EFFECTS OF SHORT-TERM RESISTANCE TRAINING ON MOTOR UNIT PROPERTIES AND NEUROMUSCULAR FUNCTION IN YOUNG AND OLD MEN

By

GARRETT HESTER

Bachelor of Science in Health and Human Performance
Northeastern State University
Tahlequah, OK
2010

Master of Science in Health and Human Performance
Oklahoma State University
Stillwater, OK
2012

Submitted to the Faculty of the
Graduate College of the
Oklahoma State University
in partial fulfillment of
the requirements for
the Degree of
DOCTOR OF PHILOSOPHY
July, 2016

EFFECTS OF SHORT-TERM RESISTANCE TRAINING ON MOTOR UNIT PROPERTIES AND NEUROMUSCULAR FUNCTION IN YOUNG AND OLD MEN

Dissertation Approved:

Dr. Jason M. DeFreitas

Dissertation Adviser

Dr. Doug B. Smith

Dr. Aric J. Warren

Dr. Robert E. Davis

ACKNOWLEDGEMENTS

My Family:

To my mom, Brenda, who consistently encouraged me to strive for my goals – I am very grateful for your early guidance which allowed me to reach this destination.

To my dad, Bill, and sisters Shawna and Deena – Thank you for your support and providing me with the assistance I needed throughout my journey.

To my fiancé, Hailey – Thank you for your continuous patience and support throughout this process. I am lucky to have such a positive and caring person in my life!

My professors and colleagues:

To Drs. Doug B. Smith, Aric J. Warren, and Robert E. Davis – Your combined insights and efforts were highly influential during this process. Thank you all for sharing your knowledge and guiding me through this process.

To Zachary K. Pope, Mitchel A. Magrini, Ryan J. Colquhoun, Carlos A. Estrada, Ryan M. Thiele, Ty B. Palmer, Eric C. Conchola, and Alejandra Barrera-Curiel – I am grateful for your assistance with this project and for having the opportunity to work with you all.

To Jason M. DeFreitas, my mentor – Your leadership has provided me with the tools needed to be successful exploring my interests and teaching. My accomplishments are possible because of your thoughtful and directed instruction. It was truly a privilege working under your guidance.

Acknowledgements reflect the views of the author and are not endorsed by committee members or Oklahoma State University.

Name: GARRETT HESTER

Date of Degree: JULY, 2016

Title of Study: EFFECTS OF SHORT-TERM RESISTANCE TRAINING ON MOTOR UNIT PROPERTIES AND NEUROMUSCULAR FUNCTION IN YOUNG AND OLD MEN

Major Field: HEALTH, LEISURE AND HUMAN PERFORMANCE

Aging is accompanied by decreases in neuromuscular functioning that can lead to decreased mobility and quality of life in older adults. The purpose of this study was two-fold: 1) To investigate the age-related differences in maximum motor unit firing rates and motor unit action potential size. 2) To examine the effects of short-term resistance training on motor unit properties and neuromuscular function in young and old men. Twenty-five young (range: 18-30) and twenty-three older adults (range: 55-88 yrs) volunteered to participate in this study and were randomly assigned to either a training or control group. The training groups performed 3 sessions of unilateral resistance training per week for 4 weeks, while the control group only performed the testing. Testing was performed for the trained and untrained leg before (PRE), after 2 weeks (MID), and after 4 weeks (POST) of training. Motor unit properties including maximum motor unit firing rates (MaxFR), motor unit action potential size (MUAP-size), and recruitment threshold (RT) were obtained through the use of decomposing surface electromyography. Linear regression was used for the MaxFR and RT as well as the MUAP-size relationship in order to obtain slope coefficients ($MaxFR_{SLOPE}$ and $MUAP\text{-}size_{SLOPE}$, respectfully). In addition, strength, peak velocity (PV), rate of velocity development (RVD), and rate of muscle activation (RER) were measured during each testing visit. Older adults exhibited a decreased $MUAP\text{-}size_{SLOPE}$ at PRE compared to younger adults, but $MaxFR_{SLOPE}$ was similar between groups. $MaxFR_{SLOPE}$ increased at MID and POST, while $MUAP\text{-}size_{SLOPE}$ increased at POST in both groups with no age-related differences being demonstrated for either. Older adults exhibited an attenuated response to the training as young training group demonstrated larger gains after 2 and 4 weeks of resistance training. While RVD (unloaded knee extension) for the trained leg remained unchanged, it increased for the untrained leg in both, the young and old training group. These findings provide preliminary evidence of aging- and training-related alterations in MU morphology Also, new information was provided that may contribute to understanding the functional implications for cross-education.

TABLE OF CONTENTS

Chapter Page

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

1.1. Introduction

It is well established that aging is associated with a decline in neuromuscular function (i.e., muscle strength, power, rate of force development (RFD)) that can lead to decreased mobility[13, 109], a lower quality of life[94], and an increased risk of mortality[74, 82] in older adults (≥60 yrs). Neuromuscular function is optimal during approximately the 3rd decade[63], begins to decrease shortly thereafter and rapidly declines in the 6th or 7th decade[52, 102, 103]. Furthermore, measures characterizing explosive neuromuscular function (e.g., power, RFD, rate of velocity development (RVD)) are augmented drastically more with aging compared to maximal strength[54, 61, 98, 101]. Indeed, lower-body strength and power were reported to decrease at a rate of 1-2% and 3.5% per annum, respectively[98]. The majority of research investigating the age-related impairments in neuromuscular function has focused on muscles of the lower-body due to the importance of the lower-body musculature for locomotion and physical functioning. While the effects of aging on the aforementioned performance characteristics and the time-line of these changes are relatively well established, there is much still to be elucidated regarding potential changes within the central nervous system (CNS) and the implications of these changes on motor control in older adults.

Considering the role of the CNS in mediating the control of human movement, impairments of the CNS due to aging likely have a strong contribution to the age-related decline in functionality. Indeed, the motor unit (MU) which consists of the motor neuron, its axon, and the muscle fibers innervated by the axon is the final common pathway between the nervous system and the muscle, and thus represents the basic functional element for human force production[45]. An increase in either the number of MUs activated (i.e., MU recruitment) or rate of impulse firings (i.e., rate coding) will increase voluntary force production during a muscle contraction. As the demand for force production increases, MUs are recruited in the order of their size such that the largest MUs are the last to be recruited[42, 76]. The functional behavior for a group of motor neurons that innervate a single muscle (i.e., motor pool) and their constituent muscle fibers vary drastically[45]. The larger, later-recruited (i.e., high-threshold, type II) MUs are believed to have muscle fibers with a greater cross-sectional area (CSA) compared to the smaller, earlier-recruited (low-threshold, type I) MUs as indicated by reports of recruitment threshold (RT; % maximum strength) being positively related to twitch tension[42, 76, 99] and MU action potential size (MUAP-size)[42, 89]. MUAP-size was demonstrated to vary as a function of single muscle fiber diameter in which larger fibers had a larger MUAP-size compared to smaller fibers[43]. During high-force contractions, low-threshold MUs will demonstrate higher firing rates as compared to higher-threshold MUs in order to sustain a high degree of force output while limiting fatigue in the higher-threshold MUs[25]. Thus, MU behavior directly influences the efferent drive delivered to the muscle. The study of MU properties is important for gaining insight on the neural control of movement.

Previous studies have used intramuscular electromyography (iEMG; e.g., fine-wire) to investigate changes in MU properties due to aging. This technique along with cadaveric studies have demonstrated an age-related reorganization of the MU pool for many muscles as a result of the denervation-reinnervation process[4]. Indeed, in addition to potential age-related changes in supraspinal function, MU properties may be altered due to a change in either the muscle unit or the motor nerve innervating the muscle unit. For example, previous research has shown that maximal MU firing rates are decreased[19, 56] in older adults and there is a reduction in the number of MUs for various muscles[15, 30]. While this work has provided a great deal of insight thus far, there are limitations associated with using iEMG. Firstly, the procedure is invasive as the electrode must be inserted via a needle for proper placement within the muscle. Secondly, traditional iEMG techniques are only able record a very small sample of MUs (i.e., 3-5) during a muscle contraction, although recent technological developments allow for the recording of ~15 MUs[36]. Thirdly, studies using this technique typically refrain from high-intensity contractions due to the potential for superposition of MU action potentials. Thus, there is a paucity of information regarding the effects of aging on MU properties across the recruitment threshold (RT) continuum during maximal force production. Given that the behavior of low- and high-threshold MUs differ during a voluntary contraction and aging changes the organization of a MU pool, it is important to investigate MUs across a larger range of the RT continuum. Especially, if the purpose is to investigate the potential contribution of MU properties to deficits in maximal strength in an aged population. Recent technological advancements allow for the non-invasive recording of ~30-50 MUs via a 5-pin surface array sensor during a maximal intensity ramp isometric contraction[24]. Thus, this technology provides a means to study properties of low- and high-

threshold MUs such as maximal MU firing rates and MUAP-size during a maximal voluntary contraction.

It is well known that aging is associated with a reduction in muscle mass (i.e., sarcopenia). Undoubtedly, the age-related reduction in muscle mass has an influential role on the decrements in neuromuscular function that are reported in older adults. Sarcopenia is due to a combination of both, a decrease in the number[67] and size[63, 84] of muscle fibers. However, it appears that type II muscle fibers are more affected compared to type I muscle fibers[67, 84] and that a decrease in the size of type II muscle fibers may be the largest contributor to the overall decrease in muscle mass[84] for the vastus lateralis (VL). iEMG has been used to record MUAP-size to investigate age-related changes in MU morphology (i.e., fiber size) based on the assumption that MUAP-size is dependent upon fiber diameter[43]. MUAP-size tends to be increased in older adults[16, 31, 73] and this is believed to be the result of an increased muscle unit due to reinnervation. However, these findings are from studies which investigated low-intensity muscle contractions. Thus, the sampled MUs may not have appropriately represented the entire MU pool and may have predominantly reflected the behavior of low-threshold MUs. The effect of age on a larger representation of the MU pool (i.e., low- and high-threshold MUs) has not been investigated. Recently, in young adults, the change in the relationship between MUAP-size and RT after resistance training was found to be closely correlated with changes in whole muscle CSA of the VL[89]. Thus, MUAP-size as derived from the decomposition of surface EMG signals may provide a non-invasive measure of changes in MU specific atrophy as a result of sarcopenia. This technology has yet to be used to compare MUAP-size between young and older adults.

Resistance training has proven effective at reversing some of the effects aging has on neuromuscular function, even after short-term exposure. For example, improvements in maximal strength[8, 19, 86] occur similarly in older adults as compared to young adults after only 2 to 4 weeks of resistance training. However, less is known regarding the effects of resistance training on explosive performance parameters. For example, there are no reports on the effects of short-term resistance training on maximum velocity characteristics of the knee extensors in young and older adults. This is unfortunate considering the role of velocity for power development and the preferential decrease in power as compared to maximal strength in older adults. Several studies have investigated the age-related differences in MU adaptations induced by resistance training using iEMG. Resistance training has been shown to increase maximal MU firing rates after only a few weeks[19, 85], and even after only 2 sessions[56, 86]. However, due to the previously mentioned constraints of iEMG, whether or not there are age-related differences in the training-induced changes in MU firing rates across the RT is unclear. Taking into consideration that MU properties may be altered during the aging process, it is important to elucidate whether or not training-induced MU adaptations are different in older adults compared to their younger counterparts.

Cross-education, a training-induced increase in strength of the untrained limb after unilateral resistance training, is a commonly observed neural adaption to resistance training. Cross-education plays an important role in clinical populations in which paralysis or limb immobilization can lead to rapid losses in strength[46]. While there is evidence that age does not negatively affect cross-education, these studies investigated the upper-body, therefore; it

is unclear if cross-education for the lower-body is similar between young and older adults. While the specific location of the mechanism responsible for cross-education has remained elusive, a neural mechanism is believed to be responsible for this adaptation. MU properties for the untrained limb after lower-body resistance training have not been investigated, thus whether or not changes in MU properties are linked to cross-education of the lower-limb muscles is unknown. Furthermore, the majority of studies investigating cross-education have focused on the cross-transfer of strength. Due to potential benefit of cross-education for older adults, the ability of explosive neuromuscular function parameters (e.g., peak velocity, RVD) to be cross-transferred needs requires investigation.

There are numerous neuromuscular function parameters known to decrease throughout the aging process, however, the underlying mechanisms responsible for these decrements are not as clear. Due to technological advancements, the ability to detect a large sample of MUs across the RT continuum provides an opportunity for gaining insight on age-related or training-related changes in the neural control of movement. In addition, the effect of age on the short-term adaptations of explosive neuromuscular capacity requires further investigation due to a paucity of research on these measures and the importance of these parameters for physical functioning in older adults.

1.2. Purpose of the Study

The purpose of this study was two-fold: 1) To investigate the age-related differences in motor unit action potential size. 2) To examine the effects of short-term resistance training on motor unit properties and neuromuscular function in young and old men.

1.3. Research Questions

This study had the potential to provide new information related to the effects of aging on motor unit properties and the age-related differences in the adaptation of motor unit properties and neuromuscular function after short-term resistance training. The following research questions had the potential to be answered by the present study and had not been answered in the literature:

- What is the effect of age on MUAP-size?

- Does unilateral resistance training affect maximal MU firing rates of the untrained leg?
    - Is this effect dependent upon age?

- Does short-term resistance training affect MUAP-size?
    - Is this effect dependent upon age?

- Are there age-related differences in the adaptability of maximal velocity characteristics in the trained leg during short-term strength training?

- Is there a cross-transfer effect for maximal velocity characteristics during short-term strength training?

- Is this effect dependent upon age?

- Is there a cross-transfer effect for rate of muscle activation during short-term strength training?
  - Is this effect dependent upon age?
  - Is this effect velocity specific?

1.4. Hypotheses

1. MUAP-size$_{SLOPE}$ will be significantly smaller in older adults compared to young adults.

2. Resistance training will increase MUAP-size$_{SLOPE}$ in the trained leg for the young adults, but not for the older adults.

3. There will be no effect of unilateral resistance training on MUAP-size$_{SLOPE}$ in the untrained limb for either age group.

4. Short-term unilateral resistance training will increase MaxFR$_{SLOPE}$ of the untrained leg in the old group only.

5. Neither the young nor old group will increase PV or RVD for IsoK$_{500}$ in the trained or untrained leg during short-term resistance training.

6. Young and older adults will increase RVD for IsoK$_{45}$ in the untrained leg.

7. RER for $IsoK_{45}$ will be increased for the untrained leg in the young and old group.

1.5. Significance of the Study

This study provided novel evidence regarding the effects of aging and resistance training on select MU properties. This information will improve our understanding of the changes in the nervous system that contribute to decreased physical functioning and how training-induced adaptations may be altered by age. Evidence was brought forth regarding the cross-transfer of novel neuromuscular performance measures after unilateral training, which may benefit clinicians who are interested in maintaining the physical capacity of a single limb. Furthermore, age-related differences in the adaptations of novel explosive performance parameters were examined in this study, thus adding to our understanding how aging affects training-induced changes. This may be especially important for practitioners whose purpose is to assist with maintaining or improving physical functioning for older clientele.

1.6. Delimitations

1. Participants had to be between 18-30 or 55 years of age and older.

2. All participants had to be healthy, and free of neuromuscular disease as self-reported on a questionnaire.

3. All participants were untrained (i.e., unfamiliar with resistance training) as self-reported on a questionnaire.

4. The participants only performed voluntary contractions.

5. Only one group of muscles (i.e., knee extensors) of the dominant leg were trained.

1.7. Limitations

1. Participants responded to either a posted advertisement or an informational announcement and chose to volunteer on a volunteer basis. Thus, the process of subject selection was not truly random.

2. The technology and equipment used to assess motor unit properties has many restrictions, including:
    a. Contractions must be isometric
    b. The force profile must be trapezoidal in shape, characterized by a slow linear increase in force followed by a linear decrease.

3. The higher force levels used in this study led to shorter action potential trains than those commonly demonstrated in most of the literature.

4. Differences in motivation levels between participants may produce varying levels of maximal exertions for maximal contractions.

5. EMG was not recorded during the training sessions to confirm that the untrained

leg remained in a rested state.

1.8. Assumptions

1. Subjects responded to the health and exercise history questionnaire accurately and honestly.

2. All subjects gave a maximal voluntary effort on all strength tests.

3. The equipment is appropriately calibrated and functioning properly.

4. There are no data collection, data analyses, data entry or statistical processing errors.

5. The samples for the younger and older groups are similarly represented in terms of the population and their relative physical fitness levels when compared to the norms for their respective age groups.

2. REVIEW OF LITERATURE

2.1. Effects of Aging on Motor Unit Properties and Neuromuscular Function

Campbell et al., 1973[16]

In this seminal study, Campbell and colleagues are among the first to demonstrate the association between aging and motor unit dysfunction. This study examined the number of MUs along with their physiological properties in a 'control' group of seventy-two young- to middle-aged individuals (3-58 yrs.) and made comparisons with an elderly group (60-96 yrs.). Muscle function of the extensor halluces brevis was impaired as indicated by a reduced maximal M-wave and peak twitch torque in the elderly group compared to the control group. In the control group, there was no indication a decrease in the number of functioning MUs through the entire age spectrum of 3-58 yrs. However, while there was a great deal of variability in the elderly group, many individuals in this group exhibited a decrease (~30%) in the number of functioning MUs and this trend was exacerbated with advancing age. MU action potential size was found to be larger in the elderly group than the control group. The authors conclude that even in the elderly remaining MUs participate in compensatory changes in order maintain muscle function. Also, while the denervation process may vary considerably among individuals, it likely does not commence until the age of 60 yrs.

Tomlinson and Irving, 1977[107]

In this cadaveric study, Tomlinson and Irving, were among the first to provide evidence of the number of MNs present within the lumbosacral segments of the human spinal cord. The number MNs were counted in forty-seven spinal cords derived from healthy individuals ranging in age from 13-95 yrs. Although no statistical tests were performed, comparisons amongst the mean differences for each 10 yr. age group (e.g., 11-20 yrs., 21-30 yrs., etc.) were made. There appeared to be minimal MU loss from 13-60 yrs. of age as indicated by the correlation between age and mean number of MUs (r = -0.067, $p > 0.70$). In contrast, when the same correlation was examined for individuals from 63-95 yrs. of age a sharp age-associated decrease in MU numbers was revealed (r = -0.543, $p < 0.01$). Furthermore, the mean number of MUs for all individuals up to 60 yrs. of age was 59,000 compared with approximately 52,000 in the seventh decade, 46,000 in the eighth and ninth decade and 41,000 in the tenth decade. They concluded that the degree MN death may be negligible until the age of 60, but is considerable beyond this point and becomes substantial in advanced years.

Larsson et al., 1979[63]

Larsson and colleagues compared the influence of age on isometric and dynamic strength, along with speed of movement, and examined the correlation between changes in mechanical performance at different ages with histochemical muscle characteristics. Both, maximum strength and maximum knee extension velocity of the quadriceps followed a similar pattern which consisted of peak values occurring in the 20-30 yrs group and a decline beginning in the 50-59 yrs group, although the relative reduction in peak velocity with age

were smaller compared to maximum strength. Type II muscle fiber area was found to decrease with age, and type II muscle atrophy was found to significantly correlate with strength loss in old age. The authors concluded that the latter finding along with the finding of a decrease in the type II/I fiber diameter ratio from the 20-29 yrs to the 80-89 yrs group are the likely mechanisms of the age-associated neuromuscular impairments.

Brown et al., 1988[15]

Brown et al. introduced the spike-triggered averaging technique in this study which examined the MUNE and MU properties in 40 healthy individuals ranging in age from 19-74 yrs. In this study the methodological approach using the spike-triggered averaging is discussed at length, however, preliminary evidence regarding the age-related reduction of MUs is provided. A slightly reduced maximal M-wave amplitude was found for the older group (>60 yrs) compared to the young group, and the MUNE was substantially lower in the old group (479 ± 220 vs. 911 ± 254). Brown and colleagues report that age-related decline calculates to approximately 10-20 MUs per year, or ~1% of total MUs per year.

Lexell et al., 1988[67]

Lexell and colleagues were the first to examine whether a reduction in muscle fiber size or muscle fiber number was the predominant cause for age-related atrophy. They found that atrophy of the vastus lateralis (VL) begins around age 25 and accelerates thereafter with approximately 10% of muscle mass being lost at the age of 50. The main cause for atrophy of the VL was a reduction in the quantity of muscle fibers regardless of fiber type, and secondly a reduction in the size of type II fibers.

Doherty et al., 1993[31]

This study used spike-triggered averaging of a sample of surface-recorded single motor unit action potentials (S-MUAP) from the biceps brachii and brachialis muscles to estimate motor unit numbers in young (22-38 yrs.) and old (60-81 yrs.) subjects. The motor unit number estimate (MUNE) was derived by dividing the maximum compound muscle action potential (maximal M-wave) by the mean S-MUAP amplitude. In addition, maximum voluntary isometric contractions (MVCs) and maximum isometric twitch contractions of the elbow flexors were recorded to evaluate the subjects' ability to maximally activate a supramaximal stimulus was given during MVC attempts. The MUNE was greatly reduced (47%) in older subjects compared to their younger counterparts, and the MUNE was significantly correlated with MVC (r = 0.521). However, the sizes of the S-MUAPs were significantly larger in older subjects (23%). The authors reported significant but less pronounced reductions in the maximum twitch contraction (33%) and MVC (33%) for the older group. Due to the fact that older subjects were able to fully activate the elbow flexors, the authors concluded that the age-related reduction in strength is likely the result of decreased muscle mass. Doherty and colleagues postulated that the age-associated similarities in S-MUAP size is the result of reorganization within the motor unit (MU) pool. Specifically, although the older group possessed a lower MUNE, collateral sprouting could increase the number of muscle fibers supplied by each remaining motor neuron causing an increase in extracellular current and therefore the S-MUAPs generated by these motor units.

Skelton et al., 1994[98]

This was the first study to examine the inter-relationships of strength, power, and functional ability in the same people. Isometric knee extensor strength and handgrip strength of ten men and ten women in each half-decade of life from age 65-89 years was assessed. In addition, functional abilities were evaluated through the performance of a chair rise, bag lift test, and box stepping test. Lower-body strength and power were found to decrease at rate of 1-2% and 3.5% per annum, respectively. In men, the age-related decline in explosive power of the lower-body was greater than the reduction in lower-body strength. The major contribution of this study was the previous finding that the age-related reduction in muscle power is more rapid than that of muscle strength.

Kamen et al., 1995[57]

Kamen and colleagues sought to determine whether maximal MU firing rates presents a potential limit to maximal force production in elderly adults. MUAPs of the first dorsal interosseous were examined in young and elderly adults during maximal and submaximal (50% MVC) isometric contractions. The average of the 5 shortest interpulse intervals (IPIs) was used to obtain maximal MU firing rates. It was determined that maximal MU firing rates during maximal contraction was lower in the elderly group compared to the young group, however; it was similar during submaximal contraction. The authors conclude that reduced maximal MU firing rates likely has a role with the well-known age-related loss of strength. Furthermore, several mechanisms are mentioned as possible reasons why neural drive from the MUs is reduced.

Izquierdo et al., 1999[52]

This studies examined age-related differences in measures of maximal and explosive strength, and investigated the relationship between these measures and balance performance. Maximal strength and rate of force development of the knee extensors was tested in young, middle-aged, and older adults. In addition, balance and explosive jumping performance was assessed with a platform. The age-related reduction in explosive strength was substantially greater than that of maximal strength. The capacity for explosive strength was not related to balance performance in the young group, however, impaired explosive strength capabilities was found to be associated with poorer balance scores. The authors conclude that resistance training programs for older individuals should not only emphasize the development of strength, but the enhancement of explosive capacity as well.

Metter et al., 2002[74]

This study examined whether muscle strength in men or rate of change in grip strength over time has an independent impact on all-cause mortality when body mass, muscle mass, and physical activity are accounted for over a 40-year period of follow-up. Lower and declining grip strength were associated with increased mortality, independent of physical activity and muscle mass. Strength was more protective than the rate of loss, when muscle mass was accounted for, for men ≥60 yrs. The authors concluded that although muscle mass and physical activity level are important, these factors do not totally account for the effect of strength on mortality.

McNeil et al., 2005[73]

McNeil et al. examined the MUNE, central activation (CA), MVC, and maximal twitch torque of the tibialis anterior in young (23-32 yrs.), old (61-69 yrs.), and very old (80-89 yrs.) healthy men. The MVC for the very old group was significantly reduced (31%) compared to the young and old group, with no difference existing between the latter two groups. Conversely, no difference among the three groups was found for central activation or peak twitch torque, although very old men had significantly slower time-to-peak torque and half-relaxation time than either the young or old men. Maximum M-wave was significantly smaller in the very old men (4.7 ± 1.0 mV) than the young (6.9 ± 1.1 mV) and old men (6.9 ± 1.0 mV), while negative-peak amplitude of the mean S-MUAP was larger in the old and very old compared to the young men. As a result of the smaller M-wave amplitude but larger S-MUAP amplitude, MUNEs were significantly reduced with age. McNeil and colleagues conclude that the enlargement of the mean S-MUAP amplitude with age provides indirect evidence of the denervation-reinnervation process associated with aged MUs. The finding of similar voluntary strength and evoked M-wave between the young and old men suggest that despite the significant MUNE loss there is a maintenance of muscle mass until a more advanced age when a critical threshold is reached. The authors postulate that upon this threshold being reached (65-80 yrs. old) the loss of larger MUs may cause substantial loss of muscle mass leading to decreased functionality.

Newman et al., 2006[82]

This longitudinal study tracked mortality rates of elderly adults for 6 years to determine whether low muscle mass would explain an association of strength with and without adjusting for hypothesized causes of sarcopenia, including physical activity, disease,

and inflammatory markers. The results demonstrated that both lower quadriceps and hand-grip strength, are strong independent predictors of mortality in older adults. Interestingly, since muscle size did not attenuate either association, the authors suggest that these associations cannot be attributed to sarcopenia. The authors suggest that tests of muscle function without accounting for muscle size may be sufficient to assess mortality risk.

Klass et al., 2008[61]

The purpose of this study was to examine the association between RTD and maximal MU firing rate in young and elderly adults during rapid submaximal contractions of the ankle dorsiflexors. Rapid strength, surface EMG, and motor unit characteristics were recorded during ballistic isometric contractions at 25, 50, and 75% MVC in you and old adults. The primary finding of this study was that older adults exhibited a slower maximal RTD (-48%) that was accompanied by a reduction in MU firing rate and number of doublet discharge. The age-related decrement observed with maximal RTD was greater than that of maximal strength (28%). A slowing of muscle contractility can explain, in part, the slower RTD demonstrated by the old adults as this group had a significantly longer contraction time (10%) than the young group during involuntary contractions. Klass and colleagues suggest that neural impairment, as indicated by reduced MU firing rate and number of doublet firings, in the old group explains part of the age-related reduction of RTD. Since antagonist co-activation was similar between groups, the authors suggest that reduced cortical drive or MN excitability are likely responsible for the observed age-related alteration in MU control.

Dalton et al., 2008[23]

In a similar study design using the spike-triggered averaging technique, the MUNE, central activation (CA), MVC, and maximal twitch torque of the soleus were examined in young (27 yrs.) and old (75 yrs.) healthy men. MVC and CA were significantly reduced in the old men compared to the young, however; peak twitch torque was similar between the two groups. In contrast, time-to-peak torque and half-relaxation times were significantly slower in the old than the young men. M-wave amplitude was found to be 38% smaller in the old men than the young. The MUNEs were similar for both groups at a contraction intensity of 10%, 20%, and 30% MVC. The authors suggest that the reduced M-wave in the old men may be due to a decrease in muscle fiber cross-sectional area (CSA) since the MUNE was similar between the two groups indicating that the number of functional MUs is similar between the old and young men. As for why there was not age-related difference in MUNE, the authors propose that the soleus may not undergo substantial the collateral reinnervation that is characteristic of the aging neuromuscular system.

Ling et al., 2009[69]

The purpose of this study was to determine whether motor unit size and firing rate change with aging during submaximal isometric contractions. MUAP size and MU firing rates during sustained isometric contractions at 10%, 20%, 30%, and 50% MVC were compared between young-, middle-, and old-aged adults. The unique aspect of this study was that researchers were able to assess S-MUAP area and MU firing rate to force ratios indicating how these measures change across intensity (10-50%). Maximum strength was highest in the 20-39 yrs. age group and progressively decreased with each of the older age groups (40-59, 60-75, ≥75 yrs.). The ≥75 yrs. age group exhibited larger S-MUAP area

compared to the younger groups, while S-MUAP area was similar amongst the younger groups. Interestingly, S-MUAP size decreased as effort increased for all age groups, except for the 20-39 yrs. age group. In regards to MU firing rate, the 60-75 yrs. and 75 yrs. and older groups, but not the 40-60 yrs. group, demonstrated higher firing rates compared to the less than 40 yrs. age group across all effort levels. Thus, it was concluded that older adults use different MU activation strategies to produce the same amount of force as compared to younger adults, and these changes become more pronounced with advanced age.

Deschenes et al., 2010[28]

The primary aim of this study was to examine whether the changes in muscle fiber profile that characterize sarcopenia (i.e., atrophy, increased percentage of type I fibers) were preceded by signs of denervation. Secondarily, to determine whether the normal activity level of a muscle influenced its susceptibility to the onset of sarcopenia. Deschenes and colleagues examined the morphology of pre- and post-synaptic neuromuscular junction (NMJ) and that of fast- and slow-twitch myofibers in young rats (10 months) and old rats (21 months). It is important to note that twenty-one months expressed relative to the average lifespan of men in the United States is the equivalent of a 62 year old man. To assess the impact that normal, daily activity levels might have on the onset of sarcopenia, the soleus and plantaris muscles were selected for study. The results showed that remodeling at both the pre- and post-synaptic NMJ preceded atrophy or fiber-type conversion of muscle. Interestingly, denervation was detected in both fast- and slow-twitch NMJs of the non-weight bearing, sparingly recruited plantaris muscle, however, little evidence of age-related denervation was observed for the regularly recruited soleus muscle. This supports the notion that the

sensitivity of age-related denervation may be influenced by neuromuscular activity. The authors also suggest that since aging affected fast- and slow-twitch NMJs similarly, large MNs may not necessarily be more likely to suffer aptosis during the aging process.

