# THE DEVELOPMENT AND ORIGIN OF MUSCLE FATIGUE AFTER HEXAGONAL-BARBELL DEADLIFT EXERCISE OF DIFFERENT LOADS AND VELOCITIES:

contributing to a knowledge-base for coaches to make evidence-informed decisions regarding athletic training programmes

by Jay Collison

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Centre for Sport and Exercise Science

School of Biological Sciences

University of Essex

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**Abstract**

Athletes perform bouts of resistance exercise to develop athletic performance and reduce the risk of injury. Fatigue in response to exercise can be associated with alterations to voluntary muscle activation or contractile function. Recent literature has proposed terms of performance fatigability and perceived fatigability to better describe the broad interpretation of fatigue during human performance. Accordingly, performance fatigability depends on the capabilities of the contractile machinery and nervous system to provide adequate activation signal to maintain the task.

The rationale for the experimental chapters in this thesis was established after conducting a review of the literature relating to fatigue and human performance (chapter 2). This review highlighted that studies of performance fatigability have generally focused on locomotor exercise such as running or cycling. However, very little work has examined the influence of resistance exercise on performance fatigability. Specifically, it was identified that performance fatigability during multi-joint resistance exercise performed with different loads until mechanical failure and modification of lifting tempo required further research. For these reasons, two experimental studies were conducted to address these gaps in knowledge.

The first study (chapter 3) examined the influence of an exhaustive bout of high-load and moderate-load hexagonal-barbell deadlift (HBD) resistance exercise on acute changes and recovery of contractile function and voluntary activation in resistance-trained males. The results indicate that both voluntary activation and contractile function were reduced after moderate-load HBD exercise, but not high-load exercise. Additionally, after a 24-h recovery period, both voluntary activation and contractile function were impaired. Interestingly, the partial recovery was due to a near return to before exercise values of contractile function and therefore the incomplete recovery was due to reduced voluntary activation.

In chapter four, the influence of a structured bout of volume load-equated HBD exercise with manipulation of lifting tempo on changes to contractile function and voluntary activation was examined. The main finding was that slow tempo and fast tempo HBD exercise resulted in similar reductions to both voluntary activation and contractile function. However, it is unknown if this remains the case when greater resistance exercise volumes are performed. Additionally, further research is required to understand the mechanisms of reduced voluntary activation during fast tempo resistance exercise. Finally, the findings from the experimental chapters are summarised and practical recommendations for the prescription of resistance exercise within athletic training programmes are presented.

In conclusion, because of the lowered reductions of contractile function and voluntary activation observed after high-load resistance exercise, it may be a preferable training modality for coaches to employ during the in-season period or during times of intense concurrent training. Additionally, changes to contractile function and voluntary activation associated with fast and slow tempo volume load-equated exercise is similar. However, more work is required to see if this remains the case when larger volumes of work are completed.

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# Chapter 1. Introduction

## 1. Thesis rationale

Resistance exercise is an effective stimulus to enhance skeletal muscle mass and strength (Folland and Williams, 2007). Maximum strength is defined as the maximum force or torque that can be exerted by skeletal muscles on an external object or resistance (Siff, 2000). Relatedly, high levels of muscular strength are important for athletic performance and are known to improve the ability to perform general and sports specific skills such as sprinting, jumping, change of direction ability, and throwing, while reducing the risk of injury (Suchomel et al., 2016). For these reasons, many athletes perform planned bouts of resistance exercise, which induce muscle fatigue.

Fatigue is described as an exercise-induced limitation of performance and measurable as a reduction in the ability of a muscle to exert force, or changes in voluntary muscle activation (Enoka and Duchateau, 2008, Gandevia, 2001). In an attempt to provide a unified taxonomy, recent literature (Enoka and Duchateau, 2016, Kluger et al., 2013) defines fatigue as a disabling symptom in which physical and cognitive function is limited by interactions between performance fatigability and perceived fatigability (Enoka and Duchateau, 2016). Performance fatigability depends on the contractile capabilities of the working muscle and capacity of the nervous system to provide adequate activation signal to maintain the task; while perceived fatigability describes the changes in subjective sensations that regulate the integrity of the exerciser (Enoka and Duchateau, 2016). Furthermore, performance fatigability is quantified as the objective rate of change in a criterion performance outcome over a discrete period of time during a fatiguing task (Enoka and Duchateau, 2016).

Muscle fatigability is influenced by the manipulation of resistance exercise variables such as load intensity (i.e. the amount of mass used per repetition for a given resistance exercise), volume, and lifting tempo. As well, ensuring that fatigue is appropriately managed is important for adaptations to training and competition

performance as well as reducing injury risk through excessive fatigue and overtraining (Hulin et al., 2016). Since the magnitude and type of fatigue will dictate the nature of subsequent training bouts, knowledge of the influence of resistance training variables on muscle fatigability may optimise the preparation of athletic training programmes.

Although the adaptive response to regular long-term resistance exercise is well researched (Folland and Williams, 2007), work on the influence of resistance exercise on muscle fatigability is scarce. Specific types of resistance exercise are prescribed to develop different physical qualities. For example, traditional recommendations are that the greatest and quickest increases in relative strength occur with training conducted at heavy loads above 80% of one-repetition maximum (1RM) with low (1-5) repetitions (Crewther et al., 2006a, Schoenfeld et al., 2016a). In contrast, resistance exercise conducted at lower loads (~60-80% 1RM) with more repetitions (8-12) has greater positive influence on muscle cross-sectional area (Wernbom et al., 2007, Schoenfeld et al., 2016a). These training schemes are commonly referred to as high-load (HL) and moderate-load (ML) resistance exercise (Schoenfeld et al., 2016a).

In addition to load, manipulation of lifting tempo may stimulate specific adaptations (Schoenfeld et al., 2015). For instance, when lifting submaximal loads <80-85% 1RM an individual can vary the concentric and eccentric tempo of the repetition for a given exercise. With this in mind, intentionally slowing repetition cadence reduces the momentum in a lift, thereby increasing tension on the working muscles (Westcott et al., 2001). It has been proposed that increasing mechanical tension throughout a lift could positively mediate intracellular anabolic signalling, promoting a greater hypertrophic response (Schoenfeld et al., 2015). In contrast, maximum effort resistance exercise in which the athlete moves the resistive load with maximum velocity has been shown to drive specific adaptations such as enhanced rate of force development, which is crucial for athletic performance (i.e. running, jumping, and throwing) (Newton et al., 1997).

To date, the majority of research exploring muscle fatigability has focused on single-joint exercise or locomotor exercise such as running or cycling (Goodall et al., 2015, Ross et al., 2010b, Thomas et al., 2015). Furthermore, as many athletes engage in concurrent training programmes, muscle fatigability and resistance exercise is of interest to strength and conditioning practitioners and sports coaches, particularly when research informs the preparation of athletic training programmes. Clearly, there are many resistance exercise variables that contribute to the design of athletic training programmes. The purpose of this thesis is to further knowledge of the influence of resistance exercise on voluntary muscle activation and contractile function.

## 2. Significance of the research

The research studies that comprise this thesis aim to further understanding on the influence of resistance exercise on voluntary muscle activation and contractile function. The findings are novel and build on previous research (Brandon et al., 2015, Howatson et al., 2015, McCaulley et al., 2009, Tran et al., 2006): first, by including an exercise with no available data; second, by examining the influence of performing resistance exercise until mechanical failure; and third, by examining the influence of manipulating concentric and eccentric lifting tempo during multi-joint exercise on voluntary muscle activation and contractile function. The rationale for the thesis and experimental chapters was developed by conducting a comprehensive review of the scientific literature relating to human performance and fatigue. The findings from the research studies contribute to a knowledge-base for coaches to make evidence-informed decisions regarding athletic training. These findings are important because performance may be optimised when research informs the preparation of athletic training programmes.

## 3. Thesis aims

This thesis aims to measure changes in voluntary muscle activation and contractile function during multi-joint high-load and moderate-load resistance exercise performed

until mechanical failure and resistance exercise performed with different time-under-tension by manipulating lifting tempo. To meet these aims the following two studies will be conducted:

Study 1: changes in voluntary muscle activation and contractile function after high-load low repetition (HL) and moderate-load high repetition (ML) resistance exercise performed until exhaustion. Specifically:

1. What are the changes in maximal voluntary contraction (MVC) torque, potentiated twitch torque (Pt), and voluntary activation (VA) after resistance exercise performed at 90 and 75% of 3 repetitions maximum to mechanical failure?
2. What is the 24-h recovery from each protocol?

Study 2: changes in voluntary muscle activation and contractile function during multi-joint resistance exercise performed with fast and slow lifting tempos. Specifically:

1. What are the changes in MVC, Pt, and VA after volume-load equated resistance exercise performed with fast and slow lifting tempos?

**4. Thesis structure**

The current chapter, along with the review of literature in chapter 2 form the theoretical basis and rationale for this thesis. Next, two experimental studies are presented designed to: (i) examine the influence of load and rest period modification on voluntary activation and contractile function and recovery (chapter 3) and (ii) examine the influence of time-under-tension on voluntary activation and contractile function (chapter 4). Finally, chapter 5 provides an integrative summary of the main findings from the two studies and recommendations for the prescription of resistance exercise for athletic training programmes.

# Chapter 2. A review of fatigue and human performance

## 1. Synopsis

The purpose of this review is to synthesise findings of studies which examine the influence of fatigue on human performance. Specifically, muscle fatigability during maximal and submaximal contractions at a single joint are initially discussed. Next, fatigability during locomotor exercise such as running and cycling involving a relatively large muscle mass is discussed. Finally, the influence of resistance exercise on muscle fatigability is considered. A secondary aim of this review is to establish the rationale for the methodological considerations and approach to the thesis.

## 2. Muscle fatigability

To understand how muscular force or torque can decrease with fatigue, it is important to understand the processes that contribute to voluntary muscle contractions. Briefly, the central nervous system (CNS) recruits individual motor units for the desired movement and after command from supra-cortical structures, descending drive from motor cortical structures activates lower motor neurons in the spinal cord which subsequently carries action potentials to the neuromuscular junction. Next, neuromuscular transmission results in action potential generation and propagation on each muscle fibre and membrane resulting in cross-bridge cycling. With this in mind, the causes of fatigue can stem from a decrease in neural activation of muscle and/or biochemical changes at or distal to the neuromuscular junction that can cause an attenuated contractile response to neural input (Bigland-Ritchie et al., 1978).

### 2.1 Mechanisms related to alterations in contractile function

Alterations to contractile function (i.e. changes occurring within the muscle cell itself) may arise from adjustments in cross-bridge cycling inhibiting muscular force production or excitation-contraction coupling (Allen et al., 2008). Several metabolic changes occur within muscles that precede loss of mechanical performance and each has the potential to play a role in loss of muscle force or torque. The following sub-

sections will briefly outline the primary mechanisms associated with impairment of contractile function.

### *2.1.1 $Na^+$ and $K^+$*

Following neuromuscular transmission, an action potential propagates along the surface membrane of the muscle fibre (sarcolemma) triggering calcium ion ($Ca^{2+}$) release from the transverse-tubules. Calcium ions bind to troponin removing tropomyosin from the actin binding site and allowing myosin to bind with actin to generate the power stroke (Debold et al., 2016). Action potential transmission may be influenced by several factors including extracellular and intracellular $Na^+$ and $K^+$ concentrations (Allen et al., 2008). In resting skeletal muscle, the chemical gradients for $Na^+$ and $K^+$ are maintained within narrow limits by the $Na^+$-$K^+$ pump through active transport of $Na^+$ out of the muscle cell and $K^+$ into the cell. However, during intense muscle activity, repeated action potentials cause a net $K^+$ efflux and subsequent increase in $K^+$ in the extracellular spaces, particularly within the narrow transverse tubular space. The increase in extracellular $K^+$ concentrations is widely believed to impair action potential transmission due to ion disturbances over the sarcolemma and a possible block in its propagation into the transverse tubules (Allen et al., 2008).

### *2.1.2 Cellular acidosis*

The understanding of muscle fatigue has moved on since it was thought that the accumulation of lactate and associated increase in hydrogen ions ($H^+$) was a primary culprit in the development of fatigue. Indeed, evidence that lactate production metabolically consumes an $H^+$ and directly opposes cellular acidosis is convincing, while the source of proton release is associated with glycolysis and ATP hydrolysis (Robergs et al., 2004). There is also strong evidence that the direct depressant effect of acidosis is greatly reduced at physiological temperatures (Pate et al., 1995, Westerblad et al., 1997). Additionally, although acidosis may have a small negative

impact on muscle performance, these effects appear to have been overestimated and likely benefits such as maintenance of membrane excitability may have been disregarded (Nielsen et al., 2001, Pedersen et al., 2004). Accordingly, if acidosis is involved in skeletal muscle fatigue, the effect may be indirect. For instance, extracellular acidosis may stimulate group III/IV muscle afferents (free nerve endings stimulated by contraction-induced mechanical and chemical stimuli) where it has been suggested that the brain may use this feedback to produce a sensation of fatigue on which motor output is regulated (Noakes, 2012, Amann et al., 2008). For these reasons, from a muscle cell perspective, a greater focus on other metabolites such as inorganic phosphate has been presented.

*2.1.3 Inorganic phosphate*

The rapid degradation of phosphocreatine (PCr) during high-intensity exercise gives rise to elevated intracellular inorganic phosphate ($P_i$) concentrations. An increase in $P_i$ has been shown to strongly correlate with a decline in muscle performance in a number of *in vitro* investigations (Cady et al., 1989, Pate et al., 1998, Potma et al., 1995). The primary mechanism by which $P_i$ may exert its effects on muscle fatigability is by inhibiting $Ca^{2+}$ release from the sarcoplasmic reticulum (Glaister, 2005), but may also include reduced force production by direct action on cross-bridge cycling and myofibrillar calcium sensitivity (Allen et al., 2008). However, these observations were made at physiologically low temperatures (10-15˚C) and there is evidence that the depressive effect of $P_i$ diminishes as the temperature is increased (much like acidosis) (Debold et al., 2004). Nevertheless, observations made in intact fibres at temperatures closer to normal physiological values (~25˚C) still report marked depressive effects on muscle performance (Bruton et al., 1998). These findings suggest that further work related to the temperature dependent effects of metabolites on fatigue is needed and raises the possibility that different mechanisms might be involved (De Ruiter and De Haan, 2000).

*2.1.4 Reactive oxygen species*

There is a growing body of evidence that reveals reactive oxygen species (ROS) may contribute to muscle fatigability as these are produced at higher rates in active muscles (Arbogast and Reid, 2004). Interestingly, ROS scavengers can also slow fatigability in preparations at 37˚C (Khawli and Reid, 1994). Studies using isolated single muscle fibres and small muscle bundles at 37˚C reveal that the increases in ROS may contribute to fatigability by reducing $Ca^{2+}$ sensitivity (Moopanar and Allen, 2005, Reid, 2008). Additionally, observations at room temperature show that ROS may accelerate fatigability by reducing maximum $Ca^{2+}$ activated force (van der Poel and Stephenson, 2002) and SR $Ca^{2+}$ release (Reid, 2001). Taken together, there is convincing evidence that ROS contribute to muscle fatigability at physiological temperatures, however, the pathway by which ROS affects myofibrillar $Ca^{2+}$ sensitivity is limited as many studies demonstrate equivocal findings (Steinbacher and Eckl, 2015).

