12-4-2012 12:00 AM

Neuromuscular Function Following Lengthening Contractions

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A thesis submitted in partial fulfillment of the requirements for the degree in Doctor of Philosophy

NEUROMUSCULAR FUNCTION FOLLOWING LENGTHENING CONTRACTIONS
(Spine title: Power-loss following muscle damage)

(Thesis format: Integrated Article)

by

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Graduate Program in Kinesiology

A thesis submitted in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy

The School of Graduate and Postdoctoral Studies
The University of Western Ontario
London, Ontario, Canada

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is accepted in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

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ABSTRACT

Unaccustomed lengthening contractions have been shown to impair muscle function - however little is known regarding this impairment on muscle power - specifically, the velocity component of power during voluntary contractions in humans. The four studies presented in my thesis investigated power-loss following lengthening contractions in healthy young and old women and young men.

The purpose of Study 1 was to determine reliability of velocity-dependent power of the dorsiflexors using the isotonic mode of the Biodex Dynamometer. I determined the isotonic mode is reliable and can be used to track changes in velocity and power following fatigue and lengthening contractions.

The purpose of Study 2 was to investigate changes in neuromuscular properties of the ankle dorsiflexors during and following repetitive lengthening contractions and throughout recovery in 21 (10 men, and, 11 women) recreationally active young adults (25.8 ± 2.3 y). The protocol for the following 3 studies involves subjects performing 5 sets of 30 lengthening contractions, with neuromuscular measures (i.e., electrically evoked twitch, tetanus, voluntary activation, voluntary contractions) recorded at baseline, during the task, and throughout recovery. Exercise induced muscle damage ultimately led to velocity-dependent (i.e., isotonic) power loss at a moderate load (i.e., 20% maximum voluntary strength).

Compared with isometric and isokinetic tasks, less is known regarding velocity-dependent muscle power and recovery in older adults following repeated lengthening contractions. In Study 3 we tested 9 old (68.3 ± 6.1 y) and 9 young women (25.1 ± 1.3 y). Old were more impaired following the task than young as shown by greater low-frequency torque depression at task termination leading to a more pronounced initial loss of power than young. However, power remained reduced in both groups during the 30 min recovery period. Older women were more susceptible to power loss than young following lengthening contractions likely owing to a greater fatigue response.

In Study 4, power curves were constructed [8 men (27 ± 3 y), 8 women (26 ± 4 y)] using various isotonic loads before and following task termination. There was a preferential loss of power at higher loads, with a relative maintenance of maximal shortening velocity shifting the power curve down and leftward. When stressed with heavier loads during dynamic contractions, force modulators arranged in parallel seem to be affected more by damage than those organized in series (velocity), which was highlighted by the attenuation of power at higher versus lower resistances.

The main findings of my thesis are that repetitive lengthening contractions fatigued and temporarily weakened the dorsiflexors, thus impairing their power producing ability immediately (i.e., fatigue + weakness) and longer term (i.e., weakness) owing to an inability to generate torque rapidly.

KEYWORDS

Muscle Damage, Shortening Velocity, Rate of Torque Development, Power, Sex, Aging, Eccentric, Isometric, Isotonic, Isokinetic

CO-AUTHORSHIP STATEMENT

This thesis contains material from published manuscripts (Chapters 2-4). On all manuscripts, Geoffrey A. Power was the first author and Brian H. Dalton, Charles L. Rice and Anthony A. Vandervoort were co-authors. William J. Booth was a co-author of Chapter 5. All experimental data presented in this thesis were collected, analyzed, and interpreted by Geoffrey A. Power.

ACKNOWLEDGMENTS

First and foremost I would like to thank my family for their unconditional love and support for all of my endeavors. Dad, your support whether it was driving to the races or asking how my school work is going keeps pushing me to reach the goals I set for myself and make you proud. Mom, I should probably blame you for my stubbornness or what has actually been key personality trait to my success in academia "tenaciousness"! Both of you have instilled in me many life lessons and taught me the meaning of hard work and I am grateful for that. Julie, you support me in: life, work and leisure all of which seem to overlap and mash into a whirlwind, sometimes I do not always see when extra attention is needed on the most important -life. Thanks for your understanding, love and support.

A special thanks to my Lab mates for helping with my experiments and providing critical feedback on my work. Specifically, Arthur, Brad, and Brian, you guys truly made this an enjoyable and memorable 4 years. Brian, I attribute much of my academic success to your guidance and help. Not only did you push me in the lab but were a fantastic training partner for cycling.

Charles, thanks for allowing me to work in your lab alongside your students. Your support through discussions and edits to my manuscripts contributed greatly to my productivity. I really like how you run your lab and this is something I will try to emulate one day. Your wit and humor made everyday a pleasure. With everything I have learned from you I will 'go boldly' into a future of research.

Tony, your supervision and mentorship has had a profound impact on my research and professional philosophies. I enjoyed our lengthy discussions which tended to meander from time to time but perhaps, this was a learning experience in itself to take a global perspective and not just get caught up in the minute details. I honestly do not think there is a better training environment than the one you provided me with, thank you.

"Only those who will risk going too far can possibly find out how far one can go."-T. S. Eliot-

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LIST OF ABBREVIATIONS

ANOVA – Analysis of variance

ATP – Adenosine triphosphate

Ca^{2+} – Calcium

CD – Contraction duration (TPT + HRT)

E-C coupling – Excitation contraction coupling

ES – Effect size

EMG - Electromyography

F-L – Force-length relationship

HRT – Half relaxation time of peak twitch torque

HSD – Honest significant difference

ICC – Interclass correlation coefficients

LFTD – Low frequency torque depression

LOA – Limits of agreement

MVC - Maximal voluntary isometric contraction

M-Wave – Compound muscle action potential

ROM – Range of motion

P_t – Peak twitch amplitude

P_d – Peak doublet amplitude

SD – Standard deviation

SE – Standard error

RMS – Root mean square

RTD – Rate of torque development

TA – Tibialis anterior muscle

TEM – Typical error

TEM_{CV} – Typical error expressed as a coefficient of variation

T-V relationship – Torque-velocity relationship

TPT – Time to peak twitch torque

VA – Voluntary activation

GLOSSARY OF TERMS

Angular Velocity – Change in angular position over time.

Force – A vector which has both magnitude and direction, the product of mass and acceleration.

Isokinetic – A dynamic muscular contraction in which the angular joint velocity is constant and the resistance (i.e., torque) is variable.

Isometric – A static muscular contraction.

Isotonic – The dynamic contraction mode in which a load is held constant and the joint angular velocity is variable as the limb moves through a range of motion.

Muscle Damage – Exercise induced dysfunction to the structure and function of skeletal muscle.

Neuromuscular Fatigue – Any exercise-induced reduction in the ability to generate torque or power regardless of whether or not the task can be sustained.

Power – The product of torque (N·m) and joint angular velocity (rad/s) expressed in watts (W); considered to be a more relevant measure of function because it incorporates both strength and contractile speed.

Torque – Also termed 'moment' is the product of the lever arm length, the magnitude of force vector, and the sine of the angle between the force vector and the lever arm vector, and is expressed in newton·meters (N·m).

Velocity-Dependent Contraction – Velocity-dependent contractions are those in which the imposed load remains relatively constant (i.e., isotonic-like) and the velocity is allowed to vary throughout the joint range of motion and is dependent upon the maximal effort of the subject.

Muscle Weakness – An inability to produce expected muscular strength.

Chapter 1– General Introduction

Skeletal muscle is a remarkable, highly organized tissue which regulates metabolic processes, is important in thermoregulation and ultimately serves as a 'molecular motor'. Muscles produce tension, pull on tendons and move bones to produce meaningful movement and locomotion. Not only do muscles pull and shorten, but when an external load overcomes the tension produced by the muscle, they lengthen actively. Lengthening muscle actions are a normal part of daily activity whether it be absorbing energy when landing from a jump or walking down a flight of stairs. For a given resistance, these contractions are less energetically demanding, cause less metabolic disturbance and generally produce greater forces than shortening or isometric contractions (2, 7, 35). Because of the greater tension associated with lengthening versus shortening or isometric contractions (28), and the high strain placed on the myofilaments, this contraction type is prone to causing muscle damage (46, 63). However, there is minimal knowledge regarding how unconstrained isotonic-like power (i.e., velocity-dependent power) production in humans is impaired following muscle damage, specifically the role of shortening velocity.

1.0 Mechanics of Lengthening Contractions

The first published investigation of lengthening contractions and muscle damage in humans was a study by Theodore Hough at the turn of the twentieth century (36). The simple study design involved participants contracting their finger against a

spring, thus shortening the muscle while stretching the spring and experiencing unaccustomed lengthening during the spring recoil. Following the task, Hough described a long lasting muscle pain which he distinguished from the short term transient pain of repetitive shortening or sustained isometric actions. Hough suggested the short term pain was associated with muscle fatigue and was due to the accumulation of metabolites while the long lasting soreness was caused ultimately by 'some sort of rupture within the muscle', which we now know as muscle damage. Indeed, lengthening muscle actions possess several unique features compared with those of other muscle actions, which lead to a greater susceptibility to muscle damage (4). First, based on the force-velocity (F-V) relationship (Figure 1) established by Katz (39), the force generated during muscle lengthening is 1.5-1.9 times greater as compared to isometric force (24). The F-V relationship dictates that, as a muscle shortens and velocity changes from zero, force generating capacity drops, owing to the decreased probability of interaction between the contractile proteins actin and myosin, and that muscle force also decreases as a function of shortening velocity (34). Conversely, during lengthening, when a muscle is stretched actively, muscle force is elevated above isometric and shortening muscle force due to a tighter packing of myofilaments increasing the contact area between actin and myosin effectively increasing bond formation leading to a firmer attachment of the cross bridge (24). As well, the engagement of passive force transmitting elements (25, 33) contributes to the elevated tension. Katz (39) observed a discontinuity in the F-V relationship during lengthening such that greater force was required for a given rate of stretch than for the same rate of

shortening. Additionally, following rapid stretching, the muscle became permanently weaker, and there was a shift in the optimal length of force production towards longer muscle lengths (39), suggesting the presence of damaged and overstretched sarcomeres (11, 32).

Furthermore, muscle activity as indicated via surface electromyography (EMG) is lower for maximal lengthening actions compared with isometric and shortening (10, 23, 26) contractions. Therefore, the combination of higher forces during lengthening and lower levels of muscle activity (i.e., less active muscle mass involved) places greater tensile strain on the involved remaining structures (26). Finally, force generation during lengthening differs from shortening whereby cross-bridges are broken mechanically rather than undergoing detachment by high-energy phosphates (ATP) (5). The forced detachment places greater strain on the myofilaments and contributes to muscle damage following lengthening contractions.

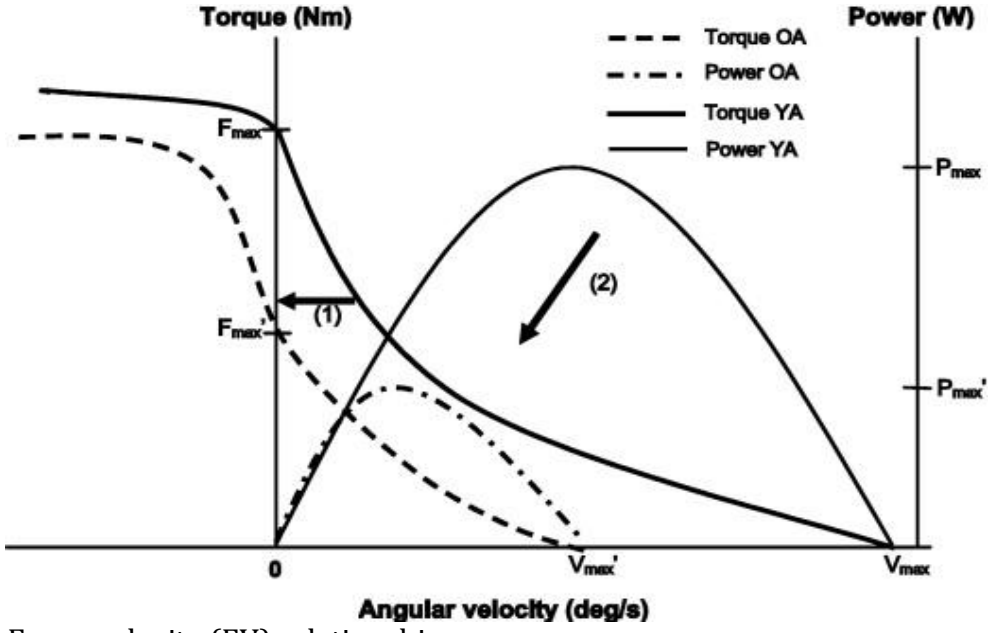

Figure 1. Force-velocity (FV) relationship

The angular velocity of movement is represented along the X-axis, with Vmax representing maximum unloaded movement velocity for a representative young and older adult. Torque and power are represented on the dual Y-axis, Fmax represents maximum voluntary torque and Pmax represents the finely tuned trade-off of angular velocity and torque to achieve peak power. Note the hyperbolic FV curve and power curve for the older adult is shifted left-ward (1) down (2) and relative to the young adult. *Adapted from Raj et al. Exp Gerontol 45: 81-90, 2010.*

1.1 Structural Changes Associated With Muscle Damage

Unaccustomed repeated lengthening contractions result in muscle damage (28). Evidence of structural and morphological changes to the muscle following lengthening contractions in humans came from Friden (29). Following unaccustomed lengthening contractions of the lower limbs, muscle biopsies were obtained from the vastus lateralis of participants. Analysis of the muscle tissue was performed via electron microscopy which identified disturbances to the ultrastructural milieu of the sarcomere (29). Damage to the sarcomere was

observed along the Z-line (Figure 2) which included: Z-line broadening, spreading of the Z-line material throughout the sarcomere, and non-uniform disturbed Z-lines throughout the fiber. These structural changes and disorganization of Z-lines contribute to impaired force production and transmission. Thus, during lengthening contractions when the muscle is under active strain over the descending limb of the length-tension (Figure 3) curve (46, 55), there is mechanical disruption of the actin-myosin bonds, and cytoskeleton of the muscle fibers. Ultimately, this damage results in a prolonged reduction in maximal voluntary force (6, 72).

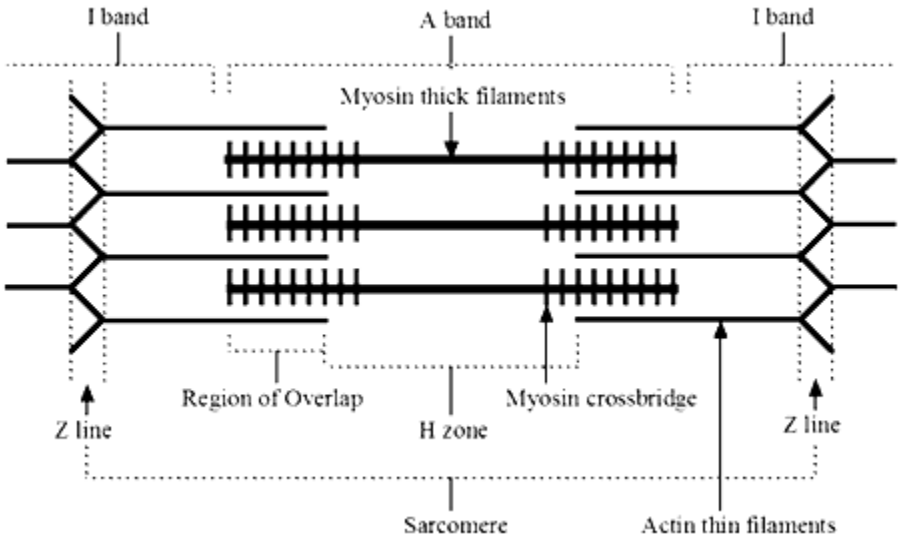

Figure 2. A schematic depicting the structure of an individual sarcomere**.**

1.2 Mechanisms of Damage Induced Force Loss

The extent of muscle damage induced force loss following lengthening contractions is determined by multiple factors, which include: the number of lengthening contractions, the initial muscle length (i.e., location on the force-length

(F-L) relationship; Figure 3), amplitude of stretch, the tension reached during stretch, and the contractile history (4, 18). Ultimately, force is reduced by a disturbance to the contractile machinery and failure to activate viable intact structures. Structurally, weakened and overstretched sarcomeres result in a shift in the peak of the F-L relationship to longer muscle lengths for optimal torque production (32). As well, sarcomeres stretched further along the descending limb of the F-L relationship may fail to re-interdigitate, thus producing lower force due to less thick and thin filament overlap (54, 55). Therefore, when isometric force is measured at the same muscle length prior to lengthening contractions and not the new optimal muscle length, force will appear to be reduced. The examination of isometric strength as a function of joint angle/muscle length reveals a disproportionate loss of strength at joint angles corresponding to short versus long muscle lengths (59). These findings lend support to the idea that longer muscle lengths are required to achieve the same myofilament overlap after muscle damage and hence one contributing factor to force loss after damage is an increase in series compliance as a result of overextended sarcomeres (11, 32).

Not only are muscle contractile structures damaged following lengthening contractions, but, impaired force production can also be attributed to failure of excitation-contraction (E-C) coupling. Excitation-contraction coupling is the cascade of events that begins with the transmission of an action potential along the sarcolemma and ends with the release of calcium (Ca^{2+}) from the sarcoplasmic reticulum and subsequent activation of the contractile machinery. Reduced Ca^{2+} release as a result of damage induced dysfunction to structural components

involved in E-C coupling, and reduced myofibular Ca^{2+} sensitivity (5, 37, 71) result in impaired force production capability (71). A reduced efficiency of the E-C coupling process is commonly observed following damage and in some cases (37) the failure to activate the contractile machinery following lengthening contractions contributes more (75%) than actual structural damage to the functional impairment/force generation.

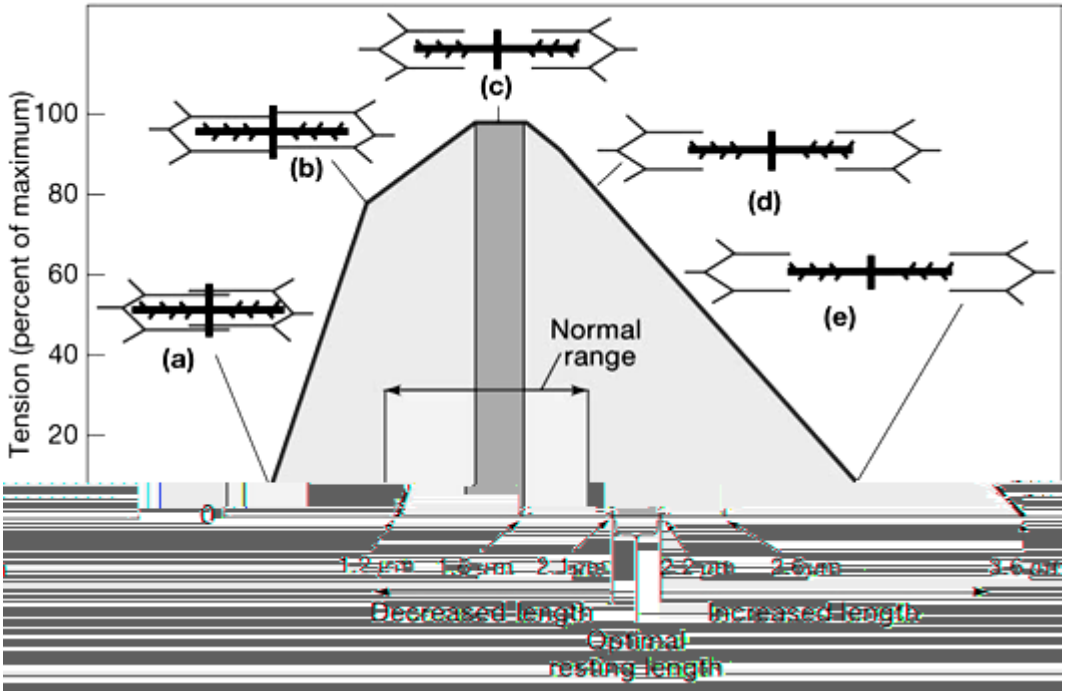

Figure 3. A schematic depicting the sarcomere length-tension (FL) relationship. At short muscle lengths, along the ascending limb of the curve, force is reduced due to too much overlap of thick and thin myofilaments. Optimal length is reached over the plateau region. On the descending limb, force is reduced owing to less interaction of myofilaments. *Adapted and modified from Martini (2001, p. 115.)*

1.3 Common Markers of Muscle Damage

The invasive nature of muscle biopsies, renders this technique less feasible to perform in some muscles to determine the incidence of muscle damage following lengthening contractions in humans. It has been suggested that hypercontracted fibers as evidence of ultrastructural damage might be caused by the biopsy procedure itself. Thus, the biopsy procedure can produce come changes mistaken for damage (18). Therefore, many non-invasive indirect measures of muscle damage are commonly used. The three most frequently used markers are: 1) subjective reports of muscle soreness, 2) blood protein assessment and 3) recovery of maximal voluntary muscle strength (18, 72). Maximal voluntary isometric torque generating capacity (MVC) is generally regarded as the best indirect measure of muscle damage and functional impairment following lengthening contractions in investigations on human subjects (72). Maximal voluntary torque is less impaired immediately following high-intensity lengthening actions than following shortening or isometric tasks (8, 43, 57). However, when assessed throughout recovery and day(s) later, MVC torque loss following shortening contractions recovers fully; whereas following lengthening contractions torque loss remains (68). Because the fatigue induced from repetitive contractions is transient and recovers relatively quickly compared with muscle damage, the incomplete recovery of both voluntary and electrically evoked torque cannot be attributed to fatigue. Thus, the remaining impairment in maximal torque capacity (i.e., MVC) represents muscle damage.

Self reported soreness and blood markers do not correlate well with measures of functional impairment. Muscle soreness and blood markers typically peak 48 hrs

following the initial insult to the muscle structures (13, 44, 53), during which time strength is already starting to recover. The mismatch between these markers and recovery of functional impairment does not lend support to the utility of soreness and blood markers as indirect measures of muscle damage. Thus, MVC performance is a relatively accurate and reliable measure, and provides the means for determining muscle function (12). The incomplete recovery of MVC torque following lengthening contractions suggests strongly that the muscle fibers are damaged (6). Nevertheless, an important issue in all studies of lengthening muscle actions is to distinguish between the reduction in force caused by fatigue and that caused by muscle damage (17). To corroborate results from an impaired MVC another useful measure in quantifying muscle damage is the shift in optimal angle of torque production to longer muscle lengths (62). The presence of overstretched, disrupted sarcomeres in series with still functional sarcomeres results in an immediate shift in optimum length of torque production to longer muscle lengths and is considered to be a reliable indicator of muscle damage (32, 62). This shift following lengthening contractions has been observed previously in the ankle dorsiflexors (45, 61).

1.4 Neuromuscular Function Following Muscle Damage

Optimal power generation is based on the finely tuned relationship between torque and velocity. As velocity increases, less torque can be generated owing to fewer cross-bridge attachments, requiring a trade-off of torque in favor of velocity to achieve peak power (3, 47). Following muscle damage, maximal isometric

dorsiflexion torque (8, 50, 57) is reduced although little is known regarding power loss. Voluntary maximal loaded shortening velocity is known to recover rapidly (< 5 min) in young adults after voluntary isometric and concentric fatigue tasks (15, 16). However, repeated lengthening contractions result in muscle damage which can take several days to recover fully (18), and it is unclear how this damage may affect velocity-dependent power production during short-term recovery. Up to now, the only available data of velocity-specific alterations in power in humans are based on studies involving isovelocity (i.e., constant speed/isokinetic) actions (12, 67). To determine the extent of concentric strength loss following muscle damage an isovelocity model relies specifically on testing the torque component of power when angular velocity is fixed and results from this paradigm are equivocal. Some report greater impairments at slow angular velocities, thus reflecting impaired torque generation (21, 52); whereas others report greater impairments at fast velocities suggesting shortening velocity is more impaired than torque generation (27, 29, 31). However, the isovelocity contraction mode constrains angular velocity artificially and therefore does not properly represent normal contractile function of the limb muscle model. Importantly, when torque is held constant and velocity can vary freely (i.e., velocity-dependent), the muscle functions more closely to *in vivo* conditions (60), and alterations in the power curve can be explored to offer insight on the mechanisms of power loss following muscle damage.

A loss of capacity to produce high torques rapidly (i.e., rate of torque development; RTD) would contribute much less to power production for lighter loads whereas at higher loads it would presumably impede power production

severely. In other words, because shortening velocity is related to the number of sarcomeres working in series whilst torque production is related to those sarcomeres in parallel (56), muscle damage would preferentially affect torque production, and would therefore result in a greater loss of power at heavier rather than lighter loads following muscle damage. This is an under studied but important area of research which needs further elucidation, and investigations of populations with specific characteristics as described below can help explain the role of shortening velocity and power loss following muscle damage.

1.5 Sex Differences in Response to Muscle Damage

In contrast to the literature on sex-differences following muscle damage in animals, reports on sex-related differences in response to damaging lengthening contractions in humans are equivocal, or show a greater impairment in women than men [for review see (18) and references therein]. Following lengthening contractions in a large sample of men (n=98) and women (n=94), Sayers & Clarkson (68) reported that a disproportionately higher number of women than men demonstrated greater initial force loss. In addition, despite similar indices of muscle damage in the elbow flexors of both sexes, Sewright *et al.*, (69) showed that immediate strength loss was more prominent in women than men. Women and men had similar markers of muscle damage, but women had a greater impairment in strength. This finding can be interpreted as E-C uncoupling playing a key role in the observed sex-difference. Additionally, muscle damage results in impaired RTD (9, 53), potentially diminishing power production. Thus, in women, muscle damage

induced dysfunction may be exacerbated due to a greater susceptibility to E-C coupling failure and lower RTD compared with men, owing to a lower Type II/Type I fiber area ratio (42).

1.6 Effects of Age on Muscle Damage

Impaired force generating capacity is a consequence of natural adult aging resulting from many factors (66) including: the loss of contractile muscle mass and motor units (22, 56, 70), decreased neural activation (1, 65), changes in muscle architecture (56) and excitation-contraction uncoupling (58). Because E-C coupling is compromised in older adults (58) and maximal unconstrained shortening velocity is indeed slower (19, 20, 51, 64) compared with younger adults, the old may be energetically disadvantaged during repetitive lengthening contractions. Therefore, older adults may experience a greater perturbation in ATP homeostasis, consequently exacerbating their fatigue response and resulting in a greater reduction in shortening velocity and subsequent velocity-dependent power following repetitive lengthening contractions than young adults. Moreover functional impairments following muscle damage have been previously attributed to impaired E-C coupling (71). A mechanical disruption of the link between the t-tubule and the sarcoplasmic reticulum impairing Ca^{2+} release (37) could further impair an already compromised system. Furthermore, dynamic concentric muscle performance following multi-joint lengthening contractions is known to be impaired (12, 67) although the underlying mechanisms are not understood entirely. Thereby, stressing a system which is already compromised in terms of E-C coupling and

shortening velocity will aid in understanding these physiological mechanisms of muscle damage on subsequent power loss.

1.7 Limb-Muscle Model

The ankle dorsiflexors were chosen as the model for my studies due to the many advantages of this particular muscle group. The fibular nerve is easily accessible at the head of the fibula for percutaneous electrical stimulation. The dorsiflexor muscle group's consistently high voluntary activation level, with minimal familiarization trials required, aids in comparing muscle damage studies between young and older adults (40, 41). The main dorsiflexor, the tibialis anterior (TA), contributes approximately 40-60% to dorsiflexion torque. The 40% value was estimated via focal tetanus to the TA, relative to fibular nerve stimulation (14, 49). However, based on the physiological cross-sectional area of the TA relative to the other dorsiflexors, Fukunaga et al. (30) suggest the TA contributes ~60% to dorsiflexion torque. The TA is a primarily slow twitch muscle, composed of ~76% (38) Type I muscle fibers. The dorsiflexors have a flat force-length relationship (14), reaching peak torque values over both the ascending and plateau regions (48). Therefore, age-related changes in fiber type and alterations in the F-L relationship should be of minor influence in interpreting the results.

