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Torque Production of the Dorsiflexors During Lengthening Contractions at Different Velocities in Young and Old

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Supervisor: Dr. Anthony Vandervoort, The University of Western Ontario A thesis submitted in partial fulfillment of the requirements for the Master of Science degree in Kinesiology © Demetri Makrakos 2014

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TORQUE PRODUCTION OF THE DORSIFLEXORS DURING LENGTHENING CONTRACTIONS AT DIFFERENT VELOCITIES IN YOUNG AND OLD

(Thesis Format: Monograph)

by

Demetri P. Makrakos

Graduate Program in Kinesiology

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science

The School of Graduate Studies and Postdoctoral Studies The University of Western Ontario London, Ontario June 11^{th} , 2014

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ABSTRACT

The force-velocity relationship of skeletal muscle dictates that with increasing velocity of muscle shortening there is an exponential decrease in force production. However, during lengthening muscle actions, with increasing velocity there is a rapid increase then plateau in force. It is unknown whether ECC strength continues to increase at very-fast velocities $(>\!\!200^{\circ}\!/s).$ **PURPOSE:** To investigate ECC strength of the dorsiflexors over a large range of velocities in younger and older men. **METHODS:** Isometric neuromuscular properties (voluntary & electrically evoked) were assessed at 40° of plantar flexion on a CYBEX dynamometer. Nine younger $(\sim 24y)$ and 9 older $(\sim 76y)$ healthy men performed, in random order, one isokinetic lengthening contraction over a 50 $^{\circ}$ range of motion (5 $^{\circ}$ DF-45 $^{\circ}$ PF) at ten angular velocities; 15, 30, 45, 60, 120, 210, 270, 300, 330, 360%. RESULTS: Participant voluntary activation was >99% in both older and younger. Older were 31% weaker than the young for ISO strength. At all velocities, old had a greater ECC:ISO ratio compared to young. Additionally, there was a velocity-dependence of strength in both groups, in which absolute ECC strength increased as velocity increased. Old and young ECC strength at 15% was 17% and 37% greater than ISO strength; while at 360% ECC strength was 49% and 88% greater, respectively. **CONCLUSION:** Both old and young had continuing increases in ECC ankle dorsiflexion strength as the velocity of the lengthening contraction increased. Furthermore, relative to isometric levels, the older group showed particularly enhanced torque production at the higher velocities of muscle lengthening.

Keywords: torque, force, velocity, eccentric, lengthening, dorsiflexors,

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CHAPTER 1: INTRODUCTION

1.1 BACKGROUND MUSCLE PHYSIOLOGY

Our muscles are what allow us to perform important everyday movements. The necessary force to achieve these everyday movements is provided by contractions of the muscles involved. A muscular contraction develops force through collective contribution of many working parts. Force development, in part, is generated by the myosin heads attaching (cross-bridge) and pulling on the actin filament (power stoke) [\(Huxley 1957\)](#page-44-0). Cross-bridges are formed during both static and dynamic muscular contractions, where a dynamic contraction can either be to produce force by the myosin pulling on the actin filament, or resist force by the myosin fighting being pulled apart from the actin. Therefore, there are three types of contractions; isometric, concentric, and eccentric. Isometric (ISO) torque is produced during a static contraction; the joint angle is fixed and not moving. Concentric (CON) torque is produced during muscle shortening, and has an inverse relationship with the angular velocity of the joint [\(Hill 1938\)](#page-44-1). Eccentric (ECC) force is produced during muscle lengthening, during which the muscle is resisting stretch, and tends to increase as velocity increases [\(Edman et al. 1978\)](#page-43-1) and plateaus at higher velocities [\(Reeves et al. 2005\)](#page-46-0).

It is known that torque is developed through the formation of cross-bridges and the initiation of power stokes. The number of cross-bridges contributing to and developing torque varies depending on how overlapped the thick and thin filaments are, where either too overlapped (short muscle length) or not overlapped enough (long muscle length) will result in less force production. Naturally, there is an optimal amount of thick and thin filament overlap for force production, making the length of the muscle (joint angle) highly influential on the maximal amount of isometric force the muscle is able produce. This relationship between muscle length and maximal isometric force production is called the force-length relationship [\(Gordon et al.](#page-43-2) [1966\)](#page-43-2).

Maximal concentric force is related to the velocity of the movement. As the force the muscle is required to produce increases, the maximum velocity of the movement decreases. This is called the force-velocity relationship [\(Hill 1938\)](#page-44-1). It is well known that more force can be produced isometrically than concentrically, and furthermore that one can produce more ECC force than ISO or CON force [\(Katz 1939;](#page-44-2) [Wilkie 1949\)](#page-47-0). This thesis investigates the maximum eccentric torque generated about the ankle joint at a range of velocities from slow to very fast.

1.2 TIBIALIS ANTERIOR

1.2.1 Anatomy and Function of the Tibialis Anterior

The tibialis anterior (TA) is found in the anterior compartment of the leg on the lateral side of the tibia. It is a circumpennate muscle originating from the lateral tibial condyle and inserting onto the base of the first metatarsal. The TA is innervated by the deep branch of the fibular nerve and is responsible for dorsiflexion and inversion of the foot. The fiber type composition of the TA is about 73% type I (slow twitch) and 27% type II (fast twitch) [\(Polgar et](#page-45-0) [al. 1973\)](#page-45-0). The TA is responsible for approximately two thirds (65%) of the dorsiflexor group [\(Holmback et al. 2003\)](#page-44-3).