Drey et al., 2013[32]

Recently, Drey et al. used a new technique, motor unit number index (MUNIX), for the first time in this pilot study for the purpose of evaluating MN loss as a cause of sarcopenia in 27 sarcopenic (determined by gender-specific skeletal muscle mass index) individuals aged 66-90 yrs. The MUNIX value is derived from mathematical derivations based on the area and power of the maximal M-wave and voluntary surface electromyography (EMG) recordings. Motor unit size index (MUSIX) was obtained by dividing the maximal M-wave amplitude by MUNIX. Although a non-sarcopenic (control group) was not used, Drey and colleagues make comparisons with values considered to be normal for MUNIX (>80) and MUISX (>100 μV). Similar to other studies using MUNE techniques, the sarcopenic individuals were found to have low MUNIX values compared to normal. In addition, a moderate negative relationship was found for MUNIX and MUSIX indicating that sarcopenic individuals with the lowest MUNIX values demonstrate higher MU action potential size. The authors suggest that these findings indicate which individuals demonstrate neuropathic (high MUSIX and normal MUNIX) and myopathic (normal MUSIX and high MUNIX) changes due to sarcopenia.

Kaya et al., 2013[59]

Kaya and colleagues used the MUNIX technique to determine whether age-related differences in muscle strength are moderated by the number of functioning motor units. Maximal pinch-grip strength along with MUNIX and MUSIX for the abductor pollicis brevis were obtained from an old group (67 yrs.) and a young group (22 yrs.). A relationship between muscle strength and MUNIX amongst the old group was found indicating that weaker individuals exhibited a smaller MUNIX value. Kaya and colleagues conclude that a portion of muscle weakness in older adults may be attributable to the loss of functioning MUs. Furthermore, the older adults did not demonstrate a significant relationship between MUSIX and muscle strength, while the young group did. The authors suggest that this could be indicative of the weaker older adults experiencing a loss of MUs without collateral reinnervation.

Nilwik et al., 2013

This study extends on the age-related atrophy literature by combining a cross-sectional and longitudinal design to examine age-related differences in VL CSA, fiber specific CSA, and muscle fiber numbers and investigate the effects of prolonged resistance training on these parameters in older adults (71 yrs). The lower quadriceps CSA (14%) in the older group is reported to be due to age-related reduction in type II muscle fiber size (29%) as the calculated number of fibers in the VL did not differ between groups. After 6 months of resistance training, it was determined that type II muscle fiber hypertrophy explained essentially the entire training induced 9% increase in quadriceps CSA. Thus, it is concluded that type II muscle fiber atrophy and hypertrophy are responsible for age-related muscle loss

and training induced gain of muscle mass with little contribution from changes in muscle fiber number.

Thompson et al., 2013[103]

The purpose of this study was to examine the influence of aging on maximal and rapid torque characteristics of the leg extensors and flexor muscle groups in young (24.9 yrs.), middle-aged (50.6 yrs.), and old (66.8 yrs.) men. Maximal strength along with early- and late-phase RTD and normalized RTD was measured during MVCs of the knee extensors and flexors to identify age-related differences. Maximal and rapid torque variables were similar middle-aged men compared to young men. A majority of the rapid torque variables for the leg extensors were higher in the young and middle-aged compared to the old men. Relative RTD was similar among all three groups suggesting that the age-related declines in muscle strength may be largely responsible for the decreases in absolute RTD.

Clark et al., 2013[21]

This study aimed to determine the extent to which potential changes in voluntary neuromuscular activation (quantified as the RER) and muscle size account for potential changes in leg press strength, leg press power, and mobility function during an approximately 2.5 year longitudinal assessment of healthy older adults. Neuromuscular activation was examined during the leg press exercise during which subjects performed 5 explosive concentric contractions using 70% MVC. Mobility function was assessed via the Short Physical Performance Battery (SPPB) which consists of 4 m walking speed, repeated chair rise time, and standing balance. While there was no change in the SPPB scores at

follow-up, this was the first study to provide longitudinal evidence of impaired neuromuscular activation capacity with aging. RER, which has been shown to be associated with physical performance in older adults[1, 20], was found to decrease at a rate of 9% per year. Power significantly decreased after 2.5 years (6%), while 1-RM strength did not change. The decrease in neuromuscular activation is likely the cause of the reduction in power as the relationship between the percent change of these two factor were closely related (r = 0.78). In support of the disassociation between muscle size and physical performance, CSA did not change over the 2.5 year duration.

Thompson et al., 2014[102]

This study examined the effects of aging on the rate of muscle activation and maximal and rapid force characteristics of the plantar flexors in young, middle-aged, and old men. Maximal strength along with early- and late-phase RTD and normalized RTD were measured during MVCs of the plantar flexors. In addition, the findings of this study indicated that maximal strength and all rapid force variables were similar between young and middle-aged men, but were substantially reduced in the older men. An interesting finding of this study was that there was no reduction observed in the old compared to young men for normalized RER; however, these values were greater for middle-aged men at all time intervals when compared to the young and old men. Relative RFD, which is thought to represent qualitative contractile characteristics, was also significantly impaired in the older adults compared to the young adults. The authors suggest that the lower relative RFD characteristics compared to the young men, and the lack of differences in the rate of muscle activation may suggest that qualitative factors as type II fiber atrophy, increased

accumulation of intramuscular fat and connective tissue, or alterations in muscle architecture may be the cause for the age-related decline in rapid force capacity.

Jenkins et al., 2014[55]

This study examined the age-related differences in voluntary and evoked, absolute and normalized RTD and RER of the knee extensors. Similar to Thompson et al.[103], RTD at all reported time intervals was lower in the older group. This was accompanied by age-related decreases in absolute RER as well. However, upon normalization of RTD to peak torque and RER to maximum M-wave amplitude, there were no age-related difference in either RTD or RER at any time interval. Therefore, it is suggested that age-related differences in maximal strength are contributing to the lower absolute RTD values which are commonly reported for older men. Furthermore, since the age-related differences in RER were explained by M-wave amplitude, that the neuromuscular influences of aging on the knee extensors may be more related to a decrease in MU number than diminished MU firing rates.

Thompson et al., 2014[101]

The purpose of this study was to examine the effects of aging on maximal and rapid velocity characteristics of the knee extensors in healthy young and older men. Both groups performed MVCs at $240°\cdot s^{-1}$ and $500°\cdot s^{-1}$ (i.e., unloaded) on an isokinetic dynamometer. RVD was obtained for both speeds, while PV was recorded for the unloaded knee extension only. The old group exhibited a lower PV and RVD at both velocities. RVD for $240°\cdot s^{-2}$ and $500°\cdot s^{-2}$ were 26.7 and 37.2% lower for the older compared to the young group. This was

substantially greater than the 10.1% age-related impairment for PV. Thompson and colleagues highlight the importance of time-dependent measures and suggest that RVD may be a more sensitive and functional neuromuscular performance measurement as compared to PV.

Drey et al., 2014[33]

This study was a follow-up to the pilot study of [32] in which the MUNIX technique was used for the first time in a sarcopenic population. The purpose of this study was to examine whether having a small quantity of MNs is accompanied by low muscle mass in elderly adults. After being clinically diagnosed as sarcopenic or non-sarcopenic, the MUNIX and MUSIX of the hypothenar muscle along with other measures of neuromuscular function were compared between elderly adults belonging to these two groups. The MUNIX technique was able to differentiate between individuals classified as sarcopenic and non-sarcopenic indicating that those diagnosed as having low muscle mass, also had a low number of functioning MUs. This finding suggest that generalized MN loss of a small muscle such as the hypothenar can reflect generalized loss of muscle mass. Furthermore, the group of sarcopenic adults with pathologic MUNIX/MUSIX were found to have lower grip strength and gait speed when compared with the group of non-sarcopenic adults. Drey and colleagues suggest the negative relationship between MUNIX and MUSIX represents reinnervation in individuals with a low quantity of MU numbers but large average MU size.

McKinnon et al., 2015[72]

The purpose of this study was to investigate the MU properties of the TA and vastus medialis (VM) in young and older adults, and to compare these results to isometric strength and power. MUNE and MU action potential size estimates were determined via spike triggered averaging for both muscles. Also, maximal isometric strength and peak power during at submaximal loads during an isotonic contraction were obtained. As hypothesized, older adults exhibited significant indications of MU loss, as well as declines in power and maximal isometric strength for the TA muscle. While there was no indication of MU loss for the VM, an age-related reduction of muscle strength and power was observed. The author suggest the discrepancy between MUNE values for the TA and VM is the result of length-dependent manner in which neurogenic changes occur with aging. That is, degeneration may be greater in more distal muscles. A much larger age-related reduction in peak power was found for the knee extensors than the dorsiflexors (44% vs. 29%), which the authors propose is due to the difference in fiber-types for these muscles. These findings further validate the importance of muscle power as a measure of neuromuscular performance in older adults, and provides a further mechanistic support for the concept of dynapenia.

Piasecki et al., 2015[87]

This study used iEMG to investigate the MUNE and organization of MUs for the VL in young (25.3 yrs) and old men (71.4 yrs). VL CSA was 33% smaller and maximum strength was 32% less in older adults. The older adults had fewer MUs and these MUs were enlarged as indicated by an increased peak-to-peak amplitude. In support of the notion that an increased MUAP amplitude is associated with more fibers due to reinnervation, the older adults also demonstrated an increased fiber count. Older adults demonstrated greater

transmission variability at the neuromuscular junction as indicated by a higher jiggle value. Importantly, these age-related alterations in MUNE, MUAP-size, and variability preceded any meaningful decrements in mobility as the older adults were not identified as being sarcopenic whether or not a measure of mobility was included.

Wu et al., 2016[110]

Recently, Wu and colleagues examined the effect of age and sex on a multitude of neuromuscular and mechanical determinants of muscles strength. A cross-sectional design was used to study the knee extensors for 24 young (23.6 yrs) and 20 older (66.5 yrs) healthy males and females. In accordance with previous research, maximum strength and RTD in males were reduced with age with RTD being more dramatically effects. (47.4% and 60.4%, respectively). Interestingly, lower VL activation (RMS) was found in the older group, however, when subcutaneous fat was accounted for (ANCOVA) this difference disappeared. Conversely, an age-related difference for VL median frequency was retained even after accounting for subcutaneous fat. In regards to muscle architecture, age-related differences were found for muscle thickness (ultrasound) and fascicle length, however; there was no difference in pennation angle between the young and older group. Thus, the age-related impairments in strength and RTD found in this study are believed to be at least partly influenced by the decreased fascicle length, muscle thickness, and muscle-tendon stiffness values reported for the older group.

Summary of "Effects of Aging on Motor Unit Properties and Neuromuscular Function"

Dramatic changes of the central nervous system (CNS) at the level of the MU are associated with aging. Previous research has investigated these changes for the purpose of improving our understanding of potential mechanisms causing age-related reductions in neuromuscular performance. The examination of the underlying physiological alterations causing age-related decrements in neuromuscular performance is necessary in order to differentiate between inevitable neuromuscular decrements and those which may be modifiable through intervention. The MU, which consists of the MN along with its motor axon and the muscle fibers innervated by the MN, is considered the final common pathway in which the generation of movement is derived[68]. Thus, the physiological alterations of the MU induced by aging has an important role in our understanding of the neuromuscular impairments associated with aging.

The aging process is accompanied by a reorganization of the MU pool for various muscles as a result of MN death and the denervation-reinnervation process. Not all but some muscle fibers become denervated by the motor axon of their constituent MN which results in either reinnervation of the muscle fiber by a surrounding MN or the loss of the muscle fiber[4]. Thus, this leads to a reduction in the number of functioning MUs contributing to the force production required for human movement. This is supported by human research using neurophysiological techniques to obtain a MUNE[15, 31, 33, 73, 87] and cadaveric studies using counting techniques[16, 107] for various muscles. It has been estimated that an approximately 1% loss per year of functioning MUs occurs with aging[15]. Specifically, the decrease in the number of functioning MUs becomes substantial in approximately the 6th decade of life[16, 73,

[107] and rapidly accelerates with advancing age[107]. The age-related reduction in the number of functioning MUs is paralleled by impaired maximal strength capacity[15, 59], and has been used to differentiate sarcopenic and non-sarcopenic individuals[32, 33].

Once a muscle fiber has been denervated by its respective MN there is the possibility of reinnervation by means of collateral sprouting. Under these circumstances, the motor axon of a neighboring MU attaches to the denervated muscle fiber, thus increasing the number of muscle fibers being innervated by the MN[31]. Therefore, while the number of functional MUs have been found to decrease with age, the number of muscle fibers innervated by each MU will increase if substantial reinnervation is present (i.e. increased innervation ratio). This is supported by indirect evidence derived from neurophysiological measurements in which the average MU action potential size or area was greater in older adults than young adults, although a reduced MUNE was exhibited by the older adults[16, 31, 73, 87]. In addition, Piasecki and colleagues found older adults to have an increased near fiber count in addition to a larger MUAP-size[87]. Importantly, these findings were found in older adults who were not identified as being sarcopenic. Thus, collateral reinnervation may act as a compensatory mechanism for age-related MU loss in middle- to older-aged adults who have not suffered severe muscle atrophy. However, upon a critical threshold in older age this mechanism is not sufficient to maintain muscular strength, and thus functionality in older individuals[73, 107].

Previous studies have used in-dwelling EMG to investigate the effects of aging MU properties. It has been consistently demonstrated that maximal MU firing rates during a maximal contraction in older adults are reduced compared to young adults[19, 56, 85, 86].

Although the underlying mechanism is unclear, the age-related decrease in maximal MU firing rates may be due to factors such as increased antagonist co-activation[85], altered biophysical properties of MUs[19], or an attempt of the nervous system to match neural control with muscle contractility[86] which is known to be slower in older adults[23, 73]. MU firing rates during ballistic isometric contractions appears to be negatively affected by age as well[61]. Klass et al.[61] demonstrated that the firing rates of the first 3 recruited MUs during a ballistic isometric contraction are reduced in older adults compared to their young counterparts. In addition, older adults exhibited a reduced number of doublet firings as well compared to young adults. Thus, older adults appear to demonstrate diminished maximal MU firing rates during both ballistic and ramp isometric contractions.

It is well known that the aging process is associated with a decrease in muscle mass. Undoubtedly, the age-related loss of functioning MUs is partly responsible as an impaired capacity for reinnervation of denervated muscle fibers is likely responsible for the net loss of muscle mass with old age[4]. There appears to be a preferential loss of the larger diameter type II muscle fibers associated with aging[63, 67, 84]. Larsson et al.[63] demonstrated that type II muscle fiber area was found to decrease with age, and that this atrophy of the type II muscle fibers correlated with strength loss in old age. Furthermore, the type II/I fiber diameter ratio was reduced in young adults (20-29 yrs old) compared to old adults (80-90 yrs old). Lexell and colleagues showed that a reduction in the number of muscle fibers for the VL was the primary cause of age-induced atrophy, while the reduction in the size of type II fibers was a secondary source. The relative proportion and quantity of type II muscle fibers for the VL is positively associated with maximum velocity capacity and explosive strength[104]. Thus, the

preferential loss of type II muscle fibers in older adults is likely partly responsible for age-related decrements in neuromuscular performance.

In general, neuromuscular function peaks during the 3rd decade of life[63], begins to decline shortly thereafter and rapidly declines in the 6th or 7th decade[52, 101, 103]. There is some evidence of decreased maximal strength in middle-aged adults compared to young adults[52], while other researchers have demonstrated no difference[102, 103]. The loss of maximal strength capacity in older adults (≥60 yrs) as compared to young adults has been well documented with reports of relative decreases ranging from ~20-45%[8, 53, 103]. A more drastic age-related decrease in explosive strength (i.e., RTD, RFD) has been reported in numerous studies[8, 54, 61, 103] with a relative decrease as much as 50-60% being reported in some studies[8, 52, 61]. Similarly, while maximum shortening velocity of the knee extensors is decreased in older adults[63, 101], comparatively a two-fold decrease for RVD has been reported in older adults[101]. Thus, it appears that while maximum strength is impaired in older adults; the ability to rapidly develop force or limb movement is dramatically affected by aging.

There are many factors related to muscle activation by the nervous system that have a role in the age-related reduction in neuromuscular performance. As previously discussed, the commonly observed reduction in maximal MU firing rates during a maximal contraction in older adults is a likely candidate for the loss of maximal strength associated with aging. It is unclear whether or not antagonist co-activation is increased in older adults with some studies reporting no difference in antagonist co-activation between young and old adults[61, 78, 97] and

other reporting higher coactivation level in old adults[52, 71]. Maximal EMG amplitude appears to remain unchanged throughout the aging process[44, 61, 83]. An impairment in the ability to rapidly activate the muscle is likely partly responsible for the age-related reductions in rapid strength and velocity characteristics. However, while several studies have examined the associated between RER and RTD, the relationship between RER and PV or RVD is less clear. Klass et al.[61] demonstrated that a 48% reduction in RTD was accompanied by a decrease in MU firing rates and a reduction in the number of doublet discharges during the initial phase of contraction in older adults. In a 2.5 year longitudinal study, Clark et al.[21] found that the decrease in power output during a leg press was closely associated to the decrease RER in older adults ($r = 0.78$)[21]. This suggest that an impairment in the ability to rapidly activate the knee extensors is at least partly responsible for the decrease in power output. In support of this, there was no change in muscle CSA or maximum strength over the 2.5 year span. On the contrary, Thompson et al. found no difference in the rate of muscle activation between young and old adults although there was an age-related decrease in early and late rapid strength characteristics[102]. The authors suggested that qualitative factors of the muscle may have been the cause for the reduction observed in older adults. An age-related reduction in maximal MU firing rates during maximal ramp contractions has been consistently reported in the literature, however, whether this is an impairment of the nervous system or a compensatory mechanism is unknown. The capacity of the nervous system to rapidly activate the muscle appears to be a limiting factor for rapid strength production.

2.2. Effects of Resistance Training on Motor Unit Properties and Neuromuscular Function

Moritani and DeVries, 1979[77]

This seminal study was one of the first to examine the time-course of neural and hypertrophic adaptations induced by resistance training. Moritani[77] used a force-EMG ratio derived from the force-EMG relationship for the elbow flexors to indicate the relative contributions of hypertrophy or neural factors to training-induced strength gains. Furthermore, CSA of the elbow flexors was estimated using a skin-fold testing technique accounting for skin and subcutaneous fat. Testing was performed on the ipsilateral and contralateral arm before and every 2 weeks during an elbow flexor training program which consisted of dumbbell exercises being performed twice per day and three times per week for 8 weeks. The results indicated a significant increase in strength of ~21.2 lb. which was brought on by both, neural factors and hypertrophy. In addition, the untrained arm demonstrated an increase in strength of ~13.4 lb. accompanied by an increase in EMG amplitude, but neither the CSA nor EMG slope coefficient was altered after training. It is concluded that this strength increase of the untrained arm may be caused by neural factors that would cause an increase in EMG amplitude, but have no effect on the EMG/force ratio. The findings indicated that neural factors were primarily responsible for the increase in strength in the trained arm during the first 2 weeks, while the contribution of hypertrophy started to become the predominant contributing factor at approximately 4 weeks.

Narici et al., 1989[80]

The aim of this study to investigate the degree of hypertrophy of the quadriceps femoris muscles and compare the size and time course of changes in CSA with muscle activation and force during strength training. Four young males performed 6 sets of 10 maximal isokinetic knee extensions of the dominant leg at $120°\cdot s^{-1}$ for 60 days. Before training and on every 20^{th} day of training muscle size, strength, and activation of both the trained and untrained leg were examined. Isometric strength as well as isokinetic strength at 60, 120, 180, 240, and $300°\cdot s^{-1}$. In addition, max EMG during an isometric MVC was recorded. The results indicated an increase in CSA of 8.5%, and an increase in MVC of 20.8% for the trained leg. Thus, the increase in CSA of the quadriceps only accounted for 40% of that for MVC. In addition, a significant increase (42.4%) was exhibited by the untrained leg. Isokinetic torque increased at the training velocity ($120°\cdot s^{-1}$) and at $60°\cdot s^{-1}$ only with no changes in strength demonstrated for the faster velocities. Maximum strength, CSA, and EMG remained unchanged for the untrained leg. The authors suggest that increased neural drive and an alteration in muscle architecture are likely responsible for the observed strength increases due to the small increase in CSA.

Carolan and Cafarelli, 1992[17]

The purpose of this study was to determine whether there is a reduction in hamstring co-activation after 8-weeks of isometric resistance training of the quadriceps. Extensor MVC and VL maximal EMG amplitude and EMG amplitude of the antagonist BF were measured before and after 1, 2, 4, and 8-weeks of training. In addition, Flexion MVC, BF maximal EMG amplitude, and EMG amplitude of the antagonist VL were measured at the same time intervals. An increase in the MVC and a decrease in the antagonist co-activation of the

trained leg was found after only 1 week of training. There were no further decreases in antagonist co-activation, while MVC was also significantly increased at week 8. Antagonist co-activation of the untrained leg was reduced at the same time period, but MVC only decreased after 2 weeks of training. There was no change in the maximal EMG amplitude of the agonist or antagonist muscles for either leg throughout the duration of the study. Carolan and Cafarelli[17] estimated that the decrease in antagonist co-activation accounted for approximately one-third of the increase in mean extension MVC after the first week of training.

Aagaard et al., 1994[3]

This study examined the changes in moment-velocity and power-velocity relationships of the knee extensors over the full spectrum of maximal extension velocity in elite soccer players after resistance training. Three different resistance training protocols were performed: 1) high resistance at low speed (HR), 2) low resistance at high speed (LR), or 3) loaded kicking (FU). The angular velocity for the HR group was in the range of 20-50°·s^{-1}, while angular velocity range for the LR group was 100-200°·s^{-1}. The FU groups used a load corresponding to approximately a 16-RM and the velocity varied unrestrictedly between 0 and 400°·s^{-1}. The primary finding of this study was that HR training induced increased in moment and power at slow and fast velocities, while the LR and FU training only produced velocity-specific improvements. Specifically, HR training improved both power and moment at velocities of 240-480°·s^{-1}. In contrast to the previous findings, maximal strength was only increased at velocities specific to the velocity used during training. Andersen et al.[7] reported an increase in the proportion of type IIb fibers in the VL

of the same subjects used in the HR group. Aagaard and colleagues conclude that either these morphological changes or an increase in RFD is responsible for the power and moment increases observed in the HR group at both low and high velocities.

Hortobagyi et al., 1997[49]

This study compared the magnitude of cross-education following training with muscle lengthening and shortening in the quadriceps muscle. Young, sedentary males performed 12 weeks of either eccentric or concentric isokinetic resistance training at $60°\cdot s^{-1}$, and performed neuromuscular testing sessions at weeks 6 and 12. Significantly greater strength improvements for the contralateral leg were found after eccentric training compared to concentric training (77% vs. 30%, respectively) when tested with the same contraction mode used during training. EMG of the contralateral VL increased 2.2 times (pre- to mid-test), 2.8 times (mid- to post-test), and 2.6 times more (pre- to post-test) in the eccentric group than the concentric group when % EMG change was pooled across concentric, eccentric, and isometric testing modes. Cross-education was greatest for the for the contraction type that was used during training. There was no training-induced changes in coactivation for either group.

Chan et al., 1998[18]

This study sought to determine if MUs possessing different contractile characteristics would adapt differently after an involuntary electrical stimulation training intervention. The contractile properties of six MUs in two young, healthy males were recorded before, during, and after 7 weeks of training. Three MUs were identified as “slower, fatigue-resistant” or

"faster, fatigable" based on their contractile properties and fatigue profile at baseline. There were relative differences in the response of the MUs from different "categories" in that the twitch and tetanic tensions of the slower MUs was increased by training, but these measures were unaffected in the faster MUs. Furthermore, fatigue resistance of the faster MUs increased after training, while it remained unchanged in the slower MUs. The fact that involuntary training was used in this study limits the interpretation of its results. Also, it is possible that such a small sample (i.e., 6 MUs) does not provide an accurate representation of the training-related changes in the MU pool.

Häkkinen et al., 1998[44]

This study implemented a 6 month strength training protocol combined with explosive exercises to examine the degree of hypertrophic, voluntary neural activation, and antagonist co-activation adaptations in middle-aged and elderly men and women. The strength training protocol consisted of two leg extensor exercises and four to five other exercises for the other main muscle groups of the body. To incorporate explosive training subjects performed 20% of the leg extensor exercises with light loads (50-60% 1-RM) in an explosive manner. The resistance training protocol enhanced maximal isometric and dynamic strength were as well as explosive force production measures of the leg extensors in both middle-aged and elderly men and women. Although the increase in force production was accompanied by increased CSA of the leg extensor muscles, the latter changes were minor compared to the changes found for voluntary activation (EMG amplitude) of the same muscles. In addition, antagonist co-activation during maximal isometric and dynamic decreased for both men and women in the elderly group, but remained unchanged in both

middle-aged groups. Furthermore, the reduction in antagonist co-activation for the elderly individuals took place during the first 1-2 months of the training protocol. Despite the reduced antagonist co-activation during maximal force contractions, antagonist co-activation during a rapid-force MVC was unaltered. However, EMG amplitude during the early phase of a rapid-force MVC and squat jump was enhanced in all groups. Furthermore, all groups showed a tendency for training-induced decreases in antagonist co-activation during a squat jump, with elderly women demonstrating a significant increase. The authors suggest that neural adaptations seem to play a greater role than training-induced hypertrophy in the strength and power improvements demonstrated after strength training.

Van Cutsem et al., 1998[108]

This seminal study examined the effects of 3 months of ballistic dynamic training on the neuromuscular adaptations of the ankle dorsiflexors muscles, as well as the associations between these adaptations and RTD. In order to distinguish neural adaptations from contractile changes, MVC torque, speed of ballistic contractions, EMG activity during MVC and voluntary ballistic contractions, and electrically induced twitch torque were analyzed. The training protocol consisted of ten sets of ten ballistic dorsiflexions performed 5 days per week at an intensity of 30-40% of maximal muscle strength. The adaptation of contraction velocity was assessed by measuring the maximal RTD during ballistic contractions at different torque levels. Both, the MVC and the speed of voluntary ballistic contraction were increased by training. The authors suggest that the latter finding was mainly mediated by neural factors since the time course of the muscle twitch induced by electrical stimulation remained unaffected. The finding of a training induced increase velocity and a decrease in

the time to peak of the ballistic contractions was accompanied by an earlier onset of EMG activity, increased maximal MU firing rates, and higher initial MU firing rates. Furthermore, the training lead to 27% increase in brief (2-5 ms) MU interspike intervals, or doublets, which were found for MUs with various RTs. The authors suggest that earlier MU activation, extra doublets, and increased maximal MU firing rates contributed to the increase in the speed of voluntary muscle contraction after dynamic training.

Prevost et al., 1999[90]

This study compared the effects of 2 days of velocity-specific training at either a slow ($30°\cdot s^{-1}$) or fast ($270°\cdot s^{-1}$) on isokinetic leg extension peak torque at $30°\cdot s^{-1}$, $150°\cdot s^{-1}$, and $270°\cdot s^{-1}$. Both groups performed 3 sets of 10 maximal concentric contractions of the knee extensors. The results indicated that the high-velocity group increased maximum strength only at the training velocity, while the slow-velocity group did not increase maximum strength at any of the tested velocities. The authors suggest that motor learning is a large contributor to increases in high-velocity strength after high-velocity training since a 22% increase was found after the 2 day training protocol. The authors highlight that an increase of this magnitude is similar to that of studies involving resistance training for several weeks.

Akima et al., 1999[6]

This study examined the effects of 2 weeks of isokinetic resistance training on isometric and isokinetic knee extension strength, muscle use, muscle fiber characteristics, and CSA of quadriceps femoris. T2-weighted magnetic resonance image was used to determine activated and non-activated regions of muscle (muscle use) before and after

training. Training consisted of 10 sets of 5 maximal isokinetic knee extensions at $120°\cdot s^{-1}$. Isometric peak torque and peak torque at the velocities of 60, 90, 120, 180, and $240°\cdot s^{-1}$ increased, but strength at $300°\cdot s^{-1}$ was unchanged. In addition, T2 values and % activated CSA increased after training, however; there was no change in hypertrophy. The authors conclude that short-term increases in strength after isokinetic training are the result of increased muscle contractile activity, but not muscle hypertrophy.

Connelly and Vandervoort, 2000[22]

This study examined the effects of a two week isokinetic strength training protocol on concentric and eccentric torque development in older adults. Training consisted of two sets of twelve repetitions at 30, 90, and $180°\cdot s^{-1}$ involving both concentric and eccentric muscle actions. Dorsiflexor peak torque increased at all three speed for concentric and eccentric muscle actions. In addition, RTD was increased at all three speeds for concentric muscle actions. EMG amplitude of the antagonist soleus remained unchanged by the training protocol, while EMG amplitude of the agonist tibialis anterior increased at all three velocities. The authors conclude that neural mechanisms are likely responsible for the rapid increases in peak torque and RTD.

Patten and Kamen, 2000[85]

This study used force control training in young and old adults to examine whether or not age-related differences exist for strength improvements and to gain an understanding of the underlying mechanisms leading to improved force control. At baseline, maximal MU firing rates at 100% MVC for the old group were significantly lower (26%) than the young

group. After 6-weeks of force modulation training, both the young and old group improved force accuracy, but only the young group increased maximal strength. Although the old group did not increase strength after the training protocol, they showed an increase in maximal MU firing rates and a decrease in antagonist co-activation during submaximal contractions. The authors conclude that, especially in the old group, the adaptations in both force regulation and MU control properties during submaximal contractions can be attributed to reduced antagonist co-activation. Furthermore, that neural strategies such as this may be compensatory mechanisms to account for MN loss and remodeling.

Rich and Cafarelli, 2000[92]

The purpose of this study was to determine whether there is a change in the average MU firing rate during submaximal isometric contraction after 8 weeks of isometric, single-leg resistance training of the knee extensor muscles. The average MU firing rate for approximately 400 MUs per leg was determined during a sustained (~10 s) isometric contraction at 50% MVC in college-aged males. In addition, RER for the first 200 ms of the signal and maximal EMG amplitude were measured during an explosive MVC. The contractile speed of the knee extensor muscles was improved as training induced a significant increase in MVC (36%), along with maximal twitch amplitude (17%), time to peak tension (9%), and maximal instantaneous rate of contraction in the trained leg. Despite the latter increases, there was no increase in maximal EMG amplitude, RER, or average MU firing rate. The authors conclude that although adaptations of the contractile properties were present, control properties of the nervous system are not altered after 8 weeks of resistance training.

