# FAILURE AND NON-FAILURE RESISTANCE EXERCISE IN TRAINED INDIVIDUALS

*by*

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# STATEMENT OF AUTHENTICATION

The work presented in this thesis is, to the best of my knowledge and belief, original except as acknowledged in the text. I hereby declare that I have not submitted this material, either in full or in part, for a degree at this or any other institution.

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(Signature)

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# ABSTRACT

The rate and magnitude of muscular strength and power improvements are reduced the longer an individual is engaged in consistent moderate to high intensity resistance exercise training. It is therefore thought that trained individuals need to 'work harder' by performing resistance exercise to failure to evoke a large increase in acute fatigue and optimise improvements in muscular strength and power following a period of training. Previous literature has demonstrated that performing resistance exercise to failure stimulates significant acute reductions and chronic improvements in muscular strength and power. However, it is not well understood whether a less stressful and potentially safer exercise modality, such as not completing exercise to the point of failure, can achieve similar or superior outcomes in trained individuals. Disagreement within the current literature that has compared failure and non-failure based resistance exercise prescription may stem from many factors, potentially related to differences in methodological design and a relatively poor understanding of the mechanisms that promote acute and chronic changes in muscular strength and power in trained individuals. Therefore, this thesis contains a series of investigations designed to address the disagreement within the present body of literature and examine gaps in the understanding of the need for trained individuals to perform resistance exercise to failure to improve muscular strength and power.

Study 1 investigated changes in muscular strength and power following an acute bout of isometric failure and non-failure based exercise of the knee extensors. Failure exercise was observed to promote greater reductions in muscular strength than a similar bout of non-failure exercise. Peripheral, rather than central mechanisms were found to facilitate reductions in

muscular strength with both exercise modalities and likely mediated the greater reduction in muscular strength following failure exercise prescription.

As isotonic contractions are more commonly performed in many real world training and competitive environments, Study 2 examined a single session of dynamic failure and non-failure exercise. This investigation demonstrated that a single bout of failure exercise was no more effective at stimulating reductions in plantar flexor strength than a similar bout of non-failure exercise. The decline in strength likely resulted from significant impairment of central neural drive to the muscle. However, a potentiation of muscular excitation-contraction coupling processes seems to have produced an acute increase in muscular power output.

The final investigation presented in this thesis (Study 3) examined changes in muscular strength and power following short term failure and non-failure training. Whilst plantar flexor power did not improve with training, failure and non-failure exercise modalities were equally effective at improving plantar flexor strength. The results demonstrated that improvements in muscular strength were likely produced from improved functionality of the muscular contractile apparatus and not from adaptations within spinal or supraspinal neural pathways.

The body of work presented in this thesis has demonstrated that both failure and non-failure based exercise evoke an increase in muscular fatigue acutely, which for the most part, was observed to promote a similar acute reduction in muscular strength between modalities. The acute increase in muscular fatigue likely facilitated the similar improvements in muscular strength observed with failure and non-failure exercise following short term training in trained populations. However, the central and peripheral fatigue mechanisms that mediated acute reductions in muscular strength following failure and non-failure exercise did not appear to have any relevance for predicting the training outcome.

# CHAPTER 1

*Introduction*

## THE PROBLEM

Resistance exercise is commonly prescribed to improve maximal and explosive force production (i.e. muscular strength and muscular power, respectively). Untrained individuals typically experience rapid improvements in muscular strength and power in the initial phases of training regardless of the resistance exercise stimulus. However, the rate and magnitude of subsequent adaptations are reduced the longer a person is engaged in consistent resistance exercise training. It is therefore assumed that improvements in muscular strength and power are optimised in individuals with many years of training experience when exercise is prescribed to maximise acute fatigue. Currently, strength and conditioning practitioners and researchers are still searching for the ideal method of resistance exercise prescription to maximise acute fatigue and optimise training outcomes in trained individuals.

Failure based exercise (i.e. when voluntary contractile force output cannot be maintained at a given intensity or throughout a specified range of motion) is one method of resistance exercise prescription that is understood to maximise acute fatigue and promote improvements in muscular strength and power in trained individuals. However, the literature that has compared failure and non-failure exercise has demonstrated that non-failure exercise is able to evoke a similar fatigue response and can in fact be more effective than failure exercise at improving muscular strength and power in trained population demographics. Disagreement between studies may, at least partially, be attributed to factors related to participant training experience prior to testing and differences in exercise volume and duration between failure and non-failure exercise modalities. Furthermore, the current confusion within the literature may be exacerbated by a lack of understanding of the mechanisms that promote fatigue and adaptation in trained individuals in response to failure and non-failure resistance exercise.

## ACUTE FATIGUE IN TRAINED INDIVIDUALS

Resistance exercise promotes an acute increase in muscular fatigue manifest as a reduction in the maximal force generating capacity of a muscle (Bigland-Ritchie & Woods, 1984). The factors that mediate acute declines in muscular force production with resistance exercise are considered from central mechanisms associated with the level of output from spinal and supraspinal neurons in the nervous system (Bigland Ritchie *et al.*, 1978; Gandevia, 2001; Taylor *et al.*, 2006) and peripheral mechanisms related to the intrinsic contractile properties of the muscle-tendon unit (Merton, 1954; Sale *et al.*, 1982; Bigland-Ritchie *et al.*, 1986; Behm & St-Pierre, 1997). The extent of central fatigue following an acute bout of resistance exercise is thought to be important for the development of muscular strength and power following a period of resistance exercise training (Moritani & DeVries, 1979; Sale, 1988; Aagaard *et al.*, 2002b). However, there is a paucity of research that has examined the underlying contribution of central fatigue to acute changes in muscular strength and power following an acute bout of failure and non-failure resistance exercise. Performing resistance exercise to failure is a highly stressful and uncomfortable method of exercise prescription (Fisher *et al.*, 2015). Therefore, it seems important for strength and conditioning researchers to determine whether a bout of non-failure exercise can evoke a similar level of central fatigue to effectively inform exercise practice for coaches and recreational weightlifters.

Central fatigue is recognised as an exercise-induced loss in voluntary force production manifest from the inability of the nervous system to maximally recruit the active motor unit pool during muscular contraction (Taylor *et al.*, 2006). Central fatigue is commonly reported as a reduction in muscle activity, observed as a decline in maximal muscle surface electromyographic (sEMG) signal amplitude. In trained individuals, a reduction in maximal

muscle activity and thus central fatigue, is thought to be at least partially responsible for the similar decline in maximal strength incurred from a bout of failure and non-failure exercise (Benson *et al.*, 2006). In contrast, failure and non-failure exercise modalities are also understood to promote similar reductions in muscular strength in trained individuals despite a potentiation of muscle activity following failure exercise and a decrease in activity following non-failure exercise (Marshall *et al.*, 2012). Disagreements between studies may be explained by a number of limitations of the sEMG technique that can affect the interpretation of sEMG signal amplitude, such as action potential (i.e. electrical signal) propagation (Yue *et al.*, 1995), detection (Mottram *et al.*, 2005; Farina *et al.*, 2010) and cancellation (Keenan *et al.*, 2005). Therefore, it is currently unclear whether the magnitude of central fatigue is different between a bout of failure and non-failure exercise in trained individuals.

Calculating the voluntary activation (VA) of a muscle via the interpolated twitch technique (i.e. the relationship between electrically evoked and voluntary force output during contraction expressed relative to evoked force at rest) is a reliable and relatively valid technique that can be used to observe central fatigue whilst avoiding the limitations associated with sEMG amplitude interpretation (Behm *et al.*, 1996; Gandevia, 2001; Shield & Zhou, 2004). An increase in central fatigue following an acute bout of resistance exercise is commonly demonstrated by a reduction in VA. Much of the research that has observed reductions in VA following resistance exercise has been conducted using untrained participants that were required to perform a single sustained maximal isometric contraction (Bigland Ritchie *et al.*, 1978; Kent-Braun, 1999; Schillings *et al.*, 2003; Place *et al.*, 2007). However, a bout of resistance exercise is typically performed over a series of submaximal dynamic muscular contractions in most real world training environments. Furthermore, there has been relatively little investigation into the changes in VA that occur following a bout of

resistance exercise in trained individuals (Behm *et al.*, 2002; Hartman *et al.*, 2011; Marshall *et al.*, 2015). Trained individuals are understood to possess a well-adapted nervous system compared to untrained persons (Nielsen *et al.*, 1993; del Olmo *et al.*, 2006). Despite reductions in VA reported previously in untrained persons, trained individuals have demonstrated the capacity to maintain complete VA after a fatiguing bout of isometric resistance exercise (Marshall *et al.*, 2015). Thus, the majority of investigations conducted using untrained participants may not be externally valid to trained population demographics. Nonetheless, a study in which trained individuals completed a single set of moderate to high intensity dynamic elbow flexion contractions to failure has demonstrated significant reductions in VA regardless of exercise volume or intensity (Behm *et al.*, 2002). Therefore, performing resistance exercise to failure may serve as a suitable exercise stimulus to maximise central fatigue in trained individuals. However, Behm and colleagues also demonstrated that peripheral fatigue significantly impaired muscular force production following failure based exercise in trained individuals, albeit with a low intensity and high volume of muscular contractions (Behm *et al.*, 2002).

Peripheral fatigue is referred to as an exercise-induced loss in voluntary force production experienced from impaired functioning of processes distal to the neuromuscular junction (Bigland-Ritchie & Woods, 1984; Buckthorpe *et al.*, 2014). Similar to central fatigue, the magnitude of peripheral fatigue incurred from a bout of resistance exercise is believed to be important for the development of muscular strength and power with resistance exercise training (Hakkinen *et al.*, 1985b; Del Balso & Cafarelli, 2007). Increases in peripheral fatigue are commonly reported using the amplitude and torque-time characteristics of an electrically evoked twitch at rest, which are believed to provide an indirect estimation of processes related to muscular excitation-contraction coupling and thus, muscular force production

(Allen *et al.*, 2008; Neyroud *et al.*, 2012; Siegler *et al.*, 2014). Peripheral fatigue, indicated by a reduction in resting twitch peak force, rate of force development, and an increase in half-relaxation time has been observed to impair muscular strength and power following an acute bout of resistance exercise in trained persons (Marshall *et al.*, 2015). As mentioned above, Behm and colleagues have previously reported that a single set of low to moderate intensity resistance exercise completed to failure can promote an increase in peripheral fatigue in trained individuals, demonstrated by a significant reduction in resting twitch peak force (Behm *et al.*, 2002). However, in this investigation, resting twitch temporal characteristics were potentiated following exercise (Behm *et al.*, 2002). Furthermore, external to laboratory settings, resistance exercise is more commonly completed over multiple sets. Given trained individuals are considered to have a greater ratio of type II (i.e. fast contracting, fast fatigable) to type I (i.e. slow contracting, fatigue resistant) muscle fibres (Hakkinen *et al.*, 1985b), it may be assumed that multiple sets of failure based exercise may stimulate greater peripheral fatigue than a similar non-failure task. However, it is unknown whether performing exercise to failure is in fact necessary to maximise peripheral fatigue in trained individuals, or if a similar bout of non-failure exercise could achieve a similar outcome. Therefore, a series of studies were performed to investigate the disagreements within the present body of literature and to examine whether performing resistance exercise to failure is required to stimulate significant peripheral as well as central fatigue in trained individuals or if non-failure exercise can serve as an efficacious alternative.

## MUSCULAR STRENGTH AND POWER ADAPTATION IN TRAINED INDIVIDUALS

Optimising improvements in muscular strength and power is of critical importance to strength and conditioning practitioners and researchers. Trained individuals have already experienced significant improvements in muscular strength and power which are largely thought to result from adaptation of central processes that has led to a greater ability to recruit the available motor unit pool during maximal muscular contraction (Hakkinen *et al.*, 1985a; Van Cutsem *et al.*, 1998). As a result, the time course for further improvement in muscular strength and power is prolonged and the magnitude of subsequent adaptations are reduced (Hakkinen *et al.*, 1985a; Hakkinen *et al.*, 1985b). The current understanding of the ideal resistance exercise stimulus required to optimise central adaptations and thus, muscular strength and power in trained populations is relatively poor. It is therefore assumed that trained individuals need to 'work harder' by performing moderate to high intensity resistance exercise to failure to maximise improvements in muscular strength and power. However, exercising to failure for long training periods is thought to increase the risk of musculoskeletal injury and has been suggested to compromise athletic performance through the negative effect of accumulative fatigue on neuromuscular functioning (i.e. overreaching) (Izquierdo *et al.*, 2006). To limit these undesirable training outcomes and to minimise feelings of discomfort during exercise and improve adherence, non-failure training has been suggested as a potentially efficacious alternative.

Many studies conducted in both trained and untrained population demographics provide support for the use of failure and non-failure exercise when these modalities are performed with moderate to high intensity loads. Some investigations have suggested that performing

exercise to failure produces greater increases in muscular strength and power than a similar period of non-failure training (Rooney *et al.*, 1994; Schott *et al.*, 1995; Drinkwater *et al.*, 2005). Conversely, other studies have demonstrated larger improvements in these variables with non-failure exercise (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). This debate has led to several reviews and meta-analyses of literature that has directly compared a period of failure and non-failure training on outcomes of muscular strength and power (Willardson, 2007; Davies *et al.*, 2016). Based on the current position, it is believed that failure and non-failure exercise modalities are equally effective at improving muscular strength when maximal strength data is analysed from both trained and untrained individuals (Davies *et al.*, 2016). However, in the relatively small body of literature conducted using trained individuals, the need to perform exercise to failure and/or not to failure to improve muscular strength and power is not as clear.

Previous studies that have compared failure and non-failure resistance training programs in trained individuals have not agreed upon the need to perform moderate to high intensity exercise to failure to maximise improvements in muscular strength and power. Only one of these investigations has found a greater benefit of exercising to failure in trained individuals following short term training (Drinkwater *et al.*, 2005). Other studies have typically demonstrated similar improvements in maximal strength and have reported that non-failure exercise is in fact more beneficial for improving maximal power in trained individuals (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). Therefore, it is not well understood whether muscular strength and power adaptations are optimised with failure or non-failure exercise training. The confusion within the literature may, in part, be owing to the development of early neural adaptations that promoted changes in muscular strength and power as a result of minimal participant training experience prior to testing (Drinkwater *et*

*al.*, 2005), the performance of exercise during the training period that was external to study design (Drinkwater *et al.*, 2005; Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010) and the lack of properly controlled failure and non-failure exercise prescription in which resistance exercise volume was not equated between programs (Izquierdo-Gabarren *et al.*, 2010). Debate may be further augmented by the lack of understanding of the mechanisms thought to improve training outcomes.

To the author's knowledge, no investigation has examined central and peripheral mechanistic adaptation following a period of resistance exercise training in trained persons. An increase in the number of recruited motor units and rate of motor unit firing, typically assumed through increased muscle sEMG amplitude characteristics, is thought to be responsible for improvements in muscular strength and power with training (Hakkinen *et al.*, 1985b; Häkkinen *et al.*, 1987; Van Cutsem *et al.*, 1998). Given higher motor unit firing frequencies are thought to be important for greater muscular power output, failure based training by nature may negatively affect explosive contractile velocity and thus the ability to activate motor units at high firing frequencies. Indeed, a period of non-failure training has been shown to promote significant increases in muscular power, with an observed increase in muscle activity leading authors to conclude that that non-failure exercise is an efficacious technique to improve motor unit recruitment (Van Cutsem *et al.*, 1998). To date, no literature has reported the use of sEMG as a measure of central adaptation following a comparison of failure and non-failure training in trained individuals. Furthermore, sEMG cannot discriminate between mechanistic adaptations affecting spinal and cortical neural input to the motor unit pool (Dimitrova & Dimitrov, 2003) that are thought to promote an increase in motor unit activation and thus, muscular strength during maximal contraction in trained individuals (Nielsen *et al.*, 1993; del Olmo *et al.*, 2006). Therefore, an understanding of

central mechanistic adaptation and how to optimise these chronic changes in neural functioning with resistance exercise may be vital to exercise practitioners and coaches required to maximise muscular strength and power in athletes and recreational weightlifters.

The underlying central mechanisms that contribute neural input to the motor unit pool have previously been observed following a period of training in untrained individuals. These central mechanisms, typically associated with spinal and supraspinal input to the motor neuron pool are often examined using the electrophysiological spinal reflex analogues known as the Hoffmann reflex (H-reflex) and V-wave that are produced from electrical stimulation of the axons of mixed (i.e. afferent and efferent fibres) peripheral nerves (Aagaard *et al.*, 2002b; Duclay *et al.*, 2008). Spinal and supraspinal adaptations, indicated by an increase in the amplitude of the H-reflex and V-wave, respectively, have been shown to predict improvements in muscular strength and power with training (Aagaard *et al.*, 2002b; Del Balso & Cafarelli, 2007; Holtermann *et al.*, 2007; Johnson *et al.*, 2014). Spinal adaptations, indicated by amplitude changes of a single H-reflex at rest, are thought to be dependent on the exercise stimulus (i.e. resistance exercise compared to endurance exercise) used during training (Kyröläinen & Komi, 1994; Maffiuletti *et al.*, 2001). Therefore, it seems important to determine if spinal adaptations promote differences in muscular strength improvements between two different resistance exercise modalities in trained individuals. Additionally, the authors did not control for post-synaptic events (Knikou, 2008) and changes in motor neuron excitability known to effect passive H-reflex recruitment (Nordlund *et al.*, 2004). Also, a single H-reflex does not provide an indication of motor unit recruitment at different activation thresholds (Klimstra & Zehr, 2008; Vila-Cha *et al.*, 2012). An observation of H-reflex recruitment across a spectrum of electrical stimulation intensities may aid understanding of whether training induced adaptations in the recruitment of small, medium or large motor units are necessary to optimise improvements in muscular strength and power in

trained individuals. Furthermore, neural input to the α-motor neuron pool can be inhibited from processes occurring pre- (i.e. at the Ia afferent terminal) and post-synaptically (i.e. at the α-motor neuron terminal) (Eccles *et al.*, 1962; Pierrot-Deseilligny *et al.*, 1976; Bussel & Pierrot Deseilligny, 1977; Iles *et al.*, 2000). Although power trained athletes have been shown to have reduced Ia afferent inhibition compared to untrained persons (Earles *et al.*, 2002), it is unknown if this inhibition modulates changes in strength and power following short term training in trained individuals. Determining an exercise modality that promotes a disinhibition of motor neurons during maximal contraction may be important for trained individuals, a demographic that is often required to have a large absolute level of muscular strength. Therefore, a training intervention was designed to address gaps in the understanding of the exercise modalities and mechanisms thought to optimise improvements in muscular strength and power in trained individuals, while at the same time, expanding upon the acute investigations conducted in this thesis to provide knowledge of whether improvements in muscular strength and power following a period of training are dependent on performing an acute bout of resistance exercise to failure and/or not to failure.

## EXPERIMENTAL AIMS AND HYPOTHESES

### Research Aim

The primary aim of this research was to examine acute (Studies 1 and 2) and chronic (Study 3) changes in muscular strength and power, and the mechanisms that promote these changes following failure and non-failure based resistance exercise prescription in trained individuals.

### Research Hypotheses

The following hypotheses were tested in this thesis:

1. A single bout of moderate to high intensity exercise performed to failure would produce a significantly greater reduction in maximal strength compared to a similar bout of non-failure exercise in trained individuals (Studies 1 and 2).
2. Acute reductions in strength following failure and non-failure based exercise would be mediated by fatigue from central and peripheral origins (Studies 1 and 2).
3. Failure based exercise would facilitate larger reductions in muscular strength and power as a result of greater central, rather than peripheral fatigue (Studies 1 and 2).
4. Moderate to high intensity failure and non-failure based exercise would evoke similar increases in muscular strength in trained individuals following short term training, with the non-failure group experiencing a significantly greater increase in muscular power (Study 3).
5. Improvements in muscular strength and power following a period of failure and non-failure training would result from an increase in motor neuron output, produced from greater spinal and supraspinal neural input to the motor unit pool (Study 3).

# THESIS OUTLINE AND SIGNIFICANCE

## Thesis Outline

Chapter 2 presents a general literature review of the use of failure and non-failure exercise and the techniques used to extract information about the sites of fatigue and adaptation within the nervous and musculoskeletal systems. Chapters 3, 4 and 5 present the individual methods, results, discussion and conclusion sections of the series of original investigations that comprise this thesis. Specifically, Chapters 3 (Study 1) and 4 (Study 2) examined fatigue from a single bout of failure and non-failure exercise in the knee extensors and plantar flexors, respectively, and Chapter 5 (Study 3) examined the adaptations produced from eight weeks of failure and non-failure training in the plantar flexors. Chapters 6 and 7 present a general discussion and conclusion of the main findings of this research, respectively.

## Significance of Thesis

The current understanding of the ideal resistance exercise stimulus required to optimise muscular strength and power adaptations in trained individuals is poor. This will be the first body of research to examine whether performing resistance exercise to failure or not to failure predicts training outcomes in individuals with years of resistance exercise experience. The series of investigations performed in this thesis will also be the first to observe the mechanisms responsible for central and peripheral fatigue and adaptation within this population demographic.

# CHAPTER 2

*General Literature Review*

## FAILURE AND NON-FAILURE EXERCISE: ACUTE INVESTIGATIONS

Reductions in maximal muscular strength and power output represent a typical fatigue response to resistance exercise. The extent of fatigue following a bout of resistance exercise is thought to be important for the development of muscular strength and power following a period of training. It assumed that acute reductions in muscular strength and power will be maximised by completing a set of repetitions to the point of failure. Therefore, in many real world training environments, resistance exercise is commonly performed to failure. This practice is not unsubstantiated as literature has demonstrated that failure based resistance exercise is an effective exercise modality for evoking significant reductions in muscular strength and power following an acute bout of exercise (Willardson, 2007; Willardson *et al.*, 2010). Nonetheless, this line of thinking has led strength and conditioning practitioners and researchers to question whether there is actually a need to perform exercise to failure, subsequently prompting investigation into the changes in muscular strength and power that occur between a single bout of failure based exercise and a similar bout of resistance exercise not performed to failure (Rooney *et al.*, 1994; Linnamo *et al.*, 1998; Drinkwater *et al.*, 2005; Benson *et al.*, 2006; Marshall *et al.*, 2012). However, within this relatively small body of literature there is considerable disagreement regarding the need to perform resistance exercise to the point of failure to stimulate acute reductions in muscular strength and power.

### Acute declines in muscular strength and power

The consensus within the current literature is that failure and non-failure exercise modalities promote significant reductions in muscular strength and power following an acute bout of resistance exercise (Rooney *et al.*, 1994; Linnamo *et al.*, 1998; Drinkwater *et al.*, 2005;

Benson *et al.*, 2006; Marshall *et al.*, 2012). The present disagreement between investigations relates to whether these studies have observed a greater decline in muscular strength and power following failure based exercise prescription (Rooney *et al.*, 1994; Linnamo *et al.*, 1998; Drinkwater *et al.*, 2005) or a similar reduction in these variables when exercise is not performed to failure (Benson *et al.*, 2006; Marshall *et al.*, 2012). The findings of this literature are summarised in Table 1.

**Table 1.** Summary of literature that has observed acute changes in muscular strength and power following a comparison of failure and non-failure exercise.

| Study | Muscle group | Participant details | Methods | Results |
|---|---|---|---|---|
| Rooney *et al.* (1994) | Elbow flexors | Untrained | Isotonic elbow flexion | ↓ 20.2 % isometric MVC; *F*<br>↓ 10.4 % isometric MVC; *NF* |
| | | Age (18-35 years) | 6RM load | |
| | | *n* = 9 | | ↓ isometric MVC *F* > *NF* |
| | | | *F:* 1 × 6 reps | |
| | | | *NF:* 6 × 1 rep, 30 sec rest b/w reps | |
| Linnamo *et al.* (1998) | Knee extensors | Untrained | Isotonic knee extension | ↓ 21.3 % isometric MVC, ↓ 28 % RFD, ↓ 13.4 % max sEMG, ↑ blood lactate c. ; *F* |
| | | Age (25.2 years) | 2 min rest b/w sets | ↓ 11.6 % isometric MVC, ↓ 13.5 % RFD, ↓ 18.5 % max sEMG, ↑ blood lactate c. ; *NF* |
| | | *n* = 16 | | |
| | | | *F:* 5 × 10 reps (10 RM load) | |
| | | | *NF:* 5 × 10 reps (40 % 10 RM load), explosive | ↓ isometric MVC *F* > *NF* |
| Drinkwater *et al.* (2005) | Pectorals/ elbow extensors | Trained (0.5-3years) | Isotonic bench press | ↓ 19.6 % bench press power; *F*<br>↓ 7.8 % bench press power; *NF* |
| | | | 6RM load | |
| | | Age (18.6 years) | | ↓ bench press power *F* > *NF* |
| | | *n* = 26 | *F:* 4 × 6 reps, 260 sec rest b/w sets | |
| | | | *NF:* 8 × 3 reps, 113 sec rest b/w sets | |

*Table 1 continued on next page*

*Table 1 continued*

| Study | Muscle group | Participant details | Methods | Results |
|---|---|---|---|---|
| Benson *et al.* (2006) | Elbow flexors | Trained (1 year)<br>Age (25.5 years)<br>*n* = 13 | Isotonic elbow flexion<br>3 min rest b/w sets<br>*F:* 3 × 10 reps (10 RM load),<br>*NF:* 2 × 10 reps (90 % 10RM load),<br>1 × 90 % 10RM until failure | ↓ 19 % isometric MVC, ↓ 17 % max sEMG, ↑ 118 % blood lactate c. ; *F*<br>↓ 18 % isometric MVC, ↓ 18 % max sEMG, ↑ 59 % blood lactate c. ; *NF* |
| Marshall *et al.* (2012) | Quadriceps/gluteals | Trained (5.5 years)<br>Age (25.0 years)<br>*n* = 14 | Isotonic back squat<br>80 % 1RM load<br>*F:* Reps to failure each set, 20 sec rest b/w sets, 20 reps accrued<br>*NFa:* 5 × 4 reps, 20 sec rest b/w sets<br>*NFb:* 5 × 4 reps, 3 min rest b/w sets | ↓ 8.2 % isometric MVC, ↓ 11.5 % isometric RFD; pooled *F*, *NFa*, *NFb*<br>↑ 8.4 % max sEMG; *F*<br>↓ ~ 11 % max sEMG; pooled *NFa* and *NFb*<br>↑ max sEMG *F* > pooled *NFa* and *NFb* |

In an acute laboratory setting, muscular strength is typically observed using the magnitude of the force produced during an isometric maximal voluntary contraction (MVC). The investigation by Rooney *et al.* (1994) was the first study to compare changes in muscular strength between a failure and comparative non-failure task. Participants completed a series of six, dynamic elbow flexion contractions with a 6 repetition maximum (RM) load in both exercise conditions. The failure task required participants to complete all six contractions without resting between repetitions, whereas 30 sec recovery was provided between individual repetitions in the non-failure task (Rooney *et al.*, 1994). The authors observed a 20.2 % reduction in muscular strength immediately following the failure task, measured using the peak force output recorded during an isometric maximal voluntary contraction (MVC) of the elbow flexors. The reduction in muscular strength at the conclusion of the failure task was significantly greater than the 10.4 % reduction in the non-failure condition (Rooney *et al.*, 1994). Hence, failure exercise was concluded to be more effective at stimulating acute reductions in muscular strength than a similar bout of non-failure exercise. This finding has since been supported by an investigation that demonstrated significantly greater reductions in knee extensor maximal strength at the conclusion of a bout of failure (21.3 % decline) compared to non-failure exercise (11.6 % decline) (Linnamo *et al.*, 1998). However, since these reports, studies by Benson *et al.* (2006) and Marshall *et al.* (2012) have observed similar reductions in maximal elbow flexor (~19 % pooled decline) and barbell back squat strength (~8 % pooled decline), respectively, between a bout of failure and non-failure based exercise. An issue not isolated to muscular strength, current literature also disagrees on the need to perform resistance exercise to failure to maximise acute reductions in muscular power.

Changes in muscular power output with fatiguing resistance exercise typically represent a reduction in the force-time characteristics of the initial, explosive phase of maximal muscular contraction. Studies by Linnamo *et al.* (1998) and Marshall *et al.* (2012) have demonstrated that muscular power output is similarly reduced between a bout of failure and non-failure resistance exercise. In contrast, one investigation has reported that a failure based task stimulates a greater reduction in muscular power than a bout of non-failure exercise. Drinkwater and colleagues had participants complete a series of dynamic bench press repetitions using 6 RM loads either to failure (4 sets × 6 repetitions) or not to failure (8 sets × 3 repetitions) (Drinkwater *et al.*, 2005). The authors reported that the 19.6 % decline in muscular power output at the conclusion of the failure condition was significantly greater than the 7.8 % decline experienced in the non-failure condition (Drinkwater *et al.*, 2005). Therefore, these current findings demonstrate that it is not well understood whether resistance exercise should be performed to failure to maximise acute reductions in muscular power or if individuals engaged in resistance exercise for athletic or recreational purposes could achieve a similar outcome by not completing repetitions to the point of failure. The present disagreement amongst investigations that have observed reductions in muscular power and strength following a failure and non-failure task may be attributed to a number of differences between, and potential limitations of, study design.

## Methodological differences and limitations

### *Determining muscular power*

Disagreement amongst the literature that has observed a greater reduction in muscular power following failure compared to non-failure exercise versus studies that have reported similar

reductions between failure and non-failure modalities may relate to differences in the methods used to measure and calculate muscular power output. Acute declines in muscular power output following failure and non-failure exercise have been observed using two techniques: rate of force development (RFD) (Linnamo *et al.*, 1998; Marshall *et al.*, 2012), which tests explosive force production isometrically at a single joint range of motion (ROM); and dynamic power output (Drinkwater *et al.*, 2005), which tests the rate at which an object is displaced through a ROM. Because Drinkwater and colleagues calculated muscular power output throughout an entire ROM, comparing the change in power to literature that observed RFD at a single point in the ROM (i.e. when tested isometrically) is potentially problematic. Furthermore, Drinkwater *et al.* (2005) used an absolute load (40 kg) to determine muscular power output during a single explosive bench press repetition. Given the load used to perform this test did not correspond to a relative percentage of maximal strength prior to testing, between-participant differences in strength level were not controlled. Measuring muscular power output using an isometric MVC and expressing RFD relative to peak force output may therefore serve as a more time efficient technique that would also allow for easier comparison to previous investigations.

*Exercise volume and inter-set recovery periods*

Resistance exercise volume (sets × repetitions × load (kg)) and recovery time between sets are two prescription variables that are commonly manipulated with training as part of a balanced program design. However, when researchers manipulate these variables whilst comparing different exercise modalities, the validity of the exercise comparison should be questioned. For example, if a non-failure task was completed with a greater exercise volume and longer inter-set recovery periods than a similar failure task, the fatigue incurred from this

bout of exercise would be expected to be less than the failure task in which participants would have 'worked' more and spent less time recovering. Therefore, it seems important to equate exercise volume and inter-set recovery periods between two exercise protocols to control for potential differences in fatigue by the conclusion of exercise that are external to study design.

To date, authors that have observed acute changes in muscular strength and power following a comparison of failure and non-failure based exercise have either controlled (Rooney *et al.*, 1994; Drinkwater *et al.*, 2005; Marshall *et al.*, 2012) or have not controlled (Linnamo *et al.*, 1998; Benson *et al.*, 2006) differences in exercise volume between groups. Following a dynamic elbow flexion task, Benson and colleagues reported similar reductions in strength between a bout of failure and non-failure exercise despite participants completing the non-failure task with a significantly greater volume of work (14 %) (Benson *et al.*, 2006). The authors reported that the greater volume of work in the non-failure condition resulted from the reduced number of repetitions completed in each set of the failure condition (Benson *et al.*, 2006). To maintain a similar volume between failure and non-failure exercise it may be more advantageous to minimally reduce exercise load between sets of failure exercise so the overall number of repetitions, and therefore, the volume of work can remain relatively similar between conditions.

In contrast to the findings of Benson *et al.* (2006), Linnamo and colleagues observed greater reductions in knee extensor strength following a failure task that was completed with a volume more than twice that of the comparative non-failure condition (Linnamo *et al.*, 1998). The greater volume of work completed in the failure protocol likely contributed to the larger reduction in muscular strength observed following failure exercise. Previous literature has

demonstrated larger increases in muscular fatigue in untrained (Walker *et al.*, 2011) and trained (Tran *et al.*, 2006) populations in response to a higher versus a lower volume of exercise. Furthermore, because the exercise load was significantly lower and participants were instructed to perform contractions in a rapid explosive fashion in the non-failure protocol (Linnamo *et al.*, 1998), changes in muscular strength were likely to have been affected by differences in exercise intensity and contraction velocity between conditions (Kanehisa & Miyashita, 1983; Hakkinen & Komi, 1986; Behm & Sale, 1993b). However, reductions in muscular strength and power were unlikely to have been affected by differences in inter-set recovery periods given Linnamo *et al.* (1998) controlled the duration of recovery periods between groups.

In the study by Rooney and colleagues, not only was the total session duration of the non-failure protocol greater than that of the failure protocol, it was also the only condition to include inter-set recovery periods (Rooney *et al.*, 1994). Ratamess *et al.* (2007) have previously demonstrated following multiple sets of a moderate to high intensity bench press task with either 30 sec, 1 min, 2 min, 3 min and 5 min rest between sets, that acute performance decrements are greater with shorter (< 1 min) compared to longer inter-set recovery periods. Therefore, the larger reduction in strength following failure exercise in the Rooney *et al.* (1994) investigation may have been a function of the difference in recovery time between tasks and not because failure based exercise prescription is more fatiguing by nature. However, a previous report has demonstrated a greater reduction in muscular power output following failure based exercise when inter-set recovery periods and total session duration were matched between failure and non-failure exercise (Drinkwater *et al.*, 2005). These observations contrast more recent findings in which similar reductions in muscular strength and power have been observed between failure and non-failure exercise despite

participants spending on average 43 sec, 80 sec and 720 sec resting between sets in the respective failure and two non-failure tasks (Marshall *et al.*, 2012). Therefore, to address the disagreement within the current literature it seems necessary for future investigations to equate inter-set recovery periods, and thus, total session duration when comparing changes in muscular strength and power between a bout of failure and non-failure exercise.

*Training experience*

Resistance exercise literature typically observes neuromuscular fatigue and adaptation using participants from two healthy population demographics, individuals who are untrained, or persons with prior resistance exercise training experience. Untrained individuals have no formal resistance exercise experience or may be recreationally physically active but have not engaged in regular strength training for at least six months prior to participation in any given study. On the other hand, it is generally accepted that trained individuals have consistently engaged in repeated bouts of resistance exercise over an extended period of time lasting months to years for the purposes of increasing muscular strength and power to improve elite and/or recreational sporting performance. It is thought that larger absolute and relative muscular strength levels in trained compared to untrained individuals (Hoeger *et al.*, 1990; Ahtiainen *et al.*, 2003) are the result of significant adaptations within the nervous system (i.e. central adaptations) that have resulted in a greater capacity to recruit the active motor unit pool during muscular contraction (Sale *et al.*, 1983b). These central adaptations are demonstrated by lower Ia afferent inhibition (Earles *et al.*, 2002), and greater spinal excitability (Nielsen *et al.*, 1993) and cortical drive to the motor unit pool (del Olmo *et al.*, 2006) in trained compared to untrained individuals. Therefore, it is possible that a

participant's prior training experience has contributed to the disagreement between studies that have compared a bout of failure and non-failure exercise.

Failure based exercise prescription has been observed to promote greater acute reductions in muscular strength and power than non-failure exercise in untrained individuals (Rooney *et al.*, 1994; Linnamo *et al.*, 1998). In contrast, performing exercise to failure is typically no more effective than non-failure exercise at evoking declines in strength and power in trained individuals (Benson *et al.*, 2006; Marshall *et al.*, 2012). These findings may be indicative of the well adapted nervous system present in trained population demographics. However, the mechanisms responsible for central fatigue have not been observed in trained individuals following a bout of failure and non-failure exercise. Given fatigue is also understood to be influenced by peripheral factors (i.e. those distal to the neuromuscular junction), it is currently not understood whether differences in fatigue between trained and untrained individuals incurred from a bout of failure and non-failure exercise are a consequence of impaired nervous or musculoskeletal system functioning.

Measurement of central and peripheral fatigue

Muscular fatigue is understood to promote a reduction in the force producing capabilities of a muscle, typically observed as a decline in maximal muscular strength and power following an acute bout of resistance exercise. Acute reductions in muscular strength and power are generally considered from fatigue of similar mechanisms with origins within the nervous (i.e. central fatigue) and musculoskeletal (i.e. peripheral fatigue) systems (Bigland Ritchie *et al.*, 1978; Buckthorpe *et al.*, 2014). Observing the mechanisms understood to stimulate acute reductions in muscular strength and power with resistance exercise may help with the present

understanding of the fatigue incurred from a bout of failure and non-failure exercise and serve to address disagreements within the current literature. Currently, no clear mechanism has been proposed to explain the acute reduction in muscular strength and power that has been demonstrated with failure and non-failure exercise, although central factors have been suggested to play a role in this response (Linnamo *et al.*, 1998; Marshall *et al.*, 2012).

*Central fatigue*

The force produced during muscular contraction that can be attributed to nervous system functioning is dependent on the magnitude of α-motor neuron output to the muscle (Sale *et al.*, 1983a; Sale, 1988; Herbert & Gandevia, 1999; Aagaard *et al.*, 2002b). Central mechanisms impair and/or facilitate neural input to the α-motor neuron pool, ultimately affecting the ability of the nervous system to 'drive' or recruit the muscle maximally during contraction (Taylor *et al.*, 2006). These mechanisms modulate muscular force production by mediating the recruitment and rate of discharge of available motor units (Maton, 1981; Enoka & Stuart, 1984) through a series of synaptic events affecting action potential depolarisation within the afferent, efferent and interneuronal pathways of the spinal cord (i.e. spinal mechanisms) and motor cortex (i.e. supraspinal mechanisms). Central fatigue is commonly reported by observing electrical activity at the level of the muscle using a non-invasive technique known as muscle surface electromyography (sEMG) (Moritani & DeVries, 1979; Hakkinen *et al.*, 1985a; De Luca, 1997) that is thought to provide a gross, downstream estimation of α-motor neuron output to the muscle (Behm, 1995). To date, literature that has compared a bout of failure and non-failure based exercise disagrees on the change in muscle activity, and thus, central fatigue evoked by these modalities.

*Surface electromyography.* Of the three investigations that have observed muscle activity following a bout of failure and non-failure exercise, two studies have reported a similar, 10-20 % reduction in maximal sEMG amplitude between groups at the conclusion of exercise (Linnamo *et al.*, 1998; Benson *et al.*, 2006). Benson *et al.* (2006) interpreted the similar decrease in both maximal muscle activity and muscular strength between failure and non-failure conditions to reflect that a reduction in neural activation was responsible for the decline in muscular strength at the conclusion of exercise. In the investigation by Linnamo *et al.* (1998), the authors reported that the similar decrease in muscle activity between conditions was indicative of impaired neuromuscular propagation. However, the greater reduction in muscular strength reported in the failure condition was not accompanied by a comparatively greater reduction in maximal muscle activity. Hence, the sEMG technique could not elucidate why reductions in muscular strength were greater with failure based exercise and/or that fatigue following a bout of exercise performed to failure is instead a consequence of impaired functioning of processes distal to the neuromuscular junction. Furthermore, Marshall *et al.* (2012) demonstrated that reductions in muscular strength and power are similar between failure and non-failure modalities, despite observing an 8 % increase in maximal sEMG amplitude in the failure condition that was significantly different to the 11 % decrease that occurred following the non-failure task. Additionally, the authors reported that muscle activity changed in some agonist muscles but not others (Marshall *et al.*, 2012). Therefore, the present disagreement between studies as well as the observed conditional reductions in muscular strength without concomitant greater declines in muscle activity, likely demonstrates that central fatigue following exercise cannot be inferred from the sEMG technique.

Surface electromyography is understood to have multiple limitations in its ability to estimate motor unit output. These limitations are often considered from interpretation issues related to signal processing (De Luca, 1997), an underestimation of motor unit output resulting from the cancellation of positive and negative phases of action potential generation (Keenan *et al.*, 2005), the ability of the sEMG technique to detect action potential activity at the cutaneous level (Mottram *et al.*, 2005; Farina *et al.*, 2010), and the inability to distinguish differences between the synchronisation of motor unit action potential generation and the signal artefact and signal-to-noise ratio (Yue *et al.*, 1995). Additionally, given the amplitude of the sEMG signal provides an estimation of net motor unit output, this technique is unable to distinguish between changes in output that are the result of impaired spinal or supraspinal neural input to the motor unit pool (Dimitrova & Dimitrov, 2003). Furthermore, studies that have observed muscle activity following failure and non-failure exercise have not controlled for changes in sEMG signal amplitude that could have been produced from action potential propagation occurring distal to the neuromuscular junction, across the muscle sarcolemma (Pasquet *et al.*, 2000).

*The interpolated twitch technique.* The interpolated twitch technique (ITT) is an alternative method that has been used to measure central fatigue following a bout of resistance exercise (Merton, 1954; Herbert & Gandevia, 1999). This technique involves delivering a supramaximal electrical stimulus to the axons of the α-motor neurons that innervate a contracting muscle. If the level of motor unit output is not sufficient to maximally drive the muscle during voluntary contraction, the stimulus will evoke a twitch (i.e. an involuntary increase in force amplitude), superimposed on the force trace (Rutherford *et al.*, 1986; Sale, 1988; Herbert & Gandevia, 1999) (Figure 1). By expressing the difference between the peak force output recorded during MVC and the peak force of the superimposed twitch relative to the peak force of a twitch evoked at rest, the ITT is understood to provide a measure of the degree an individual is able to voluntarily activate a muscle during contraction (Shield & Zhou, 2004; Taylor *et al.*, 2006). Therefore, a reduction in muscular strength with a concomitant decline in voluntary activation (VA) following exercise is believed to be indicative of a decrease in motor unit output (Gandevia, 2001). Hence, VA is thought to provide an estimation of central fatigue whilst avoiding the limitations of observing motor unit output using the sEMG technique. However, although the ITT provides a relatively reliable and valid measure of central fatigue (Behm *et al.*, 1996), the measurement technique itself is also thought to overestimate the extent of muscle activation, and can be limited by the muscle-tendon kinematics of the testing procedure and an individual's familiarity to the technique (Behm, 2009).

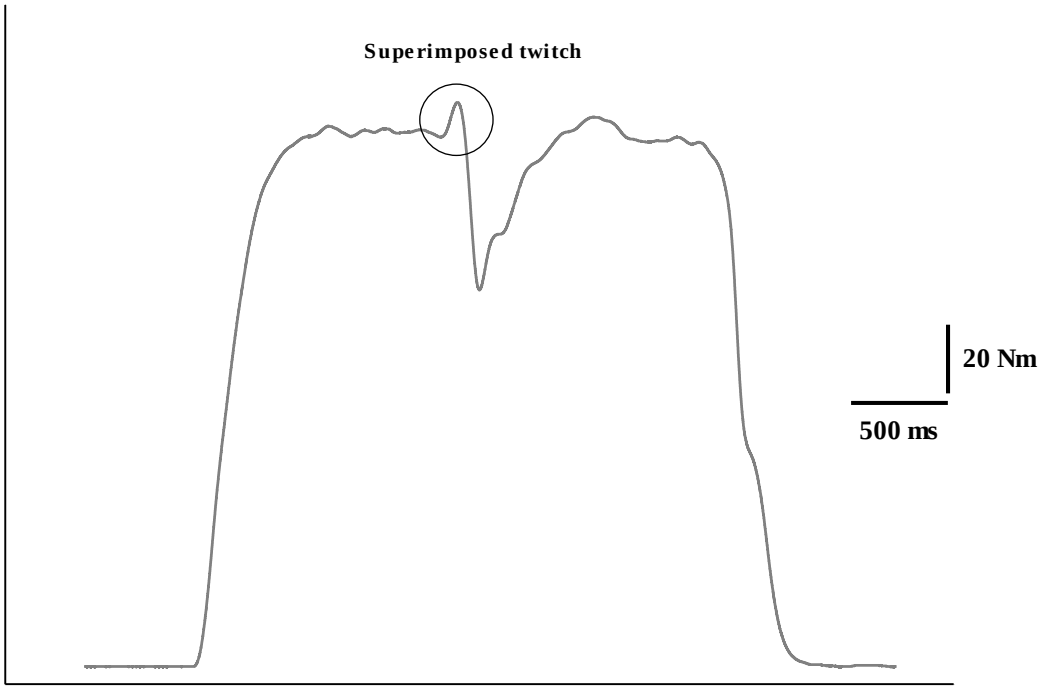


**Figure 1.** A typical maximal voluntary contraction (MVC) with superimposed twitch.

Much of the research that has used the ITT as a measure of central fatigue has reported that reductions in muscular strength following a single bout of exercise are at least partially modulated by declines in VA (McKenzie *et al.*, 1992; Gandevia *et al.*, 1996; Löscher *et al.*, 1996; Kawakami *et al.*, 2000; Nordlund *et al.*, 2004). To date, much of the literature that has observed reductions in VA following a fatiguing bout of exercise has been conducted using untrained participants (Bigland Ritchie *et al.*, 1978; McKenzie *et al.*, 1992; Gandevia *et al.*, 1996; Löscher *et al.*, 1996; Kent-Braun, 1999; Kawakami *et al.*, 2000; Nordlund *et al.*, 2004). The acute, exercise induced increase in central fatigue observed in untrained individuals is largely thought to result from the inability of the un-adapted nervous system to maintain motor neuron output in response to fatiguing muscular contraction. In contrast, trained

individuals are understood to possess a well-adapted nervous system compared to untrained persons (Nielsen *et al.*, 1993; del Olmo *et al.*, 2006). However, there is relatively little understanding of the changes in VA that occur following exercise in trained population demographics (Behm & St-Pierre, 1998; Behm *et al.*, 2002; Hartman *et al.*, 2011; Marshall *et al.*, 2015). Despite some literature that has demonstrated similar reductions in VA in trained compared to untrained persons following exercise (Behm *et al.*, 2002; Hartman *et al.*, 2011), a recent investigation has shown that trained individuals are able to maintain complete VA in the presence of significant reductions in muscular strength following a single exercise session (Marshall *et al.*, 2015). Therefore, changes in central functioning following a bout of fatiguing resistance exercise conducted by trained individuals are not as clear.

The exercise task itself and the mode of muscular contraction performed during the task are factors which may also limit conclusions drawn from the present body of literature that has used VA to report central fatigue following an acute bout of resistance exercise. Given the magnitude of central fatigue produced from a bout of resistance exercise is believed to be important for muscular strength and power development (Moritani & DeVries, 1979; Sale, 1988; Aagaard *et al.*, 2002b), many authors have used VA to examine the extent of central fatigue incurred from an exercise task designed to stimulate maximal muscular fatigue. Consequently, much of this literature has used an exercise task in which participants were required to perform a single, sustained isometric MVC for an extended period of time (i.e. a 2 min MVC) (Bigland Ritchie *et al.*, 1978; Kent-Braun, 1999; Schillings *et al.*, 2003; Place *et al.*, 2007). However, the increase in central fatigue inferred by these investigations should not be viewed as externally valid to traditional resistance exercise training, where exercise sessions are typically performed using submaximal muscular contractions, which by design, do not necessarily evoke maximal fatigue. Because performing muscular contractions to

failure is believed maximise reductions in muscular strength for a given exercise intensity (Rooney *et al.*, 1994; Linnamo *et al.*, 1998), this prescription modality is viewed as a practical alternative to sustained MVCs to induce central fatigue. This has prompted researchers to investigate changes in VA that occur when exercise is performed to failure using isometric contractions completed at a submaximal percentage of MVC.

Literature that has observed VA following a bout of submaximal isometric exercise has either performed a single sustained muscular contraction to failure (Löscher *et al.*, 1996; Neyroud *et al.*, 2012) or a series of contractions to failure (Bigland-Ritchie *et al.*, 1986). Following a sustained isometric plantar flexion contraction performed at 30 % MVC until failure, Loscher *et al.* (1996) interpreted the observed reduction in superimposed twitch amplitude to reflect that motor unit output was in fact facilitated following isometric failure based exercise. However, the work of Neyroud and colleagues contrasts this finding, whereby VA significantly declined, and thus, the reduction in muscular strength following an isometric contraction at 20 % MVC to failure likely resulted from central impairment (Neyroud *et al.*, 2012). Given resistance exercise is commonly completed over multiple repetitions in many real word training environments, some authors have also observed VA at the conclusion of a series of brief submaximal isometric contractions performed to failure. The findings of Bigland Ritchie *et al.* (1986) further contrast those of the above literature as knee extensor VA was maintained following a series of six second, 50 % MVC isometric contractions completed to failure, despite a reduction in muscular strength. Because an isometric exercise task is generally viewed to be easy to control and replicate, most of the present literature has used this mode of muscular contraction as an exercise stimulus when observing VA. However, external to laboratory testing, resistance exercise is not commonly performed isometrically in training, testing, and competitive settings. To date, relatively little

investigation has been conducted into the acute changes in VA that occur with dynamic resistance exercise.

Similar, equivocal findings have been reported within the small body of literature that has observed VA following a bout of dynamic resistance exercise (Behm *et al.*, 2002; Klass *et al.*, 2004; Gauche *et al.*, 2009; Hartman *et al.*, 2011). In an investigation by Klass *et al.* (2004), the authors reported that impaired VA was not responsible for the reduction in muscular strength at the conclusion of a plantar flexion task in which participants completed sets of 30 contractions until failure with a load corresponding to 50 % MVC. However, other studies in which dynamic exercise was performed to failure do not support this account. Following a series of 40 % MVC plantar flexion contractions completed to the point of failure, Hartman and colleagues reported that a decline in VA at the conclusion of exercise likely mediated reductions in muscular strength (Hartman *et al.*, 2011). Additionally, three different, dynamic failure based tasks in which participants completed a single set of elbow flexion contractions with either a 5 RM, 10 RM or 20 RM load have been shown to evoke similar reductions in VA that were thought to be at least partially responsible for the decline in muscular strength in each task (Behm *et al.*, 2002). Therefore, there is considerable disagreement within the field that has observed VA following dynamic and isometric failure based exercise. Furthermore, the literature is yet to report if changes in VA are similar between a bout of failure and non-failure exercise (whether dynamic or isometric), and if VA does in fact effect an acute change in muscular strength and power with these modalities.

*Peripheral fatigue*

Muscular force production during a bout of resistance exercise is also understood to be facilitated and/or impaired from processes occurring distal to the neuromuscular junction. These peripheral mechanisms regulate force production through a series of intramuscular signalling events, intrinsic to a contracting muscle, that affect muscular excitation-contraction coupling and ultimately the rate and magnitude of myofibrillar cross bridge binding (Bigland-Ritchie & Woods, 1984; Fitts, 1994; Allen *et al.*, 2008). Peripheral fatigue is commonly reported using the amplitude, rate and temporal characteristics of a single electrically evoked twitch at rest (Figure 2), that can be used to estimate whether reductions in muscular strength and/or power following a bout of resistance exercise are the result of impaired intrinsic contractile functioning at one or more stages of the excitation-contraction coupling process (Merton, 1954; Stephens & Taylor, 1972; Westerblad *et al.*, 1997; Ortenblad *et al.*, 2000; Lamboley *et al.*, 2014). Additionally, fatigue of intrinsic contractile processes, particularly high frequency cross bridge dynamics, can be observed using tetanic contractions evoked from high intensity stimulations applied in series. However, this technique is less commonly used as it has been known to cause pain and discomfort. Currently, literature that has compared a single session of failure and non-failure exercise has not observed the factors that promote an increase in peripheral fatigue following exercise. An increase in central fatigue reported in these investigations has therefore been inadvertently proposed as the likely mechanism for the observed reductions in muscular strength and power (Rooney *et al.*, 1994; Linnamo *et al.*, 1998). However, peripheral mechanisms are expected to have at least partially contributed to this response.

