Time course of muscle hypertrophy, strength, and muscle activation with intense eccentric training

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Abstract

Early strength increase with training is normally attributed to neural adaptations but recent evidence suggests that muscle hypertrophy occurs earlier than previously thought. The purpose of this study was to examine the time course of adaptation through 20 days of training and 5 days of detraining. Twenty-two untrained subjects trained one arm every 2nd day for 20 days. Subjects performed isokinetic eccentric biceps training at 90°/s (6 sets of 8 reps). Muscle thickness (reported in cm) via ultrasound, strength (reported in Nm) and muscle activation (electromyography) were measured before, during and after training (9 time points). Muscle thickness increased after 8 days of training (3.66±0.11 to 3.90±0.12; $p<0.05$) and remained above baseline until the end of training (3.97±0.12). After 5 days of detraining muscle thickness decreased (3.97±0.12 vs. 3.85±0.11; $p<0.05$), but remained higher than baseline ($p<0.05$). Muscle thickness did not change significantly in the untrained arm at any time point. Strength in the trained arm decreased after 8 days of training (65.6±4.1 to 57.5±3.5; $p<0.05$) and remained suppressed throughout the study. Muscle activation amplitude increased after 14 days of training ($p<0.05$) and remained elevated throughout the study. In conclusion, biceps muscle thickness increases very rapidly with frequent intense eccentric training although this type of training appears to impair strength. These findings provide additional evidence that muscle hypertrophy may occur much faster than has been generally accepted.

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Dedication

I dedicate this Master's thesis to my parents. Mom and Dad, thank-you for always being there for me, for always supporting my goals, and for always believing that I could achieve those goals. I would not be here today without you and all that you have done for me. I love you.

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Chapter 1

Scientific Framework

1.1 Introduction

The performance of resistance training normally results in both increased strength and muscle hypertrophy (increased muscle size) (Moritani and deVries, 1979; Colliander and Tesch, 1990; Staron et al, 1994; Higbie et al., 1996; Abe at al., 2000; Aagaard et al., 2001; Farthing and Chilibeck, 2003b; Seynnes et al., 2007). Traditionally, the increase in strength has been thought to precede muscle hypertrophy, and is presumably due almost solely to adaptations of the nervous system (Moritani and deVries, 1979; Seynnes et al., 2007). In particular, these neural adaptations appear to involve alterations in the way the target muscles are activated (Sale, 1988; Narici et al., 1989; Carolan and Caferelli, 1992; Ploutz et al., 1994; Akima et al., 1999; Rabita et al., 2000; Rutherford at al., 2001; Reeves et al., 2005); however, recent evidence suggests that neural adaptations alone may not be the cause of strength increases and that muscle hypertrophy may occur much earlier than previously thought (Seynnes et al., 2007).

Past studies that have attempted to capture the time course of neural versus hypertrophic adaptations have brought forth the idea that muscle hypertrophy is delayed early in training and that strength increases quickly without early morphological adaptation (Ikai and Fukunaga, 1970; Moritani and deVries, 1979; Narici et al., 1989; Abe et al., 2000). In contrast, Seynnes and colleagues (2007) found that after combined eccentric and concentric training, muscle hypertrophy was significant after only 20 days. The mechanistic adaptations necessary for muscle hypertrophy such as increased protein synthesis (Phillips et al., 1997; Moore et al., 2005) and satellite cell activity (Crameri et al., 2004; Dreyer et al., 2006) also occur very quickly after

resistance exercise. These more recent studies suggest that hypertrophy may have the potential to occur very early with intense resistance training.

To date, very few studies have tracked the potential for muscle hypertrophy within 20 days of training. Seynnes et al. (2007) measured muscle size 10 days after training but did not find significant growth. Other studies have neglected to even attempt measurements within the first 2 weeks of training, even though strength increases much quicker (Seynnes et al., 2007). Research into the time course of early adaptations has been limited in other aspects. Past studies have had small participant numbers (Narici et al., 1989; Seynnes et al., 2007), did not provide an optimal training prescription for hypertrophy (Ikai and Fukunaga, 1970; Abe et al., 2000), or used relatively insensitive measurement techniques (Moritani and deVries, 1979). Logically, if all factors that influence muscle hypertrophy are optimized and measurements are made early enough to see early adaptation, it is expected that hypertrophy may occur even faster than the 20 day time point which has been previously reported.

The primary objective of this investigation is to track the time course of early adaptations to training. Muscle hypertrophy, strength, and muscle activation will be measured at several time points within the first 20 days of an intense eccentric training program designed to induce rapid hypertrophic adaptation. This study will provide further insight into the relationship between hypertrophy and neural adaptations in the early stages of resistance training adaptation.

1.2 Review of Literature

1.2.1 Adaptations Early In Training

Skeletal muscle is an adaptable tissue that responds to various forms of tensional stimuli (Toigo and Boutellier, 2006). One specific stimulus that causes skeletal muscle adaptation is

resistance training. This is especially true early in training (in the first 4-5 weeks), when strength increases quickly (Moritani and deVries, 1979; Narici et al., 1989). As mentioned above, it is generally believed that this initial increase in strength is entirely due to neural adaptations although this idea is currently being challenged by studies providing evidence for early muscle hypertrophy (Seynnes et al., 2007) as well as early responses of the mechanisms of hypertrophy (Phillips, 2000; Moore et al., 2005). Still, strong evidence is available to support the idea that a number of neural adaptations are responsible for early strength increases, because strength gain almost always precedes changes in muscle size. Such evidence includes: increased agonist activation, decreased antagonist co-activation, and the phenomenon of cross-education (Sale, 1988; Carolan and Caferelli, 1992; Enoka, 1997; Farthing et al., 2007; Lee and Carroll, 2007). These neural adaptations occur with resistance training and potentially cause increases in strength, even in the absence of morphological adaptation.

1.2.1.1 Neural Adaptations

Increased Agonist Activation

One of the earliest and most prominent neural adaptations with training is increased muscle activation of the agonist or prime mover muscle. Measured via electromyography (EMG) or interpolated twitch, this adaptation has been shown across numerous studies (Sale, 1988; Narici et al., 1989; Ploutz et al., 1994; Akima et al., 1999; Rabita et al., 2000; Rutherford et al., 2001; Reeves et al., 2005; Seynnes et al., 2007). Whereas EMG measures muscle activity by picking up signal from active motor units, interpolated twitch involves delivering a supra-maximal stimulus to the agonist muscle during a maximal voluntary contraction. If the force of contraction increases with the delivery of the stimulus, muscle activation of the agonist muscle is

considered to be incomplete. It has been proposed that increased activation of the agonist may occur as a result of at least two different adaptations (Sale, 1988). Increased agonist activation may be a result of more efficient recruitment of higher threshold motor units (type II motor units) (Sale, 1988) or could also be the result of increased motor unit firing rates (Zehr and Sale, 1994; Sale, 1988; Enoka and Fuglevand 2001).

Increased agonist activation accompanied by increased muscle size may not be strong evidence for solely neural adaptation as increases in muscle size could potentially affect activation. Regardless, studies have shown increased agonist EMG activation in the apparent absence of morphological changes (Ploutz et al., 1994; Akima et al., 1999). The current study will measure agonist muscle activation via surface EMG as an indicator of neural adaptation occurring early in training. As strength is expected to increase rapidly early in training, monitoring changes in agonist activation will provide some insight into how much of this strength adaptation is attributed to increased activation of the agonist muscle.

Decreased Antagonist Activation

Most major muscles exist in an agonist/antagonist relationship in regards to the movement of a specific joint. For this reason another neural adaptation that may be responsible for early strength gain is the decreased activation of antagonist muscles, also known as co-activation (Sale, 1988; Enoka, 1997). Co-activation of the antagonist muscle may produce torque which is in opposition of the voluntary movement, resulting in reduced overall force in the desired direction. Carolan and Caferelli (1992) showed a reduction in knee flexor activation after a period of knee extension training. Specifically, this study noted that the decreased antagonist activation may have been responsible for approximately 33% of the increased force production

in knee extension after one week of training. Conversely, numerous other studies have found no changes in antagonist co-activation after training (Colson et al, 1999; Rutherford et al, 2001). Although theoretically logical, it appears that more research is needed to draw further conclusions on the affects of antagonist activation in regards to neural adaptation. The present study will add to this area by measuring changes in the muscle activation (via surface EMG) of the antagonist muscle (triceps) during training of the elbow flexors.

Cross Education

Further evidence for neural adaptation can be found in the phenomenon known as cross-education. Cross-education is defined as an increase in strength of an untrained limb due to the training of the contralateral limb. This phenomenon, first identified by Scripture and colleagues in 1894, supports the idea that neural adaptation occurs, as in the vast majority of studies strength increase in the untrained limb occurs in the absence of any morphological adaptation. To our knowledge, Brown et al. (1990) is the only study to show evidence of an increase in muscle size (mean fiber cross-sectional area from muscle biopsies) in the untrained limb after unilateral training. Cross-education has now been shown extensively in the literature across various muscle groups, training protocols, and populations (Moritani and deVries, 1979; Farthing et al., 2005; Carroll et al, 2006; Farthing et al., 2007; Lee and Carroll, 2007). In monitoring strength in both arms while only training one arm, the current study will have the capability to observe changes related to cross-education. However, it should be noted that the present study will counter-balance the training of the dominant or non-dominant arm across participants. A study by Farthing and colleagues (2005) found that cross-education occurred more strongly when training the dominant arm than the non-dominant. Thus, the cross-education effect may be blunted in the

current investigation compared to other studies which have specifically trained for cross-education.

1.2.1.2 Muscle Hypertrophy

Muscle hypertrophy is defined as an increase in muscle fiber size that occurs from training. When resistance training occurs, muscle fibers experience small tears or microtrauma. This damage causes protein degradation. After training, protein synthesis is also signaled. In order for hypertrophy to occur, protein synthesis must be greater than protein degradation (Behm, 1995). Factors such as hypertrophy may contribute to early strength increase. Traditionally, muscle hypertrophy is not predicted to occur until after at least 4 weeks of strength training, after which it is the primary contributor to further increases in strength (Moritani and deVries, 1979). However, recent research suggests that muscle hypertrophy might occur earlier than 4 weeks (Seynnes et al., 2007). The goal of this study is to further investigate the time course of hypertrophy early in strength training, which might depend somewhat on the type and speed of contraction used in training, the specific muscle group being trained, and the training prescription.

Muscle Hypertrophy and Contraction Type and Speed

With adequate volume and duration, muscular adaptation occurs with almost all forms of resistance training; however, the type of contraction used in training has an effect on the magnitude of muscle hypertrophy. Regular resistance training consists of combined concentric and eccentric contractions, but the major emphasis of this form of training is on the concentric. When the eccentric portion is emphasized and properly stressed, eccentric contractions have

been shown to be more effective than concentric contractions for inducing muscle hypertrophy (Higbie et al., 1996; Seger et al., 1998; Hortobágyi et al., 1996; Farthing and Chilibeck, 2003b). As well, eccentric contractions have been shown to increase muscle protein synthesis to a greater extent than concentric contractions (Moore et al., 2005). It has long been known that eccentric contractions have the potential to generate greater maximal force than concentric contractions (Levin and Wyman, 1927). This is one possible explanation for why eccentric contractions are the most efficient for increasing muscle size, and this idea has been confirmed in the literature, where training with higher force contractions led to the greatest gains in muscle hypertrophy (Farthing and Chilibeck, 2003b). Additionally, it has been proposed that the fiber tension and mechanical tearing of muscle fibers that occurs with eccentric contractions also plays a role in their ability to generate hypertrophy. This idea is also supported by studies showing that eccentric contractions produce large amounts of muscular damage (Stauber, 1989; Behm, 1995; Enoka, 1996; Stupka et al., 2001, Paddon-Jones et al., 2005) and result in significant muscle soreness (Nosaka and Newton, 2002; Paddon-Jones et al., 2005).

With regards to contraction velocity, eccentric contractions at both fast and slow speeds (30 to 180º/s) are very effective for increasing strength and hypertrophy (Seger et al., 1998; Farthing and Chilibeck, 2003b; Shepstone et al., 2005). High velocity eccentric contractions are the most effective for muscle hypertrophy (Farthing and Chilibeck, 2003b; Shepstone et al., 2005); however, fast eccentric contractions performed on an isokinetic dynamometer are quite unfamiliar and might have an extended learning curve. This may impede maximal exertion early in training by way of agonist muscle inhibition or by co-activation. More complex neuromuscular tasks have a delayed hypertrophy response compared to simpler tasks (Chilibeck et al., 1998). In a study designed to target the time course of early hypertrophic adaptation, full

effort, proper execution, and the potential to maximize early morphological adaptation are important. For these reasons, the present investigation will utilize eccentric training at 90°/second. Medium speed eccentric training will be used as it combines the high tension of faster eccentric contractions and yet will be more easily mastered.

Muscle Hypertrophy and Muscle Group

Resistance training in the upper body results in greater hypertrophy compared to lower body (Wilmore, 1974; Cureton et al., 1988; Chilibeck et al., 1998; Brown et al., 1990; Abe et al., 2000). Brown et al. (1990) had participants train the elbow flexors along with the knee flexors and extensors. The elbow flexors had the largest relative increase (17%) compared to the knee extensors (9.9%) and flexors (4.4%). Elbow flexors muscle thickness increased 22% in young subjects versus only 4% in the quadriceps of the same group after training (Welle et al., 1996). Abe and colleagues (2000) showed the same trend as upper body muscle trained in their study hypertrophied to a greater extent than those of the lower body. A number of mechanisms may explain the greater hypertrophy in upper-body than lower-body muscles. Lower body muscles are frequently used in everyday living (i.e. walking, standing, etc). Thus, these muscles may be habitually activated and have less of a training response than less often used upper body musculature (Cureton et al., 1988). This idea is further supported by Turner and colleagues (1997) who found a 24% increase in biceps cross-sectional area after endurance upper limb arm cycling. In this same study, lower limb cycling had negligible effects on lower limb mass. As noted by Wernbom et al. (2007) in a review, the rate of increase (0.57% per day) reported in the study by Turner et al. (1997) surpasses a large number of resistance training studies that have been performed on other muscle groups, even though the study by Turner et al. (1997) used

endurance training rather than strength training. Another explanation is that muscles of the upper body are more strongly influenced by testosterone as they are reported to have greater androgen receptor content (Kadi et al., 2000). As testosterone is known to have anabolic effects, this may allow upper body muscles to have increased hypertrophic capability.

Based on all of the past findings, the current investigation will use the biceps muscle to investigate the time course of early adaptation to training. In targeting the biceps the current investigation may expect to see an even faster significant hypertrophy response than 20 days (Seynnes et al., 2007). One final note with regards to muscle group is that the biceps is a fusiform muscle group; meaning that its fibers have no angle of pennation. Pennation angle changes have been reported as an additional form of morphological adaptation to training (pennation angle is discussed in more detail below). For fusiform muscles (no pennation), such as the biceps brachii and brachialis, changes in pennation angle are not relevant. Therefore the biceps muscle group provides a good model to examine early hypertrophy with resistance training. Using this model, an increase in strength of the elbow flexors can be attributed to neural adaptations and/or muscle hypertrophy.

Muscle Architecture and Pennation Angle

Along with size and neural proficiency, the geometry of a muscle may play an important role in its force generating characteristics (Fukunaga et al, 2001; Blazevich, 2006; Blazevich et al., 2007). Specifically, as pennation angle increases, the ability of a muscle to produce force increases (Fukunaga et al., 2001). In a recent review, Blazevich (2006) proposed three mechanisms by which increased pennation angle increases force generating capacity. Increased pennation angle is accompanied by: a) increase in physiological cross-sectional area (greater

amount of contractile tissue per a given anatomical area), b) increase in fiber force production due to optimal fiber length, and c) decreased shortening velocity. Controversy exists with regards to the effects of resistance training on muscle pennation angle. A few studies have shown that pennation angle increases after resistance training (Kawakami et al., 1995; Aagard et al., 2001; Seynnes et al., 2007). Conversely, other research has shown that resistance training has no effect on pennation angle (Rutherford and Jones, 1992; Blazevich and Giorgi, 2001; Blazevich et al., 2006). Pennation angle may be closely associated with muscle hypertrophy induced by resistance training (Aagaard et al., 2001). Kawakami et al. (1993) reported that bodybuilders had greater muscle CSA and steeper pennation angles than age-matched sedentary subjects. Thus, it appears that when examining pennate muscles, pennation angle must be considered as a potential mechanism contributing to strength increase with training. The obvious exception to this statement is when examining fusiform muscles such as the biceps, which have no pennation angle.

Muscle Hypertrophy and Training Prescription

When training to induce muscle hypertrophy adequate training volume is needed in order to bring about significant adaptations. Factors that make up the amount of volume in a training program include the number of sets performed per session, number of repetitions performed per set, and the number of sessions performed per week. Also, rest between sets and training sessions should be factored into the interplay of training prescription and muscle hypertrophy.