Bemben and Murphy, 2001[10]

Similar to Ehsani et al.[34], this article investigated the influence of age on strength and estimated muscle hypertrophy of the trained and untrained limb after 2 weeks of resistance training. A young (20 yrs.) and an older (58 yrs.) group of females trained the elbow flexors for 14 consecutive days using 70% of their maximal strength for 4 sets of 10 repetitions. Both groups increased strength similarly for the trained (28%) and untrained (12-15%) limb. It was concluded that these adaptations were due to neural mechanisms since there was no change in estimated CSA and efficiency of electrical activity decreased.

Patten et al., 2001[86]

The purpose of this study was to determine whether resistance training alters maximal MU firing rates of the trained or untrained hand, and whether this alteration is different in young (23 yrs) and old individuals (76 yrs). Maximal MU firing rates of the abductor digiti minimi (ADM) during MVC was investigating using intramuscular EMG before and 2, 14, and 42 days after commencement of a strength training program. Baseline strength was 29% and 27% greater in the young group than in the old group for the trained and untrained hand, respectively. However, strength gains for the trained and untrained hand in groups were observed as early as 14 days and continued at 42 days. At baseline, maximal MU firing rates was 20% lower in the older adults than in the young adults at each of the four testing sessions. The primary finding was that maximal MU firing rates increased significantly at 2 days in both young and older adults for the trained and untrained hand. However, a similar pattern was observed for both hands where the initial increase stabilized, especially in older

adults, and decreased back to baseline levels after 6 weeks of training. It is postulated that the initial enhancement of MU firing rates could be due to increased antagonist inhibition early in the training period. The authors conclude that maximal MU firing rates may be an important mechanism for very improvement in strength after beginning strength training, however; that the neural mechanisms involved in short-term strength changes are complicated.

Evetovich et al., 2001[35]

The purpose of this study was to examine the effects of unilateral concentric isokinetic knee extension training on maximum strength and EMG in the trained and untrained limbs. College-aged males performed 12 weeks of concentric-only isokinetic training at $90°\cdot s^{-1}$ for 3-6 sets of 10 repetitions. Peak torque and maximum EMG of the trained and untrained leg were examined every 4 weeks during maximal isokinetic testing at the training speed. PT of the trained leg demonstrated a significant increase at 4, 8, and 12 weeks compared to baseline, whereas the untrained leg was only stronger at week 12. The overall increase for the trained and untrained leg was 15.5% and 5.5%, respectively. Although training increased strength for the trained and untrained leg, neither leg demonstrated an increase in EMG. The authors propose that EMG may have been altered for other muscles involved during the leg extension task.

Aagaard et al., 2002[1]

The purpose of this study was to examine changes in maximal contractile RFD, impulse, and efferent neural drive evoked by 14 weeks of heavy, multi-joint, lower-body

resistance training. A novel contribution from this study was the finding of a training-induced concurrent increase in RFD and EMG amplitude in the initial phases of a muscle contraction (i.e., 0-50 ms). In addition, the finding of an increase in the RER for the VL, vastus medialis, and RF. Similar to RFD, impulse was found to increase during both the initial (0-50 ms) and later (100-200 ms) phases of muscle contraction. Due to the greater increases in integrated EMG and RER compared with RFD and maximal muscle strength, the authors suggest that an increased in MU firing rate at the onset of contraction is likely responsible for the training-induced increase in RFD. In support of this, only RFD relative to one-sixth of MVC was found to be significantly different compared to greater relative values, thus indicating qualitative changes such as incidence of discharge doublets or MU firing rate are responsible for the increase in RFD. However, muscle morphology was not evaluated in this study, therefore; the possible contribution from an increase in muscle mass to the increase in RFD and impulse cannot be excluded.

Newton et al., 2002[83]

The purpose of this study was to examine the effects of a 10 week periodized resistance training program on power output, along with maximal- and rapid-strength in young and old men. The training program which consisted of a mixed methods design focused on muscle hypertrophy, maximal force production, and maximal power production. A total of 3 sessions was performed per week with each session emphasizing either hypertrophy, strength, or power. Similar to previous research, the old group exhibited reduced maximal strength and power output compared to the young group. Both groups responded to the training program in a similar manner with increases in both, maximal

strength and power after the 10 week training program. Rapid-force production from 100-150, 500-1500, and 1500-2500 ms also increased in both groups, however, there was no training-induced change in rapid-force production from 0-100 ms for either group. Due to a large degree of variability, an increase in EMG was only found when the groups were collapsed. Interestingly, none of the dependent variables changed significantly prior to the 10 week post-test, thus there were no early-phase neuromuscular adaptations. In conclusion, older men were found to increase maximal strength as well as muscular power during the jump squat to a similar magnitude as their younger counterparts.

Blazevich et al., 2003[11]

This study examined muscle architecture and morphology before and after three different 5 week training programs. After a standardization phase, athletes performed either explosive back squat, forward hack squat, or sprint/jump training in addition to other supplementary lower-body exercises. Maximum strength of the knee extensors at $30°\cdot s^{-1}$ and $180°\cdot s^{-1}$ as well as MT, FA, and FL were obtained for the VL and rectus femoris after the 5 week training protocol. Specific architectural changes were demonstrated for the training groups. The sprint/jump group, who performed only high-velocity training, had a decrease in FA whereas FL increased, particularly in the VL. The subjects who performed resistance training (back squat or forward hack squat) exhibited small but consistent increases in fascicle angle in VL and RF without any changes in fascicle length. A small, non-significant increase in MT was demonstrated when all groups were pooled together. Although differences in muscle architecture were noted for the training groups, there were no training-induced differences in any of the performance measures between groups.

Farthing and Chilibeck, 2003[37]

This study examined whether cross-education is specific to low and high velocities of eccentric training, and attempted to determine the candidate neural mechanisms involved. College-aged males and females were tested before and after performing either fast ($180°\cdot s^{-1}$) or slow ($30°\cdot s^{-1}$) eccentric isokinetic training of the elbow flexors for 8 weeks. Training at the fast velocity ($180°\cdot s^{-1}$) produced velocity-specific increases in strength, while the slow group ($30°\cdot s^{-1}$) did not exhibit cross-education at either speed. On the contrary, the trained arm demonstrated strength increases at specific and non-specific testing velocities. The authors suggest that the finding of greater cross-education after the most unfamiliar type of training (fast-velocity) indicates that learning may play a large role in cross-education. Interestingly, while RTD (time to PT) did not increase for the untrained arm in the fast-trained group or the trained arm in both fast- and slow-trained groups, it did increase for concentric contractions in the untrained limb of the fast-trained group. Thus, while PT did not increase for this limb in this group, RTD did. The authors suggest that cross-education may be related to the amount of change in the trained limb since there was only a 10% increase in strength of the trained limb in the slow-trained group.

Brown and Whitehurst, 2003[14]

This study determined the effects of short-term isokinetic training at a fast and slow velocity on limb acceleration in college-aged men and women. This study used a fast velocity group which trained at $240°\cdot s^{-1}$ and a slow velocity group that trained at $60°\cdot s^{-1}$. Both groups performed 2 workouts consisting of 3 sets of 8 repetitions. Despite neither

groups demonstrated changes in torque production, improvements in RVD were found. The improvement in RVD were specific to the velocity used by the training groups. Due to the limited exposure to strength training, the authors suggest that these early increase in RVD are neurally mediated.

Kamen and Knight, 2004[56]

This study examined changes in knee extensor strength along with MU firing rates of the VL during maximal and sub-maximal contractions before and after 6-weeks of strength training in young and old adults. The training protocol consisted of three sets of ten dynamic knee extensions at 85% 1-RM and three 5 second maximal isometric contractions for three days per week. Subjects completed four testing sessions in total, two testing sessions separated by seven days prior to commencing the training protocol, a third session after 2-weeks of training, and the fourth session after the 6-week training protocol. At baseline, the young adults were significantly stronger (28%) than old adults, but both groups increased strength significantly as early as the second testing sessions and continued to increase strength at a similar rate resulting in an average increase of 33% in strength. MU firing rates were significantly higher in the young group compared to the old group at 100% and 50% MVC, but not 10% MVC throughout the entire study. A significant increase (19%) for maximal MU firing rates at 100% MVC was demonstrated during the second testing session prior to the onset of training. This increase in maximal MU firing rates corresponded with the strength increase demonstrated during the same period of time for young adults (r = 0.88) and old adults (r = 0.83). Interestingly, when all four testing sessions were incorporated into the analysis there was a significant increase in maximal MU firing rates at 100% MVC,

however, there was no increase in maximal MU firing rates when only measured between the start of the training and the end of the study. Thus, indicating a sharp increase in maximal MU firing rates very early upon exposure to a strength testing or training stimulus; however, the influence of maximal MU firing rate on strength improvements appears to lessen after a few weeks. Kamen and Knight[56] propose that either a decrease in antagonist co-activation levels or supraspinal alterations as possible underlying mechanisms influencing the observed changes in maximal MU firing rates.

Barry et al., 2005[8]

Barry and colleagues used a similar design to that of [1] to examine if older adults demonstrate similar neuromuscular adaptations during the initial phases of a muscular contraction compared to young adults after performing 4 weeks of RFD training. The isometric training consisted of four sets of six repetitions of combined flexion and supination of the elbow flexors performed in a ballistic manner. The older adults possessed a substantially lower RTD (-51.2%) at baseline compared to younger adults which was more than twice the difference in maximal strength. For both the young and old adults, there was a training-induced increased in RTD during the first 200 ms the muscular contraction. In both groups, this was accompanied by an increase EMG amplitude and RER during the first 100 ms of the contraction. This was the first study to show changes in older adults for EMG during the very early phases of muscular activation in response to resistance. However, this response was limited to only two of the four elbow flexor muscles examined. The author suggest the training-induced increases in EMG during the initial phases of muscle activation

may have been caused by an increase in MU firing rates, an earlier recruitment of MUs, or an improvement in MU synchrony.

Pucci et al., 2006[91]

The purpose of this study was to measure average MU firing rate during maximal and submaximal contractions and to determine the time course of changes in maximal force, maximal EMG amplitude, M-wave amplitude, voluntary activation, and agonist-antagonist coactivation during 3 weeks of isometric training of the quadriceps muscles in untrained, college-aged males. MVC force was significantly increase after only 4 days of training and increased by 35% after 3 weeks of resistance training. Average MU firing rate at 50, 75, and 100% MVC of the VL did not increase, despite a small but significant increase in voluntary activation. Furthermore, EMG amplitude of the antagonist did not change with training at any point. Pucci and colleagues suggest that maximal MU firing rates increase in response to dynamic but not isometric resistance training. Also, that mechanisms other than MU firing rates such as muscle synergy, synchronization, or increased protein synthesis led to the rapid increases in strength.

Del Balso and Cafarelli, 2007[27]

This study aimed to identify adaptive alterations in neural function during four weeks of isometric resistance training. Three training sessions consisting of 6 sets of 10 MVCs lasting 3-4 s of the plantar flexors were performed per week. During each visit measures of evoked H reflex and V wave responses were recorded to examine if changes in volitional drive and/or spinal excitability occurred in conjunction with increased muscle activation and

muscle strength. MVC, RER of the soleus, and maximal RTD were found to increase after just two training sessions. Both, the increase in RER and RTD and that of RTD and MVC over the twelve training sessions were found to be highly correlated. Del Balso and Cafarelli[27] suggest that either an increase in firing rates, the number of doublet firings, or an increase in MU synchronization may be the cause for training-induced increase in RTD. The increased V wave amplitude observed after training is suggested to be from increased descending volitional drive resulting from either an increase in MU recruitment and/or firing rates. It is concluded that the increase in MVC may be attributed to the observed increase in rate of activation, secondary to the increase in descending volitional drive.

Murray et al., 2007[79]

The purpose of this study was to examine the effects of a short-term velocity-specific training on torque production, RVD, and physical performance in young males. A fast velocity group trained at $400°\cdot s^{-1}$, while a slow velocity group trained at $60°\cdot s^{-1}$. Both groups performed 2 sessions per week for 4 weeks. Interestingly, neither group exhibited and increase in peak torque at any velocity. The authors suggest this may be a result of previous or concurrent resistance training participation by the sample of male kinesiology students. There were no differences between groups from pre to post for any of the measures, so the groups were collapsed for data analysis. The results indicated an overflow of adaptation for RVD since an increased was exhibited at all speeds except $180°\cdot s^{-1}$. The authors suggest that the intention of moving the limb as fast as possible resulted in the increased RVD, and that this adaptation is likely neurally mediated.

Blazevich et al., 2007[12]

This study examined measures of muscle architecture and morphology of the quadriceps femoris musculature during short-term isokinetic training. Testing of the trained and untrained leg consisting of ultrasonographic imaging as well as maximal concentric and eccentric strength testing at $60°\cdot s^{-1}$ occurred after 2.5 and 5 weeks of resistance training. Maximal concentric-eccentric knee extensions at $60°\cdot s^{-1}$ were performed 3 days per week at 4 sets of 6 repetitions the first 2.5 weeks, and 5 sets of 6 repetitions were performed the second 2.5 weeks. Eccentric strength of the trained leg exhibited a large increase (42%), while concentric strength increased significantly, but to a lesser degree (8%). In contrast, there was no training-induced strength increase for the untrained leg, except for eccentric strength for women only. Interestingly, there were no training-induced changes in muscle thickness, fascicle length, fascicle angle for any of the sites examined in the trained or untrained leg. Due to the significant, and stronger correlations found between VL muscle thickness and knee extensor torque after training as compared to this relationship before training, the authors suggest that the rapid increase in strength can be explained by alterations in recruitment strategies.

Seynnes et al., 2007[96]

The purpose of this study was to examine the time course of early muscular adaptations in young males and females. Subjects performed bilateral concentric and eccentric contractions of the knee extensors 3 times per week for 4 sets of 7 maximal repetitions. Maximal strength, EMG, quadriceps muscle cross-sectional area, along with fiber length and pennation angle were obtained before and 10, 20, and at the end of a 35 day

training program. A training-induced increase in strength occurred after only 10 days of training, while an increase CSA of quadriceps was demonstrated after 20 days of resistance training. Given that strength increased by 38% at the end of the 5 week training protocol and quadriceps CSA increased by 7%, the authors suggest that neural factors accounted for a major portion of the observed strength gain. In support of this, a significant increase in EMG of 39% was found for the VL after training. In addition, fascicle length was increased after only 10 days of training, whereas, pennation angle did not increase until the end of the training program. The authors conclude that while neural factors may be the major contributor to early training-induced increases in strength, the finding of an increase in CSA after only 3 weeks suggest that hypertrophy may contribute earlier than once thought.

Adamson et al., 2008[5]

This was the first study to examine the contralateral effects of unilateral maximal strength training on RFD. The subjects performed an 8 week single-arm elbow flexors training protocol which emphasized maximal mobilization of force and consisted of five sets at a 5-RM intensity for three sessions per week. Maximal force and peak RFD were recorded during an isotonic contractions performed at submaximal intensities (1, 2, 3, and 4 kg) and maximal isometric contractions. Anthropometric measurements indicated there was no hypertrophy induced by the training protocol in either the trained or untrained arm. Interestingly, the training increased RFD to a similar magnitude in both the trained and untrained during the dynamic and isometric contraction, however; peak force only increased during the maximal isometric contraction. Furthermore, although the 1-RM load significantly increased for both arms, the increase for the trained arm was nine-fold greater.

Laroche et al., 2008[62]

The purpose of this study was to test the ability of a combination high-velocity/high-resistance training program to enhance knee extensor muscle strength, power, neural activation of muscle, and muscle activation time in inactive women and compare the responses between young and old women. The training was performed 3 times per week for 8 weeks and consisted of 3 sets of 8 concentric-eccentric repetitions at both, $45°\cdot s^{-1}$ and $200°\cdot s^{-1}$. The older group demonstrated similar increases in both isometric and isokinetic strength compared to the young group, however; the older group exhibited a modest improvement for RTD and impulse compared to the young group. Maximal EMG and RER for the agonist remained unchanged for both the young and old group, but antagonist co-activation decreased for both groups. Laroche and colleagues suggest that the training-induced strength gains are likely modulated through reduced antagonist co-activation rather than alterations of efferent drive of the agonist. The authors suggest that future research examine training-related changes in the RER during antagonist co-activation.

Fimland et al., 2009[38]

The aim of this study was to identify the site of neural adaptations for the contralateral limb after 4 weeks MVC training of the plantar flexors. This study was the first to assess changes in H-reflex excitability during maximum contractions of the contralateral limb after unilateral strength training. The training program led to large increases in strength for the trained (44%) and untrained (32%) limb. The strength increase of the untrained limb was accompanied by increased EMG amplitude, an increased V-wave response, and an

unchanged H-reflex response. The authors conclude enhanced descending neural drive to the agonist muscles of the contralateral limb is likely responsible for the cross-education observed in this study, although several possible mechanisms for increased descending neural drive such as motorneuron excitability and/or changes in presynaptic inhibition mediating augmented motorneuron recruitment and/or rate coding are suggested.

Lee et al., 2009[65]

This study used a twitch interpolation technique involving transcranial magnetic stimulation (TMS) to investigate the site of any associated increase in central drive after unilateral strength training of the elbow flexors. This was the first study to examine whether or not an increase in cortical voluntary drive contributes to the strength increase of the untrained limb after unilateral strength training. The 8.2% strength increase in untrained limb was accompanied by an increase in voluntary activation as assessed by the cortical stimulation. The increase in voluntary activation was indicated by the observation of a small superimposed twitch for the untrained leg after unilateral strength training. Thus, the authors suggest that subjects were able to more effectively recruit more MUs that generate force by volition.

Christie and Kamen, 2010[19]

Christie and Kamen sought to assess age- and training-related differences in maximal MU firing rates and the duration of the MN afterhyperpolarization phase (AHP). Also, the correlation between maximal MU firing rates and AHP duration was assessed to explore the potential relationship between MN properties and maximal MU firing rates. A young and old

group performed isometric strength training of the of the dorsiflexors for two weeks. Maximal MU firing rates were examined during the plateau phase of slow, ramp contraction to 100% MVC. At baseline, the old group had slower maximal MU firing rates and longer AHP durations than the young group, however, both group showed an increase in maximal MU firing rates and a decrease in AHP duration after training. Furthermore, MVC force increased 20% and 17% in the old group and young group, respectively. Interestingly, only a modest relationship was found between the pre-post change in MVC force and maximal MU firing rates (r = 0.40) and no increase in central activation was demonstrated for either group. As a result, the authors speculate that an increase in contraction velocity may have been the contributing factor to increased strength. Although training-induced increase in maximal MU firing rates corresponded with a decrease in AHP duration in both groups, the relationship between these variables and their pre-post change was weak. Christie and Kamen conclude that the age-related changes in the AHP phase and maximal MU firing rates are not a necessary consequence of aging since older subjects showed similar improvement for both measures after training.

Beck et al., 2011[9]

This was the first study to use the surface EMG decomposition (dEMG) technique to examine changes in motor unit behavior after resistance training. The resistance training protocol consisted of a full-body protocol performed three times per week for 8-weeks. Each exercise was performed for 10-12 repetitions at approximately 80% 1-RM. Before and after training subjects performed MVC testing and a submaximal trapezoidal isometric task which required a linear increase in isometric leg extension force from 5% to 80% in four seconds.

The RT and average firing rate for each motor unit was examined using linear regression analysis. Although strength was significantly increased, the resistance training protocol had no effect on average firing rate or RT for the VL. Thus, resistance training did not bring the average MU firing rates of the high-threshold MUs up to a level comparable to those of the low-threshold MUs as indicated by lack of change in the linear slope of the relationship between average firing rate and RT after resistance training.

Tillin et al., 2011[106]

The purpose of this study was to investigate the contribution of agonist and antagonist activation to changes in strength and RFD in the trained and untrained leg after 4 weeks of unilateral isometric resistance training. Tillin and colleagues[106] examined the agonist EMG-force relationship and agonist EMG-antagonist EMG relationship across intensities to quantify the level of muscular activation and antagonist co-activation, respectively. College-aged males performed the training which consisted of four sets of ten unilateral isometric knee extensions during which subjects were instructed to ramp up to 75% of maximal voluntary force over 1-s, hold it for 3-s, and relax. A significant strength increase of 20% was accompanied by a significant 26% increase in agonist EMG at maximal voluntary force, while no change in agonist EMG was found to accompany strength gain of the untrained leg. Furthermore, co-activation was lower for any given level of agonist activation after training for both the trained and untrained leg. Although maximum strength was found to increase, there was no change in RFD or impulse. The authors suggest that minimal adaptations occurred at the peripheral level as indicated by no change in the position of the force-agonist EMG relationship and the magnitude of the resting twitch values. It is concluded that

enhance agonist activation and decreased antagonist co-activation are likely responsible for the observed increases in maximum voluntary strength.

DeFreitas et al., 2011[26]

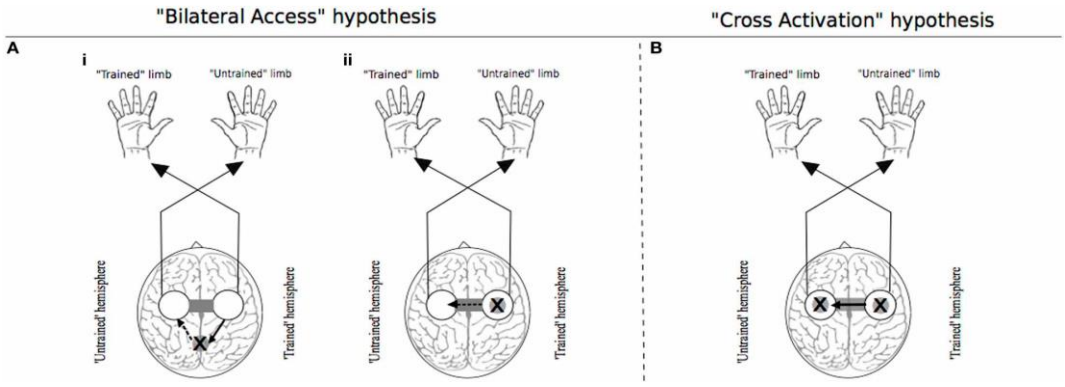
This study aimed to determine the time course of skeletal muscle hypertrophy in young males during 8 weeks of resistance training. The resistance training protocol consisted of a full-body routine with each exercise being performed for 3 sets to failure. Isometric strength and CSA of the quadriceps femoris musculature were examined on a weekly basis throughout the duration of the 8 week training program. An increase in muscle CSA was demonstrated as early as 2 training sessions after the commencement of training. DeFreitas and colleagues[26] suggested it is possible that an increase in edema could be responsible for this early increase in CSA. It was determined that CSA at week 3 was greater compared to CSA at week 1. Thus, with the assumption that edema would be the same at 3 weeks or less compared to week 1, the authors suggest that muscle hypertrophy likely occurred during weeks 3-4. Isometric strength had a similar time course of adaptation as it was found to be significantly greater at week 4.

Ruddy and Carson, 2013[93]

This review discussed the two commonly proposed hypotheses (see figure below) for the neurally mediated mechanisms contributing to the cross-education effect. In addition, evidence from studies using structural and functional brain imaging is brought forward in an effort to delineate how each of these two hypotheses could be responsible for cross-education after unilateral training. The “Bilateral Access” hypothesis states that motor “engrams” (i.e.,

stored memory) are developed through unilateral training and are accessible by both hemispheres of the brain. Thus, the untrained motor cortex is capable of accessing these engrams when the untrained limb performs a task. This access is believed to occur through high-order brain centers (i) or the corpus callosum is the branch of nerves that connect the two hemispheres of the brain. The "Cross Activation" hypothesis suggest that the bilateral cortical activity occurring during a unilateral task induces a task-specific neural adaptation of the motor circuits in the untrained hemisphere[66]. It is believed that cross-transfer via cross activation would be restricted to the homologous muscle of the untrained limb, while performance of non-homologous muscles of the untrained limb would be improved as well if the cross-transfer occurred via bilateral access.

Figure adopted from Lee et al. 2010[66]



Stock and Thompson, 2014[100]

The purpose of this study was to examine changes in the average firing rates of MUs, as well as the firing rates at recruitment, for both the VL and the rectus femoris (RF) following ten weeks of deadlift training. In contrast to other studies using the dEMG technique, Stock &

Thompson[100] examined MU properties at two force level during the post-test. This included 50% of the pre-test MVC and 50% of the post-test MVC. It was hypothesized that for subjects demonstrating a significant increase in knee extensor MVC strength, the slopes and y-intercepts would be affected during the absolute force measurements. MVC testing and trapezoidal isometric tasks at an intensity of 50% MVC were performed before and after the deadlift training protocol. Although the training increased maximal strength, there was no change in mean MU firing rates or firing rates at recruitment for either the VL or RF. This finding was the same for both post-testing at 50% pre-test MVC and 50% post-test MVC. The authors suggest that the testing timeline may not have been appropriate for changes in MU firings rates since previous reports have observed changes early after initiating resistance training. Also, it is suggested that resistance training may have a larger influence on maximal MU firing rates examined during a maximal contraction.

Ehsani et al., 2014[34]

This study examined the effect of a 2 week unilateral upper-body resistance training intervention on strength of the untrained limb in young and older adults. The results showed both, the young and older group demonstrated cross-education of the elbow flexor, however; there was no age-related difference associated with this effect. For the young group, the relative strength increases for the trained and untrained limb (31% and 24%, respectively) tended to be less compared to the older group (52% and 39%, respectively).

Scanlon et al., 2014[95]

The purpose of this study was to examine the effects of a 6 week progressive, full-body resistance training protocol on muscle morphology and architecture in healthy older adults. Ultrasonography was used to obtain CSA, physiological cross-sectional area, pennation angle, fascicle length, muscle thickness (MT), and muscle quality of the VL and RF before and after the resistance training program. CSA of the VL was found to increase, however, MT was not altered by the training. The authors suggest that a longer period of training may be necessary to induce a change in MT since CSA may be a more sensitive measure total muscle hypertrophy. There was no change found fascicle length or pennation angle of the VL or RF. Scanlon et al.[95] proposed that a longer training period is likely necessary to alter these properties. The resistance training program did improve muscle quality, measured as strength per unit of mass, but not echo intensity (EI). Similar to pennation angle, fascicle length, and MT, the authors believe a longer duration of resistance training may be necessary to increase these measures.

Pinto et al., 2014[88]

This study examined the effects of a 6 week lower-body resistance training intervention on maximum strength, functional performance, muscle hypertrophy (muscle thickness), and muscle quality (maximum strength relative to muscle thickness) in older females. After training, strength, muscle thickness, and muscle quality were significantly increased by 23.5%, 10.5%, and 14.8%, respectively. This study provided evidence of some of the earliest training-induced hypertrophy in older adults to date. In addition, the training-induced change in muscle quality was correlated with the training-induced changes in function performance.

Lixandrao et al., 2016[70]

This study investigated the time-course of VL muscle hypertrophy during 10 weeks of resistance training in older adults. An increase in CSA of the VL did not occur until the 9th week of resistance training, while maximum strength was increased significantly by 42%. The evidence on the timeline of hypertrophy is the main contribution of this study since there are no other reports on the timeline of short-term hypertrophy in older adults.

Pope et al., 2016[89]

Recently, we studied the effects of a longitudinal resistance training intervention on MUAP-size through the use of recently developed technology which allows for the decomposition of single MUs from a sEMG signal. MUAP-size and recruitment threshold (RT) of numerous VL MUs were obtained during an MVC of the knee extensors before and after an 8 week resistance training intervention. The training consisted of 3 sets of 10 repetitions for 7 dynamic resistance exercises with a load that represented each participant's 10 repetition maximum. We used separate linear regression equations to examine the effects of training on lower- (≤30% RT) and higher-threshold (≥30% RT) MUs. Resistance training had a preferential effect on higher-threshold MUs and 83.6% of the variance in the change of the relationship between MUAP-size and RT was explained by training-induced changes in whole muscle CSA. This was the first study to non-invasively investigate the longitudinal effects of resistance training on hypertrophy using an electrophysiological assessment as an indicator of MU morphology.

Summary of "Effects of Resistance Training on Motor Unit Properties and Neuromuscular Function

Numerous studies have examined the neural contributions to training-induced adaptions during short-term training (≤6 weeks). Moritani was among the first to identify a time-course which distinguished the proportional influences of "neural factors" and "muscle hypertrophy" to training-induced strength increases[77]. It was determined that neural adaptations were primarily responsible for strength increases during the first 2 weeks of resistance training, but muscle hypertrophy was the predominant mechanism at approximately 4 weeks. Previous literature has established that many neuromuscular performance parameters are improved to a similar degree in older adults as compared to young adults. Whether or not the underlying mechanisms for these training-induced improvements in neuromuscular performance are similar for young and old adults is less clear. Resistance training has proven effective at increasing maximal MU firing rates during ramp isometric contractions in young and old adults[19, 56, 85, 86]. Maximal MU firing rates have been found to increase after only two sessions of resistance training exposure and then return to baseline level during the next few weeks[56, 86]. Interestingly, a 19% increase in maximal MU firing rate exhibited during a second testing session was significantly correlated with the strength increase over the same period in young adults (r = 0.88) and older adults (r = 0.83). For young and old adults, an increase in maximal MU firing rates may have an influential role for strength increases during the very initial phases of strength training, however; other mechanisms may become predominant after that time.

It is well known that prolonged exposure to high-intensity resistance training increases the size of individual muscle fibers and thus the whole muscle. The short-term effects (≤6 weeks) of resistance training on the timeline of muscle hypertrophy are less clear. Due to the invasiveness of indwelling EMG and impracticality of tracking a single MU, there is little to no evidence regarding the effects of resistance training on MUAP-size. Recently, using a non-invasive 5-pin surface EMG sensor in combination with MU decomposition software, we demonstrated a preferential hypertrophic effects of higher-threshold MUs[89]. There are no reports regarding the effect of age on MU specific responses to resistance training, thus much of the evidence on short-term training induced hypertrophy is at the whole muscle level. Resistance training induced increases in whole-muscle CSA in young adults may occur as early as 3 weeks after the commencement of resistance training[26, 96], however; a training induced increase in hypertrophy during the early-phases of resistance training in young adults has not been a consistent finding[6, 12]. There is relatively less data on the effects of short-term resistance training on muscle hypertrophy in older adults compared to young adults. Lixandrao et al. concluded that nine weeks of resistance training was necessary to induce muscle hypertrophy in older adults[70]. However, a training related increase in muscle hypertrophy for older adults has recently been reported after only 6 weeks[88, 95]. Importantly, previous studies examining muscle hypertrophy during short-term resistance training did not include a young group to provide a direct comparison of age[70, 88, 95]. Evidence regarding the effects of resistance training on muscle hypertrophy during earlier periods of training (i.e., 2-4 weeks) in older adults is scant.