*2.1.5 Substrate depletion*

During prolonged, strenuous exercise muscle glycogen and blood glucose are key substrates for working muscles and performance is compromised when glycogen stores reach low levels (even when there is an abundance of alternative fuel sources) (Ortenblad et al., 2013). For instance, a strong relationship exists between muscle glycogen concentration and muscle fatigability during moderate-intensity, sustained (> 60-min) and high-intensity intermittent exercise (Bergstrom et al., 1967, Bangsbo et al., 1992). This relationship has been explained by compromised rates of ATP regeneration (Allen et al., 2008). However, the ‘energy deficiency theory’ has been questioned by both *in vitro* and *in vivo* observations that reveal strong associations between glycogen stores and muscle function, even after recovery periods where ATP levels would be restored (Bangsbo et al., 1992). More recently, some evidence suggests that glycogen, located within the myofibrils, may have a direct inhibitory

effect on SR $Ca^{2+}$ release affecting muscle contractility and fatigability (Ortenblad et al., 2013).

*2.1.6 Peripheral governor*

In addition to the abovementioned ideas that associate substrate depletion, accumulation of waste products, or wearing down of contractile function as contributors to fatigue, it has been proposed that individual skeletal muscles have the capacity to regulate their own activation to limit the rate of adenosine triphosphate (ATP) use at the cellular level (MacIntosh and Shahi, 2011). The peripheral governor is therefore a cellular regulated process and suggests that control of skeletal muscle contraction is not limited to the brain through motor unit recruitment and rate coding (MacIntosh et al., 2012).

Adenosine triphosphate (ATP) is the energy currency of living cells and its regulation is of particular importance as several processes including molecular motors, ion pumps, chemical signaling processes, and synthetic reactions rely on energy from ATP. The theoretical basis of the peripheral governor is formed from the idea that regulatory processes must be in place to not only replenish ATP as quickly as possible, but also regulate its use within the active muscle, where there is potential for severe disruption of homeostasis and metabolic catastrophe (MacIntosh and Shahi, 2011). With this in mind, the authors believe that each muscle cell can preserve ATP because the ATP level remains reasonably constant during exercise and does not usually fall more than 20-25% of resting values (Argov et al., 2000). This proposal is consistent with studies that demonstrate that neither glycogen depletion (Saltin and Karlsson, 1972) nor exhaustion of ATP (Baker et al., 1994) occurs at exercise termination at intensities above the critical power. As such, it is argued that the contractile response of a muscle is regulated within the muscle cell itself, decreasing ATP utilization when the ability to replenish it is challenged (Macintosh and Shahi, 2011).

The peripheral governor is proposed to operate by mechanisms that involve $Ca^{2+}$ handling. For example, there is strong evidence that decreased $Ca^{2+}$ release is a consequence of repetitive stimulation of a single muscle cell (Allen et al., 2008). This seems logical because regulating $Ca^{2+}$ release from the sarcoplasmic reticulum and intracellular $Ca^{2+}$ concentration dictates the rate of ATP use in the muscle cell via the enzymes $Ca^{2+}$ ATPase and myosin ATPase activity (Homsher, 1987, MacIntosh et al., 2012). The most likely mechanisms involved with attenuation of $Ca^{2+}$ release are: (i) muscle membrane depolarization, (ii) ryanodine receptor (RyR) inhibition, (iii) and decreased $Ca^{2+}$ availability (Macintosh and Shahi, 2011). As such, the peripheral governor is far removed from the notion of a central governor that theoretically preserves the integrity of muscle by inhibition of motor cortex drive. The authors acknowledge the importance of a central governor, which may interfere with pacing during exercise, but argue that the peripheral governor has the final say in limiting ATP use by muscles (Macintosh and Shahi, 2011).

Finally, the fact that exercise can be enhanced with psychological interventions such as motivational self-talk (Blanchfield et al., 2014b), music (Nakamura et al., 2010), placebo (Beedie et al., 2006), non-conscious visual cues (Blanchfield et al., 2014a), and competition (Viru et al., 2010) has been used to provide evidence against a cellular regulated process and muscle fatigue model of endurance performance (Smirmaul and Dantas, 2011). Nevertheless, MacIntosh and Shahi (2011) argue that highly motivated individuals who are familiar or trained with the task will continue the task until the muscles are no longer capable of achieving the target force, but agree that in cases of lower motivation or unfamiliarity with the task, performance is often limited by an unwillingness to continue.

## 2.2 Mechanisms related to alterations in voluntary muscle activation

More recently, a greater focus and culpability has been placed on neural processes that contribute to performance fatigability as well as cognitive and psychological factors that contribute to perceived fatigability in the development of fatigue (Enoka and Duchateau, 2016, Gandevia, 2001, Noakes, 2012, Marcora et al., 2009). A reduction in neural output or drive from the motor cortex to muscle arises as a decrease in voluntary muscle activation (VA) during exercise (Gandevia, 2001). That is, despite voluntary maximal effort the motor units are not driven to high enough firing rates enough to generate maximal voluntary force (Gandevia, 2001). A decrease in motor unit firing rate at the motorneuron pool is a likely mechanism for the observed reductions in VA during exercise, which may result from three distinct processes: (i) impairment in the motor cortex demonstrated through a decreased descending efferent drive or an increased perceived exertion; (ii) increased inhibition at the spinal level; or (iii) changes in the intrinsic motorneuron properties that reduce neuronal excitability at the synapse of the motor axon (Taylor and Gandevia, 2008, Enoka and Duchateau, 2016).

It is now well-established that disturbances to nervous system function and reductions in VA occur. For example Gandevia et al. (1996) found evidence after observing sustained 2-min isometric MVCs of the elbow flexors. The authors found that during the MVC, both motor point stimulation of the biceps brachii and magnetic cortical stimulation increased torque output (relative to voluntary torque) implying that the participants became progressively worse at activating the biceps brachii and cortical output became suboptimal. Additionally, the muscle was made ischaemic after the MVC to retain its metabolic state of stress resulting in an elevated discharge rate of the group III/IV muscle afferents. The authors reported dissociation between the changes in corticospinal excitability and cortical output (i.e. VA impairment). As well, VA remained incomplete until blood flow was restored (i.e. metaboreceptors stimuli

decreased) (Gandevia et al., 1996). The authors suggested that the observed reduction in VA was likely due to inadequate neural input upstream of the motor cortex (Gandevia et al., 1996).

One mechanism by which reduced VA may arise during exercise is via neural signalling from group III/IV muscle afferents that respond to contraction and/or stretch as well as metabolic changes within the working muscle (Amann, 2012, Noakes, 2012). It has been proposed that increased group III/IV muscle afferent feedback might progressively inhibit cortical output and tightly regulate exercise intensity to ensure that the metabolic milieu and thus 'peripheral fatigue' (i.e. within the muscle) does not exceed a certain level (Amann and Dempsey, 2008, Noakes, 2012). The 'critical threshold of peripheral fatigue' theory has been proposed by numerous authors that argue that the magnitude of contractile dysfunction incurred during whole-body exercise does not exceed an individual and task specific threshold (Amann and Dempsey, 2008, Gagnon et al., 2009). For instance, after severe pre-induced quadriceps fatigue, cycling performance during constant-workload (Gagnon et al., 2009) and time-protocol (TT) exercise (Amann and Dempsey, 2008) is reduced when compared to control protocols without prior pre-fatigue. However, although cortical output reduced, contractile dysfunction and perceived fatigability were similar between conditions (i.e. control). That is, although participants were significantly pre-fatigued before exercise they could not exceed their critical threshold of peripheral fatigue.

In addition to contributing to performance fatigability, further roles for group III/IV muscle afferents in limiting exercise performance have been explored in numerous studies relating to perceived fatigability (Amann, 2012, Amann and Dempsey, 2008, Mauger, 2013, Mauger et al., 2010, Noakes, 2012). For example, it is known that stimulation of group III/IV muscle afferents contributes to the acute muscle pain associated with high-intensity exercise (O'Connor and Cook, 1999). Moreover, pain is perceived by the brain to provide an individual with information regarding the relative

internal load on the body, which may be used to inform a conscious decision to adjust exercise performance such that the continuation of exercise is tolerable and does not become ‘unattractive’ or ‘painful’ (Amann et al., 2008, Mauger, 2013, Noakes, 2012). In support of this, after pharmacologically blocking afferent feedback and maintaining normal efferent function during an all-out 5 km cycle time-protocol (TT), cortical output was greater compared with a placebo (saline infusion) and control protocol (Amann et al., 2008). Additionally, during the experimental protocol, participants adopted a more aggressive pacing strategy and could exceed their critical threshold of peripheral fatigue. However, possibly due to missing feedback, performance was reduced during the second half of the TT despite continuous high cortical output. The authors concluded that blocking muscle afferents may release a ‘centrally mediated brake’ on central motor output, allowing greater fatigability (up to 44%) to occur (Amann et al., 2008). These findings were supported by Mauger et al. (2010) who found that after ingesting the analgesic acetaminophen, a higher power output for a given perceived pain and rating of perceived exertion (RPE) was achieved, resulting in a faster time to completion of a 16.1 km cycle time protocol.

The brain neurotransmitters dopamine, noradrenaline, and serotonin have also been shown to influence endurance performance at high ambient temperatures (Roelands et al., 2013). For example, high levels of dopamine activity in the brain are associated with increased exercise tolerance (Bridge et al., 2003) and performance in the heat (Roelands et al., 2012, Watson et al., 2005). Accordingly, as dopamine plays an important role in motivation, reward and attention, and memory it has been suggested that a higher power output and sustained metabolic heat production are achieved through increased drive and motivation (Roelands et al., 2008b). In contrast, manipulations of serotonin and noradrenaline appear to decrease exercise performance. For example, after ingesting a serotonin reuptake inhibitor, participants were unable to perform an ‘end spurt’ despite lower mid-protocol power outputs during

a 30-min cycle time protocol suggesting an absence of a reserve capacity or lack of motivation to increase power output (Roelands et al., 2009). Also, Piacentini et al. (2002) reported a trend towards decreased performance during a 90-min time trial at 18ºC after participants ingested a noradrenaline reuptake inhibitor (reboxetine). Additionally, a higher dose of the same drug decreased performance during a 30-min time protocol at both 18 and 30ºC (Roelands et al., 2008a). Taken together, it appears that an increase in brain dopamine concentrations, in contrast to serotonin and noradrenaline has beneficial effects on exercise performance through increased drive and motivation.

In a challenge to the central governor theory (St Clair Gibson and Noakes, 2004), Marcora (2008) proposed the psychobiological model of fatigue, based on the intensity of motivation theory (Brehm and Self, 1989). The psychobiological model proposes that task failure is caused by a conscious decision to terminate exercise, as opposed to muscle fatigue (Marcora, 2008). Specifically, exhaustion occurs when the effort required to perform the task is equal to the maximum effort the individual is willing to exert during the task or when the individual believes to have exerted a true maximal effort and continuation of the task is perceived as impossible (Marcora, 2008). The model proposes that perception of effort (i.e. the conscious decision of how hard, heavy, and strenuous exercise is) predicts time to exhaustion in several conditions and so any physiological or psychological factor affecting perception of effort will affect endurance performance (Marcora, 2008). In support of this, the influence of mental fatigue, defined as a psychobiological state caused by prolonged periods of demanding cognitive activity characterised by subjective feelings such as ‘tiredness’ and ‘lack of energy’ (Boksem and Tops, 2008), on performance has been explored (Marcora, 2009). In this study, participants cycled to exhaustion at 80% of their peak power output after 90-min of a demanding cognitive task (i.e. mental fatigue) or after 90-min of watching emotionally neutral documentaries (i.e. control). The findings

demonstrated that mental fatigue reduced time to exhaustion compared with the control condition that was unmediated by circulatory or energetic factors. The authors therefore concluded that mental fatigue impairs exercise tolerance through higher perception of effort (Marcora et al., 2009).

In summary, fatigue is described as an exercise-induced limitation of performance and measurable as a reduction in the ability of a muscle to exert force, or changes in voluntary muscle activation (Enoka and Duchateau, 2008, Gandevia, 2001). Recent literature (Enoka and Duchateau, 2016, Kluger et al., 2013) defines fatigue as a disabling symptom in which physical and cognitive function is limited by interactions between performance fatigability and perceived fatigability (Enoka and Duchateau, 2016). Performance fatigability depends on the contractile capabilities of the working muscle and capacity of the nervous system to provide adequate activation signal to maintain the task (Enoka and Duchateau, 2016). Performance fatigability is quantified as the objective rate of change in a criterion performance outcome over a discrete period of time during a fatiguing task (Enoka and Duchateau, 2016). The performance outcomes most commonly measured within the scientific literature will be briefly discussed in the following section.

## 3. Assessment of muscle fatigability

Performance fatigability is typically quantified by measuring changes in muscle force or rate of torque development during a maximal voluntary contraction (MVC). Muscle torque is measured using dynamometers, where over time torque declines despite continuing maximal effort. MVCs are typically performed before and after exercise, and the extent of torque loss observed is a classic index of muscle fatigue. Furthermore, knowledge of the mechanisms that contribute to muscle fatigability may be of interest to strength and conditioning practitioners or sports coaches when designing optimal recovery strategies. For example, strategies that aim to optimise the recovery process from reduced contractile function may prioritise nutritional as well as anti-inflammatory interventions such as cold water immersion and cryotherapy (Barnett, 2006, Reilly and Ekblom, 2005, Ihsan et al., 2016). In contrast, little research has examined how recovery from reduced nervous system function or perceived fatigability can be optimised. However, it has been proposed in two narrative reviews that both carbohydrate and sleep present two major strategies to improve recovery from perceived fatigability (Rattray et al., 2015, Marcora et al., 2009). So, additional measurements that provide estimations of contractile function and voluntary muscle activation may be useful. Accordingly, the procedures and techniques typically used within the scientific literature to estimate contractile function and voluntary muscle activation are discussed in the following sub-sections.

### 3.1 Surface electromyography

A muscle action is performed after the desired motor units are recruited following neural transmission from the spinal cord. The net motor unit activity is therefore related to the magnitude of activation signal discharged by the spinal cord, and amplitude of the surface electromyography (EMG) signal is often used as an index of voluntary muscle activation (Enoka and Fuglevand, 2001). For example, increases in EMG amplitude would suggest that more motor units are recruited or are firing faster.

However, local peripheral factors may also change the EMG amplitude as many factors including: changes in fibre type and size, and, more specifically membrane potential, intramuscular ionic concentrations and $Na^+$-$K^+$ pump content will alter the amplitude of muscle fibre action potentials (Fitts, 1994). Although an association exists between voluntary muscle activation and EMG amplitude, the results should be interpreted with caution and so voluntary activation cannot be solely assumed from changes in EMG amplitude.