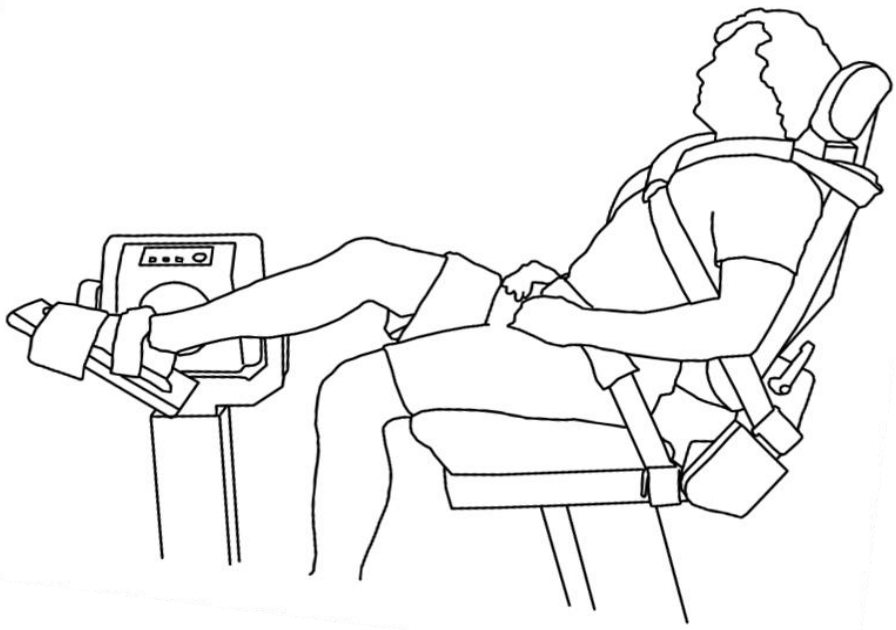

Figure 4. Participant positioned in the Biodex Multi-joint Dynamometer for testing of the ankle dorsiflexors. *Graphic art provided by Mr. Andrew Davidson.*

Testing was performed on a Biodex dynamometer (Figure 4), using the ankle attachment for dorsiflexion. All subjects were recreationally active and not systematically trained. The isotonic mode was used to perform 'velocity-dependent' contractions. A velocity-dependent movement is characterized by a participant producing a dynamic contraction as fast as possible with only minimal constraint in the angular velocity while the load or resistance is maintained at a pre-determined value (i.e., %MVC). Before the footplate is displaced during the velocity-dependent shortening contraction, the pre-programmed resistance has to be overcome by the participant. The dynamometer absorbs this increase in applied torque resulting in a directly proportional increase in angular velocity. This is in contrast to isovelocity actions (i.e., isokinetic) where the velocity is constrained and torque is recorded. However, the isovelocity contraction mode constrains angular velocity artificially and therefore does not properly represent normal contractile function of the limb muscle model. Importantly, when torque is held fairly constant and velocity can vary freely (i.e., velocity-dependent), the muscle functions more closely to in vivo conditions (60), and alterations in the power curve can be explored to offer insight on the mechanisms of power loss following muscle damage.

1.8 Purpose

Understanding the concomitant reductions in torque generating capacity and shortening velocity are important in elucidating the mechanism by which power production is reduced and neuromuscular function impaired following damaging lengthening contractions. Skeletal muscles are designed to modulate shortening

velocity based upon the load imposed (isotonic) and not vice-versa (isokinetic). The following series of investigations employed a velocity-dependent model which aimed to offer insight into the mechanisms of power loss following muscle damage. Reliable measures of strength and power output are critical for the assessment of neuromuscular function. Therefore, In Chapter 2 the purpose was to provide an initial assessment of the day-to-day reproducibility of shortening velocity and power variables, using the isotonic testing mode of the Biodex dynamometer.

Owing to the lower metabolic cost of lengthening contractions, but greater muscle damage compared with isometric or shortening contractions, it remains unclear whether velocity-dependent power loss is different between this type of exercise and repeated isometric or concentric contraction tasks. In Chapter 3, the purpose was to investigate the effect of high-intensity lengthening contractions on neuromuscular function and velocity-dependent power in young men and women. A secondary purpose was to explore further the equivocal observations in the literature regarding sex-related differences in muscle fatigue and responses to lengthening contractions. As an extension, the purpose of Chapter 4 was to investigate neuromuscular function in older and younger women with a particular emphasis on short-term recovery of velocity-dependent power following muscle damage. Finally, in Chapter 5, the purpose was to investigate velocity-dependent power loss following muscle damage, and to determine whether a sex-difference exists when assessed across multiple loads; stressing torque production and near maximal shortening velocities.

The hypotheses were that: 1) measurement methods for velocity and power following muscle damage will yield good reliability; 2) there will be a modest reduction in shortening velocity due to muscle damage, resulting in velocity-dependent power loss which will remain reduced throughout recovery in both men and women; 3) as a result of muscle damage levels that are comparable, MVC torque will be reduced similarly in both old and young women and remain reduced throughout a 30 min recovery period. However, when tested under dynamic conditions, older women will have a larger reduction in velocity-dependent power than the young owing to a greater impairment in E-C coupling and shortening velocity, which are known to be compromised in older adults and may not be observable during isometric testing. As a result of muscle damage neither group will recover by 30 min. 4) Because torque production is impaired considerably following muscle damage and the velocity at which a muscle shortens depends on the force it is resisting, it was hypothesized there would be a left and downward shift in the power curve, owing to a preferential loss of power at higher loads. However, maximal shortening velocity and shortening velocity at low loads will not be affected significantly owing likely to fewer cross-bridge interactions involved that do not stress the damaged force generators. Finally - to further highlight the role of muscle damage and impaired RTD - which is a putative major contributor to power production; I tested women, whom are known to have lower RTD than men. It was expected that following damage women will have a greater loss of power at heavier loads than men because of a greater strength loss driven by larger impairments in RTD and more reliance on the velocity component of power.

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Chapter 2 – Reproducibility of velocity-dependent power: before and after lengthening contractions[1]

2.0 Introduction

Reliability of isokinetic testing at various fixed angular velocities has been well established (6, 8, 25, 32). However, when muscle power is tested isokinetically shortening velocity is constrained artificially and does not provide a measure of muscle performance replicating daily activities, in which the load is fixed and velocity is unconstrained. A less common, but useful method used to determine power is to perform contractions under velocity-dependent conditions, whereby velocity is unconstrained and the contraction is performed at a pre-determined load. The Biodex Dynamometer can be operated in the isotonic mode to allow for a fixed resistance (i.e., % maximum voluntary isometric contraction (MVC)) and a variable unconstrained angular velocity (29, 30) dependent upon the effort of the subject. Because these contractions involve the acceleration of a constant load rather than measuring torque produced at a constant velocity, this mode may serve as a better tool than isokinetic measures during clinical, athletic and laboratory testing. Due to the recent increase in use of the isotonic mode for baseline normative measures (19, 33), training (30, 34) and fatigue studies (2, 3, 5, 18, 28) it is essential to establish the day-to-day reliability and utility of this measure.

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Power GA, Dalton BH, Rice CL, Vandervoort AA. Reproducibility of velocity-dependent power: before and after lengthening contractions. *Appl Physiol Nutr Metab* 36: 626-633, 2011.

When testing participants using the isotonic mode of the Biodex Dynamometer, the individual must first overcome the preset resistance throughout the range of motion, while any additional torque generated is translated into increases in velocity. Due to inherent mechanical limitations of the dynamometer (unable to maintain an exact constant external load), these contractions are not strictly isotonic and neither are they iso-inertial as the load is fixed (mechanically) and is determined by the constant braking of the dynamometer (14). Therefore, we have chosen to refer to these contractions as "velocity-dependent", in that these velocity-dependent movements involve an unconstrained angular velocity while the contraction is performed at a pre-determined load (i.e., %MVC).

The determination of strength and power under isokinetic conditions has been shown to be reliable (ICCs) in muscles about the ankle [0.61-0.96] (10, 11, 25, 26), knee [0.82-0.98] (8, 23, 24, 32), elbow [0.95-0.97] (16) and shoulder [0.60-0.95] (17, 20) joints, as well as during and following fatigue interventions [0.82-0.89] (23, 25). However, isotonic and isokinetic testing involve different mechanical constraints which are likely to necessitate altered neuromuscular strategies to perform each movement effectively (29, 30). Thus, reliability of the isotonic mode should be evaluated, and may result in different outcomes than isokinetic maneuvers.

Fatigue, defined as any exercise-induced reduction in muscle performance is task-dependent and multi-faceted (7), and thus should be assessed using a multitude of tasks in addition to the most common, isometric strength (3). Isometric and isokinetic tasks utilize torque as the index of fatigue whereas for

velocity-dependent contractions, velocity is the underlying parameter that largely reflects changes in power over time. Isokinetic contractions are limited by a constant fixed velocity and provide limited information regarding fatigue-induced alterations in shortening velocity, which ultimately is the major determinant of power-loss during daily activities with unconstrained velocities. It is of particular interest to explore the reliability of these measures to track group changes following a bout of unaccustomed lengthening contractions, which in addition to muscle fatigue are known to induce muscle damage (4, 21) and require a prolonged recovery (28) for the return of neuromuscular function. Because torque generation capacity is more impaired following damaging lengthening contractions than loaded shortening velocity (28), a moderately loaded contraction (i.e., 20% MVC) may provide a reliable day-to-day measure of muscle function following lengthening contractions such as those incurred during plyometric training.

The importance of accurately reproducing strength and power values is critical for the assessment of fatigue and training induced alterations in muscle function. Furthermore, the ankle dorsiflexors were chosen as the model of study due to this muscle group's consistently high voluntary activation with little need for subject familiarization (15). Therefore, the purpose of this investigation was to provide an initial determination of the day-to-day reliability of maximum shortening velocity and peak power in healthy young adults, using an isotonic testing mode and further the understanding of fatigue and recovery of shortening velocity following lengthening contractions.

2.1 Methods

2.1.1 Experimental approach to the problem: A group of healthy young men and women performed dynamic contractions on a Biodex Dynamometer operated using the 'isotonic mode'. Day-to-day reliability of velocity-dependent power (calculated at 20% MVC) was evaluated at baseline and following repeated high-intensity lengthening contractions. Data were collected approximately the same time of day on two separate testing sessions seven days apart. Intraclass correlation coefficients ($ICC_{2,1}$) with 95% confidence intervals were used to determine relative reliability, while absolute reliability measures included typical error (TEM) and typical error expressed as a coefficient of variation (TEM_{CV}). Bland-Altman plots were constructed to provide a visual representation of systematic bias and variability.

2.1.2 Subjects: Twenty four young men (n=10; 25.6 ± 2.9 y) and women (25.3 ± 1.8 y) from the university population volunteered for this study. The mean height and body mass of the men and women were: 176.4 ± 6.8 cm, 76.8 ± 7.8 kg and 166.9 ± 6.6 cm, 61.5 ± 10.7 kg, respectively. Participants were recreationally active and free from musculoskeletal disorders and were not involved in systematic resistance training of the dorsiflexors, or were competitive runners. This study received approval from the University of Western Ontario Review Board for Health Sciences Research Involving Human Subjects and conformed to the Declaration of Helsinki. Informed, oral and written consent were obtained prior to testing. Participants were asked to refrain from strenuous exercise 24 hr prior to the day of testing and to not consume caffeine on the day of testing.

2.1.3 Experimental set-up: A Biodex multi-joint dynamometer (System 3, Biodex(TM) Medical Systems, Shirley, New York) was used for testing and calibration was verified according to Biodex(TM) System 3 guidelines. All dynamic contractions were performed in the isotonic mode. The right foot was strapped tightly to the footplate with the ankle in line with the rotational axis of the dynamometer. Extraneous body movements were minimized using non-elastic shoulder, waist and thigh straps. Participants were positioned on the chair with hip and knee angles at ~110^{o} and ~140^{o}, respectively, and ankle angle at ~30^{o} plantar flexion. All isometric contractions were performed at 30^{o} of plantar flexion. Voluntary shortening contractions began from the plantar flexed position of 30^{o} and ended at the neutral ankle angle (0^{o}), thus moving through a 30^{o} range of motion. Before the footplate moved during the velocity-dependent shortening contractions, participants had to overcome the pre-programmed resistance. The dynamometer absorbs this increase in applied torque resulting in a directly proportional increase in angular velocity (29).

2.1.4 Procedures: Velocity-dependent contractions were performed at 20% of MVC. Pilot testing indicated that a 20% MVC load represents a moderate resistance in which the participant could perform fast shortening contractions without range of motion failure following high-intensity lengthening contractions. Three MVCs were performed for 3-5 s, with three min rest between all contractions (Figure 5). Participants were provided visual feedback of the torque, and exhorted during all voluntary contractions. To ensure MVCs were maximal, voluntary activation was assessed using the modified interpolated twitch technique (9).

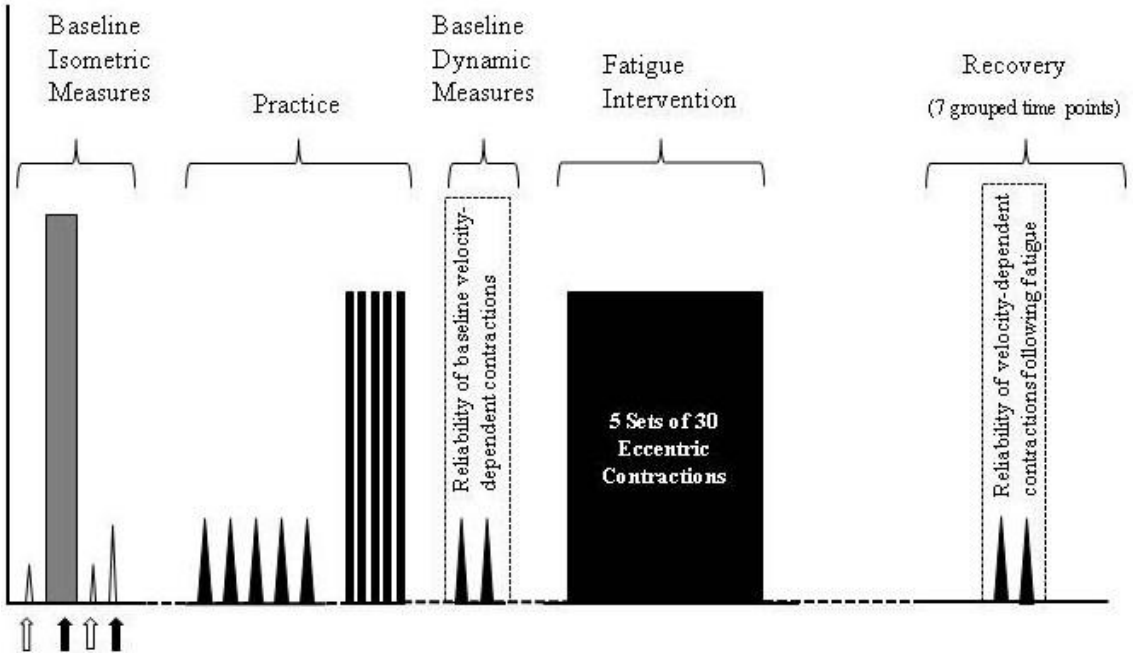


Figure 5. Schematic diagram of experimental protocol.

Baseline measures, a fatigue intervention and recovery measures were performed in the same order during two sessions separated by 7 days. Day-to-day reliability analyses were performed on peak velocity and power for the baseline velocity-dependent contractions and the recovery response of these measures following the fatigue intervention. Grey bars are maximum voluntary isometric contractions (MVC). Open triangles are electrically evoked contractions (twitch and twitch doublet). Open arrows indicate the stimuli of the electrically evoked twitches; and filled arrows are electrically evoked doublets. Filled profiles are dynamic contractions; fast velocity-dependent shortening contractions at 20% MVC (triangles), and dynamic lengthening contractions at 80% MVC (rectangles). Recovery time points: 30s, 2, 5, 10, 15, 20, 30 min.

Contractions of the tibialis anterior were electrically evoked using a bar electrode held distal to the fibular head over the deep branch of the common fibular nerve. A computer-triggered stimulator (model DS7AH, Digitimer. Welwyn Garden City, Hertfordshire, UK) was used with a setting of 400 V and a pulse width of 100 μs. The amplitude of the interpolated torque evoked during the peak plateau of the MVC (T_s) was compared with a resting twitch doublet torque evoked when the muscle was relaxed fully ~1 s following the MVC attempt (T_r). If the superimposed twitch doublet torque amplitude was visible during the MVC, the participant was encouraged further to perform an additional attempt until there was indeed minimal voluntary activation failure. Percent voluntary activation was calculated as voluntary activation (%) = $[1- (T_s/T_r)] \times 100$. Values from the MVC with the highest torque amplitude were used for data analysis.

Once MVC torque was determined to be maximal, the dynamometer was switched from the isometric to isotonic mode and a load equal to 20% MVC was programmed. The dynamometer was programmed to allow the footplate to return to 30^o of plantar flexion at the end of each shortening voluntary contraction while the participant relaxed fully. Familiarization with these 'fast' shortening contractions involved participants performing several (typically 5) velocity-dependent shortening contractions until a stable baseline value was obtained (no change in maximal shortening velocity). To ensure a maximal effort (peak velocity) contraction, all participants were instructed to move the load "as hard and as fast as possible throughout the entire range of motion". To assist participants in reaching their maximal shortening velocity, visual feedback of the velocity profile was

provided via a computer monitor, and a horizontal cursor was positioned at the previous personal best attempt. Participants rested for 3 min and then performed 2 consecutive contractions, the fastest was used to establish baseline values for maximum shortening velocity and peak power.

2.1.5 Lengthening contraction intervention: Because many natural movements are comprised of isometric, shortening and lengthening phases we challenged the system with an under-studied, but important dynamic task of lengthening contractions to explore reliability following fatigue in relation to velocity and power, and also uniquely during a period of recovery. Participants performed 5 sets of 30 lengthening dorsiflexion contractions with a load of 80% MVC and each set separated by 30 s. The contractions started at the neutral ankle angle (0^{o}) and ended at 30^{o} plantar flexion, thus moving through a 30^{o} range of motion. Participants were provided with visual feedback of velocity and instructed to resist while lowering the foot plate through the 30^{o} range of motion over a 1 s period ($\sim 30^{o}$/s). The foot was then returned to the neutral ankle position by the investigator over a period of 2 s. Following task completion on both day 1 and day 2 absolute peak velocity of the shortening contractions were determined from two contractions performed at each of seven time points throughout recovery; at 0.5 min, 2 min, 5 min, 10 min, 15 min, 20 min, and 30 min (Figure 5). The absolute peak velocity values from each of the seven recovery time points from day 1 and 2 were used to assess the reliability of the overall recovery response to the lengthening contraction protocol (see statistical analysis for specific measures) (27). This

allowed for a comprehensive analysis of the reliability of recovery following the intervention of lengthening contractions.

2.1.6 Data reduction and analysis: Torque, position and velocity data were sampled at 100 Hz and converted to digital format using a 12-bit analog-to-digital converter (model 1401 Power, Cambridge Electronic Design, Cambridge, UK). Spike 2 software was used to determine off line values for MVC torque, and voluntary maximum shortening velocity. Power was calculated as the product of torque (Nm) and the peak shortening velocity (rad/s) of the faster of two contraction attempts (as described above).

2.1.7 Statistical analysis: All statistical analyses were performed using SPSS software (version 16, SPSS Inc. Chicago, IL) and Microsoft Excel 2007 (Microsoft, Seattle, WA). Paired t-test analysis between day 1 and day 2 was performed to establish whether reproducibility bias was present for baseline measures. Reliability of baseline and recovery measures was assessed using the following statistical analyses. Bland-Altman plots were constructed to provide a visual representation of systematic bias and variability (1) by plotting the difference of day 1 and day 2 against the individual mean of day 1 and day 2 using either peak velocity or power at baseline and following the lengthening contractions. Reliability of maximum shortening velocity and peak power were assessed using the intraclass correlation coefficient $ICC_{2,1}$ which is based upon a repeated-measures ANOVA with testing session as the independent variable (31). The first subscripted number denotes the model (i.e., 2), selected because it is based upon repeated measures analysis of variance during which all participants are assessed by the same rater.

The second subscripted number signifies the form using either a single score (1) or the mean of several scores (2). The scores were peak absolute values (31). This model takes into account differences among participants, testing sessions, and error variance. Therefore, $ICC_{2,1}$ with 95% confidence intervals were used to determine the relative reliability across the 2 testing sessions of peak shortening velocity and power at baseline and following lengthening contractions. Measures of absolute reliability include: typical error (TEM), typical error expressed as a coefficient of variation (TEM_{CV}), and the limits of agreement (LOA) reflecting 95% probability limits between which the difference scores of day 1 and 2 should fall. Typical error (TEM) was calculated as the standard deviation of the difference score between the two days, divided by the square root of 2. Coefficient of variation of the typical error was calculated as the TEM divided by the average of all trials, multiplied by 100 (12). The LOA was calculated as the mean difference between the two days ± 1.96 x SD of the difference between the two days. Alpha was set at 0.05, and Table 1 is presented as means ± standard deviations (SD).

2.2 Results

Among participants, MVCs ranged from 24 to 66 N·m while individual scores were highly reproducible day to day, thus resulting in similar 20% loads (8.2 ± 2.2 and 8.3 ± 2.2 N·m) with which the loaded velocity-dependent shortening contractions were performed. The means and SDs for MVC, maximum shortening velocity, and peak power on day 1 and day 2 are presented in Table 1. There were no significant differences between day 1 and day 2 for any of these measures ($p >$

0.05). As well, voluntary activation was near maximal at baseline both days (99% ± 1%) and following (96% ± 5%; 95% ± 6%) the lengthening contractions both days ($p > 0.05$).

Intraclass correlation coefficients were calculated separately for men and women for maximum shortening velocity and power at baseline, however ICCs were not different between sexes for velocity [0.93 (men), 0.94 (women)] or power [0.97 (men), 0.98 (women)]. Thus, data were pooled to represent the reliability of velocity and power for both men and women for all subsequent analyses.

The differences between test day 1 and day 2 for maximum shortening velocity and peak power at baseline and following lengthening contractions are plotted against the average of the two testing sessions for each individual (Figure 6). The results from the Bland-Altman plots show the mean bias to be positive and relatively small for velocity and power measures indicating values were slightly higher on day 2, with fatigue data showing a greater bias towards a positive difference between the two testing sessions. For all Bland-Altman plots, the 95% limits of agreement were symmetric around the zero line, with a greater tendency towards asymmetry for the fatigue data.

Despite fluctuations in mean bias, the intraclass correlations for maximal shortening velocity and peak power at baseline (presented in Table 1) were classified as 'high' (27). The pooled recovery data over 30 min for maximum shortening velocity and peak power following the lengthening contractions also displayed high intraclass correlations for maximal shortening velocity and peak power.

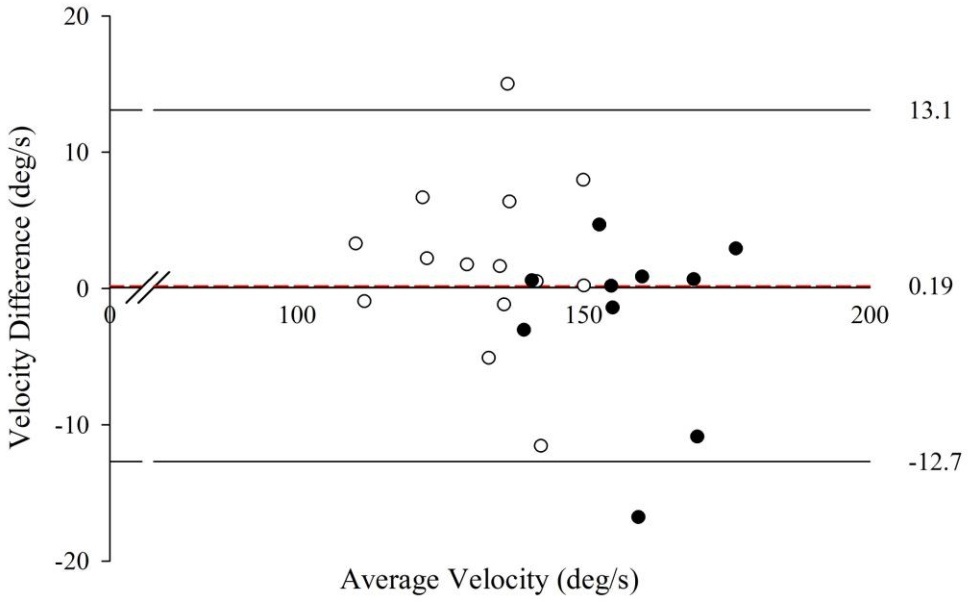
Absolute Baseline Measures and Reliability Statistics for Maximal Shortening Velocity and Peak Power

	MVC (Nm)	Velocity (deg/sec)	20% MVC (Nm)	Power (Watts)
Test Day 1	41.4 ± 11.5	143.2 ± 18.6	8.2 ± 2.2	21.1 ± 8.4
Test Day 2	41.2 ± 11.5	143.4 ± 16.8	8.3 ± 2.2	21.2 ± 7.5
	Baseline		**Recovery**	
	Velocity	**Power**	**Velocity**	**Power**
Mean difference	0.19	0.16	3.60	0.90
Between Measurement (*P*-value)	0.89	0.64	n/a	n/a
Intraclass correlation coefficient (ICC)	0.93	0.98	0.86	0.94
Lower confidence limit	0.85	0.95	0.82	0.93
Upper confidence limit	0.97	0.99	0.90	0.96
Typical error (TEM)	4.66	1.19	6.80	1.80
Typical error as a coefficient of variation (TEMcv)	3.25	5.63	5.20	8.70

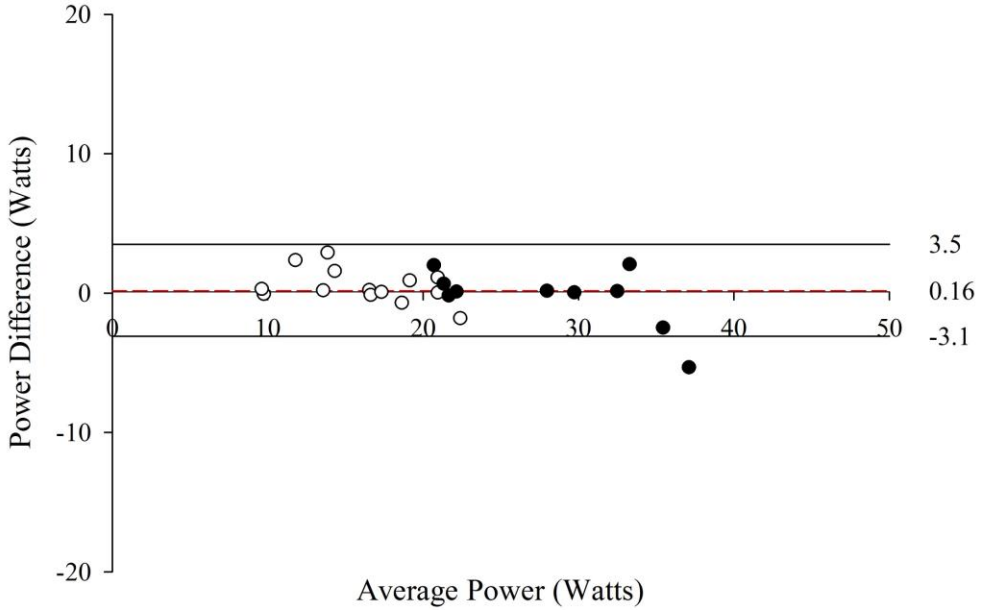
Table 1 Absolute baseline measures and reliability statistics for maximal shortening velocity and peak power.

Between measurement *p*-values are not reported for day-to-day recovery data because the data were pooled over the testing sessions and analyzed as a time effect of the fatigue intervention.