Surface EMG has been used in numerous studies to examine training-induced changes in muscle activation, however, equivocal findings have been reported likely because EMG amplitude is influenced by several factors such as the number of MUs recruited, MU firing rates, sarcolemma excitability, and the degree of amplitude cancellation. Training-induced increases in maximal EMG amplitude after short-term resistance training have been demonstrated by some researchers[27, 80, 106], while other have reported no change after training[17, 91]. The rate of muscle activation (i.e., rate of EMG rise; RER) during an isometric contraction is increased by resistance training in young adults[8, 27] and old adults[8] when training is performed in a ballistic manner. Thus, it appears that if the concentric phase of repetitions is performed in a slow manner during resistance training, the early-phase (≤100 ms) activation of the involved muscle will remain unaltered[105, 106]. In contrast to Barry et al., who examined RER of the elbow flexors after training, rapid muscle activation of the RF remained unchanged in old and young adults after lower-body resistance training[62]. Thus, it is unclear whether or not there is a differential response for RER between young and old adults after lower-body resistance training. In addition, there is substantially less data regarding the effects of resistance training on RER during dynamic contractions. Furthermore, increases in rapid strength have consistently been found to accompany training-induced increases in RER[8, 27]. Although not always demonstrated[91], a training-induced decrease in antagonist-activation has been found to accompany strength increases in young adults[17, 106] and old adults[44, 85]. In regards to velocity parameters, a velocity-specific increase in RVD of the knee extensors has been demonstrated after only two sessions of resistance training[14], while 4 weeks of training led to increases in RVD at various velocities[79]. There is little data available on the effects of short-term resistance training on

maximal limb velocity. Increases in RVD or maximal velocity may be predominantly mediated by neural factors due to the short exposure time to resistance training necessary to induce improvements. However, information regarding training-related influences on muscle activation of the agonist or antagonist during high-velocity contractions is limited. Furthermore, whether or not these mechanisms may differ between young and older adults is not known.

Cross-education, or an increase in strength of the untrained limb after unilateral resistance training is a commonly observed neural adaption to resistance training. On average, the strength increase in the untrained limb is approximately 8%[64]. Age appears to not have an effect on cross-education as studies have reported similar strength increases in the untrained limb in young and older adults[10, 34]. However, both of these studies involved upper-body training, thus we are aware of no study which directly compares the effect of age on cross-education for the lower-body. It is widely accepted that cross-education is mediated by neural factors since the increase in the untrained limb is not accompanied by muscle hypertrophy[5, 10, 37] and the untrained limb demonstrates a considerably low level of activation during unilateral training. The locus of the adaptation within the nervous system is unclear, thus many studies have taken a mechanistic approach to determine where along the motor or spinal reflex pathway the training-induced adaptation occurs. Indeed, increases in maximal EMG amplitude[38, 49] as well as an increase in V-wave response[38] and voluntary activation[65] have been demonstrated in the untrained limb after unilateral resistance training. Training-related changes in MU properties such as augmented MU firing rates could result from an increase in cortical drive[2]. While an increase in MU firing rates of the abductor

digiti minimi for the untrained limb after two sessions of resistance training has been reported[86], the potential influence of training-related increases in MU firing rates of the untrained limb during the first few weeks of unilateral training is not well established. Thus, the role of augmented MU firing rates of the untrained limb on cross-education after unilateral training is unclear. Furthermore, we are unaware of any reports on the effects of unilateral training on maximal MU firing rates of the untrained limb of the lower-body.

3. METHODS

3.1. Participants

Fifty-six young (18-30 yrs; n = 28) and old (≥ 55 yrs; n = 28) males were recruited for this study. All subjects were apparently healthy and reported having no conditions of the nervous or cardiovascular system, or musculoskeletal issues of the lower-body. Furthermore, all subjects reported having no experience with resistance training within the past 6 months. An informed consent, health and exercise status questionnaire, and physical activity readiness questionnaire were completed by all subjects prior to beginning the study. This study was approved by the University's Institutional Review Board prior to data collection.

3.2. Research Design

This study used four groups including a young training group (YTG), young control group (YCG), old training group (OTG), and old control group (OCG). Initially, 56 subjects were recruited, however, 6 subjects dropped out of the study due to time constraints and 2 subjects were excluded because of poor adherence with the training protocol, thus 48 subjects completed this study. Fourteen young adults (YTG) and 12 older adults (OTG) participated in 4 weeks of unilateral resistance training of the dominant leg and three testing sessions during which the trained (TL) and untrained leg (UL) were tested.

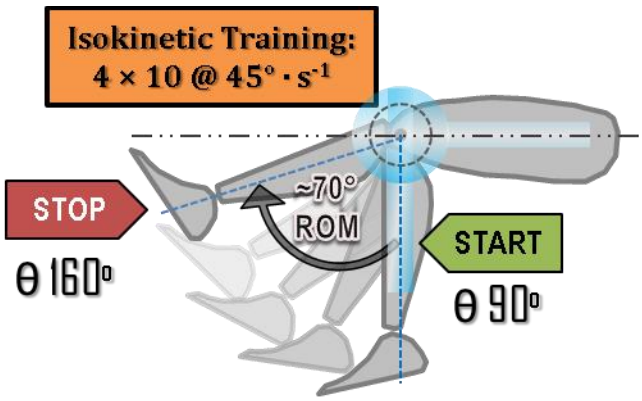
In addition, 11 young adults (YCG) and 11 older adults (OCG) served as controls which involved participating in only the testing sessions. Testing sessions were performed before (PRE), at week 2 (MID),and after week 4 (POST) of the resistance training protocol. All dependent variables were examined in both, the TL and UL. The leg order for the neuromuscular tests was randomized at PRE and remained the same for MID and POST for each subject. All testing sessions were performed at approximately the same time of day and at least 48 hours after the previous training session. Subjects were instructed to refrain from caffeine consumption within 12 hours of each testing visit. For all strength tests, subjects were carefully instructed to keep the leg musculature relaxed and to avoid a countermovement prior to contraction. An additional trial was performed if any preceding activity occurs during baseline. Strong verbal encouragement and visual bio-feedback was provided during all strength tests. The dynamometer chair settings were recorded to ensure that identical settings was used for each subsequent visit. All offline processing for torque, velocity, and surface electromyography (sEMG) signals as well as motor unit (MU) properties was performed using custom written software (LabVIEW, National Instruments, Austin, Texas).

3.3. Instrumentation and Procedures

3.3.1. Resistance Training Protocol

Subjects in the training groups performed unilateral isokinetic concentric resistance training of the dominant leg knee extensors 3 times per week for 4 weeks on a calibrated Biodex system 3 isokinetic dynamometer (Biodex Medical Systems, Inc. Shirley, NY, USA). Subjects were seated with hands across the chest, restraining straps over the trunk, pelvis,

and thigh and the input axis of the dynamometer aligned with the axis of rotation of the knee. Prior to each training session, subjects performed 10 submaximal leg extension at 45°·s^{-1} at ~50% perceived maximal effort. Training sessions consisted of maximal concentric knee extensions at 45°·s^{-1} for 4 sets of 10 repetitions with 2-3 s of rest between repetitions and a 2 min rest interval between each set. Upon determination of 90° knee flexion via manual goniometry, a 70° range of motion consisting of knee extension from 90° to 160° (180° = full extension) was used for each subject (see figure below). A target line representing the maximal torque determined during the previous testing visit was provided on a computer monitor during each session. Subjects were instructed to "reach the target line as quickly as possible by kicking out as hard and fast as possible throughout the entire range of motion". In addition, subjects were instructed to avoid a preceding countermovement and to passively return the leg to 90° knee flexion. Also, subjects were instructed and continually reminded to keep their contralateral leg relaxed during the training sessions. Consistent verbal encouragement and visual bio-feedback was provided during each session.



3.3.2. Isokinetic Strength Testing

Maximal isokinetic strength testing was performed using a calibrated Biodex System 4 isokinetic dynamometer (Biodex Medical Systems, Inc. Shirley, NY, USA). Subjects were seated with hands across the chest, restraining straps over the trunk, pelvis, and thigh and the input axis of the dynamometer aligned with the axis of rotation of the knee. Prior to isokinetic testing, subjects performed 5 warm-up contractions at ~50% perceived maximal effort at $45°\cdot s^{-1}$ and 3 maximal effort contraction at $500°\cdot s^{-1}$. Concentric isokinetic strength testing of the knee extensors at $45°\cdot s^{-1}$ ($IsoK_{45}$), and maximal velocity testing was performed at $500°\cdot s^{-1}$ ($IsoK_{500}$) using a 70° ROM which was determined using procedures identical to those of the training protocol. There was no resistance, with the exception of lever arm mass, provided during $IsoK_{500}$ as this velocity was above all subjects' maximum velocity capacities [41]. For $IsoK_{45}$ testing, subjects were instructed to kick out as "hard and fast as possible throughout the entire ROM", however, subjects were instructed to kick out as "fast as possible throughout the entire ROM" during $IsoK_{500}$ testing. Two repetitions, separated by 30 s of rest, were performed at each velocity. A third repetition was performed if the maximum values of the two repetitions differ by more than ~10%.

3.3.2.1. Torque and Velocity Acquisition and Processing

Prior to testing, passive torque resulting from leg mass was determined with the testing leg approximately 10° less than full extension. This passive torque value was used to correct for gravity and was applied to the torque signal during all isokinetic contractions. Subsequently, the torque signal was smoothed using a zero-lag, low-pass filter (15 Hz). PT was considered the highest 5ms torque value occurring during the constant velocity of $45°\cdot s^{-}$

[1]. The scaled velocity signal was smoothed using a zero-lag, low-pass filter (10 Hz) prior to determining peak velocity (PV) and rate of velocity development (RVD) for $IsoK_{500}$ and $IsoK_{45}$. PV ($°\cdot s^{-1}$) was identified as the highest velocity produced after the onset of velocity. RVD ($°\cdot s^{-2}$) was calculated as the linear slope of the velocity-time curve ($\Delta velocity/\Delta time$) from velocity onset to 2 $°\cdot s^{-1}$ below the PV attained during $IsoK_{500}$[(101)], whereas RVD for $IsoK_{45}$ was defined as the linear slope of the velocity-time curve to 43$°\cdot s^{-1}$. The onset of velocity was defined as 2 $°\cdot s^{-1}$ above baseline.

3.3.2.2 EMG Acquisition and Processing

Prior to electrode placements, the skin over the specified muscles and C7 vertebrae was shaved, abraded, and cleaned with alcohol. Global muscle activation of the VL and biceps femoris (BF) was recorded using a single differential bar electrode (DE-2.1, Delsys, Inc., Boston, MA, USA) with a fixed inter-electrode distance of 10 mm. sEMG of the VL and BF was sampled at 2 kHz using a 16-channel Bagnoli Desktop System (Delsys, Inc., Boston, MA, USA) during the $IsoK_{45}$ and $IsoK_{500}$ testing. Electrode positions were in accordance with the recommendations of the SENIAM project [(47)] and these locations were marked at PRE and every few days until the completion of the study to ensure consistent electrode placement during testing. A reference electrode (Dermatrode, American Imex, Irvine, California) was placed over the spinous process of the C7 vertebrae. During offline processing, the scaled, zero means VL and BF EMG signals were smoothed using a zero-lag, 4^{th} order Butterworth filter with a high- and low-frequency cutoff of 500 Hz and 10 Hz, respectively. Maximal EMG amplitude during $IsoK_{45}$ for the VL was identified as the highest 50 ms root mean square (RMS) value. This value was normalized to the maximum 50 ms

RMS VL EMG value during the isometric ramp task and the quotient was considered the maximal VL EMG amplitude (EMG_{MAX}; $\%EMG_{MAX}$) during $IsoK_{45}$. In addition, the VL EMG signal was smoothed with a zero-lag, low-pass filter (10 Hz) and the linear slope of the rectified EMG-time curve for a 75 ms period after the onset of contraction was calculated. The rate of EMG rise (RER; $\%EMG_{MAX} \cdot s^{-1}$) for the VL during $IsoK_{45}$ was determined by normalizing this slope to the maximum VL EMG amplitude of a similarly filtered signal during the maximal isometric ramp task. The highest 50 ms RMS value for EMG of the BF for $IsoK_{45}$ was recorded and divided by the highest 50 ms RMS value during the knee flexion MVC in order to calculate antagonist co-activation ($ANT_{CO\text{-}A}$; $\%EMG_{MAX}$) during $IsoK_{45}$. Identical processing was performed to obtain EMG_{MAX}, RER, and $ANT_{CO\text{-}A}$ during $IsoK_{500}$.

3.3.3. Isometric Testing

A maximal voluntary isometric ramp contraction (MVC) was performed using the same dynamometer and chair set-up procedures as all other strength tests. Prior to testing, subjects performed 3-4 practice ramp isometric contractions at ~40% of perceived maximal effort in accordance with the visual template provided on a monitor which was located approximately 3 feet in front of each subject. Subjects performed two, 15 second ramp MVCs which required an increase in torque from 0-100% MVC in five seconds (20%/second), maximal effort for 5 seconds, and a decrease in torque from 100-0% MVC in five seconds (20%/second). In the case of poor adherence to either the ramp-up or ramp-down portion, an additional trial was performed. Subsequently, subject performed a maximal isometric contraction for the knee flexors at a knee angle of 150° (180° = full extension).

Subjects were instructed “pull down as hard as possible” for ~3-4 s for the MVC. A warm-up trial at ~50% perceived maximal effort prior to testing. A 2 min rest interval separated all MVC trials.

Maximal voluntary isometric testing was performed after isokinetic testing using the same dynamometer and chair set-up procedures. A knee flexion angle of 120° and 150° (180° = full extension) was used for testing the knee extensors and flexors, respectively. Subsequently, subjects performed a maximal voluntary isometric contraction (MVC) of the knee flexors, followed by two MVCs of the knee extensors. Subjects were instructed to “kick out as hard as possible” and “pull down as hard as possible” for ~3-4 s for the MVC of the knee extensors and knee flexors, respectively.

3.3.3.1. Torque Acquisition and Processing

For all isometric contractions, the passive baseline torque was considered limb weight and was subtracted during off-line processing from the signal so that the baseline signal value was 0 N·m. A zero-lag, 500 ms rolling average was applied to the torque signal, and the highest 500 ms value was recorded as PT.

3.3.3.2. EMG Acquisition and Processing

During the isometric ramp task, sEMG of the VL and BF was recorded using the same EMG system and preparation procedures as that used for isokinetic testing. During offline processing, the scaled, zero means signal for the VL and BF was smoothed using a zero-lag, 4th order Butterworth filter with a high and low-frequency cut-off of 500 Hz and 10 Hz, respectively. Maximal VL EMG amplitude (EMG_{MAX}; mV) was identified as the 500 ms

RMS value corresponding to PT (250 ms on both sides). Antagonist co-activation ($ANT_{CO\text{-}A}$; $\%EMG_{MAX}$) was defined as the ratio of the highest 500 ms RMS BF EMG value corresponding to PT (250 ms on both sides) during the isometric ramp task and the highest 500 ms RMS BF EMG value during the knee flexion MVC.

3.3.4. Motor Unit Decomposition and Processing

A specialized 5-pin surface array EMG sensor (Delsys, Inc., Boston, MA, USA) was positioned next (distal) to the bar electrode over the VL during the isometric ramp task. The surface array sensor uses pairwise subtraction of the 5 pins to obtain four single differential EMG channels which are filtered with a bandwidth of 20 Hz to 1,750 Hz and sampled at 20 kHz prior to being stored for subsequent decomposition. The four separate raw EMG signals were decomposed into their constituent motor unit action potential trains (MUAPTs) using the Precision Decomposition III algorithm. The technical aspects of the algorithm have been described by [24, 81]. Nawab et al. (2010) have demonstrated the ability of this algorithm to reliably discriminate characteristics of a large number of MUs up to maximal force levels for the VL muscle [81]. The accuracy of the MUAPTs were tested using the Decompose-Synthesize-Decompose-Compare test described by [25] and validated by [51]. To reduce the potential influence of false positive and false negative firings, any MU that did not exhibit an accuracy of 90% or greater were eliminated from analysis. During off-line processing, maximal firing rate (MaxFR) and recruitment threshold (RT) were calculated for each MU. MaxFR (pulses per second; pps) was calculated as the mean of the three highest consecutive instantaneous firing rates (i.e., the reciprocal of the 3 shortest consecutive interspike intervals). RT was defined as the relative force level (%MVC) at which the MU commenced

firing. A RT range of ≥25 % of MVC was required (for PRE, MID, and POST) for each subject to be included in any of the MU analyses [89]. Thus, MUs could not be clustered together across a small RT range. MUs that were detected prior to the onset of force production were eliminated from the analyses. The action potential templates from each of the 4 sEMG channels, calculated by the decomposition algorithm, were exported and the maximum peak-to-peak amplitude (mV) was calculated for each MU during offline processing to assess motor unit action potential size (MUAP-size).

3.3.5. Statistical Analyses

3.3.5.1. Baseline Comparisons

Demographic and neuromuscular measures at baseline (i.e., PRE) for the DL were compared between all groups using a two-way factorial ANOVA (age [young vs. old] × group [TG vs. CG]). Baseline comparisons for MU properties were made using the DL variables between the YTG and OTG only. Due to smaller sample sizes and failing to meet the homogeneity of variance assumption, age-related differences for MaxFR and MUAP-size between the YTG and OTG were examined using Welch's t-test. Similarly, MVC EMG_{MAX} and $ANT\text{-}_{COA}$ failed to meet the assumption of homogeneity of variance, thus Welch's test was used.

3.3.5.2. Training-related adaptations

In regards to MU properties, linear regression was used for all to examine the relationship between MaxFR and RT as well as MUAP-size and RT for the grouped PRE,

MID, and POST data. For RT, bin widths of 20% (e.g., 0-20%, 20-40%, etc.) were used to condense the group data. The grouped linear regressions were applied to the means of each bin. The means of the individual slope and y-intercept coefficients obtained from the grouped linear regressions were used to make comparisons between the YTG and OTG. The slope coefficients for the MaxFR vs. RT and MUAP-size vs. RT ($MaxFR_{SLOPE}$ and $MUAP\text{-}size_{SLOPE}$, respectfully) in addition to the y-intercept coefficients ($MaxFR_{Y\text{-}INT}$ and $MUAP\text{-}size_{Y\text{-}INT}$, respectfully) for these relationships were used to make comparisons between means. To examine trained-related adaptations, a two-way mixed factorial ANOVA (group [YTG vs. OTG] × time [pre vs. mid vs. post]) was performed for the TL and UL separately to determine if MaxFRs or MUAP-size changed in either leg across time. For all other neuromuscular measures, a three-way mixed factorial ANOVA (age [young vs. old] × group [TG vs. CG] × time [pre vs. mid vs. post]) was performed for each leg separately to determine changes across time between young and old, and between training and control groups. When appropriate, follow-up analyses included, two-way mixed factorial ANOVAs (time × group), one-way repeated measures ANOVAs (time), and Bonferroni pairwise comparisons. Mauchley's test was used to confirm sphericity for each analysis. If the assumption of sphericity was violated, a Greenhouse-Geisser adjustment was used. Statistical analyses were performed using PASW software version 21.0 (SPSS Inc, Chicago, IL, USA). Due to violations of the homogeneity of variance assumption, an alpha level of $p < 0.025$ was adopted for determining statistical significance[60] during all analyses for MaxFRs, MUAP-size, EMG_{MAX}, and $ANT_{CO\text{-}A}$. For all other dependent variables an alpha level of $p < 0.05$ was used to determine statistical significance. Effect size (ES) was assess and reported using partial eta squared (η_p^2). All data is presented as mean ± SEM, unless otherwise noted.

4. RESULTS

4.1. Descriptives

Descriptive data presented as mean ± SD. Forty-eight males participated in this training study. Fourteen young adults (age = 21.60 ± 1.9 yrs [range: 18-25 yrs], height = 178.88 ± 7.95 cm, mass = 82.2 ± 13.98 kg) and twelve older adults (age = 64.94 ± 7.59 yrs [range: 55-78 yrs], height = 172.56 ± 4.81cm, mass = 86.97 ± 16.57 kg) performed the 4 week resistance training protocol, while eleven young adults (age = 22.42 ± 3.99 yrs [range: 18-30 yrs], height = 178.48 ± 6.74 cm, mass = 87.16 ± 16.23 kg) and eleven older adults (age = 65.75 ± 11.03 yrs [range: 55-88 yrs], height = 179.68 ± 7.89 cm, mass = 94.00 ± 16.34 kg) served as controls. There were no differences between any of the groups for body mass or height ($p > 0.05$). Only two training sessions were missed by two training group members, one from the YTG and another from the OTG. Accuracy tests were performed on the MUs detected from the VL and only those that met at least the 90% accuracy criterion were analyzed. A total of 5,675 MUs were detected for all subjects across all three testing visits. However, after accounting for the required criteria, only 3,382 MUs were used for analysis for the TL/DL (Table 1) and NTL/NDL (Table 2).

4.2. Baseline Comparisons

4.2.1. Motor Unit Properties

At PRE MUAP-size$_{SLOPE}$ was significantly greater in the YTG compared to the OTG at baseline (p = 0.021, +48.3%) (Figure 1). The pooled data for the relationship between MUAP-size and RT for the YTG and OTG at PRE is presented in Figure 2. However there were no differences (p > 0.05) between groups for MaxFR$_{SLOPE}$ (YTG = -.2739 ± .0453 mV · %MVC; OTG = -.2600 ± .0350 mV · %MVC), MaxFR$_{Y\text{-}INT}$ (YTG = 39.87 ± 2.28 pps; OTG = 43.94 ± 1.74 pps), or MUAP-size$_{Y\text{-}INT}$ (YTG = .0449 ± .0151 mV; OTG = .0786 ± .0221 mV).

ANOVA Summary Table

		Sum of Squares	df	Mean Square	F	Sig.
PRE_FR_SLOPE	Between Groups	.001	1	.001	.117	.738
	Within Groups	.131	12	.011		
	Total	.132	13			
PRE_FR_Y_INT	Between Groups	56.678	1	56.678	2.089	.174
	Within Groups	325.564	12	27.130		
	Total	382.242	13			
PRE_APSIZE_SLOPE	Between Groups	.000	1	.000	6.152	.029
	Within Groups	.000	12	.000		
	Total	.000	13			
PRE_APSIZE_Y_INT	Between Groups	.003	1	.003	1.222	.291
	Within Groups	.034	12	.003		
	Total	.038	13			

Robust Tests of Equality of Means

		Statistic[a]	df1	df2	Sig.
PRE_FR_SLOPE	Welch	.113	1	10.177	.743
PRE_FR_Y_INT	Welch	2.009	1	10.076	.187
PRE_APSIZE_SLOPE	Welch	8.039	1	8.276	.021
PRE_APSIZE_Y_INT	Welch	1.418	1	11.529	.258

a. Asymptotically F distributed.

4.2.2. MVC

For PT, there was no age × group interaction or main effect for group, but as expected there was a main effect for age indicating that MVC strength was significantly less in older adults ($p < 0.001$, ES = .415, -26.7%). At PRE, PT was 240.15 ± 9.82 N·m, 231.18 ± 11.91 N·m, 167.46 ±10.57 N·m , and 177.73 ± 12.69 N·m for the YTG, YCG, OTG, and OCG, respectively. There were no interactions or main effects for EMG_{MAX} (YTG = 165.93 ± 21.62 mV; YCG = 182.38 ± 37.42 mV; OTG = 118.24 ± 9.90 mV; OCG = 137.12 ± 24.62 mV) or $ANT_{CO\text{-}A}$ (YTG = .1860 ± .0343 $\%EMG_{MAX}$; YCG = .1062 ± .0118 $\%EMG_{MAX}$; OTG = .1383 ± .0127 $\%EMG_{MAX}$; OCG = .1750 ± .0327 $\%EMG_{MAX}$) ($p > 0.05$).

ANOVA Summary Table

EMG_{MAX}

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	28947.900	3	9649.300	1.332	.276
Within Groups	318679.552	44	7242.717		
Total	347627.452	47			

Robust Tests of Equality of Means

EMG_{MAX}

	Statistic[a]	df1	df2	Sig.
Welch	1.950	3	21.364	.152

a. Asymptotically F distributed.

ANOVA Summary Table

ANT_{CO-A}

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.047	3	.016	1.864	.150
Within Groups	.368	44	.008		
Total	.415	47			

Robust Tests of Equality of Means

ANT_{CO-A}

	Statistic[a]	df1	df2	Sig.
Welch	2.827	3	23.134	.061

a. Asymptotically F distributed.

4.2.3. $IsoK_{45}$

Similar to the MVC, for $IsoK_{45}$ PT there was no age × group interaction or main effect for group ($p > 0.05$), but as expected there was a main effect for age indicating that strength was significantly less in older adults ($p < 0.001$, ES = .299, -26.2%). $IsoK_{45}$ PT was 219.68 ± 9.70 N·m , 220.10 ± 15.18 N·m , 160.81 ± 15.56 N·m, and 163.76 ± 16.09 N·m for the YTG, YCG, OTG, and OCG, respectively. There were no significant interactions or main effects for RVD (YTG = 600.36 ± 26.60 °·s^{-2}; YCG = 627 ± 17.85 °·s^{-2}; OTG = 579.13 ± 31.71 °·s^{-2}; OCG = 560.57 ± 29.78 °·s^{-2}) or ANT_{CO-A} (YTG = .3312 ± .0756 $\%EMG_{MAX}$; YCG = .2934 ± .0728 $\%EMG_{MAX}$; OTG = .2536 ± .0279 $\%EMG_{MAX}$; OCG = .2658 ± .0423 $\%EMG_{MAX}$) ($p > 0.05$). Older adults showed a tendency for greater EMG_{MAX} (YTG = 1.0750 ± .0790 $\%EMG_{MAX}$; YCG = .9938 ± .0990 $\%EMG_{MAX}$; OTG = 1.3397 ± .1746 $\%EMG_{MAX}$; OCG = 1.1640 ± .0681 $\%EMG_{MAX}$) and RER (YTG = 7.77 ± 1.16 $\%EMG_{MAX}$·s^{-1}; YCG = 8.38 ± 1.56 $\%EMG_{MAX}$·s^{-1}; OTG = 15.08 ± 3.06 $\%EMG_{MAX}$·s^{-1}; OCG = 8.58 ± 1.18

$\%EMG_{MAX} \cdot s^{-1}$) , but statistical significance was not met ($p = 0.064$ and $p = 0.054$, respectively).

ANOVA Summary Table

Dependent Variable: PT

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	37279.737[a]	3	12426.579	5.879	.002	.301
Intercept	1628586.675	1	1628586.675	770.468	.000	.949
Age	36999.125	1	36999.125	17.504	.000	.299
Group	31.545	1	31.545	.015	.903	.000
Age * Group	17.930	1	17.930	.008	.927	.000
Error	86664.332	41	2113.764			
Total	1799582.156	45				
Corrected Total	123944.068	44				

a. R Squared = .301 (Adjusted R Squared = .250)

ANOVA Summary Table

Dependent Variable: RVD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	26082.511[a]	3	8694.170	1.062	.376	.072
Intercept	15618934.341	1	15618934.341	1907.016	.000	.979
Age	21440.620	1	21440.620	2.618	.113	.060
Group	184.155	1	184.155	.022	.882	.001
Age * Group	5703.796	1	5703.796	.696	.409	.017
Error	335800.212	41	8190.249			
Total	16178061.842	45				
Corrected Total	361882.722	44				

a. R Squared = .072 (Adjusted R Squared = .004)

ANOVA Summary Table

Dependent Variable: ANT_{CO-A}

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	.043[a]	3	.014	.337	.798	.024
Intercept	3.647	1	3.647	86.865	.000	.679
Age	.031	1	.031	.734	.397	.018
Group	.002	1	.002	.044	.835	.001
Age * Group	.007	1	.007	.166	.685	.004
Error	1.721	41	.042			
Total	5.507	45				
Corrected Total	1.764	44				

a. R Squared = .024 (Adjusted R Squared = -.047)

ANOVA Summary Table

Dependent Variable: EMG_{MAX}

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	.729[a]	3	.243	1.709	.180	.111
Intercept	58.164	1	58.164	409.397	.000	.909
Age	.516	1	.516	3.634	.064	.081
Group	.188	1	.188	1.322	.257	.031
Age * Group	.026	1	.026	.185	.670	.004
Error	5.825	41	.142			
Total	64.892	45				
Corrected Total	6.554	44				

a. R Squared = .111 (Adjusted R Squared = .046)

ANOVA Summary Table

Dependent Variable: RER

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	396.484[a]	3	132.161	3.313	.029	.195
Intercept	4418.456	1	4418.456	110.753	.000	.730
Age	156.894	1	156.894	3.933	.054	.088
Group	96.776	1	96.776	2.426	.127	.056
Age * Group	140.713	1	140.713	3.527	.067	.079
Error	1635.678	41	39.895			
Total	6431.006	45				
Corrected Total	2032.162	44				

a. R Squared = .195 (Adjusted R Squared = .136)

4.2.4. IsoK$_{500}$

Older adults demonstrated a lower PV ($p < 0.001$, ES = .354, -36.6%) and RVD ($p < 0.001$, ES = .383, -22.1%), however; there was no interaction or main effect for group present for either variable. PV was 462.84 ± 3.43 °·s^{-1}, 456.93 ± 6.54 °·s^{-1}, 422.80 ± 10.04 °·s^{-1}, 400.89 ± 16.67 °·s^{-1} for the YTG, YCG, OTG, and OCG, respectively. RVD was 3239.81 ± 97.12 °·s^{-2}, 3137.91 ± 120.13 °·s^{-2}, 2604.24 ± 133.72 °·s^{-2}, 2482.93 ± 194.71 °·s^{-2} for the YTG, YCG, OTG, and OCG, respectively. There was a main effect for group as the CG (collapsed across age) demonstrated a decreased EMG$_{MAX}$ ($p = 0.032$, ES = .108, -19.0%). EMG$_{MAX}$ was 1.33 ± 0.12 %EMG$_{MAX}$, 1.12 ± 0.13 %EMG$_{MAX}$, 1.53 ± 0.14 %EMG$_{MAX}$, 1.20 ± 0.08 %EMG$_{MAX}$ for the YTG, YCG, OTG, and OCG, respectively. No significant interactions or main effects were found for RER (YTG = 11.61 ± 1.14 %EMG$_{MAX}$·s^{-1}; YCG = 11.79 ± 1.28 %EMG$_{MAX}$·s^{-1}; OTG = 16.76 ± 2.43 %EMG$_{MAX}$·s^{-1};

OCG = 12.90 ± 1.92 $\%EMG_{MAX} \cdot s^{-1}$) or ANT_{CO-A} (YTG = .3329 ± .0765 $\%EMG_{MAX}$; YCG = .2118 ± .0360 $\%EMG_{MAX}$; OTG = .2418 ± .0309 $\%EMG_{MAX}$; OCG = .2195 ± .0357 $\%EMG_{MAX}$).