### 3.2 Interpolated twitch technique

The completeness of voluntary activation (VA) is more effectively assessed by superimposing a relatively strong electrical stimulus to the nerve trunk or intramuscular nerve branches of an active muscle during an MVC. This is known as the interpolated twitch technique (ITT) (Merton, 1954, Paillard et al., 2005). If extra torque can be produced in response to stimulation during an MVC, some motor units were not recruited or firing fast enough to produce fused contractions at the moment of stimulation (Belanger and McComas, 1981). An increase in superimposed twitch torque (SIT) indicates a reduction in VA and places the site of fatigue proximal to the neuromuscular junction (i.e. spinal, sub-cortical, or cortical) (Shield and Zhou, 2004). Both VA (%) and central activation ratio (CAR) can be estimated from the ITT. Voluntary activation is quantified by comparing the amplitude of the SIT during an MVC with the twitch evoked from the same muscle at rest (Shield and Zhou, 2004). Measurement of VA can therefore be performed before, during, and after exercise to measure the influence of the task on performance fatigability. The central activation ratio is calculated from the ratio of the MVC and the MVC plus the SIT amplitude (Kent-Braun, 1999). However, it is often and erroneously assumed that the combination of SIT and MVC will evoke the muscle's true maximum torque. Also, several recruited synergists typically determine MVC torque, while only the stimulated muscles produce the torque increment. So, the CAR value will vary with the number of synergists that

are activated, and is therefore unable to provide a valid measure of muscle activation (Shield and Zhou, 2004). For these reasons, the measurement of VA in which the amplitude of the SIT and evoked resting twitch are compared will be used in this thesis.

## 3.3 Evoked twitch responses

Contraction function is estimated by measuring the declines in twitch or tetanic torque produced by transcutaneous electrical stimulation applied to the motor nerve or intramuscular nerve branches of a muscle. The amplitude of an evoked twitch can be studied in isolation to monitor muscle contractility and excitability. Briefly, muscle contractility is impaired if the muscle torque output in response to stimulation is reduced compared to before exercise levels, indicating peripheral fatigue. However, the simultaneous presence of muscle potentiation (i.e. greater torque production for a similar $Ca^{+}$ release resulting from the phosphorylation of the myosin light chain) due to its previous contraction and fatigue can make interpretation difficult. This is due to the theory that the contractile history of a muscle influences the mechanical performance of subsequent muscle actions (Robbins, 2005). Nevertheless, this may simply be overcome by ensuring the evoked twitch is measured after contraction (Fowles and Green, 2003). As such, evoked potentiated twitch (Pt) torque is a reliable performance outcome that can provide specific information about muscle contractility. The following section will discuss the technical and practical considerations of the interpolated twitch technique.

## 3.4 Twitch interpolation: technical considerations

### *3.4.1 Site of stimulation*

Gold standard assessment of voluntary quadriceps activation is measured with electrical stimulation of the femoral nerve. However, nerve stimulation is associated with discomfort and may lead to negative anticipatory effects (Button and Behm, 2008). Also, the stimulation electrode is likely to be pushed away from the femoral

nerve during contraction because of the nearby tendon (Place et al., 2010). In contrast, direct stimulation of the intramuscular nerve branches causes less discomfort than nerve stimulation, while providing a valid and reliable measure of quadriceps activation (Place et al., 2010). Of note, regardless of the technique employed, unnecessary stimulation of the antagonists should be avoided. This can be achieved by ensuring the electrodes are not placed too far apart or too close to antagonists and by avoiding the use of excessively large electrodes (Shield and Zhou, 2004).

*3.4.2 Stimulation intensity*

Supramaximal stimulation is preferred over submaximal stimulation in studies of performance fatigability because the threshold of motor axons increases during fatiguing contractions and stimulation at a given intensity will activate progressively fewer motor units (Vagg et al., 1998). Supramaximal stimulation can be achieved by ensuring that the current intensity is 120-130% of the intensity required to achieve a plateau in peak twitch torque under resting conditions (Shield and Zhou, 2004).

*3.4.3 Number of interpolated stimuli*

It has been suggested that twin stimuli separated by 10 ms are useful for measuring VA during exhausting exercise (Shield and Zhou, 2004). This is because the evoked force increments are larger and more readily detected. Also, single stimuli are less advisable to muscle fatigue studies because alterations in excitation-contraction coupling (i.e. low-frequency fatigue characterised by loss or torque at low frequencies of stimulation) dictate that the resulting evoked responses will decline more than those evoked with multiple stimuli delivered at a high frequency (i.e. 100 Hz).

*3.4.4 Familiarisation of participants*

Participants unfamiliar with electrical stimulation generally perform lower MVCs when stimulation is anticipated compared to when it is not (Button and Behm, 2008). So,

participants should be formally tested after familiarisation and only when MVCs with expected stimulation match those without expected stimulation.

*3.4.5 Rejection criteria for MVC*

It is recommended that MVCs should be rejected and repeated if: (i) the torque trace exhibits no clear plateau prior to superimposed stimulation; (ii) the superimposed stimulus is not delivered when the voluntary torque is not at or very close to its peak for that contraction; and (iii) the participant perceives that their effort was submaximal at the time of stimulation (Shield and Zhou, 2004). Torque traces of rejected MVCs are presented in figure 1.

In summary, the contribution contractile and central nervous system function to the development of performance fatigability cannot be estimated by measuring changes in MVC torque alone. Additional measures such as the interpolated twitch technique, when performed with rigour provide an acceptable estimation of muscle activation that cannot be assumed solely from changes in EMG activity alone. Additionally, evoked potentiated twitch torque measured before and after exercise provides a robust

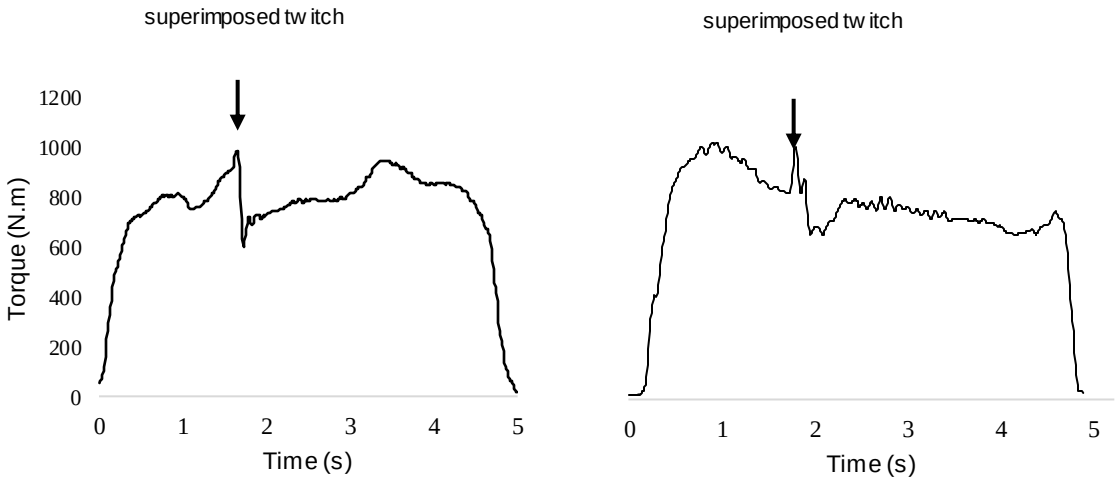


**Figure 1. Torque traces of rejected MVCs. The left panel represents a rejected MVC due to no plateau in torque prior to the superimposed stimulation. The right panel represents a rejected MVC due to the superimposed stimulus not delivered at or very close to peak torque**

estimation of contractile function. The next section provides an overview of the literature relating to fatigue and human performance.

## 4. Fatigue and human performance

Muscle contraction relies on a complex chain of events throughout the entire motor system. The concept of symmorphosis postulates that biological systems adhere to an 'economy of design' giving a close match between their various structural and functional parameters (Weibel et al., 1991). So, it is unlikely that a single parameter within the system is over engineered or has excess capacity beyond the requirements of the system. In the context of muscle fatigability, it is unlikely that only one site or mechanism is responsible for loss of performance during fatiguing contractions. The development of muscle fatigue depends on several factors including the type and duration of activity as well as the nature of muscle action and these differences have become known as the task dependency of fatigue (Enoka and Stuart, 1992). The next subsections will explore the influence of exercise mode, duration or volume, and intensity on muscle fatigability.

### 4.1 Single-joint exercise

Merton (1954) conducted seminal work in the area of fatigue when he measured changes in muscle torque during a sustained maximal isometric voluntary contraction of the isolated adductor pollicis muscle. Additionally, intermittent electrical stimulations were delivered to the ulnar nerve during the voluntary contraction (superimposed twitch) and the amplitude of the EMG signal was measured from the muscle. Merton (1954) showed that the sustained MVC torque reduced soon after exercise onset, while the evoked twitch force was inversely related to torque output. That is, the delivered stimulus did not evoke any additional output when voluntary torque approached maximal values. From these findings two conclusions were made: i) during a maximal effort, the evoked electrical stimulus does not produce an additional

input as the motor units are maximally activated; and ii) the relationship between voluntary torque and the size of the interpolated twitch meant that predictions of absolute maximal torque via linear extrapolation could be made (Merton, 1954).

Merton's (1954) original observations were later confirmed by investigations that employed a similar fatigue assessment model (Bigland-Ritchie et al., 1983, Gandevia et al., 1996, Kent-Braun, 1999, Schillings et al., 2003). For example, Kent-Braun (1999) examined the influence of a 4-min sustained maximal voluntary ankle dorsiflexion on contractile function and voluntary muscle activation (VA). The findings demonstrated that both contractile function and VA were impaired indicate by reductions in central activation ratio (CAR) and evoked twitch torque amplitude. Lastly, the authors estimated that 20% of the reduced MVC torque was due to incomplete VA (Kent-Braun, 1999). As well, Gandevia et al. (1996) attributed ~25% of the loss of MVC torque (~60%) to supraspinal mechanisms (assessed by transcranial magnetic stimulation in which a magnetic stimulus is delivered to the motor cortex) after 2-min of sustained elbow flexion. As previously discussed, corticospinal excitability recovered immediately, while VA remained impaired (during ischaemia) until normal blood flow was restored (Gandevia et al., 1996).

Like sustained isometric maximal actions, impaired contractile function and incomplete muscle activation has been observed after submaximal isometric actions (Behm and St-Pierre, 1997, Smith et al., 2007, Sogaard et al., 2006, Yoon et al., 2007). Behm and St-Pierre (1997) compared quadriceps fatigability after a long and short duration knee extension protocols at 25 and 50% of MVC performed until task failure. Time to failure was 20-min and 4-min, while reductions in MVC (40 and 30%) and VA (13 and 6%) were reported. More recently, in two similar studies (Smith et al., 2007, Sogaard et al., 2006), participants held isometric elbow flexions at 5 and 15% of MVC for 70 and 43-min. Maximal voluntary contraction was measured every 3-min and contractile function and VA were assessed via both twitch interpolation and motor cortex stimulation. The

findings from both studies show that loss of VA developed concurrently with declines in contractile function (Smith et al., 2007, Sogaard et al., 2006). Interestingly, Sogaard et al. (2006) measured the recovery of these outcome measures after exercise cessation. The authors reported that 25-min after exercise cessation, MVC had recovered by ~86% of before exercise values, while evoked twitch torque presented with minimal recovery (indicating contractile dysfunction). The authors concluded that the partial recovery of MVC torque reflects rapid recovery of VA after exercise cessation, which may be readily explained by the clearance of metabolic-by-products after a short recovery period (Amann and Dempsey, 2008, Sogaard et al., 2006).

In addition to sustained isometric actions, reduced contractile function and VA have been observed during intermittent submaximal actions. Bigland-Ritchie et al. (1986) examined the influence of submaximal (50% of MVC) intermittent isometric contractions (6-s with 4-s rest) between the quadriceps and soleus muscles until a target force could no longer be attained. For the quadriceps, participants achieved the target force for ~4-min with no decrease in VA, whereas in the soleus muscle time to failure was much longer (~35-min), but some impairment of VA was present (Bigland-Ritchie et al., 1986). Although the different results between the quadriceps and soleus might be attributable to the intrinsic properties of the muscles such as fibre type composition, the impairment of soleus VA may also be related to the longer task duration. In support of this, self-paced single-joint exercise time protocols (TT) of different durations induce distinct changes in MVC, VA, and contractile function measures (Froyd et al., 2016). In this well-controlled study, participants performed 3, 10, and 40-min TTs consisting of continuous maximal isokinetic (60 $deg \cdot s^{-1}$) knee extensions. Each 3, 10, and 40-min TT consisted of 4, 16, and 64 sets consisting of 15 contractions with muscle performance assessment (ITT) completed after each set. The results of the study were that MVC reduced to similar levels after all TTs, however evoked twitch torque decreased more after the 40-min TT compared with the 3-min

TT (-42 versus --37%). Additionally, declines in VA were present during the longer 10- and 40-min TTs, but was unchanged during and after the 3-min TT. This work clearly demonstrates that short duration tasks are associated with contractile dysfunction, whereas muscle fatigability during longer tasks may be attributed to both reduced VA and contractile dysfunction.

The influence of muscle action on muscle fatigability has also been examined in numerous studies (Babault et al., 2006, Kay et al., 2000). For example, Kay et al. (2000) compared the influence of 'all-out' isometric, concentric, and eccentric actions of the knee extensors each lasting 100-s on muscle fatigability. The authors reported that MVC torque reduced to similar levels after isometric and concentric muscle actions, while eccentric actions were largely fatigue resistant. Interestingly, EMG amplitude was unchanged during concentric and eccentric conditions, but reduced after isometric exercise only (Kay et al., 2000). More recently, Babault et al. (2006) had participants perform maximal concentric and isometric knee extensions until similar reductions in MVC were established. Voluntary activation assessed by twitch interpolation decreased progressively during concentric exercise, but faster and more profoundly during isometric exercise. In contrast, evoked twitch torque amplitude reduced soon after concentric exercise onset and remained reduced, but decreased progressively across the three sets of isometric exercise (Babault et al., 2006). So, after isometric exercise, VA declined rapidly and impairment of contractile function developed secondarily. Interestingly, the development of fatigue was the inverse during concentric exercise. Lastly, and comparable to the observations of Kay et al. (2000), EMG amplitude during MVCs did not change after concentric exercise, but was reduced after isometric actions.

The differences in the development of muscle fatigue profiles after concentric and isometric schemes could be partly explained by the increased intramuscular concentration of metabolites due to the ischaemic nature of isometric muscle actions.

As previously discussed in section 2.3, metabolite accumulation is likely involved with inhibition of central motor output (Amann, 2012, Noakes, 2012). Taken together, these findings indicate that the time course and magnitude of disturbances to VA and contractile function depend on the duration and intensity of exercise as well as contraction type and movement velocity. To this point, this review has focused on studies on single-joint exercise. The topic of fatigue and fatigability however, is likely more complex in studies of whole-body exercise where larger disturbances to general homeostasis are expected. The next section will summarise the literature relating to the influence of locomotor exercise and muscle fatigability.