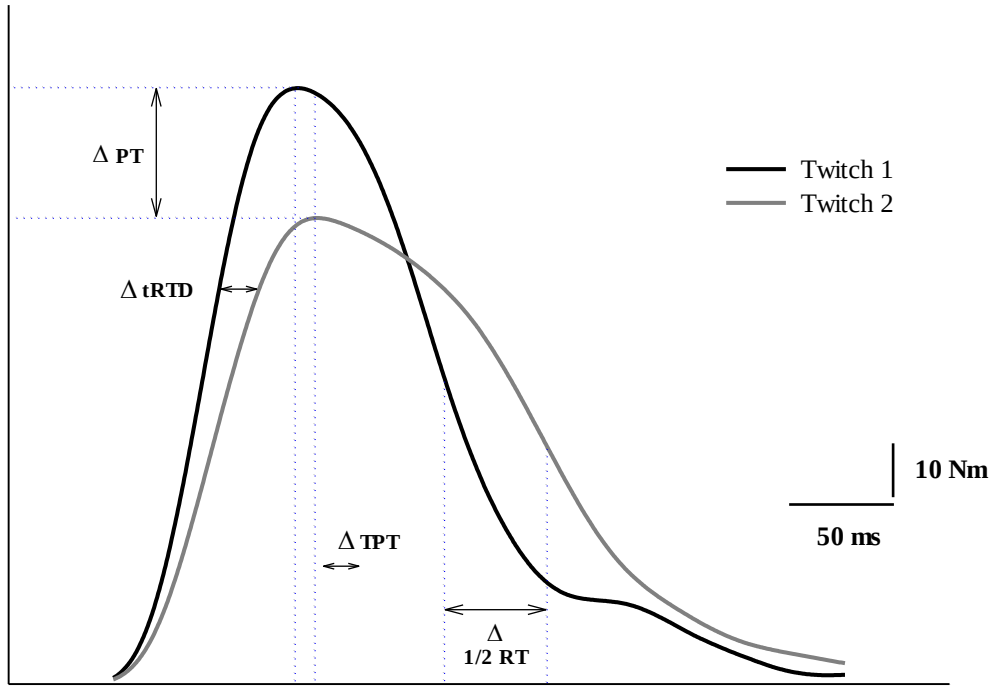


**Figure 2.** A typical pre- (Twitch 1) and post-fatigue (Twitch 2) resting twitch illustrating the change (Δ) in the amplitude (PT), rate (twitch rate of torque development; tRTD), and temporal (time to peak torque, half relaxation time; TPT and ½ RT, respectively) characteristics that occurs with fatiguing resistance exercise. A resting twitch is observed as an involuntary increase in muscular force (torque) output, produced from low to supramaximal electrical stimulation of the axons of α-motor neurons when a muscle is relaxed. By evoking a supramaximal twitch at rest, the influence of neural input to the α-motor neurons is largely negated and thus, the maximal amplitude, rate and temporal characteristics provide a global estimation of the processes that contribute to excitation-contraction coupling such as $Ca^{2+}$ release (Ortenblad *et al.*, 2000), reuptake (Lamboley *et al.*, 2014) and the rate of $Ca^{2+}$ binding to the contractile proteins (Westerblad *et al.*, 1997).

*Resting twitch.* The acute reduction in muscular strength and power with failure and non-failure exercise, and in particular, the greater decline in these variables observed following failure based exercise (Rooney *et al.*, 1994; Linnamo *et al.*, 1998; Drinkwater *et al.*, 2005) may be a product of an increase in peripheral fatigue. In the investigation by Linnamo *et al.* (1998), participants completed the failure task with an exercise volume more than twice that of the comparative non-failure condition. It is possible that the significantly greater reduction in muscular strength observed by the authors following the failure, compared to the non-failure task (21 % and 12 % decline, respectively) (Linnamo *et al.*, 1998) was the result of a greater increase in peripheral fatigue that is understood to impair muscular strength with larger volumes of failure based exercise. In the investigation by Behm *et al.* (2002), in which the authors compared a single set of 5 RM, 10 RM and 20 RM elbow flexion contractions performed to failure, peripheral fatigue, indicated by a reduction in resting twitch amplitude, was significantly greater following the 20 RM compared to the 5 RM and 10 RM tasks. Alternatively, studies that have matched exercise volume between failure and non-failure tasks have also observed greater reductions in muscular strength and power following failure based exercise (Rooney *et al.*, 1994; Drinkwater *et al.*, 2005). These findings may be indicative of the larger increase in peripheral fatigue incurred as a result of the greater contractile duration for a given failure compared to non-failure set. Motor unit recruitment has been proposed to occur in an orderly sequence during muscular contractions whereby the smallest, low force threshold (type I) motor units are recruited before the larger, high force threshold (type II) motor units (Henneman, 1957; Milner-Brown *et al.*, 1973b, 1973a). Furthermore, motor units typically innervate muscle fibres with relatively homogenous contractile properties, classified as being either slow contracting, fatigue resistant (type I) or fast contracting, fast fatigable (type II) (Burke *et al.*, 1973). Therefore, because the contractile time under tension is longer for a given failure compared to a non-failure based exercise set, a

larger decline in muscular strength and power following failure exercise may occur from the peripheral fatigue incurred from a comparatively larger recruitment of the fast fatigable, type II muscle fibres. However, whether peripheral fatigue would indeed be responsible for a greater reduction in muscular strength and power following a failure compared to non-failure task remains to be seen.

Variability in motor unit composition and associated type I and type II muscle fibre distribution between the elbow flexor (Rooney *et al.*, 1994; Benson *et al.*, 2006), pectoral (Drinkwater *et al.*, 2005) and quadriceps (Linnamo *et al.*, 1998; Marshall *et al.*, 2012) muscle groups (Johnson *et al.*, 1973) is another factor that may contribute to the disagreement between studies that have observed changes in muscular strength and power output following a bout of failure and non-failure exercise. An investigation in which participants performed isometric MVCs of the knee extensors for 10 sets × 5 repetitions reported a significant, 42 % decline in maximal strength and an up to 56 % reduction in maximal and early phase voluntary power output by the conclusion of exercise (Buckthorpe *et al.*, 2014). The reduction in voluntary strength and power occurred with a concurrent ~20 % decline in resting twitch rate and amplitude characteristics, therefore demonstrating that peripheral factors likely contributed to the fatigue incurred following exercise utilising muscles with a high percentage of type II fibres (Johnson *et al.*, 1973). These findings are extended by a recent investigation which reported a significant reduction in knee extensor voluntary strength and power that was accompanied by an up to 70 % reduction in knee extensor resting twitch rate and amplitude parameters following sustained, 40 % and 80 % MVC isometric contractions (Marshall *et al.*, 2015). In contrast, Garland and colleagues observed no change in plantar flexor resting twitch amplitude characteristics with fatiguing, 30 % and 65 % MVC sustained isometric plantar flexion contractions performed to failure (Garland *et al.*, 2003).

Compared to the knee extensors, the plantar flexor muscle group, and in particular soleus, is understood to contain a higher percentage of type I muscle fibres (Johnson *et al.,* 1973). The lesser degree of peripheral fatigue observed in the plantar flexors compared to the knee extensors would therefore be expected given the superior fatigue resilience of type I muscle fibres (Colliander *et al.*, 1988) owing to innate membrane characteristics such as the generation of smaller action potentials and slower depolarisation and conduction velocities (Buchthal *et al.*, 1973; Milner-Brown & Miller, 1986). However, the disagreement between these studies may also be a consequence of the effect of prior training experience on muscle fibre composition, and thus, peripheral fatigue.

The amount of peripheral fatigue following resistance exercise has been demonstrated to be greater in trained compared to untrained individuals. The 70 % decline in resting twitch rate and amplitude characteristics following sustained isometric exercise in trained individuals (Marshall *et al.*, 2015) contrasts the ~20 % (Buckthorpe *et al.*, 2014; Siegler *et al.*, 2014) and 48 % (Neyroud *et al.*, 2012) decline in untrained individuals following repetitive and sustained isometric contractions performed to failure, respectively. Trained individuals may experience comparatively greater levels of peripheral fatigue since untrained persons may not possess the tolerance to push themselves to the same degree of fatigue and thus, 'give up' before true failure is reached. The larger increase in peripheral fatigue observed in trained individuals may also be a function of the changes in type II muscle fibre morphology and distribution, such as an increase in type II fibre area and the distribution ratio of type II-to-type I fibres as a result of continuous resistance exercise training (Hakkinen *et al.*, 1985b). Alternatively, given the large increase in peripheral fatigue following a sustained isometric contraction (Neyroud *et al.*, 2012; Marshall *et al.*, 2015) and the relatively smaller increase observed with repetitive isometric contractions (Buckthorpe *et al.*, 2014; Siegler *et al.*, 2014),

the disagreement between these investigations may demonstrate that peripheral fatigue is a function of contraction type. Furthermore, given resting twitch amplitude has been observed to be well maintained in trained individuals following a high intensity (85 % 1 RM) dynamic loading protocol (4 sets × 3 repetitions) (Walker *et al.*, 2009), peripheral fatigue in trained individuals alone may be dependent on whether exercise is of an isometric (Marshall *et al.*, 2015) or dynamic (Walker *et al.*, 2009) nature. Additionally, because exercise sets are unlikely to be performed to failure with three repetitions of an 85 % 1 RM load, the investigation by Walker *et al.* (2009) may demonstrate that peripheral factors do not impair muscular strength and power production following non-failure based exercise. However, it is currently unknown whether the magnitude of peripheral fatigue will be different following a comparison of failure and non-failure exercise. Observing the peripheral factors responsible for reductions in muscular strength and power following these modalities in trained populations and between contraction types may also help to address the disagreements between studies that have directly compared failure and non-failure exercise. Nevertheless, whilst determining the mechanisms of fatigue following single session of failure and non-failure exercise will provide additional information on the necessity to perform resistance exercise to failure or not to failure acutely, it is unknown whether maximising acute mechanistic fatigue using either failure or non-failure based exercise is indeed necessary to optimise adaptations in muscular strength and power following a period of training.

## FAILURE AND NON-FAILURE EXERCISE: TRAINING STUDIES

Resistance exercise is commonly prescribed over training cycles lasting weeks to months for the purposes of increasing muscular strength, power and/or hypertrophy. Strength and conditioning practitioners and researchers have long sought to determine the ideal exercise stimulus that optimises these adaptations in the shortest possible time without compromising athletic performance, although no consensus has yet been reached. For the last 20 years, many practitioners have prescribed resistance exercise to the point of failure because this programming method has demonstrated effectiveness at promoting significant improvements in muscular strength following short term training (Rooney *et al.*, 1994). However, recent investigations that have compared a period of failure and non-failure based training have suggested that non-failure exercise can be used to achieve similar improvements in muscular strength and power whilst avoiding deleterious training outcomes shown to occur with failure based exercise programming (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). Therefore, the current literature within this field disagrees whether failure based resistance exercise prescription is the ideal programming method to be used when the goal of a period of training is to improve muscular strength and power. The findings of this literature are summarised in Table 2.

**Table 2.** Summary of the literature that has observed chronic changes in muscular strength, power and hypertrophy following a comparison of failure and non-failure exercise

| Study | Muscle group | Duration | Participant details | Methods | Results |
|---|---|---|---|---|---|
| Rooney *et al.* (1994) | Elbow flexors | 6 weeks | Untrained | Isotonic elbow flexion | ↑ 56.3 % 1RM, ↑ 22.1 % isometric MVC; *F*<br>↑ 41.2 % 1RM, ↑ 19.8 % isometric MVC; *NF* |
| | | | Age (18-35 years) | 6RM load | |
| | | | *n* = 14 (*F*) | | ↑ 1RM *F* > *NF* |
| | | | *n* = 14 (*NF*) | *F:* 1 × 6 reps (Weeks 1, 3, 5)<br>1 × 10 reps (Weeks 2, 4, 6) | |
| | | | | *NF:* 6 × 1 rep, 30 sec rest b/w reps<br>10 × 1 rep (Weeks 2, 4, 6) | |
| Schott *et al.* (1995) | Knee extensors | 14 weeks | Untrained | Isometric knee extension | ↑ 54.7 % isometric MVC, ↑ muscle size, ↑ metabolite c. ; *F*<br>↑ 31.5 % isometric MVC, ↔ muscle size, ↑ metabolite c. ; *NF* |
| | | | Age (22.7 years) | 70 % isometric MVC | |
| | | | *n* = 7 | | ↑ isometric MVC, muscle size, metabolite c. *F* > *NF* |
| | | | | *F* (left leg): 4 × 1 rep, 30 sec contraction,<br>1 min rest b/w sets | |
| | | | | *NF* (right leg): 4 × 10 reps, 3 sec contraction,<br>2 min rest b/w sets | |
| Folland *et al.* (2002) | Knee extensors | 9 weeks | Untrained | Isotonic knee extension | ↑ 34 % 1RM, ↑ 18.2 % isometric MVC; *F*<br>↑ 40 % 1RM, ↑ 14.5 % isometric MVC; *NF* |
| | | | Age (21 years) | 75 % 1RM load | |
| | | | *n* = 12 (*F*) | | |
| | | | *n* = 11 (*NF*) | *F:* 4 × 10 reps, 30 sec rest b/w sets | |
| | | | | *NF:* 40 × 1 rep, 30 sec rest b/w reps | |

*Table 2 continued on next page*

*Table 2 continued*

| Study | Muscle group | Duration | Participant details | Methods | Results |
|---|---|---|---|---|---|
| Drinkwater *et al.* (2005) | Pectorals/ elbow extensors | 6 weeks | Trained (0.5-3years)<br><br>Age (18.6 years)<br>*n* = 15 (*F*)<br>*n* = 11 (*NF*) | Isotonic bench press<br><br>80-105 % of 6RM load<br><br>Fixed session duration 13 min 20 sec<br><br>*F:* 4 × 6 reps, 260 sec rest b/w sets<br>*NF:* 8 × 3 reps, 113 sec rest b/w sets | ↑ 10 % 6RM, ↑ 12 % bench press power; *F*<br>↑ 5 % 6RM, ↑ 7 % bench press power; *NF*<br><br>↑ 6RM, bench press power *F* > *NF* |
| Izquierdo *et al.* (2006) | 1. Pectorals/elbow ext<br>2. Quadriceps/gluteals | 16 weeks | Trained (up to 12.5 years)<br><br>Age (24.4 years)<br>*n* = 14 (*F*)<br>*n* = 15 (*NF*) | Isotonic bench press (bp) and back squat (bs)<br><br>2 min rest b/w sets<br><br>Week 1-6 (load): 10RM bp, 80 % 10RM bs<br>*F:* 3 × 10 reps, *NF:* 6 × 5 reps<br>Week 7-11 (load): 6RM bp, 80 % 6RM bs<br>*F: 3* × 6 reps, *NF*: 6 × 3 reps<br>Week 12-16 (load): 5RM bp and bs w/ low intensity ballistic exercise<br>*F/NF:* 3 × 2-4 reps | ↑ 23 % 1RM bp, ↑ 22 % 1RM bs; *F*<br>↑ 23 % 1RM bp, ↑ 23 % 1RM bs; *NF*<br><br>↑ 27 % bp power, ↑ 26 % bs power; *F*<br>↑ 28 % bp power, ↑ 29 % bs power; *NF*<br><br>↑ bs power *NF* > *F* |

*Table 2 continued on next page*

*Table 2 continued*

| Study | Muscle group | Duration | Participant details | Methods | Results |
|---|---|---|---|---|---|
| Izquierdo *et al.* (2010) | Elbow/shoulder flexors, muscles of the back | 8 weeks | Trained (12.1 years)<br><br>Age (24.7 years)<br>*n* = 14 (*F*)<br>*n* = 15 (*NFa*)<br>*n* = 6 (*NFb*) | Isotonic bench pull<br><br>Rest b/w sets not stated<br><br>75-92 % 1RM load<br><br>*F:* 4 back exercises, 3-4 sets × 4-10 reps<br>*NFa:* 4 back exercises, 3-4 sets × 2-5 reps<br>*NFb:* 2 back exercises, 3-4 sets × 2-5 reps | ↔ 1RM bench pull, ↔ bench pull power; *F, NFb*<br>↑ 4.6 % 1RM bench pull, ↑ 6.4 % bench pull power; *NFa*<br><br>↑ 1RM bench pull, power *NFa* > *F, NFb* |
| Fisher *et al.* (2015) | Knee extensors | 6 weeks | Untrained<br><br>Age (21.4 years)<br>*n* = 9 | Isotonic knee extension<br><br>80 % 1RM load<br><br>2 min rest b/w sets<br><br>*F* (non-dominant leg): Reps to failure each set, 25 reps accrued<br>*NF* (dominant leg): 5 × 5 reps | ↑ 46 % isometric MVC; *F*<br>↑ 40 % isometric MVC; *NF* |

*Table 2 continued on next page*

*Table 2 continued*

| Study | Muscle group | Duration | Participant details | Methods | Results |
|---|---|---|---|---|---|
| Sampson *et al.* (2015) | Elbow flexors | 12 weeks | Untrained | Isotonic elbow flexion | ↑ 30.5 % 1RM, ↑ 13.3 % isometric MVC, ↑ 11.4 % muscle size, ↑ 22.1 % agonist max sEMG; pooled *F*, *NFa*, *NFb* |
| | | | Age (23.8 years) | 85 % 1RM load | |
| | | | *n* = 10 (*F*) | | ↑ and ↓ antagonist max sEMG (*F* and *NFa*, respectively) |
| | | | *n* = 10 (*NFa*) | 3 min rest b/w sets | |
| | | | *n* = 8 (*NFb*) | | ↑ antagonist max sEMG *F* > *NFa* |
| | | | | *F:* 4 × 6 reps, 2 sec concentric, 2 sec eccentric | |
| | | | | *NFa:* 4 × 4 reps, max concentric acceleration, 2 sec eccentric | |
| | | | | *NFb:* 4 × 4 reps, max concentric and eccentric acceleration | |

Muscular strength, power and hypertrophic adaptations

Changes in muscular strength following a period of failure and non-failure training have been observed using the magnitude of the force produced during an isometric MVC, and the load an individual is capable of lifting once through a ROM (i.e. the 1 RM). Seven (Rooney *et al.*, 1994; Schott *et al.*, 1995; Folland *et al.*, 2002; Drinkwater *et al.*, 2005; Izquierdo *et al.*, 2006; Fisher *et al.*, 2015; Sampson & Groeller, 2015) of the previous eight (Izquierdo-Gabarren *et al.*, 2010) investigations that have compared short term (6-16 weeks), moderate to high intensity dynamic failure and non-failure training have reported that both modalities promote significant increases in muscular strength. Three of these studies have demonstrated that the increase in muscular strength with failure based exercise at the conclusion of training (10 % to 56 %) is significantly greater than that experienced from a similar period of non-failure training (5 % to 41 %) (Rooney *et al.*, 1994; Schott *et al.*, 1995; Drinkwater *et al.*, 2005). In contrast, failure based exercise prescription has been observed to be no more effective than non-failure exercise at promoting improvements in muscular strength (23 % to 43 % pooled) with training (Folland *et al.*, 2002; Izquierdo *et al.*, 2006; Fisher *et al.*, 2015; Sampson & Groeller, 2015). To date, one investigation has demonstrated that failure based prescription is not capable of improving muscular strength with training, despite a 5 % increase observed following non-failure training (Izquierdo-Gabarren *et al.*, 2010). Currently, similar disagreement exists between studies that have observed changes in muscular power with short term failure and non-failure training.

Following six weeks of bench press training performed to failure or not to failure with loads corresponding to 80-105 % of 6 RM, Drinkwater and colleagues reported that the 12 % increase in bench press power output following a period of failure based training was

significantly greater than the 7 % increase observed in the similar non-failure group (Drinkwater *et al.*, 2005). In contrast to this investigation, the study by Izquierdo *et al.* (2006) in which moderate to high intensity upper and lower body exercise was prescribed for 2-10 repetitions over 3-6 sets, demonstrated that non-failure exercise is just as effective at increasing bench press power as failure exercise (28 % pooled increase) following 16 weeks of training. However, the increase in back squat power in the non-failure group was significantly greater than the increase observed in participants who trained to failure (29 % versus 26 %, respectively) (Izquierdo *et al.*, 2006). These findings are supported by their subsequent investigation, in which bench pull power was increased by 6 % following 8 weeks of moderate to high intensity non-failure (3-4 sets × 2-5 repetitions) but not failure exercise (3-4 sets × 4-10 repetitions) (Izquierdo-Gabarren *et al.*, 2010). Therefore, it is currently unclear whether failure or non-failure based exercise should be used when the goal of resistance exercise training is to improve muscular power. Furthermore, like muscular power, there is relatively little understanding of the hypertrophic adaptations produced from a period of failure and non-failure resistance exercise training.

Muscular hypertrophy is defined as an increase in myocyte size, typically observed as an increase in the diameter and/or structural re-organisation of muscle fascicles. Muscular hypertrophy is commonly examined *in vivo* using non-invasive imaging techniques such as ultrasound, computer tomographic scanning (CT) and magnetic resonance imaging (MRI) that can detect changes in cross-sectional area (CSA) and angulation/pennation of muscle fascicles (Kawakami *et al.*, 1993; Higbie *et al.*, 1996; McCall *et al.*, 1996; Aagaard *et al.*, 2001). To date, two investigations have observed muscular hypertrophy following a comparison of failure and non-failure based training. At the conclusion of 12 weeks of high intensity (85 % 1 RM) dynamic failure (4 sets × 6 repetitions) and non-failure training (4 sets

× 4 repetitions) of the elbow flexors, Sampson *et al.* (2015) reported that both modalities significantly increased elbow flexor CSA by ~ 11 % (pooled data). In contrast, Schott *et al.* (1995) only observed increases in knee extensor CSA following 14 weeks of moderate to high intensity (70 % 1 RM) isometric failure (four sets of one, 30 sec contraction) and not non-failure training (four sets of ten, 3 sec contractions). Together, the disagreement between the investigations that have examined not only muscular hypertrophy, but also muscular strength and power following short term failure and non-failure based training are likely to result, in part, from one or more differences and/or limitations related to study design.

## Methodological differences and limitations

### *Exercise volume and inter-set recovery periods*

Of the literature that has observed chronic changes in muscular strength and power following a comparison of failure and non-failure training, only one investigation has controlled differences in the inter-set recovery period and total session duration between groups (Drinkwater *et al.*, 2005). This study matched total session inter-set recovery period duration between failure and non-failure groups, whereby participants who performed exercise to failure (4 sets × 6 repetitions, 260 sec rest between sets) completed exercise with the same total session inter-set recovery period duration (i.e. 13 min and 20 sec) as those who did not train to failure (8 sets × 3 repetitions, 113 sec rest between sets) (Drinkwater *et al.*, 2005). At the conclusion of the six week training period, the authors reported that participants who trained to failure experienced greater improvements in muscular strength than those who did not train to failure (Drinkwater *et al.*, 2005). In contrast, when non-failure exercise is performed with longer inter-set recovery periods, and thus, greater total session duration than

failure based exercise, participants who trained to failure have demonstrated similar improvements in muscular strength compared to participants in the non-failure group (Folland *et al.*, 2002; Fisher *et al.*, 2015). In the investigation by Folland *et al.* (2002), participants were required to rest for 30 sec between 4 sets × 10 repetitions performed to failure compared to 30 sec between 40, single repetitions not performed to failure. Therefore, total session inter-set recovery period duration for one exercise totalled 1 min and 30 sec in the failure group and a very inefficient 19 min and 30 sec in the non-failure group. Likewise, Fisher *et al.* (2015) observed a similar increase in muscular strength between failure and non-failure modalities despite participants in the non-failure group completing exercise with a significantly longer average total session duration than those in the failure group (12 min and 10 sec; and 7 min and 6 sec, respectively). Previous literature has demonstrated that inter-set recovery period length has a significant influence on the development of muscular strength with short term, moderate to high intensity resistance exercise training. Following a five week lower limb strength training program in which exercise was completed over 3-5 sets, 1 RM back squat has been observed to increase more significantly following 3 min compared to 30 sec inter-set recovery periods (Robinson *et al.*, 1995). Therefore, disagreements regarding the improvement in muscular strength between the current investigations that have compared a period of failure and non-failure training may be partially attributed to differences in the duration of inter-set recovery periods between the two programming methods. However, argument between these investigations may also be attributed to differences in exercise volume between failure and non-failure modalities.

In the only study that has observed a larger increase in muscular strength following a period of non-failure compared to failure training, participants who performed exercise to failure completed training sessions with a greater total volume of exercise (Izquierdo-Gabarren *et*

*al.*, 2010). In the investigation by Izquierdo *et al.* (2010), failure and non-failure exercise was performed with the same intensity (75-92 % 1 RM) and number of sets (3-4 sets) per session, although participants in the failure group (four exercises, 4-10 repetitions per set) completed exercise with double and quadruple the volume of the two non-failure groups (four exercises, 2-5 repetitions per set; two exercises, 2-5 repetitions per set, respectively). In contrast, similar improvements in muscular strength and hypertrophy have been observed between failure and non-failure modalities despite participants who performed exercise to failure completing each session with a greater number of repetitions and longer contractile time under tension (*specific values given in* Table 2) (Sampson & Groeller, 2015). Despite this finding, by equating exercise volume between failure and non-failure groups, muscular strength has been observed to increase similarly with failure and non-failure training (Folland *et al.*, 2002; Fisher *et al.*, 2015). Disagreement is further enhanced between investigations given a matched volume of failure and non-failure exercise has been observed to promote a greater increase in muscular strength following a period of failure training (Rooney *et al.*, 1994). This latter study had participants complete training with a relatively low volume of exercise per session (one set, 6-10 repetitions) (Rooney *et al.*, 1994). This volume served as an appropriate stimulus to increase muscular strength for their untrained sample demographic, although may contribute to the disagreement between this investigation and studies that have sampled from trained individuals (Drinkwater *et al.*, 2005; Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010).

*Training experience*

Trained individuals typically require a greater volume of resistance exercise to produce larger improvements in muscular strength following a period of training (González-Badillo *et al.*, 2006; Marshall *et al.*, 2011). The effectiveness of failure and non-failure exercise at improving muscular strength and power in trained populations is equivocal (Drinkwater *et al.*, 2005; Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). Authors have reported greater strength improvement with failure based exercise (Drinkwater *et al.*, 2005), similar strength improvements between modalities (Izquierdo *et al.*, 2006), and greater strength and power improvements with non-failure exercise (Izquierdo-Gabarren *et al.*, 2010). This may result from a number of factors including, but not limited to, prior training experience and the broad definition of the term 'trained.'

In the investigation by Drinkwater *et al.* (2005), participants had on average 0.5-3 years of prior training experience before commencing testing. However, the authors reported that participants had only modest upper body strength training experience within this time. The authors speculated that the observed improvements in bench press strength could therefore be attributed to early neural adaptations (Drinkwater *et al.*, 2005) that are known to contribute to muscular strength adaptations with resistance exercise training in untrained populations (Moritani & DeVries, 1979; Häkkinen *et al.*, 1996) and may not be truly reflective of strength improvements that occur in trained individuals. This explanation supports the greater increase in muscular strength following failure based training previously observed in untrained individuals (Rooney *et al.*, 1994) and may also justify the disagreement between studies that compared failure and non-failure training, in which participants had up to 12 years of training experience (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). However, the current

disagreement regarding muscular strength and power improvement between studies may also be compounded by heterogeneity of the trained sample population demographics.

It is possible that the classification of individuals as 'trained' encompasses too broad a spectrum of training experience levels. In studies that have observed muscular strength and power following a period of failure and non-failure training, authors have sampled from individuals whose prior training experience ranges between 0.5 years (Drinkwater *et al.*, 2005) to 12 years (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). Other literature external to this prescription design has sampled from populations of trained individuals with modest weight training experience ranging from six months to one year (Willardson & Burkett, 2006; Robbins *et al.*, 2010), whilst other investigations have sampled from individuals with 5+ years of continuous strength training experience (Ahtiainen *et al.*, 2005; Marshall *et al.*, 2011) or even elite level powerlifters and weightlifters with 7+ years of training and competitive experience (Häkkinen *et al.*, 1987; Ahtiainen & Häkkinen, 2009). Following a period of training, Hakkinen and colleagues observed differences in strength gains between trained individuals who performed resistance exercise with some regularity compared to those with many years of strength training experience (Hakkinen & Komi, 1981). Therefore, the current definition of a trained individual covers a broad demographic to which muscular strength may change differently with training depending on the number of years of prior experience. A medium between the two training extremes has been proposed as a minimum of two years consistent strength training at least 2-3 times per week (Rhea *et al.*, 2002; Marshall *et al.*, 2011; Marshall *et al.*, 2012). It is believed that two years of regular strength training should be long enough to avoid the confounding influence of rapid muscular strength improvements commonly associated with initial musculoskeletal adaptations to resistance exercise in untrained individuals (Seynnes *et al.*, 2007).

*Concurrent resistance and endurance exercise training*

The performance of failure and non-failure resistance exercise training with a concurrent period of endurance exercise training is another factor that may contribute to the disagreement between studies that have examined muscular strength and power following a comparison of failure and non-failure exercise modalities. As a result of recruiting from populations of national level athletes currently engaged in sports with a strong endurance and sports specific skills focus, all three studies that have examined changes in muscular strength and power following a period of failure and non-failure resistance exercise in trained individuals have had to conduct simultaneous endurance exercise training (Drinkwater *et al.*, 2005; Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). Specifically, to maintain sports specific fitness and skill sets to compete at a high level, participants completed failure and non-failure resistance training sessions combined with endurance/skills training regimens daily (Drinkwater *et al.*, 2005), four times per week (Izquierdo *et al.*, 2006), and five to six times per week (Izquierdo-Gabarren *et al.*, 2010) for the duration of the respective intervention periods. Although these studies were conducted using resistance trained athletes, the findings of these investigations may not be externally valid to other trained population demographics, such as weightlifters, that can rely solely on a resistance training regimen for improved athletic performance. Additionally, whilst resistance exercise training was closely monitored in these investigations, the authors could not control changes in muscular strength or power within, or between, studies that may have been affected by sport specific skills and/or endurance training that was external to failure and non-failure program design. Furthermore, the greater volume of moderate to high intensity exercise completed as a result of performing combined resistance exercise and endurance/skills training might interfere with muscular strength and power development (Dudley & Djamil, 1985; Sale *et al.*, 1990;

Leveritt *et al.*, 1999) by stimulating a perpetual state of fatigue or over-reaching (Fry *et al.*, 1994; Fry & Kraemer, 1997; Hedelin *et al.*, 2000). This effect is reflected by the findings of Izquierdo *et al.* (2010) in which a significant loss in fat-free mass was observed in all failure and non-failure training groups. Therefore, the current improvements in muscular strength and power observed in trained individuals in response to a period of failure and non-failure training may be confounded by muscular atrophy or the effects of over-reaching as a consequence of performing concomitant resistance and endurance exercise training.

*Determining muscular power*

Currently, muscular power output has only been observed following a comparison of short term failure and non-failure training in trained individuals. This literature has either demonstrated that failure based training is more effective at increasing muscular power (Drinkwater *et al.*, 2005), failure and non-failure modalities are equally effective at increasing muscular power (Izquierdo *et al.*, 2006) or that non-failure exercise produces greater increases in muscular power (Izquierdo-Gabarren *et al.*, 2010) (*specific values given in* Table 2). Disagreement between studies may have resulted for a number of reasons, potentially related to the method used to determine muscular power output and/or the type of muscular contraction used during the period of failure and non-failure training.

Differences in the measurement technique used to calculate muscular power output is one possible explanation for the lack of consistency in the reporting of muscular power output following failure and non-failure training. As a measure of muscular power output, Drinkwater and colleagues had participants complete a 40 kg bench press test in an explosive manner at baseline and at the conclusion of the six week training period (Drinkwater *et al.*,

2005). Consequently, the authors did not control between-participant differences or relative increases in muscular strength and/or power that may have occurred over the duration of the training period. Izquierdo and colleagues addressed this problem in their subsequent investigations by using loads that corresponded to a constant percentage of 1 RM to test muscular power output before and after their interventions (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). However, the authors calculated muscular power output using loads equivalent to either 60 % 1 RM (Izquierdo *et al.*, 2006) or the average output from a range of loads between 15-100 % 1 RM (Izquierdo-Gabarren *et al.*, 2010), making it difficult to compare findings between investigations. Calculating the rate of force development (RFD) at a single joint angle within a ROM using an explosive isometric MVC is an alternative method that can be used to calculate muscular power output that would allow for a comparison between investigations.

Disagreements between studies regarding the effectiveness of failure and non-failure exercise at improving muscular power may also be related to the speed of muscular contractions performed during training as well as the use of low intensity exercise incorporated into the final weeks of training that is designed to stimulate a 'peaking' effect. Izquierdo *et al.* (2006) instructed participants to perform the concentric phase of each muscular contraction during training with the highest possible velocity. The authors also periodised training into mesocycles of 5-6 weeks duration. Exercise was predominantly completed with moderate to high intensity loads (5-10 RM) for the duration of the 16 week training macrocycle, with low intensity ballistic exercise incorporated into a final 'peaking' mesocycle. Performing the concentric phase of each contraction with the highest possible velocity, in addition to the lower loads used in the final mesocycle was likely to have partially contributed to the larger increase in muscular strength and power output (Kanehisa & Miyashita, 1983; Hakkinen &

Komi, 1986; Behm & Sale, 1993b; Munn *et al.*, 2005) compared to previous literature (Drinkwater *et al.*, 2005).

## Measurement of neural adaptation

Improvements in muscular strength and power following a period of training are largely thought to be dependent on adaptations within the nervous system (Moritani & DeVries, 1979; Aagaard *et al.*, 2002b). Until recently, it was unknown whether the increase in muscular strength and power with failure and non-failure based training was the result of adaptations within the nervous system. To date, one investigation has demonstrated that neural adaptations, at least partially mediate an increase in muscular strength and power with failure and non-failure training (Sampson & Groeller, 2015).

### *Gross measures of neural adaptation*

*Surface electromyography*. In the investigation by Sampson *et al.* (2015), the 22 % pooled increase in biceps brachii maximal sEMG amplitude following a 12 week period of failure and non-failure training of the elbow flexors suggested that failure and non-failure modalities can similarly improve muscular strength by facilitating an increase in motor unit output. However, it is unknown whether a greater increase in motor unit output is responsible for the larger improvements in muscular strength and power currently observed with either failure (Rooney *et al.*, 1994; Schott *et al.*, 1995; Drinkwater *et al.*, 2005) or non-failure training (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010). A previous study has demonstrated that neural adaptations likely facilitate the development of muscular power with non-failure based training (Van Cutsem *et al.*, 1998). This report may provide some explanation for the

recent trend that has demonstrated that non-failure exercise is more effective at promoting improvements in muscular power output than failure based exercise (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010).

To observe neural adaptations within the dorsiflexor muscle group following short term resistance exercise training, Van Cutsem and colleagues used fine wire needle electromyography to record motor unit action potentials, and sEMG to detect gross changes in dorsiflexor motor unit output (Van Cutsem *et al.*, 1998). Additionally, the authors used a single supramaximal electrical stimulation to evoke a resting twitch, the amplitude and temporal characteristics of which were used to determine the extent of peripheral adaptations to training. For the duration of the 12 week study period, participants were required to complete five training sessions per week, in which, they performed ten sets of ten, high velocity dorsiflexion contractions with loads corresponding to 30-40 % of 1 RM (i.e. not to failure). At the conclusion of training, the authors reported that muscular power output increased by 53 % of baseline values, with an accompanying increase in motor unit firing frequency and an earlier onset of muscle activity, despite no change in the amplitude and temporal characteristics of the resting twitch (Van Cutsem *et al.*, 1998). Therefore, this study demonstrated that central, and not peripheral factors, likely mediate an increase in power output following high velocity, low intensity non-failure training. These findings may provide some mechanistic explanation for the larger increase in power following high velocity non-failure contractions observed by Izquierdo *et al.* (Izquierdo *et al.*, 2006; Izquierdo-Gabarren *et al.*, 2010), although direct observation of the central and peripheral factors that contribute to increases in power output with controlled cadence moderate to high intensity failure and non-failure training are unknown.

Multiple studies have also demonstrated that improvements in muscular strength and power following resistance exercise training are likely produced from increased motor unit output, observed using sEMG. Hakkinen *et al.* (1985b) have observed an 11 % increase in maximal knee extensor strength and a 24 % increase in muscular power at the conclusion of a combined, low intensity ballistic and moderate to high intensity (60-80 % 1 RM) lower limb resistance training program. Given the concomitant increase in maximal quadriceps sEMG and average sEMG rate of rise characteristics, the authors inferred that an increase in neural drive was responsible for the improvement in muscular strength and power with training (Hakkinen *et al.*, 1985b). Similarly, Aagaard and colleagues reported an improvement in knee extensor strength and power following a dynamic lower limb training program (4-5 sets per exercise, 3-12 RM loads), that was likely modulated by an increase in motor unit output, observed as an increase in early phase quadriceps sEMG rate of rise (Aagaard *et al.*, 2002a). Therefore, the concurrent increase in muscle activity and muscular strength and power in these studies would suggest that motor neuron output is an important factor in development of muscular strength, particularly in the early phases of contraction (Aagaard *et al.*, 2002a). However, as reviewed earlier in this chapter, the sEMG technique has multiple limitations in its measurement of motor unit output to the muscle (Farina *et al.*, 2004).

*The interpolated twitch technique*. Using the ITT to determine VA is an alternative method that can be performed to observe adaptations in neural drive to the motor unit pool following a period of resistance exercise training. The literature typically agrees that VA is improved with strength training (Pensini *et al.*, 2002; Scaglioni *et al.*, 2002; Del Balso & Cafarelli, 2007; Nordlund Ekblom, 2010). Whilst VA has not been used to measure neural adaptation following a comparison of failure and non-failure training, Nordlund and colleagues have reported significant improvements in the level of VA following short term plantar flexor

strength training to failure (Nordlund Ekblom, 2010). Participants in this investigation were required to complete dynamic plantar flexion contractions for five sets of five repetitions with loads corresponding to 5 RM and thus, to failure (Nordlund Ekblom, 2010). Although unknown, the increase in VA and muscular strength in this investigation may demonstrate that the present improvements in muscular strength following a period of failure and non-failure training and the larger improvements following failure exercise alone (Rooney *et al.*, 1994; Schott *et al.*, 1995; Drinkwater *et al.*, 2005) are a product of greater neural drive to the motor unit pool. However, the ITT cannot differentiate whether an improvement in muscular strength and/or power is a result of neural adaptation from either spinal or supraspinal origins (Gandevia, 2001).

*Spinal and supraspinal components of neural adaptation*

*The Hoffman reflex.* The Hoffmann reflex (H-reflex) can be used to observe neural adaptations from processes occurring at a spinal level (Aagaard *et al.*, 2002b; Zehr, 2002) (Figure 3). Most literature has demonstrated that spinal excitability, typically reported using the maximal amplitude of the H-reflex, does not change following a period of resistance exercise training (Lagerquist *et al.*, 2006; Del Balso & Cafarelli, 2007; Duclay *et al.*, 2008; Fimland *et al.*, 2009a; Nordlund Ekblom, 2010). In the investigation by Del Balso *et al.* (2007), the authors observed no change in plantar flexor H-reflex recruitment despite a 20 % and 43 % increase in muscular strength and power, respectively, and a 61 % increase in soleus muscle activity following a four week plantar flexor training program (6 sets × 10 isometric MVCs, 3-4 sec in duration). Given the large increase in muscle activity was not observed with a concomitant increase in H-reflex recruitment, it is likely that either an increase in muscle activity is not mediated by spinal mechanisms, or potentially that, sEMG

incorrectly predicts motor unit output from spinal origins. In contrast to the findings of Del Balso *et al.* (2007) and other literature, few studies have proposed a link between spinal reflex excitability and the development of muscular strength and power with training (Aagaard *et al.*, 2002b; Holtermann *et al.*, 2007). Following short term plantar flexor training (5 sets × 10 isometric MVCs, four sec in duration), Holtermann and colleagues demonstrated an 18 % and 28 % increase in muscular strength and power, respectively, with a 17 % increase in maximal H-reflex amplitude (Holtermann *et al.*, 2007). The authors reported a significant positive correlation ($r = 0.59$, $p < 0.05$) between the improvement in maximal H-reflex amplitude observed during low force tonic contractions and muscular power, indicating that changes in excitability of the motor neuron pool at the spinal level predicted muscular power output following training. The disagreement between this study that reported an increase in spinal excitability with training and others that have observed no change, may be explained by differences in the methodology used to elicit the H-reflex.

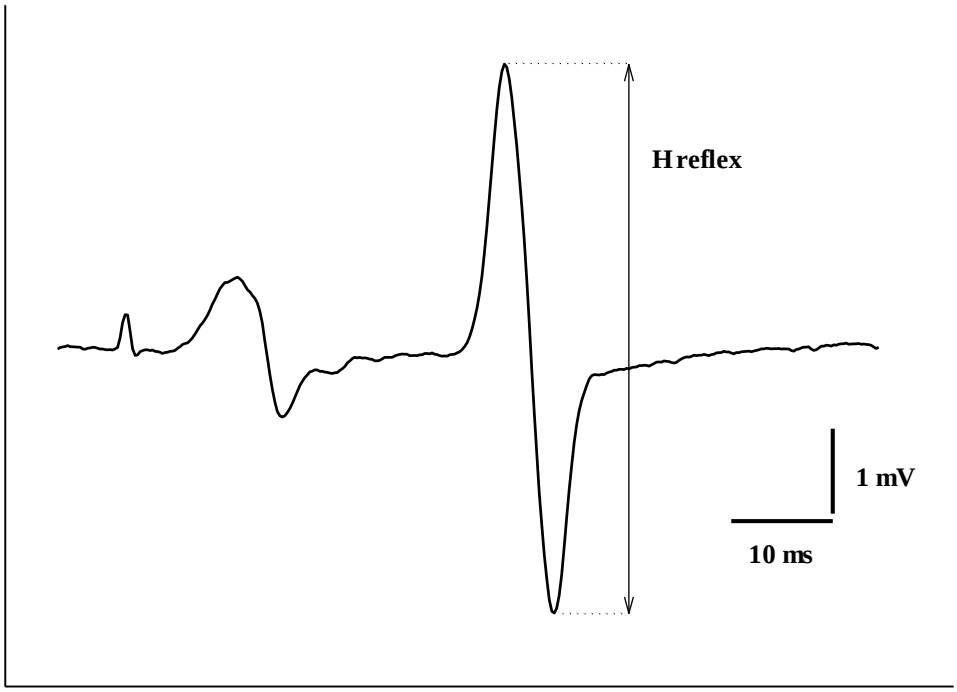


**Figure 3.** A typical H-reflex recording. An H-reflex is detected as a visible waveform on a muscle electromyographic trace when a mixed peripheral nerve is stimulated with a single pulse at low current intensities. At low current intensities, electrical stimulation preferentially activates the Ia afferent fibres given their larger axon diameter relative to α-motor neuron axons. The electrical stimulus depolarises the Ia fibres, propagating action potentials along the afferent neural arc before synapsing with spinal interneurons and α-motor neurons in the ventral horn of the spinal cord. The H-reflex increases in amplitude relatively linearly until the point of maximal afferent excitation, at which time, a maximal H-reflex occurs (Misiaszek, 2003). Beyond the point of maximal Ia afferent excitation, stimulation at higher current intensities triggers a progressively greater antidromic depolarisation of α-motor neuron axons, preventing any further increases in H-reflex amplitude.

To date, much of the literature has measured spinal excitability by evoking an H-reflex at rest (Scaglioni *et al.*, 2002; Del Balso & Cafarelli, 2007; Duclay *et al.*, 2008; Fimland *et al.*, 2009a; Dragert & Zehr, 2011). By observing spinal excitability at rest and not during tonic muscular contraction, many studies have not controlled for post-synaptic events (Knikou, 2008) and changes in motor neuron excitability known to effect passive H-reflex recruitment (Nordlund *et al.*, 2004). Furthermore, studies that have evoked an H-reflex during isometric muscular contraction have contracted using intensities of 10 % (Lagerquist *et al.*, 2006; Del Balso & Cafarelli, 2007), 20 % (Fimland *et al.*, 2009a) and 90 % (Aagaard *et al.*, 2002b) of MVC. Consequently, cortical input to the α-motor neurons has varied between studies, potentially adding to the present disagreement regarding the changes in spinal excitability that occur with resistance exercise training.

Spinal adaptations to exercise are also thought to be dependent on whether the stimulus used during training is either an endurance or resistance exercise based modality (Kyröläinen & Komi, 1994; Maffiuletti *et al.*, 2001). Therefore, the variability in resistance exercise intensity, as well as the use of either isometric (Lagerquist *et al.*, 2006; Fimland *et al.*, 2009b) or dynamic (Aagaard *et al.*, 2002b; Beck *et al.*, 2007; Nordlund Ekblom, 2010) training methods, may provide some explanation for the disagreement in the observed changes in spinal excitability within the current literature. Additionally, most of the present literature has reported changes in spinal excitability using the amplitude of a maximal H-reflex, and thus, has only provided knowledge of improvements in muscular strength and power that are produced from spinal adaptation at a single Ia afferent-to-α-motor neuron activation threshold (Klimstra & Zehr, 2008; Vila-Cha *et al.*, 2012).

The maximal H-reflex amplitude only provides an estimation of maximal Ia afferent input to the motor neuron pool, and thus the recruitment of larger motor neurons. Observing changes in Ia afferent excitability by constructing a recruitment curve for the period of ascending H-reflex recruitment (i.e. H-reflex onset to maximal amplitude, Figure 4) (Dragert & Zehr, 2011; Vila-Cha *et al.*, 2012) enables researchers to determine whether improvements in muscular strength and power following a period of resistance exercise training are produced from the recruitment of small, medium and/or large motor units (Klimstra & Zehr, 2008). An H-reflex recruitment curve may also be used to address limitations related to the non-linearity of H-reflex recruitment, allowing a comparison of the same relative level of Ia afferent input to the α-motor neurons between multiple experimental groups and from baseline assessment to the conclusion of a training period (Zehr, 2002). Literature that has observed spinal Ia afferent excitability using an H-reflex recruitment curve disagrees on the changes that occur following short term resistance exercise training. At the conclusion of three weeks of dynamic plantar flexor strength training (3 sets × 8-18 repetitions, 60-80 % 1 RM load), Villa-Cha and colleagues observed no changes in ascending H-reflex recruitment (Vila-Cha *et al.*, 2012). These findings are supported by the work of Del Balso *et al.* (2007), where plantar flexor H-reflex recruitment remained unaffected following four weeks of maximal isometric based training. In contrast, a 15 % increase in maximal dorsiflexor strength and a concomitant increase in H-reflex recruitment threshold has been observed following five weeks of dorsiflexor training (5 sets × 5 isometric MVCs, five sec in duration) (Dragert & Zehr, 2011), demonstrating that adaptation of lower threshold motor units is important for the development of muscular strength with resistance exercise training. The disagreement between studies that have observed H-reflex recruitment, and between investigations that have reported the maximal H-reflex in isolation, may be explained by processes that affect

signal transmission between the Ia afferent fibres and α-motor neurons in response to resistance exercise.

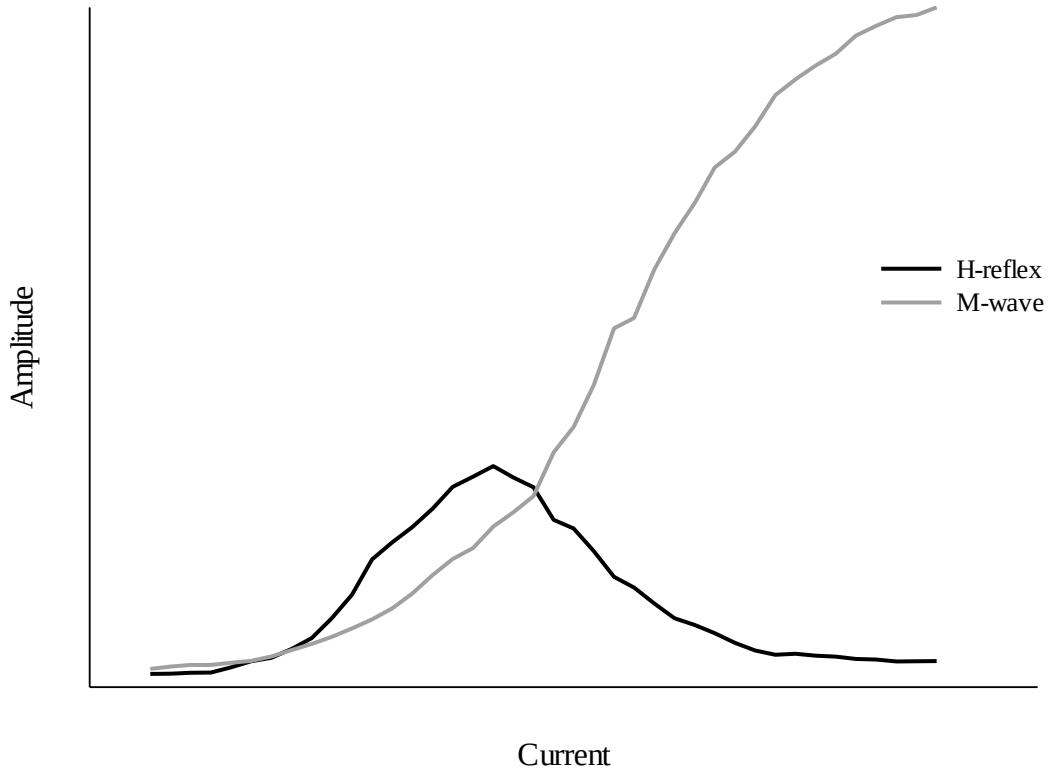


**Figure 4.** H-reflex and M-wave recruitment curves elicited using current intensities between 0 % and 100 % of the current required to evoke a maximal M-wave.

The magnitude of α-motor neuron excitation that can be attributed to spinal processes is ultimately dependent on neurotransmitter release from the Ia afferent pre-synaptic terminal and subsequent action potential depolarisation occurring on the post-synaptic membrane of an α-motor neuron. Resistance exercise is understood to promote changes in this process which can be measured by observing the degree to which signal transmission is inhibited at

the Ia afferent-to-α-motor neuron synapse (Figure 5). Neural input to the α-motor neuron pool can be inhibited from processes occurring pre- (i.e. at the Ia afferent terminal) and post-synaptically (i.e. at the α-motor neuron terminal) (Eccles *et al.*, 1962; Pierrot-Deseilligny *et al.*, 1976; Bussel & Pierrot Deseilligny, 1977; Iles *et al.*, 2000; Nordlund *et al.*, 2004). Therefore, examination of neural inhibition at the level of the spinal cord should be performed to indicate whether an increase in H-reflex amplitude is simply produced from greater Ia afferent excitation or from changes in the degree of neural inhibition to the α-motor neuron pool.

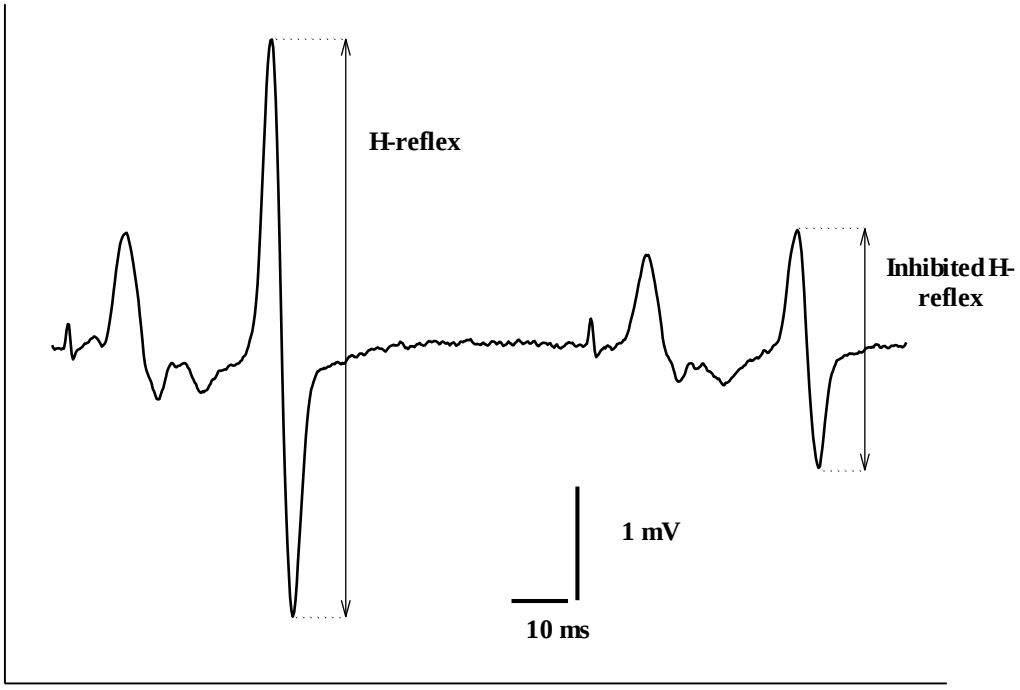


**Figure 5.** A typical inhibited H-reflex recording. Delivering two, low intensity stimuli in quick succession (i.e. a paired pulse) evokes two H-reflex waveforms. The second H-reflex is generally depressed relative to the first, and is therefore used to reflect the magnitude to which Ia afferent input to the α-motor neuron is inhibited (Hultborn *et al.*, 1996; Kipp *et al.*, 2011).