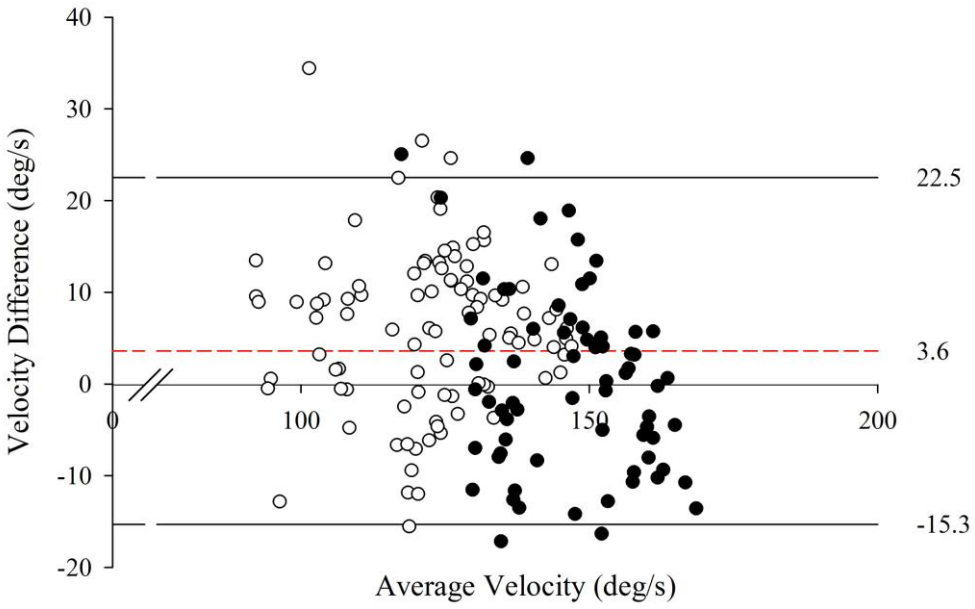
A.


20
10
0
-10
-20
Velocity Difference (deg/s)
0
100
150
200
Average Velocity (deg/s)
13.1
0.19
-12.7

B.


20
10
0
-10
-20
Power Difference (Watts)
0
10
20
30
40
50
Average Power (Watts)
3.5
0.16
-3.1

C.



D.

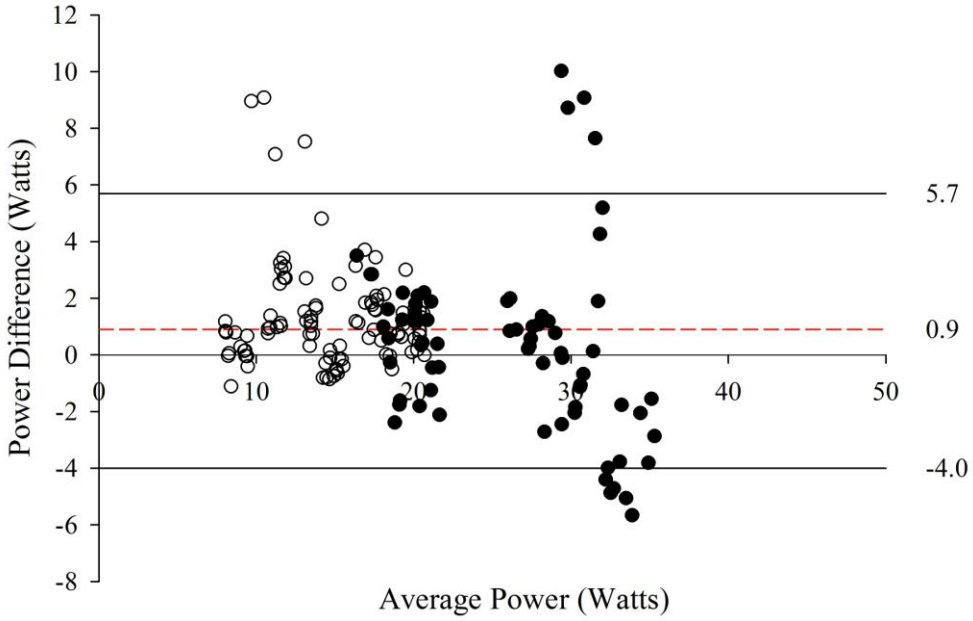


Figure 6. Bland-Altman plots

Maximum shortening velocity (deg/s) (**A**) and peak power (Watts) (**B**) at baseline and following the fatigue intervention (**C** and **D**), respectively, for women (open circles) and men (closed circles). The horizontal lines represent the mean bias (dotted line) and upper and lower 95% limits of agreement. The x-axis is the mean value of day 1 and day 2, and the y-axis is the difference score of day 2 – day 1.

Measures of absolute reliability for maximum shortening velocity and peak power are presented in Table 1. The typical error and coefficient of variation associated with shortening velocity was 4.66°/s and 3.25%, respectively. The typical error and coefficient of variation associated with peak power was 1.2 Watts and 5.63 %, respectively. Following the lengthening contractions, the typical error associated with shortening velocity was 6.8°/s and the coefficient of variation was 5.2%. The typical error associated with peak power following the lengthening contractions was 1.8 Watts and the coefficient of variation was 8.7%.

2.3 Discussion

This study analyzed the day-to-day reproducibility of maximum shortening velocity and velocity-dependent power with a load set at 20% MVC in healthy young adults before and after an intervention of repeated high-intensity lengthening contractions. Our findings demonstrate relative reliability (ICCs) to be 'high' at baseline and following lengthening contractions. Absolute reliability, as assessed, via coefficient of variation of the typical error for maximum shortening velocity and peak power resulted in an error of ~3% and ~9% at baseline and following the lengthening contractions, respectively. As suggested by Portney and Watkins (2000) intraclass correlations greater than 0.75 are considered to have good reliability. In this study, ICC confidence intervals for velocity and power at baseline ranged from 0.85-0.97 and 0.95-0.99, respectively. As well, following the lengthening contractions we obtained ICC confidence intervals ranging from 0.82-0.90 and 0.93-0.96 for velocity and power, respectively indicating high reliability.

The high reliability of this measure is encouraging and suggests the isotonic mode can be used in various settings to track group changes such as before and after training and following fatigue and lengthening contractions.

This study reported TEM and TEM_{CV}; these statistics provide an absolute and generalizable measure, respectively for comparisons of reliability between individuals of different strength and power. Typical error provides a reliability statistic free from the influence of correlations, as well; TEM_{CV} serves as a dimensionless measure which allows for the comparison across reliability studies using different testing protocols, participants and measurement tools (12). Here, lower values for TEM and TEMcv indicate high reliability. Velocity measures resulted in a TEM of 4.66°/s, and TEMcv of 3.25%, which suggest one would need a signal-to-noise ratio greater than ~5°/s and a 3.25% difference to observe a value that would not be associated with systematic error. Power measures resulted in a TEM of 1.19 Watts, and TEMcv of 5.63%, which suggest one would need a signal to noise ratio greater than 1.19 Watts and a difference of 5.63% to observe a value that would not be associated with systematic error. Visual analysis of the graphs and interpretation of the Bland-Altman analysis showed the mean absolute scores for maximum shortening velocity and power at baseline to be stable across day 1 and day 2. The mean bias for velocity (0.19°/s) and power (0.16 Watts) at baseline suggests there was no practice/learning effect from performing the previous bout. Adding the element of repeated lengthening contractions over time allows for potentially more error to affect the true score. There was however only a mean bias of 3.6°/s and 0.87 Watts for velocity and power, respectively, following the

lengthening contractions (Figure 6). The positive mean bias on day 2 following the lengthening contractions suggests there was less impairment in shortening velocity and power, thus individuals may have benefited slightly from the previous experience, such that, the muscle may have adopted a protective mechanism leading to less impairment in neuromuscular function during the second day of testing, commonly known as, the "repeated bout effect" (4, 22). For example, the muscle may have adapted to the previous bout of lengthening contractions with the addition of more sarcomeres in series (21) and thus, 'protected' from subsequent muscle damage during the second testing day one week later. However, the baseline values for maximum shortening velocity and peak power were highly consistent across days (Figure 6), suggesting the muscle had adequate time to recover from the previous bout of lengthening contractions.

In the present study, the ICC statistics were higher for power than velocity. This may be attributed to normalization of shortening velocity to a percentage of one's MVC (Power (W) = 20% MVC (N·m) x Velocity (rad/s). Reliability methods based on correlation coefficients, such as ICC, provide a measure of relative reliability. However, these reliability statistics are influenced by the range of values measured and give no indication of actual measurement values or systematic variability within the measure itself (12). Here, the ICC for maximum shortening velocity was 0.93 while the TEM_{CV} was 3.25%. Although, power had a higher ICC of 0.98 it was associated with more measurement error (5.63%), thus emphasizing the need for several statistical measures to evaluate reliability effectively. Using the isotonic mode, in which power is calculated as a percentage of MVC the additional

error can be attributed to day-to-day variability of the MVCs and hence emphasizes the importance of proper control measures to ensure a suitable maximal isometric effort is obtained prior to isotonic testing.

When performing velocity-dependent contractions strict care ought to be taken to ensure high reliability. First, the process of obtaining the isometric MVC must be controlled to achieve a maximal value; depending on the muscle group, this may require multiple familiarization attempts (9, 13). The current study investigated the ankle dorsiflexors because of the consistently high voluntary activation levels reported for this muscle group (15). Secondly, to obtain a maximal effort (peak velocity) during the velocity-dependent contractions and reduce the learning effect, participants were required to reach a consistent peak velocity (no change during five successive attempts) before performing baseline attempts. A fast, maximal effort can be achieved by providing the participant with visual feedback of the velocity profile and positioning a horizontal cursor at a previous personal best. These considerations help to minimize the likelihood of introducing systematic error into the measurement and ensures high reliability.

Holmback et al. (1999) investigated the isokinetic reliability of the ankle dorsiflexors of young men and women across a range of velocities (30 – 150°/s). The ICCs for peak torque when the participants were tested at 120 and 150°/s (similar to our isotonic velocities) ranged from 0.78-0.80 with a coefficient of variation of ~13% and a trend of increasing measurement error with increasing velocity. This is not surprising based upon a study of the mechanical reliability of the Biodex (6) which showed higher reliability values associated with slower

isokinetic velocities. In our study we found high reliability and minimal measurement error associated in determining power before and after lengthening contractions using the isotonic mode. But, it is unknown in young adults whether such reliability would be similar when performing isotonic contractions at other relative workloads which may dictate a faster or slower angular velocity, or place a greater or lesser demand on rate of torque development. Furthermore, with the increasing recognition of the isotonic mode for neuromuscular testing (2, 3, 5, 18, 28), reliability should be evaluated in other populations, such as elite athletes, individuals with athletic injuries or those with musculoskeletal disorders to ensure the utility of this testing mode.

These measures of relative and absolute reliability indicate velocity-dependent power is sufficiently reproducible when assessing baseline muscle characteristics (as in the case of a training intervention) and recovery following an intervention consisting of lengthening contractions. Acceptability of these values depends highly on the precision one requires to observe a meaningful difference. When investigating fatigue-induced changes following an exercise intervention or over the course of a training study, these day-to-day error fluctuations are relatively small and should provide reliable measures. To reduce the chance of introducing systematic error into the measurement when testing under unconstrained velocity conditions participants must be highly motivated and able to maintain high or at least consistent voluntary activation of the muscle group involved, and for some clinical populations this may require multiple practice contractions or separate familiarization days.

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Chapter 3 – Delayed recovery of velocity-dependent power loss following eccentric actions of the ankle dorsiflexors [2]

3.0 Introduction

Unaccustomed eccentric exercise is known to induce muscle damage and impair muscle function (19), although little is known regarding this impairment on concentric muscle power. Power loss is the result of fatigue-related reductions in both torque and shortening velocity, but the contributions of fatigue-related declines in shortening velocity to the reduction in power following eccentric exercise are unknown. Thus, our interest involves investigating the effects of repeated eccentric contractions on the ability of the muscle to generate velocity-dependent power.

Eccentric contractions are characterized by an external load overcoming the torque produced by the agonist resulting in a lengthening of the muscle. For a given resistance, these contractions are less energetically demanding, cause less metabolic flux and generally produce greater forces than concentric or isometric contractions (31, 32). This lengthening can place the muscle fiber under active strain over the descending limb of the length-tension curve (43, 46), resulting in mechanical disruption of the actin-myosin bonds, cytoskeletal damage, and a prolonged reduction in voluntary force evident in studies of both animals (24) and humans

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[2] A version of this chapter has been published via the *American Physiological Society*.

Power GA, Dalton BH, Rice CL, Vandervoort AA. Delayed recovery of velocity-dependent power loss following eccentric actions of the ankle dorsiflexors. *J Appl Physiol* 109: 669-676, 2010.

(50). Impaired torque production following eccentric exercise can be attributed to impaired calcium release as a result of damage induced dysfunction to structural components involved in E-C coupling (2, 59). As well, the increase in series compliance due to overstretched sarcomeres, leads to a shift to longer muscle lengths for optimal torque production (27) resulting in impaired torque production at the original muscle length.

The ability of a muscle to generate peak power is dependent on its maximum shortening velocity at a given load. Many factors contribute to maximum shortening velocity in an intact muscle, such as; rate of motor unit recruitment (58), muscle architecture (7) and fiber composition (28) (see Gordon (26) for review). Type II fibers generally produce ~4x greater power than Type I fibers. Additionally, it has been suggested that Type II muscle fibers are more susceptible to muscle damage than Type I (36), and damage may be more closely related to sarcomere length during contraction (12). Thus, the differences in fatigability following an eccentric fatigue task may depend upon the muscle group and type of contraction performed.

Neuromuscular fatigue, defined as any exercise-induced reduction in the generation of torque or power, can be manifested through both central or peripheral factors (22) and its analysis is further complicated by many influences such as species, sex and muscle fiber type differences whose interactive effect will depend on the type of contraction task utilized, muscle group involved and incidence of muscle damage (2, 6, 11, 12, 18, 19, 24, 34). The fatigue response to dynamic shortening contractions is similar between sexes (17, 54). However, in limited studies following eccentric fatiguing contractions women had greater

isometric strength loss compared with men (53, 55). Thus it is unknown whether isotonic power will be impaired differently between the sexes.

One limitation of previous studies is only using a measure of isometric torque (MVC) to assess fatigue following eccentric contractions, but because of task-specificity, MVCs may underestimate the functional deficit in muscle performance (49). Power, the product of both torque and velocity, may serve to exploit different mechanisms of fatigue to a greater extent than isometric torque. However, only isokinetic power has been reported following eccentric contractions (23, 44, 47) with modest reductions. Isokinetic measures are limited by a constant velocity with varying resistance which therefore cannot assess fatigue-induced alterations in shortening velocity following a task or exercise.

A less common method used to calculate power, but functionally relevant, are velocity-dependent contractions, in which the load is held constant and velocity varies throughout the range of motion and over time (15). Unlike impairments in force production capacity, the contributions of fatigue-related declines in shortening velocity to the reduction in power following eccentric exercise are unknown. Indeed, shortening velocity is known to recover fairly rapidly (< 5 min) after isometric and concentric contractions (15, 16), but repeated eccentric contractions result in disintegration and streaming of the Z-disks, disorganized myofilaments, and hypercontracted and overstretched sarcomeres (40), which could impair cross-bridge cycling, and hence, affect, to a greater extent, the production of shortening velocity.

Because of the lower metabolic cost of lengthening contractions, but greater muscle damage compared with isometric or shortening contractions, it remains unclear whether velocity-dependent power loss is lesser than, greater than, or similar to repeated isometric or concentric contraction tasks (16). Therefore, the purpose here was to investigate the effect of high-intensity eccentric contractions on neuromuscular function and velocity-dependent power in young men and women. We hypothesized there will be a modest reduction in shortening velocity resulting in velocity-dependent power loss which will remain reduced throughout recovery. A secondary purpose of the study was to explore further the equivocal observations in the literature about differences between the sexes in muscle fatigue and responses to eccentric exercise.

3.1 Methods

3.1.1 Participants: Ten young men (25.6 ± 2.9 y) and eleven young women (26.0 ± 1.7 y) from the university population volunteered for the study. The mean height and mass of the men and women were: 176.4 ± 6.8 cm and 76.8 ± 7.7 kg; and 164.8 ± 5.9 cm and 59.2 ± 10.1 kg, respectively. The study protocol was approved by the local University's Review Board for Health Sciences Research Involving Human Subjects and conformed to the Declaration of Helsinki. Informed, oral and written consent was obtained prior to testing.

Participants visited the laboratory on 2 occasions separated by seven days. All participants were recreationally active with no known neurological or

cardiovascular diseases. The first session was familiarization to the testing procedures, and the second during which data were collected. Participants were asked to refrain from strenuous exercise one day prior to testing and to not consume caffeine on the day of testing.

3.1.2 Experimental set-up: A Biodex multi-joint dynamometer (System 3, Biodex Medical Systems, Shirley, New York) was used for testing and calibration was verified according to Biodex System 3 guidelines. A footplate was attached to the dynamometer and positioned at an angle of approximately 45^{o} to the floor. The right foot was strapped tightly to the footplate with the lateral malleolus in line with the rotational axis of the dynamometer. Extraneous body movements were minimized using non-elastic shoulder, waist and thigh straps. Participants sat in a slightly reclined position with hip, knee, and ankle angles at ~110^{o}, ~140^{o}, and ~30^{o} plantar flexion, respectively. All isometric dorsiflexion contractions were performed at 30^{o} of plantar flexion. Concentric contractions began from the plantar flexed position of 30^{o} and ended at the neutral ankle angle (0^{o}). The eccentric contractions started at the neutral ankle angle and ended at 30^{o} plantar flexion, thus moving through a 30^{o} range of motion. The dynamic contractions were performed in the isotonic mode of the Biodex, thus allowing velocity to vary while providing inertia-free constant torque. In the isotonic mode, participants had to overcome the pre-programmed torque before the footplate would move during the concentric movements. Increases in applied torque were absorbed by the dynamometer and returned as a directly proportional increase in velocity (51). The isotonic mode is not by the proper definition strictly isotonic. The important point is that the load

(resistance) is essentially constant and velocity of movement can vary freely. This is useful when exploring the effect of velocity changes on movement and power. Therefore, throughout this paper we will refer to these contractions as velocity-dependent.

Surface electromyography (EMG) was collected from the tibialis anterior and soleus muscles using self-adhering Ag-AgCl electrodes (1.5 X 1cm; Kendall, Mansfield, MA). The skin was rubbed vigorously with alcohol prior to the application of the electrodes. A monopolar electrode set up was used with an active electrode positioned on the proximal portion of the tibialis anterior over the innervation zone (~7 cm distal to the tibial tuberosity and ~2 cm lateral to the tibial anterior border) and a reference placed over the distal tendinous portion of the tibialis anterior at the ankle. For the soleus the active electrode was positioned ~2 cm distal to the medial head of the gastrocnemius and a reference placed over the calcaneal tendon.

A computer-triggered stimulator (model DS7A, Digitimer. Welwyn Garden City, Hertfordshire, UK) provided the electrical stimulation of the dorsiflexors using a pulse width of 100 μs, 400 V, and current ranging from 20-95 mA. Contractions of the tibialis anterior were electrically evoked using a bar electrode held distal to the fibular head over the deep branch of the common peroneal nerve. Through palpation and careful observation we were confident there was no activation of the peroneal or plantar flexor muscles during the electrically evoked contractions.

3.1.3 Experimental procedures: Peak twitch torque (Pt) was determined by increasing the amplitude of the current until a plateau in M-wave amplitude was

reached (30-95 mA), followed by a further 10-15% increase in current to ensure supramaximal stimulation. This stimulation intensity was the same one used for doublet stimulation (two pulses at 10 ms interpulse interval) to assess voluntary activation. Next, 100 Hz peak torque (P100) was determined by increasing the current until there was a plateau in P100 (20-65 mA). A torque-frequency relationship was constructed using 1 s trains of the following frequencies: 1, 5, 10, 20, 30, 40, 50, and 100 Hz. Frequencies were delivered, in random order, at the current found to evoke P100 with 1 s between trains.

Then, 3 MVCs were performed of 3-5 s duration. Three minutes of rest was given between all contractions. Participants were provided visual feedback of the torque via near real time display, and verbally exhorted during all voluntary contractions. Voluntary activation was assessed during all MVCs using the modified interpolated twitch technique (29). The amplitude of the interpolated torque evoked during the MVC was compared with a resting twitch doublet torque evoked ~1 s following the MVC. Percent voluntary activation was calculated as voluntary activation (%) = [1- (interpolated twitch doublet/resting twitch doublet)] x 100. Values from the peak MVC were used for data analysis. Once MVC torque was determined, the dynamometer was switched from the isometric to isotonic mode. A load equal to 20% MVC was programmed into the Biodex and participants were instructed to perform practice concentric contractions (3-5 contractions) as fast as possible. The 20% MVC load represents a moderate resistance for dynamic contractions that all subjects could endure when it is important to have fast shortening contractions performed throughout the range of motion following a

fatiguing protocol. For example, at a load of approximately 60% of MVC many subjects cannot perform one concentric contraction through a full range of motion and the speed of movement is very slow. The Biodex was programmed such that the footplate was automatically returned to 30° of plantar flexion at the end of each concentric voluntary contraction. Following practice, two contractions were performed to establish values for peak shortening velocity at baseline.

3.1.4 Fatigue and recovery protocol: Participants performed 5 sets of 30 eccentric dorsiflexion contractions separated by 30 s, and performed with a load set at 80% MVC. Pilot testing showed 80% to be a compromise between very rapid fatigue but a sufficient contraction intensity to permit several contraction cycles to occur before achieving task failure. Participants were provided with visual feedback of velocity and instructed to resist while lowering the foot plate through the 30° range of motion over a 1 s period. The foot was then returned to the neutral ankle position by the investigator over a period of 2 s. The voluntary and electrically evoked responses of the dorsiflexors were recorded at: baseline, during the fatigue protocol, immediately following each of the 5 sets, and throughout the recovery period at 0.5 min, 2 min, 5 min, 10 min, 15 min, 20 min, and 30 min (Figure 7). Measures following the fatigue protocol included, and were performed in the following order: (1) maximum evoked twitch properties, (2) assessment of MVC and voluntary activation, (3) post-activation twitch and twitch doublet, (4) A measure of low frequency torque depression (10:50 Hz ratio), and (5) velocity-dependent concentric power.

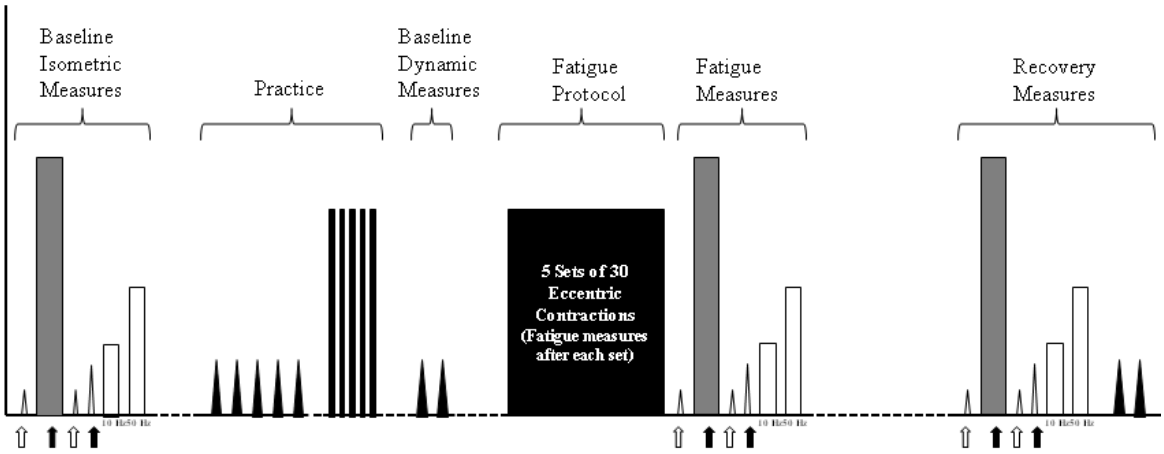

Figure 7. Schematic diagram of experimental protocol.

Grey bars are isometric maximum voluntary contractions (MVC). Open profiles are electrically evoked contractions (twitches (small triangles), doublet (large triangles), 10 Hz and 50 Hz (bars)). Filled profiles are dynamic contractions; concentric at 20% MVC (triangles), and dynamic eccentric contractions at 80% MVC (rectangles). Open arrows are electrically evoked twitches; and filled arrows are electrically evoked doublets. Recovery time points: Post (task termination), and at 0.5, 2, 5, 10, 15, 20, and 30 minutes.

3.1.5 Data reduction and analysis: Torque, position and velocity data were sampled at a rate of 100 Hz. All data were converted to digital format using a 12-bit analog-to-digital converter (model 1401 Power, Cambridge Electronic Design, Cambridge, UK). Surface EMG signals were pre-amplified (x100), amplified (x2) and band-pass filtered (10-1,000 Hz), and sampled online at 2500 Hz using Spike 2 software (version 6.10, Cambridge Electronic Design Ltd.). Surface EMG from the MVC was root mean squared (RMS) and values were used from a 1 s time period about the peak torque. All subsequent MVC RMS values were normalized to the level obtained during baseline. EMG was collected during the fatigue protocol from contractions 1-5, 13-17 and 25-30 of each set and averaged for each set. Peak RMS values of the raw surface EMG was calculated during the lowering phase through the 30^{o} range of motion and then normalized to the M-wave. Post-activation potentiation was calculated by comparing the twitch following the MVC to the baseline twitch. Power was calculated as the product of torque (N·m) and the peak shortening velocity (rad/s) of the fastest contraction attempt. Spike 2 software was used off line to determine M-wave amplitude, area, duration, the peak twitch torque (Pt), peak doublet torque (Dt), doublet time to peak twitch (DTPT), half relaxation time (DHRT) of the doublet, contraction duration (CD=DTPT+DHRT), doublet rate of torque development, and doublet maximum rate of relaxation. Low frequency torque depression was calculated using a ratio of peak 10 to peak 50 Hz evoked torques (10:50 Hz). To account for expected strength differences, all measures were normalized to baseline and presented as a percent change.

3.1.6 Statistical analysis: Using SPSS software (version 16, SPSS Inc. Chicago, IL) a two-way (sex x time) repeated measures analysis of variance was used to assess all neuromuscular data over time. Because voluntary activation values are not normally distributed, a Mann-Whitney U-test was employed to test for significance between groups. An unpaired t-test was used to assess group differences for subject characteristics. The level of significance was set at $p < 0.05$. When a significant main effect or interaction was present, Tukey's HSD post hoc test was performed to identify where significant differences existed. Tables are presented as mean ± standard deviation (SD), and figures as mean ± standard error (SE).

3.2 Results

3.2.1 Baseline measures: As expected, due to differences in anthropometrics, men had higher values for absolute measures of: peak twitch torque, MVC torque, velocity and power than women, ~ 49%, 30%, 16% and 38%, respectively (Table 2). When absolute values were compared, men were stronger than women ($p < 0.05$) at every stimulation frequency, but when the torque frequency curves (Figure 8) were normalized to 100 Hz torque there were no differences in the relationship between men and women ($p > 0.05$). Evoked torque corresponded to approximately 62% and 50% of MVC torque for the 50 Hz, and 64% and 52% of MVC torque for the 100 Hz, for the men and women, respectively.

Sex	Twitch Properties				MVC	VA	Velocity	Power
	Torque (Nm)	TPT (ms)	HRT (ms)	CD (ms)	(Nm)	(%)	(deg·s^{-1})	(W)
Men (n=10)	6.7 ± 1.8*	103.5 ± 20.1	107.6 ± 20.5	211.1 ± 19.8	47.2 ± 16.1*	99.4 ± 0.6	156.8 ± 12.2*	25.9 ± 9.0*
Women (n=11)	3.4 ± 1.0	115.5 ± 17.4	86.3 ± 28.9	201.8 ± 33.1	33.3 ± 6.9	99.8 ± 0.4	131.9 ± 13.5	15.9 ± 4.2

Table 2. Baseline contractile data.

Women had lower absolute evoked peak twitch torque, maximal voluntary isometric contraction (MVC) torque, maximum shortening velocity, and peak power than men (*$p < 0.05$). Voluntary activation (VA) was not significantly different ($p > 0.05$) between groups. Time to peak twitch (TPT), half relaxation time (HRT) and contraction duration (CD) of the twitch were not significantly different ($p > 0.05$) between groups. Mean ± SD.

