Is the Acute Neuromuscular Fatigue Produced During Resistance Training Associated with Chronic Increases in Muscle Strength and Muscle Fiber Area?

By

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ABSTRACT

The primary objective of the present study was to examine the effects of three resistance training programs that varied in either inter-set rest interval length or volume of training on the development of strength and muscle fiber size. Male subjects with a minimum of 1-year of regular resistance training experience were randomly assigned to one of three, 8-week training groups. The first set of all three programs was similar in that 10 repetitions to failure were performed. In program A (n=5) the load (78% 1-RM) remained constant for all subsequent sets. Program B (n=7) also used a constant load (80% 1-RM), however the rest interval was reduced from 3 minutes (as in Program A) to 1 minute. Subjects in this group performed additional sets to equate training volume with Program A. The training load for Program C (n=7) was progressively reduced (80% to 70% 1-RM) before each subsequent set to ensure the completion of 10 repetitions. Therefore, the volume performed was greater than that of Programs A and B. Single arm elbow flexion 1-RM increased by 12.3 +/- 3.5% in Program A, 16.5 +/-3.5% in Program B, and 14.1 +/- 4.7% in Program C. Gains in 10-RM equaled 16.3 +/-4.1%, 18.0 +/- 5.0% and 13.9 +/- 3.1% for Programs A, B and C, respectively. Although these increases in strength were significant ($p<.05$), there were no differences in the magnitude of change between the three groups. Increases in the cross-sectional area of type I and type II muscle fibers were similar after all three training programs. The second objective of this investigation was to measure the acute neuromuscular fatigue produced during a single session of each of the training protocols incorporated in the longitudinal part of this study. Force and IEMG during maximal isometric voluntary contractions (MVIC) along with blood lactate were assessed

prior to and upon the completion of each protocol. Subjects performed 3 sets of single-arm elbow flexion to failure using a training load of approximately 77.3% 1-RM in Protocol A. During Protocol B, subjects utilized the same constant resistance but the rest-intervals between each set were 1 minute. Protocol C was designed to maintain the repetitions completed per set at 10 while utilizing 3-minute rest interval. During Protocol C, the load used during the first set was equal to that used during Protocol A and was then reduced by about approximately 5% for each of the two subsequent sets. Protocol A and Protocol B resulted in similar reductions in MVIC, whereas Protocol C (24.8 +/- 7.2%) resulted in a significantly ($p<.05$) greater reduction in MVIC than Protocol A (20.2 +/- 7.7%). Protocols A and B elicited similar reductions in the force-time curve of the MVIC. A significantly greater reduction in the final 300ms of the force-time curve was observed following Protocol C (in comparison to Protocol A) ($p<.05$). There were no significant changes in IEMG after subjects performed protocols A and B. A significant time effect (with no interaction effect) in IEMG was observed following the comparison of Protocol A with Protocol C. Blood lactate increased significantly in response to all three protocols with no differences between the protocols. The third objective of this study was to compare the magnitude of resistance training-induced acute fatigue before and after the completion of 8 weeks of resistance training specific to the fatigue protocols used. The magnitude of resistance training-induced acute neuromuscular fatigue remained unchanged following the resistance training programs. The results appear to indicate that acute neuromuscular fatigue produced during resistance training may not be associated with the chronic increases in muscle strength and size.

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Dedication

To my parents, for always believing that I can.

To my grandparent, for opportunity and leading the way.

Although the prolonged performance of resistance training provides many neuromuscular benefits, resistance training is customarily performed to enhance muscular strength and increase muscle size. Programs used to produce these long-term neuromuscular adaptations are designed through the incorporation of a number of training variables. Specifically, resistance training variables include a) magnitude of training load, b) training volume, c) training to failure, d) rest intervals between sets, and e) speed of contraction (Pearson, Faigenbaum, Conley, & Kraemer, 2000; Tan, 1999). Although the inclusion of many of these variables in training programs is common, there is a paucity of longitudinal research identifying the relative importance of each of these training variables to the long-term development of strength and/or muscle hypertrophy. Further, research studies that have been performed are conflicting in their results.

MacDougall (1992) has suggested that in order to promote neuromuscular adaptations the magnitude of the resistance training load must exceed a minimal threshold of approximately 60-70% of maximal strength abilities. Additionally, the use of near maximal and above maximal training loads is implemented by weight lifters to promote gains in maximal strength (Garhammer & Takano, 1992). Dudley, Tesch, Miller and Buchanan (1991), in a 19-week resistance training study, provided additional evidence acknowledging the importance of the magnitude of the training load in developing muscle strength. During the duration of this training study it was observed that increments in leg press strength (as measured by a 3-RM) were related to the rate of increase in the magnitude of the training load. However, increases in 3-RM leg press strength over the 19 weeks were also associated with the rate of increase in the total

amount of resistance lifted per training session. As the total amount of resistance lifted in a single training session is a measure of work (Stone, Potteiger, Pierce, Proulx, O'Bryant, Johnson, & Stone, 2000), these results suggest that the training volume of a resistance training program may also influence increases in strength. In a comparison of three 12-week periodized strength training programs, Baker, Wilson, and Carlyon (1994) demonstrated that the total training volume completed during a training program was the most important factor in the long-term improvement of strength. As a result of inconsistent research observations in regard to the relative importance of the different training variables on strength development, the most effective method(s) for increasing muscle strength and size appear to be questionable. Additionally, training studies investigating the relative contribution of various training variables on the development of strength have failed to control for more than one variable and as a result it is difficult to determine the relative importance of each training variable.

The uncertainty in regard to the most effective means to develop strength and increase muscle size may be related to the lack of understanding of the physiological mechanisms or stimuli responsible for long-term neuromuscular adaptation. Komi (1986) has suggested that, although training load is critical to increases in strength and hypertrophy, the acute changes in neural, metabolic, and endocrine functioning in response to the training load, when performed systematically over time, may contribute to the development of strength. Similarly, it has been observed that the development of acute neuromuscular fatigue during each resistance training session may provide the stimulus for the development of strength and muscle cross-sectional area (Rooney, Herbert, & Balnave, 1994; Schott, McCully, & Rutherford, 1995).

Rooney et al. (1994) compared the effectiveness of two strength training programs that elicited different levels of acute muscle fatigue (as measured by a reduction in the force generating capacity of the trained muscle) on the development of long-term maximal strength. Significantly greater increases in strength were demonstrated in response to the program that produced larger decrements in maximal voluntary force. Thus, it was concluded that acute fatigue experienced during resistance training contributed to the long-term development of strength.

Immediately following a resistance training session acute neuromuscular fatigue is manifested in a temporary reduction in the maximal force-generating capacity of muscle (Behm, Reardon, Fitzgerald, & Drinkwater, in press; Kauranen, Siira, Vanharanta, 1999; Linnamo, Hakkinen & Komi, 1998). The acute decrease in the maximal force-generating capacity of muscle is the result of neuromuscular fatigue (Kent-Braun, 1999). Although neuromuscular fatigue manifests itself through a reduction in force-generating abilities, the physiological mechanisms responsible may be central (neural) and/or peripheral (muscular) (Kent-Braun, 1999; McLester Jr., 1997). Consequently, an acute training-induced decrement in muscle strength as a result of a training stimulus can occur through a combination of central and peripheral mechanisms.

Central mechanisms contributing to acute reductions in muscle performance, also referred to as central or neural fatigue, are the processes proximal to the neuromuscular junction (NMJ) that decrease neural drive (Kawakami, Amemiya, Kanehisa, Ikegawa, & Fukunaga, 2000). Specifically, these mechanisms include reduced descending drive, impaired motor neuron excitability, and increased antagonist activity (Kent-Braun, 1999).

Peripheral factors associated with muscle fatigue include those factors that impair the excitation-contraction process, consequently reducing the generation of force (McLester Jr., 1997; Tesch, Colliander & Kaiser, 1986). These processes include failure of the neuromuscular junction to transmit the electrical impulse to the muscle fiber (Green, 1990), Na^+ and K^+ imbalances reducing muscle fiber excitability, impaired Ca^{2+} release from the sarcoplasmic reticulum (Fitts & Balog, 1997), reductions in phosphocreatine (PCr) (MacDougall et al., 1999), and the accumulation of metabolites such as H^+ and P_i (McLester Jr., 1997).

The ability to perform repeated, heavy muscle contractions, as is practiced during a bout of resistance exercise, is dependent on high production rates of ATP through both PCr hydrolysis and anaerobic glycogenolysis (MacDougall et al., 1999). The performance of a bout of heavy-resistance training results in transient reductions in intramuscular PCr and glycogen (MacDougall et al., 1999; Tesch et al., 1986). MacDougall et al. (1999) observed 50% and 24% depletions in muscle PCr and muscle glycogen, respectively, following 3 sets of elbow flexion at 80% 1-RM. Additionally, a high volume training protocol elicited a similar reduction in PCr but a greater reduction in muscle glycogen in the vastus lateralis (Tesch et al., 1986). Coupled to the intramuscular consumption of PCr and glycogen are increases in metabolic by-products such as creatine (Cr), lactate, and hydrogen ions. It has been postulated that the acute changes in metabolite levels may act as a stimulus for resistance training-induced chronic adaptations, particularly muscle hypertrophy (Tesch et al., 1986).

Metabolic accumulation during resistance training has been proposed to be an important contributor to the stimulus for the development of muscle strength and

hypertrophy (Schott et al., 1995). Schott et al. observed significantly greater increases in muscle strength and hypertrophy following an isometric strength training program which produced greater acute metabolic changes than a program which elicited smaller acute metabolic changes. The acute increases in metabolite levels that contribute to fatigue were suggested to be a component of the stimulus for increasing strength and muscle size. Despite this evidence, the physiological mechanism(s) associating fatigue with neuromuscular adaptations remain(s) unclear.

Muscle hypertrophy is an increase in fiber cross-sectional area due to the addition of myofibrillar proteins (Goldspink, 1992). The accumulation of muscle proteins following extended resistance training is the result of a net increase in myofibrillar protein synthesis (Goldspink, 1992). In vitro analysis of differentiating skeletal muscle has demonstrated that myosin and actin protein synthesis are stimulated by elevated intramuscular concentrations of creatine (Ingwall, 1976). Creatine may act as a transcriptional or translational factor for protein synthesis (Ingwall, 1976), or may increase the uptake of amino acids by the contractile proteins themselves (Bessman & Savabi, 1990). Another possible mechanism contributing to muscle hypertrophy is exercise-induced satellite cell activation and proliferation (MacDougall, 1992). Satellite cells, by donating their nuclei, provide the growing muscle fibers with a greater source of DNA content (Dangott, Schultz, & Mozdiak, 2000). In the presence of creatine, satellite cell activity was significantly increased in functionally overloaded rats. Consequently, the alteration of satellite cell activity may be another possible mechanism by which high levels of intramuscular creatine promote muscle hypertrophy. If these proposed mechanisms of creatine action are applicable to the resistance-training model, the degree

of muscle hypertrophy may be related to the extent of phosphocreatine hydrolysis during a resistance training session.

The accumulation of intramuscular creatine is dependent upon the amount of PCr hydrolyzed during muscle activity as well as the re-synthesis of PCr during subsequent rest intervals (Tesch et al., 1986). Complete PCr recovery after depletion following a set of heavy resistance exercise has been suggested to take approximately 3-5 minutes (MacDougall et al., 1999). Programs that reduce the inter-set rest intervals to less than 3 minutes may limit the amount of PCr re-synthesized, thus maintaining greater levels of intramuscular creatine. Tesch et al. (1986) monitored acute changes in PCr levels following a protocol incorporating 60 second rest intervals between sets and results indicated that PCr was almost completely depleted following the training session.

With reduced levels of PCr available to re-synthesize ATP, glycolysis would become a major contributor to the production of ATP for a muscle performing resistance training. During glycolysis metabolic by-products such as lactate and hydrogen ions are also produced. It has been proposed that the lactate produced during a bout of resistance training may play a role in the release of human growth hormone (Craig & Kang, 1994; Hakkinen & Pakarinen, 1993). Hakkinen and Pakarinen (1993) observed a significant relationship between acute increases in lactate and serum growth hormone during bouts of intensive resistance training (Hakkinen & Pakarinen, 1993). Growth hormone is thought to have both a direct and indirect affect on protein synthesis (Kraemer, Marchitelli, Gordon et al., 1990). Theoretically, resistance training protocols that elicit acute increases in lactate may stimulate gains in muscle size from the release of human growth hormone.

Alternatively, it has been demonstrated that the amount and rate of glycogen utilization during a resistance training session appear to be influenced by the volume and intensity of training (Robergs et al., 1991). It has also been observed that the rate of glycolysis as well as the number of repetitions performed (volume) in successive sets is reduced when the training load is maintained throughout all sets performed (MacDougall et al., 1999; Hannie, Hunter, Kekes-Szabo, Nicholson, & Harrison, 1995; Robergs et al., 1991). As fatigue accumulates during successive sets of resistance training, there is a progressive reduction in the force generating capacity of muscle. Therefore, although a constant training load maintains the same absolute intensity it becomes relatively more intense as a result of the reduced force capacity of the fatigued muscle. Consequently, fewer repetitions are performed in successive sets which compromises the total training volume performed and possibly limits glycolytic activity. Perhaps, the amount and rate of glycogen utilization (and thus lactate produced) can be optimized during a training program in which the training load is progressively reduced over consecutive sets in order to maintain the same relative intensity and sustain training volume.

Statements of the Problem and Purpose

Currently, resistance training programs are prescribed on the basis that high volume, moderate intensity training will elicit muscle hypertrophy and high intensity, lower volume training will increase maximal strength through neural adaptations. This occurs despite the lack of evidence suggesting that neural and muscular adaptations are specific to moderate and high intensity training, respectively. Further, there is little understanding of the physiological mechanisms underlying long-term muscular and neural adaptations. Without a clear understanding of how neural and muscular

adaptations occur, it seems inappropriate to prescribe specific training programs to elicit specific adaptations.

If resistance training-induced fatigue is associated with long-term neuromuscular adaptations, resistance training programs should be designed to optimize the fatigue response. However, the specific mechanisms (central and/or peripheral) responsible for the temporary reduction in maximal force capabilities following a bout of resistance training are inadequately understood. Although the magnitude and site of acute neuromuscular fatigue following a bout of resistance training has been suggested to be influenced by a) rest intervals (Kraemer & Culver, 1987), b) training volume (Hakkinen & Pakarinen, 1993), c) magnitude of the training load (Linnamo et al., 1998), d) contraction duration (Kraemer, Marchitelli, & Gordon, 1990), and e) training to failure (Tesch, 1992), it is difficult to ascertain which factors are of primary importance as there is an absence of comparative research.

Therefore, the objectives of this investigation were to a) measure, compare and define the acute neuromuscular response following protocols designed to produce different levels of muscle (metabolic) fatigue, b) measure and compare the effectiveness of these programs in producing long-term gains in muscle strength and muscle size, and c) to determine if the acute neuromuscular response changes with training. In order to accurately describe the acute neuromuscular fatigue following a resistance training protocol, neural as well as metabolic measures of fatigue should be included. Although acute neural fatigue may not be associated with chronic increases in strength, monitoring changes in acute neural fatigue may provide a better understanding of the mechanisms underlying increases in strength. This study intended to independently compare a) the

role of different rest intervals, while controlling for relative training intensity and volume and b) the role of training volume, while controlling for rest interval length on the augmentation of strength and muscle size as well as the development of acute neuromuscular fatigue.

Research Questions

1) Are there differences in the chronic increases in single-arm forearm flexor 1-RM following resistance training programs having different inter-set rest intervals?
2) Are there differences in the chronic increases in single-arm forearm flexor 1-RM following resistance training programs differing in training volume?
3) Are there differences in the chronic increases in biceps brachii muscle fiber area following resistance training programs having different inter-set rest intervals?
4) Are there differences in the chronic increases in biceps brachii muscle fiber area following resistance training programs differing in training volume?
5) Is the magnitude of acute neuromuscular fatigue different in response to resistance training protocols with different inter-set rest intervals but equated for training volume?
6) Is the magnitude of acute neuromuscular fatigue different in response to resistance training protocols different in training volume but equal in inter-set rest-interval length?
7) Does the magnitude of resistance training-induced acute neuromuscular fatigue change after eight weeks of resistance training, in experienced resistance trained subjects?

Deflnitions

Concentric 1-RM: The maximum amount of weight that was lifted during the concentric phase of the single-arm flexion exercise.

10-RM: The maximum amount of weight that was used to perform 10 concentric coupled to eccentric repetitions of single-arm forearm flexion.

Muscle Failure: The inability to complete or properly perform another repetition of single-arm elbow flexion.

Muscle Fiber Area: The cross-sectional area of a muscle fiber.

Limitations

1. The magnitude of resistance training-induced neuromuscular fatigue was assessed by using pre- and post-protocol maximal voluntary isometric contractions.

Delimitations

1. Participants of the present study were male, aged 20-33 and had at least one year of previous resistance training experience. This may have influenced the type and magnitude of the chronic response to resistance training.
2. The duration of the resistance training component of this study was eight weeks.

Assumptions

1. Eight weeks was of sufficient duration for differences between the training programs to become evident.
2. It was assumed that any change in MVIC (and the other acute fatigue related dependant variables) was the result of the fatiguing protocol and not the result of measurement error or the amount of effort exerted by the subject.

Methodology

Subjects

Twenty-one university-aged males volunteered to participate in the study. At the onset of the study, all subjects had been participating in a regular weight training program (minimum 3 times per week) for at least one year. Prior to participating in the study, each subject provided written consent after being informed of the specific protocols being used in the investigation. The study was approved by the University of Victoria Human Ethics Committee. Two subjects failed to complete the training portion of this investigation due to causes unrelated to the study, therefore 19 subjects completed the resistance training component of this study.

Experimental Design

This investigation consisted of three related and successive components. The first component included initial strength (concentric 1-RM) testing as well as measuring the acute neuromuscular fatigue produced in response to a single bout of each of the three loading (fatiguing) protocols that were performed during the 8-week resistance training component of this study (Figure 1). Following initial strength testing, subjects were randomly assigned into one of three 8-week resistance training programs (Figure 1). The third component, which occurred upon completion of the 8-weeks of training, measured the chronic strength changes elicited by the resistance training programs and reassessed the acute neuromuscular response to the three loading protocols (Figure 1).

Strength Testing

The concentric 1-RM of the right elbow flexors was measured using a padded arm-curl bench in which the upper arms were braced on an inclined padded arm support.

Subjects were seated with their torso upright and both feet in contact with either the floor or the base of the bench. Seat height was adjusted so the subjects' axillae were at a similar height as the superior aspect of the padded arm support when their backs were straight. With the seat at the appropriate height, subjects placed both arms on the anterior surface of the inclined padded arm support. Seat height was recorded to ensure it remained constant between all testing and training sessions. Prior to testing, subjects performed a warm-up comprised of 1 set of 10 repetitions at approximately 50% 1-RM and 1 set of 3-5 repetitions at approximately 75-80% 1-RM. A 4-minute rest period separated the warm-up and first testing set (Chestnut & Docherty, 1999)

The concentric 1-RM test of the right elbow flexors began with the elbow extended and the right hand supinated. In this position, a load (dumbbell) was placed in the hand and subjects attempted to lift the load. A successful repetition was defined as one in which full elbow flexion was achieved. Throughout the entire range of motion of each attempt, subjects were instructed to keep both arms and axillae in contact with the padded arm support. Subjects were verbally encouraged to perform more than one repetition with each testing load. If more than one repetition was performed, the load was increased and another attempt at establishing the 1-RM was made. Free weights were used and the 1-RM loads were recorded to the nearest 0.5 kg. Subjects were provided with 4 minutes of rest between successive attempts. Assessment of the 1-RM of the right elbow flexors occurred prior to training and approximately 72 hours after the final training session of the 8-week resistance training program.

During the initial strength testing the maximum number of repetitions with a training load of approximately 75% 1-RM was also determined for all subjects. This was

performed to determine the 10-RM load to be utilized during the three fatiguing protocols as well as for the beginning of the 8-week training program. If the number of repetitions performed at 75% 1-RM was not within 1 repetition of 10 repetitions, the load was adjusted and another attempt was made. Rest intervals of 5 minutes were provided between trials. No more than 2 attempts were necessary to establish the 10-RM.

Fatiguing protocols

Prior to beginning the training program, subjects performed a single bout of all three of the different training protocols under investigation to assess acute neuromuscular fatigue. Fatigue protocol A consisted of three sets of repetitions to failure using a constant load of approximately 75% 1-RM with 3 minutes rest between sets. During this protocol, the number of repetitions performed per set progressively decreased in successive sets (MacDougall et al., 1999). Protocol B (using the same load that was used in protocol A) incorporated 3-5 sets of repetitions to failure, however the inter-set rest interval was reduced to 1 minute. Because of the reduced rest interval the number of repetitions performed per set decreased at a greater rate in protocol B than in protocol A (Abdessemed, Duche, Hautier, et al., 1999). To equate training volume between protocol A and B, subjects were required to perform as many sets to failure as necessary until the total number of repetitions performed approximated the number performed during protocol A. Training volume was defined as number of sets x number of reps x training load (% 1-RM). During protocol C subjects performed 3 sets to failure in which the load in the initial set was identical to that used in the first set of protocol A. However, the training load was reduced for the second and third sets to ensure that 10 repetitions to

failure were performed in these sets. A 3-minute rest interval separated each set in protocol C.

During all three protocols, subjects performed the same single-arm elbow flexion exercise that was used during strength testing. In all of the fatiguing protocols, each set was performed using continuous concentric coupled eccentric repetitions until volitional muscle failure was reached. For the objectives of this study, failure was defined as the inability to complete the next repetition or a deviation from the described exercise technique (Hakkinen, Kauhanen, & Komi, 1988). A metronome was set to assist the subjects in controlling the tempo of the concentric and eccentric phase of each repetition (1.5 s :1.5 s/ concentric: eccentric). The same investigator supervised each protocol to ensure that the exercise range of motion was consistent between the different protocols and subjects. Measures of acute neuromuscular fatigue were assessed pre- and post-protocol (Figure 2).

Measures of Acute Neuromuscular Fatigue

Maximal Voluntary Isometric Contraction (MVIC)

MVIC of the right elbow flexors was measured utilizing a Cybex II (Lumex Inc) isokinetic dynamometer. Subjects were seated in the same padded arm curl bench as used during strength testing with their left forearm flexed to 90 degrees at the elbow. The axis of rotation of the mechanical lever was aligned with the axis of rotation of the right elbow joint. Lever length was adjusted so the handle fit into the palm of each subject's right hand. This length was recorded to maintain consistency between pre- and post-measurements as well as between the three fatiguing protocols.

Measurement of MVIC occurred before and immediately after the final repetition in each of the three fatiguing protocols (Figure 2). Subjects were instructed to exert maximal force as hard and as fast as possible as well as maintain the contraction for approximately 2-3 seconds (Linnamo, Hakkinen, & Komi, 1998). During the MVIC, subjects were instructed to keep both axillae and both arms in contact with the padded arm support. Further, the left hand of the subjects was maintained in a supinated position and hung freely over the lower edge of the padded arm support in order to avoid any contribution in force development. The pre-protocol MVIC was performed 2 minutes after a warm-up set consisting of 10-12 repetitions at approximately 50% 1-RM (Figure 2). A 2-minute rest interval also separated the pre-protocol MVIC and the onset of the first set of the fatigue protocol.

Force-Time Curves (Rate of Force Development)

During each MVIC, a force-time curve was analyzed as the peak force developed in succeeding 100ms intervals from the onset of the contraction up to 500ms (Hakkinen, 1994; Hakkinen et al., 1988).

Electromyography (EMG)

Two surface electrodes (silver-silver chloride) were placed approximately 2 cm apart over the muscle belly of the right biceps brachii. A permanent marker was used to outline the electrodes to ensure that electrode position remained constant between the three fatiguing protocols. A ground electrode was placed over the styloid process of the left forearm. Skin preparation for all electrodes included removal of oils and dead skin cells by lightly abrading and cleansing the placement sites with sandpaper and prepared alcohol swabs.

EMG activity, recorded during the maximal voluntary isometric contractions, was amplified (1000X), filtered and stored on the computer (Biopac Systems Inc., MP 100). Sampling rate was set at 250 Hz. The EMG signal of the biceps brachii was rectified and integrated (iEMG) for data analysis (AcqKnowledge, 3.0). Data analysis consisted of measuring the mean amplitude of the iEMG (Linammo et al., 2000) over a 1000 ms period of the MVIC after the initial 250 ms had elapsed. Additionally, a Fourier transformation was performed over the same 1000 ms window in order to calculate mean power frequency (MPF) of the iEMG power spectrum (Linnamo, Newton, Hakkinen, Komi, Davie, et al., 2000).

Muscle Biopsy

The muscle biopsies from the biceps brachii were performed before the beginning of the 8-week resistance training program as well as 48 hours after testing the post-training 1-RM (5 days after the last day of training). Muscle samples were taken by an experienced physician using a percutaneous needle-biopsy technique with local anesthesia and manual suction (Sleivert, 1994).

Tissue samples were mounted on cork with an embedding medium (tragacanth gum) and frozen in liquid nitrogen-cooled isopentane and stored at -80 degrees Celsius (MacDougall, et al., 1999; Essen-Gustavsson & Tesch, 1989). Analysis of all samples occurred following the completiion of the 8-week resistance training study. Prior to analysis, the muscle samples were sectioned (10 - 14 um) in the transverse plane using a cryostat (HM 500 OM, Microm) set at approximately -20 degrees Celsius. Histochemical analysis was performed on all biopsy samples to determine muscle fiber type composition and fiber area. All sections were stained for myofibrillar adenosine triphosphatase

following alkaline (pH = 10.4) preincubation (Suter, Herzog, Sokolosky, Wiley, & MacIntosh, 1993). Consequently, muscle fibers could be visually differentiated as type I (light) or type II (dark) (Figure 3). The percentage of each fiber type was calculated from sections containing a minimum of 80 fibers (range = 84 - 349 fibers). Muscle fiber area of types I and type II fibers was determined by tracing individual fibers in a digital imaging program (Optimas 3). All fibers were traced by the same investigator and during this process the investigator was unaware from which subject and from which time (pre- or post-training) the section originated. A minimum of 8 (range 8 - 25) fibers for each fiber type, representative of the entire transverse section, were selected for analysis (Tesch, Thorsson, & Kaiser, 1984).

Blood Lactate

Capillary blood samples were drawn from a fingertip of the non-exercising (left) arm at rest and post-exercise. Resting samples were obtained from each subject following 10 minutes in a seated and relaxed position (Figure 2). Post-exercise blood samples were drawn 5 minutes following the final repetition of the final set in all protocols (MacDougall et al., 1999). Samples were immediately analyzed using a Lactate Pro blood lactate analyzer (KDK).

Resistance Training

Following the initial testing sessions, subjects performed an 8-week resistance training program specific to their assigned group. Each of the three training programs corresponded to one of the fatiguing protocols. Generally, each program was designed to be equal in the repetitions performed (10) and relative training load utilized in the first set with inter-program differences occurring following completion of the first set. Subjects

assigned to training program A performed 3-4 sets of repetitions to failure using the same load for all sets during a training session. This load was equal to the load in which 10 repetitions could be performed in the first set. A 3-minute rest interval separated all training sets. Subjects belonging to training program B also trained with a constant load, however the inter-set rest interval was reduced to one minute. In order to equate volume between programs B and A, subjects of program B performed more than 3-4 sets. In training program C, the training load used by the subjects was reduced prior to the performance of each successive set ensuring that the number of repetitions performed per set (10) remained constant. Three minutes of rest separated all sets in this protocol. For all programs, repetitions were performed to failure during each set and the training load was increased or decreased if the subject performed more than 12 or fewer than 8 repetitions in the first set (Sanborn, Boros, Hruby, Schilling, O'Bryant et al., 2000).

