

## **Abstract**

Influence of training history and contraction velocity on hamstring muscle coactivation during  
maximal effort knee extension

By

Robert B. Brady, CSCS

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Director of Thesis: Tibor Hortobágyi

Major Department: Kinesiology

When a muscle produces voluntary force, muscles on the opposite side of the joint, the antagonists, are also activated. While coactivation of the knee flexors during knee extension is presumed to increase joint stability by decreasing anterior shear force of the tibia on the femur, the coactivation of the hamstrings also produces what is called the antagonist torque.

Systematic exercise in the form of resistance training can reduce antagonist muscle coactivation in healthy young adults. However, the mechanical consequence of this neurological adaptation is unclear. We thus hypothesized that previously strength-trained individuals would exhibit less antagonist coactivation, resulting in a reduced antagonist torque, and that with an increase in contraction speed there would be an increase in antagonist coactivation to slow the movement but there would be less of an increase due to speed in the trained compared with untrained individuals. Therefore, the purpose of this study was to determine the effects of training status on coactivation, i.e., antagonist torque, and on the speed-sensitivity of coactivation. Subjects for this study were fitted with surface EMG electrodes on their thigh muscles, and performed maximal effort knee extensions on a dynamometer, using shortening (concentric) and

lengthening (eccentric) contractions at 30, 90, and 150°/s. As expected, trained individuals produced ~44% less coactivation at all contractions speeds. Against the hypothesis, coactivation did not increase in either group as velocity increased, as there was less than 10% difference in coactivation levels between the 3 speeds. Also against the hypothesis, as determined with an EMG-driven mathematical model, antagonist torque did not decrease with decreasing coactivation; in fact we see a trend towards the opposite for trained individuals. A borderline greater antagonist torque was noted in the trained compared to the untrained subjects even with decreased coactivation of the trained. These data suggest that antagonist muscle coactivation is less in trained healthy young adults but this reduced neural activation does not manifest itself in lower levels of antagonist torque. Therefore, leg strength training may increase muscle strength in part by reducing antagonist muscle coactivation without compromising joint stability.



Influence of training history and contraction velocity on  
hamstring muscle coactivation during maximal effort knee  
extension

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By

Robert B. Brady, CSCS

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Influence of training history and contraction velocity on hamstring muscle coactivation during  
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By

Robert Brady, CSCS

Approved By:

Director of Thesis:

---

Tibor Hortobágyi, Ph.D.

Committee Member:

---

Paul DeVita, Ph.D.

Committee Member:

---

Kristina Karvinen, Ph.D.

Committee Member:

---

Patrick Rider, MS

Chair of the Department of Exercise and Sport Science:

---

Stacey R. Altman, J.D.

Dean of the College of Health and Human Performance:

---

Glen G. Gilbert, Ph.D.

Dean of the Graduate School:

---

Paul J. Gemperline, Ph.D.

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## Chapter I. Introduction

Maximal force production is a cornerstone for human performance; however how the central nervous system controls and regulates such movements is not fully understood. Modeling studies suggest that during resistance training exercises such as the back squat, leg press, and knee extension at 70% of individuals 1 repetition maximum can produce greater than six times body weight of compressive force in the knee for the squat and leg press, and greater than five times body weight of force for knee extension (Wilk et al 1996). Shear forces of the knee can be approximately two times body weight for the squat and leg press and slightly greater than body weight for knee extension (Wilk et al 1996). Such high force productions and loads must be the result of muscle hypertrophy even then the nervous system would have to activate the increased muscle volume, implying a large role for neural adaptations contributing to strength gains. In addition, large forces would require the nervous system to create an activation strategy that prevents injury of the involved joints and muscles.

One neural adaptation associated with resistance training is a change in the individual activation of the antagonist muscle of a said task (Carolan et al 1992). The common measure of this antagonist muscle coactivation is electromyography (EMG). EMG allows us to see the neural activation patterns of muscles; however, coactivation is typically associated with the idea of increased joint stiffness, and joint stability (Devita & Hortobagyi 2000; Kubo et al 2004). Stiffness and stability are functional outcomes of coactivation as a result of forces within a joint, whereas EMG recordings are neurological in nature and are evoked from the central nervous system. The idea of this functional outcome of coactivation during maximal effort knee extension has been poorly researched and is a novel idea in the biomechanical research

community. Along with the novelty of a functional outcome of coactivation is the consequence resistance training has on these functional and neurological outcomes.

Many studies have shown that highly skilled individuals exhibit lower levels of coactivation than their sedentary counterparts, based on EMG, but the idea of resistance training is less developed. The rationale for exploring the functional and neurological outcomes of coactivation is to further understand coactivation's impact on resistance training. It has been shown that untrained individuals exhibit large inhibition during maximal effort contractions of the agonist, synergistic, and antagonist muscles about a joint (Kidgell et al 2010). It is believed that trained individuals do not exhibit this inhibition as well as have the ability to focus the activation of muscles about a joint during maximal effort contraction that allows maximal torque production along with sufficient joint stability possibly through coactivation.

Finally the duality of coactivation is of great interest. One neurological mechanism that would result in a greater net torque about a joint is decreased coactivation (Bryant et al 2010). A lower level of counteracting torque, produced by the contraction of the antagonist muscles concurrently activated with the agonists, would result in a higher net torque. However, this resulting increase in torque due to decreased coactivation coupled with increased muscle strength through hypertrophy would warrant greater joint stiffness, i.e., stability to protect the joint from increased shear force with increased torque production. Therefore determining the functional outcomes of coactivation such as calculating the agonist torque production along with the antagonist torque production would be beneficial in understanding the role of coactivation on both a neurological and mechanical level.

### **Hypothesis**

The hypothesis of this study is that resistance-trained individuals will exhibit less coactivation during maximal knee extension with quadriceps as the agonist and hamstrings as the antagonist muscles. Sub hypotheses will be that as speed increased, coactivation will increase but less in trained individuals and finally, counteracting antagonist torque will be less when antagonist muscle activation is less.

### **Purpose**

The purpose of this study is to determine if: a) resistance trained individuals produce lower levels of EMG activity in the antagonist muscles during maximal effort knee extension, b) this lower EMG level produces less counteracting torque, and if, c) training history and contraction velocity interact.

### **Delimitations**

1. All Subjects will perform lower body resistance training at least once a week along with some other form of lower body training at least 1 additional day a week
2. All subjects will be adults age 18-25
3. All subjects will be healthy and free of any orthopedic conditions, or surgical procedures of the lower extremities, which may affect outcome.
4. All subjects will be right leg dominant based on a ball kick test

### **Limitations**

1. True maximal effort of individuals is uncertain
2. EMG may be reduced due to body composition

3. Honesty of participants in training history

### **Assumptions**

1. EMG and Dynamometry will be accurate, reliable, and valid
2. All subjects will give maximal effort during each contraction and contraction mode
3. Fatigue will not be a factor due to adequate rest periods

### **Definitions**

1. Agonist Torque: The total torque produced as a result of net torque + antagonist torque
2. Antagonist Torque: The torque produced by the antagonist muscles, (hamstrings), this causes a torque in the opposite direction as the primary mover resulting in a decrease net torque as compared to agonist torque.
3. Coactivation: Denotes the concurrent activation of the hamstring muscles with the activation of the quadriceps muscle, measured with surface electromyography (EMG), during isokinetic knee extension at 30, 90, and 150 °/s.
4. Net Torque: The torque product of agonist torque minus the antagonist torque
5. Torque: Rotational force about an axis. In the present study force produced by the quadriceps during knee extension
6. Trained: Individuals who participate in 2 days or more a week in lower body training with at least 1 day being resistance training
7. Untrained: Individuals who perform no regular exercise outside of normal activities of daily living.



## **Chapter II. Literature Review**

### **The role of coactivation**

Coactivation is the concurrent activation of the agonist and antagonist muscles during a movement that creates torque about a joint. The reason for this coactivation is not entirely understood but many studies suggest one reason is joint stability (Kubo et al 2004, Snow et al 1993, Psek and Carafelli 1992, Osternig et al 1986, Hagood et al 1990). Coactivation is present in the lower extremities during locomotion, and increases with speed (Martin and Peterson 2010), and measurable during isokinetic movements such as knee extension (Kubo et al 2004, Aagaard et al 2000). Coactivation is thought to increase stability but also decreases total force production from the agonist muscle. If the quadriceps are contracting to extend the knee, the antagonist hamstrings are also activated. This coactivation counteracts and reduces the effort of the quadriceps.

The amount of coactivation varies with joint angle (Kubo et al 2004, Remaud et al 2007, Snow et al 1993). The actual angle that induces greatest coactivation is not consistent. However, these three studies all showed that the hamstring became less active towards anatomical 0 at the knee except for the final degrees where the hamstrings again contract in efforts to slow the rotation of the knee joint, as it approaches full extension. Kubo et al suggest that the reduced activation of the antagonist muscle towards knee extension is for protection of the knee whereas increased coactivation is also used for stability when the knee is flexed. The length tension relationship accounts for increased torques at specific joint angles due to myosin actin overlap as explained by the sliding filament theory, and the changing moment arm of the limb. Coactivation is greatest with increased torques and as the knee approaches full

extension the quadriceps EMG is reduced and the hamstring EMG is increased. (Kubo et al 2004).

The Velocity of Contraction also has an effect on coactivation levels. Faster contractions result in greater coactivation throughout the motion (Hagood et al 1990, Osternig et al 1986). This may be due to a reflex action that contributes to joint stability as the greater the velocity about the joint, the greater forces and an increased need for stability through coactivation. It is also warranted to note that during faster contractions the amount of coactivation during the initial movement of the leg is reduced, and as the knee nears full extension coactivation is increased. This is believed to allow initial acceleration of the limb and then deceleration of the limb towards extension to slow the shank protecting the knee from injury (Hagood et al 1990). This is evident even when the speed is increased from 30 to 90 degrees showing greater coactivation at the end of extension at 90 degrees compared to 30 degrees (Snow et al 1993). These results are amplified when the velocity ranges from 15 up to 240 degrees per second as the overall coactivation increased 128% from the slowest to the fastest speed during concentric knee extension and 113% during eccentric knee extension (Hagood et al 1990).

Coactivation is present during locomotion, and stationary (isometric) efforts such as dynamometry, etc. The amounts of coactivation are influenced by many factors such as speed of contraction, the joint angle within contraction, and mode of contraction. Coactivation appears to be greater as the agonist muscle is near maximal force production, and also increases as speed increases (Kubo et al 2004, Osternig et al 1986).

## **Training Status Influence on Coactivation**

The reduction of hamstring coactivation would lead to greater net force production by the quadriceps during maximal effort knee extension. This is important for the general population but applies even more so for individuals in training, more specifically athletes. Greater force production by the quadriceps through reduced coactivation may lead to heavier weights lifted, faster turnover of the legs when running, and also greater height during jumping movements to list a few. If training reduces the amount of coactivation of antagonist muscles then the agonist muscles not only increase force through hypertrophy and greater neural adaptations but also by not having to overcome as much resistance from the antagonist muscles activation.

There are two modes of training, aerobic and anaerobic with aerobic including cyclist, distance runners, skiers etc, and anaerobic including resistance training, power training, and most competitive team sports are anaerobic in nature such as football, and basketball. How does a training mode affect coactivation level? Anaerobically trained athletes, such as high jumpers, showed significantly less coactivation during knee extension as compared to a sedentary group of similar stature and age (Amiridis et al 1996). This reduction of coactivation may be due in part to increased efficiency of the neurologic pathways of trained individuals allowing for increased force production of the quadriceps but also allowing for joint stability in part from coactivation of the hamstrings (Amiridis et al 1996).

Carolan et al 1992, showed that even after one week of resistance training sedentary individuals may reduce the amount of coactivation during maximal knee extension. This is contributed to the increased efficiency of the neurologic pathways allowing greater force

production by the quadriceps and reduced countering torque by the hamstrings. The speed of these contractions also tends to affect athletes differently dependent on sport. Increased speed leads to increased coactivation, however, these results are less evident in athletes such as sprinters compared to endurance athletes meaning sprinters have less of an increase in coactivation as speed increases compared to endurance athletes who have a greater increase of coactivation as speed increases (Osternig et al 1986). Anaerobic athletes show reductions of coactivation compared to sedentary individuals, but still follow the same patterns of coactivation as sedentary individuals just to a lesser degree.

Aerobic athletes such as distance runners, and cyclist also show reductions of coactivation when compared to their sedentary counterparts (Garrandes et al 2007). Aerobically trained athletes also show greater amounts of coactivation initially than power/anaerobically trained athletes (Garrandes et al 2007). Aerobic athletes exercise in great volume but generally at a lower intensity as high intensity exercise cannot be supported for long periods of time due to different metabolic pathways. The increased volume of training allows aerobic athletes to not only show decreases in coactivation due to neural adaptations but also to resist fatigue and those effects on coactivation as it is shown that as people fatigue the amount of coactivation is increased (Psek and Carafelli 1992).