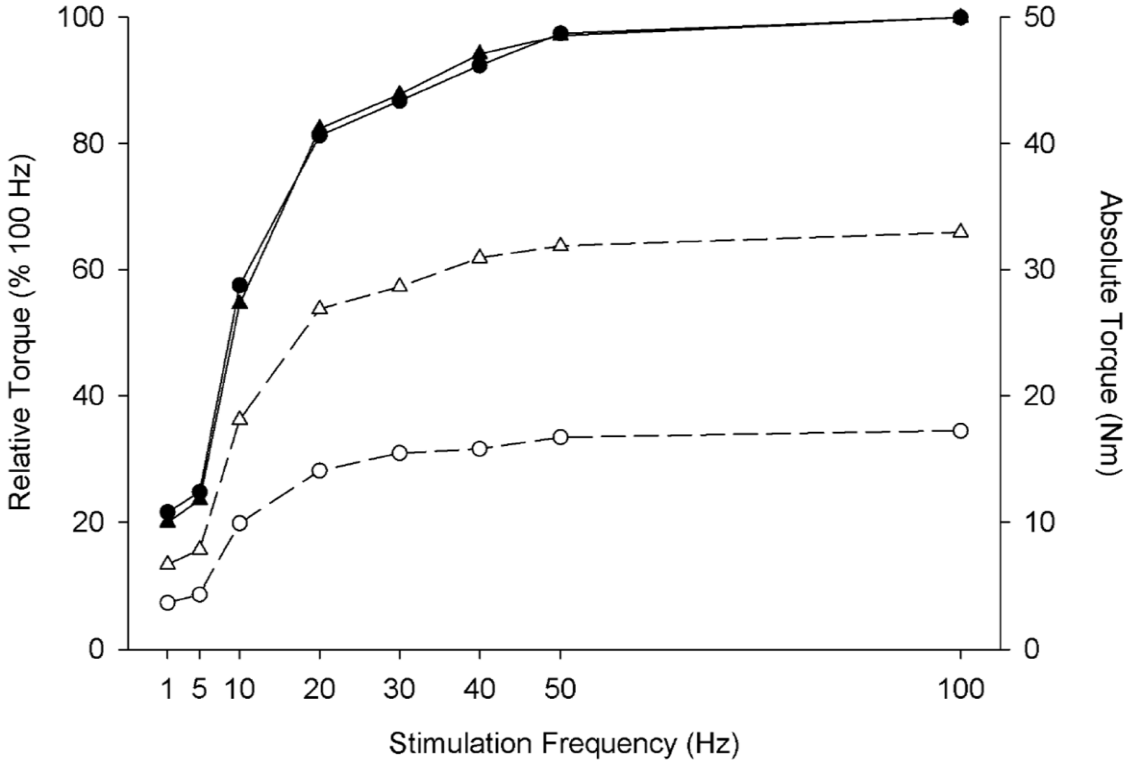

Figure 8. Torque-Frequency relationship.

Open triangle (men absolute torque), filled triangle (men relative torque), Open circle (women absolute torque), and filled circle (women relative torque). Men had higher absolute torques at all frequencies (1-100 Hz) compared to women (*p < 0.05). Relative torques were similar at all stimulation frequencies (* p > 0.05).

3.2.2 Fatigue and recovery measures: All participants were capable of completing the 5 sets of 30 eccentric contractions, although some subjects had difficulty lowering the foot plate at a constant velocity for the last few contractions of each set. This failure to maintain a constant velocity resulted in increased eccentric velocities which ranged from 37°/s to 41°/s. Despite the variation in velocity, the duty cycles were similar ($p > 0.05$) between men and women (0.32 ± 0.04).

When all neuromuscular measures were analyzed with regard to relative changes over time, no significant differences between men and women were found ($p > 0.05$). Thus, data were pooled and normalized to baseline for all subsequent analyses. Peak dorsiflexor MVC torque decreased to 85% of baseline ($p < 0.05$), following the first set of 30 eccentric contractions (Figure 9). The MVC torque progressively decreased following each successive set to 72% of baseline immediately following task termination and did not recover fully. There were no significant changes from baseline ($p > 0.05$) in RMS EMG of the agonist TA during MVCs, and voluntary activation was greater than 99% at baseline and did not change ($p > 0.05$; Figure 10) throughout fatigue and recovery. Conversely, soleus RMS EMG during MVCs increased ($p < 0.05$) to 111 ± 21% of baseline following the third set of eccentric contractions, resulting in a 13 ± 9% increase in the ratio of antagonist coactivation where it remained for up to 20 min recovery, but returned to baseline by 30 min. M-wave properties, including; peak-to-peak amplitude, duration, and area remained unchanged from baseline ($p > 0.05$). During the

eccentric contractions, RMS EMG of the agonist TA normalized to M-wave did not differ significantly ($p > 0.05$) among sets.

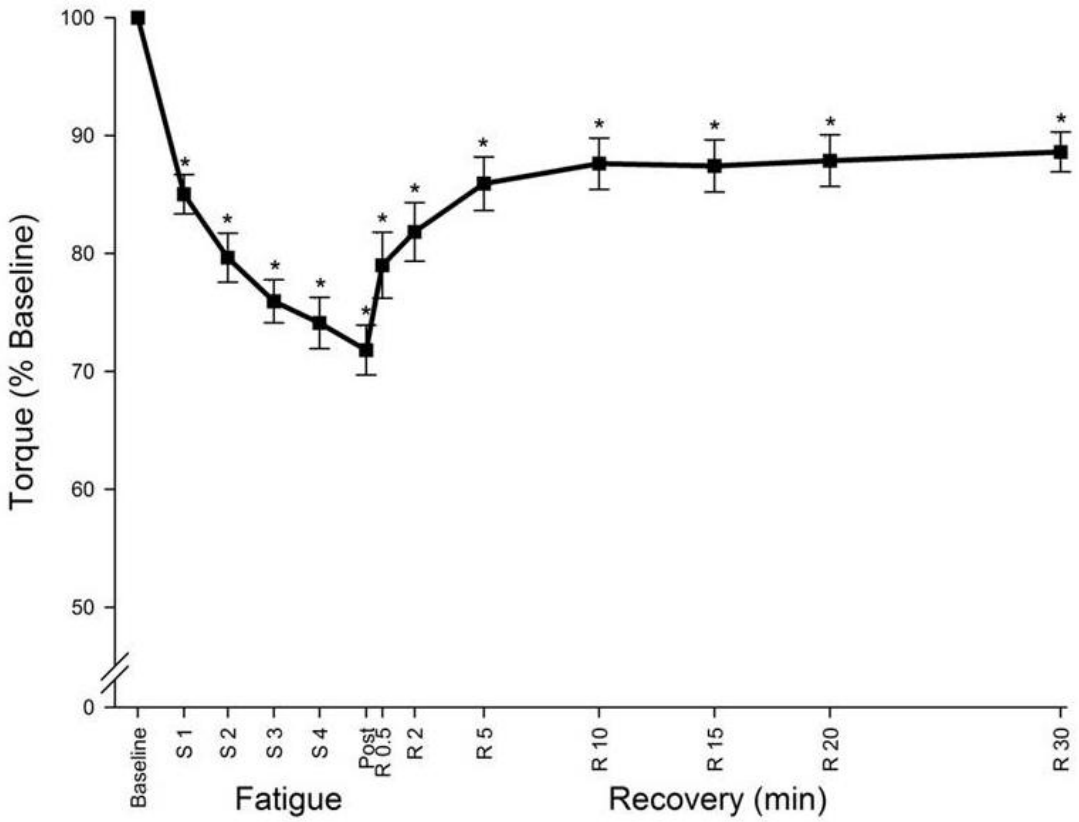

Figure 9. Maximum isometric voluntary contraction (MVC).

Maximal voluntary strength was reduced following the first set (S1) of eccentric contractions and continued to decline to ~70% of baseline at Post (task termination) and it did not recover fully (*$p < 0.05$) within 30 min. [S represents 'sets', R represents 'recovery']. Mean ± SE.

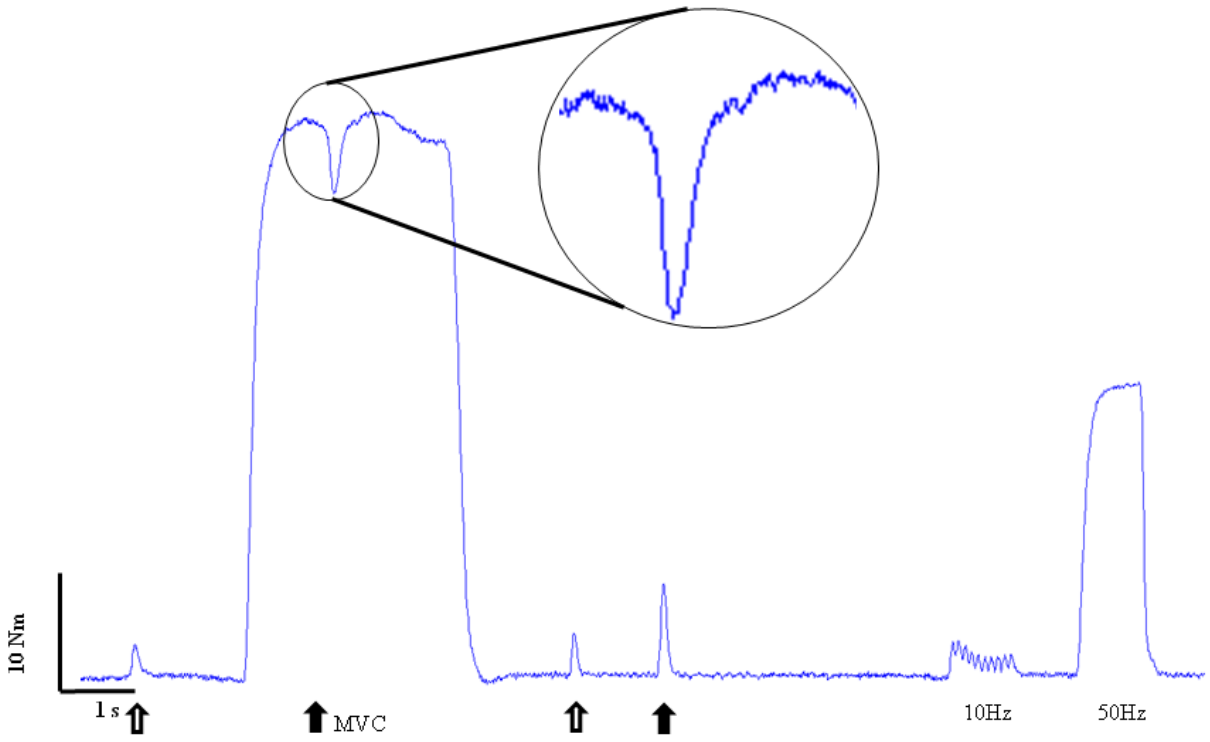

Figure 10. Torque output and activation for a representative subject at 30min of recovery.

The vertical bar on the torque tracing represents the evoked doublet. Open arrows indicate electrically evoked twitches; and filled arrows indicate electrically evoked doublets. Maximal voluntary isometric contraction (MVC) torque

Twitch potentiation increased to 130 ± 16% from baseline following the first set of 30 contractions and 140 ± 28% of baseline immediately following task termination ($p < 0.05$), gradually diminishing to the baseline value at 2 min. Once the potentiating effects of the fatigue protocol were mitigated, twitch torque was reduced to 79 ± 24% of baseline at 2 min recovery ($p < 0.05$) and continued to decrease to 65 ± 18% of baseline by 30 min of recovery. Twitch doublet torque decreased ($p < 0.05$) to 83 ± 15% of baseline following the third set of contractions and was further reduced to 63 ± 11% of baseline by 30 min. Twitch doublet contractile properties parameters including DTPT, DHRT, CD, maximum rate of relaxation and rate of torque development did not differ significantly from baseline at any time point during fatigue and recovery ($p > 0.05$). Peak torque of the 10 Hz was 13.9 ± 5.7 N·m at baseline and was reduced to 64 ± 24% of baseline immediately following the eccentric exercise ($p < 0.05$), and did not recover fully. As well, peak torque of the 50 Hz (Baseline; 24.0 ± 10.2 N·m) was reduced only to 85 ± 16% of baseline following the second set of eccentric contractions ($p < 0.05$) and to 79 ± 15% of baseline immediately following task termination, and did not recover fully. The change in the 10:50 Hz ratio was manifested by the greater reduction in 10 Hz evoked torque compared with the 50 Hz. The 10:50 Hz ratio decreased to 28% of baseline immediately following task termination and continued to decrease to 47% of baseline ($p < 0.05$) at 10 min of recovery (Figure 11) and remained reduced. This indicated there was significant low frequency torque depression following the last set of eccentric contractions.

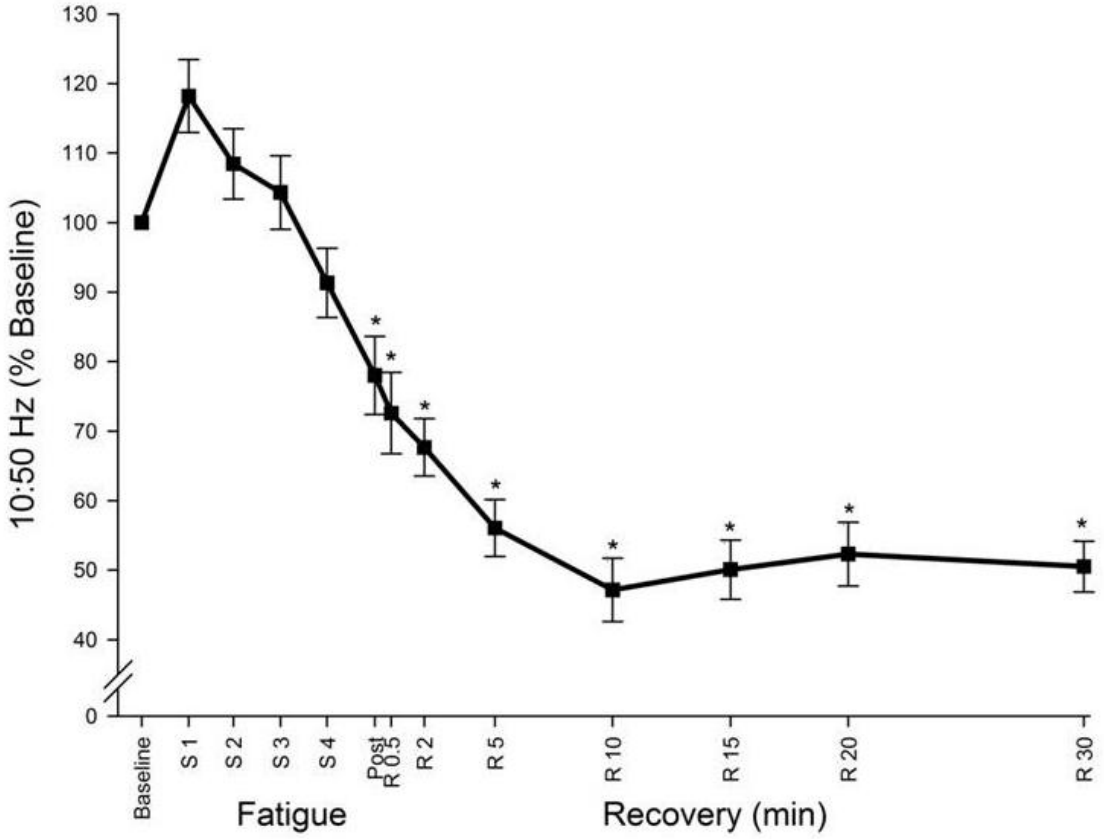

Figure 11. Low-Frequency torque depression (10:50 Hz).

A significant increase in low frequency torque depression as shown by the decreased 10:50Hz ratio was present at Post (task termination), with a continued decrease in 10:50 Hz until 10 min and remained depressed for 30 min (*$p < 0.05$). [S represents 'sets', R represents 'recovery']. Mean ± SE.

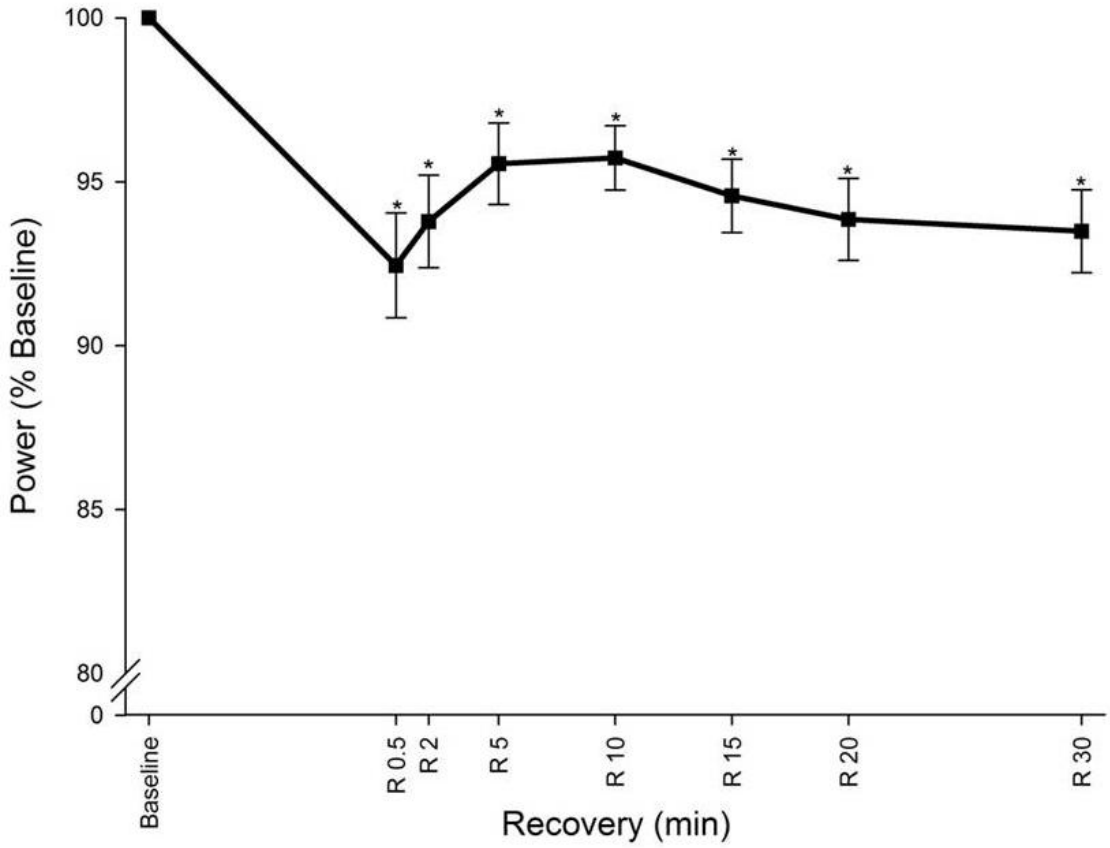

Figure 12. Velocity-dependent power

Velocity-dependent concentric power was reduced by 8% at Post (task termination) compared with baseline and did not recover fully within 30 minutes (*$p < 0.05$). Mean ± SE.

All participants were capable of completing the 30° range of motion during baseline measures and following the eccentric fatigue protocol for all velocity-dependent shortening contractions. Absolute values for baseline velocity and power measures are presented in Table 2. Maximum shortening velocity and subsequently velocity-dependent power were reduced to 92% of baseline immediately following the fatigue protocol ($p < 0.05$; Figure 12) and neither recovered fully.

3.3 Discussion

We tested the hypotheses that following a bout of high-intensity eccentric contractions of the ankle dorsiflexors, there would be a modest reduction in shortening velocity resulting in velocity-dependent power loss, which would remain reduced throughout recovery. The main findings indicate velocity-dependent power loss occurred immediately following the eccentric exercise, and did not recover fully. Furthermore, despite baseline differences the fatigue and recovery profiles were not different between men and women. These results indicate following a bout of eccentric muscle contractions there is a reduction in velocity-dependent power driven by impairment in maximum shortening velocity.

When normalized to pre-fatigue values, there was no sex-related difference for fatigue and recovery. This is an interesting finding because studies on animals support a sex-related difference in fatigability following eccentric exercise (5, 57). However, equivocal results are found in humans (8, 11, 33, 52, 53, 55). The normalized torque-frequency curves (Figure 8), and twitch contractile speeds (time

to peak twitch, half-relaxation time and contraction duration) (Table 2), were not different between the men and women. Thus, both groups may have similar muscle properties (i.e., architecture and fiber type composition) of the ankle dorsiflexors which would lead to a comparable fatigue response. In turn, these findings corroborate reports that suggest human single fiber shortening velocity is similar between sexes (38).

Voluntary ankle dorsiflexor strength was reduced by 28% following eccentric exercise and did not recover fully. Evidently, the mechanisms of fatigue in this study originate peripherally as voluntary activation (>99%) and RMS EMG of the agonist tibialis anterior did not change throughout the entire protocol, which is similar to previous reports on the ankle dorsiflexors (9, 48). However, this is not always the case when other muscles are investigated, for example voluntary activation of the elbow flexors has been shown to decrease by ~11-22% following eccentric exercise (25, 40). Thus, the ability to fully activate the dorsiflexors, even when the muscle is stressed severely or in this case undergone damaging lengthening contractions is unique.

A recent investigation (30) found muscle fiber conduction velocity in the quadriceps was decreased following eccentric exercise due to sarcolemmal damage. However, excitation failure of the sarcolemma cannot account for the torque and power depression in the ankle dorsiflexors, as similar to other reports (48), M-wave properties (area, duration, amplitude) did not change during and following task termination. This was further corroborated with the findings from the electrically evoked contractions. For example, peak twitch torque declined by 21% 2 min

following task termination. Concomitantly, twitch potentiation, which could offset the initial fatigue response in peak twitch torque, was no longer measurable at that time and peak twitch torque remained depressed. Similarly, the 10 Hz and 50 Hz evoked torques were reduced following the eccentric exercise and did not recover fully. As previously observed (9, 48) following eccentric exercise of the ankle dorsiflexors, the contractile speeds (time to peak twitch, half-relaxation time and contraction duration) of the evoked twitch doublet did not change. Because eccentric muscle actions are less metabolically demanding than other contraction types (1, 10) metabolic accumulation and alterations to blood chemistry may not have been responsible for the impairment in torque production (3). Subsequently, mechanical disruption of the link between the t-tubule and the sarcoplasmic reticulum lead to excitation-contraction (E-C) uncoupling, which remains as the likely peripheral impairment responsible for the immediate torque and power loss (2, 35, 59). The most plausible stage of E-C coupling which was impaired following the eccentric exercise was the release of calcium from the sarcoplasmic reticulum (39), evident by the decrease in electrically evoked torque at low-frequency stimulation. In addition to impaired calcium release, muscle damage or some structural impairment to the contractile machinery likely occurred, which is represented by the decrease and incomplete recovery of the 10:50 Hz ratio, and MVC. The 10:50 Hz ratio decreased immediately following eccentric exercise and continued to decrease into recovery, but at 10 min it stabilized at ~50% of baseline throughout the remainder of recovery. The change in the 10:50 Hz ratio was manifested by the greater reduction in 10 Hz than 50 Hz evoked torque. This

further supports an impairment in E-C coupling leading to low frequency torque depression (21). Ultimately, this finding was a result of the primary insult of eccentric exercise and not due to secondary effects of muscle damage which typically occur 1-2 hr after the initial injury (56).

The incomplete recovery of MVC torque following the eccentric exercise suggests strongly that damage to muscle fibers had occurred (6). Prolonged torque loss following unaccustomed eccentric exercise is often considered to be a reliable indirect marker of muscle damage (19, 40, 60). Although MVC torque is less impaired immediately following high-intensity eccentric actions than concentric or isometric exercise (37, 41, 48), when reassessed day(s) later, voluntary isometric torque loss following concentric contractions recovers, whereas following eccentric contractions torque loss is still present (53). Incomplete recovery of both voluntary and evoked torque cannot be attributed to metabolic fatigue. Thus, muscle damage and the subsequent impairment of the contractile machinery may have been responsible for the prolonged torque loss in the present study.

Velocity-dependent power, calculated here as the product of 20% MVC torque and maximum angular velocity of the contraction, was reduced by 8% following eccentric exercise and did not recover fully. These observations are unlike previous reports which used shortening velocity as the criterion measure of fatigue following contractions of the ankle dorsiflexors, and other muscles (14, 15) in which velocity-dependent power recovered within ~5 min following concentric contractions. For example, Cheng & Rice (16) fatigued the dorsiflexors to 50% of peak shortening velocity, but velocity recovered within 5 min. In the present study,

velocity-dependent power was reduced by 8%, but did not recover within 30 min. Mechanisms of impaired neuromuscular functioning differ between fatigue and damage, and can be distinguished by the time course of recovery. Thus, the reduction and prolonged recovery of power following eccentric fatigue may result from different mechanisms than during a concentric fatigue task (discussed below).

Although MVC torque yields valuable insight regarding the contractile state of the muscle, it assesses only a single aspect of muscle performance. The unique study design employed here involved testing participants using the isotonic mode of the Biodex to evaluate eccentric fatigue-induced reductions in shortening velocity which would remain masked when tested isokinetically. We observed a significant decrease of 8% and 28% in velocity-dependent power and MVC following the eccentric fatigue task, compared with baseline, respectively. Despite a 3.5 fold greater loss of torque production capacity (MVC) over shortening velocity, it would seem MVC is more sensitive to perturbations to the system following eccentric exercise. Because power was calculated at 20% MVC the observed loss of torque production capacity may only contribute minimally to the loss of power, as peak shortening velocity was reached not at the onset of movement but rather throughout the range of motion ($\sim 15^{o}$ plantar flexion). Hence, the torque developed to overcome the resistance was not as critical in determining peak power as the speed of shortening.

A metabolic explanation (20, 61) may account for the initial decrease in shortening velocity, where excessive ADP surrounding the contractile proteins actin and myosin result in slower cross-bridge cycling. However, due to the time

sensitive nature of metabolic perturbations this slowing does not account for the incomplete recovery of shortening velocity and, hence, power. The delayed recovery of power, as seen here is most likely due to damage induced EC uncoupling, resulting in reduced calcium release (4, 35), and damage to the contractile machinery imposed by the lengthening contractions. Increased sarcomere instability following eccentric exercise leads to a reduction in the number of functional sarcomeres in series, hence the number of 'force generators' are reduced (45, 46) resulting in a reduced shortening velocity, as well, a change in optimal muscle length for torque production to longer lengths (13, 42, 49). Thus, structural impairments in EC coupling and the contractile machinery imposed via the eccentric actions is responsible for power loss and reduced recovery following eccentric exercise.

Although the current study cannot determine the specific mechanisms of reduced power, we found significant E-C coupling perturbations as evidenced by the presence of low frequency torque depression. The damaging eccentric contractions impaired shortening velocity and reduced power for up to 30 min following task termination. In summary, when velocity-dependent contractions are used as the criterion measure to calculate power, we demonstrated that following eccentric exercise maximal shortening velocity was reduced, which contributed to the observed reduction in power. Further research on velocity-dependent contractions is warranted, as it relates to human movement where the load is fixed and velocity is variable.

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Chapter 4 – Power loss is greater following lengthening contractions in old versus young women[3]

4.0 Introduction

Research on age-related muscle fatigue has focused primarily on isometric and shortening contractions. Far less is known in older adults regarding neuromuscular function and short-term recovery following repeated high-intensity lengthening contractions which can provoke long lasting impairments in neuromuscular performance (11, 49). Furthermore, we are interested in the velocity component (i.e., voluntary shortening velocity) of power, following lengthening contractions. This contraction mode in which the load is fixed and velocity of movement is unconstrained allows for alterations in shortening velocity to be elucidated which in older adults already is impaired and is a strong indicator of age-related muscle fatigability (22, 41, 47, 56).

By the eighth decade of life, the senescent adult has undergone alterations to both the structure and function of the neuromuscular system that lead to impaired muscle performance (17, 44, 57). These alterations include: muscle atrophy (preferentially Type II muscle fibers), and the death and remodeling of motor units (MUs) resulting in a greater relative composition of slow type muscle fibers (57), and architectural changes to the muscle and musculotendinous unit (44).

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Power GA, Dalton BH, Rice CL, Vandervoort AA. Power loss is greater following lengthening contractions in old versus young women. *Age* 34: 737-750, 2012.

Additionally, neural changes can include greater antagonist coactivation (34) and lower maximal MU discharge rates (21). The combined consequence of these structural and neural age-related manifestations are a slowing of intrinsic muscle contractile properties (59), lower rates of torque development and reduced cross-bridge kinetics (1). Hence, older adults exhibit impairments in maximal voluntary shortening velocity, torque production and especially muscle power (41). Despite the negative implications of age-related changes to the neuromuscular system, there is a relative preservation of eccentric strength (31, 48, 58). Although older adults can experience similar (18), less (38), or more (24) muscle damage than young adults, it is unknown whether maintained eccentric strength is an advantageous mechanism with which to maintain effective neuromuscular performance during and following a bout of repeated lengthening contractions.