Subjects adhered to these parameters while performing the 2 primary training exercises involving the forearm flexors of both the left and right arms: 1) single-arm elbow flexion (SAEF)(as performed in the fatiguing protocols and 1-RM testing) and 2) standing barbell biceps curls (SBBC). Subjects trained the forearm flexors twice per week and the same investigator(s) supervised each of these training sessions. The single-arm elbow flexion exercise was always performed first, however the order in which the left and right arm performed this exercise was alternated from session to session. After 3 weeks of training the number of sets performed by subjects in group A and C increased from 3 to 4, where as subjects in group B increased the number of sets performed until the volume was similar to that performed by subjects in group A. After 5 weeks of training, a similar increase in the number of sets performed during the standing barbell

biceps curls occurred. During the final week of training the number of sets performed of both exercises was decreased to 3 in order to prepare the subjects for the post-training strength test. A daily log recording training load used for every set, number of sets performed, and repetitions per set completed was kept for each subject. Comparisons of training variables between the training programs included total training volume, total number of repetitions performed, mean training load used in the first set, mean training load used in all sets, and repetitions per set were made. For these calculations the number of repetitions performed by the left and right elbow flexors during the SAEF were averaged. Furthermore, changes over the 8 weeks to the training load that was used during the first set (10-RM) of each protocol were used to measure and compare the effectiveness of each training program.

Additionally, subjects in the three training groups were provided with supplementary exercises to train the other muscles of the upper as well as lower body. All subjects followed a standard set of protocols while performing the supplementary exercises. Supplementary training was performed twice per week. Exercises for the upper body included bench press, pec dec, lat pull down, seated row, lateral raises, and standing triceps press down. Lower body training exercises included leg press, lunges, leg extensions, hamstring curls, straight-leg deadlift and standing calf raises. All subjects performed 3-4 sets of 10-12 repetitions of each supplementary exercise with a 3-minute inter-set rest interval.

Data Treatment

In examining the effectiveness of the three training programs used in the present study, between-subject comparisons were made between Program A, Program B, and

Program C (see results and discussion section A). However, the discussion will focus on independent comparisons between Programs A and B as well as Programs A and C. Program A and Program B were equated for all training variables except for inter-set rest interval length, and the only difference between programs A and C was the way in which the training load was applied.

Within-subject comparisons were used in assessing the acute neuromuscular fatigue produced in response to each program. However, because not all subjects were able to complete all three fatigue protocols (due to time constraints rather than causes related to the study), comparisons were limited to protocol A with protocol B (N = 12) (see results and discussion section B) as well as protocol A with protocol C (N = 14) (see results and discussion section C).

To determine if the acute neuromuscular response changed following eight weeks of training, within-subject comparisons were made between the acute fatigue measured pre- and post-training but only for the protocol that corresponded to the training program performed by each subject (see results and discussion section D).

Statistical Analysis

To determine the effectiveness of the three training programs on muscular strength and the muscle fiber characteristics, a repeated measures (pre- and post-training) ANOVA was performed on each dependent variable (1-RM, 10-RM, and muscle fiber size) with training group serving as the between subjects factor. To compare the training variables of the three 8-week training programs a one-way ANOVA was used. When significant differences were indicated between the groups, a post hoc Bonferroni test was performed to determine the differences between the mean values. Alternatively, a 2 x 2

[measurement time (pre- and post-protocol) x protocol] ANOVA with repeated measures on both factors (no between subject factor) was utilized for each measure of acute neuromuscular fatigue to determine if any differences existed between the fatiguing protocols. The present study focused on possible time x protocol interaction effects, indicating a difference in the magnitude of the acute neuromuscular fatigue produced by the fatigue protocols. Because not all subjects completed all three of the fatiguing protocols, independent comparisons were made between protocol A and B as well as protocol A and C.

R1: Random assignment of subjects into groups
R2: Random ordering of protocols B and C
F.P.A: Fatigue Protocol A
F.P.B: Fatigue Protocol B
F.P.C: Fatigue Protocol C
ST: Strength Testing (1-RM & 10-RM)
B:Muscle Biopsy
TRAIN: 8-week strength training program

Figure 1. Experimental Design: Time Line.

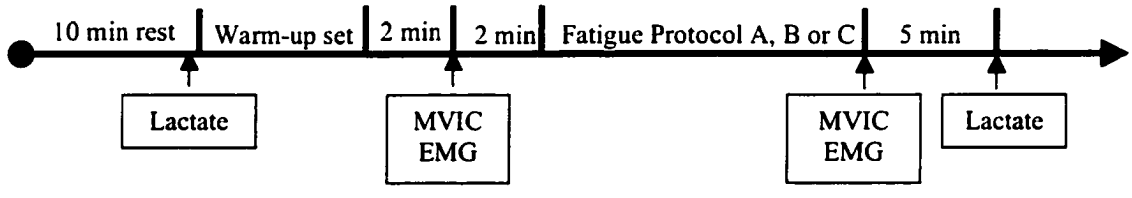


Figure 2. Time line of measures during the fatigue protocols.

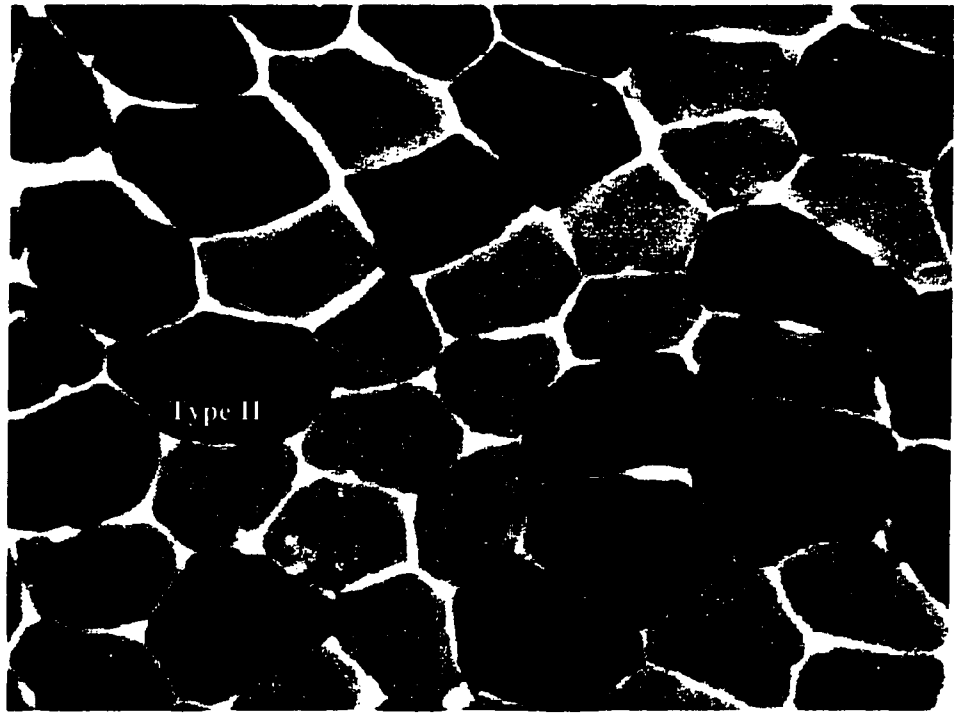


Figure 3. Myofibrillar ATPase stain of a muscle fiber sample after preincubation in a pH of 10.4. Light fibers are type I and dark fibers are type II.

Section A: Results and discussion for the effects of using different rest intervals or loading strategies on chronic changes in forearm flexor strength and muscle fiber size.

Results

The descriptive characteristic of the subjects of the different training programs are listed in Table A1.

Training Variables

The average relative training intensity (percent of initial 1-RM) used in the first set was 77.8 ±3.7%, 80.2 ±3.2%, and 81.1 ±3.9%, for programs A, B, and C, respectively (Table A2). There were no differences between the programs with regards to relative training intensity used in the first set. Since the training intensity remained constant for programs A and B, the relative mean training intensity used in all sets did not change from that used in the first set. However, because the load was reduced each set to maintain the number of repetitions, the mean relative mean training intensity used over all sets in program C equaled 74.2 ±3.7%. As a result there was a significany difference between the relative training intensities used in all sets between programs B and C ($p<.05$).

Total training volume (total number of repetitions x percent of initial 1-RM) for the single-arm elbow flexion equaled 582.6 ±38.3 units, 602.2 ±33.8 units, and 803.2 ±80.2 units for programs A, B, and C, respectively (Table A2). The volume accomplished in program C was significantly greater than that completed in program A and B ($p<.05$). Total training volume for the standing biceps curl could not be determined because initial 1-RM was not measured therefore the total number of repetitions performed was also calculated. Subjects in program A completed a mean of 374 repetitions of single-arm elbow flexion and 399.8 ±19.5 repetitions of the standing bilateral biceps curl. Subjects in program B performed 375 repetitions of single-arm

elbow flexion and 384.6 ±17.1 repetitions of standing bilateral biceps curl. There was no difference between these two programs in the total number of repetitions performed for either exercise. The number of repetitions performed by subjects in program C was significantly greater ($p<.05$) for both exercises (540 repetitions of single-arm elbow flexion and 485.6 ±50.3 repetitions of standing bilateral biceps curl).

The average number of repetitions completed in the first set of single-arm elbow flexion over the 8 weeks was 10.3 ±.8 for program A, 10.7 ±.7 for program B, and 10.3 ±.7 for program C. During the first set of the standing bilateral biceps curl, subjects in program A, B, and C performed a mean of 10.7 ±.4, 10.8 ±.8, and 10.2 ±.7 repetitions, respectively (Table A2). There was no difference between the three programs in the number of repetitions performed during the first set of either exercise. The average number of repetitions performed per set across all sets of single-arm elbow flexion and standing bilateral biceps curl was 7.5 ±.6 and 8.7 ±.7 in program A, 5.9 ±.8 and 6.8 ±.8 in program B, and 10.6 ±.7 and 10.5 ±.8 in program C, respectively. In both exercises the number of repetitions performed per set across all sets was significantly greater in program C in comparison to program A and B ($p<.05$), and in program A in comparison to program B ($p<.05$).

1-RM and 10-RM

There were no significant differences between the three groups at the onset of training. A significant main effect (training) ($p<.05$) was evident following the 8 weeks of resistance training for both 1-RM and 10-RM strength. Group A increased single-arm elbow flexion 1-RM from 20.7 ±2.7 kg to 23.3 ±3.5 kg (Figure A4). In response to programs B and C, 1-RM increased from 21.9 ±3.5 kg to 25.7 ±4.7kg and 22.4 ± 2.9kg to

25.5 ± 3.2kg, respectively (Figure A4). The corresponding relative increases in 1-RM included, 12.3 ±3.7%, 16.5 ±3.5%, and 14.0 ± 4.7% for group A, group B, and group C, respectively. No significant interaction effect was observed between the three groups.

A similar trend was observed for increases in the training 10-RM of the three groups. In group A, the 10-RM training load increased from 15.0 ±1.3kg to 17.5 ±1.7kg (16.3 ±4.1% improvement). The 10-RM load increased by 18.0 ± 5.0% (16.4 ± 2.5kg to 19.4 ±3.1kg) and 13.9 ±3.1% (17.1 ±2.1kg to 19.5 ±1.9kg) in groups B and C, respectively (Figure A5).

Muscle fiber area and fiber type

Muscle fiber area of the type I fibers increased from 66.7 ±7.4μm^2 to 71.6 ±9.9μm^2 following program A, from 62.0 ±10.7μm^2 to 69.8 ±12.3μm^2 following program B, and from 54.0 ±14.7μm^2 to 60.3 ±14μm^2 following program C (Table A3). The corresponding relative increases in type I fiber area are 6.9 ±3.4%, 12.6 ±5.1%, and 12.5 ±4.8%. All groups exhibited a significant increase in slow twitch area, with no significant differences occurring between the three groups. The type II fibers exhibited a similar pattern. Program A increased type II fiber area from 79.8 ±11.5μm^2 to 84.9 ±13.8μm^2 (6.2 ±2.1%), program B increased type II fiber area from 75.7 ±14.6μm^2 to 82.3 ±13.9μm^2 (9.3 ±10.6%), and program C increased type II fiber area from 72.6 ±15.1μm^2 to 83.2 ±19.4μm^2 (14.2 ±10.6%). All the increases in type I and type II fibers were significant ($p<.05$).

The percentage of type II fibers increased in response to programs A (from 54.5 ±7.4% to 59.3 ±2.8%) and C (from 56.7 ±9.2% to 58.7 ±3.7%. However, these increases

were not significant. The percentage of type II fibers decreased from 61.8 ±2.7% to 57.0 ±3.1% in response to program B. This reduction was not significant and there were no significant differences between the groups pre- to post-training.

Table A1. Mean (SD) height, body mass and age of the participants of the three training groups.

Training Group	Height (cm) $\bar{x}$ (SD)	Body Mass (kg) $\bar{x}$ (SD)	Age (years) $\bar{x}$ (SD)
A (n=5)	180.5 (5.6)	89.7 (18.7)	25.0 (4.9)
B (n=7)	180.1 (3.9)	80.5 (6.5)	23.7 (3.9)
C (n=7)	180.3 (4.3)	86.4 (11.2)	23.9 (3.2)

Table A2. Training variables of the three training programs (mean values over the 8 weeks).

	Program A $\bar{x}$ (SD)	Program B $\bar{x}$ (SD)	Program C $\bar{x}$ (SD)
Repetitions completed per set of SAEF in the first set	10.3 (.8)	10.7 (.7)	10.3 (.3)
Repetitions completed per set of SAEF across all sets	7.5 (.6)	5.9 (.8)*	10.6 (.7)**
Repetitions completed per set of SBBC in first set	10.7 (.4)	10.8 (8)	10.2 (.7)
Repetitions completed per set of SBBC across all sets	8.7 (.7)	6.8 (.8)*	10.5 (.8)**
RI of training load used in first set (% of initial 1-RM)	77.8 (3.7)	80.3 (3.2)	81.08 (3.9)
RI of training load used across all sets (% of initial 1-RM)	77.8 (3.7)	80.3 (3.2)	74.2 (3.7)#
Total volume performed of SAEF (repetitions x % of initial 1-RM)	582.6 (38.3)	602.2 (33.7)	803.2 (80.2)**
Total repetitions of SAEF completed	374.6(21.5)	375.4(20.7)	540.6(35.9)**
Total repetitions of SBBC completed	399.8 (19.5)	384.6 (17.1)	485.6 (50.3)**

RI=Relative Intensity (of pre-training 1-RM)
* Represents significant difference with Program A ($p<.05$)
** Represents significant difference with Program A and B ($p<.05$)
# Represents significant difference with Program B ($p<.05$)

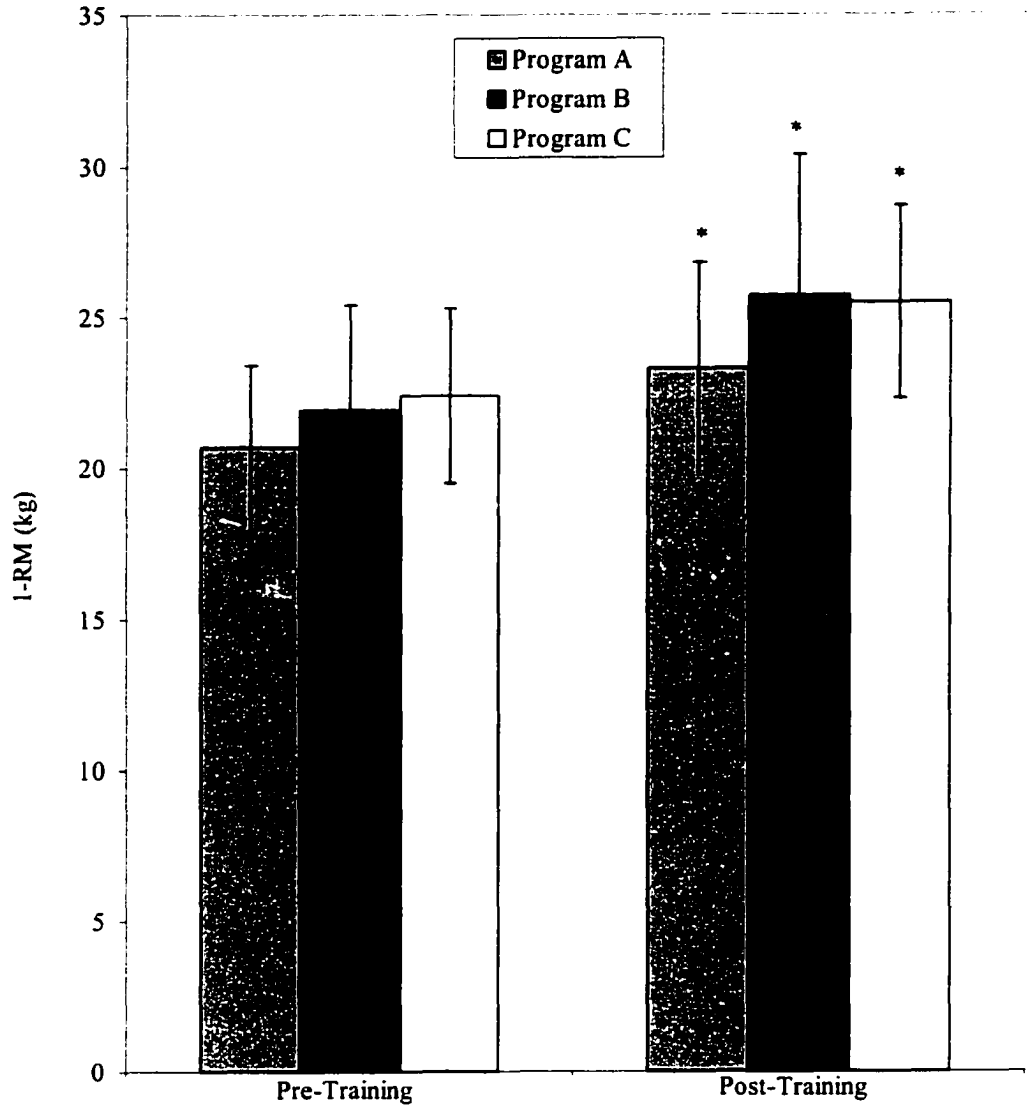


Figure A4. Mean (SD) single-arm forearm flexion 1-RM values pre- and post-training for all three training programs (* = significant difference from pre-training value within the respective training group).

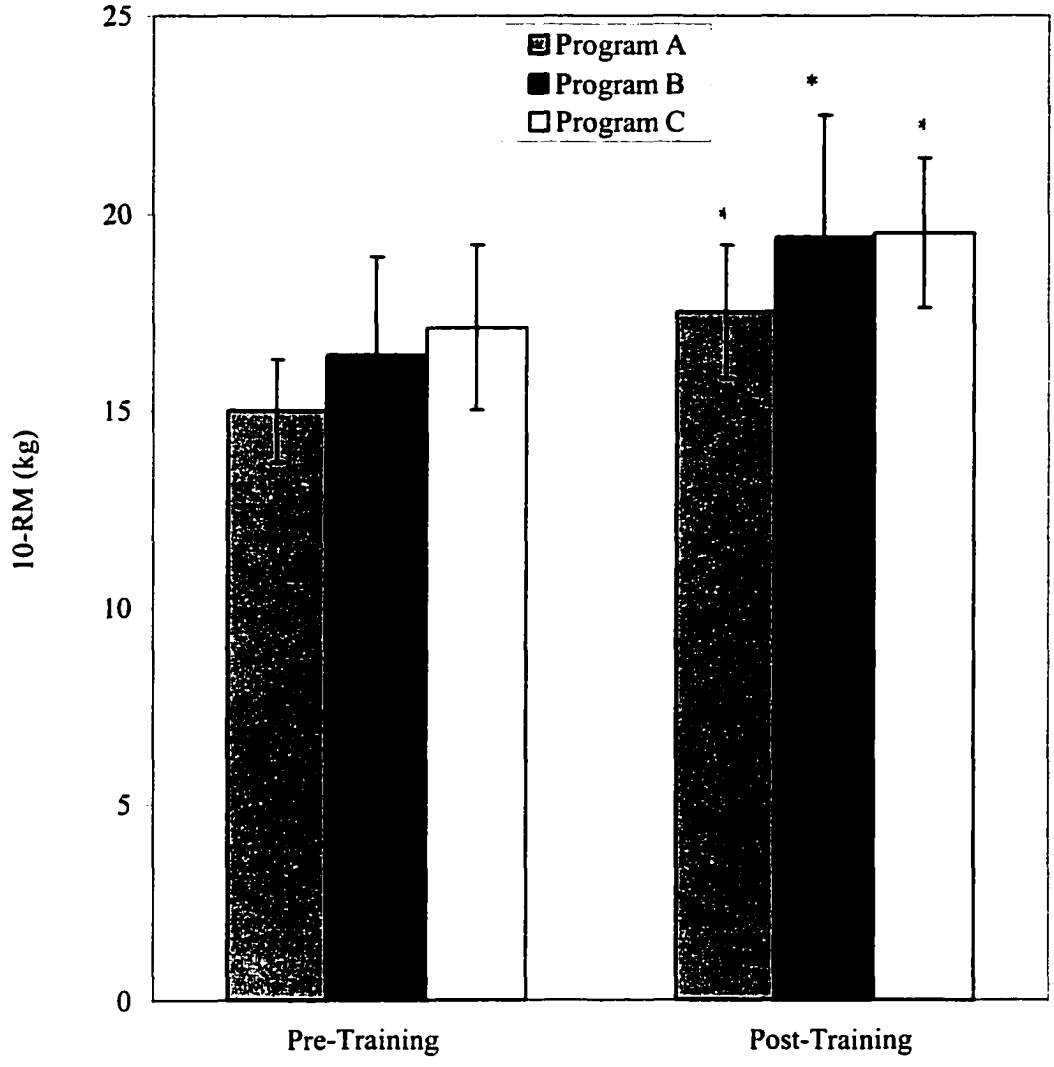


Figure A5. Mean (SD) single-arm forearm flexion 10-RM values pre- and post-training for all three training programs (* = significant difference from pre-training value within the respective training group).

TableA3. ST and FT muscle fiber characteristics (area and fiber type) before and after performance of the three, 8-week of resistance training programs.

Training Program	Type I fiber area(μm^2)		Type II fiber area(μm^2)		%Type II fibers	
	Pre-train	Post-train	Pre-train	Post-train	Pre-train	Post-train
	$\bar{x}$ (SD)	$\bar{x}$ (SD)	$\bar{x}$ (SD)	$\bar{x}$ (SD)	$\bar{x}$ (SD)	$\bar{x}$ (SD)
Program A (n=4)	66.75 (7.40)	71.59* (9.98)	79.80 (11.54)	84.93* (13.80)	54.5 (7.4)	59.3 (2.8)
Program B (n=3)	62.07 (11.06)	69.80* (12.30)	75.70 (14.01)	82.31* (13.90)	61.8 (2.6)	56.9 (3.1)
Program C (n=4)	54.00 (14.74)	60.30* (14.03)	72.61 (15.10)	83.20* (19.39)	56.7 (9.2)	58.7 (3.7)
Values collapsed from all 3 programs (n=11)	60.83 (11.69)	66.99* (12.16)	76.06 (12.66)	83.59* (14.48)	57.2 (7.4)	58.5 (3.1)

* Represents significant main effect (training) ($p<.05$).

Discussion

Strength

Major findings of this study were that single-arm elbow flexor 1-RM and 10-RM increased similarly in response to the three resistance training programs that differed in either A) rest interval length or B) training volume and intensity (Figure A4 and Figure A5). Training with a constant load (approximately 78% 1-RM) for all sets with a 3-minute rest interval improved 1-RM and 10-RM strength by 12.3% and 16.3%, respectively. Using the same loading pattern and intensity (approximately 80% of pre-training 1-RM), but with a 1-minute rest interval, program B produced 1-RM strength gains of 16.3% and 10-RM improvements of approximately 18%. A 14% increase in 1-RM and a 14% elevation in 10-RM were demonstrated by using a training program that progressively reduced the training load set while incorporating a 3-minute rest interval between sets (Program C). There were no significant differences between the three training groups with regards to the magnitude of any of the strength gains (Figure A4 and Figure A5).

The relative increases in forearm flexor 1-RM strength experienced by the training groups in the present study are similar to those observed in recreationally trained subjects following nine weeks of a 12-week resistance training study (McCall, Byrnes, Dickinson, Pattany & Fleck, 1996). Subjects in their study performed 3 sets of multiple exercises involving the forearm flexors using a 10-RM load and a 1-minute rest interval between successive sets. Additionally, 9 weeks of resistance training with loads of 90% 1-RM produced 15% improvements in forearm flexor 1-RM of well-trained male subjects (Moss, Refsnes, Abildgaard, Nicolaysen, & Jensen, 1997). Thus, it appears that the

increases in strength observed in the present study are in accordance with those reported in previous studies using subjects with a similar training background as well as the same muscle group.

Previous research comparing the effectiveness of strength training programs that have manipulated rest interval length on the development of strength are equivocal in their findings and difficult to interpret because other training variables were not controlled between the training programs. Schott, McCully, and Rutherford (1995) observed significantly greater improvements in maximal isometric strength of the quadriceps in a training group using a 30-second rest period than a group using 2 minutes of recovery between sets. The researchers suggested that the greater strength gains were a result of a greater accumulation of fatigue due to the shorter rest periods. However, in addition to the differences in rest interval length, the two groups were also different with respect to the type of contraction (intermittent versus continuous) used in training. Consequently, it was uncertain which factor (rest or contraction type) may have contributed more to the differences in strength development.

Training variables that appear to be important in the augmentation of muscular strength include the magnitude of the training load and training volume (Dudley et al., 1991; O'Hagan et al., 1995; Pincivero, Lephart & Karunakara, 1997; Wilson, 1995). It has been suggested that both of these training variables are compromised in training programs that incorporate short rest intervals, and as a result sub-optimal gains in strength are attained (Pincivero et al., 1997; Robinson et al., 1995; Wilson, 1995).

Pincivero et al. (1997) compared two isokinetic training programs using different rest periods on changes in isokinetic muscular performance following 4 weeks of

training. Subjects in both programs performed 4 sets of 10 maximal repetitions at 90 degrees per second with the only difference between the two training groups being the inter-set rest interval. One group used a 40-second rest period and the other training group used a 160-second rest interval. Significant interaction effects were observed in hamstring performance (total work and average power), with the training group comprised of longer rest intervals exhibiting greater gains than the 40-second rest interval group. No significant differences were observed between the two groups in quadriceps performance. The results of this study may have been confounded by the lack of specificity between the strength testing speeds (60 and 180°/s^{-1}) and the training speed (90°/s^{-1}). In this study, although all isokinetic repetitions during training were performed maximally, it is possible that the amount of work (volume) performed during training was different between the two training groups. Comparisons of the acute effects of these two programs revealed that peak torque and total work (over 4 sets during a single training session) of the hamstring and quadriceps muscle groups were significantly reduced with 40-second inter-set rest intervals but remained unaltered with rest periods of 160 seconds (Pincivero, Lephart, & Karunakara, 1998). Touey, Sforzo and McManis (1994) observed that both peak torque and total work of the quadriceps and hamstrings were significantly reduced to a larger extent following isokinetic (60 and 180°/s^{-1}) protocols incorporating 30 seconds of rest between sets in comparison to120-second inter-set rest intervals. Similarly, peak torque and total work of the quadriceps and hamstrings muscle groups were reduced more following a 1-minute rest interval in comparison to a 5-minute inter-set recovery period (Pincivero, McCann, & Mark, 2000). Therefore, the smaller performance gains of the 40-second rest interval training group compared to the 160-

second rest interval training group (Pincivero et al., 1997) could be the result of reduced absolute training intensity and training volume rather than less rest.

