isometric squats and countermovement jump (CMJ) height, in elite varsity rugby union players (n = 18). Force is vertical ground reaction force once body weight is subtracted. The correlation coefficient was significant (P = 0.006).

To further explore the relationship between sprint performance and explosive force during the isometric squats the athletes were separated into two groups; those that ran 5 m in <1 s (fast; n = 10) and those that ran 5 m in ≥ 1 s (slow; n = 8). These two groups displayed similar body mass (fast, 93.1 ± 9.4 vs. slow, 91.2 ± 7.7 kg; P = 0.66) absolute MVF (fast, 2996 ± 223 vs. slow, 2856 ± 450 N; P = 0.44), and MVF scaled to body mass (fast, 146 ± 26 vs. slow, 151 ± 12 N.kg^{-0.66}; P = 0.75). However, the fast group achieved a greater absolute explosive force at 50 (+62%; P = 0.046; Figure 4.7A) and 100 ms (+49%; P = 0.008; Figure 4.7A) from force onset and tended to achieve a greater absolute force at 150 ms (+27%; P = 0.084). Moreover, normalised force was also 33-67% greater at 50, 100, and 150 ms in the fast group (0.001<P<0.017; Figure 4.7B), with a tendency for the same effect at 200 ms (+20%; P = 0.085).
Chapter 4: Athletic Performance and RFD

4.4 Discussion

Chapter 3 provided good evidence of an association between explosive isometric strength and explosive athletic performance that is supported by the results of the current study. There were clear relationships between explosive strength and both sprint and jump performance, confirming the influence of explosive isometric force production on athletic performance. Furthermore, when the athletes were separated into those that could sprint 5 m in <1 s and those that couldn’t, the faster group displayed...
greater normalised explosive force in the initial phase of the squat (first 50-150 ms). When comparing the athletes and controls in this study, differences in absolute explosive force production appeared to be due to strength discrepancies, as these groups displayed similar normalised force-time curves (normalised to MVF). Finally there was also a good between trials reliability (ICC = 0.78-0.96) of absolute and normalised force measurements recorded during maximal and explosive isometric squats.

The between trials CV of absolute force measured during the isometric squats (MVF, 4%; and explosive force at 50 ms, 15%; 100ms, 7%; and 150 ms, 5%) was comparable to that observed in an isolated muscle group (the knee extensors; MVF, 2.3-3%; and explosive force at 50 ms, 13%; 100ms, 5%; and 150 ms, 5%; Chapter 3 and 5). Chapter 3 and other single joint studies have shown that initial RFD is primarily determined by agonist activation (de Ruiter et al. 2004, de Ruiter et al. 2006). This may partly explain the greater variability in explosive force at 50 ms, as high CV was also observed in agonist activation during explosive contractions in Chapter 5 (12.2%). Nevertheless, the significant ICCs of explosive force production at all time points during isometric squatting indicates a reliable method of assessing the force-time curve in a multiple joint activity, where care was taken to ensure a stable baseline force; a consistent explosive force onset across trials and participants; and minimal system compliance during the explosive efforts.

The athletes produced greater explosive squat force than the controls after the initial 150 ms from explosive force onset. However, this difference appears to be due to the athletes’ being 37% stronger than the controls, as normalised explosive squat force was comparable for the two groups at all measured time points. The influence of MVF on RFD is thought to increase with time from force onset (Chapter 3) (Andersen, Aagaard 2006), which may explain why group differences in absolute force only became evident at later time points during the explosive squats. In contrast to the current results Chapter 3 reported that explosive power athletes have a greater normalised RFD in the initial 50 ms of explosive isometric knee extensions. However, the athletes in Chapter 3 were national/international level sprinters and jumpers, and thus had a demonstrated ability for explosive athletic performance. In contrast, the athletes in the current investigation were rugby union players who might be expected to have varying levels of explosive athletic ability, depending on the physical demands of their position. For example,
forwards spend large periods of the game performing high force, slow speed activities (e.g., scrummaging, rucking and mauling), and less time performing explosive activities, such as sprinting (Roberts et al. 2008).

Whilst sprint times recorded by the athletes in the current study (5 m, 1.00 ± 0.04 s; 20 m, 2.99 ± 0.07 s) were slightly slower than those previously observed in elite sprinters (5 m, 0.97 ± 0.09 s; (Dowson et al. 1998)), they were comparable if not better than those previously measured in Norwegian elite national soccer players (20 m, 3.0 ± 0.3; (Wisloff et al. 2004)), Australian premier rugby league players (20 m, 3.25 ± 0.16 s; (Gabbett, Kelly & Sheppard 2008)), and British international and/or elite national rugby union players (5 m, 1.00 ± 0.06 s; (Dowson et al. 1998)). Furthermore, mean CMJ height in the current study (60 ± 7 cm) was comparable to that measured in Netherlands premier league volley ball players (61 ± 6 cm; (de Ruiter et al. 2007)) and greater than that previously observed in untrained (recreationally active) individuals (52 ± 9 cm; (Harman et al. 1990)). This confirms that the athletes in the current study had comparable explosive athletic ability to team sport athletes competing at a similar level. The observed relationship between CMJ height and 20 m sprint time was consistent with earlier studies that have also reported a commonality in performance in these two activities (Peterson, Alvar & Rhea 2006, Cronin, Hansen 2005). However, it is unclear why CMJ height was not related to 5 m sprint time. It is possible that technical aspects contributing to CMJ performance are more relevant to sprinting over longer distances, although this requires further investigation. Furthermore, sprint time over 20 m is known to be more reliable than over 5 m (Gabbett, Kelly & Sheppard 2008), which would improve the probability of observing an underlying relationship.

When the athletes were separated by 5 m sprint performance, those that ran 5 m in <1 s achieved a greater proportion of their MVF in the initial phase (first 50-150 ms) of explosive isometric squats, than those that ran 5 m in ≥ 1 s. However, normalised explosive force during the later phase of the contraction (200 and 250 ms) was similar for the two groups. These results show that the ability to explosively utilise the available MVF during the early phase, but not during the later phase, of isometric squats was different for fast and slow sprinters, and indicates that normalised explosive strength in this early phase may be an important determinant of sprint performance. This is consistent with the observations in Chapter 3 for an isolated muscle group;
where sprinters and jumpers displayed greater normalised explosive force production in the early, but not the late phase, of explosive isometric knee extensions compared to untrained individuals. The association of normalised explosive strength during squatting with sprint performance was supported by the significant inverse relationships between sprint performance (5 and 20 m) and normalised explosive squat force at 100 ms. The reason for greater initial normalised explosive squat force in faster runners may be due to more effective agonist activation in the short time period available in both of these situations (Chapter 3). In contrast, the similar normalised explosive force at later time points for fast and slow runners is consistent with the finding of no relationship between MVF and sprint performance, and the increased influence of MVF on later phase explosive strength (Chapter 3) (Andersen, Aagaard 2006).

Whilst normalised explosive force during the early phase (100 ms) of the contraction was most strongly related to sprint time (5 m, $r = -0.63$; 20 m $r = -0.54$), absolute explosive force after 100 ms was most strongly related to CMJ height (150 ms, $r = 0.61$). It seems likely that this discrepancy is associated with the time to develop force in these two situations. Foot contact time during the acceleration phase of sprinting is <$300$ ms, and ~$100$ ms at top speed (Weyand et al. 2000). It is unlikely that MVF will be achieved in this time (Aagaard et al. 2002a, Thorstensson et al. 1976), and therefore the proportion of MVF that can be achieved in the time available would be a logical predictor of sprint performance. In contrast, the length of time for force production during a CMJ (i.e., from the start of the countermovement to take-off) ranged from 700-1100 ms in the current study. This time is sufficient to achieve high absolute levels of force and potentially MVF. Hence CMJ height is likely to be more reliant on the overall capacity for absolute force production. This would explain the relationship between CMJ height and explosive absolute force at all measured time points after 50 ms, and between CMJ and MVF.

Although MVF (absolute and scaled to body mass) was related to CMJ height ($0.42 < r < 0.48$), previous studies have reported a stronger association between these variables ($0.78 < r < 0.86$; (Wisloff et al. 2004, Peterson, Alvar & Rhea 2006)). Furthermore, we found no relationship between MVF (absolute or scaled to body mass) and sprint performance, despite earlier studies reporting a commonality between leg strength and sprint performance (Dowson et al. 1998, Wisloff et al. 2004, Peterson,
Alvar & Rhea 2006, Alexander 1989). However, these earlier studies measured leg strength during dynamic contractions (compared to isometric contractions in the current study), and whilst measures of dynamic and isometric strength are considered to be related (Haff et al. 1997, Blazevich, Gill & Newton 2002), dynamic strength is likely to be more specific to functional athletic performance (Baker, Wilson & Carlyon 1994). Furthermore, the participants in the present study were fairly homogenous for age, gender, and athletic/training background. This may have reduced the chance of observing significant relationships between the dependent variables. Two other investigations have also reported a poor relationship between leg strength (absolute or relative to body mass) and both CMJ height and sprint performance in a fairly homogenous group of rugby players (Cronin, Hansen 2005, Baker, and Nance 1999). Clearly further work is required to understand the relationship between strength and athletic performance.

In conclusion, this study presents a reliable method of assessing explosive isometric squat performance over different time periods from force onset in a multiple joint situation. Explosive strength during isometric squatting was associated with explosive athletic performance; specifically normalised explosive force during the early phase (100 ms) of the contraction was most strongly related to sprint time, whereas absolute explosive force after 100 ms was most strongly related to CMJ height. These findings confirm the association found between explosive strength and athletic performance in Chapter 3.
CHAPTER 5

Short-Term Unilateral Resistance Training Affects the Agonist-Antagonist but not the Force-Agonist Activation Relationship
Chapter 5: Training for Maximum Strength

5.1 Introduction

Marked increases in muscle function, specifically maximum strength and rate of force development (RFD), have been found during the early phase (2-4 weeks) of a training with sustained (>3-s) high load (>70%) contractions (‘training for maximum strength’; (Del Balso, Cafarelli 2007, Narici et al. 1989, Abe et al. 2000)). Despite recent evidence of hypertrophy after just 20 days of training for maximum strength (Seynnes, de Boer & Narici 2007), early strength and RFD gains are typically attributed to neural adaptations. Specific mechanisms put forward include increased activation of the agonist muscles and decreased activation of the antagonists; however, evidence for these mechanisms is equivocal. Typically, agonist neural activation has been assessed using either surface electromyography (EMG) or the interpolated twitch technique (ITT; see Shield, Zhou (2004)). Antagonist activation has also been measured with EMG, but its relationship to agonist activation through the range of contraction intensities has not been considered in the context of training.

Studies that have used EMG to assess the effect of training for maximum strength on agonist activation during maximal contractions have reported an increase (Del Balso, Cafarelli 2007, Narici et al. 1989, Hakkinen, Komi 1983, Hakkinen et al. 1998, Kubo et al. 2006, Van Cutsem, Duchateau & Hainaut 1998) while others have not (Kubo et al. 2001, Rich, Cafarelli 2000, Narici et al. 1996, Garfinkel, Cafarelli 1992, Pucci, Griffin & Cafarelli 2006, Cannon et al. 2007, Carolan, Cafarelli 1992). Issues such as electrode relocation and between-session variability in fascia, subcutaneous fat and skin impedance may explain the inconsistent results of past research (Folland, Williams 2007a). Normalizing EMG amplitude to a supramaximal compound muscle action potential (M-wave) may remove some of these confounding factors (Gandevia 2001), but few maximum strength training studies (Rich, Cafarelli 2000, Pucci, Griffin & Cafarelli 2006, Cannon et al. 2007, Pensini, Martin & Maffiuletti 2002, Colson, Martin & Van Hoecke 2009) have employed this technique. The force-agonist EMG relationship through the range of contraction intensities may also provide insight into the link between changes in activation and force production. Previous studies have reported a rightward shift in the force-agonist EMG relationship (i.e. greater force for the same level of activation) following 2-6 months of training for maximum strength (Narici et al. 1996, Hakkinen, Komi 1983, Garfinkel, Cafarelli 1992, Moritani, deVries
1979), suggesting that peripheral adaptations were the primary determinants of long-term maximum strength gains. In contrast, it may be expected that the force-agonist EMG relationship will remain constant, although extended, over a short term (<2 months) resistance training intervention, when the primary adaptations may be neural. However, evidence for this effect is inconsistent (Rabita, Perot & Lensel-Corbeil 2000) and limited.

The ITT has also produced highly equivocal evidence of enhanced agonist activation during maximal voluntary efforts after training for maximum strength. Some studies have reported an increase (Del Balso, Cafarelli 2007, Pucci, Griffin & Cafarelli 2006, Reeves, Narici & Maganaris 2004, Scaglioni et al. 2002, Shima et al. 2002), and others have reported no change (Cannon et al. 2007, Jones, Rutherford 1987, Brown, McCartney & Sale 1990). The aforementioned studies however, defined activation as the ratio of superimposed twitch force to twitch force evoked at rest. This method assumes that the superimposed twitch is elicited at maximum voluntary force (MVF), and that the relationship between superimposed twitch force and voluntary force is linear (Folland, Williams 2007b), which are both unlikely scenarios (Taylor 2009, de Haan, Gerrits & de Ruiter 2009). The difference between MVF and theoretical maximum force (TMF; force at maximal activation) may provide a more valid measure of activation, where TMF has been extrapolated from an appropriate model of the superimposed twitch-voluntary force relationship (Folland, Williams 2007b).

Agonist and antagonist activation contribute simultaneously to net force production, therefore assessing their co-activation (via a statistical analysis of their relationship) will provide greater insight into the neuromuscular adaptations to resistance training. Decreased co-activation would theoretically increase net force production but may compromise joint integrity. These competing demands are likely to influence the nature of the adaptation to any specific training stimulus and may partly explain why previous studies have reported antagonist activation to increase (Simoneau et al. 2006, de Boer et al. 2007), decrease (Hakkinen et al. 1998, Carolan, Cafarelli 1992), or remain unchanged (Hakkinen et al. 1998, Pucci, Griffin & Cafarelli 2006) following training for maximum strength. While these studies compared antagonist and agonist activation qualitatively, they did not quantify co-activation. Consequently, their results provide limited insight into the contribution of antagonist activation to force output before and
after training. The complete agonist-antagonist EMG relationship should provide a comprehensive assessment of co-activation and its association with force changes post-training for maximum strength.

Alongside maximum strength, Chapters 3 and 4 identified RFD as an important descriptor of functional performance in activities where time to develop force is limited. As supported by the results of Chapter 3, RFD over periods >100 ms appears closely related to strength (Andersen, Aagaard 2006), but RFD over shorter periods (≤ 50 ms) appears to be more related to agonist activation (de Ruiter et al. 2004). Short-term training for maximum strength, which is aimed at increasing maximum strength through neural adaptations, might produce differential effects on RFD according to the time period of measurement, but this possibility has not been explored. Investigating this issue may offer further insight into the mechanisms that determine maximum strength and RFD, and have implications for strength training.

The purpose of this study was to investigate the contribution of agonist and antagonist activation to changes in maximum strength and RFD following 4 weeks of unilateral isometric training for maximum strength. To provide a more comprehensive assessment, maximal neural activation of the agonists was assessed with EMG normalized to $M_{max}$, and using the ITT with appropriate extrapolation. Furthermore, the force-agonist EMG and agonist EMG-antagonist EMG relationships were examined. It was hypothesised that any improvements in maximum strength would be due to increased agonist activation and decreased agonist-antagonist co-activation.

5.2 Methods

5.2.1 Participants

Nine male participants (age, 21 ± 1 yrs; height, 1.82 ± 0.05 m; mass, 81 ± 7 kg) completed the study. Participants were recreationally active (moderate exercise ≤ 3 times per week) but were not involved in any form of strength or power training. All the participants were healthy, injury free and provided written informed consent prior to their involvement in this study, which was approved by the Loughborough University ethical advisory committee.
5.2.2 Overview

Participants completed three trials before (one familiarization and two measurement trials; each trial was 2-3 days apart) and two measurement trials (2-3 days apart) after a 4 week unilateral isometric knee extensor strength training program. Measurements were taken from both legs in the 10 days prior to the start of training (pre-training) and 2-6 days after the last training session (post-training). Measurement trials were completed at a consistent time of day and involved a standard 60-90 min protocol. Specifically, knee extension/flexion force and surface EMG of the superficial knee extensors and biceps femoris were recorded in response to electrically stimulated twitch, explosive voluntary, and maximal voluntary isometric contractions. All contractions, including measurements and training, were completed in an isometric strength testing chair (Bojsen-Moller et al. 2005, Parker et al. 1990), with a constant knee and hip angle of 85° and 100°, respectively (180° representing full extension).

5.2.3 Training

Participants completed 4 training sessions per week for 4 weeks. Each session lasted 10-15 min and consisted of 4 sets of 10 unilateral isometric knee extensions using the same apparatus as the measurement trials. In each contraction, participants were instructed to ramp up to 75% of MVF (determined in the measurement trials, and re-determined at the start of the first session of each week; as detailed below) over 1 s, hold it for 3 s and relax. Participants were not given any instruction to push ‘fast’ during the contractions. Two seconds separated each contraction, and 2 min separated each set. Training was completed on one leg (trained leg) chosen at random, and participants were instructed to avoid contracting the contralateral leg (untrained leg) during each effort. Typically, participants struggled to achieve 75% of MVF by the fourth set, in which case they were instructed to extend the knee as hard as possible. Real time biofeedback of the force response, with a target marker at 75% MVF, was provided on a computer monitor, and verbal encouragement was given throughout.
5.2.4 Measurement Trials

5.2.4.1 Measurements

Participants were firmly secured in the strength testing chair with a waist belt and shoulder straps. Force was measured with a calibrated U-shaped aluminium strain gauge (linear response up to 1000 N (Jones, Parker 1989)), which was in series with an ankle strap placed 2 cm proximal to the medial malleolus, and positioned perpendicular to tibial movement during knee extension/flexion. The force signal was amplified (x500), interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), and sampled at 2000 Hz with a PC utilizing Spike 2 software (CED, Cambridge, UK). The force signal was notch filtered at 100 and 200 Hz in both directions with an infinite impulse response digital filter (q-factor of 100), to remove harmonics of the mains frequency.