## 4.2 Locomotor exercise

Locomotor exercise such as running or cycling involving a relatively large muscle mass is likely to cause larger disturbances to general homeostasis and present distinct influences on muscle fatigability compared to single-joint exercise. It is well known that reductions in quadriceps VA occur during prolonged, endurance running and cycling exercise (Decorte et al., 2012, Lepers et al., 2002, Place et al., 2004, Ross et al., 2010b). For example, during a 20-km self-paced running time protocol (TT) in experienced runners, loss of isometric knee extensor torque (-15%) was reported during the final 5-km only (assessment at 5, 10, 15, and 20-km) (Ross et al., 2010a). Interestingly, the authors attributed the loss of torque exclusively to impaired VA (-13%; ITT) as potentiated twitch torque (Pt) induced by femoral nerve stimulation was unchanged during and after the 20-km run. Additionally, during cycling TTs of 40-, 20, and 4-km, similar reductions in isometric knee extensor torque were observed (-18, -15, and -16%) (Thomas et al., 2015). However, impairment of contractile function was observed after 4-km (40% reduction in Pt) compared to the 20-km (-31%) and 40-km TTs (-29%). In contrast, the longer 20 and 40-km TTs were characterised by greater reductions of quadriceps VA (-11 and -10%) compared to the 4-km TT (-7%). Similar to studies of single-joint exercise (Froyd et al., 2016), changes in VA and contractile

function after self-paced exercise are task dependent, with greater impairment to contractile function occurring after short, high-intensity TTs, while greater muscle inactivation occurs after sustained, low-intensity TTs.

Although much work has described changes in contractile function and VA during locomotor exercise, very few studies have examined the recovery of these outcome measures (Bentley et al., 2000, Ross et al., 2010b). Accordingly, high-intensity (30-min at 80% $VO_{2max}$ followed by four x 60-s at 120% $VO_{2max}$) cycling exercise supressed VA and contractile function for a period of 6-hours (h) after exercise cessation (Bentley et al., 2000). In addition, after 20-days (d) of repetitive endurance cycling within a 22-d period, both VA and contractile function were impaired after 18-h passive recovery. Interestingly, while contractile function was restored after 2-d rest, VA remained impaired at 2-d after exercise cessation (Ross et al., 2010b). Such findings are in contrast to those observed in isolated muscle groups, where rapid recovery of VA has been observed (Gandevia, 2001). These results suggest that a different mechanism may be responsible for the persistent reductions in muscle activation. As such, the slow recovery of VA may demonstrate a transient form of chronic fatigue and may provide evidence that the central nervous system has an integral role in the impaired performance associated with overreaching (Ross et al., 2010b).

Numerous studies have examined the influence of endurance exercise on muscle fatigability, yet the influence of maximal repeated- or intermittent-sprint exercise is unclear. For instance, after 10 x 6-s 'all-out' repeated cycle sprints (30-s recovery) followed 6-min later by a further 5 x 6-s sprints (30 s recovery), reductions in MVC (-11%) were due to impairment of contractile function as VA was unchanged (Girard et al., 2013). However, loss of MVC (~ -16%) due to contractile dysfunction as well as small but significant decreases in VA (-2.7%; interpolated twitch technique) were reported after 12 x 40-m running sprints (Perrey et al., 2010) and 10 x 6-s (30-s recovery) cycle sprints (-2.5%) (Racinais et al., 2007). More recently, Goodall et al.

(2015) and Pearcey et al. (2015) observed reduced MVC (-12 and -24%) and larger reductions in VA (-8% and 6%) after repeated-sprint running. In the study conducted by Goodall et al. (2015), participants performed 12 x 30-m sprints interspersed by 30-s rest with assessment of muscle performed before, during, and after exercise. The reductions in VA observed are larger than those reported after repeated cycle sprints (Girard et al., 2013, Racinais et al., 2007) and after a similar running protocol (12 x 40-m) (Perrey et al., 2010). The differences may be due to greater metabolic disturbance (e.g. blood lactate concentrations) associated with a larger muscle mass recruited during running compared to cycling. As well, the timing of muscle fatigability assessment after exercise cessation may be a critical issue. For instance, Perrey et al. (2010) made measurements 6-8-min after exercise cessation, whereas Goodall et al. (2015) measured fatigue within 2.5-min and may have captured the presence of suppressed VA before it quickly dissipates.

The above findings indicate that muscle fatigability during locomotor exercise depends largely on the intensity and duration of exercise. Impairment to contractile function is greatest after short, high-intensity exercise, whereas reductions in VA present during the latter stages of sustained, low-intensity tasks. The influence of high-intensity intermittent exercise on muscle fatigability is unclear as the role central nervous system function is debated. Many athletes engage in concurrent training (strength and endurance) and perform resistance exercise to improve athletic performance and reduce the risk of injury (Suchomel et al., 2016). Therefore, the next section will discuss the influence of resistance exercise on muscle fatigability.

### 4.3 Resistance exercise

In contrast to endurance and intermittent exercise, the influence of resistance exercise on muscle fatigability is poorly understood owing to a paucity of literature. Similar to locomotor exercise, it is generally accepted that manipulation of resistance exercise variables is necessary to drive and maximise specific adaptations (American College

of Sports, 2009). The intensity of load is widely considered the most important and commonly manipulated of these variables (Schoenfeld et al., 2016a). Indeed, the most common loading strategies used within athletic training programmes are: high-load low repetition (HL) and moderate-load high repetitions (ML). High-load strategies have been proposed to maximise increases muscular strength (Crewther et al., 2006a, Crewther et al., 2006b, Schoenfeld et al., 2016a), whereas ML strategies are associated with the largest and quickest increases in muscle cross-sectional area (i.e. muscle hypertrophy) (Wernbom et al., 2007, Schoenfeld et al., 2016a). The majority of work relating to resistance exercise and muscle fatigability has examined these strategies.

Early observations suggest that HL and ML training protocols present distinct changes in loss of MVC and EMG amplitude (Hakkinen, 1993, Hakkinen, 1994). In these studies, resistance-trained males and females performed loaded barbell back squats: the HL protocol consisted of 20 sets of single maximal repetitions (3-min recovery); while the ML protocol consisted of ten sets of 10 repetitions at a load corresponding to 10RM (i.e. the maximum amount of mass that can be lifted for ten consecutive repetitions) with 1.5-min recovery. MVC torque and EMG amplitude were measured before, during, and immediately after exercise. The main finding was that after HL and ML training, MVC torque reduced by 24 and 47% in males and 21 and 29% in females. These large declines in MVC torque are in contrast with previous observations on endurance and intermittent exercise where loss of MVC was lower after a 20-km run (-15%) (Ross et al., 2010a); cycling TTs of 40, 20, and 4-km (-18, -15, and -16%) (Thomas et al., 2015); and intermittent maximal running and cycling exercise (-11-16%) (Girard et al., 2013, Perrey et al., 2010). Additionally, the authors reported that after both protocols EMG amplitude was reduced (implying a decline in VA). As well, blood lactate concentrations after exercise were 3.5 and 15 mmol/L versus 2.5 and 6 mmol/L for males and females. Taken together, it was suggested that ML training was

associated with greater reductions in MVC torque and EMG amplitude compared to HL exercise. However, a causative link to the source of fatigue was outside the scope of the study as VA cannot be assumed solely from changes in EMG amplitude.

More recently, Walker et al. (2012) also assessed changes in MVC and EMG amplitude during ML (five sets of 10RM) and HL (fifteen sets of 1RM) leg-press exercise in untrained males. In line with previous observations (Hakkinen, 1993, Hakkinen, 1994), the authors reported that ML training induced greater loss of MVC torque (-48 versus -30%) compared to HL training. However, loss of MVC torque after ML training was assumed due to impairment of contractile (indirectly indicated by greater after exercise blood lactate concentrations (~12 mmol/L)) and unchanged VA assumed from a reduction in EMG amplitude. In contrast, EMG amplitude reduced during and after the HL protocol only, while after exercise blood lactate concentrations were lower (~4 mmol/L) compared to ML exercise. Accordingly, it was suggested that fatigability induced by ML training was due to contractile dysfunction only and therefore differs from the study of Hakkinen (1994) that reported reduced VA after ML back squat exercise. However, it is possible that the variance in observations may be explained by the difference in duration (i.e. volume of work) between the ML protocols. For example, Walker et al. (2012) had participants perform five sets of 10RM leg press exercise, whereas Hakkinen (1994) employed ten sets of 10RM back squat exercise. From studies on both single-joint exercise (Froyd et al., 2016) and whole-body exercise (Thomas et al., 2015) it is well- established that longer task durations are associated with more disturbance to nervous system function. Relatedly, the observed differences between HL and ML resistance exercise may be related to the differences in volume of work completed.

To further explore this mechanism McCaulley et al. (2009) examined changes in MVC and EMG amplitude during HL (eleven sets of 3 repetitions at 90% 1RM) and ML (four sets of 10 repetitions at 75% 1RM) volume load-equated resistance exercise. Briefly,

volume load (VL) is defined as the product of the total number of repetitions performed and the corresponding load lifted. Similar to previous observations (Hakkinen, 1993, Hakkinen, 1994, Walker et al., 2012), ML training induces greater losses in MVC torque compared to HL training (-26 versus -17%). Additionally, ML training resulted in elevated muscle activity (EMG amplitude) compared to HL training (McCaulley et al., 2009). As well, the recovery of rate of force development, measured at 24-h and 48-h after exercise was slower after the HL protocol in comparison to ML, suggesting a greater disruption of nervous system function (McCaulley et al., 2009). The authors concluded that VL did not influence fatigability and recovery during resistance exercise and that load and rest period modification were more important variables. It was suggested that the higher intensity of loading in the HL protocol may heighten the stimulus to the nervous system and result in more greater declined in EMG amplitude compared to ML resistance exercise (McCaulley et al., 2009).

In addition to intensity of loading and volume, the influence of contraction velocity on MVC and EMG has been explored. One study (Linnamo et al., 1998), examined the influence of two protocols performed with different loads and movement angular velocities in healthy participants. The first protocol consisted of five sets of leg extension exercise at a load corresponding to 10RM and the second consisted of five sets at a reduced load corresponding to 40% of 10RM. Additionally, participants were asked to perform the task with maximum effort (i.e. as fast as possible). Loss of MVC torque was greater and the recovery slower after the higher loading protocol, while reduced EMG amplitude was observed after the reduced loading/fast velocity protocol. This study suggests that movement angular velocity may also influence fatigability.

Taken together, the findings from the abovementioned studies suggest that intensity of loading, volume of work, and contraction velocity may influence fatigability during resistance exercise. High-load resistance exercise is associated with reductions in EMG amplitude, whereas ML resistance exercise is associated with greater muscle

fatigability likely from impairment of contractile function. However, as previously discussed, estimates of VA cannot be assumed solely from changes in EMG amplitude. Therefore, the abovementioned studies are limited as methods that can robustly estimate a causative link to the development of fatigue, such as the interpolated twitch technique (ITT), have not been employed.

Although twitch interpolation is considered a more robust estimate of VA, it is surprising that very few studies have employed this technique when assessing before and after exercise performance fatigability during resistance exercise. To date, only four studies have used the ITT to estimate the completeness of muscle activation and contractile function after resistance exercise (Behm et al., 2002, Brandon et al., 2015, Tran et al., 2006, Howatson et al., 2015). Behm et al. (2002), compared the influence of one set of maximal elbow flexions at 20, 10 and 5RM in healthy males on MVC, VA, and contractile function. The authors reported reduced MVC torque and VA after all protocols that was not different between conditions. However, higher repetitions (i.e. 20RM) was associated with greater detrimental effects on evoked twitch torque amplitude compared to 10RM (~11%) and 5RM (~30%).

Later, Tran et al. (2006) assessed changes in MVC, VA, and Pt after manipulation of either concentric time-under-tension (TUT) or volume load (VL) of elbow flexion exercise. The study conducted in males consisted of three fatiguing protocols: the first was defined as high-volume (three sets of 10 repetitions) and long (5 s per repetition) concentric TUT; (ii) in the second protocol participants completed the same high-volume load but with low (40%; i.e. 2 s per repetition) of the concentric TUT compared to the first protocol; (iii) and in the third protocol, participants performed low-volume load (50%), but with equal TUT (i.e. 10 s per repetition) compared to the first protocol. In line with previous observations (Behm et al., 2002), the main finding was that high-volume and long-TUT was associated with greater muscle fatigability demonstrated by a 19% reduction in MVC torque compared to 13 and 15% after the short concentric

TUT and low volume protocols. Also, greater impairments of contractile function were observed indicated by declines in Pt (-57, 12 and 30%). Additionally, TUT was more influential than volume load on fatigability, with the low-volume long TUT protocol associated with greater fatigability than high-volume, short- time under tension. These findings suggest that prolonged tension elicits greater contractile stress, resulting in greater contractile dysfunction. As well, the authors were unable to detect any disruption to nervous system function as VA (~96%) was unchanged after all protocols. The study (Tran et al., 2006) provides valuable insight into muscle fatigability during single-joint resistance exercise of varying TUT and volume loads, however elbow flexions are unrepresentative of primary exercises employed in athletic training programmes. Therefore, more work is needed on the influence of TUT and volume load on fatigability during multi-joint exercises such as squats and deadlifts typically employed by coaches and athletes.

Most recently, Brandon et al. (2015) examined changes in MVC, VA, and contractile function after typical resistance exercise schemes involving heavy, moderate, and light barbell back squat exercise (ten sets of 5 repetitions) in elite male track and field athletes. The heavy condition load corresponded to an active muscle RPE of 16-17 (very hard) and the moderate and light loads were 75 and 50% of the heavy session load. The main findings of the study were that MVC and evoked twitch torque were significantly reduced 10-min after the heavy (-12 and 31%) and moderate (-7% and 16%), but not the light sessions. Also, VA assessed by the central activation ratio (CAR) method was unchanged between all three protocols. As such, the loss of MVC torque after ten sets of heavy and moderate back squats was attributed to contractile dysfunction only. As well, Howatson et al. (2015) studied the acute and 24-h fatigue response after heavy barbell back squat exercise in elite male and female athletes. The acute responses were in line with the previous findings (Brandon et al., 2015) as MVC torque was reduced (-11%), while CAR was unchanged. In addition, MVC torque

was still impaired (-6%) 24-h after exercise, indicating incomplete recovery (Howatson et al., 2015).