The greatest gains in hypertrophy have been shown when utilizing multiple sets as compared to single sets in exercise training (Kraemer, 1997; Kraemer et al., 2000). A meta-analysis performed by Wernbom and colleagues (2007) noted that specific to biceps training, 4-6

sets seem to produce the most muscle growth per day. Fleck and Kraemer (1997) have specific recommendations on the number of repetitions that promote maximal muscular hypertrophy. They state that a repetition range of 8-12 is optimal for muscle growth. Similarly, Chandler and Brown (2008) also advocate that 8-12 repetitions is the best range for hypertrophy. These suggested workout volumes have also been acknowledged in a recent review of various training stimuli and increased muscle cross-sectional area (Wernbom et al., 2007). This review noted that moderate volumes of work (30-60 repetitions per session) yielded the largest responses in muscle growth.

One final area to consider with regards to training volume is sessions per week. Wernbom et al. (2007) noted that with studies training the biceps, the average number of sessions per week was just fewer than 3. But, Wernbom and colleagues (2007) also noted that the one study that prescribed 4 days per week of training showed the highest rate of growth per day of any of the studies included in the review. This suggests that as long as adequate recovery is given to avoid overtraining, more training sessions may result in greater hypertrophy as the muscle may be broken down and built up more often.

In a recent review Willardson (2006) suggests that for optimizing hypertrophy rest between sets must remain short enough so that full muscle recovery cannot be achieved. With this in mind he suggests a rest period of 30-60 seconds between sets. Similarly, Fleck and Kraemer (1997) suggest short rest periods of 1-2 minutes between sets for hypertrophy specific training.

Based on the information provided above, the current investigation will employ a training program that includes 6 sets of 8 repetitions per set. Rest between sets will be one minute in length. Training sessions will occur every 2nd day for 20 days in a row. This training

frequency will allow 48 hours for full protein synthesis to occur (Phillips et al., 1997) while also utilizing the greatest number of sessions that may be performed thus maximizing the potential for early hypertrophic adaptation. One final note on training prescription is that the above recommendations are based on conventional weight training and not isokinetic eccentric training. Little is known about the optimal training prescription for isokinetic eccentric training, but this study will utilize the current recommendations for conventional weight training.

Muscle Hypertrophy and Sex Differences

Absolute changes in muscle size and strength are larger in men (Cureton et al., 1988) but this is most likely because males have larger muscle to begin with (Cureton et al., 1988; Davies et al., 1988; Abe et al., 2000). Specific studies have provided evidence that sex differences do exist (Delmonico et al., 2005; Hubal et al., 2005). Hubal and colleagues (2005) showed that with upper-body training, males had greater muscle size increases but greater strength increases were shown in women. The idea that strength may increase more in women is also supported by O' Hagan et al. (1995) and by Delmonico et al. (2005) who proposed the idea that women may have a greater potential for neural adaptation. Relative responses to resistance training have been shown to be similar between men and women (Cureton et al., 1988; Davies et al., 1988, O' Hagan et al., 1995).

With regards to the time course of muscle hypertrophy, research shows there are similar relative increases across sexes. Abe et al. (2000) showed similar relative increases in biceps muscle thickness after 4, 6 and 8 weeks of training. This study also showed similar muscle size trends across weeks for other muscle groups trained. Similar results were reported by Staron et al. (1994) who found a similar time course of changes across sexes when training the lower

body. The current investigation will include both males and females. Since the study employs a within subjects design, this controls for the confounding effect of baseline differences between subjects. Despite the fact that some studies have shown different responses across sexes, the inclusion of both sexes will also improve the generalizability of the results of this study, providing a better perspective on the adaptations that occur early in training. This is significant considering that of the few time course studies to date, two have included males only (Ikai and Fukunaga, 1970; Narici et al., 1989) and another used a majority of males (Seynnes et al., 2007).

1.2.2 Time Course of Muscle Hypertrophy

Effective strategies for inducing muscle hypertrophy have been studied extensively (Moritani and deVries, 1979; Colliander and Tesch, 1990; Staron et al., 1994; Higbie et al., 1996; Abe at al., 2000; Aagaard et al., 2001; Farthing and Chilibeck, 2003b; Seynnes et al., 2007). Interestingly though, researchers have generally accepted the idea that hypertrophy does not occur early on in training (Moritani and deVries, 1979; Sale, 1988; Seynnes et al., 2007). Very few studies have investigated the time course of muscle hypertrophy during resistance training, and these are described in detail below.

One of the first studies to investigate the time course of strength and muscle growth was performed by Ikai and Fukunaga (1970). Training and strength measurements were performed on an arm dynamometer and muscle size was measured using ultrasound pictures in which pre and post pictures were overlapped and the difference distances were calculated accordingly. Training consisted of 10 second isometric contractions of the arm flexors 3 times per day, 6 days a week, for 100 days. Measurements were made at days 20, 40, 60, and 100. Strength (as percentage change) increased significantly after 20 days and cross-sectional area (also expressed as percent

change) increased significantly after 40 days. After 20 days, cross-sectional area was 8.2% higher than pre-training but this was not significant, likely due to low subject number. This study was one of the first to introduce the idea that neural adaptations precede hypertrophic adaptations. When analyzed closely, this study also shows the potential for early hypertrophic adaptation.

Moritani and deVries (1979) investigated the neural versus hypertrophy time course every 2 weeks during an 8-week training study. Seven men and 8 women performed 10 repetitions 2 times per day, 3 days per week at an intensity of two thirds of their one repetition maximum (1RM). After measuring isometric strength, muscle activation and muscle size they found that neural factors accounted for approximately 80 percent of the increase in strength after two weeks. After four weeks they reported that neural factors still accounted for approximately 60 percent of strength increase, whereas hypertrophy became the dominant factor for strength increase somewhere between 3-5 weeks into training. This reported onset of hypertrophy was very early considering the relatively insensitive measure of hypertrophy used in this study (elbow flexors girth corrected for skinfolds).

In 1989, Narici and colleagues investigated time course of training adaptation in the quadriceps muscle of 4 male subjects between the ages of 23-34. They measured strength along with muscle size and neural activation every 20 days for 60 days of training and for 40 days of detraining. Training consisted of 6 sets of 10 maximum isokinetic knee extensions at a speed of approximately 120º/s. Cross sectional area did not significantly increase until after 60 days. They concluded that hypertrophy as a result of strength training accounted for approximately 40 percent of the observed strength gain while the remaining 60% could be accounted for by either increased neural drive or muscle architecture changes.

More recent studies provide evidence for earlier morphological change during training. Abe and colleagues (2000) studied time course of muscle thickness and strength after upper and lower body resistance training. Over a 12-week training period, strength and hypertrophy were examined every 2 weeks. Participants in this study performed 6 different exercises 3 times per week at 60–70% of one repetitious maximum (1-RM). One important factor to note with their study was that some participants trained with 3 sets per exercise while others only used 1 set. Muscle thickness was measured using B-mode ultrasound. Results of this study showed increased muscle thickness in both men and women across a number of muscle groups after 6 weeks, and a significant increase in biceps thickness in males after only 4 weeks. This study generally concluded that increased muscle thickness was not present within the first 4 weeks of training but did acknowledge that the time course of hypertrophy is still unclear. Notably, they found a non-significant trend for an increase in muscle thickness of the quadriceps after only 2 weeks. This finding is in line with studies that have investigated early muscle adaptation using muscle biopsies to measure muscle fiber cross-sectional area (CSA). Staron and colleagues (1994) found that trends for increased muscle fiber CSA could be observed after 2 weeks of training (approximately 5% increase in fiber area). Using successive biopsies every 2 weeks for 8 weeks of training Staron et al. (1994) eventually found fiber area to be increased by over 15% in type IIa and IIb fibers after 8 weeks of training, although this was not statistically significant. Unfortunately, this study was likely underpowered due to the high variability and low sample size that often accompanies muscle biopsy research.

The most recent study of the interplay between morphology and strength early in training was conducted by Seynnes and colleagues (2007). Using a unique gravity independent flywheel ergometer which combined both concentric and eccentric training, 7 participants trained their

quadriceps 3 times per week. Magnetic resonance imaging (MRI) was used for measuring total muscle CSA. Testing time points were investigated after 10, 20, and 35 days of training. The major claim of their study was that they found the earliest onset of significant muscle hypertrophy ever reported in humans, after only 20 days. Further, they state that with the size increase reported within 20 days, a rate of approximately 0.2% per day growth was occurring. This study measured muscle hypertrophy earlier, and more frequently than previous studies, but still had no measurement between days 10 and 20, leaving unanswered the question of exactly how early muscle hypertrophy occurs. Still, this study provided very strong evidence that hypertrophy occurs much faster than many physiologists previously believed.

1.2.3 Mechanistic Responses and Muscle Hypertrophy

It has been suggested that there are two fundamental adaptations necessary for muscle hypertrophy; increased protein synthesis and satellite cell proliferation (Seynnes et al., 2007). Protein synthesis is significantly increased after resistance training (Phillips et al., 1997; Moore et al., 2005). With training protein degradation also occurs and in order for hypertrophy to occur, protein synthesis must exceed protein degradation, causing a net increase in protein content (Behm, 1995). Additionally, it is believed that satellite cells are essential for increased muscle growth (Rosenblatt et al., 1994; Phelan and Gonyea, 1997). This may be linked to the myonuclear domain theory which suggests that a myonucleus of a muscle cell controls the production of mRNA and protein for a finite portion of cytoplasm in a muscle cell. Hence, for a muscle fiber to expand in size it must also add more myonuclei to maintain the myonuclei to cytoplasmic volume ratio (Cheek, 1985; Hawke, 2005). With exercise and muscle damage

satellite cells are activated and fuse to myofibers, donating new myonuclei and allowing muscle fibers to increase in size.

The exact mechanisms that lead to activation of satellite cells are not fully understood. Toigo and Boutellier (2006) discuss four potential ways that satellite cells may potentially become activated. They propose that activation may be signaled by: 1) anabolic cytokines which are present because of extracellular damage, 2) other infiltrating cells involved in combating the inflammatory process, 3) by the myofibers themselves, or 4) by other satellite cells themselves. Regardless of the way they are specifically activated, it is satellite cells that play a major part in the repair of damaged muscle fibers and subsequently are essential for the promotion of optimal muscle hypertrophy.

Research pertaining to protein synthesis and satellite cell proliferation also supports the possibility of hypertrophic adaptation very early in the time course of training. This is in line with the previously discussed recent evidence suggesting early hypertrophic adaptation at the whole muscle level may occur very early in training (Seynnes et al., 2007). Mixed muscle protein synthesis may increase as early as 3 hours after resistance exercise and remains significantly elevated for up to 48 hours (Phillips et al., 1997). Specifically, myofibrillar protein synthesis is increased as early as 4.5 hours after resistance training (Moore et al., 2005) and significant increases in total myofibrillar protein content may occur after just 1-3 training sessions (Phillips, 2000; Willoughby and Taylor, 2004). Transcription and translation of mRNA and other adaptive responses occurs even sooner, within as little as hours or even minutes after exercise commencement (Bickel et al., 1998; Staron et al., 1994; Haddad and Adams, 2002).

Similarly, it has been suggested that satellite cells are involved in the hypertrophy and muscle repair process very early after training in both humans (Crameri et al., 2004; Dreyer et

al., 2006) and in animal models (Adams et al., 1999; Parise et al., 2008). Dreyer and colleagues (2006) reported that satellite cell recruitment was significantly elevated in both younger (141%) and older adults (51%) 24 hours after an acute bout of eccentric contractions. Crameri et al. (2004) also found that satellite cells can be caused to re-enter the growth cycle after just one session of high intensity exercise. More research is needed to draw strong conclusions of the time course of satellite cell proliferation and differentiation. However, as outlined above, several studies do support the idea that muscle satellite cell adaptation also occurs very early after training, and it appears the mechanistic adaptations associated with hypertrophy occur rapidly after resistance exercise. This research provides evidence that the potential for early whole muscle hypertrophy is supported by the early cellular and molecular adaptations occurring at the site of muscular adaptation.

1.3 Statement of the Problem and Hypotheses

1.3.1 Statement of the Problem

A limited number of studies have focused on the time course of muscle hypertrophy and in many cases they were not designed or equipped to be able to truly detect the early changes that may occur with training. Past studies have used less sensitive measurement techniques, have been statistically underpowered, and utilized less than optimal training protocols. Recent research, which identified muscle hypertrophy with 3 weeks of training, made no measurements between days 10 and 20 of training and trained the quadriceps muscle (known to show a blunted hypertrophy response compared to the biceps). The purpose of this investigation will be to track the time course of muscle hypertrophy within the first 20 days of training. This study will attempt to use an effective muscle group and training protocol for hypertrophy, as well as a larger sample size in order to attempt to reveal the true potential for early muscle size increases during training.

1.3.2 Hypotheses

1. The primary hypothesis of this study is that muscle hypertrophy will occur at a time point sooner (prior to 20 days) than has been previously reported in the literature (Seynnes et al., 2007). This hypothesis challenges the idea that early strength increase is mediated solely by neural adaptation.
2. A secondary hypothesis is that strength and agonist muscle activation (measured via EMG) will increase significantly with training, and antagonist muscle activation will decrease significantly with training, similar to the findings of past research (Moritani and deVries, 1979; Carolan and Cafarelli, 1992; Abe at al., 2000).

Chapter 2

Methods

2.1 Study Design

This study used a within-subjects design consisting of two phases; a 20-day baseline phase followed by a 20-day training phase. The baseline phase was used to allow participants to serve as their own non-training controls and to show the stability of the measures across our sample over time. Strength tests, muscle thickness measures, and muscle activation assessment were performed at 9 time points: before the 20 day baseline phase, at days 0, 8, 12, 14, 16, 18, and 20 during the training period, and after 5 days of detraining (See Figure 2.1 for an illustration of the design). Day 0 corresponded to the end of the baseline period and the beginning of the training period. All muscle thickness measures were taken immediately before the training session on the specified day according to the measurement and testing schedule (See Appendix A for a sample calendar). This allowed testing and measurement of fully rested and recovered muscles. During strength testing, neuromuscular activity was measured via EMG in order to monitor changes in neural activation with progressive training.

The resistance training phase consisted of intense unilateral eccentric (lengthening) contractions of the elbow flexors every 2nd day during the training phase (designated training arm was counter-balanced for arm dominance across participants). During training, muscle thickness was measured via ultrasound on both the trained and untrained arms. This allowed subjects to serve as their own controls for the muscle thickness measures. Strength and muscle activation

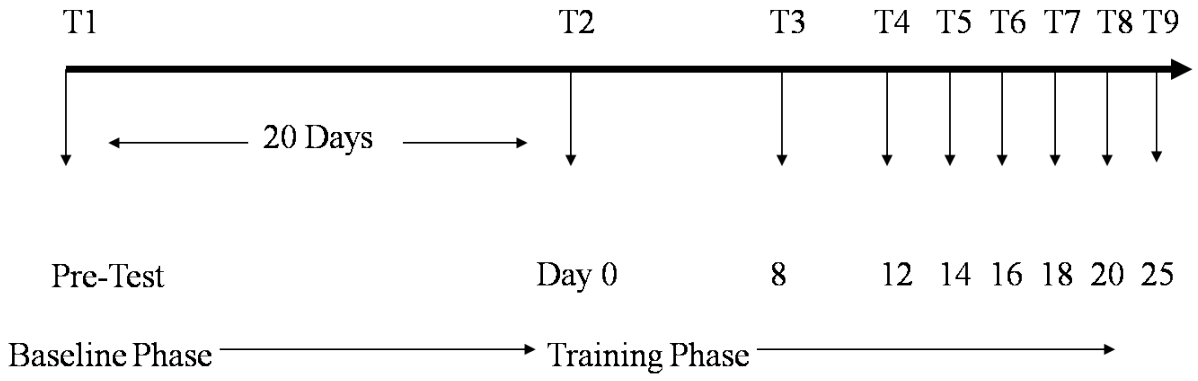

Figure 2.1 Study Design Timeline

were measured only on the training arm during the training phase. The untrained arm could not be used as a within-subjects control comparison for the trained arm due to the effect of cross-education with unilateral training (Moritani and deVries, 1979; Farthing et al., 2005; Carroll et al., 2006; Lee and Carroll, 2007). As well, testing of strength on the untrained arm might have caused morphological adaptation that could have compromised the validity of using the untrained arm as a control for hypertrophy.

2.2 Participants

Prior to the start of participant recruitment ethical approval was obtained from the University of Saskatchewan biomedical ethics board (See Appendix B for a copy of the Ethics: Certificate of Approval). Power calculations were computed using effect sizes for strength and muscle thickness measures from recent unilateral strength training studies performed by in the lab where the current study was performed (Farthing and Chilibeck, 2003b; Krentz et al., 2008). To achieve 80% power at $\alpha = 0.05$ with the repeated measures design described above, it was determined that approximately 20 participants were required (nQuery Advisor, version 3.0, Statistical Solutions, Cork, Ireland).

Twenty-three untrained participants (12 male /11 female) between the ages of 19-31 originally volunteered to participate in the study. One female participant was required to withdraw from the study due to consecutive missed training sessions. Thus, twenty-two untrained participants (12 male /10 female) completed the study. For the purpose of this study untrained was defined as both not currently training as well as having minimal previous training experience (See Table 2.1 for lifetime training experience of participants). Both males and females were included to increase the generalizability of the study. As well, past research has

shown that males and females have similar time course of adaptation (Cureton et al., 1988; Staron et al., 1994; Abe et al., 2000). Participants were recruited mainly from the University of Saskatchewan community through classroom presentations, posters, and word of mouth. Prior to beginning the study all subjects gave informed written consent (See Appendix C for copy of consent form). Participants were not allowed to participate in the study if they were currently performing regular resistance training. All participants were required to refrain from training of the biceps outside of the supervised training for the duration of the study. Participants were encouraged to maintain their normal diet. Participant characteristics are displayed in Table 2.1.