Surface EMG was recorded from the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and long head of the biceps femoris (BF), using a Delsys Bagnoli-4 EMG system (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), a single differential surface electrode configuration (1 cm inter-sensor distance, model DE-2.1, Delsys, Boston, USA) was attached over each muscle using adhesive interfaces. To normalize electrode placement across participants and trials, the same experienced investigator placed the electrodes over the belly of each muscle, parallel to the presumed orientation of the muscle fibers, at ~50, 55, 90, and 45% of the distance between the greater trochanter and lateral femoral condyle for the RF, VL, VM and BF, respectively. The reference electrode was placed on the patella of the same limb. EMG signals were amplified (x100, differential amplifier 20-450 Hz) and sampled at 2000 Hz with the same analogue to digital converter and PC as the force signal prior to being band-pass filtered in both directions between 6-500 Hz using a 2nd order Butterworth digital filter.

The femoral nerve was electrically stimulated (via a constant current, variable voltage stimulator; DS7AH, Digitimer Ltd., UK) with square wave pulses (0.1 ms duration) to elicit twitch contractions and compound muscle action potentials (M-waves). The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom adapted stimulation
probe (1 cm diameter, Electro Medical Supplies, Wantage, UK) protruding 2 cm perpendicular from the center of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve in the femoral triangle. The precise location of the cathode was determined as the position that evoked the greatest twitch response for a particular submaximal electrical current (typically 30-50 mA).

5.2.4.2 Protocol

The following protocol was first completed on one leg and then repeated on the other. The order in which legs were tested was randomized between participants but remained consistent for each individual. Once in the chair a series of twitch contractions was elicited at incremental current intensities until a simultaneous plateau in force and M-wave response of each muscle was achieved (typically between 100-160 mA). Thereafter, the electrical current was increased by 20%, and three supramaximal twitches were elicited at 12 second intervals. The average peak-peak M-wave response of these three supramaximal impulses was defined as $M_{\text{max}}$ for each muscle. Peak force, peak RFD (peak slope of the force-time curve determined by a 1 ms moving time window), and peak RFD normalized to twitch peak force (RFD/peak force) was also averaged across the three supramaximal twitch contractions.

Participants then completed a warm up of submaximal voluntary contractions, followed by 10 explosive voluntary contractions (each separated by 20 s). In each contraction participants attempted to extend their knee as ‘fast’ and as hard as possible for 1 s from a relaxed state. Explosive voluntary contractions were completed separately from maximal voluntary contractions (MVCs; detailed below) because previous work has highlighted the importance of instruction with regards to performance outcome (Sahaly et al. 2001); whereby explosive performance (RFD) was greatest when participants received the “fast and hard” instruction. Furthermore, due to the technicalities of the explosive contractions, ten efforts were completed to ensure that a valid measure of the participant’s capacity for voluntary RFD was recorded.

During each explosive contraction, participants were instructed to avoid any countermovement (knee flexion prior to knee extension). A computer monitor displayed
both force (on a sensitive scale around resting values) and the slope of the force-time curve. The latter was used to provide immediate biofeedback of performance, specifically peak slope of each contraction, and the former highlighted any countermovement. The three contractions with the largest peak slope and no discernible countermovement or pre tension (change of baseline force of < 0.5 N during the 100 ms prior to contraction onset) were used for analysis, and all RFD and EMG variables were averaged across these three explosive contractions. Analysis consisted of measurement of force- and EMG-time curves in three 50 ms time windows (0-50, 50-100, and 100-150 ms) after their respective onsets. RFD (slope of the force-time curve) was measured for each time window and reported in absolute terms and normalized to MVF (detailed below) i.e., RFD/MVF. The root mean square (RMS) of the EMG signal during each time window was calculated for each muscle. Agonist (RF, VL, and VM) EMG RMS values were normalized to $M_{\text{max}}$ (i.e. EMG RMS/$M_{\text{max}}$) and averaged across the three muscles to give a mean quadriceps value. Antagonist (BF) EMG RMS values were normalized to maximal BF RMS EMG determined during the knee flexor MVCs (detailed below).

Following the explosive contractions participants completed four knee extensor isometric MVCs (separated by ≥ 30 s), in which they were instructed to push as hard as possible for 3 seconds. Biofeedback and verbal encouragement were provided during and between each MVC. Knee extensor MVF was the greatest voluntary force achieved by a participant in any of the knee extensor MVCs or explosive contractions during that laboratory visit. Peak voluntary force in each MVC was typically between 95-100% of MVF. Following the MVCs participants completed one submaximal contraction at 20, 40, 60 and 80% of MVF (separated by 20s), during which they were requested to achieve the required force (represented with a target line on the computer monitor) and hold it steady for up to 5 s.

Neural activation was assessed by measuring the RMS EMG of each muscle during a 200 ms epoch, at MVF (100 ms either side of MVF) and during a stable segment of each submaximal contraction that was not influenced by the interference pattern of a superimposed twitch (detailed below). Agonist and antagonist RMS EMG values were normalized as described for the explosive contractions, and the former were averaged to give a mean quadriceps value. To illustrate the effects of training on the force-agonist
EMG relationship and agonist-antagonist EMG relationship, agonist (mean quadriceps) normalized EMG at the five contraction intensities (~20, 40, 60, 80, and 100% MVF) was plotted against absolute force, and antagonist normalized EMG, respectively, for each participant pre and post training. An appropriate function for these relationships was then generated.

Maximum voluntary neural activation was also determined via the ITT. Supramaximal twitch contractions (two superimposed twitches, separated by ~1 s) were evoked during a stable segment of the second and fourth MVCs and each submaximal contraction. Single twitch contractions were chosen to assess activation rather than a train of impulses, as previous work has reported no difference in activation calculated with single or multiple impulses (Folland, Williams 2007b, Behm, St-Pierre & Perez 1996, Scaglioni, Martin 2009). During extensive pilot work we found neural inhibition (Button, Behm 2008) to be greater when participants were anticipating a larger superimposed stimuli. For each voluntary contraction the average force increment of the two superimposed twitches (ST) was expressed as a percentage of average peak force of two control twitches evoked at rest 1-2 s immediately after that contraction (average coefficient of variation of peak force for the two control twitches was 1.8%). The relationship between voluntary force (VF, as a percentage of MVF) and normalized ST was plotted (Figure 5.1) using the data from all participants, measurement trials (two pre and two post) and conditions (trained and untrained legs). To determine a model that best described this relationship, criteria were set whereby the ST at VF = 0% was 100% and VF at ST = 0% was ≥ 100%. Furthermore, the model was required to meet the same criteria when fitted separately to the data from each of the four conditions (pre trained, post trained, pre untrained, post untrained). We found the best fit (of various linear and curvilinear models) was generated by a model previously proposed by Scaglioni, Martin (2009) and Scaglioni et al. (2002) which contains both linear and exponential components (Equation 5.1).

$$VF = a \left[ (1 - b)e^{cST} + b\left(1 - \frac{ST}{d}\right) \right]$$

(5.1)
Chapter 5: Training for Maximum Strength

The coefficient ‘a’ corresponds to the value of VF when ST = 0% and represents TMF as a percentage of MVF; ‘b’ weights the linear and exponential components; ‘c’ is the constant of the exponential portion; and ‘d’ corresponds to the value of ST when VF = 0%.

When applied to the data for all participants and conditions, this model generated a function for the ST-VF relationship (Equation 5.2) that had an adjusted $R^2$ value of 0.97:

$$VF = 101.5 \left[ (1 - 0.44)e^{-0.036ST} + 0.44 \left( 1 - \frac{ST}{100} \right) \right]$$  

Equation 5.2

The model (Equation 5.1) was fitted to the individual data of each leg pre and post training. The TMF (x-axis intercept) generated by each function was converted to absolute force, and the percentage of TMF achieved at MVF was defined as the level of neural activation.

To complete the trial, participants performed three isometric MVCs of the knee flexors (separated by 30 s), at a knee joint angle of 105°. This knee joint angle was required, as participants were not comfortable performing the knee flexor MVCs at an 85° knee

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**Figure 5.1.** The relationship between the voluntary force (VF; expressed as a percentage of maximum VF) and superimposed twitch force (ST; expressed as a percentage of control twitch peak force). This relationship is represented with a model first proposed by Scaglioni et al. (2002) that contains both a linear and exponential component.
joint angle. RMS EMG during a 200 ms epoch at the greatest knee flexor force (100 ms either side) was measured to give maximal BF RMS EMG.

5.2.5 Data Analysis and Statistics

For the explosive voluntary and involuntary contractions identification of force and EMG onsets were made manually using the same methods discussed in Chapter 3. Briefly; the same investigator identified signal onsets with a constant y-axis scale of ~1 N and 10 mV, for force and EMG respectively, and an x-axis scale of 500 ms. Manual identification is considered the ‘gold standard’ method for detecting signal onsets (Allison 2003, Moretti et al. 2003, Pain, Hibbs 2007, Pulkovski et al. 2008).

To assess the reliability of each dependent variable values recorded in the first and second pre-training measurement trial were compared with paired t-tests. The coefficient of variation of each dependent variable (averaged across the trained and untrained legs) between the two pre-training measurement trials was also recorded.

Before group values (mean ± SD) for the trained and untrained legs were generated, dependent variables for each participant were averaged across the two pre-training measurement trials and across the two post-training measurement trials. For each dependent variable the influence of time (pre vs. post) and leg (trained vs. untrained) was analyzed with a two way repeated measures ANOVA. Paired t-tests were then used to determine within-leg differences between pre and post training measures, and a stepwise Bonferroni correction procedure was employed for all time series data. Statistical analysis was completed using SPSS version 16, and the significance level was set at P<0.05. Dependent variables included: MVF; normalized agonist and antagonist EMG activation at MVF, the gradient and coefficients of the agonist EMG-force relationship and agonist-antagonist EMG relationship; ITT agonist activation and TMF; explosive voluntary RFD (absolute and normalized); mean quadriceps EMG activation 0-50, 50-100, and 100-150 ms from onset; twitch peak force and twitch peak RFD (absolute and normalized).
5.3 Results

5.3.1 Reliability

There was no difference in any of the dependent variables between the two pre-training measurement trials (paired t-tests, P>0.05). Therefore, changes between pre and post-training measurement trials were considered to be an effect of training. Coefficients of variation of key dependent variables between the two pre-training measurement trials are presented in Table 5.1.

Table 5.1. Coefficient of variation (CV) of key dependent variables between the two pre-training measurement trials. CV for submaximal agonist and antagonist normalised EMG is a mean of the CVs recorded at 20, 40, 60 and 80% of maximal voluntary force.

<table>
<thead>
<tr>
<th>Dependent variable</th>
<th>CV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal voluntary force (MVF)</td>
<td>3.0</td>
</tr>
<tr>
<td>Agonist normalised EMG at MVF</td>
<td>10.9</td>
</tr>
<tr>
<td>Submaximal agonist normalised EMG</td>
<td>12.7</td>
</tr>
<tr>
<td>Antagonist normalised EMG at MVF</td>
<td>19.7</td>
</tr>
<tr>
<td>Submaximal antagonist normalised EMG</td>
<td>29.7</td>
</tr>
<tr>
<td>Explosive force at 50 ms</td>
<td>12.8</td>
</tr>
<tr>
<td>Explosive force at 100 ms</td>
<td>5.3</td>
</tr>
<tr>
<td>Explosive force at 150 ms</td>
<td>4.5</td>
</tr>
<tr>
<td>Twitch peak force</td>
<td>6.0</td>
</tr>
<tr>
<td>Twitch peak RFD</td>
<td>10.6</td>
</tr>
</tbody>
</table>
5.3.2 Maximal Voluntary Contractions

MVF increased in both the trained (+20%, paired t-test, \( P = 0.001 \)) and untrained (+8%, paired t-test, \( P = 0.007 \)) legs. This increase was greater in the trained legs, resulting in a time by leg interaction effect (ANOVA, \( P = 0.002 \); Figure 5.2).

![Figure 5.2](image_url)

**Figure 5.2.** Maximal voluntary force (MVF) during isometric knee extensions of the trained (filled squares) and untrained leg (clear circles) pre and post resistance training. ** denotes a time by leg interaction effect (\( P<0.01 \)). Values are mean ± SD (n = 9).

There was a 26% increase of agonist normalized EMG at MVF in the trained leg (pre, \( 0.094 ± 0.021 \) vs. post, \( 0.119 ± 0.022 \) RMS/M\(_{\text{max}}\); Paired t-test, \( P = 0.046 \)), and this increase was similar for sub-maximal contractions at 20, 40, 60 and 80% of MVF (+26-31%; all \( P<0.02 \); Figure 5.3A). While agonist normalized EMG in the untrained leg was unchanged at MVF (pre, \( 0.102 ± 0.011 \) vs. post, \( 0.107 ± 0.025 \) RMS/M\(_{\text{max}}\); paired t-test, \( P = 0.53 \)), and at 40-80% of MVF, it did increase at 20% of MVF (+15.7%; \( P = 0.023 \); Figure 5.3B) following training. The position of the force-agonist EMG relationship, specifically the quadratic coefficients of the relationship, were unchanged post-training for both the trained (paired t-tests, \( 0.44<P<0.80 \); Figure 5.3A) and untrained (paired t-tests, \( 0.65<P<0.98 \); Figure 5.3B) legs.
Figure 5.3. The relationship between force and agonist (3 superficial quadriceps) normalized EMG during isometric knee extensions at 20, 40, 60, 80 and 100% MVF, performed pre (squares, solid line) and post (circles, dotted line) resistance training, by the trained (A) and untrained (B) leg. Relationships are represented as quadratic functions (pre-trained, $y = 1 \times 10^{-7}x^2 + 8 \times 10^{-5}x + 0.0007$; post-trained $y = 1 \times 10^{-7}x^2 + 8 \times 10^{-5}x + 0.0009$; pre-untrained, $y = 1 \times 10^{-7}x^2 + 0.0001x - 0.0026$; post-untrained, $y = 9 \times 10^{-8}x^2 + 0.0001x - 0.0051$). Values are mean ± SD (n = 9). Agonist normalized EMG of the trained leg increased at all force levels (P<0.05).

In contrast, there was no change in maximum neural activation determined via ITT in either the trained (pre, 98.6 ± 0.8 vs. post, 98.7 ± 0.9%; paired t-test, P = 0.64) or untrained (pre, 98.5 ± 0.8 vs. post, 98.5 ± 0.8%; paired t-test, P = 0.97) legs. Consequently, changes in TMF were proportional to changes in MVF in both the trained (+20%; pre, 599.2 ± 84.8 vs. post, 718.2 ± 118.2 N; paired t-test, P = 0.001) and untrained (+8%; pre, 592.3 ± 52.0 vs. post, 637.8 ± 63.9 N; paired t-test, P = 0.006) legs.
There was a tendency for the absolute level of antagonist normalized EMG at MVF to increase by 8% in the trained leg (pre, 0.115 ± 0.047 vs. post, 0.124 ± 0.044 RMS\textsubscript{max}; Paired t-test, P = 0.053), but no change occurred in the untrained leg (pre, 0.125 ± 0.076 vs. post, 0.107 ± 0.048 RMS\textsubscript{max}; paired t-test, P = 0.42). There was a downward shift in the position of the antagonist-agonist EMG relationship, with a decrease in the y-intercept (Figure 5.4) in both the trained (-51.7%; paired t-test, P = 0.014; Table 5.2) and untrained leg (-48.6%; paired t-test, P = 0.097; Table 5.2), but no change in the gradient of the relationship of either leg (paired t-tests, 0.34<P<0.59). This indicates reduced co-activation for a given level of agonist activation.

**Table 5.2.** Linear regression parameters for the relationship between agonist and antagonist normalised EMG during isometric knee extensions at different levels of force pre and post strength training, in the trained and untrained leg. Values are mean ± SD (n = 9).

<table>
<thead>
<tr>
<th>Leg</th>
<th>Linear Parameter</th>
<th>Pre</th>
<th>Post</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained</td>
<td>gradient</td>
<td>0.97 ± 0.41</td>
<td>1.04 ± 0.43</td>
<td>0.59</td>
</tr>
<tr>
<td></td>
<td>Y-intercept (RMS/RMS\textsubscript{max})</td>
<td>0.02 ± 0.01</td>
<td>0.01 ± 0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Untrained</td>
<td>gradient</td>
<td>0.95 ± 0.66</td>
<td>0.77 ± 0.33</td>
<td>0.34</td>
</tr>
<tr>
<td></td>
<td>Y-intercept (RMS/RMS\textsubscript{max})</td>
<td>0.03 ± 0.02</td>
<td>0.02 ± 0.01</td>
<td>0.10</td>
</tr>
</tbody>
</table>
5.3.3 Explosive voluntary contractions

There was no change in the absolute force achieved at 50, 100, or 150 ms during explosive isometric knee extensions, in either the trained (Figure 5.5A) or untrained leg (Figure 5.5C). Accordingly, there was no change in absolute RFD during any of the three 50 ms time windows. When force was normalized to MVF, there was a decrease in the percentage of MVF achieved at 100 and 150 ms by both the trained (100 ms, -15%; 150 ms, -12%; paired t-tests; P<0.01; Figure 5.5B), and untrained legs (100 ms, -
8%; 150 ms, -6%; paired t-tests; P<0.01; Figure 5.5D) after the intervention. This appears to be due to a decrease in normalized RFD during the 50-100 ms time window in the trained leg (pre, 7.3 ± 1.3 vs. post, 6.0 ± 1.3 MVF.s⁻¹; paired t-test, P = 0.028), and a tendency for the same effect in the untrained leg (pre, 7.1 ± 0.69 vs. post, 6.1 ± 1.0 MVF.s⁻¹; paired t-test, P = 0.074). There was also no training effect on agonist or antagonist normalized EMG during the initial 150 ms of the explosive contractions of either leg.

**Figure 5.5.** Absolute (A and C) and normalized (B and D) force (normalized to maximal voluntary force; MVF) during explosive isometric knee extensions, pre (filled squares) and post (clear circles) strength training, in the trained (A and B) and untrained leg (C and D). Data are mean ± SD of all participants (n = 9). ** denotes a difference between pre and post training values (P<0.01).
5.3.4 Twitch Contractions

Pairwise comparisons revealed no significant change in twitch peak force or absolute peak RFD in either the trained or untrained leg after the intervention (Table 5.3). However, normalized peak RFD (to twitch peak force) decreased in the trained leg by 7%, but not the untrained leg (Table 5.3). There was no change in M-wave amplitude of the RF, VL or VM, in the trained or untrained leg following training (Table 5.3).