To date, neural inhibition at the Ia afferent-to-α-motor neuron synapse has not been widely researched following a period of resistance exercise training. One investigation has demonstrated that inhibition of soleus Ia afferent transmission is increased from 6 % at baseline to 22 % at the conclusion of a four week period of explosive dorsiflexor training (3 sets × 16 isometric MVCs), potentially to facilitate muscular strength and power development (20 % and 33 % increase, respectively) in the dorsiflexor muscle group (Geertsen *et al.*, 2008). Furthermore, following a bout of failure based exercise, Baudry and colleagues have observed a progressive disinhibition of pre-synaptic output which likely delayed time to task failure (Baudry *et al.*, 2011). It is possible that the acute reduction in pre-synaptic inhibition occurring with failure based exercise may therefore have important implications for the development of muscular strength and power following a period of training. However, a recent study has demonstrated that H-reflex recruitment and pre-synaptic inhibition do not predict muscular power output acutely (Johnson *et al.*, 2014). These observations may be limited by a number of factors, such as the measurement of spinal excitability and inhibition at rest and the method used to calculate inhibition. The authors calculated Ia afferent inhibition by dividing the first reflex response by the second reflex response (i.e. (1-first reflex/second reflex) × 100). Given the second waveform is inhibited and therefore often depressed relative to the first, dividing in this manner does not reflect the degree to which the second waveform is inhibited. Furthermore, calculating inhibition in this way gives a negative value, notwithstanding the positive values presented by the authors (Johnson *et al.*, 2014). Despite the problems with determining Ia afferent inhibition to the α-motor neurons in this investigation, the authors reported that cortical input to the motor unit pool, measured using the V-wave technique, likely predicted muscular power output.

*The V-wave*. The V-wave, so called because it is evoked during voluntary muscular contraction, is thought to provide an estimation of α-motor neuron recruitment produced from spinal and supraspinal processes (Del Balso & Cafarelli, 2007) (Figure 6). The general consensus within the present literature is that V-wave amplitude is increased in response to a period of resistance exercise training, demonstrating that improvements in muscular strength and/or power are likely mediated by greater spinal and supraspinal neural input to the motor unit pool (Sale *et al.*, 1983a; Aagaard *et al.*, 2002b; Del Balso & Cafarelli, 2007; Duclay *et al.*, 2008; Fimland *et al.*, 2009a; Fimland *et al.*, 2009b; Nordlund Ekblom, 2010; Vila-Cha *et al.*, 2012). However, multiple investigations that have examined both spinal and supraspinal input to the motor unit pool using the H-reflex and V-wave, respectively, have only observed an increase in the amplitude of the V-wave following a period of resistance exercise training (Del Balso & Cafarelli, 2007; Duclay *et al.*, 2008; Fimland *et al.*, 2009a; Nordlund Ekblom, 2010). In one study, Del Balso *et al.* (2007) reported that H-reflex recruitment was maintained following a period of plantar flexor training despite a 57 % increase in soleus V-wave amplitude, likely demonstrating that cortical input to the motor unit pool contributes to improvements in muscular strength and power with training. Therefore, measures of spinal input to the motor unit pool should not be used to observe neural adaptations to resistance exercise training in isolation given spinal and supraspinal factors seem to differentially affect the development of muscular strength and power, with output from the motor cortex likely facilitating improved training outcomes. Furthermore, because supraspinal input to motor unit pool (measured using the V-wave), is generally improved with resistance exercise training, it is unknown whether cortical drive will continue to facilitate muscular strength and power development in individuals that have already experienced significant adaptation or if improvements in these variables will be dependent on different mechanisms. Currently, the

literature that has examined spinal and supraspinal adaptations following a period of resistance exercise training has sampled from untrained population demographics.

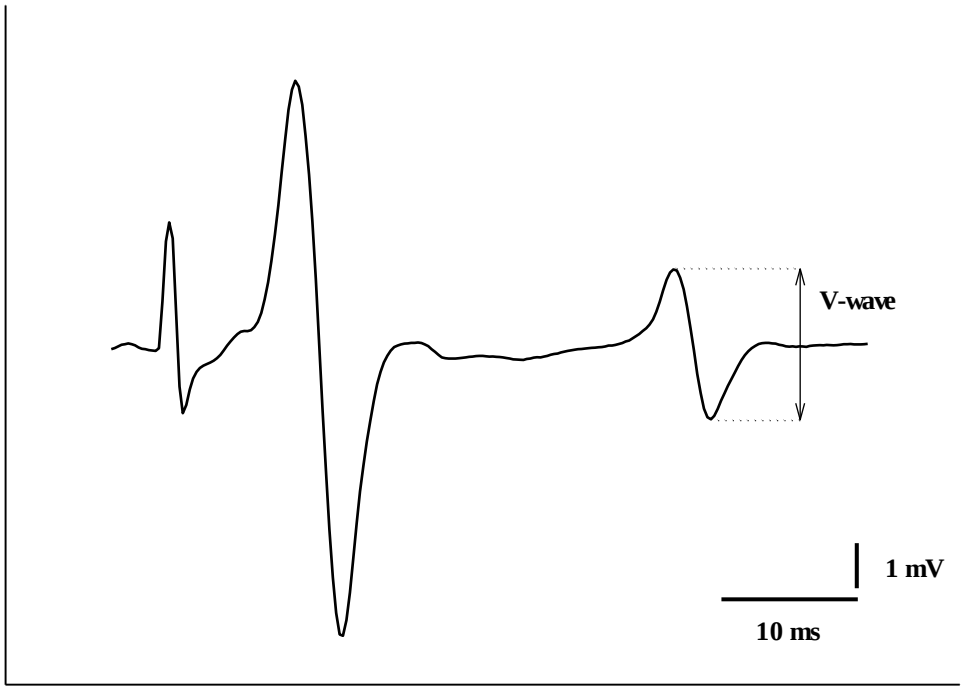


**Figure 6.** A typical V-wave recording. A V-wave is observed as a visible waveform on a muscle electromyographic trace when a single electrical stimulation is delivered to the axons of a mixed peripheral nerve during voluntary muscular contraction (Upton *et al.*, 1971; Sale *et al.*, 1982). Commonly, a V-wave is evoked from supramaximal stimulation of these axons during the phase of peak isometric force production. The maximal voluntary orthodromic neural volley produced from the motor cortex during maximal muscular contraction collides with the maximal antidromic motor volley evoked from supramaximal α-motor neuron stimulation, cancelling one another out (Aagaard *et al.*, 2002b). Subsequently, the orthodromic afferent volley, generated from the concurrent activation of Ia afferent fibres, can pass through to the muscle relatively unaffected. Therefore, the V-wave is thought to provide an estimation of α-motor neuron output produced from both spinal and supraspinal input to the motor unit pool (Del Balso & Cafarelli, 2007).

Individuals with many years of training experience have often already experienced significant changes in spinal and supraspinal neural functioning. The possibility of further neural adaptation and its role in subsequent muscular strength and power development is unknown in this demographic. Ia afferent (Nielsen *et al.*, 1993) and cortical (del Olmo *et al.*, 2006) input to the motor unit pool is understood to be greater in trained compared to untrained individuals. At the same time, trained individuals have demonstrated greater pre- (Nielsen *et al.*, 1993) and post-synaptic inhibition (Earles *et al.*, 2002) relative to untrained individuals. In contrast, Ia afferent excitability (Casabona *et al.*, 1990) and pre-synaptic inhibition (Earles *et al.*, 2002) have been reported as being lower in trained populations. Given spinal and supraspinal functioning is believed to be different between trained and untrained individuals, the current body of literature that has observed training induced changes in these variables and muscular strength and power using untrained persons may not be externally valid to trained population demographics. Therefore, it seems important for researchers to examine spinal and supraspinal adaptations in trained population demographics, and to determine an exercise modality that may be used to maximise these adaptations for continued improvement in recreational and athletic activities that require muscular strength and power for successful sporting performance.

# CHAPTER 3

*Acute Failure and Non-failure Exercise of the Knee Extensors*

## METHODS

### Subjects

Seven healthy resistance trained males ($n$ = 7) volunteered to participate in the study (age, 26.9 ± 5.1 years; height, 181.4 ± 9.4 cm; body mass, 87.2 ± 8.9 kg; training experience, 5.2 ± 2.4 years; mean ± SD). All participants were required to have regularly (at least 3 days per week) performed resistance exercise of the upper and lower body for the previous 24 months. Participants were excluded if they reported taking performance enhancing substances as per the World Anti-Doping Agency's 2012 prohibited list, had a recent history of upper or lower limb injury that may limit performance of the exercise task, or any known metabolic or neuromuscular disease. Participants were instructed to refrain from any resistance or anaerobic lower limb exercise and maintain normal dietary habits for 48 hours preceding each testing session. All participants gave written informed consent prior to testing. Experimental procedures were approved by Western Sydney University's Human Research Ethics Committee (approval number H9859) and were carried out in accordance with the Declaration of Helsinki.

### Experimental design

Participants were required to attend the laboratory for one familiarisation session followed by two experimental sessions separated by a minimum of 72 hours. The initial familiarisation served to accustom participants to procedures used to assess knee extensor maximum voluntary isometric torque output (isokinetic dynamometer, KinCom 125, Version 5.32, Chattanooga, USA), and central and peripheral fatigue using the interpolated twitch

technique (ITT). On two separate days, participants completed the failure and non-failure sessions, firstly performing the failure protocol enabling the non-failure condition to be completed with a matched contraction time under tension and thus equivalent total session duration and volume.

## Experimental procedures

### *Maximal voluntary contractions and exercise testing*

All measurements were performed on the knee extensors of the left leg. Participants sat upright in the dynamometer with the hip and knee flexed to 90º throughout testing and exercise. The centre of rotation of the dynamometer lever arm was aligned with the lateral femoral condyle. The lever arm of the dynamometer was firmly strapped to the lower leg approximately 2cm superior to the lateral malleolus. Participants were securely fastened to the dynamometer with straps placed across the torso, hips and thighs. Participant alignment was recorded to maintain consistency between sessions. All torque signals were sampled at 2,000 Hz (Powerlab 16/35, ADInstruments Australia; 16 bit analog to digital conversion) and filtered with a $4^{th}$ order 10 Hz digital low pass filter prior to analysis.

Prior to each exercise trial, participants performed a brief warm up consisting of a short series of submaximal isometric knee extension contractions at 50 % and 75 % of perceived maximal effort. Following this warm up, four maximal voluntary contractions (MVCs) were completed in a random order, separated by one minute rest, with supramaximal stimulation of the femoral nerve occurring on two of four attempts. Prior to analysis, baseline values (PRE) were obtained from the average data of the four MVCs and two accompanying resting

potentiated twitches. Fatigue was assessed with single MVCs and subsequent stimulations completed at four time points (T1, T2, T3 and T4) over the course of both exercise tasks. Participants were instructed to complete all knee extension MVCs as fast and as forcefully as possible and maintain contraction for 3-4 sec.

The fatiguing exercise task utilised in both the failure and non-failure conditions was completed with a load (Nm) equivalent to 80 % of maximal voluntary isometric torque output ($T_{MAX}$), calculated using the average $T_{MAX}$ data from the four initial MVCs. Participants completed three short (5 sec) practice contractions with this 80 % loading before commencing the main exercise protocols. The failure protocol required participants to perform four sustained isometric contractions with an 80 % $T_{MAX}$ load to the point of volitional exhaustion, defined as a 2.5 % drop in torque output for 2 sec (Marshall *et al.*, 2015). The relatively small torque range and short duration before contraction termination was chosen so exercise volume could be effectively matched between conditions and to ensure the internal validity of the non-failure protocol. MVCs were performed immediately upon failure of all four contractions (T1, T2, T3 and T4). Participants received 70 sec rest from the completion of each MVC to the beginning of the next 80 % $T_{MAX}$ contraction (total 210 sec rest within the session) (Figure 7). The non-failure protocol required participants to perform eight sustained isometric contractions at 80 % $T_{MAX}$ and cease torque production before reaching the point of volitional exhaustion, determined by halving the time to exhaustion from each corresponding failure set. Distributing contractile time under tension over two contractions instead of one therefore allowed total session volume to be equated between conditions. MVCs were performed immediately upon completion of the corresponding failure time point (i.e. contractions two (T1), four (T2), six (T3) and eight (T4)). Participants received 30 sec rest from the end of each contraction or MVC to the

beginning of the next 80 % $T_{MAX}$ contraction (total 210 sec rest within the session). No participant failed whilst completing the non-failure testing session. If a participant was deemed to have failed on an odd numbered second during the failure protocol, the first of the two analogous non-failure contractions was completed for 1 sec longer than the 2nd contraction (i.e. a fifteen second failure contraction corresponded to two non-failure contractions, each lasting eight and seven seconds, respectively). Torque output was continuously displayed throughout both exercise protocols on a 25" LCD monitor (LG™, Australia), with horizontal upper and lower bound guidelines placed ± 2.5 % around the desired 80 % $T_{MAX}$ contraction intensity. Participants were required to maintain torque output within these limits during each exercise protocol. Strong verbal encouragement was provided during all MVCs and exercise tasks.

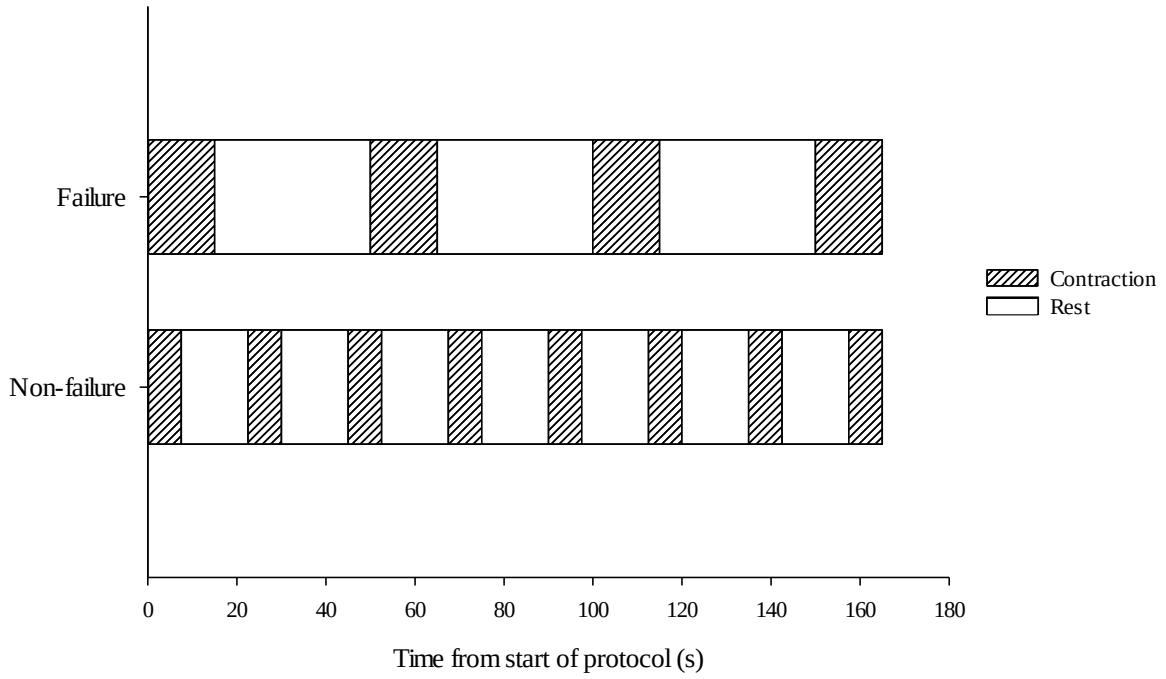


**Figure 7.** Graphical representation of the protocol design. The figure illustrates that both failure and non-failure conditions were completed with an equal total session contractile time under tension, volume and duration. For ease of viewing, the figure provides a representation of the work:rest ratio as a fraction of the total session duration.

*Electromyography*

Surface electromyograms (sEMG) were recorded from the left vastus lateralis (VL) and vastus medialis (VM) using pairs of Ag/AgCl surface electrodes (Maxsensor, Medimax Global, Australia). VL and VM electrodes (10 mm diameter, 10 mm inter-electrode distance) were applied in bipolar configuration parallel to the direction of the muscle fibres after careful skin preparation (shaving of excess hair, abrasion with fine sandpaper and cleaning the skin with isopropyl alcohol swabs) to reduce skin electrical impedance below 5 kΩ. The distal VL electrode was placed 8-12 cm superior to the lateral aspect of the patella and the distal VM electrode was placed 3-4 cm superior to the medial aspect of the patella. Placement sites were recorded for each participant with respect to anatomical landmarks to maintain consistency between sessions. The reference electrode was placed on the right patella. sEMG signals were recorded using the ML138 Octal BioAmp (common mode rejection ratio > 85 dB at 50 Hz, input impedance 200 MΩ) with a 16-bit analog-to-digital conversion, sampled at 2,000 Hz (ADInstruments, Australia). Prior to analysis, raw signals were filtered with a fourth-order Bessel filter between 20 Hz and 500 Hz and smoothed using a root mean square (RMS) calculation with a 100 ms time constant. All sEMG RMS values were normalised to the raw sEMG M-wave evoked during each analogous MVC (sEMG/M, %) to control for potential changes in axonal excitability (Pasquet *et al.*, 2000).

*Femoral nerve stimulation*

The femoral nerve was stimulated using a doublet (two, 1 ms square wave pulses with a 10 ms inter-stimulus interval; 100 Hz stimulation) applied at 400 V using a constant current stimulator (Digitimer DS7AH, Welwyn Garden City, UK). To identify nerve location for

cathodal stimulation, a rubber insulated portable cathodal probe was used to deliver low intensity stimulations (30 mA) to the femoral triangle. The femoral triangle was pre-marked with a permanent marker with optimal location determined at rest by moving the probe until the largest evoked M-wave was elicited in both VL and VM. When optimal cathodal location was identified, a single Ag/AgCl surface electrode was applied (15 mm diameter; Kendall, Covidien, USA). The anodal surface electrode was placed posterior to the greater trochanter. The level of stimulation during testing was determined by gradually increasing the current intensity in 10 mA increments with 20 sec rest between stimuli until the peak-to-peak VL and VM M-wave amplitudes and quadriceps twitch torque plateaued. The maximal stimulus intensity was recorded (range, 80-210 mA) and multiplied by 130 % to establish the supra-maximal current intensity to be applied throughout the testing session.

*Voluntary activation*

The superimposed twitch technique (Merton, 1954) was used to measure knee extensor voluntary activation (VA). A single, supra-maximal doublet stimulation was used to evoke a superimposed twitch when a visible plateau in the torque-time curve was observed for 1-2 sec during each MVC. In addition, a single, supra-maximal doublet stimulation was delivered 3-4 sec following the completion of each MVC when the participant was relaxed to evoke a resting potentiated twitch. Voluntary activation was estimated according to the following formula (Strojnik & Komi, 1998): $VA\ (\%) = 100 - [D \times (T_{SUP}/T_{MAX})/PT] \times 100$, where $D$ is the difference between the torque amplitude just before the superimposed twitch ($T_{SUP}$) and the peak torque amplitude recorded during the superimposed twitch, $T_{MAX}$ is the maximal torque amplitude recorded during the MVC, and PT is the peak torque amplitude of the resting potentiated twitch (Figure 8).

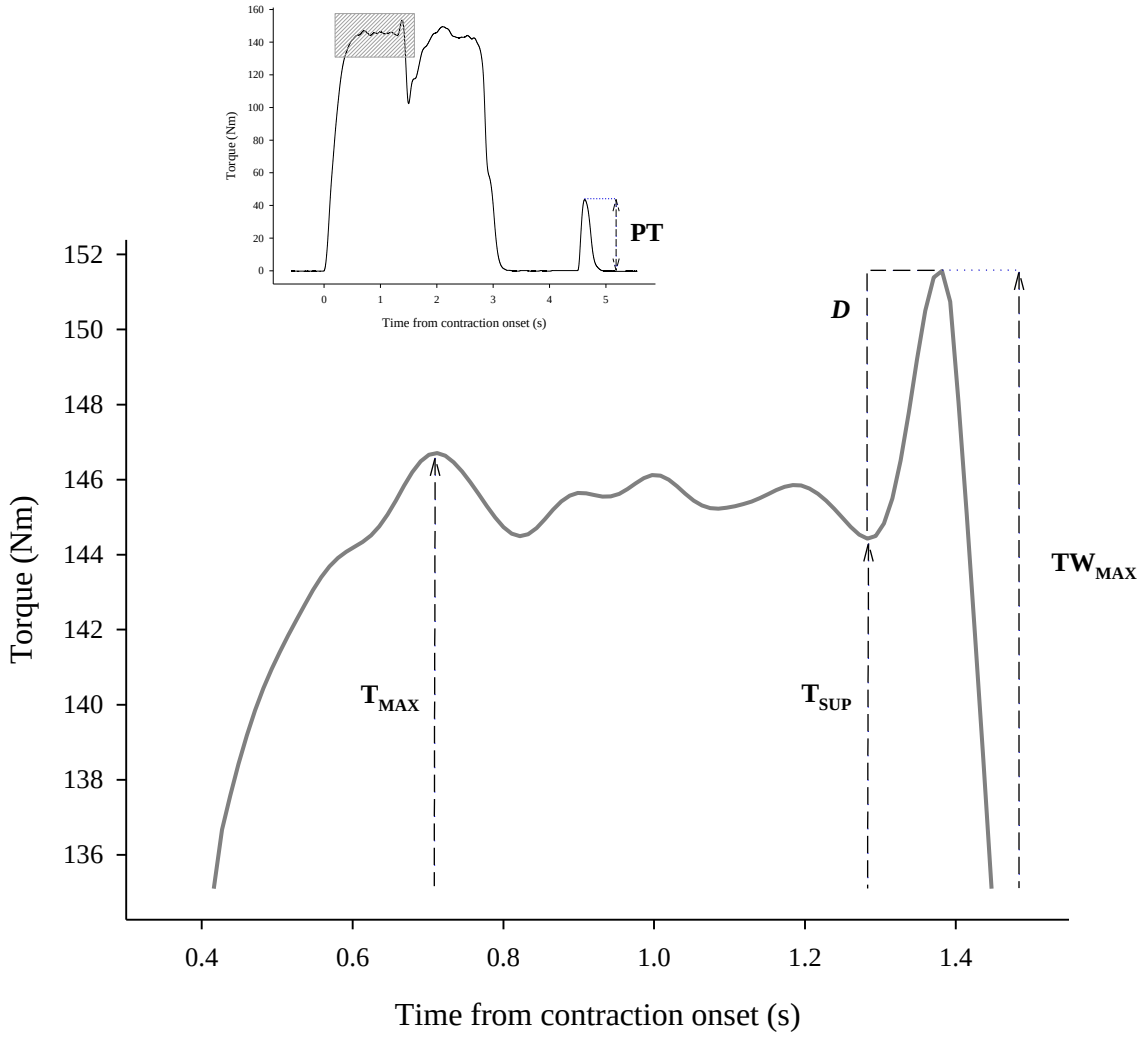


**Figure 8.** A representative maximal voluntary contraction with superimposed and resting potentiated twitch stimulations showing the ITT parameters used to calculate voluntary activation. The peak torque amplitude recorded during the MVC ($T_{MAX}$), the peak torque amplitude recorded during the superimposed twitch ($TW_{MAX}$), the peak torque amplitude recorded during the resting potentiated twitch (PT), the torque amplitude just before the superimposed twitch ($T_{SUP}$), and the difference (*D*) between the torque amplitude just before the superimposed twitch and the peak torque amplitude recorded during the superimposed twitch are illustrated on the figure.

Data processing

For all MVCs and resting potentiated twitches, torque onset was defined as the point on the torque-time curve where torque output exceeded baseline values by ≥ 1 % of the difference between baseline and peak torque amplitude. The following variables were analysed from the torque-time curve of each MVC: *1)* maximal voluntary torque output, defined as the greatest amplitude of the torque-time curve, excluding the point of stimulation ($T_{MAX,}$ Nm); *2)* normalised maximal rate of torque development ($RTD_{MAX}$), determined from the greatest average 10 ms slope of the torque-time curve (Δtorque/Δtime) throughout the first 500 ms of each MVC; and *3)* normalised average rate of torque development ($RTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms, 0-200 ms post torque onset. All rate dependent measures of voluntary torque production were normalised to $T_{MAX}$ of each analogous MVC to observe changes in RTD independent of changes to maximal torque (Holtermann *et al.*, 2007).

sEMG onset was defined 70 ms before torque onset to account for the presence of electromechanical delay (Aagaard *et al.*, 2002a). During each MVC the following variables were identified from VL and VM sEMG signals: 1) maximal sEMG activity ($VL_{MAX}$ and $VM_{MAX}$; sEMG/M, %), calculated from the greatest average 250 ms period of activity (excluding superimposed stimulation) of the RMS signal throughout each MVC; 2) maximal rate of sEMG rise ($VL_{RERmax}$ and $VM_{RERmax}$; sEMG/M, $\%.s^{-1}$), determined as the greatest average 10 ms slope (ΔsEMG/Δtime) of the RMS signal up to 200 ms post sEMG onset; 3) average rate of sEMG rise ($VL_{RERave}$ and $VM_{RERave}$; sEMG/M, $\%.s^{-1}$) of the RMS signal calculated in time intervals from 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset; 4) the maximal M-wave amplitude ($VL_{Mmax}$ and $VM_{Mmax}$; mV), determined from the peak-to-peak

amplitude of the raw sEMG signal post doublet stimulation. sEMG RER was observed up to a maximum of 75 ms post sEMG onset (instead of > 100 ms) as a decrease in RMS amplitude often occurred after this time.

The following variables were analysed from the resting potentiated twitches: *1)* resting twitch peak torque (PT, Nm), defined as the greatest amplitude of the torque-time curve; *2)* normalised resting twitch maximal rate of torque development ($tRTD_{MAX}$), defined as the greatest average 10 ms slope of the ascending limb of the twitch torque-time curve; *3)* normalised average resting twitch rate of torque development ($tRTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms post twitch torque onset; *4)* resting twitch time to peak torque (TPT, ms), defined as the time from twitch torque onset to PT; and *5)* resting twitch half relaxation time (½ RT, ms), defined as the time elapsed from PT to 50 % PT. $tRTD_{AVE}$ was observed up to a maximum of 75 ms post stimulation as reductions in twitch torque amplitude often occurred between 75 ms and 100 ms. All tRTD variables were normalised to the PT amplitude of each analogous resting potentiated twitch (Figure 9).

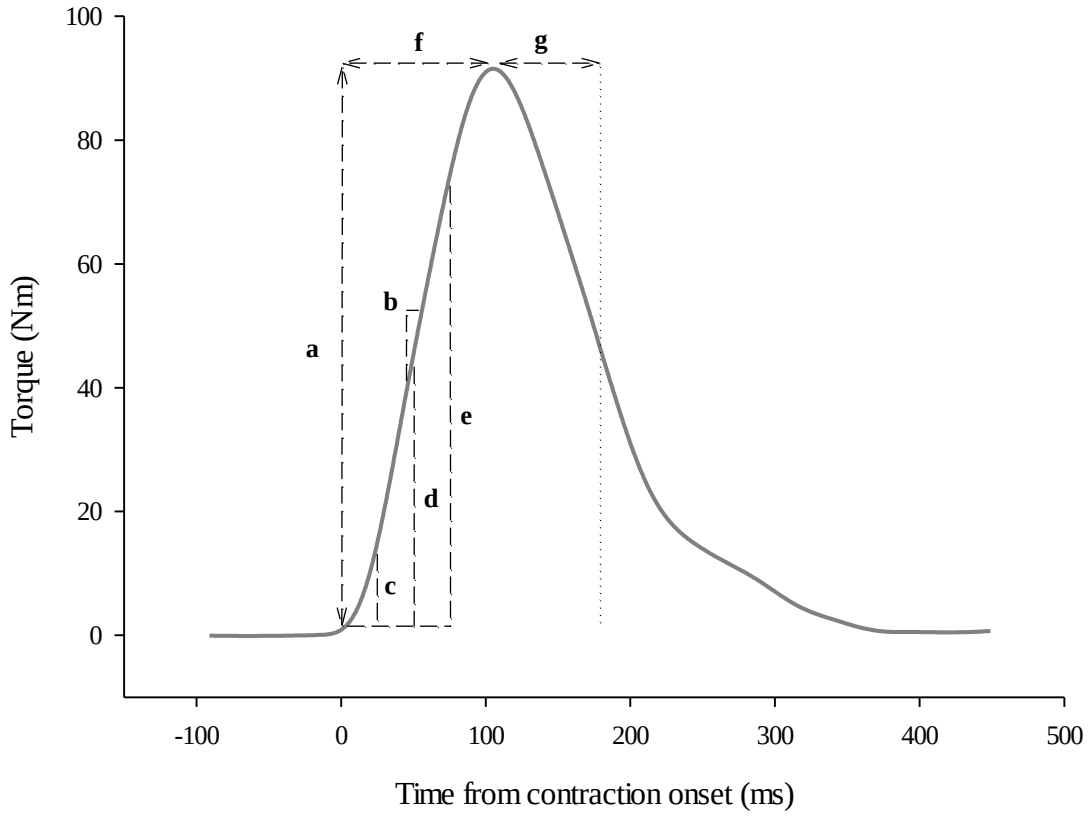


**Figure 9.** A representative resting potentiated twitch showing the parameters used for analysis. Illustrated on the figure are: *a)* resting twitch peak torque (PT); *b)* resting twitch maximal rate of torque development over the greatest 10 ms period of ascending torque production ($tRTD_{MAX}$); *c), d), e)* resting twitch average rate of torque development ($tRTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms, respectively, post twitch torque onset; *f)* resting twitch time to peak torque (TPT); *g)* resting twitch half relaxation time (½ RT).

Time under tension

Total contraction time under tension (sec) was equated between both exercise conditions to provide a measure of exercise volume between groups. Time under tension was defined as the time spent contracting above the lower bound limit (2.5 %) of the desired 80 % $T_{MAX}$ intensity. Following completion of exercise, no difference in mean time under tension was observed between failure and non-failure groups (48.9 ± 8.2 sec and 49.5 ± 11.6 sec, respectively; $p = 0.86$). Data are means ± SD.

## Reliability

Reliability analyses were completed using data from the four initial MVC recordings. The mean within-day, within-subject coefficients of variation (%) were 4.4 ± 1.7 (range 2.1 to 6.9) for $T_{MAX}$, 1.9 ± 1.7 (range 0.2 to 6.6) for VA, and 1.8 ± 1.5 (range 0.1 to 4.0) for PT. Mean between-day, within-subject coefficients of variation were 4.7 ± 4.7 (range 0.9 to 12.6) for $T_{MAX}$, 3.5 ± 4.2 (range 0.4 to 12.6) for VA, and 6.7 ± 4.1 (range 2.7 to 13.9) for PT. Data are means ± SD. The mean within-day, within-subject intra-class correlation coefficient (ICC, *r*) was 0.98 (95 % CI 0.95 to 0.99, $p < 0.001$) for $T_{MAX}$, 0.98 (95 % CI 0.94 to 0.99, $p < 0.001$) for VA, and 0.99 (95 % CI 0.97 to 1, $p < 0.001$) for PT. The mean between-day, within-subject ICC was 0.92 (95 % CI 0.58 to 0.99, $p = 0.004$) for $T_{MAX}$, 0.93 (95 % CI 0.60 to 0.99, $p = 0.003$) for VA, and 0.87 (95 % CI 0.36 to 0.98, $p = 0.011$) for PT.

## Statistical analysis

All statistical analyses were completed using IBM SPSS Statistics version 22 (SPSS Inc., Chicago, IL). All data were normally distributed, determined from Kolmogorov-Smirnov normality testing. Dependent variables were analysed using a two-way (condition × time) ANOVA for repeated measures (PRE, T1, T2, T3, T4) between conditions (failure, non-failure). In the event of a significant condition effect, post hoc repeated measures were conducted over four levels of time (TI, T2, T3, T4) with PRE data as a covariate. A separate two-tailed, paired *t*-test was applied to analyse Student's *t* distribution for total session time under tension between conditions. If Mauchly's test indicated a violation of sphericity in the ANOVA, the Greenhouse-Geisser epsilon correction was used to adjust the degrees of freedom. Post hoc comparisons were made using a Bonferroni correction, in the presence of a

significant $F$ ratio (considered significant at $p < 0.05$). The data are presented as means ± SD unless otherwise stated.

## RESULTS

Maximal torque and rate of torque development

A main time effect ($p < 0.001$) and time by condition interaction ($p = 0.002$) was observed for $T_{MAX}$. Post hoc analysis indicated $T_{MAX}$ declined from PRE at T1 by 15.0 ± 4.3 % in the non-failure condition, with a greater reduction of 23.7 ± 5.0 % ($p = 0.007$; Figure 10) observed in the failure condition.

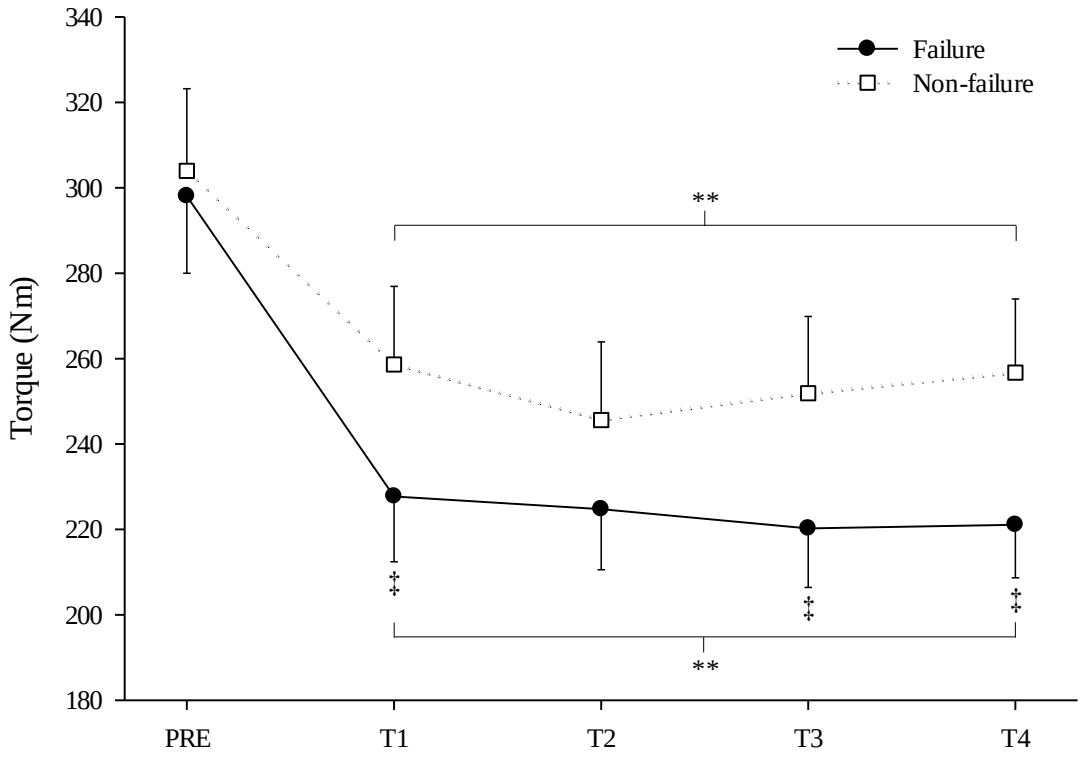


**Figure 10.** Maximal voluntary torque ($T_{MAX}$, Nm) measured during maximal voluntary knee extension contractions performed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ‡ $p < 0.01$ from non-failure condition, ** $p < 0.01$ from PRE. Data are mean and SE.

Main time effects ($p < 0.01$) were observed for normalised $RTD_{AVE}$ during time intervals 0-25 ms, 0-50 ms and 0-75 ms post torque onset. Post hoc analysis indicated $RTD_{AVE}$ declined between 24.3 % to 40.4 % ($p < 0.05$; Table 3) from PRE at T2 during time intervals 0-25 ms, 0-50 ms and 0-75 ms post torque onset. No changes were observed for $RTD_{AVE}$ during time intervals 0-100 ms and 0-200 ms post torque onset ($p \geq 0.101$) or for normalised $RTD_{MAX}$ ($p > 0.244$).

**Table 3.** Normalised maximal and average rate of torque development ($RTD_{MAX}$, $RTD_{AVE}$) measured during maximal voluntary knee extension contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions. $RTD_{AVE}$ data are presented as the average slope of the torque-time curve in time intervals of 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms and 0-200 ms post torque onset ($RTD_{AVE}$ (0-25), $RTD_{AVE}$ (0-50), $RTD_{AVE}$ (0-75), $RTD_{AVE}$ (0-100) and $RTD_{AVE}$ (0-200)). All data are normalised to the corresponding MVCs' $T_{MAX}$.

| | $RTD_{MAX}$ | | $RTD_{AVE}$ (0-25) | | $RTD_{AVE}$ (0-50) | | $RTD_{AVE}$ (0-75) | |
|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF |
| PRE | 7.9 ± 1.2 | 7.8 ± 1.2 | 4.8 ± 1.6 | 4.5 ± 1.1 | 6.1 ± 1.5 | 5.9 ± 1.2 | 6.2 ± 1.0 | 6.1 ± 0.8 |
| T1 | 6.2 ± 1.9 | 6.6 ± 1.6 | 2.8 ± 0.9 | 3.0 ± 1.2 | 4.1 ± 1.3 | 4.3 ± 1.5 | 4.7 ± 1.4 | 4.9 ± 1.3 |
| T2 | 5.6 ± 1.5 | 7.0 ± 1.5 | 2.4 ± 0.6$^{**}$ | 2.8 ± 0.7$^{**}$ | 3.4 ± 0.9$^{**}$ | 4.2 ± 0.9$^{**}$ | 4.0 ± 1.0$^{*}$ | 5.0 ± 1.0$^{*}$ |
| T3 | 6.7 ± 1.4 | 7.6 ± 1.3 | 2.8 ± 1.6 | 3.7 ± 1.6 | 4.0 ± 1.8 | 5.1 ± 1.5 | 4.7 ± 1.6 | 5.6 ± 1.1 |
| T4 | 6.9 ± 0.9 | 7.7 ± 1.5 | 2.7 ± 1.6 | 3.9 ± 1.3 | 3.9 ± 1.8 | 5.4 ± 1.4 | 4.6 ± 1.5 | 5.8 ± 1.1 |

| | $RTD_{AVE}$ (0-100) | | $RTD_{AVE}$ (0-200) | |
|---|---|---|---|---|
| MVC | F | NF | F | NF |
| PRE | 5.6 ± 0.6 | 5.5 ± 0.3 | 3.9 ± 0.3 | 3.9 ± 0.2 |
| T1 | 4.6 ± 1.2 | 4.8 ± 1.1 | 3.5 ± 0.9 | 3.6 ± 0.6 |
| T2 | 4.1 ± 1.1 | 5.0 ± 0.9 | 3.4 ± 0.7 | 3.8 ± 0.5 |
| T3 | 4.7 ± 1.2 | 5.3 ± 0.6 | 3.8 ± 0.4 | 3.9 ± 0.3 |
| T4 | 4.7 ± 1.0 | 5.4 ± 0.7 | 3.8 ± 0.3 | 4.1 ± 0.4 |

Data are presented as mean ± SD
$^{**}$ $p < 0.01$ from PRE
$^{*}$ $p < 0.05$ from PRE

Central fatigue

No changes were observed for VA ($p$ = 0.982; Figure 11; Table 4) or for measures of muscle activation using sEMG ($VL_{MAX}/VM_{MAX}$; $VL_{RERmax}/VM_{RERmax}$; and $VL_{RERave}/VM_{RERave}$ during 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset ($p \geq 0.159$; Table 5). A large effect (Cohen's effect size ($d$) = 0.82, $p \geq 0.097$) was observed for $VL_{RERave}$ 0-25 ms.

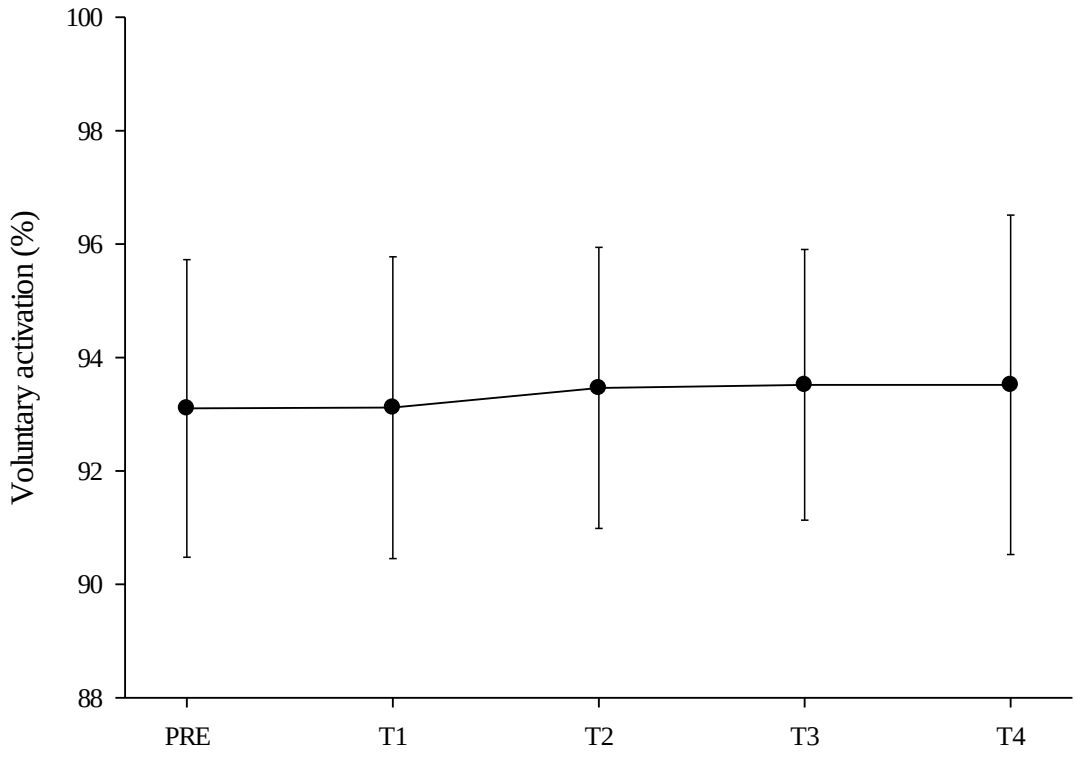


**Figure 11.** Voluntary activation (VA, %) measured during maximal voluntary knee extension contractions performed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. Data are grand mean and SE of failure and non-failure conditions.

**Table 4.** Voluntary activation (VA, %) measured during maximal voluntary knee extension contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions.

| | VA | |
|---|---|---|
| MVC | F | NF |
| PRE | 93.2 ± 8.0 | 93.1 ± 12.0 |
| T1 | 93.7 ± 9.0 | 92.5 ± 11.6 |
| T2 | 94.3 ± 6.7 | 92.6 ± 11.8 |
| T3 | 94.4 ± 7.6 | 92.6 ± 10.7 |
| T4 | 95.9 ± 5.1 | 91.1 ± 15.2 |

Data are presented as mean ± SD

**Table 5.** Vastus lateralis and vastus medialis maximal sEMG activity ($VL_{MAX}$ and $VM_{MAX}$; sEMG/M, %), maximal rate of sEMG rise up to 200 ms post sEMG onset ($VL_{RERmax}$ and $VM_{RERmax}$; sEMG/M, $\%.s^{-1}$) and average rate of sEMG rise in time intervals 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset ($VL_{RERave}$ (0-25), $VL_{RERave}$ (0-50), $VL_{RERave}$ (0-75), $VM_{RERave}$ (0-25), $VM_{RERave}$ (0-50) and $VM_{RERave}$ (0-75); sEMG/M, $\%.s^{-1}$). Data was recorded during maximal voluntary knee extension contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions. VL and VM data are expressed as a percentage of the corresponding MVCs' VL and VM maximum M-wave (sEMG/M, %), respectively.

| | $VL_{MAX}$ | | $VL_{RERmax}$ | | $VL_{RERave}$ (0-25) | | $VL_{RERave}$ (0-50) | | $VL_{RERave}$ (0-75) | |
|---|---|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF | F | NF |
| PRE | 8.5 ± 6.9 | 7.0 ± 2.9 | 181.7 ± 134.2 | 165.8 ± 82.2 | 89.7 ± 85.4 | 88.0 ± 78.7 | 86.6 ± 79.1 | 77.3 ± 49.3 | 67.7 ± 61.8 | 61.3 ± 28.5 |
| T1 | 8.0 ± 4.4 | 7.2 ± 2.8 | 177.1 ± 132.2 | 143.6 ± 90.6 | 25.1 ± 18.2 | 54.8 ± 57.6 | 47.5 ± 31.0 | 61.0 ± 57.4 | 48.9 ± 34.2 | 56.1 ± 45.7 |
| T2 | 8.6 ± 5.1 | 7.2 ± 3.1 | 218.5 ± 141.8 | 134.1 ± 71.3 | 31.3 ± 31.9 | 50.4 ± 54.4 | 59.8 ± 54.4 | 58.9 ± 48.1 | 74.5 ± 60.7 | 58.0 ± 40.2 |
| T3 | 8.5 ± 4.4 | 7.8 ± 4.3 | 164.6 ± 84.1 | 165.9 ± 94.5 | 32.3 ± 32.6 | 48.2 ± 44.3 | 54.3± 39.1 | 61.5 ± 47.7 | 57.6 ± 37.7 | 64.2 ± 44.8 |
| T4 | 9.2 ± 4.9 | 7.0 ± 3.6 | 192.3 ± 121.3 | 151.8 ± 91.9 | 26.3 ± 16.6 | 55.2 ± 50.3 | 47.0 ± 25.1 | 68.7 ± 55.8 | 62.4 ± 37.9 | 61.5 ± 38.9 |
| | $VM_{MAX}$ | | $VM_{RERmax}$ | | $VM_{RERave}$ (0-25) | | $VM_{RERave}$ (0-50) | | $VM_{RERave}$ (0-75) | |
| MVC | F | NF | F | NF | F | NF | F | NF | F | NF |
| PRE | 7.7 ± 3.6 | 8.8 ± 4.6 | 168.3 ± 55.2 | 228.0 ± 162.5 | 88.0 ± 53.4 | 131.0 ± 136.8 | 78.2 ± 46.3 | 102.1 ± 81.9 | 51.6 ± 29.4 | 60.7 ± 23.6 |
| T1 | 8.6 ± 4.8 | 10.4 ± 5.9 | 167.0 ± 120.3 | 244.5 ± 188.5 | 49.0 ± 42.2 | 80.4 ± 90.5 | 58.6 ± 29.7 | 97.3 ± 85.8 | 62.3 ± 29.8 | 94.9 ± 78.3 |
| T2 | 9.0 ± 6.4 | 7.9 ± 3.7 | 178.3 ± 102.0 | 182.0 ± 130.0 | 42.7 ± 34.8 | 52.3 ± 49.9 | 62.0 ± 45.5 | 73.7 ± 72.9 | 71.2 ± 54.7 | 68.5 ± 63.3 |
| T3 | 8.0 ± 3.4 | 9.0 ± 4.2 | 191.3 ± 102.8 | 195.9 ± 112.6 | 35.9 ± 22.4 | 56.2 ± 55.7 | 54.5 ± 23.9 | 76.5 ± 67.8 | 54.1 ± 25.7 | 78.2 ± 60.8 |
| T4 | 7.8 ± 4.1 | 8.0 ± 3.5 | 170.2 ± 80.6 | 171.1 ± 96.0 | 33.4 ± 31.5 | 51.6 ± 47.8 | 51.8 ± 34.4 | 75.0 ± 57.4 | 57.6 ± 26.1 | 66.8 ± 51.3 |

Data are presented as mean ± SD

Peripheral fatigue

A main time effect ($p < 0.001$) and time by condition interaction ($p = 0.032$) was observed for PT. Post hoc analysis indicated PT was reduced from PRE at T1 by 17.4 ± 10.1 % ($p < 0.001$), with a greater reduction from PRE at T4 (29.5 ± 8.6 %; $p = 0.016$; Figure 12; Figure 13) observed in the failure condition.

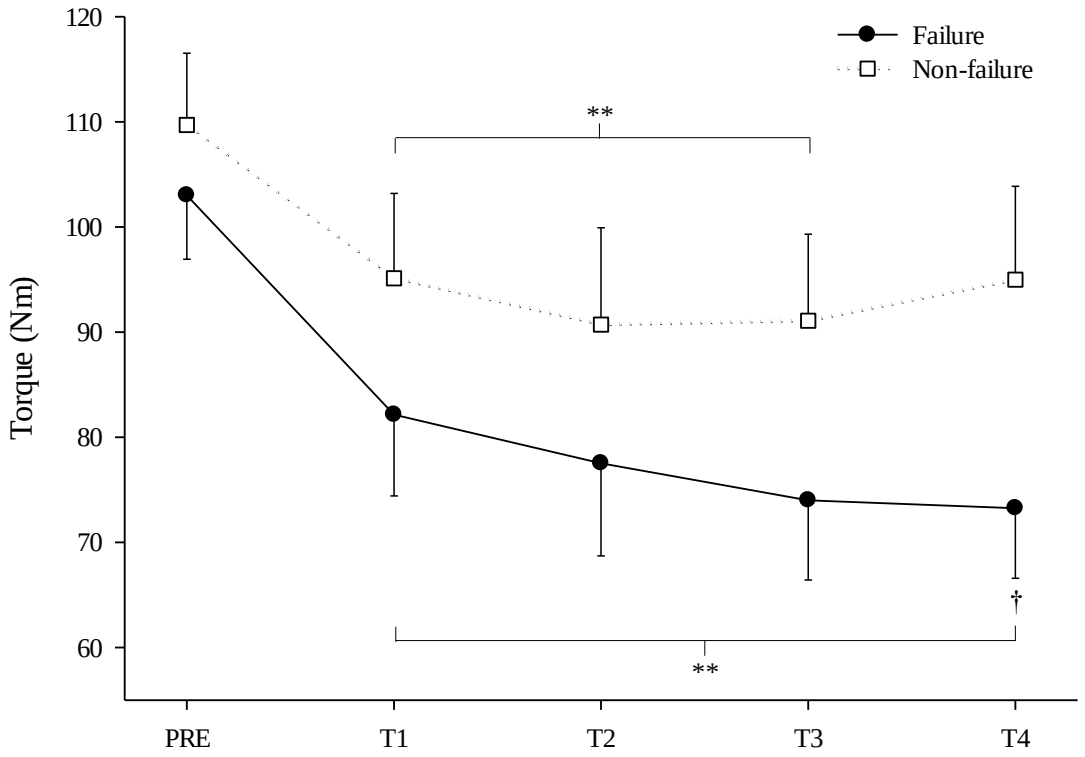


**Figure 12.** Resting twitch peak torque (PT, Nm) measured immediately following maximal voluntary knee extension contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. † $p < 0.05$ from non-failure condition, ** $p < 0.01$ from PRE. Data are mean and SE.