It seems well established that older adults are more fatigue resistant than young adults during isometric tasks (33), yet the fatigue response during and following dynamic shortening contractions is equivocal and depends upon the task. Older adults can experience less (37, 53), similar (12, 35), or more (8, 40, 47) fatigue than young. However, tasks which are performed with an unconstrained velocity component (i.e., velocity-dependent) always yield a greater fatigue response in older adults than young (22, 40, 47). Moreover, the effects of repeated lengthening contractions on age-related muscle fatigue are less well understood. The only study investigating age-related fatigability following lengthening contractions (8), reported that the reduction in maximum voluntary isometric contraction (MVC) torque did not differ between old and young adults during or following repeated

isokinetic (60°/s) lengthening contractions, but isokinetic torque loss during the lengthening contractions was greater in older adults than the young. However, power was not assessed following the protocol, and thus it is unknown whether repetitive lengthening contractions affect concentric power differently in old and young adults, and which component of power (torque or shortening velocity) is more compromised.

Voluntary maximal loaded shortening velocity is known to recover rapidly (< 5 min) in young adults after voluntary isometric and concentric fatigue tasks (14, 15). However, repeated lengthening contractions result in muscle damage which can take multiple days to recover fully (19, 51), and it is unclear how this damage may affect velocity-dependent power during short-term recovery in older adults. Impaired isometric torque production following lengthening contractions can be attributed to a mechanical disruption of the link between the t-tubule and the sarcoplasmic reticulum impairing calcium (Ca^{2+}) release (32, 60), and also, to the redistribution of sarcomere lengths [see popping sarcomere hypothesis (43)], resulting in a length-tension relationship shift to longer muscle lengths for optimal torque production. As well, dynamic performance following multi-joint lengthening contractions is known to be impaired (11, 55) although the mechanisms are not entirely understood. Recently, we (49) reported that MVC torque and velocity-dependent power did not recover fully up to 30 min following 150 lengthening contractions in healthy young men and women. Although lengthening contractions are less energetically demanding than isometric and dynamic shortening contractions (2, 54), they are known to induce muscle fatigue (8, 16, 42, 45).

Because excitation-contraction (E-C) coupling is compromised in older adults (46) and maximal unconstrained shortening velocity is indeed slower (22, 41, 47) compared with young adults, the old may be energetically disadvantaged during this task. Thus, older adults may experience a greater perturbation in ATP homeostasis, consequently exacerbating their fatigue response (33) and resulting in a greater reduction in shortening velocity and subsequent velocity-dependent power than young adults.

Therefore, the purpose here was to investigate the effect of repeated high-intensity lengthening contractions on neuromuscular function in old and young women with a particular emphasis on the short-term recovery of velocity-dependent power. As a result of similar muscle damage, MVC torque will be reduced similarly in both old and young women and remain reduced throughout a 30 min recovery period. However, when tested under dynamic conditions (velocity-dependent shortening), we hypothesize that the older women will have a larger reduction in velocity-dependent power than the young owing to a greater impairment in shortening velocity and impairments in E-C coupling, which are known to be compromised in older adults and may not be observable during isometric testing. As a result of muscle damage neither group will recover by 30 min.

4.1 Methods

4.1.1 Participants: Nine old (68.3 ± 6.1 y) and nine young women (25.1 ± 1.3 y) from the university population and local community groups, who were free from musculoskeletal disorders which would impair their ability to perform the task, volunteered for this study. All participants were recreationally active. The mean height and mass of the old and young women were: 162.0 ± 7.3 cm and 67.7 ± 8.5 kg, and 167.1 ± 7.0 cm and 63.7 ± 10.4 kg, respectively. All participants were asked to refrain from strenuous exercise one day prior to testing and to not consume caffeine on the testing day. This study was approved by the local University's Review Board for Health Sciences Research Involving Human Subjects and conformed to the Declaration of Helsinki. Informed, oral and written consent was obtained from all participants prior to testing.

4.1.2 Experimental arrangement*:* All testing was conducted on a Biodex multi-joint dynamometer (System 3, Biodex Medical Systems, Shirley, New York). For a detailed explanation and experimental timeline of the testing set-up and procedures please refer to (49). The right foot was strapped tightly to the Biodex ankle attachment footplate, aligning the lateral malleolus with the rotational axis of the dynamometer. Extraneous movements were minimized using non-elastic shoulder, waist and thigh straps. Participants sat in a slightly reclined position with the hip, knee, and ankle angles set at ~110^{o}, ~140^{o}, and ~30^{o} plantar flexion, respectively. All voluntary and evoked isometric dorsiflexion contractions were performed at an ankle joint angle of 30^{o} of plantar flexion. Shortening contractions began from the plantar flexed position of 30^{o} and ended at the neutral ankle angle

(0^{o}). The lengthening contractions commenced at the neutral ankle angle (0^{o}) and ended at 30^{o} plantar flexion, thus both dynamic actions moved through a 30^{o} range of motion. All dynamic contractions were performed using the isotonic mode of the Biodex. However, due to inherent mechanical limitations of the dynamometer (unable to maintain an exact constant external load throughout an entire range of motion), these contractions are not purely isotonic and neither are they iso-inertial as the load is fixed (mechanically) and not influenced by gravity but rather the braking of the dynamometer. And therefore, we refer to these contractions as "velocity-dependent". A velocity-dependent movement is characterized by a participant producing a dynamic contraction as fast as possible in which the angular velocity is unconstrained while the load or resistance is fixed at a pre-determined value (i.e., 20%MVC).

Surface electromyography (EMG) was collected from the tibialis anterior and soleus muscles using self-adhering Ag-AgCl electrodes (1.5 X 1 cm; Kendall, Mansfield, MA). The skin was cleaned forcefully with an alcohol swab prior to the application of the electrodes. A monopolar electrode configuration was used with the active electrode positioned on the proximal portion of the tibialis anterior over the innervation zone (~7 cm distal to the tibial tuberosity and ~2 cm lateral to the tibial anterior border) and a reference electrode was placed over the distal tendinous portion of the tibialis anterior at 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 a reference was placed over the calcaneal tendon.

Stimulated contractions of the dorsiflexors were evoked electrically using a bar electrode held distal to the fibular head over the deep branch of the common peroneal nerve. 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 50-100 μs.

4.1.3 Experimental procedures: Peak twitch torque (P_t) was determined by increasing the current until a plateau in dorsiflexor P_t and tibialis anterior M-wave amplitude were reached, and then the current was further increased by 10-15% to ensure activation of all motoneurons via supramaximal stimulation. This stimulation intensity was used for the evoked doublet (P_d) (2 pulses at a 10 ms interpulse interval), and to assess voluntary activation. Next, a 1 s train at 50 Hz was delivered to assess peak tetanic torque by increasing the current until there was a plateau in evoked torque. This was tolerated by all young women and 4 of the older women.

Three isometric MVCs were then performed; each of 3-5 s duration. Three min of rest was given between all contractions. To ensure MVC attempts were maximal, participants were provided visual feedback of the torque tracing on a computer monitor, and exhorted verbally during all voluntary efforts and voluntary activation was assessed using the modified interpolated twitch technique (26). The amplitude of the interpolated torque evoked during the peak plateau of the MVC was compared with a resting P_d evoked ~1 s following the MVC when the muscles were relaxed fully. Percent voluntary activation was calculated as voluntary activation (%) = [1- interpolated P_d /resting P_d] x 100. Values from the MVC with

the highest peak torque were used for data analysis. Next, 10 pulses and 50 pulses were delivered over a 1 s period to determine a 10 Hz to 50 Hz relationship in all 9 young and 4 old participants using the current required to evoke peak 50 Hz torque.

Once MVC torque was determined, the dynamometer was switched from the isometric to isotonic mode and a load equal to 20% MVC was programmed to allow for determination of maximal shortening velocity with this load, and velocity-dependent power. The 20% MVC resistance was chosen because it represents a moderate load for the fast shortening contractions, and one that all subjects could perform through the entire range of motion even after a bout of repeated high-intensity lengthening contractions. Before the footplate moved during the velocity-dependent shortening contractions, participants had to overcome the pre-programmed resistance. The dynamometer absorbs this increase in applied torque resulting in a directly proportional increase in angular velocity. This is a helpful feature to explore the effect of damaging lengthening contractions on alterations in velocity of unconstrained movement and power. The dynamometer was programmed to allow the footplate to return to 30^{o} of plantar flexion at the end of each shortening voluntary contraction while the participant relaxed fully. Familiarization with these 'fast' shortening contractions involved participants performing several (typically 5) velocity-dependent shortening contractions until a stable value was obtained (no change in maximal shortening velocity). To ensure a maximal effort (peak velocity) contraction, all participants were instructed to move the load "as hard and as fast as possible throughout the entire range of motion". To assist participants in reaching their maximal shortening velocity, visual feedback of

the velocity profile was provided via a computer monitor, and a horizontal cursor was positioned at the previous plateau in peak velocity. Participants rested for 3 min and then performed 2 consecutive contractions which were used to establish baseline values for maximum shortening velocity and peak power.

4.1.4 Fatigue and recovery protocol: With a load of 80% MVC, participants performed 5 sets of 30 eccentric dorsiflexion contractions with each set separated by 30 s of rest. Participants were provided visual feedback of the torque and instructed to resist the lowering of the foot plate through the 30^{o} range of motion over a 1 s period. After the lengthening contraction, the foot was returned to the neutral ankle starting position by the investigator over a 2 s (15^{o}/s) period while the participant relaxed fully. The participant was then instructed to resist the lowering of the footplate immediately again until the protocol was complete. The voluntary and electrically evoked responses of the dorsiflexors were recorded at baseline, during the fatigue protocol immediately following each of the 5 sets, and during recovery at 0.5 min, 2 min, 5 min, 10 min, 15 min, 20 min, and 30 min after task termination. Measures following the fatigue protocol included, and were performed in the following order: (1) maximum evoked twitch properties, (2) assessment of MVC and voluntary activation, (3) post-activation twitch and twitch doublet, (4) measure of low frequency torque depression (10:50 Hz ratio; LFTD), and (5) velocity-dependent shortening power.

4.1.5 Data reduction and analysis: Torque, position and velocity data were sampled at a rate of 100 Hz. All data were converted to digital format using a 12-bit analog-to-digital converter (model 1401 Power, Cambridge Electronic Design,

Cambridge, UK). Surface EMG signals were pre-amplified (x100), amplified (x2) and band-pass filtered (10-1,000 Hz), and sampled online at 2500 Hz using Spike 2 software (version 6.10, Cambridge Electronic Design Ltd.). Surface EMG from the MVC was expressed as root mean squared (RMS) and values were obtained from a 1 s time period about the peak torque. All subsequent MVC RMS values were normalized to the level obtained during baseline. Peak RMS values of the raw surface EMG during the fast shortening contractions was calculated through the 30^{o} range of motion from the onset of movement to the end of the range of motion and then normalized to the fastest baseline contraction. Power was calculated as the product of torque (Nm) and the peak shortening velocity (rad/s) of the faster of 2 contraction attempts. Post-activation potentiation was determined by calculating the ratio between the amplitude of the peak twitch torque recorded before and following the isometric MVC. Spike 2 software was used off line to determine M-wave amplitude, area, duration, the peak twitch torque (P_t), peak doublet torque (P_d), doublet time to peak twitch ($_D$TPT), half relaxation time ($_D$HRT) of the doublet, and doublet rate of torque development ($_D$MRTD). Low frequency torque depression was calculated using a ratio of peak 10 to peak 50 Hz evoked torques (10:50 Hz).

4.1.6 Statistical analysis: Using SPSS software (version 16, SPSS Inc. Chicago, IL) a two-way (age x time) repeated measures analysis of variance was performed to assess all neuromuscular data. Because voluntary activation values are not normally distributed, a Mann-Whitney U-test was employed and an unpaired T-test was used for subject characteristics and baseline measures to assess group

differences. The level of significance was set at $p<0.05$. When a significant main effect or interaction was present, Post hoc analysis using unpaired T-tests was performed with a Bonferroni correction factor to determine where significant differences existed. Effect sizes (ES) were calculated using the partial eta-squared method to explore the magnitude of apparent statistical effects. Due to the small sample size of old women for LFTD (n=4) unpaired t-tests were performed for this parameter. The table is presented as means ± standard deviations (SD), and figures as mean ± standard errors (SE) values, normalized to baseline (pre-test).

4.2 Results

4.2.1 Baseline measures: As shown in Table 3 the old women as compared with the young women were ~21% weaker for MVC torque (p=0.021, ES=0.292) despite similar high voluntary activation (~95%, p=0.682, ES=0.012). Peak loaded shortening velocity (Figure 13) was ~21% slower for the old women than the young ($p<0.001$, ES=0.522), which lead to power (calculated as the product of peak loaded shortening velocity at 20% MVC) to be ~39% less in the old compared with the young women (p=0.006, ES=0.383). Both groups had a similar P_d (p=0.685, ES=0.011), while $_DTPT$ was ~16% slower (p=0.023, ES=0.284), and $_DHRT$ was ~33% longer in old compared to young, respectively (p=0.012, ES=0.337). Despite similar P_t (p=0.735, ES=0.007) for the old (3.9 ± 1.6 N·m) and young (4.0 ± 0.9 N·m) women, the older adults had a reduced capacity for potentiation (105.8 ± 6.0%) compared to the young (124.6 ± 17.2%) (p=0.023, ES=0.339).

	Twitch Doublet Properties							
Group (n=9)	Pd (N·m)	TPT (ms)	HRT (ms)	MRTD (s-1)	MVC (N·m)	VA (%)	Velocity (deg·s-1)	Power (W)
Young	9.1 ± 2.7	148.2 ± 24.8	80.0 ± 15.2	11.3 ± 1.2	34.0 ± 4.6	99.2 ± 0.9	134.3 ± 9.0	15.8 ± 3.3
Old	8.6 ± 2.3	173.3 ± 21.9*	101.0 ± 23.1*	10.0 ± 1.1*	28.2 ± 4.9*	98.9 ± 2.1	109.1 ± 16.1*	11.2 ± 2.7*

Table 3. Voluntary and evoked participant baseline characteristics.

Old women had slower absolute evoked doublet twitch torque (P_d) contractile properties for time to peak twitch ($_D$TPT) (p=0.023, ES=0.284), half-relaxation time ($_D$HRT) (p=0.012, ES=0.337), and maximum rate of torque development ($_D$MRTD) (p=0.031, ES=0.258) compared to young. Maximal voluntary isometric contraction (MVC) torque (p=0.021, ES=0.292), maximum shortening velocity (p=0.001, ES=0.522), and peak power (p=0.006, ES=0.383) were lower in the old than young women. Voluntary activation (VA) (p=0.682, ES=0.012) and doublet twitch torque (p=0.685, ES=0.011) was not significantly different between groups. * Denotes a significant difference between old and young women.

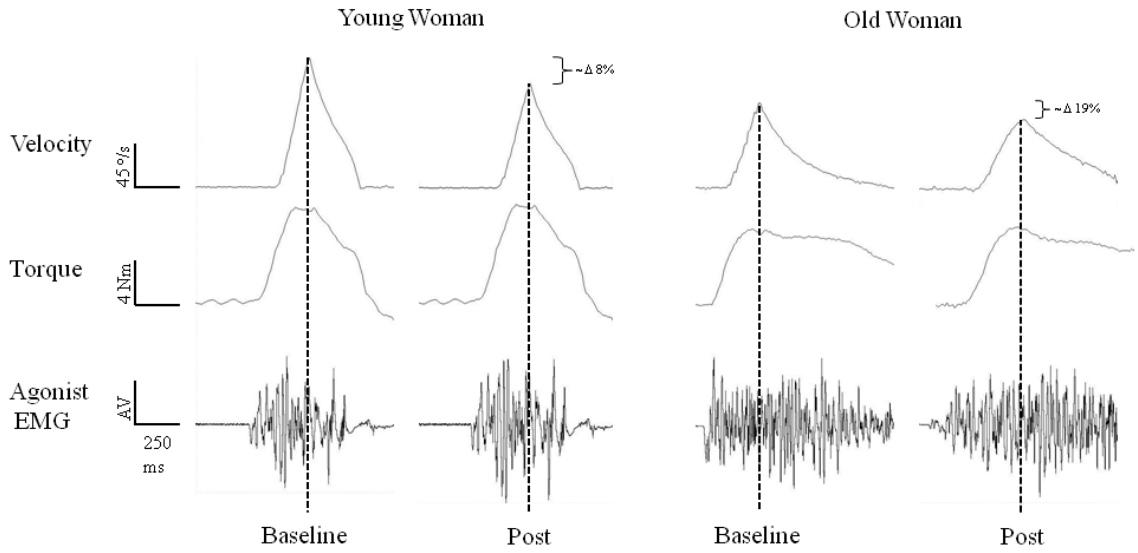

Figure 13. Representative unprocessed data

A young and older woman performing a fast velocity-dependent shortening contraction at baseline and 30 s following (Post) the lengthening contraction task. The EMG amplitude is presented with arbitrary values (AV). The dashed vertical line indicates peak velocity.

4.2.2 Fatigue and recovery measures: All participants were capable of completing all contractions, although as reported previously using this contraction mode some subjects had difficulty lowering the foot plate at a steady pace for the last few contractions of each set (49). This failure to maintain a constant velocity resulted in increased eccentric velocities which ranged from 36°/s to 42°/s. Despite the variation in velocity, the duty cycles were similar (p=0.295, ES=0.680) between old and young women 0.33 ± 0.07. For the velocity-dependent shortening contractions, all participants were capable of completing the 30° range of motion during baseline measures and following the lengthening contraction task. Neuromuscular fatigue measures were analyzed with regard to relative changes over time. For maximum loaded shortening velocity and subsequently peak power (Figure 14), there were main effects for time (p<0.001, ES=0.681) and age (p=0.007, ES=0.396) and an interaction (p=0.004, ES=0.244). Thus, at task termination the old women had a greater loss of power (~19%) than the young (~8%). This difference persisted until 10 min of recovery and did not recover by 30 min post intervention.

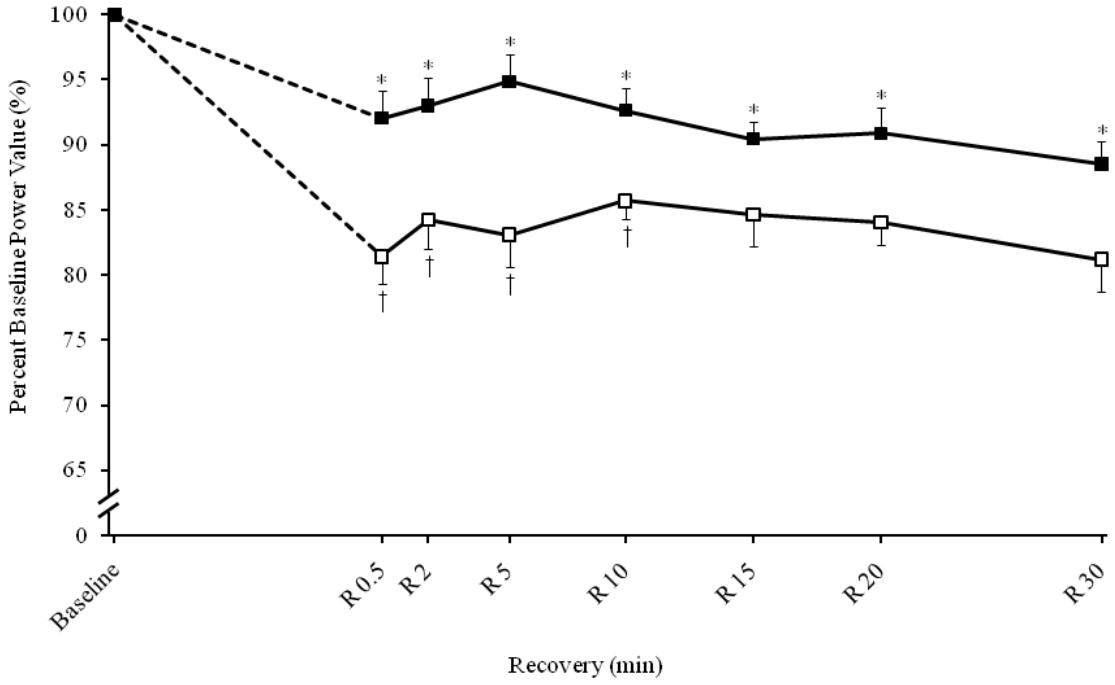

Figure 14. Velocity-Dependent Power

Short-term recovery of velocity-dependent power calculated at 20% MVC and maximal shortening velocity normalized to 100% of baseline values for old (open symbols) and young women (solid symbols). The dashed lines represent the lengthening contraction intervention during which time only isometric measures were obtained. Significant effects for Time (*p<0.05) and Age (†p<0.05). Values are means ± SE.

For dorsiflexor MVC torque there was only a significant effect for time (p<0.001, ES=0.696). Isometric MVC torque decreased similarly in the old and young by ~19% following the first set of 30 eccentric contractions and following each successive set it continued to decrease until it was reduced by ~28% immediately following task termination. By the end of the 30 min recovery period the MVC regained 9% but was still significantly less than baseline (Figure 15). Voluntary activation was maintained greater than 95% at baseline and did not change (p=0.910, ES=0.022) throughout fatigue and recovery. The incomplete recovery of MVC by 30 min post intervention suggests similar muscle damage had occurred in young and old women.

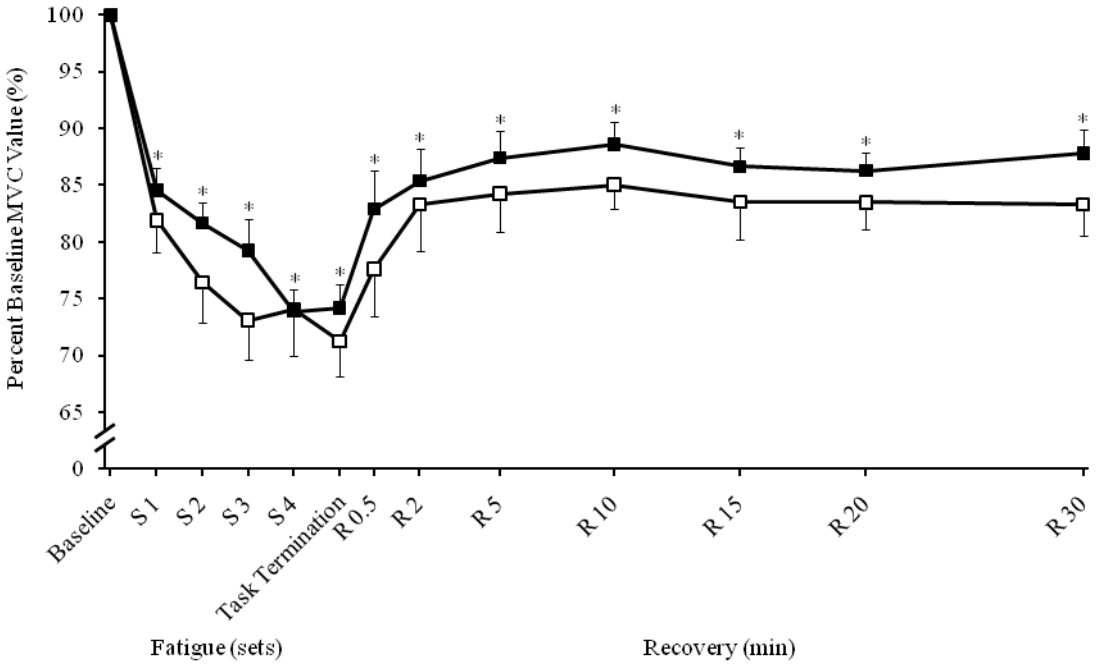

Figure 15. Maximum voluntary isometric contraction (MVC)

Maximal voluntary isometric strength during and following lengthening contractions normalized to 100% of baseline values for old (open symbols) and young women (solid symbols). Significant effects for Time (*p<0.05). Values are means ± SE.

Low frequency torque depression (10:50 Hz) presented a significant effect for time (p<0.001, ES=0.960) and age (p=0.032, ES=0.225). Over time, the alterations in the 10:50 Hz ratio were manifested by the greater reduction in 10 Hz evoked torque compared with the 50 Hz. This indicated there was significant low frequency torque depression following the lengthening contractions for both groups. Low frequency torque depression persisted in both groups throughout the 30 min recovery period. However, at task termination the 10:50 Hz ratio was reduced by 40% in the four old, but only 20% in the young, suggesting there was an initial greater impairment in E-C coupling in the old women. This age-related difference was present up to 10 min in the recovery period (Figure 16) at which time both groups were reduced by 50% and did not change during the final 20 min of the recovery period.

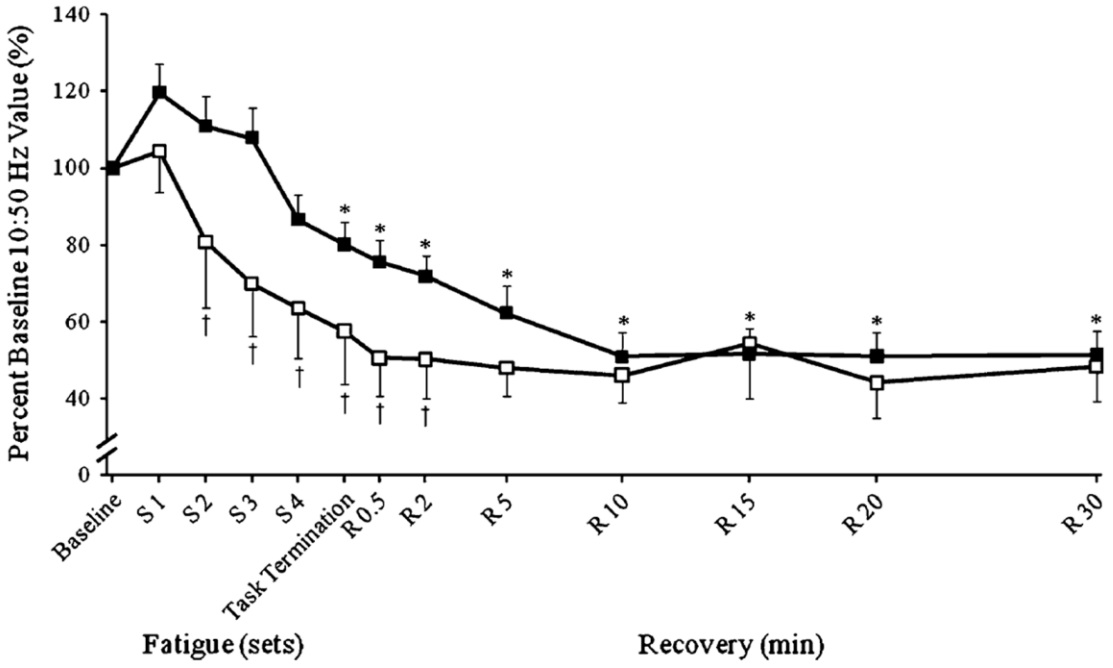

Figure 16. Low Frequency torque depression (10:50 Hz)

Low frequency torque depression during and following lengthening contractions normalized to 100% of baseline values for old (n=4) (open symbols) and yo ung (n=9) women (solid symbols). The decrease in the 10:50 Hz ratio was driven primarily by the progressive decline in 10 Hz torque (40% decrease at task termination, and 60% decrease by 30 min of recovery) with a minimal decrease in 50 Hz (20% decrease at task termination and throughout recovery). Significant effects for Time (*p<0.05) and Age (†p<0.05). Values are means ± SE

There were main effects for time (p<0.001, ES=0.646) and age (p=0.005, ES=0.437) and an interaction (p<0.001, ES=0.409) for P_t (Figure 17). Twitch torque decreased by ~21% in the old women following the first set of 30 lengthening contractions, while the young women had a potentiation of Pt, which increased to ~130% of baseline following Set 1. At task termination the values for the old women were reduced by 50%, whereas P_t for the young women was not different from baseline values. Once the potentiating effects of the fatigue protocol were mitigated in the young, both groups were reduced similarly (~ 50%) 5 min into recovery. For the P_d torque there was only a significant effect for time (p<0.001, ES=0.648). P_d continued to decrease (Figure 17) during the lengthening contractions and remained reduced in both groups by ~40% throughout the 30 min recovery period For doublet twitch contractile speeds there were only main effects for time for $_D$TPT (p<0.001, ES=0.544), and $_D$HRT (p<0.001, ES=0.475), meaning doublet twitch contractile properties slowed similarly in both groups by ~15-20%. However, there was a main effect of time (p<0.001, ES=0.356) and age (p=0.003, ES=0.429) and an interaction (p=0.05, ES=0.118) for the $_D$MRTD which was reduced ~15% greater in old women than young women at task termination but was no longer significantly different between groups 30 s later.