Table 5.3. Maximal evoked twitch contractions of the knee extensors, pre and post strength training in the trained and untrained leg. Values are mean ± SD of all participants (n = 9).

<table>
<thead>
<tr>
<th></th>
<th>Trained</th>
<th>Untrained</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Peak force (N)</td>
<td>141 ± 39</td>
<td>144 ± 33</td>
</tr>
<tr>
<td>Peak RFD (N.s⁻¹)</td>
<td>3977 ± 970</td>
<td>3753 ± 649</td>
</tr>
<tr>
<td>Normalised peak RFD</td>
<td>28.8 ± 5.1</td>
<td>26.7 ± 4.7</td>
</tr>
<tr>
<td>(Peak force.s⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rectus Femoris M_max (mV)</td>
<td>1.4 ± 0.7</td>
<td>1.4 ± 0.4</td>
</tr>
<tr>
<td>Vastus Lateralis M_max (mV)</td>
<td>2.6 ± 0.8</td>
<td>2.4 ± 0.6</td>
</tr>
<tr>
<td>Vastus Medialis M_max (mV)</td>
<td>8.2 ± 3.1</td>
<td>7.1 ± 3.0</td>
</tr>
</tbody>
</table>

5.4 Discussion

This study investigated the effects of a four week unilateral isometric maximum strength training intervention on the maximum strength, RFD, agonist and antagonist activation of the trained and untrained limbs. Maximum strength gains in the trained leg (20%) were similar to increases in agonist normalized EMG at MVF (26%). The position of the force-agonist EMG relationship and the magnitude of the resting twitch remained unchanged, indicating negligible muscular adaptation. Despite an increase in antagonist normalized EMG at MVF in the trained leg, which appears to be a consequence of greater joint loading, co-activation was lower for any given level of agonist activation (i.e. a downward shift of the agonist-antagonist activation relationship occurred). In contrast to this accumulated evidence for neural changes,
agonist activation as determined by the ITT was unaffected by training. Maximum strength gains of 8% in the untrained leg may be due to a downward shift of the agonist-antagonist EMG relationship, and there was no evidence of increased agonist activation (normalized EMG or ITT). Training for maximum strength was not effective at improving explosive strength (i.e., RFD) in either leg.

Maximum strength changes in the trained leg, +20% in 16 training sessions, were comparable to other studies that have used a similar training intervention (Kubo et al. 2001, Del Balso, Cafarelli 2007, Pucci, Griffin & Cafarelli 2006, Carolan, Cafarelli 1992). It appears that maximum strength gains in our study were largely due to enhanced agonist activation as shown by the increase in agonist EMG normalized to \( M_{\text{max}} \), and the consistent position of the force-agonist EMG relationship (i.e., no change in force for the same neural drive). On the contrary, activation determined via ITT was unaffected by training. Previous studies have reported no change (Cannon et al. 2007, Jones, Rutherford 1987, Brown, McCartney & Sale 1990) or typically modest increases (\( \leq 5\% \)) in activation with ITT following resistance training (Del Balso, Cafarelli 2007, Pucci, Griffin & Cafarelli 2006, Reeves, Narici & Maganaris 2004, Scaglioni et al. 2002, Shima et al. 2002). These investigations however, have used the ratio of superimposed twitch force to control twitch force, irrespective of MVF, so their results do not necessarily reflect the maximal level of voluntary activation, which is only achieved at MVF. Care was taken in this study to define an appropriate ST-VF relationship (Folland, Williams 2007b) for each individual (de Haan, Gerrits & de Ruiter 2009) in order to extrapolate up to TMF. Furthermore, conditions of the study included a low compliance strength chair, prior potentiation of the superimposed twitch (Folland, Williams 2007b) and nerve stimulation (Scaglioni, Martin 2009). Nevertheless, the validity of quantifying neural activation with the ITT remains controversial (Taylor 2009, de Haan, Gerrits & de Ruiter 2009). There are suggestions that the ITT is insensitive at high voluntary forces (Herbert, Gandevia 1999) and that the ST-VF relationship may become increasingly asymptotic and potentially confound extrapolation up to TMF (Kooistra, de Ruiter & de Haan 2007). Considering the similar increases in force and agonist normalized EMG and a consistent force-agonist EMG relationship, it seems likely that the ITT was insensitive to changes in agonist activation after strength training.
Evidence for changes in maximum agonist EMG after training for maximum strength is controversial (Kubo et al. 2001, Narici et al. 1996, Narici et al. 1989, Hakkinen, Komi 1983, Hakkinen et al. 1998, Kubo et al. 2006, Garfinkel, Cafarelli 1992, Carolan, Cafarelli 1992). However, none of these earlier studies involved normalization to M\text{max}, so their results may have been confounded by EMG reliability issues such as relocating electrodes and variable impedance of fascia, subcutaneous fat and skin (Folland, Williams 2007a). Two more recent studies of short duration (9 training sessions), that did normalize agonist EMG to M\text{max}, reported no change in neural drive following training (Pucci, Griffin & Cafarelli 2006, Cannon et al. 2007). However, after 16 training sessions in this study and 18 training sessions in a previous report (Cannon et al. 2007) agonist normalized EMG did increase. Therefore, while early strength gains to resistance training appear to be primarily due to enhanced agonist activation, a sufficient training volume may be required to detect this adaptation. It is important to note that while there may be limitations to the information that surface EMG can provide due to signal cancelation (Keenan et al. 2005), the results of this study support the use and sensitivity of EMG normalized to M\text{max} as a global measure of neural activation.

To fully understand the co-ordinated changes in agonist and antagonist activation after training maximum strength and their combined influence on force production, the complete agonist-antagonist EMG relationship was assessed. There was a downward shift in the entire agonist-antagonist EMG relationship, representing reduced co-activation post training for any given level of agonist activation. Despite this shift in the agonist-antagonist EMG relationship with training, antagonist-normalized EMG at MVF increased in the trained leg. Previous research has reported inconsistent effects of resistance training on antagonist activation (Pucci, Griffin & Cafarelli 2006, Carolan, Cafarelli 1992, Simoneau et al. 2006, de Boer et al. 2007, Hakkinen et al. 1998), but these studies did not quantify co-activation. It is thus unclear how their observed changes in antagonist activation influenced force production. The increase in antagonist activation in this study appears to be a consequence of increased agonist activation and force and is likely to be a protective mechanism required to maintain joint integrity (Cochrane et al. 2006). Nevertheless, the shift in the agonist-antagonist EMG relationship indicates a less than proportionate increase in antagonist activation at MVF after training that, if replicated throughout all of the knee flexor muscles, may have
contributed to the observed gains in strength. The high coefficient of variation of antagonist normalized EMG (20-30%) was not unexpected (Krishnan, Williams 2009). This variation is an artefact of the low recorded antagonist normalized EMG values (~0.12 of RMS$_{\text{max}}$ at MVF), and reflects only a 2-3% change in the level of antagonist activation. Nevertheless, pre and post training measurements were averaged across two trials to improve their reliability.

Changes in maximum strength were similar, but of a smaller magnitude, in the untrained leg compared to the trained leg. Previous studies have also observed enhanced maximum strength in the untrained limb following training maximum strength (Adamson et al. 2008, Lee, Gandevia & Carroll 2009, Shima et al. 2002, Carolan, Cafarelli 1992, Fimland et al. 2009). Although there was no change in agonist or antagonist normalized EMG at MVF of the knee extensors, there was a tendency for the agonist-antagonist EMG relationship of the untrained leg to shift downwards (i.e. lower co-activation). It seems likely that neural adaptations account for the maximum strength gains in the untrained limb, but they are too subtle to provide significant changes and may only become evident when assessing the entire agonist-antagonist EMG relationship.

Despite increased maximum strength there was no change in RFD during the explosive voluntary contractions in either the trained or untrained legs. Consequently, normalized RFD decreased during the 50-100 ms time window in both legs following resistance training. This is surprising given that maximum strength appears to have a strong influence on RFD, particularly during the later phases of the contraction (Chapters 3 and 4, and Andersen, Aagaard (2006)). However, maximum strength gains in this study appear to be due to neural adaptations specific to maximum force production that did not transfer to the rising force-time curve. Furthermore, there did not appear to be peripheral adaptations (as discussed below) that may have otherwise benefited RFD. There is preliminary evidence that including an explosive component into the strength training contractions (i.e., push ‘fast and hard’), will provide the necessary stimuli for improvements in this aspect of force production (Gruber et al. 2007, Del Balso, Cafarelli 2007, Barry, Warman & Carson 2005, Van Cutsem, Duchateau & Hainaut 1998); however, the neuromuscular adaptations to this type of training are not fully understood. It is also unclear how training for explosive strength will influence
maximum strength. Further research should investigate the effects of training for maximum vs. explosive strength on neuromuscular performance.

Although neural adaptations appear to explain the majority of changes observed in this study, the surprising decrease in normalized twitch RFD in the trained leg suggests that there were possible adaptations to the muscle’s contractile properties. The time course of the twitch response, and therefore normalized twitch RFD, is expected to be influenced by cross bridge cycling rate (Andersen, Aagaard 2006), which is greatest in muscle fibers that express type IIX myosin heavy chain isoforms (Larsson, Moss 1993). A training-induced transition of MHC expression from type IIX to IIA (fast-to-slow) is well documented (Baumann et al. 1987, Pette, Staron 1997) and has been reported after just four weeks of resistance training (Jurimae et al. 1996, Staron et al. 1994). If this effect occurred in the current study it could have contributed to the drop in evoked and voluntary normalized RFD, although this possibility cannot be confirmed without the collection of tissue samples. It is important to note that this training intervention may also have induced hypertrophy, as this adaptation has been observed previously over a similar time period (Seynnes, de Boer & Narici 2007). However, while cross-sectional area of the quadriceps muscles was not recorded in this study, the consistent force-agonist EMG relationship and twitch amplitude of the trained leg suggests that peripheral adaptations were minimal.

In conclusion, for the trained leg, maximum strength gains appear to be due to greater agonist activation during the maximum voluntary effort with no change in the position of the force-agonist activation relationship. The increase in force and agonist activation appears to have caused an increase in antagonist activation at MVF; however, there was an overall decrease in co-activation for any given level of agonist activation and force post training. The ITT was insensitive to changes in agonist activation. Finally, the current short-term maximum strength training programme was not effective at improving explosive strength due to neural adaptations that were specific to the peak of the force-time curve.
CHAPTER 6

Short-Term Training for Explosive Muscle Performance Affects Agonist Neural Drive and the Mechanical Properties of the Muscle-Tendon Unit
6.1 Introduction

Chapters 3 and 4 showed that explosive muscular force production, often measured as the rate of force development (RFD), is functionally important during explosive sports activities, where time to develop force is limited. RFD is therefore also likely to be important during injury prevention mechanisms (Gruber et al. 2007, Krosshaug et al. 2007, Domire, Boros & Hashemi 2010). Consequently, a greater understanding of how explosive force production can be improved with strength training and the contributory neural, mechanical and architectural adaptations has implications for sports performance and health.

Training to enhance maximum voluntary force (MVF) production, via sustained high load contractions (‘training for maximum strength’), has proved successful at increasing this component of strength, but there is equivocal evidence for its effects on explosive force production (Aagaard et al. 2002a, Suetta et al. 2004, Andersen et al. 2010, Blazevich et al. 2009, Kubo et al. 2001, Kubo et al. 2007b, Rich, Cafarelli 2000). Chapter 5 found that after four weeks of training for maximum strength there was no change in RFD or agonist neural drive over the first 150 ms of explosive contractions, despite a concurrent increase in MVF and agonist neural drive at MVF. This suggests that short-term improvements in explosive and maximal force production may require distinct training stimuli that elicit different adaptations.

Training with short (~1 second) high RFD contractions, which place a substantial emphasis on producing explosive rather than maximal force (‘training for explosive strength’), may provide the specific stimuli required to improve this aspect of force production. Furthermore, the smaller loading volume associated with training for explosive strength, and the possibility of RFD gains after just 4 weeks (Gruber et al. 2007, Barry, Warman & Carson 2005), suggests that functional improvements may be obtained without the level of fatigue and discomfort typically associated with training for maximum strength. However, the neural, peripheral (mechanical and architectural) adaptations to training for explosive strength, and their effects on RFD, have not been widely investigated. Early evidence has linked RFD gains after training for explosive strength to increased motor unit firing rate (Van Cutsem, Duchateau & Hainaut 1998) and agonist surface EMG in the early phase of explosive contractions (Gruber et al.
2007, Barry, Warman & Carson 2005). However, these surface EMG studies did not normalise recordings to a maximal M-wave, so their results may have been confounded by variations in signal conduction and electrode relocation (Folland, Williams 2007a, Gandevia 2001). Furthermore, evoked supramaximal contractions (e.g. 8 pulses at 300Hz), that generate the maximum possible RFD (de Ruiter et al. 2004, de Ruiter et al. 1999), may provide a reference measure for calculating the extent to which volitional contractions are utilising the available force generating capacity, but this method has not been employed in the context of training for explosive strength.

Whilst there is early evidence for increased neural drive following short-term training for explosive strength, it is unclear if mechanical and architectural adaptations might also occur. Any changes in muscle size, architecture or MTU stiffness could potentially also contribute to improvements in voluntary and involuntary RFD after training. Previous studies of training for maximum strength have reported changes in muscle architecture (specifically fascicle length and pennation angle) after 4-5 weeks (Seynnes, de Boer & Narici 2007, Blazevich et al. 2007), and increased tendon stiffness after 9-10 weeks (Seynnes et al. 2009, Kubo et al. 2010), but the volume of loading in this situation is likely to be greater than that experienced when purely training for explosive force production. This might suggest that mechanical and architectural adaptations are unlikely to occur with short-term training for purely explosive strength; however, this hypothesis has not yet been tested. Clearly, further investigation is required to understand the neuromuscular adaptations to explosive strength training.

The purpose of this study was to investigate changes in RFD after just 4 weeks of training for explosive, rather than maximum force production, and provide a detailed assessment of the neural, mechanical and architectural adaptations contributing to the observed changes in function. It was hypothesised that the training would improve explosive strength due to neural adaptations, but that mechanical and/or architectural adaptations would not occur.
6.2 Methods

6.2.1 Participants
Ten male participants (age, 20 ± 2 yrs; height, 182 ± 7 cm; mass, 74 ± 7 kg) completed the study. Participants were recreationally active (moderate exercise ≤ 4 times per week), but not involved in any form of strength or power training. All the participants were healthy, injury free and provided written informed consent prior to their involvement in this study, which was approved by the Loughborough University ethical advisory committee.

6.2.2 Overview
Participants completed a familiarisation session and two measurement sessions before (each separated by 2-3 days), and two measurement sessions (separated by 2-3 days) after, 4 weeks of isometric explosive strength training of the knee extensors. Training was completed on one leg (trained leg) chosen at random, but all pre and post-training measurements were taken from the trained and untrained leg. Pre-training and post-training measurement sessions took place in the 10 days prior to the start of training and 2-6 days after the last training session. The first of the pre- and post-training measurement sessions consisted of recording knee extension/flexion force and surface EMG of the superficial knee extensors and bicep femoris throughout electrically evoked, explosive voluntary, and maximal voluntary isometric knee extensions. The second of the pre- and post-training measurement sessions involved recording ultrasonic images of the vastus lateralis at rest and during ramped maximal voluntary isometric knee extensions in order to assess mechanical and architectural properties of the muscle-tendon unit (MTU). All measurement and training sessions, were completed in an isometric strength testing chair, (Bojsen-Moller et al. 2005, Parker et al. 1990) with knee and hip angles of 85° and 100°, respectively (180° representing full extension).

6.2.3 Training
The aim of the training programme was to enhance explosive rather than maximum force production, whilst minimising the volume of loading and fatigue. Participants
completed 4 training sessions per week for 4 weeks. Each session consisted of a brief warm-up of submaximal isometric knee extensions, followed by 4 sets (separated by 2 min) of 10 unilateral isometric knee extensions (separated by 5 s) using the same apparatus as the measurement trials. Prior to each contraction participants were instructed to completely relax before pushing “fast and hard” for ~1 s, in an attempt to achieve at least 90% of their maximal voluntary force (MVF) as quickly as possible, and without prior countermovement. MVF was determined via the method detailed below in the pre-training measurement sessions and re-determined at the start of the first training session each week. Participants were instructed to avoid contracting the untrained leg during each effort. For biofeedback, a computer monitor displayed the force-time curve with a cursor at 90% of MVF, the slope of the force-time curve (established with a 1 ms constant epoch), and the baseline of the force-time curve. The latter was used to confirm that a countermovement had not occurred.

6.2.4 Measurement Sessions

6.2.4.1 Measurements

Participants were firmly secured in the strength testing chair with a waist belt and shoulder straps. Force was measured with a calibrated U-shaped aluminium strain gauge (linear response up to 1000 N (Jones, Parker 1989)), which was in series with an ankle strap placed 2 cm proximal to the medial malleolus, and positioned perpendicular to tibial movement during knee extension/flexion. The force signal was amplified (x500), interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). The force signal was notch filtered at 100 and 200 Hz in both directions with an infinite impulse response digital filter (q-factor of 100), to remove harmonics of the mains frequency.

Surface EMG was recorded from the rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM), and long head of the bicep femoris (BF), using a Delsys Bagnoli-4 EMG system (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), a single differential surface electrode configuration (1 cm inter-sensor distance, model DE-2.1, Delsys, Boston, USA) was
attached over each muscle using adhesive interfaces. To standardise electrode placement across participants and sessions, the same experienced investigator placed the electrodes over the belly of each muscle, parallel to the presumed orientation of the muscle fibres, at ~50, 55, 90, and 45% of the distance between the greater trochanter and lateral femoral condyle for the RF, VL, VM and BF, respectively. The location of each electrode during pre-testing was outlined on the skin surface with a permanent marker, and marks were re-applied on each successive visit to the laboratory (i.e. every 2-3 days) to ensure identical placement post-testing. EMG signals were amplified (x100, differential amplifier 20-450 Hz) and sampled at 2000 Hz with the same analogue to digital converter and PC as the force signal, prior to being band-pass filtered (6-500 Hz) in both directions using a 2nd order Butterworth digital filter.