The ability of a muscle to contract repeatedly under high loads depends on the supply of ATP in the contracting muscle (MacDougall et al., 1999; Weiss, 1991; Wenger & Reed, 1976). During resistance training, ATP requirements are met through the anaerobic breakdown of PCr and glycogen (MacDougall et al., 1998, Robergs et al., 1991, Tesch et al, 1987). As soon as ATP consumption exceeds production, muscular force can no longer be maintained at the same intensity (Wenger & Reed, 1976). In resistance training, this is considered the point of muscle failure (Weiss, 1991), and is primarily dependent on the magnitude of the resistance used. For the muscle to restore the high-energy phosphagens, so it is capable of exerting force at the required level, rest must be provided (Weiss, 1991). MacDougall et al. (1999) suggested that ATP and PCr are completely replenished 3 minutes following a bout of heavy resistance training. Rest periods shorter than the time necessary to completely re-synthesize ATP and PCr (as well as remove metabolic by-products) are suggested to impair the ability of a muscle to develop force (Pincivero et al., 1997; Robinson et al., 1995; Weiss, 1991) thus, limiting the training load used and/or the training volume accomplished during training.

In the present study, the subjects of programs A and B performed dynamic constant external resistance (DCER) training (Weiss, Coney, & Clark, 1999). Specifically, the amount of force or tension a muscle generates is determined by the magnitude of the external resistance used. The external resistance used was similar for programs A and B (approximately 80% 1-RM). Despite this similarity, larger impairments in muscle performance as a result of the 1-minute rest intervals in program

B were evident in the progressively greater reduction in the number of repetitions performed per set than that experienced with 3 minutes of rest (Table A2). Unlike previous studies, the training volumes between programs A and B in the present study were equated through the performance of additional sets by subjects in program B to account for the progressively greater reduction in repetitions performed per set. Consequently, the effectiveness of program B (in comparison to program A) in developing strength may be a result of performing similar training volumes as well as the use of similar relative training intensities.

During a 5-week resistance training study, significantly greater increases in 1-RM squat (7% compared to 2%) were observed following a program utilizing a 180-second rest interval between sets than a program incorporating only 30 seconds of rest before performing the next set (Robinson et al., 1995). Both training groups performed the same number of sets while using a 10-RM load. Robinson et al. (1995) suggested that the longer rest period of 180 seconds allowed for significantly greater training intensities (10-RM load) during each training session, therefore producing significantly greater strength gains. However, because of the discrepancies in training intensity as well as rest interval duration between these two training groups, it is difficult to conclude which factor was more responsible for the differences in strength improvements. In addition to these two training groups, a third training group incorporating 90-second rest intervals was included. Gains in strength experienced by the 90-second rest interval group were similar to those of the 180-second rest interval group. Further, the relative training intensity used by the 90-second rest interval group was not different to that used by the 180-second group. These findings support the observations of the present study in which protocol A

and B elicited similar increases in strength while using a similar relative training intensity.

Kulling, Hardison, Jacobson, & Edwards (1998) compared two, 12-week resistance training programs that were equated for all training variables with the exception of the duration of rest between sets. Significantly greater increases in leg press and bench press 1-RM of untrained subjects were observed in the training group that used 30 seconds of rest than the group that utilized 90 seconds of rest between sets. Perhaps if the duration of the present study had been longer than 8 weeks, the difference in 1-RM improvements would have been significant.

As stated previously, training volume and training intensity are considered important aspects of a training program designed to increase strength (Dudley et al., 1991; Baker, Wilson, & Carlyon, 1994; Pincivero et al., 1997, Stone et al., 2000). Because only small differences between the average training intensity used for all sets were evident between programs A and C (Table A2), examination and comparison of the changes in strength produced by these two programs may determine the importance of training volume in the development of muscle strength. Generally, studies investigating the effects of volume and intensity on increases in strength have compared periodized training programs or single- with multiple-set programs. Comparison of the results of the present study to those obtained in periodized models may be inappropriate because periodized training involves manipulating volume and intensity over the course of a training period (Willoughby, 1993) rather than during each training session, as in program C. In the present study, subjects in program C progressively reduced the relative intensity of the training load for each successive set following the completion of the

previous set to ensure that approximately 10 repetitions were performed per set. As a result the total training volume performed during program C was significantly greater in comparison to the total training volume accomplished while using a constant load (program A). However, despite the progressive reduction in training load during program C, there was no statistical difference between the average relative training intensity used for all sets between training groups A and C (Table A2).

Despite a significantly greater training volume, program C produced similar improvements in forearm flexor strength as program A. These results appear to confound previous suggestions regarding the importance of training volume in promoting increases in strength (Dudley et al., 1991). However, the effectiveness of low volume (1 set per muscle group), moderate volume (2 sets per muscle group) and high volume (4 sets per muscle group) resistance training on the development of maximal squat and bench press strength in moderately trained subjects was examined over 10 weeks (Ostrowski, Wilson, Weatherby, Murphy, & Lyttle, 1997). During the 10-week training study, subjects trained three times per week with each program utilizing equal relative training intensities and completing the same number of repetitions per set. Squat and bench press 1-RM increased in all groups with no differences between the three groups. Therefore, it was suggested that a minimum threshold level for training volume may exist, in which training volume performed exceeding the threshold fails to contribute to greater improvements in strength (Ostrowski et al., 1997). Accordingly, increases in upper and lower body 1-RM strength in recreationally trained adults were similar in response to performing either 1 set or 3 sets with an 8 to 12 RM load to failure (Hass, Gararella, De Hoyos, & Pollock, 1998). The authors concluded that 1 set of resistance training to

failure was as effective as 3 sets to failure in adults with previous strength training experience. Although each program in the present study consisted of performing multiple sets, one of the similarities between the three programs was the composition of the first set. During the first set of each program, subjects completed a similar number of repetitions to failure and trained with comparable relative training intensities (Table A2). If a minimum threshold for training volume does exist, this point may have been attained in the first set for all programs and consequently the training volume achieved beyond this point (even with the different composition of training variables of each program) failed to offer an additional stimulus.

One other similarity of the three training programs was that each set was performed until failure regardless of relative training intensity and number of repetitions performed. Training to muscle failure on each set is speculated to be a key component of a training program (Tesch, 1992) and represents maximal effort and maximal muscle activation (O'Hagan et al., 1995). It has been demonstrated that the IEMG signal of the forearm flexors (Kauhanen, Hakkinen, & Komi, 1989) and the quadriceps muscle groups (Hakkinen, Kauhanen, & Komi, 1988) increased during a set of dynamic submaximal (60% 1-RM) loading to failure. Similar increases in IEMG were observed in the pectoralis major and triceps brachii during one set of bench press using a 6-RM load (Keogh, Wilson & Weatherby, 1999). The increase in IEMG is believed to reflect the recruitment of new motor units or an increase in firing frequency of active motor units as a result of an acute reduction in the force capacity of fatigued muscle fibers (Hakkinen et al., 1988). It is possible that the above-mentioned changes in neuromuscular behavior occurred in the present study because all programs trained to failure with similar sub-

maximal loads. O'Hagan et al. (1995) suggested that a key physiological mechanism involved in the strength training stimulus is the force developed per active muscle fiber. It is possible that training to muscle failure in each set may progressively increase the force generated per active muscle fiber because fatigued fibers contribute less to the overall development of force. Alternatively, the recruitment of previously inactive fibers following the development of fatigue in previously contracting fibers increases the overall pool of fibers that are required to develop tension, which may be important in the development of strength.

Hakkinen, Alen, & Komi (1985) observed that increases in maximal isometric strength of the quadriceps during a 24-week resistance training study were associated with the degree of IEMG activity. If changes in dynamic neuromuscular behavior occurred during training in the present study and the changes were similar between programs because each program trained to failure with a similar training intensity, this mechanism may account for the similar increases in strength observed between the programs. In the investigation performed by Ostrowski et al. (1997) the similar increases in upper and lower body strength that were observed following three training programs of different volumes may have been influenced by all three of the groups training to failure.

Muscle Fiber Morphology

The three training groups in the present study produced similar and significant increases in the fiber area of both type II and type I muscle fibers (Table A3). The pre-training values as well as the degree of muscle fiber hypertrophy appear to be in agreement with results from previous investigations. The initial (pre-training) areas of type I and type II muscle fibers were greater than the pre-training values reported in

untrained subjects (MacDougall, Sale, Always, & Sutton, 1984; O'Hagan, Sale, MacDougall, & Garner, 1995) and were also greater than the range of pre-training muscle fiber areas of recreationally trained subjects reported by McCall et al. (1996). These discrepancies with the present study are probably a reflection of differences in the pre-training state of the subjects. However, the pre-training areas of the type I and type II fiber reported in the present study were less than those of intermediate and competitive body builders (Alway, Grumbt, Stray-Gunderson, & Gonyea, 1992; MacDougall et al., 1984).

The average relative increases in type I and type II fiber area (with the values collapsed from all three programs) was 10.5% and 10%, respectively (Table 3). The relative increase in type I fiber area is similar to the increases displayed in 11 recreationally trained subjects following a 12-week resistance training program using 10-RM training loads and 1-minute rest intervals (McCall et al., 1996). However, the relative increases in type II fiber area in the present study are less than those observed by McCall et al. (1996). Greater increases in type II muscle fiber hypertrophy were also observed following 20 weeks of training in subjects with an undefined pre-training history (O'Hagan et al., 1995). Alternatively, the absolute post-training muscle fiber areas achieved by the subjects in the current study approached (within one standard deviation) the range reported in intermediate body builders (MacDougall et al., 1984).

Increases in muscle size are thought to be best produced by training programs of a high training volume that utilize 8-12 repetitions per set and short inter-set rest periods (Houston, 1999). This study was designed to independently determine A) the effectiveness of different rest intervals, while controlling for training volume and

intensity (Program A vs. Program B), and B) the role of training volume, while controlling for rest interval duration (Program A vs. Program C) on the development of muscle size.

Despite much of the strength training literature endorsing the use of short rest intervals to increase muscle size, to date there are no other investigations comparing the effectiveness of strength training programs utilizing different inter-set rest intervals on changes in muscle size. The results of the present study suggest that short (1-minute) rest intervals were as effective as longer (3-minute) rest intervals in eliciting muscle hypertrophy. With regards to training volume, it would appear that a greater training volume does not provide a greater stimulus for muscle hypertrophy as program C, which performed a significantly greater training volume than program A, did not produce significantly greater increases in muscle size. These finding support the results observed by Ostrowski et al. (1997) in which increases in muscle CSA of the rectus femoris were similar following low-, moderate-, and high-volume training. Additionally, one set of 8-12 reps to failure produced similar increases in muscle thickness of experienced subjects during the first 13 weeks of a 25 week resistance training study (Pollock, Abe, De Hoyos, Garzarella, Hass, & Werber, 1998). However, during the final 12 weeks of this study the 3 set training group proved more effective in increasing muscle size. Perhaps, the duration (8 weeks) of the present study was not long enough to allow the differences to develop between the training groups despite using subjects with previous training experience.

Another objective of this study was to gain a better understanding of the mechanisms underlying muscle hypertrophy, with particular reference to fatigue. Schott

et al. (1995) have suggested that increases in muscle size are related to the acute metabolic fatigue accumulated through resistance training. Therefore, it was also the purpose of this study to clarify the role of fatigue in the development of muscle hypertrophy. If fatigue is an important stimulus for muscle hypertrophy, as indicated by Schott et al. (1995), it was speculated that the 1-minute rest interval program (program B) would increase muscle size to a greater extent than program A. Accordingly, it was hypothesized that the use of shorter rest periods would result in a greater acute accumulation of free creatine (Cr) (due to limited re-phosphorylation time) and H^+ (because of a greater dependence on anaerobic glycolysis) than a program using a 3-minute rest-interval. It would appear that both programs elicited similar changes in H^+, as there were no differences between changes in blood lactate between these two programs (Section B). However, the effect of either program on acute changes in free Cr was unknown because it was not measured. Therefore, the physiological mechanisms underlying the similar levels of fatigue (as measured by changes in MVIC and blood lactate) produced by the protocols incorporated in each program may have also been similar.

When compared to program C, the protocols used during program A resulted in less acute fatigue as measured by an acute reduction in MVIC (Section C). Further, differences in the acute changes in blood lactate were approaching significance with the protocols incorporated by program C resulting in greater increases in blood lactate. If fatigue is important in the development of muscle size (Schott et al., 1995), program C should have elicited a greater increase in muscle fiber area. Although the changes in muscle fiber area were greater following program C, the differences with program A

were not significant. Thus it would appear the results of the present study fail to support those of Schott et al. (1995), in which fatigue is thought to be important to the development of muscle size.

Unlike previous research (MacDougall, 1992; McCall et al., 1996), the increases in fiber area following programs A and B did not demonstrate a greater degree of type II hypertrophy. The short rest intervals of program B may have placed a greater demand on the type I fibers due to their greater resistance to fatigue than a portion of the type II fiber pool. The contribution of the highly fatigable pool of the type II fibers during training may have been limited following the first set because of the brief rest intervals. Thus, the activation of type I fibers may have increased and produced a larger degree of hypertrophy than the type II fibers.

The interpretations of the present study should be made with some caution due to the short duration of the present study, the small sample size in each program, and errors inherent with the muscle biopsy technique. As mentioned previously, the short (8-week) duration of the present resistance training study may not have been sufficient to permit differences between the three programs to emerge.

Despite the absence of a significant interaction effect, subjects in program B (12.6 & 9.4%) and C (12.5 & 14.2%) experienced a greater relative increase in type I and type II fiber area than subjects in program A (7.0 & 6.2%). It is likely that the small sample size of each program as well as the larger standard deviations in the fiber areas contributed to the lack of an interaction effect. It is recognized that small sample sizes in combination with large variability limit data interpretations and limit statistical power (McCall et al., 1996). However, McCall et al. (1996) reported standard deviations of

24% and 27% of the mean for type I and type II post-training fiber areas, respectively and this variance is slightly larger than the variance observed in the present study. Therefore, with a larger sample size it is plausible that a significant interaction effect between the three programs would have occurred.

Interpretation of the results of the present study may also be limited due to methodological considerations inherent to the muscle biopsy procedure and subsequent analysis. The accuracy and reliability of determining muscle fiber area appear to be affected by the number of muscle fibers that are sampled (Alway, Grumbt, Gonyea, & Stray-Gunderson, 1989; Lexell, Taylor, & Sjorstrom, 1985; McCall, Byrnes, Dickinson, & Fleck, 1998). It has been suggested that a minimum of 200 fibers of each type must be measured to accurately determine fiber area in elite body builders (Alway et al., 1989). McCall et al. (1998) investigated the effectiveness of different sample sizes (in intervals of 5 starting at 5 fibers up to 100 fibers) in pre- and post-training muscle biopsy samples of the biceps brachii on the accuracy of measuring mean fiber area. Although samples of 5 and 10 fibers produced strong correlations with the mean fiber area attained with measuring 100 fibers, these correlations did not become significant in both type I and II fibers until 50 measurements of each fiber type were performed. Therefore, it was concluded that 50 muscle fibers of each fiber type (ST and FT) should be measured to accurately represent the area of each fiber type within an individual muscle biopsy. The average area of each muscle fiber type, when fewer than 50 muscle fibers were measured, is generally overestimated (McCall et al., 1998). In the present study because a range of only 8 – 25 fibers per fiber type were analyzed, the accuracy of the reported fiber areas of type I and type II fibers is questionable.

The accuracy of single site muscle biopsy procedures on representing whole muscle fiber types, individual fiber area, and changes in these parameters as a result of training is uncertain (Lexell et al., 1985). The amount of muscle hypertrophy has been demonstrated to vary throughout the length of a muscle (Narici, Hoppeler, Kayser, Landoni, Claassen, et al., 1996; Narici, Roi, Landoni, Minnetti, & Cerretelli, 1989). Therefore, a biopsy from a single site (as performed in the present study) may not be an accurate reflection of whole muscle increases in size. Additionally, Lexell et al. (1985) observed that sampling at multiple (2-3) biopsy sites of the vastus lateralis reduced the variance in fiber area associated with single site procedures. Although multiple sampling may be possible with a larger muscle group, performing multiple biopsies in a smaller muscle group like the biceps brachii seems unrealistic due to concerns for the subject.

In order for accurate analysis and comparison of fiber size to be made, true transverse sections of the biopsy sample are required (Anderson, 1997). If oblique sections are used, in which the muscle fibers profiles may be slightly elongated, the areas that are measured will be misleading. Although only fibers representative of a true cross-section were analyzed as recommended by Tesch et al., (1984), and all analyses were performed with subject identity and sampling time unknown to the investigator as recommended by McCall et al., (1996), it is possible that some fiber measurements may have occurred outside true transverse sections.

Conclusion

The three training programs examined in the present study produced similar gains in 1-RM and 10-RM strength. Similarities between the three programs, with regard to training variables, that may account for these results include the number of repetitions

performed during the first set, the relative training intensity used during the first set as well as the performance of each set until muscle failure. Although all three training programs elicited significant increases in muscle fiber area, methodological considerations limited between-group comparisons and interpretations.

Section B: Results and discussion for the effects of rest interval duration on the acute neuromuscular response to resistance training.

Results

Descriptive characteristics of the subjects who completed this component of the present study are listed in Table B1.

Training volume performed

The twelve subjects that completed Protocol A and B performed 17.9 ± 3.4 and 17.0 ± 2.1 units of training volume (Table B2). There was no difference in the amount of training volume performed between these protocols.

MVIC

The force produced during MVIC decreased from 104.4 ± 11.1 Nm to 83.1 ± 11.7 Nm following protocol A and from 110.3 ± 14.4 to 84.0 ±13.1 Nm in response to protocol B (Figure B4). There were no pre-protocol differences between the two groups in the magnitude of force produced. MVIC was significantly reduced following both protocols (main effect, $p<0.05$) but no interaction effect was observed.

Force-time curves

Following protocol A and B, the force-time curve was shifted significantly downwards at all time periods (main effect, $p<.05$) (Figure B5). There were no differences between the two protocols in the magnitude of decrease in absolute force at any of the time periods.

IEMG

There were no significant differences pre- to post-protocol as well as between protocols in changes in IEMG mean amplitude and mean power frequency when comparing protocol A and B (Figure B6 & B7).

Blood Lactate

Protocols A and B both elicited significant increases in blood lactate (main effect, $p<0.05$). Pre-protocol blood lactate levels were 1.4 ± 0.38 mmol/l and 1.6 ± 0.36 mmol/l during protocols A and B, respectively (Figure B8). Blood lactate increased to 3.5 ± 1.2mmol/l following protocol A and to 3.6 ± 1.3 mmol/l following protocol B (Figure B8). There was no significant difference between the magnitude of increase in blood lactate between protocols A and B.

Table B1. Characteristics of the subjects who completed fatigue protocols A and B (N = 12).

Subject Characteristics	Mean (SD)
Body mass (kg)	87.3 (12.1)
Height (cm)	180.5 (4.3)
Age (yrs)	25.1 (3.7)

Table B2. Mean (and SD) total repetitions, time under tension and number of sets completed during fatigue protocols A and B (N = 12).

Training Variable	Protocol A	Protocol B
Total repetitions	22.8 (4.4)	21.8 (2.4)
Mean Repetitions per set	7.6 (1.5)	5.1 (1.4)*
Total training volume	17.9 (3.4)	17.0 (2.1)
Time under tension (s)	89.1 (16.1)	87.6 (15.7)
Number of sets	3.0 (0.0)	4.3 (1.1)
Training intensity (all sets)	77.3 (2.8)	77.3 (2.8)

* Represents significant difference from protocol A ($p<.05$).

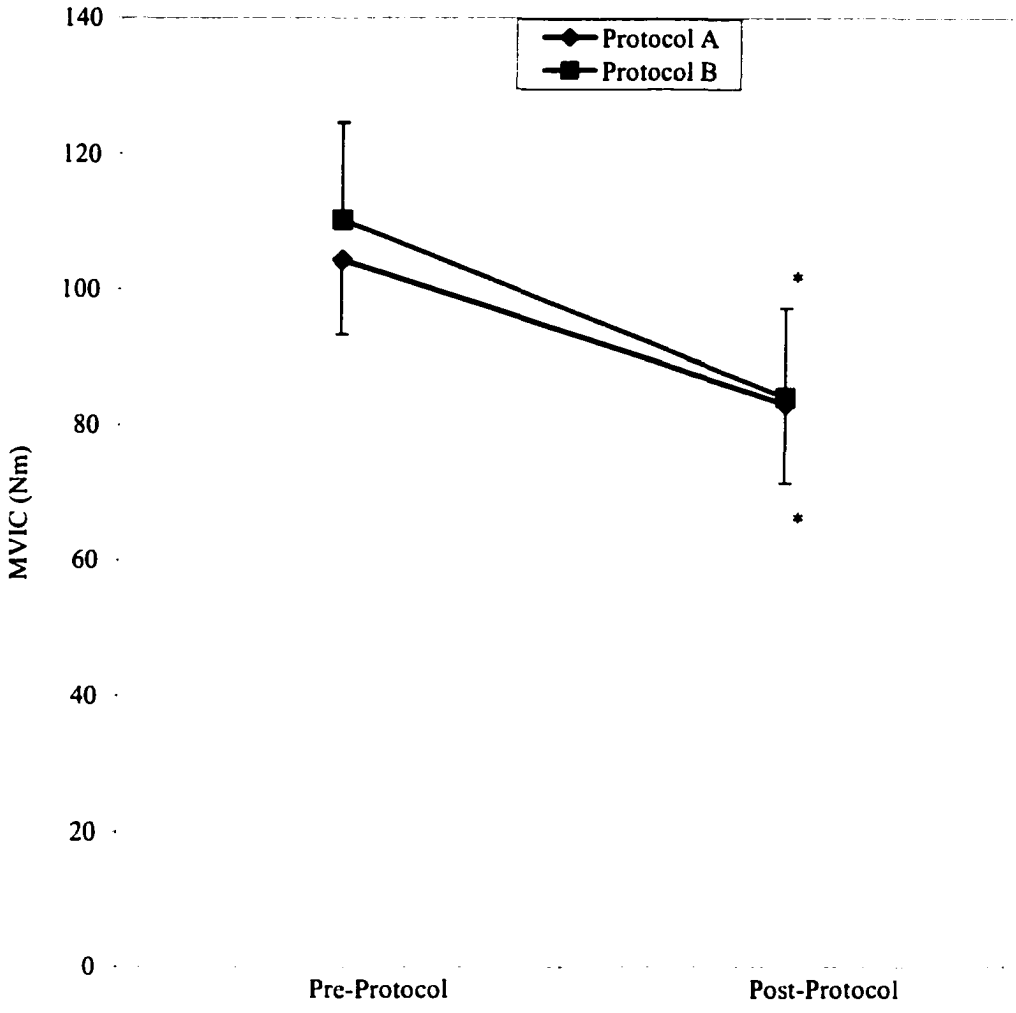


Figure B4. Mean (SD) force produced during single-arm flexor maximal voluntary isometric contractions performed pre- and post-protocol (* represents significant difference from respective pre-protocol value. $p<.05$).

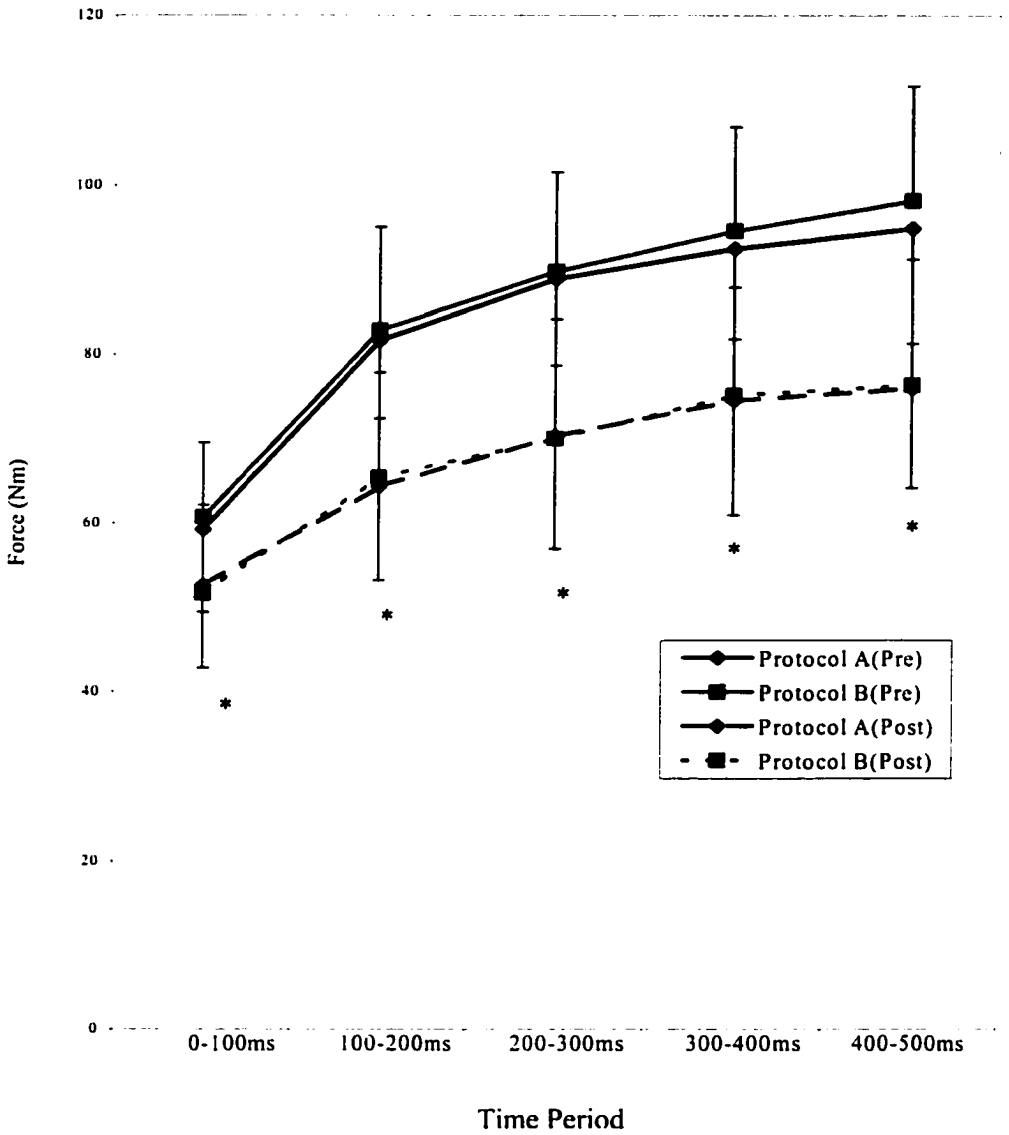


Figure B5. Mean (SD) force-time curves during maximal voluntary isometric contractions performed before and after protocol A and Protocol B (* represents significant difference from corresponding pre-protocol value during the same time period. $p<.05$).