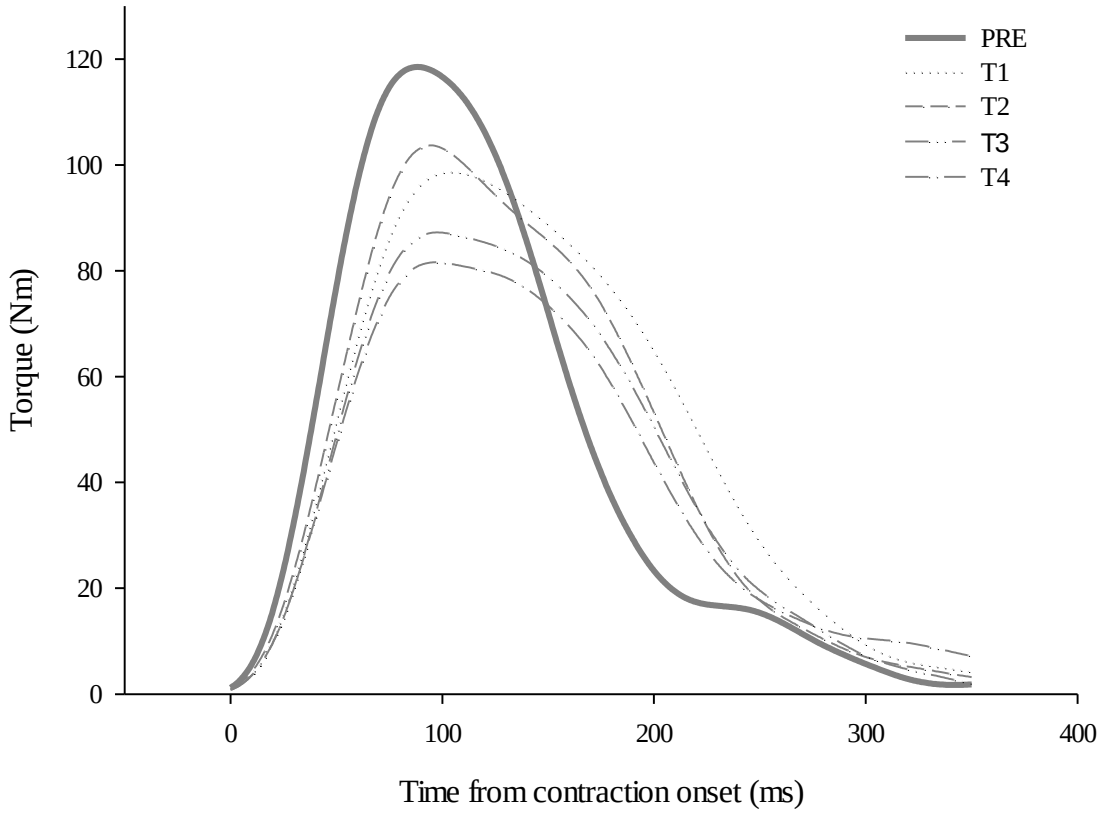


**Figure 13.** Resting potentiated twitches observed before (PRE) and during (T1, T2, T3 and T4) the failure protocol of a representative participant. The figure illustrates the increase in peripheral fatigue experienced with failure based exercise of the knee extensors.

Main time effects ($p < 0.001$) were observed for normalised $tRTD_{MAX}$. Post hoc analysis indicated $tRTD_{MAX}$ decreased from PRE at T1 by 7.6 ± 6.9 % ($p = 0.005$, Figure 14). $tRTD_{MAX}$ remained decreased between 7.1 ± 6.7 % and 5.2 ± 5.4 % from PRE at T2 and T3, respectively ($p < 0.05$). Main time effects ($p < 0.05$) were observed for normalised $tRTD_{AVE}$. Post hoc analysis indicated $tRTD_{AVE}$ in time intervals 0-50 ms and 0-75 ms post twitch torque onset decreased from PRE at T1 by 8.5 ± 9.6 % ($p = 0.043$) and 7.1 ± 7.1 % ($p = 0.016$), respectively (Table 6). $tRTD_{AVE}$ 0-75 ms post twitch torque onset remained decreased between 7.9 ± 7.1 % and 6.4 ± 6.4 % from PRE at T2 and T3 ($p < 0.05$), respectively. No change was observed for $tRTD_{AVE}$ 0-25 ms post twitch torque onset ($p \geq 0.132$).

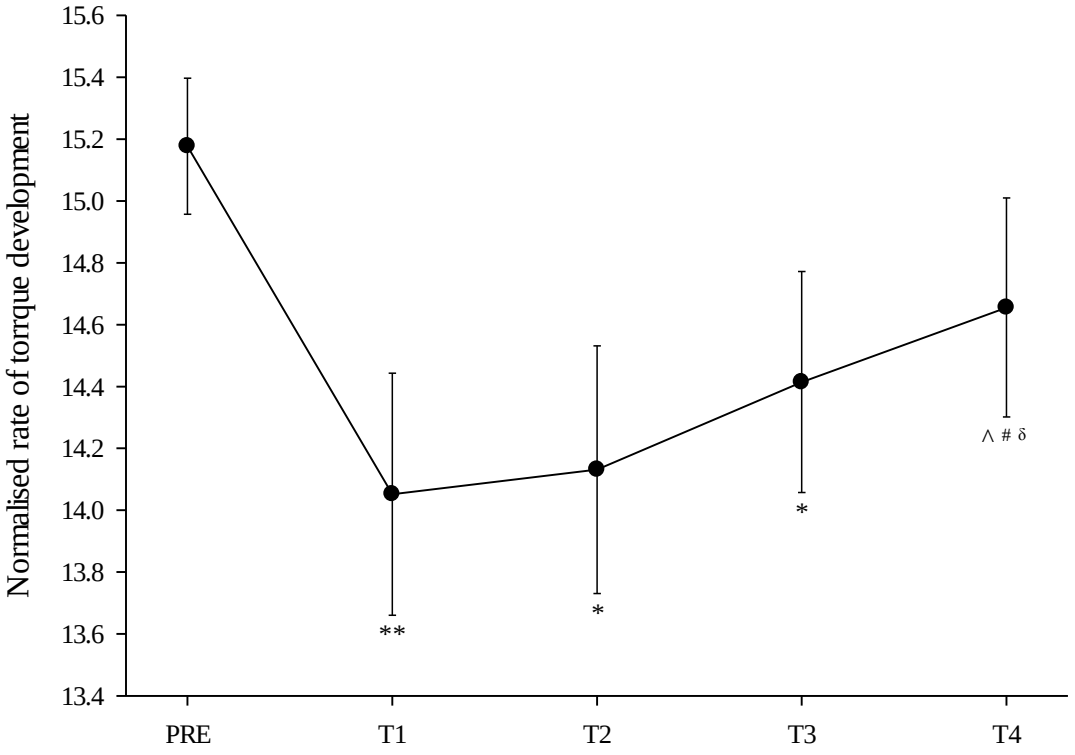


**Figure 14.** Resting twitch maximal rate of torque development ($tRTD_{MAX}$) normalised to the peak torque (PT) of the corresponding resting twitch, measured immediately following maximal voluntary knee extension contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p < 0.01$ from PRE, * $p < 0.05$ from PRE, ^ $p < 0.05$ from T1, # $p < 0.05$ from T2, δ $p < 0.05$ from T3. Data are grand mean and SE of failure and non-failure conditions.

**Table 6.** Normalised resting twitch maximal and average rate of torque development ($tRTD_{MAX}$, $tRTD_{AVE}$) measured immediately following maximal voluntary knee extension contractions completed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions. $tRTD_{AVE}$ data are presented as the average slope of the torque-time curve in time intervals of 0-25 ms, 0-50 ms and 0-75 ms post resting twitch torque onset ($tRTD_{AVE}$ (0-25), $tRTD_{AVE}$ (0-50), $tRTD_{AVE}$ (0-75)). All data are normalised to the peak torque (PT) of the corresponding resting twitch.

| | $tRTD_{MAX}$ | | $tRTD_{AVE}$ (0-25) | | $tRTD_{AVE}$ (0-50) | | $tRTD_{AVE}$ (0-75) | |
|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF |
| PRE | 15.1 ± 0.6 | 15.2 ± 1.1 | 7.3 ± 1.0 | 7.8 ± 1.5 | 10.6 ± 1.0 | 11.0 ± 1.5 | 11.4 ± 0.5 | 11.5 ± 0.8 |
| T1 | 13.5 ± 1.5$^{**}$ | 14.6 ± 1.5$^{**}$ | 6.3 ± 0.9 | 7.5 ± 1.4 | 9.2 ± 1.3$^{*}$ | 10.5 ± 1.6$^{*}$ | 10.2 ± 1.1$^{*}$ | 11.1 ± 1.0$^{*}$ |
| T2 | 13.7 ± 1.7$^{*}$ | 14.5 ± 1.5$^{*}$ | 6.4 ± 0.6 | 6.7 ± 2.3 | 9.4 ± 1.1 | 9.8 ± 2.2 | 10.4 ± 1.1$^{*}$ | 10.7 ± 1.3$^{*}$ |
| T3 | 14.0 ± 1.4$^{*}$ | 14.8 ± 1.5$^{*}$ | 6.5 ± 0.8 | 6.8 ± 2.2 | 9.5 ± 1.1 | 10.0 ± 2.2 | 10.6 ± 0.9$^{*}$ | 10.9 ± 1.2$^{*}$ |
| T4 | 14.2 ± 1.3$^{\wedge\#\delta}$ | 15.1 ± 1.5$^{\wedge\#\delta}$ | 6.6 ± 0.8 | 7.4 ± 1.5 | 9.7 ± 1.2 | 10.7 ± 1.7 | 10.7 ± 0.9$^{\#}$ | 11.3 ± 1.0$^{\#}$ |

Data are presented as mean ± SD
$^{**}$ $p < 0.01$ from PRE
$^{*}$ $p < 0.05$ from PRE
$^{\wedge}$ $p < 0.05$ from T1
$^{\#}$ $p < 0.05$ from T2
$^{\delta}$ $p < 0.05$ from T3

A main time effect ($p$ < 0.001) was observed for TPT. Post hoc analysis indicated TPT increased from PRE at T1 by 14.1 ± 10.3 % ($p$ = 0.001; Figure 15; Table 7). TPT remained increased between 6.3 % to 13.2 % ($p$ < 0.05) from PRE at all subsequent time points.

A main time effect ($p$ = 0.016) was observed for $VM_{Mmax}$. Post hoc analysis indicated $VM_{Mmax}$ was reduced from T1 and T2 at T4 ($p$ < 0.05; Table 7). No changes were observed for $VL_{Mmax}$ ($p$ = 0.239).

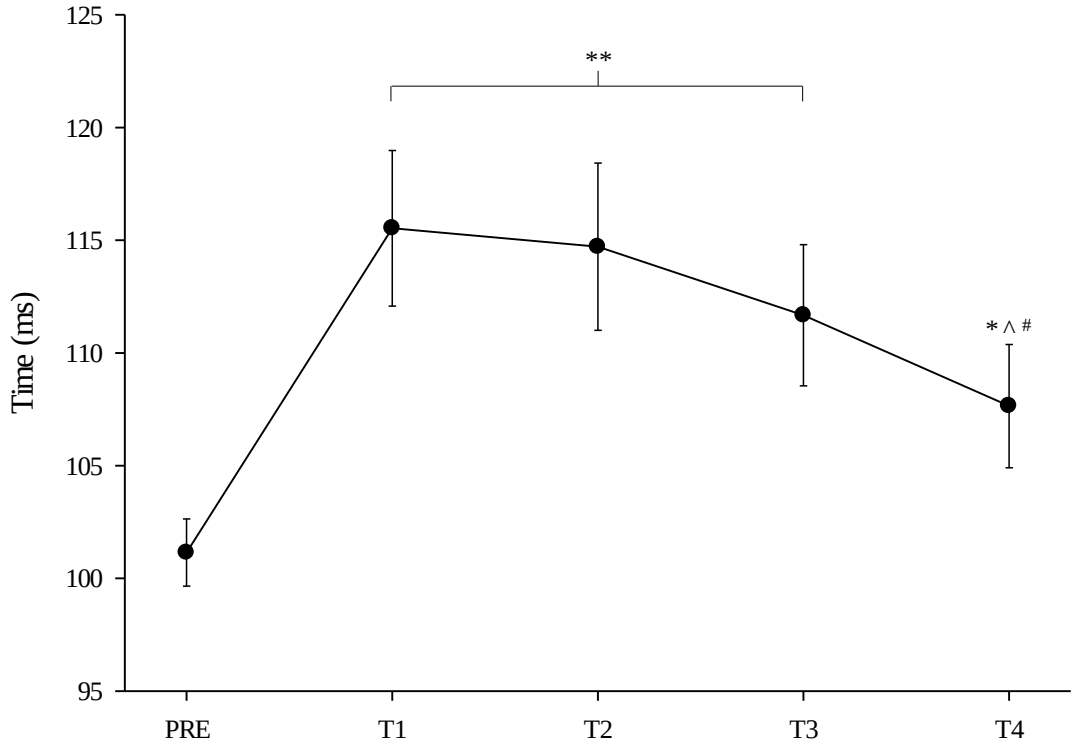


**Figure 15.** Resting twitch time to peak torque (TPT, ms) measured immediately following maximal voluntary knee extension contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p$ < 0.01 from PRE, * $p$ < 0.05 from PRE, ^ $p$ < 0.05 from T1, # $p$ < 0.05 from T2. Data are grand mean and SE of failure and non-failure conditions.

**Table 7.** Resting twitch time to peak torque (TPT, ms); and vastus lateralis and vastus medialis maximum M-wave ($VL_{Mmax}$, $VM_{Mmax}$; mV) evoked during maximal voluntary knee extension contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions.

| | TPT | | $VL_{Mmax}$ | | $VM_{Mmax}$ | |
|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF |
| PRE | 101.4 ± 4.7 | 100.9 ± 7.4 | 8.5 ± 5.3 | 9.1 ± 5.2 | 11.5 ± 5.5 | 9.3 ± 5.8 |
| T1 | 120.4 ± 15.4$^{**}$ | 110.6 ± 11.0$^{**}$ | 7.1 ± 4.4 | 8.7 ± 5.1 | 9.4 ± 5.9 | 7.4 ± 5.0 |
| T2 | 117.8 ± 16.8$^{**}$ | 111.6 ± 13.2$^{**}$ | 7.1 ± 4.9 | 8.9 ± 6.3 | 9.9 ± 6.2 | 7.6 ± 5.2 |
| T3 | 115.3 ± 12.8$^{**}$ | 108.1 ± 12.1$^{**}$ | 6.8 ± 4.3 | 8.9 ± 6.1 | 10.3 ± 5.9 | 8.0 ± 4.6 |
| T4 | 111.1 ± 10.8$^{*\wedge\#}$ | 104.1 ± 10.7$^{*\wedge\#}$ | 7.1 ± 4.8 | 9.0 ± 5.9 | 10.9 ± 5.8$^{\wedge\#}$ | 8.3 ± 5.4$^{\wedge\#}$ |

Data are presented as mean ± SD
$^{**}$ $p < 0.01$ from PRE
$^{*}$ $p < 0.05$ from PRE
$^{\wedge}$ $p < 0.05$ from T1
$^{\#}$ $p < 0.05$ from T2

A main time effect ($p < 0.001$) and time by condition interaction ($p = 0.024$) was observed for ½ RT. Post hoc analysis indicated that the 61.6 ± 17.1 % increase in ½ RT from PRE to T1 observed in the failure condition was greater than the non-failure condition ($p = 0.009$; Figure 16).

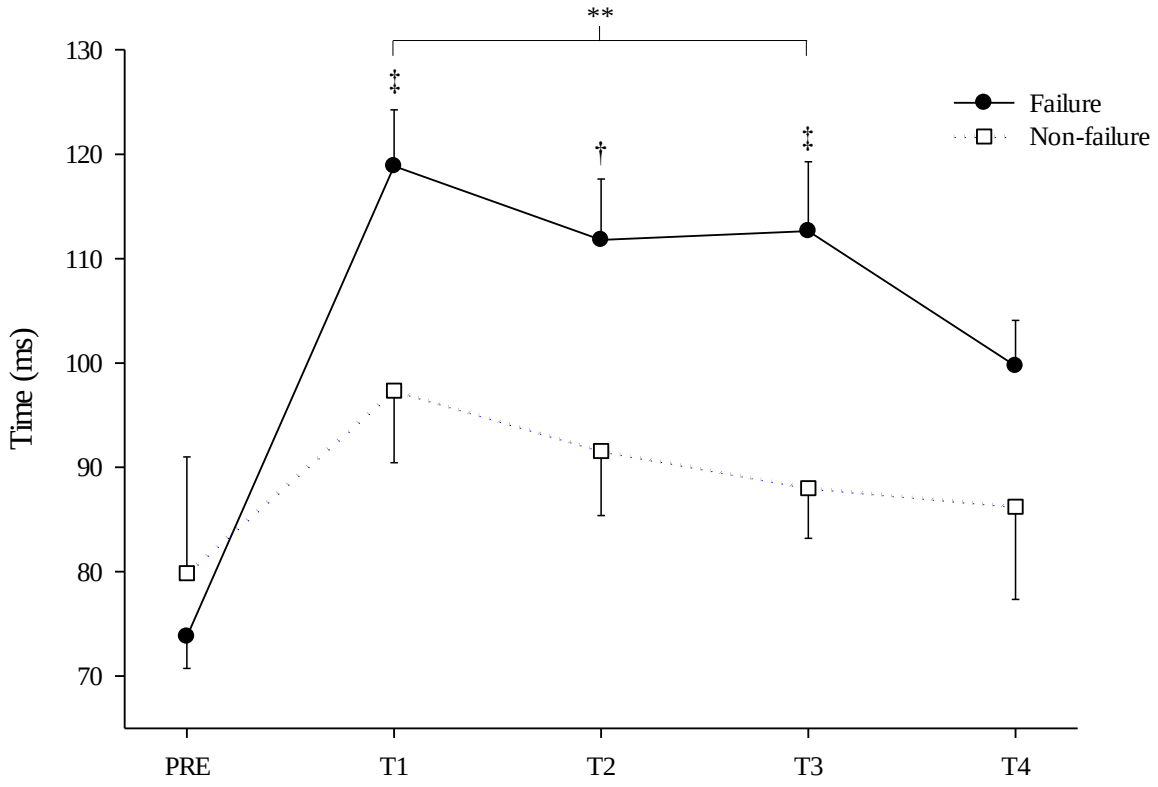


**Figure 16.** Resting twitch half-relaxation time (½ RT, ms) measured immediately following maximal voluntary knee extension contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ‡ $p < 0.01$ from non-failure condition, † $p < 0.05$ from non-failure condition, ** $p < 0.01$ from PRE. Data are mean and SE.

## DISCUSSION

The main finding of this investigation was that greater reductions in knee extensor maximal torque output ($T_{MAX}$) were observed when moderate to high intensity (80 % MVC) isometric knee extension contractions were performed to failure. Furthermore, peripheral mechanisms indicated by a decline in resting twitch peak torque (PT) and an increase in half relaxation time (½ RT), likely facilitated the larger reduction in torque output following muscular contractions performed to failure. Contrary to the hypothesised outcomes, measures of neural drive and muscle activation were unaffected following exercise, demonstrating that central factors did not mediate the observed declines in maximal strength in either condition.

The greater reduction in knee extensor $T_{MAX}$ in the failure condition supports the hypothesis and is in agreement with previous data. In untrained individuals, Rooney and colleagues reported a comparable, greater decline in maximal force output as a result of performing a short series of dynamic high intensity elbow flexion contractions to failure, compared to a similar non-failure exercise bout (Rooney *et al.*, 1994). In contrast to the present results, recent investigations in trained populations have observed similar reductions in maximal force output following moderate to high intensity failure and non-failure isotonic squat (Marshall *et al.*, 2012) and elbow flexion (Benson *et al.*, 2006) exercise tasks. The approximate 8 % (Marshall *et al.*, 2012) and 19 % (Benson *et al.*, 2006) pooled decline in maximal isometric force output in response to isotonic failure and non-failure exercise reported in these studies was also not as large as the 26 % reduction in maximal torque output observed at the conclusion of a bout of failure exercise in the present investigation. Given the distribution of type I and type II muscle fibres is comparable between the knee extensors and elbow flexors (Johnson *et al.*, 1973), it may be speculated that changes in maximal torque

output would be similar between investigations. Therefore, the present findings may suggest that moderate to high intensity isometric contractions promote a greater reduction in maximal torque output than similar isotonic exercise and/or that testing maximal force isometrically is not the most appropriate method of assessing fatigue incurred isotonically. Additionally, an increase in central fatigue, observed as a reduction in maximal muscle activation was thought to be responsible for the decline in maximal force output reported previously following isotonic failure and non-failure exercise (Benson *et al.*, 2006; Marshall *et al.*, 2012). Conversely, trained individuals maintained central neural drive (measured using voluntary activation (VA) and maximal muscle activity) at the conclusion of exercise in this study. Reductions in maximal knee extensor torque output following isometric failure and non-failure exercise were instead observed to be the product of impaired peripheral functioning.

Peripheral fatigue, indicated by reductions in PT, was significantly increased during moderate to high intensity isometric failure and non-failure exercise in this study. The 29.5 % reduction in PT observed in the failure condition is in agreement with Neyroud and colleagues who reported a similar 48 % decrease in PT following two, low intensity (20 % MVC) isometric knee extension contractions to failure in untrained individuals (Neyroud *et al.*, 2012). However, the present data contrast previous literature in which PT was well preserved when exercise was performed to failure (Garland *et al.*, 2003; Walker *et al.*, 2009). Following a high intensity (85 % 1RM) isotonic knee extension loading protocol (4 sets × 3 repetitions), Walker and colleagues did not observe a change in PT from baseline values in trained individuals (Walker *et al.*, 2009). Extending the trend relating to the level of central fatigue following failure and non-failure exercise (mentioned above), the current changes in PT with fatiguing exercise in trained populations may also reflect task specific (i.e. isometric versus

isotonic exercise) modulation of peripheral fatigue that is not *per se*, necessarily dependent on whether exercise is performed to failure.

The 29.5 % decline in PT in the failure condition was significantly greater than the 14.2 % decline observed in the non-failure condition at the completion of exercise. The greater reduction in PT after failure exercise likely occurred as a result of the greater volume of work accumulated in each individual contraction, relative to each non-failure contraction. However, this effect only became evident after enough volume had been accrued over the course of the exercise bout, given comparable reductions in PT were observed between conditions after the first three failure and six non-failure contractions. Peripheral fatigue has previously increased as a function of the volume of exercise when muscular contractions have been performed to failure in trained individuals. Behm and colleagues documented an approximate 32 % greater decrease in PT following a single set of elbow flexion contractions using a 20 RM load versus a 5 RM and 10 RM loading protocol (Behm *et al.*, 2002). Although direct observation of the mechanisms responsible for the reduction in PT was beyond the scope of the present investigation, the 61.6 % prolongation of resting twitch ½ RT in the failure condition extends prior understanding of the peripheral factors responsible for the reduction in PT experienced following exercise performed to failure. Increased relaxation time is thought to result from impaired $Ca^{2+}$ uptake/removal from the myoplasm and/or $Ca^{2+}$ dissociation from troponin causing cross bridge detachment (Westerblad *et al.*, 1997). The present findings suggest that impairment in one or both of these mechanisms likely contributed to the greater reduction in PT and thus, the increase in peripheral fatigue observed in the failure condition which was subsequently responsible for the larger reduction in maximal torque output.

The hypothesis that proposed a bout of failure based resistance exercise would stimulate greater reductions in maximal strength than a similar bout of non-failure exercise because of a larger increase in central fatigue was not supported here. Given maximal VA and muscle activity were maintained with exercise, the observed reduction in torque output was not the product of impaired central drive. Therefore, the present data may suggest that isometric contractions are not an ideal method of inducing central fatigue, the task itself was not of satisfactory duration or intensity to promote central impairment, and/or demonstrate that the mechanisms that mediate central drive in trained individuals have adapted to facilitate voluntary force production in the face of significant peripheral impairment.

The observed impairment in excitation-contraction coupling, despite the preservation of central drive in this study contrasts reductions in both PT and VA observed with fatiguing isometric knee extension exercise in untrained populations (Neyroud *et al.*, 2012), although supports data in trained individuals (Behm & St-Pierre, 1998; Marshall *et al.*, 2015). Marshall and colleagues reported that central neural drive (measured using VA) was maintained following low (40 % MVC) and high intensity (80 % MVC) isometric contractions of the knee extensors despite a worsening of peripheral fatigue, indicated by a 30 % to 70 % reduction in PT (Marshall *et al.*, 2015). Additionally, the authors attributed the increase in peripheral fatigue to a prolongation of resting twitch rate and temporal characteristics (Marshall *et al.*, 2015), similar to those observed in the present investigation. Therefore, the developing trend demonstrates that trained individuals are capable of maintaining central drive to the knee extensors during submaximal isometric contraction to cope with the increase in peripheral fatigue. Further research should therefore look to examine mechanisms responsible for peripheral impairment such as blood lactate accumulation, which has also been associated with acute reductions in strength with failure and non-failure exercise

(Linnamo *et al.*, 1998; Benson *et al.*, 2006). An increase in blood lactate concentration is understood to impair $Ca^{2+}$ kinetics and thus, cross-bridge binding within the muscle contractile apparatus (Metzger & Moss, 1990) which may explain the reduction in resting twitch PT and torque-time parameters observed in this investigation. Furthermore, a concurrent observation of blood lactate and myofibrillar $Ca^{2+}$ concentrations would help to provide understanding of whether the specific peripheral mechanisms that modulate strength production in trained individuals are the product of $Ca^{2+}$ release (Ortenblad *et al.*, 2000), reuptake (Lamboley *et al.*, 2014) or the rate of binding to the contractile proteins (Westerblad *et al.*, 1997).

An interesting finding from this study was that vastus lateralis (VL) and vastus medialis (VM) maximal M-wave ($M_{max}$) amplitudes were not uniformly affected by exercise. $VM_{Mmax}$ decreased up to 17.6 % in both failure and non-failure conditions despite $VL_{Mmax}$ remaining unaffected, indicating that action potential depolarisation at the neuromuscular junction and/or transmission along the sarcolemma was impeded in VM although not in VL. Compared to VL, the VM muscle is composed of a larger percentage of type I motor units and their associated fibres (Johnson *et al.*, 1973). VM would therefore be expected to have superior fatigue resilience (Colliander *et al.*, 1988) owing to innate membrane characteristics such as the generation of smaller action potentials and slower depolarisation and conduction velocities (Buchthal *et al.*, 1973; Milner-Brown & Miller, 1986). Thus, the greater reduction in $VM_{Mmax}$ may in part be explained by a block in signal transmission from altered $Na^+/K^+$ gradients, rather than muscle fibre type. However, such conclusions are beyond the scope of this study and offer potential directions for future investigation. Because the M-wave did not decrease in both muscles and minimal impairments were seen in VM, membrane excitability was unlikely to be the primary determinant of voluntary force production in this study. Given

the prolongation in resting twitch temporal characteristics, the present results suggest the likely cause for the greater reduction in maximal torque output observed in the failure condition was the result of processes such as $Ca^{2+}$ handling occurring in the later phases of the excitation-contraction coupling process.

To conclude, declines in maximal torque generating capacity were more pronounced in trained individuals when moderate to high intensity isometric knee extension contractions were performed to failure, compared to a similar series of non-failure contractions. The present findings are the first to demonstrate that central drive remains well preserved following failure and non-failure exercise despite a worsening of peripheral fatigue. The data also demonstrate that reductions in maximal torque output observed in both conditions manifest from impaired intrinsic contractile functioning. Furthermore, the greater reduction in maximal torque output in the failure condition likely resulted from impaired $Ca^{2+}$ kinetics and maximal cross-bridge binding, observed as a prolongation in ½ RT and a decline in PT, respectively.

# CHAPTER 4

*Acute Failure and Non-failure Exercise of the Plantar Flexors*

## METHODS

### Subjects

Eight healthy resistance trained males ($n$ = 8) volunteered to participate in the study (age, 23.3 ± 2.3 years; height, 179.0 ± 7.2 cm; body mass, 89.1 ± 10.7 kg; training experience, 3.4 ± 1.1 years; calf raise one repetition maximum (RM), 64.1 ± 10.4 kg; mean ± SD). All participants were required to have regularly (at least 3 days per week) performed resistance exercise of the upper and lower body for the previous 24 months and be able to complete a dynamic 1 RM seated calf raise contraction ≥ 60 % body mass. Participants were excluded if they reported taking performance enhancing substances as per the World Anti-Doping Agency's 2012 prohibited list, had a recent history of upper or lower limb injury that may limit performance of the exercise task, or any known metabolic or neuromuscular disease. Participants were instructed to refrain from any resistance or anaerobic lower limb exercise and maintain normal dietary habits for 48 hours preceding the testing sessions. Each participant gave written informed consent prior to testing. All procedures were approved by Western Sydney University's Human Research Ethics Committee (approval number H9859) and were carried out in accordance with the Declaration of Helsinki.

### Experimental design

Participants were required to attend the laboratory for one familiarisation session followed by two experimental sessions separated by a minimum of 72 hours. The initial familiarisation served to determine participant entry into the study based on 1 RM testing and accustom participants to procedures used to assess plantar flexor voluntary isometric torque (isokinetic

dynamometer, KinCom 125, Version 5.32, Chattanooga, USA), and central and peripheral fatigue using the interpolated twitch technique (ITT). Participants completed the failure and non-failure exercise conditions on two separate days, firstly performing the failure protocol. The study was purposefully designed in this way as it enabled total session exercise volume and duration to be equated between conditions.

Experimental procedures

*Range of motion assessment*

An electro-goniometer (MLTS700, ADInstruments, Australia) was used to determine the range of motion required for successful completion of 1 RM and all repetitions during both exercise protocols. The centre of the goniometer was aligned with the right lateral malleolus and secured to the lateral aspect of the right shank and fifth metatarsal. Weight equalling an approximate 1 RM load was added to the seated calf raise machine (adjustable seated calf raise machine, ForceUSA) and participants performed a controlled contraction to full eccentric range of motion (ROM) until the raw signal (degrees, º) plateaued for 2-3 sec. The weight was removed and participants completed an unloaded contraction to full concentric ROM until the raw signal plateaued for 2-3 sec. The difference between concentric and eccentric ROM was used as a measure of joint ROM.

*1 RM testing*

Participants were required to complete a bilateral seated calf raise 1 RM ≥ 60 % body mass during familiarisation. Pilot testing indicated a 1 RM seated calf raise contraction ≥ 60 %

body mass was reasonable to expect from a sample of resistance trained individuals. Prior to 1 RM testing, participants completed a short series of dynamic contractions with a load approximately equalling 50 % and 75 % of predicted 1 RM. Following this warm up, 1 RM was assessed. Participants were required to complete a single dynamic eccentric-concentric contraction with a 2:1 sec cadence and achieve 90 % of full joint ROM (determined from ROM assessment) for 1 RM attempts to be deemed successful. The highest weight (kg) attained prior to failing was recorded as the participants' 1 RM. Weight was increased in 2.5-5 kg increments until 1 RM was achieved, always within 3-5 attempts. To ensure adequate recovery, participants received 3 min rest between 1 RM attempts. Strong verbal encouragement was provided throughout all attempts.

*Maximal voluntary contractions and exercise testing*

All measurements were performed on the plantar flexors of the left leg. Participants sat upright in the dynamometer with the hip and knee flexed to 90º throughout all maximal voluntary contractions (MVCs). The centre of rotation of the dynamometer lever arm was aligned with the lateral malleolus. The lever arm of the dynamometer was aligned with and firmly secured around the metatarsophalangeal joint. Participants were securely fastened to the dynamometer with straps placed across the torso, hips and thighs. Participant alignment was recorded to maintain consistency between sessions. All torque signals were sampled at 2,000Hz (Powerlab 16/35, ADInstruments Australia; 16 bit analog to digital conversion) and filtered with a $4^{th}$ order 10 Hz digital low pass filter prior to analysis.

Prior to each exercise trial, participants performed a brief warm up consisting of a short series of submaximal isometric plantar flexion contractions at 50 % and 75% of perceived maximal

effort. Following this warm up, four MVCs were performed in a random order, separated by one minute rest, with supramaximal stimulation of the posterior tibial nerve occurring on two of four attempts. Prior to analysis, baseline values (PRE) were obtained from the average data of the four MVCs and two accompanying resting potentiated twitches. Fatigue was assessed with single MVCs and subsequent stimulations completed at four time points (T1, T2, T3 and T4) over the course of both exercise tasks. Participants were instructed to complete all MVCs as fast and as forcefully as possible and maintain contraction for 3-4 sec.

Both exercise protocols required participants to complete dynamic, bilateral plantar flexion contractions with a load (kg) corresponding to 80 % 1 RM. Individual repetitions were performed with a 2:1 sec eccentric to concentric contraction cadence. Before commencing each exercise protocol, the electro-goniometer was attached to the right leg and ROM assessed according to the above description. A short warm up performed with 50 % and 75 % of the 80 % 1 RM load preceded each condition. The failure protocol required participants to perform four sets of plantar flexion contractions to the point of volitional exhaustion, defined as an inability to reach 90 % ROM for two consecutive repetitions. MVCs were performed immediately upon failure of all four sets (T1, T2, T3 and T4). Participants received 70 sec rest from the completion of each MVC to the beginning of the next set (total 210 sec rest within the session). The non-failure protocol required participants to perform eight sets of plantar flexion contractions. The number of repetitions performed in each non-failure set was determined by halving the number of repetitions completed in each analogous failure set, therefore distributing an equal contraction volume over two sets instead of one. MVCs were performed immediately upon completion of the corresponding failure time point (i.e. sets two (T1), four (T2), six (T3) and eight (T4)). Participants received 30 sec rest from the end of each set or MVC to the beginning of the next set (total 210 sec rest within the

session). No participant failed whilst completing the non-failure testing session. If a participant was deemed to have failed on an odd numbered repetition in the failure protocol, the first of the two analogous non-failure sets contained a single repetition more than the second set (i.e. a nine repetition failure set corresponded to two non-failure sets, each with five and four repetitions, respectively). Goniometer signal output was continuously displayed throughout both exercise protocols on a 25" LCD monitor (LG™, Australia), with a guideline placed at 90 % of full ROM. Strong verbal encouragement was provided at all times. All MVCs were completed within 30 sec of the conclusion of exercise sets because participants needed to be relocated from the seated calf raise machine to the isokinetic dynamometer. This was not believed to have affected any results observed from this study.

*Electromyography*

Surface electromyograms (sEMG) were recorded from the left soleus (SOL) and medial gastrocnemius (MG) using pairs of Ag/AgCl surface electrodes (Maxsensor, Medimax Global, Australia). SOL and MG electrodes (10 mm diameter, 10 mm inter-electrode distance) were applied in bipolar configuration parallel to the direction of the muscle fibres after careful skin preparation (shaving of excess hair, abrasion with fine sandpaper and cleaning the skin with isopropyl alcohol swabs) to reduce skin electrical impedance below 5 kΩ. SOL electrodes were placed at ⅔ of the line between the medial condyle of the femur and the medial malleolus, with GM electrodes positioned on the most prominent bulge of the muscle. Placement sites were recorded for each participant with respect to anatomical landmarks to maintain consistency between sessions. The reference electrode was placed on the left medial malleolus. sEMG signals were recorded using the ML138 Octal BioAmp (common mode rejection ratio > 85 dB at 50 Hz, input impedance 200 MΩ) with a 16-bit

analog-to-digital conversion, sampled at 2,000 Hz (ADInstruments, Australia). Prior to analysis, raw signals were filtered with a fourth-order Bessel filter between 20 Hz and 500 Hz and smoothed using a root mean square (RMS) calculation with a 100 ms time constant. All sEMG RMS values were normalised to the raw sEMG M-wave evoked during each analogous MVC (sEMG/M, %) to control for potential changes in axonal excitability (Pasquet *et al.*, 2000).

*Posterior tibial nerve stimulation*

The posterior tibial nerve was stimulated using a doublet (two, 1 ms square wave pulses with a 10 ms inter-stimulus interval; 100 Hz stimulation) applied at 400 V using a constant current stimulator (Digitimer DS7AH, Welwyn Garden City, UK). To identify nerve location for cathodal stimulation, a rubber insulated portable cathodal probe was used to deliver low intensity stimulations (20 mA) to the popliteal fossa. The popliteal fossa was pre-marked with a permanent marker with optimal location determined at rest by moving the probe until the largest evoked peak-to-peak M-wave was elicited in SOL and MG. When optimal cathodal location was identified, a single Ag/AgCl surface electrode was applied (15 mm diameter; Kendall, Covidien, USA). The anode was specially made from aluminium foil (8.5 × 5.5 cm rectangle), covered in a layer of conductive gel (Ten20 Conductive Paste, Weaver and Company, USA) and secured 2 cm superior to the patella. The level of stimulation during testing was determined by gradually increasing the current intensity in 10 mA increments with 20 sec rest between stimuli until SOL and MG M-wave amplitudes and plantar flexor twitch torque plateaued. The maximal stimulus intensity was recorded (range, 100-180 mA) and multiplied by 130 % to establish the supra-maximal stimulation intensity to be applied throughout the exercise testing session.

*Voluntary activation*

The superimposed twitch technique (Merton, 1954) was used to measure plantar flexor voluntary activation (VA). A single, supra-maximal doublet stimulation was used to evoke a superimposed twitch when a visible plateau in the torque-time curve was observed for 1-2 sec during each MVC. In addition, a single, supra-maximal doublet stimulation was delivered 3-4 sec following the completion of each MVC when the participant was relaxed to evoke a resting potentiated twitch. Voluntary activation was estimated according to the following formula (Strojnik & Komi, 1998): VA (%) = 100 – [$D \times (T_{SUP}/T_{MAX})/PT] \times 100$, where *D* is the difference between the torque amplitude just before the superimposed twitch ($T_{SUP}$) and the peak torque amplitude recorded during the superimposed twitch, $T_{MAX}$ is the maximal torque amplitude recorded during the MVC, and PT is the peak torque amplitude of the resting potentiated twitch.

Data processing

For all MVCs and resting potentiated twitches, torque onset was defined as the point on the torque-time curve where torque output exceeded baseline values by ≥ 1 % of the difference between baseline and peak torque amplitude. The following variables were analysed from the torque-time curve of each MVC: *1)* maximal voluntary torque output, defined as the greatest amplitude of the torque-time curve, excluding the point of stimulation ($T_{MAX,}$ Nm); *2)* normalised maximal rate of torque development ($RTD_{MAX}$), determined from the greatest average 10 ms slope of the torque-time curve (Δtorque/Δtime) throughout the first 500 ms of each MVC; and *3)* normalised average rate of torque development ($RTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms, 0-200 ms post torque onset. All rate dependent

measures of voluntary torque production were normalised to $T_{MAX}$ of each analogous MVC to observe changes in RTD independent of changes to maximal torque (Holtermann *et al.*, 2007).

sEMG onset was defined 70 ms before torque onset to account for the presence of electromechanical delay (Aagaard *et al.*, 2002a). During each MVC the following variables were identified from SOL and MG sEMG signals: 1) maximal sEMG activity ($SOL_{MAX}$ and $MG_{MAX}$; sEMG/M, %), calculated from the greatest average 250 ms period of activity (excluding superimposed stimulation) of the RMS signal throughout each MVC; 2) maximal rate of sEMG rise ($SOL_{RERmax}$ and $MG_{RERmax}$; sEMG/M, $\%.s^{-1}$), determined as the greatest average 10 ms slope ($\Delta$sEMG/$\Delta$time) of the RMS signal up to 200 ms post sEMG onset; 3) average rate of sEMG rise ($SOL_{RERave}$ and $MG_{RERave}$; sEMG/M, $\%.s^{-1}$) of the RMS signal calculated in time intervals from 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset; 4) the maximal M-wave amplitude ($SOL_{Mmax}$ and $MG_{Mmax}$; mV), determined from the peak-to-peak amplitude of the raw sEMG signal post doublet stimulation. sEMG RER was observed up to a maximum of 75 ms post sEMG onset (instead of $>$ 100 ms) as a decrease in RMS amplitude often occurred after this time.

The following variables were analysed from the resting potentiated twitches: *1)* resting twitch peak torque (PT, Nm), defined as the greatest amplitude of the torque-time curve; *2)* normalised resting twitch maximal rate of torque development ($tRTD_{MAX}$), defined as the greatest average 10 ms slope of the ascending limb of the twitch torque-time curve; *3)* normalised resting twitch average rate of torque development ($tRTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms post twitch torque onset; *4)* resting twitch time to peak torque (TPT, ms), defined as the time from twitch torque onset to PT; and *5)* resting twitch

half relaxation time (½ RT, ms), defined as the time elapsed from PT to 50 % PT. $tRTD_{AVE}$ was observed up to a maximum of 75 ms post stimulation as reductions in twitch torque amplitude often occurred between 75 ms and 100 ms. All tRTD variables were normalised to the PT amplitude of each analogous resting potentiated twitch.

Reliability

Reliability analyses were completed using data from the four PRE MVC recordings. The mean within-day, within-subject coefficients of variation (%) were 4.2 ± 2.3 (range 1.5 to 8.4) for $T_{MAX}$, 4.5 ± 4.4 (range 0 to 14.5) for VA, and 2.5 ± 2.5 (range 0.1 to 9.4) for PT. Mean between-day, within-subject coefficients of variation were 11.0 ± 9.2 (range 1.3 to 25.3) for $T_{MAX}$, 3.9 ± 4.3 (range 0.1 to 12.4) for VA, and 8.3 ± 10.2 (range 0.3 to 31.9) for PT. Data are means ± SD. The mean within-day, within-subject intra-class correlation coefficient (ICC, *r*) was 0.99 (95 % CI 0.97 to 1, $p < 0.001$) for $T_{MAX}$, 0.66 (95 % CI 0.08 to 0.88, $p = 0.019$) for VA, and 0.98 (95 % CI 0.95 to 0.99, $p < 0.001$) for PT. The mean between-day, within-subject ICC was 0.63 (95 % CI -1.32 to 0.93, $p = 0.126$) for $T_{MAX}$, 0.65 (95 % CI -0.35 to 0.92, $p = 0.075$) for VA, and 0.74 (95 % CI -0.34 to 0.95, $p = 0.056$) for PT.

Statistical analysis

All statistical analyses were completed using IBM SPSS Statistics version 22 (SPSS Inc., Chicago, IL). All data were normally distributed, determined from Kolmogorov-Smirnov normality testing. Dependent variables were analysed using a two-way (condition × time) ANOVA for repeated measures (PRE, T1, T2, T3, T4) between conditions (failure, non-

failure). In the event of a significant condition effect, post hoc repeated measures were conducted over four levels of time (TI, T2, T3, T4) with PRE data as a covariate. If Mauchly's test indicated a violation of sphericity in the ANOVA, the Greenhouse-Geisser epsilon correction was used to adjust the degrees of freedom. Post hoc comparisons were made using a Bonferroni correction, in the presence of a significant $F$ ratio (considered significant at $p < 0.05$). The data are presented as means ± SD unless otherwise stated.

## RESULTS

Maximal torque and rate of torque development

A main time effect ($p < 0.001$) was observed for $T_{MAX}$. Post hoc analysis indicated $T_{MAX}$ decreased from PRE at T1 by 10.6 ± 7.1 % ($p = 0.002$; Figure 17; Table 8). $T_{MAX}$ remained decreased between 13.4 % to 15.0 % ($p < 0.001$) from PRE at all subsequent time points.

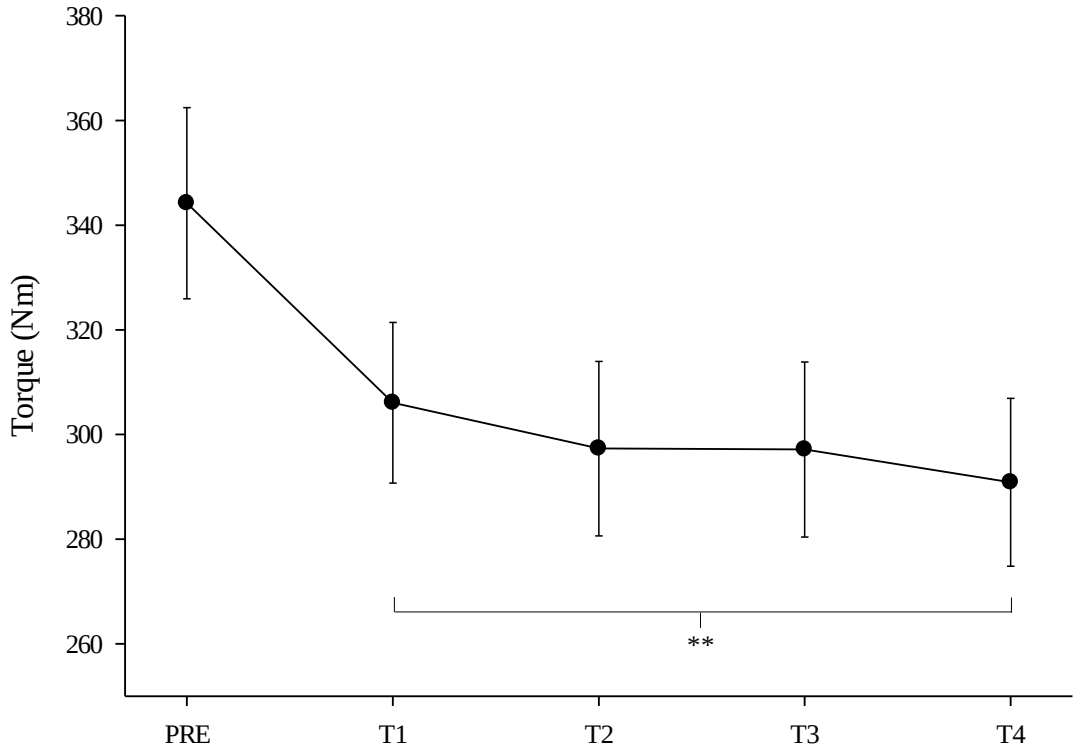


**Figure 17.** Maximal voluntary torque ($T_{MAX}$, Nm) measured during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p <$ 0.01 from PRE. Data are grand mean and SE of failure and non-failure conditions.

**Table 8.** Maximal voluntary torque ($T_{MAX}$, Nm); and normalised maximal and average rate of torque development ($RTD_{MAX}$, $RTD_{AVE}$) measured during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions. $RTD_{AVE}$ data are presented as the average slope of the torque-time curve in time intervals of 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms and 0-200 ms post torque onset ($RTD_{AVE}$ (0-25), $RTD_{AVE}$ (0-50), $RTD_{AVE}$ (0-75), $RTD_{AVE}$ (0-100) and $RTD_{AVE}$ (0-200)). All rate dependent measures of torque development are normalised to the corresponding MVCs' $T_{MAX}$.

| | $T_{MAX}$ | | $RTD_{MAX}$ | | $RTD_{AVE}$ (0-25) | | $RTD_{AVE}$ (0-50) | |
|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF |
| PRE | 338.7 ± 50.8 | 349.7 ± 93.7 | 4.2 ± 1.1 | 4.2 ± 1.0 | 1.4 ± 0.6 | 1.4 ± 0.6 | 2.0 ± 0.9 | 2.0 ± 0.9 |
| T1 | 299.6 ± 32.3** | 312.5 ± 83.3** | 4.9 ± 1.4* | 4.8 ± 1.3* | 1.5 ± 0.7 | 1.8 ± 0.7 | 2.2 ± 1.1 | 2.6 ± 1.0 |
| T2 | 291.8 ± 39.1** | 302.8 ± 89.0** | 4.7 ± 1.3** | 4.9 ± 1.0** | 0.9 ± 0.8 | 1.5 ± 0.7 | 1.4 ± 1.1 | 2.2 ± 1.1 |
| T3 | 286.9 ± 34.4** | 307.3 ± 90.3** | 4.7 ± 1.8 | 4.8 ± 1.4 | 1.4 ± 0.9 | 1.1 ± 0.6 | 2.1 ± 1.3 | 1.5 ± 0.9 |
| T4 | 280.6 ± 26.3** | 301.1 ± 88.7** | 4.6 ± 1.2* | 4.6 ± 1.1* | 1.3 ± 0.9 | 1.3 ± 0.8 | 1.9 ± 1.3 | 1.8 ± 1.2 |

| | $RTD_{AVE}$ (0-75) | | $RTD_{AVE}$ (0-100) | | $RTD_{AVE}$ (0-200) | |
|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF |
| PRE | 2.5 ± 1.1 | 2.6 ± 1.0 | 2.8 ± 1.1 | 2.9 ± 1.0 | 2.9 ± 0.6 | 3.0 ± 0.6 |
| T1 | 2.8 ± 1.3 | 3.2 ± 1.1 | 3.2 ± 1.3 | 3.6 ± 1.1 | 3.2 ± 0.6 | 3.3 ± 0.8 |
| T2 | 1.9 ± 1.3 | 2.8 ± 1.4 | 2.4 ± 1.4 | 3.1 ± 1.4 | 2.9 ± 0.8 | 3.2 ± 0.7 |
| T3 | 2.7 ± 1.7 | 2.0 ± 1.2 | 3.0 ± 1.8 | 2.3 ± 1.4 | 2.9 ± 1.3 | 3.0 ± 0.8 |
| T4 | 2.4 ± 1.6 | 2.3 ± 1.4 | 2.8 ± 1.6 | 2.6 ± 1.5 | 2.9 ± 0.9 | 3.0 ± 1.0 |

Data are presented as mean ± SD
** $p < 0.01$ from PRE
* $p < 0.05$ from PRE

A main time effect ($p$ = 0.023) was observed for normalised $RTD_{MAX}$. Post hoc analysis indicated $RTD_{MAX}$ increased from PRE at T1 by 15.4 ± 16.8 % ($p$ = 0.017; Figure 18; Table 8). No changes were observed for normalised $RTD_{AVE}$ during time intervals of 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms and 0-200 ms post torque onset ($p \geq 0.100$; Table 8).

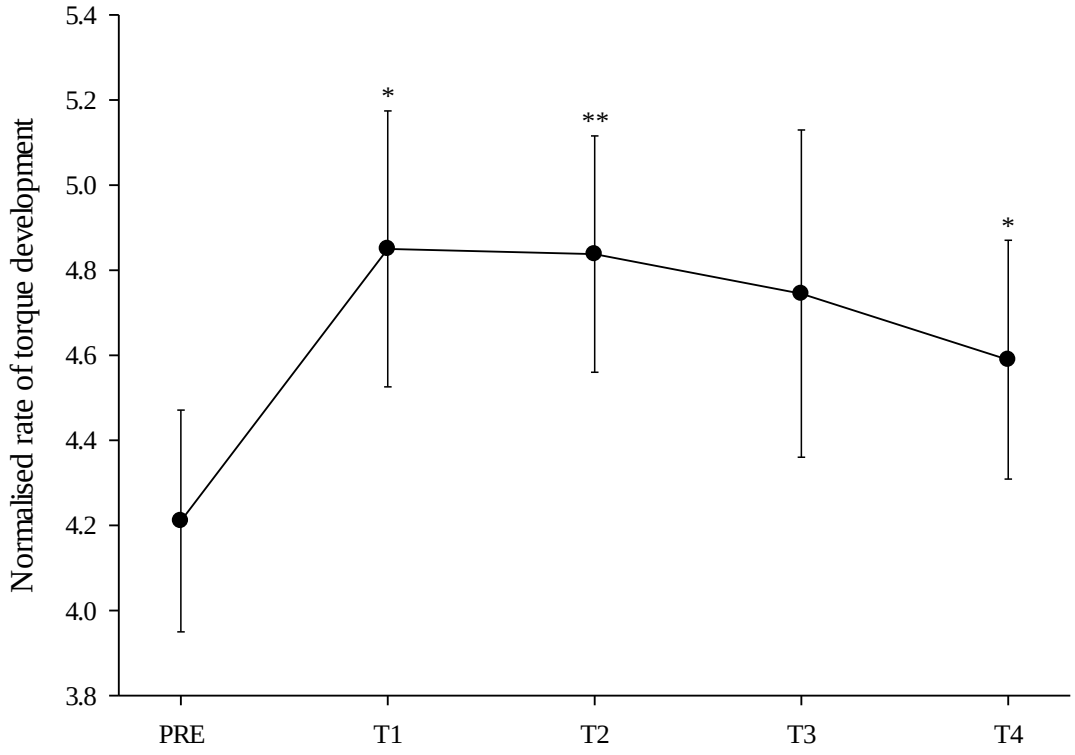


**Figure 18.** Maximal rate of torque development ($RTD_{MAX}$) normalised to the corresponding MVCs' $T_{MAX}$, measured during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p < 0.01$ from PRE, * $p < 0.05$ from PRE. Data are grand mean and SE of failure and non-failure conditions.

