The Effects of Load and Training Pattern on Acute Neuromuscular Responses.

by

Tyler Logan Goodale
B.Sc. Zoology, University of Manitoba, 1999

A Thesis Submitted in Partial Fulfillment of the Requirements for the Degree of

MASTER OF SCIENCE

In the School of Physical Education

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Abstract

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The purpose of this study was to examine the effects of two different repetition maximum (RM) loads and training patterns on acute neuromuscular responses. Twenty one resistance-trained males performed 4 protocols of dynamic constant external resistance exercise, involving elbow flexors, that manipulated either load (5RM, 10RM) or training pattern (TP). For TP the subjects completed 4 sets of exercise with 3 min rest between exercise performing each set to fatigue which produced a decrease in the number of repetitions with each subsequent set (DTP); the second testing pattern involved performing the same number of sets and repetitions to the DTP but in a reverse order so that the repetitions ascend with each subsequent set (ATP). Volume load (VL) was equated for both patterns but the set in which the greatest VL was achieved occurred at different times in the workout, either in the 1st (DTP) or 4th (ATP) set of the exercise. Both TPs were conducted with a 5RM and 10RM load. Fatigue was assessed by changes in maximum voluntary isometric contraction (MVIC), motor unit activation (MUA), muscle twitch characteristics (peak twitch [PT], time to peak twitch [TPT], and ½ relaxation time [½RT]). All protocols produced significant changes pre to post fatigue ($p \leq .05$) for MVIC, MUA, PT, ½RT, and TPT. 5RM/ATP, 10RM/DTP, and 10RM/ATP protocols produced significant changes in MUA. PT was found to be significantly different across loads. The results

indicate that central fatigue is independent of load and pattern whereas peripheral fatigue appears to be dependent on load.

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Symbols and Abbreviations

½ RT	Half Relaxation Time
5RMD	Five Repetition Maximum Descending Training Pattern
5RMA	Five Repetition Maximum Ascending Training Pattern
10RMA	Ten Repetition Maximum Descending Training Pattern
ATP	Ascending Training Pattern
DTP	Descending Training Pattern
Ca^{2+}	Calcium
E-C	Excitation Contraction Coupling
EMG	Electromyogram
H+	Hydrogen Ion
HFF	High Frequency Fatigue
ITT	Interpolated Twitch Technique
LFF	Low Frequency Fatigue
MUA	Motor Unit Activation
MVIC	Maximum Voluntary Isometric Contraction
PT	Peak Twitch
R-LC	Myosin Regulatory Light Chain
RM	Repetition Maximum
SR	Sarcoplasmic Reticulum
SMS	Supramaximal Stimulation
TMS	Trans-cranial Magnetic Stimulation
TPT	Time to Peak Twitch

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I would like to thank my advisor, Dave, for his mentorship, understanding and patience in helping me through this experience. Without your help this project never would have been possible and would never have been completed. You have helped to shape my vision in sports science and I have appreciated all your help from day one of this experience. It has been a great experience working with you on everything from this project to training programs for Rugby Canada. Without you I would never have had a start in this business and I owe a lot to where I am today to you. Thanks for taking a chance on the kid from Manitoba who just showed up at your door one day.

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Many thanks to Dan and Quan for being great friends and a support group. You have made the whole process fun from the start, whether we were in class, working on our projects or hanging out together. I couldn't have asked for a greater group of peers!

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Thank you.

1 INTRODUCTION

It is well established that skeletal muscle increases in size and strength as a result of prolonged resistance exercise (Chestnut & Docherty, 1999; Kawakami, Abe, Kuno, & Fukunaga, 1995). However, the physiological mechanisms underlying these changes remain unclear. One proposed mechanism suggests that the temporary neuromuscular fatigue associated with performing a bout of resistance exercise may be associated with long term muscular adaptations (Rooney, Herbert, & Balnave, 1994; Schott, McCully, & Rutherford, 1995).

There are many theories about the merits of different training practices. It is important from both a practical and theoretical perspective to understand the training practices that will elicit optimal adaptation. One training variable which is still not well understood is the effect of fatigue on neuromuscular adaptation. Few studies have looked at the effects of varying levels of fatigue on strength and hypertrophy (Rooney et al., 1994; Folland, Irish, Roberts, Tarr, & Jones, 2002; Drinkwater, Lawton, Lindsell, Pyne, Hunt & McKenna, 2004; Izuierdo, Ibanez, Gonzalez-Badillo, Hakkinen, Ratamess, Kraemer, French, Eslava, Altadill, Asiain & Gorostiaga, 2006).

Rooney et al. (1994) investigated the role of fatigue in strength training. Strength increases produced by a training protocol in which subjects rested between contractions were compared to a protocol in which subjects did not rest. Subjects were randomly allocated to either a no rest group, a rest group, or a control group. Subjects in the two training groups trained the biceps brachii by lifting a 6RM weight 6 times, three days per week for 6 weeks. Subjects in the no-rest group performed repeated lifts without resting whereas subjects in the rest-group rested for 30 s between lifts. Both training groups performed the same number of

lifts at the same relative intensity. The results indicated that subjects who trained without rest experienced significantly greater increases in dynamic strength (mean 56.3%,SD 6.8%) than subjects who trained with the 30s rest interval (mean 41.2%, SD 6.6%). Rooney et al. (1994) concluded that greater short term strength increases are achieved when subjects lift weights without resting, eliciting a greater amount of fatigue.

Rooney et al. (1994) did not attempt to identify the physiological mechanisms that were responsible for the significant strength increases in the no-rest group. It was suggested that the increases in strength from short-term training were the result of neural mechanisms. High intensity fatiguing protocols may bring about greater activation of motor units than high intensity non-fatiguing protocols (Enoka, 1988; Sale, 1987; 1992). It is possible that the degree to which motor units are activated determines the magnitude of the strength training stimulus. Studies examining the effect of fatigue on submaximal isometric contractions found that as muscles fatigue they experience a progressively greater level of activation (Maton, 1981). Rooney et al. postulated that the fatiguing high intensity contractions provided a better way of activating high threshold motor units than non-fatiguing high intensity muscle contractions.

More recently Folland et al. (2002) compared the training effects of a high fatigue to a low fatigue training regimen, on neuromuscular adaptation. Subjects were assigned to either a high-fatigue protocol (four sets of 10 repetitions at 75% of 1RM with 30 s rest in between sets) or a low fatigue protocol (40 repetitions at 75% of 1RM with 30 s rest in between repetitions). The high fatigue protocol was designed to maximize metabolic stress. All fatiguing protocols were completed on a leg extension machine designed to isolate the quadriceps muscle group. Both groups trained for a total of nine weeks. Pre-, post- and mid-

testing sessions were conducted on isometric strength, torque and torque velocity. At the mid-point of training, the high fatigue group had 50% greater gains in isometric strength than the low-fatigue group. After nine weeks of training both groups showed similar improvements in strength measures with isometric strength increasing 3.9% in the high-fatigue group compared to a 4% improvement in the low-fatigue group. Folland et al. concluded that strength training does not need to involve the discomfort associated with high-fatigue training protocols in order to produce significant strength gains. One possible explanation for these results is that the low-fatiguing protocol enabled subjects to complete all repetitions at a greater load. Due to the nature of the high-fatigue protocol, the weight had to be decreased in subsequent sets to allow for the completion of the prescribed repetitions. This resulted in the high-fatigue group lifting on average 71% of their 1RM, whereas the low fatigue group lifted an average of 75% of their 1RM. This may indicate that a balance must be achieved between volume and intensity in order to achieve optimal strength gains.

Drinkwater et al., (2004) examined whether repetition failure is required for strength development. Testing and training were conducted on elite athletes divided into 3 training groups using different sets x reps and rest intervals: 4x6 with a 120s rest; 8x3 with a 72s rest; and 12x3 with a 72s rest. All subjects trained between 90-100% of their 6RM to ensure repetition failure was attained by the end of each training session. All athletes had their 3RM and 6RM bench press and maximal power output in the bench throw tested pre- and post-training. Significant improvement in strength was found in all groups regardless of the amount of repetition failure attained. The 4x6 and 12x3 groups experienced greater amount of repetition failure than the 8x3 group but did not experience any significant greater strength gains. It is difficult to interpret the results of this study due to the varying volume loads

between groups and different work to rest ratios. However, it should be noted that, although all training variables were not equated between groups, all groups attained significant strength improvements, indicating that elite athletes can attain strength gains when training with loads between 90-100% of 6RM.

More recently Izquierdo et al., (2006) examined the effect of training to failure versus not training to failure on hormonal changes and strength and power gains. An 11 week training study was conducted in which subjects were divided into a training-to-fatigue group and a non-fatigue group. Subjects trained twice per week with a total body training program. For the majority of the study the sets and reps were kept at 3 sets with 6 to 8 reps. Testing on power output, strength levels and endocrine response were conducted pre- and post-training. The study standardized both volume load and work to rest ratios between training groups. The results indicated that similar strength and power gains were found pre- to post-training regardless of whether or not training was taken to fatigue. Differences between groups were found in tests for muscular endurance in which the failure-group outperformed the non-failure group. In addition the endocrine response for the non-failure group showed a hormonal milieu that was considered to be more conducive to strength and power gains. The training-to-failure group demonstrated very high levels of catabolic stress hormones, depressed levels of growth hormones, and other anabolic growth factors. After the 11 wk study was conducted the researchers tapered the athletes for 5 weeks and found that the non-failure group displayed greater power output during the peaking strategy than the fatigue group, indicating that for athletes training to peak, training programs that involve training-to-failure may be sub-optimal.

The cause or mechanism (locus) of fatigue is important in order to understand the contribution of fatigue as a strength training stimulus. Fatigue can be viewed as either central or peripheral in origin. Central fatigue refers to a reduction in voluntary activation of muscle during exercise. Fatigue that is produced as a result of impairments in force generating capacities at or distal to the neuromuscular junction has been termed peripheral fatigue (Gandevia, 2001).

Many techniques have been used to determine the origin of fatigue. Recently Behm, Reardon, Fitzgerald, and Drinkwater (2002) used the interpolated twitch technique (ITT) method to monitor fatigue deficits caused by training at various intensities. This technique involves application of a supra-maximal stimulus to muscle. The application can be done when the muscle is in either a relaxed or contracted state. When in the relaxed state a number of variables can be measured. Peak twitch, time to peak twitch and twitch half relaxation time can all be used to infer what is happening within the muscle. Depression of peak twitch is considered to indicate impairment in excitation-contraction coupling (EC) (Behm et al., 2002). Time to peak twitch and twitch half relaxation time can be used as an indication of calcium kinetics, specifically calcium release and sequestering from the sarcoplasmic reticulum (Behm et al. 2002).

The ITT can also be superimposed on a maximal voluntary isometric contraction (MVIC). The superimposed twitch on the voluntary activation has been used to monitor the degree of voluntary motor unit activation. Any depression in this activity post fatigue can be interpreted as an indication of neurological deficit in the recruitment of motor units (Behm et al., 2002). When combined with electromyography (EMG) it is possible to measure general muscle activation including recruitment and synchronization patterns.

Based on the theory of a repetition maximum continuum, training loads of 2-6 RM have typically been used by strength and conditioning practitioners to induce fatigue with the goal of increasing strength through neural mechanics (Fleck & Kraemer, 1998). Strength coaches also typically use training loads of 8-15 RM to elicit a more peripheral response in the muscle, leading to both metabolic and biochemical changes within the muscle. In addition the structural damage and subsequent repair to the muscle lead to an increase in force producing contractile proteins.

To date very little research has been conducted differentiating the fatigue response induced by different repetition maximums. Behm el al., (2002) conducted an acute training study comparing the effect of 5, 10 and 20RM loads on neuromuscular responses. No significant differences between the training loads were found with respect to effects on the central nervous system. However peripheral responses were related to higher training volumes. Campos, Luecke, Wendeln, Toma, Hagerman, Murray, Ragg, Ratamess, Kraemer and Straton (2002) attempted to differentiate the adaptive response of the neuromuscular system to an 8 week training program with groups training at various RM loads. The research conducted by the Campos et al., (2002) group supported the theory of a repetition maximum continuum where higher intensity lower volume loads (3-5RM) lead to greater strength and power adaptations compared to a lower intensity higher volume loads (20-28RM) which lead to a greater muscular endurance response. All training groups found significant increases in Type IIa skeletal muscle fibres due to the conversion of Type IIb fibres. Although all groups saw a similar shift in fibre type the higher volume training groups experienced a significantly greater level of muscular hypertrophy in both Type IIb and Type I skeletal muscle fibres. Because

strength is related to muscular size it was postulated that the significant strength gains experienced by the high intensity low volume training group were induced by neural changes.

To date many of the training programs designed by strength and conditioning professionals still require further investigation into the mechanism by which they induce neuromuscular adaptations. It has been suggested that acute peripheral fatigue during resistance training contributes to the development of strength and hypertrophy (Schott et al., 1995). If peripheral adaptations are an important factor in strength development and fatigue is an important variable which influences peripheral responses, then further studies are necessary to determine the extent to which fatigue contributes to acute and long-term neuromuscular responses. Very few studies have examined either the effect of fatigue or load on training adaptations. Therefore many of our current training prescriptions lack sound research-based support. In order to optimize training adaptation for athletes the variables involved in the prescription of training programs must be investigated further.

1.1 Purpose of Experiment

To date no study has examined varying training patterns and loads with respect to their effect on acute neuromuscular responses. In the prescription of training programs the effects of fatigue are still not well understood. There are a number of strength training variables that are thought to be important to the training response such as volume, intensity, tempo, density of work, and the degree of fatigue. The importance of neuromuscular fatigue to the strength training stimulus is still not well understood and therefore is a point of contention amongst sports scientists and practitioners.