1.2.2 Muscle Model of Choice

There are many factors that contribute to the TA being a highly suitable muscle to study. Nerve stimulation of the TA is made easy due to the common fibular nerve, posterior to the fibular head, running superficially. Studies have also shown that the voluntary activation (VA) is consistently high in the TA, meaning most subjects are able to fully activate the muscle with limited practice, eliminating effort and familiarization as a confounding variables [\(Klass et al.](#page-45-1) [2005,](#page-45-1) [2007;](#page-45-2) [Power et al. 2011\)](#page-46-1).

With regards to antagonist accessibility, nerve stimulation and EMG recording is quite feasible on the soleus (SOL). It is important to record EMG from both the agonist and antagonist muscle groups so we can decide whether changes in torque are attributed to changes in EMG or attributed to other mechanisms. Using the muscle of the leg makes this simpler than other areas of study on the body.

1.3 AGE RELATED CHANGES

As the neuromuscular system ages, there are changes that will inevitably occur. Muscle from older people (>~65y), on average, becomes slower and weaker [\(Power et al. 2013\)](#page-46-2). This change is most pronounced in CON and the least pronounced in ECC contractions, with CON having been shown to drop $30 - 40$ %, whereas eccentric contractions lose only $20 - 25$ % between the third and tenth decades [\(Porter et al. 1995;](#page-45-3) [Poulin et al. 1992;](#page-46-3) [Roig et al. 2010;](#page-46-4) [Vandervoort 2002\)](#page-47-1). Even though ECC is maintained more than CON, absolute force produced during both types of contractions is less in old compared to young, and there are many factors responsible for this change. The most important ones, for my purposes, involve the death of motor neurons [\(McNeil et al. 2005;](#page-45-4) [Power et al. 2013\)](#page-46-2), motor unit remodeling [\(Roos et al. 1997\)](#page-47-2) and the increase in passive elements [\(Kent-Braun et al. 2000\)](#page-44-4).

1.3.1 Motor Neuronal Death

To produce a movement, the brain sends a signal down the spinal cord to the motor neuron pool, which sends a signal to all of the muscle fibers it controls. The motor neuron and all of the muscle fibers it innervates is called a motor unit. As the neuromuscular system ages, some

motor neurons will eventually die, rendering the motor unit dysfunctional. This phenomenon has been studied using many different techniques to compare the number of functional motor units in young to old, one of those being the motor unit number estimate technique (MUNE). Using MUNE, it has been confirmed that the number of motor units decrease with age [\(Campbell et al.](#page-43-3) [1973;](#page-43-3) [McNeil et al. 2005\)](#page-45-4).

1.3.2 Change in Motor Unit Structure and Function

When motor neurons die, the muscle fibers that were innervated by it are now 'orphaned' and are no longer able to contract. However, this orphaned state doesn't always last indefinitely. Other motor units can connect with the orphaned muscle fibers and include them into their motor unit; this is called collateral reinnervation. This reorganization results in there being less motor units. There is a preferential loss of muscle fibers from fast motor units, as well as a decrease in fiber size of the remaining fibers. Both these factors result in smaller motor units with slower contractile properties (acting closer to slow twitch, type I muscle fibers). As the neuromuscular system ages, motor neuron death occurs and is followed by collateral reinnervation, leading to smaller motor units and slower contractile properties: see review by [Roos et al. \(1997\)](#page-47-2) and [Campbell et al. \(1973\)](#page-43-3). Slower contractile properties also mean slower cross-bridge kinetics, such that the rate of force development is slower in older adults [\(Hook et al. 2001\)](#page-44-5). This slower rate of force development contributes to the decrease in concentric force seen in older adults.

1.3.3 Passive Elements

There are both contractile and non-contractile components that are part of the musculotendinous unit and contribute to total force production. Contractile components involve cross-bridge based mechanisms actin and myosin, whereas non-contractile components are structural proteins that transfer force to the joints. When muscle is stretched, the passive

components resist and absorb part of the force. The resisting and absorbing during lengthening allows the muscle to resist loads and produce forces via a mechanism not active during isometric or concentric contractions. There is evidence suggesting that titin could be the mechanism not active during muscle shortening, and plays an important role in increasing force during lengthening [\(Herzog et al. 2012\)](#page-43-4). This property of passive components is in part how we are able to produce more force eccentrically. It has been found that older adults have an increase in passive element content [\(Kent-Braun et al. 2000;](#page-44-4) [Narici et al. 2003\)](#page-45-5) which could be the reason older adults have a better maintenance of ECC strength compared to ISO and CON.

1.4 LENGTHENING VELOCITY

Numerous studies have looked at the torque-velocity relationship during concentric contractions, many reaching velocities above 500°/s, whereas studies testing eccentric contractions mostly test at much slower velocities. We know that as a percentage of ISO torque, older adults are weaker concentrically and stronger eccentrically than young [\(Porter et al. 1995;](#page-45-3) [Poulin et al. 1992;](#page-46-3) [Power et al. 2012;](#page-46-5) [Vandervoort 2002\)](#page-47-1); owing to the maintenance of ECC torque in old. This maintenance of eccentric torque in old is consistent at both the lower and higher velocities of lengthening. Until recently, torque had only been recorded during lengthening velocities of around 200 \degree /s in large muscle groups [\(Babault et al. 2001,](#page-43-5) Poulin et al. [1992;](#page-46-3) [Roig et al. 2010\)](#page-46-4) as well as the dorsiflexors [\(Klass et al. 2005;](#page-45-1) [Porter et al. 1997;](#page-46-6) [Sasaki](#page-47-3) [and Ishii 2010\)](#page-47-3) of young and old adults. A more recent study has tested high velocities of lengthening [\(Pain et al. 2013\)](#page-45-6) but only in young and not in the dorsiflexors. To my knowledge, there has yet to be a published study looking at high velocity lengthening contractions greater than 200°/s in older adults.