Short term resistance training reduces coactivation in the lower extremities. This reduction of coactivation allows anaerobic athletes to produce greater net force. Although never directly examined, it is possible that decreased coactivation observed in a single joint task (knee extension) would be also present in the target tasks such as high jumping (Amiridis et al 1996), running, and weight lifting (Carolan et al 1992). Coactivation though still present is

reduced with training among other joints such as the wrist (Shimose et al 2011). This indicates some form of neural adaptation possibly leading to increased force as well efficiency in stabilizing the joint saving metabolic costs (Shimose et al 2011).

### **Effects of fatigue on coactivation**

Fatigue of muscles may cause a difference in the amount of coactivation at the knee joint. As muscle fatigue they lose the ability to produce or maintain the maximal voluntary force capable of being produced by the agonist muscle (Garrandes et al 2007). The difference in coactivation may be one component of decreased torque about the joint if fatigue causes increased coactivation as found by Colson et al 2007. Whether it is walking up a flight of stairs, or during a marathon, our bodies experience fatigue to some degree almost daily. Fatigue causes an increase in coactivation during knee extension in both trained and untrained individuals (Garrandes et al 2007, Psek and Carafelli 1992). There also is a difference in the fatiguing affect of coactivation between endurance trained and power-trained people. As expected endurance trained athletes tend to resist fatigue therefore resist-increased coactivation due to fatigue (Garrandes et al 2007).

Fatigue will not be measured in the current study, using previous literature on studies that include fatiguing conditions, and those that do not, (such as the present study), may provide insight into the central nervous systems control of force through coactivation. Fatigue causes a greater amount of coactivation during knee extension meaning not only is participants fighting against the fatigue of the muscles, but also fighting the increased activation of the antagonist muscle (Garrandes et al 2007). As participants are training, or going through a fatiguing protocol the increase of coactivation as much as 59% increase in bicep femoris

activation during knee extension (Psek and Carafelli 1992). The increase in EMG of both the agonist and antagonist muscles as the fatiguing exercises are performed may be due to increased demands on the agonist to work harder while maintaining a sub maximal effort in turn requiring greater coactivation to match these increases in agonist EMG for joint stability along with protection to prevent damage that may be caused by uneven pressure along the articular surfaces of the joint (Psek and Carafelli 1992).

Fatigue causes reduction of torque for both trained and untrained individuals. Also endurance trained and power trained individuals show decreases in maximal torque when the muscles are fatigued (Garrandes et al 2007). Power trained athletes however fatigue at a faster rate showing greater levels of EMG at sub maximal efforts faster when compared to their endurance trained counterparts. Power athletes however tend to show less coactivation during maximal contractions when fresh compared to cyclist and tri-athletes (Garrandes et al 2007). These differences in coactivation pre and post fatiguing exercise protocols are most likely due to the differing neurological adaptations to training history.

Endurance trained athletes resist fatigue longer, but when fatigue does become a factor coactivation is increased just as in power athletes, and sedentary individuals. The effects of training have an effect on coactivation, which means that fatigue must be accounted for when planning a protocol involving coactivation depending whether the experiment wished to have fatigue as a factor or to not have fatigue as a factor. Increased levels of coactivation again due to fatigue increase joint stability and regulate pressures placed on the articular surfaces of the joint (Garrandes et al 2007)

### **Contraction mode effects on coactivation**

The central nervous system incorporates different neural activation strategies for different contraction modes ie, concentric, eccentric and isometric. Concentric Knee extension contractions induce greater EMG based coactivation ratios (antagonist/agonist) such as (24%) compared to eccentric contractions (8%) at the initial 10-20 degree range of maximal effort knee extension (Aagaard et al 2000)

### **Mechanical effect of increased antagonist coactivation**

Coactivation inherently causes a decrease in the net force produced by the agonist muscle. As the knee extends, the coactivation of the hamstrings resist that extension. Trained individuals are able to produce greater torques about the knee via increased neural control (Amiridis et al 1996), a combination of muscle unit recruitment, rate coding, and also synchronous firing of motor units. Hypertrophy is also a cause of increased torque and requires a greater amount of time to develop compared to neural adaptation (Coyle et al 1981). One neural adaptation that takes place in as little as one week is a decrease in coactivation (Carolan et al 1992).

Being able to calculate the theoretical force being produced by the antagonist muscle coactivation gives us a better understanding of the actual amount of force our muscles may produce. Muscles create greater torques during eccentric movements as compared to concentric contractions. Eccentric knee extension accounts for greater ratios of quadriceps to hamstring strength meaning the hamstrings contracting eccentrically may be able to contribute significantly greater amounts of stabilization of the knee joint during both fast and slow concentric knee extensions (Aagaard et al 2000). The equations were derived from the

maximal eccentric contraction of the hamstrings relationship of Torque and EMG activity as it relates to the EMG of the hamstrings during concentric knee extension resulting in an antagonist torque being produced. Using these equations we are able to quantify the force produced by the antagonist muscle during knee extension leading to a differentiation between the net and gross torques produced by the agonist muscle (Aagaard et al 2000).

Coactivation reduces the anterior displacement of the tibia away from the femur as the hamstrings actively pull against the proximal end of the tibia when they activate during knee extension. As the knee approaches full knee extension (10-30 degrees), the moment causes an increase in this shearing effect leading to increased coactivation of the hamstrings at this time. This increased coactivation may be viewed as a mechanism to slow the knee (Osternig et al 1986), or increase the stability of the joint at that moment (Aagaard et al 2000). These resulting increases in coactivation lead to a decrease in net torque at these knee angles. The torques may be quantified by calculating the antagonist torque and its magnitude of force opposing the agonist quadriceps giving us a better estimate of actual gross force being produced by the agonist muscle.



## Chapter III. Methods

### **Subject Characteristics**

Two groups of volunteers were included in the experimental protocol. One group consisted of consist of 11 individuals that have a history of resistance training the lower extremities a minimum of 1 day a week for at least 6 months prior to the start of this study. The other group contained 12 individuals that were completely sedentary with no regular exercise regimen of any design.

Table 1: Subject Characteristics:

Subject Characteristics	Males	Females	Age	Height (cm)	Weight (kg)
Trained	6	4	22.8 (1.8)	178 (8.4)	79 (13.1)
Untrained	6	5	21.2 (2.5)	171 (11.8)	66 (12.3)

#### **Inclusion Criteria:**

- 1: Healthy young adults between the ages of 18-25 with no musculoskeletal injuries of the lower extremities requiring surgical repair such as ACL injury etc.
- 2: No pain associated with the limbs that may alter activities of daily living
- 3: Capable of performing the protocol without pain or exemplary difficulty
- 4: Written informed consent signed
- 5: Right Leg Dominant

#### **Exclusion Criteria:**

- 1: Orthopedic surgery of the lower extremities
- 2: Resistance training of lower body not part of regular exercise regimen for trained group
- 3: Age less than 18 or greater than 25

## **Study Design**

Comparison based on EMG recordings of the agonist and antagonist muscle activation of the knee extensors and knee flexors during maximal effort concentric knee extension. The data is used to determine the level of coactivation of the dominant limb between the two groups.

## **Equipment**

EMG used to create EMG-torque ratios, along with coactivation ratios (Myopac MPRD-101 receiver and belt pack EMG system, Konigsberg Instruments, Inc. Pasadena, CA). Torque data were collected using the Humac Norm, isokinetic dynamometer (Computer Sports Medicine, Inc, Ma). Qualisys Track Manager (Qualisys, Sweden) software was used for data collection. Frequency of data collection was 960 Hz. Visual 3D will also be used for data analysis.

## **Experimental Protocol**

Participants of this study were recruited from the ECU campus and surrounding areas such as local wellness centers, gyms, etc. Instruction on proper attire and protocol was given before participants came to the lab. Once in the lab, participants gave informed consent after a thorough explanation of the protocol, and associated risks.

Participants were then seated and the skin was prepped for electrode placement including shaving of the hair, exfoliation of dead skin and oils with an abrasive scrub, and a final cleaning with alcohol wipes, protocol used to reduce impedance from the skin. Four pre-gelled (Ag, Ag/Cl) electrodes and 1 ground electrode were placed on the vastus medialis and vastus

lateralis of the quadriceps, and the biceps femoris, and semitendinosus of the hamstrings muscles. The ground electrode was placed on the lateral condyle of the femur.

Once electrodes are placed a stationary squat followed by a standing right leg curl will be performed to determine quality of electrode placement. Electrodes then secured in place using a neoprene quadriceps wrap. Maximal voluntary contractions then performed for knee extension followed by knee flexion. Participants were then placed on a stationary bike for a five-minute warm up prior to starting protocol.

The participants are then seated on the Humac Norm and restrained in place using chest straps and a thigh strap to prevent movement of the right thigh. Participants were positioned such that the axis of rotation for the Humac Norm was lined up directly with the lateral condyle of the femur such that the axis of rotation of the Humac lined up with the axis of rotation about the knee. The subjects then began the protocol. Subjects performed maximal effort concentric and eccentric knee extensions at 30, 90, and 150 degrees per second both concentrically and eccentrically. The order of these speeds and contractions were randomized. Subjects were given 3 practice trials at each speed and instructed to give 50% effort for each practice trial. 5-7 maximal effort trials were obtained at each speed and contraction mode with a minimum of 90-second rest between repetitions in an effort to negate the fatigue effect. The 3 most similar trials based on torque throughout the range of motion were chosen for data analysis.

### **Data Analysis**

Raw EMG data collected in Qualysis Track Manager software. Data was then filtered with a 10-300 Hz band pass filter and rectified. Filtered Data was then exported to Microsoft Excel (Microsoft Corp, Seattle, Wa). Position data was used to create 10-degree bins. These 10

degree bins take into account the length tension properties of muscles and differences between concentric and eccentric contractions. Mean EMG was then calculated for each 10 degree bin for each muscle. Quadriceps and hamstring moments are calculated for both agonist and antagonist muscles for concentric knee extension using an EMG to force constant as developed by Aagaard et al. 2000

$$M_1 = K_1 \cdot \text{EMG}_{Q,\text{agon}} - K_2 \cdot \text{EMG}_{H,\text{antag}}$$

$M_1$  is the net moment during concentric knee extension and  $K_1$  and  $K_2$  are EMG to force constants. This equation shows the total extensor concentric moment by subtracting the antagonist flexor moment from the antagonist extensor moment. Total net eccentric moment is calculated using:

$$M_2 = K_2 \cdot \text{EMG}_{H,\text{agon}} - K_1 \cdot \text{EMG}_{Q,\text{antag}}$$

Where  $M_2$  is the net moment during eccentric knee extension for the hamstrings. Total flexor eccentric moment is calculated by subtracting antagonist extensor moment from agonist flexor moment. By solving for K in the equation, EMG to force constants may be isolated into:

$$K_1 = (A_1 + A_2) / (B_2 - B_1)$$

$$K_2 = A_1 / (1 - B_1)$$

Where  $A_1$  is the ratio of the net eccentric moment to the agonist Hamstring EMG activity ( $M_2 / \text{EMG}_{H,\text{agon}}$ ),  $A_2$  is the ratio of net concentric moment to the antagonist Hamstring EMG activity ( $M_1 / \text{EMG}_{H,\text{antag}}$ ),  $B_1$  is the ratio of antagonist Quad EMG to agonist Hamstring EMG ( $\text{EMG}_{Q,\text{antag}} / \text{EMG}_{H,\text{agon}}$ ), and  $B_2$  is ration of agonist Quad EMG to antagonist Hamstring ( $(\text{EMG}_{Q,\text{agon}} / \text{EMG}_{H,\text{antag}})$ ). Using angular position data, extensor and flexor moments for each position bin may be calculated using:

$$M_{Q_{\text{ext}}}(\theta) = K_1(\theta) \cdot \text{EMG}_Q(\theta)$$

$$M_{H_{\text{flex}}}(\theta) = K_2(\theta) \cdot \text{EMG}_H(\theta)$$

Which state the net quadriceps extensor moment at given angular position can be calculated by multiplying EMG activity of the quadriceps by EMG to torque constant, and net hamstring flexor moment at given angular position may be calculating by multiplying EMG activity of the hamstrings by the EMG to torque constant.

### **Statistical Analysis**

An analysis of variance with repeated measures will consist of training status (trained, untrained) by velocity (30,90,150<sup>°/s</sup>) to determine if there is a training status interaction with velocity of EMG activity of antagonist hamstring muscles, and the reduction of torque produced about the knee due to increased EMG level of antagonist hamstring coactivation during concentric knee extension. Any p value less than 0.05 will be followed by a Tukey's post-hoc contrast at p<0.05.