Before undertaking long term studies on the effects of fatigue on chronic neuromuscular adaptations it is necessary to understand the acute response of the neuromuscular system to

different fatigue-training protocols. Within physiological research it is important to not only state a change has occurred but also why a change may have occurred. With a better understanding of the neurological and metabolic response to acute training bouts, long term training programs can be better designed to maximize adaptation.

This study compared two different training patterns while keeping volume load equal. The study also compared the acute neuromuscular responses to training with high (5RM) and moderate (10RM) loads.

1.2 Research questions

1. Did a descending training pattern (DTP) where the highest initial volume load is completed in the earlier sets of multiple training sets induce greater acute neuromuscular fatigue than an ascending training pattern (ATP) where the highest volume load is completes in the latter sets of multiple training sets?
2. Did equal training volume, regardless of when the greatest volume load is completed during multiple training sets, result in a similar neuromuscular fatigue deficit?
3. Was there a difference in the origin of fatigue (central or peripheral) between a DTP and an ATP?
    i. Did a DTP pattern result in greater neural inactivation then an ATP?
    ii. Did a DTP vs. an ATP result in greater peripheral fatigue?
4. Was there a difference in the origin of fatigue (central or peripheral) due to the magnitude of load (5RM vs. 10RM)?

1.3 Operational Definitions

1. Fatigue: A temporary decline in maximal force production.
    a. Central fatigue will include fatigue at the neuromuscular junction and proximal to it.
    b. Peripheral fatigue will be considered as fatigue that occurs distal from the neuromuscular junction.

2. Trained subjects: Individuals that have performed upper body resistance training approximately three times a week for a minimum of one year prior to beginning the study.
3. Muscle activation: The extent to which a muscle is activated as measured by the ITT.
4. Maximal Voluntary Isometric Contraction (MVIC): The greatest force that an individual is able to generate.
5. 10RM: The maximal load that an individual is able to successfully arm curl 10 times.
6. 5RM: The maximal load that an individual is able to successfully arm curl 5 times.
7. Volume load: Training volume quantified by an approximation of mechanical work calculated by multiplying the number of repetitions by the load

1.4 Delimitations and Limitations

1. Participants were male.
2. Participants were trained individuals.
3. Participants were of university age.
4. Peripheral fatigue was assessed using evoked contractile properties.
5. Time under tension was not equated between training loads.
6. Teleo-anticipatory nature of the ascending training pattern (already knowing repetition scheme for testing session).

1.5 Assumptions

1. All subjects provide maximal effort.
2. Changes in ITT values indicated changes in neural activation and were not the result of error in measurement or the effort of the subjects.
3. The ITT is a valid measure of neural activation.

4. Peak twitch, peak ½ relaxation time and time to peak twitch are valid measures of peripheral fatigue.
5. Maximal isometric strength measures provided accurate fatigue levels induced from dynamic fatiguing protocols.

2 METHODS

2.1 Participants

21 university-aged males with a minimum of one year of continuous upper body resistance training participated in the study. Written consent was obtained from all subjects prior to participation and approval of the study was granted by the University of Victoria Human Ethics Committee. Subjects were initially briefed on the purpose and possible risks associated with the study. Prior to beginning the study, all subjects were asked to refrain from performing any resistance training targeting the biceps brachii for the duration of the study.

2.2 Experimental Design

The experiment consisted of two different training patterns across two different loads designed to manipulate fatigue. Subjects were required to participate in a minimum of four familiarization sessions. A fifth familiarization session was conducted if there was a greater than 5% difference in the acquired 5RM, 10RM or supramaximal stimulus (SMS). After satisfactory completion of the familiarization sessions, subjects performed each fatiguing protocol on separate days with approximately 48-72hrs between testing sessions.

2.3 Familiarization

As seen in figure 2.1, subjects reported to the lab and rested for 5min to ensure that they were in a relaxed state. Following the initial rest period, subjects began a warm-up consisting

of three sets of 10 repetitions of elbow flexion at a load of 50% of the estimated 1RM , separated by 3min rest periods. Testing of the SMS began three minutes after the completion of the warm-up. Following the SMS test, subjects performed two ITT evaluations separated by one minute in order to familiarize subjects with the protocols. Testing of the 10RM commenced approximately three minutes post-ITT at an initial load of 75% of estimated 1RM. The load was adjusted by 2.5-5lbs increments to ensure a true 10RM was obtained. A cadence of a 3 s eccentric phase and 2 s concentric phase was used during the warm-up to identify the 10RM load. Five minute rest periods between trials were provided to minimize fatigue (Brandendurg, 2001). Testing of the 5RM was completed on the second familiarization session. On the second day, testing commenced approximately 3 minutes post-ITT at an initial load of 85% of estimated 1RM. The load was adjusted by 2.5-5lbs increments to ensure a true 5RM was obtained. A cadence of a 3s eccentric and 2s concentric phase was used during the warm up and to acquire the 5RM load. Five minute rest periods between trials were provided to minimize fatigue (Brandendurg, 2001).

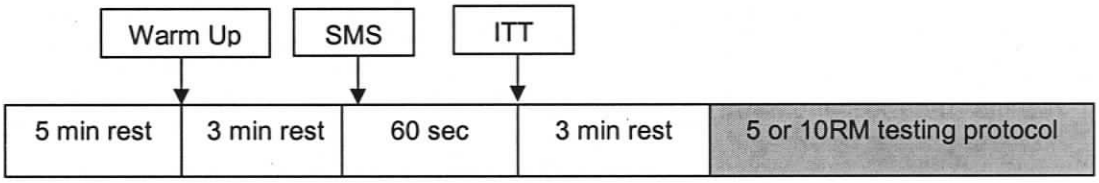

Figure 2.1 Timeline of the familiarization session.

2.4 Experimental Session

Subjects rested 5min upon arrival at the lab to establish baseline values which were consistent between sessions. Following the initial rest period, subjects performed a similar warm-up to that in the familiarization sessions. The SMS and ITT were performed pre- and post-fatiguing protocol (see figure 2.2). Prior to beginning the fatigue protocol, the subjects

performed an additional single set warm-up at 50% of the estimated 10 RM (Brandendurg, 2001). The SMS and ITT were administered 30s post fatiguing protocol because this timeframe has been associated with the greatest neural deficits (Behm et al., 2002).

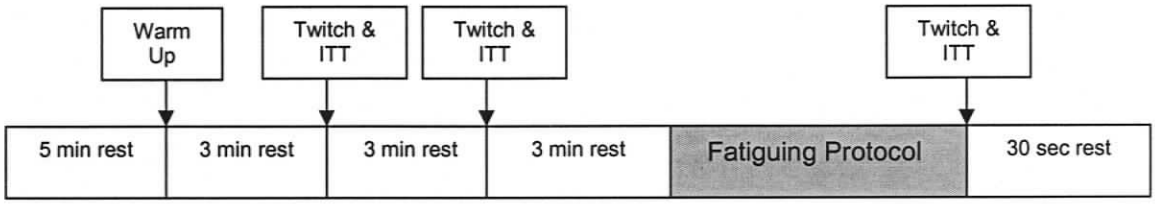

Figure 2.2 Timeline of testing session.

2.5 Fatiguing Protocols

All fatiguing protocols consisted of signal arm bicep curls of the dominant arm in a standing position. Subjects stood with their backs against a wall in order to minimize the contribution of other muscles to the movement. One complete repetition consisted of moving the arm through the full range of elbow motion. Subjects were instructed to keep in time with a pre-set metronome. All fatiguing protocols were supervised by the principal investigator to ensure that consistent technique was maintained between testing sessions and subjects. The various fatigue protocols were designed to manipulate the set in which the greatest volume load was applied (DTP vs. ATP). The DTP involved subjects performing 4 sets of training with 3 min rest between sets. Even with 3 min between sets, subjects were unable to maintain 10 repetitions for each of the 4 sets. Normally subjects demonstrated a steady decrease in the number of repetitions from the first to last set (e.g. 1st set 10, 2nd set 8, 3rd set 6, 4th set 5). This is a descending repetition scheme in which the greatest volume load was conducted in the initial training sets.

After at least 48-72 hours subjects returned to the lab and completed the ATP. This involved reversing the order of repetitions completed in the DTP. In the example, as described

above, the subject would perform only 5 repetitions on the first set, 6 on the second, 8 on the third and 10 on the last set. The total volume across all four sets was the same but the sets in which the greatest volume was performed were different.

Subjects executed both the DTP and the ATP with both a 5RM and 10RM load. The order of load was randomized but the DTP was conducted first in order to establish the repetition pattern for the ATP.

2.6 Set up for the Electrical Stimulator

Both the ITT and supramaximal stimulus testing were performed on a modified preacher curl apparatus. The skin was thoroughly prepared via sanding of the designated area followed by cleansing with isopropyl alcohol. Subjects were seated in a modified preacher curl, the seat was adjusted so the legs were at a 90 degree angle, and chest was flush against the arm rest pad with the arm resting on a additional pad and fully supinated and flexed at 90 degree (see figure 2.3) The joint angle was set using a goniometer. Once the desired position was obtained, the wrists of the subjects were inserted into a padded loop attached to a strain gauge (Omegadyne Ltd. Model 101-500, range 0-500lbs). Height of the strain gauge was adjusted and recorded so that the line of pull was perpendicular to that of the bicep. The high tension wire connecting the wrist loop to the strain gauge was adjusted in order to eliminate any slack and this distance was recorded for each individual. For electrode placement, the cathode (20x15mm) was lowered over the biceps brachii midway between the anterior edge of the deltoid and the proximal elbow crease with the elbow flexed at 90 degrees. The anode (15 x 15mm) was placed over the distal tendon in the elbow groove (Allen, Gandevia, and McKenzie, 1995; Allen, McKenzie and Gandevia, 1998). Placement of the electrodes was

designed to target the musculocutaneous nerve. Stimulation was given via a Digitimer LTD. Constant Current High Voltage Stimulator (Model Number DS7A). Voltage was set at a 100V rectangular pulse. Once the electrodes were in place, metal clamps were lowered until they pressed the upper arm firmly against the upper arm pad. The height of each clamp was measured and recorded for each individual.

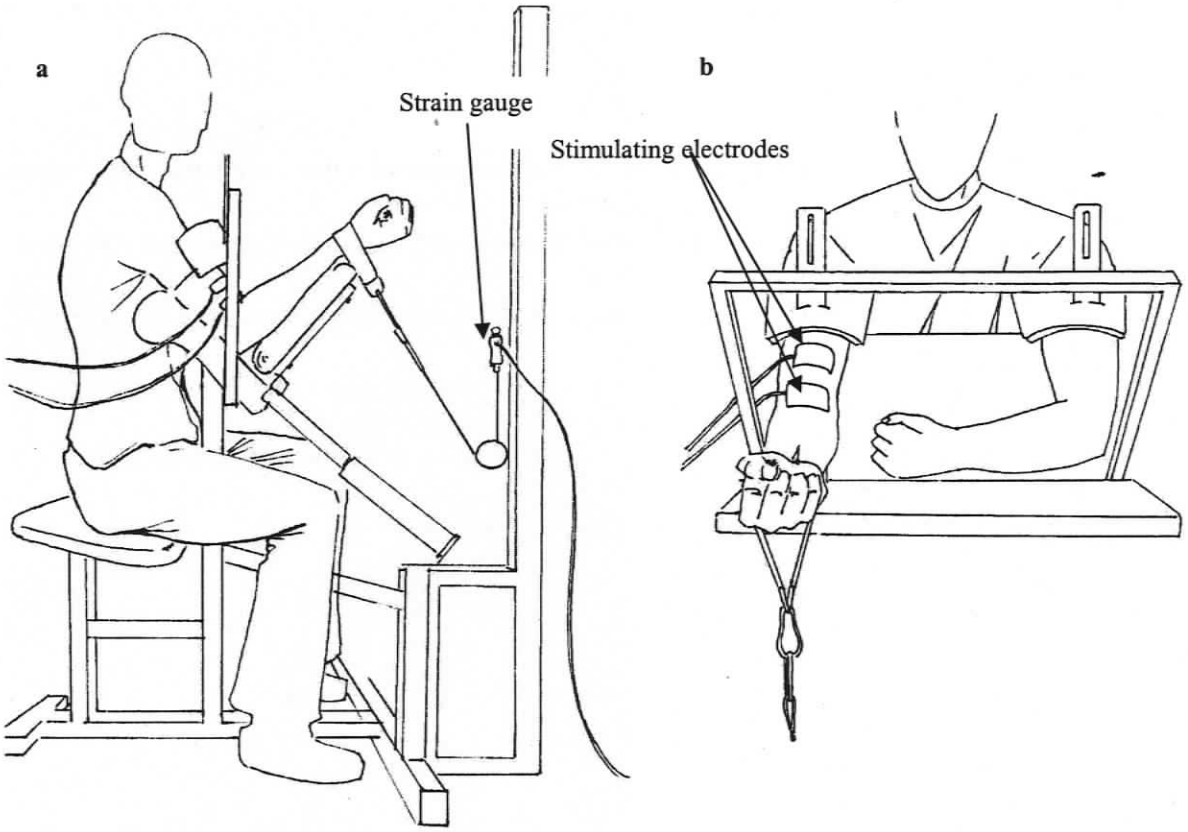

Figure 2.3 Body position on the modified preacher curl apparatus from the side (a) and front (b). (Tran, Docherty, & Behm 2006)

2.7 Supramaximal Stimulus (SMS)

The SMS was determined from an initial low (amperage) stimulation of a constant 100V rectangular pulse. Amperage was progressively increased on consecutive trials until no

further increase in twitch amplitude was detected. Stimulation was conducted in 10s intervals. Torque values were sent through a strain gauge (Omegadyne Ltd. Model 101-500, range 0-500lbs) and amplified by a high gain amplifier (Biopac Systems Inc. DA100 and MP100), with the resulting force displayed with the Acknowledge 3.5 software (Aquasonic). The minimum external electrical stimulus that can elicit the greatest muscle contractile force, during a resting condition, was considered the SMS.