The purpose of my study is to determine if there is a difference in torque production between young and old adults during lengthening velocities higher than 200°/s, thereby extending knowledge about the aging human motor system and the eccentric force velocity relationship. We expect the older adults will have a larger amount of torque production relative to isometric torque as lengthening velocity increases.

CHAPTER 2: METHODS

2.1 PARTICIPANTS

All young (n=9, 24.8 ± 3.2 y, 180.0 ± 6.9 cm, 82.7 ± 10.0 kg) and old (n=9, 76.0 ± 5.5 y, 175.3 ± 5.8 cm, 83.0 ± 9.4 kg) participants were instructed to refrain from any unaccustomed or strenuous physical activity for 24 h, alcohol for 6 h, and caffeine consumption for 2 h prior to testing. All participants were recreationally active and free from any known neuromuscular or musculoskeletal disorders. Young were recruited from the university population and old from a university run exercise group for older adults. Informed written consent was obtained from all participants prior to testing. Participants returned to our lab two days after this testing session for related testing not included in this thesis, and none reported any soreness as a result of the lengthening contraction protocol. This study was approved by The University of Western Ontario Health Science Research Ethics Board and conformed to the Declaration of Helsinki.

2.2 EXPERIMENTAL SETUP

A HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA) was used to test the dorsiflexors muscle during lengthening. The non-dominant foot was fastened to the ankle attachment footplate, and the ipsilateral thigh, just above the knee, was immobilized by the thigh stabilizer pad. If the non-dominant side was injured (knee or ankle) then the dominant foot was used (3 old, 1 young). The contralateral foot rested on the footrest attachment, and remained in the same position throughout the testing session. Participants were seated in a slightly reclined position, with the knee angle at 140° (where 180° is considered straight). The contralateral hip angle was 110° and the ipsilateral hip angle was 70° , due to the knee being raised and immobilized by the thigh stabilizer pad (see **Figure 1**). Proper knee and hip alignment was achieved through manual modification of the chair position and angles were measured using a

goniometer. The ankle axis of rotation was lined up with the rotational axis of the dynamometer. The foot was secured to the footplate by two Velcro straps around the distal portion of the foot. To reduce extraneous movements, a horizontal strap across the waist and two vertical straps across the chest were used to secure the participant. Ankle range of motion (ROM) was set at 45° dorsiflexion (PF) – 5° plantar flexion (DF); totalling for 50° ROM.

Figure 1: Experimental Setup

2.3 RECORDING

Electromyography (EMG) signals were collected using self-adhering Ag – AgCl surface electrodes (1.5 x 1 cm; Kendal, Mansfield, MA) arranged in a monopolar setup. The active electrode for the TA was placed over the muscle belly (7 cm distal from the tibial tuberosity and 2 cm lateral to the tibial anterior border) and the reference electrode over the distal tendinous portion of the TA just proximal to the level of the malleoli. The active electrode for the soleus was positioned 2 cm distal to the lower border of the medial head of the gastrocnemius and the corresponding reference electrode was placed over the calcaneal tendon (Power et al. 2012). The surface EMG signals collected by the electrodes were pre-amplified $(x100)$, amplified $(x2)$, band-pass filtered $(10 - 1,000 \text{ Hz})$, and sampled at 2500 Hz using Spike 2 software (Version 7.07, Cambridge Electronic Design Ltd). Torque, velocity, and position channel information was sampled at a rate of 2500 Hz, converted to digital format using a 12-bit analog-to-digital converter (model 1401 Power, Cambridge Electronic Design, Cambridge, UK) and using Spike 2 software (Version 7.07) a live feed was displayed for participants for feedback on a large computer monitor.

2.4 BASELINE MEASUREMENTS

Passive resting tension of the dorsiflexors and plantar flexors was measured after participant setup was complete and prior to any electrically evoked or voluntary contractions. Participants were asked to relax while passive resting tension was measured at 0, 10, 20, 30, and 40° PF. EMG was also monitored to make sure the participant was fully relaxed. The ankle was moved from $0 - 40^{\circ}$ PF in 10° intervals in a step-like pattern, returning to 0° after each 10° progression towards 40° PF, and pausing for 5-7 seconds at each of the five angles (see **Figure**

2). The torque value at each angle was recorded as the mean torque between 2-5 seconds after the ankle was moved to that angle.

For tibialis anterior stimulation, the bar electrode was positioned over the deep branch of the common fibular nerve, with one electrode posterior and one anterior to the fibular head. For soleus stimulation, the bar electrode was pressed into the popliteal fossa, up against and perpendicular to the medial tendon of the hamstring. Compound muscle action potentials (Mwaves) were measured from the tibialis anterior and soleus muscle to normalize agonist and antagonist EMG collected during subsequent voluntary efforts. Maximum M-waves were determined by gradually increasing the current until a plateau in M-wave peak to peak amplitude was reached; then the current was further increased by at least 15% to supramaximally stimulate, ensuring all motor axons were activated. The stimulator current remained at this intensity for the remainder of the testing session, and was the intensity used to assess voluntary activation. Mwaves were evoked electrically with a standard clinical bar electrode (Empi, St. Paul, Minnesota, USA) coated in conductive gel. A computer-triggered stimulator (model DS7AH, Digitimer, Welwyn Garden City, Hertfordshire, UK) set at 400 V provided the electrical stimulation using a pulse width of 100 µs.

A 3-5 s duration isometric dorsiflexion baseline MVC was performed to ensure there was sufficient time for subjects to reach peak MVC torque. During MVC, participants were provided visual feedback of the torque tracing on a computer monitor, and were exhorted verbally. Voluntary activation was used to determine the degree to which participants were capable of fully, or close to fully, activating their dorsiflexors and was assessed using the interpolated twitch technique. The amplitude of the interpolated twitch torque evoked during the peak plateau of the MVC was compared with a resting peak twitch torque $(P_t: 1 Hz)$ evoked 1 s

following the MVC when the muscles were relaxed. Percent voluntary activation was calculated as voluntary activation (%) = [1- interpolated P_t /resting P_t] x 100%. Values from the baseline MVC with the highest peak torque are reported.