## **Chapter IV. Results**

The purpose of this study was to determine if a) resistance trained individuals produce lower levels of EMG activity in the antagonist muscles during maximal effort knee extension, b) this lower EMG level produces less counteracting torque, and c) if the training history and contraction velocity interact. The main finding of the present study was that trained individuals exhibited ~44% less “coactivation” than did untrained individuals. Coactivation of the hamstring muscles did not vary with contraction speed of the quadriceps muscle because the 10% difference in coactivation between trained and untrained individuals across the 3 speeds was statistically not significant and other differences were even smaller. While trained vs. untrained individuals produced approximately 46%, 88%, and 92% greater antagonist torque, as predicted by a mathematical model, at 30, 90, and 150 °/s, respectively, these between-group differences were statistically not significant. This Chapter is separated into 4 sections: 1) coactivation ratio during concentric contraction, 2) net torque during concentric contraction, 3) agonist torque during concentric contraction, 4) antagonist torque during concentric contraction, 5) measured vs calculated torque validity and 6) Aagaard’s Model. Figure A shows sample EMG and torque tracing from a trained individual and Figure B shows a tracing from an untrained individual.

Figure A: Trained individuals torque, vastus lateralis, vastus medialis, biceps femoris, and semitendinosus

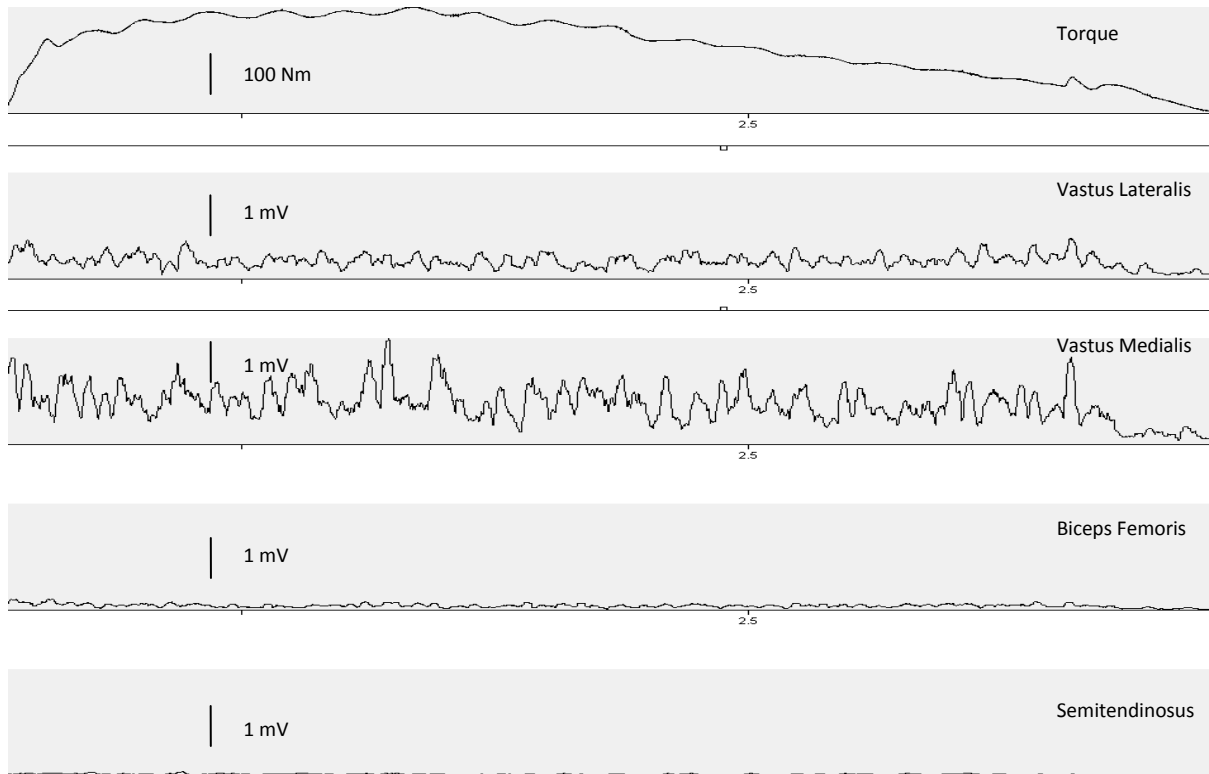
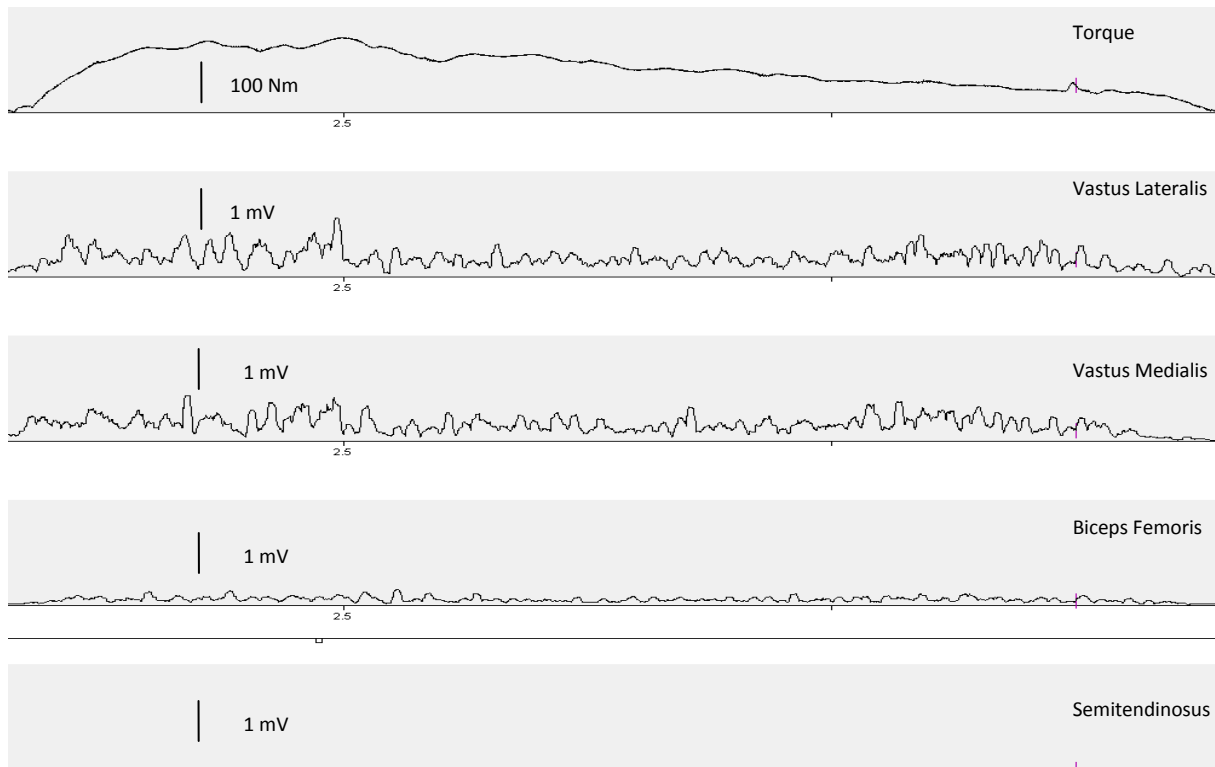


Figure B: Untrained individuals torque, vastus lateralis, vastus medialis, biceps femoris, and semitendinosus



## Coactivation ratio during concentric quadriceps contraction

Training Status Main Effect - Figure 1a shows the main effect of training status on hamstring/quadriceps coactivation ratio averaged across each bin and bout. Trained vs. untrained subjects had 44% less coactivation ( $F=14.1$ ,  $p=.001$ ).

Speed Main Effect – Figure 1 b shows that there was no main effect of speed on hamstring/quadriceps coactivation ratio. There was less than 10% difference in coactivation between any combination of speeds ( $F=1.67$ ,  $p=.201$ )

Group by speed Interaction Effect – There was no group by speed interaction effect ( $F=.031$ ,  $p=.970$ ).

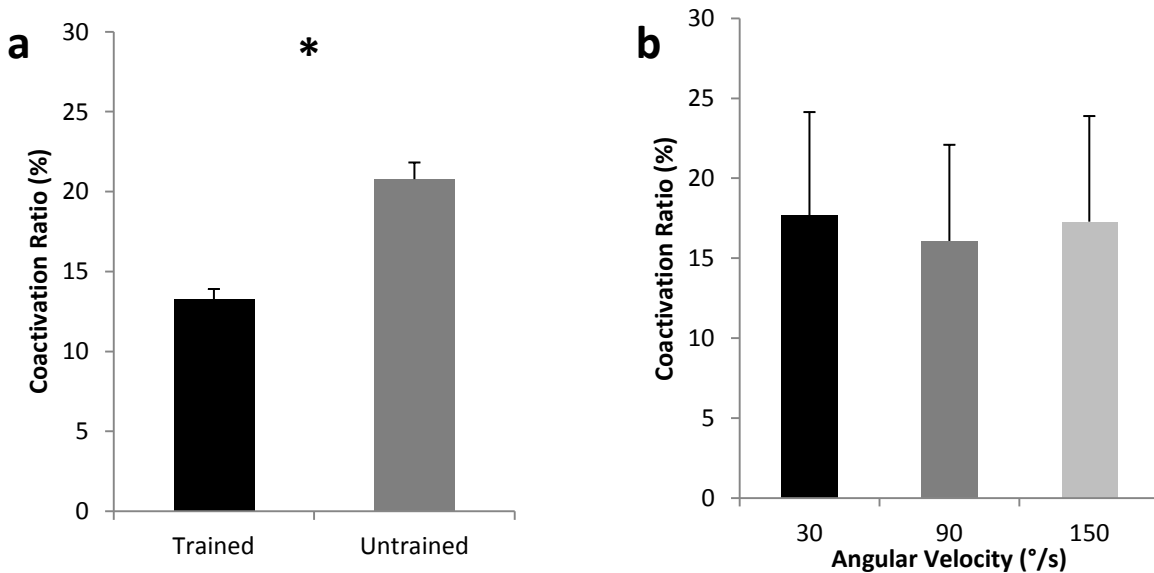


Figure 1: Training Status (a) and speed (b) main effects. (\*) indicates  $p < .05$  difference between trained and untrained.

## Peak Net torque during concentric contraction

Training Status Main Effect - Figure 2a shows the group main effect in peak net torque production. Trained were  $\sim 34\%$  stronger than untrained ( $F=5.87$ ,  $p=.026$ ). Peak net torque is



the torque as a result of the agonist torque – the antagonist torque as calculated with the Aagaard model.

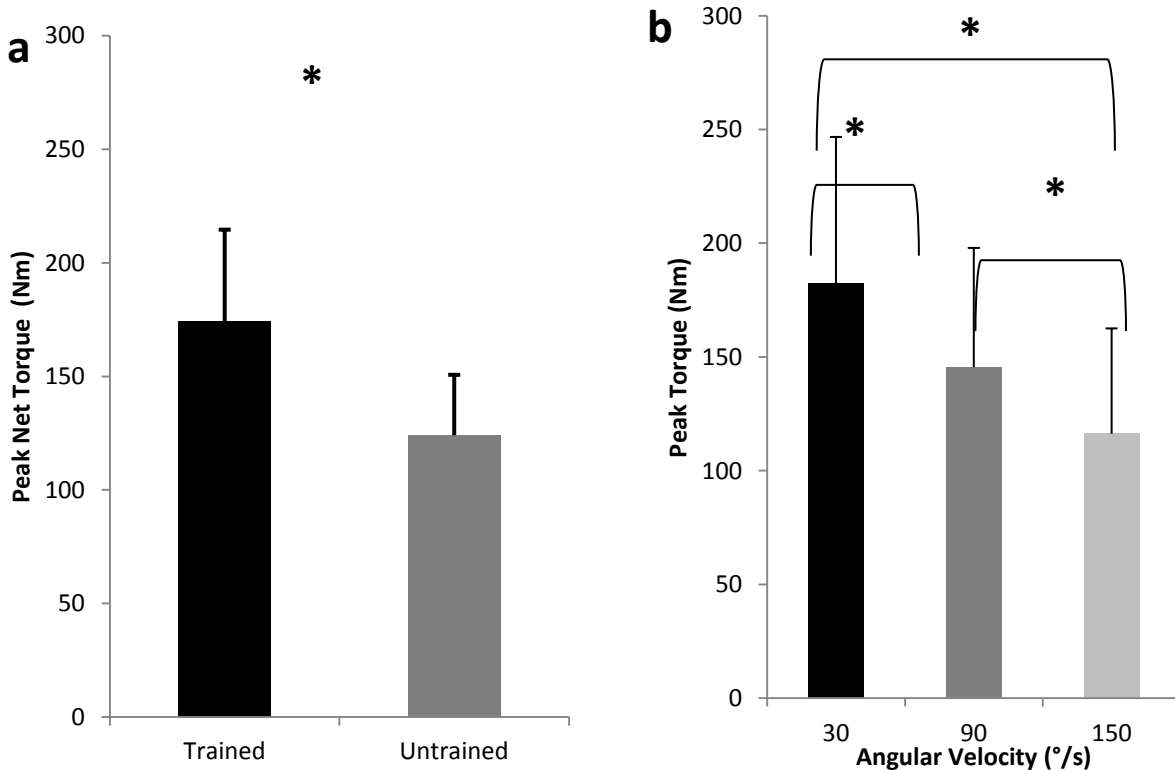
Speed main Effect - Figure 2b shows the speed main effect on peak net torque production.