2.8 Interpolated Twitch Technique (ITT)

The ITT was conducted to measure the extent of muscle activation. Pre-ITT consisted of two maximal contractions separated by 3 min rest. Post-ITT consisted of one MVIC at 1 min post fatigue. The shorter post ITT was used to minimize the effects of recovery (Behm et al., 2002). Each contraction was 3s in duration, two doublets were delivered at 1.5s and 3s of the contraction. Participants were instructed to cease contracting after the second doublet. A third doublet was delivered at rest following each contraction (Figure 2.4). A doublet was used (2 twitches interspaced by 10 ms) to increase the signal to noise ratio (McKenzie & Gandevia, 1991).

Muscle activation was calculated using the following formula (Gandevia, 2001).

$$\text{Voluntary activation} = 100(1-\text{Tinterpolated}/\text{Tcontrol})$$

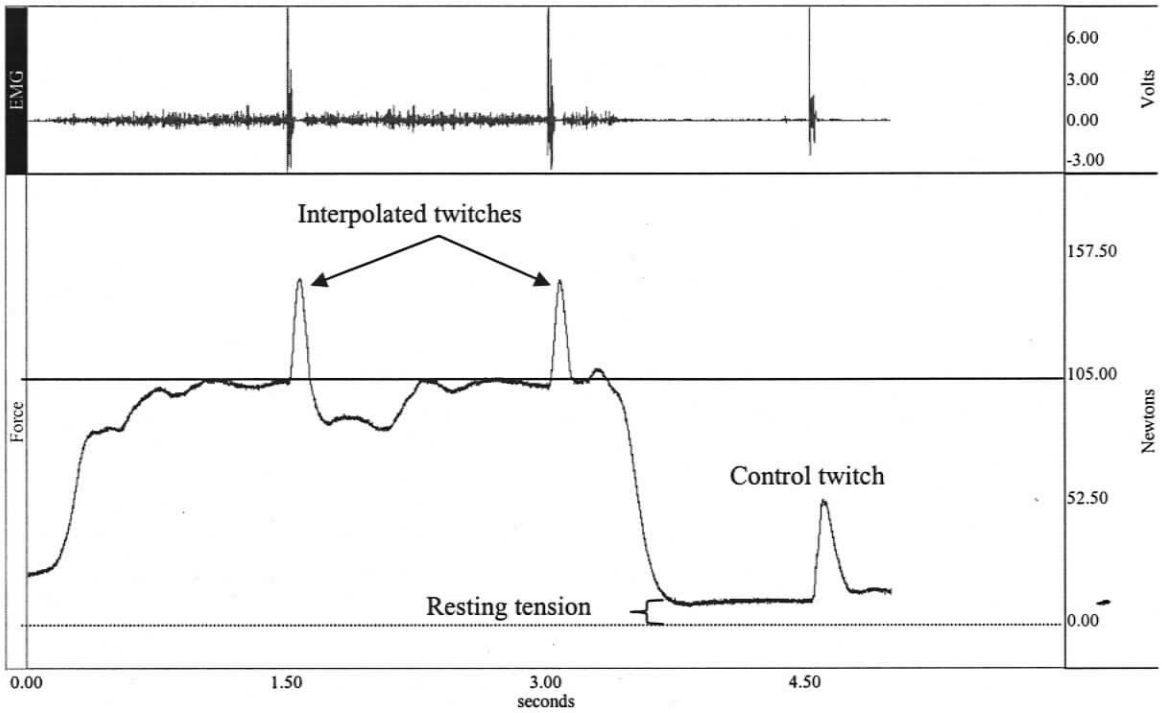

Figure 2.4 Raw data of the interpolated twitch technique of a participant during a MVIC isometric contraction with 3 doublet twitches interspaced by 1.5 s. The top and bottom lines represents EMG activity and force, respectively.

2.9 Maximal Voluntary Isometric Contraction (MVIC)

Participants performed 2 pre-MVIC, separated by 3 min rest periods, and one post-MVIC. The average of the peak pre-MVIC forces were measured (non-twitched forces). Maximal voluntary isometric contractions were measured 1 min post-fatigue protocol. All MVIC attempts were 3s in duration (Tran et al., 2006).

2.10 Twitch Contractile Properties

Participants were instructed to remain fully rested and to close their eyes to prevent anticipation of the stimulus. All measures were performed during a fully relaxed state in order to control for neural activation. Thus, the twitch contractile properties are representative of peripheral changes. All twitch contractile properties were measured from a single stimulation

(singlet). Peak twitch (PT) is considered to reflect excitation-contraction (E-C) coupling. Time to peak twitch (TPT) and half relaxation time (½RT) represent calcium (Ca^{2+}) kinetics, specifically sequestering and reuptake at the sarcoplasmic reticulum (SR), respectively (Ørtenblad, Sjøgaard, & Madsen, 2000). Peak twitch was the greatest force evoked by the singlet, TPT was the time from the onset of stimulation to the PT, and ½RT is the time taken for PT to decrease by half its amplitude (Figure 2.5).

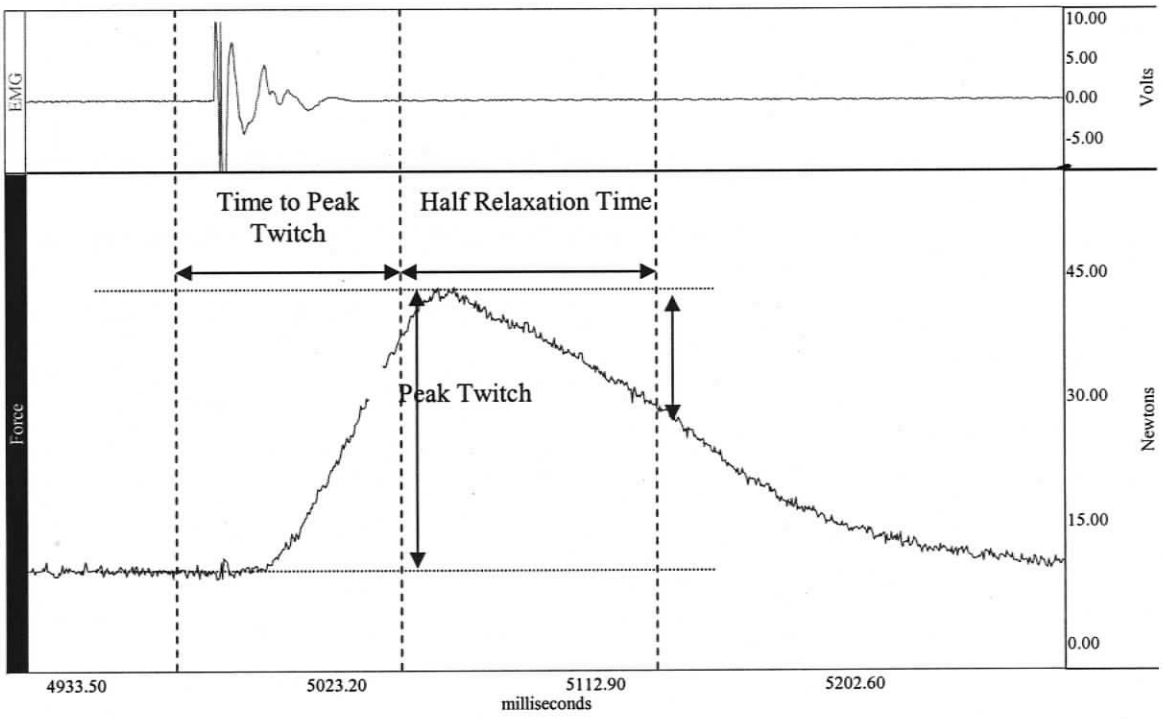

Figure 2.5 Time to peak twitch, peak twitch, and half relaxation time measurements on a single twitch elicited during rest. The top and bottom lines represent EMG activity and force, respectively.

2.11 Statistical Analysis

Means and standard errors were used to describe all data. Data were analyzed using a 3 way analysis of variance (ANOVA) with repeated measures (2x2x2). ANOVA levels included

RM (5 and 10), time (pre- post test), and training pattern (ascending and descending). F ratios were considered significant at $p \leq 0.05$. Effect size via the Cohen's D method were calculated across all protocols, effect sizes were classified as either trivial (<0.25), small (0.25-0.50), moderate (0.50-1.0) or large (>1.0) (Rhea, 2004). If significant interaction effects were present, post hoc analysis using students t-test were conducted using the Bonferroni Inequality to control for the level of type I error ($p \leq 0.01$). An n= 21 gave adequate statistical power as calculated by G*Power (Dr Grant Devilly, Department of Criminology, University of Melbourne, Australia). All statistical procedures were performed using SPSS for Windows version 14 statistical software (SPSS, LEAD Technologies Inc., USA).

3 RESULTS

3.1 Subject characteristics

The physical characteristics of the 21 male participants are listed in Appendix B. The age of the participants ranged from 19 to 37yrs (24.67±3.45yrs), weight ranged from 70.0kg to 114.2kg (86.40±11.01kg), 10RM weight ranged from 11.25kg to 19.25kg (14.32±2.19kg) and 5RM weight ranged from 14.75kg to 25.00kg (19.46±2.53kg).

3.2 Fatigue

As seen in Figure 3.1 there was a significant main effect (time) for all protocols. A similar decrease in force production was found across both loads and training patterns due to the training protocols. Post hoc analysis indicated (5RM descending (5RMD), 5RM ascending (5RMA), 10RM descending (10RMD), and 10RM ascending (10RMA)) significant decreases in isometric force output from pre- to post-values ($p<0.05$). Moderate effect sizes were found across all protocols (0.834-1.151).

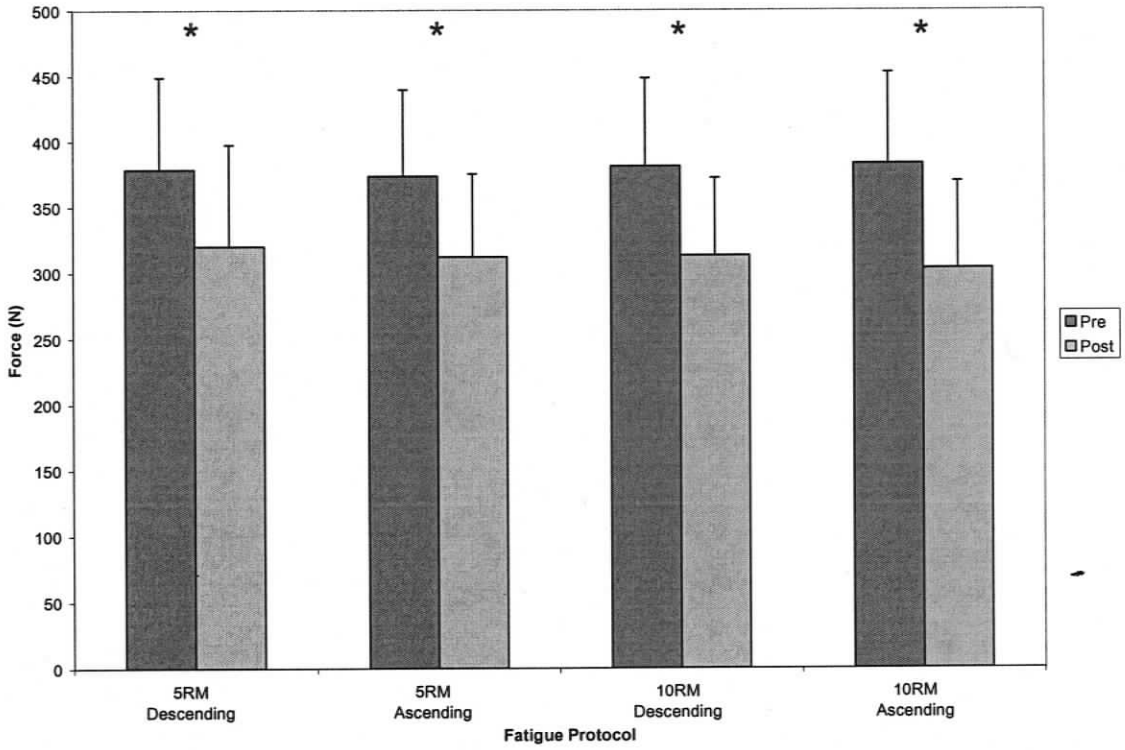

Figure 3.1 Maximum voluntary isometric contraction measured pre- and 1 min post-completion of each fatiguing protocol. Vertical lines represent standard deviation of the means. Asterisk (*) denotes significant difference from pre- to post-training protocol (p=<0.05).

3.3 Motor Unit Activation

Mean muscle activation values, across all protocols, indicated that participants were able to achieve full or near full activation (98.03 ± 1.59%). There was a significant main effect (time) for all protocols (Figure 3.2). A decrease in motor unit activation due to the fatiguing protocol was demonstrated across all loads and training patterns. Ascending training patterns lead to the largest decreases in motor unit activation from pre- to post-training protocol. Post hoc analysis indicated (5RMA, 10RMD, and 10RMA) significant decreases in motor unit

activation from pre- to post-values ($p<0.05$). Moderate to large effect sizes were found across protocols (1.065-3.136).