The femoral nerve was electrically stimulated (DS7AH, Digitimer Ltd., UK) with single square wave pulses (0.1 ms duration) to elicit compound muscle action potentials (M-waves), twitch contractions, and octet contractions. The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom adapted stimulation probe (1 cm diameter, Electro Medical Supplies, Wantage, UK) protruding 2 cm perpendicular from the centre of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve in the femoral triangle. The precise location of the cathode was determined as the position that evoked the greatest twitch response for a particular submaximal electrical current (typically 30-50 mA).

An ultrasound machine (Toshiba, Power Vision 6000, SSA-37OA; Mount International Ultrasound Services Ltd) with a linear array probe (7.5-MHz B-mode, with a scanning width and depth of 60 and 50 mm, respectively) was used to collect video images of the VL. The probe was held longitudinally over the VL at 50% of the distance between the greater trochanter and the lateral femoral condyle. An echo-absorbent marker (elastic band), taped over the skin surface perpendicular to the direction of the probe, provided a reference point within the video images of any probe movement relative to the skin. The position of the probe in the pre-training measurement session was outlined on the surface of the skin with permanent marker, and these marks were re-applied on each successive visit to the laboratory (i.e. every 2-3 days) to ensure identical placement post-testing. Images were recorded with a digital video recorder (Sony Walkman, GV-
D900E) at 25 Hz, and synchronised with force recordings via a custom built remote trigger. When pressed, the trigger generated a pulse on the force trace and a simultaneous high frequency sound wave which was amplified, via a microphone, and recorded in real-time with the ultrasonic images onto the digital video through a common mixer. EMG signals from the BF were collected throughout the ultrasound measurement trials. During off-line analysis, the processed force and EMG traces were exported to a Microsoft Excel spreadsheet at 25 Hz to match the sampling frequency of the video. Video recordings were imported into manual digitisation software (with a resolution of 0.16 mm; Avi Digitiser Version 1.2 Beta, RF Spectrum Modelling Ltd) for further analysis. This digitisation software provided an oscilloscope to identify the 1st frame during which the high frequency sound wave occurred.

6.2.4.2 Protocol

The protocol was completed first with one leg and then repeated with the other leg. A consistent, but randomly assigned order was used for each individual. The protocol consisted of the following procedures:

**Evoked contractions:** Twitch contractions were evoked (via single pulses) at incremental current intensities until a simultaneous plateau in force and M-wave response of each muscle was achieved (typically between 100-160 mA). Thereafter, the electrical current was increased by 20% and three supramaximal twitches were elicited at 12 second intervals. Variables recorded and averaged across the three supramaximal twitch contractions included peak-peak M-wave (M\text{max}) response of each agonist muscle, peak force, time-to-peak force, force at 50 ms from force onset, and involuntary maximal electromechanical delay (EMD\text{max}; defined as the time difference between the first agonist EMG signal to deflect away from baseline, and force onset). At the same supramaximal current as the twitch contractions, 3 octet contractions (8 pulses at 300 Hz) were evoked (at 12-s intervals), to measure the maximal capacity of the MTU for RFD (de Ruiter et al. 2004, de Ruiter et al. 1999). Variables recorded and averaged across the three supramaximal octet contractions included peak force, time-to-peak force, and force at 50 and 100 ms from force onset. Octet forces were also normalised to MVF. Octet data from only seven participants was obtained as a result of the discomfort caused by this type of stimulation.
Explosive voluntary contractions: Following the evoked contractions, participants completed a warm up of submaximal voluntary contractions, followed by 10 explosive voluntary contractions (each ~20 s apart). Prior to each contraction participants were provided with the same instruction as that given during the training efforts. Explosive voluntary contractions were completed separately from maximal voluntary contractions (MVCs; detailed below) because previous work has highlighted the importance of instruction with regards to performance outcome (Sahaly et al. 2001). The three explosive voluntary contractions with the largest peak slope and no discernible countermovement or pre tension (change of baseline force of < 0.5 N during the 100 ms prior to contraction onset) were used for analysis. Analysis consisted of measuring force at 50, 100 and 150 ms from force onset and RFD between these time points. Force and RFD variables are reported in absolute terms and relative to MVF (detailed below). Voluntary force at 50 and 100 ms was also calculated as a percentage of octet force at 50 and 100 ms, respectively (voluntary/octet), to provide a measure of agonist neural drive during the up-slope of the voluntary force-time curve. Furthermore, the root mean square (RMS) of the EMG signal of each muscle was measured in three, 50 ms time windows (0-50, 50-100, and 100-150 ms) from EMG onset (first agonist muscle to be activated). Agonist (RF, VL, and VM) RMS EMG values were normalised to M_{max} and averaged across the three muscles to give a mean agonist value. Antagonist (BF) RMS EMG values were normalised to maximal BF RMS EMG determined during the knee flexor MVCs (detailed below). Voluntary EMD_{max} was defined as the time difference between EMG and force onset, and is reported in absolute terms and as a percentage of involuntary EMD_{max}. All explosive voluntary force and EMG variables were averaged across the three explosive voluntary contractions chosen for analysis.

Maximal voluntary contractions: Following the explosive contractions participants completed three knee extensor isometric MVCs (separated by ≥ 30 s). Biofeedback and verbal encouragement were provided during and between each MVC. Knee extensor MVF was the greatest voluntary force achieved by a participant, in any of the knee extensor MVCs or explosive contractions during that laboratory visit. RMS EMG of each agonist muscle was recorded during a 500 ms epoch at MVF (250 ms either side of MVF), normalised to M_{max} and averaged across the three quadriceps muscles. Participants then completed 3 knee flexor isometric MVCs, at the same hip and knee
joint angle as the knee extensor contractions. RMS EMG during a 500 ms epoch at the
greatest knee flexor force (250 ms either side) was measured to give maximal BF RMS
EMG.

Ultrasound procedures: Ultrasound images were recorded while participants were at
rest, and then throughout 2 ramped contractions (separated by ~2 min). The ramped
contractions involved participants steadily increasing force production from rest to
MVF over an 8 s period. If peak force differed between the two ramped contractions by
more than 10% participants were asked to perform another effort. On completion of the
ramped knee extensor MVCs, participants completed 3 knee flexor MVCs to establish
the greatest flexor force and maximal BF RMS EMG as outlined above. The best video
image of the two ramped knee extensor MVCs was used for further analysis. Muscle
thickness, pennation angle and fascicle length were measured in 10 consecutive frames
recorded at rest, and averaged across these 10 frames (Figure 6.1A). Muscle thickness
was defined as the mean distance between the superficial and deep aponeurosis
measured at both ends of the 60 mm image. Pennation angle was defined as the angle
between a muscle fascicle, chosen at random, and the deep aponeurosis. The full length
of the muscle fascicles was not visible in the images, so fascicle length was
extrapolated using trigonometry, from pennation angle, and muscle thickness. Whilst
there are errors associated with this extrapolation method, due to the curvature of the
muscle fascicles and aponeurosis, these errors are typically within the region of 2-7%

To assess MTU stiffness, the displacement of two visible cross points between a muscle
fascicle and the aponeurosis, relative to the echo-absorbent marker, were tracked and
digitised throughout the ramped MVC with the clearest images (Figure 6.1B). The
average displacement of the two cross-points across 5 frames was recorded at rest and
at 10% increments of internal quadriceps force up to MVF (see below); i.e., 1 frame at,
and 2 frames either-side of each 10% increment. The relationship between aponeurosis
displacement and internal quadriceps force was plotted and the slope of this relationship
from 10-50% and 50-90% of internal quadriceps MVF was defined as MTU stiffness at
low and high force levels, respectively. Maximal aponeurosis displacement and
displacement at 50% of internal quadriceps MVF were also assessed.
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Figure 6.1. Ultrasonic images of the vastus lateralis (VL) and vastus intermedialis (VI) muscles at rest (A) and during a maximal voluntary isometric knee extension (B). VL muscle thickness (mean distance between superficial and deep aponeurosis at both ends of the image), pennation angle ($\theta$; angle between a muscle fascicle and the deep aponeurosis), and fascicle length (extrapolated using trigonometry) were determined at rest (A). The displacement of two cross-points between VL muscle fascicles and the deep aponeurosis (P1 and P2), relative to the echo-absorbent marker, was tracked throughout ramped isometric knee extensions up to the maximal voluntary effort (B). The average displacement of these cross-points was plotted against internal quadriceps force to measure muscle-tendon stiffness.

Internal quadriceps force was estimated using previously reported methods (Bojsen-Moller et al. 2005). Briefly, measured force was corrected for the negative contribution from antagonist co-activation as follows: Antagonist (BF) RMS EMG (250 ms moving epoch) during the ramped contractions, as a ratio of maximal BF RMS EMG (during the knee flexor MVCs), was multiplied by maximal knee flexor force, and summed with measured extensor force. Corrected extensor force was then multiplied by the external moment arm (distance along the tibia between the anterior edge of the tibial condyle and the centre of the ankle strap) to provide an external moment of force. Finally, the external moment of force was divided by the internal knee moment arm,
derived as a function of hip and knee angle from Visser et al. (1990), to provide internal quadriceps force.

Soft tissue compression during isometric knee extensor MVCs causes knee angle changes and subsequent passive aponeurosis displacement (Stafilidis et al. 2005, Arampatzis et al. 2007a, Bojsen-Moller et al. 2003). We were unable to correct for passive aponeurosis displacement due to erroneous electro-goniometer data collected during the measurement sessions. However, we believe that for this repeated measures study design any changes in soft tissue compression pre to post training were likely to be minimal, and the within-participant comparison of trained vs. untrained leg would be expected to control for any possible changes in passive aponeurosis displacement. Mechanical variables measured for one participant using ultrasound were not included in the statistical analysis, because his deep aponeurosis was poorly defined in the video images.

6.2.5 Data Analysis and Statistics

For the explosive voluntary and involuntary contractions force and EMG onsets were identified manually, which is considered the ‘gold standard’ method for detecting signal onsets (Allison 2003, Moretti et al. 2003, Pain, Hibbs 2007, Pulkovski et al. 2008). This method is discussed in detail in Chapter 3. Briefly; the same investigator identified onsets with a constant y-axis scale of ~1 N and 10 mV, for force and EMG respectively, and an x-axis scale of 500 ms.

The reliability of each mechanical and architectural variable (including twitch and octet force characteristics, muscle-tendon stiffness, aponeurosis displacement, muscle thickness, pennation angle, and fascicle length) was assessed by calculating intra-class correlation coefficients (ICC) for repeated measures (i.e., pre-training and post-training) in the untrained leg. To investigate the coefficient of variation in mechanical variables as a result of manual digitisation errors, six ultrasound recordings were re-analysed by the same investigator a week after the original analysis. Chapters 3 and 5 report good repeatability of the volitional parameters assessed in this study.
For each dependent variable the influence of time (pre vs. post) and leg (trained vs. untrained) was analysed with a two way repeated measures ANOVA. Paired t-tests were then used to determine within leg differences between pre and post training measures, and a stepwise Bonferroni correction procedure was employed for all time series data. Statistical analysis was completed using SPSS version 16, and the significance level was set at P<0.05. Pearson’s product moment correlations were used to assess the strength of bivariate relationships between changes in key dependent variables.

### 6.3 Results

#### 6.3.1 Voluntary Force

MVF increased in both the trained (+11%; Paired t-test, P<0.001) and untrained (+4%; Paired t-test, P = 0.007) leg. This increase was greater in the trained leg resulting in a time by leg interaction effect (ANOVA, P = 0.002; Figure 6.2).

![Figure 6.2](image)

**Figure 6.2.** Maximal voluntary force (MVF) during isometric knee extensions of the trained leg (filled squares) and untrained leg (clear circles) pre and post training for explosive strength. Data are mean ± SD (n = 10). ** denotes a time by leg interaction effect (P<0.01).

During the explosive contractions force achieved by the trained leg at 50, 100 and 150 ms after force onset was greater following training (+70, +16 and +14%, respectively; Figure 6.3A). This was primarily due to a greater absolute RFD (+70 %) during the initial 50 ms after training, with no change in the absolute RFD from 50 ms onwards.
The changes in absolute explosive voluntary force at 150 ms were highly correlated with the changes in MVF \( (r = 0.82; \ P = 0.004) \). For the untrained leg there was a tendency for RFD over the first 50 ms (and thus force at 50 ms) to increase following training \(+28\%; \text{ Paired t-test, } P = 0.09\) , but there was no change in RFD or force achieved at any other time (Table 6.1; Figure 6.3B).

Normalised explosive voluntary force (normalised to MVF) of the trained leg was greater at 50 ms, but unchanged at 100 and 150 ms, following training (Figure 6.3C). These results reflect an increase in normalised RFD of the trained leg from 0-50 ms, but a decrease from 50-100 ms and no change from 100-150 ms, after training (Table 6.1). There was no change in RFD or force of the untrained leg when normalised to MVF (Figure 6.3D; Table 6.1).

**Table 6.1.** Absolute and normalised rate of force development (RFD) parameters recorded in the trained and untrained leg during explosive isometric knee extensions pre and post training for explosive strength.

Data are means ± SD \((n = 10)\). A training effect is denoted by * \((P<0.05)\) or ** \((P<0.01)\).

<table>
<thead>
<tr>
<th>RFD</th>
<th>Trained leg</th>
<th>Untrained leg</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>Absolute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-50 ms (N.s(^{-1}))</td>
<td>1809 ± 669</td>
<td>2781 ± 942**</td>
</tr>
<tr>
<td>50-100 ms (N.s(^{-1}))</td>
<td>3948 ± 612</td>
<td>3867 ± 562</td>
</tr>
<tr>
<td>100-150 ms (N.s(^{-1}))</td>
<td>1610 ± 377</td>
<td>1725 ± 278</td>
</tr>
<tr>
<td>Normalised</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-50 ms (MVF.s(^{-1}))</td>
<td>3.80 ± 1.46</td>
<td>5.17 ± 1.61*</td>
</tr>
<tr>
<td>50-100 ms (MVF.s(^{-1}))</td>
<td>8.19 ± 0.74</td>
<td>7.26 ± 0.85*</td>
</tr>
<tr>
<td>100-150 ms (MVF.s(^{-1}))</td>
<td>3.35 ± 0.75</td>
<td>3.27 ± 0.69</td>
</tr>
</tbody>
</table>

MVF; maximal voluntary force, time period depicts time over from force onset which RFD was measured.
6.3.2 Evoked Force

Octet peak force increased in the trained leg after training, but there was no change in time to peak force (Table 6.2). Octet force of the trained leg at 50 and 100 ms after force onset was greater following training (Table 6.2 and Figure 6.4A) with increased evoked RFD over both the 0-50 (pre, 3858 ± 226 vs. post, 4144 ± 260 N.s⁻¹; Paired t-test, P = 0.039) and 50-100 ms time windows (pre, 2161 ± 478 vs. post, 2452 ± 310 N.s⁻¹; Paired t-test, P = 0.05). In contrast, when normalised to MVF, octet force at 50 and 100 ms was unchanged following training (Figure 6.4C). This reflected the significant correlation between the changes in MVF and octet force at both 50 (r = 0.80; P = 0.031) and 100 ms (r = 0.83; P = 0.022) after force onset. The untrained leg showed an unchanged mechanical response to the octet stimulation following training (Figure 6.4B).
and D; Table 2). Twitch peak force, time to peak force, and RFD over the first 50 ms were similar pre and post training for both the trained and untrained leg (Table 6.2).

Figure 6.4. Absolute (A and B) and normalised (C and D) force (normalised to maximal voluntary force; MVF) during evoked supramaximal octet contractions of the knee extensors, pre (filled squares) and post (open circles) training for explosive strength, in the trained (A and C) and untrained leg (B and D). Data are mean ± SD (n = 7). A training effect is denoted by * (P<0.05), or ** (P<0.01).


### 6.3.3 Neural Drive

At MVF agonist normalised EMG was unchanged in the trained (pre, 10.1 ± 2.6 vs. post, 11.2 ± 2.1% M_{max}; Paired t-test, P = 0.24) and untrained leg (pre, 10.6 ± 1.8 vs. post, 10.5 ± 2.2% M_{max}; Paired t-test, P = 0.95). In contrast, during the explosive voluntary contractions there was a 66% increase in agonist normalised EMG of the trained leg during the 0-50 ms time window (Figure 6.5A), following training. There was also a tendency for agonist normalised EMG of the trained leg to increase during the 50-100 ms period (+18 %; Paired t-test; P = 0.077), but there was no change in agonist normalised EMG between 100-150 ms (Figure 6.5A). The individual change in normalised explosive voluntary force that occurred at each time point after training was correlated with the change in agonist normalised EMG over the same time period (50 ms, r = 0.75, P = 0.013; 100 ms, r = 0.73, P = 0.016; 150 ms, r = 0.69; P = 0.029).

Consistent with the agonist normalised EMG results, voluntary/octet force in the trained leg increased by 55% at 50 ms, but was unchanged at 100 ms, following training (Figure 6.6A). Furthermore, the individual change in voluntary/octet force after training was very highly correlated with the change in normalised explosive voluntary force at
50 (r = 0.99; P<0.001) and 100 ms (r = 0.87; P = 0.011). For the explosive contractions of the untrained leg agonist normalised EMG and voluntary/octet force were similar pre and post training (Figure 6.5B and 6.6B).

Antagonist normalised EMG during the explosive contractions was also similar pre and post training for both the trained (0.11<P<0.33) and untrained legs (0.54<P<0.83). Training did not affect voluntary EMD$_{max}$ (~16 ms in both legs and conditions), involuntary EMD$_{max}$ (~7 ms in both legs and conditions) or voluntary EMD$_{max}$ relative to involuntary EMD$_{max}$ (~230% in both legs and conditions).

**Figure 6.5.** Agonist normalised EMG of the trained (A) and untrained (B) leg during explosive isometric knee extensions performed pre (light bars) and post (dark bars) training for explosive strength. Activation was measured with the root mean square of the EMG signal normalised to maximal M-wave (% M$_{max}$), and analysed in three, 50 ms time windows; 0-50, 50-100, and 100-150. Values were averaged across the three superficial quadriceps muscles and group data are expressed as mean ± SD (n = 10). A training effect is denoted by * (P<0.05).
Figure 6.6. Force at 50 and 100 ms from force onset during explosive voluntary isometric knee extensions, as a percentage of octet force at 50 and 100 ms, respectively (Voluntary/Octet), pre (light bars) and post (dark bars) training for explosive strength, in the trained (A) and untrained leg (B). Data are means ± SD (n = 7). A training effect is denoted by * (P<0.05).