Figure 2: Experimental protocol schematic showing torque recordings of the dorsiflexors. TA (tibialis anterior). SOL (soleus). Figure 2: Experimental protocol schematic showing torque recordings of the dorsiflexors. TA (tibialis anterior). SOL (soleus). MVC (maximal voluntary contractions). ITT (interpolated twitch technique). MVC (maximal voluntary contractions). ITT (interpolated twitch technique).

2.5 LENGTHENING VELOCITIES

The range of motion was increased by 5° in both directions for the lengthening contractions; from 0° PF – 40° PF to 5° DF – 45° PF. This change in ROM is due to dynamometer requiring more time during the faster velocities to accelerate up to the programmed velocity and decelerate near the end range of motion to stop the foot. If not changed, the faster velocities would be mechanically unattainable by the dynamometer. The 10 different velocities the muscle was lengthened at were 15, 30, 45, 60, 120, 210, 270, 300, 330, and 360°/s. Each velocity of lengthening was performed only once. Velocities were pseudorandomized such that the only consistencies between participants were that 330 and 360°/s were performed last. This was done so the participant performed eight lengthening velocities before attempting the two fastest ones, giving them practice, which is important given that the faster velocities are the novel part of this protocol (**Figure 2**).

Each trial consisted of a 1-2 second isometric contraction at 5° DF followed directly by a lengthening contraction as the foot moved at the programmed velocity through the ROM, ending at 45° PF. The participants were encouraged verbally for both the isometric and eccentric portions of the contraction. One minute of rest was given between each contraction.

2.6 Data and Statistical Analysis

Data were analysed off-line with Spike2 software after the testing session was complete. All position and torque channels were filtered in Spike2 with a $2nd$ order Butterworth filter. This effectively smoothed the torque tracings without changing the location or amplitude of peak torque, and reduced noise in the position channel. The peak torque value from the lengthening contraction was taken at the time during which the dynamometer arm was moving at the programmed velocity. At higher velocities acceleration and deceleration of the foot took up a larger portion of the contraction and if peak torque occurred during either of those phases, it was disregarded; peak torque at the programmed velocity was taken instead. Using the absolute value of peak torque, relative peak torque was calculated as: ((ECC torque / MVC torque) – 1) x 100. Once the peak torque value was chosen, the position channel was used to determine ankle angle at time of peak torque.

The torque passively applied to the dynamometer arm at 0° PF was used as the reference for 10, 20, 30 and 40°PF. The difference between passive torque at 0°PF and passive torque at 10, 20, 30 or 40°PF was considered the amount of passive tension in Nm. This difference was then divided by ISO torque, to be presented as a percentage of ISO.

The EMG was processed as root mean square amplitude (RMSamp) for both TA and SOL. For analysis of EMG, time 0 represents the start of lengthening; where negative time is during the MVC prior to lengthening and positive time is during lengthening. The time intervals chosen for analysis were - 0.5 seconds to 0, 0 to 0.18, and 0 to time of peak torque. In the faster velocities this 0.18 seconds would represent a greater portion of the lengthening contraction, where as in the slower velocities this would represent a smaller portion (**Figure 3**). The reason the interval of 0.18 seconds was chosen is because that's the amount of time it takes to reach peak torque at 360°/s. I wanted to compare EMG leading up to peak torque while controlling for time, so 0.18 was the maximum amount of time I could compare EMG across the entire range of velocities; i.e., at faster velocities any EMG recordings past 0.18 seconds would be after peak torque has already been achieved.

SPSS software (version 20, SPSS Inc. Chicago, IL) was used for all statistical analyses. Un-paired t-tests were performed for all baseline measurement comparisons between young and old. A two-way analysis of variance (ANOVA) with repeated measures was performed to compare peak torque, ankle joint angle, and EMG during lengthening between young and old

across velocities (Age x Absolute torque, Age x Relative torque, Age x Ankle angle, and Age x EMG RMSamp). A two-way analysis of variance (ANOVA) with repeated measures was also performed to compare passive tension at rest between young and old across ankle angles (Age x Passive tension). The level of significance was set at $P < 0.05$. Post hoc analysis used un-paired t-tests with a modified bonferroni correction factor to determine where significant differences between factors existed. All data in tables are presented as mean ± standard deviation (SD), and all data in figures are presented mean \pm standard error (SE).

Figure 3: Raw torque, TA EMG, and velocity data at $30^{\circ}/s$ (A), $120^{\circ}/s$ (B), and $330^{\circ}/s$ (C). Cursors 1, 0, 2, and 3 represent 0.5 seconds before onset of lengthening, onset of lengthening, 0.18 seconds after onset of lengthening, and peak torque, respectively. In figure C, cursor 2 represents both peak torque and 0.18 seconds after onset of lengthening.

CHAPTER 3: RESULTS

3.1 BASELINE MEASUREMENTS

Baseline Measurements are all shown in **Table 1**. The old and young participants were separated by \sim 50 years of age, but not significantly different in height and weight. The isometric MVC was significantly different between old and young, with the old being 31% weaker than young, 29.1 ± 6.7 and 42.1 ± 8.5 respectively. Old and young had no significant differences in VA. All participants included in the data set, except one old (~ 96%), had a VA of >99%, therefore participants were able to voluntarily maximally activate their tibialis anterior and were giving a full effort. This allows us to assume most participants were able to give their full effort during the lengthening contractions as well.

The ITT showed that during the isometric MVC participants were near maximally activated, and EMG during lengthening was not different than during isometric; therefore I know participants were giving a full effort during the lengthening contraction.