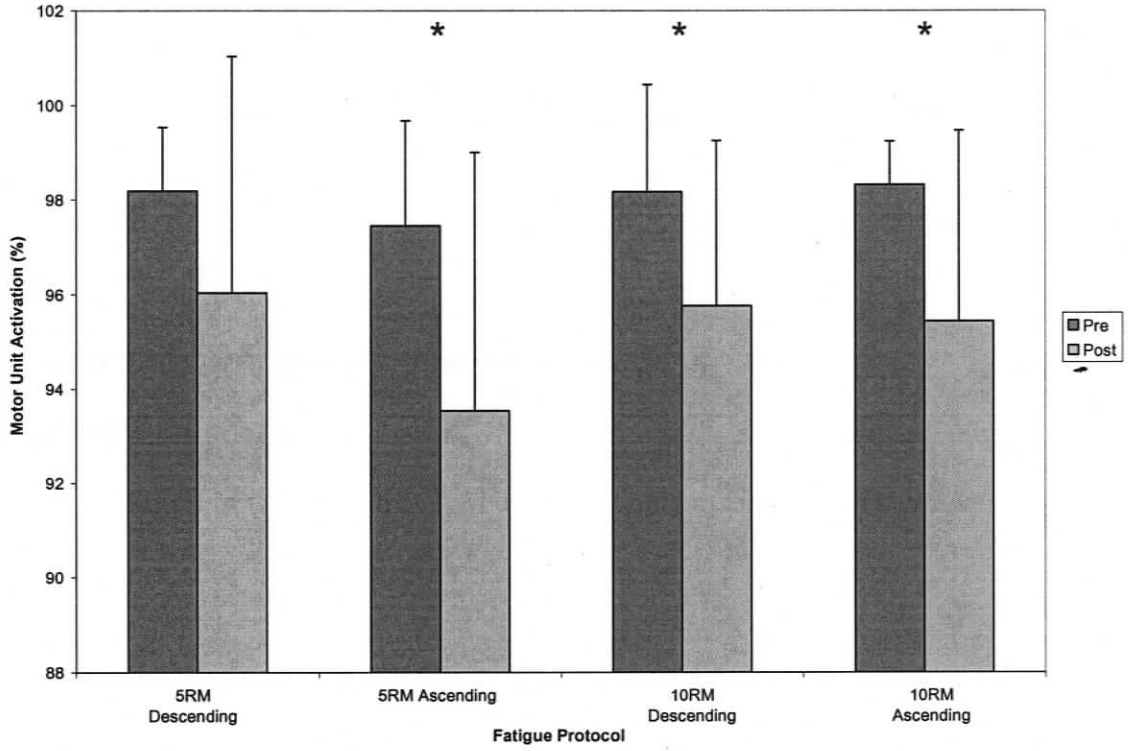

Figure 3.2 Motor unit activation measured via the ITT pre- and 1 min post-completion of each fatiguing protocol. Vertical lines represent standard deviation of the means. Asterisk (*) denotes significant difference from pre to post ($p<0.05$).

3.4 Twitch Characteristics

3.4.1 Peak Twitch

As seen in Figure 3.3 there was a significant interaction effect (load x pattern) and post hoc comparisons indicated that 5RMD and 5RMA produced significant increases in peak twitch force when compared to 10RMD and 10RMA, respectively ($p<0.05$). Depending on RM load, peak twitch either increased pre- to post-fatigue (5RM) or decreased pre- to post-fatigue (10RM). Small to moderate effect sizes were found across protocols (0.4024-0.978).

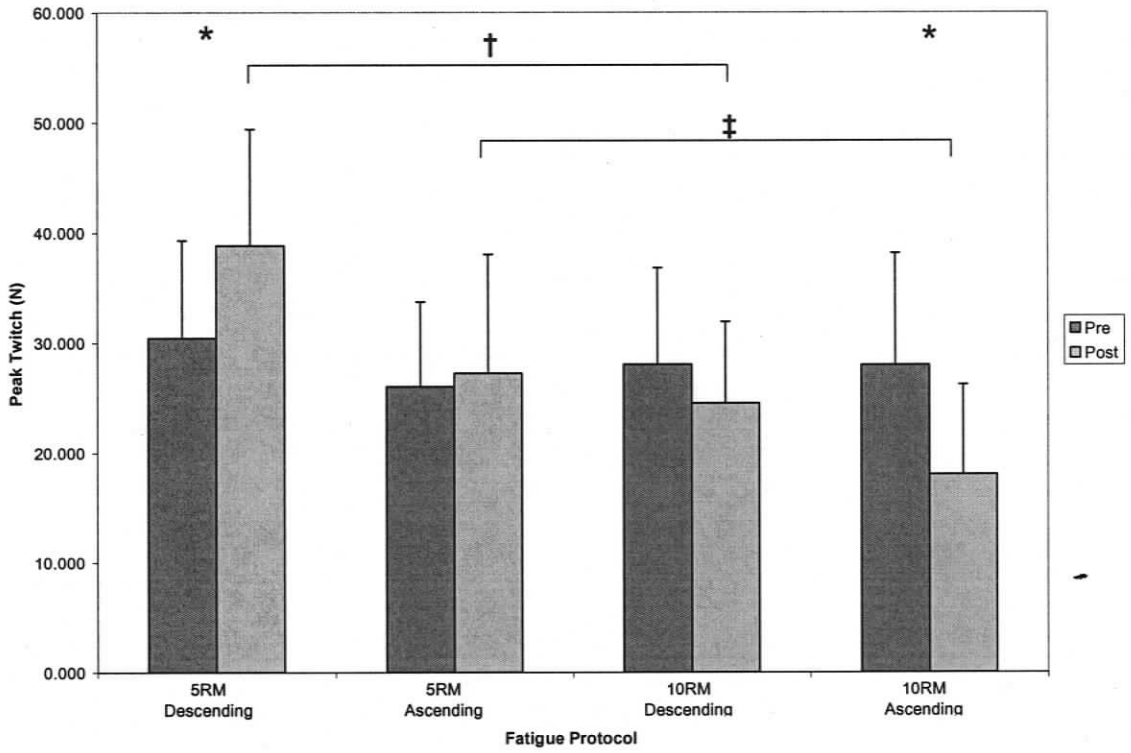

Figure 3.3 Peak twitch forces measured pre- and post-fatiguing protocol. Vertical lines represent standard deviation of the means. Asterisk (*) denotes significant difference from pre to post ($p<0.05$). (†) denotes significant interaction effect (load x pattern) between 5RMD and 10RMD. (‡) denotes significant interaction effect (load x pattern) between 5RMA and 10RMA.

3.4.2 Time to Peak Twitch

There was a significant main effect for time for all protocols (see Figure 3.4). A decrease in the time required to achieve peak twitch was observed across all loads and training patterns. Post hoc analyses indicated (5RMD, 5RMA, 10RMD, and 10RMA) significant decreases in time to peak twitch from pre- to post-values ($p<0.05$). Moderate to large effect sizes were found across protocols (1.219-1.764).

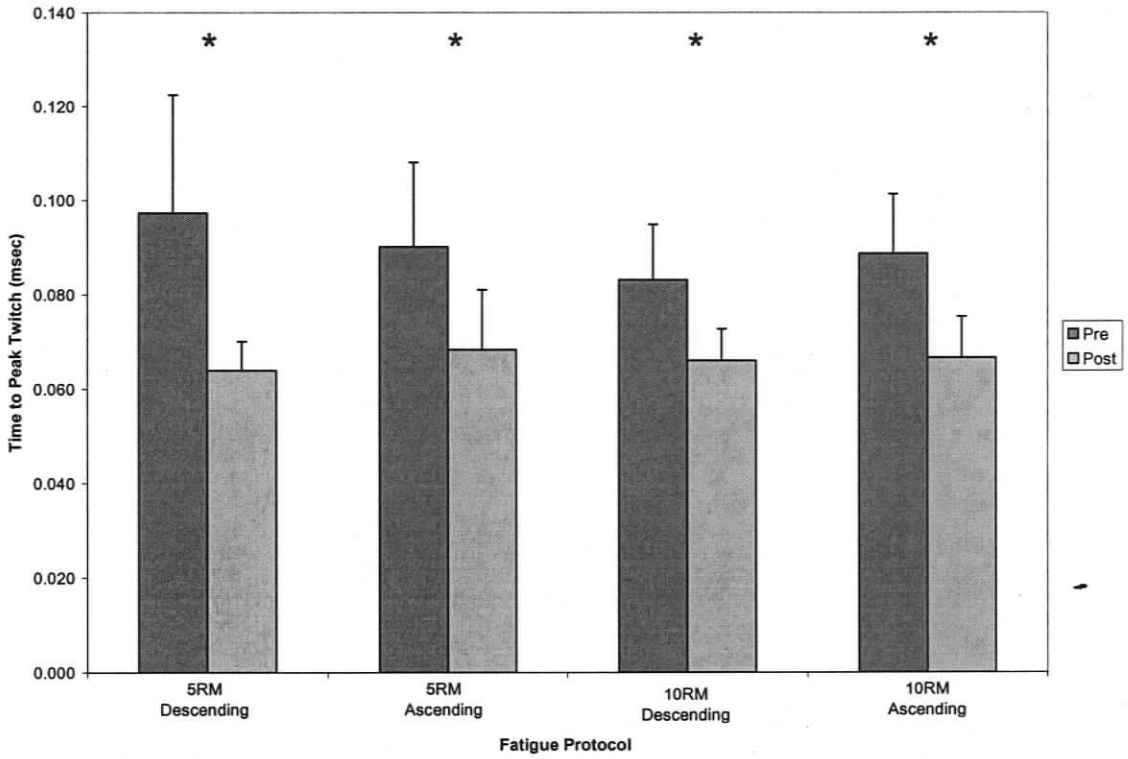

Figure 3.4 Time to peak twitch measured pre- and post-fatiguing protocol. Vertical lines represent standard deviation of the means. Asterisk (*) denotes significant difference from pre- to post-values ($p<0.05$).

3.4.3 Half Relaxation Time

As seen in Figure 3.5 there was a significant main effect (time) for all protocols. Less time was required to achieve half relaxation across all loads and training patterns due to the fatiguing protocol. Post hoc analyses indicated (5RMD, 5RMA, 10RMD, and 10RMA) significant decreases in half relaxation time from pre- to post-values ($p<0.05$). Large effect sizes were found across protocols (1.402-1.622).

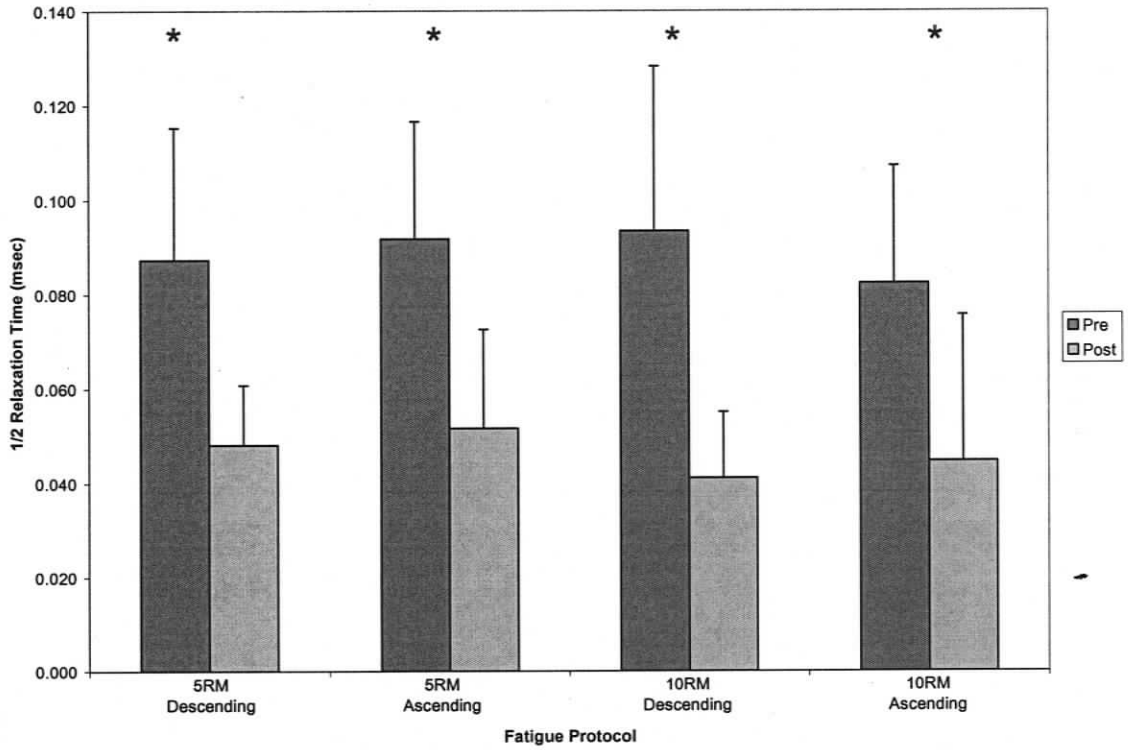

Figure 3.5 Half relaxation time measured pre- and post-fatiguing protocol. Vertical lines represent standard deviation of the means. Asterisk (*) denotes significant difference from pre to post ($p<0.05$).