Table 2.1- Participant Characteristics

Data listed as Means ± Standard Error.

Age (Years)	21.4 ± 0.6
Height (cm)	171.4 ± 1.8
Pre-Training Weight (kg)	69.8 ± 2.3
Post-Training Weight (kg)	69.9 ± 2.3
Lifetime Resistance Training Experience (months)	7.4 ± 2.3

2.3 Procedures

2.3.1 Test Protocol

Participants were tested at 9 time points over the course of 45 days of baseline, training, and detraining. Prior to any initial testing at baseline, age and handedness were self-reported. Each participant's past resistance training experience was determined via a resistance training experience questionnaire (See Appendix D Resistance Training Experience Questionnaire). One month of resistance training experience was defined as training three days per week for an entire month (4 weeks). Participants were also asked about any current supplements or medications they were taking which could influence their response to resistance training. No participants were currently taking any performance enhancing supplements (i.e. creatine, protein, etc). Prior to the start of training at Day 0, each participant's height and weight was recorded. At the end of training each participant's weight was again recorded in order to account for any possible weight fluctuations over the training phase.

2.3.2 Muscle Thickness

Muscle thickness was measured in both arms at baseline and on days 0, 8, 12, 14, 16, 18, 20, and 25. Muscle thickness measurements always preceded strength and muscle activation measures. Confounding effects of testing order were controlled by counter-balancing testing arm order across participants. The coefficient of variation for muscle thickness in this study was 2.14%. Thickness was measured using B-mode ultrasound (Aloka SSD-500, Tokyo, Japan**)** according to previous methods found reliable in the lab where the current study took place (Farthing and Chilibeck, 2003a; Farthing et al., 2005; Candow et al., 2006; Krentz et al., 2007).

Muscle thickness measured via ultrasound has been shown to be a valid measure of muscle size (Miyatani et al., 2000; Miyatani et al., 2002; Sanada et al., 2005). Muscle thickness has been shown to significantly predict muscle volume of the upper arm via MRI (r =0.96) (Miyatani et al., 2000) and the knee extensors (r = 0.91) (Miyatani et al., 2000). As well, Sanada and colleagues (2005) found strong correlations between muscle thickness via ultrasound and MRI measurements in a several muscles (including the arm, trunk, body, thigh, and lower leg) across 72 subjects.

In the present study, thickness was measured on the bulk of the biceps, approximately two thirds of the way distally down the arm between the acromion process of the shoulder and the olecranon of the elbow. Once this point was established a detailed land marking procedure (using overhead transparencies) was employed to ensure exact placement of the ultrasound probe for each subsequent measurement time point (Farthing and Chilibeck, 2003a). During the training phase, once the measurement site had been established it was marked with permanent marker and continually retraced to ensure precise land-marking from measure to measure throughout the study.

Positioning of participants during muscle thickness measures remained constant for all time points. Participants were instructed to lay their arms as flat as possible on a table so that their arms were parallel to the table with their triceps resting. Participants were also instructed to fully relax their biceps before and during measurements.

Four measurements were taken on each arm and the average of the two closest values was used as the thickness value. To ensure precision, a fifth measurement was taken when two pairs of values were equidistant apart or if all values were equal to or greater than 1mm apart.

2.3.2 Strength

Strength was assessed at baseline and at days 0, 8, 12, 14, 16, 18, 20, and 25 using an isokinetic dynamometer (Humac Norm, CSMi, Stoughton, MA). Use of isokinetic dynamometry allowed precise control of the contraction type and velocity used, while accurately measuring torque production. The coefficient of variation for strength was 6.1%. Unilateral strength of each arm was assessed at the beginning and end of the baseline phase (baseline and day 0) and at the end of the training and detraining phases (day 20 and day 25). Participants performed 4 maximum unilateral repetitions each separated with 1 minute rest and the peak repetition was used for comparison. Before the start of the testing repetitions, participants were familiarized with the movement until they felt comfortable enough to perform maximal contractions and the primary researcher was satisfied they could safely and successfully exert maximal effort. Throughout the training period of the study (days 8, 12, 14, 16, 18), strength was only assessed on the training arm and was recorded while participants performed their regular training protocol. The highest value of eccentric torque during the first training set was used as that testing day's peak torque value.

Strength was assessed using medium speed eccentric (90 º/s, 1.57 rad/s) contractions of the elbow flexors performed on the Humac Norm isokinetic dynamometer. Testing strength with these specific contractions was done in order to remain consistent with the type of training performed during the study. During testing, range of motion was set at 110°. The lengthened position was set so that the participant's arm was just above the fully straightened position at the elbow joint. Participants were seated in a reclined position with their backs supported at approximately 60° from supine. Participants' feet were placed against a metal support attached to the seat. Dynamometer chair settings were recorded at the start of the study and remained

consistent throughout testing and training for each participant. This allowed the dynamometer positioning and comfort to be controlled across the study.

During testing, participants were allowed access to their test scores for each repetition. Participants were instructed to try their best for each repetition and encouragement was provided by the researcher throughout the repetition.

2.3.3 Muscle Activation

A four-lead EMG system (Bagnoli-4, Delsys Inc., Boston, MA) was used to assess activation of the biceps and triceps. Prior to positioning the electrodes, the skin was prepared by shaving and cleaning the area with alcohol to reduce skin impedance. The coefficient of variation for a maximally activated muscle for this measure was 20.04%. The EMG main amplifier unit included single differential electrodes with a bandwidth of 20 ±5 Hz to 450 ±50 Hz, a 12 dB/octave cutoff slope, and a maximum output voltage frequency range ± 5 V. The overall amplification or gain per channel was 1K. The system noise was <1.2 μV rms for the specified bandwidth. The electrodes were two silver bars (10 X 1mm diameter) spaced 10mm apart, with a common mode rejection ratio (CMRR) of 92 dB.

Muscle activation was assessed at baseline and at days 0, 8, 12, 14, 16, 18, 20 and 25 via EMG. Both arms were assessed at the beginning and end of the baseline phase (baseline and day 0) and at the end of the training and detraining phases (day 20 and day 25). Consistent with strength measures, only the training arm was assessed on days 8, 12, 14, 16, and 18. Activation was measured on the agonist (biceps) and antagonist (triceps) muscles. For the biceps the electrode was placed in the middle of the marked area where muscle ultrasound was measured. For the triceps, the electrode was placed on the bulk of the muscle and on the midline of the

segment, approximately 1/3 of the distance down from the acromium process to the olecranon process. Muscle activation was measured on all repetitions of the testing protocol and on the entire first set of training during the training protocol. EMG data from the repetition with the highest peak torque was used for comparison. The land marking scheme used to ensure accurate ultrasound land marking was also applied and used for EMG placement. A reference electrode was applied to the kneecap and served as a common ground for the signal. Raw data was collected in volts and later converted to root mean squared (RMS) using the accompanying computer software (EMGworks, version 3.1) in order to determine the amplitude of activation. RMS is mathematically defined as the square root of the mean of a number of squared values. Thus in this case the RMS is the square root of the mean of the squares for a specified window length (0.125s) of raw values acquired by the EMG electrode.

2.3.4 Muscle and Joint Soreness

All participants were asked to complete a recall soreness questionnaire at the end of the study in order to obtain information about the occurrence and magnitude of soreness experienced during the study. Participants were instructed to indicate which of the listed sites (biceps, elbow, forearm, shoulder, hand/wrist) they experienced soreness in as a result of training. They were then instructed to rate the magnitude of this soreness across each of the three weeks on a scale of 0-9 with 0 being no soreness and 9 being intense soreness. This soreness ratings scale was used by Krentz and colleagues (2008) in a study investigating the effects of ibuprofen and intense training. As well, Krentz and colleagues had participants give a rating of biceps soreness daily, but then pooled the soreness changes across weeks. The soreness ratings questionnaire used in this study was based on that method.

2.3.5 Unilateral Training Program

The training program consisted of medium speed unilateral eccentric (90 º/s, 1.57 rad/s) contractions of the elbow flexors performed on the Humac Norm isokinetic dynamometer every second day for 20 days. Participants were counter-balanced to train either their dominant or non-dominant arm. Eccentric contractions at fast, medium and slow speeds (30, 90, and 180º/s) [0.52, 1.57, 3.14 rad/s] are very effective for increasing strength and hypertrophy (Seger et al., 1998, Farthing and Chilibeck, 2003b); however, fast eccentric contractions performed on an isokinetic dynamometer and are quite novel and have an extended learning curve, potentially impeding maximal exertion early in training. They also elicit more cross-education than slow eccentric contractions (Farthing and Chilibeck, 2003a). For this study full effort and proper execution were crucial from the start of training, due to the short duration of the study and the early measurement time points. For these reasons, medium speed eccentric contractions were used, as they combine the high tension of faster eccentric contractions and were more easily mastered than faster contractions.

A progressive overload design was utilized in which subjects started with 3 sets of 8 contractions on their first training sessions. This progression was continued by adding one set to each training session until participants reached 6 sets. At this point no more sets were added. Rest between sets was one minute in length. Positioning, range of motion, and encouragement was kept consistent for training as previously described for the strength testing protocol.

2.3.6 Statistical Analyses

All data analysis was performed with SPSS, version 15.0 for Windows. To determine if there were differences in muscle thickness, strength, and muscle activation over the baseline phase three analyses were performed. These analyses were conducted with the purpose of evaluating the stability of the measures during the control interval. Muscle thickness and strength were each tested using a 2 x 2 repeated measures ANOVA with factors of arm (trained and untrained) and time (baseline and Day 0). Any changes in muscle activation through the baseline phase were tested for with a 2 x 2 x 2 repeated measures ANOVA with factors of arm (trained and untrained), time (baseline and Day 0), and muscle (biceps and triceps).

After baseline analyses were completed, muscle thickness (baseline and training phase) was analyzed using a 2 x 9 repeated measures ANOVA with factors of arm (trained and untrained) and time (9 levels). Strength for the trained arm was analyzed using a one factor (Time: 9 levels) repeated measures ANOVA. Strength for the untrained control arm was analyzed using a one factor (Time: 4 levels) repeated measures ANOVA. Muscle activation for the trained arm was analyzed using a 2 x 9 repeated measures ANOVA with factors of muscle (biceps and triceps) and time (9 levels of time). Muscle activation for the untrained control arm was analyzed using a 2 x 4 repeated measures ANOVA with factors of muscle (biceps and triceps) and time (4 levels). The dependent variables of strength, muscle activation, and muscle thickness were analyzed separately because of their different designs (i.e. ARM factor the for muscle thickness data, MUSCLE factor [biceps, triceps] for muscle activation data, and different levels of time for each arm). In addition, a separate analysis for each variable was desirable for this study because it allowed close examination of the independent changes in each variable early in strength training. Biceps muscle soreness was analyzed using a one factor (Time: 3 levels) repeated measures ANOVA and joint soreness was expressed as frequencies. Simple main

effects and post hoc multiple comparisons with Bonferroni adjustment were performed when appropriate. Significance was set at $\alpha<0.05$. All values are expressed as means ± standard error.

CHAPTER 3

RESULTS

3.1 Muscle Thickness

There were no significant differences between the trained and untrained arm at baseline or prior to the start of training, $F(1, 21)=0.564$, $p>0.05$. There were also no significant differences for muscle thickness over time between baseline and Day 0, $F(1, 21)=0.741$, $p>0.05$. For the complete data set (baseline and training phases), there was a significant arm by time interaction, Greenhouse-Geisser (GG) adjusted $F(5.3, 111.4)=57.714$, $p<0.001$. Simple main effects analysis revealed a significant time main effect for the trained arm, GG $F(4.3, 89.5)=64.546$, $p<0.001$. Post hoc multiple comparisons (Bonferroni adjusted) revealed that muscle thickness significantly increased after 8 days of training (Day 0: 3.66±0.11 to Day 8: 3.90±0.12; $p<0.05$) (Refer to Figure 3.1 for a graph of Muscle Thickness changes for the trained and untrained arms). There was a trend for further muscle thickness increase from Day 12 to Day 16 (3.92±0.12 to 3.98±0.11; $p=0.08$). Muscle thickness remained significantly higher than Day 0 for all time points until the end of training (Day 20: 3.97±0.12; $p<0.05$). After 5 days of detraining muscle thickness significantly decreased (3.97±0.12 vs. 3.85±0.10; $p<0.05$), but remained higher than Day 0 ($p<0.05$) as well as significantly higher than the untrained arm ($p<0.001$). There were no significant changes in muscle thickness in the untrained arm at any time points through the training phase, GG $F(5.6,116.9)=1.725$, $p>0.05$. Refer to Appendix E for the statistical output tables for the analysis of the muscle thickness data.

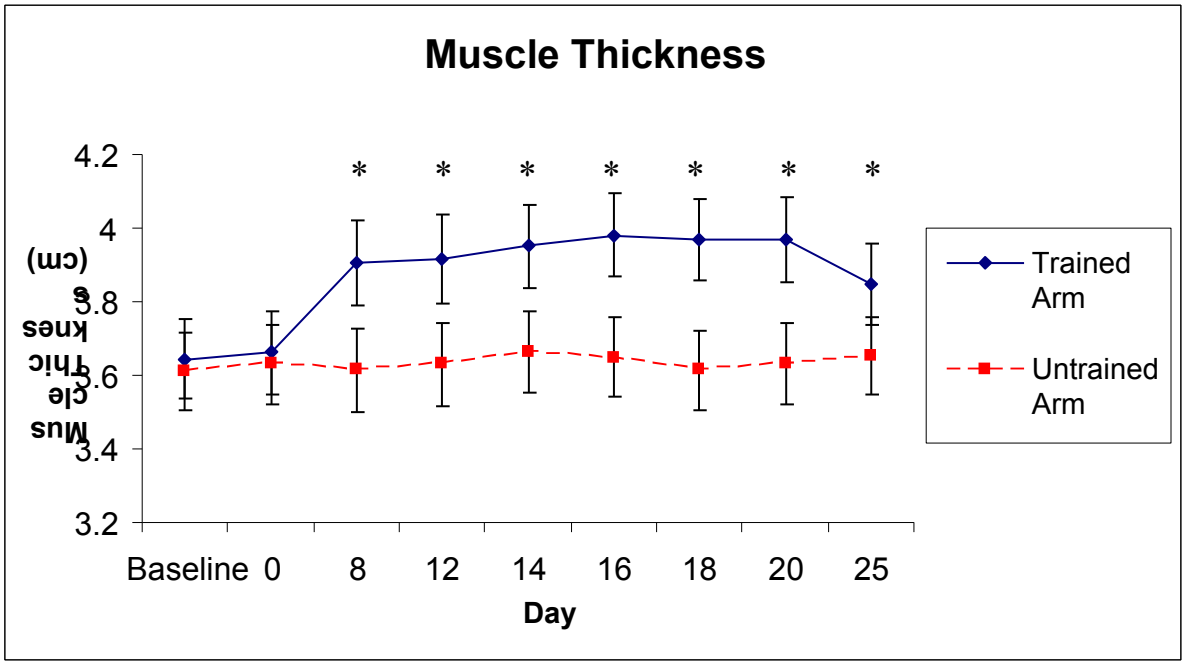

Figure 3.1: Muscle Thickness. Values are expressed as means ± standard error.
* Indicates time points are significantly different than Day 0 for the trained arm; $p < 0.01$. Bonferroni adjusted.
** Indicates time point is significantly less than Day 20 for the trained arm; $p < 0.01$. Bonferroni adjusted.

3.2 Strength

There were no significant differences between the trained and untrained arms prior to baseline or at the start of training, $F(1, 21)=0.183$, $p>0.05$. There were no significant differences for strength between baseline and Day 0 for either the trained or untrained arm, $F(1, 21)=0.403$, $p>0.05$. As previously discussed, different designs were used for the trained and untrained arm. The trained arm had 9 measurement time points (baseline and training phase) while the untrained arm has only 4 (baseline and training phase). There was a significant main effect of time in the trained arm, GG $F(4.5, 94.5)=16.179$, $p<0.001$. Post hoc multiple comparisons (Bonferroni adjusted) revealed strength in the training arm decreased after 8 days of training (Day 0: 65.6±4.1 to Day 8: 57.5±3.5; $p<0.05$) and remained suppressed throughout the study. There were no significant changes in strength in the untrained arm at any time points throughout the study, GG $F(1.5, 32.5)=1.516$, $p>0.05$. Refer to figures 3.2 and 3.3. See Appendix E for the statistical output tables for the analysis of the strength data.