Torque was 22% greater between 30-90, 22% greater between 90-150, and 44% greater between 30-150 ( $^{\circ}/s$ ). ( $F=85.7$ ,  $p=.000$ ).

Group by Speed Interaction Effect – Figure 2c shows the group by speed interaction ( $F=3.5$ ,

$p=.039$ ). Tukeys post hoc revealed that peak net torque was 23% higher at 30 than 90 for

trained and 21% higher in the untrained. 30 was 46%, and 43% greater than 150 for trained and untrained respectively. Finally, 90 was 23% and 21% greater than 150 for trained and untrained respectively.



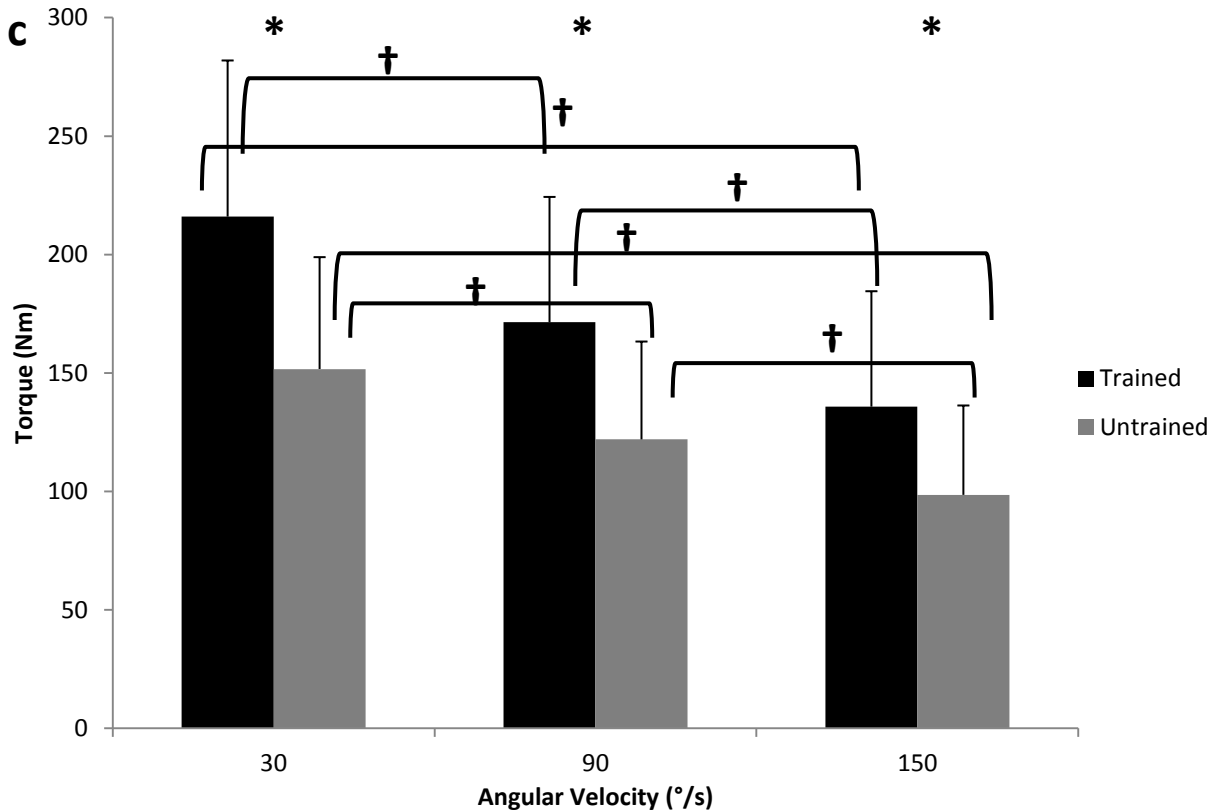


Figure 2: Group main effect (a), speed main effect (b), and group by speed interaction effect (c). (\*) indicates  $p < .05$  between groups and within speeds. † indicates an differences within group between speeds with  $p < .05$  as indicated by Tukeys HSD Post Hoc Analysis

### Peak Agonist Torque during Concentric Contraction

Training Status Main Effect – Figure 3a shows the group main effect of agonist torque ( $F=7.2$ ,  $p=.015$ ). Trained individuals generated 45% greater agonist torque than their untrained counterparts. Agonist torque is the torque produced by the quadriceps muscle only, and is calculated by the net torque + the antagonist torque.

Speed Main Effect – Figure 3b shows the speed main effect on agonist torque being greater at 30 than 90 by 18%, 90 than 150 by 15%, and 30-150 by 32% ( $F=53.5$ ,  $p=.000$ ).

Group by Speed Interaction Effect – There was no group by speed interaction effect ( $F=0.2$ ,  $p=.77$ )

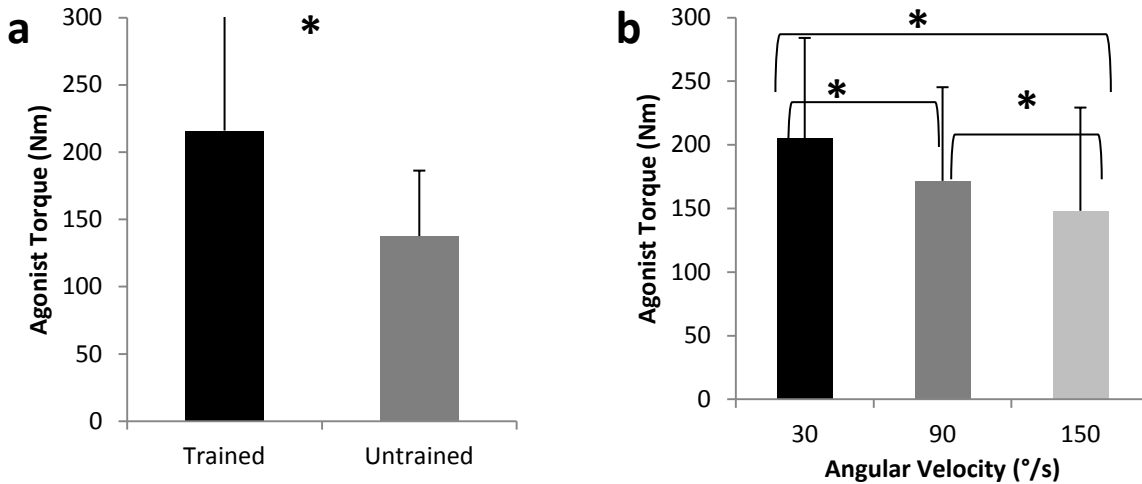


Figure 3: Shows group main effect (a), and speed main effect (b) on agonist torque production. (\*) indicates  $p < .05$

### Antagonist Torque during Concentric Contraction

Training Status Main Effect – As seen in figure 4a there was an almost significant group main effect of antagonist torque production ( $F=3.5$ ,  $p=.075$ ). Antagonist torque is the torque produced by the hamstrings during knee extension, resulting and a decreased net torque value in relation to agonist torque.

Speed Main Effect - Also in figure 4b there was no significant speed main effect on antagonist torque production ( $F=1.99$ ,  $p=.151$ ).

Group by Speed Interaction Effect - Figure 4c shows there was a trend for a group by speed interaction effect. ( $F=3.12$ ,  $p=.056$ )

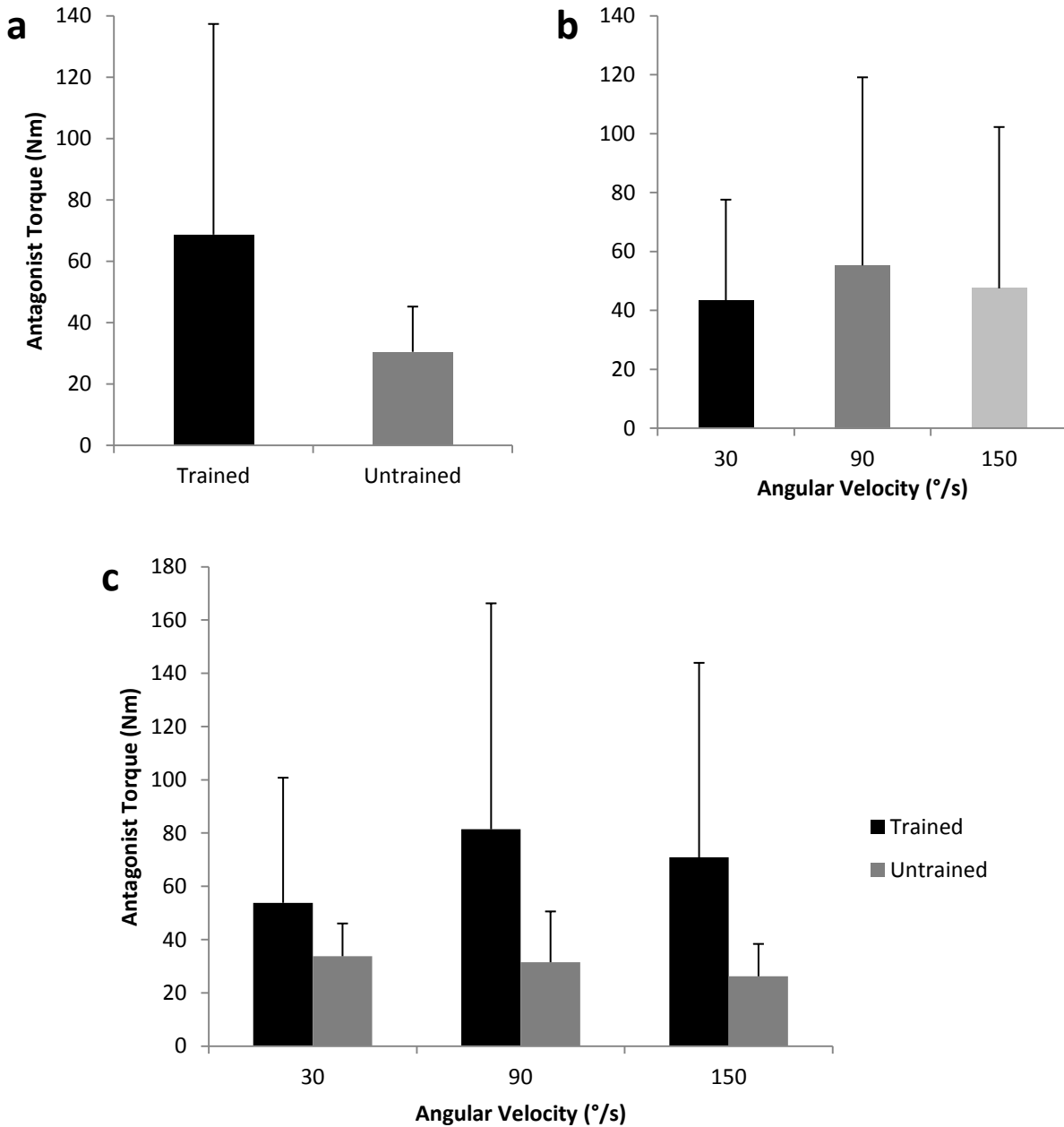


Figure 4: Shows Group main effect (a), speed main effect (b) and group by speed interaction for antagonist torque (c).

### Peak Hamstring Torque during Eccentric Knee Extension

Training Status Main Effect – As seen in figure 5a there was a group main effect where trained individuals produced ~39% greater torque than the untrained ( $F=5.28, p=.033$ )

Speed Main Effect - Also in figure 5b there was no significant speed main effect on hamstring torque production ( $F=1.71$ ,  $p=.195$ ).