4 DISCUSSION

The purpose of the study was to examine the acute neuromuscular responses to resistance training protocols that varied in load and training pattern. Subjects trained in an acute manner with multiple sets of 5RM and 10RM loads across two separate training patterns; one in which the volume load was highest in the initial sets (descending pattern) and one in which the repetitions for each set were reversed with the highest number of repetitions occurring in the final sets (ascending pattern). Volume load was equated within patterns for the 5RM and 10RM training loads. The major finding of this study was that manipulation of the training load influenced the acute markers of fatigue. The force producing capacities of the subjects significantly decreased pre- to post-training. Voluntary force production levels decreased on average by 17.6%. Muscle contractile properties were found to be dependent upon the training load whereas motor unit activation was independent of both training load and pattern. 5RM loads resulted in potentiation of muscle contractile properties whereas 10RM loads resulted in the depression of these properties. Training programs designed to induce muscular failure with varying loads will, therefore, lead to fatigue as a result of different mechanisms. In regard to neural fatigue, the magnitude of decline in central drive was similar for both the heavier (5RM) and lighter loads (10RM). Consequently, training loads typically thought not to induce neural fatigue (8-15RM) may actually result in similar neural decrements as those induced by more traditional neural fatigue loads (1-6RM).

4.1 Muscle Contractile Properties

4.1.1 Response to 5RM and 10RM Loads

In regard to Peak Torque (PT) kinetics it was found that heavier loads (5RM) produced a potentiated response within the muscle compared to the lighter load (10RM). Potentiation in

PT following low volume-high intensity training is consistent with other research (Nordlund, Thorstensson, & Cresswell, 2004; Behm, Button, Barbour, Butt & Young, 2004; Pasquet, Carpentier, Duchateau & Hainaut, 2000). The twitch force potentiation that can coexist with decrements in force has been suggested to be a fatigue resisting phenomenon. Twitch potentiation has been attributed to regulatory light chain (R-LC) phosphorylation (Grange, Vandenboom & Ho, 1993; Vandenboom & Houston, 1996) which increases the number of force producing crossbridges exposed to Ca^{2+} through increased Ca^{2+} sensitivity of the cross bridges. Phosphate binding induces a conformational change in the myosin molecule near the head and hinge location. This conformational change can increase the rate at which the myosin crossbridge moves from a non-force producing state to a force producing state (Hodgson, Docherty & Robbins, 2005).

The depression of peak twitch response is in agreement with other studies (Nordlund et al., 2004; Klass, Guissard, & Duchateau, 2003; Behm, Reardon, Fitzgerald & Drinkwater, 2002; Pasquet et al., 2000) who also found PT depression with higher training volumes. Alteration of E-C coupling can be a result of a decrease in Ca^{2+} released from the SR due to structural disruption of the SR, decreased Ca^{2+} ATPase activity, and/or a decrease in Ca^{2+} release channels due to ionic flux and/or structural damage (Pasquet et al, 2000; Behm et al., 2002; Klass el al., 2003). Impaired E-C coupling could be due to a reduction in the sensitivity of Ca^{2+} to its crossbridge binding site as a result of fatigue-related metabolic changes (Pasquet et al., 2000; Klass et al, 2003). The decrease in PT could also have been due to acute muscle damage caused by the training sets and the depletion of energy stores within the muscle due to a greater time under tension when compared to the 5RM loading protocol (Pasquet et al,).

Significant decreases in TPT and ½ RT were found to occur between the pre- and post-training protocols although no significant interactions were found between loads or training patterns. As a result of the 5RM load increasing PT, it might be expected that TPT and ½ RT would increase. However, potentiation probably increased the rate constant of crossbridge formation which would increase the rate of force development. This would support the theory (Grange, Vandenboom & Ho, 1993; Vandenboom & Houston, 1996) that potentiation is a result of R-LC phosphorylation increased Ca^{2+} binding sensitivity rather than an increase in the release of Ca^{2+} from the sarcoplasmic reticulum (SR). An increase in the release of Ca^{2+} from the SR would be expected to increase TPT and ½ RT due to a greater release time of stored Ca^{2+} and sequestering time of the free Ca^{2+} (Grange, Vandenboom & Ho, 1993; Vandenboom & Houston, 1996). The decrease in TPT and ½ RT with higher repetition training loads (10RM) are in agreement with Behm et al. (2002) who also found that a depression in PT resulted in a decrease in TPT and ½ RT. The decrease in TPT and ½ RT can be attributed to a decrease in the amount of Ca^{2+} released from the SR due to various metabolic disturbances (such as H+ accumulation, increase in ADP, and lactate concentration), free radical build up, decreased energy stores, ionic disruptions, and structural damage to the SR and its release gates (Favero, 1999) resulting in less Ca^{2+} available to bind with the troponin C regulatory protein and therefore less Ca^{2+} needed to be sequestered back to the SR.

Muscle stiffness has been observed to increase due to fatigue. Increased stiffness can arise due to changes in the viscosity of skeletal muscle tissue, agonist/antagonist interactions, edema, and reflex activity (Behm et al., 2002). An increase in muscle stiffness can lead to impairment in the sequestering of Ca^{2+}. Increased muscle stiffness can result from residual Ca^{2+} remaining bound to the troponin C regulatory protein resulting in a prolonged

maintenance of the cross-bridge state. Increased residual cross-bridges post-fatigue will increase the series elasticity of the active muscle tension and can therefore accelerate the rates of twitch tension and relaxation. This increased stiffness may help to maintain force output even in the presence of fatigue induced biochemical conditions which are not conducive to muscular contraction.

4.1.2 Responses to Ascending and Descending Training Patterns

No significant differences were found in muscle contractile properties due to variation in training patterns. Since volume load was equated within patterns it appears that there is no difference in the contractile fatigue response due to variation in the set in which the greatest volume of work is conducted when using multiple sets in training.

Although no significant differences were found, a trend was displayed with respect to training patterns. Descending training patterns produced a potentiation-trend for muscle twitch characteristic. This trend is most apparent when comparing effect sizes for the two training pattern treatments. When fatigued with heavier loads (5RM) muscle twitch characteristic effect size for the descending training patterns resulted in a 6x greater response when compared with the ascending effect size (5RMD = 0.949, 5RMA = 0.155). When fatigued with moderate loads (10RM) muscle twitch characteristic effect size for the descending training patters resulted in a 2x greater response when compared with the ascending effect size (10RMD = - 0.402, 10RMA = -0.978,). This trend could be due to the result of the highest volume of work being completed closer to the post-testing in the ascending training pattern compared to the descending pattern. The greater potentiation trend found in the 5RM patterns when compared to the 10RM patterns (6x greater vs. 2x greater) may also be a result of the 5RM training patterns training at a lower volume but higher intensity then the 10RM patterns.

As the 10RM training patterns resulted in a greater total number of repetitions when compared to the 5RM training patterns the time under tension in the 10RM training patterns was greater than experienced in the 5RM training patterns. Training protocols with higher time under tension haves been shown to induce greater levels of peripheral fatigue (Tran et al., 2006).

4.2 Motor Unit Activation

All participants were able to achieve full or near full muscle activation (98.03 ± 1.59%) of the elbow flexors. These results agree with other studies that have reported high levels of muscle activation of the elbow flexors (Allen et al. 1998 [99.1%]; and Gandevia, Herbert, & Leeper 1998 [98%]). Fatiguing protocols (5RMA, 10RMD, 10RMA) resulted in significant changes in muscle activation (95.196 ± 4.493) pre- to post-training which is consistent with some research (McKenzie, Bigland Ritchie, Gorman, & Gandevia, 1992; Behm, Baker, Kelland & Lomond, 2001; Nordlund et al., 2004; and Babault, Desbrosses, Fabre, Michaut & Pousson, 2005) but not consistent with the findings of other research (Bigland-Ritchie, Furbush, & Woods, 1996; Behm & St. Pierre, 1998; Gandevia et al. 1998; Pasquet et al., 2000; Plaskett & Cafarelli 2001; Behm et al, 2002; Klass et al., 2004; Tran et al. 2006). Due to the muscle group studied and the high volume of sets conducted in the fatiguing protocol, decreases in motor unit activation were more readily apparent in this study. The results suggest that, within the context of the present study, individuals were unable to maintain full or near full activation during the development of fatigue which was induced by dynamic efforts of the elbow flexors, suggesting some form of central fatigue. The mechanisms of central fatigue are numerous and include: metabolite accumulation that may inhibit the α-motoneurons; decreased cortical drive to the motoneuron pool; increased activity of group III and IV afferents; presynaptic inhibition of Ia afferents; stretch reflex

disfacilitation; responsiveness of the Golgi tendon organ; and increased recurrent inhibition (Gandevia, 1998, Nordlund et al., 2004; Babault et el., 2005). Some mechanism of central fatigue would appear to be contributing to a decrease in force producing capabilities but the limitations in methodology of the current study prevent identification of any specific source .

4.3 Conclusions

This study found that the magnitude of the load had a differential effect on peripheral fatigue with a moderate load (10RM) producing more peripheral fatigue than heavier loads (5RM). However, the development of central fatigue was independent of the load. PT was found to be potentiated when fatigue was induced with heavy training loads (5RM) compared to moderate training loads (10RM). Moderate training loads resulted in a depression in PT when compared to training with heavier loads (5RM). Motor unit activation was found to be depressed equally, regardless of the training load used (5RM or 10RM), indicating the development of some aspect of central fatigue. This may indicate that strength adaptations are a result of stress being placed on both the peripheral and central components of the neuromuscular system.

Although there was a trend towards greater peripheral fatigue when the highest volume of work was completed in the final sets of training, training pattern did not have any significant affect on the acute neuromuscular responses as measured in this study.

Neural fatigue was similar after training with both a lighter load (10RM) and a heavier load (5RM) indicating that the acute responses to these two loads were similar. Any chronic adaptations that may occur as a result of training with heavier loads may not be due solely to neural adaptations as is often believed by many strength and conditioning practitioners. It has

yet to be determined if any of the acute neuromuscular differences found in the current study produce any chronic neuromuscular adaptations.

5 LITERATURE REVIEW

5.1 Introduction

Muscle fatigue is defined as an exercised-induced reduction in force generating capabilities and may be central or peripheral in origin. Central fatigue refers to a reduction in voluntary activation of muscle during exercise. Fatigue that is produced as a result of impairments in force generating capacities at or distal to the neuromuscular junction has been termed peripheral fatigue (Gandevia, 2001). In dynamic (isotonic) training, by this definition, fatigue may also be referred to as muscular failure. Muscular failure is the point when the weight can no longer be moved, indicating that the force generated can no longer overcome the resistance (failure to complete the next repetition) (MacDougall, Ray, Sale, McCartney, Lee, & Garner, 1999). Failure to maintain the force criterion is due to disruptions within the central and peripheral components (Kent-Braun, 1999).

Recently muscle fatigue has also been described as either high frequency fatigue (HFF) or low frequency fatigue (LFF). HFF is characterized by a rapid loss of force that recovers very quickly once muscle activation is reduced or stopped (Jones, 1996). It has been suggested that HFF is most likely due to disturbances in muscle excitation related to ionic imbalances that are readily reversible. LFF is characterized by a loss of force primarily at lower stimulation frequencies, from which recovery is slow and may persist for hours or days, despite the absence of any disturbances within the muscle (Jones, 1996). It has been suggested that LFF may be due to a wide variety of mechanisms such as metabolic, biochemical and sarcoplasmic reticulum disruption. In order to include the phenomenon of LFF into a working definition certain researchers have accepted the definition of muscular fatigue as a response

that is less that the expected or anticipated contractile response for a given stimulation (Bigland-Ritchie, Furbush, & Woods, 1986).

There is a large amount of evidence indicating that fatigue is not caused by a single factor, but that the mechanisms underlying the force reduction are task specific (Hunter, Duchateau, & Enoka, 2004, Maluf & Enoka, 2005). This means that fatigue can be induced by a combination of processes that contribute in different ways to the decline in forces according to the details of the task (Enoka & Stuart, 1992, Gandevia 2001). Factors such as the training intensity, volume, and duration of the exercise (Lloyd, Gandevia & Hales 1991, Sacco, Thickbroom, Byrnes, and Mastaglia 2000), type of load (Hunter, Lepers, MacGillis, & Enoka 2003), and the fibre type composition of the active muscle (Kawakami, Amemiya, Kanehisa, Ikegawa & Fukunaga 2000, Loscher, Cresswell & Thorstensson 1996)), as well as the type of contraction (Pasquet, Carpentier, Duchateau & Hainaut 2000, Tesch, Dudley, Duvoisin, Hater & Harris 1990), induces specific neural and muscular changes.

5.2 Central Fatigue

Central fatigue is defined as the development of neuromuscular fatigue occurring primarily within the central nervous system and the neuromuscular junction (Green, 1990). Mechanisms implicated in neural fatigue include supraspinal failure (transmission down the spine), disrupted feedback mechanism, recurrent inhibition (Renshaw cells, golgi tendon organ [GTO], antagonist co-activation), and branch point failure (to generate an action potential) of the motor neuron. Disturbances within these neural pathways often alter the excitability of the motorneuron, resulting in a decreased ability (or inability) to conduct a repetitive action potential. The net effect of central fatigue results in low signaling communication to the periphery and decreased motor unit activation (MUA) (Green, 1990; Kent-Braun, 1999).