Table 1: Participant characteristics and baseline measurements

There were 9 subjects in each group for all measures. Isometric maximum voluntary contraction (MVC). Voluntary activation (VA). Standard deviation (SD). * denotes significant difference between age.

3.2 LENGTHENING VELOCITIES

The absolute peak torque during lengthening was only significantly different between old and young at the three slowest velocities: 15, 30, and 45°/s. As the velocity of the lengthening contraction increased, the difference in absolute peak torque decreased and was statistically nonsignificant between old and young: at 15°/s and 360°/s old peak torque was 80% and 87% that of young, respectively (**Figure 4**). On the other hand, relative peak torque was significantly greater in old compared to young at all velocities. The old relative peak torque was approximately double the young at most velocities. At 15°/s young and old ECC peak torque was 17% and 37% greater than MVC torque, respectively, and at 360°/s young and old ECC peak torque was 49% and 88% greater than MVC torque, respectively. At most velocities, old had double the percent increase that young had (**Figure 5**).

Figure 4: Absolute peak torque during dorsiflexor lengthening over ten velocities. Data are expressed as group mean ± standard error. * denotes a significant difference between age (P < 0.05).

Figure 5: Peak torque during dorsiflexor lengthening as a percentage increase from isometric torque (shown on graph as 0), over ten velocities. Data are expressed as group mean ± standard error. * denotes a significant difference between age (P < 0.05).

When comparing old with young at the same angular velocity, there are no significant differences in ankle angle where peak torque was achieved. The mean ankle angle at peak torque of all trials, not controlling for velocity, is $27.0^{\circ} \pm 8.3$ for young and $25.2^{\circ} \pm 9.9$ for old (**Table 2**). Therefore, peak torque was achieved around 25°PF in both old and young. In the older group the passive tension at 20°PF and 30°PF, the angles surrounding the angle peak torque was achieved at, was 14.1% and 18.0% of ISO torque, respectively. In young the passive tension at 20°PF and 30°PF was 12.7% and 15.3% of ISO torque, respectively. At 0, 10, 20, and 30 passive tension in the older group was greater than the younger, but not significantly different. Passive tension was only found to be significantly different at 40°PF, where old had 6.1% greater passive torque than young (**Table 3**).

	Young		Old	
Velocity $(^{\circ}/s)$	Mean	SD	Mean	SD
15	29.9	9.7	26.4	9.9
30	28.3	9.8	27.9	13.8
45	29.5	6.9	26.2	11.9
60	29.6	8.3	26.1	12.0
120	32.1	9.1	27.8	10.6
210	26.6	9.3	25.3	9.3
270	22.5	9.6	24.4	11.2
300	23.5	7.4	21.0	9.6
330	22.1	4.1	20.7	4.2
360	25.7	3.0	26.0	4.3
Total	27.0	8.3	25.2	9.9

Table 2: Ankle joint angle at time of peak torque

Mean values are in degrees plantar flexion. 'Total' represents the mean of all ankle joint angles during peak torque regardless of velocity. Standard deviation (SD).

Ankle Angle (°PF)	Young		Old	
	Mean	SD	Mean	SD
$\boldsymbol{0}$	0.0	0.0	0.0	0.0
10	8.8	2.1	9.8	4.3
20	12.7	3.2	14.1	5.8
30	15.3	3.6	18.0	5.7
40	18.7	3.9	24.8*	6.7

Table 3: Passive Tension

Means are normalized to 0, and represent the percentage of MVC torque. Standard deviation (SD). Plantar flexion (PF)* denotes significant difference between age.

When comparing the RMSamp during the first 0.18 seconds of the lengthening contraction in old, the TA EMG was not significantly different across velocities. When comparing the RMSamp during the first 0.18 seconds of the lengthening contraction in young, the TA EMG was only significantly different between the 30 (4.9 \pm 2.7) and 330°/s (5.2 \pm 2.6) conditions. SOL EMG was consistent across velocities for the 0.18 second interval in both old and young.

When comparing the RMSamp during the whole lengthening contraction in old, the TA EMG was only significantly different between 45 (5.5 \pm 2.9) and 60 \degree /s (5.0 \pm 2.6), and 60 (5.0 \pm 2.6) and $120^{\circ}/s$ (5.6 \pm 3.1). When comparing the RMSamp during the whole lengthening contraction in young, the TA EMG was only significantly different between the 15 (5.6 ± 3.0) and $360^{\circ}/s$ (5.2 \pm 2.8) conditions. The SOL EMG in both old and young, like during the 0.18

second interval, was not significantly different across velocities when comparing the whole lengthening contraction. Therefore, neither agonist nor antagonist activation levels differed substantially among the various comparisons, and are not responsible for the torque related changes experienced across velocities and between age groups. **Figure 3** shows the time intervals from which the data was extracted and the comparisons made for EMG.

CHAPTER 4: DISCUSSION

4.1 MAJOR FINDINGS

One of the major findings in this study is that the older adults had greater increases than young in ECC torque, relative to MVC, as the velocity of the lengthening contraction increased. The old group had increases, at some velocities, twice as large as the young group. It was necessary to present the ECC torques relative to MVC because absolute values could be misleading. Old had significantly lower absolute torque at 0 to 45°/s. It is also noteworthy that there was a trend (**Figure 4**), in both old and young, for the torque to increase as the velocity of lengthening increased. This trend was independent of and uninfluenced by any other measure I collected. Motivation would not be an influence either as each participant was verbally encouraged in the same manner. Furthermore, the ITT measurements indicate that all participants were able to maximally activate their TA. EMG, in both groups and in both TA and SOL, is unlikely to explain changes in torque because EMG did not change as the velocity increased. The ankle angle that peak torque was reached at was similar between groups. Even if the angles were slightly different, there is essentially a plateau in isometric torque when stretching further than 10° PF [\(Marsh et al. 1981\)](#page-45-7); meaning a plateau in the torque length relationship. So even if there was a slight difference in the angle old and young were producing peak torque during lengthening, I could eliminate any active mechanisms of torque production as a possible contributor. I am unable to pin the results on any additional measures I collected, but there may be some mechanism at work during lengthening (discussed below), advantaging the older group and allowing them to produce a relatively greater amount of torque than young.