ANOVA Summary Table

Dependent Variable: PV

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	29612.964[a]	3	9870.988	8.588	.000	.380
Intercept	8693340.089	1	8693340.089	7563.651	.000	.994
Age	26403.835	1	26403.835	22.973	.000	.354
Group	2213.519	1	2213.519	1.926	.173	.044
Age * Group	732.378	1	732.378	.637	.429	.015
Error	48273.018	42	1149.358			
Total	8863914.132	46				
Corrected Total	77885.982	45				

a. R Squared = .380 (Adjusted R Squared = .336)

ANOVA Summary Table

Dependent Variable: RVD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	6174659.280[a]	3	2058219.760	9.400	.000	.402
Intercept	368016186.942	1	368016186.942	1680.788	.000	.976
Age	5700956.648	1	5700956.648	26.037	.000	.383
Group	339461.884	1	339461.884	1.550	.220	.036
Age * Group	56638.958	1	56638.958	.259	.614	.006
Error	9196092.199	42	218954.576			
Total	389913096.289	46				
Corrected Total	15370751.479	45				

a. R Squared = .402 (Adjusted R Squared = .359)

ANOVA Summary Table

Dependent Variable: EMG_{MAX}

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	1.074[a]	3	.358	2.146	.109	.133
Intercept	76.904	1	76.904	460.923	.000	.916
Age	.232	1	.232	1.388	.245	.032
Group	.849	1	.849	5.086	.029	.108
Age * Group	.034	1	.034	.203	.654	.005
Error	7.008	42	.167			
Total	85.585	46				
Corrected Total	8.082	45				

a. R Squared = .133 (Adjusted R Squared = .071)

ANOVA Summary Table

Dependent Variable: RER

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	195.131[a]	3	65.044	1.900	.144	.120
Intercept	8049.560	1	8049.560	235.177	.000	.848
Age	112.097	1	112.097	3.275	.078	.072
Group	38.816	1	38.816	1.134	.293	.026
Age * Group	46.587	1	46.587	1.361	.250	.031
Error	1437.561	42	34.228			
Total	9637.005	46				
Corrected Total	1632.692	45				

a. R Squared = .120 (Adjusted R Squared = .057)

ANOVA Summary Table

Dependent Variable: ANT_{CO-A}

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	.115[a]	3	.038	1.242	.306	.082
Intercept	2.894	1	2.894	93.536	.000	.690
Age	.020	1	.020	.644	.427	.015
Group	.059	1	.059	1.902	.175	.043
Age * Group	.028	1	.028	.903	.348	.021
Error	1.300	42	.031			
Total	4.407	46				
Corrected Total	1.415	45				

a. R Squared = .082 (Adjusted R Squared = .016)

4.3. Adaptations of the Trained Leg

4.3.1. Motor Unit Properties

There was no time × group (YTG vs. OTG) interaction for $MaxFR_{SLOPE}$, however; a main effect for time (ES = .349) was present indicating a significant increase in $MaxFR_{SLOPE}$ at MID (p = 0.024, +43.0%) and POST (p = 0.009, +42.6%) as compared to PRE (see pooled data in Figure 3). The relationship between MaxFR and RT at PRE, MID, and POST for an individual young and old training group subject is displayed in Figure 4. There was no interaction (p = 0.327) or main effect for time (p = 0.057) present for $MaxFR_{Y\text{-}INT}$. No time × group interaction (p = 0.0894, ES = .004) was present for $MUAP\text{-}size_{SLOPE}$, but there was a main effect for time. When the YTG and OTG were collapsed together, $MUAP\text{-}size_{SLOPE}$ was significantly increased at POST (p = 0.013, ES = .333, +42.3%) (Figure 5). The pooled data for the MUAP-size vs. RT relationship at PRE, MID, and POST for the YTG and OTG is

displayed in Figure 6. There was neither a time × group interaction ($p = 0.832$) nor main effect ($p = 0.065$) of time demonstrated for MUAP-size$_{Y\text{-}INT}$.

ANOVA Summary Table

Measure: MaxFR$_{SLOPE}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.123	2	.061	6.431	.006	.349
	Greenhouse-Geisser	.123	1.762	.070	6.431	.008	.349
	Huynh-Feldt	.123	2.000	.061	6.431	.006	.349
	Lower-bound	.123	1.000	.123	6.431	.026	.349
Time * GROUP	Sphericity Assumed	.002	2	.001	.092	.912	.008
	Greenhouse-Geisser	.002	1.762	.001	.092	.891	.008
	Huynh-Feldt	.002	2.000	.001	.092	.912	.008
	Lower-bound	.002	1.000	.002	.092	.767	.008
Error(Time)	Sphericity Assumed	.229	24	.010			
	Greenhouse-Geisser	.229	21.143	.011			
	Huynh-Feldt	.229	24.000	.010			
	Lower-bound	.229	12.000	.019			

ANOVA Summary Table

Measure: MaxFR$_{Y\text{-}INT}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	206.438	2	103.219	3.241	.057	.213
	Greenhouse-Geisser	206.438	1.801	114.644	3.241	.063	.213
	Huynh-Feldt	206.438	2.000	103.219	3.241	.057	.213
	Lower-bound	206.438	1.000	206.438	3.241	.097	.213
Time * GROUP	Sphericity Assumed	74.646	2	37.323	1.172	.327	.089
	Greenhouse-Geisser	74.646	1.801	41.454	1.172	.324	.089
	Huynh-Feldt	74.646	2.000	37.323	1.172	.327	.089
	Lower-bound	74.646	1.000	74.646	1.172	.300	.089
Error(Time)	Sphericity Assumed	764.331	24	31.847			
	Greenhouse-Geisser	764.331	21.608	35.372			
	Huynh-Feldt	764.331	24.000	31.847			
	Lower-bound	764.331	12.000	63.694			

ANOVA Summary Table

Measure: MUAP-size$_{SLOPE}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	2.593E-005	2	1.296E-005	5.981	.008	.333
	Greenhouse-Geisser	2.593E-005	1.312	1.976E-005	5.981	.020	.333
	Huynh-Feldt	2.593E-005	1.532	1.693E-005	5.981	.015	.333
	Lower-bound	2.593E-005	1.000	2.593E-005	5.981	.031	.333
Time * GROUP	Sphericity Assumed	1.918E-007	2	9.588E-008	.044	.957	.004
	Greenhouse-Geisser	1.918E-007	1.312	1.461E-007	.044	.894	.004
	Huynh-Feldt	1.918E-007	1.532	1.252E-007	.044	.921	.004
	Lower-bound	1.918E-007	1.000	1.918E-007	.044	.837	.004
Error(Time)	Sphericity Assumed	5.202E-005	24	2.167E-006			
	Greenhouse-Geisser	5.202E-005	15.746	3.304E-006			
	Huynh-Feldt	5.202E-005	18.380	2.830E-006			
	Lower-bound	5.202E-005	12.000	4.335E-006			

ANOVA Summary Table

Measure: MUAP-size$_{Y\text{-}INT}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.024	2	.012	3.070	.065	.204
	Greenhouse-Geisser	.024	1.426	.017	3.070	.086	.204
	Huynh-Feldt	.024	1.699	.014	3.070	.075	.204
	Lower-bound	.024	1.000	.024	3.070	.105	.204
Time * GROUP	Sphericity Assumed	.001	2	.001	.186	.832	.015
	Greenhouse-Geisser	.001	1.426	.001	.186	.758	.015
	Huynh-Feldt	.001	1.699	.001	.186	.797	.015
	Lower-bound	.001	1.000	.001	.186	.674	.015
Error(Time)	Sphericity Assumed	.093	24	.004			
	Greenhouse-Geisser	.093	17.110	.005			
	Huynh-Feldt	.093	20.384	.005			
	Lower-bound	.093	12.000	.008			

4.3.2. MVC

PT at PRE, MID, and POST for the trained and untrained leg for the YTG and OTG is displayed in Figure 7. There was a significant time × age (p = 0.009, ES = .110) and time × group (p < 0.001, ES = .291) interaction for PT. There was no change in PT across time for either the YCG or OCG (p = 0.081). The YTG demonstrated a significant increase in PT at MID (p < 0.001, +16.4%) and POST (p = 0.001, +25.1%) compared to PRE, as well as a significant increase from MID to POST (p < 0.001, +7.4%). The OTG only increased PT at MID (p = 0.006, +10.5%) and POST (p = 0.001, +17.7%) as compared to PRE. In addition, the increase in strength form PRE to MID was significantly greater for the YTG compared to the OTG (p = 0.036, ES = .170). There was a significant time × age × group interaction (p = 0.024, ES = .081) and significant time × group interaction (p = 0.001, ES = .147) for EMG_{MAX}. There was no change in EMG_{MAX} across time for either the YCG or OCG (p = 0.313). The YTG increased EMG_{MAX} at MID (p = 0.009, +41.5%) and POST (p = 0.001, +55.5%), while the OTG only increased EMG_{MAX} at POST (p = 0.019, +31.9%). The increase in EMG_{MAX} from PRE to POST by the YTG was nearly statistically greater than the increase in the OTG (p = 0.026). There were no significant interactions or main effects for $ANT_{CO\text{-}A}$ (p > 0.05).

ANOVA Summary Table

Measure: PT

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	16637.446	2	8318.723	31.955	.000	.421
	Greenhouse-Geisser	16637.446	1.717	9688.695	31.955	.000	.421
	Huynh-Feldt	16637.446	1.902	8746.928	31.955	.000	.421
	Lower-bound	16637.446	1.000	16637.446	31.955	.000	.421
Time * Age	Sphericity Assumed	2835.839	2	1417.919	5.447	.006	.110
	Greenhouse-Geisser	2835.839	1.717	1651.430	5.447	.009	.110
	Huynh-Feldt	2835.839	1.902	1490.906	5.447	.007	.110
	Lower-bound	2835.839	1.000	2835.839	5.447	.024	.110
Time * Group	Sphericity Assumed	9404.501	2	4702.250	18.063	.000	.291
	Greenhouse-Geisser	9404.501	1.717	5476.642	18.063	.000	.291
	Huynh-Feldt	9404.501	1.902	4944.298	18.063	.000	.291
	Lower-bound	9404.501	1.000	9404.501	18.063	.000	.291
Time * Age * Group	Sphericity Assumed	847.640	2	423.820	1.628	.202	.036
	Greenhouse-Geisser	847.640	1.717	493.617	1.628	.206	.036
	Huynh-Feldt	847.640	1.902	445.636	1.628	.204	.036
	Lower-bound	847.640	1.000	847.640	1.628	.209	.036
Error(Time)	Sphericity Assumed	22908.886	88	260.328			
	Greenhouse-Geisser	22908.886	75.557	303.200			
	Huynh-Feldt	22908.886	83.692	273.729			
	Lower-bound	22908.886	44.000	520.656			

ANOVA Summary Table

Measure: EMG_{MAX}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	27588.513	2	13794.256	8.154	.001	.156
	Greenhouse-Geisser	27588.513	1.951	14144.176	8.154	.001	.156
	Huynh-Feldt	27588.513	2.000	13794.256	8.154	.001	.156
	Lower-bound	27588.513	1.000	27588.513	8.154	.007	.156
Time * Age	Sphericity Assumed	1435.996	2	717.998	.424	.655	.010
	Greenhouse-Geisser	1435.996	1.951	736.211	.424	.650	.010
	Huynh-Feldt	1435.996	2.000	717.998	.424	.655	.010
	Lower-bound	1435.996	1.000	1435.996	.424	.518	.010
Time * Group	Sphericity Assumed	25703.682	2	12851.841	7.597	.001	.147
	Greenhouse-Geisser	25703.682	1.951	13177.855	7.597	.001	.147
	Huynh-Feldt	25703.682	2.000	12851.841	7.597	.001	.147
	Lower-bound	25703.682	1.000	25703.682	7.597	.008	.147
Time * Age * Group	Sphericity Assumed	13183.952	2	6591.976	3.897	.024	.081
	Greenhouse-Geisser	13183.952	1.951	6759.195	3.897	.025	.081
	Huynh-Feldt	13183.952	2.000	6591.976	3.897	.024	.081
	Lower-bound	13183.952	1.000	13183.952	3.897	.055	.081
Error(Time)	Sphericity Assumed	148862.453	88	1691.619			
	Greenhouse-Geisser	148862.453	85.823	1734.530			
	Huynh-Feldt	148862.453	88.000	1691.619			
	Lower-bound	148862.453	44.000	3383.238			

ANOVA Summary Table

Measure: ANT_{CO-A}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.014	2	.007	1.417	.248	.031
	Greenhouse-Geisser	.014	1.710	.008	1.417	.248	.031
	Huynh-Feldt	.014	1.893	.007	1.417	.248	.031
	Lower-bound	.014	1.000	.014	1.417	.240	.031
Time * Age	Sphericity Assumed	.019	2	.009	1.882	.158	.041
	Greenhouse-Geisser	.019	1.710	.011	1.882	.165	.041
	Huynh-Feldt	.019	1.893	.010	1.882	.161	.041
	Lower-bound	.019	1.000	.019	1.882	.177	.041
Time * Group	Sphericity Assumed	.025	2	.013	2.524	.086	.054
	Greenhouse-Geisser	.025	1.710	.015	2.524	.095	.054
	Huynh-Feldt	.025	1.893	.013	2.524	.089	.054
	Lower-bound	.025	1.000	.025	2.524	.119	.054
Time * Age * Group	Sphericity Assumed	.014	2	.007	1.359	.262	.030
	Greenhouse-Geisser	.014	1.710	.008	1.359	.262	.030
	Huynh-Feldt	.014	1.893	.007	1.359	.262	.030
	Lower-bound	.014	1.000	.014	1.359	.250	.030
Error(Time)	Sphericity Assumed	.440	88	.005			
	Greenhouse-Geisser	.440	75.232	.006			
	Huynh-Feldt	.440	83.308	.005			
	Lower-bound	.440	44.000	.010			

4.3.3. IsoK$_{45}$

There were no significant interactions or main effects for RVD ($p > 0.05$) (Figure 8). A significant time × age × group interaction ($p = 0.017$, ES = .095) and time × group interaction ($p < 0.001$, ES = .350) was demonstrated for PT. Although there were relative decreases in the CGs (-15.5%), neither grouped demonstrated a significant decrease ($p = 0.180$). Training-induced increases in PT were demonstrated at MID ($p = 0.007$, +7.9%) and POST ($p = 0.001$, +13.2%) in the YTG, while PT in the OTG did not increase over time ($p = 0.129$, ES = .185, POST = +7.1%). There were no significant interactions or main effects for EMG_{MAX} RER, or $ANT_{CO\text{-}A}$ ($p > 0.05$).

ANOVA Summary Table

Measure: PT

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	874.664	2	437.332	1.589	.210	.037
	Greenhouse-Geisser	874.664	1.802	485.390	1.589	.213	.037
	Huynh-Feldt	874.664	2.000	437.332	1.589	.210	.037
	Lower-bound	874.664	1.000	874.664	1.589	.215	.037
Time * Age	Sphericity Assumed	2.639	2	1.319	.005	.995	.000
	Greenhouse-Geisser	2.639	1.802	1.464	.005	.992	.000
	Huynh-Feldt	2.639	2.000	1.319	.005	.995	.000
	Lower-bound	2.639	1.000	2.639	.005	.945	.000
Time * Group	Sphericity Assumed	12148.895	2	6074.447	22.068	.000	.350
	Greenhouse-Geisser	12148.895	1.802	6741.957	22.068	.000	.350
	Huynh-Feldt	12148.895	2.000	6074.447	22.068	.000	.350
	Lower-bound	12148.895	1.000	12148.895	22.068	.000	.350
Time * Age * Group	Sphericity Assumed	2357.115	2	1178.557	4.282	.017	.095
	Greenhouse-Geisser	2357.115	1.802	1308.067	4.282	.021	.095
	Huynh-Feldt	2357.115	2.000	1178.557	4.282	.017	.095
	Lower-bound	2357.115	1.000	2357.115	4.282	.045	.095
Error(Time)	Sphericity Assumed	22571.257	82	275.259			
	Greenhouse-Geisser	22571.257	73.881	305.507			
	Huynh-Feldt	22571.257	82.000	275.259			
	Lower-bound	22571.257	41.000	550.518			

ANOVA Summary Table

Measure: RVD

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	15788.539	2	7894.270	1.625	.203	.038
	Greenhouse-Geisser	15788.539	1.988	7940.841	1.625	.203	.038
	Huynh-Feldt	15788.539	2.000	7894.270	1.625	.203	.038
	Lower-bound	15788.539	1.000	15788.539	1.625	.210	.038
Time * Age	Sphericity Assumed	3597.521	2	1798.761	.370	.692	.009
	Greenhouse-Geisser	3597.521	1.988	1809.372	.370	.690	.009
	Huynh-Feldt	3597.521	2.000	1798.761	.370	.692	.009
	Lower-bound	3597.521	1.000	3597.521	.370	.546	.009
Time * Group	Sphericity Assumed	22281.647	2	11140.823	2.294	.107	.053
	Greenhouse-Geisser	22281.647	1.988	11206.547	2.294	.108	.053
	Huynh-Feldt	22281.647	2.000	11140.823	2.294	.107	.053
	Lower-bound	22281.647	1.000	22281.647	2.294	.138	.053
Time * Age * Group	Sphericity Assumed	9983.159	2	4991.579	1.028	.362	.024
	Greenhouse-Geisser	9983.159	1.988	5021.026	1.028	.362	.024
	Huynh-Feldt	9983.159	2.000	4991.579	1.028	.362	.024
	Lower-bound	9983.159	1.000	9983.159	1.028	.317	.024
Error(Time)	Sphericity Assumed	398291.198	82	4857.210			
	Greenhouse-Geisser	398291.198	81.519	4885.864			
	Huynh-Feldt	398291.198	82.000	4857.210			
	Lower-bound	398291.198	41.000	9714.419			

ANOVA Summary Table

Measure: EMG_{MAX}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.178	2	.089	1.145	.323	.027
	Greenhouse-Geisser	.178	1.964	.090	1.145	.322	.027
	Huynh-Feldt	.178	2.000	.089	1.145	.323	.027
	Lower-bound	.178	1.000	.178	1.145	.291	.027
Time * Age	Sphericity Assumed	.058	2	.029	.371	.691	.009
	Greenhouse-Geisser	.058	1.964	.029	.371	.687	.009
	Huynh-Feldt	.058	2.000	.029	.371	.691	.009
	Lower-bound	.058	1.000	.058	.371	.546	.009
Time * Group	Sphericity Assumed	.099	2	.049	.637	.532	.015
	Greenhouse-Geisser	.099	1.964	.050	.637	.529	.015
	Huynh-Feldt	.099	2.000	.049	.637	.532	.015
	Lower-bound	.099	1.000	.099	.637	.429	.015
Time * Age * Group	Sphericity Assumed	.104	2	.052	.672	.513	.016
	Greenhouse-Geisser	.104	1.964	.053	.672	.511	.016
	Huynh-Feldt	.104	2.000	.052	.672	.513	.016
	Lower-bound	.104	1.000	.104	.672	.417	.016
Error(Time)	Sphericity Assumed	6.360	82	.078			
	Greenhouse-Geisser	6.360	80.507	.079			
	Huynh-Feldt	6.360	82.000	.078			
	Lower-bound	6.360	41.000	.155			

ANOVA Summary Table

Measure: RER

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	33.702	2	16.851	.781	.461	.019
	Greenhouse-Geisser	33.702	1.987	16.964	.781	.460	.019
	Huynh-Feldt	33.702	2.000	16.851	.781	.461	.019
	Lower-bound	33.702	1.000	33.702	.781	.382	.019
Time * Age	Sphericity Assumed	72.466	2	36.233	1.680	.193	.039
	Greenhouse-Geisser	72.466	1.987	36.475	1.680	.193	.039
	Huynh-Feldt	72.466	2.000	36.233	1.680	.193	.039
	Lower-bound	72.466	1.000	72.466	1.680	.202	.039
Time * Group	Sphericity Assumed	39.661	2	19.830	.920	.403	.022
	Greenhouse-Geisser	39.661	1.987	19.963	.920	.402	.022
	Huynh-Feldt	39.661	2.000	19.830	.920	.403	.022
	Lower-bound	39.661	1.000	39.661	.920	.343	.022
Time * Age * Group	Sphericity Assumed	51.981	2	25.991	1.205	.305	.029
	Greenhouse-Geisser	51.981	1.987	26.165	1.205	.305	.029
	Huynh-Feldt	51.981	2.000	25.991	1.205	.305	.029
	Lower-bound	51.981	1.000	51.981	1.205	.279	.029
Error(Time)	Sphericity Assumed	1768.323	82	21.565			
	Greenhouse-Geisser	1768.323	81.455	21.709			
	Huynh-Feldt	1768.323	82.000	21.565			
	Lower-bound	1768.323	41.000	43.130			

ANOVA Summary Table

Measure: ANT_{CO-A}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.090	2	.045	3.117	.050	.071
	Greenhouse-Geisser	.090	1.689	.053	3.117	.059	.071
	Huynh-Feldt	.090	1.882	.048	3.117	.053	.071
	Lower-bound	.090	1.000	.090	3.117	.085	.071
Time * Age	Sphericity Assumed	.082	2	.041	2.824	.065	.064
	Greenhouse-Geisser	.082	1.689	.048	2.824	.075	.064
	Huynh-Feldt	.082	1.882	.043	2.824	.069	.064
	Lower-bound	.082	1.000	.082	2.824	.100	.064
Time * Group	Sphericity Assumed	.055	2	.028	1.905	.155	.044
	Greenhouse-Geisser	.055	1.689	.033	1.905	.163	.044
	Huynh-Feldt	.055	1.882	.029	1.905	.158	.044
	Lower-bound	.055	1.000	.055	1.905	.175	.044
Time * Age * Group	Sphericity Assumed	.000	2	.000	.011	.989	.000
	Greenhouse-Geisser	.000	1.689	.000	.011	.979	.000
	Huynh-Feldt	.000	1.882	.000	.011	.986	.000
	Lower-bound	.000	1.000	.000	.011	.916	.000
Error(Time)	Sphericity Assumed	1.186	82	.014			
	Greenhouse-Geisser	1.186	69.231	.017			
	Huynh-Feldt	1.186	77.163	.015			
	Lower-bound	1.186	41.000	.029			

4.3.4. IsoK$_{500}$

No significant interactions or main effects were demonstrated for PV, RVD, EMG_{MAX}, RER, or ANT_{CO-A} ($p > 0.05$). The means for PV and RVD for the trained/dominant leg at PRE, MID, and POST in the YTG, OTG, YCG, and OCG are displayed in Figure 9.

4.4. Adaptations of the Untrained Leg

4.4.1. Motor Unit Properties

For $MaxFR_{SLOPE}$ and $MaxFR_{Y-INT}$, there were no time × group interactions ($p = 0.322$ and $p = 0.167$, respectively) or main effects for time ($p = 0.178$ and $p = 0.240$, respectively) resulting from resistance training (Figure 12). MUAP-$size_{SLOPE}$ demonstrated relatively strong time × group interaction ($p = 0.031$, ES = .206), however, in consideration of the strict alpha level adopted this effect was considered non-significant. However, a main effect for time was found for MUAP-$size_{SLOPE}$ and pairwise comparisons indicated a significant increase at POST ($p = 0.001$, ES = .352, +34.2%) (Figure 10). The relationship between MUAP-size and RT at PRE, MID, and POST for the pooled data (all training group subjects) is displayed in Figure 11. No significant interaction ($p = 0.108$) or main effect for time ($p = 0.043$) was demonstrated for MUAP-$size_{Y-INT}$.

ANOVA Summary Table

Measure: $MaxFR_{SLOPE}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.161	2	.081	1.829	.178	.109
	Greenhouse-Geisser	.161	1.608	.100	1.829	.187	.109
	Huynh-Feldt	.161	1.892	.085	1.829	.180	.109
	Lower-bound	.161	1.000	.161	1.829	.196	.109
Time * Group	Sphericity Assumed	.104	2	.052	1.175	.322	.073
	Greenhouse-Geisser	.104	1.608	.064	1.175	.316	.073
	Huynh-Feldt	.104	1.892	.055	1.175	.321	.073
	Lower-bound	.104	1.000	.104	1.175	.295	.073
Error(Time)	Sphericity Assumed	1.323	30	.044			
	Greenhouse-Geisser	1.323	24.121	.055			
	Huynh-Feldt	1.323	28.379	.047			
	Lower-bound	1.323	15.000	.088			

ANOVA Summary Table

Measure: $MaxFR_{Y\text{-}INT}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	157.727	2	78.864	1.499	.240	.091
	Greenhouse-Geisser	157.727	1.778	88.697	1.499	.241	.091
	Huynh-Feldt	157.727	2.000	78.864	1.499	.240	.091
	Lower-bound	157.727	1.000	157.727	1.499	.240	.091
Time * Group	Sphericity Assumed	199.771	2	99.886	1.899	.167	.112
	Greenhouse-Geisser	199.771	1.778	112.340	1.899	.173	.112
	Huynh-Feldt	199.771	2.000	99.886	1.899	.167	.112
	Lower-bound	199.771	1.000	199.771	1.899	.188	.112
Error(Time)	Sphericity Assumed	1578.263	30	52.609			
	Greenhouse-Geisser	1578.263	26.674	59.168			
	Huynh-Feldt	1578.263	30.000	52.609			
	Lower-bound	1578.263	15.000	105.218			

ANOVA Summary Table

Measure: MUAP-size$_{SLOPE}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	1.954E-005	2	9.771E-006	8.165	.001	.352
	Greenhouse-Geisser	1.954E-005	1.826	1.070E-005	8.165	.002	.352
	Huynh-Feldt	1.954E-005	2.000	9.771E-006	8.165	.001	.352
	Lower-bound	1.954E-005	1.000	1.954E-005	8.165	.012	.352
Time * Group	Sphericity Assumed	9.341E-006	2	4.671E-006	3.903	.031	.206
	Greenhouse-Geisser	9.341E-006	1.826	5.115E-006	3.903	.036	.206
	Huynh-Feldt	9.341E-006	2.000	4.671E-006	3.903	.031	.206
	Lower-bound	9.341E-006	1.000	9.341E-006	3.903	.067	.206
Error(Time)	Sphericity Assumed	3.590E-005	30	1.197E-006			
	Greenhouse-Geisser	3.590E-005	27.393	1.311E-006			
	Huynh-Feldt	3.590E-005	30.000	1.197E-006			
	Lower-bound	3.590E-005	15.000	2.393E-006			

ANOVA Summary Table

Measure: MUAP-size$_{Y\text{-}INT}$

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.026	2	.013	4.456	.020	.229
	Greenhouse-Geisser	.026	1.750	.015	4.456	.026	.229
	Huynh-Feldt	.026	2.000	.013	4.456	.020	.229
	Lower-bound	.026	1.000	.026	4.456	.052	.229
Time * Group	Sphericity Assumed	.014	2	.007	2.401	.108	.138
	Greenhouse-Geisser	.014	1.750	.008	2.401	.116	.138
	Huynh-Feldt	.014	2.000	.007	2.401	.108	.138
	Lower-bound	.014	1.000	.014	2.401	.142	.138
Error(Time)	Sphericity Assumed	.088	30	.003			
	Greenhouse-Geisser	.088	26.248	.003			
	Huynh-Feldt	.088	30.000	.003			
	Lower-bound	.088	15.000	.006			

4.4.2. MVC

PT at PRE, MID, and POST for the untrained leg of the YTG and OTG is displayed in Figure 7. A time × group interaction was found for PT (p = 0.001, ES = .181) indicating improvements over time for the two training groups with no effect of age. Neither of the control groups increased PT over the 4 weeks (p = 0.532). There was no time × group interaction (p = 0.814) demonstrated when the YTG and OTG were compared across time indicating that both groups increased PT similarly. When collapsed, both groups improved PT at MID (p = 0.010, +7.2%) and POST (p < 0.001, +14.2%) compared to PRE as well as from MID to POST (p = 0.004, +6.5%). A main effect for time (p = 0.001) was demonstrated for EMG_{MAX} indicating an increase at POST regardless of age or group. However, there were no significant interactions (p = 0.466-0.561). No significant interactions or main effects were found for $ANT_{CO\text{-}A}$ (p > 0.05).