5.3 Peripheral Fatigue

Peripheral fatigue occurs at the muscle and is the result of disturbances to either excitation or contraction processes (Kent-Braun, 1999). Peripheral fatigue during excitation processes involves the failure of the T-tubule (to conduct a regenerative action potential), decreased excitability of the sarcoplasmic reticulum, and failure of the coupling between the T-tubule and the sarcoplasmic reticulum. The net effect of failure at any one of these sites is reduced Ca^{2+} concentration in the cytosol, which is necessary for activation of muscular contraction (Green, 1990). Peripheral fatigue due to contraction processes are a result of failure at the level of the regulatory proteins (troponin and tropomyosin) and the force-generating proteins (actin and myosin). Decreased force production is due to either a decreased affinity of the regulatory protein for Ca^{2+} (decreased force production for a given concentration of Ca^{2+}) and/or failure of the cross-bridging cycle (Green, 1990).

The ability to perform repeated contractions is dependent on the metabolic processes that fuel the action. Accumulation of lactic acid, as a result of glycolytic metabolism, has been implemented as a contributor to peripheral fatigue (Kent-Braun, 1999). The decrease in pH hinders the excitation-coupling process by decreasing the concentration of Ca^{2+} released into the cytosol and/or interfering with Ca^{2+}-troponin binding.

Another peripheral fatigue mechanism involves the decrease of adenosine triphosphate (ATP), and phosphocreatine (PCr) stores along with adenosine diphosphate (ADP) adenosine monophosphate (AMP) and inorganic phosphate (P_i) stores. ATP is the direct energy source for almost all processes including muscular contraction. PCr is used in the re-synthesis of ATP, and depletion of these stores results in an inability to maintain the force criterion (Foss, & Keteyian, 1998; Kent-Bruan, 1999; Linnamo et al., 1998; MacDougall et al., 1999).

To date various techniques have been used to localize the mechanism of muscular fatigue within human skeletal muscle tissue; electromyogram (EMG), blood lactate measures, twitch properties, M wave, H reflex, ITT, trans-cranial magnetic stimulation (TMS), and muscle biopsies. Each of these methods measures unique aspects of central and/or peripheral fatigue.

5.4 Measuring Peripheral Fatigue

Biopsies can be used to assess multiple factors of muscular fatigue. Biopsies allow for an examination of structural damage to the muscle itself and for a measure of the concentrations of various biochemical products within the muscle such as metabolic by-products, ionic concentrations and neurotransmitter flux (Davis & Bailey, 1997).

Blood lactate is typically measured from capillary sources. Lactate production is a by-product of anaerobic glycolysis, and during periods of high exercise intensity lactate will accumulate within the working muscle which can result in the shut down of both biochemical energy pathways and muscle cross-bridge formation. The concentration of lactate within blood can, therefore, be used to gauge exercise-induced fatigue of the anaerobic energy systems (Ahtiainen, Pakarinen, Kraemer & Hakkinen, 2004). Blood lactate results can be questioned to represent actual lactate levels within skeletal muscle. Drawing from a capillary source (a systemic measure) may not accurately represent localized muscular levels of lactate accumulation.

Twitch proprieties have traditionally been used to interpret calcium kinetics within the muscle cell. These interpretations are based on animal studies examining muscle twitch characteristics while various aspects of calcium kinetics have been impaired. Peak twitch, time to peak twitch and half relaxation time are used to investigate the effects of fatigue on

excitation-contraction coupling (E-C) as well as relaxation and recovery of the muscle to a pre-firing state (Behm, Baker, Kelland & Lomond, 2001, Behm, Reardon, Fitzgerald & Drinkwater, 2002). The use of twitch properties as a representation of peripheral muscle fatigue has come under question. Our knowledge to date of muscle twitch properties has been extrapolated from animal studies where isolated muscle fibres have been examined. It is still considered theoretical whether or not these animal studies accurately represent what is occurring in fatiguing human skeletal muscle.

5.5 Measuring Central Fatigue

The H-reflex is a measure of the electrical activity at the muscle. It is used to assess spinal motorneuron excitability and transmission efficacy in Ia afferent synapses (Aaggaard, Simonsen, Andersen, Magnusson, & Dyhre-Poulsen, 2002). The H-reflex is considered to be the electrical equivalent of the stretch reflex. (Zehr, 2002). A decrease in the recorded H-reflex would occur as a result of decreased excitation of the alpha motoneuron. This would be a representation of central spinal fatigue. The H-reflex is a highly variable measure and requires very strict experimental protocols for its experimentation to be accurate. For research that is examining neurological fatigue it a vital measure as it can aid the researcher in localizing the origin of neurological muscular failure.

The M-Wave is a recorded representation of the compound action potential evoked by a stimulus to the motor nerve (Cupido, Galea & McComas, 1996). Changes in the M Wave would indicate impairment of central neural drive and action potential propagation within the muscle itself due possibly to membrane transmission failure brought about by ionic or neurotransmitter flux (Behm & St.Perre, 1997, Pasquet, Carpentier, Duchateau, & Hainaut, 2000). Similar to the H-reflex, M-wave interpretation is highly variable depending on

experimental protocol. It is also a vital measure of neural fatigue and combined with other neural measure aids in determining the locus of fatigue.

EMG data records a combination of the recruitment of motor units, their firing frequency, and the synchronization of these impulses. EMG was one of the earliest methods used to assess neural drive to a muscle (Behm, 1995). Increases or decreases in EMG activity brought about by fatiguing exercise would represent one or a combination of the following impairment of motor unit recruitment decreased firing frequency or loss of motor unit synchronization (Moritani, Muro, & Nagata, 1986). With more modern methods of neurological testing available EMG is no longer the sole measure of fatigue available. EMG is still used quite extensively in concert with measures such as the H-reflex, M-wave, ITT and TMS as both a fatigue measure and as a tool to refine the other measuring techniques (determining wave or reflex amplitude or temporal location of stimulus volleys).

TMS can be used to examine the motor output from the motor cortex in human subjects. Stimulation over the motor cortex evokes both excitatory and inhibitory response in EMG recordings of contracting muscles (Taylor, Butler, Allen, & Gandevia, 1996). TMS can evoke a short latency motor evoked potential which is a compound muscle action potential in EMG recordings. This latency period arises from direct and trans-synaptic activation of the cortico-spinal neurons and is influenced by the excitability of the motor cortex and the alpha-motoneuron pool. The latency period is due to inhibition of the motor cortical output so the duration of the latency period can reflect the strength of inhibition with the motor cortex. The prolongation of the latency period with respect to muscular fatigue suggests a net increase in inhibition to cortico-spinal neurons. This method allows for the measure of fatigue at the level

of the motor cortex (Taylor et al., 1996, Gandevia, Allen, Butler, Taylor, 1996, Taylor, Allen, Butler, & Gandevia, 2000).

5.6 Twitch Interpolation

When a maximal electrical stimulus is applied to the nerve trunk or intramuscular nerve branches of an active muscle during a maximal voluntary contraction (MVC), those motor units that have not already been recruited respond by generating a twitch response (Belanger and McComas, 1981). Those motor units firing at submaximal rates and whose motoneurons are not in a refractory state respond with a twitch like increment in force (Belanger and McComas, 1981). With increasing neural drive to the muscle, fewer units are available for recruitment, the twitch-like response of others diminishes and the superimposed twitch becomes smaller and ultimately undetectable if the muscle can be fully activated. This technique reflects the central drive to a muscle allowing for the magnitude of any change to be determined, in addition to the peripheral force-generating capacity of the muscle.

The detection of voluntary activation deficits indicates that the central nervous system is contributing to the decrease in force production during and after fatiguing exercise (Gandevia, 1998). Gandevia suggests that the contribution of the central nervous system to fatigue most likely depends on a number of factors including subject motivation, neural control processes affecting the maximal voluntary activation of a muscle, peripheral muscle properties, and the interaction between the intensity and duration of the fatiguing exercise.

5.7 Fatigue and Twitch Interpolation

5.7.1 Plantar Flexors

Many studies using the ITT to investigate the effects of muscular fatigue have been conducted on the plantar flexors. Many of these fatigue studies are of the prolonged or

cumulative fatigue type, in which the muscle is fatigued over an extended period of time at various levels of MVC.

Simpson, Burke, and Davis (2004) investigated the cumulative effect of intermittent MVCs on voluntary activation deficits in the soleus muscle. The soleus muscle was fatigued by having the subject perform 100 isometric plantar flexion contractions at maximal contraction intensities. MVCs were performed in 5 bouts of 20 repetitions with a 2 min rest between each set. Each repetition consisted of a 10 s sustained MVC, followed by a 5s rest period. Voluntary activation of the soleus muscle decrease by 10% after the first 2 min of maximal exercise with a subsequent decrease of 9% occurring after 25 min of maximal exercise. These finding could indicate that the systematic decreases in voluntary activation levels and MVC forces are results of cumulative central fatigue effects. Decreases in activation levels could also be a result of the extended nature of the fatiguing protocol and the untrained nature of the subjects.

Nordlund, Thorstensson and Creswell (2004) also examined central and peripheral fatigue in relation to the level of activation during repeated maximal voluntary isometric plantar flexions. Fatigue was induced by subjects performing nine bouts of 10 MVCs. Each MVC was held for 2s with 1s rest between each MVC and a 10s rest was given between bouts. The fatigue protocol lead to both a significant decrease in force production (-29.0% ± 4.9), level of activation (-12.6% ± 2.8) and resting twitch (-16.2% ± 5.9). Peripheral fatigue measures were also found to correlate with the decrease in plantar flexor strength (r^2=0.57). Greater levels of muscle activation pre fatigue were found to correlate with greater levels of peripheral fatigue post-fatigue. It is believed that this may be related to the fact that a higher level of activation resulted in a faster depletion of energy stores and a more rapid

accumulations of metabolites leading to the impairment of the release and reuptake of Ca^{2+} from the sarcoplasmic reticulum. These results may also be related to the size principal as individuals who are more able to completely activate a muscle fibre will recruit the larger high threshold fibres to a greater extent leading to a more pronounced fatigue deficit within the muscle.

Klass, Guissard, and Duchateau (2004) examined the limiting mechanisms of force production in the triceps surae after repetitive dynamic contractions. Fatigue was induced by performing multiple sets of 30 plantar flexions at a rate of 50 movements per minute; each set was separated by a 15 second rest. The fatiguing protocol ended when the subject was unable to complete 50% of the non-fatigued plantar flexion range of motion for three consecutive contractions. ITT measures, resting twitch proprieties and M wave and H reflex were measure post fatigue. Unlike Simpson et al., (2004) a non significant 2% deficit in voluntary activation along with no change in M wave amplitude was found post-fatigue indicating that central fatigue did not explain the loss of force producing capabilities, indicating that prolonged fatigue protocols may not contribute to neural fatigue. A significant change in H-reflex response was found suggesting that pre-synaptic inhibition did increase post fatigue. A significant decrease in twitch torque was also observed indicating an alteration in excitation contraction coupling caused by a either a decrease in Ca^{2+} release by the sarcoplasmic reticulum and/or by a reduction in the sensitivity of the contractile proteins to Ca^{2+} related metabolic changes that occur during fatigue.

Pasquet, Carpentier, Duchateau, and Hainaut (2000) attempted to differentiate muscle fatigue during concentric and eccentric contractions using untrained subjects. The fatigue protocol involved 5 sets of 30 maximal voluntary concentric or eccentric contractions

separated by 1 min of rest. Muscle contractions were carried out at a constant velocity (50 degrees/sec), rate (1/3.5 sec), and range of motion (30 degrees). Fatigue was achieved when the subject could no longer achieve a significant force output in order to displace the testing mechanism. Similar to Klass et al., (2004) neural activation did not change significantly from pre- to post-testing for either concentric or eccentric fatiguing protocols. Decreases in voluntary force production were attributed to peripheral measures as a significant decrease in peak twitch and twitch half relaxation time were found indicating impairment in the E-C coupling process. In this study concentric fatigue appeared to induce a more rapid fatigue state which was demonstrated by a more rapid and pronounced twitch depression over eccentric fatiguing protocols. A significant increase in half relaxation time was thought to be caused by H^+ accumulation during concentric contraction which acted to inhibit Ca^{2+} pumps and myofibrillar ATPase activity resulting in impaired Ca^{2+} reuptake by the sarcoplasmic reticulum and a decreased rate of cross bridge formation.

A difficulty in interpreting and comparing many of these studies is the various fatiguing protocols that were used. Although some researchers did find central fatigue deficits (Simpson et al., 2004 & Nordlund et al., 2004) others did not (Klass et al., 2004 & Pasquet et al., 2000). Unfortunately, it is not possible to interpret whether these different findings were due to physiological properties of the muscles examined because all four studies used different volumes and intensities to induce fatigue. It does appear that prolonged fatiguing protocols will lead to responses that are more peripheral in origin as oppose to central.

Behm and St-Pierre (1998) studied fatigue mechanisms in trained and untrained subjects to ascertain if the effects of fatigue varied upon individuals depending upon training history. The main difference in this study to the above studies is that contraction intensities

were not maximal, contraction intensity of 50% of MVC of plantar flexion were used for 16 s contraction periods separated by 4 s rest periods. Contraction cycles were continuous till subjects could no longer maintain the target force for the entire 10 s period. The study determined that even though trained individuals were able to attain a higher pre-fatigue force measurement there was no significant difference in muscle activation, peak twitch torque, and ½ relaxation time with respect to training status. Untrained individuals did exhibit a great level of antagonist activity during MVCs. This may indicate that trained individuals are able to decrease antagonist muscle activity allowing for a greater contraction of the agonist muscle group, a proposed neural adaptation to prolonged strength training. Trained subjects also demonstrated a superior ability to resist the effects of fatigue when compared to untrained individuals. This fatigue resistant ability was demonstrated by trained individuals having the ability to maintain a larger percentage of their MVC post-fatigue.