Group by Speed Interaction Effect - There was no group by speed interaction effect. ( $F=.481$ ,  $p=.662$ )

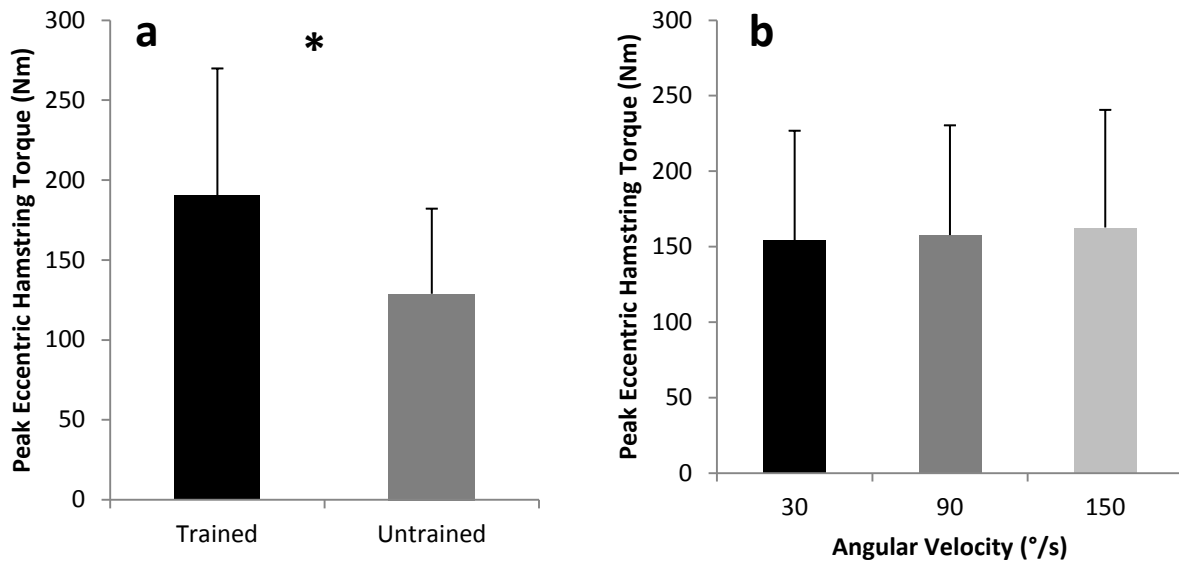
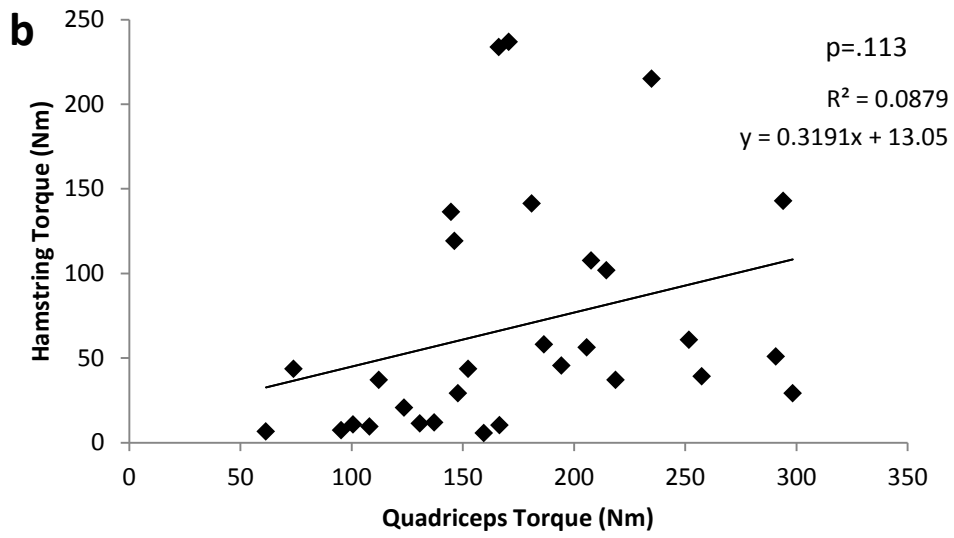
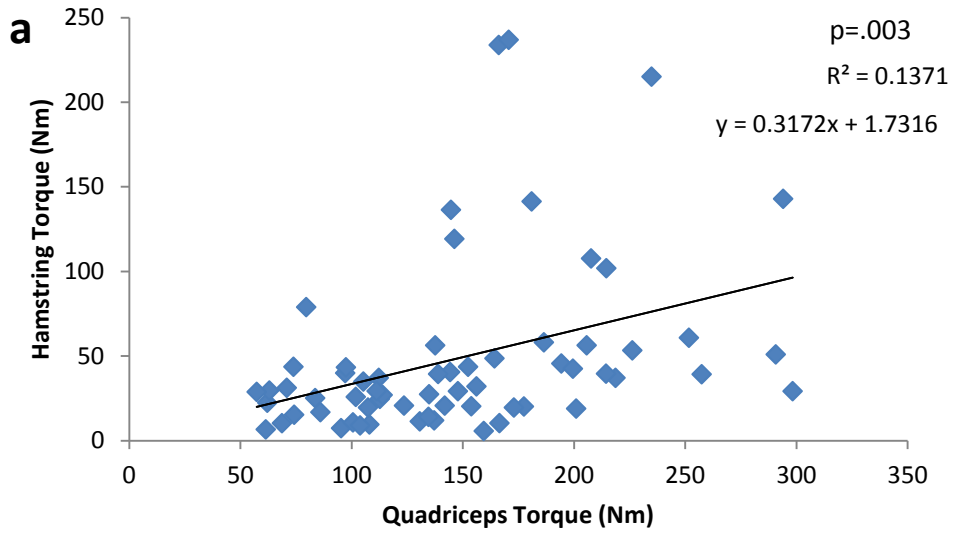


Figure 5: Shows group main effect (a) and speed main effect (b)

### Correlation Analyses

Quadriceps torque x hamstring torque correlation: Figure 6 shows quadriceps torque x hamstring torque. The weak positive correlations suggest that as quadriceps torque increases, hamstring torque also increases. However, utilizing a Pearson coefficient some correlations were deemed significant at an alpha level .05.



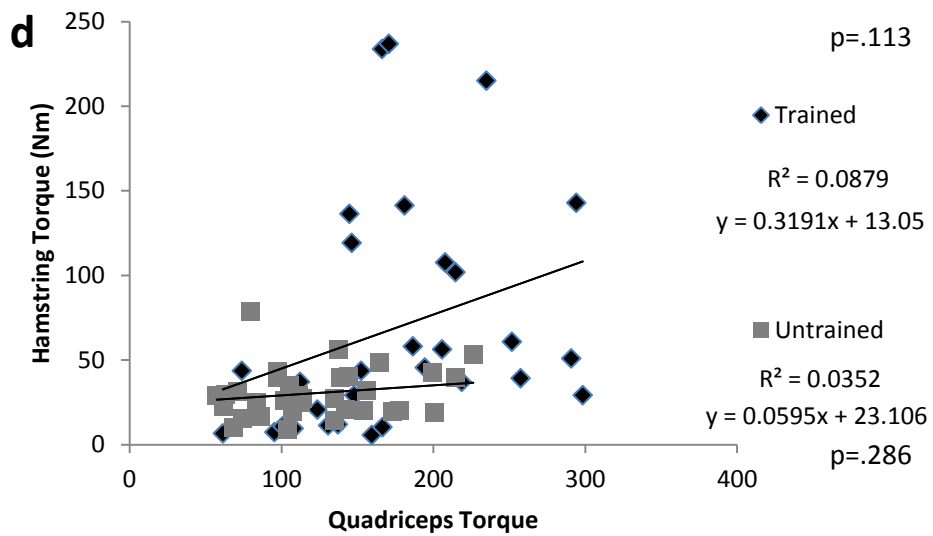
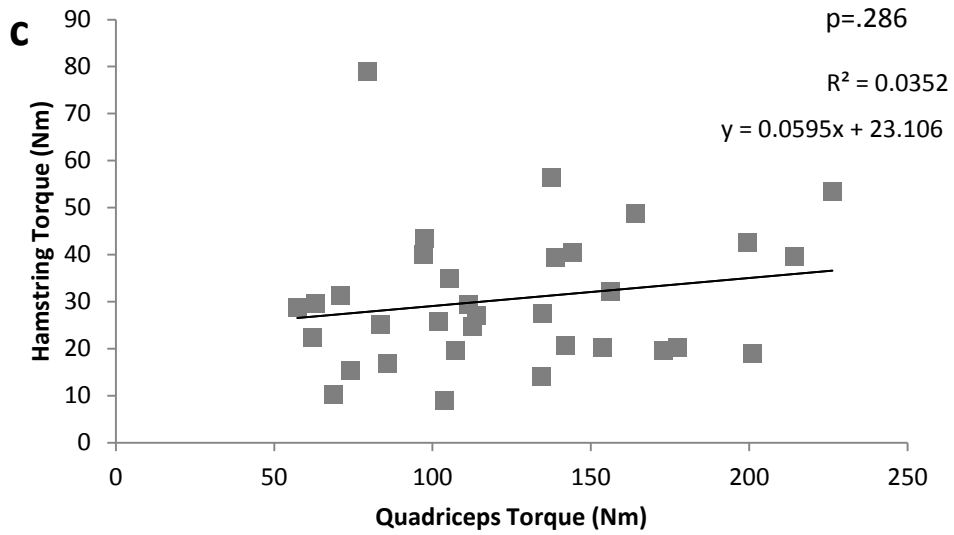
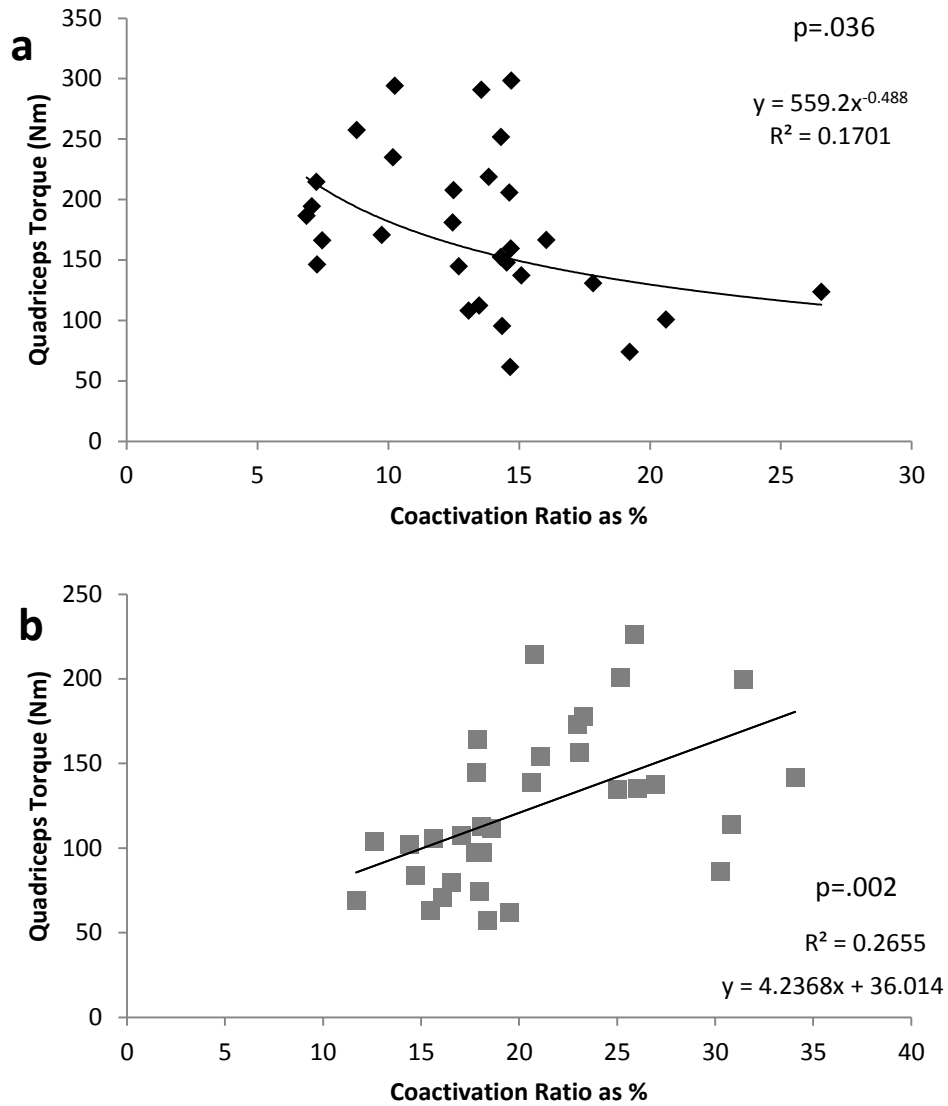


Figure 6: Trained and untrained quad x ham torques (a), trained quad x ham torque correlation (b), untrained quad x ham torque correlation (c), and trained vs untrained quad by ham correlation (d).

Coactivation ration x quadriceps torque correlation: Figure 7 shows the correlations of coactivation ratio and quadriceps net torque. Note the negative relation for increased coactivation ratio yields decreased quadriceps net torque for trained, and vice versa for the untrained.