Central fatigue

A main time effect ($p < 0.001$) was observed for VA. Post hoc analysis indicated VA decreased from PRE at T1 by 7.0 ± 7.7 % ($p = 0.024$; Figure 19; Figure 20; Table 9). VA remained decreased between 7.7 % to 9.5 % ($p < 0.05$) from PRE at all subsequent time points.

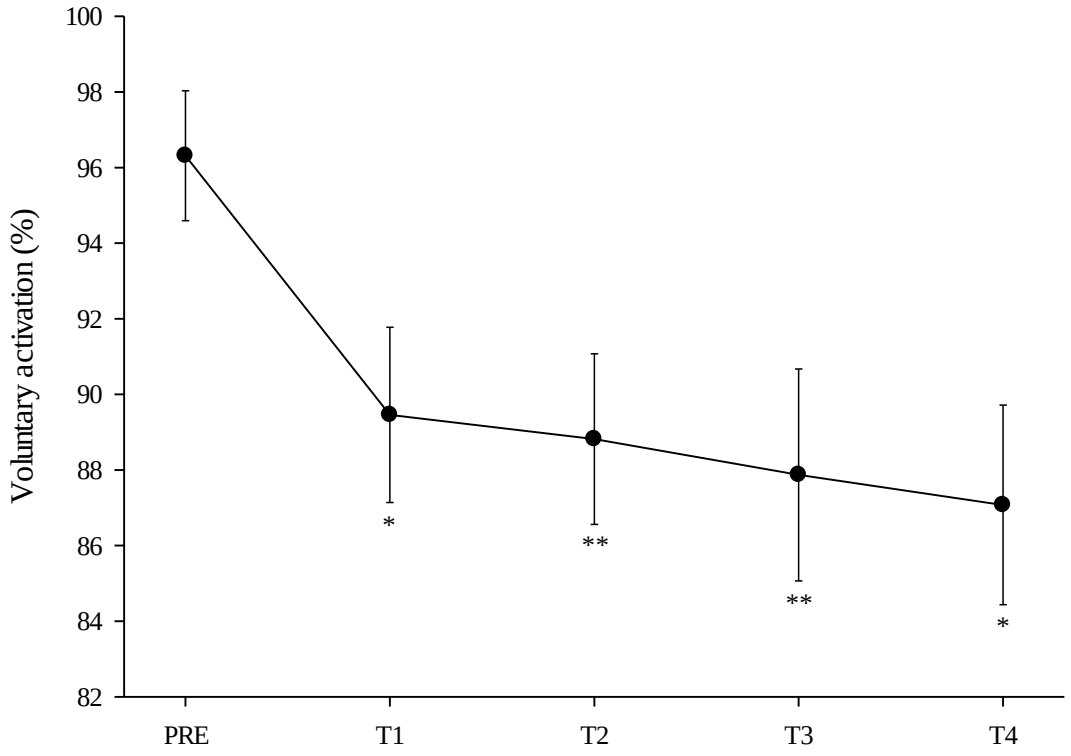


**Figure 19.** Voluntary activation (VA, %) measured during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p < 0.01$ from PRE, * $p < 0.05$ from PRE. Data are grand mean and SE of failure and non-failure conditions.

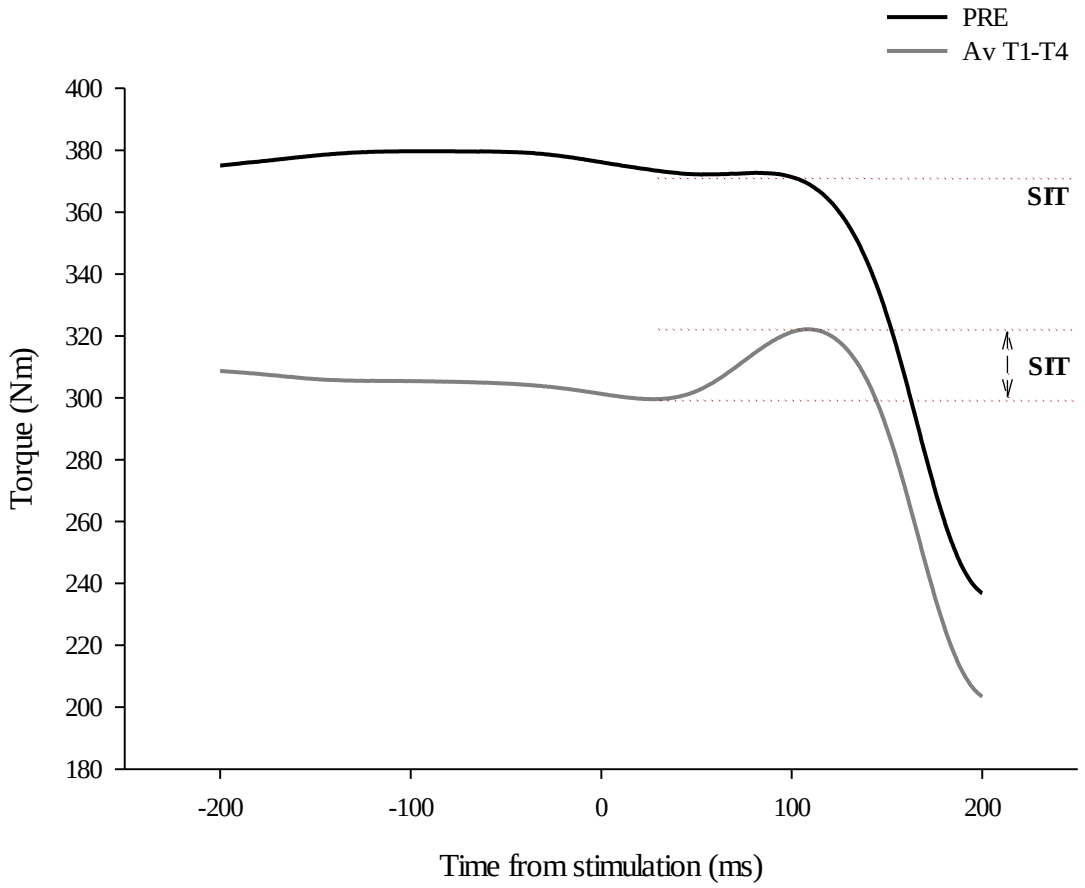


**Figure 20.** A representative maximal voluntary contraction with superimposed twitch (SIT) from a single subject's pooled conditional data, demonstrating the difference in SIT amplitude observed at baseline (PRE) to the average at all other time points (Av T1-T4). The figure illustrates the increase in central fatigue experienced with failure and non-failure based exercise of the plantar flexors.

**Table 9.** Voluntary activation (VA, %) measured during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions.

| | VA | |
|---|---|---|
| MVC | F | NF |
| PRE | 98.1 ± 3.3 | 94.5 ± 9.1 |
| T1 | 93.0 ± 7.4$^{*}$ | 86.0 ± 10.1$^{*}$ |
| T2 | 91.6 ± 8.0$^{**}$ | 86.0 ± 9.6$^{**}$ |
| T3 | 90.6 ± 10.8$^{**}$ | 85.1 ± 11.7$^{**}$ |
| T4 | 88.1 ± 10.1$^{*}$ | 86.0 ± 11.6$^{*}$ |

Data are presented as mean ± SD
$^{**}$ $p < 0.01$ from PRE
$^{*}$ $p < 0.05$ from PRE

Main time effects ($p < 0.01$) were observed for $SOL_{RERave}$ during 0-25 ms, 0-50 ms and 0-75 ms time intervals post sEMG onset. Post hoc analysis indicated $SOL_{RERave}$ decreased from PRE at T4 during time intervals 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset by 49.2 ± 32.0 % ($p = 0.028$), 39.1 ± 33.2 % ($p = 0.028$) and 34.5 ± 30.7 % ($p = 0.042$) respectively (Table 10). No changes were observed for $SOL_{MAX}$ ($p \geq 0.248$; Figure 21; Table 10) or $SOL_{RERmax}$ ($p \geq 0.593$; Table 10).

A main time effect ($p = 0.004$) was observed for $MG_{MAX}$. Post hoc analysis indicated $MG_{MAX}$ decreased from PRE at T2 and T3 by 25.1 ± 25.6 % ($p = 0.044$) and 24.3 ± 19.3 % ($p = 0.043$) respectively (Figure 21, Table 10). Main time effects ($p < 0.01$) were observed for $MG_{RERave}$ during time intervals 0-25 ms and 0-75 ms post sEMG onset. Post hoc analysis indicated $MG_{RERave}$ during 0-25 ms post sEMG onset declined from PRE at T2 and T4 by 39.2 ± 58.9 % ($p = 0.021$) and 39.1 ± 60.4 % ($p = 0.033$), respectively (Table 10). Post hoc analysis indicated $MG_{RERave}$ 0-75 ms post sEMG onset declined from PRE at T2 and T4 by 31.8 ± 33.6 % ($p = 0.015$) and 31.3 ± 58.4 % ($p = 0.008$), respectively. No changes were observed for $MG_{RERmax}$ ($p \geq 0.534$) or $MG_{RERave}$ 0-50 ms ($p \geq 0.054$; Table 10). A large effect (Cohen's effect size ($d$) = 0.85), was observed for $MG_{RERave}$ 0-50 ms.

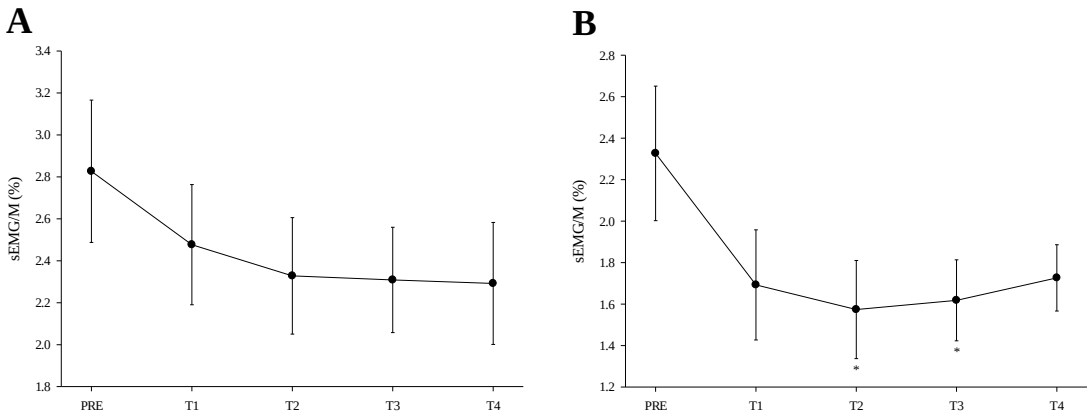


**Figure 21.** Maximal soleus ($SOL_{MAX}$; *A*) and medial gastrocnemius ($MG_{MAX}$; *B*) sEMG activity measured during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. $SOL_{MAX}$ and $MG_{MAX}$ data are expressed as a percentage (%) of the corresponding MVCs' SOL and MG maximal M wave (sEMG/M, %), respectively. * $p < 0.05$ from PRE. Data are grand mean and SE of failure and non-failure conditions.

**Table 10.** Soleus and medial gastrocnemius maximal sEMG activity ($SOL_{MAX}$ and $MG_{MAX}$; sEMG/M, %), maximal rate of sEMG rise up to 200 ms post sEMG onset ($SOL_{RERmax}$ and $MG_{RERmax}$; sEMG/M, $\%.s^{-1}$) and average rate of sEMG rise in time intervals 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset ($SOL_{RERave}$ (0-25), $SOL_{RERave}$ (0-50), $SOL_{RERave}$ (0-75), $MG_{RERave}$ (0-25), $MG_{RERave}$ (0-50) and $MG_{RERave}$ (0-75); sEMG/M, $\%.s^{-1}$). Data was recorded during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions. SOL and MG data are expressed as a percentage of the corresponding MVCs' SOL and MG maximum M-wave (sEMG/M, %), respectively.

| | $SOL_{MAX}$ | | $SOL_{RERmax}$ | | $SOL_{RERave}$ (0-25) | | $SOL_{RERave}$ (0-50) | | $SOL_{RERave}$ (0-75) | |
|---|---|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF | F | NF |
| PRE | 3.0 ± 1.6 | 2.7 ± 1.1 | 87.8 ± 83.3 | 74.0 ± 29.9 | 18.3 ± 14.0 | 20.0 ± 8.8 | 24.6 ± 12.9 | 29.9 ± 14.2 | 27.4 ± 14.3 | 31.9 ± 13.1 |
| T1 | 2.4 ± 1.0 | 2.6 ± 1.3 | 66.7 ± 38.3 | 59.9 ± 26.4 | 18.7 ± 16.2 | 19.4 ± 12.5 | 22.8 ± 13.3 | 28.8 ± 14.4 | 23.5 ± 13.8 | 29.8 ± 15.4 |
| T2 | 2.3 ± 0.9 | 2.4 ± 1.3 | 52.3 ± 30.4 | 62.6 ± 30.6 | 8.5 ± 8.2 | 13.8 ± 7.8 | 12.6 ± 9.9 | 24.8 ± 12.9 | 16.0 ± 11.4 | 28.4 ± 17.4 |
| T3 | 2.1 ± 0.7 | 2.5 ± 1.3 | 37.9 ± 21.4 | 66.4 ± 33.9 | 7.6 ± 4.2 | 12.1 ± 12.4 | 12.8 ± 8.3 | 18.9 ± 15.0 | 14.8 ± 9.7 | 21.6 ± 16.2 |
| T4 | 2.3 ± 1.0 | 2.3 ± 1.4 | 57.4 ± 33.0 | 61.3 ± 37.3 | 9.0 ± 6.0* | 9.2 ± 11.0* | 15.3 ± 8.4* | 16.2 ± 13.7* | 18.6 ± 9.8* | 18.9 ± 15.5* |

| | $MG_{MAX}$ | | $MG_{RERmax}$ | | $MG_{RERave}$ (0-25) | | $MG_{RERave}$ (0-50) | | $MG_{RERave}$ (0-75) | |
|---|---|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF | F | NF |
| PRE | 2.5 ± 1.5 | 2.1 ± 1.1 | 45.6 ± 22.2 | 48.6 ± 21.2 | 18.9 ± 8.5 | 18.7 ± 12.9 | 21.9 ± 9.3 | 23.9 ± 13.8 | 22.6 ± 9.3 | 24.9 ± 12.7 |
| T1 | 1.9 ± 1.5 | 1.5 ± 0.5 | 46.7 ± 31.2 | 40.1 ± 17.1 | 18.2 ± 14.5 | 15.1 ± 12.0 | 22.2 ± 17.5 | 18.4 ± 11.5 | 21.7 ± 16.0 | 18.1 ± 9.4 |
| T2 | 1.7 ± 1.3* | 1.5 ± 0.5* | 36.0 ± 19.1 | 48.0 ± 31.9 | 5.5 ± 2.8* | 11.7 ± 7.9* | 9.8 ± 5.5 | 19.8 ± 14.0 | 11.7 ± 5.5* | 19.6 ± 12.2* |
| T3 | 1.8 ± 0.9* | 1.5 ± 0.7* | 37.1 ± 26.4 | 42.3 ± 31.7 | 12.5 ± 9.2 | 13.3 ± 14.8 | 14.9 ± 9.3 | 16.6 ± 19.7 | 13.3 ± 7.3 | 14.8 ± 16.6 |
| T4 | 2.0 ± 0.7 | 1.5 ± 0.4 | 41.9 ± 23.0 | 40.9 ± 21.2 | 10.2 ± 6.0* | 8.5 ± 7.8* | 13.8 ± 6.8 | 15.2 ± 10.8 | 13.6 ± 4.7** | 14.4 ± 10.8** |

Data are presented as mean ± SD
** $p < 0.01$ from PRE
* $p < 0.05$ from PRE

Peripheral fatigue

Main time effects ($p < 0.01$) were observed for normalised $tRTD_{MAX}$ and $tRTD_{AVE}$. Post hoc analysis indicated $tRTD_{MAX}$ increased from PRE at T1 by 3.7 ± 4.5 % ($p = 0.037$, Figure 22, Table 11). $tRTD_{MAX}$ remained increased between 5.0 % to 6.8 % ($p < 0.01$) from PRE at all subsequent time points. Post hoc analysis indicated $tRTD_{AVE}$ during time intervals 0-50 ms and 0-75 ms post twitch torque onset increased from PRE at T3 by 10.2 ± 14.7 % ($p = 0.048$) and 7.7 ± 8.3 % ($p = 0.007$), respectively (Table 11). No changes were observed for $tRTD_{AVE}$ 0-25 ms post twitch torque onset ($p \geq 0.055$, $d = 0.85$). No change was observed for PT ($p \geq 0.161$, Table 11).

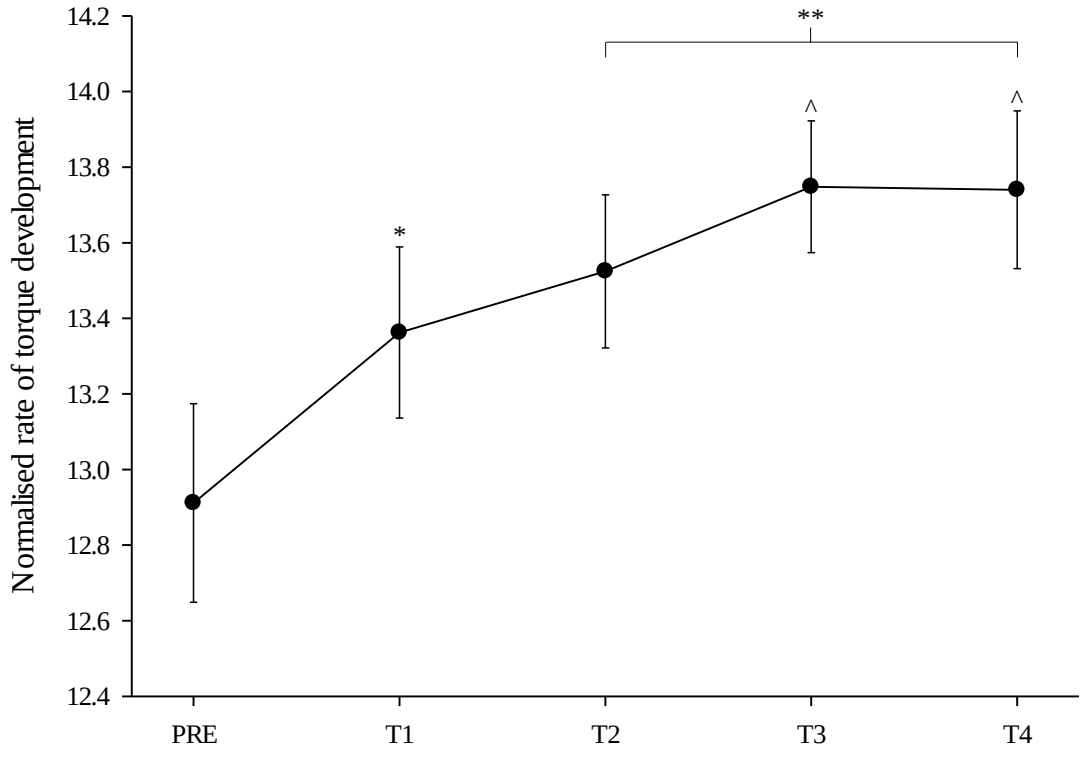


**Figure 22.** Resting twitch maximal rate of torque development ($tRTD_{MAX}$) normalised to the peak torque (PT) of the corresponding resting twitch, measured immediately following maximal voluntary plantar flexion contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p < 0.01$ from PRE, * $p < 0.05$ from PRE, ^ $p < 0.05$ from T1. Data are grand mean and SE of failure and non-failure conditions.

**Table 11.** Resting twitch peak torque (PT, Nm); and resting twitch normalised maximal and average rate of torque development ($tRTD_{MAX}$, $tRTD_{AVE}$) measured immediately following maximal voluntary plantar flexion contractions completed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions. $tRTD_{AVE}$ data are presented as the average slope of the torque-time curve in time intervals of 0-25 ms, 0-50 ms and 0-75 ms post twitch torque onset ($tRTD_{AVE}$ (0-25), $tRTD_{AVE}$ (0-50), $tRTD_{AVE}$ (0-75)). All data are normalised to the peak torque (PT) of the corresponding resting twitch.

| | PT | | $tRTD_{MAX}$ | | $tRTD_{AVE}$ (0-25) | | $tRTD_{AVE}$ (0-50) | | $tRTD_{AVE}$ (0-75) | |
|---|---|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF | F | NF |
| PRE | 100.3 ± 20.6 | 104.1 ± 18.8 | 13.1 ± 1.0 | 12.8 ± 1.2 | 6.1 ± 1.6 | 5.6 ± 1.0 | 8.8 ± 1.7 | 8.4 ± 1.1 | 9.9 ± 1.3 | 9.6 ± 1.0 |
| T1 | 106.1 ± 12.6 | 106.9 ± 13.1 | 13.3 ± 0.9$^{*}$ | 13.4 ± 0.9$^{*}$ | 6.5 ± 0.7 | 6.1 ± 1.1 | 9.4 ± 0.9 | 9.0 ± 1.2 | 10.3 ± 0.7 | 10.1 ± 0.9 |
| T2 | 105.7 ± 16.5 | 107.1 ± 15.3 | 13.4 ± 0.8$^{**}$ | 13.6 ± 0.8$^{**}$ | 6.3 ± 0.7 | 6.5 ± 0.7 | 9.2 ± 0.8 | 9.4 ± 0.8 | 10.3 ± 0.6 | 10.4 ± 0.7 |
| T3 | 105.7 ± 16.9 | 107.6 ± 16.5 | 13.8 ± 0.7$^{**\wedge}$ | 13.7 ± 0.7$^{**\wedge}$ | 6.6 ± 0.9 | 6.1 ± 1.0 | 9.5 ± 0.9$^{*}$ | 9.1 ± 1.1$^{*}$ | 10.5 ± 0.6$^{**}$ | 10.3 ± 0.8$^{**}$ |
| T4 | 99.3 ± 16.6 | 108.5 ± 15.1 | 13.9 ± 0.8$^{**\wedge}$ | 13.6 ± 0.9$^{**\wedge}$ | 6.8 ± 1.1 | 6.3 ± 0.8 | 9.7 ± 1.1$^{**}$ | 9.3 ± 0.9$^{**}$ | 10.6 ± 0.8$^{**\wedge}$ | 10.3 ± 0.8$^{**\wedge}$ |

Data are presented as mean ± SD
$^{**}$ $p < 0.01$ from PRE
$^{*}$ $p < 0.05$ from PRE
$^{\wedge}$ $p < 0.05$ from T1

A main time effect ($p < 0.001$) was observed for TPT. Post hoc analysis indicated TPT decreased from PRE at T2 by 7.3 ± 6.1 % ($p = 0.006$; Figure 23; Table 12). TPT remained decreased between 9.7 ± 6.3 % and 10.3 ± 5.7 % from PRE at T3 and T4, respectively ($p < 0.01$).

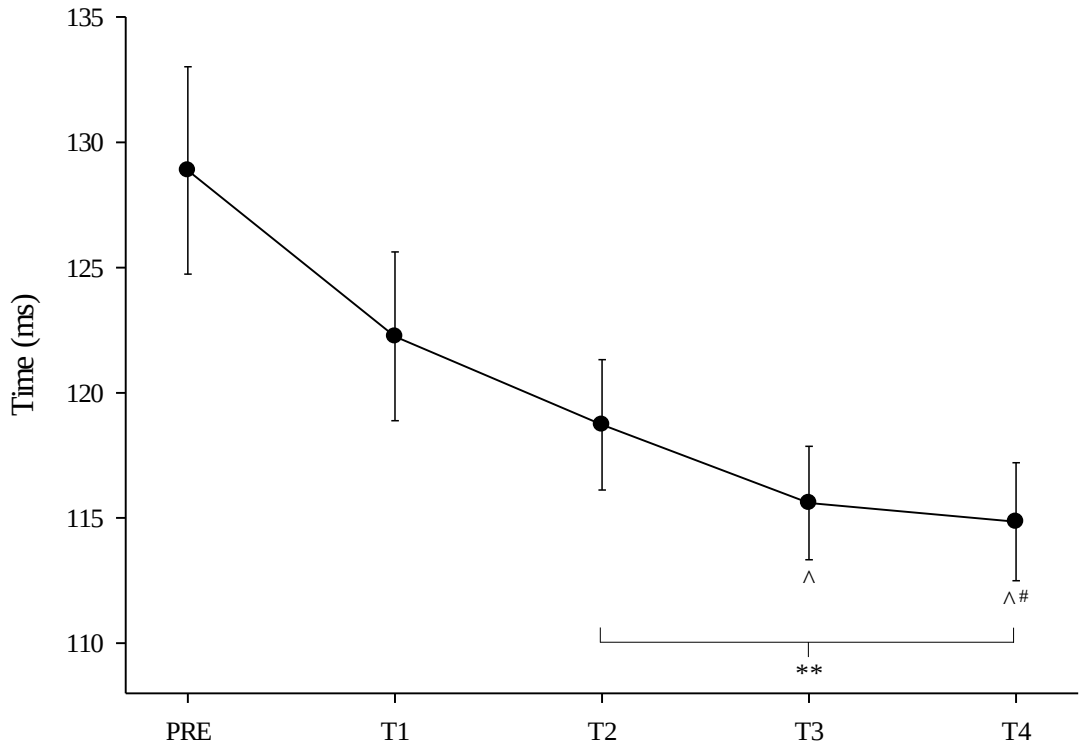


**Figure 23.** Resting twitch time to peak torque (TPT, ms) measured immediately following maximal voluntary plantar flexion contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p < 0.01$ from PRE, ^ $p < 0.05$ from T1, # $p < 0.05$ from T2. Data are grand mean and SE of failure and non-failure conditions.

A main time effect ($p$ = 0.008) was observed for ½ RT. Post hoc analysis indicated ½ RT decreased from PRE at T2 by 15.5 ± 14.7 % ($p$ = 0.020; Figure 24; Table 12). ½ RT remained decreased between 20.3 ± 12.8 % and 22.2 ± 11.5 % from PRE at T3 and T4, respectively ($p$ < 0.01).

No changes were observed for $SOL_{Mmax}$ ($p \geq 0.693$) and $MG_{Mmax}$ ($p \geq 1.000$, Table 12).

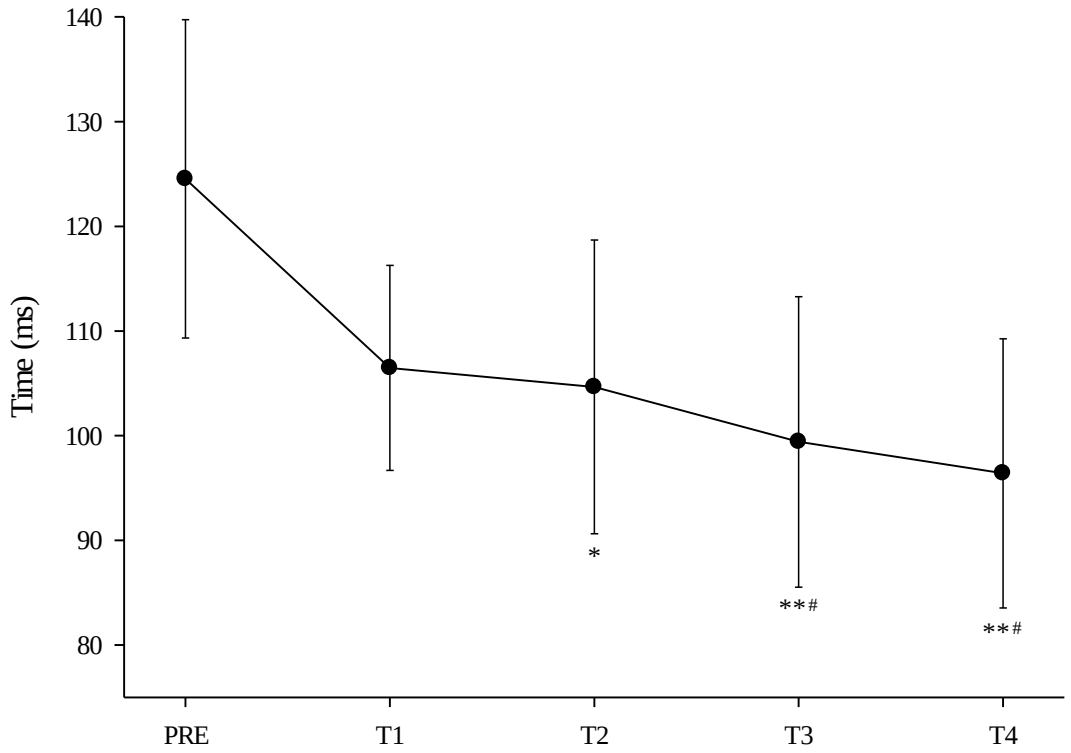


**Figure 24.** Resting twitch half-relaxation time (½ RT, ms) measured immediately following maximal voluntary plantar flexion contractions completed before (PRE) and during (T1, T2, T3 and T4) failure and non-failure conditions. ** $p$ < 0.01 from PRE, * $p$ < 0.05 from PRE, # $p$ < 0.05 from T2. Data are grand mean and SE of failure and non-failure conditions.

**Table 12.** Resting twitch time to peak torque (TPT, ms) and half-relaxation time (½ RT, ms) measured immediately following maximal voluntary plantar flexion contractions; and soleus and medial gastrocnemius maximum M-wave ($SOL_{Mmax}$, $MG_{Mmax}$; mV) evoked during maximal voluntary plantar flexion contractions performed before (PRE) and during (T1, T2, T3 and T4) failure (F) and non-failure (NF) conditions.

| | TPT | | ½ RT | | $SOL_{Mmax}$ | | $MG_{Mmax}$ | |
|---|---|---|---|---|---|---|---|---|
| MVC | F | NF | F | NF | F | NF | F | NF |
| PRE | 127.5 ± 18.1 | 130.3 ± 15.9 | 121.6 ± 67.3 | 127.4 ± 58.0 | 6.9 ± 1.9 | 7.3 ± 2.5 | 7.7 ± 3.1 | 8.8 ± 3.3 |
| T1 | 123.5 ± 14.9 | 121.0 ± 12.8 | 109.4 ± 20.2 | 103.4 ± 53.4 | 7.2 ± 1.9 | 6.8 ± 2.6 | 8.8 ± 3.0 | 9.9 ± 3.2 |
| T2 | 119.7 ± 11.1$^{**}$ | 117.8 ± 10.4$^{**}$ | 110.9 ± 60.1$^{*}$ | 98.3 ± 55.2$^{*}$ | 7.1 ± 2.1 | 7.0 ± 2.8 | 9.1 ± 3.3 | 9.4 ± 3.1 |
| T3 | 114.9 ± 9.1$^{**\wedge}$ | 116.3 ± 9.6$^{**\wedge}$ | 104.8 ± 59.9$^{**\#}$ | 94.1 ± 54.3$^{**\#}$ | 7.3 ± 1.9 | 7.0 ± 3.0 | 8.1 ± 3.3 | 10.0 ± 3.1 |
| T4 | 113.8 ± 9.9$^{**\wedge\#}$ | 115.9 ± 9.5$^{**\wedge\#}$ | 99.1 ± 51.2$^{**\#}$ | 93.7 ± 55.1$^{**\#}$ | 7.1 ± 1.9 | 7.3 ± 2.9 | 7.3 ± 3.2 | 9.2 ± 2.0 |

Data are presented as mean ± SD
$^{**}$ $p < 0.01$ from PRE
$^{*}$ $p < 0.05$ from PRE
$^{\wedge}$ $p < 0.05$ from T1
$^{\#}$ $p < 0.05$ from T2

# DISCUSSION

This investigation did not support the hypothesis that acute reductions in maximal strength would be greater following failure than non-failure resistance exercise. The results in fact demonstrated that moderate to high intensity (80 % 1 RM) isotonic contractions of the plantar flexors produced a similar decline in maximal torque output regardless of whether exercise was completed to failure or not. However, given the present downregulation of voluntary activation (VA) and muscle surface electromyographic activity (sEMG/M) observed following failure and non-failure exercise, some evidence was found to support the hypothesis that central (neural) factors would be responsible for the decline in maximal strength following exercise. An unexpected finding was that peripheral factors did not mediate the change in muscular strength, and in contrast to the hypothesis, were potentiated following failure and non-failure exercise.

The similar reduction in maximal torque output following isotonic failure and non-failure exercise observed in this study supports previous research conducted in trained populations. Both Marshall *et al.* (2012) and Benson *et al.* (2006) observed comparable acute declines in maximal torque output following traditional multi-set isotonic exercise completed to failure or not to failure using moderate to high intensity loading. Given the similar decline in maximal torque output following failure and non-failure isotonic exercise observed here in the plantar flexors and previously in the knee extensors (Marshall *et al.*, 2012) and elbow flexors (Benson *et al.*, 2006), performing isotonic exercise to failure has demonstrated a tendency to be no more effective at stimulating reductions in maximal torque output regardless of the muscle group tested. Furthermore, the current trend seems to suggest that performing isotonic exercise to failure is not as fatiguing (relative to non-failure exercise) as

originally thought and that when moderate to high intensity loads are prescribed, neither exercise modality is capable of generating larger reductions in maximal torque output than the other. Additionally, central fatigue, observed via reductions in maximal muscle activity following failure and non-failure exercise in this study supports the decline in muscle activity observed in the aforementioned investigations (Benson *et al.*, 2006; Marshall *et al.*, 2012). These previous reports were limited in their ability to differentiate between the central and peripheral mechanisms that mediate muscular force production. The results of this study have extended prior understanding of these mechanisms, now demonstrating that central factors observed as reductions in both VA and muscle activity are primarily responsible for acute reductions in muscular strength following dynamic failure and non-failure exercise in trained individuals.

The 15.0 % reduction in maximal torque output observed following moderate to high intensity failure and non-failure isotonic exercise in this study was the result of a downregulation of central input to the plantar flexor motor neuron pool, indicated by a 9.5 % decrease in VA and an up to 49.2 % decrease in maximum and rate dependent measures of muscle activation (sEMG/M). The findings of this investigation are in agreement with Hartman and colleagues who observed similar reductions in maximum plantar flexor VA and sEMG activity following a series of moderate intensity (40 % MVC) contractions performed to failure in trained individuals (Hartman *et al.*, 2011). However, the results of this study contrast those of Klass *et al.* (2004) that showed central motor drive (measured using maximum VA and muscle sEMG activity) is maintained following moderate intensity (50 % MVC) explosive plantar flexion contractions performed to failure in untrained populations. Given measures of central drive have not shown dependence on training experience following isotonic plantar flexion contractions (Hartman *et al.*, 2011), disagreement between the present

and previous data are potentially related to differences in contractile cadence and joint range of motion (Klass *et al.*, 2004) that are known to affect the magnitude of the central response to exercise (Morel *et al.*, 2015).

The reduction in maximal sEMG/M amplitude in MG ($MG_{MAX}$) but not in SOL ($SOL_{MAX}$) in this study suggests that plantar flexor central drive is muscle specific. Similarly, Nordlund *et al.* (2004) observed similar differences in MG and SOL intramuscular EMG/M following ninety plantar flexion MVCs. Interestingly, using the V-wave (Upton *et al.*, 1971; Aagaard *et al.*, 2002b) as a reflection of central drive to the plantar flexor motor unit pool in untrained individuals, a recent investigation has demonstrated that drive to MG and SOL is muscle specific with fatiguing submaximal (55 % MVC) exercise performed to failure (Siegler *et al.*, 2014). The doublet stimulation method used to determine VA in the present investigation was not sensitive enough to detect changes in central drive between the synergistic plantar flexor muscle group. Supramaximal doublet α-motor neuron stimulation was chosen to minimise the deleterious effects of contraction coupling failure on resting potentiated twitch amplitude (and thus the calculation of voluntary activation) occurring with fatiguing exercise (McKenzie *et al.*, 1992; Shield & Zhou, 2004). Future research should therefore consider evoking a V-wave during MVC to estimate whether reductions in central drive with fatiguing exercise are muscle specific in trained individuals. Furthermore, given type I and type II muscle fibre distribution is vastly different between MG and SOL (Johnson *et al.*, 1973), the V-wave or other measures central drive such as transcranial magnetic stimulation, may be used to discern whether mechanisms of central drive are dependent on motor unit/muscle fibre morphology in trained populations.

Additionally, VA is thought to reflect both spinal and supraspinal processes contributing to central impairment (Gandevia, 2001), and as such, is limited in its interpretation of central drive. Nordlund and colleagues have observed reductions in the 'level of activation' (a calculation method similar to that of VA) following isometric plantar flexion exercise (described above) (Nordlund *et al.*, 2004). This decline was accompanied by decreased Ia afferent excitability, indicated by reductions in the maximum SOL H-reflex-to-M-wave ratio and increased pre-synaptic inhibition (Nordlund *et al.*, 2004). Although speculative, these results suggest that impaired spinal afferent excitability may have partially contributed to the increase in central fatigue observed in the present study. Given central factors exhibited the greatest influence on plantar flexor torque production following failure and non-failure exercise in this investigation, prospective research should aim to clarify the development of central fatigue in the plantar flexors by employing techniques such as direct motor cortex and Ia afferent stimulation that can be used to observe mechanisms responsible for impaired input to the α-motor unit pool.

Although no difference in peripheral fatigue was observed between failure and non-failure conditions, this study did not find evidence to support the hypothesis that peripheral along with central fatigue would contribute to a decline in maximal strength following exercise. Maximum M-wave amplitude and resting twitch peak torque (PT) remained unaffected by both exercise modalities, demonstrating that neither failure of action potential depolarisation at the neuromuscular junction/transmission across the sarcolemma or processes related to excitation-contraction coupling explained the reduction in maximal torque output following failure and non-failure exercise. The maintenance of maximum M-wave amplitude following isotonic plantar flexion exercise performed to failure supports previous findings (Hartman *et al.*, 2011). However, Hartman and colleagues did not examine peripheral functioning distal to

the muscle cell membrane (Hartman *et al.*, 2011) (typically reported using the torque-time characteristics of a resting potentiated twitch) and thus, their investigation did not provide a thorough examination of the peripheral factors known to modulate voluntary force production with fatiguing exercise. The present data contrast previous reports that showed concurrent increases in both peripheral and central fatigue (indicated by reductions in PT and VA, respectively) promoted a decline in maximal torque output following isometric plantar flexion exercise in untrained populations (Kawakami *et al.*, 2000; Nordlund *et al.*, 2004; Siegler *et al.*, 2014). Therefore, the maintenance of PT and improved resting twitch torque-time properties in this study may infer that peripheral fatigue of the plantar flexors is a function of the exercise task itself, and/or that peripheral mechanisms are well adapted in trained individuals to facilitate voluntary and explosive force production in the presence of significant downregulation of central drive during fatiguing dynamic contractions.

In this study the maintenance of PT, shortening of resting twitch time to peak torque (TPT) and half relaxation time (½ RT) by 10.3 % and 22.2 %, respectively; and the greater than 6.8 % increase in measures of electrically evoked involuntary explosive torque production ($tRTD_{MAX}$ and $tRTD_{AVE}$) contrasted the hypothesised increase in peripheral fatigue that would typically be expected following exercise. Potentiation of these measures of intrinsic contractile functioning can produce reductions (Klass *et al.*, 2004) or increases (Behm & St-Pierre, 1997) in plantar flexor PT and thus maximal cross-bridge binding. In contrast, the findings of this study support the work of Davies *et al.* (1983) who observed no change in plantar flexor PT and similar reductions in TPT (24 %) and ½ RT (22 %) following a bout of exercise. The authors attributed the potentiation intrinsic contractile functioning to an elevation of muscle temperature. Although speculative, it is possible that the failure and non-failure protocols used in this investigation facilitated an increase in muscle temperature that

promoted the up-regulation of excitation-contraction coupling and involuntary torque production through the effects of increased ATP hydrolysis and the attachment of M.ADP.Pi to actin (Offer & Ranatunga, 2015). Furthermore, other literature has suggested that a reduction in TPT and ½ RT may be attributed to shorter $Ca^{2+}$ transients (Strojnik & Komi, 1998), whereas increased maximal and average tRTD manifest through myosin light chain phosphorylation, making the myosin interactions with actin more sensitive to $Ca^{2+}$ and stimulating more rapid binding (Sweeney *et al.*, 1993). Additionally, the effect of myosin light chain potentiated force generating capacity does not promote increases in maximal force production during high intensity isometric tetanic stimulation, despite increasing the maximal rate of force production at high or low stimulation frequencies (Vandenboom *et al.*, 1993). This mechanism may explain why voluntary rate of torque development increased despite the observed reductions in maximal voluntary torque output. The results also suggest that not only did the potentiation of intrinsic contractile functioning increase explosive torque production, but that it mediated the increase in central fatigue seen through reductions in early phase rate of rise of muscle activation that would have likely contributed to a reduction in voluntary rate of torque development.

This investigation demonstrated that in resistance trained populations, a single bout of moderate to high intensity plantar flexion exercise performed to failure is no more effective at stimulating reductions in voluntary torque output than a similar bout of exercise not performed to failure. The present data suggest that failure of torque generating capacity during fatiguing voluntary skeletal muscle contractions was the result of considerable impairment of central neural drive, observed as reductions in voluntary activation and muscle activity. The novel finding of a potentiation of intrinsic contractile functioning likely explained the observed increase in voluntary rate of torque development. However, further

investigation is required to determine if this finding has important implications for the development of muscular power in trained individuals following a period of failure and non-failure training.

# CHAPTER 5

*Chronic Failure and Non-failure Exercise of the Plantar Flexors*

## METHODS

### Subjects

Sixteen healthy resistance trained volunteers ($n$ = 16; 12 men and 4 women) participated in the study (age, 22.1 ± 3.1 years; height, 170.9 ± 9.1 cm; body mass, 75.0 ± 9.4 kg; training experience, 3.5 ± 2.1 years; calf raise one repetition maximum (RM), 66.1 ± 14.7 kg; mean ± SD). All participants were required to have regularly (at least 3 days per week) completed resistance exercise of the upper and lower body for the previous 24 months and be capable of performing dynamic 1 RM barbell back squat and seated calf raise contractions ≥ 130 % and 60 % of body mass, respectively. Participants were excluded if they reported taking performance enhancing substances as per the World Anti-Doping Agency's 2012 prohibited list, had a recent history of upper or lower limb injury (within the last 3 months), or any known metabolic or neuromuscular disease. Participants were instructed to refrain from any resistance exercise external to the study design and maintain normal dietary habits for the course of the intervention. Each participant gave written informed consent prior to testing. All procedures were approved by Western Sydney University's Human Research Ethics Committee (approval number H10408) and were carried out in accordance with the Declaration of Helsinki.

### Experimental design and training

Participants trained for a total of 8 weeks. Assessments were conducted at baseline and at the conclusion of the training period. Familiarisation sessions served to determine inclusion and initial training loads based on seated calf raise 1 RM and 10 RM testing, respectively, and to

accustom participants to posterior tibial nerve stimulation during isometric plantar flexion contractions.

Participants completed four training sessions per week. Training was prescribed in a two-way bodypart split format (A program: legs and shoulders; B program: chest, back and arms) to ensure each muscle group was trained twice per week (Rhea *et al.*, 2003). The second performance of the A and B program each week was completed with a reduced training volume to avoid potential deleterious effects of overreaching. Prior to completing the primary working sets, participants performed 1-2 warm up sets at approximately 50 % and 75 % of the training load. A traditional multi-set prescription (3-8 sets per exercise) was used throughout the program, with six primary exercises performed each session using 6-12 RM loading (Appendix I).

Participants were randomly assigned to either a repetition failure or non-failure grouping. A RM loading scheme (for example, 10 reps × 10 RM load) was used throughout the training period because it was easier to prescribe exercise and match training volumes between experimental groups using this method compared to a percentage of 1 RM based prescription (for example, 10 reps × 75 % 1 RM). The failure condition required participants to complete exercise sets to volitional exhaustion, defined as the inability to move a load through the full range of motion (ROM) with a 2:1 sec eccentric-to-concentric contraction cadence, in a controlled manner without assistance, or when participants felt they could no longer continue. If participants failed, consequently performing > 2 repetitions less than the desired RM range, the weight was decreased 2.5-5 kg to maintain the appropriate volume for subsequent sets. All failure sets were completed with 180 sec inter-set recovery periods. Participants in the non-failure group completed repetitions with the same RM training range as the failure group,

although performed an equal number of repetitions over two sets instead of one. This allowed training volume to be equated between groups (i.e. a single 10 RM failure set corresponded to 2 sets × 5 repetitions with a 10 RM load in the non-failure condition). All non-failure sets were completed with 90 sec inter-set recovery periods.

Participants were required to attend the institution's gymnasium facility to complete seated calf raise testing and training sessions (A program) under direct supervision of the primary researcher. The seated calf raise (adjustable seated calf raise machine, ForceUSA) was the only plantar flexion exercise performed throughout the training period. Participants were instructed to refrain from engaging in high intensity resistance exercise external to the study design for the duration of the training period. Participants consumed a commercially sourced (Bulk Nutrients, Australia) protein (Whey protein isolate, 40 g) and carbohydrate (maltodextrin, 30 g) dietary supplement immediately upon the completion of each exercise session to control for variations in post workout nutritional intake (Burke *et al.*, 2004; Cribb & Hayes, 2006). Participants were instructed to maintain normal dietary habits over the course of the training period and complete detailed nutritional diaries during weeks one, four and eight to monitor energy consumption.

Experimental procedures

*Plantar flexor maximal strength*

Dynamic plantar flexor strength was tested using a 1 RM and 10 RM bilateral seated calf raise exercise. To determine eligibility, participants were required to complete a 1 RM eccentric-concentric seated calf raise contraction with a load ≥ 60 % body mass. Pilot testing

demonstrated that this was reasonable to expect from a resistance trained population. An electro-goniometer (MLTS700, ADInstruments, Australia) was used to determine the ROM required for successful completion of 1 RM. The centre of the goniometer was aligned with the right lateral malleolus and secured to the lateral aspect of the right shank and fifth metatarsal. Weight equalling an approximate 1 RM load was added to the seated calf raise machine (adjustable seated calf raise machine, ForceUSA) and participants performed a controlled contraction to full eccentric range of motion until the raw signal (degrees, º) plateaued for 2-3 sec. The weight was removed and participants completed an unloaded contraction to full concentric ROM until the raw signal plateaued for 2-3 sec. The difference between concentric and eccentric values was used as a measure of joint ROM.

Prior to 1 RM testing, participants completed a short series of dynamic plantar flexion contractions with a load approximately equalling 50 % and 75 % of predicted 1 RM. Following this warm up, 1 RM was assessed. 1 RM was deemed successful if participants completed the single repetition with a 2:1 sec eccentric-to-concentric cadence and achieved 90 % of full joint ROM. The highest weight (kg) achieved prior to failing was recorded as the participants' 1 RM. Weight was increased in 2.5-5 kg increments until 1 RM was achieved, always within 3-5 attempts. Strong verbal encouragement was provided and participants received 3 min rest between 1 RM attempts to ensure adequate recovery.

Training of the plantar flexors was completed with loads corresponding to seated calf raise 10 RM, regardless of group randomisation. 10 RM was assessed at baseline (T0), to determine initial training loads; at the end of weeks three (T1) and six (T2), as a measure of exercise progression; and at the conclusion of the training period in week 9 (T3), as a measure of training induced changes in muscular strength. 10 RM testing followed similar procedures to

those outlined above for 1 RM assessment. Briefly, if participants failed ≥ ± 2 repetitions outside the 10 RM range, the load was increased or decreased by 2.5-5 kg until a valid 10 RM load was determined.

*Squat maximal strength*

Participants were required to complete a 1 RM barbell back squat ≥ 130 % of body mass to determine study eligibility. Participants first completed a warm up consisting of a short series of repetitions with a load approximately equalling 50 % and 75 % of estimated 1 RM, followed by 1-2 repetitions at an approximate 80 % 1 RM load. The highest weight (kg) achieved prior to failure was recorded as the 1 RM. Weight was increased in 2.5-10 kg increments until 1 RM was achieved, always within 3-5 attempts. Participants were required to perform 1 RM attempts with a 2:1 sec eccentric-to-concentric contraction cadence and descend to a 'parallel' depth in which the inguinal fold was perpendicular to the superior aspect of the patella. Squat depth was verified by the primary research investigator throughout all trials. Attempts were considered valid if participants completed the lift to the required depth in a controlled manner and without assistance. Participants received 3-5 min rest between 1 RM attempts to ensure adequate recovery. Strong verbal encouragement was provided on all trials.

*Maximal voluntary contractions and toque recording*

All testing was performed on the plantar flexors of the left leg. Participants sat upright in an isokinetic dynamometer (KinCom 125, Version 5.32, Chattanooga, USA) with the hip, knee and ankle flexed to 90º throughout all maximal voluntary contractions (MVCs). The centre of

rotation of the dynamometer lever arm was aligned with the lateral malleolus. The lever arm of the dynamometer was aligned with and firmly secured around the metatarsophalangeal joint. Participants were securely fastened to the dynamometer with straps placed across the torso, hips and thighs. Participant alignment was recorded to maintain consistency between sessions.

Prior to completing maximal voluntary contractions (MVCs), participants performed a warm up consisting of a series of short submaximal isometric plantar flexion and dorsiflexion contractions at 50 % and 75% of perceived maximal effort. Following this warm up, three plantar flexion and dorsiflexion MVCs were completed and the maximal torque output ($T_{MAX}$, Nm) was averaged across the respective muscle groups to determine the 10 % $T_{MAX}$ contraction intensity required for subsequent testing. No data from these three MVCs was used for analysis. All torque signals were sampled at 4,000Hz (Powerlab 16/35, ADInstruments Australia; 16 bit analog to digital conversion) and filtered with a $4^{th}$ order 10Hz digital low pass filter prior to analysis. Participants were instructed to complete all MVCs as fast and as forcefully as possible and maintain contraction for 3-4 sec. Strong verbal encouragement was provided throughout and a minimum of one minute rest was given between MVC attempts.

*Electromyography*

Surface electromyograms (sEMG) were recorded from the left soleus (SOL), medial gastrocnemius (MG) and tibialis anterior (TA) muscles using pairs of Ag/AgCl surface electrodes (Maxsensor, Medimax Global, Australia). SOL, MG and TA electrodes (10 mm diameter, 10 mm inter-electrode distance) were applied in bipolar configuration parallel to

the direction of the muscle fibres after careful skin preparation (shaving of excess hair, abrasion with fine sandpaper and cleaning the skin with isopropyl alcohol swabs) to reduce skin electrical impedance below 5 kΩ. SOL electrodes were placed at ⅔ of the line between the medial condyle of the femur and the medial malleolus; GM electrodes were positioned on the most prominent bulge of the muscle; and TA electrodes were placed at ⅓ of the line between the head of the fibula and the medial malleolus. Placement sites were recorded for each participant with respect to anatomical landmarks to maintain consistency between sessions. The reference electrode was placed on the left medial malleolus. sEMG signals were recorded using an ML138 Octal BioAmp (common mode rejection ratio > 85 dB at 50 Hz, input impedance 200 MΩ) with a 16-bit analog-to-digital conversion, sampled at 4,000 Hz (ADInstruments, Australia). Prior to analysis, raw signals were filtered with a fourth-order Bessel filter between 20 Hz and 500 Hz and smoothed using a root mean square (RMS) calculation with a 100 ms time constant. All sEMG RMS values were normalised to the raw sEMG M-wave evoked during each analogous MVC (sEMG/M, %) to control for potential changes in axonal excitability (Pasquet *et al.*, 2000).