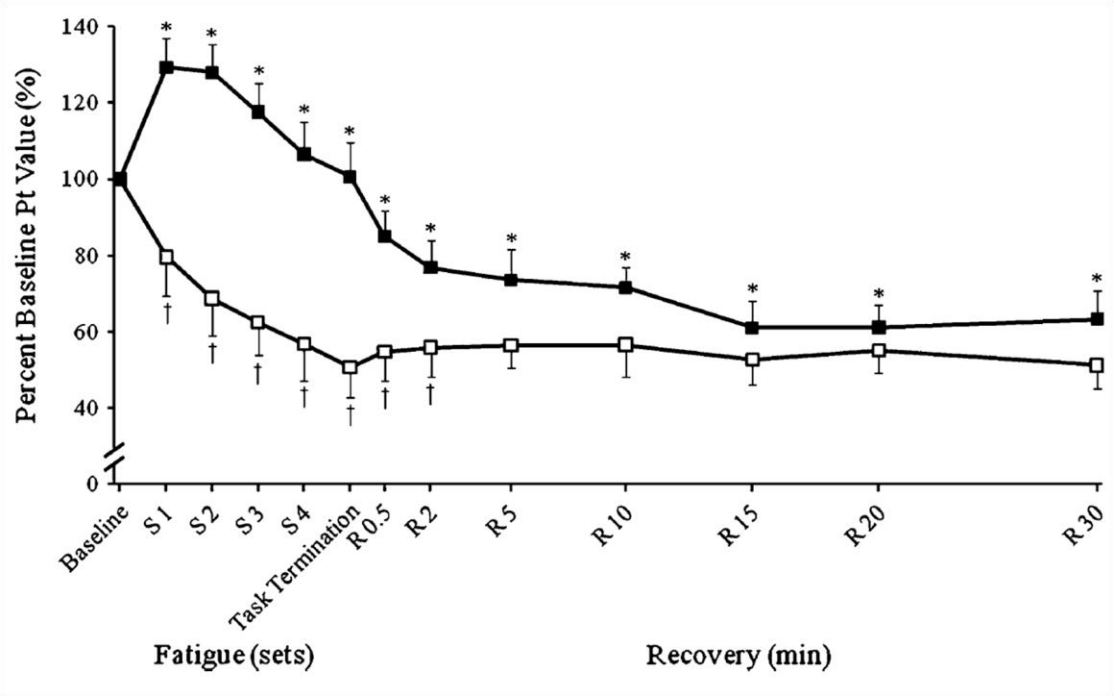

Figure 17. Peak twitch torque (P_t)

Peak twitch torque during and following lengthening contractions normalized to 100% of baseline values for old (open symbols) and young women (solid symbols). Significant effects for Time (*p<0.05) and Age (†p<0.05).

Tibialis anterior M-wave properties, including: peak-to-peak amplitude, duration, and area showed a main effect for time (p=0.004, ES=0.190), meaning M-wave properties were reduced similarly in both old and young women by ~10-15% at task termination, and returned to baseline value by the end of the 30 min recovery period. For both the old and young women there were no significant changes from baseline for (p=0.064, ES=0.151) RMS EMG of the agonist tibialis anterior during MVCs throughout the protocol. As well, RMS EMG of the soleus muscle did not differ for time or age from baseline (p=0.222, ES=0.0125). During the velocity-dependent shortening contractions RMS EMG of the agonist tibialis anterior or antagonist soleus showed no effect for time (p=0.135, ES=0.125) or age (p=0.426, ES=0.070) meaning there was no difference in 'neural drive' from baseline contractions.

4.3 Discussion

This investigation tested the hypothesis that neuromuscular function of the dorsiflexors following repeated lengthening contractions would be impaired more in the old women than young. Specifically, velocity-dependent power would be reduced more in the old than young and neither would remain depressed throughout the 30 min period of recovery following task termination. Indeed, peak power was reduced by 19% for the older women after the lengthening contractions, whereas the young women only incurred an 8% decrement at task termination, and neither recovered. In contrast, isometric MVC torque was reduced similarly (28%) in both the old and young and did not recover fully. Despite similar muscle damage

as indicated by incomplete recovery of MVC torque (61), these findings suggest old women have greater decrements in velocity-dependent power than their younger counterparts following repeated lengthening contractions. Therefore, the greater power-loss in the old than young women is driven more by fatigue mechanisms influencing impairments in whole muscle loaded shortening velocity following lengthening contractions than those affecting torque generation per se.

4.3.1 Baseline. The old women in this study were weaker and slower (Table 3) for whole muscle shortening velocity, leading to a greater reduction in power when compared with young women. The 39% reduction in velocity-dependent power compared with the young is greater than that reported previously for velocity-dependent contractions of the dorsiflexors (25%) of old men (41) and similar to the plantar flexors (38%) of old men (22), and elbow flexors (41%) (56) and knee extensors (45%) (47) of old women. As well, older women rely more on the velocity component of power than torque production when compared with old men and younger adults (56). Valour et al. (2003) reported that when peak muscle power was compared among various loads (i.e., % MVC) older women reached peak power at a lower percentage of MVC torque than older men and women. In the current study we used a relative load of 20% MVC which relies strongly on the velocity component of power (49). Factors discussed below that impair whole muscle shortening velocity in older women may greatly impair their ability to generate power more so than older men and younger individuals.

4.3.2 Lengthening contraction intervention. In the current study, following 150 high-intensity lengthening contractions the old women incurred (up to 10 min)

a greater loss of velocity-dependent power (19%) than the young (8%) following task termination, whilst both the old and young women experienced similar reductions in isometric MVC torque at task-termination (28%). This is similar to findings from isovelocity fatigue studies in which older adults incur a greater decline in eccentric isokinetic torque than young, while still maintaining isometric strength (8). Interestingly, the reduction in MVC torque at 30 min recovery (~19%) is similar to the reduction following the first 30 lengthening contractions (Figure 15), suggesting the primary insult of muscle damage occurred during the first set of contractions and the further decrease in MVC torque to task termination can be attributed to fatigue processes (16, 42). Despite similar reductions in isometric MVC torque following the lengthening contractions, low-frequency torque depression was greater in the old than the young women (~25% difference) following the second set of lengthening contractions and for up to 5 min into recovery, and neither recovered during the 30 min period of recovery .

The development of fatigue can manifest through central or peripheral mechanisms (4, 26), or both. In the current study voluntary activation and RMS EMG amplitude of the tibialis anterior during the isometric MVCs was not reduced from baseline and did not differ between age groups. In accord with previous investigations utilizing velocity-dependent contractions, RMS EMG amplitude of the agonist tibialis anterior during velocity-dependent shortening contractions did not differ throughout the study (49) or between young and old. Hence, the main site of fatigue is likely peripheral in nature. Voluntary activation failure can account for torque loss following muscle damage in other limb muscles (50) however,

maintained voluntary activation to the tibialis anterior is a common finding following lengthening contractions (8, 45, 49). Furthermore, in the present study, M-wave parameters (i.e., p-p amplitude, area, duration) were reduced similarly in old and young indicating that muscle damage may have disturbed sarcolemmal excitability in both age groups equally. However, findings are equivocal; some studies show a decrease in M-wave properties (30) while others using similar lengthening contraction protocols do not (45, 49). The reason for this disparity among studies is unclear, but it may be related to rest intervals between contractions or because of different aged populations tested.

4.3.3 Fatigue and muscle damage. Although lengthening contractions are less energetically demanding than isometric and dynamic shortening contractions (2, 54), they are known to induce muscle fatigue in addition to muscle damage (8, 16, 42, 45). A commonly accepted indirect measure of muscle damage is the reduction and incomplete recovery of isometric MVC torque (6, 19, 61). The concomitant existence of fatigue and damage may account for the greater initial decline in MVC torque than either factor alone, however because MVC torque did not recover fully, this observation may represent muscle weakness (26) and suggest muscle damage occurred. The long term deficits in force production may be due to damage induced impairments in E-C coupling (32, 60). In the present study, it seems the old had an initial greater perturbation in E-C coupling as shown by the reduced twitch torque and greater low-frequency torque depression compared to the young (Figures 16 and 17). As well, following lengthening contractions a shift to longer muscle lengths for optimal torque production represents an increase in series compliance of the

muscle (29, 64). The presence of overstretched, disrupted sarcomeres in series with still functional sarcomeres results in an immediate shift in optimum length and is considered to be a reliable indicator of muscle damage, as it relates to the number of overstretched sarcomeres (9, 13). An immediate shift in muscle length for optimal torque production following 120 lengthening contractions has been previously observed in the ankle dorsiflexors (39). With our study design utilizing a velocity-dependent contraction task we were not able to record optimal muscle torque-length per se, however based on the same muscle tested and a similar protocol of repeated lengthening contractions we would expect a similar increase in the optimal muscle length-tension relationship as is known to be induced by muscle damage.

The mechanisms responsible for force loss that occur following muscle damage have been reviewed extensively (3, 20), whereas the processes responsible for impairments in shortening velocity have received little attention (16, 42, 64). Data from our study highlight that the effects of fatigue on loaded shortening velocity are independent of muscle damage and the coexistence of fatigue and damage is evident by the time course of the transient effects of fatigue and long-lasting effects of damage. Hence, the combined effects of fatigue and muscle damage more greatly affect the production of shortening velocity and subsequently power than either variable alone following this task. Indeed, voluntary maximal shortening velocity is known to recover rapidly (< 5 min) in young adults after isometric and concentric fatigue tasks (14, 15). Interestingly, following repeated lengthening contractions the velocity component of power does not recover fully (49, 64). In a recent study of young men and women, following repeated lengthening

contractions, power remained reduced up to 30 min following task termination (49). Therefore, long lasting muscle damage appears to limit power production (10, 49) following lengthening contractions 30 min into recovery.

Both the old and young women possibly incurred a similar amount of muscle damage (i.e., prolonged reduction in isometric MVC), yet the old were more fatigable than young as indicated by the greater power-loss up to 10 min into the recovery period. Once the transient effects of fatigue were recovered both groups had a similar reduced power and for this reason, we can argue both groups experienced similar impairments in muscle function owing to muscle damage. However, the old women incurred more fatigue than the young women which can account for the greater power-loss immediately following the lengthening contractions. The loss of power in the old women in the current study following 150 lengthening contractions is less than that observed in studies using protocols of shortening contractions (8, 22, 40). For example, in older men, McNeil *et al*. (2007) found a 20% loss of power for the dorsiflexors following 25 fast shortening contractions and Dalton *et al*. (2010) found a 26% reduction following 50 fast shortening plantar flexion contractions. The greater mechanochemical efficiency for lengthening compared to isometric and shortening contractions result in less perturbation of intracellular high-energy phosphate (P_i) energetics (16, 54). Thus, despite the greater number of contractions in this study than the others, the disparate results can be explained by the task-dependent nature of fatigue (23).

4.3.4 Young vs. old metabolic (dis)advantage. It is well known that older adults are more fatigue resistant than young when performing isometric tasks,

owing to their slower contracting muscle and lower motor unit discharge rates required to reach fused tetanus as indicated by a shift to the left in the force-frequency relationship (5). That is to say, under isometric conditions the lower glycolytic flux in old compared with young is less energetically costly (lower ATP required) with a greater energy turn over through oxidative processes (36), resulting in less metabolic acidosis and accumulation of inorganic phosphates thus mitigating the reduction in isometric MVC torque (33). However, when 'stressed' with repeated dynamic shortening contractions this apparent fatigue resistance in older adults is abolished and in some situations older adults are more fatigable than young (12). This is found exclusively during tasks which allow velocity to be unconstrained (i.e., velocity-dependent) (22, 40, 47). Furthermore, it appears based on the greater power-loss incurred by the old women in this study we now show older adults may be 'energetically' disadvantaged following repeated lengthening contractions, thus further exacerbating fatigue mechanisms related to whole muscle shortening velocity and the subsequent generation of power. The greater accumulation of muscle metabolites during the lengthening contraction protocol in older women impairs E-C coupling and may limit crossbridge function while performing a subsequent fast shortening contraction.

A greater initial impairment in E-C coupling is supported further by the reduced 10:50 Hz ratio and an already impaired capacity for potentiation may have disadvantaged the older adults for the performance of subsequent 'fast' velocity-dependent contractions (Figure 14) compared with the young. By contrast, the young had a greater twitch potentiation and were less influenced by LFTD in the

first 5 min into recovery (Figures 16 and 17). Post-activation potentiation, due to myosin light-chain phosphorylation, can compensate for impaired E-C coupling by increasing myofibrillar calcium sensitivity in spite of the presence of LFTD (28, 52). In our current study, this suggests the young had less of a reduction in myofibrillar calcium sensitivity (27) compared with old, meaning they were less adversely affected by cellular mechanisms of fatigue. This could include: increased H^+ and P_i which directly reduce force output, and can result in a decline in the number and/or force per unit of the strongly bound cross bridges (25) as well as impaired ADP dissociation from the myosin head (25) limiting peak shortening velocity. Following lengthening contractions a failure of the dihydropyridine receptors to stimulate sarcoplasmic reticulum Ca^{2+} release (32), and reduced myofibrillar Ca^{2+} sensitivity together with minimal potentiation capability might have heightened the effects of the 'potentially greater' metabolite accumulation in older adults effect on the impaired generation of velocity-dependent power (25, 62, 63). Whereas, velocity-dependent power in both the old and young women reached a similar value by 10 min into recovery, the greater potentiation in young may have helped offset the initial perturbations in E-C coupling, thus mitigating the reduction in shortening velocity (7) and power at task termination. The greater power loss in older women is likely a result of greater LFTD and E-C coupling failure in the muscles of older compared with young women, as this is also supported by our observation of a greater reduction in doublet twitch rate of torque development in the old women than the young at task termination.

In summary, the damaging lengthening contractions impaired shortening velocity and thus power in both the old and young women, with a greater reduction in the old for up to 10 min into recovery at which time subsequently both remained reduced for the duration of the 30 min recovery period. The observations were not related to neural drive changes but to peripheral alterations primarily affecting E-C coupling. The mechanisms responsible for the reduction in shortening velocity following muscle damage may include decreases in the number of functioning sarcomeres in series, Ca^{2+} kinetics and myofibular Ca^{2+} sensitivity. The greater fatigue in older women can be attributed to their blunted potentiation, a factor in the young which may have helped offset initial fatigue-induced impairments in shortening velocity. Furthermore, our findings highlight the value of investigating changes in the velocity-component of power (i.e., shortening velocity) following perturbations to the neuromuscular system.

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Chapter 5 – A leftward shift in the torque-velocity relationship following muscle damage results in a preferential loss of power at higher loads

5.0 Introduction

Unique to unaccustomed lengthening contractions is muscle damage (25). Mechanical strain imposed upon the muscle fiber during active lengthening (40) results in the disruption of actin-myosin bonds, cytoskeletal damage and impaired excitation contraction (E-C) coupling (4, 28, 46). As well, a redistribution of sarcomere lengths (33) increases series compliance and shifts the torque-length relationship to longer muscle lengths for optimal torque production (21). Damage to the muscle is detrimental to its function (41) by attenuating torque generating capacity, shortening velocity, and as a result, whole muscle concentric power (8, 37, 39, 43). We reported previously that following a bout of damaging lengthening contractions, power was reduced in young men and women similarly and remained reduced throughout short term recovery (35), however power was tested only at a moderate load (i.e., 20% maximum voluntary isometric contraction; MVC). Thus, it remains unclear whether the torque-velocity (T-V) relationship and hence power production over the entire working range of the muscle is altered as a result of muscle damage and whether a sex-related difference is an underlying factor in performance decrements.

Power generation is based on the relationship between torque and velocity. As velocity increases, less torque can be generated owing to fewer cross-bridge attachments, requiring an optimal trade off of torque and velocity to achieve peak

power (2, 29). Following muscle damage, dorsiflexion MVC and shortening velocity (load: 20% MVC) were reduced by 28% and 8%, respectively, in men and women (35). Because muscle damage resulted in a greater loss of maximal torque generating capacity than shortening velocity at a moderate load, and because whole muscle shortening velocity depends on the load resisting the movement (29), it is conceivable that power will be reduced preferentially at higher rather than lower loads. This muscle strategy will reflect impaired torque-generating capacity and the ability to initiate the movement rapidly (rate of torque development; RTD) for adequate power production. As a compensatory mechanism to muscle damage and associated muscle weakness, peak power will 'shift' to lighter loads thus relying on the velocity component of power for optimal performance rather than torque per se. This hypothesis of a preferential loss of concentric power at higher loads following muscle damage is partially supported by studies involving isovelocity (i.e., isokinetic) actions (8). To determine the extent of concentric strength loss following muscle damage an isovelocity model relies specifically on testing the torque component of power when angular velocity is fixed. Some studies report greater impairments at slow angular velocities, thus reflecting impaired torque generation (12, 19, 31) whereas others report greater impairments at fast velocities suggesting shortening velocity is more impaired than torque generation (15, 17, 20). However, the isovelocity contraction mode artificially constrains angular velocity and therefore does not properly represent normal contractile function of the limb. Importantly, when torque is constant and velocity can vary freely, the muscle functions more closely to in vivo conditions (37), and alterations in the power curve

can be explored to offer insight on the mechanisms of power loss following muscle damage.

In contrast to the literature on sex-differences following muscle damage in animals, reports on sex-related differences in response to damaging lengthening contractions in humans are equivocal, or show a greater impairment in women than men [for review see (10)]. Following lengthening contractions in a large sample of men (n=98) and women (n=94), Sayers & Clarkson (2001) reported that a disproportionately higher number of women than men demonstrated greater initial force loss. In addition, despite similar indices of muscle damage in the elbow flexors of both sexes, Sewright *et al.* (2008) showed that immediate strength loss was more prominent in women than men. Because women and men had similar markers of muscle damage, but women had a greater impairment in strength, this finding can be interpreted as E-C uncoupling playing a key role in the observed sex-difference. Muscle damage results in impaired RTD (26), potentially diminishing power production. Thus, in women, muscle damage induced dysfunction may be exacerbated due to a lower RTD compared with men, owing to a lower Type II/Type I fiber area ratio (24) and the potential for greater susceptibility to E-C coupling failure.

Therefore, the purpose of our study was to investigate the effect of repeated high-intensity lengthening contractions on velocity-dependent power loss, and to determine whether a sex-difference exists when assessed across multiple loads. Because torque production is impaired substantially following muscle damage and the velocity at which a muscle shortens depends on the force it is resisting, we

hypothesized there would be a left and downward shift in the power curve with a preferential loss of power at higher loads. However, maximal shortening velocity and shortening velocity at lower loads will be impaired minimally owing likely to fewer cross-bridge interactions, thus avoiding damaged force generators. Finally, to further highlight the role of muscle damage and impaired RTD, which we suspect is a major contributor to power production; we tested women, whom are known to have lower RTD than men. We expect that following damage women will have a greater loss of power at heavier loads than men because of a greater strength loss driven by larger impairments in RTD and more reliance on the velocity component of power.

5.1 Methods

5.1.1 Participants: Eight men (27 ± 3 y, 178.1 ± 7.3 cm, 81.4 ± 10.1 kg) and 8 women (26 ± 4 y, 170.6 ± 6.8 cm, 63.9 ± 6.8 kg) volunteers, who were recreationally active and free from musculoskeletal disorders, were recruited for the study. All participants were asked to refrain from strenuous exercise 1 day prior to and 2 days following baseline testing and not consume caffeine prior to testing. This study was approved by the local University's Review Board for Health Sciences Research Involving Human Subjects and conformed to the Declaration of Helsinki. Informed oral and written consent was obtained from all participants prior to testing.

5.1.2 Experimental arrangement: All testing was conducted on a Biodex multi-joint dynamometer (System 3, Biodex Medical Systems, Shirley, New York). The right foot was fastened tightly to the ankle attachment footplate with inelastic

straps, aligning the lateral malleolus of the ankle with the rotational axis of the dynamometer. Extraneous movements were minimized using non-elastic shoulder, waist and thigh straps. Participants sat in a slightly reclined position with the hip, knee, and ankle angles set at 110°, 140°, and 30° plantar flexion, respectively. All voluntary and evoked isometric dorsiflexion contractions were performed at an ankle joint angle of 30° of plantar flexion, which pilot testing and previous investigations of the dorsiflexors (30, 38) showed to be the optimal angle of torque production. Shortening contractions began from the plantar flexed position of 30° and ended at the neutral ankle angle (0°). To maximize the stretch placed on the muscle, lengthening contractions occurred from the neutral ankle angle until 30° of plantar flexion thus all dynamic actions moved through a 30° range of motion.

5.1.3 Electromyography (EMG): Electromyography of the tibialis anterior was collected using a custom-made insulated stainless steel fine wire electrode (50μm, California Fine Wire Company, Grover Beach, CA) with ~5 mm of insulation removed from the recording tip. The electrode was inserted using a 30 G sterilized hypodermic needle (B-D PrecisionGlide; Becton Dickinson and Company, Franklin Lakes, NJ). Self-adhering Ag-AgCl surface electrodes (1.5 x 1 cm; Kendall, Mansfield, MA) were used to collect surface EMG from the antagonist soleus. Prior to electrode placement and insertion, the skin was cleansed with pre-soaked alcohol swabs. A monopolar electrode configuration was used with the active electrode positioned in the proximal portion of the tibialis anterior at the innervation zone (~7 cm distal to the tibial tuberosity and ~2 cm lateral to the tibial anterior border) (7) and a reference surface electrode was placed over the distal tendinous portion of the

tibialis anterior at the malleoli. The surface active electrode for the soleus was positioned ~2cm distal to the lower border of the medial head of the gastrocnemius and a reference was placed over the calcaneal tendon at the malleoli. The ground electrode for both tibialis anterior and soleus EMG configurations was positioned over the patella.

5.1.4 Electrical stimulation: Stimulated contractions of the dorsiflexors were evoked electrically using a bar electrode firmly held distal to the fibular head over the deep branch of the common fibular nerve. 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. Peak twitch torque (P_t) was determined by increasing the current until a plateau in dorsiflexor P_t and tibialis anterior compound muscle action potential (M-wave) peak to peak amplitude were reached. Then, the current was further increased by at least 15% to ensure activation of all motor axons via supramaximal stimulation. This stimulation intensity was used for the evoked doublet (P_d) (2 pulses at a 10 ms interpulse interval) to assess voluntary activation. Finally, a 10 Hz (5 pulses over 0.5 s) and 50 Hz stimulus (25 pulses over 0.5 s) was delivered to assess peak tetanic torque by increasing the current until there was a plateau in evoked 50Hz torque.

5.1.5 Maximal voluntary isometric contraction (MVC): Three isometric MVCs were performed, each of 3-5 s in duration. Three min of rest was given between all contractions. To ensure MVC attempts were maximal, participants were provided visual feedback of the torque tracing on a computer monitor, and exhorted verbally during all voluntary efforts. Voluntary activation was assessed using the modified

interpolated twitch technique. The amplitude of the interpolated torque evoked during the peak plateau of the MVC was compared with a resting P_d evoked ~1s following the MVC when the muscles were relaxed fully. Percent voluntary activation was calculated as voluntary activation (%) = [1 - interpolated P_d /resting P_d] x 100%. Values from the MVC with the highest peak torque were used for data analysis. Next, electrical stimulations at tetanic frequencies were delivered to determine a 10Hz to 50Hz relationship using the current required to evoke peak 50Hz torque.

5.1.6 Power curve determination: Once MVC torque was determined, the dynamometer was switched from the isometric to isotonic mode. However, due to inherent mechanical limitations of the dynamometer (i.e., unable to maintain an exact constant external load throughout an entire range of motion), these contractions are neither purely isotonic, nor are they iso-inertial as the load is fixed (mechanically) and velocity of contraction is determined by the effort of the participant. Therefore we refer to these contractions as "velocity-dependent" (37). A velocity-dependent movement is characterized by a participant producing a dynamic contraction as fast as possible without any constraint in the angular velocity while the load or resistance is maintained at a pre-determined value (i.e., %MVC). Before the footplate will move during the velocity-dependent shortening contraction, the pre-programmed resistance has to be overcome by the participant. The dynamometer absorbs this increase in applied torque resulting in a directly proportional increase in angular velocity. Velocity-dependent contractions allow us

to explore the effect of damaging, lengthening contractions on alterations in velocity of unconstrained movement and power.

The dynamometer was programmed to allow the footplate to return to 30^{o} of plantar flexion at the end of each shortening voluntary contraction while the leg muscles were relaxed fully. Familiarization with these shortening contractions involved participants performing 5 velocity-dependent shortening contractions at a moderate load (20% MVC) until a stable value was obtained (no change in maximal shortening velocity). To ensure a maximal effort (peak velocity) contraction, all participants were instructed to move the load "as hard and as fast as possible throughout the entire range of motion", and provided verbal encouragement and visual feedback of the velocity profile via a computer monitor. Participants rested for 3 min and then performed 2 consecutive velocity-dependent contractions at each of the 8 pre-determined loads with 30 s between attempts, with the peak value of each contraction used to establish baseline values for maximum shortening velocity and peak power. Power curves were then constructed from those values from the contractions with the highest peak velocity obtained from 1N·m to 70% MVC. Power curves were plotted and fitted by the Hill equation (23) (SigmaPlot 12, Systat Software Inc. Washington USA.). To investigate changes in the series compliance of the muscle, optimal angle of torque production during a slow (30^{o}/s) isokinetic contraction was performed. Participants first practiced 2 of these contractions and following 3 min rest performed 2 more for baseline values. The attempt with the greater torque value was used for analysis.

5.1.7 Damage and recovery protocol: Participants performed 5 sets of 30 eccentric isokinetic dorsiflexion contractions at 60°/s with each set separated by 30s of rest. Participants were provided visual feedback of the torque and instructed to resist maximally the lowering of the foot plate through the full 30° range of motion. The foot was then returned to the neutral ankle position by the dynamometer over a 1 s period while the leg muscles were relaxed fully. The voluntary and electrically evoked responses of the dorsiflexors were recorded at baseline, during the protocol immediately following each of the 5 sets, immediately following task termination, and during recovery at: 2.5 min, 5 min, 10 min, 15 min, 20 min, 30 min, 24 hr and 48 hr. Neuromuscular measures following the protocol included, and were performed in the following order: (1) maximum evoked twitch properties, (2) assessment of MVC and voluntary activation, (3) post-activation twitch and twitch doublet, (4) measure of low frequency torque depression (10:50 Hz ratio), (5) torque –velocity relationships (1N·m-70% MVC) and (6) determination of optimal angle of torque production (30°/s isokinetic contraction) (see experimental protocol: Figure 18). Muscle soreness was assessed subjectively by the participant using a 100 mm visual analog scale, with 'no soreness' (0 mm) and 'severe soreness' (100 mm) serving as the left and right anchors, respectively.

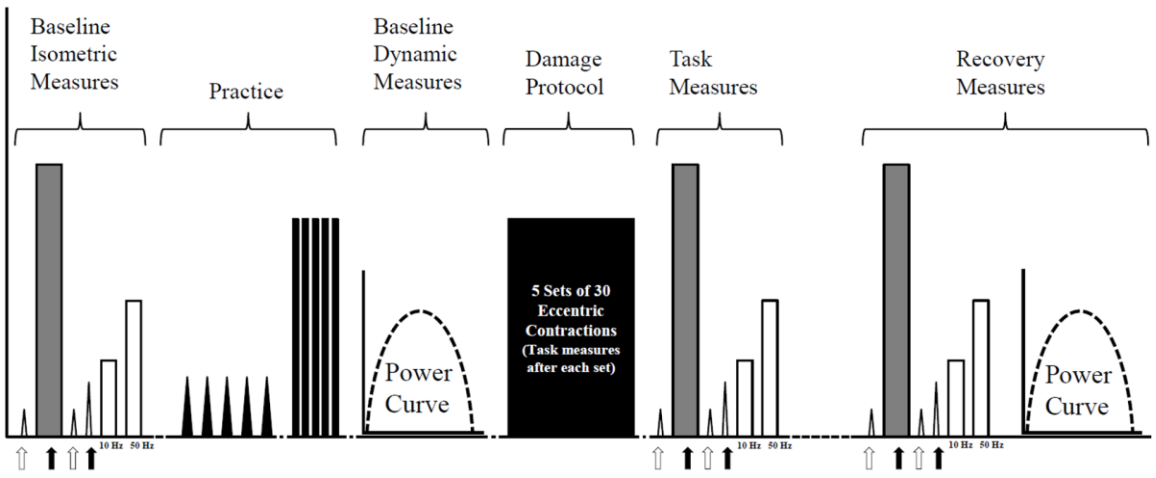

Figure 18. Schematic diagram of experimental protocol.