4.2 ISOMETRIC TORQUE

The way I was able to show ECC torque as being increased was by normalizing it to the participant's MVC. The main findings of this study are reliant on the MVC torque being an accurate measure. When comparing to like studies in the literature, this study's average value of 42.1 N•m for young MVC torque is similar to Klass et [al. \(2005\)](#page-45-1), [Power et al. \(2010\)](#page-46-7), and [Sasaki](#page-47-3) [and Ishii \(2010\)](#page-47-3) which were 43.5, 47.2, and 44.2 respectively. I also found this study's average value of 29.1 N•m for old MVC torque to be similar to [Baudry et al. \(2007\)](#page-43-6) and [McNeil et al.](#page-45-4) [\(2005\)](#page-45-4) which were 28.6 and 29.5 respectively.

4.2 ECCENTRIC TORQUE

Having shown that the MVC torque produced by both young and old is consistent with the literature, the next major concern is whether the ECC forces are similar to those achieved in other studies. The novel part of this study is that the high angular velocities old participants' muscles were lengthened at, to my knowledge, had not been reached previously; and in young, it has not been reached by many. What I can compare is whether the results at lower velocities of lengthening are consistent with other studies; if yes, I can extrapolate that the torque readings during the higher velocities of stretch are valid as well.

Studies already conducted in young show that dorsiflexors ECC torque at $100^{\circ}/s$ is approximately $30 - 40\%$ greater than ISO torque [\(Klass et al. 2005\)](#page-45-1). My results fall into this range; at 120°/s young ECC torque was 35% greater than ISO torque. There have been few studies testing young at higher velocities in the dorsiflexors. One study shows ECC to be 27% greater than ISO [\(Reeves and Narici 2003\)](#page-46-8) and another shows ECC to be 50% greater [\(Hasson et](#page-43-7) [al. 2011\)](#page-43-7); both at 200°/s. Other studies have tested young ECC torque at velocities close to and as high as the ones this study reached, but they are in different muscle groups. A recent study by

[Pain et al. \(2013\)](#page-45-6) investigated voluntary and stimulated ECC torque at 400 \degree /s in the knee extensors; they reported that voluntary ECC torque at 400°/s was about 20% greater than the ISO force, and the stimulated was about $50 - 70\%$ greater. It has been shown that the dorsiflexors are easier to fully activate than larger multi-joint muscle groups [\(Kent-Braun and Ng 1999;](#page-44-6) [Power et](#page-46-7) [al. 2010\)](#page-46-7), so they would act more like the stimulated condition; in which case the 50% increase at 360°/s in this study is consistent with Pain et al (2013).

When lengthening the dorsiflexors in old at 100 \degree /s, [Klass et al. \(2005\)](#page-45-1) found ECC torque to be 40% greater than ISO torque; similar to the 60% greater ECC torque recorded in this study. [Hasson et al. \(2011\)](#page-43-7) measured ECC torque at 200°/s in older adults and found a 40% increase from ISO torque. The ECC torque in this study at 200°/s was 75% higher than ISO torque for the older group. Thus my age-related effect is more pronounced than other studies in the dorsiflexors and even when I looked at studies testing old ECC torque at 200°/s in other muscle groups their ECC torque was still only about 40% higher than ISO torque [\(Hortobagyi et al.](#page-44-7) [1995;](#page-44-7) [Reeves et al. 2005\)](#page-46-0). To my knowledge, no study has examined old ECC torque at the higher velocities that this study lengthened at: $\geq 200^{\circ}/s$.

4.3 ELECTROMYOGRAPHY

One explanation for seeing a larger change in the older group's torque as velocity was increased may be as a result of changing neural drive to the muscles performing or resisting the movement. If the neural input to the TA and other DF muscles in old became relatively higher than young as the velocity of the lengthening contraction increased, then that could explain the larger amount of torque. Likewise, if the neural input to the antagonist plantar flexors in old was relatively lower than young as the velocity increased, then that could also help explain the larger torque increases.

However, the results of this study do not show any change in neural input as the velocity of the lengthening contraction changes: I did not find a change in either the TA or SOL EMG in young and old as the velocity increased. The old did have higher ECC torque as the velocity increased, so if neural input were to be a contributing factor, I would have seen a change in either the TA or SOL EMG as velocity changed. A similar study to this one, which measured ECC torque in young and old, found that the TA EMG when normalized to the M-wave was not different between groups and did not change across velocities [\(Klass et al. 2005\)](#page-45-1); agonist neural drive is therefore not considered to be responsible for the maintenance of ECC torque in old [\(Roig et al. 2010\)](#page-46-4). Unfortunately, [Klass et al. \(2005\)](#page-45-1) did not have antagonist data to speculate on SOL co-activation. A study that examined the elbow flexors of young and old that did record antagonist activation though reported both young and old to have similar levels of co-activation across velocities [\(Pousson et al. 2001\)](#page-46-9). Interestingly, it has also been shown that there is more antagonist activation during concentric compared to eccentric contractions [\(Kellis and](#page-44-8) [Baltzopoulos 1999\)](#page-44-8), an observation that points out the unique nature of eccentric muscle performance.