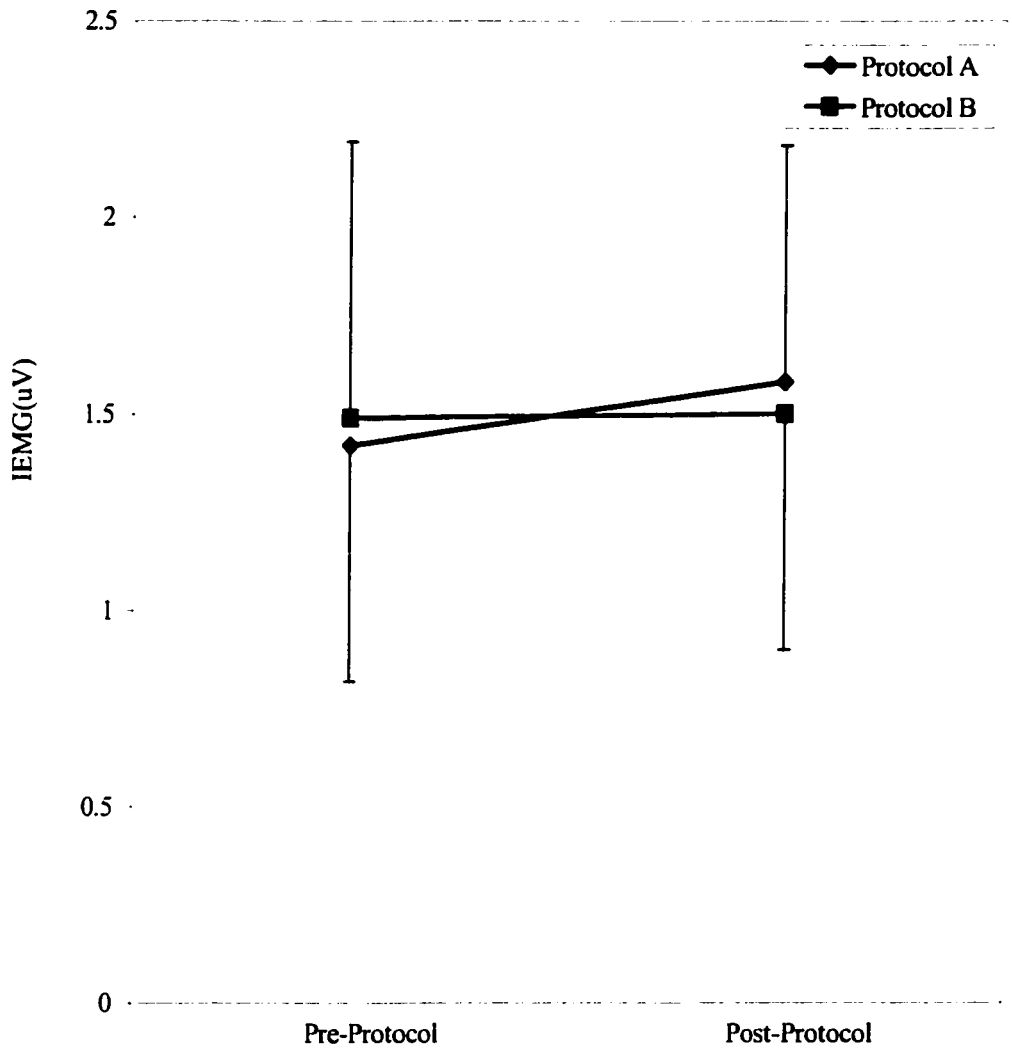


Figure B6. Mean (SD) values for the Biceps Brachii mean IEMG activity recorded during maximal voluntary isometric contractions performed prior to and upon completion of fatigue protocol A and Fatigue protocol B.

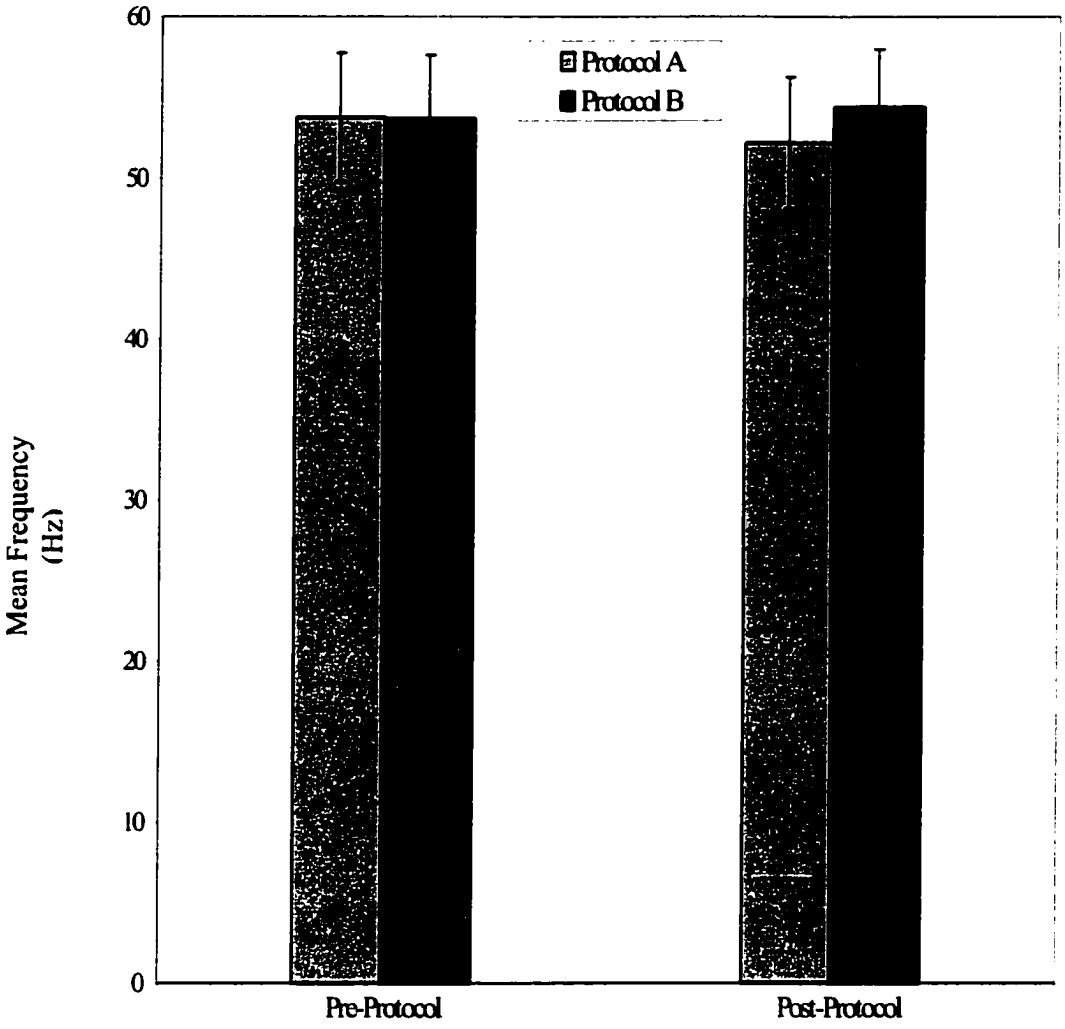
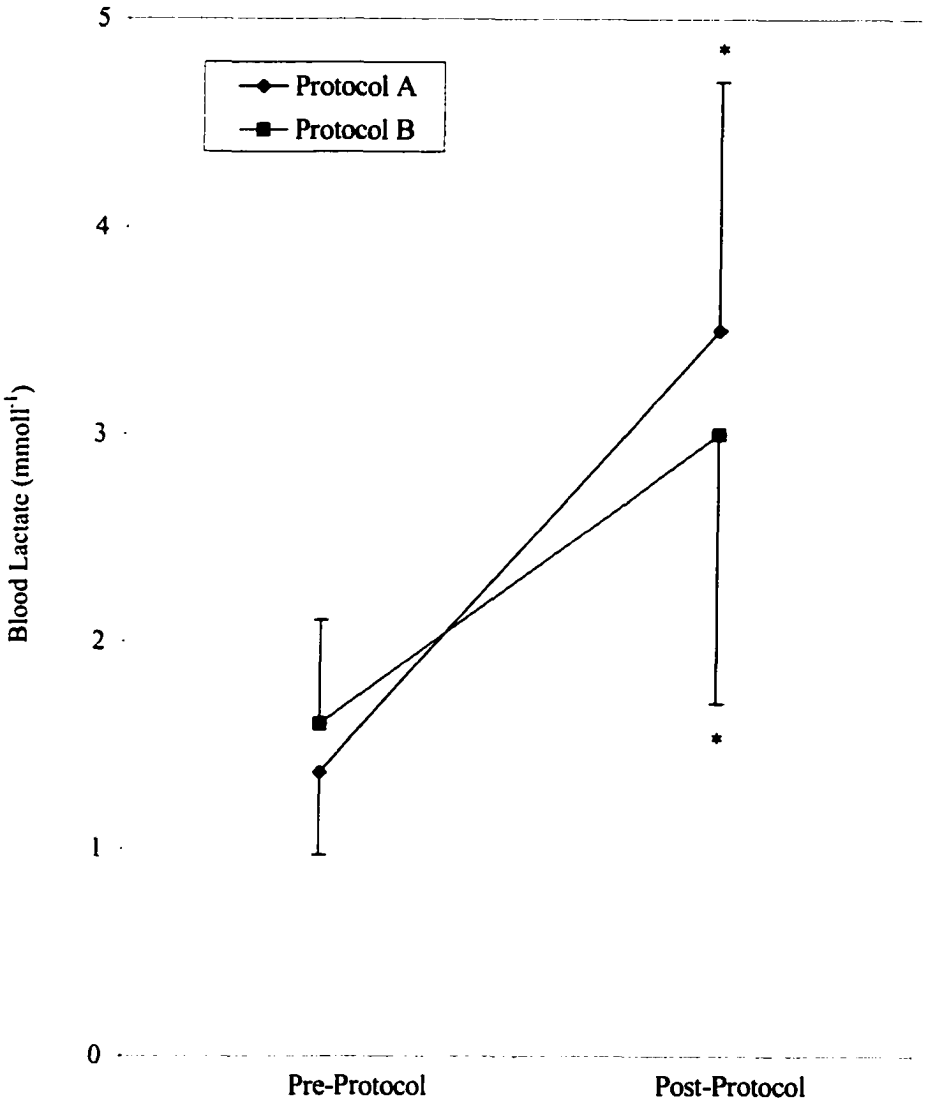


Figure B7. Mean (SD) values for the Biceps Brachii mean frequency of the power spectrum of the IEMG activity recorded during maximal voluntary isometric contractions performed prior to and upon completion of fatigue protocol A and Fatigue protocol B.



FigureB8. Mean (SD) blood lactate values before and five minutes following the performance of fatigue protocols A and B (* represents significant difference from respective pre-protocol value. $p<.05$).

Discussion

The primary findings of the present study indicated that acute neuromuscular fatigue associated with resistance training is similar between protocols of equal training volume but different in inter-set rest interval length. Reductions in MVIC and explosive force (force-time curve) as well as increases in blood lactate were similar during protocols A and B and neither program elicited a significant change in average IEMG amplitude pre- to post-protocol.

Strength Performance during protocol

The mean total number of repetitions accomplished during protocol A and protocol B equaled approximately 23 and 22, respectively (Table B2). However, while only 3 sets were performed during protocol A, an average of 4.3 sets was necessary during protocol B to equate the volume that was achieved during protocol A (Table B2). The difference in the number of sets performed between protocol A and B was significant and consequently, the reduction in the number of repetitions performed per set was greater in protocol B.

The greater drop-off in performance (reps/set) of protocol B supports the observations of other investigations measuring muscle performance during resistance training sessions using different rest intervals (Abdessemed et al., 1999; Ballantyne & Sale, 1999; Pincivero, Lephart, & Karunakara, 1998). A greater decrement in the number of repetitions performed per set of leg press was observed during a protocol incorporating 1-minute rest intervals than during a protocol providing three minutes of rest between sets (Ballantyne & Sale, 1999). Abdessemed et al. (1999) had subjects perform 10 sets of 6 repetitions of bench press with a constant load of 70% 1-RM using 1-, 3-, and 5-minute

rest intervals. Significantly greater reductions in the mean power per repetition of bench press was observed following the protocol using a 1-minute rest interval than when using three or five minutes of rest. However, these differences did not emerge until after the third set and only occurred in the final 3 of the 6 repetitions performed in each set. Furthermore, only 4 of 10 subjects were able to complete all 10 sets when 1-minute rest intervals were incorporated, which would seem to indicate that the 1-minute protocol was more fatiguing than the 3- and 5-minute protocols. Similarly, peak torque and total work of the quadriceps and hamstring muscle groups during 4 sets of 10 maximal isokinetic repetitions were significantly reduced during a protocol incorporating 40 seconds of rest but remained unaffected with 160 seconds of rest (Pincivero et al., 1998).

The ability of a muscle to repeat high force contractions, as in a set of resistance training, is dependent on the production of ATP from the breakdown of PCr and muscle glycogen (MacDougall et al., 1999; Wenger & Reed, 1976). Following a set of heavy resistance training it has been observed that complete restoration of PCr levels does not occur until about three minutes after the performance of the final repetition (MacDougall et al., 1999). Previously, it has been determined that acute intra-muscular PCr levels approached complete exhaustion following a high-volume resistance training protocol utilizing 1-minute inter-set rest periods (Tesch et al., 1986). Tesch et al. (1986) also observed a gradual decrease in the number of repetitions performed per set during each of the four lower body exercises. Additionally, the mean power produced per repetition was reduced to a greater extent during the final three repetitions of a set during a resistance training protocol incorporating 1-minute rest periods than when 3- or 5-minute rest intervals were used (Abdessemed et al., 1999). It was concluded that limited PCr

availability during the 1-minute protocol accounted for the reduction in power during the final three repetitions per set (Abdessemed et al., 1999). Therefore, during the present study, PCr levels following 1-minute of rest may have been reduced to a greater extent prior to the performance of successive sets when compared to PCr levels after 3 minutes of rest because of the limited time for re-phosphorylation. Reduced PCr levels at the onset of each set in comparison to the preceding set (with the use of 1-minute rest intervals) (Abdessemed et al., 1999) may have accounted for the progressively greater drop-off in repetitions completed per set when compared to protocol A. During the three minutes of recovery provided during protocol A PCr levels should have approached or reached resting levels, therefore other mechanisms may have been responsible for the progressive decrease in performance observed during this protocol.

With less PCr available at the onset of each successive set, subjects during protocol B may have relied more heavily on anaerobic glycolysis for the re-synthesis of ATP. Modest but significant depletions in muscle glycogen have been observed following various heavy resistance training protocols (MacDougall et al., 1999; Pascoe et al., 1993; Robergs et al., 1991; Tesch et al., 1986; Tesch, Ploutz-Snyder, Ystrom, Castro, & Dudley, 1998). Following 3 sets of single-arm elbow flexion with three minutes of rest between sets, MacDougall et al. (1999) concluded that the breakdown of muscle glycogen was a significant contributor in the re-synthesis of ATP. As protocol A of the present study was similar to that investigated by MacDougall et al. (1999) it would appear reasonable to suggest that significant muscle glycogen depletion occurred during this protocol. A greater depletion of muscle glycogen may have occurred during protocol B if less PCr was available for ATP production at the onset of each successive set and

therefore placing a greater emphasis on glycogenolysis. Although muscle glycogen depletion is evident during resistance training, previous studies investigating the extent of muscle glycogen utilization during resistance training have demonstrated that muscle glycogen stores are not fully exhausted (MacDougall et al., 1999; Pascoe et al., 1993; Robergs et al., 1991; Tesch et al., 1986; Tesch, et al., 1998). Thus, the role that diminished (but not exhausted) muscle glycogen stores play during fatigue is considered insignificant (Tesch et al., 1986). Even if protocol B elicited greater reductions in muscle glycogen than protocol A, it is uncertain whether the magnitude of muscle glycogen depletion would have accounted for the greater impairments in muscle performance. Tesch et al. (1986) suggested that the magnitude of glycogen depletion was not sufficient to account for reduction in performance observed during successive sets with 1-minute rest intervals. Because changes in intra-muscular levels of muscle glycogen were not analyzed during the present study, conclusions regarding muscle glycogen utilization cannot be established.

Another potential mechanism responsible for the acute decrement in strength performance observed in both protocols may be the progressive slowing in the rate of muscle glycogen utilization over the course of multiple sets of resistance training (MacDougall et al., 1999; Robergs et al., 1991). Associated with increasing demands on anaerobic glycolysis to re-supply ATP is the production of lactic acid (Tesch et al., 1986; Wenger & Reid, 1976). Decreases in intra-muscular pH during high-intensity contractions, that are associated with the production of lactic acid, may slow the rate of glycolysis through the inhibition of glycolytic enzymes and thus limit ATP production (Hannie, Hunter, Kekes-Szabo, Nicholson, & Harrison; Tesch et al., 1986; Wenger &

Reid, 1976). The production and accumulation of muscle lactate during protocol B (in comparison with protocol A) may have been magnified due to a greater possible dependence on glycolysis and shorter rest periods that would limit the removal of lactate and H^+.

Increases in blood lactate measurements are suggested to reflect increases in muscle lactate and pH (Tesch et al., 1986). If a slowing of glycolysis, as a result of decreasing muscle pH, was responsible for the greater drop-off in repetitions performed per set, blood lactate should have been higher following protocol A than B. However, the increases in blood lactate following protocol B were similar to those after protocol A. These observations confirm those by Ballantyne & Sale (1999), in which increases in blood lactate were similar in response to 5 sets of leg press using a load of 85% 1RM performed with either 1- or 3-minute rest intervals. Likewise, Hannie et al. (1995) compared two types of recovery on bench press performance during successive sets. Although one of the protocols resulted in a greater reduction in the number of repetitions performed per set over 4 sets, this decrement (like in the present study) was not associated with a greater increase in blood lactate. In contrast, Abdessemed et al. (1999) found significantly greater elevations in blood lactate concentrations following a resistance training protocol utilizing 1-minute rest intervals than when 3-minute rest intervals were used. A significant relationship between the number of repetitions performed with 65% of 10-RM and blood lactate concentrations was observed following the completion of 6 sets of 10 repetitions with greater reductions in performance associated with higher blood lactate levels (Corder, Potteiger, Nau, Figoni, & Hershberger, 2000). The lack of difference in blood lactate response between the two

protocols in the present study may be attributed to the greater reduction in the number of repetitions performed per set during protocol B. Fewer repetitions completed in the final sets of protocol B (in comparison to protocol A) would have placed less metabolic demand on the muscle and consequently less lactate may have been produced. However, because blood lactate concentration is a balance in the accumulation and production as well as removal, distribution, and utilization of lactic acid (Corder et al., 2000), actual differences between protocols A and B in lactate accumulation and production may not have been measured.

In the present study, blood lactate was assessed prior to and 5 minutes following the completion of the final repetition of the last set. It is possible that a difference between the two protocols would have existed but was not detected due to sample times used. Tesch et al. (1986) observed that the highest blood lactate concentrations occurred midway through a high-volume training protocol incorporating 1-minute rest intervals. Perhaps blood samples administered while each protocol was in progress (rather than 5 minutes after) may have identified a difference between the two protocols. Alternatively, early and significant production and accumulation of lactate during protocol B, accounting for the progressively greater reductions in strength performance, might have also compromised subsequent lactate production because fewer repetitions were performed during subsequent sets which would account for the similar increases in blood lactate observed in both protocols.

Reductions in muscle pH may also impair excitation-contraction coupling, consequently limiting the amount of Ca^{2+} released from the sarcoplasmic reticulum (McLester Jr., 1997). Less available intra-muscular Ca^{2+} would activate fewer cross-

bridge binding sites and therefore reduce the number of active cross-bridges. Because force is related to the number of active cross-bridges, fewer cross-bridges would translate into less force and reduced performance.

MVIC and IEMG

Despite a greater reduction in strength performance while performing protocol B, subjects demonstrated a similar reduction in MVIC following both protocols. In comparison to pre-protocol measures, MVIC was reduced by 20.3% and 23.3% following protocol A and B, respectively (Figure B4). The magnitudes of these decreases are in agreement with previous studies measuring the resistance training-induced acute decrements in forearm flexor MVIC of trained subjects (Behm et al., in press; Kauhanen et al., 1989). In recreationally trained subjects, Behm et al. (in press) observed an average 21.4% reduction in MVIC of the forearm flexors 30 seconds following one set of a single-arm elbow flexion exercise performed with 5-, 10-, and 20-RM loads. Maximal isometric force producing capabilities of the forearm flexors of well trained males were diminished by approximately 15 to 20% following a single set of approximately 15 repetitions to failure against an accommodating resistance of 60% 1-RM (Kauhanen et al., 1989).

MacDougall et al. (1999) indicated that the extent of the decrease in muscle force generating ability experienced when performing a set of resistance training until failure is related to the training load used. Thus, if failure occurred with a training intensity of 80% 1-RM, the muscle is not fully fatigued and only a 20% reduction in maximal force-generating capacity should be experienced. Accordingly, training to failure with similar loading intensities used in protocols A (77.3% 1-RM) and B (77.3% 1-RM) may account

for the similar reductions in MVIC observed in the present study. This rationale assumes that the fatigue mechanisms that influence repetitive dynamic force production also affect maximal isometric force production.

Factors underlying the capacity to develop maximal force during an isometric contraction include the number of active of cross-bridges and the force generated per cross-bridge (Westerblad, Allen, Bruton, Andrade, & Lannergren, 1998). Any impairment in the neural, metabolic, and/or mechanical events associated with cross-bridge kinetics will compromise maximal isometric force (Westerblad et al., 1998). Although an acute reduction in MVIC suggests an impairment in neuromuscular performance, it fails to identify the specific underlying mechanisms and locations that may be involved (Vollestad, 1997).

The absence of any observable change in average IEMG amplitude (Figure B6) in association with the decrement in MVIC observed in the present study supports the findings of previous studies investigating the acute neuromuscular fatigue produced from resistance training (Hakkinen et al., 1988; Kauhanen et al., 1989; Linnamo, Newton, Hakkinen, Komi, Davie et al., 2000). Like the present investigation, these studies failed to demonstrate a significant change in IEMG despite significant reductions in MVIC following fatiguing resistance training protocols. These results combined with those of the present study suggest that neural drive was not affected by either protocol A or B and the reductions in maximal isometric force producing abilities were independent of changes in neural drive. In contrast, significant reductions in IEMG have been observed in parallel with decreases in MVIC following the performance of various resistance training protocols (Behm et al., in press; Hakkinen, 1994; Linnamo, Hakkinen, & Komi,

1998). The results from these studies indicate that fatigue-induced acute decrements in MVIC were partially attributed to reductions in neural drive. However, although IEMG and MVIC were reduced post-protocol, the time line for the recovery of IEMG and MVIC differed (Behm et al., in press, Linnamo et al., 1998). Following a single set of an elbow flexion exercise using loads of 5-, 10- and 20-RM, IEMG activity of the biceps brachii returned to pre-exercise values following 3 minutes of recovery whereas MVIC remained significantly reduced (Behm et al., in press). Similarly, Linnamo et al. (1998) observed that maximal isometric force remained impaired for 2 days following the performance of 5 sets of leg extensions using a 10-RM load while IEMG had recovered within one hour post-protocol. These findings support the observations of the present study, which suggest the acute decrements in MVIC induced by resistance training protocols are not associated with changes in neural drive. Therefore, it would appear that peripheral factors are responsible for the acute reductions in MVIC

The significant and similar increases in blood lactate in response to both fatiguing protocols indicate that peripheral factors may be associated with the reduction in MVIC (Hakkinen, 1993, 1994). As previously mentioned, the elevations in blood lactate in response to the two fatiguing protocols in the present study were similar to the increases in blood lactate experienced in well trained individuals following one set of a singe-arm flexion exercise using a load of 80% 1-RM but were less than those produced after 3 sets (MacDougall et al., 1999). Increases in blood lactate concentration suggest that energy demands of the contracting muscle have been partially met through anaerobic glycolysis (Abdessemed et al., 1999; Tesch et al., 1986). It was thought that protocol B would elicit greater increases in blood lactate than protocol A because the shorter inter-set rest

intervals would compromise the replenishment of PCr and thus the contracting muscle would have to primarily rely on glycolysis, rather than the breakdown of PCr, to re-synthesize ATP. Shorter recovery times would also limit the amount of lactate removed from the working muscle. The lack of difference in the increases of blood lactate appears to suggest this may not have been the case. However, protocol B produced a greater decrease in the number of repetitions performed per set compared to protocol A. Fewer repetitions performed per set would have reduced the demand on anaerobic glycolysis and may account for the lack of differences in blood lactate concentrations produced by the two protocols. These results support previous observations in which increases in blood lactate were similar following resistance training protocols incorporating 1- and 3-minute rest intervals (Ballantyne & Sale, 1999). Like the present study, the 1-minute rest interval protocol produced a greater reduction in the number of repetitions performed per set over five sets (Ballantyne & Sale, 1999).

Measures of blood lactate are suggested to reflect metabolic changes within the working muscle (Tesch et al., 1986). A reduction in muscle pH will reduce glycolytic enzyme activity as well as delay the re-phosphorylation of PCr (Tesch et al., 1986). Consequently, the re-synthesis of ATP will be compromised and maximal force development will be impaired. In the present study, the average elapsed time between the final repetition and the MVIC was 10.1 seconds. If PCr was completely exhausted upon completion of the final repetition, PCr levels would have reached approximately 15% of resting levels by the onset of the MVIC. Therefore, it is uncertain that inadequate PCr levels were a cause of the decrements in MVIC. During the brief period separating the

final repetition and MVIC, minimal lactate would have been removed and contributed to the acute reduction in maximal force generation (MacDougall et al., 1999).

The accumulation of hydrogen ions (H^+) may also alter muscle membrane resting potential (Wenger & Reed, 1976). Consequently, the excitability of the sarcolemma and the permeability of the sarcolemma to Na^+ and K^+ may be reduced. Both of these would limit the amplitude and frequency of action potential propagation, therefore compromising excitation-contraction coupling. It has also been suggested that an accumulation of H^+ may interfere with the binding of Ca^{2+} to the actomyosin active binding site (Wenger & Reed, 1976). Consequently, the number of cross-bridges formed would be reduced producing a decrement in maximal force generating abilities. Sahlin & Ren (1989) observed the return of maximal isometric force generating capacity to pre-fatigue levels within 2 minutes of recovery following the performance of continuous isometric contractions at 60% of maximum. Although, muscle performance was restored in the two minutes, muscle pH remained below resting values (Sahlin & Ren, 1989). Therefore, it was suggested that because force recovers more quickly than intra-muscular pH, the accumulation of H^+ may not be a mechanism underlying the reduction of maximal isometric force in muscular fatigue (Sahlin & Ren, 1989). Because dynamic contractions, such as those used in the present study, may elicit a different degree and type of fatigue than during isometric contractions, H+ is considered a potential mechanism that contributed to the acute reductions in MVIC.

Additionally, strenuous activity appears to disrupt Na^+ and K^+ balance as a result of the high frequency of action potential discharge required for force development (Medbo, Jebens, Vikne, Refsnes, & Gramvik, 2001; Vollestad, 1997). De Luca (1997)

has added that action potential conduction velocity and propagation may be impaired with Na^+, K^+ and H^+ imbalances. The frequency component of the EMG power spectrum appears to be sensitive to changes in muscle fiber conduction velocity, such that with fatigue the EMG power spectrum shifts towards lower frequencies (De Luca, 1997). In the present study, mean power frequency during the MVIC did not change from pre-fatigue levels in both protocol A and B. These results support those observed by Linnamo et al. (2000) in which no changes were observed in median power frequency during maximal isometric contractions prior to and immediately following the completion of 5 sets of leg extensions using a 10-RM load.

Accompanying the utilization of intra-muscular PCr and ATP are concurrent increases in P_i concentration (McLester Jr., 1997; Westerblad et al., 1998). The release of P_i during the cross-bride power stroke is thought to be critical to force development (McLester Jr., 1997). An accumulation of intra-muscular P_i, which may prevent the release of P_i from the cross-bridge head, is believed to reduce the force generated per cross-bridge, thus potentially impairing maximal voluntary force (McLester Jr., 1997; Westerblad et al., 1998). Increases in P_i were observed in response to continuous and intermittent isometric fatiguing protocols with greater increases in P_i subsequent to the continuous protocol incorporating a 1-minute rest interval (Schott, McCully, & Rutherford, 1995). Acute changes in P_i following resistance training protocols, like those utilized in the present study, are unknown. However, if there were an accumulation of P_i in response to the protocols of the present study, it is thought that the degree of Pi accumulation would be similar between the two protocols. Despite the use of different rest intervals, both protocols were performed to muscle failure. Consequently, the degree

to which ATP and PCr were utilized may have been similar between the two protocols and, therefore, resulted in similar levels of P_i accumulations.