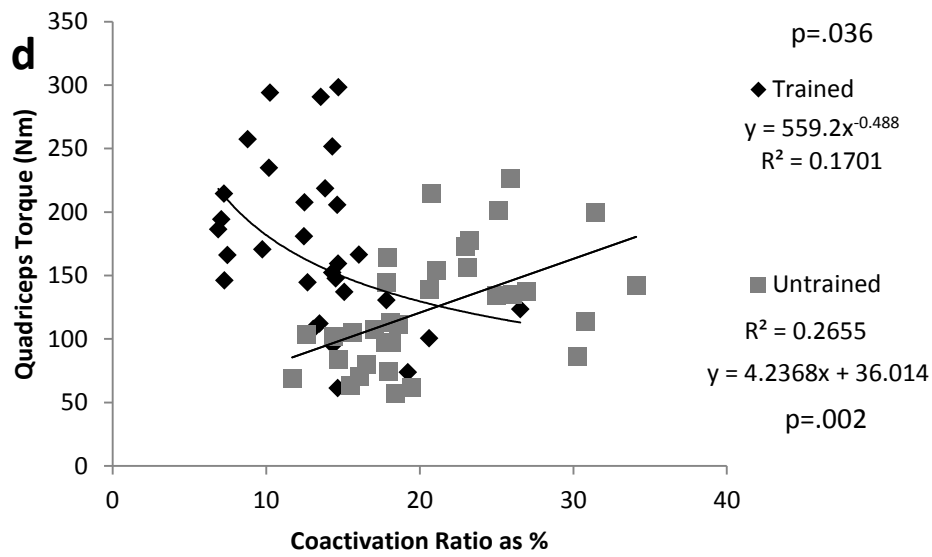
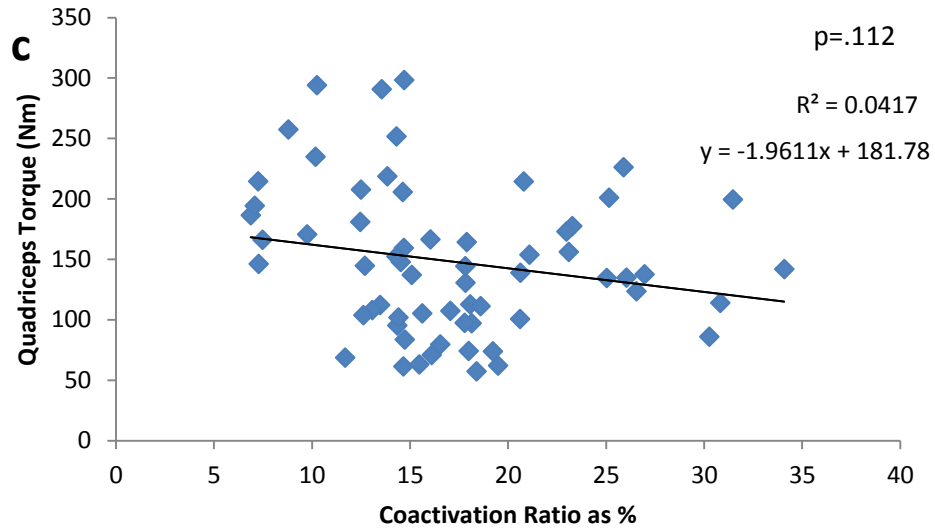


Figure 7 shows the combined relationship of trained and untrained coactivation ratio x quadriceps net torque (a), the trained only correlation (b), untrained correlation (c), and trained vs untrained correlation (d)

### Measured-Calculated Torque Validity

Validity - Figure 8 shows that measured torque for trained individuals at 90°/s and calculated torque were only 2% different. This is consistent with all groups, at all speeds

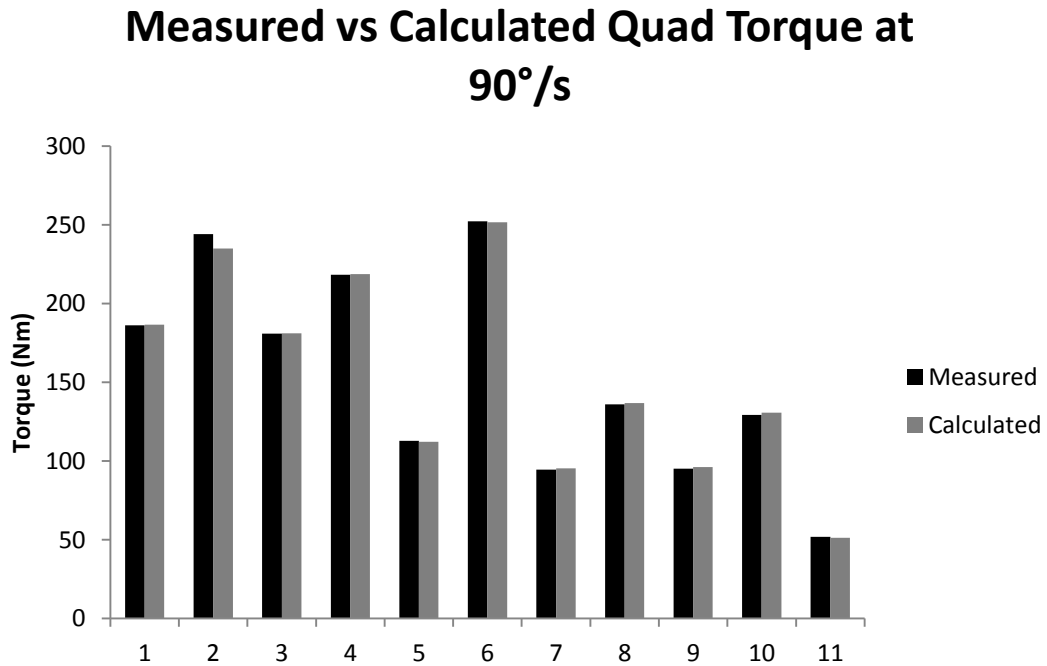


Figure 8 shows measured and calculated peak torques for Trained Individuals at 90°/s

## **Chapter V. Discussion**

The purpose of this study was to determine if a) resistance trained individuals produce lower levels of EMG activity in the antagonist muscles during maximal effort knee extension, b) this lower EMG level produces less counteracting torque, and c) if the training history and contraction velocity interact. The main finding was that trained vs. untrained individuals had ~44% less coactivation. This was consistent across all 3 speeds. This depression in coactivation did however trend towards an actual increase in antagonist torque ( $p=.075$ ). These findings were again constant across speeds. And finally, contraction speed did not affect coactivation levels differently in the two groups as there was no group by speed interaction. The question of the population within this subject pool being heterogeneous between the two groups is a question that is not easily answered. We qualified individuals as trained that resistance trained the lower body at least once a week, along with some other mode of lower body exercise at least a second day of the week. The trained individuals in this study produced greater torque with the knee extensors (260 Nm), and the untrained individuals produced a lesser knee extensor torque (155 Nm) than another study combining both a similar number of men and women in both the trained and untrained group at 30°/s with trained averaging (221 Nm) and untrained averaging (183 Nm), (Poulis et al, 2009). This adds validity that our groups were indeed different, and helps us assume the trained are indeed trained, and the untrained, are untrained as defined in the present study.

The speed x velocity relationship was present within this study further validating the methods. There was a significant decrease in trained individual's peak torque values of about 22% between both 30-90 and 90-150 °/s velocities. These findings are consistent with

the torque-velocity relationship (Enoka, 2008, p. 241, and de Haan, A. 1998). Speed had an effect on peak torque production for both the trained and untrained individuals.

### **Neural Adaptation to Resistance Training: Coactivation Ratio**

According to the hypothesis trained individuals executed the knee extension task with 44% significantly lower hamstring muscle coactivation. These findings are in agreement with some but not all cross-sectional studies (Aagaard et al 2000, Simoneau et al 2009) and also agree with decreases in coactivation following chronic knee extension strength training in previously untrained individuals (Carolan and Cafarelli, 1992). We also see a reversal of these findings in that several studies show that with training there is an increase in coactivation especially in the elderly. This could make it even more difficult to perform regular activities of daily living such as standing from a chair (Fujita et al, 2011).

The interpretation of reduction in coactivation in the context of the present study is not straight forward. While practicing basic skills associated with the knee extension task in strength training would tend to reduce counteracting torque effect from the hamstrings (Aagaard et al 2000, Kellis & Baltzopoulos 1999 & 1998, Simoneau et al 2009). The significantly higher measured torque of trained individuals would seemingly require greater stability at the knee, often associated with greater coactivation in order to reduce anterior shear forces of the tibia on the patella tendon (Snow et al 1987, Kubo et al 2004, Aagaard et al 2000). Of these two opposing mechanisms, if correct, then perhaps the skill component has a greater effect because the coactivation was less and longitudinal strength training studies also tend to report reductions in antagonist torque as part of the neural adaptation phenomenon, especially in young adults (Amiridis et al 1996, Carolan et al 1992, Enoka 1997, and Sale 1988, Tillin et al

2010). It has also been documented that as contraction intensity increases, coactivation levels increase, possibly to increase stability of the knee, or other neural adaptations to prevent injury, and especially to slow the shank at the extreme of the knee extension range of motion (Clark et al 2010, Hakkinen et al 2000, Macaluso et al 2002). Surprisingly the current study did not find an overall increase in coactivation with increasing contraction speed in either the trained or untrained group as previously shown ( Hagood et al 1990, Osternig et al 1986). This may be due to a relatively small range of speeds from 30-150°/s in the current study, compared to other studies such as Hagood et al in 1990 testing 15-240°/s. Furthermore, a neural strategy may have been present within subjects due to the novelty of the isokinetic dynamometry task. There was a noticeable increase in coactivation during the final 20 degrees of extension, which is in agreement with past studies; however, this issue was not investigated in this study (Kubo et al 2004, Remaud et al 2007). This increased activation is thought again to be a protective mechanism, preventing the knee from forcefully extending past its natural range of motion.

Correlation analysis of coactivation ratio vs. quadriceps net torque revealed an interesting finding. While all correlations are low, we see that as a group there is no correlation between an increase in coactivation ratio and peak knee extensor torque. The interesting finding is that trained individuals tend to have a decreased torque with higher coactivation, while the untrained have an increasing torque with increasing coactivation. This result may imply that while trained individuals are able to isolate the activation of the knee extensors exclusively with focused EMG, any increase in activation of the antagonist muscle yields a reduction in torque as the quadriceps are already maximally activated. While untrained individuals are not able to isolate the knee extensor muscles as well, with increasing torque we

see a less focused EMG activation, leading to not only increased activation of the knee extensors, but also the antagonist knee flexors. This may indicate that a result of training has a skill component, or a learning component to focus activation of the agonist muscles, while only activating the antagonist in such a way to support proper knee function. Also it appears that regardless of training status, there is a wall of data points at approximately 8% (hamstring/quadriceps) coactivation ratio that may indicate that regardless of training history we have a floor effect in terms of a minimal amount of antagonist coactivation during maximal effort knee extension. This finding is novel, and has not been addressed in previous literature to our knowledge. This finding may indicate that regardless of training, or skill level, coactivation is required to some extent for one reason or another. The reasoning for coactivation is still not clear, yet the recurring theme is joint stiffness, and stability.

This finding is in agreement with trained individuals trending in a positive manner where increases in quadriceps net torque, is also met with an increase in hamstring net torque. The resultant increased hamstring torque would be a byproduct of increased coactivation. So while trained individuals produce lower coactivation ratios, they produce the same amount, if not more antagonist torque that both aids the knee for stability, but may also hinder the net extensor torque about the knee.

The utilization of the coactivation ratio (antagonist EMG/agonist EMG) has both distinct advantages and disadvantages. While there is no universal measure for coactivation, utilizing the coactivation ratio of the average of the biceps femoris and semitendinosus EMG activity, divided by the average EMG of the vastus lateralis and medialis throughout the motion gives us a quantitative ratio of both muscles during the task at hand. This ratio takes into account the

neural drive from the CNS for both the agonist and antagonist muscles with similar impedance of the action potential due to factors such as adipose tissue, and skin debris. This method has been used in similar studies such as Carolan and Cafarelli, 1992, and Psek et al, 1993. This method of measuring coactivation also has disadvantages. One such disadvantage is the possibility of cross-talk. Cross-talk is the volume of neural activity from the agonist muscle that may infringe upon other surrounding muscles including synergist, and antagonist muscles and may increase the EMG signal of said muscles. Cross talk was not measured, however when proper interelectrode distance is utilized the effects of cross talk are negligible (Fuglevand et al, 1992). A recent study suggests that while antagonist EMG measures antagonist activity, it does not account for different activation patterns during eccentric conditions, such as an antagonist muscle experiences, as well not accounting for different architectural patterns (Simoneau et al, 2012). This is especially relevant when normalizing antagonist EMG to a value derived from the antagonist muscle as the agonist during a similar motion ie normalizing hamstring EMG during a knee extension, to the hamstring EMG during a knee flexion task.

### **Mechanical Adaptations to Knee Extensor Resistance Training: Increased Peak Torque**

The data on peak torque are consistent with regular adaptations of resistance training in that the resistance trained group indeed was stronger than the untrained group. Allowing this study to be used to examine the main hypothesis of whether there is a difference in the neural control strategy of force production due to strength level and activation of the muscles acting as the antagonist. The utilization of isokinetic dynamometry and EMG in previous studies have shown that incorporating an EMG to torque model, the net torque production of the agonist knee extensors is hindered by the counteracting antagonist torque produced by the knee

flexors (Aagaard et al 2000, Amiridis et al 1996, Baratta et al 1988). Recent studies also shed light on the possibility that not only neural but mechanical behavior of the fascicles in the antagonist muscle could have an effect (Simoneu et al 2012).