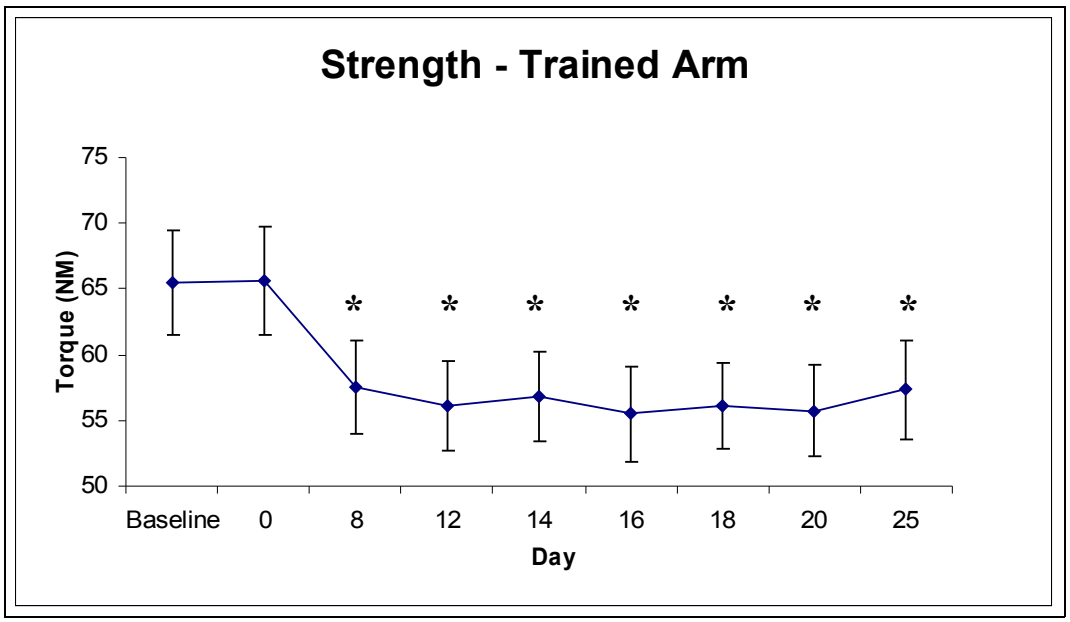

Figure 3.2 Strength – Trained Arm. Values are expressed as means ± standard error.
* Indicates time points are significantly different than Day 0; $p < 0.01$. Bonferroni adjusted.

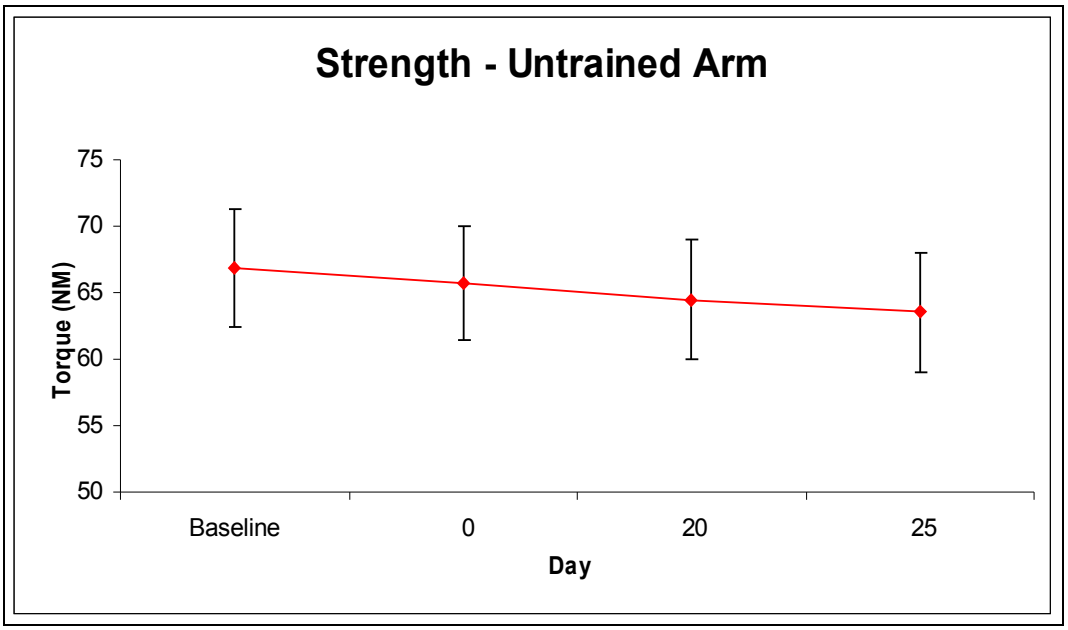

Figure 3.3 Strength – Untrained Arm Values are expressed as means ± standard error.

3.3 Muscle Activation

There were no significant differences for muscle activation of the biceps or triceps between the trained and untrained arms prior to baseline or at the start of training, $F(1, 21)=0.176$, $p>0.05$. There were no significant differences for muscle activation between baseline and Day 0 for either the trained or untrained arm, $F(1, 21)=0.079$, $p>0.05$. As mentioned previously, separate analyses were necessary for the trained and untrained arm for the complete data set (baseline and training phase). There was a significant muscle by time interaction for the trained arm, GG $F(4.8, 101.5)=4.273$, $p<0.01$. There was a significant main effect of time for the biceps of the trained arm, GG $F(4.8, 100.1)=3.569$, $p<0.01$. Biceps muscle activation in the training arm increased after 14 days of training (Day 0: 0.781 ± 0.069 to Day 14: 0.934 ± 0.088mV; $p<0.05$) and was still elevated at the end of the study (Day 25: 0.894 ± 0.074 mV; $p<0.05$). There was a significant main effect of time in the triceps of the trained arm, GG $F(3.2, 68.1)=3.431$, $p<0.05$. Triceps muscle activation was significantly reduced at the end of training (Day 0: 0.067 ± 0.006 to Day 20: 0.045 ± 0.003 mV; $p<0.05$) There were no significant changes over time in muscle activation of either the biceps or triceps in the untrained arm, GG $F(2.4, 50.4)=0.359$, $p>0.05$. Refer to figures 3.4, 3.5, 3.6 and 3.7 below. See Appendix E for the statistical output tables for the analysis of the EMG data.

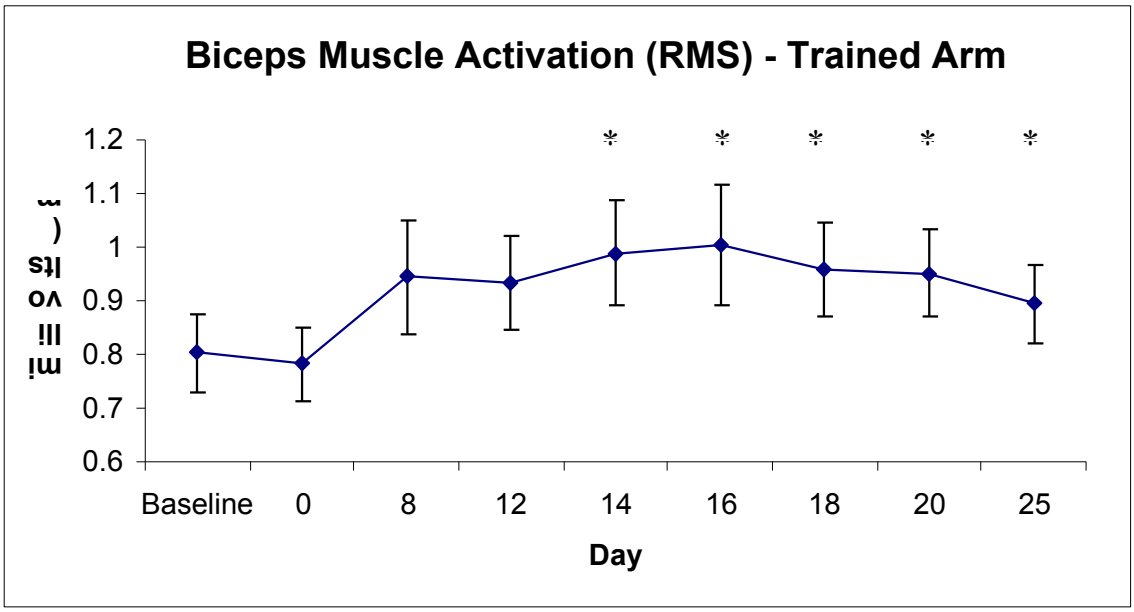

Figure 3.4 Muscle Activation – Biceps Trained Arm. Values are expressed as means ± standard error.

* Indicates time points are significantly different than Day 0; $p < 0.01$. Bonferroni adjusted.

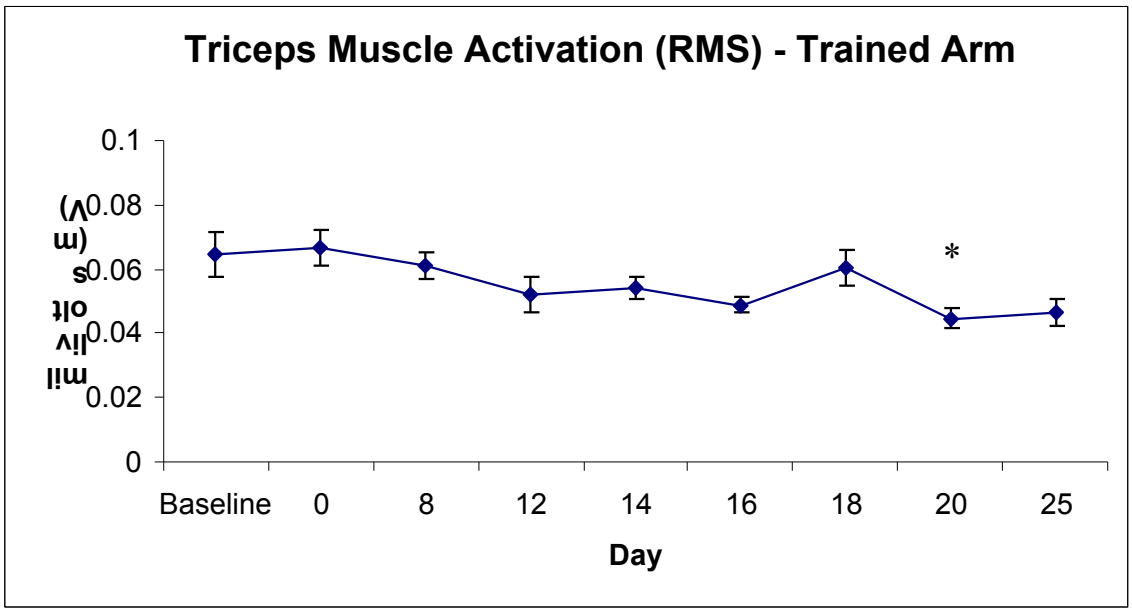

Figure 3.5 Muscle Activation – Triceps Trained Arm. Values are expressed as means ± standard error.

* Indicates time point is significantly different than Day 0; $p < 0.01$. Bonferroni adjusted.

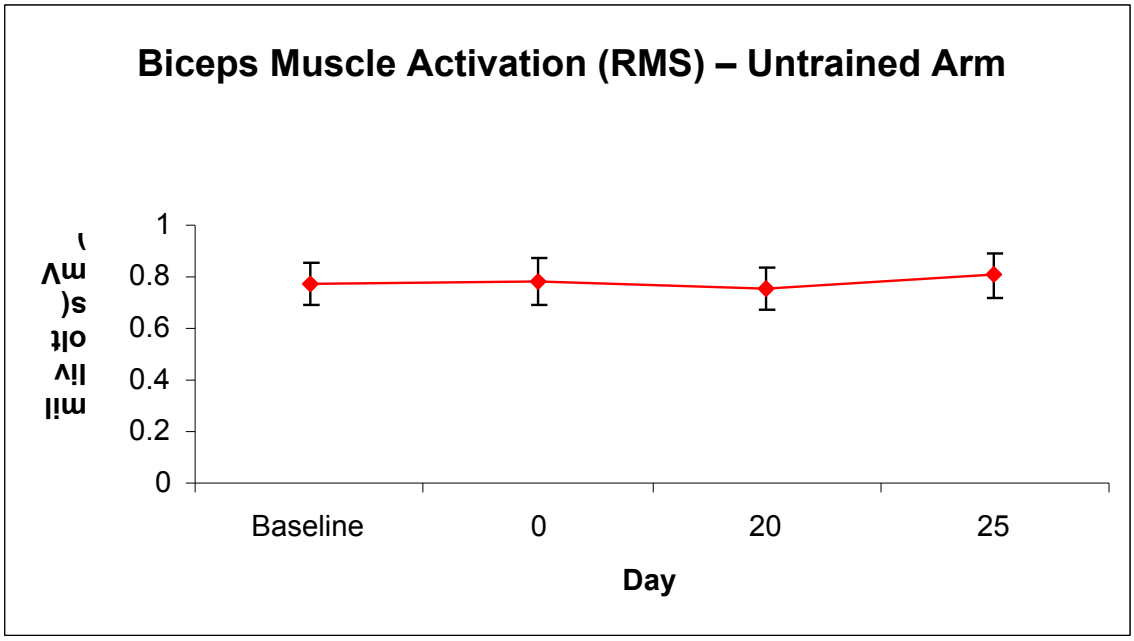

Figure 3.6 Muscle Activation – Biceps Untrained Arm. Values are expressed as means ± standard error.

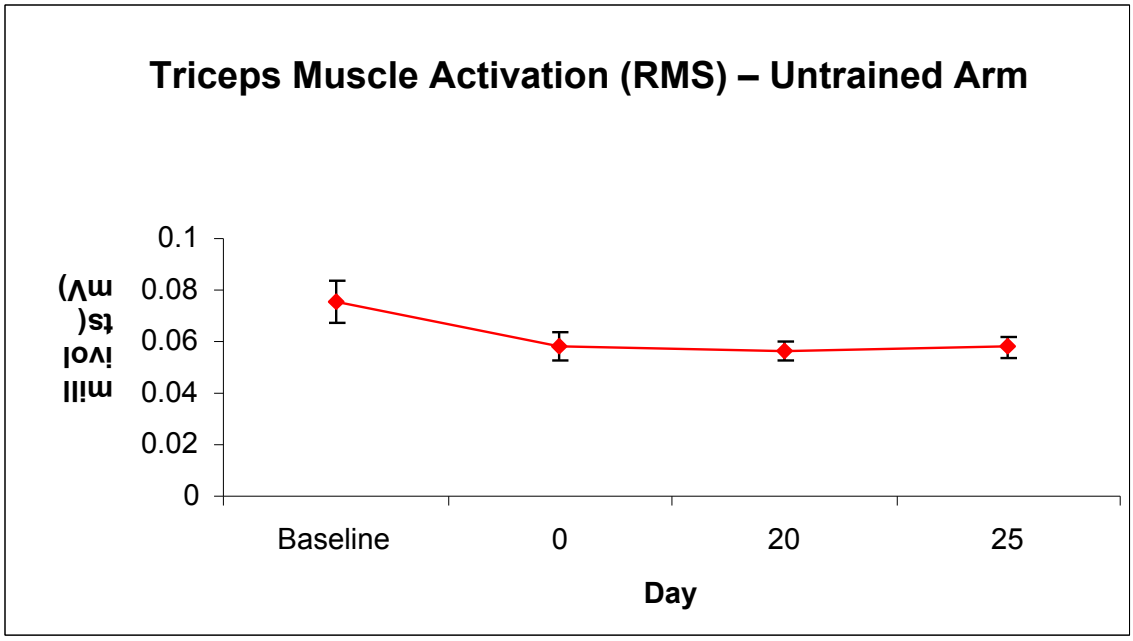

Figure 3.7 Muscle Activation – Triceps Untrained Arm. Values are expressed as means ± standard error.

3.4 Muscle/Joint Soreness

There was a time main effect for biceps muscle soreness, $F(2, 42)=62.839$, $p<0.05$. Post hoc multiple comparisons (Bonferroni adjusted) revealed soreness significantly decreased from week 1 to week 2 (5.32 ± 0.41 to 2.5 ± 0.44; $p<0.05$) and then again from week 2 to week 3 (2.5 ± 0.44 to 0.73 ± 0.25; $p<0.05$). This data is presented in figure 3.8 below. Frequency of soreness reported for arm muscle and joints is reported in Table 3.1. Across the study, 91% of participants reported some degree of biceps muscle soreness. For other joints the percentages were as follows: 68% participants reported some degree of elbow soreness, 50% reported some shoulder soreness, 23% reported some form of forearm soreness, and 27% reported hand or wrist soreness. See Appendix F for a detailed summary of muscle/joint soreness ratings for each participant across each week of the study.