Raastad & Hallen (2000) observed a 12-14% decrease in maximal force production 5 minutes following the completion of a heavy (3- to 6-RM) resistance training protocol. The researchers proposed that a depression in Ca^{2+} release from the sarcoplasmic reticulum (SR) was largely responsible for impairing maximal force production because other force-reducing metabolic disturbances may have recovered in the 5 minutes separating the final repetition and the maximal strength test. A reduction in SR Ca^{2+} release may be due to a change in excitation-contraction (E-C) coupling (Raastad & Hallen, 2000) or a change in muscle metabolite levels (increased P_i and reduced ATP) (Westerblad et al., 1998).

Explosive Force

The two fatiguing protocols used in the present study resulted in similar and significant downward shifts in the force-time curves indicating a reduction in explosive force production (Figure B5). Similar reductions have been observed in the force-time curves of the quadriceps following one set of repetitions to failure with a load of 60% 1-RM (Kauhanen et al., 1989) and following 10 sets of squats using a 10-RM load (Hakkinen, 1994). Since IEMG was not analyzed during this period, it is difficult to determine if neural factors were responsible for the decrements in explosive force observed in the present study. Explosive force is dependent on the number of cross-bridges as well as the rate at which cross-bridges can recycle (Westerblad et al., 1998). A proposed peripheral factor that may influence the explosive force is H^+ accumulation because of the inhibition of myofibrillar ATPase (Westerblad et al., 1998). An inhibition

of myofibrillar ATPase ultimately limits the release of ADP from the myosin head, which is necessary for cross-bridge detachment and re-attachment (McLester Jr., 1997).

Conclusion

Significantly greater reductions in forearm flexor strength performance (reps/set) during the resistance training protocols were observed when 1-minute rest intervals between sets were used than when three minutes of rest separated sets. It appeared that peripheral mechanisms were responsible for the greater reduction in performance elicited with 1-minute rest intervals. When fatigue was assessed following both protocols, MVIC was significantly reduced, but there were no differences between protocols A and B with regards to the magnitude of the decrement. Peripheral mechanisms, particularly reductions in muscle pH, were attributed to the impairment of maximal force generating capacity because both protocols elicited significant increases in blood lactate whereas IEMG remained unchanged. Future research should be directed at identifying the specific peripheral mechanisms that may be associated with resistance-training induced fatigue.

Section C: Results and Discussion for the acute neuromuscular response to resistance training protocols of different training volumes but equated for rest interval length.

Results

Descriptive characteristics of the subjects who completed this component of the present study are listed in Table C1.

Acute neuromuscular response

Comparisons of the acute measures of neuromuscular performance were made between Protocols A and B as well as A and C because not all of the subjects were able to complete all three of the fatiguing protocols. Twelve subjects completed the requirements for protocols A and B whereas 14 subjects completed both protocols A and C.

Training volume performed

Training volume performed during protocol C (21.7 ± 2.7 units) was significantly greater ($p<0.05$) than that performed by the same subjects during protocol A (17.3 ± 3.5 units) (Table C2).

MVIC

When comparing protocols A and C, MVIC decreased from 104.6 ± 12.8 Nm to 83.3 ± 11.9 Nm and from 108.9 ±12.6 Nm to 81.5 ± 8.1 Nm, respectively (Figure C4). Both protocols produced significant decrements in MVIC (main effect, $p<.05$) with the reductions in MVIC elicited during protocol C being significantly greater than those produced from protocol A (measurement time x protocol interaction effect, $p=.05$).

Force-time curves

The force time-curve shifted significantly downwards at all time periods following protocols A and C ($p<.05$) (main effect, Figure C5). At time periods 200-300ms, 300-400ms, and 400-500ms the decrease in absolute force produced was significantly greater after protocol C (measurement time x protocol interaction effect, $p<.05$) (Figure C5).

IEMG

A significant main (measurement time) effect was observed when comparing the pre- to post-protocol changes in IEMG mean amplitude between protocol A and C, but there was no interaction effect (Figure C6). There were no significant differences in mean frequency of the power spectrum pre- to post-protocol in either of the fatigue protocols (Figure C7).

Blood Lactate

Blood lactate increased from 1.3 ± 0.35 mmol/l to 3.4 ± 1.2 mmol/l during protocol A and from 1.6 ± 0.48 mmol/l to 4.1 ± 1.3 mmol/l during protocol C (Figure C8). Although both increases were significant (main effect, $p<.05$) the differences between the two protocols was not ($p=0.057$)

Table C1. Characteristics of the subjects who completed fatigue protocols A and C (N = 14).

Subject Characteristics	Mean (SD)
Body mass (kg)	87.4 (12.1)
Height (cm)	181.1 (4.3)
Age (yrs)	25.2 (3.7)

Table C2. Mean (and SD) total repetitions, time under tension and repetitions per each of the 3 sets completed during fatigue protocols A and C (N = 14).

Training Variable	Protocol A	Protocol C
Total repetitions (all sets)	22.3 (4.3)	29.9 (3.7)*
Total training volume	17.3 (3.5)	21.6 (2.7)*
Training intensity (first set)	77.3 (2.8)	77.5 (2.5)
Training intensity (second set)	77.3 (2.8)	72.9% (3.2)
Training intensity (third set)	77.3 (2.8)	68.0 (3.3)
Repetitions in first set	9.9 (2.0)	10.6 (1.6)
Repetitions in second set	7.0 (1.6)	9.4 (1.5)
Repetitions in third set	5.4 (1.2)	9.9 (1.4)
Time under tension (s)	89.9 (15.6)	109.7 (12.0)*

* represents significant difference from protocol A ($p<.05$)

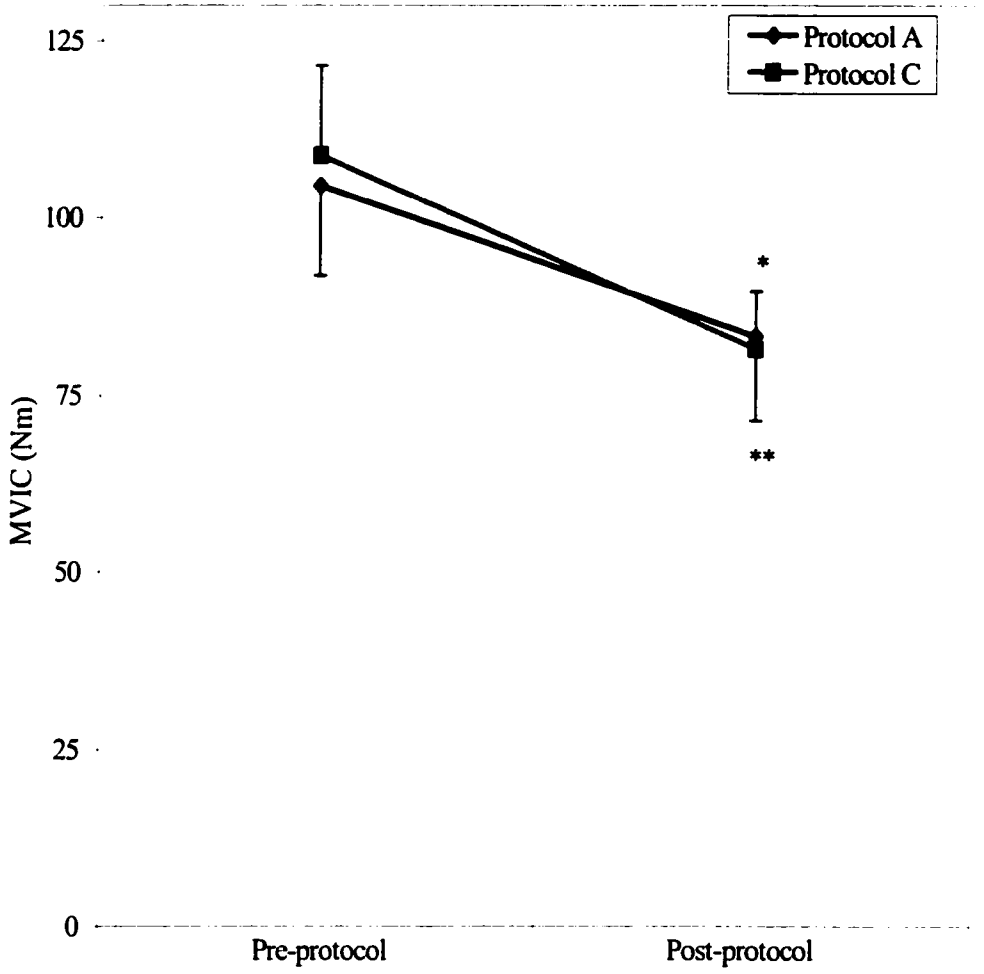


Figure C4. Mean (SD) MVIC of the right forearm flexors before and immediately after performing 3 sets of approximately 10 repetitions of single-arm flexion (* = significant difference from corresponding pre-protocol value; ** = significant difference from corresponding pre-protocol value as well as an interaction effect. $p<.05$).

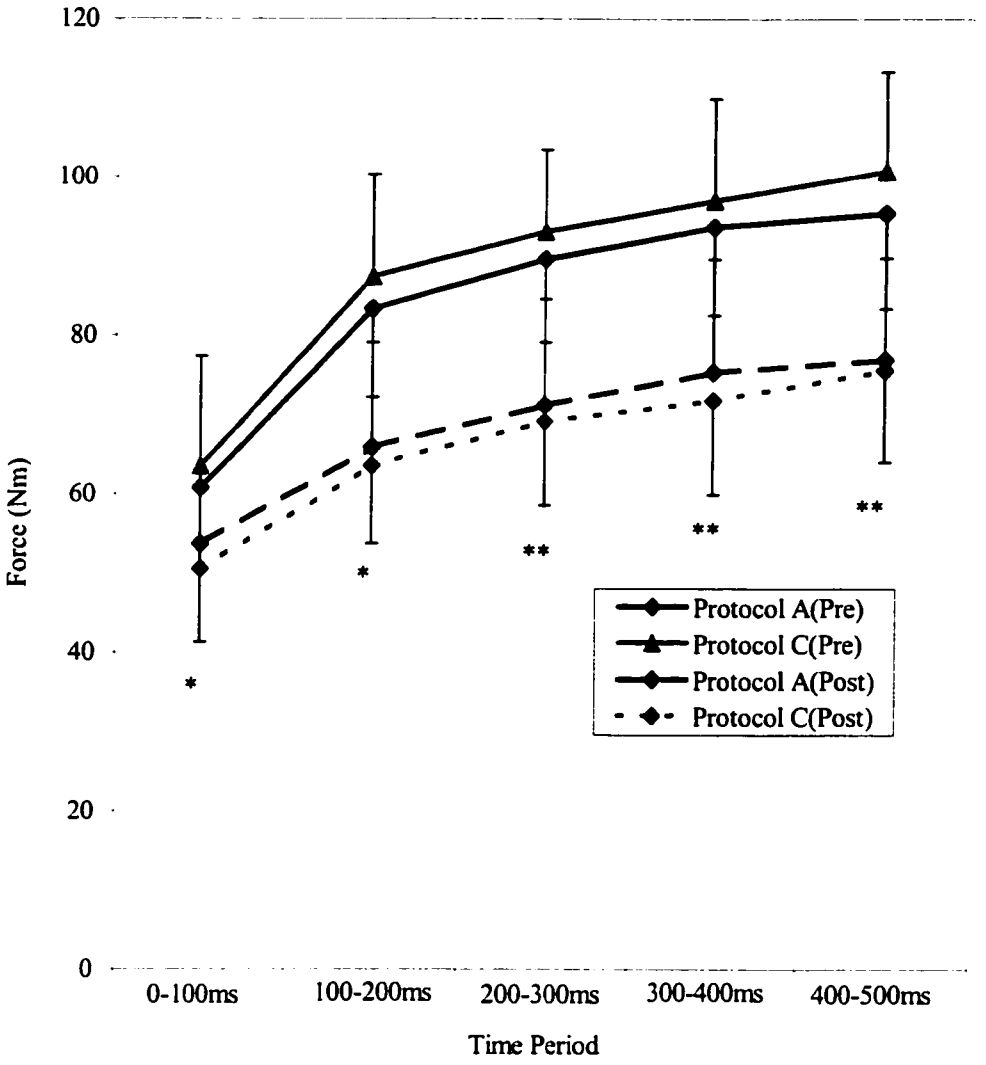


Figure C5. Mean (SD) force-time curves during maximal voluntary isometric contractions performed before and after protocol A and Protocol C (* represents significant difference from corresponding pre-protocol value during the same time frame; ** represents significantly larger decrease following protocol C. $p<.05$).

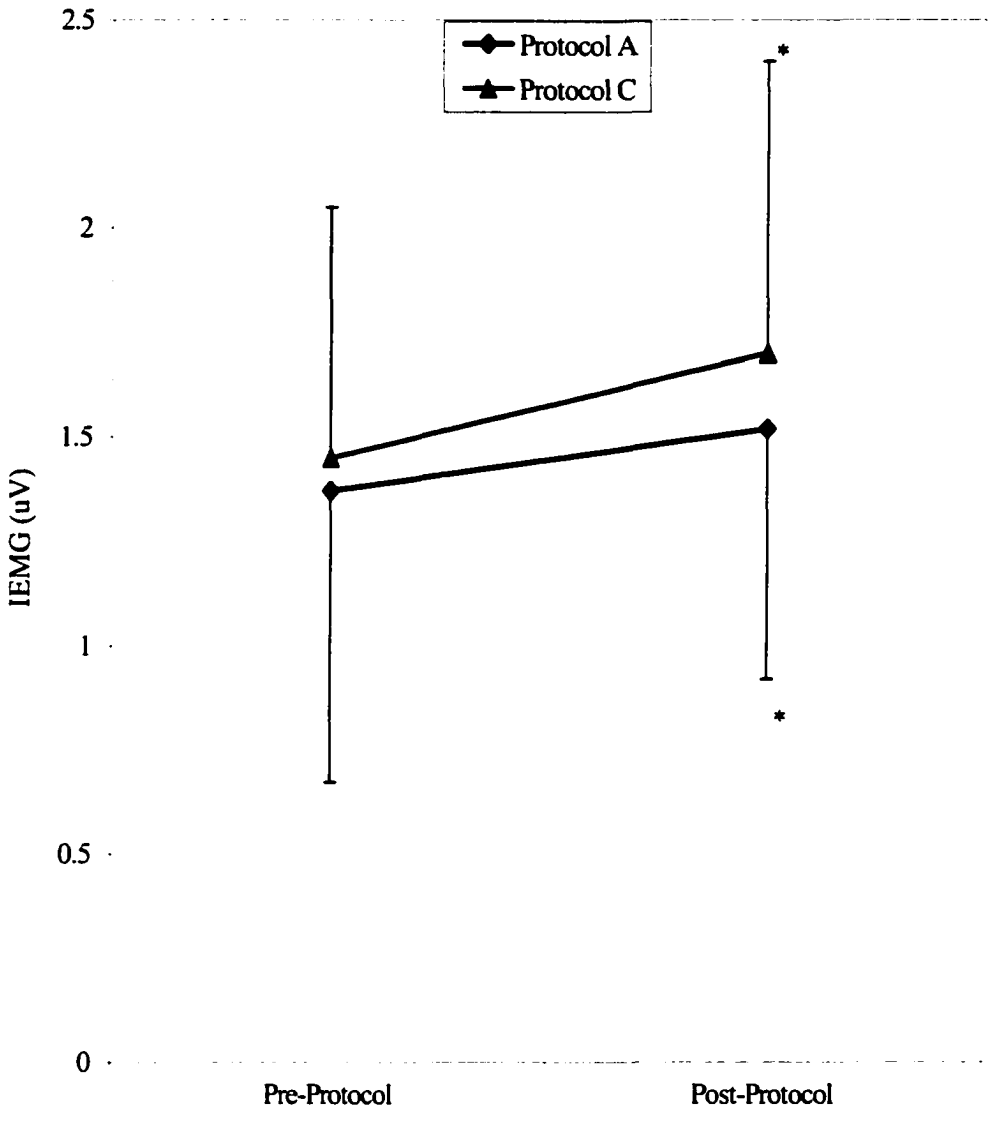


Figure C6. Mean (SD) values for the Biceps Brachii mean IEMG activity recorded during maximal voluntary isometric contractions performed prior to and upon completion of fatigue protocol A and Fatigue protocol C (* represents significant time effect, $p<.05$).

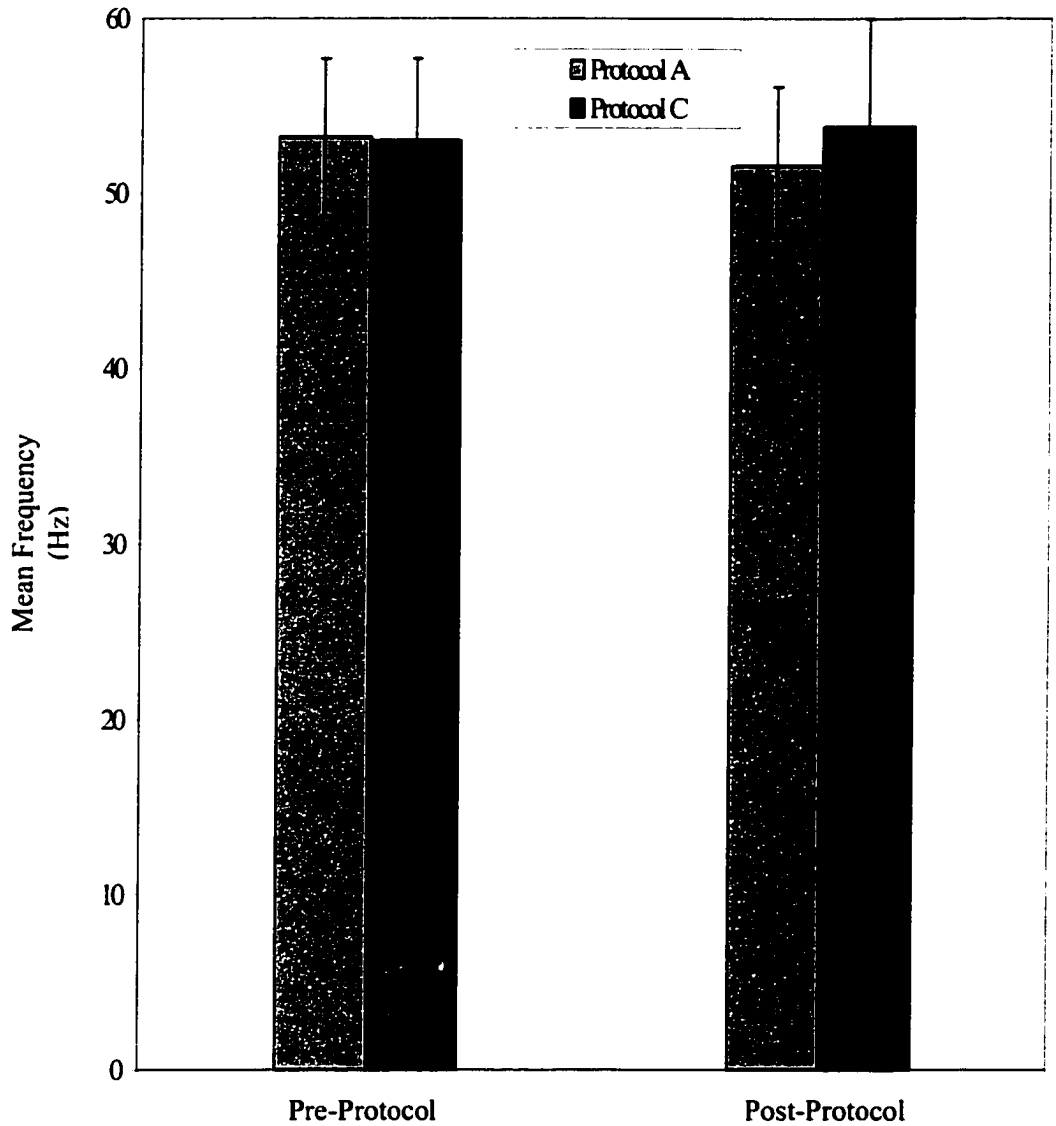


Figure C7. Mean (SD) values for the Biceps Brachii mean frequency of the power spectrum of the IEMG activity recorded during maximal voluntary isometric contractions performed prior to and upon completion of fatigue protocol A and Fatigue protocol C.

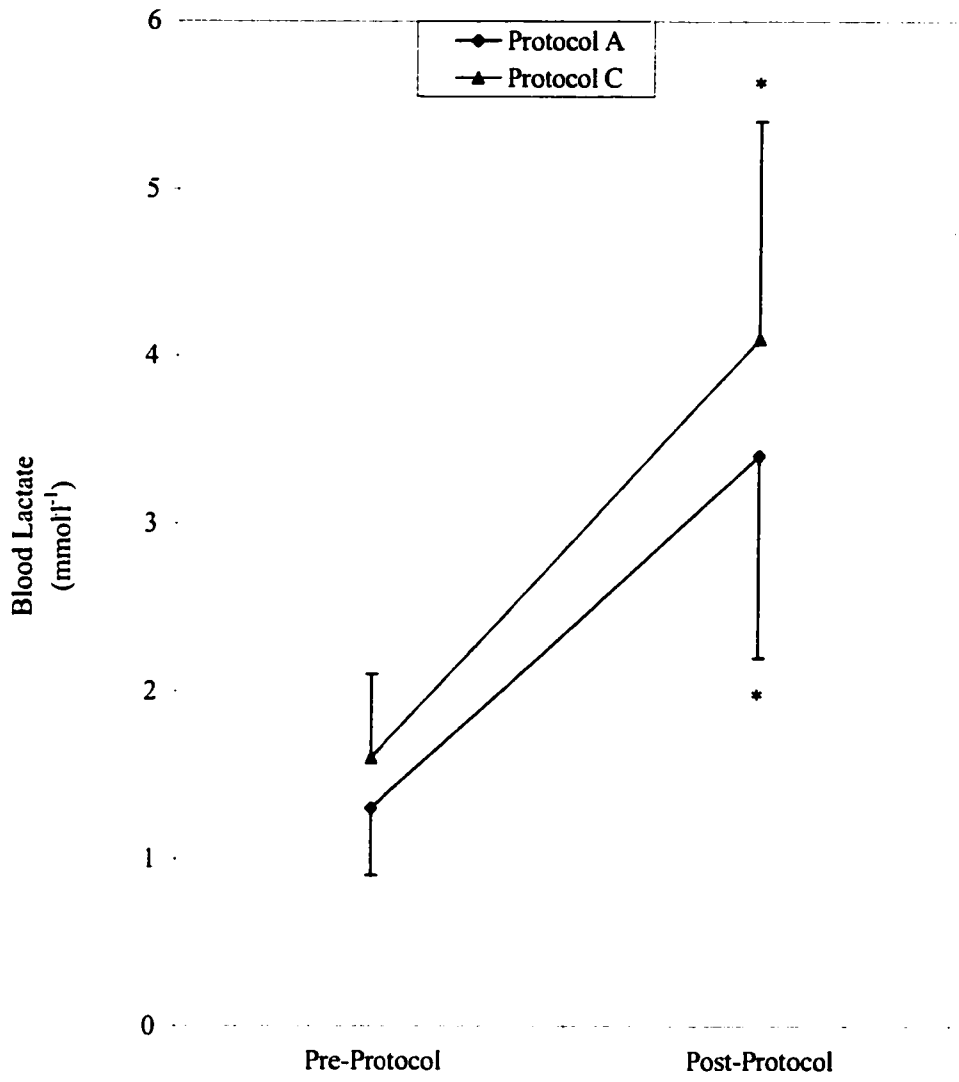


Figure C8. Mean (SD) blood lactate values before and five minutes following the performance of fatigue protocols A and C (* represents significant difference from respective pre-protocol value. $p<.05$).

Discussion

The purpose of this investigation was to compare and examine the acute neuromuscular fatigue produced during 3 sets of a constant load protocol and a 10-RM (adjusted load) protocol. The primary findings of this study were that protocol C elicited significantly greater reductions in MVIC and explosive force than protocol A. Despite these findings, there were no significant differences between the two protocols in the changes in blood lactate and IEMG.

Strength Performance in Protocol

During protocol A, in which a constant training load was used during all three sets of the single-arm elbow flexion exercise, a gradual reduction in the number of repetitions performed per set was observed over the three sets whereas the number of repetitions performed per set in protocol C remained constant (Table C2). However, in order to maintain the number of repetitions performed per set at approximately 10, a reduction in relative training intensity by about 5% was necessary prior to performing both the second and third sets during protocol C (Table C2). Despite the progressive reduction in relative training intensity the total training volume accomplished (reps x sets x relative training intensity) was greater during protocol C (Table C2).

The gradual reduction in the number of repetitions performed to failure over consecutive sets during protocol A is indicative of accumulative neuromuscular fatigue (MacDougall et al., 1999). Over 3 sets of single-arm elbow flexion using 3-minute rest intervals and a constant load of approximately 80% 1-RM, MacDougall et al. (1999) observed a decrease in the mean number of repetitions performed to failure per set from 11.7 to 7.2. This absolute reduction in the number of repetitions performed per set is

very similar to that observed during protocol A in the present study (Table C2). Alternatively, the reduction in training load prior to performing sets two and three accompanied with the maintenance in the number of repetitions performed was also indicative of fatigue.

MacDougall et al. (1999), performed muscle biopsies to identify the mechanism(s) underlying the decrease in strength performance. They concluded that in heavy resistance training protocols using 3-minute rest intervals the progressive reduction in the number of repetitions completed per set was primarily the result of an increase in muscle H^+ concentration with a minor contribution made by a depletion in PCr (MacDougall et al., 1999). MacDougall et al. (1999) suggested that 3 minutes of rest, although enough time to restore PCr levels, was not enough time to completely remove muscle lactate (H^+). Consequently, each subsequent set would have begun with muscle lactate and H^+ concentrations greater than resting levels as well as above that of preceding sets and therefore accounting for the drop-off in repetitions performed. Because of the similarities between the protocol used by MacDougall et al. (1999) and Protocol A of the present study, it seems plausible that similar mechanisms were responsible for the decrement in forearm flexor performance over the three sets.

An increase in muscle H^+ concentration may inhibit the binding of Ca^{2+} to troponin C, consequently limiting the number of cross-bridges formed and compromising the magnitude of force produced (McLester Jr., 1997). An accumulation of H^+ within the contracting muscle may also inhibit glycolytic enzymes causing a slowing in the rate of glycolysis as well as delay the rate of PCr re-phosphorylation, both of which would limit the supply of ATP (MacDougall et al., 1999; Robergs et al., 1991; Tesch et al., 1986;

Wenger & Reed, 1976). Because the ability to repeat high force contractions is dependent on the supply of ATP (MacDougall et al., 1999; Weiss, 1991), a reduction in ATP supply would limit the capacity of the muscle to exert force, possibly accounting for the progressive decrease in the number of repetitions performed per set with a constant load (as observed in protocol A). This decrement in the number of repetitions performed per set was not evident during protocol C. In addition to reducing the force requirements of the muscle, the five-percent reduction in training load would have also decreased ATP demand. Some support for this was provided by Robergs et al. (1991) in which a resistance training protocol of a lower intensity produced a slower rate of glycogen utilization. Therefore, in the present study the progressive reduction in training intensity may have reduced the rate at which ATP was required. Even if glycolysis was slowed because of an increase in H^+ concentration, the decreased ATP demands would have been satisfied and permitted the completion of the similar amount of repetitions per set. Additionally, if the rate of anaerobic glycolysis was progressively reduced during each set during protocol C, this would translate into a slower rate of lactate production and possibly account for the constant number of repetitions performed per set during protocol C.