5.7.2 Elbow Flexors

A number of research projects have been conducted upon the elbow flexors muscle group. The studies performed using the elbow flexors have combined both isometric and dynamic isotonic muscular contractions along with high and low training volumes.

Gandevia, Herbert and Leeper (1998) examined voluntary activation of the elbow flexors during maximal concentric contractions. They compared the level of activation achieved in the elbow flexors through isometric and dynamic isotonic contraction as well examining the effects of fatigue on voluntary activation of the elbow flexors. Eleven subjects participated in the comparison study and five subjects participated in the fatigue study. The fatiguing protocol in the fatigue experiment involved subjects performing arm curls with a load between 6-12RM. Subjects performed weighted elbow flexion movement to a set

cadence. At the point where the subject could no longer complete one concentric portion of the lift on their own concentric-only, assistance was provided until the subject completed another 10-12 repetitions. It was found that there was no statistical difference in the level of voluntary activation decrease when comparing maximal isotonic contractions and to maximal isometric contractions. Another finding of this study was that maximal voluntary contraction levels during dynamic concentric contractions were independent of movement speed. No significant decreases in maximal voluntary activation levels were found post-fatigue which suggests that the primary site of fatigue during the fatiguing protocol was located within the muscle and the result of peripheral muscle fatigue factors rather than central mechanisms. It is very difficult to interpret the results of this experiment due to a number of reasons; subjects were untrained individual, volume load was not standard across trials and the overall training volume of this study was quite low (1 set) compared to how most athletes train (3-4 sets).

Behm, Reardon, Fitzgerald and Drinkwater (2002) studied the effects of 5, 10 and 20 RM's on the recovery of both voluntary and evoked contractile properties. It was found that a single set to failure using a 5RM load did not produce greater muscle inactivation then a 10RM or 20RM load. This finding indicates that in this study a 5RM strength load did not induce a greater amount of neural fatigue then either a 10RM or 20RM hypertrophy load. Evoked contractile properties were found be affected to a greater degree by the higher number of repetitions conducted in a fatiguing session, therefore, the higher the number of repetitions in a fatiguing protocol the greater the decrease in calcium function within the muscle. The 20RM fatigue protocol lead to greater deficits in all evoked contractile properties; PT, TPT and ½RT over the 5RM and 10RM fatigue protocols. Higher repetition fatiguing protocols, therefore, result in a more severe decrement in calcium mechanics within the skeletal muscle

cell than lower repetition fatiguing protocols. Like the Gandevia et al., (1998) study only one set of training was used to induce fatigue, it is therefore difficult to extrapolate these results to a true training environment due to the low training volume conducted.

McKenzie, Bigland-Ritchie, Gorman, and Gandevia (1992) examined both central and peripheral fatigue of the human diaphragm and elbow flexors. The elbow flexors were fatigued with three fatigue sequences consisting of 10 MVCs of 10s duration separated by 10 s rest. This fatigue protocol resulted in a 57.9 ± 3.0% decrease in torque whereas voluntary activation significantly decreased from 98.4 ± .4% to 86.8 ± 2.2% pre- to post-fatigue The researchers felt that their results indicated that the elbow flexors are susceptible to aspects of central fatigue; unfortunately, the authors did not test for any aspects of peripheral fatigue and were not able to quantify the contribution of peripheral measures to the overall significant decrease in force producing capability post fatigue. This research indicates that central fatigue may be related to high intensity high volume short rest period training protocols. This protocol did lead to one of the most significant percentage decreases in MUA of any ITT based study.

Behm, Baker, Kelland and Lomond (2001) examined the effects of long term muscle damage on both strength and fatigue. Untrained individuals were used as subjects for this experiment. Testing took place prior to the fatiguing protocol and 1, 3, 5, and 7 days after the exercise session. A high volume fatiguing protocol was used where subjects performed 7 sets of 10 repetitions of elbow flexion at 70% of their 1RM maximum with one minute rest in between sets. Like the Mckenzie et al., (1992) study a significant degree of muscle inactivation (11.1%) was found on day 1 post-fatigue. Although this indicated evidence of central fatigue there was no correlation with strength deficits. These findings also support those of Mckenzie et al., who found significant neural impairment with a high intensity high

volume training program with short rest period. Measures of peripheral muscle damage did correlate with strength deficits on day 1 post-fatigue. Impairment in twitch amplitude indicated the possibility of disruption in E-C coupling mechanisms. Dysfunction in the sarcoplasmic reticulum function has been found to be associated with twitch impairment. Disruption of SR function can lead to altered Ca^{2+} kinetics within skeletal muscle resulting in decreased force production.

Jakobi and Rice (2002) investigated the differences between levels of muscle activation with respect to both age and muscle group. Although no fatigue was induced this study demonstrated that old (83 ± 4 yrs) men were unable to activate their elbow flexors compared to young (24 ± 1 yrs) men. On average older males experience a 11% decrease in muscle activation compared to the young men. The aging process could reflect a form of neural fatigue due to the fact there was a loss of ability to fully activate all motor units within a muscle group with older subjects.

5.7.3 Knee Extensors

A smaller amount of research has been conducted on the knee extensors compared to the elbow flexors using the ITT method. A number of these studies used exercise of a dynamic nature to induce muscular fatigue whereas some studies still relied on isometric contractions as a method of inducing fatigue.

Behm and St. Pierre (1997) investigated the effect of exercise duration on voluntary force production and evoked contractile properties in both the quadriceps and plantar flexors. Active subjects were divided into 4 experimental groups. Each of the four groups was subjected to different MVIC intensities. Two experimental groups performed voluntary contractions at 25% and 50% MVIC with the quadriceps muscle group while the other two

experimental groups performed voluntary contractions at 50% and 70% of MVIC with the plantar flexor muscle group. The difference in contraction intensity per muscle group was due to pilot work results that indicated that the time to fatigue was similar between the two muscle groups at these varying intensities. This was attributed to fibre type make-up of the respective muscles. Groups were then designated as short duration fatigue (quads 50% and plantar flexors 75%) or long duration fatigue (quads 25% and plantar flexors 50%) groups. Continuous contractions at the designated percentage of MVIC were then conducted for 10 sec periods with a 4 s rest between contractions. Fatigue was attained when a subject could no longer maintain the desired percentage of MVIC for the total 10 s contraction duration. The results indicated that the exercise duration exerted its greatest effect on muscle activation and ½RT. Long duration fatigue groups experienced the greatest decrease in muscle activation (12%) versus short duration fatigue groups (5.6%) independent of muscle group. Exercise duration had a major effect on ½RT in the LDF group indicating impairment in Ca^{2+} sequestering by the SR. Because Ca^{2+} sequestering is an active process requiring ATP, the authors believed that this process was most likely to be disrupted by metabolic imbalances caused by prolonged exercise protocols.

Bigland-Ritchie et al., (1986) examined central and peripheral factors that contributed to fatigue in intermittent sub-maximal muscular contractions for the quadriceps muscle group. The fatiguing protocol for this experiment consisted of 6 s voluntary contractions at 50% of MVC followed by 4 s rest. This cycle was continued until the subject was no longer able to attain the targeted % of MVC in two consecutive attempts; on average it took 4.4 ± .6 min before subjects were unable to attain the targeted % of MVC. For the quadriceps muscle group, central fatigue played no role in the diminishment of force production this conclusion

was researched due no apparent decrement in the percentage of motor unit activation. The researchers concluded that peripheral measures or low frequency fatigue contributed to the decrease in force-producing capability caused by a medium-length intermittent fatiguing protocol due to the decrease in the single twitch response measured between low frequency stimulation trains.

Babault, Desbrosses, Fabre, Michaut, and Pousson (2005) examined the development of fatigue during maximal concentric and isometric knee extensions. They had individual fatiguing protocols for each type of muscular contraction. For the concentric protocol subjects performed three sets of 30 maximal concentric leg extensions on an iso-kinetic dynamometer at a joint speed of 60 degrees/second. During the isometric fatiguing session subjects performed three MVCs. The MVCs lasted for as long as it took the torque levels to drop to similar levels as induced by the concentric fatiguing protocol. In this way the researchers attempted to control the decrement in torque between the concentric protocol and the isometric protocol. Post-hoc statistical analysis indicated that neither protocol produced significantly more fatigue than the other. Both levels of activation and twitch properties decreased to a similar extent between both protocols. They did discover that the course of fatigue was different between protocols. The level of muscular activation was found to be decreased to a significantly greater extent ($p<0.05$) by isometric contractions than concentric contractions between the two initial fatigue sets but were similar after the final set of fatiguing exercise. Twitch properties were found to be reduced to a greater extent via the concentric fatiguing protocol early between sets of fatigue compared to the isometric fatiguing protocol ($P<0.05$). It appears that the development of fatigue in its particular locus, peripheral versus central, is inversely related depending on the contraction type. The researchers felt that the continuous

nature of the isometric contractions allowed for metabolites to accumulate within the small diameter afferent fibres effecting alpha motor-neuron inhibition and leading to a reduction in supra-spinal descending neural drive. This also led to an increase in metabolites so that aspects of peripheral fatigue were not seen until later in the testing session compared to the concentric protocol.

5.8 Limitations in the Literature

To date a large majority of studies using the ITT as an evaluation method of central fatigue have focused on the plantar flexors muscle group. The majority of these studies are of a long duration or cumulative fatigue type, where the individual muscle group is fatigued using an extreme fatiguing measure over an extended period of time. These studies have given us varying perspectives of the mechanisms of fatigue within skeletal muscle yet many of the findings are contradictory to one another muddying our overall understanding of the underlying causes of neuromuscular fatigue.

A smaller number of projects have been conducted on larger muscle groups such as the quadriceps and biceps brachii. These studies have attempted to introduce dynamic fatiguing exercise into the fatigue protocols. These studies appear to have more application in their interpretation to applied sports science research as the majority of fatiguing exercise conducted by athletic populations are dynamic in nature. Still many of these studies have failed to standardize testing and fatiguing protocols and therefore yield highly varying results to the mechanism of fatigue.

Future paths of study which will yield results more applicable to applied sports science research must begin look to and take into account the following:

1. Incorporate a greater amount of dynamic exercise in fatiguing protocols

2. Standardize fatiguing methods and manipulate only one training variable at a time
3. Measure both central and peripheral mechanisms of fatigue so the locus of fatigue can be more readily postulated.
4. Work on athletic populations
5. Examined training methods that are typical to athletic populations
6. Incorporate other measures of central fatigue such as M-Wave and H-reflex
7. Begin to monitor subjects over longer periods of time, training studies vs. acute studies

5.9 Summary

The ITT has demonstrated itself to be an effective and reliable measure of central fatigue, researchers must therefore look to continue to use this technique to expand the breath of our knowledge within the domain of neuromuscular fatigue in an applied setting. By expanding our knowledge in the area of muscular fatigue the underlying principles to program design and the methods to which athletes adapt to these programs will be better understood. A greater understanding of the training effect will allow for greater specificity, efficiency and efficacy in program design. Continuing to expand our knowledge of training variables will benefit the sport scientist as well as the athlete.

REFERENCES

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

Ahthiainen, J.P, Parkarinen, A., Kraemer, W.J., & Hakkinen, K. (2004). Acute hormonal responses to heavy resistance exercise in strength athletes versus non-athletes. *Canadian Journal of Applied Physiology 29,* 527-543.

Anderson, T., & Kearney, J.T. (1982). Effects of three resistance training programs on muscular strength and absolute and relative endurance. *Research Quarterly in Exercised and Sport, 53,* 1-7.

Babault, N., Desbrosses, K., Fabre, M.S., Michaut, A., & Pousson, M. (2005). Neuromuscular fatigue development during maximal concentric and isometric knee extensions. *Journal of Applied Physiology, 100,* 78-785.

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

Behm, D.G., Button, D.C., Barbour, G., Butt, J,C, & Young, W.B. (2004). Conflicting effects of fatigue and potentiation on voluntary force. *Journal of Strength and Conditioning Research, 18,* 365-372.

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

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

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

Belanger, A.Y. & McComas, A.J. (1981). Extent of motor unit activation during effort. *Journal of Applied Physiology, 51,* 1131-1135.

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

Campos, G.E.R, Luecke, T.J., Wendeln, H.K, Toma, K., Hagerman, F.C., Murray, T.F., Ragg, K.E., Ratamess, N.A., Kraemer, W.J., & Staron, R.S. (2002). Muscular adaptations to response to three different resistance training regimens: specificity of repetition maximum training zones. *European Journal of Applied Physiology, 88,* 50-60.

Chestnut, J., & Docherty, D. (1999). The effects of 4 and 10 repetition maximum weight-training protocols on neuromuscular adaptations in untrained men. *Journal of Strength and Conditioning Research, 13*(4), 353-359.

Cupido, C.M., Galea, V., & McComas, A.J. (1996). Potentiation and depression of the M wave in human biceps brachii. *Journal of Physiology, 491,* 541-550.

Davis, M.J., & Bailey, S.P. (1997). Mechanisms of central nervous system fatigue during exercise. *Medicine and Science in Sports and Exercise, 29,* 45-57.

Drinkwater, E.J., Lawton, T.W., Lindsell, R.P., Pyne, D.B., Hunt, P.H, McKenna, M.J. (2004). Repetition failure is a key determinant of strength development in resistance training. *Medicine and Science in Sports and Exercise, 38,* S53.