4.4 PASSIVE TENSION

The results of my study show that during a stretch of the musculotendinous unit at rest, older adults have more passive tension than young adults. A statistical difference occurred at the longer muscle length (40° PF), and there was a consistent trend for old to have more passive tension at 10°, 20°, and 30° PF compared to young. The passive tension measurement was obtained during a relaxed muscular state where the majority of the resistive torque produced by the muscle would be due to the non-contractile components, Therefore we should look at the non-contractile components of aged muscle units more closely. In animal muscle, passive tension is increased with age as a result of an increase of connective tissue in either the parallel or series elastic component [\(Alnaqeeb et al. 1984\)](#page-43-8). Studies in humans similarly suggest that the connective and non-contractile tissues in muscle increase with age [\(Kent-Braun et al. 2000\)](#page-44-4). We can assume the increase in passive tension I found in old was due to the increase of noncontractile elements and their contribution to the larger viscoelastic effect old would experience at longer muscle lengths [\(Power et al. 2013\)](#page-46-2). In the next section on potential mechanisms I explore the possibility that this increase in connective tissue and the non-contractile components could be responsible for the differences we see between young and old when comparing ECC relative to MVC.

4.5 POTENTIAL MECHANISMS

The amount of force generated by a stretched muscle is a combination of contributions from both the active components and the passive tissue components within it [\(Rassier et al.](#page-46-10) [1999\)](#page-46-10). When I measured passive tension at rest, the actin and myosin were weakly bound and not actively contributing to force production. However, prior to the lengthening contraction I had participants perform an isometric MVC, thus engaging the active components (actin and myosin cross-bridges). The MVC before lengthening would allow the active component to contribute to the overall total force production during lengthening. We know that older adults have an overall shift in muscle composition from fast twitch to slow twitch muscle fiber components. This alteration is one of the causes that leads to slower cross-bridge cycling in older adults [\(Hook et](#page-44-5) [al. 2001;](#page-44-5) [Miller et al. 2013\)](#page-45-8). It could be possible that thick and thin filament detachment takes longer as well if the myosin takes longer to attach to actin, maybe it takes longer to detach as well. We know twitch contractile characteristics, more specifically half relaxation time, slows with aging [\(Roos et al. 1999\)](#page-47-4). This prolongation means that the active enhancement of resistance

to lengthening, force generated by the cross-bridges, could be contributing to the ECC torque for a longer period of time in old compared to young. It is also understood that the muscle will produce less force near the end of its ROM, due to the amount of thick and thin filament overlap not being optimal for generating force. One might think that the peak torque occurred at a joint angle where the thick and thin filament were past the optimal angle for force production; however it has been shown that around 30° PF is where the dorsiflexors are strongest isometrically [\(Maganaris 2001\)](#page-45-9). It is important too that we recognize the [Maganaris \(2001\)](#page-45-9) study was isometric, because force generation isometrically requires the active components of muscle to form cross-bridges. The peak torque was reached at the optimal ankle angle for cross-bridge force to contribute to overall force, yet, there is still an increase from ISO to ECC torque. There must be an additional mechanism increasing the force even further during lengthening, as the active components are fully engaged and are already contributing as much force as possible.

As the length of a muscle increases, the contribution of the passive components (connective tissue and non-contractile elements) to the overall force production increases [\(Rassier et al. 2005\)](#page-46-11). Participants were reaching their peak torque at longer muscle lengths during high velocity, and therefore passive muscle elements had a greater opportunity to influence overall torque production. Furthermore, older adults have an increase in passive element content [\(Kent-Braun et al. 2000\)](#page-44-4) so older adults' torque during lengthening may be more affected by the involvement of the passive muscle components in muscle compared to young. Therefore, a conclusion was reached that the force enhancement seen during lengthening is likely due to additional resistance from passive structural elements [\(Edman and Tsuchiya 1996\)](#page-43-9). It is thought that the passive structural element that is mostly responsible for increases in force during lengthening is a structural protein called titin that might also be affected by a muscle's

activation history [\(Granzier and Labeit 2007;](#page-43-10) [Herzog et al. 2012;](#page-43-4) [Kellermayer and Granzier](#page-44-9) [1996b;](#page-44-9) [Monroy et al. 2012\)](#page-45-10). Increased levels of calcium have been shown to increase titin-actin interaction [\(Kellermayer and Granzier 1996a\)](#page-44-10). By having the participants perform a one to two second isometric contraction directly before lengthening, the muscle would have been flooded with calcium [\(Cannell and Allen 1984\)](#page-43-11), providing a good environment for titin-actin binding, thereby increasing resistance during lengthening.

4.6 SUMMARY

To my knowledge, ECC torque has not been compared between young and old adults at the high velocities of up to 360°/s that this study lengthened the dorsiflexor muscle group at. The ECC torque expressed relative to baseline MVC increased in old more than young as the velocity of the lengthening contraction increased. This difference cannot be explained by EMG, as the RMSamp of the agonist and antagonist muscles did not change in either young or old as the velocity increased, and the literature has shown similar results [\(Klass et al. 2005;](#page-45-1) [Pousson et al.](#page-46-9) [2001\)](#page-46-9). Passive tension at rest between young and old was found to be significantly different at the ankle position of 40° PF; with a consistent trend for passive tension to be higher in old at 10°, 20°, and 30° PF. The angle that young and old hit peak torque during lengthening was around 25° PF, and thus stretched connective tissue could be of influence. When comparing young to old, I found old to generate significantly more ECC torque production as a percentage of ISO torque at every lengthening velocity. I believe this finding is likely a result of greater noncontractile elements in the old muscle [\(Kent-Braun et al. 2000\)](#page-44-4), thereby increasing tension during lengthening. Of these passive elements, I recognize titin as one of the potential major

contributors responsible for generating the additional tension and resistance in older adults [\(Herzog 2014](#page-43-12); [Herzog et al. 2012\)](#page-43-4), resulting in the ability to produce more relative eccentric torque than young.