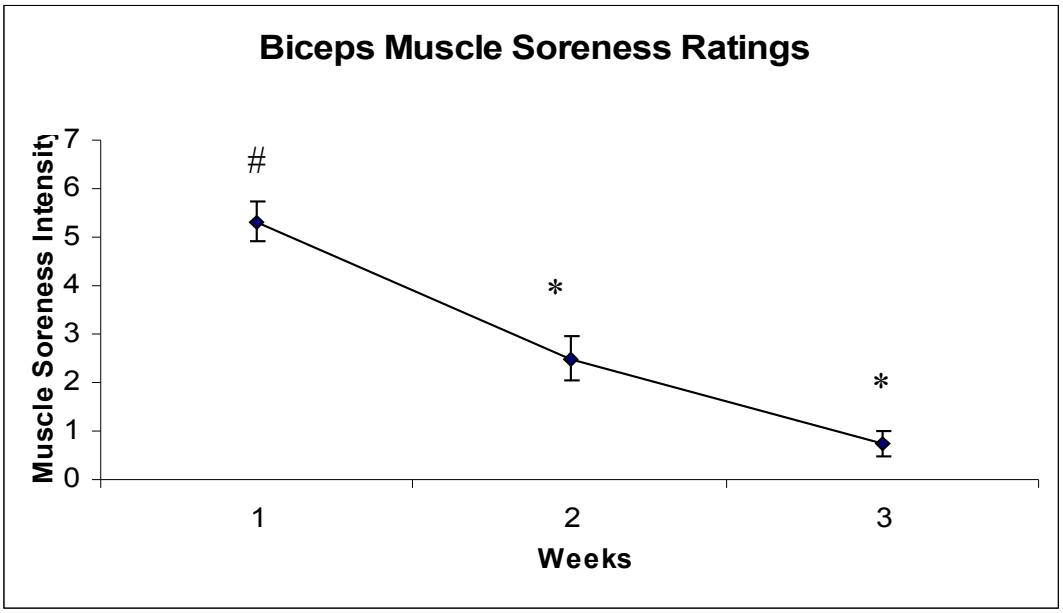

Figure 3.8 Biceps Muscle Soreness. Values are expressed as means ± standard error.
# Indicates time point was significantly different than 0; $p<0.05$. Adjusted with Bonferroni.
* Indicates time points are significantly different than previous week; $p<0.05$. Adjusted with Bonferroni.

Table 3.1 Muscle and Joint Soreness Frequency

Location	# of participants out of 22	% of total participants
Biceps	20	91%
Elbow	15	68 %
Forearm	11	50 %
Shoulder	5	23 %
Wrist / hand	6	27 %

Chapter 4

Discussion

The main finding of the current investigation was that muscle thickness increased very early in training. This finding was accompanied by increased agonist muscle activation and decreased antagonist muscle activation, and decreased strength. This is the first study to our knowledge, to show increased muscle size and improved coordination of muscle activation along with decreased strength over the course of a multi-week training study.

The major hypothesis of this study was that with intense eccentric training of the biceps, muscle hypertrophy would occur faster than has ever been reported (20 days). This hypothesis was supported as muscle thickness increased after only 8 days of training (4 training sessions). This rapid increase in muscle thickness is in opposition to the majority of research which emphasizes the current belief that muscle hypertrophy does not occur until approximately 4 weeks into training (Moritani and deVries, 1979; Abe et al., 2000). The finding of increased muscle thickness after 8 days is somewhat consistent with the study by Seynnes and colleagues (2007) who reported increased muscle thickness after 20 days. In their study there was a small increase in muscle size after 10 days (~2%) but this was not significant. There are a number of possible explanations for the faster increase in muscle size in the current study compared to the study by Seynnes et al. (2007). Seynnes and colleagues had only 7 participants in their study compared to the 22 participants that completed the current investigation, which likely provided the current study with much more statistical power. The study by Seynnes et al. (2007) had participants train the quadriceps muscle 3 days per week, with a volume of 4 sets of 7 repetitions. The current investigation had participants train more frequently (every 2nd day), with a higher

training volume (6 sets of 8 repetitions), and it targeted the biceps muscle, which has been shown to have a stronger hypertrophic response to training than the quadriceps (Brown et al., 1990; Welle et al., 1996). Still, both the current investigation and Seynnes et al. (2007) support the notion that hypertrophy may occur much earlier in training than previously thought. In addition, both studies prescribed strength training programs that involved eccentric contractions. Intense eccentric training might involve an earlier and more rapid phase of muscle growth when compared to more conventional resistance training (Moritani and deVries, 1979; Staron et al., 1994; Abe et al., 2000).

The notion that the hypertrophy process is initially delayed appears to have come from early work by Ikai and Fukunaga (1970) as well as Moritani and deVries (1979). Both of these studies tracked the time course of early strength and hypertrophy adaptations with training and found that strength increased more rapidly than muscle size. Closer examination of the study by Ikai and Fukunaga reveals that they did find substantial increases in muscle size after only 20 days of training (8.2%) but this was not significant. This is not surprising considering they only had 5 participants. This scenario is similar to research by Staron et al. (1994) and Abe et al. (2000), who both reported increases in muscle size (~5%, although non-significant) after only two weeks of training. These studies suggest that muscle size has the potential for increasing very early in training. This is interesting considering that these same studies are often cited to support the notion that the potential for hypertrophic adaptation is not present until after 4 weeks of training.

To our knowledge, no study has ever attempted to measure adaptations to strength training as early and as often as were measured in the present investigation. Including the baseline and detraining phases, 9 separate measurements were done on muscle thickness,

strength, and muscle activation on 22 participants. Time course studies are quite labour intensive and present significant challenges for the participants and research team, but they have the potential to provide valuable insight into our understanding of muscular adaptations with training. In particular, the early and frequent measurements conducted in the current study provide initial evidence that muscle hypertrophy can occur much earlier than 20 days.

4.1 Role of Inflammation?

In the current investigation, muscle thickness increased significantly after 8 days (Figure 3.1). This rapid increase in muscle size is much earlier than the fastest ever muscle hypertrophy response (20 days) reported in the literature (Seynnes et al., 2007). When examining this result and the fact that strength was also inhibited (Figure 3.2), the possibility that inflammation may have contributed to the observed increase in muscle thickness cannot be ruled out. Some amount of inflammation was likely present in the muscle especially very early in the training phase. Nosaka and Clarkson (1996) investigated the time course of inflammation with eccentric exercise and found that peak inflammation occurred 4-5 days after the exercise session. Traditionally it has been accepted that early inflammation may be accounted for by fluid accumulation, but this accumulated fluid may only be the cause of inflammation for a maximum of two days (Ryan and Majno, 1977). After two days, factors such as production of connective tissue or protein synthesis may account for additional swelling (Ryan and Majno, 1977; Smith, 1991; Nosaka and Clarkson, 1996). Nosaka and Newton (2002) examined whether repeated bouts of eccentric training would exacerbate damage incurred from previous bouts. They concluded that subsequent eccentric training did not exacerbate damage in muscles recovering from eccentric training. The findings of these studies seem to support the argument that

inflammation resulting from muscle damage was likely not the primary reason for the increase in muscle size in the current study. Inflammation may have partially contributed to the increase in muscle thickness early in training, but if repeated bouts of training do not cause exacerbating amounts of damage then it is logical to expect that over time the effect of inflammation may have eventually lessened. This may also be supported by the fact that biceps soreness was significantly reduced as training progressed (Figure 3.8). However, the relationship between inflammation and muscle soreness may not be as strong as previously thought. There is evidence to suggest a disconnect between the two factors, whereby muscle soreness may be gone but inflammation is still present in a muscle (Nosaka and Clarkson, 1996).

Another possible argument that may partially dispel the notion that inflammation was responsible for the increase in muscle thickness is known as the repeated bout effect. The repeated bout effect implies that after the performance of an initial bout of eccentric exercise a muscle adapts and is less susceptible to muscle damage when performing future bouts of eccentric exercise (Nosaka et al., 2001; Peake et al., 2005). In the current study, all participants performed baseline eccentric testing before the start of their first training session. Although the initial testing session consisted only of 4 maximal eccentric reps, along with familiarization and practice reps, this small amount of eccentric exercise may have prepared the biceps to better handle the subsequent eccentric training. Howatson et al. (2007) reported that the repeated bout effect was similar when comparing an initial exercise session that consisted of either 10 or 45 eccentric repetitions. This suggests that even a small amount of eccentric exposure was adequate to induce the repeated bout effect. Thus, it is reasonable to expect that the repeated bout effect was present in the current investigation and that the pre-baseline eccentric testing repetitions may

have had a protective effect that resulted in less muscle damage once participants began eccentric training.

Another argument against the possibility that inflammation was a confounding factor is in regards to the post eccentric exercise inflammatory response. Nosaka and Clarkson (1996) found that inflammation after eccentric exercise was highest after 4-5 days. This indicates that peak inflammation in the current investigation may have occurred prior to the first measurement of muscle thickness on Day 8 of training. In another eccentric exercise study, Nosaka and Newton (2002) reported that a second bout of eccentric exercise 48 hours after the first bout caused no additional damage and did not affect recovery. This study supports the idea that repeated training sessions (every 2nd day) in the present study likely did not lead to further muscle damage and inflammation.

4.2 Effects of the Detraining Phase

The finding that muscle size was reduced after 5 days of detraining (from day 20 to day 25) is noteworthy. Muscle thickness significantly decreased after 5 days of detraining but was still significantly larger than at Day 0 (Figure 3.1). There are several possible conclusions that can be drawn from this finding. Initially, it is easy to conclude that this decrease is related to a reduction in inflammation. If this is the case, the fact that the detraining value is still significantly higher than pre-training again supports the idea that significant muscle hypertrophy did occur within the training phase. However, before ruling out any factors aside from inflammation it is important to consider past detraining research when accounting for the decrease in size. Andersen and colleagues (2005) reported that all of the muscle hypertrophy experienced during 3 months of training was lost after 3 months of detraining. Similar results were reported by

Häkkinen et al. (2000) after 24 weeks of training followed by 24 weeks of detraining. These results indicate that in a period of detraining, all muscle size gains may be lost as rapidly as they were gained. By this explanation it is possible that 5 days of training, which represents 25% of the total training duration, was enough to induce some atrophy in the current study. The size decrease observed after detraining was 0.12 cm (3.97 to 3.85cm). This represents a reduction of just over 35% of the total increase in muscle size during the 20 days of training. Considering this relatively proportional decrease, it is plausible that the observed decrease is at least in part attributable to disuse atrophy. This idea is further supported when considering that in situations of muscle unloading (limb suspension, casting, etc) it has been suggested that atrophy seems to occur within a few days (de Boer et al., 2007).

4.3 Strength

A secondary hypothesis of the current investigation was that with training, strength would increase. Unexpectedly, the findings of this study did not support this hypothesis. Strength was significantly decreased at day 8 and remained suppressed for the entire study, even after 5 days of detraining (Figure 3.2). This finding is difficult to contrast with the literature, because previous isokinetic eccentric training studies did not re-assess strength until at least 5 weeks or more after the start of training (Seger et al., 1998; Farthing and Chilibeck, 2003b; Shepstone et al., 2005). It is very possible that an initial decline in strength in the first 3 weeks of training was also present in previous eccentric training studies, but was not detected because no early measurements were taken. The only other time course study that included eccentric training is Seynnes et al. (2007) who did report an increase in strength to accompany an increase in muscle size after 20 days. However, the current study employed isokinetic eccentric training, whereas

Seynnes et al. (2007) used a unique gravity independent flywheel ergometer. The flywheel system may not have provided the same intensity of eccentric contraction and therefore did not result in as much muscle damage. As well, it is very possible that the current study's large volume of training and limited rest may have also caused the observed decrease in strength. Regardless, the Seynnes et al. (2007) study represents an effective training protocol for optimizing both strength and hypertrophy.

Although a strength decrease was not the expected outcome, there are several possible explanations for this result. Several studies have shown decreased strength after bouts of eccentric exercise (Tokmakidis et al., 2003; Jamurtas et al., 2005; Chapman et al., 2006). The difference between these studies and the current investigation is that these studies were acute response studies in which responses to single sessions of eccentric exercise were observed. In contrast, the current investigation is a chronic response training study where repeated bouts of training were performed over a 20 day period. For this reason it might be expected that even though strength may be initially inhibited with eccentric exercise, strength would eventually recover and exceed pre-training scores. In the present study this was not the case. When interpreting this result it is important to consider the role that pain and muscle or joint soreness may have played. The current investigation obtained information on the muscle and joint soreness experienced by participants during the study. Results showed that 20 of the 22 participants reported some sort of muscle or joint soreness during the course of the study. In fact even as biceps muscle soreness began to dissipate, surrounding joint pain may have inhibited strength. This information highlights the importance of factors other than just site specific muscle recovery that must be considered in program design. For example, the target muscle site (e.g. biceps) may adequately adapt to a particular training volume, whereas the same training

volume may be too intense for surrounding tendons, joints, and ligaments. The finding of decreased strength in the presence of an increase in muscle thickness also brings up another possibility. If adequate muscle recovery was occurring, yet strength was still decreased, is this evidence that certain training stimuli that may be optimal for hypertrophic adaptation but detrimental to strength and neural adaptation? This idea warrants further discussion.

Strength was not significantly different in the untrained arm throughout the study. This is notable considering past studies have shown significant cross-education after unilateral training (Moritani and deVries, 1979; Farthing et al., 2005; Carroll et al., 2006; Farthing et al., 2007; Lee and Carroll, 2007). One reason for the absence of cross-education may be due to the counter-balancing of arms performed in this study. Farthing and colleagues (2005) found that right-handed individuals who trained their non-dominant arm experienced significantly less cross-education than those who trained their dominant arm. In the current investigation participants were randomized to train either their dominant or non-dominant arms which may have limited the amounts of cross-education experienced in those who trained their non-dominant arms. As well, Farthing and Chilibeck (2003a) found cross-education after fast (180°/s) eccentric training but not after slow (30°/s) eccentric training, and they suggested that the novel nature of the fast training may have contributed to the results. The present study used 90°/s and this speed may not have been novel enough to induce cross-education. The short training period may have also been a reason why cross-education was not present in this study as past studies have used substantially longer training periods (Moritani and deVries, 1979; Farthing and Chilibeck, 2003a; Farthing et al., 2005; Farthing et al., 2007) and it remains unclear how early cross-education may occur.

4.4 Agonist / Antagonist Activation

Another secondary hypothesis of the current study was that agonist (biceps) muscle activation would increase with training and that antagonist (triceps) muscle co-activation would decrease over the training phase. Both of these changes have been proposed in the literature as signs that neural adaptation is taking place (Sale, 1988; Narici et al., 1989; Carolan and Caferelli, 1992; Ploutz et al., 1994; Akima et al., 1999; Rabita et al., 2000; Rutherford et al., 2001). The findings of the current investigation supported both of these secondary hypotheses. Agonist (biceps) activation significantly increased on day 14 of training (Figure 3.4), and antagonist (triceps) co-activation was decreased by day 20 of training (Figure 3.5). These results support the idea that neural adaptations occurred with training; however it is puzzling that strength was still decreased. This further contributes to the idea that joint and or muscle pain may have played a large role in the strength decrease observed in training. It appears that training allowed participants to better coordinate the eccentric movement, a finding that would normally be associated with increased force output. In this situation, even though more force should have theoretically been expected in the desired direction, this was not the eventual outcome. This current study is limited by the fact that we cannot directly examine the forces exerted by the biceps and triceps muscles during the strength task. Even if the coordination of biceps and triceps muscle activation was improved, it appears that somewhere along the kinetic chain of movement force output exerted on the dynamometer handle was impaired. Another consideration is that despite increased agonist activation over the training period there still may have been inhibition of the agonist. In other words, the true maximal activation of the agonist muscle could have been much greater than the highest activation level reported in this study (Figure 3.4). Unfortunately, we are unable to confirm or refute this hypothesis because we did not assess maximal voluntary activation using interpolated twitch.

Although increased activation of the agonist normally accompanies increased strength or force output, this may not always be the case. During muscle fatigue, agonist muscle activation may increase as the muscle is trying to recruit more motor units in order to overcome fatigue of the already active fibres (Masuda et al., 1999). Thus, in the current investigation it is plausible that fatigue may have caused a decrease in strength while concurrently causing an increase in RMS activation of the agonist.

Increased agonist activation was expected and is consistent with a number of previous studies (Sale, 1988; Narici et al., 1989; Ploutz et al., 1994; Akima et al., 1999; Rabita et al., 2000; Rutherford et al., 2001; Reeves et al., 2005; Seynnes et al., 2007). In contrast, decreased co-activation of the antagonist muscle is a more novel finding. Although it has been suggested as a possible and very plausible early neural adaptation to strength training, there has been limited research to actually support the hypothesis that decreasing antagonist activation accompanies a strength increase with training. Despite that fact that decreased co-activation of the antagonist was evident in the current investigation, strength was still decreased. However, it should be noted that the activation of the antagonist muscle was minimal at the beginning of the study (Figure 3.5); therefore even a significant reduction in activation may not have played a huge role in strength production. In summary, the muscle activation data provides more evidence that intense eccentric training enhances agonist / antagonist movement coordination. Additionally, these results further suggest that reduced force output observed in the study was probably affected by factors other than changes in the activation of the primary agonist and antagonist.

4.5 Implications and Future Research

As previously mentioned, the main finding of the present investigation is that hypertrophy appears to occur much sooner than previously thought. Accompanying this conclusion, there are still many questions that are left unanswered. Along with the increased size there was increased agonist muscle activation and decreased strength. These findings may suggest that there is dissociation between changes in muscle thickness, strength, and neural adaptations. Previous research has concluded that these 3 factors are closely related and this is certainly the case in many situations (Moritani and deVries, 1979; Sale, 1988; Narici et al., 1989; Seynnes et al., 2007). But, it appears that in certain situations, namely intense eccentric training as demonstrated in this study; that there may be an altered time course of early adaptations. Certain training stimuli may result in increased muscle size and be able to enhance neural adaptation and movement coordination but may also be so damaging to the muscles and surrounding tissues that strength is not enhanced. This is completely in opposition to most conventional training protocols which show positive relationships between the changes in muscle thickness, strength, and muscle activation (Moritani and deVries, 1979; Sale, 1988; Narici et al., 1989; Seynnes et al., 2007). Further, the current study suggests that it is possible for a muscle to get larger even if its training does not result in more strength output. For example, a muscle that is smaller in size but well trained (e.g. a muscle of a light weight power lifter) may be stronger than the same larger muscle of an untrained person. The current findings suggest the opposite idea; that a muscle may become larger even if it does not become stronger with training. This would suggest that form and function may not be highly correlated in all training situations.