As expected, trained individuals produced ~34% greater torque than the untrained individuals participating in this study with the knee extensors. While we expected a larger difference, not just the magnitude of torque plays a role in the antagonist muscle behavior, but the practice of simple motor skills, as well as repetitive exercise contractions. Trained also produced greater eccentric torques with the knee flexors ~ 39% greater, with this number staying consistent throughout each speed. Although this issue is currently controversial some studies did show neural adaptations in the brain (Farthing et al, 2007) and in the corticospinal pathway (Hortobagyi et al, 2011), other studies found no adaptations with simple motor skills and loads normally used in resistance training programs (Jensen et al, 2005).

Resemblance of the data to the predicted force-velocity curve would increase the internal validity of the study. Indeed, as expected torque decreased with increasing quadriceps contraction speed ~ 22% between each condition in both groups for concentric contractions. Eccentric Contractions, however, did not significantly change across the speeds.

We did find an interaction effect of group by speed in this study for peak net torque of the knee extensors. However, Tukey's Post Hoc Analysis revealed minor differences between groups, therefore, we deemed this finding significant but not meaningful.

### **Mechanical Adaptations to Knee Extensor Resistance Training: Agonist Torque**

Agonist torque, is the theoretical torque produced by the quadriceps during a concentric knee extension as if there was 0 negative torque produced by the antagonist



hamstrings. Therefore the resultant equation is  $\text{Agonist torque} = \text{net torque} + \text{antagonist torque}$ . The equation as described by the Aagaard model has the distinct advantage of the EMG-torque relationship being derived from the actual torque at given EMG during agonistic contraction of the hamstrings during an eccentric contraction. This negates the need for mathematical estimations of moment arms, muscle architecture, etc. This model allows us to seemingly estimate agonist, and antagonist torques in the most accurate way utilizing solely EMG driven models. In the current study agonist torque values were 45% greater in the trained than untrained, with that figure being consistent across all 3 speeds. Agonist torque was ~18% greater at 30 than 90 °/s, and 15% greater at 90 than 150 °/s. We see a greater difference between the trained and untrained when comparing the agonist torque leading us to believe that the antagonist forces are greater in the trained group, resulting in a closer net torque production as there is a 45% greater agonist torque from trained, and only a 34% greater net torque for the trained. Greater agonist forces lead to a greater anterior shear force on the knee as the patella acting as a lever arm extender for the knee extensors allows for even greater force production (Hagood et al, 1990). This resultant increased torque by the agonist quadriceps would prospectively require greater antagonist torque to stabilize and stiffen the knee.

### **Mechanical Adaptations to Knee Extensor Resistance Training: Antagonist Torque**

Antagonist torque is the theoretical torque produced by the hamstrings during a concentric knee extension. Antagonist torque is attributed to creating posterior shear force on the tibia, and increasing compressive forces within the knee (Kellis et al 1999). Antagonist torque is the mechanical output produced by the neural mechanism known as coactivation.

Increasing antagonist torque would in theory increase knee stability, but at the same time reduce net knee torque. It has previously been suggested that one mechanism of increased torque with training is a decrease in coactivation (Carroll et al, 2002, Duchateau et al, 2002). However, in this study we see that the trend is that trained exhibit ~75% greater antagonist torque than the untrained, 69 Nm for trained, and 30 Nm. These findings, namely the untrained group, were consistent with the untrained individuals in a previous study by Aagaard in 2000. This difference was borderline significant ( $p=.075$ ), potentially due to the large standard deviation within the trained group (~68 Nm) and suggest with greater net torque production, we also see greater agonist and antagonist torques, that would coincide with greater joint stability needed when lifting heavier loads, or producing greater forces, as trained individuals generally do compared to their untrained counterparts. There were no statistically significant differences but we did see a borderline group main effect of training status ( $p=.075$ ). Interestingly trained subjects had lower activation of the antagonist in relation to the quadriceps based on EMG, while producing borderline statistically greater torques. This indicates that trained are able to produce greater torques, with lesser amounts of EMG. This is a novel finding not previously recorded or mentioned in any other literature to my knowledge.

### **Ecological Validity**

The ecological validity of the data is unclear. The similarities between isokinetic contractions and other dynamic muscle contractions such as squats, and other real life tasks are uncertain. Previous studies have compared open kinetic chain and closed kinetic chain exercises and finds conflicting results. Escamilla et al 1998, showed that squats had significantly greater hamstring coactivation almost 2 fold, whereas Kvist et al 2001 showed open kinetic

chain exercises produced greater antagonist activity. It has also been shown that closed kinetic chain exercise produced no anterior shear force on the knee and greater compressive force of the knee along with greater posterior shear tension. Open kinetic chain exercises produced anterior shear force about the knee as well as less posterior shear tension (Escamilla et al 1998). The present study further adds to the literature attempting to understand underlying neural mechanisms that may alter the way our bodies do things based on resistance training. While some of our results are consistent with prior literature, we have also been the first to our knowledge to incorporate the EMG driven model to estimate antagonist force in trained and untrained young adults. While the results currently trend to trained individuals producing greater antagonist torque we cannot say that yet. However with the addition of more trained and untrained subjects this finding may be deemed significant. Trained individuals moving greater weights, and producing greater antagonist torque would be both a positive and negative for the individual. While they produce greater antagonist torque, seemingly increasing stability at the knee, the individual also has to overcome the weight being used, but also that opposing force of their antagonist muscles. This could lead to increased energy expenditure, and possibly hinder performance to some degree.

#### **Aagaard's Model**

Utilizing the model derived by Aagaard et al in 2000, has one distinct advantage, and that is the EMG-torque ratio is based off an eccentric contraction for the hamstrings as the hamstrings act in an eccentric manner during a concentric knee extension. Other models such as Baratta et al in 1998 utilized an EMG-torque ratio utilizing EMG during a concentric contraction for the hamstring. Normalizing EMG for antagonist muscles acting in an eccentric

fashion, to EMG obtained when said muscles are the agonist acting in a concentric fashion does not take into account the differences in muscle properties present at various contraction modes ie concentric, eccentric, or isometric. Also, eccentric contractions have a smaller emg-torque ratio than concentric which could further exacerbate the error in other models. The hamstrings are capable of producing approximately 40-50% of the concentric torque compared to quadriceps concentric torque (Aagaard et al 2000). However, the hamstrings are capable of producing approximately 90-130% eccentric torque when compared to the quadriceps during concentric knee extension (Aagaard et al 2000). Finally, the Aagaard model also takes into account the length tension relationship by partitioning each contraction into  $10^\circ$  bins, accounting for differences in actin myosin overlap, as well as changing moment arms throughout the range of motion for both the concentric, and eccentric knee extension tasks. Therefore there is a greater likely hood that the calculated antagonist torque of the hamstrings is more accurate utilizing this EMG driven model than others utilizing EMG-torque ratios during concentric action of the hamstrings.

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## Appendix A: Consent Form



### EAST CAROLINA UNIVERSITY

University & Medical Center Institutional Review Board Office  
11-09 Brody Medical Sciences Building • 600 Moye Boulevard • Greenville, NC 27834  
Office 252-744-2914 • Fax 252-744-2284 • www.ecu.edu/irb

TO: Patrick Rider, MA, CSCS, Dept. of EXSS, ECU—360 Ward Sports Medicine Building  
FROM: UMCIRB *kk*  
DATE: April 5, 2011  
RE: Expedited Category Research Study  
TITLE: “Quadriceps and Hamstring Coactivation During Maximal Knee Extension”

#### UMCIRB #11-0208

This research study has undergone review and approval using expedited review on 11.4.11. This research study is eligible for review under an expedited category number 4. The Chairperson (or designee) deemed this **unfunded** study **no more than minimal risk** requiring a continuing review in **12 months**. Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The investigator must adhere to all reporting requirements for this study.

The above referenced research study has been given approval for the period of **4.4.11** to **4.3.12**. The approval includes the following items:

- Internal Processing Form (dated 3.16.11)
- Research Opportunity (received 3.31.11)
- Informed Consent (version date 3.31.11)

The Chairperson (or designee) does not have a potential for conflict of interest on this study.

**The UMCIRB applies 45 CFR 46, Subparts A-D, to all research reviewed by the UMCIRB regardless of the funding source. 21 CFR 50 and 21 CFR 56 are applied to all research studies under the Food and Drug Administration regulation. The UMCIRB follows applicable International Conference on Harmonisation Good Clinical Practice guidelines.**

**Informed Consent to Participate in Research**

Information to consider before taking part in research that has no more than minimal risk

Title of Research Study: **Quadriceps and hamstring coactivation during maximal knee extension**  
Principal Investigator: Patrick Rider  
Institution/Department or Division: Exercise and Sport Science  
Address: 332 Ward Sports Medicine Building  
Telephone #: 252.737.4616

Researchers at East Carolina University (ECU) study problems in society, health problems, environmental problems, behavior problems and the human condition. Our goal is to try to find ways to improve the lives of you and others. To do this, we need the help of volunteers who are willing to take part in research.

**Why is this research being done?**

The purpose of this research is to determine the effects of resistance training on quadriceps and hamstrings coactivation, and the resulting torque production in both muscle groups. The decision to take part in this research is yours to make. By doing this research, we hope to learn how strength training impacts the nervous control during a knee extension task.

**Why am I being invited to take part in this research?**

You are being invited to take part in this research because you are right-legged, age 18 to 35-years-old, free of orthopedic and neurological conditions, a non-smoker, and either do not lift weights more than once a week, or resistance train your lower extremities at least twice per week. If you volunteer to take part in this research, you will be one of about 30 people to do so.

**Are there reasons I should not take part in this research?**

I understand I should not volunteer for this study if I am a smoker, under 18 years of age, suffered a serious injury to my legs, or had or have neurological condition (stroke, Parkinson's disease).

**What other choices do I have if I do not take part in this research?**

You can choose not to participate.

**Where is the research going to take place and how long will it last?**

The research procedures will be conducted in the room 332 Ward Sports Medicine Building, Biomechanics Laboratory. The study consists of 1 visit lasting approximately 2 hours where you will perform all of the testing procedures after receiving proper warm up and instruction.

**What will I be asked to do?**

You are being asked to do the following:

At the beginning of the testing session, electrodes (like the ones used in a chest EKG) will be placed on the target muscles on the legs. These electrodes will measure muscle activity. The testing session will assess my ability to produce maximal force on a computerized device. I will have ample opportunity to practice and become familiar with the task. To measure maximal force, I will press against a pad on a computer-controlled device. The computer controls the speed of the movement. I will perform each task a maximum of 7 times, with 2 minutes of rest between each trial. The speed that the computerized device moves my leg will be randomized between a slow, medium or fast setting.

**What possible harms or discomforts might I experience if I take part in the research?**

UMCIRB Number: 11-0208

Consent Version # or Date: 3.31.11  
UMCIRB Version 2010.05.01

UMCIRB  
APPROVED  
FROM 4.4.11  
TO 4.3.12

\_\_\_\_\_  
Participant's Initials

*Title of Study:* Quadriceps and hamstring coactivation during maximal knee extension

As with any strong effort or working out in a gym and lifting weights, there is a possibility for muscle strain to occur. A thorough familiarization and warming up will minimize the risks for muscle strain and soreness. Due to the short time span of the trials, as well as the ample rest times between trials, there is no more than minimal risks for any healthy young adult.

**What are the possible benefits I may experience from taking part in this research?**

This study will hopefully reveal a novel interpretation for changes in muscle activation levels due to resistance exercise. We hope to use these findings to understand more precisely how humans control movements. As a student, you will also participate in cutting-edge technology research on muscle and nervous system function and participation in the study provides an educational experience.

**Will I be paid for taking part in this research?**

There will not be any payment for taking part in this research.

**What will it cost me to take part in this research?**

It will not cost you any money to be part of the research.

**Who will know that I took part in this research and learn personal information about me?**

To do this research, ECU and the person listed below may know that you took part in this research and may see information about you that is normally kept: Patrick Rider, the main investigator.

**How will you keep the information you collect about me secure? How long will you keep it?**

Data files will be kept for 5 years after the study is completed. The investigator will keep your personal data in strict confidence by having your data coded. Instead of your name, you will be identified in the data records with an identity number. Your name and code number will not be identified in any subsequent report or publication. The main investigator will be the only person who knows the code associated with your name and this code will be kept in strict confidence. The computer file that matches your name with the ID number will be encrypted and the main investigators will be the only staff that knows the password to this file. The data will be used for research purposes.

**What if I decide I do not want to continue in this research?**

If you decide you no longer want to be in this research after it has already started, you may stop at any time. You will not be penalized or criticized for stopping. You will not lose any benefits that you should normally receive.