MVIC

Following the performance of protocols A and C, MVIC was reduced by approximately 20% and 25%, respectively, with the magnitude of decrement in MVIC experienced after protocol C being significantly greater than that induced following protocol A (Figure C4). It appears that the significantly greater reductions in MVIC

following protocol C may be a result of the greater overall training volume performed despite the gradual decrease in relative training intensity (Table C2).

When resistance training is performed to failure, the degree of fatigue experienced by the muscle is postulated to be related to the intensity of the training load (MacDougall et al., 1999). For example, muscle failure attained with a load of 80% 1-RM, would be accompanied by a 20% decline in maximal force generating capacity. The results of the present study support this notion because muscle failure in the third set of protocol C (68% 1-RM) occurred with a lower relative training intensity than in protocol A (77% 1-RM), and protocol C elicited a greater reduction in MVIC. However, this inference assumes that the mechanisms underlying a reduction in dynamic performance are similar to those impairing maximal isometric force capabilities.

Associated with the reductions in MVIC of both groups was a significant time (but not an interaction) effect for the increases in IEMG amplitude observed following both fatigue protocols. The results from this study support those observed by Bigland-Ritchie, Furbush, & Woods (1986), in which IEMG of the quadriceps increased while maximal isometric force decreased during maximal voluntary contractions that were performed every minute during a sub-maximal (50% of maximum) and intermittent (6 seconds on, 4 seconds off) fatigue protocol. With these findings, the researchers concluded that maximal isometric force capabilities were not compromised due to inadequate muscle excitation or action potential transmission. The increases from pre- to post-protocol in mean IEMG amplitude observed in the present study may suggest that neither protocol was capable of producing neural fatigue (Figure C6). Further, the absence of any change pre- to post-protocol in mean frequency of the power spectrum of

the IEMG signal following either protocol of the present study also suggests that action potential conduction velocity remained unchanged (Figure C7) (Linnamo et al., 2000).

The decrements in MVIC along with the opposing increases in average IEMG amplitude observed in the present study support findings of previous studies investigating the acute neuromuscular fatigue produced from resistance training (Hakkinen et al., 1988; Kauhanen et al., 1989; Linnamo, Newton, Hakkinen, Komi, Davie et al., 2000). In these studies, MVIC was significantly reduced following each resistance training protocol, whereas the IEMG signal remained unchanged. These studies, accompanied with the results of the present study, suggest that resistance training-induced reductions in maximal voluntary isometric force producing abilities were not limited by changes in neural drive. In contrast, significant reductions in IEMG have been observed in parallel with decreases in MVIC following the performance of various resistance training protocols (Behm et al., in press; Hakkinen, 1994; Linnamo, Hakkinen, & Komi, 1998). The results from these studies indicate that acute reductions in neural drive are partly responsible for the fatigue-induced decrements in MVIC. However, although IEMG and MVIC were reduced post-protocol, the time course for recovery of IEMG and MVIC differed (Behm et al., in press, Linnamo et al., 1998). Following a single set of an elbow flexion exercise using loads of 5-, 10- and 20-RM, IEMG activity of the biceps brachii returned to pre-exercise values following 3 minutes of recovery whereas MVIC remained significantly reduced (Behm et al., in press). Similarly, Linnamo et al. (1998) observed that maximal isometric force remained impaired for 2 days following the performance of 5 sets of leg extensions using a 10-RM load while IEMG had recovered within one hour post-protocol. These findings support the observations of the present study, which

suggest the acute decrements in MVIC induced by resistance training protocols are not the result of reductions in neural drive. Therefore, it would appear that peripheral factors are primarily responsible for the acute reductions in MVIC.

The progressive reduction in relative training intensity during protocol C permitted the performance of a greater total number of repetitions, higher training volume, and longer time under tension (duration of activity) than protocol A (Table 2). Tesch et al. (1986) postulated that the duration of a resistance training protocol influences the fatigue response. Behm et al. (in press) demonstrated significantly greater reductions in evoked twitch torque subsequent to a moderate-intensity/high-volume/long duration resistance (20-RM) training protocol than a high-intensity/low-volume/shorter duration (5-RM) protocol. Because evoked force is a measure of peripheral integrity, it was concluded that peripheral impairments in excitation-contraction (E-C) coupling were responsible for the greater reductions in evoked force following the 20-RM protocol. Furthermore, it appears that peripheral fatigue may be related to the amount of training volume and the duration of muscle activity performed during a resistance training protocol (Behm et al., in press). However, in the study conducted by Behm et al. (in press) there were no significant differences between the protocols with regards to the magnitude of reduction in maximal voluntary force generating capacities. Behm et al. (in press) suggested that the higher discharge rate during an MVIC (in comparison to the evoked force) restored the greater loss of force experienced during the 20RM protocol. Therefore, although impairments in excitation-contraction coupling may have contributed to acute reductions in MVIC it is uncertain if this mechanism contributed to the larger decrements in MVIC observed following the performance of protocol C.

Disruptions in E-C coupling may occur through one or a combination of a number of mechanisms that include a) reduced neural drive, b) reduced excitability of the muscle membrane (including T-tubules) (Fitts & Balog, 1996; Wenger & Reed, 1976), c) reduced Ca^{2+} release from the sarcoplasmic reticulum (SR) (Westerblad, Allen, Bruton, Andrade, & Lannergren, 1998), and d) H^+ competing with Ca^{2+} for the binding site on troponin C (McLester Jr, 1997).

Blood lactate was measured in the present study to obtain an indication of peripheral metabolic disturbances, especially muscle lactate and H^+ concentration (Tesch et al., 1986). The greater increases in blood lactate in response to protocol C than in response to protocol A approached significance ($p=.057$). These results are in accordance with those observed by Keogh, Wilson, & Weatherby (1999), in which greater increases (although non-significant) in blood lactate were elicited in response to a resistance training protocol that reduced the load following each repetition (rather than after each set) than compared to a constant load protocol. Further, MacDougall et al. (1999) demonstrated greater increases in blood lactate following three sets of single-arm elbow flexion compared to one set but there was no indication to the level of significance of the difference. Since blood lactate is not only a reflection of the production and accumulation of muscle lactate but also its distribution and removal, it is possible that actual increases in muscle lactate following protocol C were greater than the measured changes in blood lactate. Therefore, muscle lactate and associated decreases in muscle pH may account for the significantly greater reductions in MVIC following protocol C. However, Robergs et al. (1991) observed that the amount of glycogen depleted during resistance training is positively related with the training volume that is accomplished

whereas the rate of glycogen depletion is associated with intensity. Because of the progressive reductions in relative training intensity during protocol C, the rate of glycogen utilization may have been slowed which may have resulted in the production of less lactate and account for the results observed in the present study.

Significant negative correlations between resistance training-induced increases in blood lactate and decreases in maximal isometric force have been observed and indicate that intra-muscular accumulations of H^+ are primarily responsible for impaired force development (Hakkinen, 1994; Linnamo et al., 1998). The accumulation of H^+ within the muscle may impair muscle functioning at a series of sites within the contractile process including a) inhibition of glycolytic enzymes, therefore limiting the supply of ATP (Tesch et al., 1986), b) slowing restoration of PCr (Tesch et al., 1986), c) inhibition of Ca^{2+} binding to troponin C resulting in fewer active cross-bridges, and d) inhibition of the SR Ca^{2+} ATPase resulting in a depletion of releasable Ca^{2+} (Green, 1990; Fitts & Balog, 1996).

Significant reductions in maximum isokinetic force of the quadriceps were demonstrated 5 minutes following multiple sets of high- and moderate-intensity resistance training (Raastad & Hallen, 2000). Because recovery from metabolic disturbances may have been complete or near complete in the 5 minutes separating the final repetition and maximal isokinetic force test, Raastad & Hallen (2000) suggested that a reduction in Ca^{2+} release from the SR was responsible for the decrements in maximal force observed. In the present study, it would seem that this proposed mechanism along with force-reducing metabolic disturbances may account for the post-protocol reductions in MVIC because post-protocol MVIC was assessed immediately (within 15 seconds)

after the final repetition. However, it is unknown if Ca^{2+} kinetics are influenced by performing a greater amount of training volume.

Additionally, an accumulation of P_i (as a result of the breakdown of ATP and PCr) has been suggested to be responsible for a reduction in maximal force generating capacity (McLester Jr, 1997; Westerblad et al., 1998). In vitro studies have demonstrated that the restoration of intra-muscular P_i levels to resting values resulted in the recovery of maximum evoked force (Westerblad et al., 1998). It is unknown if accumulations of P_i impair in vivo maximal voluntary force capabilities. However, increases in the P_i/PCr ratio following dynamic and isometric fatiguing protocols may indicate that P_i does accumulate within the muscle during intense activity. The role Pi assumes in resistance training-induced fatigue remains uncertain because the degree of Pi accumulation following a bout of heavy resistance training is unknown.

Explosive Force

The two fatiguing protocols of the present study produced significant downward shifts in the force-time curves with the shift produced by protocol C significantly greater over the final 300ms of the 500ms period (Figure C5). These results indicate that protocol C elicited significantly greater reductions in explosive force production. Explosive force is dependent on the rate of cross-bridge cycling, which itself appears to be influenced by the rate of ATP hydrolysis (Fitts & Balog, 1996). It has been postulated that the dissociation of ADP during the actomyosin formation may be the limiting factor associated with ATP hydrolysis and rate of cross-bridge cycling (Fitts & Balog, 1996; McLester, 1997). An inhibition of myofibrillar ATPase may ultimately limit the release

of ADP from the myosin head that is necessary for cross-bridge detachment and re-attachment (McLester Jr., 1997). A proposed factor that may inhibit myofibrillar ATPase and compromise explosive force generation is the accumulation of H^+ (Westerblad et al., 1998).

It is unknown if any neural mechanisms contributed to the observed shifts in the force-time curves because the IEMG signal was not processed during this time.

Conclusion

Maintaining a constant load during resistance training in which sets are performed to failure results in a progressive reduction in the number of repetitions performed per set. A minor reduction to the relative training load prior to performing upcoming sets proved to be effective at maintaining the number of repetitions per set. By maintaining a constant number of repetitions completed per set, the training volume accomplished during a protocol C was greater than that achieved when relative training load remained unchanged. Significantly greater decreases in MVIC and explosive force were observed following protocol C than protocol A, suggesting that the amount of volume and training duration are associated with resistance training-induced impairments in maximum force generating capabilities. It appears that peripheral, rather than neural, mechanisms accounted for the greater impairments in muscle performance following protocol C.

Section D: Results and discussion for the resistance training-induced acute neuromuscular fatigue before and after 8 weeks of resistance training that was specific to fatigue protocol.

Results

Five of the subjects who participated in program A completed both the pre- and post-training fatigue protocols involving protocol A. Four subjects adhering to program B and four in program C completed the corresponding fatigue protocols pre- and post-training.

Strength Performance

The average amount of training volume performed by the subjects of program A during fatigue protocol A prior to training was 19.2 units. Following training, subjects performed 17.6 units of training volume (Table D2). There was no statistical difference between these two values. The mean relative training intensity used during protocol A was 73.8% 1-RM and 76.9% 1-RM for the pre- and post-training tests, respectively, and there was no statistical difference between these intensities (Table D2).

Subjects participating in program B performed 18.0 units of training volume during the pre-training protocol B and 16.5 units of volume following the training program (Table D2). There was no statistical difference between these two values. Similar relative training intensities (approximately 77% 1-RM) were used on the two different occasions that protocol B was performed (Table D2).

Prior to training, subjects belonging to program C accomplished 20.6 units of training volume during the performance of protocol C. Following the training program, subjects performed 19.3 units of training volume (Table D2). There was no statistical difference between these two values. The relative training intensity used by subjects of program C was approximately 77% 1-RM during the pre- and post-training fatigue protocols. Relative intensity from the first to the third set was reduced by 10.8% during

the pre-training protocol C. Following training this reduction was 8.8% and the difference with the reduction during pre-training was not significant.

MVIC and IEMG

There were no significant differences in the pre-protocol MVIC and IEMG value pre- and post-training. Subjects participating in program A experienced an 18.5% and 16.7% reduction in MVIC during fatigue protocol A before and after training, respectively (Figure D4). There was no difference between the reductions in MVIC pre- and post-training. Pre- and post-training reductions in MVIC experienced by subjects in program B were similar (approximately 21.5%) (Figure D4). A 22.9% reduction in MVIC was observed pre-training during protocol C and a similar 24.6% reduction occurred following training (Figure D4). IEMG remained unchanged during protocol A, B and C prior to and upon completion of Program A, B, and C, respectively (Figure D5).

Table D1. Mean (SD) height, body mass and age of the participants of the three training groups.

Training Group	Height (cm) $\bar{x}$ (SD)	Body Mass (kg) $\bar{x}$ (SD)	Age (years) $\bar{x}$ (SD)
A (n=5)	180.5 (5.6)	89.7 (18.7)	25.0 (4.9)
B (n=4)	178.1 (3.4)	85.3 (3.6)	26.3 (3.4)
C (n=4)	182.6 (4.0)	87.9 (8.7)	24.8 (3.1)

Table D2. Relative training intensity (mean & SD) used and training volume performed by subjects of the three training programs during the corresponding fatigue protocols (pre- and post-training).

Variable	Program A (n=5)		Program B (n=4)		Program C (n=4)	
	Pre-T	Post-T	Pre-T	Post-T	Pre-T	Post-T
Relative Training Intensity used during first set (%1-RM)	73.8	76.9	77.1	77.4	77.1	77.8
Training Volume (units)	19.2	17.6	18.0	16.5	20.6	19.3

Pre-T = Pre-Training
Post-T = Post-Training

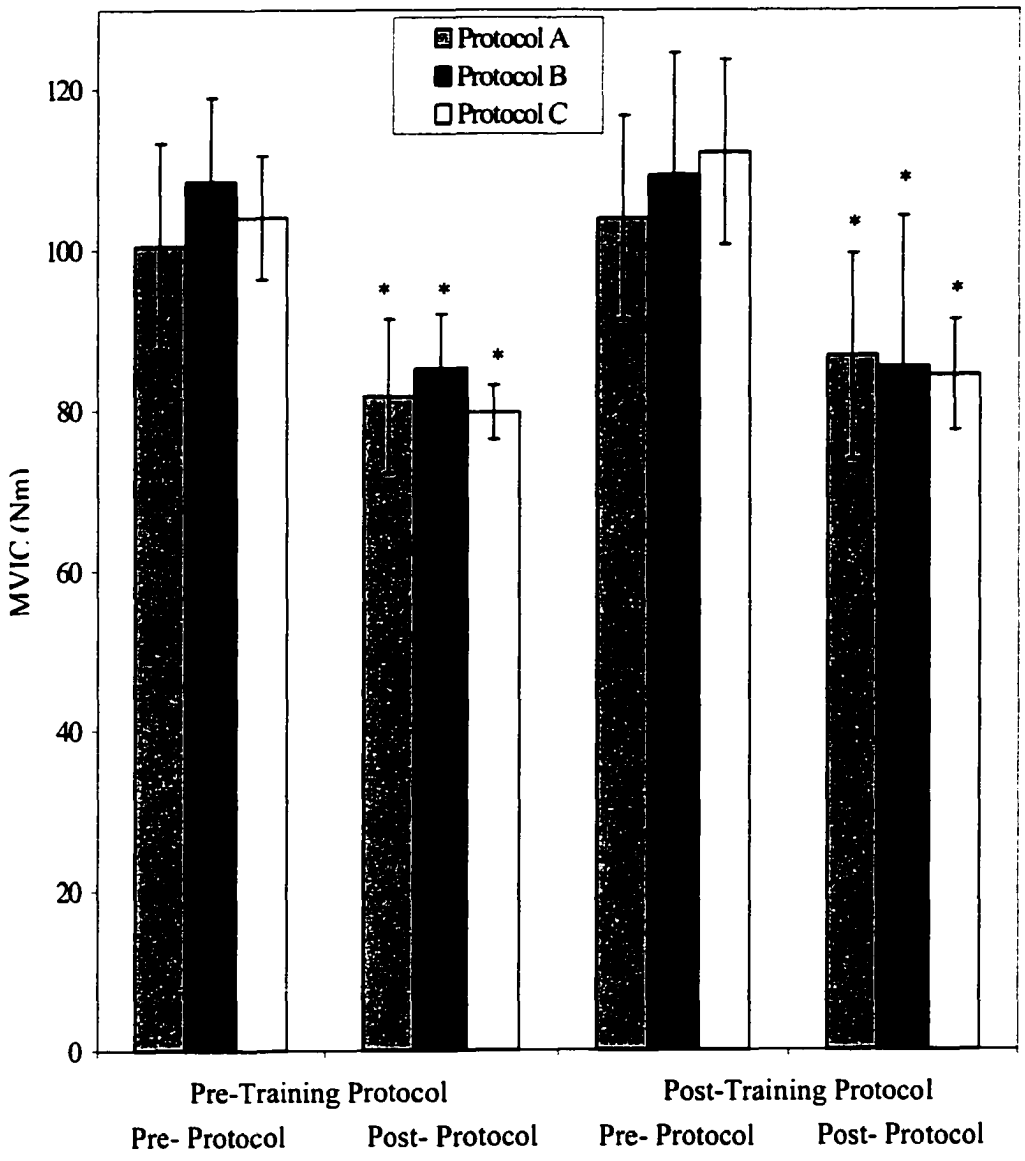


Figure D4. Mean (SD) MVIC of the forearm flexors during the corresponding pre-and post-training fatigue protocols for subjects who participated in program A (N = 5), program B (N = 4), and program C (N = 4) (* represents significant difference from corresponding pre-protocol value).

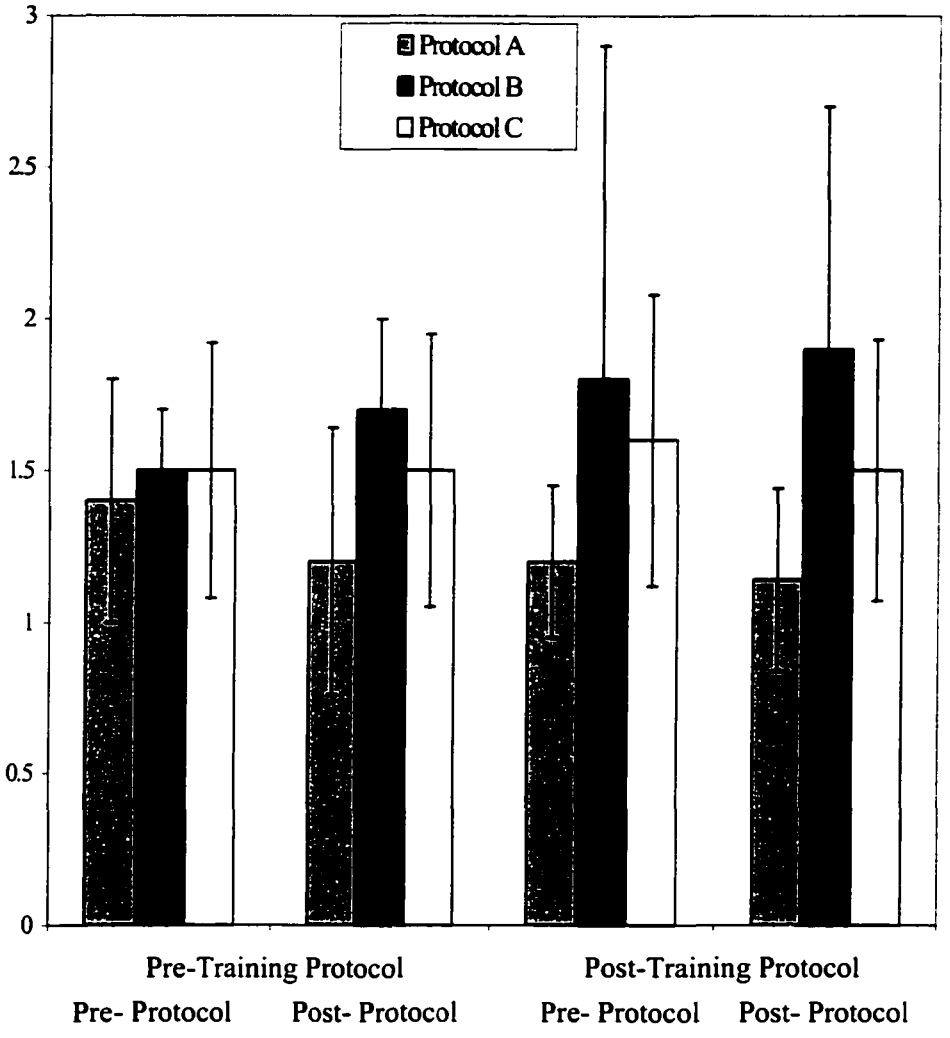


Figure D5.Mean (SD) IEMG of the biceps brachii during the corresponding pre-and post-training fatigue protocols for subjects who participated in program A (N = 5), program B (N = 4), and program C (N = 4).

Discussion

Previous research has suggested that the acute fatigue experienced during resistance training is important in the development of muscle strength and hypertrophy (Rooney et al., 1994; Schott et al., 1995). In both of these studies, the acute fatigue produced in response to the training programs used was only assessed prior to training and therefore, it remains uncertain if the acute fatigue response changed as training progressed (Rooney et al., 1994; Schott et al., 1995).

Pullinen, Huttenen, & Komi (2000) have proposed that prolonged participation in a resistance training program reduces the acute response to resistance training. Alternatively, resistance training-induced neuromuscular adaptations have been suggested to impair submaximal muscle performance because of increased fatigability (Hickson, Hidaka, & Foster, 1994). Thus, if the acute resistance training-induced fatigue changes over the course of a training program, the effectiveness of a program in developing strength may be compromised. Therefore, it was the purpose of this component of the study to determine if the resistance training-induced acute fatigue was affected by 8 weeks of resistance training. Because there was no difference in the pre- compared to post-training fatigue response experienced between subjects of the different training programs the results will not be discussed independently.

Strength Performance During Fatiguing Protocols

There was no difference between the pre-training and post-training fatigue protocols with reference to the amount of training volume that was performed (Table D2). Previous research comparing the acute muscular performance of subjects using the same relative (% 1-RM) load before and after participation in strength training programs

is limited and the research that has been conducted has only concentrated on the performance during one set rather than the performance over multiple sets. Of the studies investigating changes in acute muscle fatigability before and after participation in a resistance training program, many have compared the number of repetitions completed when using the same absolute testing load (pre- and post-training) (Hickson et al., 1994; Medbo et al., 2001). In previously untrained male subjects 16 weeks of training produced an increase in the mean number of repetitions performed at 80% pre-training 1-RM from 8 to 15 and from 8 to 21 during bench press and squat exercises, respectively (Hickson et al., 1994). In well-trained subjects a 29% increase in the number of repetitions performed during one set using 70% of the pre-training 1-RM was observed following 3 months of intense resistance training (Medbo et al., 2001). Similarly, acute increases in blood lactate during a sub-maximal fatigue test that controlled for the number of repetitions and absolute load was significantly lower following eight weeks of resistance training using loads between 5- and 10-RM (Pierce, Rozenek, & Stone, 1993). It was assumed that a post-training increase in muscle performance reflects an increase in muscle endurance and a reduced fatigability (Medbo et al., 2001). However, these conclusions are questionable because the post-training load used in the above studies was less of a neuromuscular demand than the pre-training protocol load due to an increase in maximal strength that also resulted from the training. Therefore, the improvements in performance following training may reflect the increase in strength rather than reduced fatigability.

An alternate method for assessing resistance training-induced changes in muscle fatigability is to use the same relative load for both the pre- and post-training fatigue

protocols (Hickson et al., 1994). In the present study, the load utilized during both the pre- and post-training fatigue protocols, as well as during the eight weeks of resistance training was equal to the resistance at which 10 repetitions could be completed on the first set. Following these guidelines, there was no significant differences in the percentage of 1-RM used during the pre- and post-training tests for any of the training programs (Table D2). The results of the present study suggest that there was no difference between the pre- and post-training relative fatigue produced during the performance of each protocol. Similar to the present study, Hickson et al. (1994) observed no change in the number of repetitions performed during bench press and leg press using 40%, 60% and 80% 1-RM before and after 16 weeks of 5-RM resistance training. However, although the relative intensity remained constant, the absolute load utilized for the post-training test increased. Despite the increase in absolute load for the post-training test, the training volume as defined in this study was equal to that performed pre-training.

A greater post-training absolute load has been suggested to have higher energy demands and result in a faster development of fatigue than the lower pre-training absolute loads (Hickson et al., 1994). The similarities in the training volume performed between the pre- and post-training protocols during the present study would appear to indicate that the magnitude of acute fatigue produced was similar between the testing occasions. If the use of a greater absolute load but similar relative load imposed a greater demand on the muscle, the performance of an equal training volume while using a greater absolute load during the post-training fatigue protocols may indicate that training adaptations took place.

Prior to the 8 weeks of resistance training, subjects in program C reduced the relative training load during the three sets of fatigue protocol C by approximately 11%. Following the 8-week training period, this reduction was only 9% and the difference with the pre-training drop was approaching significance (p=.086) (Table D2). The post-training decrease in the amount in which the training load had to be reduced over three sets in order to perform 10 repetitions per set also indicates that training adaptations may have occurred. These results are in agreement with previous observations, in which a decrease in the amount the training load had to be reduced over 5 sets of 12-RM was observed over a short-term training study (Pullinen et al., 2000).

MVIC and IEMG

The pre-protocol to post-protocol reductions in MVIC and concomitant responses in mean IEMG amplitude were not different prior to and after the 8 weeks of resistance training in any of the training programs (Figure D4 and D5, respectively). These results support those observed during the performance of the fatigue protocols and appear to indicate that the magnitude of fatigue experienced during the pre- and post-training protocols was similar. It is difficult to determine if these results are supported by previous research as few studies investigated how training influences the acute fatigue response (MVIC and IEMG) and the studies that have been performed were of a short duration. In untrained subjects the magnitude of the acute reduction in MVIC of the quadriceps following 6 sets of 12 bilateral leg extensions was similar during all five of five training sessions completed over a 2-week duration (Pullinen et al., 2000). Because of the untrained status of the subjects and the short duration used in the study by Pullinen et al. (2000), comparisons with the present study are difficult.

Mechanisms Responsible

The mechanism(s) thought to be responsible for a post-training increase in the number of repetitions completed using an absolute training load may be similar to those mechanisms that would account for a post-training performance of an equal training volume despite an increase in absolute training volume. Following three months of intense resistance training of the lower body, a significant increase in the number of repetitions performed with 70% of the pre-training 1-RM was demonstrated in well-trained subjects (Medbo et al., 2001). The increase in the number of repetitions performed was correlated to an increase in Na^+-K^+ ATPase concentration (Medbo et al., 2001). An increase in Na^+-K^+ ATPase concentration was suggested to increase the effectiveness of restoring the K^+ gradient during intense muscle activation (a possible source of fatigue), allowing for enhanced muscle performance following the resistance training program.

A 5-month resistance training program, using previously untrained subjects performing 8-10 repetitions produced an increase in the intra-muscular concentrations of ATP, CP and glycogen (MacDougall, Ward, Sale, & Sutton, 1977). It was proposed that because the energy utilized during resistance training is supplied by anaerobic mechanisms, the increase in these energy stores may be responsible for an increase in the number of repetitions that can be performed during a set of exercise following a training program. However, it is difficult to relate these chronic increases in substrate levels to trained individuals (as used in the present study) because the subjects had no previous resistance training experience at the onset of this study.