6.3.4 Mechanical and Architectural Properties of the Muscle-Tendon Unit

MTU stiffness at high force levels (50-90% MVF) increased in the trained leg, but not the untrained leg, following training (Figure 6.7; Table 3), reflecting a time-by-leg interaction effect (ANOVA, P = 0.009). However, there was no change in maximal aponeurosis displacement in the trained leg following training (Table 6.3). At low force levels (10-50% MVF) MTU stiffness in the trained leg was unchanged, despite a tendency for aponeurosis displacement at 50% MVF to be greater (+43%; P = 0.058; Table 6.3), following training.
Figure 6.7. The relationship between internal knee extensor force and aponeurosis displacement of the vastus lateralis in the trained (A) and untrained (B) leg pre (filled squares) and post (open circles) training for explosive strength. The slope of this relationship between 50-90% of maximal voluntary force is a measure of muscle-tendon stiffness at high force levels. Data are means ± SD (n = 9). * denotes a change in muscle-tendon stiffness with training (P<0.01).

There was a tendency for muscle thickness to increase in the trained leg (+7%; Paired t-test, P = 0.054; Table 6.3), but pennation angle and fascicle length were unaffected by training (Table 6.3). However, there was a negative correlation between the changes in fascicle length and normalised voluntary RFD from 0-50 ms (r = -0.68; P = 0.044). In the untrained leg there were no changes in any of the mechanical or architectural properties of the MTU (Table 6.3). The coefficients of variation for the repeated analysis of the same ultrasound recordings were: MTU stiffness between 50-90% MVF, 8.3%; maximal aponeurosis displacement, 3.3%; muscle thickness, 0.2%; pennation angle, 3.6%; and fascicle length, 3.6%.
Table 6.3. Mechanical and architectural properties of the muscle-tendon unit (MTU) of the trained and untrained leg, measured at the vastus lateralis pre and post training for explosive strength. The intra-class correlation coefficients (ICC) are shown for the repeated measurements of the untrained leg. Data are means ± SD (n = 9). A training effect or significant ICC is denoted by * (P<0.05) or ** (P<0.01).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Trained leg</th>
<th>Untrained leg</th>
<th>ICC</th>
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<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
</tr>
<tr>
<td>MTU stiffness 50-90% MVF (N.mm⁻¹)</td>
<td>468 ± 77</td>
<td>627 ± 92**</td>
<td>532 ± 118</td>
</tr>
<tr>
<td>MTU stiffness 10-50% MVF (N.mm⁻¹)</td>
<td>414 ± 134</td>
<td>359 ± 117</td>
<td>410 ± 158</td>
</tr>
<tr>
<td>Maximal displacement (mm)</td>
<td>14.8 ± 4.3</td>
<td>16.9 ± 7.0</td>
<td>15.3 ± 3.3</td>
</tr>
<tr>
<td>Displacement at 50% of MVF (mm)</td>
<td>9.2 ± 3.5</td>
<td>12.5 ± 6.1</td>
<td>9.8 ± 2.1</td>
</tr>
<tr>
<td>Muscle thickness (mm)</td>
<td>25.8 ± 3.5</td>
<td>27.5 ± 4.1</td>
<td>26.0 ± 2.7</td>
</tr>
<tr>
<td>Pennation angle (°)</td>
<td>17.4 ± 3.2</td>
<td>18.0 ± 3.9</td>
<td>16.8 ± 2.8</td>
</tr>
<tr>
<td>Fascicle length (mm)</td>
<td>87.6 ± 9.6</td>
<td>91.8 ± 12.2</td>
<td>92.5 ± 16.2</td>
</tr>
</tbody>
</table>

6.4 Discussion

The present study investigated the neuromuscular responses to four weeks of training for explosive, rather than maximum force production. Increased voluntary RFD in the first 50 ms was primarily due to enhanced agonist neural drive in the same time period, whilst explosive voluntary force production beyond 100 ms increased in proportion to MVF. The surprising increase in octet RFD reflected an enhanced capacity of the MTU for RFD, and demonstrates that peripheral adaptations occurred as a result of the training. Peripheral adaptations included increased MTU stiffness between 50-90% MVF; however this is unlikely to explain increased octet RFD, which was measured up to only 62% MVF. Instead, changes in absolute octet RFD were proportional to and correlated with changes in MVF. This finding and the tendency for increased muscle thickness suggests that hypertrophy may explain the enhanced capacity of the MTU for explosive and maximal force production.

Voluntary explosive force at 50, 100 and 150 ms after force onset increased in the trained leg due to the 70% improvement in voluntary RFD during the first 50 ms time
window. Two other studies have observed increased RFD after just four weeks of explosive strength training (Gruber et al. 2007, Barry, Warman & Carson 2005). In contrast, Chapter 5 found that four weeks of training for maximum strength, without a specific emphasis on performing the contractions explosively, had no effect on RFD. Moreover, evidence for increased RFD with training for maximum strength over periods longer than four weeks is equivocal (Aagaard et al. 2002a, Sueta et al. 2004, Andersen et al. 2010, Blazevich et al. 2009, Kubo et al. 2001, Kubo et al. 2007b, Rich, Cafarelli 2000). It therefore appears that short duration explosive contractions provide a more effective stimulus for improving RFD than sustained high load contractions. This has potential implications for training prescription, as the smaller loading volume experienced with purely explosive strength training may evoke substantial functional benefits (increased explosive and maximum strength) without the discomfort and fatigue typically associated with maximum strength training.

When normalised to MVF explosive voluntary force at 50 ms increased following training but was unchanged at 100 and 150 ms after force onset. Consequently, the 11% gains in maximum strength account for improved force production at time points ≥ 100 ms, but not during the initial phase (first 50 ms) of explosive contractions. This is consistent with the increased influence of MVF in the later phase of explosive force production, which was observed in Chapters 3 and 4 and has been previously reported by an earlier study (Andersen, Aagaard 2006). The differential response of normalised RFD showing an increase in the first 50 ms (+53%), but a decrease in the second 50 ms (-11%) following training is remarkably similar to the difference that is reported in Chapter 3 between explosive power athletes with untrained individuals (i.e. normalised RFD of athletes was greater for 0-50 ms, but lower during 50-100 ms). As the rise in normalised force during an explosive contraction is towards a fixed asymptote at 100%, an improved RFD during the first 50 ms appears to reduce the scope for high RFD during the subsequent phase.

The increase in initial normalised RFD appears to have been due to neural and/or peripheral adaptations specific to explosive force production. The 55% increase in voluntary/octet force after 50 ms indicates an increased ability to utilise the MTU’s capacity for RFD, and the similar 66% increase in agonist EMG normalised to M_max confirms a change in neural drive during the first 50 ms of explosive voluntary
contractions. There are earlier reports of increased surface EMG after four weeks of training for explosive strength (Gruber et al. 2007, Barry, Warman & Carson 2005), but these studies did not normalise EMG to $M_{\text{max}}$, or measure voluntary/octet force. The results of the present study provide unequivocal evidence of enhanced agonist neural drive in the early phase of explosive voluntary contractions, following short-term training for explosive strength. Specific neural adaptations are likely to include increased firing frequency and incidence of doublet discharge in the early phase of explosive contractions (Van Cutsem, Duchateau & Hainaut 1998).

Peripheral adaptations were assessed via supramaximal octet contractions, and results showed a surprising increase in octet force at 50 and 100 ms from force onset. This is a unique finding and reflects an increased capacity of the MTU for RFD, after explosive strength training. Interestingly, octet force at 50 and 100 ms increased in proportion to MVF (i.e. normalised octet force was unchanged), and these changes were highly correlated ($r \leq 0.80$), suggesting a common peripheral mechanism that does not appear to account for the substantial increase in the initial normalised voluntary RFD.

Despite the short duration of this training study and the limited loading volume of the explosive contractions, there was evidence of a hypertrophic response to the training, with a tendency for increased muscle thickness. It therefore appears likely that hypertrophy contributed to the increased capacity of the MTU for both explosive and maximal force production. Whilst hypertrophy has been observed after just 3 weeks with maximum strength training (Seynnes, de Boer & Narici 2007), this is the first study to suggest a similar effect after training for explosive strength. Further investigation, possibly with a larger sample size and using more sensitive methods to measure morphological adaptations (i.e., MRI) is required to corroborate these findings.

The surprising increase in MTU stiffness at high force levels in the trained leg occurred 4-5 weeks earlier than typically expected when training for maximum strength (Kubo et al. 2001, Kubo et al. 2007b, Kubo et al. 2006, Seynnes et al. 2009, Kubo et al. 2010, Reeves, Narici & Maganaris 2003). This is an interesting observation considering mechanical strain magnitude and volume are likely to be lower with explosive, as opposed to maximum, strength training, and both parameters have a positive influence on the biochemical (Arnoczky et al. 2002) and biomechanical (Kubo, Kanehisa &
Fukunaga 2001, Yamamoto et al. 2005) response of connective tissue to loading. However, explosive contractions provide a greater mechanical strain rate which, due to the viscoelastic properties of the MTU, predisposes the system to greater mechanical stress and strain energy for the same mechanical strain (McElhaney 1966), and could potentially explain the rapid changes in MTU stiffness observed in this study. Although MTU stiffness is thought to positively influence RFD (Bojsen-Moller et al. 2005, Wilson, Murphy & Pryor 1994), the stiffer mechanical response between 50-90% MVF following training does not appear to explain the increased maximal capacity of the MTU for RFD, as octet force at 50 and 100 ms was ~40 and ~62% MVF respectively. Nevertheless, a stiffer MTU at high mechanical loads is thought to reduce the risk of soft tissue injury (Buchanan, Marsh 2002, Garrett 1990), so this adaptation after just four weeks of training for explosive strength may have important implications for injury prevention.

MTU stiffness at low force levels did not change following training, although the force-displacement curve appeared to shift to the right (Figure 6.7A) with a tendency for aponeurosis displacement at 50% MVF to increase. These results may be an artefact of the poor reliability of the mechanical measurements from 0-50% MVF (ICC = 0.29-0.39), although the consistent measurements on the untrained leg tend to negate this explanation. It is possible that the separate components of the MTU may differentially contribute to MTU stiffness as force increases, and could show distinct mechanical responses to training (e.g., increased tendon stiffness at high force, and decreased muscle-aponeurosis stiffness at low force). Interestingly, there was a negative correlation between individual changes in fascicle length and normalised voluntary RFD in the first 50 ms time window ($r = -0.68$). A similar relationship was reported by a recent study (Blazevich et al. 2009), and although MTU stiffness was not measured, the authors suggested that longer fascicles may increase series compliance, which could negatively influence RFD in the initial phase of a contraction. Further research is required, using measurements at both the aponeurosis and tendon, to investigate the possibility of a differential mechanical response of the separate components of the MTU to strength training.
The only measured effect in the untrained leg was a small 4% increase in MVF, with a tendency for voluntary explosive force at 50 ms to increase ($P = 0.09$). This suggests that the explosive contractions of the current training intervention did not provide a sufficient stimulus for the level of cross-over effects typically observed with conventional maximum strength training (Lee, Gandevia & Carroll 2009, Shima et al. 2002, Carolan, Cafarelli 1992, Fimland et al. 2009) (Chapter 5). There was also no change in any of the twitch force parameters of the trained or untrained leg, which is consistent with the equivocal evidence for changes in twitch force parameters following any type of strength training (Del Balso, Cafarelli 2007, Pucci, Griffin & Cafarelli 2006, Duchateau, Hainaut 1984, Lee, Gandevia & Carroll 2009, Shima et al. 2002, Brown, McCartney & Sale 1990). Considering the significant improvements in octet RFD in the present study, it appears that the mechanical response to a single supramaximal electrical impulse is insensitive to subtle adaptations of the MTU. Finally, there was no change in voluntary or involuntary EMD$_{max}$ after training, supporting the evidence of Chapter 3 that EMD is not influenced by the explosive force capacity of the neuromuscular system.

In conclusion, training for explosive, rather than maximal, force production improved voluntary and involuntary explosive force, maximal strength, and MTU stiffness at high force levels after just four weeks. Increased agonist neural drive and maximum strength accounted for improved voluntary explosive force production in the initial and late phases, respectively. The increased maximal capacity of the MTU for RFD in response to an evoked octet appeared to be due to hypertrophy. The increase in muscle-tendon stiffness at high forces after explosive strength training may be an important adaptation for reducing the risk of soft-tissue injury.
CHAPTER 7

Contraction Type Influences the Human Ability to Utilise the Available Torque Capacity of Skeletal Muscle during Explosive Efforts
7.1 Introduction

The capacity of the neuromuscular system for explosive force/torque production, typically measured as rate of torque development (RTD), is considered functionally more important than maximal voluntary torque (MVT) during explosive human movements such as sprinting, jumping, or restabilising the body following a loss of balance (Newton, Kraemer 1994, Aagaard et al. 2002a, de Ruiter et al. 2004, Suetta et al. 2004). This assumption was supported by the results of Chapters 3 and 4. Whilst the influence of contraction type (i.e. isometric, concentric or eccentric) on MVT in-vivo has been documented extensively via the MVT-velocity relationship (Yeadon, King & Wilson 2006, Pain, Forrester 2009, Webber, Kriellaars 1997, Dudley et al. 1990, Aagaard et al. 2000, Seger, Thorstensson 1994), little is known about the capability for explosive torque production during different types of contractions.

The majority of past studies (including the previous Chapters) have investigated RTD during isometric contractions (de Ruiter et al. 2004, Barry, Warman & Carson 2005, Andersen, Aagaard 2006, Aagaard et al. 2002b), and occasionally during the acceleration phase of isoinertial dynamic contractions (Haff et al. 1997, Wilson, Murphy & Pryor 1994, Adamson et al. 2008). However, the latter provides an experimentally inconsistent situation, as the movement dynamics (acceleration, velocity and displacement) are not controlled and combine with the inertial properties of the system in a non-linear manner (e.g., an increase in force will cause a change in velocity, which in turn will affect the amount of subsequent force that can be produced). This gives rise to torques that vary within and between trials and participants, and confound RTD measurements. In contrast performing explosive concentric and eccentric contractions at a constant acceleration from stationary, and thus with known displacement and velocity, may provide a more controlled situation in which to investigate RTD during the acceleration phase of dynamic contractions.

A further complication with measuring RTD during dynamic contractions is that joint angle (which influences torque producing capacity) will change throughout the effort, and this change is in opposite directions for concentric and eccentric contractions. Consequently, it is not possible to match joint angle throughout the different types of contractions, apart from at a single time point/angle. The discrete influence of joint
angle on explosive torque production can be evaluated by comparing isometric contractions at different angles; however, isolating the influence of the type of contraction is problematic. One approach is to normalise the explosive torque produced at any time point during the different types of contractions to the MVT available at that specific joint angle and angular velocity. This also enables us to investigate whether explosive torque production changes in proportion to MVT. Another approach is to normalise explosive voluntary torque to the maximum contractile capacity for explosive torque production during an evoked octet contraction (8 supramaximal pulses at 300 Hz; (de Ruiter et al. 2004, de Ruiter et al. 1999, Deutekom et al. 2000)) in identical contractile conditions. This also provides an experimental approach that can dissociate between the neural and peripheral limitations of explosive torque production during different types of contraction.

Experimental studies have reported that it takes >300 ms to achieve MVT during explosive isometric contractions from rest (Aagaard et al. 2002a, Thorstensson et al. 1976), and >100 ms when there is tension in the muscle prior to explosive torque onset (de Ruiter et al. 2006). In contrast, successful computer simulation models of explosive dynamic human movements, initiated from a pre-activated state, often rely on MVT being achieved within 70 ms of torque onset (Yeadon et al. 2010, Wilson, Yeadon & King 2007). These reports suggest a possible disparity in explosive torque capabilities between isometric and dynamic contractions; however, there has not been a direct comparison of RTD during different types of contractions. There is also limited evidence of the effect of joint angle on RTD. During the initial 40 ms of explosive isometric contractions in humans torque production has been reported to change with joint angle, but only in proportion to MVT (de Ruiter et al. 2004). In contrast, animal studies have found a faster time to peak force with decreasing muscle length (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980, Rack, Westbury 1969), and this appears to primarily affect the later phases of explosive contractions (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980). Clearly, further work is required to understand the influence of the contractile conditions on explosive torque production, and if this changes in proportion to maximum torque production.

Chapters 3 and 4 show that human ability to utilise the muscles torque producing capacity explosively during isometric contractions is influenced by athletic
performance/training status. As athletic performance/training status has also been found to influence the shape of the MVT-velocity relationship, and thus the relative capacity for MVT production during different types of contractions (Amiridis et al. 1996), it could also affect the capacity for explosive torque production during different types of contractions. Preliminary insight into this possibility may be provided by comparing the explosive torque production across different types of contractions for individuals with a range of explosive athletic performance/strength training backgrounds.

The primary aim of this study was to compare explosive torque production during concentric, eccentric and isometric contractions, and examine the neural and peripheral limitations to explosive torque production in these different contractile conditions. Two isometric angles were also studied to examine the discrete influence of joint angle on explosive torque production. It was hypothesised that the contraction type and joint angle would both influence the ability to utilise the available torque capacity of the muscle in an explosive situation.

7.2 Methods

7.2.1 Ethical Approval

The procedures involved in this study were approved by the Loughborough University ethical advisory committee, and conformed to standards set by the latest revision of the Declaration of Helsinki. All of the participants in this study were healthy, injury free and provided written informed consent prior to their involvement.

7.2.2 Participants

Fourteen male participants (age, 24 ± 6 yrs; height, 1.78 ± 0.05 m; and mass, 75 ± 5 kg) completed the study: six were recreational to national level explosive power athletes (sprinters and jumpers) involved in regular strength and power training (≥ 3 sessions per week for ≥2 years); and eight were low to moderately active individuals (≤ 4 x aerobic activity a week) not involved in any strength and power training.
7.2.3 Overview

Participants visited the laboratory on 3 occasions each separated by 3-5 days. On each visit participants completed a series of voluntary and evoked contractions of the knee extensors on an isovelocity dynamometer. The first session involved: a series of isometric maximal voluntary contractions (MVCs) at different knee joint angles; electrically evoked concentric, eccentric and isometric octet contractions; and familiarisation with explosive voluntary concentric, eccentric and isometric contractions. In the second session surface EMG was collected from the three superficial quadriceps (agonist) muscles whilst participants completed explosive voluntary concentric, eccentric and isometric contractions, and during electrically evoked supramaximal twitch contractions to elicit compound muscle action potentials (M-waves). In the final session participants completed a series of concentric and eccentric isovelocity MVCs.