Grey bars are isometric maximum voluntary contractions (MVC). Open torque profiles are electrically evoked contractions (twitches, doublet, 10, 50 Hz). Filled profiles are dynamic contractions; concentric at 20% MVC (triangles), and dynamic eccentric contractions at 80% MVC (rectangles). Power curves indicate dynamic contractions performed under the following loads: 1N·m, 10%, 20% 30% 40% 50% 60% 70% of MVC. Open arrows are electrically evoked twitches; and filled arrows are electrically evoked doublets. Recovery time points: immediately, 2.5, 5, 10, 15, 20, 30 min, 24 and 48 hr.

5.1.8 Data reduction and analysis: Torque, position and velocity data were sampled at a rate of 100Hz. All data were converted to digital format using a 12-bit analog-to-digital converter (model 1401 Power, Cambridge Electronic Design, Cambridge, UK). Electromyographic signals were pre-amplified (x100), amplified (x2) and band-pass filtered (10-1,000Hz), and sampled online at 2500Hz using Spike 2 software (version 7.07, Cambridge Electronic Design Ltd). Dorsiflexor EMG from the MVC attempt was expressed as a root mean square (RMS) value over a 1 s epoch about the peak torque and soleus EMG during that period was used to calculate co-activation as soleus:tibialis anterior EMG x 100%. All subsequent MVC RMS values were normalized to the level obtained during baseline. For EMG analysis during the dynamic tasks and to estimate the maximal rate of neuromuscular activation the RMS value was calculated from the onset of voluntary EMG activity to the point at which peak RTD was achieved. Then, the signal was integrated as a function of time and a slope of best fit was derived to assess the rate of activation (mV/s) (11). Muscle power (W) was calculated as the product of torque (N·m) and the peak shortening velocity (rad/s) of the faster of the 2 contractions. Post-activation potentiation was determined by calculating the ratio between the amplitude of the peak twitch torque recorded before and following the isometric MVC. Spike 2 software was used off line to determine the peak twitch torque (P_t), peak doublet torque (P_d), doublet time to peak twitch ($_D$TPT), doublet half relaxation time ($_D$HRT), 50Hz HRT, rate of torque development (RTD) and optimal angle of torque production during a slow (30^o/s) isokinetic shortening

contraction. Low frequency torque depression was calculated using a ratio of peak 10Hz to peak 50Hz evoked torques (10:50Hz).

5.1.9 Statistical analysis: Using SPSS software (version 16, SPSS Inc. Chicago, IL) a two-way (sex x time) repeated measures ANOVA was performed to assess all neuromuscular data. Because voluntary activation values were not normally distributed, a Mann-Whitney U-test was employed. Unpaired t-tests were used for subject characteristics and baseline measures to assess group differences. The level of significance was set at $p<0.05$. When a significant main effect or interaction was present, post hoc analysis using unpaired t-tests was performed with a Modified Bonferroni correction factor to determine where significant differences existed. The tabulated and text data are presented as means ± standard deviations (SD); and the data represented in the figures are presented as means ± standard errors (SE), normalized to baseline (pre-test).

5.2 Results

5.2.1 Baseline measures: As shown in Table 4 women were 34% weaker than men for MVC torque ($p<0.01$) despite similar values for voluntary activation (~95%, P=0.68) and optimal angle of torque production ($p=0.78$). Women had a 20% lower isometric MVC_{RTD} ($p<0.01$) than men. Maximal "unloaded" (1 N·m) shortening velocity (Figure 19) was 13% slower ($p<0.01$), and peak power (Table 4) was reached at a 19% lighter load ($p<0.01$), and was 49% less in women than men ($p<0.01$). Women had a lower RTD across loads compared to men ($p<0.05$), but

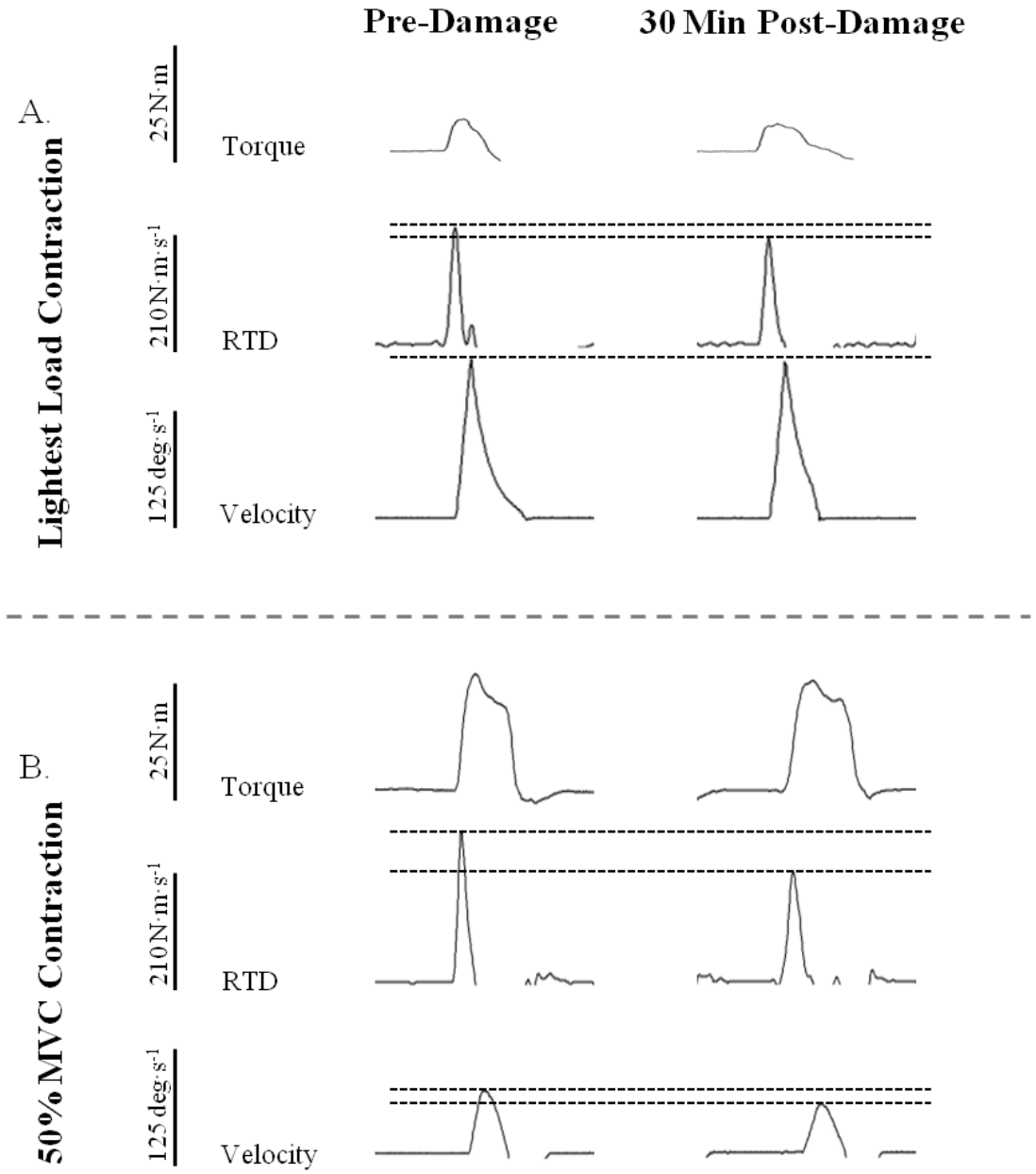

Figure 19. Unprocessed data

A representative male participant depicting a (A.) lightest load contraction and (B.) 50% MVC velocity-dependent contraction. Maximal voluntary isometric contraction (MVC) torque. Rate of torque development (RTD).

Neuromuscular Properties of the Dorsiflexors

	Electrically Evoked Isometric Properties								
Group (n=8)	P_d (N·m)	10Hz (N·m)	50Hz (N·m)	$50Hz_{HRT}$ (ms)	MVC (N·m)	MVC_{RTD} ($N·m·s^{-1}$)	V_{MAX} ($deg·s^{-1}$)	PP (W)	$\%MVC_{PP}$ (%)
Male	14.2 ± 5.0*	15.4 ± 5.9*	30.3 ± 9.8*	137.8 ± 13.7	46.8 ± 11.2*	244.5 ± 43.9*	167.5 ± 12.4*	36.6 ± 10.5*	46.3 ± 0.2*
Female	9.8 ± 1.6	10.3 ± 1.7	18.6 ± 3.2	138.7 ± 7.0	30.7 ± 3.9	194.6 ± 21.3	145.6 ± 10.3	18.5 ± 5.1	37.5 ± 0.2

Table 4. Voluntary and electrically evoked neuromuscular properties of the dorsiflexors.

Women had lower absolute evoked peak doublet twitch torque (P_d; $p<0.05$), 10Hz peak torque ($p<0.05$), 50Hz peak torque ($p<0.05$) compared with men, but 50Hz half relaxation time (50 Hz_{HRT}, $p=0.87$) was not different. Maximal voluntary isometric contraction (MVC) torque ($p<0.01$), Rate of torque development for MVC (MVC_{RTD}; $p<0.01$), maximum shortening velocity (V_{MAX}; $p<0.01$), and peak power (PP; $p<0.01$) were less in women than men. The percentage at which peak power ($\%MVC_{PP}$) was achieved was lower for women than men ($p<0.01$). * Denotes significant sex difference.

similar levels of rate of activation of EMG (p=0.54). Rate of torque development and rate of activation of EMG at each relative load are presented in Tables 5 and 6, respectively. Women had a 31% lower P_d than men (p<0.05), whereas groups did not differ for doublet contractile properties ($_D$TPT; p=0.58 and $_D$HRT p=0.86; Table 4), and both sexes had a similar capacity for twitch potentiation (124 ± 16.0%) (p=0.98).

Intensity	Baseline		Post-Damage		Percent Change		30 Min Recovery		Percent Change	
(%MVC)	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men
Unloaded	159.8±8.7	212.9±32.3	147.6±21.1	205.5±34.7	7.6%	3.5%	142.2±17.2†	197.9±26.8	11.0%	7.0%
10	159.6±12.7	226.4±46.1	150.0±20.2	214.5±34.8	6.0%	5.3%	136.8±24.6†	201.8±35.0	14.3%	10.9%
20	164.4±12.6	252.5±57.3	166.5±12.8	214.2±43.4	-1.2%	15.2%	144.0±23.5†	231.1±51.1	12.4%	8.5%
30	182.1±17.4*	259.4±53.9	169.2±11.3	257.2±44.3	7.1%	0.8%	154.9±18.6†	226.5±44.1	14.9%	12.7%
40	180.3±18.8*	270.6±59.3	170.8±23.6	244.5±58.7	5.3%	9.6%	156.5±20.1†	239.5±45.6	13.2%	11.5%
50	186.9±21.2*	276.1±45.4*	167.5±24.4	227.9±38.9	10.4%	17.5%	157.9±21.6†	229.7±47.8	15.5%	16.8%
60	192.4±21.2*	275.2±56.5*	171.9±33.2†	241.7±55.8	10.7%	12.2%	154.6±16.8†	251.7±52.9	19.6%‡	8.5%
70	191.1±19.6*	272.6±51.8*	158.8±15.1†	257.1±50.6†	16.9%‡	5.7%	148.2±22.2†	240.4±43.6†	22.5%‡	11.8%
MVC	194.6±21.3*	244.5±43.9*	127.3±32.2†	224.5±43.6†	34.6%‡	8.3%	141.2±16.1†	213.0±44.3†	27.4%‡	13.0%

Table 5. Dynamic rate of torque development RTD; $N \cdot m \cdot s^{-1}$ values preceding and succeeding muscle damage

Women had lower RTD across all loads compared to men at baseline ($p<0.01$). Following muscle damage, both sexes experienced impaired RTD ($p<0.05$). Women had a greater loss of RTD at higher loads (60, 70% MVC) than men ($p<0.01$). * = Difference in RTD across loads at baseline † = Effect of Time relative to baseline ‡ = Sex difference

Intensity	Baseline		Post-Damage		30 Min Recovery	
(%MVC)	Women	Men	Women	Men	Women	Men
Unloaded	2.98±1.67	3.50±2.11	3.83±1.67	2.48±1.13	2.21±0.87	2.84±1.72
10	2.55±1.36	3.38±2.29	3.18±1.12	3.92±2.36	3.31±1.35	3.45±1.76
20	2.42±0.60	2.74±1.79	2.75±1.37	2.64±1.52	3.40±1.07	3.01±1.78
30	3.10±2.01	2.66±1.23	2.31±1.07	2.70±1.86	2.58±0.76	3.50±1.79
40	3.48±0.98	2.41±1.82	2.81±1.55	2.62±1.45	2.63±1.37	2.13±0.93
50	3.25±2.22	2.64±1.96	2.53±1.29	2.40±0.85	2.54±0.99	2.19±1.38
60	2.24±1.18	1.79±1.11	2.82±1.86	2.25±1.50	2.66±0.82	2.60±1.80
70	2.41±1.22	2.15±0.68	1.70±1.30	2.64±0.94	3.28±1.44	1.88±1.20

Table 6. Rates of neuromuscular activation (mV/s) preceding and succeeding muscle damage.

Rate of activation of the tibialis anterior was maintained similar to baseline in both men and women (p=0.61) throughout the task and recovery (p=0.83).

5.2.2 Markers of muscle damage: Participants reported no muscle soreness preceding the lengthening contraction task (0 mm) and mild to no muscle soreness within the first 30min of recovery (13.1 mm). Soreness peaked 24 hr post task termination (27.6 mm) and returned to mild soreness (9.4 mm) within 48 hr, with no detectable differences between groups ($p>0.05$). For dorsiflexor MVC torque there was a significant time x sex interaction ($p<0.05$, ES=0.15). Isometric MVC torque decreased similarly in the men and women during the task (30%), but women had a delayed recovery compared with men from 10 min-24 hr. Women recovered to only 81.3 ± 5.5%, of baseline whereas men recovered to 89.2 ± 8.1% by 24 hr following task termination (Figure 20A), suggesting women incurred more damage induced dysfunction than men. By 48 hr both men and women had recovered. Voluntary activation in women and men was well-maintained throughout the task (greater than 95%, $p=0.91$, ES=0.02) and recovery. The optimal angle of torque production showed a main effect for time (range: 2-8^{o}; $p<0.01$, ES=0.46) in both men and women and did not recover by 24 hr. The incomplete recovery of MVC by 24 hr post lengthening contractions and the shift to longer muscle lengths for the optimal angle of torque production indicates muscle damage occurred in both sexes.

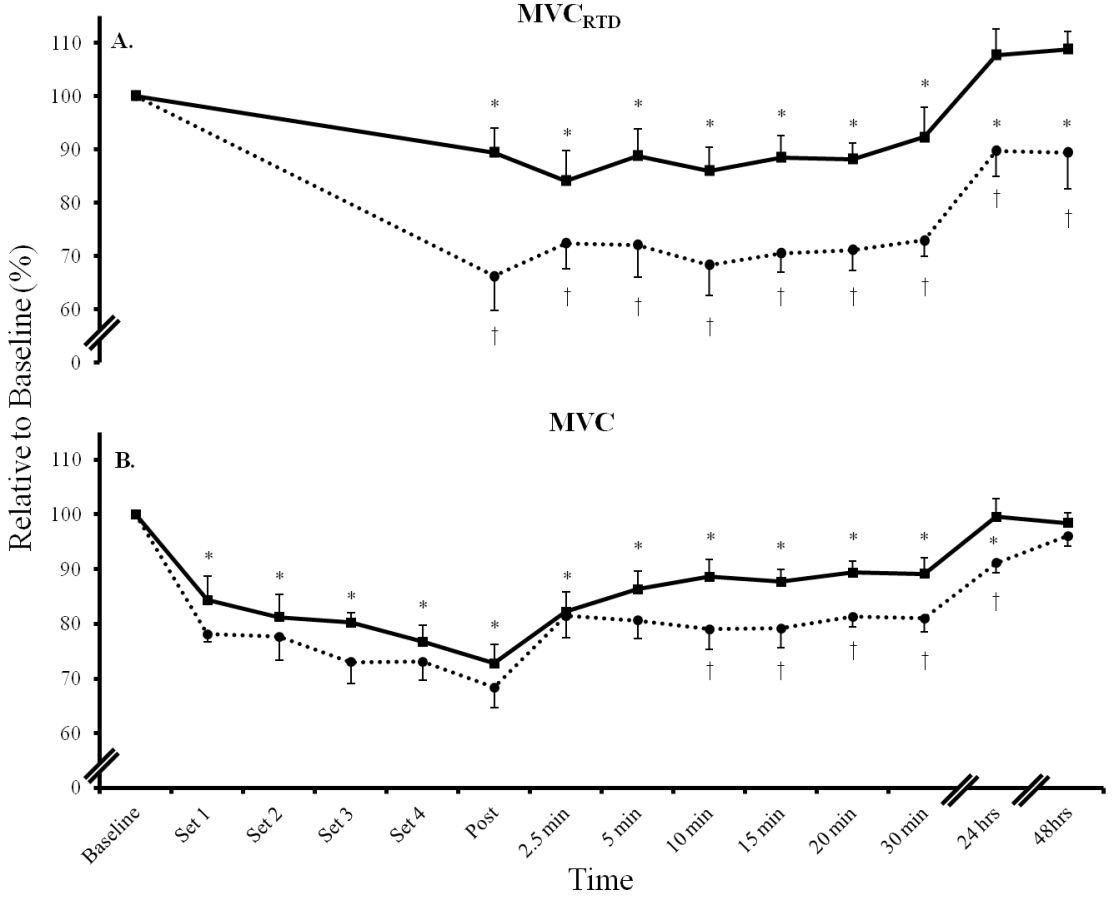

Figure 20. (**A.**) Maximal rate of torque development and (**B.**) maximal voluntary isometric contraction before and following muscle damage.

Maximal rate of torque development was impaired more in women than men, with the men recovering by 24 hrs while women did not recover fully by 48 hrs. Maximal voluntary strength was reduced similarly in men (solid line; closed box) and women (dotted line; open circle), with a reduced recovery in women. Effect of time (*), effect of sex (†), values are means ± SE

5.2.3 Lengthening contraction task and recovery measures: All participants were able to complete the lengthening contraction task, and eccentric torque for both men and women decreased similarly during this protocol (p=0.27, ES=0.11). Neuromuscular measures were analyzed and compared with baseline and reported as a relative change over time. For maximum shortening velocity (Figure 21A) and peak power (Figure 21B), there were main effects for time (p<0.01, ES=0.42 and p<0.01, ES=0.50, respectively), and a trend of a main effect for sex during recovery for shortening velocity (p=0.07, ES=0.22) and peak power *(*p=0.06, ES=0.24), respectively. At task termination both men and women had a similar 3% loss of maximal shortening velocity and a 10% loss of peak power. The reduction in maximal shortening velocity persisted until 30 min of recovery, whereas peak power did not recover until 24 hr.

For velocity-dependent power loss across loads (Figure 22A-H) there was a time × sex × load interaction (p<0.01, ES=0.12), with main effects for each variable: time (p<0.01, ES=0.38), sex (p<0.01, ES=0.17) and load (p<0.01, ES=0.23). Women had a greater loss of power at heavier loads than men, and both sexes showed a preferential loss of power at the heavier loads resulting in a shift down and to the left for the power curves (Figure 23). Both sexes recovered similarly when tested at lighter loads (<30%MVC), but women had a slower recovery than men for heavier loads (p<0.01, ES=0.33) recovering by 48 hrs, whereas men recovered by 24 hrs. For RTD determined across loads (Table 5) there was a main effect for time (p<0.01 ES=0.63) and sex (p<0.05, ES=0.29) with RTD recovering in women by 48 hr and in men by 24 hr. Women had a greater loss of RTD at higher loads (60, 70% MVC) than

men (p<0.01). As reported in Table 6, the tibialis anterior EMG rate of neuromuscular activation remained similar to baseline throughout recovery (p=0.76, ES=0.07) and did not differ between men and women (p=0.66, ES=0.08), suggesting impairments in neuromuscular function occurred distal to the neuromuscular junction.

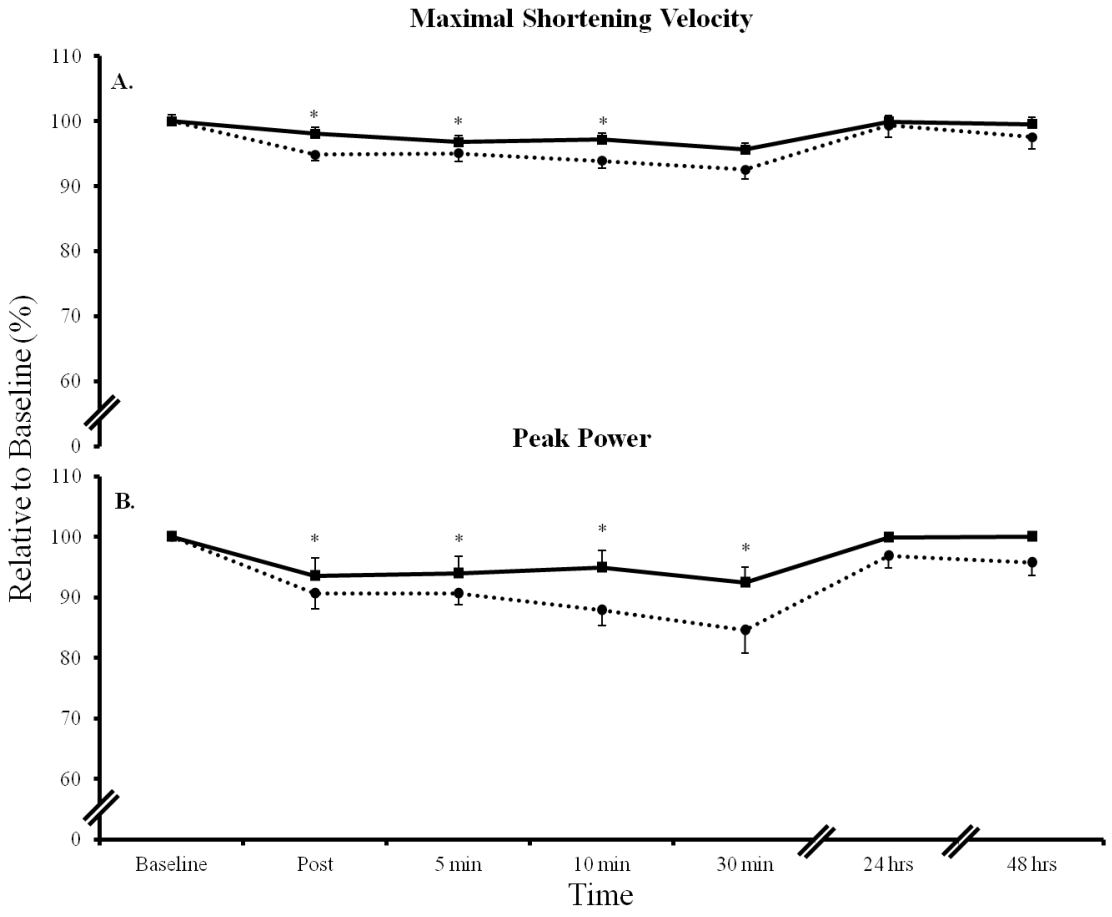

Figure 21. (A.) Maximal shortening velocity and **(B.)** peak power.

Maximal shortening velocity was impaired minimally in both men (solid line; closed box) and women (dotted line; open circle) and recovered fully by 30 min. Peak power shifted to lighter loads and was reduced similarly in men and women by 10% and recovered fully by 24 hrs. Effect of time (*), values are means ± SE

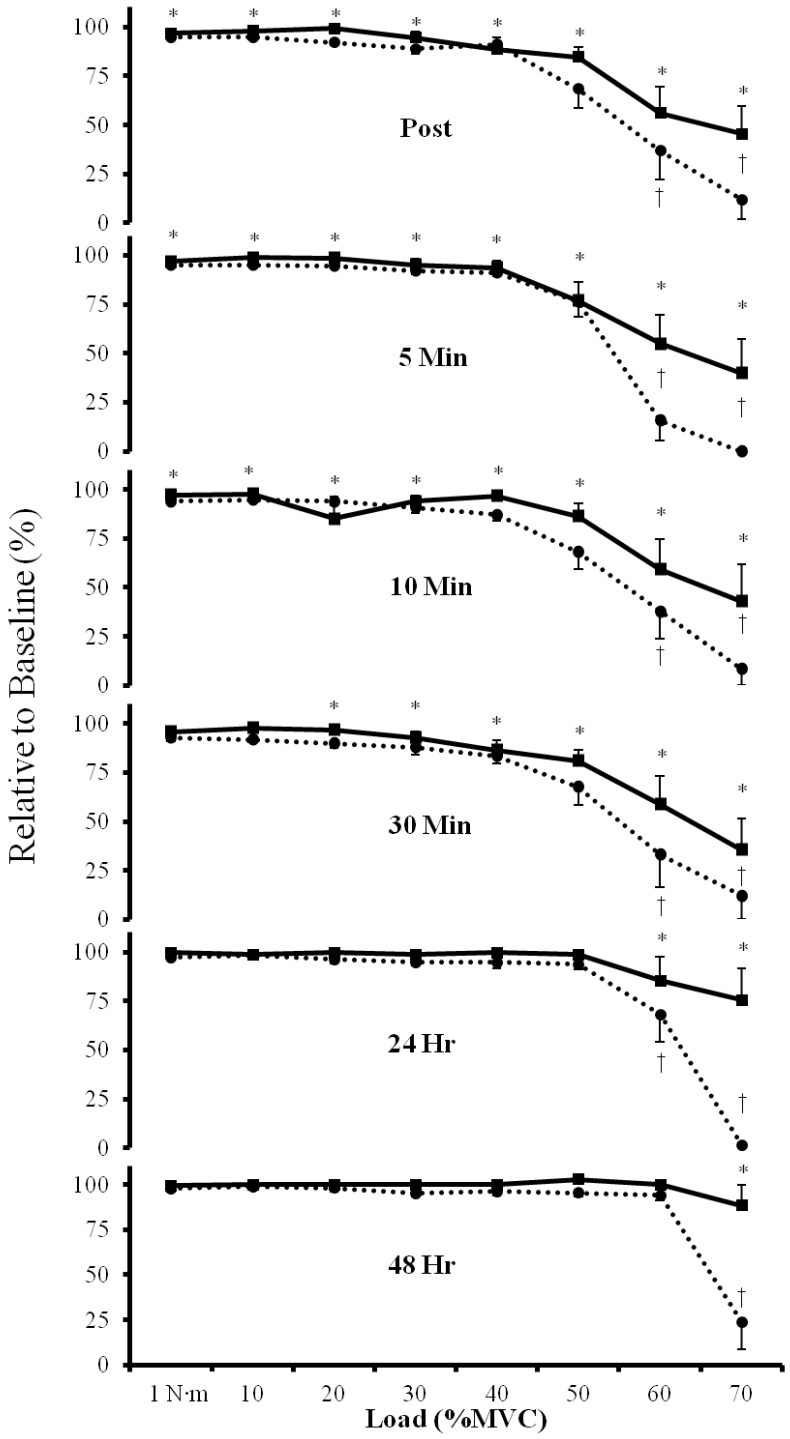

Figure 22. Power loss across multiple loads following muscle damage.