Another mechanism that may influence the fatigability of a muscle is the capillary supply. A greater capillary supply to the working muscle would result in a more effective metabolite clearance and nutrient supply (Kadi, Eriksson, Holmner, Butler, Browne, & Thornell, 1999). Following 12 weeks of resistance training of the forearm flexors using 10-RM loads it was demonstrated that the number of capillaries per fiber area remained constant in previously trained subjects (McCall et al., 1996). Similar observations were observed in untrained subjects following 12 weeks of maximal isokinetic training (Hather, Tesch, Buchanan, & Dudley, 1991; Tesch, Thorsson, & Colliander, 1990).

Behm et al. (in press) observed a significant increase in antagonist activity during an MVIC performed within one minute of completing one set of single-arm forearm flexion. Antagonist coactivation decreases the strength that can be generated by the agonist muscle(s) (Gabriel, Basford, & An, 2001). Eight weeks of isometric training of the quadriceps of previously untrained subjects reduced hamstring coactivation by 20% during knee extension. If a training-induced reduction in antagonist activation occurred during the present study this may balance the greater neuromuscular demands of a greater absolute post-training test load and account for the similar amount of fatigue measured pre- and post-training. However, it is uncertain if dynamic resistance training produces a reduction in antagonist coactivation of already trained subjects.

Conclusion

The magnitude of acute fatigue was similar before and following the completion of eight weeks of resistance training regardless of whether training consisted of using a constant load with 3-minute rest intervals, a constant load with 1-minute rest intervals, or an adjusting (10-RM) load with 3-minute rest intervals. The performance of a similar

amount of training volume over three sets with a greater absolute training load than that used during the pre-training test indicated that training adaptations may have occurred to augment muscle performance.

References

Abdessemed, D., Duche, P., Hautier, C., Poumarat, C., & Bedu, M. (1999). Effect of recovery duration on muscular power and blood lactate during the bench press exercise. **International Journal of Sports Medicine, 20**, 368-373.

Alway, S. E., Grumbt, W. H., Stray-Gunderson, J., & Gonyea, W. J. (1992). Effects of resistance training on elbow flexors of highly competitive bodybuilders. **Journal of Applied Physiology, 72(4),** 1512-1521.

Alway, S. E., Grumbt, W. H., Gonyea, W. J. & Stray-Gunderson, J. (1989). Contrasts in muscle and myofibers of elite male and female bodybuilders. **Journal of Applied Physiology, 67(1),** 24-31.

Ballantyne, C. S., & Sale, D. G. (1999). Effects of different rest intervals on fatigue in resistance training. **Journal of Strength and Conditioning Research, 13(4),** 422.

Behm, D. G., Reardon, G., Fitzgerald, J., & Drinkwater, E. (In press). The effects of 5, 10, 20 repetition maximum on the recovery of voluntary and evoked contractile properties. **Journal of Strength and Conditioning Research.**

Bessman, S.P. & Savabi, F. (1990). The role of the phosphocreatine energy shuttle in exercise and hypertrophy. In A. W. Taylor, P. D. Gollnick, H. J. Green, C. D. Ianuzzo, E. G. Noble, G. Metivier, & J. R. Sutton (eds). **Biochemistry of Exercise VII,** Human Kinetics, Champaign, IL, 167-177.

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

Brandenburg, J. P., Docherty, D., & Benson, C. R. (2000). Acute neuromuscular fatigue following two loading protocols of different intensities (abstract). Proceedings of the Canadian Society for Exercise Physiology Annual Scientific Conference (pp. 58). Canmore, Alberta, Canada.

Chestnut, J.L. & 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,** 353-359.

Corder, K. P., Potteiger, J. A., Nay, K. L., Figoni, S. E., & Hershberger, S. L. (2000). Effects of active and passive recovery conditions on blood lactate, rating of perceived exertion, and performance during resistance exercise. **Journal of Strength and Conditioning Research, 14(2),** 151-156.

Craig, B. W. & Kang, H. Y. (1994). Growth hormone release following single versus multiple sets of back squats: total work versus power. **Journal of Strength and Conditioning Research, 8**(4), 270-275.

Dangott, B., Schultz, E., & Mozdziak, P. E. (2000). Dietary creatine monohydrate upplementation increases mitotic activity during compensatory hypertrophy. **International Journal of Sports Medicine, 21,** 13-16.

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

Dudley, G. A., Tesch, P. A., Miller, M. A., & Buchanan, P. (1991). Importance of eccentric actions in performance adaptations to resistance training. **Aviation, Space, and Environmental Medicine, 62,** 543-550.

Essen-Gustavsson, B. & Tesch, P. A. (1990). Glycogen and triglyceride utilization in relation to muscle metabolic characteristics in men performing heavy-resistance exercise. **European Journal of Applied Physiology, 61**, 5-10.

Fitts, R. H., & Balog, E. M. (1996). Effect of intracellular and extracellular ion changes on E-C coupling and skeletal muscle fatigue. **Acta Physiologica Scandinavica, 156,** 169-181.

Goldspink, G. (1992). Cellular and molecular aspects of adaptation in skeletal muscle. In P. V. Komi (Ed). **Strength and Power in Sport,** Blackwell Scientific Publications, Oxford, 211-229.

Green, H. J. (1990). Manifestations and sites of neuromuscular fatigue. In A. W. Taylor, P. D. Gollnick, H. J. Green, D. Ianuzzo, E. G. Noble, G. Metivier, & J. R. Sutton (Eds). **Biochemistry of Exercise VII**, Human Kinetics, Champaign, IL, 13-35.

Hakkinen, K. (1993). Neuromuscular fatigue and recovery in male and female athletes during heavy resistance exercise. **International Journal of Sports Medicine, 14,** 53-59.

Hakkinen, K. (1994). Neuromuscular fatigue in males and females during strenuous heavy resistance training. **Electromyography and Clinical Neurophysiology, 34,** 205-214.

Hakkinen, K., Kauhanen, H., & Komi. P. V. (1988). Effects of fatiguing loading with a variable resistance equipment on neural activation and force production of the knee extensor muscles. **Electromyography and Clinical Neurophysiology, 28,** 79-87.

Hakkinen, K., & Pakarinen, A. (1993). Acute hormonal responses to two different fatiguing heavy-resistance protocols in male athletes. **Journal of Applied Physiology, 74**(2), 882-887.

Hannie, P. Q., Hunter, G. R., Kekes-Szabo, T., Nicholson, C., & Harrison, P. C. (1995). The effects of recovery on force production, blood lactate, and work performed during bench press exercise. **Journal of Strength and Conditioning Research, 9(1),** 8-12.

Hass, C. J., Garzarella, L., De Hoyos, D. V., & Pollock, M. L. (1998). Effects of training volume on strength and endurance in experienced resistance trained adults. **Medicine and Science in Sports and Exercise, 30(5),** S115.

Hickson, R. C., Hidaka, K., & Foster, C. (1994). Skeletal muscle fiber type, resistance training , and strength-related performance. **Medicine and Science in Sports and Exercise, 26(5),** 593-598.

Houston, M. E. (1999). Gaining weight: The scientific basis of increasing skeletal muscle mass. **Canadian Journal of Applied Physiology, 24**(4), 305-316.

Ingwall, J. S. (1976). Creatine and the control of muscle-specific protein synthesis in cardiac and skeletal muscle. **Circulatory Research, 38**(S), 115-123.

Kauhanen, H., Hakkinen, K., & Komi, P. V. (1989). Neural activation and force production of arm flexor muscle during normal and fatigue loading against constant and variable resistance. **Scandinavian Journal of Sports Science, 11(2),** 79-86.

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

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

Keogh, J. W. L., Wilson, G. J., & Weatherby, R. P. (1999). A cross-sectional comparison of different resistance training techniques in the bench press. **Journal of Strength and Conditioning Research, 13(3),** 247-258.

Kraemer, W. J., Fleck, S. J., & Evans, W. J. (1996). Strength and power training: Physiological mechanisms of adaptation. **Exercise and Sports Science Reviews, 24**, 363-397.

Kraemer, W. J., Marchitelli, L. J., Gordon, S. E., Harman, E. A., Dziados, J. E., Mello, R., Frykman, P. N., McCurry, D., & Fleck, S. J., (1990). Hormonal and growth

factor responses to heavy resistance exercise protocols. **Journal of Applied Physiology, 69**(4), 1442-1450.

Kraemer, W. J., Noble, B. J., Clark, M. J., & Culver, B. W. (1987). Physiologic responses to heavy resistance exercise with very short rest periods. **International Journal of Sports Medicine, 8**(4), 247-252.

Kulling, F. A., Hardison, B. H., Jacobson, B. H., & Edwards, S. W. (1998). The effect of two different rest periods between sets in a resistance training program. **Medicine Science and Sports and Exercise, 30(5),** S165.

Lexell, J., Taylor, C., & Sjorstrom, M. (1985). Analysis of sampling errors in biopsy techniques using data from whole muscle cross sections. **Journal of Applied Physiology, 59(4),** 1228-1235.

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

Linnamo, V., Newton, R. U., Hakkinen, K., Komi, P. V., Davie, A., McGuigan, M., Triplett-McBride, T. (2000). **Journal of Electromyography and Kinesiology, 11,** 417-424.

MacDougall, J. D. (1992). Hypertrophy or hyperplasia. In P. V. Komi (Ed). **Strength and Power in Sport,** Blackwell Scientific Publications, Oxford, 230-237.

MacDougall, J. D., Ray, S., Dale, D. G., McCartney, N., Lee, P., & Garner, S. (1999). Muscle substrate utilization and lactate production during weightlifting. **Canadian Journal of Applied Physiology, 24**(3), 209-215.

MacDougall, J. D., Sale, D. G., Alway, S. E., & Sutton, J. R. (1984). Muscle fiber number in biceps brachii in bodybuilders and control subjects. **Journal of Applied Physiology, 57(5),** 1399-1403.

MacDougall, J. D., Ward, G. R., Sale, D. G., & Sutton, J. R. (1977). Biochemical adaptation of human skeletal muscle to heavy resistance training and immobilization. **Journal of Applied Physiology, 43(4),** 700-703.

McCall, G. E., Byrnes, A., Dickinson, A., & Fleck, S. J. (1998). Sample size required for the accurate determination of fiber area and capillarity of human skeletal muscle. **Canadian Journal of Applied Physiology, 23(6),** 594-599.

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

McLester Jr, J. R. (1997). Muscle contraction and fatigue: The role of adenosine 5'-diphosphate and inorganic phosphate. **Sports Medicine, 23**(5), 287-305.

Medbo, J. I., Jebens, E., Vikne, H., Refsnes, P. E., & Gramvik, P. (2001). Effect of strenuous strength training on the Na-K pump concentration in skeletal muscle of well-trained men. **European Journal of Applied Physiology, 84,** 148-154.

Moss, B. M., Refsnes, P. E., Abildgaard, A., Nicolaysen, K., & Jensen, J. (1997). Effects of maximal effort strength training with different loads on dynamic strength, cross-sectional area, load-power, and load-velocity relationships. **European Journal of Applied Physiology, 75,** 193-199.

Narici, M.V., Hoppeler, H., Kayser, B., Landoni, L., Claassen, H., Gavardi, C., Conti, M., & Cerretelli, P. (1996). Human quadriceps cross-sectional area, torque, and neural activation during 6 months strength training. **Acta Physiologica Scandinavica, 157,** 175-186.

Ostrowski, K. J., Wilson, G. J., Weatherby, R., Murphy, P. W., & Lyttle, A. D. (1997). The effect of weight training volume on hormonal output and muscular size and function. **Journal of Strength and Conditioning Research, 11(1),** 148-154.

Paasuke, M., Ereline, J., & Gapeyeva, H. (1999). Neuromuscular fatigue during repeated exhaustive submaximal static contractions of knee extensor muscles in endurance-trained, power-trained and untrained men. **Acta Physiologica Scandinavica, 166,** 519-526.

Pierce, K., Rozenek, R., & Stone, M. H. (1993). Effects of high volume weight training on lactate, heart rate, and perceived exertion. **Journal of Strength and Conditioning Research, 7(4),** 211-215.

Pincivero, D. M., Lephart, S. M., & Karunakara, R. J. (1997). Effects of rest interval on isokinetic strength and functional performance after short term high intensity training. **British Journal of Sports Medicine, 31,** 229-234.

Pincivero, D. M., Lephart, S. M., & Karunakara, R. J. (1998). Effects of intrasession rest interval on strength recovery and reliability during high intensity exercise. **Journal of Strength and Conditioning Research, 12(3),** 152-156.

Pincivero, D. M., McCann, D. M., & Mark, J. (2000). Effects of rest interval and gender on quadriceps and hamstring torque during maximal intensity muscle contractions. **Canadian Journal of Applied Physiology, 25(5),** 398.

Pollock, M. L., Abe, T., De Hoyos, D. V., Garzarella, L., Hass, C. J., & Werber, G. (1998). Muscular hypertrophy response to six months of high- or low-volume resistance training. **Medicine and Science in Sports and Exercise, 30(5),** S116.

Pullinen, T., Huttunen, P, & Komi, P. V. (2000). Plasma catecholamine responses and neural adaptation during short-term resistance training. **European Journal of Applied Physiology, 82,** 68-75.

Raastad, T., & Hallen, J. (2000). Recovery, of skeletal muscle contractility after high- and moderate-intensity strength exercise. **European Journal of Applied Physiology, 82,** 206-214.

Robergs, R. A., Pearson, D. R., Costill, D. L., Fink, W. J., Pascoe, D. D., Benedict, M. A., Lambert, C. P., & Zachwieja, J. J. (1991). Muscle glycogenolysis during different intensities of weight-resistance exercise. **Journal of Applied Physiology, 70**(4), 1700-1706.

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

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

Sanborn, K., Boros, R., Hruby, J.,Schilling, B., O'Bryant, H. S., Johnson, R. L., Hoke, T., Stone, M. E., & Stone, M. H. (2000). Short-term performance effects of weight training with multiple sets not to failure vs. a single set to failure in women. **Journal of Strength and Conditioning Research, 14(3),** 328-331.

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

Sleivert, G. S. (1994). **Neural and muscular factors influence maximal power generation**. Unpublished doctoral dissertation, University of Victoria, Victoria.

Suter, E., Herzog, W., Sokolosky, J., Wiley, J. P., & MacIntosh, B. R. (1993). Muscle fiber type distribution as estimated by Cybex testing and by muscle biopsy. **Medicine and Science in Sports and Exercise, 25(3),** 363-370.

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., Colliander, E. B., Kaiser, P. (1986). Muscle metabolism during intense, heavy-resistance exercise. **European Journal of Applied Physiology, 55**, 362-366.

Tesch, P. A., Ploutz-Snyder, L. L., Ystrom, L., Castro, M. J., & Dudley, G. A. (1998). Skeletal muscle glycogen loss evoked by resistance exercise. **Journal of Strength and Conditioning Research, 12(2),** 67-73.

Tesch, P. A., Thorsson, A. & Colliander, E. B. (1990). Effects of eccentric and concentric resistance training on skeletal muscle substrates, enzyme activities, and capillary supply. **Acta Physiologica Scandinavica, 140,** 575-580.

Touey, P. R., Sforzo, G. A., & McManis, B. G. (1994). Effects of manipulating rest periods on isokinetic muscle performance. **Medicine and Science in Sports and Exercise, 26(5),** S30.

Vollestad, N. K. (1997). Measurement of human muscle fatigue. **Journal of Neuroscience Methods, 74,** 219-227.

Weiss, L. (1991). The obtuse nature of muscular strength: The contribution of rest to it development and expression. **Journal of Applied Sport Science Research, 5(4),** 219-227.

Weiss, L., Coney, H. D., & Clark, F. C. (1999). Differential functional adaptations to short-term low-, moderate-, and high-repetition weight training. **Journal of Strength and Conditioning Research, 13(3),** 236-241.

Wenger, H. A., & Reed, A. T. (1976). Metabolic factors associated with muscular fatigue during aer4obic and anaerobic work. **Canadian Journal of Applied Sports Science, 1,** 43-48.

Westerblad, H., Allen, D. G., Bruton, J. D., Andrade, F. H., & Lannergren, J. (1998). Mechanisms underlying the reduction in isometric force in skeletal muscle fatigue. **Acta Physiologica Scandinavica, 162,** 23-260.

Willoughby, D. S. (1993). The effects of mesocycle-length weight training programs involving periodization and partially equated volumes on upper and lower body strength. **Journal of Strength and Conditioning Research, 7(1),** 2-8.

Appendix A: Review of Literature

Introduction

Fatigue as a result of resistance-type exercise is recognized as either a temporary decrease in the maximum force generating capacity of muscle (Kent-Braun, 1999), failure to maintain a required sub-maximal level of force (Tesch, Komi, Jacobs, Karlsson, & Viitasalo, 1983) or the inability to complete the next repetition (MacDougall, Ray, Sale, McCartney, Lee, & Garner, 1999). Acute decrements in muscle performance are produced by physiological disturbances at various sites between the CNS and the contractile elements within the muscle (Kent-Braun, 1999). Mechanisms responsible for fatigue are considered to be of central and/or peripheral origin (Tesch et al., 1983; McLester Jr., 1997; Kent-Braun, 1999). Central mechanisms, also referred to as central or neural fatigue, are the processes proximal to the neuromuscular junction (NMJ) that limit neural drive (Kawakami, Amemiya, Kanehisa, Ikegawa, & Fukunaga, 2000). Peripheral factors associated with muscle fatigue include those factors that impair the excitation-contraction process, consequently reducing the generation of force (McLester Jr., 1997; Tesch et al., 1983).

Central Factors

Possible sites of central fatigue include: (a) motivational factors (Vollestad, 1997), (b) excitatory and sensory inputs to the motor cortex (Paasuke, Ereline, & Gapeyeva, 1999; Vollestad, 1997), (c) magnitude of descending (pre-motor neuron) drive (Fitts & Balog, 1996), (d) excitability of the motor neurons (Paasuke et al., 1999, Vollestad, 1997), and (e) antagonist co-activation (Behm, Reardon, Fitzgerald, & Drinkwater, In Press). Any of these mechanisms, whether acting independently or in combination, will reduce the amount of force produced by the agonist muscle.

Peripheral Factors

Possible peripheral factors contributing to neuromuscular fatigue include (a) impaired neuromuscular junction transmission (Paasuke et al., 1999; Green, 1997), (b) reduced sarcolemma excitability (Paasuke et al., 1999; De Luca, 1997), (c) decreased Ca^{2+} release from the sarcoplasmic reticulum which will disrupt excitation-contraction coupling (Fitts & Balog, 1996), (d) the accumulation of H^+ which will interfere with cross-bridge formation (Kent-Braun, 1999; Wenger & Reed, 1976), (e) the accumulation of P_i which will reduce the amount of force per cross-bridge (Westerblad, Allen, Bruton, Andrade, & Lannergren, 1998), and (f) availability of muscle substrates (Paasuke et al., 1999).

The temporary decrement in the force generating capacity of muscle during resistance training, as a result of the accumulation of acute neuromuscular fatigue, often results in muscle failure or the inability to complete another repetition (MacDougall et al., 1999). The relative contribution of central and peripheral factors in eliciting muscle failure is unknown and may depend on the structure of the training protocol (Hakkinen, 1993). The purpose of this review is to summarize research studies that have measured neuromuscular muscle fatigue following different resistance training sessions and to identify possible similarities and patterns in the available literature.

Muscle Performance

A common method used to assess the magnitude of fatigue induced by resistance training is to compare the force exerted during a maximal (isometric or isokinetic) voluntary contraction performed before and after a resistance training protocol. A reduction in the force generating capacity of muscle is an indicator of the degree of acute neuromuscular

fatigue accompanying resistance training (Kent-Braun, 1999). Significant decrements in MVIC have been observed in the quadriceps and forearm flexors muscle groups following the performance of a single set of resistance training (Hakkinen, Kauhanen, & Komi, 1989; Kauhanen, Hakkinen, & Komi, 1988). Although it is apparent that resistance training induces acute decrements in maximal force generating capacity, few studies have actually compared the acute decrement in maximal force generating ability in response to different training protocols. Therefore, the effect of different training variables on the development of acute fatigue is uncertain.

Recently, Raastad and Hallen (2000) compared the acute changes in peak isokinetic force in well-trained athletes following two different resistance training protocols. Significantly greater reductions in leg extension isokinetic torque were observed following a protocol using 100% of 3-RM in comparison to a protocol using 70% of 3-RM. Although it appeared that intensity was a key contributor to greater resistance training-induced fatigue induced by the 100% of 3-RM protocol, these conclusions were difficult to reach as the protocols were not equated for the training volume performed or repetitions to failure.

MacDougall et al. (1999) suggested that the acute decrease in the maximum force generating capacity is determined by relative intensity of the resistance training load if repetitions are performed to failure. For example, when a muscle can no longer perform a repetition at a load equal to 80% 1-RM, the force generating capacity is reduced by 20% for that exercise. Thus, training loads of a lower intensity would elicit greater decrements in force production if performed to failure. Behm et al. (2000) compared the magnitude of the resistance training-induced reduction in force during a maximal

voluntary isometric contraction (MVIC) following protocols that were performed to failure using different relative training intensities. MVIC's of the elbow flexors were performed before and after a single set of a 5-RM, 10-RM or 20-RM elbow flexion task. All three protocols experienced a reduction in maximal isometric force that was between 18.1 - 21.4%. Although maximal isometric force was reduced following all three protocols, the differences between the decrements were not significant indicating that intensity may not be the key variable associated with resistance training-induce fatigue.

Decrements in MVIC were similar following resistance training protocols of different intensities but equated for: (a) sets performed to failure, (b) training volume, and (c) total duration of exercise (Brandenburg, Docherty & Benson, 2000). Specifically, three sets of single-arm elbow flexion repetitions to failure at approximately 75% 1-RM produced an average 19% decrease in the MVC of the forearm flexors whereas 6 sets of repetitions to failure at approximately 85% 1-RM elicited a 22.2% decrement. The difference between these decrements was not significant. The lack of difference may be attributable to the similarities in sets performed to failure, training volume, and contraction-duration of the two protocols. MVIC was also measured following the third set of the protocol utilizing 85% 1-RM. After 3 sets, MVIC was reduced by only 14.8% compared to the 22.2% reduction after 6 sets, even though the training load was kept constant. The comparable decrements in MVC between the two different protocols, as well as the difference in MVC drop-off between the third and sixth set utilizing the same resistance, appear to suggest that training volume is an important training variable with regards to acute reductions in MVC.

Indirect evidence supporting the role of training volume as a key contributing factor to neuromuscular fatigue is provided by comparing two different studies performed by Hakkinen (1994, 1993). Well-trained subjects experienced a 47% reduction in MVIC of the leg extensors following a high-volume training session consisting of 10 sets of 10-RM squat-lift exercises (Hakkinen, 1994). Following a low volume training session, comprised of 20 sets of squats using 1-RM loads, MVIC was reduced by only 24% in subjects with similar training experience (Hakkinen, 1993). Until a study investigates the effect of different training volumes on acute decrements in MVIC while equating for other training variables, the role of training volume will remain uncertain.

Any interpretation of the above research studies should be made with caution as fatigue was produced during dynamic contractions but measured using a maximal isometric contraction. Additionally, there is a delay during the switching of contraction modes between the training protocol and isometric testing. The elapsed time during this delay may allow for some recovery and as a result the MVIC may not truly reflect the fatigue created by the training protocol.

An alternate method used to determine the magnitude of fatigue elicited by a resistance training protocol is to observe the performance of the exercise over series of successive sets. During a training session, muscle performance can be evaluated by either measuring the drop-off in number of repetitions performed in consecutive sets while the training load remains constant or recording the reduction in training load necessary to avoid a drop-off in the number of repetitions performed per set over successive sets. Utilizing trained subjects, MacDougall et al. (1999) observed a progressive decrease in the number of repetitions performed to failure over three sets

using a constant load of 80% 1-RM and 3 minutes of rest between sets. Subjects completed an average of 11.7 repetitions in the first set, 9.2 repetitions in the second set and 7.2 repetitions in the final set. Similarly, subjects unaccustomed to resistance training experienced an average decrease of 10.6 repetitions over 4 sets separated by 2 minutes while utilizing a load of 65% 1-RM (Hannie, Hunter, Kekes-Szabo, Nicholson & Harrison, 1995).

As fatigue accumulates over the course of an exercise, the relative intensity of a training load (i.e. 80% 1-RM) increases because the force generating capacity of the muscle is reduced. Consequently, investigators have adjusted the training load between sets to ensure that repetitions per set are maintained at a specified number. Hakkinen and Pakarinen (1993) conducted a study in which the training load was reduced after each set to ensure the predetermined number of repetitions were performed. Training load was decreased by 10.3% in order to maintain the performance of 20 sets of a 1-RM whereas a 24.6% reduction in training was necessary to ensure the completion of 10 repetitions for 10 sets.

Abdessemed, Duche, Hautier, Poumarat and Bedu (1999) compared the power output of each repetition during a bench press exercise in which the duration of rest intervals was different between protocols. Subjects performed 10 sets of 6 repetitions at 70% 1-RM utilizing either 1, 3 or 5 minute rest periods. Power output during each of the 6 repetitions per set was maintained over 10 sets in the 3- and 5-minute rest condition. Similar results were demonstrated in the 1-minute rest protocol during the first 3 sets. However, from sets 4 to 10, a significant reduction in power output occurred during the final 3 repetitions per set. Although these results suggest that rest interval length is an

important contributor to the acute reduction in muscle performance, the training protocols were not equated for sets performed to failure.

Although resistance training-induced reductions in MVIC and dynamic muscle performance are an indication of neuromuscular fatigue, these measures are limited as they fail to provide information with regards to the sites and mechanisms of fatigue (Green, 1990).

Mechanisms of resistance training-induced fatigue

Muscle Substrate Availability

In order for high-force, repetitive muscle performance to be sustained, ATP production must be maintained (MacDougall et al., 1999). Muscle glycogenolysis and phosphocreatine (PCr) breakdown appear to be the primary sources for ATP re-synthesis during resistance training (MacDougall et al.; Tesch et al., 1986). Muscle glycogen has been the most extensively studied muscle substrate following bouts of resistance training. Robergs et al. (1991) investigated the effect of different resistance training intensities on the depletion of muscle glycogen. The researchers compared glycogen depletion between resistance training sessions utilizing training loads of 70% and 35% 1-RM. Using 70% of 1-RM, subjects performed 6 repetitions of leg extension per set for 6 sets. With the load 30% 1-RM, subjects performed 6 sets of 12 repetitions. Although it was not reported, it is unlikely that either protocol elicited muscle failure. Muscle glycogen was depleted by about 20% after 3 sets and by approximately 40% of the pre-exercise levels following the completion of 6 sets of both protocols. The lack of differences produced by either protocol may be attributed to the similar volumes of work that were performed. These observations led the researchers to conclude that the magnitude of muscle

glycogen depletion is dependent on the volume of work performed and the rate of glycogen utilization is a function of the training intensity.

A 30% reduction in muscle glycogen was observed following the performance of approximately 9 sets of 6 leg extension repetitions utilizing a load of 70% 1-RM (Pascoe, Costill, Fink, Robergs, & Zachwieja, 1993). Although this training protocol was designed to elicit muscle failure on the last repetition of the final set and the training volume was greater than that used in the study by Robergs et al. (1991), less glycogen was depleted. Muscle glycogen content of the vastus lateralis (VL) was reduced by an average of 40% following the performance of a high volume resistance training session. The training session consisted of performing 5 sets of four lower body resistance training exercises (Tesch et al. 1986). Although the volume and duration of the training session was greater than those used in the study by Robergs et al. (1991) the magnitude of muscle glycogen depletion was similar. These results along with those observed by Pascoe et al. (1993) fail to support the relationship between training volume and glycogen utilization. It is difficult to form conclusions based on the above studies, as differences in subject training experience, inter-set rest interval length and biopsy procedures make comparisons between these studies difficult.