The studies of Brandon et al. (2015), Howatson et al. (2015), and Tran et al. (2006) suggest that load, time-under-tension, and rest period duration, are critical factors that influence muscle fatigability during resistance exercise. The abovementioned training variables are commonly manipulated within athletic training programmes and knowledge of how they influence fatigability is useful when research informs the design of athletic training programmes. Contrary to previous studies that estimated VA via changes in EMG amplitude (Hakkinen, 1993, Hakkinen, 1994, Walker et al., 2012), data from studies using electrical stimulation techniques demonstrate that impairment of contractile function, and not disruption to nervous system function, contribute to the loss of MVC torque observed during resistance exercise (Brandon et al., 2015, Howatson et al., 2015, Tran et al., 2006). However, just one study (Behm et al., 2002), has observed loss of muscle activation after single-joint resistance exercise. The studies of Brandon et al. (2015) and Howatson et al. (2015) provide valuable insight into the influence of typical resistance exercise bouts employed by athletes and coaches on performance fatigability. However, the use of a closed-loop task design (i.e. a fixed set and repetition scheme) may not allow participants to reach mechanical failure where the active muscles are unable to produce the expected force to complete a repetition due to muscle fatigue. Accordingly, it has been proposed that careful prescription of resistance exercise performed until mechanical failure may help advanced athletes exceed ‘plateaus’ (Willardson, 2007). For instance, a dose-response relationship exists, to which greater volumes of resistance exercise are associated with greater increases in both strength (Krieger, 2009) and muscle mass (Schoenfeld et al., 2016b). Additionally, resistance training to mechanical failure improves upper body strength (bench press) in resistance-trained junior elite basketball players (Drinkwater et al., 2005). Specifically, muscular strength was

measured after two different volume-equated protocols performed 3 times per week for 6 weeks. The protocols were characterised as repetitions to failure (four sets of 6 repetitions) and non-failure (eight sets of 3 repetitions), with similar loads (~85–105% 1-RM). As well, the rest period differed between protocols, with 260 s between sets for repetitions to failure and 113 s for non-failure. The results showed greater fatigability (measured before and after exercise using a customised bench throw power test) and a twofold increase in strength for the failure group compared with non-failure. These results suggest that, in strength trained males, fatigability caused by muscular failure may be related to greater muscle activation, which would explain the greater increases in muscle strength when performing repetitions to failure. In support of this, EMG amplitude is greater when repetitions are performed until failure compared with submaximal repetitions of the same intensity (Looney et al., 2016). Further possible justifications include: an ability to induce more mechanical stress, increase anabolic hormone levels; and transitional fibres (Willardson, 2007). As such, appropriate application of mechanical failure resistance exercise can be justified for athletes who wish to change or enhance the training stimulus. However, to date, no work exists that has examined the influence of training to failure on performance fatigability during resistance exercise.

**5. Summary**

Muscle fatigue is defined as an exercise-induced limitation of performance and measurable as a reduction in the ability of a muscle to exert force, or changes in voluntary muscle activation (Enoka and Duchateau, 2008, Gandevia, 2001). Recent literature (Enoka and Duchateau, 2016, Kluger et al., 2013) defines fatigue as a disabling symptom in which physical and cognitive function is limited by interactions between performance fatigability and perceived fatigability (Enoka and Duchateau, 2016). Performance fatigability depends on the contractile capabilities of the working muscle and capacity of the nervous system to provide adequate activation to maintain

the task. Perceived fatigability describes the changes in subjective sensations that regulate the integrity of the exerciser (Enoka and Duchateau, 2016).

Performance fatigability is quantified as the objective rate of change in a criterion performance outcome over a discrete period of time during a fatiguing task (Enoka and Duchateau, 2016). The development of performance fatigability during physical activity is highly task dependent and related to exercise type, duration and intensity as well as contraction type and velocity. As well, the mechanisms that contribute to performance fatigability may influence the time course of recovery and affect subsequent training bouts. Relatedly, ensuring that fatigue is appropriately managed is important for adaptations to training and competition performance as well as reducing injury risk. As such, knowledge of muscle fatigability during resistance exercise may help inform the preparation of athletic training programmes.

Early studies examining the influence of single-joint exercise on performance fatigability found that sustained maximal isometric muscle actions induce fatigue attributed to disruption of nervous system function and impairment of contractile function. Additionally, voluntary activation (VA) is recovered quickly following restoration of normal blood flow (Gandevia et al., 1996). In contrast, submaximal isometric exercise of moderate duration induces impairment of contractile function, while VA progressively declines if exercise is sustained (Behm and St-Pierre, 1997, Yoon et al., 2007). Performance fatigability during locomotor exercise is largely in parallel with isolated muscle studies. For example, prolonged endurance exercise is associated with early contractile dysfunction, while impairment to VA may develop during the latter stages of exercise (Thomas et al., 2015). However, influence of high-intensity intermittent exercise on performance fatigability remains unclear (Girard et al., 2013, Goodall et al., 2015).

The findings from studies of isolated muscle groups and locomotor exercise are interesting and suggest that a similar approach to investigating performance fatigability after resistance exercise may be useful for athletes and coaches. Like locomotor exercise, performance fatigability induced by resistance exercise is highly task-dependent and related to the intensity of load, contraction type, rest period duration, and time-under-tension. Early studies have been limited by the assessment methodology employed, while evidence using more robust techniques such as the interpolated twitch technique (ITT) is scarce. Studies employing the ITT have found that performance fatigability is greater when training is performed with longer time-under-tension (Tran et al., 2006). However, this study employed single-joint exercise (i.e. elbow flexions) and warrants further investigation in multi-joint resistance exercise (e.g. squats and deadlifts) more commonly employed by athletes.

The loss of MVC torque observed after high-load (HL) resistance exercise in elite athletes (barbell back squats) is due to impaired contractile function (Brandon et al., 2015, Howatson et al., 2015). Although representative of typical resistance exercise schemes employed by athletes and sports coaches, the research conducted so far has focused on closed-loop design (i.e. predetermined sets and repetitions), which may not allow participants to reach mechanical failure. As well, performing resistance exercise until muscle failure may maximise muscular adaptions and is a technique that advanced athletes may use to exceed 'plateaus' in training. So, employing an open-loop design may provide a novel and alternative focus of work. Relatedly, it is possible that disruption to nervous system function may develop if resistance exercise is performed with sufficient duration. For example, (Behm and St-Pierre, 1997) reported that fatigue-induced muscle inactivation after submaximal muscle actions may be duration dependent increasing with prolonged fatiguing contractions. Accordingly, it is also important to measure performance fatigability within minutes of exercise cessation to capture any changes to nervous system function (Gandevia, 2001).

Finally, the recovery of fatigue after resistance exercise performed until exhaustion is unknown and warrants investigation.

In conclusion, one of the key objectives of the coach is to plan optimal concurrent training programmes that combine: strength, endurance, speed, and technical training, while ensuring fatigue is appropriately managed. It is therefore important that sports coaches understand the influence of resistance exercise on muscle fatigability. However, very few studies have explored the influence of resistance exercise on performance fatigability, and in particular resistance exercise performed until mechanical failure and of different lifting tempos warrants further research. Finally, understanding the type of fatigue present is important and may have important implications for the prescription of recovery interventions.

# Chapter 3. Changes in contractile function and voluntary activation and 24-h recovery after exhaustive high-load and moderate-load hexagonal-barbell deadlifts

## Abstract

***Purpose:*** To examine changes and recovery of contractile function and voluntary activation after maximal high-load and moderate-load hexagonal-barbell deadlift (HBD) exercise. ***Methods:*** Eight resistance trained males completed two exhaustive resistance exercise protocols using a randomised cross-over design. The protocols included: high-load low repetitions (HL) consisting of sets of 3 repetitions performed until volitional exhaustion at 90% 3RM (3-min recovery) and moderate-load high repetitions (ML) consisting of sets of 10 repetitions at 75% 3RM (1.5-min recovery). Maximal voluntary contraction (MVC), voluntary activation (VA), and evoked potentiated twitch torque (Pt) of the quadriceps were determined using the interpolated twitch technique before, immediately after, and 24-h after exercise. Bar displacement data were measured during the exercise protocol. ***Results:*** The external load was greater during HL compared to ML (170.9 ± 27.1 vs. 150.9 ± 17.8 kg), while total concentric work, mean power output, velocity, and after exercise blood lactate concentrations were lower (18.1 ± 8.2 vs 29.8 ± 13.8 kJ; 550 ± 111 vs 648 ± 51 W; 0.34 ± 0.05 vs 0.45 0.06 m/s; 4.71 ± 2.3 vs 12.2 ± 2.4 mmol/L). Changes in MVC torque (-15.4 ± 10.7%), Pt (-18 ± 14.4%), and VA (-13.1 ± 14.35%) were observed immediately after ML HBD exercise, but not HL exercise. Loss of MVC (-12.8 ± 7.7%) was present up to 24-h after ML exercise, which was associated with reduced VA and near full recovery of contractile function. ***Conclusion:*** Moderate-load HBD exercise performed until mechanical failure resulted in losses of contractile function and voluntary activation, while MVC was preserved after HL exercise. After ML exercise, loss of MVC was present up to 24-h after exercise, which was largely due to suppressed voluntary activation.


## 1. Introduction

Muscle fatigue is defined as an exercise-induced limitation of performance and measurable as a reduction in the ability of a muscle to exert force or changes in voluntary muscle activation (VA) (Enoka and Duchateau, 2008, Gandevia, 2001). The primary causes of fatigue are associated with changes in the activation signal generated by the nervous system (Gandevia, 2001) and/or impairments of the contractile machinery within the muscle cell (Allen et al., 2008). More recently, fatigue has been defined as a disabling symptom in which physical function is limited by interactions between performance fatigability and perceived fatigability (Enoka and Duchateau, 2016, Kluger et al., 2013). Performance fatigability relates to the contractile capabilities of the working muscle and capacity of the nervous system to provide adequate activation to maintain the task, while perceived fatigability describes

the changes in subjective sensations that regulate the integrity of the exerciser (Enoka and Duchateau, 2016).

The majority of work examining the rate-limiting adjustments during fatiguing contractions has focused on locomotor exercise such as running or cycling (Amann and Dempsey, 2008, Decorte et al., 2012, Girard et al., 2013, Goodall et al., 2015, Rampinini et al., 2014, Ross et al., 2010a, Ross et al., 2010b, Thomas et al., 2015). This work demonstrates that the contribution of contractile dysfunction to performance fatigability is greater after high-intensity activity of shorter durations (Thomas et al., 2015), while the contribution of reduced voluntary activation is relatively higher after sustained, low-intensity activity (Ross et al., 2010a). However, very little is known on the influence of resistance exercise on performance fatigability. This is surprising since many athletes perform concurrent or mixed training programmes as high-levels of muscular strength are important for improving general and sport specific skills, while reducing risk of injury (Suchomel et al., 2016).

Limited data show that high-load (>85% 1RM) and moderate-load (<80% 1RM) resistance exercise appears to exert different effects on contractile function (Brandon et al., 2015, Howatson et al., 2015, McCaulley et al., 2009). For example, Brandon et al. (2015) compared changes in maximal voluntary contraction (MVC) torque, VA, and evoked potentiated twitch torque (Pt) before and 10-min after heavy, moderate, and light barbell back squats (ten sets of 5 repetitions) in elite male athletes. Maximal voluntary contraction of the quadriceps declined after the heavy (-12%) and moderate (-7%), but not light sessions. Additionally, after the heavy and moderate sessions, Pt reduced indicating impairment of contractile function, while VA was unchanged after exercise suggesting that neural drive was preserved. These findings were supported by another study (Howatson et al., 2015) where similar losses of MVC (-11%) were observed after heavy barbell back squats, which was similarly attributed to reductions

in contractile function. Finally, the authors reported that MVC remained suppressed (-6%) at 24-hours (h) after exercise (Howatson et al., 2015).

The studies of Brandon et al. (2015) and Howatson et al. (2015) provide valuable insight into performance fatigability and recovery during resistance exercise schemes employed by elite athletes. Such knowledge is essential when research informs the preparation of athletic training programmes as management of fatigue is important for adaptations to training and competition performance as well as reducing injury risk (Hulin et al., 2016). However, more work is needed to address gaps in knowledge. For example, a dose-response relationship exists, to which greater volumes of resistance exercise are associated with greater increases in both strength (Krieger, 2009) and muscle mass (Schoenfeld et al., 2016b). Relatedly, resistance exercise to mechanical failure improves strength (bench press) compared to non-mechanical failure exercise in resistance-trained junior elite basketball players (Drinkwater et al., 2005). For these reasons, performing resistance exercise until muscle failure is a technique that advanced athletes may use to exceed 'plateaus' in strength development.

With this in mind, this study aims to examine changes and recovery of contractile function and voluntary activation induced by a single bout of multi-joint resistance exercise performed until mechanical failure with high-load (HL) (3 repetitions per set) or moderate-load (ML) (10 repetitions per set). Changes in MVC, VA, and Pt were measured before, at exhaustion, and 24-h after exercise. It was hypothesised that a larger volume of work would be completed for the ML protocol compared to HL, which would induce greater loss of MVC torque. It was also hypothesised that the exhaustive nature of exercise would result in reductions in both contractile function and voluntary activation. As well, it was hypothesised that at 24-h, the recovery of fatigue would be incomplete for both protocols, but less after ML exercise.

## 2. Method

### 2.1 Participants

Eight resistance-trained males (mean ± SD; age 27.4 ± 5.7 years, height 1.76 ± 0.1 cm, body mass 77.4 ± 11.3 kg) who had been engaging in resistance exercise for at least 2 years and training ≥ 2 sessions per week volunteered to participate in this study. All participants were accustomed to performing a hexagonal-barbell deadlift (HBD) with correct technique and laboratory exercise testing and were free of cardiorespiratory, neurological, or neuromuscular disorders (PAR-Q) (Thomas et al., 1992). Initial HBD 3 repetitions maximum (3RM) was 181 ± 26.4 kg (2.3 ± 0.2 normalised per kg of body mass). All participants were informed of the purpose of the study, experimental procedures, and associated risks prior to participation and exercise testing. All participants gave verbal and written informed consent, and the protocol was approved by the University of Essex ethics committee, in the spirit of the Helsinki Declaration.

### 2.2 Experimental design

A within-group repeated measures design in which participants performed two protocols, ML and HL, in a randomised and counterbalanced order, separated by 5-8 d was employed. For each protocol, before and after exercise measurements of knee extensor MVC torque, voluntary activation, potentiated twitch torque, and capillary (ear lobe) blood lactate concentrations were taken. Three to seven days prior to the start of the study participants completed muscular strength testing to evaluate each participant's 3RM for the HBD. On the same day, participants completed a familiarisation session. Participants were instructed to avoid any lower-body exercise 48-h prior to any visit and any exercise the day before a visit.

**Table 1. Modified APRE protocol for HL and ML with set 2 adjustment**

| Repetitions | Load intensity (% protocol weight) |
|---|---|
| **Warm up** | |
| 10 x | Empty bar (40 kg) |
| 10 x | 50% |
| 6 x | 75% |
| **HL** | |
| Maximum | 100% |
| Repetitions for set 2 adjustment | Set 2 adjustment (%) |
| 1 | -10 |
| 2 | -5 |
| 3-4 | No change |
| 5-7 | +3 |
| 8+ | +12 |
| **ML** | |
| Maximum | 100% |
| Repetitions for set 2 adjustment | |
| 0-3 | -20% |
| 4-7 | -10% |
| 8-9 | -5% |
| 10-12 | No change |
| 13-17 | +10% |
| 17+ | +30% |

*APRE = auto regulating progressive resistance exercise; 3RM = 3 repetitions maximum

2.3 Muscular strength testing and familiarisation session

Prior to dynamic muscular strength testing, participants performed non-specific, individual joint mobilisation followed by a specific warm up consisting of submaximal HBD repetitions of a single set of 10, 5, 3, and 1 repetition(s) at 50, 70, 80, and 90% of estimated 3RM. After the warm up, participants rested for 3-5-min and performed three attempts (3-5 min recovery) to determine actual 3RM. The HBD exercise was performed by stepping inside the barbell and squatting down to grasp the handles with a shoulder-width stance. A repetition was considered successful if the barbell was not lowered at any point during the ascent, and upon completion an upright posture was achieved with the hip and knee joint at full extension. A 3RM and working percentages (i.e. 90 and 75% of 3RM) were chosen to align with common practice among strength

and conditioning practitioners. After dynamic strength testing, participants were accustomed with all laboratory procedures. Attention was given to the quadriceps MVC, with participants repeating the procedure until they could maintain a stable plateau in torque for 3-4 s. Also, participants were accustomed with direct muscle stimulation and care was taken to ensure participants could match MVCs produced without stimulation with those while expecting stimulation (Shield and Zhou, 2004).