The isometric and isovelocity MVCs were used to determine joint angle and angular velocity specific MVT, for normalisation of explosive voluntary torque measured under concentric, eccentric and isometric conditions. Likewise, concentric, eccentric and isometric explosive voluntary torque was also normalised to electrically evoked octet torque in the same contractile conditions. Finally, the M-waves recorded in the second session were used for normalisation of surface EMG data collected during the concentric, eccentric and isometric explosive contractions of the same session.

7.2.4 Measurements

7.2.4.1 Dynamometer

Shoulder and waist straps secured participants firmly in the seat of the dynamometer (Con-Trex; CMV AG, Switzerland) with hip angle fixed at 95°. The analogue torque and crank angle (representing knee angle) signals were interfaced with an analogue to digital converter (CED micro 1401, CED, Cambridge, UK), and sampled at 2000 Hz with a PC utilising Spike 2 software (CED, Cambridge, UK). Torque and angle signals were low pass filtered at 21 and 12 Hz, respectively, using a 4th order zero-lag Butterworth digital filter. Knee angular velocity was derived from the knee angle signal by numerical differentiation with a 1 ms epoch. Verbal encouragement was given
throughout all voluntary contractions, and biofeedback was provided via a computer
monitor.

7.2.4.2 Concentric, Eccentric and Isometric Explosive Voluntary Contractions

Explosive voluntary contractions were performed in four conditions; concentric (CON),
eccentric (ECC), and isometrically at 101° (ISO101) and 155° (ISO155) knee joint
angles (Figure 7.1). During the concentric and eccentric conditions the crank arm was
slowly moved (~10°.s⁻¹) through the range of motion (94-161°) to the start position for
CON (94°) or ECC (161°). On reaching the start position the crank arm accelerated
from stationary, at a constant 2000°.s⁻², to a peak velocity of 450°.s⁻¹, moving 52° (94-
146° in CON and 161-109° in ECC) in 225 ms, before rapidly decelerating (-6000°.s⁻²)
to stop 15° later at the opposite end of the range of motion (Figure 7.1 and 7.2). In the
CON and ECC conditions participants performed ~15 maximum voluntary explosive
contractions (separated by ~30 s), when they were instructed to push as ‘fast and hard’
as possible at the start of the acceleration phase, from a completely relaxed state, and to
keep pushing for the entire range of motion. The crank angle signal was displayed on
the computer monitor with a cursor placed at the start position to indicate when the
participant should start pushing. During extensive pilot testing we found that
participants typically started generating torque 50-70 ms into the acceleration phase due
to a delayed response to the biofeedback. During three passive trials (no muscle
activation) of the CON and ECC conditions, torque due to the acceleration and weight
of the shank was recorded. In offline analysis (using Matlab; The MathWorks inc.,
Natick, MA, USA), the three passive torque-time curves in each condition were time
aligned and averaged to provide a mean passive torque-time profile. This was then time
aligned with, and subtracted from, each active trial in that condition, to calculate the
torque due to muscle activation (Figure 7.2).
Figure 7.1. A schematic of the hip and knee angles during explosive knee extensions performed on an isovelocity dynamometer, in four separate conditions; concentric (CON) and eccentric (ECC) and two isometric positions (101° (ISO101) and 155° (ISO155)). During the dynamic conditions the crank arm accelerated at 2000°.s⁻¹ from a knee angle of 94° to 146° (CON) and from 161° (ECC) to 109° (ECC), before decelerating over a further 15° of motion.
In both ISO101 and ISO155 participants completed ≥ 6 voluntary explosive contractions (separated by ~30 s), where they were instructed to push as ‘fast and hard’ as possible for 1 s, from a completely relaxed state. The baseline of the torque-time curve was displayed on the computer monitor to confirm that participants were completely relaxed prior to torque onset. Due to the static nature of ISO101 and ISO155, participants typically required fewer attempts at these conditions than the concentric or eccentric conditions, to ensure that ≥5 contractions met the criteria for
further analysis (see below). These two isometric conditions were used to examine the independent influence of joint angle on RTD. Furthermore, these specific joint angles were selected as occurring during the early phase (~75 ms into the acceleration phase) of CON (101°) and ECC (155°) explosive contractions, to consider if joint angle effects were influencing the comparison of CON and ECC conditions.

During offline analysis of the corrected torque data, contractions performed in the CON and ECC conditions were disregarded if they did not meet the following criteria: baseline torque within ± 2 Nm to ensure minimal muscle activity prior to the explosive effort; a change in baseline torque < 2 Nm in the 200 ms prior to torque onset; and torque onset occurred 20-75 ms from the start of the acceleration phase. Contractions performed in the ISO101 and ISO155 conditions were disregarded if torque baseline changed by > 1 Nm in the 200 ms prior to torque onset. From those contractions in each condition that met the selection criteria the three with the greatest proportion of MVT (see below) at 100 ms from torque onset were chosen for further analysis, which involved measuring torque at 25 ms intervals up to 150 ms. Torque onset was defined as the point at which the first derivative of the torque-time curve (determined with a 1 ms epoch) crossed zero for the last time. For comparison of explosive voluntary torques across the different types of contraction absolute torques were normalised, firstly to maximal voluntary torque (MVT): ISO101 and ISO155 torque values were normalised to measured isometric MVT at the same knee angle (see below); CON and ECC torque values were normalised to interpolated dynamic MVT at the same knee angle and angular velocity (interpolated from a dynamic MVT function; see below). Secondly, voluntary explosive torque at 50 ms from torque onset in each condition was calculated as a percentage of evoked explosive torque at 50 ms (see below) in identical contractile conditions (voluntary/evoked). The voluntary/evoked ratio was also calculated after each had been normalised to the relevant interpolated or measured MVT value, to control for any discrepancies in joint kinematics at the 50 ms time point between the voluntary and evoked trials.

7.2.4.3 Surface Electromyography

Surface electromyography (EMG) was recorded from the rectus femoris (RF), vastus lateralis (VL), and vastus medialis (VM) in the second session using a Delsys Bagnoli-4
Chapter 7: Influence of Contraction Type

EMG system (Delsys, Boston, USA). Following preparation of the skin (shaving, lightly abrading and cleansing with 70% ethanol), single differential electrodes (1 cm inter-sensor distance, model DE-2.1, Delsys, Boston, USA) were attached parallel to the presumed orientation of the muscle fibres at ~50, 55, and 80% of the distance between the greater trochanter and lateral femoral condyle over the belly of the RF, VL, and VM, respectively. EMG signals were amplified (x100, differential amplifier 20-450 Hz) and sampled at 2000 Hz with the same analogue to digital converter and PC as the torque and angle signals, prior to being band-pass filtered (6-500 Hz) with a 4th order zero-lag Butterworth digital filter.

During the explosive voluntary contractions agonist activation was assessed by measuring the root mean square (RMS) amplitude of the EMG signal of each muscle in three consecutive 50 ms time windows (0-50, 50-100, and 100-150 ms) from EMG onset (first agonist muscle to be activated). Agonist (RF, VL, and VM) RMS EMG values were normalised to \( M_{\text{max}} \) (see below) and averaged across the three muscles to give a mean agonist value. EMG onset was detected manually with x and y-axis scales of 500 ms and 20 mV, respectively. As discussed in Chapter 3, manual identification is considered the gold standard method of detecting signal onsets. All explosive voluntary torque and EMG variables were averaged across the three contractions chosen for analysis in each condition.

7.2.4.4 Electrical Stimulation

The femoral nerve was electrically stimulated (DS7AH, Digitimer Ltd., UK) with square wave pulses (0.1 ms duration) whilst participants were voluntary passive to elicit explosive octet contractions (via 8 pulses at 300 Hz) or compound muscle action potentials (M-waves; via a single pulse). The anode (carbon rubber electrode, 7 x 10 cm; Electro-Medical Supplies, Greenham, UK) was taped to the skin over the greater trochanter. The cathode, a custom adapted stimulation probe (1 cm diameter, Electro Medical Supplies, Wantage, UK) protruding 2 cm perpendicular from the centre of a plastic base (4 x 5 cm), was taped to the skin over the femoral nerve. At a knee angle of 101° a series of single pulses were elicited at incremental current intensities until a maximal current intensity (simultaneous plateau in torque and M-wave response of each
muscle) was achieved. Thereafter, supramaximal octet contractions and M-waves were elicited at 20% above the maximal current intensity.

Supramaximal octet contractions at 300 Hz are thought to measure the maximum capacity of the muscle-tendon unit for RTD (de Ruiter et al. 2004, de Ruiter et al. 1999, Deutekom et al. 2000). Three supramaximal octet (‘evoked’) contractions were elicited during each of the four conditions (CON, ECC, ISO101, and ISO155). In the CON and ECC conditions evoked contractions were elicited at 4° (~60 ms) into the acceleration phase, so that evoked torque onset would occur at a similar knee angle and angular velocity to that expected in the voluntary explosive contractions. The torque recorded during the evoked contractions was corrected for the acceleration and weight of the shank, using those methods described above, to calculate the torque due to evoked muscle activity. For comparison across all four conditions torque was measured at 25 ms intervals up to 75 ms (75 ms was the shortest time to peak torque - CON), but an additional 100 ms measurement was recorded during ISO101 and ISO155. Torque at each time point was averaged across the three evoked contractions in each condition. In ISO101 and ISO155 evoked peak torque, time-to-peak torque, and half relaxation time were also averaged across the three supramaximal octet contractions. Furthermore, evoked torque recorded in both ISO101 and ISO155 was normalised to evoked peak torque for the same contraction.

The peak-to-peak amplitude of supramaximal M-waves (M_{max}) is affected by joint angle (Tucker, Turker 2007). Therefore, three M_{max} were elicited at both 101 and 155° knee angles, and the average M_{max} at each angle was used to normalise volitional agonist EMG in these conditions. Three M_{max} were also elicited at 3°, 11°, and 25° into the acceleration phase of CON and ECC conditions. Extensive pilot work had shown that these positions were typically in the centre of the consecutive 50 ms time windows after volitional EMG onset, and thus average M_{max} at each position was used to normalise volitional agonist EMG during the 0-50, 50-100, 100-150 ms time windows, respectively.
7.2.4.5 Isometric Maximal Voluntary Contractions

Participants completed 3 sets of four isometric MVCs. The four contractions in each set were performed at 4 different knee angles; 101, 119, 136, and 155° (180° being full knee extension), and separated by ≥ 90 seconds. During each MVC participants were instructed to push as hard as possible for 3-5 s. The largest measured extensor torque at each knee angle was defined as maximal voluntary torque (MVT) at that angle. MVT at 101 and 155° was used for normalisation of explosive torque in ISO101 and ISO155, respectively, whilst MVT at all four angles was used to establish a torque – angle relationship (defined as a quadratic function) that set the estimates and bounds of the dynamic MVT function (see below; Table 7.2). The four knee angles chosen to assess MVT represented positions at 75 and 150 ms into the acceleration phase of CON (101°, 75 ms; 119°, 150 ms) and ECC (155°, 75 ms; 136°, 150 ms), ensuring that the torque – angle relationship accurately reflected the range of motion over which CON and ECC were performed.

7.2.4.6 Dynamic Maximal Voluntary Torque Function

To establish dynamic MVT as a function of joint angle and angular velocity (used to interpolate dynamic MVT for normalisation of explosive CON and ECC torque values), participants completed a cycle of four reciprocal eccentric-concentric isovelocity MVCs at three angular velocities; 100, 250, and 400°.s⁻¹. This protocol has been used previously to ensure that there is time for maximal voluntary activation, and thus MVT, to be achieved and sustained throughout the entire range of motion (Yeadon, King & Wilson 2006, Pain, Forrester 2009, King, Yeadon 2002, Forrester, Pain 2010). Total range of motion was set at ~100° (70-170°), which provided an isovelocity range of ~75, 62, and 40° at 100, 250 and 400°.s⁻¹, respectively. Following familiarisation at each velocity, participants were instructed to extend their knee as hard as possible throughout the entire cycle. If peak eccentric torque of at least two eccentric efforts in one cycle were not ≥ 90% of the largest recorded isometric MVT for that participant, the cycle was repeated. Active torque values were corrected for the effects of gravity using a 6th order polynomial to describe the passive torque – angle relationship determined during trials where the knee was moved through the range of motion at 10°.s⁻¹, whilst participants were voluntarily passive. For each velocity the largest gravity corrected
torque per degree of isovelocity movement was used to establish a dynamic MVT function, defined as the product of torque - angular velocity (Equations 7.1-7.6), differential activation - angular velocity (Equation 7.7) and torque - angle (Equation 7.8) functions. The nine parameters of the dynamic MVT function (Table 7.1) were obtained by minimising the weighted RMS difference between interpolated and measured values using a simulated annealing algorithm (Corana et al. 1987), with the initial parameter estimates and bounds as given in Table 7.2. A weighting for the RMS difference score function which forced ~85% of the measured values below the surface representing the dynamic MVT function (Figure 7.3) was used, as errors in the measured data were thought to be predominantly one-sided (i.e., due to submaximal effort; (Forrester et al. 2010; In Press). The average weighted RMS difference between measured and interpolated dynamic MVT values of all participants was 6 ± 2 Nm, which was equivalent to 1.3 ± 0.3% of maximum eccentric torque, and demonstrated that the interpolated MVT values were an accurate representation of the measured data.

Torque (T) – angular velocity for concentric velocities ($\omega \geq 0$); described by a Hill curve (Yeadon, King & Wilson 2006):

$$T = \frac{C}{(\omega_c + \omega)} - T_c$$

(7.1)

Where,

$$T_c = \frac{T_0 \omega_c}{\omega_{max}}$$

(7.2)

$$C = T_c (\omega_{max} + \omega_c)$$

(7.3)

Torque – angular velocity for eccentric velocities ($\omega \leq 0$); described by a rectangular hyperbola (Yeadon, King & Wilson 2006):

$$T = \frac{E}{(\omega_E - \omega)} + T_{ecc}$$

(7.4)

Where,

$$\omega_E = \frac{(T - T_c) \omega_{max} \omega_c}{4.3 T_0 (\omega_{max} + \omega_c)}$$

(7.5)

$$E = -(T_{max} - T_0) \omega_E$$

(7.6)

Where 4.3 represents the ratio of the eccentric to concentric function slopes at $\omega = 0$, as interpolated by Huxley (1957).
Differential activation - angular velocity; described by a ramp style function (Forrester et al. 2010; In Press):

\[
a = a_{min} + \frac{(a_{max} - a_{min})}{1 + \exp\left(-\frac{(\omega - \omega_1)}{\omega_r}\right)} \quad (7.7)
\]

Torque – angle; described by a quadratic function (King, Wilson & Yeadon 2006):

\[
\frac{T(\theta)}{T(\theta_{opt})} = 1 - r(\theta_{opt} - \theta)^2 \quad (7.8)
\]

Table 7.1. The nine parameters of the dynamic maximal voluntary torque function

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>(T_0)</td>
<td>Maximum isometric torque (Nm)</td>
</tr>
<tr>
<td>(T_{ecc})</td>
<td>Maximum eccentric torque (Nm)</td>
</tr>
<tr>
<td>(\omega_{max})</td>
<td>Maximum concentric velocity (°.s(^{-1}))</td>
</tr>
<tr>
<td>(\omega_c)</td>
<td>Vertical asymptote of Hill hyperbola describing concentric torques</td>
</tr>
<tr>
<td>(a_{min})</td>
<td>Minimum activation; where maximum activation ((a_{max})) is equal to 1.0</td>
</tr>
<tr>
<td>(\omega_r)</td>
<td>Point of inflexion in differential activation function (°.s(^{-1}))</td>
</tr>
<tr>
<td>(\omega_1)</td>
<td>Rate of activation increase (°.s(^{-1}))</td>
</tr>
<tr>
<td>(\theta_{opt})</td>
<td>Optimal angle of the quadratic torque - angle function (°)</td>
</tr>
<tr>
<td>(r)</td>
<td>Width (standard deviation) of quadratic torque - angle curve from (\theta_{opt}) (°)</td>
</tr>
</tbody>
</table>
Table 7.2. Initial estimate and upper and lower bounds of the nine parameters defining the dynamic maximal voluntary torque function

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Initial estimate and bounds</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_0$</td>
<td>$T_0$ extrapolated from the quadratic function of the four measured isometric MVTs $\pm 10%$</td>
<td>To limit $T_0$ to within $10%$ of that derived from measured isometric values.</td>
</tr>
<tr>
<td>$T_{ecc}$</td>
<td>$T_0$ multiplied by 1.4</td>
<td>Based on Dudley et al. (1990).</td>
</tr>
<tr>
<td>$\omega_{\text{max}}$</td>
<td>$1300 \pm 500^\circ.s^{-1}$</td>
<td>Based on experimental values ($1000-1600^\circ.s$) reported by Forrester et al. (2010; In Press) $\pm 200^\circ.s^{-1}$.</td>
</tr>
<tr>
<td>$\omega_c$</td>
<td>$\omega_{\text{max}}$ multiplied by 0.375 $\pm 0.125$</td>
<td>Pertuzon, Bouisset (1972); Edman, Elzinga &amp; Noble (1978).</td>
</tr>
<tr>
<td>$a_{\text{min}}$</td>
<td>$0.77 \pm 0.22$</td>
<td>Used by Forrester et al. (2010; In Press) and based on the reported relationship between knee angular velocity and quadriceps EMG (Seger, Thorstensson 1994, Westing, Cresswell &amp; Thorstensson 1991).</td>
</tr>
<tr>
<td>$\omega_r$</td>
<td>$45 \pm 45^\circ.s^{-1}$</td>
<td></td>
</tr>
<tr>
<td>$\omega_1$</td>
<td>$\pm 90^\circ.s^{-1}$</td>
<td></td>
</tr>
<tr>
<td>$\theta_{\text{opt}}$</td>
<td>$\theta_{\text{opt}}$ extrapolated from the quadratic function of the four measured isometric MVTs $\pm 10^\circ$</td>
<td>To limit $\theta_{\text{opt}}$ to within $10^\circ$ of that derived from measured isometric values.</td>
</tr>
<tr>
<td>$r$</td>
<td>$95 \pm 85^\circ$</td>
<td>Based on normal joint range of motion</td>
</tr>
</tbody>
</table>
Figure 7.3. An example of maximal voluntary torque (MVT) values measured during isovelocity contractions of the knee extensors at six velocities (black circles). The surface of the optimised nine parameter function describing dynamic MVT relative to knee angle and angular velocity was used to interpolate angle and velocity specific MVT values for normalisation of explosive torque values. The RMS difference between measured and interpolated values was weighted so that ~85% of the measured values were forced below the surface.