Enoka, R.M., & Stuart, D.G.(1992). Neurobiology of muscle fatigue. *Journal of Applied Physiology, 72,* 1631-1648.

Favero, T.G. (1999). Sarcoplasmic reticulum Ca^{2+} release and muscle fatigue. *Journal of Applied Physiology, 87,* 471-483.

Fleck, S.J,, & Kraemer, W.J. (1997). *Designing resistance training programs*. Human Kinetics, Champaign, Illinois.

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,* 370-373.

Foss, M., & Keteyian, S. (1998). *Fox's Physiology Basis for Exercise and Sport.* (6^{th} ed.). New York: McGraw-Hill.

Gandevia, S.C. (1998). Neural control in human muscle fatigue: Changes in muscle afferents, motoneurons and motor cortical drive. *Acta Physiologica Scandinavica, 162,* 275-283.

Gandevia, S.C. (1998). Voluntary activation of human elbow flexor muscles during maximal concentric contractions. *Journal of Physiology, 512,* 595-602.

Gandevia, S.C. (2001). Spinal and Supraspinal factors in human muscle fatigue. *Physiological Reviews, 81,* 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,* 529-536.

Gandevia, S.C., Herbert, R.D., & Leeper, J.B. (1998). Voluntary activation of human elbow flexor muscles during maximal concentric contractions. *Journal of Physiology, 215,* 595-602.

Gollnicks, H. Green, D. Ianuzzo, E. Noble, G. Metivier, & J. Suttons (Eds). *Biochemistry of Exercise VII* (pp. 13-35). Champaign, IL: Human Kinetics.

Grange, R.W, Vandenboom, R., & Ho Ston, M.E. (1993). Physiological significance of myosin phosphorylation in skeletal muscle. *Canadian Journal of Applied Physiology, 18,* 229-243.

Green, H.J. (1990). Manifestations and sites of neuromuscular fatigue. In: *Biochemistry of Exercise*, Taylor, E.W., Gollnick P.D. et al (Ed.). Champaign, IL: Human Kinetics, 15-35.

Häkkinen, K., & Pakarinen, A. (1993). Acute hormonal response to two different fatiguing heavy-resistance protocols in male athletes. *Journal of Applied Physiology, 74*, 882-887.

Hodgson, M., Docherty, D., & Robbins, D. (2005). Post activation potentiation underlying physiology and implications for motor performance. *Sports Medicine, 35,* 585-595.

Hunter, S.K, Lepers, R., MacGillis, C.J. & Enoka, R.M. (2003) Activation among the elbow flexor muscles differ when maintaining arm position during a fatiguing contraction. *Journal of Applied Physiology, 94,* 2439-2447.

Hunter, S.K, Duchateau, J. & Enoka, R.M. (2004) Muscle fatigue and the mechanisms of task failure. *Exercise and Sport Science Reviews, 32,* 44-49.

Izquierdo, M., Ibanez, J., Gonzalez-Badillo, J.J, Haikkinen, K., Ratamess, N.A., Kraemer, W.J., French, D.N., Eslava, J., Altadill. A., Asiain, X., Gorostiaga, 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,* 1647-1656.

Jakobi, J.M., & Rice, C.L. (2002). Voluntary muscle activation varies with age and muscle group. *Journal of Applied Physiology, 93,* 457-462.

Jones, D.A. (1996) High and low frequency fatigue revisited. *Acta Physiologica Scandinavia, 156*, 265-270.

Kawakami, Y., Abe, T., Yuno, S., Fukunage, T. (1995) Training induced changes in muscle architecture and specific tensions. *European Journal of Applied Physiology, 72*, 37-43.

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,* 1969-1975.

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

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

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

Lloyd, A.R., Gandevia, S.C., & Hales, J.P. (1991). Muscle performance, voluntary activation, twitch properties and perceived effort in normal subjects and patients with the chronic fatigue syndrome. *Brain, 114,* 85-98.

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

Maluf, K.S. & Enoka, R.M. (2005) Task failure during fatiguing contractions performed by humans. *Journal of Applied Physiology, 99,* 389-396.

MacDougall, J., Ray, S., Sale, D., McCartney, N., Lee, P., & Garner, S. (1999). Muscle substrate utilization and lactate production during weightlifting. *Canadian Society for Exercise Physiology, 24*, 209-215.

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

Moritani, T., Muro, M., & Nagata, A. (1986). Intramuscular and surface electromyogram changes during muscle fatigue. *Journal of Applied Physiology, 60,* 1179-1185.

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

Pasquet, B., Carpentier, A., Duchateau, J., & Hainaut, K. (2003). Muscle fatigue during concentric and eccentric contractions. *Muscle and Nerve, 23,* 1727-1735.

Plasket, C. J., & Cafarelli, E. (2001). Caffeine increases endurance and attenuates force sensation during submaximal isometric contractions. *Journal of Applied Physiology, 91*, 1535-1544.

Rhea, M.R. (2004). Determining the magnitude of treatment effects in strength training research through the use of the effect size. *Journal of Strength and Conditioning Research, 2004, 18,* 918-920.

Rooney, K., Herbert, R., & Balnave, R. (1994). Fatigue Contributes to the Strength Training Stimulus. *Medicine and Science in Sport and Exercise, 26*, 1160-1164.

Sacco, P., Thickbroom, G.W., Byrnes, M.L., & Mastaglia, F.L. (2000). Changes in corticomotor excitability after fatiguing muscle contractions, *Muscle and Nerve, 23,* 1840-1846.

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

Simpson, M., Burke, J.R., Davis, J.M (2004). Cumulative effects of intermittent maximal contractions on voluntary activation deficits. *International Journal of Neuroscience, 114,* 671-692.

Stone, W.J., & Coulter, S.P. (1994). Strength/endurance effects from three resistance training protocols with women. *Journal of Strength and Conditioning Research, 8,* 231-234.

Tan, B. (1999). Manipulating resistance training program variables to optimize maximum strength in men: a review. *Journal of Strength and Conditioning Research, 13*, 289-304.

Taylor, J.L., Butler, J.E., Allen, G.M., & Gandevia, S.C. (1996). Changes in motor cortical excitability during human muscle fatigue. *Journal of Physiology, 490,* 519-528.

Taylor, J.L., Allen, G.M., Butler, J.E., & Gandevia, S.C. (2000). Supraspinal fatigue during intermittent maximal voluntary contractions of the human elbow flexors. *Journal of Applied Physiology, 89,* 305-313.

Tesch, P.A., Dudley, G.A, Duvoisin, M.R., Hather, B.M., & Harris, R.T. (1990). Force and EMG signal patterns during repeated bouts of concentric or eccentric muscle actions. *Acta Physiologica Scandinavia, 138,* 263-271.

Toigo, M., & Boutellier, U. (2006). New fundamental resistance exercise determinants of molecular and cellular muscle adaptations. *European Journal of Applied Physiology, 97,* 643-663.

Tran, Q., 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,* 402-410.

Vandenboom, R., & Houston, M.E. (1996). Phosphorylation of myosin and twitch potentiation in fatigued skeletal muscle. *Canadian Journal of Physiology and Pharmacology, 74,* 1315-1321.

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

TABLE OF APPENDICES

Appendix A - Informed Consent Form- this document was read and signed by each participant prior to beginning the study to ensure that each individual understood their personal rights when participating in the study.

UNIVERSITY OF VICTORIA
OFFICE OF THE VICE-PRESIDENT, RESEARCH
HUMAN RESEARCH ETHICS COMMITTEE

Participant Consent Form

5.9.1 The effects of varying levels of fatigue on acute neuromuscular responses.

You are being invited to participate in a study entitled **The effects of varying levels of fatigue on acute neuromuscular responses** that is being conducted by Tyler Goodale who is a graduate student in the School of Physical Education at the University of Victoria and you may contact him if you have further questions by either phone (361-3966) or email (tygoodale@hotmail.com). As a graduate student, I am required to conduct research as part of the requirements for a degree in Master's of Science. It is being conducted under the supervision of Dr. David Docherty. You may contact my supervisor at 721-8375 or docherty@uvic.ca.

The purpose of this research project is to examine the effects of varying levels of fatigue on acute neuromuscular responses following dynamic constant external resistance (DCER) elbow flexion, using dumbbells, at a two loads (5RM and 10RM).

To date no study has examined the acute neuromuscular effects of going to fatigue in multiple sets compared to going to fatigue in only the final set of multiple set training. With respect to the prescription of training programs the effects of fatigue are still not well understood. There are a number of strength training variables thought to be important to training such as volume, intensity, tempo, and the degree of fatigue to long term neuromuscular response. The importance of neuromuscular fatigue to the strength training stimulus is still not well understood.

Before undertaking long term training studies on the effects of fatigue it is necessary to understand the acute response of the neuromuscular system to different fatigue training protocols. Within physiological research it is important to not only state a change has occurred but also why a change may have occurred. With a better understanding of the neurological and metabolic response to acute training bouts, long term training programs can be better designed to maximize the adaptation.

You are invited to participate in this study because you have participated in a minimum of one year of upper body resistance training, approximately three times per week, prior to beginning the study.

If you agree to voluntarily participate in this research, your participation will include performance of various exercise protocols and the administration of the interpolated twitch technique (ITT). The ITT involves a brief electrical stimulus to the biceps brachii during a fully relaxed and maximal voluntary contracted state. Comparison between the twitches obtained during these states will give an estimate of muscle activation.

The experiment will consist of four exercise protocols of DCER elbow flexion designed to manipulate the levels of fatigue. You will be required to participate in a minimum of four familiarization sessions. A fifth familiarization session will be conducted if there is a greater than 5% difference in the acquired 5 or

10RM. After satisfactory completion of the familiarization sessions, participants will perform each exercise protocol on separate days with approximately 24-72hrs between testing sessions. You will perform the ITT to measure muscle activation pre and post each exercise protocol.

Participation in this study may cause some inconvenience to you because of the time commitment, approximately 8 hours for 8 sessions over a period of 2-3 weeks. Although very unlikely, you may also experience some discomfort during the mild electrical stimulus during the ITT.

Following the testing sessions, you may experience some minor muscle soreness. Gentle stretching of the affected muscles should alleviate the stiffness. If the muscle soreness persists for more than 48 hours or if you experience discomfort greater than normally encountered during a regular training session please inform the investigator (361-3966) and the test will be terminated. You can either reschedule another appointment to complete the test or withdraw from the study.
Your participation in this research must be completely voluntary. If you do decide to participate, you may withdraw at any time without consequence or explanation. If you do withdraw from the study your data will not be used in the study.

In terms of protecting your anonymity, your data will be stored by assigning a code number to the data sheet rather than a name. Only the principal investigator and the supervising professor will have access to the data.

Your confidentiality and the confidentiality of the data will be protected. All information collected during the study will only be accessible by the principal investigator or supervisor and personal results will not shared without your consent.

Data from this study consisting of raw data and electronic files (recorded onto a CD) will be stored in a personal locked file cabinet for a minimum of 5 years. Upon completion of the retention period, the documents will be shredded and the CD destroyed.

It is anticipated that the results of this study will be shared via a thesis paper and published article.

In addition to being able to contact the researcher at the above phone numbers, you may verify the ethical approval of this study, or raise any concerns you might have, by contacting the Associate Vice-President, Research at the University of Victoria (250-472-4362).

Your signature below indicates that you understand the above conditions of participation in this study and that you have had the opportunity to have your questions answered by the researchers.

_____	_____	_____
Name of Participant	*Signature*	*Date*

A copy of this consent will be left with you, and a copy will be taken by the researcher.

Appendix B – Table of Subject Physical Characteristics

Subject	Age (yrs)	Weight (kg)	10RM (kg)	5RM (kg)
1	26	78.3	12.50	20.50
2	27	96.0	18.25	22.75
3	27	97.5	16.00	21.50
4	26	82.9	12.50	19.50
5	25	83.0	16.00	22.75
6	21	85.0	12.50	19.25
7	22	96.0	16.00	20.50
8	23	84.0	11.50	20.00
9	28	114.2	11.50	16.00
10	28	86.0	13.75	18.25
11	21	90.0	14.75	21.50
12	23	76.0	12.50	16.00
13	24	88.0	19.25	25.00
14	19	85.0	14.75	19.50
15	22	72.5	11.25	14.75
16	21	87.0	13.75	17.00
17	25	106.0	14.75	18.25
18	35	70.0	13.75	18.25
19	26	87.0	17.00	21.00
20	24	73.0	12.50	17.00
21	25	72.0	14.75	19.50
Mean	**24.67**	**86.40**	**14.32**	**19.46**
SD	**3.45**	**11.01**	**2.19**	**2.53**

Appendix C – Table of Effect Sizes for Dependant Variables

Protocol	Effect Size
5RM DTP Peak Twitch	0.949
5RM DTP Time To Peak Twitch	-1.329
5RM DTP Half Relaxation Time	-1.402
5RM DTP Tension	-0.834
5RM DTP Activation	-1.589
5RM ATP Peak Twitch	0.155
5RM ATP Time To Peak Twitch	-1.219
5RM ATP Half Relaxation Time	-1.622
5RM ATP Tension	-0.932
5RM ATP Activation	-1.760
10RM DTP Peak Twitch	-0.402
10RM DTP Time To Peak Twitch	-1.454
10RM DTP Half Relaxation Time	-1.506
10RM DTP Tension	-1.019
10RM DTP Activation	-1.065
10RM ATP Peak Twitch	-0.978
10RM ATP Time To Peak Twitch	-1.764
10RM ATP Half Relaxation Time	-1.521
10RM ATP Tension	-1.151
10RM ATP Activation	-3.136