*Electrical stimulation*

*Posterior tibial nerve*. All electrical stimulations (single square wave pulses, 1 ms duration applied at 400 V) were provided by a constant current stimulator (Digitimer DS7AH, Welwyn Garden City, UK). A rubber insulated portable cathodal probe was used to deliver low intensity stimulations (20 mA) to the popliteal fossa to identify nerve location for cathodal stimulation. The popliteal fossa was pre-marked with a permanent marker with optimal cathodal location determined at rest by moving the probe until the largest evoked peak-to-peak M-wave was elicited in SOL. A single Ag/AgCl surface electrode (15 mm

diameter; Kendall, Covidien, USA) was applied to the point of optimal cathodal stimulation. The anode was custom made from aluminium foil (8.5 × 5.5 cm dispersal pad), covered in a layer of conductive gel (Ten20 Conductive Paste, Weaver and Company, USA) and firmly taped to the thigh 2 cm superior to the patella.

*Common peroneal nerve*. To identify nerve location, the cathodal probe was used to deliver low intensity stimulations (5 mA) to the common peroneal nerve, posterior to the head of the fibula. The fibula head and surrounding area was pre-marked with permanent marker with optimal cathodal location determined at rest by moving the probe until the largest evoked peak-to-peak M-wave was elicited in TA. A single surface electrode (15 mm diameter) was applied to the point of optimal cathodal stimulation. The anode was custom made from aluminium foil (3 × 3 cm dispersal pad), covered in a layer of conductive gel and firmly taped to the medial aspect of the shank 4 cm inferior to the medial tibial tuberosity.

*H-reflex recruitment*

SOL and MG H-reflex characteristics were observed by measuring H-reflex and M-wave recruitment at different stimulus (current) intensities to provide information on spinal reflex excitability and thus motor unit activation occurring at different thresholds across the spectrum of the afferent volley (Klimstra & Zehr, 2008; Vila-Cha *et al.*, 2012). H-reflex threshold was initially determined by progressively increasing the current intensity in 1 mA increments (from 0 mA) until a visible H-reflex waveform was observed on the raw SOL sEMG signal. Above the H-reflex threshold, the current intensity was increased in 10 mA increments until a plateau in the peak-to-peak amplitude of the SOL M-wave ($M_{MAX}$) was observed. Recruitment curves were constructed using 40 stimulation sweeps separated on a

logarithmic scale (Brinkworth *et al.*, 2007) between the current intensities corresponding to 80 % SOL H-reflex threshold and SOL $M_{MAX}$. Three single stimulations of equal current intensity were delivered at each sweep and the values (mA) manually recorded to allow recruitment to be normalised to current intensity in the subsequent data analysis. All individual stimulations were accompanied by a 10-15 sec inter-stimulus latency period wherein participants were not required to maintain plantar flexion contraction. Rest intervals between stimulations were used in an attempt to reduce the likelihood of homosynaptic mediated postactivation depression affecting subsequent recruitment amplitudes (Hultborn *et al.*, 1996). All H-reflex and M-wave measurements were recorded whilst participants performed low intensity (10 % $T_{MAX}$) isometric plantar flexion contractions to minimise post-synaptic events (Knikou, 2008) and to control for changes in motor neuron excitability known to effect passive H-reflex recruitment (Nordlund *et al.*, 2004). Real-time torque feedback for H-reflex recruitment and spinal inhibition testing was continuously displayed on a 25" LCD monitor (LG™, Australia).

*Spinal inhibition*

Testing of spinal reflex inhibition was completed to provide an understanding of adaptations to afferent excitability and supraspinal drive. Inhibition at the spinal level is facilitated by multiple factors including homosynaptic mediated postactivation depression (HPAD) and gamma-aminobutyric-acid (GABA) mediated primary afferent depolarisation (GPAD), pre-synaptically (Nordlund *et al.*, 2004); and recurrent homonymous and heteronymous inhibition, post-synaptically (Bussel & Pierrot Deseilligny, 1977; Iles *et al.*, 2000). During the inhibition protocols, H-reflexes were evoked at a constant percentage (20 %) of $M_{MAX}$ (Aagaard *et al.*, 2002b; Holtermann *et al.*, 2007) to ensure synaptic input received by the α-

motor neurons was consistent and therefore activated the same motor neuron pool between paired stimulus trains and following the training period (Capaday, 1997; Zehr, 2002). The current intensity was chosen to reflect ascending H-reflex recruitment whilst avoiding possible modulations of HPAD and GPAD occurring during the period of descending H-reflex recruitment (Nordlund *et al.*, 2004). Inhibition was assessed using a paired pulse stimulation technique (Kipp *et al.*, 2011). At least 10 (Hopkins *et al.*, 2000) paired stimulus trains were delivered and averaged prior to analysis for all measures of pre- and post-synaptic inhibition. All paired stimulus trains were evoked whilst participants completed 10 % $T_{MAX}$ isometric plantar flexion contractions and were separated by a 10-15 sec inter-stimulus latency period. The four inhibition protocols were completed in random order.

*Homosynaptic mediated post activation depression*. Two stimuli with an inter-stimulus interval of 100 ms were delivered to the posterior tibial nerve in the popliteal fossa. The first (test reflex) and second (conditioned reflex) stimulations were evoked at a current intensity corresponding to 20 % SOL $M_{MAX}$. Each stimulation produced an H-reflex in the SOL muscle. The amplitude of the conditioned reflex was typically smaller relative to the test reflex (Johnson *et al.*, 2014) and thus reflected the degree of post activation depression (i.e. inhibition). The peak-to-peak amplitude of the test and conditioned reflexes were expressed as a percentage of $M_{MAX}$ and averaged across the stimulus trains (Figure 25). The average conditioned reflex was then expressed as a percentage of the average test reflex to reflect the degree of HPAD (Baudry *et al.*, 2011): HPAD (%) = 100 – (Conditioned reflex/Test reflex) × 100.

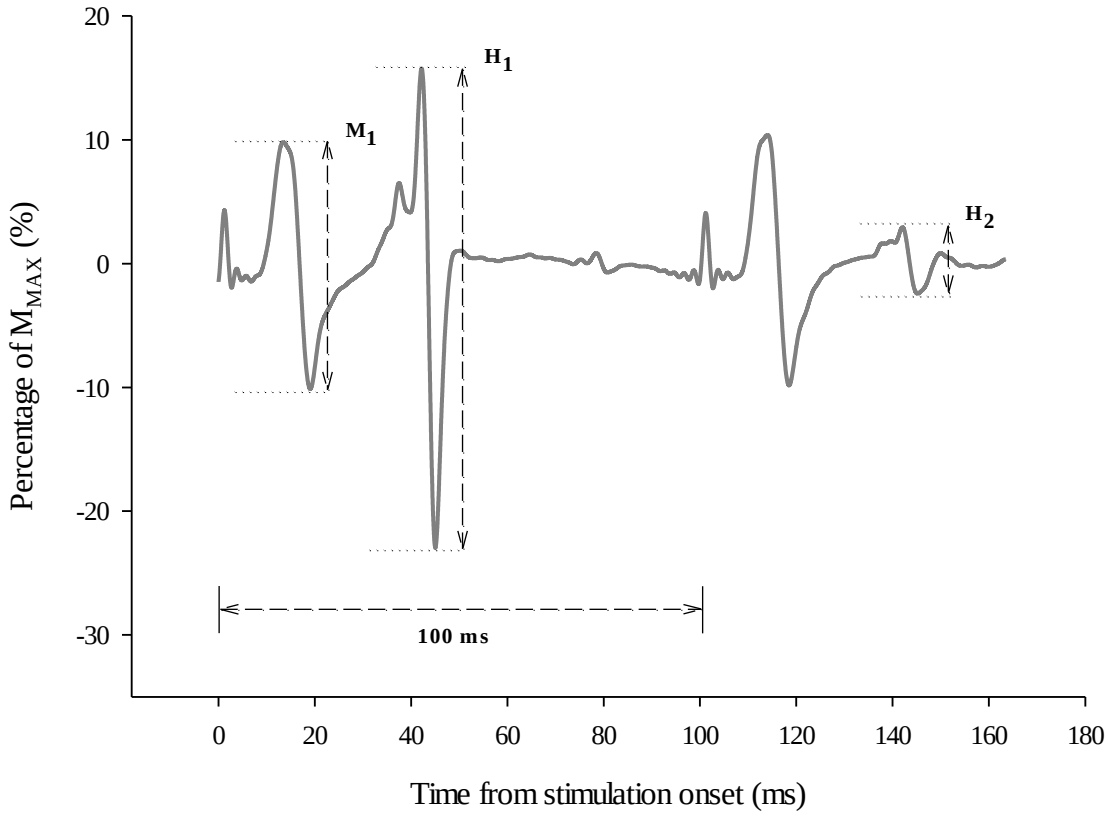


**Figure 25.** A representative HPAD trial in the SOL muscle, expressed as a percentage of the maximal M-wave. HPAD was calculated using the amplitude of the test ($H_1$) and conditioned ($H_2$) reflexes. Also illustrated are the time (100 ms) between stimulations and the amplitude of the M-wave produced when stimulating at 20 % of $M_{MAX}$ ($M_1$).

*GABA mediated primary afferent depolarisation*. A single stimulation was delivered to the common peroneal nerve posterior to the head of the fibula followed 100 ms later by a single stimulation to the posterior tibial nerve in the popliteal fossa. The first (test reflex) and second (conditioned reflex) stimulations were evoked at current intensities corresponding to 20 % TA $M_{MAX}$ and 20 % SOL $M_{MAX}$, respectively. The peak-to-peak amplitude of the conditioned reflex was expressed as a percentage of $M_{MAX}$ and averaged across the stimulus trains. The average conditioned reflex amplitude was then expressed as a percentage of the

normalised average test reflex amplitude (obtained from HPAD trains) to reflect the degree of GPAD.

*Recurrent homonymous inhibition.* Two stimuli with an inter-stimulus interval of 10 ms were delivered to the posterior tibial nerve in the popliteal fossa. The first (test reflex) and second (conditioned reflex) stimulations were evoked at current intensities corresponding to 20 % and 100 % SOL $M_{MAX}$, respectively. By eliciting the two stimulations in such short succession, the orthodromic afferent volley from the first stimulation and the antidromic motor volley from the second stimulation collide, cancelling one another out allowing the conditioned reflex (H′) from the second stimulation to pass through the α-motor neuron axon to the muscle relatively unaffected (Pierrot-Deseilligny *et al.*, 1976; Knikou, 2008). The peak-to-peak amplitude of the H′ reflex was expressed as a percentage of $M_{MAX}$ and averaged across stimulus trains. The average H′ reflex amplitude was then expressed as a percentage of the average normalised test reflex amplitude (obtained from HPAD trains) to reflect the degree of recurrent homonymous inhibition.

*Recurrent heteronymous inhibition.* A single stimulation was delivered to the common peroneal nerve posterior to the head of the fibula followed 10 ms later by a single stimulation to the posterior tibial nerve in the popliteal fossa. The first (test reflex) and second (conditioned reflex) stimulations were evoked at current intensities corresponding to 20 % TA $M_{MAX}$ and 100 % SOL $M_{MAX}$, respectively. The peak-to-peak amplitude of the conditioned H′ reflex from the second stimulation was expressed as a percentage of $M_{MAX}$ and averaged across stimulus trains. The average H′ reflex amplitude was then expressed as a percentage of the average normalised test reflex amplitude (obtained from HPAD trains) to reflect the degree of recurrent heteronymous inhibition.

Prior to GPAD and recurrent heteronymous inhibition testing, TA H-reflex threshold was determined by progressively increasing the stimulation intensity (from 0 mA) in 0.5 mA increments until an H-reflex waveform was visible on the raw TA sEMG signal. The intensity of stimulations was then increased in 2 mA increments until a visible plateau in the peak-to-peak amplitude of the TA M-wave was observed ($M_{MAX}$). TA H-reflex threshold and $M_{MAX}$ were recorded whilst participants performed 10 % $T_{MAX}$ isometric dorsiflexion contractions.

*V-wave*

V-waves were evoked and averaged in the SOL and MG muscles across five MVCs to provide an estimation of adaptations occurring to supraspinal neural drive (Upton *et al.*, 1971; Aagaard *et al.*, 2002b). The maximal stimulus intensity used to evoke $M_{MAX}$ (range, 60-150 mA) was multiplied by 150 % to establish supramaximal stimulation intensity. A single supramaximal stimulus was applied to the posterior tibial nerve in the popliteal fossa during MVC when a visible plateau in the torque trace was observed for 1-2 sec.

*Voluntary activation*

The superimposed twitch technique (Merton, 1954) was used to measure plantar flexor voluntary activation (VA). The concomitant superimposed twitch evoked from the V-wave recording procedure (described above) was used for analysis. An additional supramaximal resting potentiated twitch was evoked 3-4 sec following completion of each MVC when the participant was relaxed. Voluntary activation was estimated according to the following formula (Strojnik & Komi, 1998): VA (%) = $100 - [D \times (T_{SUP}/T_{MAX})/PT] \times 100$, where *D* is

the difference between the torque amplitude just before the superimposed twitch ($T_{SUP}$) and the peak torque amplitude recorded during the superimposed twitch, $T_{MAX}$ is the maximal torque amplitude recorded during the MVC, and PT is the peak torque amplitude of the resting potentiated twitch.

Data processing

*Maximal voluntary contractions*

Data from the five MVCs with an evoked M-wave (including concomitant resting potentiated twitch data) were averaged and used for analysis.

*Maximal torque and rate of torque development*

For all MVCs and resting potentiated twitches, torque onset was defined as the point on the torque-time curve where torque output exceeded baseline values by ≥ 1 % of the difference between baseline and peak torque amplitude. The following variables were analysed from the torque-time curve of each MVC: *1)* maximal voluntary torque output, defined as the greatest amplitude of the torque-time curve, excluding the point of stimulation ($T_{MAX,}$ Nm); *2)* normalised maximal rate of torque development ($RTD_{MAX}$), determined from the greatest average 10 ms slope of the torque-time curve (Δtorque/Δtime) throughout the first 500 ms of each MVC; and *3)* normalised average rate of torque development ($RTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms, 0-200 ms post torque onset. All rate dependent measures of voluntary torque production were normalised to $T_{MAX}$ of each analogous MVC

to observe changes in RTD independent of changes to maximal torque (Holtermann *et al.*, 2007).

*Electromyography*

sEMG onset was defined 70 ms before torque onset to account for the presence of electromechanical delay (Aagaard *et al.*, 2002a). During each MVC the following variables were identified from SOL and MG sEMG signals: 1) maximal sEMG activity ($SOL_{MAX}$ and $MG_{MAX}$; sEMG/M, %), calculated from the greatest average 250 ms period of activity (excluding superimposed stimulation) of the RMS signal throughout each MVC; 2) maximal rate of sEMG rise ($SOL_{RERmax}$ and $MG_{RERmax}$; sEMG/M, $\%.s^{-1}$), determined as the greatest average 10 ms slope ($\Delta$sEMG/$\Delta$time) of the RMS signal up to 200 ms post sEMG onset; 3) average rate of sEMG rise ($SOL_{RERave}$ and $MG_{RERave}$; sEMG/M, $\%.s^{-1}$) of the RMS signal calculated in time intervals from 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset; and 4) the maximal M-wave amplitude ($SOL_{Mmax}$ and $MG_{Mmax}$; mV), determined from the peak-to-peak amplitude of the raw sEMG signal post doublet stimulation. sEMG RER was observed up to a maximum of 75 ms post sEMG onset (instead of > 100 ms) as a decrease in RMS amplitude often occurred after this time.

*Resting twitch*

The following variables were analysed from the resting potentiated twitches: *1)* resting twitch peak torque (PT, Nm), defined as the greatest amplitude of the torque-time curve; *2)* absolute ($tRTD_{MAX}$, $Nm.s^{-1}$) and normalised resting twitch maximal rate of torque development, defined as the greatest average 10 ms slope of the ascending limb of the twitch torque-time

curve; *3)* absolute ($tRTD_{AVE}$, $Nm.s^{-1}$) and normalised resting twitch average rate of torque development during the time periods 0-25 ms, 0-50 ms, 0-75 ms post twitch torque onset; *4)* resting twitch time to peak torque (TPT, ms), defined as the time from twitch torque onset to PT; and *5)* resting twitch half relaxation time (½ RT, ms), defined as the time elapsed from PT to 50 % PT. $tRTD_{AVE}$ was observed up to a maximum of 75 ms post stimulation as reductions in twitch torque amplitude often occurred between 75 ms and 100 ms. All tRTD variables were normalised to the PT amplitude of each analogous resting potentiated twitch.

*Evoked potentials*

M-waves were measured as the peak-to-peak amplitude of the raw sEMG signal between 5 ms and 15 ms post stimulation, with the H-reflex (including HPAD, GPAD and recurrent inhibition test and condition reflexes) and V-waves observed between 25 ms and 50 ms post stimulation. The peak-to-peak amplitudes of H-reflex (including inhibition), M-wave and V-wave recordings were normalised to the concomitant peak-to-peak $M_{MAX}$ waveform prior to analysis ($H/M_{MAX}$, $M/M_{MAX}$ and $V/M_{MAX}$, respectively; %) to reduce inter-subject variability and control for the influence of contraction intensity on M-wave amplitude (Pensini & Martin, 2004).

For the curve fit analysis, H-reflex recruitment was normalised to the average $M_{MAX}$ (mV) value determined from the single stimulation sweep with the three largest peak-to-peak M-waves. The current values for all recruitment curves were normalised to the current at 50 % of $M_{MAX}$ to define the stimulus value used in the curve fit analysis and to allow recruitment curves to be compared at the same relative current intensities (Klimstra & Zehr, 2008; Vila-Cha *et al.*, 2012). Prior to data analysis, the ascending limb of the $H/M_{MAX}$ recruitment curve

was fit using a general least squares model predicted from a custom three parameter sigmoid function. This method has been proposed to be more reliable at approximating parameters of ascending H-reflex recruitment than other mathematical analysis techniques, described previously (Klimstra & Zehr, 2008). The maximal H-reflex amplitude, current intensity at 50 % of the $H_{MAX}$ value and the slope of the sigmoid function were input into the general least squares model and the sum of the squared variance between the observed and curve fit data was computed for the ascending limb of the H-reflex recruitment curves. Recruitment curves with an *r*-square > 0.90 were used for analysis (Vila-Cha *et al.*, 2012).

Ascending H-reflex recruitment parameters were predicted according to the following sigmoid function: $H(i) = H_{MAX}/1 + e^{m(i50 - i)}$, where H(*i*) is the H-reflex amplitude at a given current intensity (*i*); $H_{MAX}$ is the average $H/M_{MAX}$ value from the single stimulation sweep with the three largest H-reflexes, $\%M_{MAX}$; *m* is the slope of ascending H-reflex recruitment at 50 % of the $H_{MAX}$ value and *i*50 is the current intensity at 50 % of the $H_{MAX}$ value. The following parameters were identified from the curve fit analysis (Figure 26): *a*) the maximum H-reflex amplitude, defined as the average of the three largest H-reflex amplitudes of the sigmoid curve fit ($H_{MAX}$, $\%M_{MAX}$); *b*) half of the maximum H-reflex amplitude, defined as the value corresponding to half of the of the maximum H-reflex amplitude of the sigmoid curve fit (50 % $H_{MAX}$, $\%M_{MAX}$); *c*) the slope of the ascending limb of the H-reflex recruitment curve fit at 50 % $H_{MAX}$ ($H_{SLP}$, $mV.s^{-1}$); *d*) the current at the H-reflex threshold, defined as the value at which the linear fit of the general least squares model (developed using $H_{SLP}$ and *i* at 50 % $H_{MAX}$ values) intercepts the *x*-axis (*i* at $H_{THR}$, %*i* at $50\%M_{MAX}$); *e*) the current at 50 % $H_{MAX}$, produced as an output parameter (*i*50) from the sigmoid function (*i* at 50 % $H_{MAX}$, %*i* at $50\%M_{MAX}$); *f*) the current at $H_{MAX}$, defined as the value at which the linear fit of the general least squares model (developed using $H_{SLP}$ and *i* at 50 % $H_{MAX}$

values) intercepts $H_{MAX}$ (*i* at $H_{MAX}$, %*i* at 50%$M_{MAX}$) (Klimstra & Zehr, 2008). * *Note: A detailed description of the calculation steps can be found on the next page*. In addition to the standard fit ascending H-reflex recruitment curve, predicted PRE and POST training curves were calculated using the relative current intensities corresponding to PRE training ascending H-reflex recruitment values. This analysis method was used as it is thought to be more sensitive for detecting training induced changes in H-reflex recruitment (Dragert & Zehr, 2011). To differentiate the standard and predicted recruitment parameters, the predicted variables are described using '@' (Klimstra & Zehr, 2008). The following predicted parameters were analysed: @$H_{THR}$, @50 % $H_{MAX}$ and @$H_{MAX}$; %$M_{MAX}$.

* The following equations were used to calculate the parameters of interest from the ascending limb of the sigmoid fit.

Calculating $H_{SLP}$:

*Step 1:* Finding the slope at 50 % $H_{MAX}$ ($H_{SPL50}$)

$$H_{SLP50} = (H_2 - H_1)/(i_2 - i_1)$$

*Step 2:* Finding slope of the sigmoid function ($m$)

$$H_{SLP50} = m(H_{MAX})/4$$

$$m = (H_{SLP50} \times 4)/H_{MAX}$$

*Note:* equation also used to calculate '$m$' component of H($i$).

*Step 3:* Finding $H_{SLP}$

$$H_{SLP} = m(H_{MAX})/4$$

Calculating $i$ at $H_{THR}$:

*Step 1:* Finding the $y$-intercept

$y$-intercept = 50 % $H_{MAX}$ – ($H_{SLP}$ × $i$ at 50 % $H_{MAX}$)

*Note: i* at 50 % $H_{MAX}$ is the same as $i$50

*Step 2:* Finding $i$ at $H_{THR}$ ($x$-intercept)

$i$ at $H_{THR}$ = (-1 × $y$-intercept)/$H_{SLP}$

Calculating $i$ at $H_{MAX}$:

$i$ at $H_{MAX}$ = ($H_{MAX}$ – $y$-intercept)/$H_{SLP}$

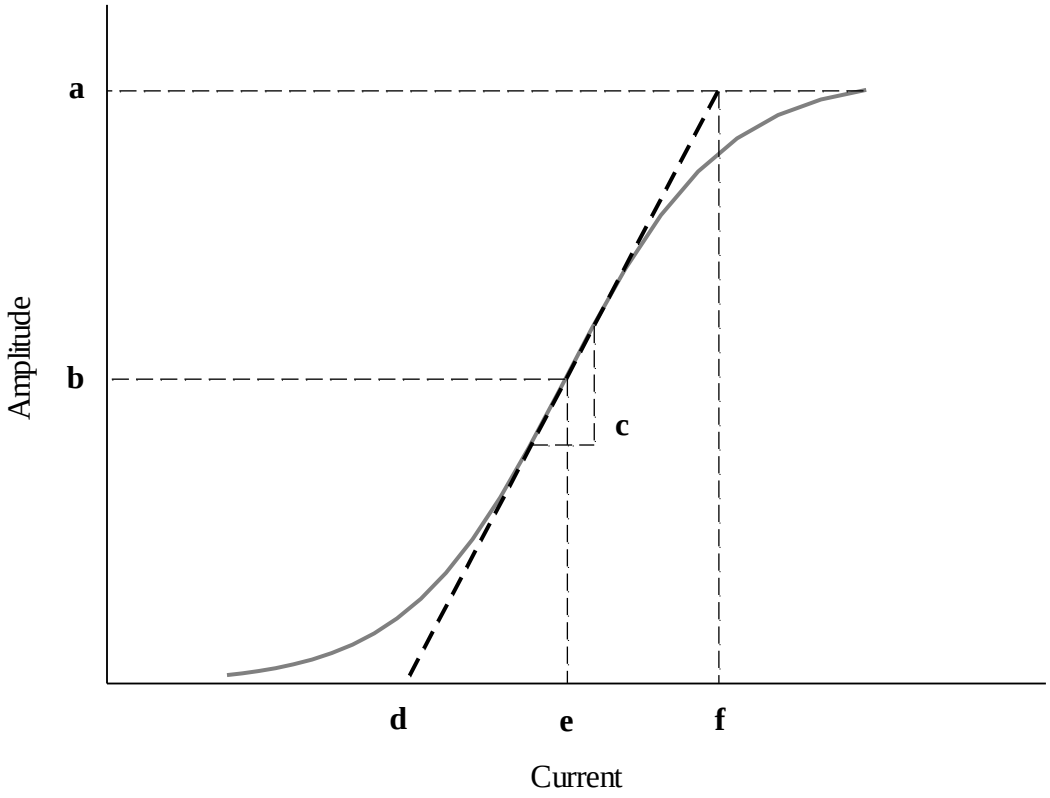


**Figure 26.** Predicted sigmoid function of ascending H-reflex recruitment (solid grey line) with linear fit of the general least squares model (bold dashed line). The recruitment parameters used for analysis were: the maximal H-reflex amplitude ($H_{MAX}$, *a*); the amplitude of the H-reflex at half of the maximal H-reflex amplitude (50 % $H_{MAX}$, *b*); the slope of the ascending limb of H-reflex recruitment curve at half of the maximal H-reflex amplitude ($H_{SLP}$, *c*); the current at H-reflex threshold (*i* at $H_{THR}$, *d*); the current at half of the maximal H-reflex amplitude (*i* at 50 % $H_{MAX}$, *e*); the current at the maximal H-reflex amplitude (*i* at $H_{MAX}$, *f*).

## Reliability

Reliability analyses were completed using data from the five plantar flexion MVCs completed in the T0 and T3 testing sessions. The mean within-day, within-subject coefficients of variation (%) were 5.4 ± 2.7 (range 2.2 to 11.9) for $T_{MAX}$, 44.8 ± 13.5 (range 22.6 to 73.7) for SOL $V/M_{MAX}$, 4.9 ± 2.3 (range 1.6 to 10.3) for PT, 15.1 ± 7.2 (range 2.0 to 28.7) for $SOL_{MAX}$, and 15.6 ± 7.5 (range 1.7 to 33.6) for $MG_{MAX}$. Data are means ± SD. The mean within-day, within-subject intra-class correlation coefficient (ICC, *r*) was 0.99 (95 % CI 0.99 to 1, $p < 0.001$) for $T_{MAX}$, 0.88 (95 % CI 0.78 to 0.94, $p < 0.001$) for SOL $V/M_{MAX}$, 0.99 (95 % CI 0.97 to 0.99, $p < 0.001$) for PT, 0.97 (95 % CI 0.93 to 0.99, $p < 0.001$) for $SOL_{MAX}$, and 0.96 (95 % CI 0.91 to 0.98, $p < 0.001$) for $MG_{MAX}$.

## Statistical analysis

All statistical analyses were completed using IBM SPSS Statistics version 22 (SPSS Inc., Chicago, IL). All data were normally distributed, determined from Kolmogorov-Smirnov normality testing. To examine initial differences in age, height, body mass and training experience between groups, independent samples t-tests were performed on baseline data. Data between groups was considered different in the presence of a significant *F* ratio. To identify group differences in body mass following training, the change in body mass from T0 to T3 was analysed with a univariate analysis of variance (ANOVA). Between group differences were considered significant if the 95 % CI's did not cross zero (Fisher *et al.*, 2015). Subsequently, *F* ratios were examined with post hoc, Bonferroni corrected paired sample t-tests performed when then the 95 % CI's did not cross zero. The change in seated calf raise training volume (from Week 1 to Week 2…, Week 8) and 10 RM (from T0 to T1,

T2 and T3) was analysed between groups with a repeated measures analysis of covariance (ANCOVA). Baseline data (from Week 1 and T0 for training volume and 10 RM, respectively) and training experience values were used as covariates to account for any influence of initial score variance on training outcomes (Mangine *et al.*, 2015). If Mauchly's test indicated a violation of sphericity in the ANCOVA, the Greenhouse-Geisser epsilon correction was used to adjust the degrees of freedom. In the event of a significant *F* ratio, post hoc comparisons were made using a Bonferroni adjustment. Changes in strength, explosive torque production and central and peripheral functioning in response to training were analysed using a univariate ANCOVA run from the T0 to T3 change in seated calf raise 1 RM and all dependent variables measured during MVC, resting twitch, H-reflex recruitment and spinal inhibition protocols. Baseline data (collected at T0) and training experience were used as covariates in the analysis. Main time effects were observed in the presence of a significant *F* ratio from Bonferroni corrected paired sample t-tests, performed when 95 % CI's did not cross zero. *F* ratios completed during all analyses were considered significant at $p < 0.05$. V-waves could not be detected in one participant from each group, therefore, their data was excluded from the V-wave analysis. The data are presented as means ± SD unless otherwise stated.

In addition to parametric testing, data was further analysed using effect sizes. Within group changes (from T0 to T3) were analysed using Cohen's *d*, where $d = 0.2$ is a small effect, $d = 0.5$ is a moderate effect and $d = 0.8$ is a large effect (Cohen, 1992). Between group differences over time (T0 to T3) were analysed using partial eta squared ($\eta_p^2$), where $\eta_p^2 = 0.01$ is a small effect, $\eta_p^2 = 0.059$ is a medium effect and $\eta_p^2 = 0.138$ is a large effect (Mangine *et al.*, 2015).

## RESULTS

### Body composition and training experience

Participant characteristics are presented in Table 13. No differences between failure and non-failure groups were observed at baseline for age ($p = 0.704$), height ($p = 0.794$), body mass ($p = 0.798$) or training experience ($p = 0.352$). No group by time interaction ($p = 0.948$, $\eta_p^2 = 0.000$) or main time effects ($p = 0.069$, $d = 0.096$) were observed for body mass at the completion of the training period.

**Table 13.** Participant characteristics and strength level during the training period

| | F ($n = 8$) | NF ($n = 8$) |
|---|---|---|
| Age, years | 21.8 ± 3.1 | 22.4 ± 3.4 |
| Height (cm) | 171.5 ± 6.1 | 170.3 ± 11.8 |
| Body mass (kg) | | |
| T0 | 75.7 ± 8.5 | 74.4 ± 10.7 |
| T3 | 76.6 ± 9.3 | 75.3 ± 9.8 |
| Training experience, years | 2.9 ± 0.8 | 3.4 ± 1.5 |
| Calf raise 1RM (kg) | | |
| T0 | 65.3 ± 6.3 | 66.9 ± 20.5 |
| T3 | 77.9 ± 8.5** | 81.5 ± 25.0** |
| Calf raise 10RM (kg) | | |
| T0 | 50.0 ± 6.4 | 53.8 ± 14.4 |
| T1 | 53.8 ± 6.0** | 58.8 ± 13.2** |
| T2 | 55.9 ± 6.9**^ | 61.3 ± 13.7**^ |
| T3 | 57.9 ± 7.2**^# | 62.2 ± 14.4**^# |

F failure group, NF non-failure group, T0 baseline measurement, T1 after three weeks of training, T2 after six weeks of training, T3 in week nine (conclusion of training period). Data are presented as mean ± SD

** $p < 0.01$ from T0

^ $p < 0.05$ from T1

# $p < 0.05$ from T2

Training volume

All participants completed 100% of the prescribed sessions over the eight week training period. No participants withdrew from the study. Average weekly seated calf raise training volumes are presented in Table 14. Absolute seated calf raise training volume significantly increased ($p$ = 0.001, $\eta_p^2$ = 0.242) during the training period. Post hoc analysis indicated an initial 6.2 % increase by Week 3 (mean increase of 218.0 ± 201.2 kg/week; 95 % CI = 108.4 to 327.5 kg/week; $p$ = 0.027; Table 14) that remained significant for the duration of the training period ($p$ < 0.05). No group by time interaction was observed between failure and non-failure groups ($p$ = 0.185, $\eta_p^2$ = 0.110). Similar main time effects ($p$ = 0.006, $\eta_p^2$ = 0.269) and an absence of group by time interactions ($p$ = 0.056, $\eta_p^2$ = 0.177) were observed when seated calf raise volume per week was normalised to body mass.

**Table 14.** Total average seated calf raise training volume per week for the duration of the eight week training period

| | F | NF |
|---|---|---|
| Volume (kg/week) | | |
| Week 1 | 3241.9 ± 434.2 | 3753.1 ± 1019.8 |
| Week 2 | 3320.6 ± 436.6 | 3800.0 ± 1005.3 |
| Week 3 | 3540.3 ± 471.2$^{*}$ | 3890.6 ± 984.6$^{*}$ |
| Week 4 | 3637.2 ± 382.7$^{**}$ | 4112.5 ± 926.0$^{**}$ |
| Week 5 | 3588.4 ± 433.5$^{*}$ | 4112.5 ± 926.0$^{*}$ |
| Week 6 | 3620.3 ± 443.3$^{**}$ | 4131.3 ± 918.5$^{**}$ |
| Week 7 | 3646.3 ± 454.5$^{**}$ | 4243.8 ± 896.0$^{**}$ |
| Week 8 | 3736.9 ± 445.4$^{**}$ | 4296.9 ± 963.8$^{**}$ |

Volume (kg) calculated by multiplying: sets × repetitions × load (kg), completed at the conclusion of each training session. F failure group, NF non-failure group. Data are presented as mean ± SD

$^{**}$ $p < 0.01$ from Wk 1

$^{*}$ $p < 0.05$ from Wk 1

*Note: Weekly training volume was significantly greater than preceding weeks' volume for many time points. These significance indicators are not shown here because the table would be too hard to follow.*

Maximal strength

Absolute seated calf raise 1 RM increased 20.5 % by the conclusion of the training period (mean increase of 13.6 ± 9.0 kg; 95 % CI = 8.2 to 19.0 kg; $p < 0.001$; Table 13). A similar increase was observed when seated calf raise 1 RM was normalised to body mass ($p < 0.001$; Figure 27). No group differences were observed for absolute ($p = 0.749$, $\eta_p^2 = 0.009$) or normalised ($p = 0.724$, $\eta_p^2 = 0.011$) measures of seated calf raise 1 RM. Absolute seated calf raise 10 RM significantly increased ($p = 0.037$, $\eta_p^2 = 0.286$). Post hoc analysis indicated an initial 8.4 % increase by T1 (mean increase of 4.4 ± 3.7 kg; 95 % CI = 2.5 to 6.2 kg; $p = 0.001$) that remained significant at T2 ($p = 0.002$) and T3 ($p = 0.001$). No group by time interaction was observed between failure and non-failure groups ($p = 0.404$, $\eta_p^2 = 0.065$). Similar main time effects ($p = 0.033$, $\eta_p^2 = 0.295$) and an absence of group by time interactions ($p = 0.300$, $\eta_p^2 = 0.092$) were observed for normalised seated calf raise 10 RM (Figure 27).

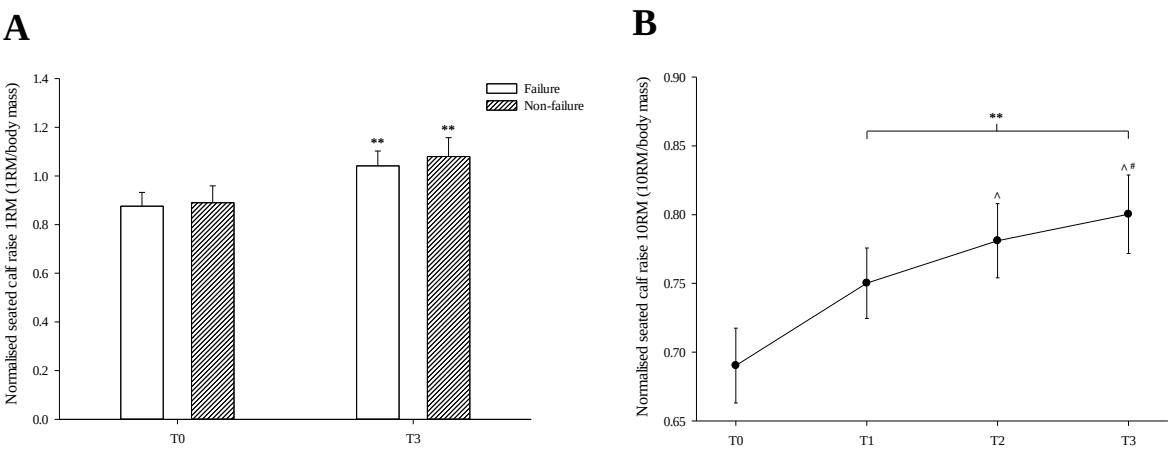


**Figure 27.** Seated calf raise 1 RM normalised to body mass (kg) at baseline (T0) and at the conclusion of the training period (T3) for failure and non-failure groups (*A*); seated calf raise 10 RM normalised to body mass at baseline (T0), after three weeks of training (T1), after six weeks of training (T2) and at the conclusion of the training period (T3) for the failure and non-failure groups (grand mean, *B*). $^{**}$ $p < 0.01$ from T0, $^{\wedge}$ $p < 0.05$ from T1, $^{\#}$ $p < 0.05$ from T2. Data are mean and SE.

Maximal torque and rate of torque development

Plantar flexor $T_{MAX}$ increased 15.1 % (mean increase 32.8 ± 26.1 Nm; 95 % CI = 20.2 to 45.4 Nm; $p < 0.001$; Figure 28) at the conclusion of the training period. No group by time interaction was observed for $T_{MAX}$. No main time effects or group by time interactions were observed for absolute or normalised measures of plantar flexor voluntary RTD (Table 15).

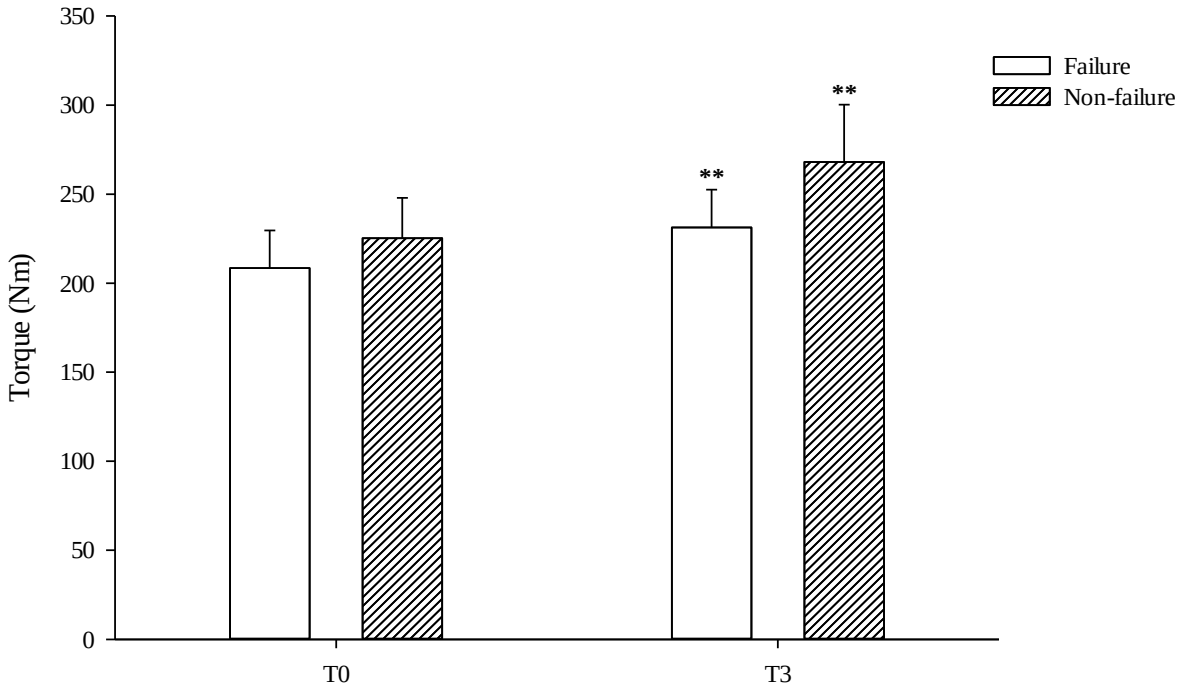


**Figure 28.** Maximal torque ($T_{MAX}$, Nm) measured during plantar flexor MVCs at baseline (T0) and at the conclusion of the training period (T3) for failure and non-failure groups. ** $p < 0.01$ from T0. Data are mean and SE.

**Table 15.** Maximal torque ($T_{MAX}$, Nm); and normalised maximal ($RTD_{MAX}$) and average rate of torque development measured during maximal voluntary plantar flexion contractions performed by both failure (F) and non-failure (NF) exercise groups at baseline (PRE; T0) and after the training period (POST; T3). $RTD_{AVE}$ data are presented as the average slope of the torque-time curve in time intervals of 0-25 ms, 0-50 ms, 0-75 ms, 0-100 ms and 0-200 ms post torque onset. All rate dependent measures of torque development are normalised to the corresponding MVCs' $T_{MAX}$.

| | | Group means | | Time by group interaction | | | Time effect | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PRE | POST | *F* | *p* value | $\eta_p^2$ | *p* value | *d* | 95% CI (pooled) | |
| | | | | | | | | | Lower | Upper |
| $T_{MAX}$ (Nm) | F | 208.5 ± 59.7 | 231.3 ± 60.0** | 2.222 | 0.162 | 0.156 | 0.000 | 0.474 | 20.201 | 45.388 |
| | NF | 225.3 ± 63.9 | 268.1 ± 91.1** | | | | | | | |
| RTD (Normalised to $T_{MAX}$) | | | | | | | | | | |
| MAX | F | 3.4 ± 0.8 | 3.3 ± 0.7 | 1.889 | 0.194 | 0.136 | 0.232 | -0.268 | -0.540 | 0.095 |
| | NF | 3.4 ± 1.0 | 3.0 ± 0.8 | | | | | | | |
| 0-25ms | F | 1.0 ± 0.4 | 0.9 ± 0.3 | 0.267 | 0.615 | 0.022 | 0.083 | -0.337 | -0.247 | 0.013 |
| | NF | 1.0 ± 0.4 | 0.9 ± 0.4 | | | | | | | |
| 0-50ms | F | 1.4 ± 0.6 | 1.2 ± 0.4 | 0.022 | 0.884 | 0.002 | 0.067 | -0.341 | -0.355 | 0.009 |
| | NF | 1.5 ± 0.6 | 1.3 ± 0.5 | | | | | | | |
| 0-75ms | F | 1.8 ± 0.7 | 1.6 ± 0.6 | 0.023 | 0.883 | 0.002 | 0.065 | -0.335 | -0.433 | 0.010 |
| | NF | 1.8 ± 0.7 | 1.6 ± 0.6 | | | | | | | |
| 0-100ms | F | 2.1 ± 0.8 | 1.9 ± 0.7 | 0.270 | 0.613 | 0.022 | 0.075 | -0.323 | -0.473 | 0.018 |
| | NF | 2.1 ± 0.8 | 1.9 ± 0.7 | | | | | | | |
| 0-200ms | F | 2.4 ± 0.7 | 2.3 ± 0.6 | 1.407 | 0.258 | 0.105 | 0.174 | -0.289 | -0.412 | 0.056 |
| | NF | 2.5 ± 0.7 | 2.2 ± 0.6 | | | | | | | |

Data are presented as mean ± SD
** $p < 0.01$ from PRE

Central adaptation

$SOL_{MAX}$ and $MG_{MAX}$ increased 13.9 % (mean increase 0.2 ± 0.3 %; 95 % CI = 0.0 to 0.4 %; $p$ = 0.012) and 19.1 % (mean increase 0.3 ± 0.4 %; 95 % CI = 0.1 to 0.5 %; $p$ = 0.012), respectively, at the conclusion of the training period (Figure 29). Collectively, no main time or group by time interactions were observed for sEMG rate of rise characteristics (Table 16). Central drive to the motor neuron pool, indicated by SOL and MG $V/M_{MAX}$ ratio and plantar flexor VA measurements did not experience main time or group by time interactions in response to training (Table 16). Similarly, spinal excitability, analysed using ascending $H/M_{MAX}$ recruitment did not change with training (Table 17). Additionally, no adaptations were observed for measures of SOL or MG pre-synaptic inhibition (Table 18).

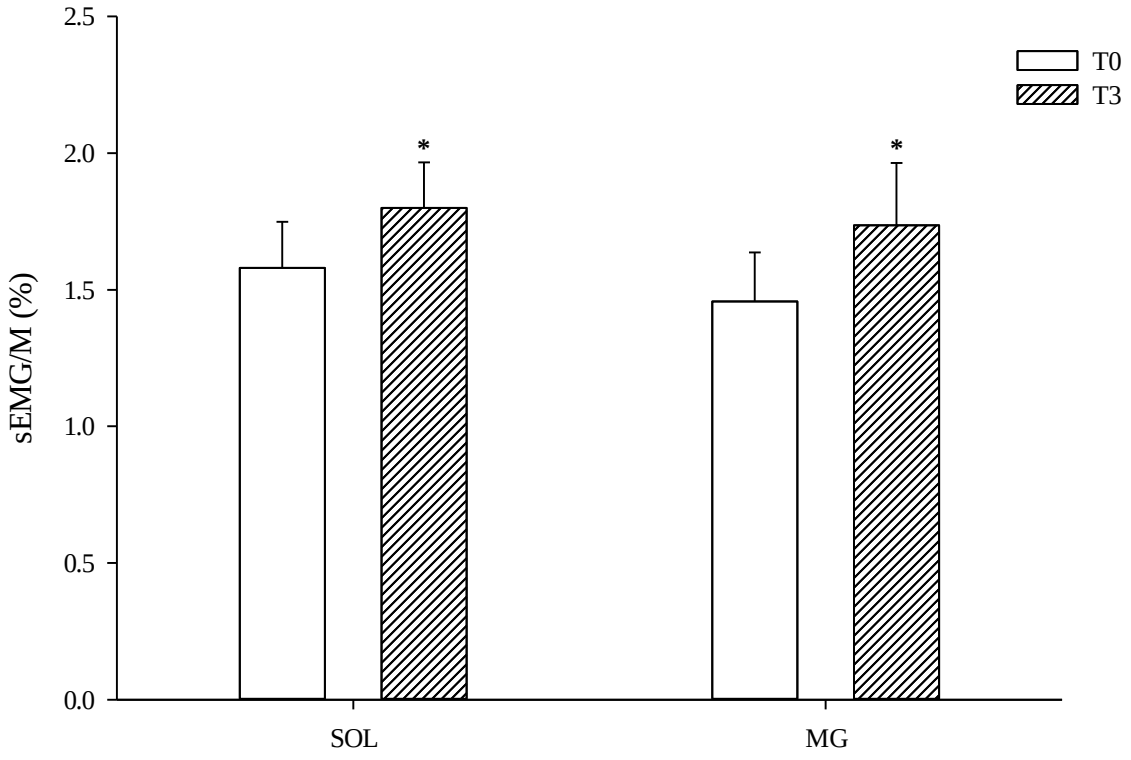


**Figure 29.** Maximal soleus ($SOL_{MAX}$) and medial gastrocnemius ($MG_{MAX}$) sEMG activity measured during maximal voluntary plantar flexion contractions performed at baseline (T0) and at the conclusion of the training period (T3). $SOL_{MAX}$ and $MG_{MAX}$ data are expressed as a percentage of the corresponding MVCs' SOL and MG maximum M-wave (sEMG/M, %), respectively. $^{*}$ $p < 0.05$ from T0. Data are grand mean and SE for failure and non-failure groups.