7.2.4.7 Statistical Analysis

The influence of condition (CON, ECC, ISO101, and ISO155) on all dependent variables measured in explosive voluntary and evoked contractions was analysed with a repeated measures ANOVA (4 conditions). Paired t-tests and a stepwise Bonferroni correction were then used to determine paired differences between conditions at specific time points. Pearson’s product moment correlations were used to assess bivariate relationships in normalised explosive torque production between the conditions. Statistical analysis was completed using SPSS version 17, and the significance level was set at P<0.05.

7.3 Results

7.3.1 Kinematics of the Explosive Contractions

During the dynamic explosive contractions, voluntary torque onset in the CON and ECC conditions occurred at similar angular displacements and angular velocities (Table 7.3). In both CON and ECC explosive voluntary torque onset typically occurred 5-10
ms earlier in the acceleration phase than evoked torque onset, as denoted by the overall tendency for angular displacement and velocity to be greater at torque onset in the evoked contractions (Table 7.3). Voluntary EMG onset occurred at an angle of 96 ± 1° and an angular velocity of 74 ± 21°.s⁻¹ during the CON trials and at 159 ± 1° and -60 ± 39°.s⁻¹ during the ECC trials. Relative to these onsets $M_{\text{max}}$ was recorded at 22, 67, and 121 ms into the CON trials, and 18, 67, and 130 ms into the ECC trials. This confirmed that $M_{\text{max}}$ was recorded in the centre of each of the three consecutive 50 ms time windows from voluntary EMG onset in both CON and ECC conditions, and thus provided an appropriate reference for normalisation of volitional EMG during these periods.

**Table 7.3.** Knee joint angular displacement and angular velocity (kinematic parameters) at torque onset in explosive voluntary and evoked knee extensions completed in concentric (CON) and eccentric (ECC) conditions. P-values for paired differences between voluntary and evoked contractions are reported. Data are means ± SD (n = 14).

<table>
<thead>
<tr>
<th>Kinematic Parameter</th>
<th>Voluntary</th>
<th>Evoked</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CON Angle (°)</td>
<td>3.6 ± 1.2</td>
<td>4.4 ± 0.8</td>
<td>0.055</td>
</tr>
<tr>
<td>CON Velocity (°.s⁻¹)</td>
<td>117 ± 24</td>
<td>129 ± 16</td>
<td>0.037</td>
</tr>
<tr>
<td>ECC Angle (°)</td>
<td>3.1 ± 1.6</td>
<td>4.8 ± 1.6</td>
<td>0.708</td>
</tr>
<tr>
<td>ECC Velocity (°.s⁻¹)</td>
<td>-93 ± 38</td>
<td>-123 ± 31</td>
<td>0.086</td>
</tr>
</tbody>
</table>

**7.3.2 Volitional Parameters**

Absolute explosive voluntary torque was affected by condition at each of the six measured time points from torque onset (ANOVA, $P<0.001$; Figure 7.4A). These effects are consistent with the differential joint kinematics of the separate conditions, where ISO101 was performed at a joint angle close to $\theta_{\text{opt}}$, and thus recorded the highest torque values after the initial 50 ms; CON torque was greater than ISO101, ISO155 and ECC in the initial phase (first 50 ms) when angular velocity was relatively low and joint angle was near $\theta_{\text{opt}}$; and ECC torque was greater than ISO155 and CON in the later phase of the contraction (>100 ms), as angular velocity increased and the joint angle moved closer to $\theta_{\text{opt}}$. 
Normalised explosive voluntary torque (relative to measured/interpolated MVT at the relevant joint angle and angular velocity) was also influenced by condition at each measured time point from torque onset (ANOVA, P<0.001; Figure 7.4B; Table 7.4). Normalised CON torque was >60% larger than all other conditions at all measured time points after 25 ms. Remarkably, after 125 ms explosive voluntary CON torque equalled MVT, and had exceeded MVT by 150 ms, being 119% MVT. Normalised torque was similar in the ISO101, ISO155, and ECC conditions during the initial 75 ms of these explosive contractions, but during the later stages of contraction ISO155 was greater than ECC (75-150 ms), and ISO101 (125-150 ms).

Figure 7.4. Absolute (A) and normalised (B) torque for 150 ms after torque onset during explosive voluntary knee extensions in four conditions: isometric at knee joint angles of 101° and 155° (ISO101 and ISO155, respectively); concentric (CON); and eccentric (ECC). CON and ECC conditions were completed at a constant 2000°.s⁻², and torque was corrected for the acceleration and weight of the shank. Normalised torque is expressed as a percentage of maximal voluntary torque (MVT) at the relevant joint angle and angular velocity. Data are means ± SD on highest and lowest data points (n = 14).
Table 7.4. Normalised torque at 25 ms intervals from torque onset during explosive voluntary knee extensions in four conditions: isometric at knee joint angles of 101° and 155° knee angle (ISO101 and ISO155, respectively); concentric (CON); and eccentric (ECC). Data are means ± SD (n = 14). Paired differences are denoted by capital (P<0.01) or lower case (P<0.05) letters; A (> ISO101 and ISO155), B (> all other conditions) C (> ECC), or D (> ISO101 and ECC).

<table>
<thead>
<tr>
<th>Time (ms)</th>
<th>ISO101</th>
<th>ISO155</th>
<th>CON</th>
<th>ECC</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>2 ± 1</td>
<td>2 ± 1</td>
<td>3 ± 1&lt;sup&gt;A&lt;/sup&gt;</td>
<td>4 ± 2&lt;sup&gt;A&lt;/sup&gt;</td>
</tr>
<tr>
<td>50</td>
<td>12 ± 5</td>
<td>11 ± 4</td>
<td>23 ± 6&lt;sup&gt;B&lt;/sup&gt;</td>
<td>11 ± 4</td>
</tr>
<tr>
<td>75</td>
<td>31 ± 9&lt;sup&gt;C&lt;/sup&gt;</td>
<td>29 ± 9&lt;sup&gt;C&lt;/sup&gt;</td>
<td>54 ± 8&lt;sup&gt;B&lt;/sup&gt;</td>
<td>24 ± 7</td>
</tr>
<tr>
<td>100</td>
<td>46 ± 11</td>
<td>50 ± 12&lt;sup&gt;C&lt;/sup&gt;</td>
<td>79 ± 10&lt;sup&gt;B&lt;/sup&gt;</td>
<td>40 ± 10</td>
</tr>
<tr>
<td>125</td>
<td>58 ± 12</td>
<td>65 ± 12&lt;sup&gt;D&lt;/sup&gt;</td>
<td>101 ± 13&lt;sup&gt;B&lt;/sup&gt;</td>
<td>55 ± 10</td>
</tr>
<tr>
<td>150</td>
<td>67 ± 11</td>
<td>74 ± 10&lt;sup&gt;d&lt;/sup&gt;</td>
<td>119 ± 20&lt;sup&gt;B&lt;/sup&gt;</td>
<td>64 ± 9</td>
</tr>
</tbody>
</table>

MVT, maximal voluntary torque as a function of knee angle and angular velocity

Normalised ISO101 torque correlated with normalised ISO155 torque at all measured time points from torque onset (r = 0.56-0.79; 0.001<P<0.038). Subsequently, normalised torque data were collapsed across the two isometric conditions in order to assess bivariate relationships between the different types of contraction. Normalised CON torque was not correlated to normalised ECC or isometric torque at any time point from torque onset, and normalised ECC torque was only moderately correlated to isometric normalised torque at the 75 and 100 ms time points (r = 0.56-0.61; 0.02<P<0.036). The individual patterns of explosive voluntary performance across the three types of contraction are shown for one time point (75 ms) in Figure 7.5. The best and worst performing individuals varied across the types of contraction. Figure 7.5 also demonstrates that all participants performed more effective concentric than isometric or eccentric explosive contractions, and all but two participants (black and white squares) performed better isometrically than eccentrically.
Figure 7.5. Torque at 75 ms from torque onset normalised to maximal voluntary torque (MVT) at the same joint angle and angular velocity, for explosive voluntary knee extensions performed during three different types of contractions: concentric (CON); isometric (ISO); and eccentric (ECC). Data from each individual participant are plotted as a unique symbol (n = 14). Isometric values are the mean of two isometric conditions performed at different knee angles (101 and 155°).
Absolute voluntary/evoked torque at 50 ms after torque onset was dependent upon the contractile condition (ANOVA, both P<0.001). Paired comparisons revealed that voluntary/evoked torque in CON (77 ± 17%) was substantially greater than all other conditions (P<0.001; Figure 7.6); ISO101 (46 ± 14%) tended to be greater than ISO155 (36 ± 13%; P = 0.054), and both isometric conditions were greater than ECC (23 ± 9%; P ≤ 0.002). These results were identical when voluntary and evoked torques were both first normalised to MVT prior to calculating the voluntary/evoked percentage, which controlled for any discrepancy in joint kinematics at torque onset between voluntary and evoked conditions.

Figure 7.6. Absolute voluntary torque at 50 ms after torque onset as a percentage of absolute evoked torque at the same time point (voluntary/evoked), during explosive knee extensions in four conditions: isometric at knee joint angles of 101º and 155º (ISO101 and ISO155, respectively); concentric (CON); and eccentric (ECC). Data are means ± SD (n = 14). Paired differences are denoted by capital letters (P<0.01); B (> all other conditions), C (> ECC).
There was also a condition effect on the agonist normalised EMG during each 50-ms time window (0-50, 50-100, 100-150 ms) and over the whole 0-150 ms (ANOVA, P<0.001). Over the whole 0-150 ms agonist normalised EMG was 10.1 ± 1.7 (CON), 9.0 ± 1.3 (ISO101), 7.3 ± 1.3 (ISO155), and 4.7 ± 1.5 (ECC) % M\text{max}, and all conditions were significantly different from each other (Paired t-tests, P<0.032). Paired comparisons, for the first 50 ms time window were similar to those for voluntary/evoked torque at 50 ms, where agonist normalised EMG differed between all of the conditions and was greatest in the CON followed by ISO101, ISO155, and ECC (P<0.05; Figure 7.7). Paired differences between conditions during the 50-100 and 100-150 ms time windows were less pronounced, but agonist normalised EMG remained greatest in CON and ISO101 and lowest in ECC.

**Figure 7.7.** Agonist EMG over 0-50 (dark grey bars), 50-100 (light grey bars), and 100-150 ms (white bars) from EMG onset during explosive voluntary knee extensions in four conditions: isometric at a 101 and 155° knee angle (ISO101 and ISO155, respectively); concentric (CON); and eccentric (ECC). Agonist EMG is an average of the three superficial quadriceps muscles once normalised to maximal M-wave (M\text{max}). Data are means ± SD (n = 14). Paired differences for each EMG time window are denoted by capital (P<0.01) or lower case (P<0.05) letters; B (> all other conditions), C (> ECC), E (> ISO155 and ECC).

### 7.3.3 Evoked Parameters

As expected given the differential joint kinematics in each condition, absolute evoked torque at 25, 50 and 75 ms after torque onset was affected by condition (ANOVA, P<0.001; Figure 7.8), with evoked ECC and ISO101 torque greater than ISO155 and CON at all measured time points. Evoked torque in ISO101 and ISO155 normalised to
evoked peak torque in the same condition was similar over the first 50 ms, but greater in ISO155 at 75 (+5%; Paired t-test, P = 0.004) and 100 ms (+14%; Paired t-test, P<0.001) after torque onset (Figure 7.9). Despite greater peak torque in ISO101, time-to-peak torque and half relaxation time were shorter in ISO155 (Table 7.5).

**Figure 7.8.** Absolute torque recorded during evoked explosive voluntary knee extensions (supramaximal octet, 8 pulses at 300 Hz) in four conditions; isometric at knee joint angles of 101º and 155º (ISO101 and ISO155, respectively), concentric (CON), and eccentric (ECC). CON and ECC conditions were completed at a constant 2000°.s⁻², and torque was corrected for the acceleration and weight of the shank. Data are means ± SD (n = 14).

**Figure 7.9.** Normalised torque during evoked isometric knee extensions (supramaximal octet, 8 pulses at 300 Hz) at a 101° (ISO101) and 155° (ISO155) knee angle, expressed as a percentage of peak torque (PT) during the same contraction. Data are means ± SD (n = 14). Paired differences are denoted by *(P<0.05) or *** (P<0.001).
Table 7.5. Torque parameters recorded during the supramaximal evoked isometric knee extensions completed at a knee angle of 101º (ISO101) and 155º (ISO155). Data are means ± SD (n = 14). The P-value denotes differences between the two conditions.

<table>
<thead>
<tr>
<th></th>
<th>ISO101</th>
<th>ISO155</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak torque (Nm)</td>
<td>148 ± 25</td>
<td>98 ± 19</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time-to-peak torque (ms)</td>
<td>137 ± 9</td>
<td>112 ± 13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Half relaxation time (ms)</td>
<td>208 ± 21</td>
<td>174 ± 12</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

7.4 Discussion

The results of the current study provide novel evidence that the ability of humans to utilise the available torque producing capacity of a muscle in an explosive situation is influenced by the type of contraction. Whether expressed relative to the available MVT or the maximum capacity for RTD during evoked contractions explosive voluntary performance was clearly superior during concentric than isometric or eccentric actions, and this was consistent across a group of participants with a wide spectrum of explosive performance abilities. The proportion of MVT expressed during explosive concentric efforts was >60% larger than for isometric or eccentric conditions after the first 25 ms of the contraction. Furthermore, participants concentrically achieved 77% of their evoked torque after 50 ms, compared to 36-46% isometrically and 23% eccentrically. This greater concentric ability to utilise the available contractile capacity of the muscle indicates enhanced agonist activation that was supported by the higher EMG amplitude throughout the explosive contraction.

7.4.1 Effects of Contraction Type

The absolute voluntary and evoked torque-time curves appear to conform to the torque – angle – angular velocity relationship, where torque during different phases of the contraction was greatest in those conditions where angular velocity was low or negative, and knee angle was near $\theta_{opt}$. Overall torque development was highest for ECC during the evoked contractions, but highest for ISO101 during the volitional contractions; this discrepancy is likely to reflect the differences typically observed
between the torque/force – velocity relationships measured \textit{in-vitro} and voluntarily \textit{in-vivo} (i.e. eccentric to isometric torque/force for the same muscle length is normally $>1.4$ \textit{in-vitro} and $0.9-1.1$ \textit{in-vivo} (Pain, Forrester 2009, Webber, Kriellaars 1997, Dudley et al. 1990). Clearly the absolute voluntary and evoked torque-time curves are primarily determined by the joint kinematics (angle and angular velocity) of each condition, and consequently provide limited information for comparing explosive torque capabilities between different types of contraction.

When normalised to MVT at the same joint angle and angular velocity there was a clear effect of the type of contraction on explosive voluntary torque production; such that from 25 ms normalised concentric torque was consistently $>60\%$ larger than during isometric or eccentric conditions. In fact, MVT was achieved after only 125 ms in CON, whilst torque in the other conditions did not exceed 73\% of MVT even after 150 ms. Previous studies have reported that it takes $>300$ ms to achieve MVT in explosive isometric contractions performed from rest (Aagaard et al. 2002a, Thorstensson et al. 1976), and it is likely that this would have been the case in both the isometric and ECC conditions of the current study, had it been possible to measure torque beyond 150 ms. However, our results provide unique evidence that during explosive concentric contractions MVT can be achieved in $<125$ ms.

The greater concentric ability to utilise the available torque generating capacity was confirmed by the proportion of evoked torque achieved after 50 ms (CON, 77\%; ISO101, 46\%; ISO155 36\%; ECC, 23\%). As these values are relative to the maximal involuntary capacity for explosive torque production in the same contractile conditions they also imply substantial differences in neural drive to the agonist muscle. The greater agonist normalised EMG over the first 50 ms, as well as over the whole 150 ms from EMG onset, for CON supports this notion. The mechanistic explanation for this effect requires further investigation, but may be associated with neural inhibition during the isometric and ECC conditions that prevents full utilisation of the high, and potentially harmful, rates of loading available in these contractions. Moreover, the condition effects on agonist activation we have observed occurred within the first 50 ms of crank arm acceleration, which is considered to be the minimum latency period for a reflex response to mechanical perturbation (Zhou et al. 1995). Therefore, even though the participants were given the same instruction to push “fast and hard” during each
condition, our results support earlier evidence that the neural strategy employed at the start of the muscle contraction is pre-defined by the central nervous system according to the type of contraction (Grabiner, Owings 2002).

The more effective neural strategy in CON appears to explain why this condition was considerably more conducive to explosive performance than any other condition. MVT was also exceeded by up to 19% in the voluntary CON condition, suggesting that the greatest peak torque response in maximum voluntary concentric contractions is achieved when the focus is on producing explosive, rather than sustained maximal torque. This was an unexpected finding that was not replicated in any of the other conditions, and appears to be a consequence of the more effective neural strategy observed in the CON condition.

Whilst this is the first study to compare agonist activation during different types of explosive contractions, previous studies have assessed agonist activation at MVT and reported greater activation in concentric than eccentric contractions (Pain, Forrester 2009, Aagaard et al. 2000, Seger, Thorstensson 1994, Westing, Cresswell & Thorstensson 1991, Babault et al. 2001), and in isometric than dynamic conditions (Forrester et al. 2010; In Press, Babault et al. 2001). Any differences in agonist activation at MVT between contraction types in this study could clearly have influenced the comparison of explosive voluntary torques when normalised to MVT. This may explain the marginal differences in normalised explosive torque between ECC and the isometric conditions (particularly ISO101), despite distinct levels of agonist activation indicated by both voluntary/evoked torque and EMG.