Using a within subject design, MacDougall et al. (1999) investigated substrate utilization following two resistance-training sessions of different training volumes. Trained subjects performed either 1 set or 3 sets of elbow flexion repetitions to failure at a load of 80% 1-RM. Muscle glycogen content after 1 set of exercise was depleted by only 12%. Three sets of elbow flexion produced a significantly greater (24%) reduction in muscle glycogen, thus supporting the evidence that suggests muscle glycogen

utilization is influenced by training volume. However, because muscle glycogen stores are far from being completely exhausted as a result of resistance training, it is doubtful that they contribute to reductions in muscle performance.

PCr is a metabolic substrate that is thought to contribute to impairments in muscle performance (MacDougall et al., 1999; Tesch et al., 1986). Intramuscular levels of PCr were significantly depleted following the performance of 1 set (by 62%) and 3 sets (by 50%) of single-arm elbow flexion with a load of 80% 1-RM. There were no differences between the two protocols with regard to the amount of PCr depleted. The lack of difference between the two conditions may be attributable to the 3-minute rest period that was used during the 3 set protocol. Three minutes of rest would have allowed for near complete replenishment of PCr to resting levels (MacDougall et al., 1999). However, despite the return of PCr levels to near resting a progressive reduction in the number of repetitions performed per set was observed over the 3 set protocol. Thus, it was concluded that limited PCr availability contributed to muscle failure in the first set but muscle failure in subsequent sets was the result of other mechanisms, specifically reduced intramuscular pH.

Blood Lactate

Increased muscle acidity has been suggested to be a primary cause of reduced muscle performance following high volume resistance training (Tesch et al., 1986). Decreases in muscle pH influence glycolysis, PCr re-synthesis, Ca^{2+} reabsorption by the sarcoplasmic reticulum, Ca^{2+} binding to troponin, cross-bridge formation (McLester Jr., 1997) as well as muscle fiber excitability (Kent-Braun, 1999), all of which impair muscle function.

Blood lactate, produced during resistance training, is commonly measured as an indicator of the peripheral contributions to acute neuromuscular fatigue. Although blood lactate accumulation is dependent on the production, distribution, and elimination of lactate within the muscle, it is thought to reflect increases in muscle lactate (Tesch et al., 1983, 1986). Hakkinen (1994) suggested that the overall volume and intensity of resistance training influences the acute blood lactate response. Further, changes in blood lactate also appear to be influenced by the duration of the exercise as well as length of rest intervals between sets (Kraemer, Marchitelli, Gordon, Harmon, Dziados et al., 1990; Kraemer, Fleck, Dziados, Harman, Marchitelli, et al., 1993).

Hakkinen and Pakarinen (1993) compared training protocols characteristic of maximal strength and hypertrophy training. Subjects performed 20 sets of a squat-lift exercise at a 1-RM load for the maximal strength training session whereas the hypertrophy training session consisted of performing 10 sets utilizing a 10-RM load. Although the mean blood lactate concentration increased significantly in response to both protocols, the elevation was greater following the hypertrophy (10-RM) protocol. As the protocols differed in intensity of the training load, exercise duration and training volume, it is difficult to determine the influence of each of these training variables on the lactate response.

In a series of investigations with similar experimental designs, Kraemer et al. (1990, 1993) examined the changes in blood lactate concentration elicited by training sessions traditionally utilized to develop maximal strength and muscle size. Unlike the investigation by Hakkinen and Pakarinen (1993), additional training sessions were performed that controlled for load intensity, amount of work performed and rest interval

duration, thus attempting to identify the importance of each of these training variables on the production of peripheral fatigue.

The subjects in these studies, who were males (1990) or females (1993) with a recreational resistance training background, performed two primary training sessions: a) a maximal strength training session and b) a hypertrophy training session. Subjects performed 3-5 sets of 8 different exercises at a 5-RM load with 3 minutes rest between sets for the maximal strength training session. The hypertrophy training session involved performing 3 sets of the same exercises utilizing a 10-RM load with 1-minute rest intervals. In order to determine the influence of specific training variables on fatigue, two secondary protocols, which controlled for either load or rest interval length, were performed and compared to each primary training session.

Comparisons were made between the primary hypertrophy sessions and a session that manipulated training load (5-RM with 1-minute rest intervals between sets) as well as a session that altered the rest period (10-RM with 3 min of rest between sets). The work performed in each of these three training sessions (hypertrophy-series) was equated. To control for the same variables in the maximal strength training protocol, subjects performed additional training sessions in which load (10-RM with 3-minute rest periods) or rest (5-RM with rest intervals of 1 min) were manipulated. Although the work performed between these three protocols (strength-series) was equivalent, it was less than the worked performed in the three training sessions comprising the hypertrophy series.

Blood lactate concentrations were significantly elevated mid- and immediately post-exercise in response to all 6 of the protocols performed. When the two primary training sessions were compared, blood lactate concentrations were greatest following the

primary hypertrophy protocol. An understanding of the influence of the different training variables is difficult to establish since the two primary training sessions were not equated for intensity, exercise duration, work performed and rest interval duration.

When comparisons were made within the hypertrophy and strength series in which intensity, volume and exercise duration were equal between protocols, increases in blood lactate were consistently greater in the protocols incorporating the 1-minute rest condition than in the 3-minute rest condition. When intensity was the manipulated training variable, increases in blood lactate concentration were inconsistent.

Intensity/Training Volume/Exercise Duration

Abernethy and Wehr (1995) measured the blood lactate response following 3 sets of leg press utilizing either a 5-RM or 15-RM load. Both training protocols produced significant increases in blood lactate following the first and third set of exercise. Further, the elevations in blood lactate following the 15-RM protocol were greater than the increases following the 5-RM protocol. Although the intensity of the training load was less during the 15-RM protocol, exercise duration and training volume were greater, thus suggesting the association of either of these variables in eliciting peripheral muscle fatigue.

Greater increases in blood lactate were demonstrated after 5 sets of leg extensions utilizing a 10-RM load than after an explosive training session using the same variables except with a lower load (Linammo, Hakkinen, & Komi, 1998). Similar to Abernethy and Wehr (1995) the differences in the blood lactate concentrations were attributed to higher average power output and longer working periods. Increases in blood lactate concentration were significantly greater following 1 set of bilateral leg extension at 60%

1-RM than at 80% 1-RM (Pullinen, Nicol, MacDonald, & Komi, 1999). Greater exercise duration and work performed during the 60% 1-RM condition may account for the observed results.

Recently it was demonstrated that increases in blood lactate were similar following 3 sets of repetitions to failure of single-arm elbow flexion utilizing approximately 75% 1-RM and 6 sets of repetitions to failure utilizing approximately 85% 1-RM (Brandenburg, 2000). Although the intensity was different, the training volume and duration of exercise were equated between the protocols, thus supporting the role of these training variables in eliciting peripheral fatigue.

Additional support was provided by MacDougall et al. (1999) in which subjects performed either 1 or 3 sets of a single-arm elbow flexion task at 80% 1-RM. Blood lactate increased in response to both training sessions, however greater increases were observed following 3 sets of single-arm elbow flexion. Because the two protocols utilized the same intensity, the difference in the blood lactate response was attributed to the greater exercise duration and training volume of the 3 set protocol.

Regan and Potteiger (1999) compared the blood lactate concentrations after 3 isokinetic-training protocols of 20 contractions using different velocities. The smallest increase in blood lactate occurred following the protocol incorporating the shortest exercise duration (12 s) and lowest total work, thus providing additional support for the association between training volume and exercise duration with the acute blood lactate response. Alternatively, concentrations of blood lactate demonstrated similar increases in response to isokinetic resistance training sessions different in duration (30 vs. 60 s) but equal in total work performed (Regan & Potteiger, 1999). Perhaps training volume rather

than exercise or contraction duration is the primary determinant of peripheral muscle fatigue.

Training volume, defined as sets x repetitions x load (Tesch, 1992), may reflect the number of cross-bridges that are formed and utilized during a resistance training protocol. During the kinetics of cross-bridge formation and dissociation ATP is required. Consequently, a high volume training program requiring more cross-bridges may also be more metabolically demanding because of a greater ATP demand.

Rest Intervals

In addition to Kraemer et al. (1990, 1993), Abdessemed et al. (1999) demonstrated that blood lactate concentrations were greater with shorter rest intervals. Abdessemed et al. investigated the effect of utilizing 1-, 3- and 5-minute rest periods on blood lactate during the performance of 10 sets of 6 repetitions at a load equaling 70% of 1RM. Each of the three rest conditions produced significant increases in blood lactate after 10 sets of exercise. There were no differences in blood lactate levels between the 3- and 5-minute rest conditions. Further, increases in blood lactate after the initial three sets were similar between the three rest conditions. The 1-minute rest protocol, however, resulted in significantly greater elevations in blood lactate concentrations from the forth to the tenth set. It was postulated that the 1-minute rest condition did not permit complete PCr re-synthesis and therefore depended on glycogenolysis for the re-synthesis of ATP. Increases in glycolytic activity in the 1-minute rest protocol would account for the greater increases in blood lactate concentration. In addition to the differences in rest interval length, the protocols were not equated for sets performed to failure.

Muscle Activation

The amplitude of the integrated electromyographic (iEMG) signal recorded during the performance of an MVIC (before and after a resistance training protocol) has been observed to significantly decrease (Behm et al., In Press; Linnamo et al., 1998; Kauhanen et al., 1989; Hakkinen et al., 1988) or remain unchanged (Linnamo et al., 2000). In these studies, the reductions in iEMG were associated with concomitant decreases in MVIC force. Thus, it would appear that resistance training-induced reductions in muscle activity (iEMG) limit maximal force generating capacity. However, in many of these studies the recovery of MVIC force occurred prior to the recovery of the iEMG signal, thus complicating this relationship (Behm et al., In Press; Kauhanen et al., 1989; Hakkinen et al., 1988).

Electromyography (EMG) is often used to assess the contribution of central factors to resistance training-induced neuromuscular fatigue. However, because EMG recording electrodes are placed superficial to the active muscle, EMG cannot distinguish between central factors as well as neuromuscular junction transmission and sarcolemma excitability (Green, 1990, Vollestad, 1997). A more effective method to determine the contribution of central factors to an acute reduction in muscle force generating ability is interpolated twitch technique (ITT). ITT involves electrically stimulating the motor nerve of a muscle while that muscle is performing a maximal voluntary contraction (Kent-Braun, 1999;Sale, 1988). The difference between the force achieved while maximally contracting voluntarily and the force achieved while the electrical stimulation is superimposed represents the degree of centrally mediated neuromuscular inactivation. To determine the contribution of central factors to resistance training-induced

neuromuscular fatigue, ITT is performed prior to and immediately upon completion of a resistance training protocol. An increase in the force elicited by the electrical stimulation suggests central factors contribute to the acute impairment in muscle performance.

Recently, Behm et al. (In Press) used ITT to determine the acute neuromuscular response after performing one set of a 5-, 10- or 20-RM loading protocol. Although all three loading conditions resulted in significant muscle inactivation, as measured by an increase in ITT, the differences in the magnitudes of muscle inactivation were not significant. The researchers hypothesized that the 5-RM protocol would produce the largest increases in ITT. Failure of this to occur may be attributed to the greater time under tension and training volume of the 10- and 20-RM protocols. It is also uncertain how the previous resistance training experience of the subjects influenced the acute changes in muscle activation.

In the above studies, subjects trained utilizing dynamic muscle actions but IEMG (MVIC) and ITT were tested under isometric conditions. Perhaps the extent of muscle activation may be specific to the type of muscle action performed, thus valid changes in muscle activation achieved during dynamic loading may require measures incorporating dynamic muscle actions (Behm et al., 2000). Further, a period of time may have elapsed between the final repetition of the loading protocol and the onset of the MVIC during which some recovery may have occurred. Recovery would facilitate MVIC and IEMG performance and ultimately underestimate the degree of fatigue elicited by the loading protocols. Additionally, the underestimation of muscle inactivation may be a result of the muscle(s) selected for IEMG analysis. The exercises performed during the resistance-training protocols and maximal isometric contractions involved the activation of multiple

muscles. IEMG was only recorded on a select muscle or muscles during the MVIC. Perhaps greater levels of muscle inactivation were present but not in the muscle(s) tested (Behm et al., 2000).

The role of fatigue in long-term neuromuscular adaptations

Although the central and peripheral factors associated with resistance training produce an acute and transient decrement in muscle performance, they also appear to be associated with long-term gains in muscle strength and hypertrophy (Rooney, Herbert & Balnave, 1994).

Rooney et al. (1994) compared the increases in strength following 6 weeks of two different resistance-training programs. One of the programs consisted of subjects performing continuous repetitions without a pause whereas in the second program, a 30-second pause separated successive repetitions. Although both training groups utilized the same relative training intensity and performed the same number of repetitions, the increases in dynamic strength of the group that performed continuous repetitions exceeded those of the intermittent protocol. Further, acute decreases in isometric force, measured during initial training sessions, were greater following the continuous protocol than after the non-continuous protocol. Thus, the researchers concluded that greater amounts of acute neuromuscular fatigue accumulated during the continuous training protocol providing a stimulus that produced greater increases in strength.

In a study comparing two resistance training programs, increments in strength and hypertrophy were larger following the program that elicited greater acute metabolic changes as measured during a training session (Schott, McCully & Rutherford, 1995). The researchers compared the effects of 14 weeks of continuous isometric training with

those of intermittent isometric training. The continuous group performed 4 isometric contractions of a 30-second duration using an intensity of 70% MVC and the intermittent group performed 4 sets of 10 isometric contractions with each contraction lasting 3 seconds. Acute metabolic changes (pH and Pi: PCr ratio) within the muscle, measured during initial training sessions, were larger in the continuous condition although the intensity and total exercise duration were equivalent to the intermittent protocol. Increases in strength and hypertrophy following the 14 weeks of training were greater in response to the continuous training protocol. Consequently, peripheral fatigue was identified as a stimulus for strength and hypertrophic adaptations.

Conclusion

Increases in muscle strength following resistance training programs are primarily attributed to increases in muscle size and/or increases in neural activity (Chestnut & Docherty, 1999; Garfinkel & Cafarelli, 1992; Kawakami et al., 1995; McCall et al., 1996; Narici et al., 1989). Despite a paucity of long-term research, the specific type of adaptation, however, appears to be influenced by the manipulation of the training variables that compose a resistance training program (Houston, 1999; Tan, 1999; Kraemer, Fleck & Evans, 1996). However, it does appear that certain training variables (i.e. training volume and rest interval length) influence the acute neuromuscular response to a bout of resistance training. If the central and peripheral factors that contribute to acute decrements in muscle performance are also associated with the long-term resistance training adaptations, it would be useful to identify and compare resistance training variables that might be key to the development of strength and muscle size.

References

Abernethy, P. J., & Wehr, M. (1997). Ammonia and lactate response to leg press work at 5 and 15 RM. **Journal of Strength and Conditioning Research, 11(1),** 40-44.

Abdessemed, D., Duche, P., Hautier, C., Poumarat, C., & Bedu, M. (1999). Effect of recovery duration on muscular power and blood lactate during the bench press exercise. **International Journal of Sports Medicine, 20,** 368-373.

Atha, J. (1981). Strengthening muscle. **Exercise and Sport Science Reviews, 9,** 1-74.

Behm, D. G., Reardon, G., Fitzgerald, J., & Drinkwater, E. (2000). The effects of 5, 10, 20 repetition maximum on the recovery of voluntary and evoked contractile properties. **Journal of Strength and Conditioning Research,** (In press).

Brandenburg, J. P., Docherty, D., & Benson, C. R. (2000). Acute neuromuscular fatigue following two loading protocols of different intensities (abstract). Proceedings of the Canadian Society for Exercise Physiology Annual Scientific Conference (pp. 58). Canmore, Alberta, Canada.

Chestnut, J.L. & 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,** 353-359.

Fitts, R. H., & Balog, E. M. (1996). Effect of intracellular and extracellular ion changes on E-C coupling and skeletal muscle fatigue. **Acta Physiologica Scandinavica, 156,** 169-181.

Gandevia, S. C. (1992). Some central and peripheral factors affecting human motoneuronal output in neuromuscular fatigue. **Sports Medicine, 13**(2), 93-98.

Garfinkel, S., & Cafarelli, E. (1992). Relative changes in maximal force, EMG, and muscle cros-sectional area after isometric training. **Medicine and Science in Sports and Exercise, 24**(11), 1220-1227.

Green, H. J. (1990). Manifestations and sites of neuromuscular fatigue. In A. W. Taylor, P. D. Gollnick, H. J. Green, D. Ianuzzo, E. G. Noble, G. Metevier & J. R. Sutton (Eds). **Biochemistry of exercise VII**, Human Kinetics, Champaign, IL, 13-36.

Hakkinen, K. (1993). Neuromuscular fatigue and recovery in male and female athletes **International Journal of Sports Medicine, 14**during heavy resistance exercise., 53-59.

Hakkinen, K. (1994). Neuromuscular fatigue in males and females during strenuous heavy resistance loading. **Journal of Neurophysiology, 34**, 205-214.

Hakkinen, K., Kauhanen, H., & Komi. P. V. (1988). Effects of fatiguing loading with a variable resistance equipment on neural activation and force production of the knee extensor muscles. **Electromyography and Clinical Neurophysiology, 28,** 79-87.

Hakkinen, K., & Pakarinen, A. (1993). Acute hormonal responses to two different fatiguing heavy-resistance protocols in male athletes. **Journal of Applied Physiology, 74**(2), 882-887.

Hannie, P. Q., Hunter, G. R., Kekes-Szabo, T., Nicholson, C., & Harrison, P. C. (1995). The effects of recovery on force production, blood lactate, and work performed during bench press exercise. **Journal of Strength and Conditioning Research, 9**(1), 8-12.

Houston, M. E. (1999). Gaining weight: The scientific basis of increasing skeletal muscle mass. **Canadian Journal of Applied Physiology, 24**(4), 305-316.

Kauhanen, H., Hakkinen, K., & Komi, P. V. (1989). Neural activation and force production of arm flexor muscle during normal and fatigue loading against constant and variable resistance. **Scandinavian Journal of Sports Science, 11(2),** 79-86.

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

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

Kamen, G., & Caldwell, G. E. (1996). Physiology and interpretation of the electromyogram. **Journal of Clinical Neurophysiology, 13**(5), 366-384.

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

Komi, P. V. (1986). Training of muscle strength and power: Interaction of neuromotoric, hypertrophic, and mechanical factors. **International Journal of Sports Medicine, 7**, 10-15 (supplement).

Kraemer, W. J., Fleck, S. J., Dziados, J. E., Harman, E. A., Marchitelli, L. J., Gordon, S. E., Mello, R., Frykman, P. N., Koziris, L. P., & Triplett, N. T. (1993). Changes in

hormonal concentrations after different heavy-resistance exercise protocols in women. **Journal of Applied Physiology, 75**, 594-604.

Kraemer, W. J., Fleck, S. J., & Evans, W. J. (1996). Strength and power training: Physiological mechanisms of adaptation. **Exercise and Sports Science Reviews, 24**, 363-397.

Kraemer, W. J., Marchitelli, L. J., Gordon, S. E., Harman, E. A., Dziados, J. E., Mello, R., Frykman, P. N., McCurry, D., & Fleck, S. J., (1990). Hormonal and growth factor responses to heavy resistance exercise protocols. **Journal of Applied Physiology, 69**(4), 1442-1450.

Kraemer, W. J., Noble, B. J., Clark, M. J., & Culver, B. W. (1987). Physiological responses to heavy-resistance exercise with very short rest periods. **International Journal of Sports Medicine, 8**(4), 247-252.

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

Linnamo, V., Newton, R. U., Hakkinen, K., Komi, P. V., Davie, A., McGuigan, M., Triplett-McBride, T. (2000). **Journal of Electromyography and Kinesiology, 11**, 417-424.

MacDougall, J. D., Ray, S., Dale, D. G., McCartney, N., Lee, P., & Garner, S. (1999). Muscle substrate utilization and lactate production during weightlifting. **Canadian Journal of Applied Physiology, 24**(3), 209-215.

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

McLester Jr, J. R. (1997). Muscle contraction and fatigue: The role of adenosine 5'-diphosphate and inorganic phosphate. **Sports Medicine, 23**(5), 287-305.

Narici, M. V., Roi, G. S., Landoni, L., Minetti, A. E., & Cerretelli, P. (1989). Changes in force, cross-sectional area and neural activation during strength training and detraining of the quadriceps. **European Journal of Applied Physiology, 59**, 310-319.

Paasuke, M., Ereline, J., & Gapeyeva, H. (1999). Neuromuscular fatigue during repeated exhaustive submaximal static contractions of knee extensor muscles in endurance-trained, power-trained and untrained men. **Acta Physiologica Scandinavica, 166**, 519-526.

Pascoe, D. D., Costill, D. L., Fink, W. J., Robergs, R. A., & Zachwieja, J. J. (1993). Glycogen resynthesis in skeletal muscle following resistive exercise. **Medicine and Science in Sports and Exercise, 25**(3), 249-354.

Psek, J. A., & Cafarelli, E. (1993). Behavior of coactive muscles during fatigue. **Journal of Applied Physiology, 74**(1), 170-175.

Pullinen, T., Nicol, C., MacDonald, E., & Komi, P. V. (1999). Plasma catecholamine responses to four resistance exercise tests in men and women. **European Journal of Applied Physiology, 80**, 125-131.

Regan, W. E., & Pottetger, J. A. (1999). Isokinetic exercise velocities and blood lactate concentrations in strength/power and endurance athletes. **Journal of Strength and Conditioning Research, 13**(2), 157-161.

Raastad, T., & Hallen, J. (2000). Recovery, of skeletal muscle contractility after high- and moderate-intensity strength exercise. **European Journal of Applied Physiology, 82,** 206-214.

Robergs, R. A., Pearson, D. R., Costill, D. L., Fink, W. J., Pascoe, D. D., Benedict, M. A., Lambert, C. P., & Zachwieja, J. J. (1991). Muscle glycogenolysis during different intensities of weight-resistance exercise. **Journal of Applied Physiology, 70**(4), 1700-1706.

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

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

Tesch, P. A. (1992). Training for bodybuilding. In P. V. Komi (Ed). **Strength and Power in Sport**, Blackwell Scientific, Oxford, 370-380.

Tesch, P. A., Colliander, E. B., Kaiser, P. (1986). Muscle metabolism during intense, heavy-resistance exercise. **European Journal of Applied Physiology, 55**, 362-366.

Tesch, P. A., Komi, P. V., Jacobs, I., Karlsson, J., & Viitasalo, J. T. (1983). Influence of lactate accumulation of EMG frequency spectrum during repeated concentric contractions. **Acta Physiologica Scandinavica, 119**, 61-67.

Wenger, H. A., & Reed, A. T. (1976). Metabolic factors associated with muscular fatigue during aer4obic and anaerobic work. **Canadian Journal of Applied Sports Science, 1,** 43-48.

Westerblad, H., Allen, D. G., Bruton, J. D., Andrade, F. H., & Lannergren, J. (1998). Mechanisms underlying the reduction in isometric force in skeletal muscle fatigue. **Acta Physiologica Scandinavica, 162,** 23-260.

Appendix B: Informed Consent

Your are being invited to participate in a study entitled *The acute neuromuscular response to different resistance training protocols and its role in long-term adaptations* that is being conducted by Jason Brandenburg, Sam Stinson and Aaron Randell. Jason Brandenburg, Sam Stinson and Aaron Randell are graduate students in the department of Physical Education at the University of Victoria and you may contact them if you have further questions by calling 712-8635 (office) or 384-8200 (residence).

As graduate students, this research is part of the requirements for a degree in Sport and Exercise Studies and it is being conducted under the supervision of Dr. David Docherty. You may contact the supervisor at 721-8375.

The objectives of this research project are to: A) measure, compare and define the immediate neuromuscular response (inciuding changes in blood lactate, muscle substrate and degree of muscle activation) following different resistance training protocols designed to optimize muscle (metabolic) fatigue and B) measure and compare the effectiveness of these programs in producing long-term gains in muscle strength and muscle size.

Research of this type is important because although it is known that the neuromuscular system responds to long-term resistance training with increases in muscle strength and muscle size, the specific stimulus for these changes is uncertain. It has been proposed that the neuromuscular fatigue experienced during resistance training may be associated with long-term adaptations. This hypothesis has never been systematically tested. This research project may provide some information to either support or refute this hypothesis.

You are being asked to participate in this study because you have a minimum of one year of previous resistance training experience.

If you agree to voluntarily participate in this research, your participation will include participating in an 8-week resistance-training program. One week prior to the training and one week following the training program you will be expected to undergo an evaluation of acute neuromuscular fatigue. During this time, you will perform 3 different resistance training protocols. Each protocol will be separated by 48 hours. Prior to and immediately upon completion of each of the three training protocols neuromuscular performance of each subject will be assessed. Measurements of neuromuscular performance will include A) maximal isometric force, B) electromyography, C) blood lactate, and D) interpolated twitch technique. Additionally, during one of the protocols you will undergo a muscle biopsy procedure before and after training.

Participation in this study may cause some inconvenience to you, including requiring you to volunteer 3-5 hours of their time each week for 10 weeks. Additionally, you may experience some temporary soreness from the muscle biopsy. Interpolated twitch involves a brief (less than 1 second) electric stimulation superimposed on a maximal voluntary contraction during which you may experience some discomfort.

There are some potential risks to you by participating in this research and they could include temporary pain and infection as a result of the muscle biopsies and finger pricks. You could also experience injury or muscle soreness due to the resistance training program. To prevent or to deal with these risks the following steps will be taken: A) any risks associated with the biopsies will be minimized because a medical doctor will be performing the procedures using stringent sterile techniques, B) a local anesthetic (through injection) will be administered prior to the muscle biopsies in order to reduce the pain that is experienced by you, C) blood samples will be handled following Occupational Health and Safety procedures and D) any risk of injury associated with performing the resistance training will be minimized, as you have previous resistance training experience and because training will be monitored by the investigators. In the unlikely event that an infection or injury should result, you will be asked to withdraw from the study and be directed to the University of Victoria Health Services for medical treatment.

The potential benefits of your participation in this research include an increase in muscle strength and muscle size as a result of performing the resistance training program. You will be introduced to various techniques and procedures that are utilized to assess neuromuscular functioning. Additionally, you will be informed of your muscle fiber type from the muscle biopsy analysis.

Your participation in this research must be completely voluntary. If you do decide to participate, you may withdraw at any time without any consequences or any explanation. If you do withdraw from the study your data will be destroyed at your request. If the data is not destroyed, the data will only be included in the analysis if agreed upon by you.

To make sure that you continue to consent to participate in this research, we will remind you that your participation in this study is voluntary and that you maintain the right to withdraw at anytime without explanation and without penalty.

In terms of protecting your anonymity, you will be assigned a number and all data collected and analyzed will be with reference to that number.

Your confidentiality and the confidentiality of the data will be kept under lock and key and access to the data will be limited to only those investigators mentioned in this form.

Data from this study will be disposed of within 5 years. Data contained on hardcopies will be shredded and any electronic data contained on computer files (disks) will be deleted.

It is anticipated that the results of this study will be shared with others in the following ways: A) dissertation, B) conference presentations, and C) published article(s).

In addition to being able to contact the researcher [and, if applicable, the supervisor] 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 (250-721-7968).

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.

_____ _____

Participant Signature Date

A COPY OF THIS CONSENT WILL BE LEFT WITH YOU, AND A COPY WILL BE TAKEN BY THE RESEARCHER