2.4 Exercise protocol

The ML protocol was conducted using a load corresponding to 75% of 3RM and comprised of an open-ended number of sets comprising ten repetitions with 90 s rest periods, while the HL protocol was conducted at 90% of 3RM and comprised sets of three repetitions with 180 s rest. To allow for daily and weekly fluctuations in participant's individual strength performance, a modified version of the auto regulating progressive resistance exercise (APRE) method (Mann et al., 2010) was employed. Specifically, during the first set, participants performed 'maximally' (i.e. as many repetitions as they could) with the prescribed weight. Next, the weight used for the remaining sets was based on the performance during the first maximal set using an adjustment table (Table 1). For the remaining sets, participants then performed 10 or 3 repetitions at the adjusted weight until they reached volitional exhaustion or could not complete the required repetitions within a set identified as an inability to extend the knee and hip joints or within the time limit of four s per repetition. Participants were instructed to perform the concentric portion of the lift with maximal effort (i.e. 'as fast as possible'), while controlling the eccentric action to avoid bouncing the weights. Strong verbal encouragement from the same two investigators was given throughout each protocol. Maximal voluntary contraction testing and measurements of blood lactate concentrations were taken before and after each protocol.

## 2.5 Before exercise

Upon entering the laboratory, participants were positioned upright on an isokinetic dynamometer (KinCom, Chattanooga, IL) ensuring the knee joint (i.e. lateral femoral epicondyle) and dynamometer axes were accurately aligned under contracted conditions, and stabilised using leg, waist, and chest straps to minimise movement during testing. The lever arm was attached to the shank 10-cm above the medial malleolus with an ankle strap. The seat height and distance from the axis were recorded to ensure accurate repeat positioning and to reduce day-to-day variation. During all tests, participants were required to have their arms crossed over their chest. All measurements were performed on the dominant limb (all right) with the knee joint angle fixed at 70° of flexion (0° corresponding to full knee extension).

Maximal voluntary contraction (MVC), voluntary activation (VA), and potentiated twitch (Pt) torque of the knee extensors were obtained to quantify muscle performance. Accordingly, the knee extensors were considered an appropriate muscle group to investigate because the HBD exercise is associated with greater peak knee moments when compared with the straight-barbell deadlift as the resistive load is positioned closer to the athlete's centre of mass (Swinton et al., 2011). A decline in MVC torque at exhaustion from before exercise levels indicates muscle fatigue, while adjustments in Pt and VA indicate contractile dysfunction or incomplete muscle activation. To determine MVC, participants performed three maximal isometric contractions of the knee extensors interspersed by 1-min rest after a standardised warm up of 10 submaximal repetitions. Strong verbal encouragement was given to all participants during each MVC to provide motivation. An electrical stimulus was delivered during the plateau of each MVC and 3-5 s following relaxation of each MVC. Potentiated twitches were used since these have been shown to be more sensitive to fatigue than un-potentiated twitches (Shield and Zhou, 2004). Two self-adhesive rectangular (5 x 9 cm) electrodes (Valutrode®, Axelgaard Manufacturing Co., Ltd, CA, USA) placed on

the leg connected to a high-voltage, constant current, stimulator (Digitimer Ltd, Stimulator model DS7AH, Welwyn Garden City, UK) which delivered pair rectangular pulses of 200 μs at 100 Hz. The electrodes were placed on clean, shaved skin 5-10 cm below the inguinal crease and 5-10 cm above the superior border off the patella over the belly of the *vastus lateralis, rectus femoris*, and *vastus medialis.* Briefly, direct muscle stimulation (as opposed to nerve stimulation) was employed since it may cause less discomfort and ensures delivery of a supramaximal stimulus (Place et al., 2010). Supramaximal stimulation was ensured by increasing stimulation intensity by 25 mA until a plateau occurred in twitch amplitude. The stimulation intensity was then increased by 25%. The same stimulation intensity was used for the same participant throughout the intervention. Voluntary activation was derived from the ratio of the superimposed twitch and the twitch produced in a relaxed potentiated muscle and expressed as:

$$VA = 100\ (1 - superimposed\ twitch\ /\ Pt)$$

[Eq. 1]

A correction was applied to the equation if the superimposed stimulation was delivered slightly before or after peak MVC (Strojnik and Komi, 1998). Next, a specific HBD warm-up was performed using a modified ARPE method (see Table 1). After the warm-up, participants were seated and given 3-min rest before the start of the protocol. During the rest period, capillary blood samples were obtained from the earlobe and collected into capillary tubes (20 μL) and placed into 1 mL haemolysing solution for assessment of blood lactate at rest (EKF Diagnostics, Biosen C-line, Germany).

2.6 During exercise

During the protocol, bar displacement data from each repetition of every set were collected using a linear positioning transducer (LPT) (GymAware, Kinetic Technology, Canberra, ACT). The LPT consisted of a central processing unit and a retractable,

measuring cable that was attached to the bar by a Velcro strap, and calibrated before each exercise session per the manufacturer's specifications. The GymAware used variable rate sampling with level crossing to detect data points that are down sampled to 50 Hz. During all testing, the LPT was connected to a tablet device to provide an instantaneous graphic display, which was shown to the participants to provide feedback on mean barbell velocity (m/s). Data acquisition was later obtained from the manufacturer's online portal software. In brief, from the time and displacement data, velocity can be calculated (i.e. displacement / time) and then acceleration (i.e. change in velocity / time). Outcome measures included: power (W), velocity (m/s), and total concentric work (kJ). The measurement error of the LPT is low and both relative and absolute reliability are within acceptable limits (Crewther et al., 2011).

### 2.7 After exercise

Within 1-2-min of task failure, MVC testing was repeated to capture the magnitude of fatigue induced by the exercise task before it dissipates. Lastly, ~5-min after exercise cessation a final capillary earlobe blood sample was taken.

### 2.8 Statistical analysis

From the three MVC measurements before and after the exercise protocol, the greatest MVC and respective potentiated twitch were selected for data analysis. Statistical analysis was carried out using SPSS 20.0 (IBM Corp, Armonk, NY). Differences in load lifted for the first set, adjusted load lifted (for subsequent sets), completed sets, total repetitions, mean repetition power output and velocity as well as total concentric work between the two protocols were analysed by paired t-tests. To compare differences between protocols a two-way repeated measures ANOVA was performed (protocol x time) for MVC, Pt, and VA. When the assumption of sphericity was violated, degrees of freedom were corrected (Greenhouse-Geisser). In case of a significant main effect a *post-hoc* test was applied. Significance level was 0.05. The critical value of P was two-tailed.

## 3. Results

### 3.1 Resistance exercise characteristics

The means, standard deviations, p value, effect sizes, and confidence intervals of the exercise characteristics of the HL and ML protocols are presented in Table 2. There were differences in post set 1 external load, number of sets, repetitions performed, total concentric work, and mean repetition power output and velocity. There was an interaction effect (protocol x time) ($F(1,7)=71.6$, $p=0.0001$) and main effect for time ($F(1,7)=46.7$, $p=0.0001$) for before and after blood lactate concentrations ($F(1,7)=71.6$, $p=0.0001$) (Table 3).

### 3.2 MVC, Pt, and VA measurements

Before, after, and 24-h maximal voluntary contraction, voluntary activation, and potentiated twitch torque from the HL and ML protocols are presented in Table 3.

There was a main effect of time for MVC ($F(2,14)=5.24$, $p=0.020$). The main effect of protocol for MVC was not significant ($p=0.530$). The interaction effect (time x protocol) was significant ($F(2,14)=6.91$, $p=0.008$).

A one-way repeated measures ANOVA was conducted to compare the effect of ML and HL exercise on MVC. There was a significant effect of ML on MVC ($F(2,14)=12.4$, $p=0.002$), but not HL ($p=0.441$). *Post hoc* Tukey's HSD tests demonstrated that for ML, MVC was reduced after exercise and at 24-h after exercise ($p=<0.05$).

In addition, one-way ANOVAs demonstrated that both Pt ($F(2,14)=6.26$, $p=0.017$) and VA ($F(2,14)=4.51$, $p=0.049$) were reduced across the time periods for ML exercise, but Pt ($p=0.191$) and VA ($p=0.300$) were unchanged across time periods for HL exercise. *Post hoc* Tukey's HSD tests demonstrated that at each time point for ML exercise, Pt was reduced after exercise only and VA was reduced after exercise and at 24-h after exercise ($p=<0.05$).

**Table 2. Load lifted, completed sets and repetitions, power, velocity, and total concentric work for high-load and moderate-load**

| | HL | ML | *t* | df | *p* | *d* | 95% CI |
|---|---|---|---|---|---|---|---|
| Set 1 load (kg) * | 161.3 ± 24.5 | 142.2 ± 12.7 | 17.4 | 7 | .0001 | 0.98 | -0.1 – 2.0 |
| Adjusted load (kg) * | 170.9 ± 27.1 | 150.9 ± 17.8 | 11.7 | 7 | .001 | 0.87 | -0.2 – 1.8 |
| Completed sets * | 9.5 ± 5.6 | 4.3 ± 2.6 | 3.2 | 7 | .01 | 1.19 | 0.1 – 2.2 |
| Total repetitions * | 33.4 ± 16.7 | 52 ± 23.7 | 3.2 | 7 | .02 | -0.91 | -1.9 – 0.2 |
| Power (W) * | 550.3 ± 111.4 | 647.6 ± 50.5 | 2.4 | 7 | .049 | -2.0 | -2.1 - 0.0 |
| Velocity (m/s) * | 0.34 ± 0.05 | 0.45 ± 0.06 | 2.3 | 7 | .001 | -2.0 | -3.1 – 0.7 |
| Total concentric work (kJ) * | 18.1 ± 8.2 | 29.8 ± 13.8 | 2.8 | 7 | .0253 | -2.0 | -3.1 – -0.7 |

Values are mean ± SD before, after, 24-h, $n = 8$
* Significant effect of protocol
*d* = Cohen'd *d* effect size; HL = high-load-low-repetitions; ML = moderate-load-high-repetitions; kg = kilograms; W = watts; m/s = metres per second; kJ = kilojoules

**Table 3. Before, after, and 24-h assessment values from high-load (HL) and moderate-load (ML)**

| | | HL | ML |
|---|---|---|---|
| MVC (N.m) [a) b)] | Before | 1138 ± 294 | 1200 ± 247 |
| | After | 1079 ± 306 | 1020 ± 272* |
| | 24-h | 1097 ± 274 | 1054 ± 273* |
| Pt (N.m) | Before | 598 ± 121 | 623 ± 118 |
| | After | 551 ± 107 | 504 ± 100* |
| | 24-h | 595 ± 101 | 582 ± 122 |
| VA (%) | Before | 81 ± 10 | 90 ± 10 |
| | After | 78 ± 11 | 79 ± 19* |
| | 24-h | 80 ± 11 | 81 ± 10* |
| Blood lactate (mmol/L) [a) b)] | Before | 2.8 ± 0.8 | 2.8 ± 1.3 |
| | After | 4.9 ± 2.5 | 12.7 ± 2.5* |

Values are mean ± SD before, after, 24-h, *n* = 8.
a) Significant main effect from before to 24-h, p=<0.05
b) Significant interaction effect (protocol x time), p=<0.05
* Within protocol post-hoc difference compared with before-exercise values
MVC = maximal voluntary contraction; Pt = potentiated twitch torque; VA = voluntary activation

## 4. Discussion

Performance fatigability relates to the contractile capabilities of the working muscle and capacity of the nervous system to provide adequate activation signal to maintain the task. This is the first study to characterise changes in performance fatigability and recovery after performing open-loop exhaustive, hexagonal-barbell deadlift (HBD) exercise at different loads in resistance-trained men. As expected there were differences between the moderate-load high-repetition (ML) and high-load low repetitions (HL) protocols in sets and repetitions performed, power output, barbell velocity, and total concentric work performed. The main finding was that intensity of load and rest period modification influenced contractile function and voluntary activation (VA). Muscle fatigue due to reduced contractile function and VA was present after moderate-load HBD exercise, but not after high-load HBD exercise indicated by preserved MVC torque. Additionally, maximal voluntary contraction (MVC) torque was reduced at 24-h after ML exercise, which was attributed to suppressed voluntary activation (VA). The fact that MVC was preserved after HL HBD exercise has significant implications for the prescription of periods of intensified resistance exercise within athletic training programmes.

In this study, performance fatigability was quantified by comparing MVC torque at exhaustion and 24-h after exercise with before exercise-values. The ML protocol induced a 15 ± 11% reduction in MVC torque at exhaustion. This result is consistent with previous findings after single bouts of lower-body multi-joint resistance exercise. For example, Brandon et al. (2015) reported 13 and 9% reductions in MVC after ‘heavy’ (equivalent to muscle active RPE 16-17) and ‘moderate’ (75% of heavy session load) barbell back squats (ten sets of 5 repetitions). Additionally, Howatson et al. (2015) reported an 11% loss of MVC torque for males and females after a ‘strength session’ (equivalent to muscle active RPE 16-17) comprised of barbell back squats, split squats, and upper-body press (four sets of 5 repetitions for each exercise).

The fact that MVC was reduced after ML exercise and not HL is surprising, but may be associated with the lower concentric work (-39 ± 35%) performed during HL exercise suggesting lower metabolic stress. Additionally, it has been demonstrated that when volume-equated resistance exercise is performed load and rest period duration has greater influence on MVC (McCaulley et al., 2009). As well, blood lactate concentrations were higher after ML HBD exercise compared with HL, which was most likely due to the shorter rest period (1.5-min versus 3-min) between sets. As such, ML exercised seemed to cause greater disturbance to homeostasis resulting in greater losses of MVC torque.

In addition to MVC testing, this study employed direct muscle stimulation techniques to gain insight into the rate-limiting adjustments responsible for task failure during resistance exercise. Briefly, when VA is incomplete, the stimulus activates the motorneurons that were not recruited or firing fast enough and augments the torque generated by the muscle. Conversely, when there is complete or nearly complete VA, there is little to no increase in torque when the stimulus is delivered (Enoka and Duchateau, 2008, Merton, 1954). Also, a decline in potentiated twitch torque (Pt) amplitude measured ~5-s after an MVC indicates impairment of contractile function. Briefly, limitations of the interpolated twitch technique (ITT) are recognised. For example, the accuracy of the ITT is limited when the electrical stimuli delivered during the voluntary activation test is triggered manually (at the point the investigator perceived to be peak torque) while watching the participant's real-time torque curves. In addition, it was rare for the stimulus to be delivered at peak torque because MVCs are characteristically somewhat unsteady. Therefore, some measurement error is typically present in estimates of VA regardless of the equations used.