The greater capacity for explosive performance during CON, rather than ECC or isometric, was consistent for all the participants despite their heterogeneity, and was not influenced by an individual’s level of explosive performance. Explosive performance during the different types of contractions was predominantly unrelated, and this lack of commonality might suggest a specificity of explosive performance according to the type of contraction.
7.4.2 Effects of Joint Angle

The comparison of explosive performance for different types of contraction may have been confounded by the influence of joint angle, which could not be matched at repeated time points during the CON, ECC and isometric contractions. This possibility was assessed by measuring isometric RTD at two joint angles. Absolute explosive voluntary and evoked torque-time curves for IS0101 and ISO155 conformed to the MVT-angle relationship, where torque at all measured time points from torque onset was greater in ISO101 (nearer $\theta_{opt}$). However, when normalised to MVT at the same knee angle, voluntary torque was similar in ISO101 and ISO155 during the initial phase of the contraction (first 100 ms), but greater in ISO155 beyond 100 ms. These results suggest that differences in joint angle did not confound comparisons between the type of contraction in the first 100 ms, but may have contributed to greater normalised torque in the later phase of CON compared to ECC, when the knee was accelerating into more extended (CON) or flexed (ECC) positions.

The improved capacity for normalised voluntary torque production in ISO155 does not appear to be due to agonist activation, as agonist normalised EMG in the 100-150 ms time window, as well as over the whole 150 ms from EMG onset, was 21-23% greater in ISO101. Earlier studies have also reported reduced agonist activation during voluntary contractions at more extended knee angles (Aagaard et al. 2000, Forrester, Pain 2010, Croce, Miller 2006, Becker, Awiszus 2001, Kubo et al. 2004) and this effect is thought to be a neural mechanism that protects the knee joint near full extension, where loading of the anterior cruciate ligament is greatest (Senter, Hame 2006).

In a similar pattern to that observed in the normalised voluntary torque-time curves, normalised evoked torque (relative to peak evoked torque) was comparable for the two isometric conditions in the early phase of the contraction, but greater in ISO155 in the later phase (after 50 ms). This was associated with a shorter time to peak torque in ISO155, suggesting a mechanical explanation for improved normalised explosive torque in the extended position. Our results are consistent with earlier in-vitro studies that found shorter muscle lengths to have a faster time to peak tension (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980, Rack, Westbury 1969), and a steeper normalised tension-time curve during the later phase of the contraction (Rassier,
MacIntosh 2002, Wallinga-de Jonge et al. 1980). However, this is the first study to measure a similar effect in-vivo during both explosive voluntary and evoked contractions. The faster time to peak force at shorter muscle lengths has been attributed to: lower Ca$^{2+}$ release or a reduced affinity of troponin C for Ca$^{2+}$ (Rassier, MacIntosh 2002) resulting in less efficient excitation-contraction coupling (Rack, Westerby 1969); and/or overlapping of the actin filaments, which would interfere with cross-bridge formation (Gordon, Huxley & Julian 1966). Nevertheless, it is unclear why a faster time to peak torque at shorter muscle lengths would only affect the normalised torque-time curve during the later stages of the contraction.

In conclusion, the type of contraction influences the ability to utilise the muscles torque producing capacity explosively, with concentric contractions being considerably more conducive to explosive performance than any other type of contraction, due to more effective neural activation. Finally, a faster time to peak torque at more extended knee angles appears to increase the slope of the normalised voluntary and evoked torque-time curves at high, but not low torque levels.
CHAPTER 8

General Discussion
8.1 Implications for Athletic Performance

The capacity for RFD is typically considered important for explosive athletic performance, but up until now, the association between RFD and athletic performance has been poorly documented. The findings of Chapters 3 and 4 support an association between athletic performance and RFD, where explosive strength was: (i) greater in explosive power athletes compared to untrained individuals (Chapter 3), and (ii) positively related to both sprint and CMJ performance (Chapter 4). Furthermore, the results of Chapters 3 and 4 suggest that the nature of the association between athletic performance and RFD appears to depend on three factors: (i) the level of agonist activation in the initial phase of the contraction; (ii) the maximum strength of the individual; and (iii) the force-time characteristics of the explosive athletic activity being analysed.

As expected, explosive power athletes had greater maximum strength in the knee extensors than untrained individuals (Chapter 3), and it was not surprising therefore that they also displayed greater absolute RFD, as all other factors being equal absolute RFD depends on MVF. However, the athletes were also able to achieve a greater proportion of their maximum strength within the first 50 ms of explosive contractions. This effect was explained by superior agonist activation in the athletes during the initial phase of the contraction (Chapter 3). This provided good evidence that the ability to achieve high levels of agonist activation in the initial phase of the contraction, and thus utilise high proportions of the available MVF in this time, may have a direct influence on performance during functional athletic performance. This conclusion was further supported by the strong relationship observed between sprint performance and the proportion of MVF achieved during the first 150 ms of explosive isometric squats (Chapter 4), despite there being no relationship between sprint performance and MVF. In contrast explosive isometric squat force during the initial phase of the contraction was not related to CMJ performance. Instead CMJ performance was related to absolute explosive voluntary force after the initial phase of the contraction (Chapter 4). Furthermore, considering that absolute explosive voluntary force after the initial phase of the contraction appears to be predominantly determined by maximal force capacity of the muscle (Chapter 3 and Andersen, Aagaard (2006)), it is not surprising that CMJ was also related to MVF. The contrasting influence that absolute and normalised
explosive force appears to have on sprint and CMJ performance is likely to be explained by the differences in the force-time characteristics of these athletic activities (force production time of sprinting, <300 ms vs. CMJ, >300 ms). Therefore, collectively the results of Chapters 3 and 4 suggest that: (i) the level of agonist activation and thus proportion of MVF achieved in the initial phase of an explosive contraction, is the main limiting factor of athletic activities with a short period for force production; whilst (ii) the capacity for maximal force production is the main limiting factor of athletic activities with longer periods of force production.

An interesting question following the conclusions of Chapters 3 and 4, is to what extent the main factors influencing explosive athletic performance (specifically, agonist activation in short periods of explosive force production and maximum strength over longer periods of force production) are innate or can be trained. An insight into the latter is provided in Chapters 5 and 6 and will be discussed below.

8.2 Influence of Strength Training

One of the most novel findings of the current thesis was the differential effects that short-term training for maximum strength vs. short-term training for explosive strength appears to have on RFD. Training for maximum strength improved this aspect of force production, and therefore theoretically would be expected to improve explosive force production, particularly for later points of the rising force-time curve. However, RFD over all time periods from force onset remained unchanged after training for maximum strength. In contrast, training for purely explosive strength increased MVF, absolute explosive force production at all measured time points, and the proportion of MVF achieved within the initial phase of the contraction. The explanation for the differential effects of short-term maximum and explosive strength training appear to be as follows:

(i) The gains in maximum strength with this training modality were due to neural adaptations (specifically increased agonist activation and decreased co-activation), that were highly specific to the peak of the force-time curve, and therefore did not benefit RFD over any time period from force onset.
(ii) Training for maximum strength did not appear to induce peripheral changes (as denoted by the consistent force-agonist activation relationship and twitch peak force), that would potentially have benefited RFD.

(iii) Training for explosive strength improved agonist activation in the very initial phase (0-50 ms) of the contraction, which caused the increase in normalised RFD in the same time period.

(iv) Training for explosive strength also induced peripheral adaptations (denoted by increased evoked octet force and a tendency for increased muscle thickness) that would contribute to the concomitant increase in MVF and absolute explosive voluntary force, particularly at later time points in the contraction.

Chapter 6 therefore shows that it is possible to improve the ability to utilise the available force capacity of the muscle during the initial phase of the contraction within just 4 weeks, but only when training for explosive strength. Furthermore, whilst maximum strength gains after 4 weeks of explosive strength training (+11%) are smaller than those observed following maximum strength training (+20%; Chapter 5), the former appears to induce peripheral adaptations that benefit absolute explosive force over longer periods of force production. This last finding is surprising, given the short time course of the training, and small loading volume of the explosive compared to the maximum strength training. It is possible that the greater mechanical strain rate, typically associated with explosive contractions, may provide a more effective stimulus for short-term peripheral adaptations (as discussed in Chapter 6), but this requires further investigation. It should however, be noted that the measurements employed in the maximum strength training study were more indirect measures of peripheral adaptations, and may not have been as sensitive as those employed in the explosive strength training study.

The natural progression of this work would be to investigate the differential effects of these different training modalities over longer and shorter periods of time. Nevertheless, the results of this thesis suggest that explosive strength training may provide greater functional benefits (e.g., RFD over all time period of the rising force-time curve) in a shorter period of time, and potentially, without the same level of fatigue and discomfort typically associated with the high loading volumes of maximum strength training.
8.3 Intrinsic Contractile Properties

The intrinsic contractile properties of the muscle did not appear to contribute to greater voluntary RFD of explosive power athletes compared to untrained individuals (Chapter 3). Whilst explosive force production during twitch and 100 Hz tetanic contractions was greater in the explosive power athletes than untrained individuals, these differences were negated when force was normalised to peak twitch force or MVF, for twitch and tetanic contractions, respectively. Theoretically, muscle fibres displaying a greater proportion of type II MHC isoforms, would be expected to display faster normalised contractile properties (normalised for influence of maximum strength; (Harridge et al. 1996)). Therefore, considering that explosive power athletes are thought to have a higher proportion of type II MHC than untrained individuals (Mero et al. 1981), it is surprising that there were no differences between the normalised contractile properties of these groups. If this were the case for the athletes in Chapter 3 it might suggest that the evoked force response of whole-muscle in vivo is determined predominantly by maximum strength, and not necessarily by its fibre type composition. Whilst the relationship between histochemical or molecular characteristics and the muscle’s intrinsic contractile properties has been well documented for skinned muscle fibres and single motor units (Bottinelli et al. 1999, Harridge et al. 1996, Burke et al. 1973, Garnett et al. 1979, Larsson, Moss 1993), the evidence of this association in human whole-muscle in vivo is equivocal (Harridge et al. 1996).

Chapter 6 provides further support for a strong association between maximum strength and the evoked force response of a muscle. Previous studies have reported a shift in myosin isoform expression from type IIX to type IIA (fast-to-slow) with strength training (Andersen et al. 2010, Jurimae et al. 1996, Staron et al. 1994). If this effect had occurred following the explosive strength training in Chapter 6, it would have been expected to negatively influence explosive force production during evoked contractions. On the contrary, 4 weeks of explosive strength training increased evoked octet force production. Furthermore, this increase was directly proportional to the improvements in MVF. Given the consistent evidence of Chapters 3 and 6, the evoked contractile properties of human muscle in-vivo do not appear to explain the discrepancies in RFD
between different athletic groups or following training, beyond that already explained by maximum strength.

8.4 Muscle-Tendon Unit Stiffness

Explosive strength training appears to increase MTU stiffness at high force levels within just 4 weeks (Chapter 6). MTU stiffness was not measured in the maximum strength training study (Chapter 5); however, the earliest observations of increased MTU stiffness when training for maximal force production occurred after 9-10 weeks (Seynnes et al. 2009, Kubo et al. 2010). As discussed in Chapter 6, it is unclear why a stiffer response to loading would be observed in a shorter period of time with explosive compared to maximum strength training, but it may be due to the greater mechanical strain rate expected to occur when training with purely explosive contractions. A higher strain rate predisposes the system to greater mechanical stress and strain energy for the same mechanical strain, and this may provide a greater stimulus for adaptation that results in a stiffer MTU, although this requires further investigation. Nevertheless, given the proposed health benefits of MTU stiffness at high force levels (protecting soft tissue from strain injury; (Buchanan, Marsh 2002)), sport and exercise health practitioners should consider implementing an explosive component into their clients’ training programmes.

A stiffer mechanical response during an explosive contraction is thought to benefit RFD (Wilkie 1949). However, the increase in MTU stiffness at high force levels in Chapter 6 did not appear to explain the improved capacity of the MTU for RFD (e.g., increased absolute evoked octet RFD). This is because MTU stiffness increased for forces between 50-90% of MVF, whilst evoked octet force was recorded over lower force levels (up to ~60% of MVF), with MTU stiffness showing a tendency to decrease throughout most of this range (10-50%). Whilst previous studies have observed a strong correlation between MTU stiffness and RFD (Bojesen-Moller et al. 2005, Wilson, Murphy & Pryor 1994, Walshe, Wilson & Murphy 1996, Ditroilo, Watsford & De Vito 2010), these studies did not normalise either parameter to maximum strength, which appears to have a common association with both MTU stiffness (Wilson, Murphy & Pryor 1994, Arampatzis et al. 2007a) and RFD (Andersen, Aagaard 2006). Future work
should normalise both voluntary and evoked RFD measurements to maximum strength and consider MTU stiffness over a relevant range of forces to better establish the interaction between these variables.

The direct influence of athletic performance on MTU stiffness was not measured in this series of studies. However, a stiffer MTU should theoretically benefit evoked RFD normalised to MVF, but there were no observed differences between the normalised contractile properties of explosive power athletes and untrained individuals (Chapter 3). Therefore, the influence of athletic performance on evoked RFD may depend more on discrepancies in maximum strength than MTU stiffness, although this requires further investigation.

### 8.5 Type of Contraction

Chapter 7 is the first study to show that the human ability to utilise the available torque capacity of skeletal muscle in an explosive effort is influenced by the type of contraction. Specifically, the proportion of MVT expressed during explosive concentric efforts was >60% larger than for isometric or eccentric conditions after the first 25 ms of the contraction. Furthermore, participants concentrically achieved 77% of their evoked torque after 50 ms, compared to 36-46% isometrically and 23% eccentrically. This latter finding also suggests a more effective activation of the agonist muscles during the concentric condition, which was supported by greater EMG normalised to $M_{\text{max}}$, not only during the initial 50 ms, but throughout the entire concentric condition (i.e., 0-150 ms). Therefore concentric contractions are more conducive to explosive performance because of a more effective neural strategy in this situation. This effect does not appear to be influenced by athletic/training background, as the results were consistent across a group of participants with a wide spectrum of explosive performance abilities.

The influence of contraction type on agonist activation during explosive contractions (Chapter 7) was similar to reports that found agonist activation at MVT was highest for concentric contractions (Pain, Forrester 2009, Aagaard et al. 2000, Seger, Thorstensson 1994, Westing, Cresswell & Thorstensson 1991, Babault et al. 2001), and greater in
isometric compared to eccentric contractions (Forrester et al. 2010; In Press, Babault et al. 2001). Therefore, normalising explosive torque to angle and angular velocity specific MVT may in theory control for the expected discrepancies in agonist activation between the different types of contraction. This might explain the marginal differences in normalised explosive torque between the isometric (particularly at a 101º knee angle) and eccentric conditions, despite distinct levels of agonist activation. In contrast, normalised explosive torque in the concentric condition was considerably greater than that in the other conditions, providing indirect evidence that agonist activation in this situation was disproportionately greater than that typically expected in concentric contractions at MVT. This could also explain why concentric explosive torque exceeded 100% of MVT. From this data it appears that the greatest peak torque response in maximum voluntary concentric contractions may be achieved when the focus is on producing explosive, rather than sustained maximal torque. The reciprocal eccentric-concentric protocol used for determining dynamic MVT, is thought to give the participants time to achieve and sustain maximal activation, and thus MVT, throughout the entire range of motion (Yeadon, King & Wilson 2006, Pain, Forrester 2009, King, Yeadon 2002). It is possible that inhibition during the preceding eccentric phase may have reduced mean activation throughout the whole reciprocal effort (thus confounding concentric MVT), although evidence showing considerably greater concentric than eccentric activation during a similar reciprocal protocol would suggest that this was not the case (Forrester, Pain 2010). Further research is required to establish the optimal method for measuring dynamic MVT.

8.6 Muscle Length / Joint Angle

The influence of muscle length on the entire force-time curve has only been previously assessed in animal models, that showed a faster time to peak force at shorter muscle lengths resulting in a steeper normalised force-time curve (normalised to peak force) at later phases of the contraction (Rassier, MacIntosh 2002, Wallinga-de Jonge et al. 1980). Chapter 7 is the first study to show a similar effect in humans in vivo during both explosive voluntary and evoked contractions. The mechanisms for this are unclear, and may be associated with disruption to the excitation-contraction coupling process at shorter muscle lengths, that influences cross-bridge formation and/or cycle rate at high
but not low levels of the rising force-time curve. It is also unclear how this effect may be influenced by athletic performance or training. Whilst the participants in Chapter 7 displayed a broad spectrum of explosive performance abilities, it has been shown previously that distinct athletic groups (specifically runners, cyclists, and untrained) display different fascicle lengths in the quadriceps, and thus for the same joint angle, are working over a different portion of their force-length relationship (Herzog, ter Keurs 1988). It is therefore possible that training for a specific athletic event may influence the effect of joint angle on the ability to utilise the available torque capacity of the muscle.

8.7 Future Directions

Possible suggestions for the future directions of this work are as follows:

1) Provide a comprehensive cross-sectional analysis (with a large cohort) of all determinants of RFD, including measurements of muscle fibre composition, to establish how all of these parameters interact to influence both voluntary and evoked RFD.

2) Investigate further the influence of contractile velocity on the capacity for explosive force production. This could be achieved by measuring explosive force at different constant velocities, and normalising to a dynamic MVT function.

3) Measure patterns of neural activation during different types of explosive contractions (i.e., isometric, concentric, and eccentric) and compare this to neural activation at MVT for the same contractile condition. This could be done by modelling EMG amplitude at MVT as a function of joint angle and angular velocity (Forrester, Pain 2010), and may help to provide a better understanding of the more effective neural strategy adopted in explosive concentric contractions.
4) Investigate the influence of strain rate on the acute stiffness response of the MTU, and provide a better understanding of the influence of strain rate on mechanical adaptations to training. This research could be further progressed by comparing the chronic mechanical adaptations of the MTU to different loading volumes, frequencies and strain rates (specifically, training for maximum and explosive strength over longer periods).

5) Investigate the differential effects of maximum and explosive strength training over a longer period of time (>4 weeks), and compare these effects in different populations, e.g., young and old. This research would have important implications for designing exercise programmes to improve sports performance and/or health.
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