ANOVA Summary Table

Measure: PT

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	5841.453	2	2920.726	10.896	.000	.198
	Greenhouse-Geisser	5841.453	1.560	3745.717	10.896	.000	.198
	Huynh-Feldt	5841.453	1.717	3402.793	10.896	.000	.198
	Lower-bound	5841.453	1.000	5841.453	10.896	.002	.198
Time * Age	Sphericity Assumed	182.305	2	91.153	.340	.713	.008
	Greenhouse-Geisser	182.305	1.560	116.900	.340	.659	.008
	Huynh-Feldt	182.305	1.717	106.197	.340	.680	.008
	Lower-bound	182.305	1.000	182.305	.340	.563	.008
Time * Group	Sphericity Assumed	5200.342	2	2600.171	9.700	.000	.181
	Greenhouse-Geisser	5200.342	1.560	3334.618	9.700	.001	.181
	Huynh-Feldt	5200.342	1.717	3029.330	9.700	.000	.181
	Lower-bound	5200.342	1.000	5200.342	9.700	.003	.181
Time * Age * Group	Sphericity Assumed	517.601	2	258.801	.965	.385	.021
	Greenhouse-Geisser	517.601	1.560	331.902	.965	.367	.021
	Huynh-Feldt	517.601	1.717	301.516	.965	.374	.021
	Lower-bound	517.601	1.000	517.601	.965	.331	.021
Error(Time)	Sphericity Assumed	23588.876	88	268.055			
	Greenhouse-Geisser	23588.876	68.618	343.771			
	Huynh-Feldt	23588.876	75.533	312.298			
	Lower-bound	23588.876	44.000	536.111			

ANOVA Summary Table

Measure: EMG_{MAX}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	12790.382	2	6395.191	7.156	.001	.140
	Greenhouse-Geisser	12790.382	1.911	6693.304	7.156	.002	.140
	Huynh-Feldt	12790.382	2.000	6395.191	7.156	.001	.140
	Lower-bound	12790.382	1.000	12790.382	7.156	.010	.140
Time * Age	Sphericity Assumed	1041.175	2	520.587	.582	.561	.013
	Greenhouse-Geisser	1041.175	1.911	544.855	.582	.553	.013
	Huynh-Feldt	1041.175	2.000	520.587	.582	.561	.013
	Lower-bound	1041.175	1.000	1041.175	.582	.449	.013
Time * Group	Sphericity Assumed	1072.661	2	536.331	.600	.551	.013
	Greenhouse-Geisser	1072.661	1.911	561.332	.600	.544	.013
	Huynh-Feldt	1072.661	2.000	536.331	.600	.551	.013
	Lower-bound	1072.661	1.000	1072.661	.600	.443	.013
Time * Age * Group	Sphericity Assumed	1378.070	2	689.035	.771	.466	.017
	Greenhouse-Geisser	1378.070	1.911	721.154	.771	.460	.017
	Huynh-Feldt	1378.070	2.000	689.035	.771	.466	.017
	Lower-bound	1378.070	1.000	1378.070	.771	.385	.017
Error(Time)	Sphericity Assumed	78646.882	88	893.715			
	Greenhouse-Geisser	78646.882	84.081	935.375			
	Huynh-Feldt	78646.882	88.000	893.715			
	Lower-bound	78646.882	44.000	1787.429			

ANOVA Summary Table

Measure: ANT_{CO-A}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.003	2	.001	.321	.726	.007
	Greenhouse-Geisser	.003	1.686	.002	.321	.689	.007
	Huynh-Feldt	.003	1.866	.002	.321	.711	.007
	Lower-bound	.003	1.000	.003	.321	.574	.007
Time * Age	Sphericity Assumed	.004	2	.002	.445	.642	.010
	Greenhouse-Geisser	.004	1.686	.002	.445	.609	.010
	Huynh-Feldt	.004	1.866	.002	.445	.629	.010
	Lower-bound	.004	1.000	.004	.445	.508	.010
Time * Group	Sphericity Assumed	.008	2	.004	.894	.413	.020
	Greenhouse-Geisser	.008	1.686	.005	.894	.398	.020
	Huynh-Feldt	.008	1.866	.004	.894	.407	.020
	Lower-bound	.008	1.000	.008	.894	.350	.020
Time * Age * Group	Sphericity Assumed	.006	2	.003	.739	.481	.017
	Greenhouse-Geisser	.006	1.686	.004	.739	.460	.017
	Huynh-Feldt	.006	1.866	.003	.739	.472	.017
	Lower-bound	.006	1.000	.006	.739	.395	.017
Error(Time)	Sphericity Assumed	.385	88	.004			
	Greenhouse-Geisser	.385	74.197	.005			
	Huynh-Feldt	.385	82.089	.005			
	Lower-bound	.385	44.000	.009			

4.4.3. $IsoK_{45}$

There was a significant time × group ($p < 0.001$, ES = .283) interaction for PT. When collapsed, PT in the CGs significantly decreased at MID ($p = 0.009$, -7.8%) and POST ($p < 0.001$, -13.0%). This was largely influenced by the YCG which decreased significantly decreased more than the OCG from MID to POST (OCG = -.8%; YCG = -10.8%). Neither the YTG nor OTG increased strength in the untrained leg ($p = 0.641$ and $p = 0.098$, respectfully). A significant time × group interaction was revealed for RVD ($p = 0.016$, ES =

.100) (Figure 13). There was no change in RVD for the YCG and OCG (p = 0.064), although a moderate decrease was demonstrated (-6.6%). Post hoc analyses revealed there was no change across time in the YTG (p = 0.669, ES = .036), while the OTG increased RVD from MID to POST (p = 0.014, ES = .365, +3.4%). Furthermore, there was neither a significant interaction (p = 0.248, ES = .064,) nor main effect for time (p = 0.253, ES = .069) when RVD was compared between YTG and OTG across time. The means for RER at PRE, MID, and POST for the YTG, OTG, YCG, and OCG are displayed in Figure 14. There were no significant interactions or main effects for EMG_{MAX}, RER, or $ANT_{CO\text{-}A}$ (p > 0.05).

ANOVA Summary Table

Measure: PT

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	1156.095	2	578.047	3.104	.050	.070
	Greenhouse-Geisser	1156.095	1.603	721.064	3.104	.062	.070
	Huynh-Feldt	1156.095	1.781	649.276	3.104	.057	.070
	Lower-bound	1156.095	1.000	1156.095	3.104	.086	.070
Time * Age	Sphericity Assumed	1445.785	2	722.892	3.882	.024	.086
	Greenhouse-Geisser	1445.785	1.603	901.745	3.882	.034	.086
	Huynh-Feldt	1445.785	1.781	811.970	3.882	.029	.086
	Lower-bound	1445.785	1.000	1445.785	3.882	.056	.086
Time * Group	Sphericity Assumed	6033.262	2	3016.631	16.200	.000	.283
	Greenhouse-Geisser	6033.262	1.603	3762.985	16.200	.000	.283
	Huynh-Feldt	6033.262	1.781	3388.351	16.200	.000	.283
	Lower-bound	6033.262	1.000	6033.262	16.200	.000	.283
Time * Age * Group	Sphericity Assumed	551.394	2	275.697	1.481	.234	.035
	Greenhouse-Geisser	551.394	1.603	343.908	1.481	.235	.035
	Huynh-Feldt	551.394	1.781	309.669	1.481	.235	.035
	Lower-bound	551.394	1.000	551.394	1.481	.231	.035
Error(Time)	Sphericity Assumed	15269.627	82	186.215			
	Greenhouse-Geisser	15269.627	65.736	232.287			
	Huynh-Feldt	15269.627	73.004	209.161			
	Lower-bound	15269.627	41.000	372.430			

ANOVA Summary Table

Measure: RVD

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	142.584	2	71.292	.097	.908	.002
	Greenhouse-Geisser	142.584	1.999	71.340	.097	.908	.002
	Huynh-Feldt	142.584	2.000	71.292	.097	.908	.002
	Lower-bound	142.584	1.000	142.584	.097	.757	.002
Time * Age	Sphericity Assumed	2054.076	2	1027.038	1.393	.254	.034
	Greenhouse-Geisser	2054.076	1.999	1027.730	1.393	.254	.034
	Huynh-Feldt	2054.076	2.000	1027.038	1.393	.254	.034
	Lower-bound	2054.076	1.000	2054.076	1.393	.245	.034
Time * Group	Sphericity Assumed	6380.749	2	3190.375	4.329	.016	.100
	Greenhouse-Geisser	6380.749	1.999	3192.526	4.329	.017	.100
	Huynh-Feldt	6380.749	2.000	3190.375	4.329	.016	.100
	Lower-bound	6380.749	1.000	6380.749	4.329	.044	.100
Time * Age * Group	Sphericity Assumed	836.488	2	418.244	.567	.569	.014
	Greenhouse-Geisser	836.488	1.999	418.526	.567	.569	.014
	Huynh-Feldt	836.488	2.000	418.244	.567	.569	.014
	Lower-bound	836.488	1.000	836.488	.567	.456	.014
Error(Time)	Sphericity Assumed	57490.057	78	737.052			
	Greenhouse-Geisser	57490.057	77.947	737.549			
	Huynh-Feldt	57490.057	78.000	737.052			
	Lower-bound	57490.057	39.000	1474.104			

ANOVA Summary Table

Measure: EMG_{MAX}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.180	2	.090	1.597	.209	.037
	Greenhouse-Geisser	.180	1.886	.096	1.597	.210	.037
	Huynh-Feldt	.180	2.000	.090	1.597	.209	.037
	Lower-bound	.180	1.000	.180	1.597	.213	.037
Time * Age	Sphericity Assumed	.177	2	.089	1.573	.214	.037
	Greenhouse-Geisser	.177	1.886	.094	1.573	.215	.037
	Huynh-Feldt	.177	2.000	.089	1.573	.214	.037
	Lower-bound	.177	1.000	.177	1.573	.217	.037
Time * Group	Sphericity Assumed	.158	2	.079	1.398	.253	.033
	Greenhouse-Geisser	.158	1.886	.084	1.398	.253	.033
	Huynh-Feldt	.158	2.000	.079	1.398	.253	.033
	Lower-bound	.158	1.000	.158	1.398	.244	.033
Time * Age * Group	Sphericity Assumed	.187	2	.093	1.654	.198	.039
	Greenhouse-Geisser	.187	1.886	.099	1.654	.199	.039
	Huynh-Feldt	.187	2.000	.093	1.654	.198	.039
	Lower-bound	.187	1.000	.187	1.654	.206	.039
Error(Time)	Sphericity Assumed	4.624	82	.056			
	Greenhouse-Geisser	4.624	77.307	.060			
	Huynh-Feldt	4.624	82.000	.056			
	Lower-bound	4.624	41.000	.113			

ANOVA Summary Table

Measure: RER

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	54.040	2	27.020	1.775	.176	.042
	Greenhouse-Geisser	54.040	1.899	28.462	1.775	.178	.042
	Huynh-Feldt	54.040	2.000	27.020	1.775	.176	.042
	Lower-bound	54.040	1.000	54.040	1.775	.190	.042
Time * Age	Sphericity Assumed	31.473	2	15.736	1.034	.360	.025
	Greenhouse-Geisser	31.473	1.899	16.576	1.034	.357	.025
	Huynh-Feldt	31.473	2.000	15.736	1.034	.360	.025
	Lower-bound	31.473	1.000	31.473	1.034	.315	.025
Time * Group	Sphericity Assumed	82.540	2	41.270	2.712	.072	.062
	Greenhouse-Geisser	82.540	1.899	43.473	2.712	.075	.062
	Huynh-Feldt	82.540	2.000	41.270	2.712	.072	.062
	Lower-bound	82.540	1.000	82.540	2.712	.107	.062
Time * Age * Group	Sphericity Assumed	91.513	2	45.757	3.007	.055	.068
	Greenhouse-Geisser	91.513	1.899	48.199	3.007	.058	.068
	Huynh-Feldt	91.513	2.000	45.757	3.007	.055	.068
	Lower-bound	91.513	1.000	91.513	3.007	.090	.068
Error(Time)	Sphericity Assumed	1247.948	82	15.219			
	Greenhouse-Geisser	1247.948	77.845	16.031			
	Huynh-Feldt	1247.948	82.000	15.219			
	Lower-bound	1247.948	41.000	30.438			

ANOVA Summary Table

Measure: ANT_{CO-A}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.048	2	.024	2.911	.060	.066
	Greenhouse-Geisser	.048	1.927	.025	2.911	.062	.066
	Huynh-Feldt	.048	2.000	.024	2.911	.060	.066
	Lower-bound	.048	1.000	.048	2.911	.096	.066
Time * Age	Sphericity Assumed	.004	2	.002	.233	.793	.006
	Greenhouse-Geisser	.004	1.927	.002	.233	.785	.006
	Huynh-Feldt	.004	2.000	.002	.233	.793	.006
	Lower-bound	.004	1.000	.004	.233	.632	.006
Time * Group	Sphericity Assumed	.009	2	.004	.531	.590	.013
	Greenhouse-Geisser	.009	1.927	.005	.531	.583	.013
	Huynh-Feldt	.009	2.000	.004	.531	.590	.013
	Lower-bound	.009	1.000	.009	.531	.470	.013
Time * Age * Group	Sphericity Assumed	.000	2	.000	.021	.979	.001
	Greenhouse-Geisser	.000	1.927	.000	.021	.976	.001
	Huynh-Feldt	.000	2.000	.000	.021	.979	.001
	Lower-bound	.000	1.000	.000	.021	.884	.001
Error(Time)	Sphericity Assumed	.683	82	.008			
	Greenhouse-Geisser	.683	79.017	.009			
	Huynh-Feldt	.683	82.000	.008			
	Lower-bound	.683	41.000	.017			

4.4.4. IsoK$_{500}$

There were no interactions or main effects present for PV (Figure 15). A significant time × group interaction was demonstrated for RVD (p = 0.024, ES = .091), while no other interactions were present (p > 0.05) (Figure 15). There was no change for RVD in the YCG or OCG (p = 0.424, POST = -2.2%). When both training groups were collapsed, RVD was significantly greater at POST compared to PRE (p = 0.012, ES = .135, +4.0%), however; there were no differences in the increase of RVD between YTG and OTG across time (p = 0.964). EMG$_{MAX}$ remained unchanged as there were no significant interactions or main effects (p > 0.05). A main effect for time indicated that the YCG and OCG significantly decreased RER at POST (p = 0.012, -23.3%) Neither the YTG (p = .318) nor OTG (p = 0.391) demonstrated a significant change in RER. There were no interactions or main effects present for ANT$_{CO-A}$ (p > 0.05).

ANOVA Summary Table

Measure: PV

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	265.747	2	132.873	1.379	.258	.032
	Greenhouse-Geisser	265.747	1.888	140.792	1.379	.258	.032
	Huynh-Feldt	265.747	2.000	132.873	1.379	.258	.032
	Lower-bound	265.747	1.000	265.747	1.379	.247	.032
Time * Age	Sphericity Assumed	148.461	2	74.230	.770	.466	.018
	Greenhouse-Geisser	148.461	1.888	78.654	.770	.459	.018
	Huynh-Feldt	148.461	2.000	74.230	.770	.466	.018
	Lower-bound	148.461	1.000	148.461	.770	.385	.018
Time * Group	Sphericity Assumed	354.276	2	177.138	1.838	.165	.042
	Greenhouse-Geisser	354.276	1.888	187.695	1.838	.168	.042
	Huynh-Feldt	354.276	2.000	177.138	1.838	.165	.042
	Lower-bound	354.276	1.000	354.276	1.838	.182	.042
Time * Age * Group	Sphericity Assumed	234.010	2	117.005	1.214	.302	.028
	Greenhouse-Geisser	234.010	1.888	123.978	1.214	.301	.028
	Huynh-Feldt	234.010	2.000	117.005	1.214	.302	.028
	Lower-bound	234.010	1.000	234.010	1.214	.277	.028
Error(Time)	Sphericity Assumed	8095.035	84	96.369			
	Greenhouse-Geisser	8095.035	79.276	102.113			
	Huynh-Feldt	8095.035	84.000	96.369			
	Lower-bound	8095.035	42.000	192.739			

ANOVA Summary Table

Measure: RVD

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	24742.820	2	12371.410	.651	.524	.015
	Greenhouse-Geisser	24742.820	1.736	14249.679	.651	.504	.015
	Huynh-Feldt	24742.820	1.934	12793.022	.651	.519	.015
	Lower-bound	24742.820	1.000	24742.820	.651	.424	.015
Time * Age	Sphericity Assumed	8807.067	2	4403.533	.232	.794	.005
	Greenhouse-Geisser	8807.067	1.736	5072.092	.232	.763	.005
	Huynh-Feldt	8807.067	1.934	4553.604	.232	.786	.005
	Lower-bound	8807.067	1.000	8807.067	.232	.633	.005
Time * Group	Sphericity Assumed	159121.849	2	79560.924	4.185	.019	.091
	Greenhouse-Geisser	159121.849	1.736	91640.131	4.185	.024	.091
	Huynh-Feldt	159121.849	1.934	82272.322	4.185	.020	.091
	Lower-bound	159121.849	1.000	159121.849	4.185	.047	.091
Time * Age * Group	Sphericity Assumed	2869.524	2	1434.762	.075	.927	.002
	Greenhouse-Geisser	2869.524	1.736	1652.592	.075	.904	.002
	Huynh-Feldt	2869.524	1.934	1483.658	.075	.922	.002
	Lower-bound	2869.524	1.000	2869.524	.075	.785	.002
Error(Time)	Sphericity Assumed	1596782.356	84	19009.314			
	Greenhouse-Geisser	1596782.356	72.928	21895.372			
	Huynh-Feldt	1596782.356	81.232	19657.142			
	Lower-bound	1596782.356	42.000	38018.628			

ANOVA Summary Table

Measure: EMG_{MAX}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.177	2	.089	1.071	.347	.025
	Greenhouse-Geisser	.177	1.919	.092	1.071	.345	.025
	Huynh-Feldt	.177	2.000	.089	1.071	.347	.025
	Lower-bound	.177	1.000	.177	1.071	.307	.025
Time * Age	Sphericity Assumed	.396	2	.198	2.394	.097	.054
	Greenhouse-Geisser	.396	1.919	.207	2.394	.100	.054
	Huynh-Feldt	.396	2.000	.198	2.394	.097	.054
	Lower-bound	.396	1.000	.396	2.394	.129	.054
Time * Group	Sphericity Assumed	.049	2	.024	.293	.747	.007
	Greenhouse-Geisser	.049	1.919	.025	.293	.737	.007
	Huynh-Feldt	.049	2.000	.024	.293	.747	.007
	Lower-bound	.049	1.000	.049	.293	.591	.007
Time * Age * Group	Sphericity Assumed	.089	2	.045	.538	.586	.013
	Greenhouse-Geisser	.089	1.919	.046	.538	.579	.013
	Huynh-Feldt	.089	2.000	.045	.538	.586	.013
	Lower-bound	.089	1.000	.089	.538	.468	.013
Error(Time)	Sphericity Assumed	6.955	84	.083			
	Greenhouse-Geisser	6.955	80.586	.086			
	Huynh-Feldt	6.955	84.000	.083			
	Lower-bound	6.955	42.000	.166			

ANOVA Summary Table

Measure: RER

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	21.897	2	10.949	.680	.509	.016
	Greenhouse-Geisser	21.897	1.620	13.513	.680	.480	.016
	Huynh-Feldt	21.897	1.796	12.189	.680	.495	.016
	Lower-bound	21.897	1.000	21.897	.680	.414	.016
Time * Age	Sphericity Assumed	22.887	2	11.444	.711	.494	.017
	Greenhouse-Geisser	22.887	1.620	14.124	.711	.467	.017
	Huynh-Feldt	22.887	1.796	12.740	.711	.480	.017
	Lower-bound	22.887	1.000	22.887	.711	.404	.017
Time * Group	Sphericity Assumed	161.680	2	80.840	5.022	.009	.107
	Greenhouse-Geisser	161.680	1.620	99.774	5.022	.014	.107
	Huynh-Feldt	161.680	1.796	89.998	5.022	.011	.107
	Lower-bound	161.680	1.000	161.680	5.022	.030	.107
Time * Age * Group	Sphericity Assumed	56.653	2	28.327	1.760	.178	.040
	Greenhouse-Geisser	56.653	1.620	34.961	1.760	.185	.040
	Huynh-Feldt	56.653	1.796	31.536	1.760	.182	.040
	Lower-bound	56.653	1.000	56.653	1.760	.192	.040
Error(Time)	Sphericity Assumed	1352.195	84	16.098			
	Greenhouse-Geisser	1352.195	68.059	19.868			
	Huynh-Feldt	1352.195	75.452	17.921			
	Lower-bound	1352.195	42.000	32.195			

ANOVA Summary Table

Measure: ANT_{CO-A}

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Time	Sphericity Assumed	.061	2	.031	2.308	.106	.052
	Greenhouse-Geisser	.061	1.948	.031	2.308	.107	.052
	Huynh-Feldt	.061	2.000	.031	2.308	.106	.052
	Lower-bound	.061	1.000	.061	2.308	.136	.052
Time * Age	Sphericity Assumed	.001	2	.001	.047	.954	.001
	Greenhouse-Geisser	.001	1.948	.001	.047	.951	.001
	Huynh-Feldt	.001	2.000	.001	.047	.954	.001
	Lower-bound	.001	1.000	.001	.047	.829	.001
Time * Group	Sphericity Assumed	.040	2	.020	1.516	.225	.035
	Greenhouse-Geisser	.040	1.948	.021	1.516	.226	.035
	Huynh-Feldt	.040	2.000	.020	1.516	.225	.035
	Lower-bound	.040	1.000	.040	1.516	.225	.035
Time * Age * Group	Sphericity Assumed	.022	2	.011	.832	.439	.019
	Greenhouse-Geisser	.022	1.948	.011	.832	.436	.019
	Huynh-Feldt	.022	2.000	.011	.832	.439	.019
	Lower-bound	.022	1.000	.022	.832	.367	.019
Error(Time)	Sphericity Assumed	1.112	84	.013			
	Greenhouse-Geisser	1.112	81.837	.014			
	Huynh-Feldt	1.112	84.000	.013			
	Lower-bound	1.112	42.000	.026			

Tables

Table 1. Number of MUs analyzed for the trained leg in the training groups and dominant leg in the control groups.

Young	**PRE**	**MID**	**POST**	**Total**
Training Group	154	137	125	416
Control Group	60	62	71	193
Total	214	199	196	609
Old				
Training Group	210	226	192	628
Control Group	116	104	107	327
Total	326	330	299	955

Table 2. Number of MUs analyzed for the untrained leg in the training groups and non-dominant leg in the control groups.

Young	**PRE**	**MID**	**POST**	**Total**
Training Group	160	170	165	495
Control Group	90	122	113	325
Total	250	292	165	707
Old				
Training Group	243	274	247	764
Control Group	115	111	121	347
Total	358	385	368	1,111

Figures

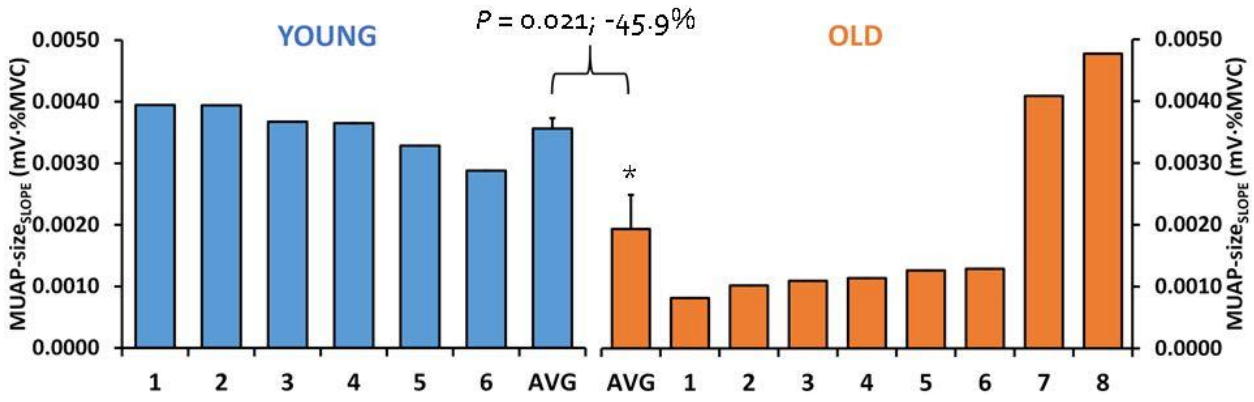


Figure 1. Individual and average slope coefficients for MUAP-size vs. RT relationship (MUAP-size$_{SLOPE}$) in young and old group. * indicates significantly lower compared to young training group. Error bars represent SEM.

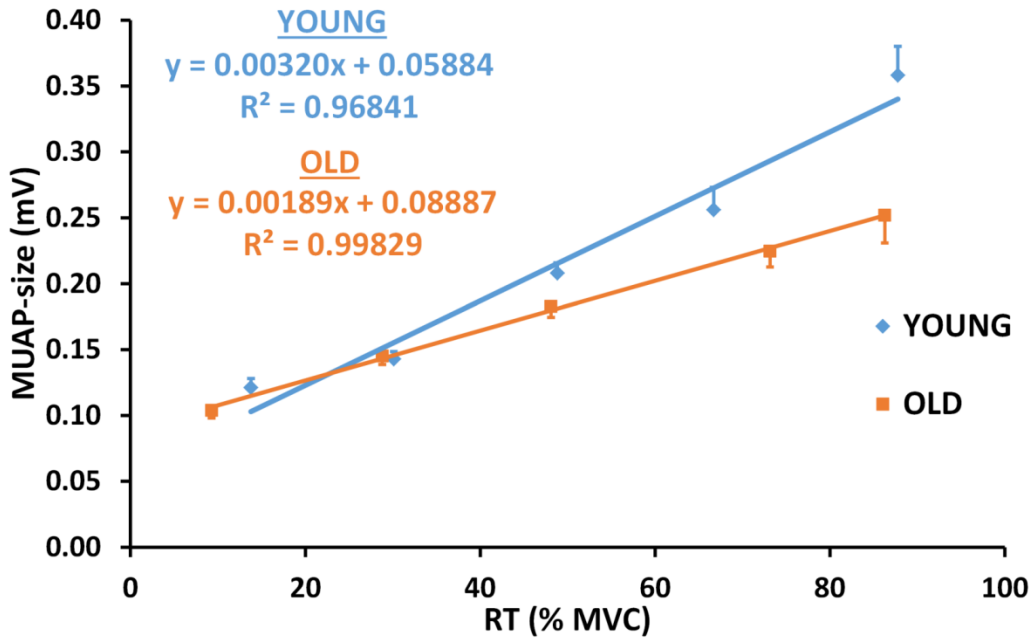


Figure 2. Relationship for pooled data (all training group subjects) between MUAP-size and RT at PRE for the young training group and old training group. Error bars represent SEM.

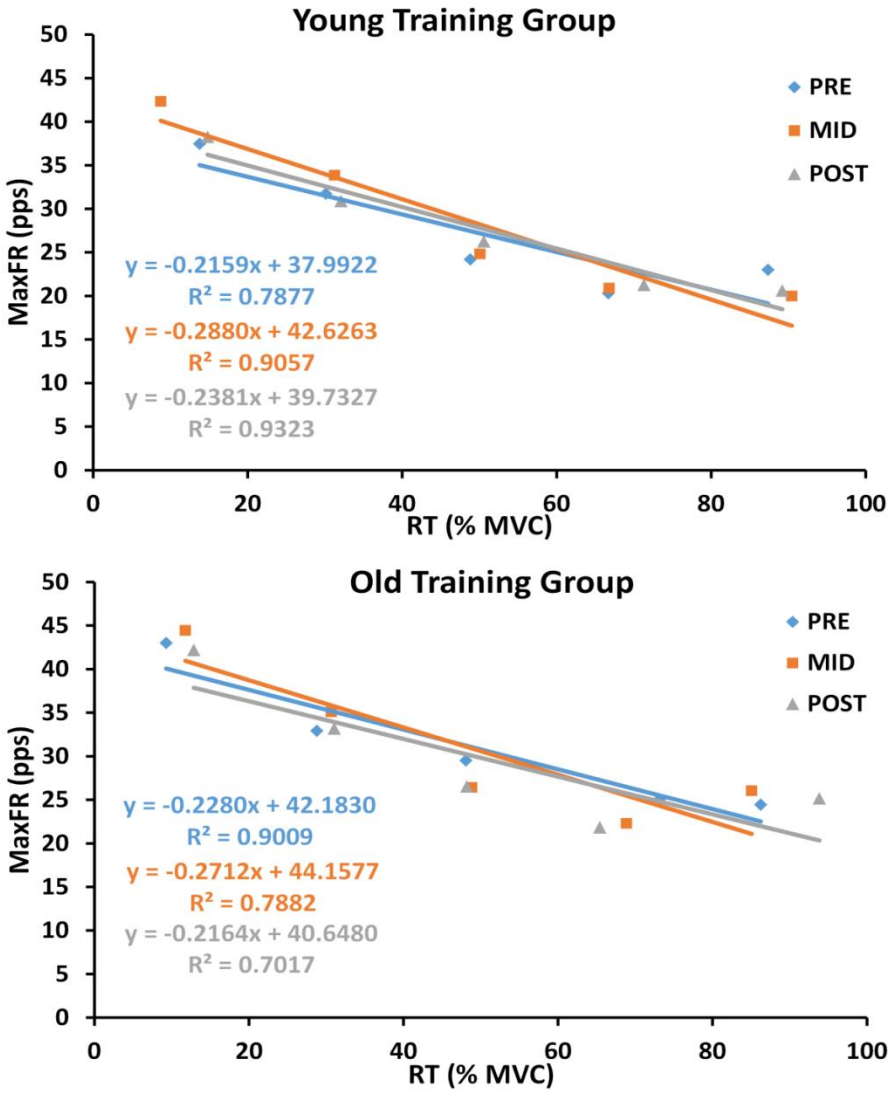


Figure 3. Relationship for pooled data (all training groups subjects) between MaxFR and RT at PRE, MID, and POST in the trained leg for the young and old training group.

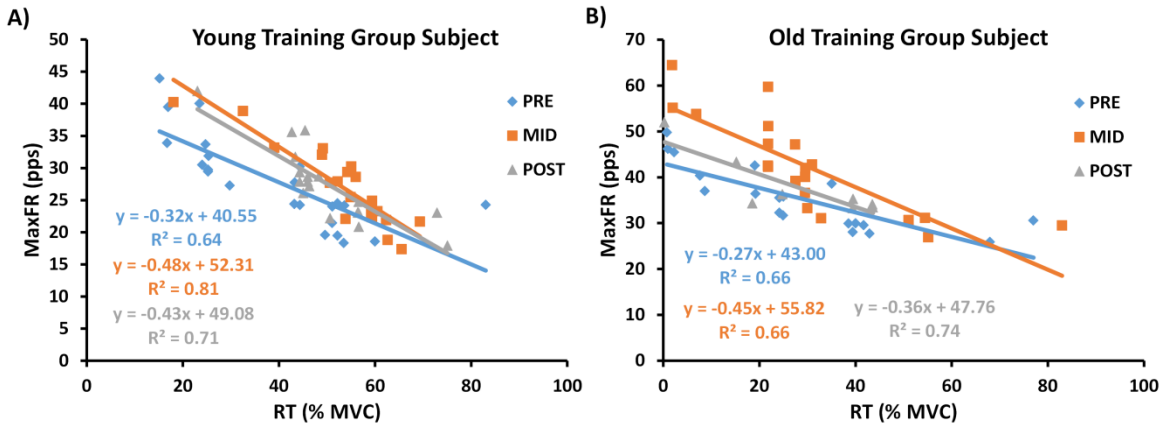


Figure 4. Relationship between MaxFR and RT at PRE, MID, and POST for the trained leg of a single subject from the YTG (A) and OTG (B).