**Who should I contact if I have questions?**

The people conducting this study will be available to answer any questions concerning this research, now or in the future. You may contact the Principal Investigator at 252.737.4616 (days, between 8 am to 5 pm).

If you have questions about your rights as someone taking part in research, you may call the Office for Human Research Integrity (OHRI) at phone number 252-744-2914 (days, 8:00 am-5:00 pm). If you would like to report a complaint or concern about this research study, you may call the Director of the OHRI, at 252-744-1971

**I have decided I want to take part in this research. What should I do now?**

The person obtaining informed consent will ask you to read the following and if you agree, you should sign this form:

- I have read (or had read to me) all of the above information.
- I have had an opportunity to ask questions about things in this research I did not understand and have received satisfactory answers.
- I know that I can stop taking part in this study at any time.
- By signing this informed consent form, I am not giving up any of my rights.

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UMCIRB  
APPROVED  
FROM H. H. 11  
TO H. B. 12

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Participant's Initials

*Title of Study:* Quadriceps and hamstring coactivation during maximal knee extension

- I have been given a copy of this consent document, and it is mine to keep.

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Participant's Name (PRINT)	Signature	Date
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**Person Obtaining Informed Consent:** I have conducted the initial informed consent process. I have orally reviewed the contents of the consent document with the person who has signed above, and answered all of the person's questions about the research.

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Person Obtaining Consent (PRINT)	Signature	Date
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UMCIRB Number: 11-0208

Consent Version # or Date: 3-31-11  
UMCIRB Version 2010.05.01

UMCIRB  
APPROVED  
FROM 4-4-11  
TO 4-3-12

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Participant's Initials

\*\*\*\*\*IMPORTANT INFORMATION\*\*\*\*\*

### Continuing Review/Closure Obligation

As a investigator you are required to submit a continuing review/closure form to the UMCIRB office in order to have your study renewed or closed before the date of expiration as noted on your approval letter. This information is required to outline the research activities since it was last approved. You must submit this research form even if you there has been no activity, no participant s enrolled, or you do not wish to continue the activity any longer. The regulations do not permit any research activity outside of the IRB approval period. Additionally, the regulations do not permit the UMCIRB to provide a retrospective approval during a period of lapse. Research studies that are allowed to be expired will be reported to the Vice Chancellor for Research and Graduate Studies, along with relevant other administration within the institution. The continuing review/closure form is located on our web site at [www.ecu.edu/irb](http://www.ecu.edu/irb) under forms and documents. The meeting dates and submission deadlines are also posted on our web site under meeting information. Please contact the UMCIRB office at 252-744-2914 if you have any questions regarding your role or requirements with continuing review.  
<http://www.hhs.gov/ohrp/humansubjects/guidance/contrev0107.htm>

### Required Approval for Any Changes to the IRB Approved Research

As a research investigator you are required to obtain IRB approval prior to making any changes in your research study. Changes may not be initiated without IRB review and approval, except when necessary to eliminate an immediate apparent hazard to the participant. In the case when changes must be immediately undertaken to prevent a hazard to the participant and there was no opportunity to obtain prior IRB approval, the IRB must be informed of the change as soon as possible via a protocol deviation form.  
<http://www.hhs.gov/ohrp/humansubjects/guidance/45cfr46.htm#46.103>

### Reporting of Unanticipated Problems to Participants or Others

As a research investigator you are required to report unanticipated problems to participants or others involving your research as soon as possible. Serious adverse events as defined by the FDA regulations may be a subset of unanticipated problems. The reporting times as specified within the research protocol, applicable regulations and policies should be followed.  
<http://www.hhs.gov/ohrp/policy/AdvEvtGuid.htm>

**Appendix B: Questionnaire**

Co-activation in Isokinetic Knee Extension 2011  
Preliminary Questionnaire

Name: \_\_\_\_\_

Age: \_\_\_\_\_

Do you regularly Exercise: Y N

If yes, when was the last time you exercised?

In what type of exercise do you participate? (Running, walking, cycling, resistance training etc)

How often do you exercise?

How Long have you been resistance training?

Personal Best:

Bench \_\_\_\_\_ Squat \_\_\_\_\_ Leg Press \_\_\_\_\_

Have you ever undergone any surgery on your lower extremities?

Untrained or Trained

**Appendix C: Data Collection Sheet**

Co-activation in Isokinetic Knee Extension 2010  
Data Sheet

Name: \_\_\_\_\_ Date: \_\_\_\_\_

Age: \_\_\_\_\_ Ht: \_\_\_\_\_ (m) Weight: \_\_\_\_\_ (kg)

Ball Test: R L

Training Hisotry:

Sport	Football	Track	Other	None
Training Type	Distance Run	Weight Lifting	Swimming	Cycling
	Walking	Rec. Activity	Other	
Frequency	1 day per week	2 days per week	3 days per week	>3 days per week
Personal Bests	100m/40 yrd Dash:	Bench:	Squat:	Leg Press:

Protocol Order:

Con: 30 \_\_\_\_ 90 \_\_\_\_ 150 \_\_\_\_ Ecc: 30 \_\_\_\_ 90 \_\_\_\_ 150 \_\_\_\_

**Appendix D: SPSS Tables**

Table 2: Statistical Data for Coactivation Ratio during Concentric Knee Extension

**Tests of Within-Subjects Effects**

Measure:MEASURE\_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Speed	Sphericity Assumed	31.179	2	15.590	1.677	.201	.081	3.354	.331
	Greenhouse- Geisser	31.179	1.743	17.887	1.677	.205	.081	2.923	.307
	Huynh-Feldt	31.179	2.000	15.590	1.677	.201	.081	3.354	.331
	Lower-bound	31.179	1.000	31.179	1.677	.211	.081	1.677	.233
Speed * Group	Sphericity Assumed	.576	2	.288	.031	.970	.002	.062	.054
	Greenhouse- Geisser	.576	1.743	.330	.031	.956	.002	.054	.054
	Huynh-Feldt	.576	2.000	.288	.031	.970	.002	.062	.054
	Lower-bound	.576	1.000	.576	.031	.862	.002	.031	.053
Error(Speed)	Sphericity Assumed	353.297	38	9.297					
	Greenhouse- Geisser	353.297	33.120	10.667					
	Huynh-Feldt	353.297	38.000	9.297					
	Lower-bound	353.297	19.000	18.595					

a. Computed using alpha = .05

**Tests of Between-Subjects Effects**

Measure:MEASURE\_1

Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Intercept	18224.047	1	18224.047	289.986	.000	.939	289.986	1.000
Group	888.936	1	888.936	14.145	.001	.427	14.145	.946
Error	1194.048	19	62.845					



Table 3: Statistical Data for Net Torque Production during Concentric Knee Extension

**Tests of Within-Subjects Effects**

Measure:MEASURE\_1

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>	
Speed	Sphericity Assumed	46732.441	2	23366.220	86.026	.000	.819	172.051	1.000
	Greenhouse-Geisser	46732.441	1.702	27460.553	86.026	.000	.819	146.399	1.000
	Huynh-Feldt	46732.441	1.950	23960.830	86.026	.000	.819	167.782	1.000
	Lower-bound	46732.441	1.000	46732.441	86.026	.000	.819	86.026	1.000
Speed * Group	Sphericity Assumed	1931.679	2	965.840	3.556	.038	.158	7.112	.626
	Greenhouse-Geisser	1931.679	1.702	1135.078	3.556	.047	.158	6.051	.575
	Huynh-Feldt	1931.679	1.950	990.418	3.556	.040	.158	6.935	.618
	Lower-bound	1931.679	1.000	1931.679	3.556	.075	.158	3.556	.433
Error(Speed)	Sphericity Assumed	10321.527	38	271.619					
	Greenhouse-Geisser	10321.527	32.334	319.213					
	Huynh-Feldt	10321.527	37.057	278.531					
	Lower-bound	10321.527	19.000	543.238					

a. Computed using alpha = .05

**Tests of Between-Subjects Effects**

Measure:MEASURE\_1

Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Intercept	1399808.667	1	1399808.667	206.289	.000	.916	206.289	1.000
Group	39820.858	1	39820.858	5.868	.026	.236	5.868	.633
Error	128927.745	19	6785.671					

Table 4: Statistical Data for Agonist Torque Production During Concentric Knee Extension

**Tests of Within-Subjects Effects**

Measure:MEASURE\_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Speed	Sphericity	34532.736	2	17266.368	53.520	.000	.738	107.040	1.000
	Assumed								
	Greenhouse-Geisser	34532.736	1.949	17715.861	53.520	.000	.738	104.325	1.000
	Huynh-Feldt	34532.736	2.000	17266.368	53.520	.000	.738	107.040	1.000
	Lower-bound	34532.736	1.000	34532.736	53.520	.000	.738	53.520	1.000
Speed * Group	Sphericity	170.451	2	85.225	.264	.769	.014	.528	.089
	Assumed								
	Greenhouse-Geisser	170.451	1.949	87.444	.264	.764	.014	.515	.088
	Huynh-Feldt	170.451	2.000	85.225	.264	.769	.014	.528	.089
	Lower-bound	170.451	1.000	170.451	.264	.613	.014	.264	.078
Error(Speed)	Sphericity	12259.327	38	322.614					
	Assumed								
	Greenhouse-Geisser	12259.327	37.036	331.012					
	Huynh-Feldt	12259.327	38.000	322.614					
	Lower-bound	12259.327	19.000	645.228					

a. Computed using alpha = .05

**Tests of Between-Subjects Effects**

Measure:MEASURE\_1

Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Intercept	1965578.286	1	1965578.286	145.822	.000	.885	145.822	1.000
Group	96581.333	1	96581.333	7.165	.015	.274	7.165	.719
Error	256105.936	19	13479.260					

a. Computed using alpha = .05

Table 5: Statistical Data for Antagonist Torque Production During Concentric Knee Extension

**Tests of Within-Subjects Effects**

Measure:MEASURE\_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Speed	Sphericity	1701.252	2	850.626	1.987	.151	.095	3.973	.385
	Assumed								
	Greenhouse-Geisser	1701.252	1.732	982.413	1.987	.158	.095	3.440	.356
	Huynh-Feldt	1701.252	1.990	854.852	1.987	.151	.095	3.954	.384
	Lower-bound	1701.252	1.000	1701.252	1.987	.175	.095	1.987	.268
Speed * Group	Sphericity	2673.633	2	1336.817	3.122	.056	.141	6.244	.566
	Assumed								
	Greenhouse-Geisser	2673.633	1.732	1543.929	3.122	.064	.141	5.406	.524
	Huynh-Feldt	2673.633	1.990	1343.458	3.122	.056	.141	6.213	.565
	Lower-bound	2673.633	1.000	2673.633	3.122	.093	.141	3.122	.389
Error(Speed)	Sphericity	16271.097	38	428.187					
	Assumed								
	Greenhouse-Geisser	16271.097	32.902	494.526					
	Huynh-Feldt	16271.097	37.812	430.314					
	Lower-bound	16271.097	19.000	856.374					

a. Computed using alpha = .05

**Tests of Between-Subjects Effects**

Measure:MEASURE\_1

Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Intercept	154591.394	1	154591.394	23.876	.000	.557	23.876	.996
Group	22949.108	1	22949.108	3.544	.075	.157	3.544	.432
Error	123019.876	19	6474.730					

a. Computed using alpha = .05

Table 6: Statistical Data for Hamstring Torque during Eccentric Knee Extension

**Tests of Within-Subjects Effects**

Measure:MEASURE\_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Speed	Sphericity Assumed	794.592	2	397.296	1.708	.195	.082	3.416	.336
	Greenhouse-Geisser	794.592	1.718	462.458	1.708	.200	.082	2.934	.310
	Huynh-Feldt	794.592	1.972	402.910	1.708	.195	.082	3.368	.334
	Lower-bound	794.592	1.000	794.592	1.708	.207	.082	1.708	.237
Speed * Group	Sphericity Assumed	223.684	2	111.842	.481	.622	.025	.962	.123
	Greenhouse-Geisser	223.684	1.718	130.186	.481	.594	.025	.826	.117
	Huynh-Feldt	223.684	1.972	113.423	.481	.619	.025	.948	.122
	Lower-bound	223.684	1.000	223.684	.481	.496	.025	.481	.101
Error(Speed)	Sphericity Assumed	8839.934	38	232.630					
	Greenhouse-Geisser	8839.934	32.646	270.785					
	Huynh-Feldt	8839.934	37.471	235.917					
	Lower-bound	8839.934	19.000	465.260					

a. Computed using alpha = .05

**Tests of Between-Subjects Effects**

Measure:MEASURE\_1

Transformed Variable:Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Intercept	1564161.795	1	1564161.795	122.768	.000	.866	122.768	1.000
Group	67321.685	1	67321.685	5.284	.033	.218	5.284	.588
Error	242074.666	19	12740.772					

a. Computed using alpha = .05