The -18 ± 14% decrease in potentiated twitch torque (Pt) after ML HBD exercise demonstrates that the ability of the muscle to translate the neural stimulus into a force response was impaired (Ross et al., 2010b). The accumulation of metabolites such as

the elevations in blood lactate observed has been associated with blunted $Ca^{2+}$ release or altered sensitivity of the myofibril complex to $Ca^{2+}$ which may be responsible for this impaired excitation-contraction coupling process (Allen et al., 2008). It is also evident that an impairment of neural drive, indicated by reduced VA, was also elicited by ML resistance exercise. The -13.1 ± 14.4% reduction in VA observed in this study has not previously been detected by electrical stimulation techniques after multi-joint resistance exercise (Howatson et al., 2015, Brandon et al., 2015). This may be partially explained by the open-loop task design employed in this study, which allowed participants to exercise until volitional exhaustion or task failure. It has been suggested that reduced VA after exhaustive exercise may serve to protect the neuromuscular system because continuing to drive the muscles would put them in a catastrophic state (Noakes, 2012). Indeed, group III and IV muscle afferents (stimulated by muscle damage and inflammation) can act at a supraspinal level with inhibitory effects on VA after contractile dysfunction exceeds a 'critical threshold' (Amann and Dempsey, 2008). So, the impairments to contractile function may have, via afferent feedback, reduced motor unit firing to avoid increasing damage to the muscle fibres.

In this study, the recovery of fatigue was assessed by repeating MVC testing 24-h after exercise. Maximal voluntary contraction remained suppressed for the ML protocol (-12.8 ± 7.7%) at 24-h after exercise compared with before exercise-values. These findings are greater than previous reported findings (i.e. -6%) (Howatson et al., 2015) and may be related to the exhaustive nature of the bout. The partial recovery was associated with a near return to before exercise Pt amplitude, indicating recovery of contractile function. Accordingly, the incomplete recovery of MVC torque may be explained by the persistence of reduced VA (-10.1 ± 8.3%) after ML HBD exercise. The finding that contractile function was recovered after 24-h of rest, while VA remained depressed after this time, is indicative that a form of persistent centrally mediated impairment. Similar findings have been reported up to 60-h after consecutive

repetitive bouts of prolonged cycling exercise (Ross et al., 2010b). Specifically, after 20 prolonged cycling stages interspersed by 2 rest days, peripheral neuromuscular function was restored, while central nervous system function was still impaired. Although the chronic changes in the ability to produce voluntary force occurred after consecutive bouts of prolonged exercise, this study demonstrates that these changes are present after a single bout of exhaustive resistance exercise. Therefore, it may be relevant to emphasise recovery of nervous system function as in some cases this may take longer to recover than contractile function (Rattray et al., 2015).

This study provides novel information on resistance exercise prescription for resistance-trained males and suggests that athletes undertaking a bout of moderate-load high repetition HBD exercise induces greater losses in MVC torque compared to high-load low repetition HBD exercise, which may require a period greater than 24-h to observe complete recovery of fatigue. Additionally, recovery of nervous system function should be prioritised as VA was slower to recover than contractile function (potentiated twitch torque) (Rattray et al., 2015). An important finding was that high-load HBD exercise results in no significant loss to MVC torque, even when performed until task failure. This is interesting for athletes and coaches who are planning in-season concurrent training programmes or periods of intensified training.

To the author's knowledge, this is the first study that has employed HBD exercise at different loads in an open-loop design to examine changes in contractile function and voluntary activation with recovery measurements after resistance exercise. The HBD exercise was chosen because of its similar biomechanical and neuromuscular demands with several athletic movements (e.g. knee and hip extension during running and jumping), but its relative safety and technical simplicity compared to a straight-barbell deadlift and squat variations. In addition, the knee extensors were considered an appropriate muscle group to investigate because the HBD exercise is associated with greater peak knee moments when compared with the straight-barbell deadlift as

the resistive load is positioned closer to the athlete's centre of mass (Swinton et al., 2011). For these reasons, it is a common exercise employed in athletic training programmes to strengthen the legs, hips, back, and trunk musculature, while providing an excellent evaluation of lower body strength. The protocol employed in this study was designed to align with common practice within athletic training prescription. For example, the HL protocol included sets of 3 repetitions performed at 90% of 3RM, with 3-min rest, whereas the ML protocol included sets of 10 repetitions performed at 75% of 3RM, with 1.5-min rest. It is acknowledged that the exhaustive nature of the task is uncommon, but can be related to periods of intensified training. With this in mind though, the total number of sets and repetitions performed were similar to previous investigations that utilised a closed-loop task design (McCaulley et al., 2009). For instance, in this study participants performed barbell back squats consisting of 11 sets of 3 repetitions at 90% 1RM and 4 sets of 10 repetitions at 75% 1RM. This may be explained by the fact that the investigators reduced the resistive load by 5% when participants reached failure within a set.

**Practical applications**

One practical application that can be derived from these results is that because of lowered fatigue after high-load HBD exercise, it may be a preferable training modality for coaches to employ during the in-season period or during times of intense concurrent training. In this way, optimal adaptations can be maintained whilst minimising the negative impact of residual fatigue from resistance exercise. Further research may reveal if this remains the case between equated volumes of HL and ML training such as 10 sets of 3 repetitions versus 3 sets of 10 repetitions training schemes.

**Conclusion**

In conclusion, this study provides important information for athletes, coaches, and practitioners seeking to use the HBD exercise and training to mechanical failure. A

single bout of exhaustive moderate-load HBD exercise induces loss of MVC torque that is persistent even after a period of recovery. The loss of MVC torque can be attributed to impaired contractile function and reduced VA. Also, reduced VA was observed after 24 h, while potentiated twitch torque was largely restored; indicating VA was slower to recover than contractile function. The fact that fatigue was lowered after HL HBD exercise has important implications for the prescription of resistance exercise.

# Chapter 4. Changes in contractile function and voluntary activation during multi-joint resistance exercise performed with fast and slow lifting tempos

## Abstract

***Purpose:*** To examine changes in contractile function and voluntary activation during resistance exercise performed with maximal effort or fast tempo (FT) and slower tempo (ST). ***Methods:*** Eight resistance trained males completed both FT and ST hexagonal-barbell deadlift (HBD) exercise protocols each consisting of eight sets of 6 repetitions at 60% 3RM using a randomised cross-over design. During the FT protocol, each repetition was performed with maximal effort, while each repetition during ST was performed with a 3-1-3 lifting tempo. Changes in maximal voluntary contraction (MVC), voluntary muscle activation (VA), evoked potentiated twitch torque (Pt) of the quadriceps were determined using the twitch interpolation technique before, during (after set 4), and after exercise. Bar displacement data were measured during the exercise protocol. ***Results:*** Mean power output (p=0.0001), barbell velocity (p=0.0001), and total concentric work (p=0.0146) were higher for the FT protocol compared to the ST protocol (995 ± 166 vs 233 ± 52 W; 0.87 ± 0.05 vs 0.19 ± 0.05 m/s; 4.8 ± 0.8 vs 3.7 ± 1.1 kJ). MVC torque (p=0.0001), Pt (p=0.013), and VA (p=0.037) were reduced compared to before exercise values after FT (-7.8 ± 9.2%; -5.2 ± 9.2%, -8.7 ± 12.2%) and ST (-11.2 ± 8.4%, -13.3 ± 8.1%, -2.2 ± 4.2%). Blood lactate concentrations were greater after ST (8.6 ± 3 mmol/L) compared with FT (4.2 ± 2.5 mmol/L) (p=0.0001). ***Conclusion:*** Changes in contractile function and voluntary activation similar after both fast tempo and slow tempo HBD exercise (8 sets of 6 repetitions at 60% 3RM). Future studies may reveal if this remains the case when performing greater resistance exercise volumes.


## 1. Introduction

It is well accepted that resistance exercise is a potent stimulus for developing skeletal muscle mass and strength (Folland and Williams, 2007). Additionally, manipulation of resistance training variables such as load and repetition duration or contraction velocity stimulate specific adaptations (Schoenfeld et al., 2015). For instance, when lifting submaximal loads such as below 85% 1RM, an individual can vary the lifting tempo of each repetition for a given exercise. It has been proposed that intentionally slowing repetition cadence reduces the momentum in a lift, thereby increasing mechanical tension on the working muscles (Westcott et al., 2001). Hypothetically, increasing mechanical tension throughout a lift could positively mediate intracellular anabolic signalling, promoting a greater hypertrophic response (Schoenfeld et al., 2015). In contrast, maximum effort resistance exercise in which the athlete moves the resistive load with maximum effort (i.e. velocity) has been shown to drive specific

adaptations such as enhanced rate of force development, which is crucial for improving general and sports specific skills such as running, jumping, and throwing (Newton et al., 1997). Performing bouts of resistance exercise will induce muscle fatigue and ensuring that fatigue is appropriately managed is important for adaptations to training and competition performance as well as reducing injury risk (Hulin et al., 2016). Therefore, knowledge of the development of fatigue after resistance exercise performed with different lifting tempos has important implications for the prescription of athletic training programmes as the magnitude of fatigue will dictate the nature of subsequent training bouts.

Previous research that has measured changes in contractile function and voluntary muscle activation (VA) during resistance exercise has largely focused on the influence of load intensity. Yet many of these studies have not controlled for volume load (VL), defined as the product of the total number of repetitions performed and the corresponding load lifted (Benson et al., 2006, Brandon et al., 2015, Hakkinen, 1993, Hakkinen, 1994, Howatson et al., 2015). Additionally, very few studies have examined the influence of lifting tempo (i.e. contraction velocity) (Tran et al., 2006). With this in mind, Tran et al. (2006) studied the effects of manipulating lifting tempo and therefore the time the muscle is under tension (TUT) as well as VL for elbow flexion exercise. The study conducted in males consisted of three fatiguing protocols: the first was defined as high-volume (three sets of 10 repetitions) and long (5 s per repetition) concentric TUT; (ii) in the second protocol participants completed the same high-volume load but with low (40%; i.e. 2 s per repetition) of the concentric TUT compared to the first protocol; whereas the in third protocol participants performed low-volume load (50%) but with equal TUT (i.e. 10 s per repetition) compared to the first protocol. The findings of the study were that all three session induced reductions in MVC torque (-19 ± 1.9%, -13 ± 1.6%, -15 ± 2.8%) that were associated with contractile dysfunction indicated by reduced potentiated twitch (Pt) amplitude (-57 ± 5.1%, -12 ± 6.5%, -30 ±

8.6%). As well, initial VA was unchanged after each protocol indicating preserved neural drive. As such, the low-volume long TUT protocol resulted in greater reductions in contractile function than high-volume, short TUT. These findings suggest that prolonged tension elicits greater contractile stress, resulting in greater contractile dysfunction compared with volume load.

The study by Tran et al. (2006) provides valuable insights into changes to contractile function and VA during resistance exercise and suggests that TUT is more influential than volume load. These findings are in agreement with previous observations that showed resistive load was more influential than volume of work completed (McCaulley et al., 2009). However, the use of a single-joint exercise (i.e. elbow flexions) is unrepresentative of primary exercises employed in athletic training programmes and therefore investigating contractile function and VA after multi-joint exercises, which may cause larger disturbances to general homeostasis may be of more interest and warrants further investigation. This study aims to describe the influence of a single, structured bout of volume load controlled slow tempo training (ST) and fast tempo training (FT) on changes in contractile function and voluntary activation. Changes in MVC, VA, and Pt measurements were made before, during, and immediately after exercise. It was hypothesised that both protocols would induce muscle fatigue, however the ST protocol would induce larger fatigability due to longer TUT.

## 2. Method

### 2.1 Participants

Eight resistance-trained males (means ± SD; age 23 ± 2.7 years, height 1.77 ± 0.04 cm, 79.5 ± 6.9 kg) who had been engaging in resistance exercise for at least 2 yr and training > 2 sessions per week volunteered to participate in this study. All participants were well accustomed to performing the hexagonal barbell deadlift (HBD) with correct technique and laboratory exercise testing and, were free of cardiorespiratory, neurological, or neuromuscular disorders. Initial HBD 3 repetition maximum (3RM) was 192 ± 19.3 kg (2.4 ± 0.2 normalised per kg of body mass). All participants were informed of the purpose of the study, experimental procedures, and associated risks prior to participation and exercise testing. All participants gave verbal and written informed consent, which was approved by the University of Essex ethics committee, in the spirit of the Helsinki Declaration.

### 2.2 Experimental design

A within-group repeated measures design in which participants performed two protocols, ST and FT, in a randomised and counterbalanced order separated by 5-8 d was employed. For each protocol, before, during (after set 4) and after exercise measurements of MVC torque, voluntary muscle activation, potentiated twitch torque, and blood lactate concentrations were included. Three to seven days prior to the start of the study participants completed muscular strength testing to evaluate each participant's 3RM performance for the HBD exercise. On the same day, participants completed a familiarisation session. Participants were instructed to avoid any lower-body exercise 48 h prior to any visit and any exercise the day before a visit.

### 2.3 Muscular strength testing and familiarisation

Maximal strength testing was conducted using the protocol described in the previous experimental chapter. After strength testing, participants were accustomed with all laboratory procedures. Particular attention was paid to the maximal isometric voluntary

contraction (MVC) of the knee extensors, with participants repeating the procedure until they were able to maintain a stable plateau in torque for 3-4 s. As well, participants were also fully familiarised with direct muscle stimulation and care was taken to ensure participants could match MVCs produced without stimulation with those while expecting stimulation (Shield and Zhou, 2004).

2.4 Exercise protocol

Resistance exercise tempo is expressed as three digits where the first number represents time (s) to complete the concentric action, the second number is the isometric transition phase between concentric and eccentric actions, and the third is the time to complete the eccentric action. The slow tempo (ST) protocol was performed with a tempo of 3-1-3, while the fast tempo (FT) was performed with maximal effort while controlling the eccentric action to avoid bouncing the weights. For both protocols eight sets of 6 repetitions with 2-min recovery between sets were performed at 60% of 3RM. During the ST protocol, participants were instructed to keep time with a metronome and/or timer displayed via a personal laptop; while a linear positioning transducer (LPT) (GymAware, Kinetic Technology, Canberra, ACT) connected to a tablet device displaying real-time feedback of mean barbell velocity for each repetition was shown to participants during the FT protocol. Measurements of blood lactate concentrations and muscle force were taken before, during (after set 4), and after each protocol.

2.5 Before exercise

Maximal voluntary contraction, voluntary muscle activation (VA), and potentiated quadriceps twitch (Pt) torque were obtained to quantify muscle performance as previously described in the previous chapter. After MVC testing, a specific warm up for the HBD exercise was conducted by performing two sets of 10 repetitions, first with the empty barbell (40kg) and second with 50% of 3RM, followed by a single set of 6 repetitions at 75% of 3RM. After the warm-up, participants were seated and given 3-

min rest before the start of the protocol. During the rest period, capillary blood samples were obtained from the earlobe and collected into capillary tubes (20 µL) and placed into 1 mL haemolysing solution for assessment of blood lactate at rest (EKF Diagnostics, Biosen C-line, Germany).

2.6 During exercise

Muscle function was assessed midway during the protocol after set 4, where a single, acceptable MVC was performed to minimise the effect of fatigue. In addition, throughout the protocol, bar displacement data from each repetition of every set was measured using the LPT previously described.

*2.7* After exercise

Within 1-2-min of task failure, MVC testing was repeated to capture the magnitude of fatigue induced by the exercise before it dissipates (Froyd et al., 2016). Lastly, ~5-min after exercise cessation a further capillary earlobe blood sample was taken.