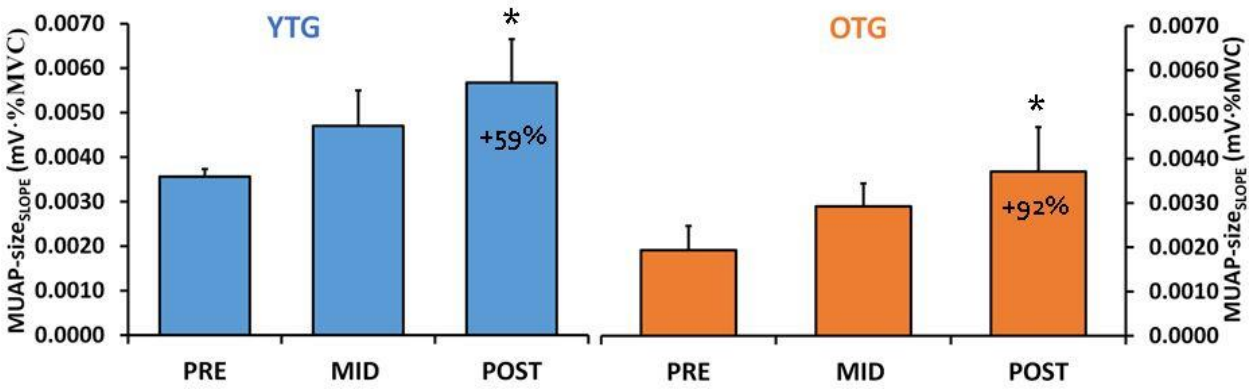


Figure 5. Average slope coefficients at PRE, MID, and POST for MUAP-size vs. RT relationship (MUAP-size$_{SLOPE}$) in the trained leg for the young training group (YTG) and old training group (OTG). * Indicates significant main effect (when groups were collapsed) MUAP-size$_{SLOPE}$ was increased at POST (p = 0.013) compared to PRE. Error bars represent SEM.

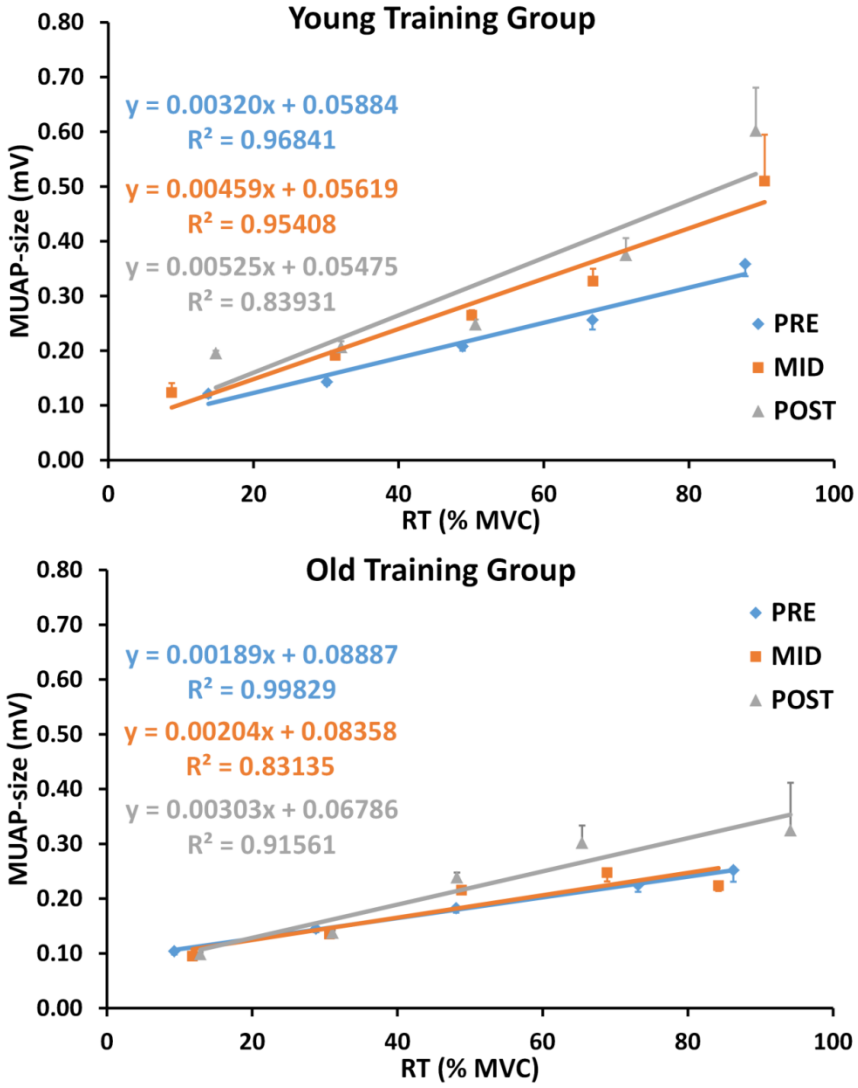


Figure 6. Relationship for pooled data (all training group subjects) between MUAP-size and RT at PRE, MID, and POST in the trained leg for the YTG and OTG. Error bars represent SEM.

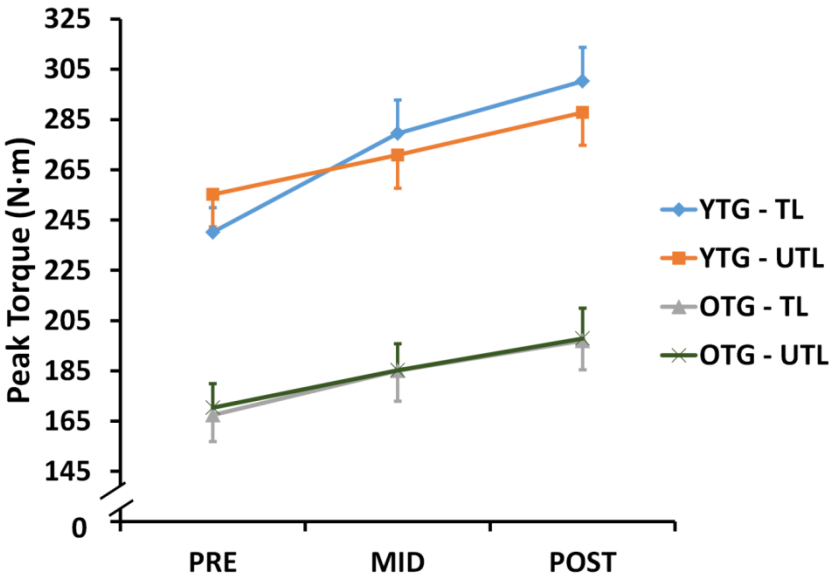


Figure 7. Peak torque at PRE, MID, and POST for the trained leg (TL) and untrained leg (UTL) of the young training group (YTG) and old training group (OTG). Error bars represent SEM.

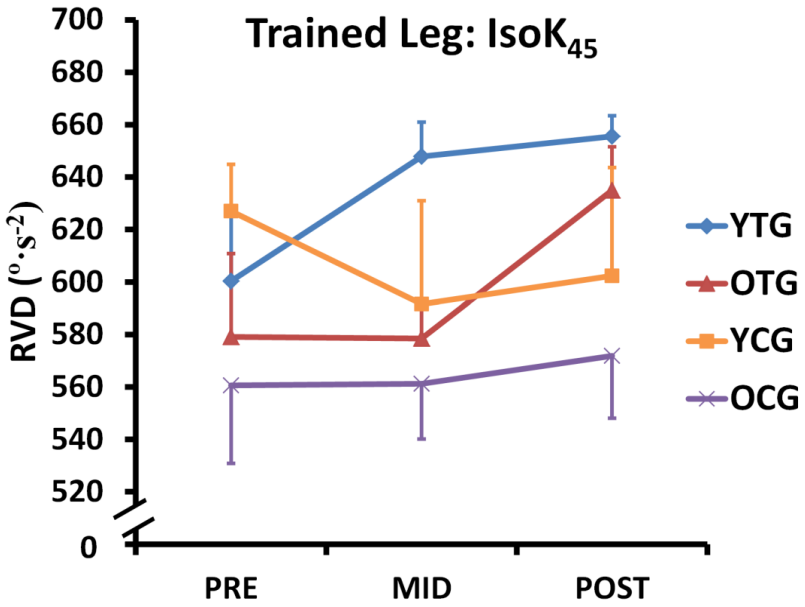


Figure 8. Rate of velocity development (RVD) at PRE, MID, and POST for the TL/DL. Error bars represent SEM.

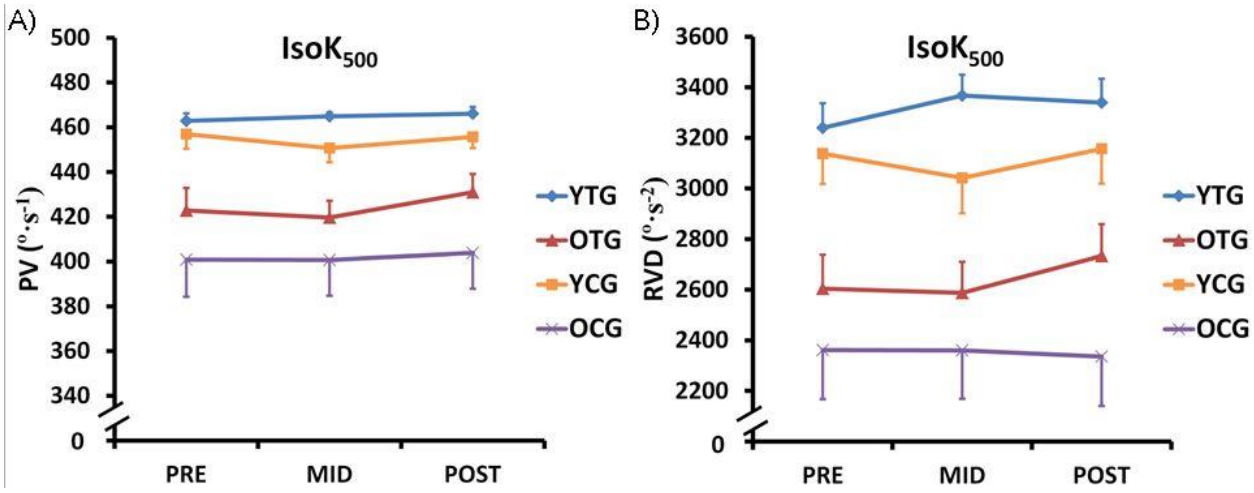


Figure 9. PV (A) and RVD (B) for the trained/dominant leg at PRE, MID, and POST in the young training group (YTG), old training group (OTG), young control group (YCG), and old control group (OCG). Error bars represent SEM.

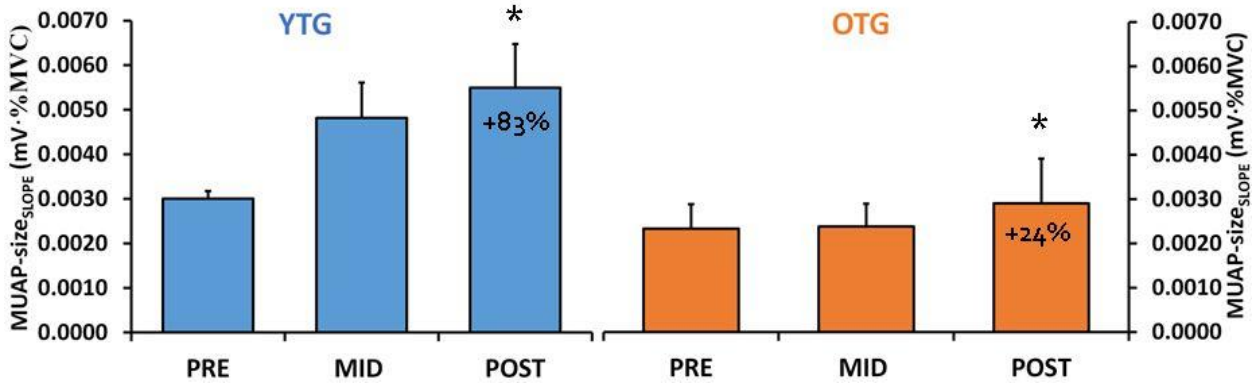


Figure 10. Average slope coefficients for MUAP-size vs. RT relationship (MUAP-size$_{SLOPE}$) in the untrained leg for the young training group (YTG) and old training group (OTG). Error bars represent SEM. * indicates significant main effect (when groups were collapsed) MUAP-size$_{SLOPE}$ was increased at POST compare to PRE (p = 0.001).

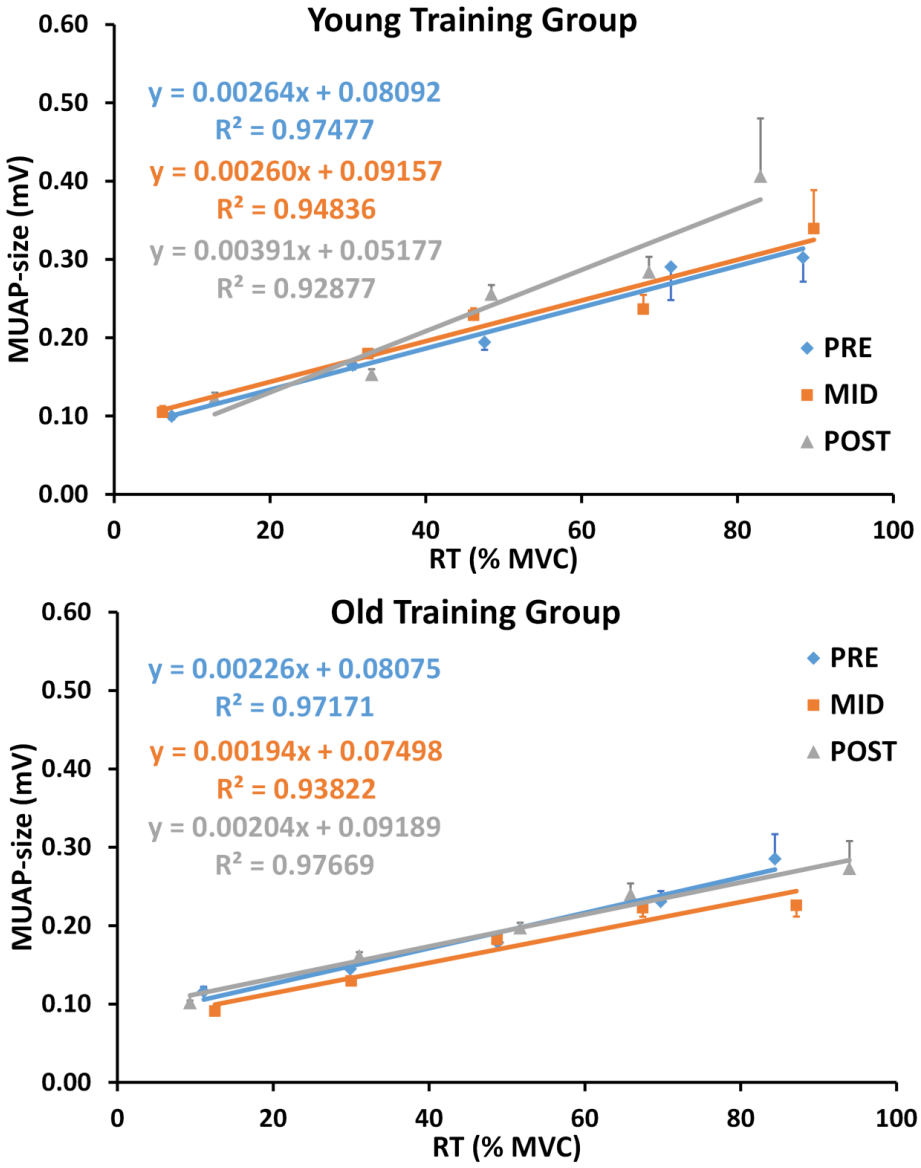


Figure 11. Relationship for pooled data (all training group subjects) between MUAP-size and RT at PRE, MID, and POST in the untrained leg for the young training group and old training group. Error bars represent SEM.

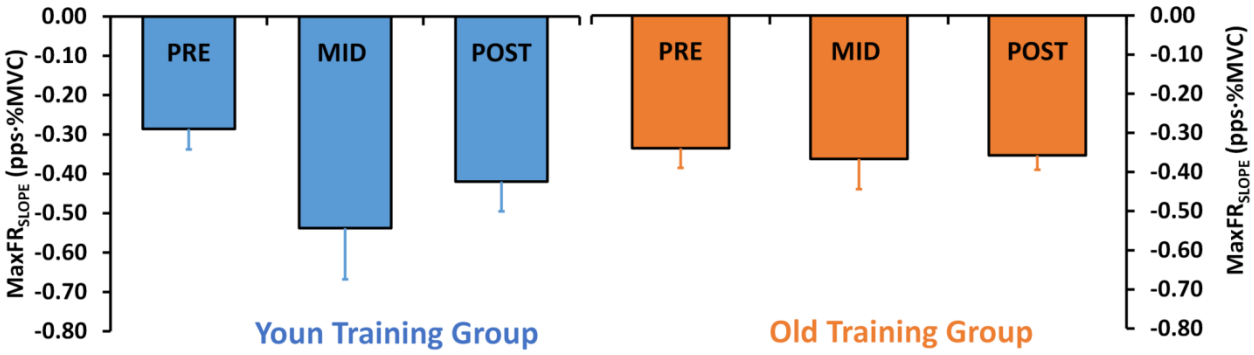


Figure 12. Average slope coefficients for MaxFR vs. RT relationship (MaxFR$_{SLOPE}$) in the untrained leg for the young training group and old training group. Error bars represent SEM.

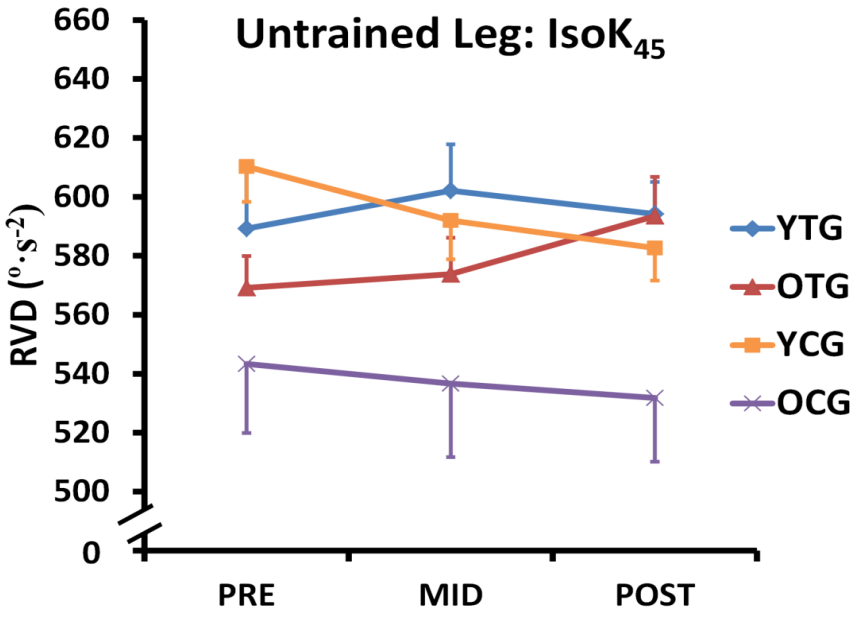


Figure 13. RVD at PRE, MID, and POST for the untrained leg in the young training group (YTG), old training group (OTG), young control group (YCG), and old control group (OCG). Error bars represent SEM.

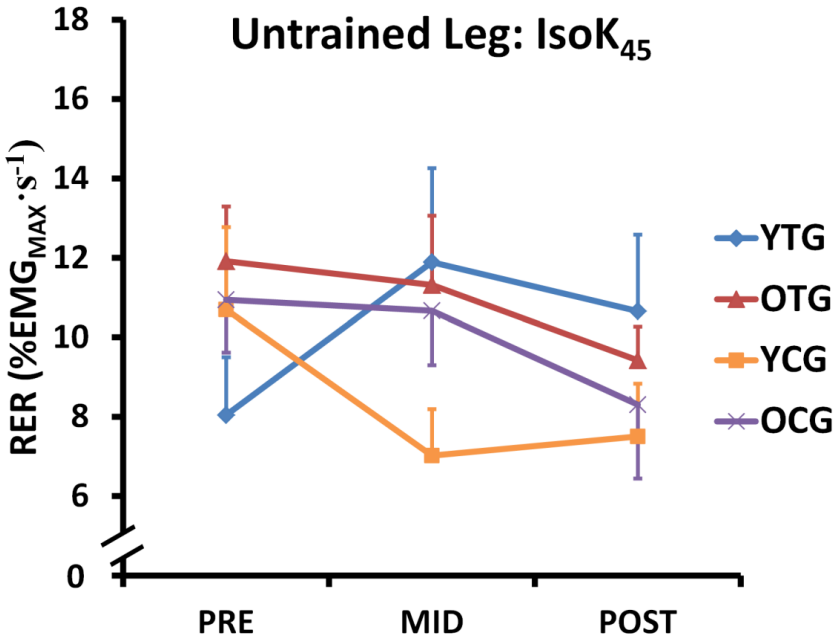


Figure 14. RER at PRE, MID, and POST for the untrained leg in the young training group (YTG), old training group (OTG), young control group (YCG), and old control group (OCG). Error bars represent SEM.

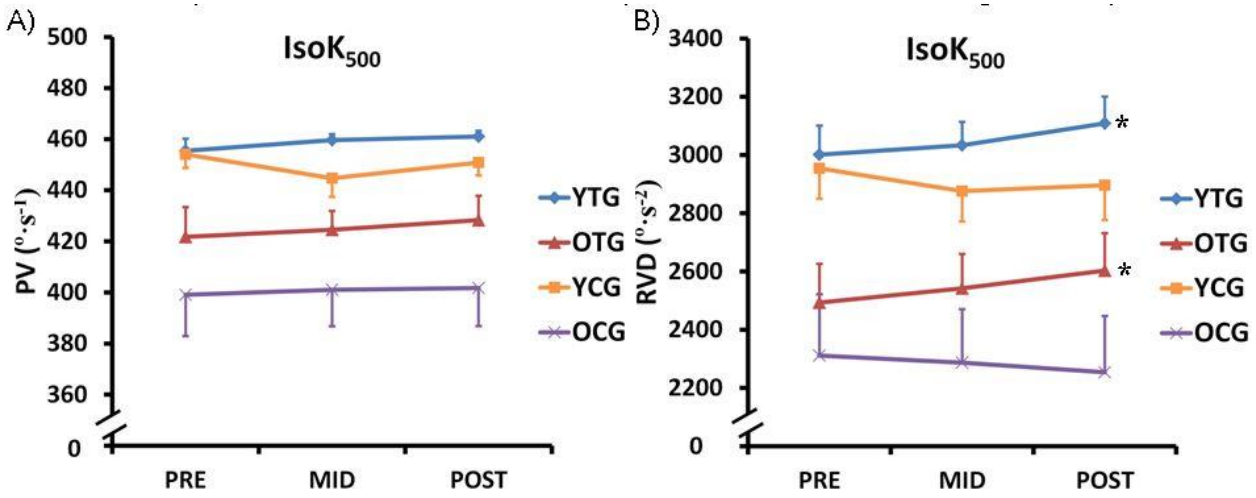


Figure 15. PV (A) and RVD (B) at PRE, MID, and POST for the untrained/non-dominant leg in the young training group (YTG), old training group (OTG), young control group (YCG), and old control group (OCG). * indicates significant main effect (when training groups were collapsed) significant at POST compared to PRE. Error bars represent SEM.

5. DISCUSSION

5.1. Implications and Significance

The focus of this study was to examine age-related differences in select MU properties and the training-induced adaptations of these properties in addition to other neuromuscular parameters. This study used recently developed technology in an effort to answer questions regarding MU properties and aging that remain elusive. In addition, the ability of unique neuromuscular measures to be enhanced in the untrained limb after unilateral training was investigated.

The effects of aging on MU firing rates and MUAP-size has been demonstrated in previous studies, with the former being more established. Unlike previous reports[19, 56, 86], the results of the present study did not demonstrate a reduction in maximum MU firing rates in older adults. An increased level of antagonist co-activation has been proposed as having an influential role on the firing rates of the agonist, however; antagonist co-activation was similar in older adults as compared to young adults in the present study. Previous studies have directly compared the firing rates (pulses per second) of MUs before and after training. We recorded and compared the slope coefficient of the MaxFR vs. RT relationship ($MaxFR_{SLOPE}$) which may not be as sensitive since this value can be influenced by differences in the RT of MUs.

In addition, the older adults in the current study were as young as 55 and the mean age (64 yrs) was less than that of previous studies reporting age-related differences in maximum MU firing rates[19, 85, 86]. While it is unclear, it is possible the sample used in the present study did not represent the same population investigated in previous reports.

One of the more interesting findings of the current study was that MUAP-size$_{SLOPE}$ was greater in young adults (+48.3%) as compared to older adults (see Figure 1) which was in accordance with our hypothesis. In other words, for every unit increase in RT (%MVC) the average increase in action potential amplitude for younger adults was 48.3% higher compared to older adults. A qualitative examination of the pooled data (Figure 1) indicates the largest discrepancies in MUAP-size are with the later recruited MUs (i.e., >60% MVC). This lends support to the notion that aging may be associated with a preferential loss in CSA of type II (i.e., high-threshold) muscle fibers in the VL due to aging[63, 67]. In contrast to these findings, previous researchers found MUAP-size to be larger in older adults[16, 31, 73]. Greater MUAP-size in older adults is believed to be the result of reinnervation in which additional fibers are innervated by the motor nerve of the MU and thus an increase in muscle fiber area occurs[30]. However, subjects performed light-intensity contractions in these studies, thus it is possible the sample of MUs obtained were predominately low-threshold MUs whose properties are likely the most influenced, as compared to high-threshold MUs, by reinnervation[58]. In the present study subjects performed maximal voluntary contractions and MUs were recruited as high as 96% MVC, thereby providing a large sample of low- and high-threshold MUs. Importantly, the detection of such a broad range of MUs may result in the detection of MUs at a variety of depths which has been found to attenuate amplitude[40],

however, Brown and Stein[75] demonstrated that MUs (i.e., their constituent fibers) are distributed in a relatively uniform manner across a broad regions and are interposed with fibers from other MUs. Furthermore, the decomposition of surface EMG has been recently used to examine MUAP-size across the RT and no correlation was found for MUAP duration (a measure of MU depth) and MU RT[50]. Although, it should be noted the aforementioned study investigated MUAP-size for MUs of a small hand muscle, thus the composition of tissue (e.g., subcutaneous fat) differs from the VL, and there is likely greater inter-individual variability for the VL region. Furthermore, a complication with making cross-sectional comparisons (i.e., young vs. old) for MUAP-size, is the potential age-related differences in the amount of subcutaneous fat for the region of the VL whereas it would be expected that older adults possess greater levels of subcutaneous fat.

There was a training-induced increase for $MaxFR_{SLOPE}$ and $MUAP\text{-}size_{SLOPE}$ for both the YTG and OTG in the trained leg (i.e., collapsed across training groups) with no age-related differences being indicated. $MaxFR_{SLOPE}$ was significantly greater (i.e., steeper slope) after 2 weeks (+43.0%) and after 4 weeks (+42.6%) of resistance training (Figures 2 and 3). This indicates an alteration in MU rate coding as a neural adaptation during the early phases of resistance training. These findings support previous research demonstrating changes in maximum MU firing rates for older and younger adults during the first few weeks of resistance training[19, 56], although others have reported no change in firing rates after isometric training[86, 91]. It appears that dynamic resistance may be more effective at augmenting maximum MU firing rates, but not necessarily average MU firing rates. We failed to accept the alternative hypothesis that unilateral training would increase MU firing

rates in the untrained limb. In contrast with one report of increased maximum MU firing rates after two days of resistance training[86], maximum MU firing rates were unaltered in the untrained limb. The hypothesis was based on the finding that unilateral training increases cortical voluntary activation in the contralateral limb[65] which could increase synaptic input to the spinal motor neurons and increases MU firing rates. Another possible mechanism for the strength increase in the untrained limb would be a training-induced increase in the number of MUs capable of being recruited. This would increase the number of muscle fibers contributing to force production, but not necessarily the MU firing rates.

MUAP-size$_{SLOPE}$ was significantly increased after 4 weeks of resistance training for the trained leg (+42.3%) and untrained leg (+34.2%) with no age-related differences being identified (Figure 1). This finding only partially supports the hypothesis as it was believed MUAP-size$_{SLOPE}$ would only be increased in the trained leg for the young training group only, but would not be altered for the either group in the untrained limb. MUAP amplitude is associated with the membrane area of the muscle fiber for which the potential is being recorded[43] such that an increase in muscle fiber area would likely augment MUAP-size due to greater current generation. Considering this relationship and our recent report that the change in the relationship between MUAP-size and RT was largely (83.6%) explained by training-induced changes in whole muscle[89], in the current study it was believed MUAP-size$_{SLOPE}$ would be indicative of changes in MU specific hypertrophy. Indeed, the change in MUAP-size$_{SLOPE}$ for the trained leg at 4 weeks after resistance training is in line with previous research demonstrating the early phases of training-induced hypertrophy[26, 96]. Furthermore, an examination of the pooled data in Figure 6 indicates that the largest training-

induced changes in the MUAP-size were demonstrated by the higher-threshold MUs (i.e., >60% MVC) for both the young and old training group. This indirectly suggests that the presumed larger, higher-threshold MUs exhibited the largest increases in muscle fiber hypertrophy. However, while a measure of hypertrophy was not obtained, it is presumed hypertrophy did not occur in the untrained limb especially after such short-term training[5, 10, 37]. Therefore, the finding that the untrained limb increased MUAP-size$_{SLOPE}$ nearly as much as the trained limb was an interesting and unexpected finding. An increase in sarcolemma excitability through increased sodium pump activity[48] could increase MUAP-size, however, sodium pump activity of the untrained limb would not necessarily be expected to change, at least acutely[39]. Otherwise, since MUAP-size$_{SLOPE}$ is influenced by the RT range of the MUs comprising the regression, it is possible that at POST a greater number of higher-threshold units were detected as compared to PRE and thus increased the slope coefficient. This may be evidenced by the increased variability observed for MUAP-size$_{SLOPE}$ and the non-linear increase for both groups at MID and POST for exhibited by the untrained limb in both groups as compared to the response of the trained limb.