The knowledge and ideas derived from this study have significant implications for future training prescription. The aim of the study was to design a training protocol effective for rapid hypertrophy. But, it is noteworthy for all exercise professionals involved in the prescription of

resistance training programs, that an intense eccentric training protocol may not be beneficial for increasing strength. Specifically, a training regimen containing intense eccentric training, although potentially great for inducing muscle growth, should be used with adequate amounts of rest in situations where strength is required in the near future (competition or pre-competition phases). In contrast, if strength is not as important and the addition of muscle mass is the primary goal, a degree of eccentric training should be included. This may be specifically beneficial for situations after atrophy from injury or disuse, in populations at risk for sarcopenia, or for sports such as bodybuilding.

Along with training implications, the current investigation also draws many questions that warrant further study. Future research should attempt to distinguish between muscle hypertrophy and inflammation in the time course of early adaptation to better understand the role that each plays in muscle size increases. This could be accomplished by tracking markers of inflammation and muscle damage either through blood samples or by taking muscle biopsies. Future studies should also attempt to design programs that can optimize hypertrophy while showing concurrent increases in strength. This may be accomplished with less intense training protocols or with decreased weekly training volume in order to allow more time for recovery. The current study shows that intense eccentric training decreases strength, but it must be considered that the volume of training and limited rest was probably a major reason for this result. Eccentric training performed less frequently and with reduced training volume may still be effective for hypertrophy but may be much less detrimental to strength. This type of training should be explored in more detail in future research.

It is also warranted that future studies attempt to show early hypertrophic adaptation across different populations and with different methodology. This study was performed with

untrained college aged individuals. It would be interesting to see if similar responses in muscle growth could be obtained in trained individuals or in older adults. Additionally, the current study had participants train using an isokinetic dynamometer. This is a very specialized and exclusive training apparatus it would be beneficial to find results of early hypertrophy using more conventional and accessible training protocols (free weights or conventional machines).

Finally, future research should continue to explore the mechanistic adaptations to early hypertrophy. By looking at responses such as protein synthesis and gene expression after single exercise sessions, the most effective training stimuli can be uncovered and put into practice. As well, the tracking of these cellular and molecular adaptations during time course studies, although difficult, provides important information about the mechanisms involved in adapting muscle.

Chapter 5

Summary and Conclusions

5.1 Summary

Adaptations early in resistance training (prior to 4 weeks) have long been thought to be mediated by neural related factors. Recent research has reported hypertrophy within 20 days of training the quadriceps (Seynnes et al., 2007). The purpose of this study was to track hypertrophy, strength, and muscle activation during 20 days of eccentric exercise in an attempt to further understand the interplay of hypertrophy and strength very early in training. The primary hypothesis of the investigation was that muscle hypertrophy would occur sooner than 20 days, the current fastest reported significant muscle hypertrophy. This hypothesis was supported as increased muscle thickness was found after only 8 days (4 training sessions). This finding must be taken in perspective though as inflammation may have been partially responsible for the early increase in muscle size. Still, with the intense eccentric training protocol utilized in the current investigation, it is likely that muscle hypertrophy was the predominant factor responsible for the increase in muscle thickness observed in this study.

The secondary hypotheses of the study were that strength would increase with training, agonist (biceps) muscle activation would increase, and antagonist muscle (triceps) co-activation would decrease with training. The hypothesis of strength increase was not supported. In fact strength decreased as a result of training and never recovered, even after 5 days of detraining. This finding was also accompanied by joint and muscle pain which may be a partial explanation for the decreased strength observed in the study. Both of the muscle activation hypotheses were supported, suggesting that improved muscle activation coordination occurred as a result of

training. This finding is usually accompanied with increased strength. Since this was not the case, it can be speculated that even though there were significant neural adaptations, the full potential of these adaptations may still have been blunted.

5.2 Conclusions

The results of this study confirm that muscle thickness increases very rapidly with intense eccentric training. Although this cannot be solely attributed to muscle growth, this is very strong evidence that muscle hypertrophy may occur much sooner than most exercise scientists have traditionally accepted. The results of the study also suggest that successive intense eccentric training performed every second day, decreases strength in previously untrained individuals. Additionally, this type of training causes increased agonist activation and decreased antagonist co-activation, both forms of early neural adaptations that reflect improved neural coordination of movement.

5.2 Limitations

There are several limitations to the current investigation. The obvious limitation is that no measures of inflammation, swelling, or internal muscle biochemistry were taken. For this reason we can only speculate as to what was going on inside the muscle during training. This information would have been useful but was not feasible for the scope of the current investigation. As previously mentioned, this is an area that should be explored by future research.

Another limitation of the study was that the findings of the study are limited to a specific population. This study used only untrained college aged students, most of who were recruited

from the College of Kinesiology at the University of Saskatchewan. Thus, caution should be exercised when trying to generalize these findings to other populations. Similarly, another limitation of this research is that it was performed using isokinetic dynamometry in a supervised laboratory setting. This raises questions about the real world applications and generalizability of the results.

Finally, a limitation of this study was that it was not blinded. The primary investigator supervised all the training sessions and made all of the muscle thickness measurements for the study. An ideal design would have been to have the muscle thickness measurements taken by a researcher blinded to the training and non-training arms of the participants but again this was not practical or feasible for this project.

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Appendices

Appendix A Sample Training Phase Calendar

S	M	T	W	TH	F	S
Prior Baseline testing 20 day Baseline Period U # 1 Test # 1	Day 0 U # 2 Test # 2 Train # 1	Day 1	Day 2 Train # 2	Day 3	Day 4 Train # 3	Day 5
Day 6 Train # 4	Day 7	Day 8 U # 3 Train # 5	Day 9	Day 10 Train # 6	Day 11	Day 12 U # 4 Train # 7
Day 13	Day 14 U #5 Train # 8	Day 15	Day 16 U # 6 Train # 9	Day 17	Day 18 U # 7 Train # 10	Day 19
Day 20 U # 8 Test # 3					Day 25 U # 9 Test # 4	

U= ultrasound and strength recorded

Appendix B Ethics: Certificate of Approval

UNIVERSITY OF SASKATCHEWAN

Biomedical Research Ethics Board (Bio-REB)

Certificate of Approval

PRINCIPAL INVESTIGATOR	DEPARTMENT	Bio #
Jonathan F Farthing	Kinesiology	08-05

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT
College of Kinesiology
105 Gymnasium Place
Saskatoon SK S7N 5C2

STUDENT RESEARCHERS
Joel Krentz

SPONSORING AGENCIES
NATURAL SCIENCES & ENGINEERING RESEARCH COUNCIL OF CANADA (NSERC)

TITLE: Time Course of Morphological Adaptations with Intense Strength Training Over a 20 Day Period

APPROVAL DATE	EXPIRY DATE	APPROVAL OF
31-Jan-2008	30-Jan-2009	Researcher Summary as submitted (07-Jan-2008) Research Particpant Information and Consent Form (07-Jan-2008) Recruitment Poster

Full Board Meeting ☐

Delegated Review ☒

CERTIFICATION
The study is acceptable on scientific and ethical grounds. The principal investigator has the responsibility for any other administrative or regulatory approvals that may pertain to this research study, and for ensuring that the authorized research is carried out according to governing law. This approval is valid for the specified period provided there is no change to the approved protocol or consent process.

FIRST TIME REVIEW AND CONTINUING APPROVAL
The University of Saskatchewan Biomedical Research Ethics Board reviews above minimal studies at a full-board (face-to-face) meeting. Any research classified as minimal risk is reviewed through the delegated (subcommittee) review process. The initial Certificate of Approval includes the approval period the REB has assigned to a study. The Status Report form must be submitted within one month prior to the assigned expiry date. The researcher shall indicate to the REB any specific requirements of the sponsoring organizations (e.g. requirement for full-board review and approval) for the continuing review process deemed necessary for that project. For more information visit http://www.usask.ca/research/ethics_review/.

REB ATTESTATION
In respect to clinical trials, the University of Saskatchewan Research Ethics Board complies with the membership requirements for Research Ethics Boards defined in Division 5 of the Food and Drug Regulations and carries out its functions in a manner consistent with Good Clinical Practices. This approval and the views of this REB have been documented in writing.

Michel Desautels, Ph.D., Chair
University of Saskatchewan
Biomedical Research Ethics Board

Jan 31, 2008
Signature Date

Please send all correspondence to:

Ethics Office
University of Saskatchewan
Room 305 Kirk Hall, 117 Science Place
Saskatoon SK S7N 5C8
Telephone: (306) 966-4053 Fax: (306) 966-2069

Appendix C Consent Form

Research Participant Information and Consent Form

Title: Time course of morphological adaptations with intense strength training over a 20 day period

Names of Researchers: Principal Investigators: Joel Krentz, B.Sc., Master's Candidate (Graduate student supervised by Dr. Jonathan Farthing), College of Kinesiology, University of Saskatchewan, phone: 966-1123, Jonathan Farthing, Ph.D., Assistant Professor, College of Kinesiology, University of Saskatchewan, phone: 966-1068

You are being invited to participate in a research study because we want to determine how the biceps muscle adapts early in strength training. This will allow us to better understand which adaptations are responsible for increased strength with training.

Voluntary Participation: Before you decide, it is important for you to understand what the research involves. This consent form will tell you about the study, why the research is being done, what will happen to you during the study and the possible benefits, risks and discomforts.

If you wish to participate, you will be asked to sign this form. Your participation is entirely voluntary, so it is up to you to decide whether or not to take part in this study. If you do decide to take part in this study, you are free to withdraw at any time without giving any reasons for your decision and your refusal to participate will not affect your relationship with any of the researchers or the University of Saskatchewan, and will not affect your academic standing if you are a student at the university. Please take time to read the following information carefully and to discuss it with your family, friends, fellow employees, employer, and doctor before you decide.

Purpose of the study: The purpose of this study is to investigate how the biceps muscle adapts early on in an "Eccentric" strength training program. "Eccentric" contractions are be performed by resisting while your muscle is forced into a lengthened position. By tracking the adaptations of the biceps with training we hope to better understand the various factors that lead to increased strength. Adaptations that will be measured are muscle thickness (i.e. size of your biceps muscle), strength, muscle activation, and the angle that your muscle fibers are arranged (i.e. pennation angle).

Possible benefits of the study: You may get stronger as a result of training your biceps muscle. As well, you will get an assessment of strength, muscle thickness, muscle activation for the biceps muscle by participating in the study. These benefits are not guaranteed.

Procedures:
If you agree to be in this study the following will happen:

Initially you will be invited to come into our lab to perform a pre-baseline assessment. In this session we will measure the strength, muscle thickness, muscle fibre angle, and muscle

activation of both of your biceps. You will then come back to our lab after 20 days and perform the same tests. This will complete the "baseline phase" of the study. The next phase will be the "training phase" and will also last 20 days. It will begin immediately after the baseline phase. In this phase you will come into the lab every second day to train the biceps of only one of your arms with eccentric contractions and to have your strength, muscle thickness, muscle fibre angle, and muscle activation measured. Each session will take about 30 minutes to complete. After the training phase is over, one final testing session will be completed on both of your biceps. In total, you will visit the lab 12 times over a period of about 6 weeks.

Muscle thickness and fibre angle will be measured using a muscle ultrasound device. This procedure requires the placement of a jelly like substance on your arm and allows us to get images inside your arm that can tell us the thickness and arrangement of the fibers. The procedure is not harmful or painful.

The natural electrical activity of your muscle will be measured during the strength tests. This involves the placement of stickers, called electrodes, on the skin over your muscle. A wire attached to the electrode measures the electrical activity during muscular contraction. This gives an indication of your ability to activate your biceps muscle.

Strength testing and training sessions will be completed using a machine (isokinetic dynamometer) that controls the speed of contraction through a determined range of motion. Strength will also be assessed while you are training your biceps during the eccentric contractions. Muscle contractions will be at a medium speed (lasting approximately 1 second each) and will be at a maximal level.

All testing procedures and strength training sessions will take place in the lab and will be supervised by a member of the research team.

Foreseeable risks, side effects or discomfort:

The exercise tests and training will be at maximal intensity and therefore will result in some discomfort and muscle fatigue. Training and testing may result in stiff muscles. There is also a small risk of muscle injury during maximal strength training, but this will be minimized by proper warm up (i.e. stretching) and training supervision.

Training will take place on only one of your arms. This may result in one arm gaining more strength and muscle size than the other arm. The time period of training is short (3 weeks) in comparison to an average training period, and it is unlikely that you end up with one arm noticeably larger than the other. However, you will be able to come in to the lab and train your other arm after you have completed the study in order to correct any slight muscular imbalance.

There may be some discomfort on your skin from the adhesive tape that temporarily sticks the electrodes to your skin, but this is rare.

In order to ensure the muscle size and fibre angle measurements are taken on the exact same spot each time, a semi-permanent mark will be placed on your biceps on each testing occasion for the

entire 3 week training phase. A non-toxic marker from our exercise physiology lab, which is safe for use on human skin, will be used for the study. You will be asked to avoid completely scrubbing off the skin markings until after the training phase is finished.

There may be unforeseen and unknown risks during the study, or after the study has been completed.

Alternatives to this study:
You do not have to participate in this study to have bicep strength levels assessed. You can pay a fee to have strength assessment completed for you by this research lab at another time designated by you and the lab coordinators, or by another fitness facility.

Research-Related Injury: There will be no cost to you for participation in this study. You will not be charged for any research procedures. In the event you become ill or injured as a result of participating in this study, necessary medical treatment will be made available at no additional cost to you. By signing this document you do not waive any of your legal rights.

Confidentiality: While complete subject anonymity cannot be guaranteed, every effort will be made to ensure that the information you provide for this study is kept entirely confidential. The testing procedures will take place in an enclosed space in the Physical Activity Complex. Your name will not be attached to any information, nor mentioned in any study report, nor be made available to anyone except the research team. It is the intention of the research team to publish results of this research in scientific journals and to present the findings at related conferences and workshops, but your identity will not be revealed.

Voluntary Withdrawal: Your participation in this research is entirely voluntary. You may withdraw from this study at any time. If you decide to enter the study and to withdraw at any time in the future, there will be no penalty or loss of benefits to which you are otherwise entitled.

If you choose to enter the study and then decide to withdraw at a later time, all data collected about you during your enrolment in the study will be retained for analysis.

If you have questions concerning the study you can contact Mr. Joel Krentz at 966-1123 or Dr. Jonathan Farthing at 966-1068. Dr. Farthing's number can be called collect if you are phoning long distance. If you have questions about your rights as a research subject, you should contact the Chair of the Biomedical Research Ethics Board, University of Saskatchewan at (306) 966-4053. Again, this number can be called collect if you are phoning long distance.

By signing below, I confirm the following:

- I have read or have had this read to me and understood the research subject information and consent form.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have had satisfactory responses to my questions.