**Table 16.** Soleus and medial gastrocnemius maximal sEMG activity ($SOL_{MAX}$ and $MG_{MAX}$; sEMG/M, %), maximal rate of sEMG rise up to 200 ms post sEMG onset ($SOL_{RERmax}$ and $MG_{RERmax}$; sEMG/M, %.s$^{-1}$) and average rate of sEMG rise in time intervals 0-25 ms, 0-50 ms and 0-75 ms post sEMG onset ($SOL_{RERave}$ 0-25, $SOL_{RERave}$ 0-50, $SOL_{RERave}$ 0-75, $MG_{RERave}$ 0-25, $MG_{RERave}$ 0-50 and $MG_{RERave}$ 0-75; sEMG/M, %.s$^{-1}$). sEMG data are expressed as a percentage of the corresponding MVCs' SOL and MG maximum M-wave (sEMG/M, %), respectively. Soleus and medial gastrocnemius V-wave expressed as a percentage of the corresponding MVCs' SOL and MG maximum M-wave (SOL $V/M_{MAX}$ and MG $V/M_{MAX}$; %), respectively. Plantar flexor voluntary activation (VA, %). Data was recorded during maximal voluntary plantar flexion contractions performed by both failure (F) and non-failure (NF) exercise groups at baseline (PRE; T0) and after the training period (POST; T3).

| | | Group means | | Time by group interaction | | | Time effect | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PRE | POST | *F* | *p* value | $\eta_p^2$ | *p* value | *d* | 95% CI (pooled) | |
| | | | | | | | | | Lower | Upper |
| $SOL_{MAX}$ (%) | F | 1.8 ± 0.8 | 2.0 ± 0.7* | 0.205 | 0.659 | 0.017 | 0.012 | 0.328 | 0.046 | 0.394 |
| | NF | 1.4 ± 0.4 | 1.6 ± 0.6* | | | | | | | |
| $SOL_{RERmax}$ (%.s$^{-1}$) | F | 39.8 ± 24.3 | 41.6 ± 22.9 | 1.161 | 0.302 | 0.088 | 0.781 | -0.031 | -6.151 | 4.792 |
| | NF | 33.6 ± 21.0 | 30.4 ± 21.8 | | | | | | | |
| $SOL_{RERave}$ 0-25ms (%.s$^{-1}$) | F | 9.9 ± 7.8 | 8.1 ± 5.3 | 0.185 | 0.675 | 0.015 | 0.681 | -0.093 | -2.960 | 1.879 |
| | NF | 6.1 ± 4.2 | 6.8 ± 5.5 | | | | | | | |
| $SOL_{RERave}$ 0-50ms (%.s$^{-1}$) | F | 15.4 ± 13.9 | 14.5 ± 11.5 | 0.033 | 0.858 | 0.003 | 0.758 | -0.040 | -3.703 | 2.800 |
| | NF | 11.1 ± 8.9 | 11.2 ± 10.8 | | | | | | | |
| $SOL_{RERave}$ 0-75ms (%.s$^{-1}$) | F | 16.7 ± 13.5 | 16.0 ± 12.1 | 0.005 | 0.945 | 0.000 | 0.746 | -0.040 | -3.684 | 2.778 |
| | NF | 12.7 ± 9.5 | 12.5 ± 11.3 | | | | | | | |
| $MG_{MAX}$ (%) | F | 1.8 ± 0.8 | 2.2 ± 1.0* | 1.094 | 0.316 | 0.084 | 0.012 | 0.340 | 0.064 | 0.493 |
| | NF | 1.1 ± 0.5 | 1.3 ± 0.4* | | | | | | | |
| $MG_{RERmax}$ (%.s$^{-1}$) | F | 34.5 ± 19.9 | 37.7 ± 22.8 | 0.809 | 0.386 | 0.063 | 0.593 | 0.080 | -4.914 | 8.002 |
| | NF | 23.7 ± 15.9 | 23.6 ± 15.3 | | | | | | | |
| $MG_{RERave}$ 0-25ms (%.s$^{-1}$) | F | 11.9 ± 8.1 | 11.2 ± 10.8 | 1.065 | 0.323 | 0.081 | 0.794 | 0.035 | -2.193 | 2.762 |
| | NF | 5.2 ± 4.9 | 6.4 ± 5.9 | | | | | | | |
| $MG_{RERave}$ 0-50ms (%.s$^{-1}$) | F | 13.6 ± 9.0 | 13.8 ± 11.7 | 0.003 | 0.958 | 0.000 | 0.778 | 0.033 | -2.237 | 2.844 |
| | NF | 7.9 ± 7.7 | 8.2 ± 6.6 | | | | | | | |
| $MG_{RERave}$ 0-75ms (%.s$^{-1}$) | F | 13.0 ± 8.1 | 14.6 ± 12.1 | 1.764 | 0.209 | 0.128 | 0.999 | 0.000 | -3.176 | 3.172 |
| | NF | 9.4 ± 8.5 | 7.8 ± 5.4 | | | | | | | |
| SOL $V/M_{MAX}$ (%) | F | 21.1 ± 10.2 | 20.3 ± 13.7 | 0.327 | 0.580 | 0.032 | 0.799 | 0.044 | -3.860 | 4.763 |
| | NF | 9.2 ± 2.5 | 11.0 ± 4.7 | | | | | | | |
| MG $V/M_{MAX}$ (%) | F | 27.3 ± 14.6 | 27.1 ± 17.8 | 0.002 | 0.964 | 0.000 | 0.765 | 0.058 | -5.738 | 7.371 |
| | NF | 11.9 ± 6.5 | 13.8 ± 7.0 | | | | | | | |
| VA (%) | F | 94.2 ± 5.2 | 96.0 ± 5.6 | 0.575 | 0.463 | 0.046 | 0.271 | 0.187 | -0.830 | 2.964 |
| | NF | 92.7 ± 6.8 | 93.1 ± 5.3 | | | | | | | |

Data are presented as mean ± SD
* $p < 0.05$ from PRE

**Table 17.** Parameters processed from the ascending limb of soleus (SOL) and medial gastrocnemius (MG) H-reflex recruitment curves. Data are recorded at baseline (PRE; T0) and at the conclusion of the training period (POST; T3) in failure (F) and non-failure (NF) groups. The maximum H-reflex amplitude ($H_{MAX}$, $\%M_{MAX}$); the slope of the H-reflex recruitment curve fit at 50 % $H_{MAX}$ ($H_{SLP}$, $mV.s^{-1}$); the current (*i*) at H-reflex threshold (*i* at $H_{THR}$, %*i* at $50\%M_{MAX}$); the current at 50 % $H_{MAX}$ (*i* at 50 % $H_{MAX}$, %*i* at $50\%M_{MAX}$); the current at $H_{MAX}$ (*i* at $H_{MAX}$, %*i* at $50\%M_{MAX}$); the predicted current at H-reflex threshold ($i@H_{THR}$, $\%M_{MAX}$), the predicted current at 50 % $H_{MAX}$ ($i@50$ % $H_{MAX}$, $\%M_{MAX}$); and the predicted current at $H_{MAX}$ ($i@H_{MAX}$, $\%M_{MAX}$).

| | | Group means | | Time by group interaction | | | Time effect | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PRE | POST | *F* | *p* value | $\eta_p^2$ | *p* value | *d* | 95% CI (pooled) | |
| | | | | | | | | | Lower | Upper |
| SOL | | | | | | | | | | |
| $H_{MAX}$ ($\%M_{MAX}$) | F | 36.9 ± 12.1 | 39.3 ± 13.5 | 0.181 | 0.678 | 0.015 | 0.560 | 0.127 | -4.3 | 7.4 |
| | NF | 31.8 ± 9.5 | 32.4 ± 12.3 | | | | | | | |
| $H_{SLP}$ ($mV.s^{-1}$) | F | 1.6 ± 0.8 | 2.5 ± 1.8 | 0.685 | 0.424 | 0.054 | 0.140 | 0.404 | -0.3 | 1.3 |
| | NF | 1.8 ± 0.9 | 2.0 ± 1.6 | | | | | | | |
| *i* at $H_{THR}$ (%*i* at $50\%M_{MAX}$) | F | 39.8 ± 13.3 | 39.1 ± 16.5 | 0.021 | 0.888 | 0.002 | 0.716 | -0.052 | -5.3 | 3.8 |
| | NF | 34.7 ± 14.1 | 33.9 ± 11.4 | | | | | | | |
| *i* at 50 % $H_{MAX}$ (%*i* at $50\%M_{MAX}$) | F | 52.6 ± 13.4 | 49.4 ± 17.3 | 0.103 | 0.753 | 0.009 | 0.321 | -0.131 | -7.1 | 2.7 |
| | NF | 45.5 ± 19.6 | 44.4 ± 16.4 | | | | | | | |
| *i* at $H_{MAX}$ (%*i* at $50\%M_{MAX}$) | F | 65.4 ± 14.6 | 59.7 ± 19.0 | 0.378 | 0.550 | 0.031 | 0.191 | -0.180 | -9.6 | 2.3 |
| | NF | 56.4 ± 25.3 | 54.8 ± 21.7 | | | | | | | |
| $i@H_{THR}$ ($\%M_{MAX}$) | F | 18.7 ± 7.5 | 20.3 ± 6.9 | 0.262 | 0.618 | 0.021 | 0.542 | 0.104 | -2.1 | 3.6 |
| | NF | 17.3 ± 7.1 | 17.3 ± 8.8 | | | | | | | |
| $i@50$ % $H_{MAX}$ ($\%M_{MAX}$) | F | 26.6 ± 6.7 | 25.7 ± 8.0 | 0.264 | 0.617 | 0.021 | 0.653 | -0.058 | -3.3 | 2.2 |
| | NF | 22.8 ± 9.8 | 22.6 ± 11.9 | | | | | | | |
| $i@H_{MAX}$ ($\%M_{MAX}$) | F | 34.5 ± 8.4 | 31.0 ± 9.6 | 2.083 | 0.175 | 0.148 | 0.185 | -0.158 | -4.8 | 1.1 |
| | NF | 28.2 ± 12.6 | 27.9 ± 15.1 | | | | | | | |
| MG | | | | | | | | | | |
| $H_{MAX}$ ($\%M_{MAX}$) | F | 38.3 ± 18.4 | 40.6 ± 19.0 | 0.676 | 0.427 | 0.053 | 0.547 | 0.085 | -4.1 | 7.1 |
| | NF | 22.0 ± 12.2 | 22.6 ± 11.6 | | | | | | | |
| $H_{SLP}$ ($mV.s^{-1}$) | F | 1.4 ± 1.5 | 2.4 ± 1.6 | 2.304 | 0.155 | 0.161 | 0.128 | 0.303 | -0.1 | 1.1 |
| | NF | 1.7 ± 1.5 | 1.7 ± 1.7 | | | | | | | |
| *i* at $H_{THR}$ (%*i* at $50\%M_{MAX}$) | F | 62.9 ± 28.7 | 42.3 ± 18.3 | 0.312 | 0.587 | 0.025 | 0.274 | -0.363 | -17.3 | 1.6 |
| | NF | 38.2 ± 16.3 | 43.1 ± 15.3 | | | | | | | |
| *i* at 50 % $H_{MAX}$ (%*i* at $50\%M_{MAX}$) | F | 91.9 ± 50.0 | 54.9 ± 27.4 | 0.000 | 0.998 | 0.000 | 0.226 | -0.436 | -29.2 | -1.2 |
| | NF | 46.6 ± 19.9 | 53.3 ± 19.7 | | | | | | | |
| *i* at $H_{MAX}$ (%*i* at $50\%M_{MAX}$) | F | 116.1 ± 66.1 | 61.5 ± 25.7 | 0.103 | 0.754 | 0.008 | 0.150 | -0.523 | -37.9 | -8.2 |
| | NF | 55.0 ± 23.4 | 63.4 ± 25.1 | | | | | | | |
| $i@H_{THR}$ ($\%M_{MAX}$) | F | 33.5 ± 18.6 | 35.2 ± 20.0 | 0.081 | 0.781 | 0.007 | 0.463 | 0.062 | -2.3 | 4.3 |
| | NF | 19.0 ± 8.2 | 19.3 ± 9.9 | | | | | | | |
| $i@50$ % $H_{MAX}$ ($\%M_{MAX}$) | F | 46.0 ± 25.0 | 43.9 ± 24.5 | 0.233 | 0.638 | 0.019 | 0.555 | -0.039 | -4.1 | 2.4 |
| | NF | 23.2 ± 10.0 | 23.6 ± 11.8 | | | | | | | |
| $i@H_{MAX}$ ($\%M_{MAX}$) | F | 58.4 ± 32.7 | 52.6 ± 29.3 | 0.525 | 0.483 | 0.042 | 0.171 | -0.099 | -6.5 | 1.1 |
| | NF | 27.5 ± 11.7 | 27.9 ± 13.8 | | | | | | | |

Data are presented as mean ± SD

**Table 18.** Measures of soleus (SOL) and medial gastrocnemius (MG) pre-synaptic inhibition recorded at baseline (PRE; T0) and at the conclusion of the training period (POST; T3) in failure (F) and non-failure (NF) groups. Homosynaptic mediated post activation depression (HPAD, %) and gamma-aminobutyric-acid mediated primary afferent depolarisation (GPAD, %).

| | | Group means | | Time by group interaction | | | Time effect | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PRE | POST | *F* | *p* value | $\eta_p^2$ | *p* value | *d* | 95% CI (pooled) | |
| | | | | | | | | | Lower | Upper |
| $SOL_{HPAD}$ (%) | F | 52.8 ± 31.2 | 51.3 ± 30.5 | 0.020 | 0.890 | 0.002 | 0.705 | -0.098 | -17.064 | 11.389 |
| | NF | 60.1 ± 31.2 | 56.1 ± 23.2 | | | | | | | |
| $SOL_{GPAD}$ (%) | F | 18.2 ± 23.2 | 20.5 ± 17.7 | 0.031 | 0.864 | 0.003 | 0.496 | 0.248 | -5.983 | 14.528 |
| | NF | 13.2 ± 10.4 | 19.5 ± 17.4 | | | | | | | |
| $MG_{HPAD}$ (%) | F | 39.7 ± 26.4 | 28.6 ± 34.3 | 1.991 | 0.186 | 0.153 | 0.694 | -0.111 | -20.325 | 12.212 |
| | NF | 43.7 ± 32.0 | 47.1 ± 28.7 | | | | | | | |
| $MG_{GPAD}$ (%) | F | 13.5 ± 13.9 | 10.0 ± 10.9 | 1.574 | 0.234 | 0.116 | 0.751 | 0.112 | -5.820 | 8.615 |
| | NF | 10.2 ± 10.3 | 16.6 ± 14.7 | | | | | | | |

Data are presented as mean ± SD

Peripheral adaptation

A 9.1 % increase (mean increase 3.8 ± 4.1 Nm, 95 % CI = 1.9 to 5.7 Nm; $p = 0.002$; Figure 30; Table 19) in resting twitch peak torque (PT) was observed with training. A similar 9.2 % increase (mean increase 50.6 ± 67.0 $Nm.s^{-1}$; 95 % CI = 11.7 to 89.5 $Nm.s^{-1}$; $p = 0.009$; Figure 31) was observed for absolute $tRTD_{MAX}$. Main time effects also occurred for average absolute tRTD in time intervals 0-50 ms ($p = 0.018$) and 0-75 ms ($p = 0.005$) post twitch torque onset. No group by time interactions were observed for absolute measures of tRTD. No main time effects or group by time interactions were observed for measures of normalised tRTD, TPT, ½ RT, or for SOL and MG maximal M-wave amplitudes (Table 19).

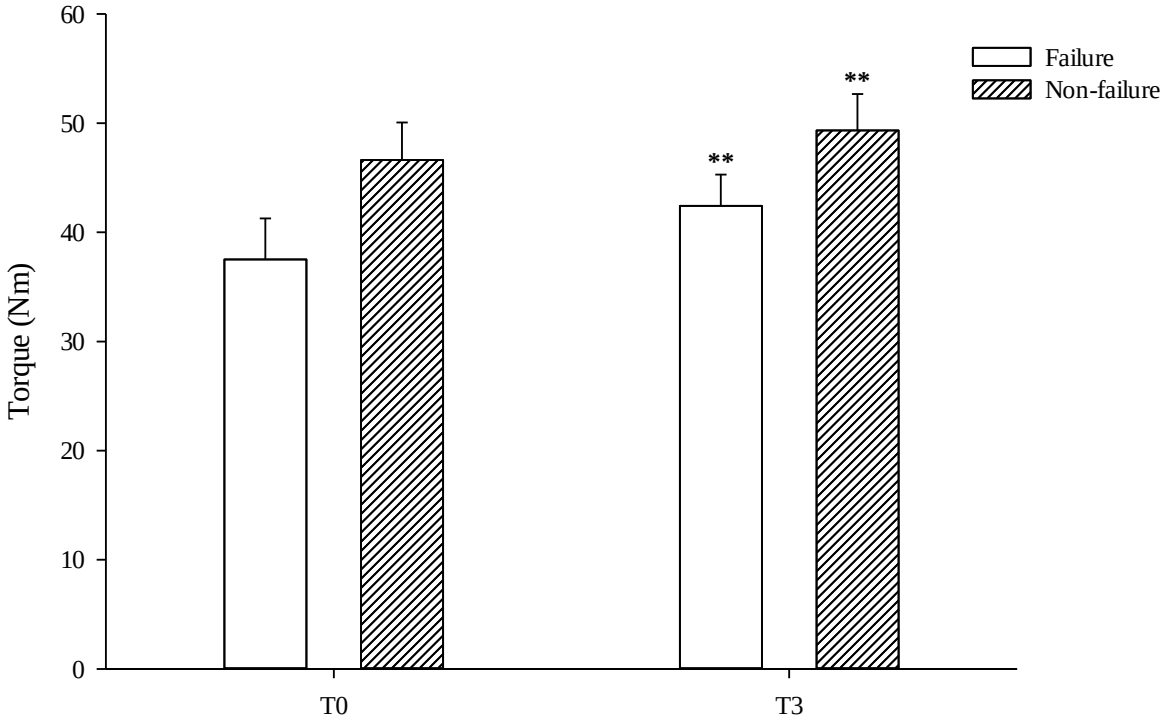


**Figure 30.** Resting twitch peak torque (PT, Nm) measured immediately following plantar flexor MVCs at baseline (T0) and at the conclusion of the training period (T3) for failure and non-failure groups. ** $p < 0.01$ from T0. Data are mean and SE.

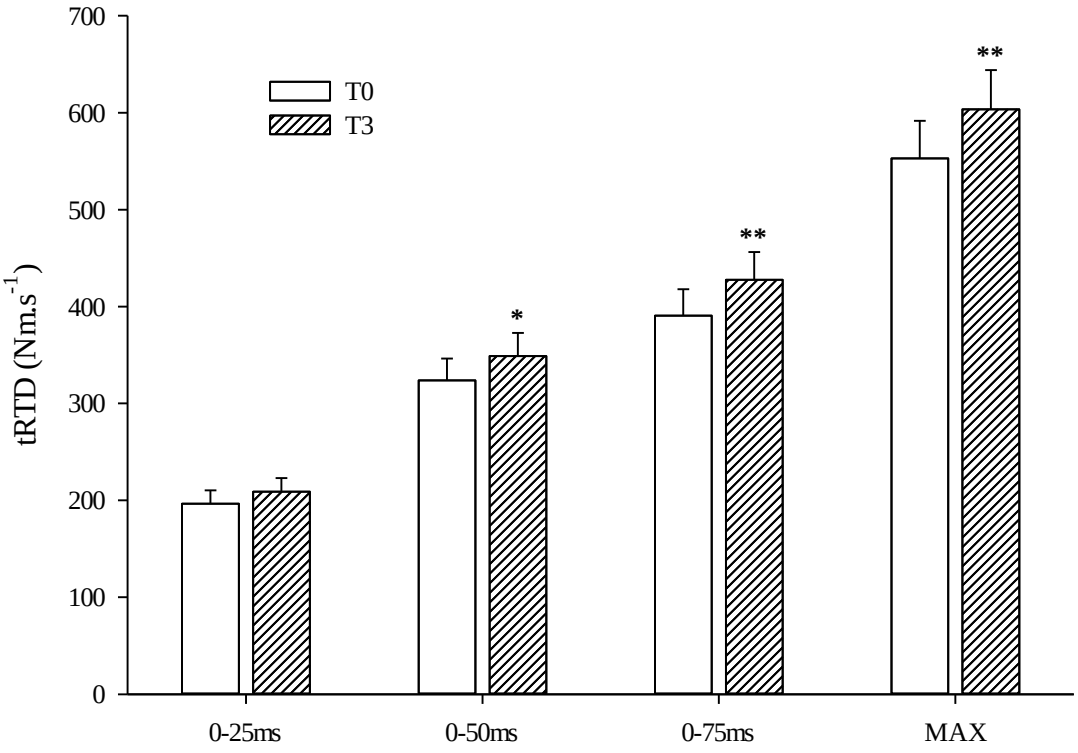


**Figure 31.** Absolute measures of resting twitch rate of torque development observed at baseline (T0) and at the conclusion of the training period (T3). Maximal ($tRTD_{MAX}$) and average twitch rate of torque development ($tRTD_{AVE}$) during the time periods 0-25 ms, 0-50 ms, 0-75 ms post twitch torque onset ($Nm.s^{-1}$). $^{**}$ $p < 0.01$ from T0, $^{*}$ $p < 0.05$ from T0. Data are grand mean and SE of failure and non-failure groups.

**Table 19.** Plantar flexor resting twitch parameters and maximal M-waves recorded from failure (F) and non-failure (NF) groups at baseline (PRE; T0) and after the training period (POST; T3). Resting twitch peak torque (PT, Nm); resting twitch normalised maximal rate of torque development ($tRTD_{MAX}$); resting twitch normalised average rate of torque development presented in time intervals of 0-25 ms, 0-50 ms and 0-75 ms post twitch torque onset ($tRTD_{AVE}$); resting twitch time to peak torque (TPT, ms); resting twitch half-relaxation time (½ RT, ms); soleus and medial gastrocnemius maximum M-wave ($SOL_{Mmax}$, $MG_{Mmax}$; mV). All rate dependent measures of twitch torque development are normalised to the corresponding PT.

| | | Group means | | Time by group interaction | | | Time effect | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PRE | POST | *F* | *p* value | $\eta_p^2$ | *p* value | *d* | 95% CI (pooled) | |
| | | | | | | | | | Lower | Upper |
| PT (Nm) | F | 37.5 ± 10.6 | 42.4 ± 8.1** | 0.024 | 0.880 | 0.002 | 0.002 | 0.378 | 1.905 | 5.720 |
| | NF | 46.6 ± 9.7 | 49.3 ± 9.4** | | | | | | | |
| tRTD (Normalised to PT) | | | | | | | | | | |
| MAX | F | 13.5 ± 1.8 | 13.1 ± 1.0 | 0.100 | 0.758 | 0.008 | 0.525 | -0.125 | -0.583 | 0.262 |
| | NF | 12.7 ± 1.1 | 12.8 ± 1.1 | | | | | | | |
| 0-25ms | F | 4.6 ± 0.5 | 4.6 ± 0.3 | 0.630 | 0.443 | 0.050 | 0.818 | 0.040 | -0.152 | 0.189 |
| | NF | 4.4 ± 0.5 | 4.5 ± 0.6 | | | | | | | |
| 0-50ms | F | 7.7 ± 0.8 | 7.6 ± 0.6 | 0.441 | 0.519 | 0.035 | 0.958 | 0.008 | -0.250 | 0.263 |
| | NF | 7.3 ± 0.8 | 7.4 ± 0.9 | | | | | | | |
| 0-75ms | F | 9.6 ± 1.1 | 9.3 ± 0.7 | 0.161 | 0.696 | 0.013 | 0.523 | -0.110 | -0.368 | 0.171 |
| | NF | 9.0 ± 0.9 | 9.0 ± 0.8 | | | | | | | |
| TPT (ms) | F | 129.3 ± 27.6 | 124.8 ± 14.5 | 1.222 | 0.291 | 0.092 | 0.572 | -0.119 | -7.562 | 3.192 |
| | NF | 131.6 ± 16.2 | 131.9 ± 13.0 | | | | | | | |
| ½ RT (ms) | F | 105.4 ± 41.0 | 93.1 ± 20.0 | 3.227 | 0.098 | 0.212 | 0.537 | -0.117 | -15.460 | 7.741 |
| | NF | 111.4 ± 29.6 | 116.0 ± 37.6 | | | | | | | |
| $SOL_{Mmax}$ (mV) | F | 10.0 ± 2.4 | 11.2 ± 4.3 | 0.735 | 0.408 | 0.058 | 0.459 | 0.146 | -1.032 | 2.022 |
| | NF | 9.1 ± 3.4 | 8.9 ± 3.3 | | | | | | | |
| $MG_{Mmax}$ (mV) | F | 10.3 ± 4.0 | 10.2 ± 4.2 | 0.506 | 0.490 | 0.040 | 0.374 | 0.152 | -0.498 | 1.691 |
| | NF | 10.9 ± 4.6 | 12.2 ± 3.0 | | | | | | | |

Data are presented as mean ± SD
** $p < 0.01$ from PRE

## DISCUSSION

The main finding of this investigation supports the hypothesis that moderate to high intensity failure and non-failure exercise would evoke a similar increase in muscular strength in trained individuals following short term training. The observed increase in muscle activation and resting twitch rate and amplitude characteristics in both failure and non-failure groups suggest that the increase in maximal plantar flexor strength was a product of improved central and peripheral functioning. However, the maintenance spinal and supraspinal neural input to the α-motor neuron pool appears to have demonstrated that improvements in muscular strength were not mediated by an increase in motor unit output to the muscle. Interestingly, the hypothesised greater increase in muscular power in the non-failure group was not corroborated by the present findings, despite the observed improvements in muscular strength.

The pooled 20.5 % and 15.1 % group increase in absolute measures of seated calf raise 1 RM and plantar flexor $T_{MAX}$ at T3, respectively, demonstrate that failure and non-failure methods of exercise prescription are equally effective at increasing maximal plantar flexor strength in trained individuals when prescribed with moderate to high intensity loads. These findings are in agreement with Izquierdo and colleagues, who observed an analogous increase in upper and lower body 1 RM strength between failure and non-failure exercise of matched volume and intensity following a 16 week intervention in trained individuals (Izquierdo *et al.*, 2006). However, the similar increase in maximal strength observed with failure and non-failure exercise contrasts previous short term (6-8 week) training accounts in athletes with moderate traditional upper body dynamic strength training experience (Drinkwater *et al.*, 2005; Izquierdo-Gabarren *et al.*, 2010). Whilst Izquierdo *et al.* (2010) found the magnitude of upper

body strength improvement to be greater when not training to failure, Drinkwater *et el.* (2005) observed larger strength improvements when exercise was performed to failure. Current disagreement in trained populations may be explained by differences in study design (i.e. training volume not being matched between repetition failure and non-failure groups), the level of training experience, muscle groups tested, the method of determination and definition of maximal strength, and whether strength and endurance training were used concurrently throughout the study period. The results of this study also support the recent trend in untrained populations (Folland *et al.*, 2002; Fisher *et al.*, 2015) and individuals with minimal training experience (Sampson & Groeller, 2015), that a similar increase in maximal strength occurs whether training to failure or not to failure. Therefore, the majority of available literature may be interpreted to reflect similar training outcomes when training to failure or not to failure, regardless of prior resistance training experience.

The results of this investigation extend previous findings in trained populations, demonstrating that improvements in muscular strength following short term failure and non-failure training were attributed to an approximate 9 % increase in maximal and rate dependent measures of plantar flexor resting twitch torque production and an approximate 16.5 % increase in SOL and MG maximal sEMG/M activity. The increase in PT and absolute $tRTD_{MAX}$, $tRTD_{AVE}$ 0-50 ms and $tRTD_{AVE}$ 0-75 ms suggest that the increase in plantar flexor strength following failure and non-failure exercise was partially accounted for by improved mechanics within the muscular contractile apparatus, likely related to improved sarcoplasmic and tubular $Ca^{2+}$ kinetics such as an increased release rate of $Ca^{2+}$ within the sarcomere (Ortenblad *et al.*, 2000) and potentially, an increase in the sensitivity (binding) of the contractile/regulatory proteins troponin and tropomyosin to $Ca^{2+}$. Furthermore, the increase in PT with training could be interpreted to reflect stronger binding of active cross bridges within

the myofibril and/or an increase in the number of available cross bridges. However, the present investigation is limited in its interpretation of the peripheral mechanisms that increased muscular strength. It is possible that improved mechanics within the muscle's contractile apparatus and subsequent increases in muscular strength were simply the result of myofibrillar hypertrophy. Therefore, further investigation should observe changes in muscle fibre pennation angle and cross sectional area to determine whether peripheral adaptations were produced from muscular hypertrophy.

The pooled 13.9 % and 19.1 % increase in $SOL_{MAX}$ and $MG_{MAX}$ sEMG/M activity, respectively, supports the hypothesis that central factors would contribute to improvements in muscular strength following short term failure and non-failure training. Gross neural activation, indicated by increased maximal sEMG amplitude has long been reported as a likely proponent of muscular strength improvement following periods of strength training in trained populations (Hakkinen *et al.*, 1985a; Hakkinen *et al.*, 1985b; Hakkinen & Komi, 1986). In contrast, muscular strength improvements in trained individuals are not always observed with concomitant increases in maximal sEMG amplitude (Baker *et al.*, 1994; Ahtiainen *et al.*, 2005; Marshall *et al.*, 2011). While differences in study design and methodology may contribute to the disagreement between these investigations, many factors (*for review see* Farina *et al.* (2004)), particularly those related to the interpretation of sEMG signal amplitude (i.e. action potential propagation, cancellation and detection) (Yue *et al.*, 1995; Farina *et al.*, 2010) may limit conclusions of neural changes drawn from muscle activation recordings using sEMG. As such, the increase in maximal sEMG amplitude observed in this investigation should be interpreted with caution.

Despite an increase in sEMG/M ratio neither plantar flexor VA, $V/M_{MAX}$, or measures of H-reflex recruitment or inhibition changed in response to eight weeks of moderate to high intensity failure or non-failure exercise training. The increase in sEMG/M amplitude could have been the product of a number of confounding factors related to sEMG amplitude interpretation, mentioned previously. Hence, it is possible that this study did not find evidence to support the hypothesis that spinal and supraspinal adaptations would contribute to improvements in muscular strength. Moreover, the stasis of plantar flexor VA, $V/M_{MAX}$ and Ia afferent excitability, despite increases in maximal sEMG/M ratio observed here suggests previous conclusions of 'central adaptations' observed as increased sEMG amplitudes following a period of strength training in trained populations are misguided. However, the present results still support the belief that trained individuals are able to recruit their available motor units more effectively than untrained individuals (del Olmo *et al.*, 2006). The maintenance of pre-training levels of plantar flexor VA and $V/M_{MAX}$ observed at the conclusion of the training period could suggest that cortical drive is already maximised in individuals with extensive resistance training experience given improvements in plantar flexor VA (Pensini *et al.*, 2002; Nordlund Ekblom, 2010) and V-wave (Sale *et al.*, 1983a; Aagaard *et al.*, 2002b; Del Balso & Cafarelli, 2007; Duclay *et al.*, 2008; Fimland *et al.*, 2009a; Fimland *et al.*, 2009b; Nordlund Ekblom, 2010; Vila-Cha *et al.*, 2012) are commonly observed in untrained populations following strength training. Additionally, Lee and colleagues have demonstrated that improvements in wrist extensor strength of untrained persons are the product of increased cortical and/or corticospinal output, measured using transcranial magnetic stimulation (TMS) (Lee *et al.*, 2009). Therefore, the TMS technique could be used in the future to investigate whether an increase in muscle activity and strength with failure and non-failure exercise in trained individuals is the result of increased neural output proximal to spinal α-motor neurons. It is also possible that moderate to high intensity

failure and non-failure exercise did not facilitate supraspinal adaptations in this study, the eight week training period was not long enough for trained individuals to experience significant adaptations within the motor cortex and/or the increase in strength may have resulted from enhanced cortical drive to muscle synergists not observed in this study.

For the first time, this investigation has demonstrated that plantar flexor Ia afferent excitability does not change following training in a resistance trained population. The maintenance of $H/M_{MAX}$ (Lagerquist *et al.*, 2006; Del Balso & Cafarelli, 2007; Duclay *et al.*, 2008; Fimland *et al.*, 2009a; Nordlund Ekblom, 2010) and measures of ascending H-reflex recruitment (Vila-Cha *et al.*, 2012) at the conclusion of the training period are representative of most strength training literature (< 8 weeks duration) conducted in untrained plantar flexors. Previous studies have reported greater $H/M_{MAX}$ (Maffiuletti *et al.*, 2001) and analogous tendon tap reflex amplitudes (Kyröläinen & Komi, 1994) in endurance than power trained athletes. Therefore, it is possible that the type of training employed in this investigation was not of an ideal nature to stimulate improvements in spinal functioning. Furthermore, it has also been suggested that H-reflex modulation is specific to the testing procedure (Zehr, 2002) and, as such, adaptations to H-reflex recruitment may not have been detected as the isometric testing procedure was not specific to the dynamic training task. Alternatively, similar to supraspinal factors, an increase in spinal neural input to the motor unit pool may not be as prevalent in trained individuals as significant spinal adaptations, observed as an increase in $H/M_{MAX}$ and GPAD, have already occurred with training (Nielsen *et al.*, 1993). Interestingly, small to moderate effect sizes without concomitant significant *p* values, indicated a general trend of improvement for ascending MG H-reflex recruitment in this study (Table 17). While potentially highlighting the need for a larger sample size, the recruitment curve mapping procedure was specific to the SOL muscle. Therefore, the

findings of this study should reflect more closely the adaptations within the SOL muscle and not those of synergistic agonists such as the MG. Additionally, the absence of training induced changes in plantar flexor HPAD and GPAD appears to suggest that changes in pre-synaptic inhibition at the spinal level did not contribute to the increase in muscular strength observed in this study.

The results of the present study demonstrate that moderate to high intensity failure and non-failure exercise neither improves nor dampens voluntary explosive torque production in trained individuals. Therefore, no evidence was found to support the hypothesis that short term non-failure resistance exercise training would produce a greater improvement in muscular power than a similar failure based exercise program. This finding contrasts the significantly larger increase in lower body concentric power output observed in trained individuals following a period of non-failure exercise training lasting 16 weeks (Izquierdo *et al.*, 2006). Despite similarly equating exercise volume and intensity between failure and non-failure groups, a number of differences in study design may provide a possible explanation for the disagreement between this investigation and the work of Izquierdo *et al.* (2006). Firstly, participants in this study were instructed to complete exercise repetitions in a controlled manner (2:1 sec, eccentric to concentric contraction ratio) without any emphasis on explosive movement. Conversely, those trained by Izquierdo and colleagues contracted as fast as possible throughout the concentric phase of muscular contraction (Izquierdo *et al.*, 2006), an exercise method that has previously been demonstrated to induce significant increases in explosive force production with training (Van Cutsem *et al.*, 1998). Furthermore, the absence of any observable change in explosive force production following training in the present investigation may be attributed to lack of emphasis and intent of participants to contract explosively (Behm & Sale, 1993a). Secondly, in the study by Izquierdo *et al.* (2006),

the authors determined muscular power output using velocity and displacement of a weighted barbell lifted explosively through a concentric range of motion (Izquierdo *et al.*, 2006), in contrast to the maximal isometric contractions completed in this investigation. Lastly, the final five weeks of their 16 week training period included a variety of low intensity ballistic movements designed to stimulate a 'peaking' effect (Izquierdo *et al.*, 2006). This type of low intensity ballistic exercise has previously been used to increase muscular power in trained individuals when traditional multi-set moderate to high intensity strength training has failed (Hakkinen & Komi, 1986). As such, the lack of change in explosive torque production despite an increase in maximal strength in this study may simply be the result of the velocity of muscular contractions performed during training. Given spinal (Holtermann *et al.*, 2007) and supraspinal (Johnson *et al.*, 2014) processes have demonstrated a significant correlation to RTD in untrained populations, further research should look to clarify whether central functioning contributed to the lack of change in explosive torque production observed here and if ballistic strength training is a more effective technique for the development of muscular strength and power in trained populations.

It must be acknowledged that a number of potential limitations exist in the present research. This investigation was specifically designed to explore the central and peripheral adaptations that occur when exercise volume, intensity and duration are equated between failure and non-failure exercise methods. Because strength improvements may occur within an eight week timeframe, 10 RM testing in weeks three and six was a necessary requirement of study design to determine appropriate training progression. Consequently, participants in the non-failure group completed a maximum of 2-4 repetitions to failure over the course of the training period. Given this is a relatively low number of repetitions compared to the total number accumulated over the duration of the training period it would not be expected to confound the

results. Measures of spinal pre- and post-synaptic neural inhibition were completed in this study to observe a number of spinal processes understood to facilitate muscular strength development with training. The data from recurrent homonymous and heteronymous spinal reflex inhibition testing was omitted from this thesis as sEMG trace recordings did not produce consistent conditioned H′ reflex amplitudes within participants or between groups. Previous research has documented greater recurrent inhibition in strength/power trained individuals (Earles *et al.*, 2002) and in males (Johnson *et al.*, 2012). Given these population demographics are mostly similar to the participants of this investigation, the inability to detect recurrent inhibition may simply be a function of the sampled population. While speculative, it may therefore be inferred that recurrent inhibition is maximally increased with resistance exercise training. If however, the inability to detect recurrent inhibition was the result of measurement error, the lack of change in all other measures of spinal and supraspinal functioning likely suggests that modifications of post-synaptic inhibition contributing to increases in muscular strength would be negligible. Lastly, paired pre- and post-synaptic inhibition stimulus trains are typically completed with concomitant singular stimulations that evoke an H-reflex used in the normalisation process of the conditioned reflex (Knikou, 2008). While the use of this single stimulation may help account for possible variations in Ia afferent excitability over time throughout the testing session, this study normalised all conditioned reflex responses to the average $H_1$ reflex response garnered from HPAD testing. For reasons relating to participant time constraints and comfort, this method of normalisation was deemed a suitable alternative to assess inhibition at a spinal level.

In conclusion, moderate to high intensity failure and non-failure exercise is equally effective for increasing plantar flexor strength in trained individuals. This investigation has demonstrated for the first time that short term failure and non-failure training does not appear

to produce adaptations within spinal or supraspinal pathways in trained individuals. Rather, improvements in maximal strength are more likely to result from improved functionality of the muscular contractile apparatus observed as increases in PT and tRTD. Furthermore, the lack of change in explosive torque production suggests muscular strength and power adaptations occur independently of one another when moderate to high intensity exercise is performed to failure or not to failure. Although this may be a function of the velocity of contractions completed during training, continued research should look to clarify whether a combination of controlled and explosive failure and/or non-failure exercise is more conducive to muscular strength and power development.

# CHAPTER 6

*General Discussion*

## SUMMARY OF FINDINGS

Study 1 (Chapter 3) observed a greater reduction in maximal torque output when an acute bout of moderate to high intensity isometric knee extension exercise was performed to failure than when not performed to failure. Given that resting twitch rate, amplitude and temporal characteristics were impaired in response to exercise in the absence of a downregulation of central drive to the motor unit pool, it was concluded that mechanisms of peripheral fatigue were responsible for the greater reduction in muscular strength following failure based exercise. Study 2 (Chapter 4) expanded upon Study 1 to an application of dynamic moderate to high intensity failure and non-failure exercise of the plantar flexors. Despite sharing many similarities with Study 1, Study 2 observed comparable declines in maximal torque output between failure and non-failure modalities that likely resulted from a significant downregulation of central input to the plantar flexor motor unit pool and not from impaired muscular contractile functioning. Study 3 (Chapter 5) was then designed to observe the outcome of short term prescription of failure and non-failure based exercise on measurements of muscular strength and power. Specifically, plantar flexor strength, but not power, increased in both the failure and non-failure group following the eight week training period. Despite the observed increase in muscle activation, the lack of change in spinal and supraspinal input to the motor unit pool likely demonstrated that improvements in plantar flexor strength were mediated by functional adaptations intrinsic to the muscle.

## THE DISAGREEMENT BETWEEN STUDY 1 AND STUDY 2

Some support was found for the hypotheses tested within the series of acute investigations presented in this thesis. A single bout of moderate to high intensity failure based exercise promoted a greater reduction in muscular strength than a similar bout of non-failure exercise in trained individuals. However, this finding was only observed following isometric contractions of the knee extensors and not with dynamic exercise of the plantar flexors (Study 1 and Study 2, respectively). The hypotheses for Studies 1 and 2 were based on previous findings that demonstrated that central and peripheral fatigue mediated reductions in muscular strength following dynamic failure based exercise of the elbow flexors (Behm *et al.*, 2002). The authors observed reductions in voluntary activation (VA) regardless of whether repetitions were performed to failure with a high (5 RM (repetition maximum)) or low (20 RM) exercise intensity, although the decline in resting twitch peak torque (PT) was dependent on the exercise volume (Behm *et al.*, 2002). In this thesis, acute reductions in muscular strength following failure and non-failure exercise were not mediated by concurrent impairments in central and peripheral functioning and central mechanisms did not facilitate larger reductions in muscular strength and power following failure based exercise. Further discussion is required to address why a reduction in VA and muscle activity was only observed following dynamic failure and non-failure exercise of the plantar flexors (Study 2), whereas excitation-contraction coupling was only impaired following isometric exercise of the knee extensors.

Muscle groups tested

The results from Study 1 and Study 2 demonstrate that moderate to high intensity failure and non-failure exercise promote significant acute reductions in knee extensor and plantar flexor strength in trained individuals. The reduction in resting twitch peak torque (PT) and rate of torque development (tRTD) and a prolongation of twitch time to peak torque (TPT) and half-relaxation time (½ RT) likely indicates that impairment of processes related to muscular excitation-contraction coupling facilitated the reduction in knee extensor strength following failure and non-failure exercise. Conversely, a decrease motor unit output observed as a reduction in VA and muscle activity was primarily responsible for the decline in muscular strength observed in the plantar flexors given the observed increase in tRTD and decrease in TPT and ½ RT indicated a potentiation of contractile functioning with failure and non-failure exercise. These findings support and extend previous comparisons of failure based exercise in the knee extensors and plantar flexors (Bigland-Ritchie *et al.*, 1986; Behm & St-Pierre, 1997).

Bigland-Ritchie *et al.* (1986) observed reductions in muscular strength following a continuous series of six second, submaximal (50 % MVC) isometric contractions performed to failure in the knee extensors and plantar flexors. The authors reported no change in VA following exercise in the knee extensors, although observed a reduction in VA and muscle activity in the plantar flexors (Bigland-Ritchie *et al.*, 1986). Therefore, the results of the present investigation support these findings and extend them to an understanding of the concurrent peripheral fatigue incurred from a bout of failure exercise in the knee extensors and plantar flexors. In a later study by Behm and colleagues, participants completed a series of ten second, submaximal isometric contractions of the knee extensors (25 % and 50 %

MVC) and plantar flexors (50 % and 75 % MVC) until the desired force output in each protocol could not be maintained (Behm & St-Pierre, 1997). Supporting the findings from the acute investigations presented in this thesis, the authors observed no change in knee extensor muscle activity and a prolongation of TPT following moderate intensity exercise. Similarly, plantar flexor muscle activity was reduced and PT was potentiated with exercise (Behm & St-Pierre, 1997). Therefore, the present literature suggests that plantar flexor strength is primarily mediated by central factors, whereas peripheral mechanisms account for reductions in strength in the knee extensors, a conclusion that is supported by differences in the motor unit composition of these two muscle groups.

Motor units typically innervate muscle fibres with relatively homogenous contractile properties. Muscle fibres are broadly classified as being either slow contracting, fatigue resistant (type I) or fast contracting, fast fatigable (type II) (Burke *et al.*, 1973). The velocity of muscular contraction is determined by the presence of myosin isozymes that have either high (type II) or low (type I) ATPase activity within the myofibril (Close, 1965; Barany, 1967). The knee extensors are considered to have a relatively high type II fibre distribution (approximately 50-70 %) compared to the plantar flexors, and in particular, the soleus muscle (approximately 13 %) (Johnson *et al.*, 1973) which is the primary agonist during plantar flexion contraction when the knee is in 90 degrees (º) of flexion. Therefore, impaired functioning within the muscle contractile apparatus would be expected to be greater in the knee extensors compared to the plantar flexors. Additionally, Vittasalo *et al.* (1981) has suggested that the ability of a muscle to relax is dependent on the distribution of type II fibres, which was further demonstrated in Study 1 by the prolongation of ½ RT following failure based exercise of the knee extensors.

Mode of contraction and task specificity

The differences in muscular strength and power observed between Study 1 and Study 2 and the mechanisms that promoted these differences may also be a function of the type of contraction performed during the exercise task and testing procedure. Although an acute bout of failure and non-failure exercise was performed in Studies 1 and 2, the exercise stimulus in the respective investigations consisted of a series of either isometric or dynamic contractions. A primary difference between the two investigations was that significant central impairment (indicated by reductions in VA and muscle activity) was observed following the dynamic exercise performed in Study 2, despite no change observed in these measures following isometric exercise in Study 1. This finding supports the work of Tax *et al.* (1989), who demonstrated that central factors, observed as a greater increase in motor unit recruitment threshold and firing frequency, are more likely to influence dynamic force production during dynamic compared to isometric muscular contraction. Furthermore, Jessop *et al.* (2013) observed differences in central functioning between isometric (5 sets × 20 repetitions, five second isometric hold) and dynamic (5 sets × 50 repetitions) body weight plantar flexion and dorsiflexion contractions. At the conclusion of the plantar flexion exercise session, the authors reported that neural inhibition at the spinal level remained unaffected with isometric contractions although was disinhibited with dynamic contractions (Jessop *et al.*, 2013). Conversely, spinal functioning was disinhibited with isometric and was not affected with dynamic dorsiflexion contractions (Jessop *et al.*, 2013). Hence, central functioning was affected by both the type of contractions performed during exercise and also the muscle groups observed, which may also explain the difference in central fatigue observed between muscle groups in Study 1 and Study 2.

It is also thought that the type of muscular contraction used to measure acute changes in strength should be specific to the exercise task. As an individual becomes accustomed to a particular movement with exercise they exhibit greater transfer of muscular strength when tested with a similar, compared to a dissimilar, mode of muscular contraction (Rasch & Morehouse, 1957). Known as the principle of task specificity, this concept is understood to be an important determinant of muscular strength and power development following a period of training (Sale & MacDougall, 1981; Rutherford & Jones, 1986; Sale, 1987) and is also believed to predict training outcomes following a period of failure and non-failure training (Rooney *et al.*, 1994). Therefore, the disagreement between the acute investigations presented here may be partially attributed to the fact that Study 1 was completed with an isometric task and testing procedure, whereas muscular strength and power were determined isometrically following a dynamic exercise task in Study 2.

Previous literature has demonstrated that acute changes in muscular strength and power are dependent on whether the exercise task is of an isometric or dynamic nature. Following a bout of isometric exercise of the knee extensors, Schmitz and colleagues observed a significantly greater reduction in maximal isometric strength compared to maximal dynamic power output (Schmitz *et al.*, 2002). Furthermore, maximal dynamic power output declined more than maximal isometric strength at the conclusion of a dynamic knee extension exercise session (Schmitz *et al.*, 2002), therefore demonstrating that fatigue was specific to the type of muscular contraction performed during exercise.

Whilst muscular strength and power testing was conducted at the neutral muscle length of 90º of knee extension and plantar flexion in Study 1 and Study 2, respectively, the dynamic exercise task in Study 2 was completed throughout a full range of motion (ROM, i.e. plantar

flexor muscle length changed approximately 50º-70º during concentric and eccentric phases of contraction). It is thought that the level of central drive to the motor unit pool (Babault *et al.*, 2003; Beltman *et al.*, 2004) and/or the contractile properties of a muscle (Gandevia & McKenzie, 1988; Newman *et al.*, 2003) determine the force a muscle is capable of producing for a given length (i.e. joint angle). Morel and colleagues observed central and peripheral fatigue following a series of maximal isometric and dynamic contractions of the knee extensors (Morel *et al.*, 2015). Using a maximal isometric MVC as a measure of muscular strength, the authors reported a significantly greater reduction in maximal torque output following dynamic compared to isometric exercise that was likely the result of impaired intrinsic contractile functioning indicated by a reduction in resting twitch PT (Morel *et al.*, 2015). However, reductions in VA and muscle activity were primarily responsible for the decline in strength observed following isometric exercise. Extending these findings, Beltman *et al.* (2004) demonstrated that voluntary neural drive is reduced when a muscle maximally lengthens compared to when maximally shortened or contracted isometrically. Furthermore, when a muscle is relaxed and passively contracted through a ROM, the magnitude of the peripheral response to contraction is greater with lengthening compared to concentric and isometric contraction modes (Beltman *et al.*, 2004). Lastly, Babault *et al.* (2003) measured central and peripheral fatigue mechanisms across an array of knee extensor muscle lengths when participants contracted isometrically and dynamically. The results indicated that neural drive during isometric and dynamic contractions is dependent on muscle length, whereas resting twitch PT and TPT responses are dependent on contraction type regardless of muscle length (Babault *et al.*, 2003). Therefore, observing changes in muscular strength and the mechanisms that promote these changes at a single muscle length may not provide a valid indication of fatigue incurred throughout the entire shortening and lengthening phases of dynamic contraction. Additionally, given the exercise task and testing procedure in Study 1

were completed at the same muscle length, comparing findings to those of Study 2 in which the task and testing procedure were completed at different muscle lengths, is also problematic.

Study 1 investigated acute changes in muscular strength and power in response to an isometric failure and non-failure exercise task and testing procedure. The nature of this protocol provided a relatively controlled set-up for inducing fatigue and at the same time, enabled rapid determination of the fatigue produced from a bout of failure and non-failure exercise. However, given the contractions performed during the exercise task were of an isometric and not dynamic nature, the protocol design was not ecologically valid to many real world training and competitive environments. Hence, Study 2 was designed to address this concern as well as to examine changes in muscular strength and power in a muscle group with a different motor unit distribution. Study 1 and Study 2 were both designed in a way that enabled exercise volume and total session duration to be matched between failure and non-failure protocols. These studies were also similar in that they were completed with loads corresponding to either 80 % of maximal isometric or dynamic force output. Therefore, it seems likely that the acute differences in muscular strength and power as well as the difference in fatigue mechanisms that promoted these changes were at least partially attributed to the physiological properties of the muscle groups tested and the mode of muscular contraction performed during the exercise task and testing procedures.

In support of the hypothesis, muscular strength increased similarly in trained individuals between failure and non-failure exercise modalities at the conclusion of the eight week training period. This result is in agreement with findings from which this hypothesis was based (Izquierdo *et al.*, 2006), although contrasts other reports in trained populations (Drinkwater *et al.*, 2005; Izquierdo-Gabarren *et al.*, 2010). Furthermore, the maintenance of muscular power output after the intervention contrasts the hypothesised greater increase in muscular power following non-failure based training. As discussed in Chapter 5, this finding was likely a result of a number of factors including, but not limited to, the velocity of contractions performed during training, the method used to determine muscular power and the use of low intensity ballistic exercise designed to stimulate a 'peaking' effect by the end of training (Izquierdo *et al.*, 2006). Additionally, the increase in muscle activity suggests central factors facilitated improvements in muscular strength with short term failure and non-failure training, yet the lack of change in spinal and supraspinal functioning does not support this finding nor the hypothesis that improvements in muscular strength following short term failure and non-failure training would result from an increase in spinal and supraspinal input to the motor unit pool. Importantly, the collective results of this thesis have demonstrated that moderate to high intensity failure and non-failure methods of resistance exercise prescription stimulate an acute reduction in muscular strength which appears to be required for the development of muscular strength in trained populations following short term training. However, this thesis found some evidence to suggest that the mechanisms that stimulated acute reductions in muscular strength with failure and non-failure based exercise do not necessarily predict muscular strength adaptation.

The studies presented in this thesis constitute the first body of research to observe acute and chronic changes in muscular strength following failure and non-failure exercise in trained populations. To date, one study has observed acute and chronic changes in muscular strength with failure and non-failure based exercise, although this investigation was conducted using untrained participants (Rooney *et al.*, 1994). The authors reported that failure and non-failure exercise of the elbow flexors produced acute reductions in muscular strength, which declined significantly more following failure based exercise. Improvements in muscular strength were subsequently observed with both exercise modalities following a six week training period, with a greater increase in strength observed in the participants that trained to failure (Rooney *et al.*, 1994). The similar acute reduction and chronic improvement in plantar flexor strength between failure and non-failure modalities in Studies 2 and 3, respectively, contrasts the findings of this previous investigation. However, muscle fibre type distribution is understood to be vastly different between the plantar flexors (in particular soleus) and the elbow flexors but relatively similar between the elbow flexors and knee extensors (Johnson *et al.*, 1973). Given the results of Study 1 in the knee extensors more closely resemble the acute findings of Rooney *et al.* (1994), further investigation is required to determine whether failure based exercise will in fact facilitate greater improvements in muscular strength compared to non-failure exercise in a muscle group with a high percentage of type II fibres in trained individuals, or if the disagreement between investigations is potentially related to the level of training experience of the sampled population.

Acute and chronic changes in muscular power have previously been observed following failure and non-failure exercise in trained populations. Drinkwater and colleagues reported a significantly greater reduction in maximal bench press power output following failure compared to non-failure exercise that subsequently resulted in a larger increase in muscular

power in participants engaged in failure based training (Drinkwater *et al.*, 2005). These findings contrast those of the acute investigations presented in this thesis in which maximal power was maintained in the knee extensors (Study 1) and increased similarly in the plantar flexors (Study 2) following a bout of failure and non-failure exercise in trained individuals. Furthermore, a short period of failure and non-failure training of the plantar flexors did not produce any significant improvements in muscular power output regardless of the prescription modality. As mentioned previously in this thesis, multiple factors, such as differences in the training experience of the sample population, the muscle groups tested, and the method used to measure and calculate muscular power could have contributed to the disagreement in findings compared to those of Drinkwater *et al.* (2005). Furthermore, the performance of concurrent strength and endurance training and participants in the failure group rarely completing exercise sets to failure may have confounded the results reported by the authors. Additionally, the authors did not examine the factors that likely facilitated greater improvements in muscular power output following failure based training.

The studies presented in this thesis are the first series of investigations to observe whether muscular power is mediated by changes central and/or peripheral functioning following failure and non-failure exercise. Whilst central and peripheral fatigue mechanisms did not appear to influence muscular power output in Study 1, Study 2 demonstrated that a potentiation of intrinsic contractile functioning likely facilitated muscular power output in the plantar flexors with failure and non-failure exercise. However, despite an improvement in intrinsic contractile processes at the conclusion of the training period, neither failure nor non-failure exercise was observed to increase plantar flexor power with training (Study 3). Therefore, the mechanisms specific to plantar flexor power production in an acute setting did not facilitate adaptations with training. Given a reduction in VA and muscle activity was

observed with fatiguing exercise of the plantar flexors, and spinal and supraspinal measures of plantar flexor neural adaptation did not change with training, it is likely that an increase in motor unit output did not contribute to the improvement in muscular strength at the conclusion of the training period. Rather, the acute potentiation of muscular excitation-contraction coupling processes appeared more vital to the development of muscular strength following short term failure and non-failure training in trained individuals.

# CHAPTER 7

*General Conclusion*

## SUMMARY

The aim of the studies that comprise this thesis was to examine acute and chronic changes in muscular strength and power, and the mechanisms that promote these changes following failure and non-failure based exercise in trained individuals. The results of the body of work presented here have demonstrated that both failure and non-failure based exercise evoke an increase in muscular fatigue acutely, which for the most part, was observed to promote a similar acute reduction in muscular strength between modalities. The acute increase in muscular fatigue likely facilitated the similar improvements in muscular strength observed with failure and non-failure exercise following short term training in trained populations. However, the central and peripheral fatigue mechanisms that mediated acute reductions in muscular strength following failure and non-failure exercise did not appear to have any relevance for predicting the training outcome.

## ORIGINALITY OF RESEARCH

This was the first series of investigations to examine acute and chronic changes in muscular strength and power following a comparison of failure and non-failure exercise in trained populations. Furthermore, this research has effectively controlled differences in exercise volume and total session duration that have previously fuelled disagreements within the failure versus non-failure exercise field. The work presented in this thesis is also the first to extensively examine the central and peripheral mechanisms responsible for acute and chronic changes in muscular strength and power with failure and non-failure exercise. Additionally, this research has improved the relatively poor understanding of the mechanisms that facilitate

and/or impair muscular force production following a single bout of resistance exercise and at the conclusion of short term training in trained individuals.

The results of the first two investigations demonstrated that acute changes in muscular strength and power as well as the mechanisms responsible for these changes appear to be dependent on the mode of muscular contraction and the muscle group fatigued during a bout of resistance exercise, and not just whether exercise is performed to failure or not to failure. Failure exercise, through significant peripheral impairment, was more effective at stimulating acute reductions in muscular strength with isometric exercise of the knee extensors in trained individuals. On the other hand, central mechanisms likely mediated a similar reduction in muscular strength with dynamic failure and non-failure exercise of the plantar flexors, despite peripheral factors facilitating muscular power output acutely in this muscle group. However, even though failure and non-failure exercise stimulated an acute and subsequent chronic improvement in muscular excitation-contraction coupling, this mechanism did not facilitate muscular power development with training. Instead, the work presented in this thesis has demonstrated similar improvements in muscular strength following short term failure and non-failure training in trained individuals, which appear to result from peripheral mechanistic adaptation of processes related to muscular excitation-contraction coupling and not from central adaptations associated with an increase in motor unit output.