CHAPTER 5: LIMITATIONS AND FUTURE DIRECTIONS

5.1 LIMITATIONS

In my protocol, each of the 9 participants only performed one lengthening contraction at each velocity. This made for a total of 9 torque readings at each velocity for each age group. It would have been interesting to have collected data on more than one lengthening contraction per velocity per person. However, it would have been difficult to do this in one session of testing, due to the eccentric contractions potentially causing fatigue plus damage to the muscle and affecting further torque readings. I pilot tested two lengthening contractions per velocity during the same session, and the second contraction at most velocities was worse. Furthermore, at the higher velocities, which was the novel part of the study, the torque was much lower.

Another potential limitation is that I was using isometric torque produced at 40°PF to normalize the individuals' torque during lengthening, i.e., for calculating ECC relative to ISO values. In retrospect, isometic MVCs should have also been performed at 20 or 30°PF, because that is the ankle angle where most participants reached peak torque during lengthening. Using ISO torque at 30°PF to calculate ECC torque relative to ISO might have been a more precise comparison; that way I would be comparing ECC and ISO at the same ankle angle.

5.2 FUTURE DIRECTIONS

It would be interesting to take a biopsy from the TA of each participant and analyse the relative amount of non-contractile content versus muscle tissue for the young and old groups. A comparison could then be made between the non-contractile contributions to torque during lengthening, to see if the participants with the higher torque during lengthening also have a higher amount of passive elements in their muscle. The biopsy method needed to make this

comparison might be challenging because the TA has a relatively smaller diameter; however, it could still potentially be done in willing volunteers with good muscle definition.

My study only looked at young and old men. Another possible avenue would be to try this same protocol with women. There have been previous studies that compared eccentric torque at slower velocities between young and old groups of men and women [\(Baudry et al. 2007;](#page-43-6) [Holmback et al. 2001;](#page-44-11) [Porter et al. 1995;](#page-45-3) [Vandervoort et al. 1990\)](#page-47-5), but not at the speeds achieved in the current investigation. It would also be interesting to test people of different activity levels, and compare whether ECC torque in sedentary individuals is the same as active individuals.

The use of ultrasound imaging during muscle lengthening would also be another potential addition to my protocol in a future study. The main purpose of using ultrasound during the contraction would be to record changes in fasicle lengths and compare them to the increases in eccentric torque that my study found to be especially pronounced at high velocities of lengthening in older men.

CHAPTER 6: BIBLIOGRAPHY

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APPENDIX A: Ethical Approval Documentation

Use of Human Participants - Ethics Approval Notice

Principal Investigator: Dr. Charles Rice Review Number: 18097 Review Level: Full Board Approved Local Adult Participants: 100 Approved Local Minor Participants: 0 Protocol Title: Neuromuscular control of human movement Department & Institution: Anatomy & Cell Biology, University of Western Ontario Sponsor: Natural Sciences and Engineering Research Council

Ethics Approval Date: July 22, 2011

Expiry Date: August 31, 2015

Documents Reviewed & Approved & Documents Received for Information:

This is to notify you that the University of Western Ontario Health Sciences Research Ethics Board (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced study on the approval date noted above. The membership of this HSREB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request form.

Member of the HSREB that are named as investigators in research studies, or declare a conflict of interest, do not participate in discussions related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The UWO HSREB is registered with the U.S. Department of Health & Hunjan Services under the IRB registration number IRB 00000940.

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Research Contributions:

Summary: Articles Published: **2** Abstracts Published and Presented: **6** (presenting author: 3)

Articles Published in Refereed Journals

- 1. Power GA, **Makrakos DP**, Rice CL, Vandervoort AA. (2013). Enhanced Force Production in Old Age is Not A Far Stretch: An Investigation of Residual Force Enhancement and Muscle Architecture. *Physiological Reports Volume 1, Issue 1*.
- 2. Power GA, **Makrakos DP**, Stevens DE, Herzog W, Rice CL, Vandervoort AA (2014) Torque depression following muscle shortening in old and young men. *In Press, Experimental Gerontology*

Abstracts Published and Presented:

- *1.* **Makrakos DP**, Power GA, Stevens DES, Rice CL, Vandervoort AA. Need For Speed? The Velocity-Dependence of Eccentric Dorsiflexor Strength in Older Men. (2014). *American College of Sports Medicine*, Orlando, FL, USA. May 28th – 31st 2014. *Poster Presentation*
- 2. **Makrakos DP**, Power GA, Rice CL, Vandervoort AA. (2013). The Need for Speed: A Research Proposal on the Effects of Velocity During Eccentric Contractions. *Exercise Neuroscience Group*, Oshawa, ON, Canada. June $13th - 14th$ 2013. *Oral Presentation*
- 3. **Makrakos DP**, Power GA, Rice CL, Vandervoort AA. (2013). Residual Force Enhancement of the Knee Extensors in Older Adults: a Good Stretch! *American* College of Sports Medicine, Indianapolis, IN, USA. May 28th – June 1st 2013. *Thematic Poster Presentation*
- 4. Power GA**, Makrakos DP**, Stevens DES, Herzog W, Rice CL, Vandervoort AA. (2014). Shortening induced torque depression: Implications for power-loss in the elderly? *Proceedings of the XXIVth World Congress of Biomechanics*, Boston, MA, USA. July $6th - 10th$ 2014. *Invited Presentation*
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- 6. Paturel JR, Power GA, **Makrakos DP**, Vandervoort AA, and Rice CL. (2013) Age-related effects of isotonic and isokinetic fatigue when matched for peak power in knee extensors. *American College of Sports Medicine*, Indianapolis, IN, USA. May 28th – June 1st 2013. *Poster Presentation*