- I understand that all of the information collected will be kept confidential and that the result will only be used for scientific objectives.
- I understand that my participation in this study is voluntary and that I am completely free to refuse to participate or to withdraw from this study at any time without changing in any way affect my academic standing or my relationship with members of the research team.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I understand that there is no guarantee that this study will provide any benefits to me.
- I have read this form and I freely consent to participate in this study.
- I have been told that I will receive a dated and signed copy of this form

Participant's Signature:_____ Date: _____

Individual conducting the consent process:_____

Date: _____

Appendix D Resistance Training Experience Questionnaire

Name _____ **Height _____**

Weight _____

Pre-Screening Questions

1. **How many months in your lifetime have you performed resistance training**

(1 month = 3 x per week for the whole month) _____

2. **How many months in the last year have you performed resistance training**

(1 month = 3 x per week for the whole month) _____

3. **How many months have you regularly trained your biceps in your lifetime**

(1 month = 3 x per week for the whole month) _____

4. **How many days have you regularly trained your biceps in the last 2 months**

(1 day = minimum 3 sets of bicep training) _____

1. **Are you currently taking any medications or pills that to your knowledge might impact your normal response to resistance training? (i.e. hormone replacement, antibiotics, contraceptive pills, etc.)**

Yes or No

2. Are you currently taking any dietary supplements that to your knowledge might impact your normal response to resistance training? (i.e. creatine, protein, vitamins, etc.) Yes or No

Appendix E Statistical Tables

2 x 9 (arm x time) Factorial ANOVA for Muscle Thickness Data

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
time	Pillai's Trace	.901	15.953[a]	8.000	14.000	.000
	Wilks' Lambda	.099	15.953[a]	8.000	14.000	.000
	Hotelling's Trace	9.116	15.953[a]	8.000	14.000	.000
	Roy's Largest Root	9.116	15.953[a]	8.000	14.000	.000
arm	Pillai's Trace	.592	30.496[a]	1.000	21.000	.000
	Wilks' Lambda	.408	30.496[a]	1.000	21.000	.000
	Hotelling's Trace	1.452	30.496[a]	1.000	21.000	.000
	Roy's Largest Root	1.452	30.496[a]	1.000	21.000	.000
time * arm	Pillai's Trace	.917	19.366[a]	8.000	14.000	.000
	Wilks' Lambda	.083	19.366[a]	8.000	14.000	.000
	Hotelling's Trace	11.066	19.366[a]	8.000	14.000	.000
	Roy's Largest Root	11.066	19.366[a]	8.000	14.000	.000

a. Exact statistic

b.
Design: Intercept
Within Subjects Design: time+arm+time*arm

Mauchly's Test of Sphericity[b]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.021	69.692	35	.001	.603	.804	.125
arm	1.000	.000	0	.	1.000	1.000	1.000
time * arm	.092	43.225	35	.174	.663	.914	.125

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: time+arm+time*arm

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	1.667	8	.208	29.733	.000
	Greenhouse-Geisser	1.667	4.820	.346	29.733	.000
	Huynh-Feldt	1.667	6.431	.259	29.733	.000
	Lower-bound	1.667	1.000	1.667	29.733	.000
Error(time)	Sphericity Assumed	1.178	168	.007		
	Greenhouse-Geisser	1.178	101.227	.012		
	Huynh-Feldt	1.178	135.046	.009		
	Lower-bound	1.178	21.000	.056		
arm	Sphericity Assumed	5.627	1	5.627	30.496	.000
	Greenhouse-Geisser	5.627	1.000	5.627	30.496	.000
	Huynh-Feldt	5.627	1.000	5.627	30.496	.000
	Lower-bound	5.627	1.000	5.627	30.496	.000
Error(arm)	Sphericity Assumed	3.875	21	.185		
	Greenhouse-Geisser	3.875	21.000	.185		
	Huynh-Feldt	3.875	21.000	.185		
	Lower-bound	3.875	21.000	.185		
time * arm	Sphericity Assumed	1.393	8	.174	57.714	.000
	Greenhouse-Geisser	1.393	5.307	.262	57.714	.000
	Huynh-Feldt	1.393	7.312	.190	57.714	.000
	Lower-bound	1.393	1.000	1.393	57.714	.000
Error(time*arm)	Sphericity Assumed	.507	168	.003		
	Greenhouse-Geisser	.507	111.438	.005		
	Huynh-Feldt	.507	153.545	.003		
	Lower-bound	.507	21.000	.024		

Simple Main Effects Analysis: One-way ANOVA for the Trained Arm Muscle Thickness and Multiple Pairwise Comparisons (Bonferroni Adjusted)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	3.002	8	.375	64.546	.000
	Greenhouse-Geisser	3.002	4.264	.704	64.546	.000
	Huynh-Feldt	3.002	5.485	.547	64.546	.000
	Lower-bound	3.002	1.000	3.002	64.546	.000
Error(time)	Sphericity Assumed	.977	168	.006		
	Greenhouse-Geisser	.977	89.536	.011		
	Huynh-Feldt	.977	115.185	.008		
	Lower-bound	.977	21.000	.047		

Pairwise Comparisons

Measure: MEASURE_1

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.018	.023	1.000	-.103	.068
	3	-.261*	.028	.000	-.362	-.160
	4	-.273*	.023	.000	-.359	-.187
	5	-.307*	.024	.000	-.395	-.220
	6	-.337*	.025	.000	-.430	-.243
	7	-.323*	.023	.000	-.408	-.239
	8	-.325*	.023	.000	-.409	-.240
	9	-.203*	.018	.000	-.268	-.137
2	1	.018	.023	1.000	-.068	.103
	3	-.243*	.034	.000	-.367	-.119
	4	-.255*	.031	.000	-.368	-.142
	5	-.290*	.027	.000	-.387	-.192
	6	-.319*	.031	.000	-.434	-.204
	7	-.306*	.029	.000	-.413	-.199
	8	-.307*	.025	.000	-.400	-.213
	9	-.185*	.025	.000	-.276	-.094
3	1	.261*	.028	.000	.160	.362
	2	.243*	.034	.000	.119	.367
	4	-.012	.020	1.000	-.085	.061
	5	-.047	.029	1.000	-.153	.060
	6	-.076	.029	.611	-.183	.032
	7	-.062	.027	1.000	-.162	.037
	8	-.064	.025	.702	-.156	.029
	9	.058	.026	1.000	-.039	.155
4	1	.273*	.023	.000	.187	.359
	2	.255*	.031	.000	.142	.368
	3	.012	.020	1.000	-.061	.085
	5	-.035	.019	1.000	-.106	.036
	6	-.064	.018	.080	-.131	.004
	7	-.051	.017	.302	-.115	.013
	8	-.052	.018	.338	-.119	.015
	9	.070	.019	.061	-.002	.141
5	1	.307*	.024	.000	.220	.395
	2	.290*	.027	.000	.192	.387
	3	.047	.029	1.000	-.060	.153
	4	.035	.019	1.000	-.036	.106
	6	-.029	.015	1.000	-.083	.025
	7	-.016	.012	1.000	-.061	.029
	8	-.017	.014	1.000	-.070	.036
	9	.105*	.018	.000	.038	.171
6	1	.337*	.025	.000	.243	.430
	2	.319*	.031	.000	.204	.434
	3	.076	.029	.611	-.032	.183
	4	.064	.018	.080	-.004	.131
	5	.029	.015	1.000	-.025	.083
	7	.013	.012	1.000	-.030	.056
	8	.012	.022	1.000	-.068	.092
	9	.134*	.024	.001	.045	.222
7	1	.323*	.023	.000	.239	.408
	2	.306*	.029	.000	.199	.413
	3	.062	.027	1.000	-.037	.162
	4	.051	.017	.302	-.013	.115
	5	.016	.012	1.000	-.029	.061
	6	-.013	.012	1.000	-.056	.030
	8	-.001	.017	1.000	-.063	.061
	9	.120*	.019	.000	.049	.192
8	1	.325*	.023	.000	.240	.409
	2	.307*	.025	.000	.213	.400
	3	.064	.025	.702	-.029	.156
	4	.052	.018	.338	-.015	.119
	5	.017	.014	1.000	-.036	.070
	6	-.012	.022	1.000	-.092	.068
	7	.001	.017	1.000	-.061	.063
	9	.122*	.012	.000	.077	.166
9	1	.203*	.018	.000	.137	.268
	2	.185*	.025	.000	.094	.276
	3	-.058	.026	1.000	-.155	.039
	4	-.070	.019	.061	-.141	.002
	5	-.105*	.018	.000	-.171	-.038
	6	-.134*	.024	.001	-.222	-.045
	7	-.120*	.019	.000	-.192	-.049
	8	-.122*	.012	.000	-.166	-.077

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

Simple Main Effects Analysis: One-way ANOVA for the Untrained Arm Muscle Thickness

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	.058	8	.007	1.725	.096
	Greenhouse-Geisser	.058	5.567	.010	1.725	.127
	Huynh-Feldt	.058	7.806	.007	1.725	.098
	Lower-bound	.058	1.000	.058	1.725	.203
Error(time)	Sphericity Assumed	.708	168	.004		
	Greenhouse-Geisser	.708	116.907	.006		
	Huynh-Feldt	.708	163.932	.004		
	Lower-bound	.708	21.000	.034		

One-way ANOVA (time) for Trained Arm Strength and Multiple Pairwise Comparisons (Bonferroni Adjusted)

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
time	Pillai's Trace	.808	7.362[a]	8.000	14.000	.001
	Wilks' Lambda	.192	7.362[a]	8.000	14.000	.001
	Hotelling's Trace	4.207	7.362[a]	8.000	14.000	.001
	Roy's Largest Root	4.207	7.362[a]	8.000	14.000	.001

a. Exact statistic

b.
Design: Intercept
Within Subjects Design: time

Mauchly's Test of Sphericity[b]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.025	66.738	35	.001	.563	.735	.125

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: time

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	2935.616	8	366.952	16.179	.000
	Greenhouse-Geisser	2935.616	4.501	652.195	16.179	.000
	Huynh-Feldt	2935.616	5.881	499.195	16.179	.000
	Lower-bound	2935.616	1.000	2935.616	16.179	.001
Error(time)	Sphericity Assumed	3810.384	168	22.681		
	Greenhouse-Geisser	3810.384	94.524	40.311		
	Huynh-Feldt	3810.384	123.495	30.855		
	Lower-bound	3810.384	21.000	181.447		

Pairwise Comparisons

Measure: MEASURE_1

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-.136	1.396	1.000	-5.276	5.003
	3	8.000*	1.616	.002	2.052	13.948
	4	9.409*	1.834	.002	2.659	16.159
	5	8.682*	1.591	.001	2.827	14.537
	6	10.000*	1.240	.000	5.437	14.563
	7	9.409*	1.840	.002	2.638	16.181
	8	9.773*	1.664	.000	3.646	15.900
	9	8.182*	1.540	.001	2.514	13.850
2	1	.136	1.396	1.000	-5.003	5.276
	3	8.136*	1.677	.003	1.965	14.308
	4	9.545*	1.727	.001	3.189	15.902
	5	8.818*	1.937	.006	1.688	15.948
	6	10.136*	1.419	.000	4.912	15.361
	7	9.545*	1.788	.001	2.963	16.128
	8	9.909*	1.955	.002	2.712	17.106
	9	8.318*	1.639	.002	2.286	14.351
3	1	-8.000*	1.616	.002	-13.948	-2.052
	2	-8.136*	1.677	.003	-14.308	-1.965
	4	1.409	1.380	1.000	-3.669	6.488
	5	.682	1.339	1.000	-4.249	5.612
	6	2.000	1.138	1.000	-2.188	6.188
	7	1.409	1.745	1.000	-5.016	7.834
	8	1.773	1.494	1.000	-3.729	7.274
	9	.182	1.456	1.000	-5.178	5.541
4	1	-9.409*	1.834	.002	-16.159	-2.659
	2	-9.545*	1.727	.001	-15.902	-3.189
	3	-1.409	1.380	1.000	-6.488	3.669
	5	-.727	1.075	1.000	-4.686	3.232
	6	.591	1.289	1.000	-4.153	5.335
	7	.000	1.508	1.000	-5.550	5.550
	8	.364	1.343	1.000	-4.580	5.307
	9	-1.227	1.240	1.000	-5.790	3.336
5	1	-8.682*	1.591	.001	-14.537	-2.827
	2	-8.818*	1.937	.006	-15.948	-1.688
	3	-.682	1.339	1.000	-5.612	4.249
	4	.727	1.075	1.000	-3.232	4.686
	6	1.318	1.070	1.000	-2.620	5.256
	7	.727	1.262	1.000	-3.920	5.375
	8	1.091	.989	1.000	-2.549	4.731
	9	-.500	1.038	1.000	-4.320	3.320
6	1	-10.000*	1.240	.000	-14.563	-5.437
	2	-10.136*	1.419	.000	-15.361	-4.912
	3	-2.000	1.138	1.000	-6.188	2.188
	4	-.591	1.289	1.000	-5.335	4.153
	5	-1.318	1.070	1.000	-5.256	2.620
	7	-.591	1.173	1.000	-4.908	3.726
	8	-.227	1.122	1.000	-4.358	3.904
	9	-1.818	.977	1.000	-5.416	1.779
7	1	-9.409*	1.840	.002	-16.181	-2.638
	2	-9.545*	1.788	.001	-16.128	-2.963
	3	-1.409	1.745	1.000	-7.834	5.016
	4	.000	1.508	1.000	-5.550	5.550
	5	-.727	1.262	1.000	-5.375	3.920
	6	.591	1.173	1.000	-3.726	4.908
	8	.364	.955	1.000	-3.151	3.878
	9	-1.227	1.267	1.000	-5.892	3.437
8	1	-9.773*	1.664	.000	-15.900	-3.646
	2	-9.909*	1.955	.002	-17.106	-2.712
	3	-1.773	1.494	1.000	-7.274	3.729
	4	-.364	1.343	1.000	-5.307	4.580
	5	-1.091	.989	1.000	-4.731	2.549
	6	.227	1.122	1.000	-3.904	4.358
	7	-.364	.955	1.000	-3.878	3.151
	9	-1.591	.854	1.000	-4.736	1.555
9	1	-8.182*	1.540	.001	-13.850	-2.514
	2	-8.318*	1.639	.002	-14.351	-2.286
	3	-.182	1.456	1.000	-5.541	5.178
	4	1.227	1.240	1.000	-3.336	5.790
	5	.500	1.038	1.000	-3.320	4.320
	6	1.818	.977	1.000	-1.779	5.416
	7	1.227	1.267	1.000	-3.437	5.892
	8	1.591	.854	1.000	-1.555	4.736

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

One-way ANOVA (time) for Untrained Arm Strength

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
time	Pillai's Trace	.114	.815[a]	3.000	19.000	.501
	Wilks' Lambda	.886	.815[a]	3.000	19.000	.501
	Hotelling's Trace	.129	.815[a]	3.000	19.000	.501
	Roy's Largest Root	.129	.815[a]	3.000	19.000	.501

a. Exact statistic

b. Design: Intercept

Within Subjects Design: time

Mauchly's Test of Sphericity[b]

Measure:MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.186	33.203	5	.000	.516	.550	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b. Design: Intercept

Within Subjects Design: time

Tests of Within-Subjects Effects

Measure:MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	139.034	3	46.345	1.516	.219
	Greenhouse-Geisser	139.034	1.549	89.761	1.516	.234
	Huynh-Feldt	139.034	1.649	84.310	1.516	.234
	Lower-bound	139.034	1.000	139.034	1.516	.232
Error(time)	Sphericity Assumed	1925.716	63	30.567		
	Greenhouse-Geisser	1925.716	32.528	59.203		
	Huynh-Feldt	1925.716	34.631	55.607		
	Lower-bound	1925.716	21.000	91.701		

2 x 9 (muscle x time) Factorial ANOVA for Trained Arm EMG

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
time	Pillai's Trace	.647	3.203[a]	8.000	14.000	.027
	Wilks' Lambda	.353	3.203[a]	8.000	14.000	.027
	Hotelling's Trace	1.830	3.203[a]	8.000	14.000	.027
	Roy's Largest Root	1.830	3.203[a]	8.000	14.000	.027
muscle	Pillai's Trace	.846	115.581[a]	1.000	21.000	.000
	Wilks' Lambda	.154	115.581[a]	1.000	21.000	.000
	Hotelling's Trace	5.504	115.581[a]	1.000	21.000	.000
	Roy's Largest Root	5.504	115.581[a]	1.000	21.000	.000
time * muscle	Pillai's Trace	.709	4.264[a]	8.000	14.000	.009
	Wilks' Lambda	.291	4.264[a]	8.000	14.000	.009
	Hotelling's Trace	2.437	4.264[a]	8.000	14.000	.009
	Roy's Largest Root	2.437	4.264[a]	8.000	14.000	.009

a. Exact statistic

b.
Design: Intercept
Within Subjects Design: time+muscle+time*muscle

Mauchly's Test of Sphericity[b]

Measure: MEASURE_1

					Epsilon[a]		
Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.037	59.943	35	.007	.591	.784	.125
muscle	1.000	.000	0	.	1.000	1.000	1.000
time * muscle	.047	55.493	35	.018	.604	.807	.125

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: time+muscle+time*muscle

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	4.70E-007	8	5.88E-008	2.939	.004
	Greenhouse-Geisser	4.70E-007	4.729	9.95E-008	2.939	.018
	Huynh-Feldt	4.70E-007	6.271	7.50E-008	2.939	.009
	Lower-bound	4.70E-007	1.000	4.70E-007	2.939	.101
Error(time)	Sphericity Assumed	3.36E-006	168	2.00E-008		
	Greenhouse-Geisser	3.36E-006	99.307	3.38E-008		
	Huynh-Feldt	3.36E-006	131.691	2.55E-008		
	Lower-bound	3.36E-006	21.000	1.60E-007		
muscle	Sphericity Assumed	7.36E-005	1	7.36E-005	115.581	.000
	Greenhouse-Geisser	7.36E-005	1.000	7.36E-005	115.581	.000
	Huynh-Feldt	7.36E-005	1.000	7.36E-005	115.581	.000
	Lower-bound	7.36E-005	1.000	7.36E-005	115.581	.000
Error(muscle)	Sphericity Assumed	1.34E-005	21	6.37E-007		
	Greenhouse-Geisser	1.34E-005	21.000	6.37E-007		
	Huynh-Feldt	1.34E-005	21.000	6.37E-007		
	Lower-bound	1.34E-005	21.000	6.37E-007		
time * muscle	Sphericity Assumed	6.08E-007	8	7.60E-008	4.273	.000
	Greenhouse-Geisser	6.08E-007	4.835	1.26E-007	4.273	.002
	Huynh-Feldt	6.08E-007	6.456	9.42E-008	4.273	.000
	Lower-bound	6.08E-007	1.000	6.08E-007	4.273	.051
Error(time*muscle)	Sphericity Assumed	2.99E-006	168	1.78E-008		
	Greenhouse-Geisser	2.99E-006	101.531	2.94E-008		
	Huynh-Feldt	2.99E-006	135.581	2.20E-008		
	Lower-bound	2.99E-006	21.000	1.42E-007		