As expected, younger adults in the current study demonstrated greater dynamic (IsoK45) and isometric (MVC) strength as compared to their older counterparts (26.7% and 26.2%, respectively). Surprisingly, a greater relative decrement for PV was revealed in older adults as compared to RVD for IsoK$_{500}$ (36.6% and 22.1%, respectively) which opposes previous research demonstrating a larger relative decrease in rapid measures of neuromuscular measures[52, 101]. While age-related differences in the adaptations to resistance training have been heavily researched, there is much less known regarding the early-phases

of resistance training (i.e., ≤4 weeks). To the best of the author's knowledge, the current study is the first to examine adaptations in maximal velocity characteristics (i.e., RVD, PV) of the knee extensors. As hypothesized, neither the young training group nor old training group increased PV or RVD for $IsoK_{500}$ in the trained limb, although the same was expected for the untrained limb as well but both the young and old training group increased RVD for $IsoK_{500}$ after 4 weeks of training. In addition, a surprising finding was that RVD for $IsoK_{45}$ increased from MID to POST (3.4%) for the OTG only in the untrained limb, despite their being no change in strength for $IsoK_{45}$. While the increase in RVD was hypothesized for the untrained limb, the same result was expected for the trained limb but neither training group increased RVD for $IsoK_{45}$ in this limb. Due to the short-term nature of the training, the training-induced increases in RVD for both $IsoK_{45}$ and $IsoK_{500}$ are likely the result of an adaptation within the CNS. An increase in RER for the training speed ($IsoK_{45}$) was hypothesized to accompany the change in RVD reflecting a potential increase in initial MU firing rates[61] or doublet firings[108] as the candidate mechanisms responsible for the increased performance. The finding that RVD can be enhanced after as few as two training sessions[90] in conjunction with our findings indicates that neural adaptations are responsible for training-related changes in this measure. While it is unclear, it is conceivable that after training subjects were able to recruit more MUs[29] during the early phases of the dynamic tests after resistance training.

In general, the findings of the present study indicated an attenuated response to resistance training in older adults compared to their younger counterparts. For example, while the old training group increased MVC strength after 2 and 4 weeks of training, the

increase in strength at week 2 for the young training group was larger in magnitude compared to the old training group. In addition, the young training group increased strength significantly from week 2 to week 4. The larger strength increase at week 2 demonstrated by the young group was accompanied by an increase in EMG_{MAX}, whereas EMG_{MAX} did not increase in the old training group until after week 4 of training. Since MU firing rates changed similarly between groups after 2 weeks of training and there were no differences between groups in the training-induced response of MUAP-$size_{SLOPE}$, the underlying mechanism for the age-related differences in EMG and strength responses is unclear.

There was no increase in strength in the old training group at the training speed, yet the young training group increased strength at 2 and 4 weeks compared to PRE. This finding differs from previous literature demonstrating that older adults are capable of exhibiting resistance training adaptations during the early phases (i.e., ≤4 weeks) of training[8, 10, 34]. It is unclear why the older training group failed to increase strength at the training speed as isokinetic training has produced plantarflexion strength gains in as early as 2 weeks[22]. The most probable rationale is due to the low sample size for this group (n = 11) and greater variability as compared to the young training group. Nevertheless, the reduced capacity for short-term strength gains is reflected by the dramatic differences in relative strength gains where the increase at 2 weeks (7.9%) in the young group was similar to the non-significant increase observed in the old group at 4 weeks (7.1%). Taken together, a diminished response to resistance training was exhibited by the old training group, it would appear the capacity for cross-education is not moderated by age.

Conclusions

This study provided novel evidence regarding changes in MU properties in older adults compared to young adults. The average slope coefficients for the relationship between MUAP-size and RT were less compared to their younger counterparts. These results indicated that older adults exhibited lower MUAP-size amplitudes, specifically in the higher-threshold MUs. Both, $MaxFR_{SLOPE}$ and $MUAP\text{-}size_{SLOPE}$ were found to be augmented for young and older adults after resistance training. We proposed that the latter finding may be indicative of a preferential hypertrophic response of the larger, higher-threshold MUs but limitations are considered in order to avoid over interpretation. Indeed, the finding of increased $MUAP\text{-}size_{SLOPE}$ for the untrained leg may pose as an indication that confounding factors influenced the $MUAP\text{-}size_{SLOPE}$ measure.

Older adults exhibited an attenuated response to resistance training as demonstrated by larger strength gains in the young adults. The larger initial response by young adults was accompanied by an earlier increase in muscle activation, but the reason for these differences are unknown. The finding in the current study that unilateral training increases RVD for the training speed and an unloaded knee extension in the untrained leg were novel findings that contribute to functional implications for cross-education.

REFERENCES

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

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

3. Aagaard P., Simonsen E.B., Trolle M., Bangsbo J. and Klausen K., Effects of different strength training regimes on moment and power generation during dynamic knee extensions. *European journal of applied physiology and occupational physiology,* 1994. 69(5): p. 382-386.

4. Aagaard P., Suetta C., Caserotti P., Magnusson S.P. and Kjær M., Role of the nervous system in sarcopenia and muscle atrophy with aging: strength training as a countermeasure. *Scandinavian journal of medicine & science in sports,* 2010. 20(1): p. 49-64.

5. Adamson M., MacQuaide N., Helgerud J., Hoff J. and Kemi O.J., Unilateral arm strength training improves contralateral peak force and rate of force development. *European journal of applied physiology,* 2008. 103(5): p. 553-559.

6. Akima H., Takahashi H., Kuno S.-Y., Masuda K., Masuda T., Anno I., Itai Y. and Katsuta S., Early phase adaptations of muscle use and strength to isokinetic training. *Medicine & Science in Sports & Exercise,* 1999. 31(4): p. 588-594.

7. Andersen J.L., Klitgaard H., Bangsbo J. and Saltin B., Myosin heavy chain isoforms in single fibres from m. vastus lateralis of soccer players: effects of strength-training. *Acta physiologica scandinavica,* 1994. 150(1): p. 21-26.

8. Barry B.K., Warman G.E. and Carson R.G., Age-related differences in rapid muscle activation after rate of force development training of the elbow flexors. *Experimental brain research,* 2005. 162(1): p. 122-132.

9. Beck T.W., DeFreitas J.M. and Stock M.S., The effects of a resistance training program on average motor unit firing rates. *Clinical Kinesiology,* 2011. 65(1): p. 1.

10. Bemben M. and Murphy R., Age related neural adaptation following short term resistance training in women. *Journal of sports medicine and physical fitness,* 2001. 41(3): p. 291.

11. Blazevich A.J., Gill N.D., Bronks R. and Newton R.U., Training-Specific Muscle Architecture Adaptation after 5-wk Training in Athletes. *Medicine & Science in Sports & Exercise,* 2003. 35(12): p. 2013-2022.

12. Blazevich A.J., Gill N.D., Deans N. and Zhou S., Lack of human muscle architectural adaptation after short-term strength training. *Muscle & Nerve,* 2007. 35(1): p. 78-86.

13. Borges L.S., Fernandes M.H., Schettino L., DA Silva C.R. and Pereira R., Handgrip explosive force is correlated with mobility in the elderly women. *Acta of bioengineering and biomechanics/Wroclaw University of Technology,* 2014. 17(3): p. 145-149.

14. Brown L.E. and Whitehurst M., The effect of short-term isokinetic training on force and rate of velocity development. *The Journal of Strength & Conditioning Research,* 2003. 17(1): p. 88-94.

15. Brown W.F., Strong M.J. and Snow R., Methods for estimating numbers of motor units in biceps-brachialis muscles and losses of motor units with aging. *Muscle & nerve,* 1988. 11(5): p. 423-432.

16. Campbell M., McComas A. and Petito F., Physiological changes in ageing muscles. *Journal of Neurology, Neurosurgery & Psychiatry,* 1973. 36(2): p. 174-182.

17. Carolan B. and Cafarelli E., Adaptations in coactivation after isometric resistance training. *Journal of Applied Physiology,* 1992. 73(p. 911-911.

18. Chan K.M., Andres L.P., Polykovskaya Y. and Brown W.F., The effects of training through high-frequency electrical stimulation on the physiological properties of single human thenar motor units. *Muscle & nerve,* 1999. 22(2): p. 186-195.

19. Christie A. and Kamen G., Short-term training adaptations in maximal motor unit firing rates and afterhyperpolarization duration. *Muscle & Nerve,* 2010. 41(5): p. 651-660.

20. Clark D.J., Patten C., Reid K.F., Carabello R.J., Phillips E.M. and Fielding R.A., Muscle performance and physical function are associated with voluntary rate of neuromuscular activation in older adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2011. 66(1): p. 115-121.

21. Clark D.J., Pojednic R.M., Reid K.F., Patten C., Pasha E.P., Phillips E.M. and Fielding R.A., Longitudinal decline of neuromuscular activation and power in healthy

older adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2013. 68(11): p. 1419-1425.

22. Connelly D.M. and Vandervoort A.A., Effects of isokinetic strength training on concentric and eccentric torque development in the ankle dorsiflexors of older adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2000. 55(10): p. B465-B472.

23. Dalton B.H., McNeil C.J., Doherty T.J. and Rice C.L., Age-related reductions in the estimated numbers of motor units are minimal in the human soleus. *Muscle & nerve,* 2008. 38(3): p. 1108-1115.

24. De Luca C.J., Adam A., Wotiz R., Gilmore L.D. and Nawab S.H., Decomposition of surface EMG signals. *Journal of neurophysiology,* 2006. 96(3): p. 1646-1657.

25. De Luca C.J. and Contessa P., Hierarchical control of motor units in voluntary contractions. *Journal of neurophysiology,* 2012. 107(1): p. 178-195.

26. DeFreitas J.M., Beck T.W., Stock M.S., Dillon M.A. and Kasishke II P.R., An examination of the time course of training-induced skeletal muscle hypertrophy. *European journal of applied physiology,* 2011. 111(11): p. 2785-2790.

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

28. Deschenes M.R., Roby M.A., Eason M.K. and Harris M.B., Remodeling of the neuromuscular junction precedes sarcopenia related alterations in myofibers. *Experimental gerontology,* 2010. 45(5): p. 389-393.

29. Desmedt J. and Godaux E., Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle. *The Journal of Physiology,* 1977. 264(3): p. 673-693.

30. Doherty T.J., Vandervoort A.A. and Brown W.F., Effects of Ageing on the Motor Unit: A Brief Review. *Canadian Journal of Applied Physiology,* 1993. 18(4): p. 331-358.

31. Doherty T.J., Vandervoort A.A., Taylor A.W. and Brown W.F., Effects of motor unit losses on strength in older men and women. *Journal of Applied Physiology,* 1993. 74(2): p. 868-874.

32. Drey M., Grösch C., Neuwirth C., Bauer J. and Sieber C., The motor unit number index (MUNIX) in sarcopenic patients. *Experimental gerontology,* 2013. 48(4): p. 381-384.

33. Drey M., Krieger B., Sieber C.C., Bauer J.M., Hettwer S., Bertsch T. and Group D.S., Motoneuron loss is associated with sarcopenia. *Journal of the American Medical Directors Association,* 2014. 15(6): p. 435-439.

34. Ehsani F., Nodehi-Moghadam A., Ghandali H. and Ahmadizade Z., The comparison of cross–education effect in young and elderly females from unilateral training of the elbow flexors. *Medical journal of the Islamic Republic of Iran,* 2014. 28(p. 138.

35. Evetovich T.K., Housh T.J., Housh D.J., Johnson G.O., Smith D.B. and Ebersole K.T., The effect of concentric isokinetic strength training of the quadriceps femoris on electromyography and muscle strength in the trained and untrained limb. *The Journal of Strength & Conditioning Research,* 2001. 15(4): p. 439-445.

36. Farina D., Negro F., Muceli S. and Enoka R.M., Principles of motor unit physiology evolve with advances in technology. *Physiology,* 2016. 31(2): p. 83-94.

37. Farthing J.P. and Chilibeck P.D., The effect of eccentric training at different velocities on cross-education. *European journal of applied physiology,* 2003. 89(6): p. 570-577.

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

39. Fowles J., Green H., Tupling R., O'brien S. and Roy B., Human neuromuscular fatigue is associated with altered Na+-K+-ATPase activity following isometric exercise. *Journal of Applied Physiology,* 2002. 92(4): p. 1585-1593.

40. Fuglevand A.J., Winter D.A., Patla A.E. and Stashuk D., Detection of motor unit action potentials with surface electrodes: influence of electrode size and spacing. *Biological cybernetics,* 1992. 67(2): p. 143-153.

41. Glenn J.M., Gray M. and Binns A., The effects of loaded and unloaded high-velocity resistance training on functional fitness among community-dwelling older adults. *Age Ageing,* 2015. p.

42. Goldberg L.J. and Derfler B., Relationship among recruitment order, spike amplitude, and twitch tension of single motor units in human masseter muscle. *Journal of neurophysiology,* 1977. 40(4): p. 879-890.

43. Hakansson C., Conduction velocity and amplitude of the action potential as related to circumference in the isolated fibre of frog muscle. *Acta Physiologica Scandinavica,* 1956. 37(1): p. 14-34.

44. Häkkinen K., Kallinen M., Izquierdo M., Jokelainen K., Lassila H., Mälkiä E., Kraemer W., Newton R. and Alen M., Changes in agonist-antagonist EMG, muscle

CSA, and force during strength training in middle-aged and older people. *Journal of Applied Physiology,* 1998. 84(4): p. 1341-1349.

45. Heckman C. and Enoka R.M., Motor unit. *Comprehensive Physiology,* 2012. p.

46. Hendy A.M., Spittle M. and Kidgell D.J., Cross education and immobilisation: mechanisms and implications for injury rehabilitation. *Journal of science and medicine in sport,* 2012. 15(2): p. 94-101.

47. Hermens H.J., Freriks B., Merletti R., Stegeman D., Blok J., Rau G., Disselhorst-Klug C. and Hägg G., European recommendations for surface electromyography. *Roessingh Research and Development, Enschede,* 1999. p.

48. Hicks A. and McComas A., Increased sodium pump activity following repetitive stimulation of rat soleus muscles. *The Journal of Physiology,* 1989. 414(1): p. 337-349.

49. Hortobagyi T., Lambert N.J. and Hill J.P., Greater cross education following training with muscle lengthening than shortening. *Medicine & Science in Sports & Exercise,* 1997. 29(1): p. 107-112.

50. Hu X., Rymer W.Z. and Suresh N.L., Motor unit pool organization examined via spike-triggered averaging of the surface electromyogram. *Journal of neurophysiology,* 2013. 110(5): p. 1205-1220.

51. Hu X., Rymer W.Z. and Suresh N.L., Accuracy assessment of a surface electromyogram decomposition system in human first dorsal interosseus muscle. *Journal of neural engineering,* 2014. 11(2): p. 026007.

52. Izquierdo M., Aguado X., Gonzalez R., Lopez J. and Häkkinen K., Maximal and explosive force production capacity and balance performance in men of different ages. *European journal of applied physiology and occupational physiology,* 1999. 79(3): p. 260-267.

53. Izquierdo M., Gorostiaga E., Garrues M., Anton A., Larrion J. and Haekkinen K., Maximal strength and power characteristics in isometric and dynamic actions of the upper and lower extremities in middle-aged and older men. *Acta Physiologica Scandinavica,* 1999. 167(p. 57-68.

54. Izquierdo M., Häkkinen K., Gonzalez-Badillo J.J., Ibanez J. and Gorostiaga E.M., Effects of long-term training specificity on maximal strength and power of the upper and lower extremities in athletes from different sports. *Eur J Appl Physiol,* 2002. 87(3): p. 264-271.

55. Jenkins N.D., Buckner S.L., Cochrane K.C., Bergstrom H.C., Palmer T.B., Johnson G.O., Schmidt R.J., Housh T.J. and Cramer J.T., Age-related differences in rates of

torque development and rise in EMG are eliminated by normalization. *Experimental gerontology,* 2014. 57(p. 18-28.

56. Kamen G. and Knight C.A., Training-related adaptations in motor unit discharge rate in young and older adults. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2004. 59(12): p. 1334-1338.

57. Kamen G., Sison S.V., Du C. and Patten C., Motor unit discharge behavior in older adults during maximal-effort contractions. *Journal of Applied Physiology,* 1995. 79(6): p. 1908-1913.

58. Kawamura Y., O'Brien P., Okazaki H. and Dyck P.J., Lumbar motoneurons of man II: the number and diameter distribution of large-and intermediate-diameter cytons in "motoneuron columns" of spinal cord of man. *Journal of Neuropathology & Experimental Neurology,* 1977. 36(5): p. 861-870.

59. Kaya R.D., Nakazawa M., Hoffman R.L. and Clark B.C., Interrelationship between muscle strength, motor units, and aging. *Experimental gerontology,* 2013. 48(9): p. 920-925.

60. Keppel G. and Wickens T., Design and Analysis, A Researcher's Handbook. 2004. p.

61. Klass M., Baudry S. and Duchateau J., Age-related decline in rate of torque development is accompanied by lower maximal motor unit discharge frequency during fast contractions. *Journal of Applied Physiology,* 2008. 104(3): p. 739-746.

62. Laroche D.P., Roy S.J., Knight C.A. and Dickie J.L., Elderly women have blunted response to resistance training despite reduced antagonist coactivation. *Medicine and science in sports and exercise,* 2008. 40(9): p. 1660-1668.

63. Larsson L., Grimby G. and Karlsson J., Muscle strength and speed of movement in relation to age and muscle morphology. *Journal of Applied Physiology,* 1979. 46(3): p. 451-456.

64. Lee M. and Carroll T.J., Cross Education. *Sports Medicine,* 2007. 37(1): p. 1-14.

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

66. Lee M., Hinder M.R., Gandevia S.C. and Carroll T.J., The ipsilateral motor cortex contributes to cross-limb transfer of performance gains after ballistic motor practice. *The Journal of physiology,* 2010. 588(1): p. 201-212.

67. Lexell J., Taylor C.C. and Sjöström M., What is the cause of the ageing atrophy?: Total number, size and proportion of different fiber types studied in whole vastus

lateralis muscle from 15-to 83-year-old men. *Journal of the neurological sciences,* 1988. 84(2): p. 275-294.

68. Liddell E.G.T. and Sherrington C.S., Recruitment and some other Features of Reflex Inhibition. *Proceedings of the Royal Society of London. Series B, Containing Papers of a Biological Character,* 1925. 97(686): p. 488-518.

69. Ling S.M., Conwit R.A., Ferrucci L. and Metter E.J., Age-associated changes in motor unit physiology: observations from the Baltimore Longitudinal Study of Aging. *Archives of physical medicine and rehabilitation,* 2009. 90(7): p. 1237-1240.

70. Lixandrao M.E., Damas F., Traina Chacon-Mikahil M.P., Cavaglieri C.R., Ugrinowitsch C., Bottaro M., Vechin F.C., Conceicao M.S., Paes de Barros Berton R. and Libardi C.A., Time-course of resistance training-induced muscle hypertrophy in elderly. *J Strength Cond Res,* 2015. p.

71. Macaluso A., Nimmo M.A., Foster J.E., Cockburn M., McMillan N.C. and De Vito G., Contractile muscle volume and agonist-antagonist coactivation account for differences in torque between young and older women. *Muscle & Nerve,* 2002. 25(6): p. 858-863.

72. McKinnon N.B., Montero-Odasso M. and Doherty T.J., Motor unit loss is accompanied by decreased peak muscle power in the lower limb of older adults. *Exp Gerontol,* 2015. p.

73. McNeil C.J., Doherty T.J., Stashuk D.W. and Rice C.L., Motor unit number estimates in the tibialis anterior muscle of young, old, and very old men. *Muscle & nerve,* 2005. 31(4): p. 461-467.

74. Metter E.J., Talbot L.A., Schrager M. and Conwit R., Skeletal muscle strength as a predictor of all-cause mortality in healthy men. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2002. 57(10): p. B359-B365.

75. Milner-Brown H. and Stein R., The relation between the surface electromyogram and muscular force. *The Journal of physiology,* 1975. 246(3): p. 549.

76. Milner-Brown H., Stein R. and Yemm R., The orderly recruitment of human motor units during voluntary isometric contractions. *The Journal of physiology,* 1973. 230(2): p. 359.

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

78. Morse C.I., Thom J.M., Davis M.G., Fox K.R., Birch K.M. and Narici M.V., Reduced plantarflexor specific torque in the elderly is associated with a lower activation capacity. *European Journal of Applied Physiology,* 2004. 92(1-2): p. 219-226.

79. Murray D.P., Brown L.E., Zinder S.M., Noffal G.J., Bera S.G. and Garrett N.M., Effects of velocity-specific training on rate of velocity development, peak torque, and performance. *The Journal of Strength & Conditioning Research,* 2007. 21(3): p. 870-874.

80. Narici M., Roi G., Landoni L., Minetti A. and Cerretelli P., Changes in force, cross-sectional area and neural activation during strength training and detraining of the human quadriceps. *European Journal of Applied Physiology and Occupational Physiology,* 1989. 59(4): p. 310-319.

81. Nawab S.H., Chang S.-S. and De Luca C.J., High-yield decomposition of surface EMG signals. *Clinical Neurophysiology,* 2010. 121(10): p. 1602-1615.

82. Newman A.B., Kupelian V., Visser M., Simonsick E.M., Goodpaster B.H., Kritchevsky S.B., Tylavsky F.A., Rubin S.M. and Harris T.B., Strength, but not muscle mass, is associated with mortality in the health, aging and body composition study cohort. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2006. 61(1): p. 72-77.

83. Newton R.U., Hakkinen K., Hakkinen A., McCormick M., Volek J. and Kraemer W.J., Mixed-methods resistance training increases power and strength of young and older men. *Medicine and science in sports and exercise,* 2002. 34(8): p. 1367-1375.

84. Nilwik R., Snijders T., Leenders M., Groen B.B., van Kranenburg J., Verdijk L.B. and van Loon L.J., The decline in skeletal muscle mass with aging is mainly attributed to a reduction in type II muscle fiber size. *Experimental gerontology,* 2013. 48(5): p. 492-498.

85. Patten C. and Kamen G., Adaptations in motor unit discharge activity with force control training in young and older human adults. *European journal of applied physiology,* 2000. 83(2-3): p. 128-143.

86. Patten C., Kamen G. and Rowland D.M., Adaptations in maximal motor unit discharge rate to strength training in young and older adults. *Muscle & Nerve,* 2001. 24(4): p. 542-550.

87. Piasecki M., Ireland A., Stashuk D., Hamilton-Wright A., Jones D. and McPhee J., Age-related neuromuscular changes affecting human vastus lateralis. *The Journal of physiology,* 2015. p.

88. Pinto R.S., Correa C.S., Radaelli R., Cadore E.L., Brown L.E. and Bottaro M., Short-term strength training improves muscle quality and functional capacity of elderly women. *AGE,* 2014. 36(1): p. 365-372.

89. Pope Z.K., Hester G.M., Benik F.M. and DeFreitas J.M., Action potential amplitude as a non-invasive indicator of motor unit specific hypertorphy. *Journal of neurophysiology,* 2016. p. jn. 00039.2016.

90. Prevost M.C., Nelson A.G. and Maraj B.K., The Effect of Two Days of Velocity-Specific Isokinetic Training on Torque Production. *The Journal of Strength & Conditioning Research,* 1999. 13(1): p. 35-39.

91. Pucci A., Griffin L. and Cafarelli E., Maximal motor unit firing rates during isometric resistance training in men. *Experimental physiology,* 2006. 91(1): p. 171-178.

92. Rich C. and Cafarelli E., Submaximal motor unit firing rates after 8 wk of isometric resistance training. *Medicine & Science in Sports & Exercise,* 2000. 32(1): p. 190.

93. Ruddy K.L. and Carson R.G., Neural pathways mediating cross education of motor function. 2013. p.

94. Sayer A.A., Syddall H.E., Martin H.J., Dennison E.M., Roberts H.C. and Cooper C., Is grip strength associated with health-related quality of life? Findings from the Hertfordshire Cohort Study. *Age and ageing,* 2006. 35(4): p. 409-415.

95. Scanlon T.C., Fragala M.S., Stout J.R., Emerson N.S., Beyer K.S., Oliveira L.P. and Hoffman J.R., Muscle architecture and strength: Adaptations to short-term resistance training in older adults. *Muscle & nerve,* 2014. 49(4): p. 584-592.

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

97. Simoneau E., Martin A. and Van Hoecke J., Muscular performances at the ankle joint in young and elderly men. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2005. 60(4): p. 439-447.

98. Skelton D.A., Greig C.A., Davies J.M. and Young A., Strength, power and related functional ability of healthy people aged 65–89 years. *Age and ageing,* 1994. 23(5): p. 371-377.

99. Stephens J. and Usherwood T., The mechanical properties of human motor units with special reference to their fatiguability and recruitment threshold. *Brain research,* 1977. 125(1): p. 91-97.

100. Stock M.S. and Thompson B.J., Effects of Barbell Deadlift Training on Submaximal Motor Unit Firing Rates for the Vastus Lateralis and Rectus Femoris. *PloS one,* 2014. 9(12): p. e115567.

101. Thompson B.J., Conchola E.C., Palmer T.B. and Stock M.S., Effects of aging on maximal and rapid velocity capacities of the leg extensors. *Experimental gerontology,* 2014. 58(p. 128-131.

102. Thompson B.J., Ryan E.D., Herda T.J., Costa P.B., Herda A.A. and Cramer J.T., Age-related changes in the rate of muscle activation and rapid force characteristics. *Age,* 2014. 36(2): p. 839-849.

103. Thompson B.J., Ryan E.D., Sobolewski E.J., Conchola E.C. and Cramer J.T., Age related differences in maximal and rapid torque characteristics of the leg extensors and flexors in young, middle-aged and old men. *Experimental gerontology,* 2013. 48(2): p. 277-282.

104. Thorstensson A., Grimby G. and Karlsson J., Force-velocity relations and fiber composition in human knee extensor muscles. *Journal of applied physiology,* 1976. 40(1): p. 12-16.

105. Tillin N.A. and Folland J.P., Maximal and explosive strength training elicit distinct neuromuscular adaptations, specific to the training stimulus. *European journal of applied physiology,* 2014. 114(2): p. 365-374.

106. Tillin N.A., Pain M.T. and Folland J.P., Short-term unilateral resistance training affects the agonist–antagonist but not the force–agonist activation relationship. *Muscle & Nerve,* 2011. 43(3): p. 375-384.

107. Tomlinson B. and Irving D., The numbers of limb motor neurons in the human lumbosacral cord throughout life. *Journal of the neurological sciences,* 1977. 34(2): p. 213-219.

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

109. Visser M., Goodpaster B.H., Kritchevsky S.B., Newman A.B., Nevitt M., Rubin S.M., Simonsick E.M. and Harris T.B., Muscle mass, muscle strength, and muscle fat infiltration as predictors of incident mobility limitations in well-functioning older persons. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences,* 2005. 60(3): p. 324-333.

110. Wu R., Delahunt E., Ditroilo M., Lowery M. and De Vito G., Effects of age and sex on neuromuscular-mechanical determinants of muscle strength. *Age (Dordr),* 2016. 38(3): p. 57.

APPENDICES

Appendix A – IRB Approval Letter

Oklahoma State University Institutional Review Board

Date:	Monday, October 06, 2014
IRB Application No	ED14129
Proposal Title:	Neural and Muscular Adaptations During 4-weeks of Resistance Training
Reviewed and Processed as:	Expedited

Status Recommended by Reviewer(s): Approved Protocol Expires: 10/5/2015

Principal Investigator(s):

Garrett Hester
001B SWC
Stillwater, OK 74078

Frank Benik
180 CRC
Stillwater, OK 74078

Zachary Pope
192 CRC
Stillwater, OK 74078

Jason DeFreitas
198 CRC
Stillwater, OK 74078

The IRB application referenced above has been approved. It is the judgment of the reviewers that the rights and welfare of individuals who may be asked to participate in this study will be respected, and that the research will be conducted in a manner consistent with the IRB requirements as outlined in section 45 CFR 46.

☒ The final versions of any printed recruitment, consent and assent documents bearing the IRB approval stamp are attached to this letter. These are the versions that must be used during the study.

As Principal Investigator, it is your responsibility to do the following:

1.Conduct this study exactly as it has been approved. Any modifications to the research protocol must be submitted with the appropriate signatures for IRB approval. Protocol modifications requiring approval may include changes to the title, PI advisor, funding status or sponsor, subject population composition or size, recruitment, inclusion/exclusion criteria, research site, research procedures and consent/assent process or forms
2.Submit a request for continuation if the study extends beyond the approval period. This continuation must receive IRB review and approval before the research can continue.
3.Report any adverse events to the IRB Chair promptly. Adverse events are those which are unanticipated and impact the subjects during the course of the research; and
4.Notify the IRB office in writing when your research project is complete.

Please note that approved protocols are subject to monitoring by the IRB and that the IRB office has the authority to inspect research records associated with this protocol at any time. If you have questions about the IRB procedures or need any assistance from the Board, please contact Dawnett Watkins 219 Cordell North (phone: 405-744-5700, dawnett.watkins@okstate.edu).

Sincerely,

Hugh Crethar, Chair
Institutional Review Board

VITA

Garrett Hester

Candidate for the Degree of

Doctor of Philosophy

Dissertation: EFFECTS OF SHORT-TERM RESISTANCE TRAINING ON MOTOR UNIT PROPERTIES AND NEUROMUSCULAR FUNCTION IN YOUNG AND OLD MEN

Major Field: Health, Leisure and Human Performance

Biographical:

Education:

Completed the requirements for the Doctor of Philosophy in Health, Leisure and Human Performance at Oklahoma State University, Stillwater, Oklahoma in July, 2016.

Completed the requirements for the Master of Science in Health and Human Performance at Oklahoma State University, Stillwater, Oklahoma in 2012.

Completed the requirements for the Bachelor of Science in Health and Human Performance at Northeastern State University, Tahlequah, Oklahoma in 2010.

Experience:

Graduate Teaching Assistant; Aug '13 – Present
Department of Health and Human Performance
Oklahoma State University

Professional Memberships:

American College of Sports Medicine; 2009 – Present
National Strength and Conditioning Association; 2011 – Present
Central States Region, American College of Sports Medicine; 2012 – Present