2.8 Statistical analysis

From the three MVC measurements before and after the exercise protocol, the greatest MVC and respective twitch were selected for data analysis. Statistical analysis was carried out using SPSS 20.0 (IBM Corp, Armonk, NY). Differences between the two protocols were analysed by paired t-tests. To compare differences between protocols a two-way repeated measures ANOVA was performed (protocol x time) for MVC, Pt, and VA. When the assumption of sphericity was violated, degrees of freedom were corrected (Greenhouse-Geisser). In case of a significant interaction effect a *post-hoc* test was applied. Significance level was 0.05. The critical value of P was two-tailed.

## 3. Results

### 3.1 Resistance exercise characteristics

The means, standard deviations, p value, effect sizes, and confidence intervals of the exercise characteristics of the FT and ST protocols are presented in Table 4. There were differences in mean power output, velocity, total concentric work, and repetition height.

### 3.2 MVC, Pt, and VA measurements

Before, during, and after MVC, Pt, VA, and blood lactate concentration values from the FT and ST protocols are presented in Table 5.

Main effects of time were found for blood lactate ($F(1.1,14)=51.2$, $p=0.0001$), MVC ($F(2,14)=15.69$, $p=0.0001$), Pt ($F(1.1,7.6)=9.94$, $p=0.013$), and VA ($F(2,14)=2.22$, $p=0.037$). *Post hoc* tests revealed that MVC declined during ($p=0.046$) and after exercise ($p=0.007$). Pt declined after exercise only ($p=0.034$). Blood lactate increased during ($p=0.0003$) and after exercise ($p=0.001$). There was an interaction effect (protocol x time) for blood lactate ($F(1.14,14)=14.31$, $p=0.0001$), but not for MVC $p=0.929$, Pt $p=0.061$ or VA $p=0.194$.

**Table 4. Power output, velocity, total concentric work, and repetition height for fast tempo (FT) and slow tempo (ST)**

| | FT | ST | *t* | df | *p* | *d* | 95% CI |
|---|---|---|---|---|---|---|---|
| Power (W)* | 995.4 ± 166.2 | 232.6 ± 51.9 | 7 | 13.8 | .0001 | 6.2 | 3.6 – 8.1 |
| Velocity (m/s)* | 0.87 ± 0.05 | 0.19 ± 0.05 | 7 | 34.1 | .0001 | 13.6 | 17.4 – 8.3 |
| Total concentric work (kJ)* | 4.57 ± 0.8 | 3.65 ± 1.1 | 7 | 3.4 | .0146 | .99 | -0.1 – 2 |
| Rep height (m)* | 0.53 ± 0.03 | 0.38 ± 0.04 | 7 | 10.24 | .0001 | 4.2 | 2.3 – 5.7 |

Values are mean ± SD before, after, 24-h, $n = 8$
* Significant effect of protocol
*d* = Cohen's *d* effect size; FT = fast tempo; ST = slow tempo; W = watts; m/s = metres per second; kJ = kilojoules; m = metres

**Table 5. Before, after, and 24-h assessment values for fast tempo (FT) and slow tempo (ST)**

| | | FT | ST |
|---|---|---|---|
| MVC (N.m) [a)] | Before | 1317 ± 303 | 1277 ± 250 |
| | During | 1238 ± 289* | 1196 ± 288* |
| | After | 1202 ± 255* | 1139 ± 281* |
| Pt (N.m) [a)] | Before | 640 ± 77 | 645 ± 74 |
| | During | 605 ± 58 | 602 ± 66 |
| | After | 602 ± 44* | 557 ± 67* |
| VA (%) [a)] | Before | 91 ± 13 | 90 ± 13 |
| | During | 83 ± 14 | 86 ± 17 |
| | After | 83 ± 17 | 88 ± 14 |
| Blood lactate (mmol/L) [a) b)] | Before | 1.9 ± 0.7 | 2 ± 0.9 |
| | During | 3.5 ± 1.7* | 7 ± 2.7* |
| | After | 4.2 ± 2.5* | 8.6 ± 3* |

Values are mean ± SD before, during, and after, *n* = 8.
a) Significant time effect from before to after, p=<0.05
b) Significant interaction effect (protocol x time), p=<0.05
* Within protocol post-hoc difference compared with before-exercise values
MVC = maximal voluntary contraction; Pt = potentiated twitch torque; VA = voluntary activation

## 4. Discussion

This is the first study to characterise performance fatigability during volume load equated hexagonal-barbell deadlift (HBD) exercise performed with different lifting tempos in resistance-trained men. As expected there were differences between the fast tempo (FT) and slow tempo (ST) protocols in power output and barbell velocity. The main finding was that a single, structured bout of FT and ST HBD exercise resulted in a progressive decline in MVC torque, which was associated with impairments of both voluntary activation and contractile function. This study provides novel information on resistance exercise prescription for resistance-trained males and suggests that undertaking FT and ST training may have similar influences on contractile function and voluntary activation (VA).

In this present study, changes in MVC torque, VA, and contractile function were measured during (after set 4) and immediately after exercise with before exercise levels. Both FT and ST induced declines in MVC torque (-5.7 ± 8.4% vs -6.2 ± 11.8%) after the fourth set, while FT resulted in a -7.8 ± 9.2% reduction at exercise cessation compared with -11.2 ± 8.4% after the ST protocol. These findings are slightly lower than previous studies that report ~ -13-19% reductions in MVC after single-bouts of elbow flexions of varying loads and time-under-tension (TUT) (Tran et al., 2006), but are similar to observations made after lower-body multi-joint resistance exercise (~ -9–13%) (Brandon et al., 2015, Howatson et al., 2015).

Interestingly, contractile function was not significantly impaired during (fourth set) FT and ST HBD exercise. However, the progressive reduction in potentiated twitch torque (Pt) (-5.2 ± 9.2% vs -13.3 ± 8.1) after FT and ST HBD exercise demonstrates that neuromuscular transmission leading to muscle contraction was impaired after exercise only (Ross et al., 2010b). In addition to contractile dysfunction, impairment of nervous system function was observed after both protocols. Maximal voluntary activation (VA) (~ 90%) of the quadriceps in the non-fatigued state was in line previous observations

(Ross et al., 2010b, Stoter et al., 2016). Voluntary activation was reduced after both FT and ST HBD exercise suggesting a reduced number of motorneurons were voluntarily recruited. Fast and slow tempo HBD exercise resulted in after exercise impairments of VA (-8.7 ± 12.2% vs -2.2 ± 4.2%). The reductions in VA observed in this study have not previously been detected with electrical stimulation techniques after maximal effort multi-joint resistance exercise (Brandon et al., 2015, Howatson et al., 2015). It has been suggested that reduced VA during exercise may serve to protect the neuromuscular system via muscle afferent feedback systems (Amann and Dempsey, 2008). This may readily explain the impairment of VA after ST, however the comparatively low after exercise blood lactate concentrations observed after FT suggest a different mechanism may be involved. Speculation about the mechanism responsible for impaired VA after this type of exercise is difficult as no data currently exists. However, it has been proposed that the high-frequency motor-unit firing pattern associated with performing movement with maximal intent may be related to impairment of nervous system function (Behm and Sale, 1993). Clearly, more work is required and future studies aimed at identifying the mechanisms responsible for impaired VA after fast tempo training is recommended.

As expected there were differences between the FT and ST protocols in power output and barbell velocity. Interestingly, although the resistance exercise was volume controlled, there was a difference in total concentric work performed between the protocols. This may be explained by the method used to calculate concentric work by the linear positioning transducer used for this study. Briefly, the LPT calculates mechanical work by multiplying the force required to move the load, by the distance travelled by the load. In practice, it is assumed that the force involved is equal to the load being lifted and that all repetitions are performed with the same range of motion and so mechanical work may be estimated by multiplying the load by the number of repetitions, referred to as volume load (Painter et al., 2012). However, during the FT

protocol, due to the nature of lifting with maximum intent, the increased momentum of the load frequently lifted the participant's heels from the ground. So, the increased distance travelled by the load explains the difference in total concentric work performed.

The exercise in this study was characterised by multi-joint resistance exercise performed using a hexagonal-barbell deadlift (HBD) with different tempo schemes. The protocol design employed in this study comprised of 8 sets of 6 repetitions performed at 60% of 3RM, with 2-min rest. Importantly, the FT protocol was performed with maximal effort, whereas the ST protocol was performed with a 3-1-3 tempo producing longer time-under-tension per repetition. These schemes were designed to align with common practice within athletic training prescription. For example, although much debate exists regarding the mechanisms that promote skeletal muscle growth after resistance training. (Campos et al., 2002, Kraemer and Ratamess, 2005, Morton et al., 2016, Schoenfeld, 2013, Tanimoto and Ishii, 2006, Tanimoto et al., 2008), slow tempo (3-1-3) training conducted with moderate-loads ~50% 1 repetition maximum (RM) is as effective for muscular hypertrophy and strength gains as normal tempo training (1-0-1) conducted with moderate-loads ~80% 1RM in single-joint (Tanimoto and Ishii, 2006) and multi-joint (Tanimoto et al., 2008) resistance exercise. Practitioners may prescribe slow tempo training during rehabilitation programmes or to develop connective tissue strength as well as improvng control and body awareness. In contrast, resistance training with intention to move the load with maximal effort compared to slow tempo training has been shown to drive specific adaptations such as enhanced rate of force development (Behm and Sale, 1993) and greater increases in jump and sprint performance (Smith and Melton, 1981).

The main limitation of this study and other twitch interpolation studies is the number of factors that may affect the accuracy of the twitch interpolation technique contributing to some measurement error typically present in estimates of VA. Additionally,

measurement of contractile function and VA at 24 hours after exercise may have allowed useful conclusions on the recovery of these outcomes after performing this type of exercise. However, a follow up measurement was not considered due to the non-exhaustive nature of the study design and low reductions in MVC expected allowing for full recovery at 24 hours after exercise. Future studies should address the effect of structured bouts of consecutive concurrent training.

**Practical applications**

One practical application that can be derived from these results is that changes in voluntary activation and contractile function are similar after performing eight sets of 6 repetitions of FT and ST HBD exercise. Future studies may reveal if this remains the case when performing greater resistance exercise volumes. Additionally, more work is needed to identify the mechanisms responsible for loss of nervous system function after fast tempo resistance exercise.

**Conclusion**

In conclusion, this study provides novel information for athletes, coaches, and practitioners seeking to use the hexagonal-barbell deadlift (HBD) exercise. Both impairment of voluntary activation and contractile dysfunction contributed to the performance fatigability observed after fast tempo and slow tempo HBD exercise. This work provides further understanding on the influence of resistance exercise on performance fatigability and may help coaches with the preparation of athletic training programmes.

# Chapter 5. Thesis summary and recommendations

## 1. Thesis summary

Athletes perform bouts of resistance exercise to enhance athletic performance and reduce the risk of injury (Suchomel et al., 2016). The hexagonal-barbell deadlift (HBD) exercise is commonly employed by strength and conditioning practitioners to develop strength and skeletal muscle mass in athletes, however there are no available data that describe the changes to contractile function and voluntary activation (VA) associated with performing this exercise. An important outcome of athletic training is to ensure that fatigue is appropriately managed, and therefore the aim of this thesis was to study the influence of resistance exercise on contractile function and VA using methods employed from previous fatigue and human performance research. These were used to conduct novel studies on high-load and moderate-load resistance exercise as well as volume-load equated slow and fast tempo resistance exercise in resistance-trained males. This thesis contributes to a knowledge-base for coaches to make evidence-informed decisions regarding athletic training programmes.

The first chapter provided a broad overview of the literature relating to fatigue and human performance. From this review, it was evident that a well-established literature exists relating to the influence of single-joint and locomotor exercise such as running and cycling on fatigue and fatigability. However, studies relating to performance fatigability after resistance exercise are generally limited by the assessment model employed such as surface electromyography (EMG). For instance, increases in EMG amplitude would suggest that more motor units are recruited or are firing faster. However, local peripheral factors such as changes to membrane potential also change the EMG and so the contribution of central mechanisms to fatigue cannot be assumed solely from changes in EMG amplitude. Furthermore, understanding of the type of fatigue has important implications for the prescription of recovery interventions. In this context, very few studies have employed more robust techniques such as nerve or direct muscle electrical stimulation. From these studies, it was evident that a paucity

of literature existed relating to the influence of multi-joint resistance exercise performed until mechanical failure or with different concentric and eccentric tempos on VA and contractile function. For these reasons, two experimental studies were conducted which importantly employed electrical stimulation techniques to evaluate these outcome measures.

Chapter three presents a novel study that sought to examine the influence of an exhaustive bout of multi-joint resistance exercise on contractile function and voluntary activation in resistance-trained males. The study also examined the influence of load and rest period modification by comparing a traditional high-load low repetition (HL) protocol (3-min recovery) with a moderate-load (ML) high repetition protocol (1.5-min recovery). Additionally, the study is the first to employ a hexagonal-barbell deadlift (HBD) exercise, which is commonly used by strength and conditioning practitioners. The main finding of the study was that disruption to contractile function and voluntary activation were observed after ML HBD exercise, but not after HL exercise. Additionally, after a 24-h recovery period, MVC torques remained suppressed. Interestingly, the partial recovery of MVC was due a near return to before exercise levels of contractile function and therefore the incomplete recovery was due to an impairment of voluntary activation.

Chapter four again presents a novel study that employed the HBD exercise and measures of contractile function and VA. In this study, the influence of a structured bout of volume load-equated HBD exercise with manipulation lifted tempo was examined. Modification of lifting tempo was chosen as this is commonly manipulated within athletic training programmes to drive specific adaptations such as enhanced rate of force development or to promote greater hypertrophic gains. The main finding of the study was reductions in contractile function and VA were similar after performing fast or slow tempo HBD exercise. However, more work is needed to see if this remains the case with greater resistance exercise volumes.

## 2. Practical applications

Strength and conditioning practitioners and coaches may consider the following recommendations of how to apply these findings when preparing athletic training programmes:

1. Loss of MVC torque due to changes in contractile function and voluntary activation are expected after moderate-load HBD exercise, but not high-load exercise.
2. When conducting ML HBD exercise to exhaustion, a period of more than 24-h is likely required to observe full recovery, which is largely attributed to impairment of voluntary activation.
3. Selection of fast or slow tempo volume load-equated HBD exercise (8 sets x 6 repetitions) results in similar changes to contractile function and voluntary activation.

## 3. Recommendations for future research

The research presented in this thesis has developed understanding on the influence of resistance exercise on the development and recovery of voluntary muscle activation and contractile function. However, a range of research questions remain to be answered to further understand the preparation of athletic training programmes. The following areas require further exploration:

1. While the influence of individual locomotor and resistance exercise sessions on contractile function and VA are understood, more information is required to establish the effect of consecutive bouts of concurrent training.
2. Fatigue is a multi-dimensional construct, and while much is known on performance fatigability, more research is needed to establish the influence of resistance exercise on fatigue and perceived fatigability.

3. While the influence of load is well-established after resistance exercise, further research is required to establish the differences between equated volumes of high-load and moderate load exercise such as 10 sets of 3 repetitions versus 3 sets of 10 repetitions training schemes.
4. More work is needed to reveal if contractile function and VA are similar when a greater volume of fast and slow tempo volume-equated exercise is performed

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