Simple Main Effects Analysis: One-way ANOVA for Trained Arm Agonist (biceps) EMG and Multiple Pairwise Comparisons (Bonferroni Adjusted)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	1.07E-006	8	1.33E-007	3.569	.001
	Greenhouse-Geisser	1.07E-006	4.765	2.24E-007	3.569	.006
	Huynh-Feldt	1.07E-006	6.334	1.68E-007	3.569	.002
	Lower-bound	1.07E-006	1.000	1.07E-006	3.569	.073
Error(time)	Sphericity Assumed	6.28E-006	168	3.74E-008		
	Greenhouse-Geisser	6.28E-006	100.065	6.28E-008		
	Huynh-Feldt	6.28E-006	133.011	4.72E-008		
	Lower-bound	6.28E-006	21.000	2.99E-007		

Pairwise Comparisons

Measure: MEASURE_1

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	2.18E-005	.000	1.000	.000	.000
	3	.000	.000	1.000	.000	.000
	4	.000	.000	1.000	.000	8.98E-005
	5	.000	.000	.130	.000	2.30E-005
	6	.000	.000	.380	.000	6.22E-005
	7	.000	.000	.877	.000	8.07E-005
	8	.000	.000	.121	.000	1.69E-005
	9	-9.10E-005	.000	.752	.000	4.31E-005
2	1	-2.18E-005	.000	1.000	.000	.000
	3	.000	.000	.766	.000	7.81E-005
	4	.000	.000	.165	.000	2.45E-005
	5	.000*	.000	.002	.000	-5.50E-005
	6	.000	.000	.110	.000	2.21E-005
	7	.000	.000	.268	.000	4.32E-005
	8	.000*	.000	.007	.000	-3.14E-005
	9	.000*	.000	.047	.000	-8.48E-007
3	1	.000	.000	1.000	.000	.000
	2	.000	.000	.766	-7.81E-005	.000
	4	1.03E-005	.000	1.000	.000	.000
	5	-4.42E-005	.000	1.000	.000	.000
	6	-5.88E-005	.000	1.000	.000	.000
	7	-1.48E-005	.000	1.000	.000	.000
	8	-7.61E-006	.000	1.000	.000	.000
	9	5.02E-005	.000	1.000	.000	.000
4	1	.000	.000	1.000	-8.98E-005	.000
	2	.000	.000	.165	-2.45E-005	.000
	3	-1.03E-005	.000	1.000	.000	.000
	5	-5.46E-005	.000	1.000	.000	.000
	6	-6.91E-005	.000	1.000	.000	.000
	7	-2.52E-005	.000	1.000	.000	.000
	8	-1.80E-005	.000	1.000	.000	.000
	9	3.99E-005	.000	1.000	.000	.000
5	1	.000	.000	.130	-2.30E-005	.000
	2	.000*	.000	.002	5.50E-005	.000
	3	4.42E-005	.000	1.000	.000	.000
	4	5.46E-005	.000	1.000	.000	.000
	6	-1.46E-005	.000	1.000	.000	.000
	7	2.94E-005	.000	1.000	.000	.000
	8	3.66E-005	.000	1.000	.000	.000
	9	9.45E-005	.000	1.000	-6.35E-005	.000
6	1	.000	.000	.380	-6.22E-005	.000
	2	.000	.000	.110	-2.21E-005	.000
	3	5.88E-005	.000	1.000	.000	.000
	4	6.91E-005	.000	1.000	.000	.000
	5	1.46E-005	.000	1.000	.000	.000
	7	4.40E-005	.000	1.000	.000	.000
	8	5.12E-005	.000	1.000	.000	.000
	9	.000	.000	1.000	-9.98E-005	.000
7	1	.000	.000	.877	-8.07E-005	.000
	2	.000	.000	.268	-4.32E-005	.000
	3	1.48E-005	.000	1.000	.000	.000
	4	2.52E-005	.000	1.000	.000	.000
	5	-2.94E-005	.000	1.000	.000	.000
	6	-4.40E-005	.000	1.000	.000	.000
	8	7.22E-006	.000	1.000	.000	.000
	9	6.51E-005	.000	1.000	.000	.000
8	1	.000	.000	.121	-1.69E-005	.000
	2	.000*	.000	.007	3.14E-005	.000
	3	7.61E-006	.000	1.000	.000	.000
	4	1.80E-005	.000	1.000	.000	.000
	5	-3.66E-005	.000	1.000	.000	.000
	6	-5.12E-005	.000	1.000	.000	.000
	7	-7.22E-006	.000	1.000	.000	.000
	9	5.78E-005	.000	1.000	-5.38E-005	.000
9	1	9.10E-005	.000	.752	-4.31E-005	.000
	2	.000*	.000	.047	8.48E-007	.000
	3	-5.02E-005	.000	1.000	.000	.000
	4	-3.99E-005	.000	1.000	.000	.000
	5	-9.45E-005	.000	1.000	.000	6.35E-005
	6	.000	.000	1.000	.000	9.98E-005
	7	-6.51E-005	.000	1.000	.000	.000
	8	-5.78E-005	.000	1.000	.000	5.38E-005

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

Simple Main Effects Analysis: One-way ANOVA for Trained Arm Antagonist (triceps) EMG and Multiple Pairwise Comparisons (Bonferroni Adjusted)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	1.13E-008	8	1.42E-009	3.431	.001
	Greenhouse-Geisser	1.13E-008	3.244	3.50E-009	3.431	.019
	Huynh-Feldt	1.13E-008	3.907	2.90E-009	3.431	.013
	Lower-bound	1.13E-008	1.000	1.13E-008	3.431	.078
Error(time)	Sphericity Assumed	6.94E-008	168	4.13E-010		
	Greenhouse-Geisser	6.94E-008	68.127	1.02E-009		
	Huynh-Feldt	6.94E-008	82.046	8.46E-010		
	Lower-bound	6.94E-008	21.000	3.30E-009		

Pairwise Comparisons

Measure: MEASURE_1

(I) time	(J) time	Mean Difference (I-J)	Std. Error	Sig.[a]	95% Confidence Interval for Difference[a]	
					Lower Bound	Upper Bound
1	2	-1.75E-006	.000	1.000	-2.18E-005	1.83E-005
	3	3.77E-006	.000	1.000	-2.97E-005	3.72E-005
	4	1.27E-005	.000	1.000	-2.30E-005	4.84E-005
	5	1.07E-005	.000	1.000	-2.18E-005	4.33E-005
	6	1.61E-005	.000	1.000	-1.24E-005	4.46E-005
	7	4.64E-006	.000	1.000	-3.02E-005	3.95E-005
	8	2.01E-005	.000	.518	-7.62E-006	4.78E-005
	9	1.81E-005	.000	1.000	-1.36E-005	4.99E-005
2	1	1.75E-006	.000	1.000	-1.83E-005	2.18E-005
	3	5.52E-006	.000	1.000	-1.91E-005	3.01E-005
	4	1.44E-005	.000	1.000	-1.43E-005	4.32E-005
	5	1.25E-005	.000	1.000	-1.33E-005	3.82E-005
	6	1.79E-005	.000	.225	-3.78E-006	3.95E-005
	7	6.40E-006	.000	1.000	-2.16E-005	3.44E-005
	8	2.18E-005*	.000	.033	1.01E-006	4.27E-005
	9	1.99E-005	.000	.342	-5.77E-006	4.56E-005
3	1	-3.77E-006	.000	1.000	-3.72E-005	2.97E-005
	2	-5.52E-006	.000	1.000	-3.01E-005	1.91E-005
	4	8.92E-006	.000	1.000	-1.35E-005	3.13E-005
	5	6.94E-006	.000	1.000	-9.19E-006	2.31E-005
	6	1.24E-005	.000	.171	-2.06E-006	2.68E-005
	7	8.77E-007	.000	1.000	-1.80E-005	1.97E-005
	8	1.63E-005*	.000	.006	3.22E-006	2.94E-005
	9	1.44E-005	.000	.059	-2.90E-007	2.91E-005
4	1	-1.27E-005	.000	1.000	-4.84E-005	2.30E-005
	2	-1.44E-005	.000	1.000	-4.32E-005	1.43E-005
	3	-8.92E-006	.000	1.000	-3.13E-005	1.35E-005
	5	-1.98E-006	.000	1.000	-1.81E-005	1.41E-005
	6	3.44E-006	.000	1.000	-1.88E-005	2.57E-005
	7	-8.04E-006	.000	1.000	-3.02E-005	1.41E-005
	8	7.39E-006	.000	1.000	-1.05E-005	2.53E-005
	9	5.46E-006	.000	1.000	-1.23E-005	2.32E-005
5	1	-1.07E-005	.000	1.000	-4.33E-005	2.18E-005
	2	-1.25E-005	.000	1.000	-3.82E-005	1.33E-005
	3	-6.94E-006	.000	1.000	-2.31E-005	9.19E-006
	4	1.98E-006	.000	1.000	-1.41E-005	1.81E-005
	6	5.43E-006	.000	1.000	-1.08E-005	2.17E-005
	7	-6.06E-006	.000	1.000	-2.76E-005	1.55E-005
	8	9.38E-006	.000	1.000	-5.71E-006	2.45E-005
	9	7.45E-006	.000	1.000	-4.37E-006	1.93E-005
6	1	-1.61E-005	.000	1.000	-4.46E-005	1.24E-005
	2	-1.79E-005	.000	.225	-3.95E-005	3.78E-006
	3	-1.24E-005	.000	.171	-2.68E-005	2.06E-006
	4	-3.44E-006	.000	1.000	-2.57E-005	1.88E-005
	5	-5.43E-006	.000	1.000	-2.17E-005	1.08E-005
	7	-1.15E-005	.000	1.000	-3.16E-005	8.64E-006
	8	3.95E-006	.000	1.000	-7.21E-006	1.51E-005
	9	2.02E-006	.000	1.000	-1.24E-005	1.64E-005
7	1	-4.64E-006	.000	1.000	-3.95E-005	3.02E-005
	2	-6.40E-006	.000	1.000	-3.44E-005	2.16E-005
	3	-8.77E-007	.000	1.000	-1.97E-005	1.80E-005
	4	8.04E-006	.000	1.000	-1.41E-005	3.02E-005
	5	6.06E-006	.000	1.000	-1.55E-005	2.76E-005
	6	1.15E-005	.000	1.000	-8.64E-006	3.16E-005
	8	1.54E-005	.000	.161	-2.42E-006	3.33E-005
	9	1.35E-005	.000	.203	-2.62E-006	2.96E-005
8	1	-2.01E-005	.000	.518	-4.78E-005	7.62E-006
	2	-2.18E-005*	.000	.033	-4.27E-005	-1.01E-006
	3	-1.63E-005*	.000	.006	-2.94E-005	-3.22E-006
	4	-7.39E-006	.000	1.000	-2.53E-005	1.05E-005
	5	-9.38E-006	.000	1.000	-2.45E-005	5.71E-006
	6	-3.95E-006	.000	1.000	-1.51E-005	7.21E-006
	7	-1.54E-005	.000	.161	-3.33E-005	2.42E-006
	9	-1.93E-006	.000	1.000	-1.66E-005	1.27E-005
9	1	-1.81E-005	.000	1.000	-4.99E-005	1.36E-005
	2	-1.99E-005	.000	.342	-4.56E-005	5.77E-006
	3	-1.44E-005	.000	.059	-2.91E-005	2.90E-007
	4	-5.46E-006	.000	1.000	-2.32E-005	1.23E-005
	5	-7.45E-006	.000	1.000	-1.93E-005	4.37E-006
	6	-2.02E-006	.000	1.000	-1.64E-005	1.24E-005
	7	-1.35E-005	.000	.203	-2.96E-005	2.62E-006
	8	1.93E-006	.000	1.000	-1.27E-005	1.66E-005

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

a. Adjustment for multiple comparisons: Bonferroni.

2 x 4 (muscle x time) Factorial ANOVA for Untrained Arm EMG

Multivariate Tests[b]

Effect		Value	F	Hypothesis df	Error df	Sig.
time	Pillai's Trace	.103	.724[a]	3.000	19.000	.550
	Wilks' Lambda	.897	.724[a]	3.000	19.000	.550
	Hotelling's Trace	.114	.724[a]	3.000	19.000	.550
	Roy's Largest Root	.114	.724[a]	3.000	19.000	.550
muscle	Pillai's Trace	.790	79.028[a]	1.000	21.000	.000
	Wilks' Lambda	.210	79.028[a]	1.000	21.000	.000
	Hotelling's Trace	3.763	79.028[a]	1.000	21.000	.000
	Roy's Largest Root	3.763	79.028[a]	1.000	21.000	.000
time * muscle	Pillai's Trace	.064	.430[a]	3.000	19.000	.734
	Wilks' Lambda	.936	.430[a]	3.000	19.000	.734
	Hotelling's Trace	.068	.430[a]	3.000	19.000	.734
	Roy's Largest Root	.068	.430[a]	3.000	19.000	.734

a. Exact statistic

b.
Design: Intercept
Within Subjects Design: time+muscle+time*muscle

Mauchly's Test of Sphericity[b]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
time	.592	10.325	5	.067	.800	.910	.333
muscle	1.000	.000	0	.	1.000	1.000	1.000
time * muscle	.685	7.467	5	.189	.845	.971	.333

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: time+muscle+time*muscle

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
time	Sphericity Assumed	1.50E-008	3	5.00E-009	.359	.783
	Greenhouse-Geisser	1.50E-008	2.400	6.25E-009	.359	.738
	Huynh-Feldt	1.50E-008	2.731	5.49E-009	.359	.764
	Lower-bound	1.50E-008	1.000	1.50E-008	.359	.555
Error(time)	Sphericity Assumed	8.77E-007	63	1.39E-008		
	Greenhouse-Geisser	8.77E-007	50.405	1.74E-008		
	Huynh-Feldt	8.77E-007	57.361	1.53E-008		
	Lower-bound	8.77E-007	21.000	4.18E-008		
muscle	Sphericity Assumed	2.26E-005	1	2.26E-005	79.028	.000
	Greenhouse-Geisser	2.26E-005	1.000	2.26E-005	79.028	.000
	Huynh-Feldt	2.26E-005	1.000	2.26E-005	79.028	.000
	Lower-bound	2.26E-005	1.000	2.26E-005	79.028	.000
Error(muscle)	Sphericity Assumed	6.00E-006	21	2.86E-007		
	Greenhouse-Geisser	6.00E-006	21.000	2.86E-007		
	Huynh-Feldt	6.00E-006	21.000	2.86E-007		
	Lower-bound	6.00E-006	21.000	2.86E-007		
time * muscle	Sphericity Assumed	1.99E-008	3	6.65E-009	.567	.639
	Greenhouse-Geisser	1.99E-008	2.535	7.87E-009	.567	.611
	Huynh-Feldt	1.99E-008	2.912	6.85E-009	.567	.634
	Lower-bound	1.99E-008	1.000	1.99E-008	.567	.460
Error(time*muscle)	Sphericity Assumed	7.39E-007	63	1.17E-008		
	Greenhouse-Geisser	7.39E-007	53.234	1.39E-008		
	Huynh-Feldt	7.39E-007	61.150	1.21E-008		
	Lower-bound	7.39E-007	21.000	3.52E-008		

Appendix F Muscle and Joint Soreness Breakdown

	Bicep			Elbow			Forearm			Shoulder			Wrist/ Hand		
Subject Number	Week 1	Week 2	Week3	Week 1	Week 2	Week3	Week 1	Week 2	Week3	Week 1	Week 2	Week3	Week 1	Week 2	Week3
1	0	0	0	4	0	0	0	0	0	0	0	0	0	6	7.5
2	7	5	1	0	0	0	5	2	1	0	0	0	0	0	0
3	6	3	1	4	2	0	3	0	0	0	0	0	0	0	0
4	7	6	4	5	5	5	0	0	0	8	7	5	0	0	0
6	6	1	0	0	0	0	0	0	0	0	0	0	3	5	6
7	6	3	0	3	4	4	0	0	0	0	0	0	3	3	3
8	6	0	0	3	0	0	5	0	0	0	0	0	0	0	0
9	9	4	0	4	2	0	4	2	0	6	2	0	0	0	0
10	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0
11	6	3	0	0	0	0	0	0	0	0	0	0	0	0	0
12	4	6	3	1	3	6	2	2	2	0	0	0	1	2	3
13	6	0	0	0	0	0	5	0	0	0	0	0	0	0	0
14	6	3	1	3	1	1	3	2	1	5	3	1	3	1	1
15	5	2	0	8	5	2	0	0	0	0	0	0	0	0	0
16	4	0	0	0	3	0	0	0	0	0	0	0	0	0	0
17	7	4	0	0	0	0	0	0	0	0	0	0	0	0	0
18	9	6	3	7	3	0	6	2	0	0	0	0	0	0	0
19	5	3	0	2	2	0	3	3	3	4	0	0	0	0	0
20	3	2	1	0	0	0	0	0	0	0	0	0	0	0	0
21	0	0	0	5	0	0	0	0	0	0	0	0	3	3	0
22	6	4	2	3	0	0	0	4	0	0	0	0	0	0	0
23	5	0	0	0	0	8	7	0	0	6	0	0	0	0	0