## PRACTICAL APPLICATIONS / LIMITATIONS / FUTURE DIRECTIONS

When performed with equated exercise volume and session duration, failure and non-failure exercise modalities are equally effective at increasing muscular strength in trained individuals. Additionally, improvements in central functioning could not be attained with short term failure and non-failure exercise in trained individuals. Therefore, exercise prescription in trained populations should focus on the development of muscular strength by stimulating improvements in peripheral functioning.

The results presented here suggest that there is no need to perform resistance exercise to failure when the goal of a period of training is to improve muscular strength, provided that non-failure based exercise is completed with a similar total session volume and inter-set recovery period duration. Therefore, non-failure exercise may be prescribed by practitioners as an efficacious, time effective and less stressful (Fisher *et al.*, 2015) alternative to failure based programming that can be used to add variety to training and improve exercise adherence without a loss of muscular strength.

As mentioned previously, acute reductions in muscular strength and the mechanisms that mediated these reductions were different between the knee extensors and plantar flexors. This thesis only observed adaptations from failure and non-failure exercise within the plantar flexor muscle group. Given peripheral mechanisms were found to facilitate strength improvement in the plantar flexors, and the knee extensors are composed of a relatively greater percentage of type II fibres (Johnson *et al.*, 1973), future research should also examine whether adaptations produced from failure and non-failure exercise are different in the knee extensors. Furthermore, this may enhance the ecological validity of the research

presented in this thesis given the plantar flexors generally function as a synergistic muscle group to many larger compound movement actions typically used during training and competition that activate type II muscle fibres.

The reduction in VA and muscle activity in Study 2 also demonstrated that central processes likely facilitated reductions in muscular strength with failure and non-failure exercise. However, neither VA nor sEMG can differentiate between the spinal and supraspinal components of central fatigue. Because spinal (i.e. H-reflex recruitment and inhibition) and supraspinal (i.e. V-wave) input to the α-motor neurons remained unaffected with training, an extension of the investigations in this thesis would be to quantify drive from the upper motor neurons using motor cortex stimulation techniques, such as transcranial magnetic stimulation. This may also help to explain why increases in muscle activity were observed with training, despite no change in VA or V-wave amplitude. Future research should also examine changes within single motor units to help clarify changes to maximal and not just rate dependent measures of motor unit recruitment.

The results of the studies presented in this thesis have demonstrated that changes in muscular strength with failure and non-failure exercise are largely dependent on processes within the muscular contractile apparatus. Observing the torque-time characteristics of a potentiated twitch evoked at rest can only provide an estimation of the mechanisms involved in excitation-contraction coupling. It is therefore important to directly examine the changes myofibrillar $Ca^{2+}$ kinetics that may occur in response to exercise. Although speculative, the improvement in contractile functioning with training may also suggest that muscular hypertrophy has contributed to the observed increases in muscular strength. Therefore, future investigations should consider the use of muscle imaging techniques such as ultrasound and

magnetic resonance imaging to explore whether increases in muscular strength with failure and non-failure exercise are the result of muscular hypertrophy.

In conclusion, trained individuals do not need to perform resistance exercise to failure to facilitate improvements in muscular strength following short term training. The growing trend within the research field that has compared failure and non-failure training now seems to suggest that both modalities are equally effective at stimulating improvements in muscular strength. Additionally, muscular fatigue in trained individuals seems to be exercise and muscle group specific. Finally, the investigations within this thesis have challenged the traditional belief that trained individuals need to maximise central fatigue to optimise improvements in muscular strength. It appears that the increase in central fatigue that impaired plantar flexor strength acutely did not have any relevance for predicting strength adaptations with training.

# REFERENCES


Aagaard, P., Andersen, J. L., Dyhre-Poulsen, P., Leffers, A. M., Wagner, A., Peter Magnusson, S., Halkjær-Kristensen, J., & Simonsen, E. B. (2001). A mechanism for increased contractile strength of human pennate muscle in response to strength training: Changes in muscle architecture. *Journal of Physiology, 534*(2), 613-623.

Aagaard, P., Simonsen, E. B., Andersen, J. L., Magnusson, P., & Dyhre-Poulsen, P. (2002a). Increased rate of force development and neural drive of human skeletal muscle following resistance training. *Journal of Applied Physiology, 93*(4), 1318-1326.

Aagaard, P., Simonsen, E. B., Andersen, J. L., Magnusson, P., & Dyhre-Poulsen, P. (2002b). Neural adaptation to resistance training: Changes in evoked V-wave and H-reflex responses. *Journal of Applied Physiology, 92*(6), 2309-2318.

Ahtiainen, J. P., & Häkkinen, K. (2009). Strength athletes are capable to produce greater muscle activation and neural fatigue during high-intensity resistance exercise than nonathletes. *Journal of Strength and Conditioning Research, 23*(4), 1129-1134.

Ahtiainen, J. P., Pakarinen, A., Alen, M., Kraemer, W. J., & Häkkinen, K. (2003). Muscle hypertrophy, hormonal adaptations and strength development during strength training in strength-trained and untrained men. *European Journal of Applied Physiology, 89*(6), 555-563.

Ahtiainen, J. P., Pakarinen, A., Alen, M., Kraemer, W. J., & Häkkinen, K. (2005). Short vs. long rest period between the sets in hypertrophic resistance training: Influence on muscle strength, size, and hormonal adaptations in trained men. *Journal of Strength and Conditioning Research, 19*(3), 572-582.

Allen, D. G., Lamb, G. D., & Westerblad, H. (2008). Skeletal muscle fatigue: Cellular mechanisms. *Physiological Reviews, 88*(1), 287-332.

Babault, N., Pousson, M., Michaut, A., & Van Hoecke, J. (2003). Effect of quadriceps femoris muscle length on neural activation during isometric and concentric contractions. *Journal of Applied Physiology, 94*(3), 983-990.

Baker, D., Wilson, G., & Carlyon, R. (1994). Periodization: The effect on strength of manipulating volume and intensity. *Journal of Strength and Conditioning Research, 8*(4), 235-242.

Barany, M. (1967). ATPase activity of myosin correlated with speed of muscle shortening. *Journal of General Physiology, 50*(6), Suppl:197-218.

Baudry, S., Maerz, A. H., Gould, J. R., & Enoka, R. M. (2011). Task- and time-dependent modulation of ia presynaptic inhibition during fatiguing contractions performed by humans. *Journal of Neurophysiology, 106*(1), 265-273.

Beck, S., Taube, W., Gruber, M., Amtage, F., Gollhofer, A., & Schubert, M. (2007). Task-specific changes in motor evoked potentials of lower limb muscles after different training interventions. *Brain Research, 1179*(1), 51-60.

Behm, D. G. (1995). Neuromuscular implications and applications of resistance training. *Journal of Strength and Conditioning Research, 9*(4), 264-274.

Behm, D. G. (2009). The interpolated twitch technique may be valid and reliable but limited [1]. *Journal of Applied Physiology, 107*(1), 362.

Behm, D. G., Reardon, G., Fitzgerald, J., & Drinkwater, E. (2002). The effect of 5, 10, and 20 repetition maximums on the recovery of voluntary and evoked contractile properties. *Journal of Strength and Conditioning Research, 16*(2), 209-218.

Behm, D. G., & Sale, D. G. (1993a). Intended rather than actual movement velocity determines velocity-specific training response. *Journal of Applied Physiology, 74*(1), 359-368.

Behm, D. G., & Sale, D. G. (1993b). Velocity Specificity of Resistance Training. *Sports Medicine: Evaluations of Research in Exercise Science and Sports Medicine, 15*(6), 374-388.

Behm, D. G., & St-Pierre, D. M. M. (1997). Effects of fatigue duration and muscle type on voluntary and evoked contractile properties. *Journal of Applied Physiology, 82*(5), 1654-1661.

Behm, D. G., & St-Pierre, D. M. M. (1998). Fatigue mechanisms in trained and untrained plantar flexors. *Journal of Strength and Conditioning Research, 12*(3), 166-172.

Behm, D. G., St-Pierre, D. M. M., & Perez, D. (1996). Muscle inactivation: Assessment of interpolated twitch technique. *Journal of Applied Physiology, 81*(5), 2267-2273.

Beltman, J. G. M., Sargeant, A. J., Van Mechelen, W., & De Haan, A. (2004). Voluntary activation level and muscle fiber recruitment of human quadriceps during lengthening contractions. *Journal of Applied Physiology, 97*(2), 619-626.

Benson, C., Docherty, D., & Brandenburg, J. (2006). Acute neuromuscular responses to resistance training performed at different loads. *Journal of Science and Medicine in Sport, 9*(1-2), 135-142.

Bigland-Ritchie, B., Furbush, F., & Woods, J. J. (1986). Fatigue of intermittent submaximal voluntary contractions: Central and peripheral factors. *Journal of Applied Physiology, 61*(2), 421-429.

Bigland-Ritchie, B., & Woods, J. J. (1984). Changes in muscle contractile properties and neural control during human muscular fatigue. *Muscle and Nerve, 7*(9), 691-699.

Bigland Ritchie, B., Jones, D. A., Hosking, G. P., & Edwards, R. H. T. (1978). Central and peripheral fatigue in sustained maximum voluntary contractions of human quadriceps muscle. *Clinical Science and Molecular Medicine, 54*(6), 609-614.

Brinkworth, R. S. A., Tuncer, M., Tucker, K. J., Jaberzadeh, S., & Türker, K. S. (2007). Standardization of H-reflex analyses. *Journal of Neuroscience Methods, 162*(1-2), 1-7.

Buchthal, F., Dahl, K., & Rosenfalck, P. (1973). Rise time of the spike potential in fast and slowly contracting muscle of man. *Acta Physiologica Scandinavica, 87*(2), 261-269.

Buckthorpe, M., Pain, M. T. G., & Folland, J. P. (2014). Central fatigue contributes to the greater reductions in explosive than maximal strength with high-intensity fatigue. *Experimental Physiology, 99*(7), 964-973.

Burke, L. M., Kiens, B., & Ivy, J. L. (2004). Carbohydrates and fat for training and recovery. *Journal of Sports Sciences, 22*(1), 15-30.

Burke, R. E., Levine, D. N., Tsairis, P., & Zajac, F. E., III. (1973). Physiological types and histochemical profiles in motor units of the cat gastrocnemius. *The Journal of physiology, 234*(3), 723-748.

Bussel, B., & Pierrot Deseilligny, E. (1977). Inhibition of human motoneurones, probably of renshaw origin, elicited by an orthodromic motor discharge. *Journal of Physiology, 269*(2), 319-339.

Capaday, C. (1997). Neurophysiological methods for studies of the motor system in freely moving human subjects. *Journal of Neuroscience Methods, 74*(2), 201-218.

Casabona, A., Polizzi, M. C., & Perciavalle, V. (1990). Differences in H-reflex between athletes trained for explosive contractions and non-trained subjects. *European Journal of Applied Physiology and Occupational Physiology, 61*(1-2), 26-32.

Close, R. (1965). The relation between intrinsic speed of shortening and duration of the active state of muscle. *The Journal of physiology, 180*(3), 542-559.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159.

Colliander, E. B., Dudley, G. A., & Tesch, P. A. (1988). Skeletal muscle fiber type composition and performance during repeated bouts of maximal, concentric contractions. *European Journal of Applied Physiology and Occupational Physiology, 58*(1-2), 81-86.

Cribb, P. J., & Hayes, A. (2006). Effects of supplement timing and resistance exercise on skeletal muscle hypertrophy. *Medicine and Science in Sports and Exercise, 38*(11), 1918-1925.

Davies, C. T. M., & Young, K. (1983). Effect of temperature on the contractile properties and muscle power of triceps surae in humans. *Journal of Applied Physiology Respiratory Environmental and Exercise Physiology, 55*(1 I), 191-195.

Davies, T., Orr, R., Halaki, M., & Hackett, D. (2016). Erratum to: Effect of Training Leading to Repetition Failure on Muscular Strength: A Systematic Review and Meta-Analysis(Sports Med, Doi:10.1007/s40279-015-0451-3). *Sports Medicine, 46*(4), 605-610.

De Luca, C. J. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics, 13*(2), 135-163.

Del Balso, C., & Cafarelli, E. (2007). Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. *Journal of Applied Physiology, 103*(1), 402-411.

del Olmo, M. F., Reimunde, P., Viana, O., Acero, R. M., & Cudeiro, J. (2006). Chronic neural adaptation induced by long-term resistance training in humans. *European Journal of Applied Physiology, 96*(6), 722-728.

Dimitrova, N. A., & Dimitrov, G. V. (2003). Interpretation of EMG changes with fatigue: Facts, pitfalls, and fallacies. *Journal of Electromyography and Kinesiology, 13*(1), 13-36.

Dragert, K., & Zehr, E. P. (2011). Bilateral neuromuscular plasticity from unilateral training of the ankle dorsiflexors. *Experimental Brain Research, 208*(2), 217-227.

Drinkwater, E. J., Lawton, T. W., Lindsell, R. P., Pyne, D. B., Hunt, P. H., & McKenna, M. J. (2005). Training leading to repetition failure enhances bench press strength gains in elite junior athletes. *Journal of Strength and Conditioning Research, 19*(2), 382-388.

Duclay, J., Martin, A., Robbe, A., & Pousson, M. (2008). Spinal reflex plasticity during maximal dynamic contractions after eccentric training. *Medicine and Science in Sports and Exercise, 40*(4), 722-734.

Dudley, G. A., & Djamil, R. (1985). Incompatibility of endurance- and strength-training modes of exercise. *Journal of Applied Physiology, 59*(5), 1446-1451.

Earles, D. R., Dierking, J. T., Robertson, C. T., & Koceja, D. M. (2002). Pre- and post-synaptic control of motoneuron excitability in athletes. *Medicine and Science in Sports and Exercise, 34*(11), 1766-1772.

Eccles, J. C., Schmidt, R. F., & Willis, W. D. (1962). Presynaptic inhibition of the spinal monosynaptic reflex pathway. *The Journal of physiology, 161*(2), 282-297.

Enoka, R. M., & Stuart, D. G. (1984). Henneman's 'size principle': current issues. *Trends in Neurosciences, 7*(7), 226-228.

Farina, D., Holobar, A., Merletti, R., & Enoka, R. M. (2010). Decoding the neural drive to muscles from the surface electromyogram. *Clinical Neurophysiology, 121*(10), 1616-1623.

Farina, D., Merletti, R., & Enoka, R. M. (2004). The extraction of neural strategies from the surface EMG. *Journal of Applied Physiology, 96*(4), 1486-1495.

Fimland, M. S., Helgerud, J., Gruber, M., Leivseth, G., & Hoff, J. (2009a). Functional maximal strength training induces neural transfer to single-joint tasks. *European Journal of Applied Physiology, 107*(1), 21-29.

Fimland, M. S., Helgerud, J., Solstad, G. M., Iversen, V. M., Leivseth, G., & Hoff, J. (2009b). Neural adaptations underlying cross-education after unilateral strength training. *European Journal of Applied Physiology, 107*(6), 723-730.

Fisher, J. P., Blossom, D., & Steele, J. (2015). A comparison of volume-equated knee extensions to failure, or not to failure, upon rating of perceived exertion and strength adaptations. *Applied Physiology, Nutrition and Metabolism, 41*(2), 168-174.

Fitts, R. H. (1994). Cellular mechanisms of muscle fatigue. *Physiological Reviews, 74*(1), 49-94.

Folland, J. P., Irish, C. S., Roberts, J. C., Tarr, J. E., & Jones, D. A. (2002). Fatigue is not a necessary stimulus for strength gains during resistance training. *British Journal of Sports Medicine, 36*(5), 370-373.

Fry, A. C., & Kraemer, W. J. (1997). Resistance exercise overtraining and overreaching: Neuroendocrine responses. *Sports Medicine, 23*(2), 106-129.

Fry, A. C., Kraemer, W. J., Borselen, F. V., Lynch, J. M., Marsit, J. L., Roy, E. P., Triplett, N. T., & Knuttgen, H. G. (1994). Performance decrements with high-intensity resistance exercise overtraining. *Medicine and Science in Sports and Exercise, 26*(9), 1165-1173.

Gandevia, S. C. (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiological Reviews, 81*(4), 1725-1789.

Gandevia, S. C., Allen, G. M., Butler, J. E., & Taylor, J. L. (1996). Supraspinal factors in human muscle fatigue: Evidence for suboptimal output from the motor cortex. *Journal of Physiology, 490*(2), 529-536.

Gandevia, S. C., & McKenzie, D. K. (1988). Activation of human muscles at short muscle lengths during maximal static efforts. *Journal of Physiology, 407*, 599-613.

Garland, S. J., Walton, D., & Ivanova, T. D. (2003). Effect of force level and training status on contractile properties following fatigue. *Canadian Journal of Applied Physiology, 28*(1), 93-101.

Gauche, E., Couturier, A., Lepers, R., Michaut, A., Rabita, G., & Hausswirth, C. (2009). Neuromuscular fatigue following high versus low-intensity eccentric exercise of biceps brachii muscle. *Journal of Electromyography and Kinesiology, 19*(6), e481-e486.

Geertsen, S. S., Lundbye-Jensen, J., & Nielsen, J. B. (2008). Increased central facilitation of antagonist reciprocal inhibition at the onset of dorsiflexion following explosive strength training. *Journal of Applied Physiology, 105*(3), 915-922.

González-Badillo, J. J., Izquierdo, M., & Gorostiaga, E. M. (2006). Moderate volume of high relative training intensity produces greater strength gains compared with low and high volumes in competitive weightlifters. *Journal of Strength and Conditioning Research, 20*(1), 73-81.

Hakkinen, K., Alen, M., & Komi, P. V. (1985a). Changes in isometric force and relaxation time, electromyographic and muscle fibre characteristics of human skeletal muscle during strength training and detraining. *Acta Physiologica Scandinavica, 125*(4), 573-585.

Häkkinen, K., Kallinen, M., Linnamo, V., Pastinen, U. M., Newton, R. U., & Kraemer, W. J. (1996). Neuromuscular adaptations during bilateral versus unilateral strength training in middle-aged and elderly men and women. *Acta Physiologica Scandinavica, 158*(1), 77-88.

Hakkinen, K., & Komi, P. V. (1981). Effect of different combined concentric and eccentric muscle work regimens on maximal strength development. *Journal of Human Movement Studies, 7*(1), 33-44.

Hakkinen, K., & Komi, P. V. (1986). Training-induced changes in neuromuscular performance under voluntary and reflex conditions. *European Journal of Applied Physiology and Occupational Physiology, 55*(2), 147-155.

Hakkinen, K., Komi, P. V., & Alen, M. (1985b). Effect of explosive type strength training on isometric force- and relaxation-time, electromyographic and muscle fibre characteristics of leg extensor muscles. *Acta Physiologica Scandinavica, 125*(4), 587-600.

Häkkinen, K., Komi, P. V., Alén, M., & Kauhanen, H. (1987). EMG, muscle fibre and force production characteristics during a 1 year training period in elite weight-lifters. *European Journal of Applied Physiology and Occupational Physiology, 56*(4), 419-427.

Hartman, M. J., Ryan, E. D., Cramer, J. T., & Bemben, M. G. (2011). The effects of fatigue of the plantar flexors on peak torque and voluntary activation in untrained and resistance-trained men. *Journal of Strength and Conditioning Research, 25*(2), 527-532.

Hedelin, R., Kentta, G., Wiklund, U., Bjerle, P., & Henriksson-Larsen, K. (2000). Short-term overtraining: Effects on performance, circulatory responses, and heart rate variability. *Medicine and Science in Sports and Exercise, 32*(8), 1480-1484.

Henneman, E. (1957). Relation between size of neurons and their susceptibility to discharge. *Science, 126*(3287), 1345-1347.

Herbert, R. D., & Gandevia, S. C. (1999). Twitch interpolation in human muscles: Mechanisms and implications for measurement of voluntary activation. *Journal of Neurophysiology, 82*(5), 2271-2283.

Higbie, E. J., Cureton, K. J., Warren Iii, G. L., & Prior, B. M. (1996). Effects of concentric and eccentric training on muscle strength, cross- sectional area, and neural activation. *Journal of Applied Physiology, 81*(5), 2173-2181.

Hoeger, W. W., Hopkins, D. R., Barette, S. L., & Hale, D. F. (1990). Relationship between repetitions and selected percentages of one repetition maximum: A comparison between untrained and trained males and females. *Journal of Strength and Conditioning Research, 4*(2), 47-54.

Holtermann, A., Roeleveld, K., Engstrøm, M., & Sand, T. (2007). Enhanced H-reflex with resistance training is related to increased rate of force development. *European Journal of Applied Physiology, 101*(3), 301-312.

Hopkins, J. T., Ingersoll, C. D., Cordova, M. L., & Edwards, J. E. (2000). Intrasession and intersession reliability of the Soleus H-reflex in supine and standing positions. *Electromyography and Clinical Neurophysiology, 40*(2), 89-94.

Hultborn, H., Illert, M., Nielsen, J., Paul, A., Ballegaard, M., & Wiese, H. (1996). On the mechanism of the post-activation depression of the H-reflex in human subjects. *Experimental Brain Research, 108*(3), 450-462.

Iles, J. F., Ali, A., & Pardoe, J. (2000). Task-related changes of transmission in the pathway of heteronymous spinal recurrent inhibition from soleus to quadriceps motor neurones in man. *Brain, 123*(11), 2264-2272.

Izquierdo-Gabarren, M., González De Txabarri Expósito, R., García-PallarIs, J., Sánchez-Medina, L., De Villarreal, E. S. S., & Izquierdo, M. (2010). Concurrent endurance and strength training not to failure optimizes performance gains. *Medicine and Science in Sports and Exercise, 42*(6), 1191-1199.

Izquierdo, M., Ibañez, J., González-Badillo, J. J., Häkkinen, K., Ratamess, N. A., Kraemer, W. J., French, D. N., Eslava, J., Altadill, A., Asiain, X., & Gorostiaga, E. M. (2006).

Differential effects of strength training leading to failure versus not to failure on hormonal responses, strength, and muscle power gains. *Journal of Applied Physiology, 100*(5), 1647-1656.

Jessop, T., Depaola, A., Casaletto, L., Englard, C., & Knikou, M. (2013). Short-term plasticity of human spinal inhibitory circuits after isometric and isotonic ankle training. *European Journal of Applied Physiology, 113*(2), 273-284.

Johnson, M. A., Polgar, J., Weightman, D., & Appleton, D. (1973). Data on the distribution of fibre types in thirty-six human muscles. An autopsy study. *Journal of the Neurological Sciences, 18*(1), 111-129.

Johnson, S. T., Kipp, K., & Hoffman, M. A. (2012). Spinal motor control differences between the sexes. *European Journal of Applied Physiology, 112*(11), 3859-3864.

Johnson, S. T., Kipp, K., Norcross, M. F., & Hoffman, M. A. (2014). Spinal and supraspinal motor control predictors of rate of torque development. *Scandinavian Journal of Medicine and Science in Sports*.

Kanehisa, H., & Miyashita, M. (1983). Specificity of velocity in strength training. *European Journal of Applied Physiology and Occupational Physiology, 52*(1), 104-106.

Kawakami, Y., Abe, T., & Fukunaga, T. (1993). Muscle-fiber pennation angles are greater in hypertrophied than in normal muscles. *Journal of Applied Physiology, 74*(6), 2740-2744.

Kawakami, Y., Amemiya, K., Kanehisa, H., Ikegawa, S., & Fukunaga, T. (2000). Fatigue responses of human triceps surae muscles during repetitive maximal isometric contractions. *Journal of Applied Physiology, 88*(6), 1969-1975.

Keenan, K. G., Farina, D., Maluf, K. S., Merletti, R., & Enoka, R. M. (2005). Influence of amplitude cancellation on the simulated surface electromyogram. *Journal of Applied Physiology, 98*(1), 120-131.

Kent-Braun, J. A. (1999). Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. *European Journal of Applied Physiology and Occupational Physiology, 80*(1), 57-63.

Kipp, K., Johnson, S. T., Doeringer, J. R., & Hoffman, M. A. (2011). Spinal reflex excitability and homosynaptic depression after a bout of whole-body vibration. *Muscle and Nerve, 43*(2), 259-262.

Klass, M., Guissard, N., & Duchateau, J. (2004). Limiting mechanisms of force production after repetitive dynamic contractions in human triceps surae. *Journal of Applied Physiology, 96*(4), 1516-1521.

Klimstra, M., & Zehr, E. P. (2008). A sigmoid function is the best fit for the ascending limb of the Hoffmann reflex recruitment curve. *Experimental Brain Research, 186*(1), 93-105.

Knikou, M. (2008). The H-reflex as a probe: Pathways and pitfalls. *Journal of Neuroscience Methods, 171*(1), 1-12.

Kyröläinen, H., & Komi, P. V. (1994). Neuromuscular performance of lower limbs during voluntary and reflex activity in power- and endurance-trained athletes. *European Journal of Applied Physiology and Occupational Physiology, 69*(3), 233-239.

Lagerquist, O., Zehr, E. P., & Docherty, D. (2006). Increased spinal reflex excitability is not associated with neural plasticity underlying the cross-education effect. *Journal of Applied Physiology, 100*(1), 83-90.

Lamboley, C. R., Murphy, R. M., McKenna, M. J., & Lamb, G. D. (2014). Sarcoplasmic reticulum Ca2+ uptake and leak properties, and SERCA isoform expression, in type I and type II fibres of human skeletal muscle. *Journal of Physiology, 592*(6), 1381-1395.

Lee, M., Gandevia, S. C., & Carroll, T. J. (2009). Unilateral strength training increases voluntary activation of the opposite untrained limb. *Clinical Neurophysiology, 120*(4), 802-808.

Leveritt, M., Abernethy, P. J., Barry, B. K., & Logan, P. A. (1999). Concurrent strength and endurance training. A review. *Sports Medicine, 28*(6), 413-427.

Linnamo, V., Häkkinen, K., & Komi, P. V. (1998). Neuromuscular fatigue and recovery in maximal compared to explosive strength loading. *European Journal of Applied Physiology and Occupational Physiology, 77*(1-2), 176-181.

Löscher, W. N., Cresswell, A. G., & Thorstensson, A. (1996). Excitatory drive to the α-motoneuron pool during a fatiguing submaximal contraction in man. *Journal of Physiology, 491*(1), 271-280.

Maffiuletti, N. A., Martin, A., Babault, N., Pensini, M., Lucas, B., & Schieppati, M. (2001). Electrical and mechanical Hmax-to-Mmax ratio in power- and endurance-trained athletes. *Journal of Applied Physiology, 90*(1), 3-9.

Mangine, G. T., Hoffman, J. R., Gonzalez, A. M., Townsend, J. R., Wells, A. J., Jajtner, A. R., Beyer, K. S., Boone, C. H., Miramonti, A. A., Wang, R., LaMonica, M. B., Fukuda, D. H., Ratamess, N. A., & Stout, J. R. (2015). The effect of training volume and intensity on improvements in muscular strength and size in resistance-trained men. *Physiol Rep, 3*(8).

Marshall, P. W. M., Finn, H. T., & Siegler, J. C. (2015). The magnitude of peripheral muscle fatigue induced by high and low intensity single-joint exercise does not lead to central motor output reductions in resistance trained men. *PLoS ONE, 10*(10).

Marshall, P. W. M., McEwen, M., & Robbins, D. W. (2011). Strength and neuromuscular adaptation following one, four, and eight sets of high intensity resistance exercise in trained males. *European Journal of Applied Physiology, 111*(12), 3007-3016.

Marshall, P. W. M., Robbins, D. A., Wrightson, A. W., & Siegler, J. C. (2012). Acute neuromuscular and fatigue responses to the rest-pause method. *Journal of Science and Medicine in Sport, 15*(2), 153-158.

Maton, B. (1981). Human motor unit activity during the onset of muscle fatigue in submaximal isometric isotonic contraction. *European Journal of Applied Physiology and Occupational Physiology, 46*(3), 271-281.

McCall, G. E., Byrnes, W. C., Dickinson, A., Pattany, P. M., & Fleck, S. J. (1996). Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. *Journal of Applied Physiology, 81*(5), 2004-2012.

McKenzie, D. K., Bigland-Ritchie, B., Gorman, R. B., & Gandevia, S. C. (1992). Central and peripheral fatigue of human diaphraghm and limb muscles assessed by twitch interpolation. *Journal of Physiology, 454*, 643-656.

Merton, P. A. (1954). Voluntary strength and fatigue. *The Journal of physiology, 123*(3), 553-564.

Metzger, J. M., & Moss, R. L. (1990). pH modulation of the kinetics of a Ca2+-sensitive cross-bridge transition in mammalian single skeletal muscle fibres. *Journal of Physiology, 428*, 751-764.

Milner-Brown, H. S., & Miller, R. G. (1986). Muscle membrane excitation and impulse propagation velocity are reduced during muscle fatigue. *Muscle and Nerve, 9*(4), 367-374.

Milner-Brown, H. S., Stein, R. B., & Yemm, R. (1973a). Changes in firing rate of human motor units during linearly changing voluntary contractions. *The Journal of physiology, 230*(2), 371-390.

Milner-Brown, H. S., Stein, R. B., & Yemm, R. (1973b). The orderly recruitment of human motor units during voluntary isometric contractions. *The Journal of physiology, 230*(2), 359-370.

Misiaszek, J. E. (2003). The H-reflex as a tool in neurophysiology: Its limitations and uses in understanding nervous system function. *Muscle and Nerve, 28*(2), 144-160.

Morel, B., Clémençon, M., Rota, S., Millet, G. Y., Bishop, D. J., Brosseau, O., Rouffet, D. M., & Hautier, C. A. (2015). Contraction velocity influence the magnitude and etiology of neuromuscular fatigue during repeated maximal contractions. *Scandinavian Journal of Medicine and Science in Sports, 25*(5), e432-e441.

Moritani, T., & DeVries, H. A. (1979). Neural factors versus hypertrophy in the time course of muscle strength gain. *American Journal of Physical Medicine, 58*(3), 115-130.

Mottram, C. J., Jakobi, J. M., Semmler, J. G., & Enoka, R. M. (2005). Motor-unit activity differs with load type during a fatiguing contraction. *Journal of Neurophysiology, 93*(3), 1381-1392.

Munn, J., Herbert, R. D., Hancock, M. J., & Gandevia, S. C. (2005). Resistance training for strength: Effect of number of sets and contraction speed. *Medicine and Science in Sports and Exercise, 37*(9), 1622-1626.

Newman, S. A., Jones, G., & Newham, D. J. (2003). Quadriceps voluntary activation at different joint angles measured by two stimulation techniques. *European Journal of Applied Physiology, 89*(5), 496-499.

Neyroud, D., Maffiuletti, N. A., Kayser, B., & Place, N. (2012). Mechanisms of fatigue and task failure induced by sustained submaximal contractions. *Medicine and Science in Sports and Exercise, 44*(7), 1243-1251.

Nielsen, J., Crone, C., & Hultborn, H. (1993). H-reflexes are smaller in dancers from The Royal Danish Ballet than in well-trained athletes. *European Journal of Applied Physiology and Occupational Physiology, 66*(2), 116-121.

Nordlund Ekblom, M. M. (2010). Improvements in dynamic plantar flexor strength after resistance training are associated with increased voluntary activation and V-to-M ratio. *Journal of Applied Physiology, 109*(1), 19-26.

Nordlund, M. M., Thorstensson, A., & Cresswell, A. G. (2004). Central and peripheral contributions to fatigue in relation to level of activation during repeated maximal voluntary isometric plantar flexions. *Journal of Applied Physiology, 96*(1), 218-225.

Offer, G., & Ranatunga, K. W. (2015). The endothermic ATP hydrolysis and crossbridge attachment steps drive the increase of force with temperature in isometric and shortening muscle. *Journal of Physiology, 593*(8), 1997-2016.

Ortenblad, N., Sjogaard, G., & Madsen, K. (2000). Impaired sarcoplasmic reticulum Ca2+ release rate after fatiguing stimulation in rat skeletal muscle. *Journal of Applied Physiology, 89*(1), 210-217.

Pasquet, B., Carpentier, A., Duchateau, J., & Hainaut, K. (2000). Muscle fatigue during concentric and eccentric contractions. *Muscle and Nerve, 23*(11), 1727-1735.

Pensini, M., & Martin, A. (2004). Effect of voluntary contraction intensity on the H-reflex and V-wave responses. *Neuroscience Letters, 367*(3), 369-374.

Pensini, M., Martin, A., & Maffiuletti, N. A. (2002). Central versus peripheral adaptations following eccentric resistance training. *Int J Sports Med, 23*(8), 567-574.

Pierrot-Deseilligny, E., Bussel, B., Held, J. P., & Katz, R. (1976). Excitability of human motoneurones after discharge in a conditioning reflex. *Electroencephalography and Clinical Neurophysiology, 40*(3), 279-287.

Place, N., Maffiuletti, N. A., Martin, A., & Lepers, R. (2007). Assessment of the reliability of central and peripheral fatigue after sustained maximal voluntary contraction of the quadriceps muscle. *Muscle and Nerve, 35*(4), 486-495.

Rasch, P. J., & Morehouse, L. E. (1957). Effect of static and dynamic exercises on muscular strength and hypertrophy. *J Appl Physiol, 11*(1), 29-34.

Ratamess, N. A., Falvo, M. J., Mangine, G. T., Hoffman, J. R., Faigenbaum, A. D., & Kang, J. (2007). The effect of rest interval length on metabolic responses to the bench press exercise. *European Journal of Applied Physiology, 100*(1), 1-17.

Rhea, M. R., Alvar, B. A., Ball, S. D., & Burkett, L. N. (2002). Three sets of weight training superior to 1 set with equal intensity for eliciting strength. *Journal of Strength and Conditioning Research, 16*(4), 525-529.

Rhea, M. R., Alvar, B. A., Burkett, L. N., & Ball, S. D. (2003). A meta-analysis to determine the dose response for strength development. *Medicine and Science in Sports and Exercise, 35*(3), 456-464.

Robbins, D. W., Young, W. B., Behm, D. G., Payne, W. R., & Klimstra, M. D. (2010). Physical performance and electromyographic responses to an acute bout of paired set strength training versus traditional strength training. *Journal of Strength and Conditioning Research, 24*(5), 1237-1245.

Robinson, J. M., Stone, M. H., Johnson, R. L., Penland, C. M., Warren, B. J., & David Lewis, R. (1995). Effects of different weight training exercise/rest intervals on strength, power, and high intensity exercise endurance. *Journal of Strength and Conditioning Research, 9*(4), 216-221.

Rooney, K. J., Herbert, R. D., & Balnave, R. J. (1994). Fatigue contributes to the strength training stimulus. *Medicine and Science in Sports and Exercise, 26*(9), 1160-1164.

Rutherford, O. M., & Jones, D. A. (1986). The role of learning and coordination in strength training. *European Journal of Applied Physiology and Occupational Physiology, 55*(1), 100-105.

Rutherford, O. M., Jones, D. A., & Newham, D. J. (1986). Clinical and experimental application of the percutaneous twitch superimposition technique for the study of human muscle activation. *Journal of Neurology, Neurosurgery and Psychiatry, 49*(11), 1288-1291.

Sale, D. G. (1987). Influence of exercise and training on motor unit activation. *Exercise and Sport Sciences Reviews, 15*, 95-151.

Sale, D. G. (1988). Neural adaptation to resistance training. *Medicine and Science in Sports and Exercise, 20*(5), S135-S145.

Sale, D. G., & MacDougall, D. (1981). Specificity in strength training: A review for the coach and athlete. *Canadian Journal of Applied Sport Sciences, 6*(2), 87-92.

Sale, D. G., MacDougall, J. D., Jacobs, I., & Garner, S. (1990). Interaction between concurrent strength and endurance training. *Journal of Applied Physiology, 68*(1), 260-270.

Sale, D. G., MacDougall, J. D., Upton, A. R. M., & McComas, A. J. (1983a). Effect of strength training upon motoneuron excitability in man. *Medicine and Science in Sports and Exercise, 15*(1), 57-62.

Sale, D. G., McComas, A. J., MacDougall, J. D., & Upton, A. R. M. (1982). Neuromuscular adaptation in human thenar muscles following strength training and immobilization. *Journal of Applied Physiology Respiratory Environmental and Exercise Physiology, 53*(2), 419-424.

Sale, D. G., Upton, A. R. M., McComas, A. J., & MacDougall, J. D. (1983b). Neuromuscular function in weight-trainers. *Experimental Neurology, 82*(3), 521-531.

Sampson, J. A., & Groeller, H. (2015). Is repetition failure critical for the development of muscle hypertrophy and strength? *Scand J Med Sci Sports*.

Scaglioni, G., Ferri, A., Minetti, A. E., Martin, A., Van Hoecke, J., Capodaglio, P., Sartorio, A., & Narici, M. V. (2002). Plantar flexor activation capacity and H reflex in older adults: Adaptations to strength training. *Journal of Applied Physiology, 92*(6), 2292-2302.

Schillings, M. L., Hoefsloot, W., Stegeman, D. F., & Zwarts, M. J. (2003). Relative contributions of central and peripheral factors to fatigue during a maximal sustained effort. *European Journal of Applied Physiology, 90*(5-6), 562-568.

Schmitz, R. J., Arnold, B. L., Perrin, D. H., Granata, K. P., Gaesser, G. A., & Gansneder, B. M. (2002). Effect of isotonic and isometric knee extension exercises on mechanical and electromyographical specificity of fatigue. *Isokinetics and Exercise Science, 10*(4), 167-175.

Schott, J., McCully, K., & Rutherford, O. M. (1995). The role of metabolites in strength training - II. Short versus long isometric contractions. *European Journal of Applied Physiology and Occupational Physiology, 71*(4), 337-341.

Seynnes, O. R., De Boer, M., & Narici, M. V. (2007). Early skeletal muscle hypertrophy and architectural changes in response to high-intensity resistance training. *Journal of Applied Physiology, 102*(1), 368-373.

Shield, A., & Zhou, S. (2004). Assessing voluntary muscle activation with the twitch interpolation technique. *Sports Medicine, 34*(4), 253-267.

Siegler, J. C., Marshall, P., Pouslen, M. K., Nielsen, N. P. B., Kennedy, D., & Green, S. (2014). The effect of pH on fatigue during submaximal isometric contractions of the human calf muscle. *European Journal of Applied Physiology, 115*(3), 565-577.

Stephens, J. A., & Taylor, A. (1972). Fatigue of maintained voluntary muscle contraction in man. *The Journal of physiology, 220*(1), 1-18.

Strojnik, V., & Komi, P. V. (1998). Neuromuscular fatigue after maximal stretch-shortening cycle exercise. *Journal of Applied Physiology, 84*(1), 344-350.

Sweeney, H. L., Bowman, B. F., & Stull, J. T. (1993). Myosin light chain phosphorylation in vertebrate striated muscle: Regulation and function. *American Journal of Physiology - Cell Physiology, 264*(5 33-5), C1085-C1095.

Tax, A. A. M., van der Gon, J. J. D., Gielen, C. C. A. M., & van den Tempel, C. M. M. (1989). Differences in the activation of m. biceps brachii in the control of slow isotonic movements and isometric contractions. *Experimental Brain Research, 76*(1), 55-63.

Taylor, J. L., Todd, G., & Gandevia, S. C. (2006). Evidence for a supraspinal contribution to human muscle fatigue. *Clinical and Experimental Pharmacology and Physiology, 33*(4), 400-405.

Tran, Q. T., Docherty, D., & Behm, D. (2006). The effects of varying time under tension and volume load on acute neuromuscular responses. *European Journal of Applied Physiology, 98*(4), 402-410.

Upton, A. R., McComas, A. J., & Sica, R. E. (1971). Potentiation of "late" responses evoked in muscles during effort. *Journal of Neurology Neurosurgery and Psychiatry, 34*(6), 699-711.

Van Cutsem, M., Duchateau, J., & Hainaut, K. (1998). Changes in single motor unit behaviour contribute to the increase in contraction speed after dynamic training in humans. *Journal of Physiology, 513*(1), 295-305.

Vandenboom, R., Grange, R. W., & Houston, M. E. (1993). Threshold for force potentiation associated with skeletal myosin phosphorylation. *American Journal of Physiology - Cell Physiology, 265*(6 34-6), C1456-C1462.

Viitasalo, J. T., & Komi, P. V. (1981). Effects of fatigue on isometric force- and relaxation-time characteristics in human muscle. *Acta Physiologica Scandinavica, 111*(1), 87-95.

Vila-Cha, C., Falla, D., Correia, M. V., & Farina, D. (2012). Changes in H reflex and V wave following short-term endurance and strength training. *Journal of Applied Physiology, 112*(1), 54-63.

Walker, S., Peltonen, J., Ahtiainen, J. P., Avela, J., & Häkkinen, K. (2009). Neuromuscular fatigue induced by an isotonic heavy-resistance loading protocol in knee extensors. *Journal of Sports Sciences, 27*(12), 1271-1279.

Walker, S., Taipale, R. S., Nyman, K., Kraemer, W. J., & Häkkinen, K. (2011). Neuromuscular and hormonal responses to constant and variable resistance loadings. *Medicine and Science in Sports and Exercise, 43*(1), 26-33.

Westerblad, H., Lännergren, J., & Allen, D. G. (1997). Slowed relaxation in fatigued skeletal muscle fibers of Xenopus and mouse: Contribution of [Ca2+](i) and cross-bridges. *Journal of General Physiology, 109*(3), 385-399.

Willardson, J. M. (2007). The application of training to failure in periodized multiple-set resistance exercise programs. *Journal of Strength and Conditioning Research, 21*(2), 628-631.

Willardson, J. M., & Burkett, L. N. (2006). The effect of rest interval length on the sustainability of squat and bench press repetitions. *Journal of Strength and Conditioning Research, 20*(2), 400-403.

Willardson, J. M., Norton, L., & Wilson, G. (2010). Training to failure and beyond in mainstream resistance exercise programs. *Strength and Conditioning Journal, 32*(3), 21-29.

Yue, G., Fuglevand, A. J., Nordstrom, M. A., & Enoka, R. M. (1995). Limitations of the surface electromyography technique for estimating motor unit synchronization. *Biological Cybernetics, 73*(3), 223-233.

Zehr, E. P. (2002). Considerations for use of the Hoffmann reflex in exercise studies. *European Journal of Applied Physiology, 86*(6), 455-468.

# APPENDICES

**APPENDIX I:**

**FAILURE AND NON-FAILURE TRAINING PROGRAMS (STUDY 3)**

## FAILURE PROGRAM

*Note:* The 'RM' value in each set indicates the number of repetitions performed per set and the load. i.e. 10 RM = 10 reps × 10 RM load.

**Week 1**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 6RM | 6RM | 8RM | 8RM |
| Standing overhead barbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 8RM | 6RM | |
| Lateral dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Rack pull | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 6RM | 6RM | 8RM | 8RM |
| Lat pull down superset with | 10RM | 10RM | 10RM | 10RM |
| close grip underhand pulldown | 10RM | 8RM | 6RM | 6RM |
| Dumbbell chest flys | 8RM | 8RM | 8RM | |
| Single arm dumbbell rows | 10RM | 8RM | 6RM | 4RM |
| Overhead tricep extension superset with | 10RM | 8RM | 6RM | |
| tricep pushdown | 6RM | 8RM | 10RM | |
| Dumbbell bicep curls | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 4

**Week 2**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 6RM | 6RM | 8RM | 8RM |
| Standing overhead barbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 8RM | 6RM | |
| Lateral dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Rack pull | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 6RM | 6RM | 8RM | 8RM |
| Lat pull down superset with | 10RM | 10RM | 10RM | 10RM |
| close grip underhand pulldown | 10RM | 8RM | 6RM | 6RM |
| Dumbbell chest flys | 8RM | 8RM | 8RM | |
| Single arm dumbbell rows | 10RM | 8RM | 6RM | 4RM |
| Overhead tricep extension superset with | 10RM | 8RM | 6RM | |
| tricep pushdown | 6RM | 8RM | 10RM | |
| Dumbbell bicep curls | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 4

**Week 3**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 6RM | 6RM | 8RM | 8RM |
| Standing overhead barbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 8RM | 6RM | |
| Lateral dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Rack pull | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 6RM | 6RM | 8RM | 8RM |
| Lat pull down superset with | 10RM | 10RM | 10RM | 10RM |
| close grip underhand pulldown | 10RM | 8RM | 6RM | 6RM |
| Dumbbell chest flys | 8RM | 8RM | 8RM | |
| Single arm dumbbell rows | 10RM | 8RM | 6RM | 4RM |
| Overhead tricep extension superset with | 10RM | 8RM | 6RM | |
| tricep pushdown | 6RM | 8RM | 10RM | |
| Dumbbell bicep curls | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 4

**Week 4**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 4RM | 6RM | 8RM | 8RM |
| Standing overhead barbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 8RM | 6RM | |
| Lateral dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Rack pull | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 4RM | 6RM | 8RM | 8RM |
| Lat pull down superset with | 10RM | 10RM | 10RM | 10RM |
| close grip underhand pulldown | 10RM | 8RM | 6RM | 6RM |
| Dumbbell chest flys | 8RM | 8RM | 8RM | |
| Single arm dumbbell rows | 10RM | 8RM | 6RM | 4RM |
| Overhead tricep extension superset with | 10RM | 8RM | 6RM | |
| tricep pushdown | 6RM | 8RM | 10RM | |
| Dumbbell bicep curls | 12RM | 12RM | 12RM | |

* Only perform 3 sets on Day 4

**Week 5**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 4RM | 6RM | 8RM | 8RM |
| Seated dumbbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 10RM | 10RM | |
| Front dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Leg curls | 10RM | 8RM | 6RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 4RM | 6RM | 8RM | 8RM |
| Seated row | 10RM | 8RM | 6RM | 4RM |
| Dumbbell incline chest press | 6RM | 6RM | 6RM | |
| Single arm lat pull down | 10RM | 10RM | 10RM | |
| Barbell skull crusher | 10RM | 8RM | 6RM | |
| Barbell bicep curls superset with | 10RM | 8RM | 6RM | 6RM |
| alternating arm dumbbell hammer curls | 6RM | 6RM | 8RM | 10RM |

* Only perform 3 sets on Day 4

**Week 6**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 4RM | 6RM | 8RM | 8RM |
| Seated dumbbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 10RM | 10RM | |
| Front dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Leg curls | 10RM | 8RM | 6RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 4RM | 6RM | 8RM | 8RM |
| Seated row | 10RM | 8RM | 6RM | 4RM |
| Dumbbell incline chest press | 6RM | 6RM | 6RM | |
| Single arm lat pull down | 10RM | 10RM | 10RM | |
| Barbell skull crusher | 10RM | 8RM | 6RM | |
| Barbell bicep curls superset with | 10RM | 8RM | 6RM | 6RM |
| alternating arm dumbbell hammer curls | 6RM | 6RM | 8RM | 10RM |

* Only perform 3 sets on Day 4

**Week 7**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 4RM | 6RM | 6RM | 8RM |
| Seated dumbbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 10RM | 10RM | |
| Front dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Leg curls | 10RM | 8RM | 6RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 4RM | 6RM | 6RM | 8RM |
| Seated row | 10RM | 8RM | 6RM | 4RM |
| Dumbbell incline chest press | 6RM | 6RM | 6RM | |
| Single arm lat pull down | 10RM | 10RM | 10RM | |
| Barbell skull crusher | 10RM | 8RM | 6RM | |
| Barbell bicep curls superset with | 10RM | 8RM | 6RM | 6RM |
| alternating arm dumbbell hammer curls | 6RM | 6RM | 8RM | 10RM |

* Only perform 3 sets on Day 4

**Week 8**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Seated calf raise | 10RM | 10RM | 10RM | 10RM |
| Barbell back squat | 4RM | 6RM | 6RM | 8RM |
| Seated dumbbell shoulder press | 10RM | 8RM | 6RM | |
| Seated leg extension | 10RM | 10RM | 10RM | |
| Front dumbbell shoulder raise | 10RM | 10RM | 10RM | |
| Leg curls | 10RM | 8RM | 6RM | |

* Only perform 3 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4* |
|---|---|---|---|---|
| Bench press | 4RM | 6RM | 6RM | 8RM |
| Seated row | 10RM | 8RM | 6RM | 4RM |
| Dumbbell incline chest press | 6RM | 6RM | 6RM | |
| Single arm lat pull down | 10RM | 10RM | 10RM | |
| Barbell skull crusher | 10RM | 8RM | 6RM | |
| Barbell bicep curls superset with | 10RM | 8RM | 6RM | 6RM |
| alternating arm dumbbell hammer curls | 6RM | 6RM | 8RM | 10RM |

* Only perform 3 sets on Day 4

## NON-FAILURE PROGRAM

*Note:* The ‘RM’ value in each set indicates the load. i.e. ‘5 × 10 RM’ corresponds to 5 repetitions performed with a 10 RM load.

**Week 1**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Standing overhead barbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Lateral dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Rack pull | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Lat pull down superset with | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| close grip underhand pulldown | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| Dumbbell chest flys | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | | |
| Single arm dumbbell rows | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Overhead tricep extension superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| tricep pushdown | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM | | |
| Dumbbell bicep curls | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 4

**Week 2**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Standing overhead barbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Lateral dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Rack pull | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Lat pull down superset with | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| close grip underhand pulldown | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| Dumbbell chest flys | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | | |
| Single arm dumbbell rows | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Overhead tricep extension superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| tricep pushdown | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM | | |
| Dumbbell bicep curls | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 4

**Week 3**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Standing overhead barbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Lateral dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Rack pull | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Lat pull down superset with | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| close grip underhand pulldown | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| Dumbbell chest flys | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | | |
| Single arm dumbbell rows | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Overhead tricep extension superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| tricep pushdown | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM | | |
| Dumbbell bicep curls | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 4

**Week 4**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Standing overhead barbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Lateral dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Rack pull | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Lat pull down superset with | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| close grip underhand pulldown | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| Dumbbell chest flys | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | | |
| Single arm dumbbell rows | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Overhead tricep extension superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| tricep pushdown | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM | | |
| Dumbbell bicep curls | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | 6 x 12RM | | |

* Only perform 6 sets on Day 4

**Week 5**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Seated dumbbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Front dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Leg curls | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Seated row | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Dumbbell incline chest press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | | |
| Single arm lat pull down | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Barbell skull crusher | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Barbell bicep curls superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| alternating arm dumbbell hammer curls | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM |

* Only perform 6 sets on Day 4

**Week 6**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Seated dumbbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Front dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Leg curls | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 4 x 8RM | 4 x 8RM |
| Seated row | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Dumbbell incline chest press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | | |
| Single arm lat pull down | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Barbell skull crusher | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Barbell bicep curls superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| alternating arm dumbbell hammer curls | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM |

* Only perform 6 sets on Day 4

**Week 7**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM |
| Seated dumbbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Front dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Leg curls | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM |
| Seated row | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Dumbbell incline chest press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | | |
| Single arm lat pull down | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Barbell skull crusher | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Barbell bicep curls superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| alternating arm dumbbell hammer curls | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM |

* Only perform 6 sets on Day 4

**Week 8**

Days 1 and 3

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Seated calf raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM |
| Barbell back squat | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM |
| Seated dumbbell shoulder press | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Seated leg extension | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Front dumbbell shoulder raise | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Leg curls | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |

* Only perform 6 sets on Day 3

Days 2 and 4

| | Set 1 | Set 2 | Set 3 | Set 4 | Set 5 | Set 6 | Set 7* | Set 8* |
|---|---|---|---|---|---|---|---|---|
| Bench press | 2 x 4RM | 2 x 4RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM |
| Seated row | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 2 x 4RM | 2 x 4RM |
| Dumbbell incline chest press | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | | |
| Single arm lat pull down | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | 5 x 10RM | | |
| Barbell skull crusher | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | | |
| Barbell bicep curls superset with | 5 x 10RM | 5 x 10RM | 4 x 8RM | 4 x 8RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM |
| alternating arm dumbbell hammer curls | 3 x 6RM | 3 x 6RM | 3 x 6RM | 3 x 6RM | 4 x 8RM | 4 x 8RM | 5 x 10RM | 5 x 10RM |

* Only perform 6 sets on Day 4