Men (solid line; closed box) and women (dotted line; closed circle). Effect of time (*), effect of sex (†), values are means ± SE

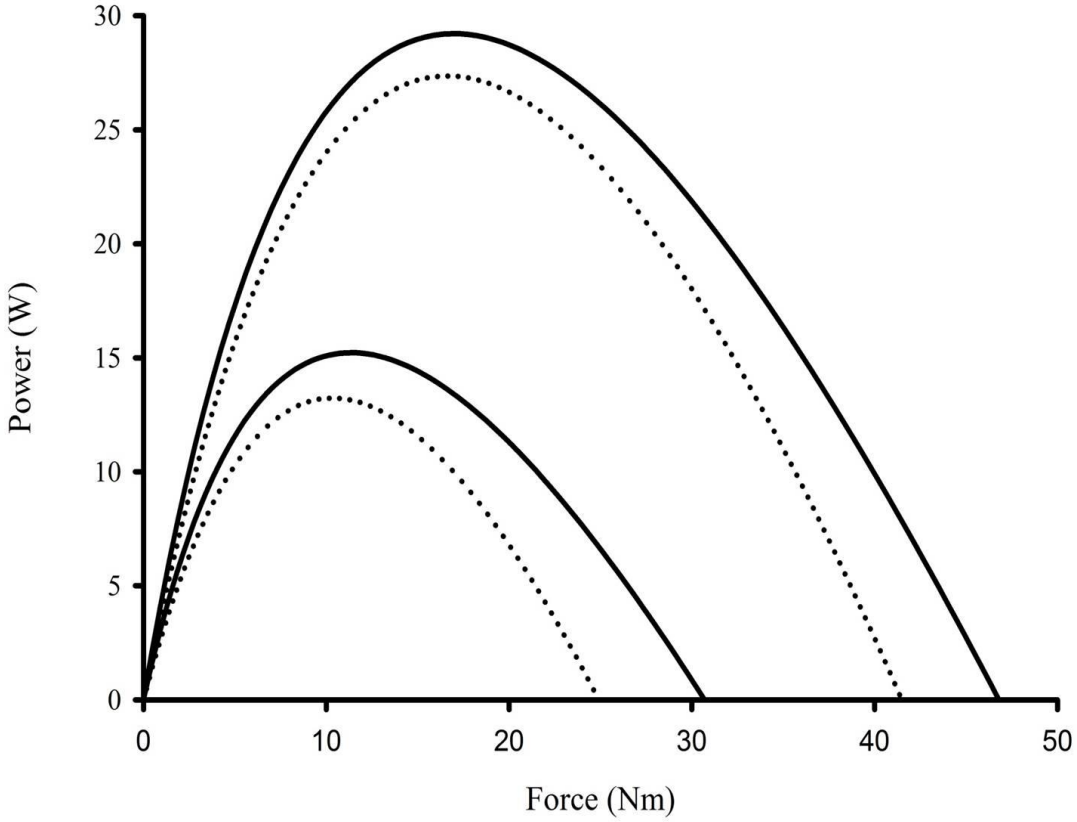

Figure 23. Power curves

Men (upper curve) and women (lower curve), preceding (solid line) and succeeding (dotted line) damaging lengthening contractions.

Rate of torque development for the isometric MVC showed a significant time × sex interaction (p<0.05, ES=0.13) and decreased more in women (66.2 ± 18.1% of baseline) than men (89.5 ± 12.9% of baseline). Rate of torque development for the MVC remained reduced in both sexes throughout the short-term 30 min recovery period, but men recovered by 24 hrs while women remained reduced throughout recovery (figure 20B). There was a significant effect for time (p<0.01, ES=0.57), but not sex (p=0.48, ES=0.04) for low frequency torque depression (10:50Hz). Men and women had a similar decrease to 83.6 ± 16.7% of baseline by 30 min and recovered within 48 hr. The alterations in the 10:50 Hz ratio was manifested by the greater reduction in 10 Hz evoked torque compared with the 50 Hz (Figure 24A-C). During the task, 50 Hz decreased (P<0.05, ES=0.16) similarly in women and men and recovered by 24 hr. The 10 Hz torque was significantly reduced throughout recovery in both men and women (p<0.05, ES=0.03). Women experienced an incomplete recovery (p<0.01, ES=0.03) up to 48 hr whereas men recovered by 24 hr (Figure 24B). Reductions in the 10:50 Hz ratio indicated there was significant low frequency torque depression following the lengthening contractions for both men and women, but a greater impairment in women throughout recovery.

For twitch torque (Figure 25) there was a main effect for time (p<0.01, ES=0.68). Twitch torque was potentiated and increased to 127 ± 17.7% immediately post task in both men and women. By 30 min recovery the potentiating effects decayed and twitch torque was reduced to 82.4 ± 17.5% of baseline. Electrically evoked contractile speeds only showed main effects of time for

$_{D}$TPT (p<0.01, ES=0.56), and $_{D}$HRT (p<0.01, ES=0.21), thus slowed similarly in both groups by 15-20% compared with baseline.

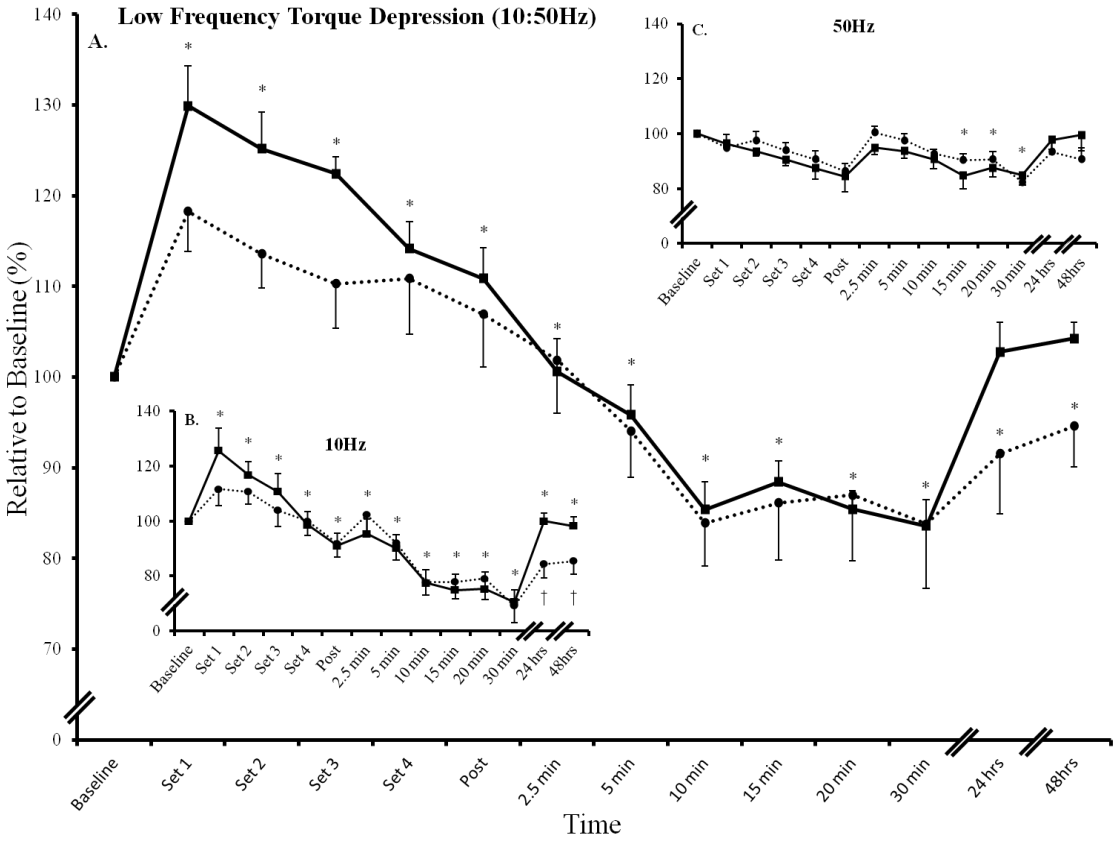

Figure 24. (A.) Low frequency torque depression as a combined consequence of impaired **(B.)** 10 Hz and **(C.)** 50 Hz torque.

A significant increase in low-frequency torque depression in men (solid line; closed box) and women (dotted line; open circle), as shown by the decreased 10:50 Hz was present throughout recovery, with a trend towards greater LFTD in women (p=0.072). The decreased ratio was driven by the 10 Hz component which also presented a sex difference throughout recovery. Effect of time (*), effect of sex (†), values are means ± SE

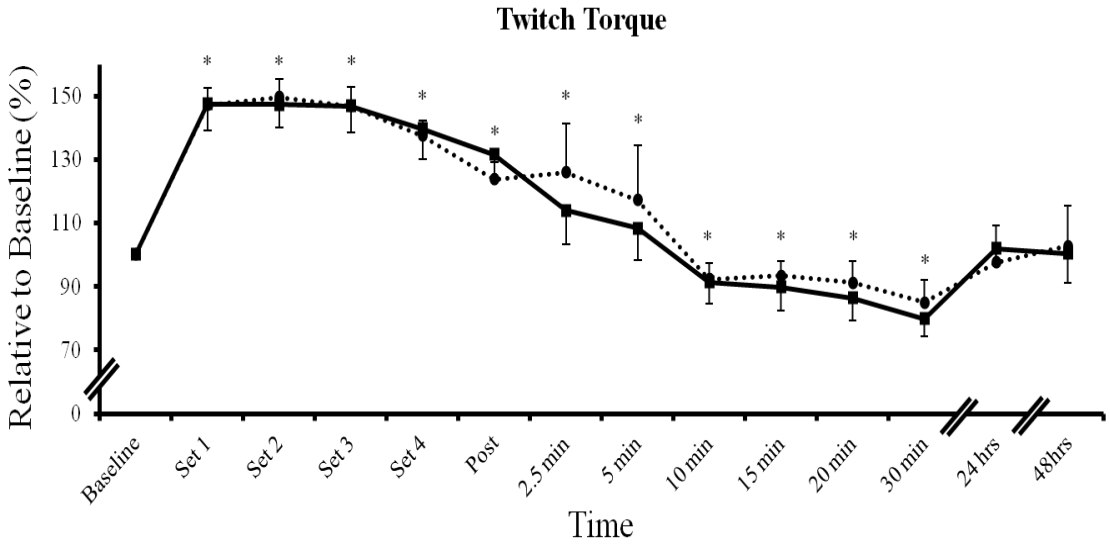

Figure 25. Peak twitch torque.

Peak twitch torque was potentiated similarly in women and men throughout the lengthening contraction task and was impaired similarly throughout recovery. Effect of time (*). Values are means ± SE

5.3 Discussion

We investigated the effects of repeated high-intensity lengthening contractions on velocity-dependent power loss. Muscle damage induced a left and downward shift in the power curve with a preferential loss of power at higher loads (Figures 22, 23). Meanwhile, maximal shortening velocity was impaired minimally owing to the transient effects of fatigue which recovered fully within 30 min. In line with our hypothesis, an effect of sex was observed in which women had a 50% greater loss of power than men when tested at loads >50% MVC, and this effect persisted throughout recovery. However, peak power was reduced similarly (10%) in both sexes and recovered fully within 24 hr. The greater loss of power at heavier loads in women than men appears to be driven by those factors affecting strength loss and not maximal shortening velocity as reflected fundamentally by the inability to generate torque rapidly (i.e., rate of torque development).

5.3.1 Strength loss Although baseline MVC torque was 34% greater in men, relative strength loss was similar in both groups throughout the task and within the first 10 min of recovery, but thereafter, recovery of strength between the groups was divergent. Men recovered fully within 24 hr, whereas women did not recover until 48 hr following lengthening contractions (Figure 20A). This finding is consistent with sex-related differences in response to damaging lengthening contractions observed previously (44, 45), although others have found no sex difference (26, 42). In the present study, men recovered to 89% of baseline during the 30 min recovery period, but women recovered to only 81% of baseline. Presumably, sex-differences in response to repeated dynamic contractions

contributed to the similar initial fatigue-induced decline in MVC in men and women and their different recovery profiles. In the present study, because women are more fatigue resistant during repetitive dynamic tasks (22, 27) the initial decline in men could have been attributed to a greater fatigue response. The divergent recovery profiles indicate that women experienced a greater level of damage induced impairment in muscle function leading to a delayed recovery of MVC compared with the men.

Previously, we reported no change in neuromuscular activation (RMS EMG) or voluntary activation following exercise-induced muscle damage (35-37), and these results are in line with our current study in which the rate of neuromuscular activation did not differ from baseline for either sex (Table 6). This indicates peripheral mechanisms are fundamentally responsible for decrements in performance following lengthening contractions in this model. Both sexes had a similar increase in muscle soreness and increase in muscle series compliance as indicated by the shift in optimal angle for torque production to longer lengths (2-8^{o}). Although structurally, men and women seem to have experienced a similar level of muscle damage, there remained a greater degree of strength loss in the women compared with the men. To account for this disparity, it is reasonable to suggest that E-C coupling was more impaired in women than men, which is supported by a 15% greater reduction in the 10 Hz torque for the women by 24 hrs recovery (Figure 24B). The main contributor to E-C uncoupling following muscle damage is impaired Ca^{2+} release (13, 28). Thus, sex-related differences in E-C uncoupling may also be related to a greater impairment in Ca^{2+} channel regulation following muscle

damage in women than men in response to differing sex hormone levels as suggested for cardiac myocytes in mice (16). Although a direct link between cardiac myocytes and skeletal muscle fibers has not been made for this pathway it is conceivable that the sex-difference in E-C uncoupling following damage could be attributed to less Ca^{2+} released from the female SR owing to a smaller safety factor. Although muscle damage was similar between sexes, the women were less able than the men to engage a sufficient number of viable force generators.

5.3.2 Velocity and power Strength loss and the velocity reductions at each relative load altered the T-V relationship and thus power production following the damaging lengthening contractions. The weakened muscle required a higher percentage of initial MVC to accomplish relative movements successfully. Peak power was reduced similarly in men and women, whereas power assessed at heavier loads was attenuated to a greater extent than when power was assessed at lighter loads in both sexes. Following the damaging contractions, power did not recover at heavier loads (i.e., > 60% MVC) for either sex, but for the unloaded and lighter loads (<20% MVC), power recovered within 30 min with no sex difference. The shift in peak power to lighter loads allowed the muscle to compensate for the damage associated muscle weakness by relying more on the velocity component for power generation with less dependence on RTD and damaged force generators.

Dynamic performance is dependent highly upon ballistic force generation which is related to the ability to generate torque rapidly and can be quantified by voluntary RTD (1, 5). Following the lengthening contractions in our study, men had a 20% reduction in RTD during the MVC whilst women exhibited a 35% decrease.

Because voluntary activation and RMS EMG amplitude were unchanged, the impairment following muscle damage was dependent on factors distal to the neuromuscular junction. Failed activation of intact force-generators (i.e., E-C coupling failure (6, 46)), and disruption of the force transmitting structures resulted in the inability to generate force rapidly. The manner in which torque is generated (i.e., developed quickly) appears to play a critical role in muscular performance following damage, such that, there may be a critical threshold value of RTD to perform the task successfully. During the dynamic contractions, across varying loads, women had a 15-18% loss of RTD compared with the 7-9% loss in men. Therefore, for loaded contractions representing a higher percentage of one's MVC, if the individual's maximal RTD is impaired below the requisite needed to initiate movement rapidly, power will be impaired greatly. On the contrary, performing fast movements with light to moderate loads that represent a low percentage of one's MVC, the critical threshold for RTD to move the load quickly is not stressed and performance is unimpeded by muscle damage.

Muscular power is defined by the T-V relationship, however the torque-length relationship governs the ability of a muscle to develop force throughout a range of motion, thereby contributing prominently to the generation of maximal power production (2, 29). The increase in muscle series compliance as shown by the change in optimal angle of torque production to longer muscle lengths could have contributed to power loss, particularly the ability to generate torque at higher loads. Women reached peak power at a lower %MVC than men, therefore, the additional loss of strength following muscle damage created an even greater loss of

power at heavier loads compared with men. Thus, mechanisms of dynamic force generation are affected more in women than men following damage. The inability to generate high torques rapidly at the onset of exercise, owing to increased series compliance and E-C uncoupling appears to be the underlying factors responsible for the loss of power following lengthening contractions, particularly at higher loads in women.

Power loss at lighter loads recovered quickly, thus representing a fatigue-related impairment in maximal shortening velocity. Neuromuscular fatigue can result from both metabolic and non-metabolic factors such as the accumulation of metabolites (3) and central nervous system impairments (18). Ultimately, fatigue has transient effects on power production as indicated by the recovery of power (< 20% MVC) by 30 min, whereas muscle damage creates longer lasting impairments (9, 32). Because of the relatively transient nature of metabolic perturbations, their slowing effect on maximal shortening velocity does not contribute to the prolonged impairment in velocity-dependent power.

5.3.3 Velocity specific alterations in power The change in maximum velocity reached at each load following muscle damage is consistent with velocity-specific alterations in the T-V relationship. As reported previously (13), when velocity-dependent power is tested at a moderate load following damage, the compromised torque production of the muscle is less stressed (< 20% MVC) as the velocity of contraction was fast with little need for high torque generation. The current study emphasizes that at higher loads, velocity is indeed compromised greatly because the weakened muscle was unable to produce the requisite torque to move the load

quickly and powerfully. These results indicate that a relationship exists between the impairment in force generation capacity following muscle damage and velocity-dependent power production. This is observed during isovelocity movements in which the constrained velocity is high enough to not impede torque production (19). However, when tested at faster velocities the isovelocity model does not permit the relative impact of force and velocity on power production to be discriminated.

Velocity-specific alterations in power following muscle damage have been recently disregarded (14). It was reported that power-loss was affected equally across a range of pedaling cadences which corresponded in knee extension velocities between 2.5-5.7 rad/s (i.e., 140-325°/s) (14). Due to the fast knee extension velocities, it is apparent this mode of testing did not stress the torque component of power and therefore any velocity-specific alteration in power could not have been observed. Previously, we (35) suspected the decline in velocity-dependent power was driven by the decline in maximal shortening velocity. However, when power was tested across multiple loads in the present study we can conclude confidently that torque impairment, particularly the inability to generate torque rapidly, was driving the loss of velocity-dependent power following muscle damage. Therefore, torque loss contributes minimally to power production for lighter loads whereas at higher loads torque loss impedes power production severely. Perhaps, because shortening velocity is related to the number of sarcomeres working in series whilst torque production is related to those sarcomeres in parallel (34), damage preferentially affects torque production, and

hence why we observed a greater loss of power at heavier rather than lighter loads following muscle damage.

Repetitive lengthening contractions fatigued and temporarily weakened the dorsiflexors, thus impairing their ability to generate torque rapidly. This effect of muscle damage was most evident via a preferential loss of power for those loads representing a higher percentage of maximal isometric strength. Although strength decreased similarly between sexes, women displayed a reduced recovery related to a greater and longer lasting failure in E-C coupling, presumably Ca^{2+} release, thus further impairing power generation at higher loads owing to a greater impairment in torque development than the men. We conclude that the inability to generate torque rapidly at the onset of exercise appears to be one of the underlying mechanisms responsible for the loss of power following muscle damage, particularly at higher loads.

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Chapter 6 – General discussion and summary

The studies presented in Chapters 2 to 5 of my thesis exploit the unique modality of muscle damage testing by focusing on the velocity component of power generation during movement. The specific aim was to challenge the human neuromuscular system in a population of young and old women and men. I used a less-studied, but important, dynamic task of lengthening contractions to explore the effects of muscle damage on factors responsible for subsequent impairments in shortening velocity and power generation.

The main findings of my thesis are that repetitive lengthening contractions fatigued and temporarily weakened the dorsiflexors, thus impairing their power producing ability owing to an inability to generate torque rapidly. In Chapter 3 I showed that muscle fatigue is transient, and recovers relatively quickly, while the effects of muscle damage can be longer lasting. This effect of muscle damage was most evident via a preferential loss of shortening velocity and power for those loads representing a higher percentage of maximal voluntary isometric strength (MVC). Although strength decreased similarly between sexes, women showed a delayed recovery related to a greater and longer lasting failure in excitation-contraction (E-C) coupling, thus further impairing power generation at higher loads owing to a lower voluntary rate of torque development (RTD) than the men (Chapter 5). The old experienced greater fatigue than the young. This greater fatigue response in older women is likely attributable to their limited capacity for post-activation potentiation; a factor in the young which may have helped offset the initial transient

fatigue-induced impairments in shortening velocity (Chapter 4; Figure 17). It appears based on the greater power loss incurred by the older women they may be energetically disadvantaged (18) following repeated lengthening contractions, thus further exacerbating fatigue mechanisms related to whole muscle shortening velocity (1) and the subsequent generation of power (Chapter 4). I emphasize - that the inability to generate torque rapidly and initiate movement quickly at the onset of exercise appears to be an underlying mechanism responsible for the power-loss following muscle damage - particularly at higher loads (Figure 26).

For this particular limb muscle model, central neural factors do not appear to contribute to impaired neuromuscular function following lengthening contractions. Voluntary activation, as assessed using the interpolated twitch technique, and surface and indwelling wire RMS EMG amplitude of the dorsiflexors and tibialis anterior, respectively, were unchanged. Therefore, impairments in power following muscle damage seem to be dependent upon factors distal to the neuromuscular junction. Isometric torque loss following muscle damage can be due to voluntary activation failure (17). However, a maintenance of high voluntary activation for the tibialis anterior is a consistent and reliable (15) finding (*see Chapter 2*) following lengthening contractions (3, 11, 13, 14, 16). Furthermore, M-wave parameters (i.e., p-p amplitude, area, duration) were reduced similarly in old and young indicating that muscle damage may have disturbed sarcolemmal excitability in both age groups equally. However, results are equivocal; M-wave properties can be impaired following lengthening contractions (8) while others using similar lengthening contraction protocols do not show impairments (3, 11).

In Chapter 4 the older women were weaker and slower for whole muscle shortening velocity, leading to a greater reduction in power when compared with young women. Both the old and young women possibly incurred a similar amount of muscle damage (i.e., prolonged reduction in isometric MVC), yet the old were more fatigable than young as indicated by the greater power loss up to 10 min into the recovery period. Once the transient effects of fatigue recovered both age groups exhibited a similar reduction in power and for this reason, I argue that both groups experienced similar functional impairments owing to muscle damage. However, the old women incurred more fatigue than the young women which accounted for the greater power loss immediately following the lengthening contractions.

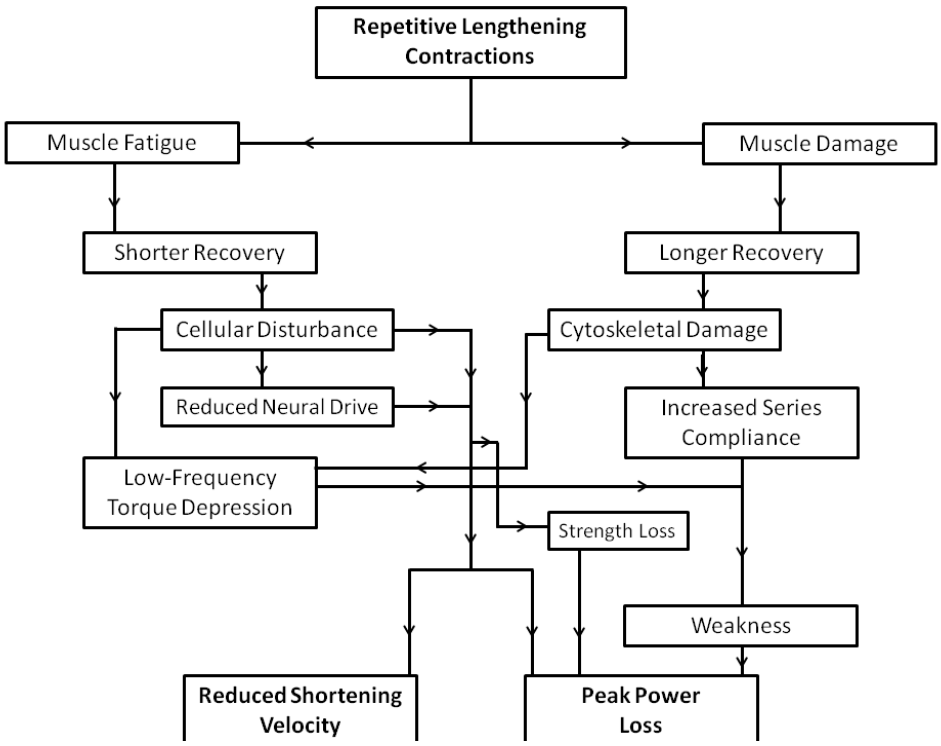

Figure 26. Factors related to repetitive lengthening contractions contributing to reduced shortening velocity and power loss.

As shown in Chapter 5, the increased muscle series compliance as evidenced by the change in optimal angle of torque production to longer muscle lengths could have contributed to the loss of power production capacity in the dorsiflexors. Muscle function was tested at the original muscle length not the new angle of optimal torque production. Therefore, the increase in series compliance could be a confounding factor for the observed weakness. Because the young women were weaker and reached peak power at a lower percentage of MVC than men, the additional loss of strength following muscle damage created an even greater loss of power at heavier loads compared with men. Structurally, men and women seem to have experienced a similar level of muscle damage. Although, it appears mechanisms of dynamic torque generation are affected more in women than men following damage (see below).

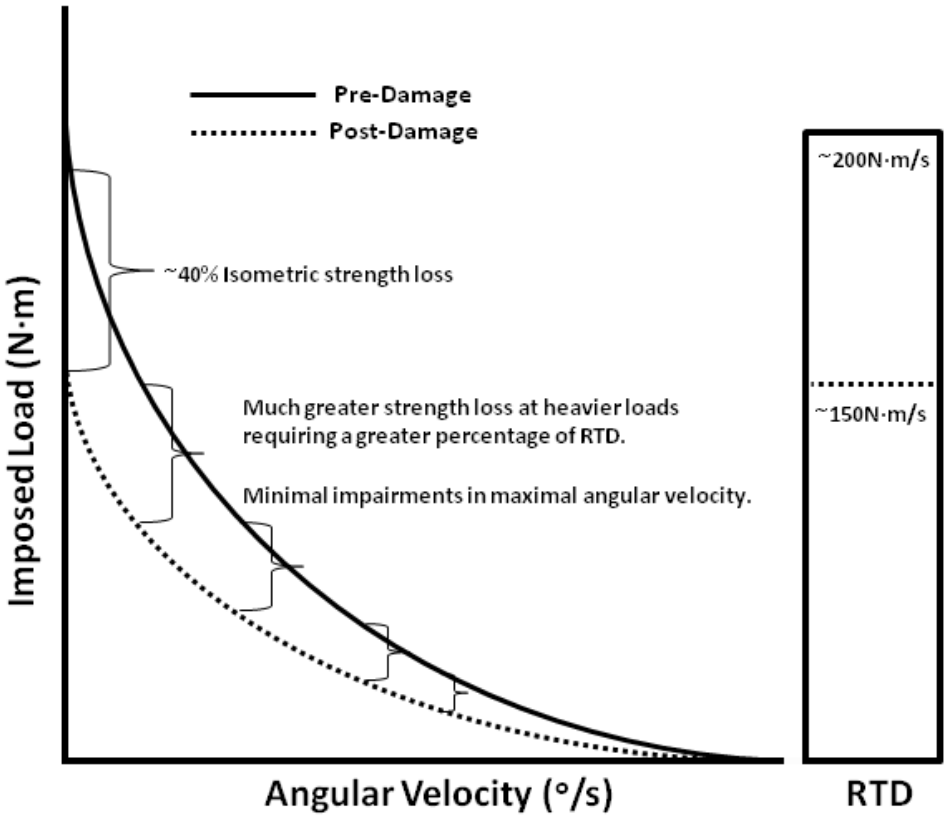

Figure 27. Force velocity and rate of torque development. Theoretical pre- (solid line) and post-damage (dotted line) force velocity relationships as related to rate of torque development (RTD). As shown by the curves angular velocity is less impaired at lower loads which require minimal RTD, while angular velocity is impaired severely at higher loads which require a much greater RTD to perform the task.

Inadequate activation of intact force-generators (i.e., E-C coupling failure (2, 19)), and disruption of force transmitting structures resulted in the inability to generate force rapidly (4, 12) which is critical for optimal performance during the heavier loaded contractions. The main contributor to E-C uncoupling following muscle damage is impaired Ca^{2+} release (6, 9). It is conceivable that the sex-difference in E-C uncoupling following damage could be attributed to less Ca^{2+} released from the female sarcoplasmic reticulum owing to a smaller safety factor

(Chapter 5). Consequently, a mechanical disruption of the link between the t-tubule and the sarcoplasmic reticulum could further impair an already compromised system. Although muscle damage was similar between sexes, the women were less able than the men to engage a sufficient number of viable force generators. The manner in which torque is generated (i.e., developed quickly) appears to play a critical role in muscular performance following damage, such that, there may be a critical threshold value of RTD to perform the task successfully. Therefore, for loaded contractions representing a higher percentage of one's maximal strength, if maximal RTD is impaired below the requisite needed to initiate movement rapidly, power will be impaired greatly. On the contrary, when performing fast movements with light to moderate loads that represent a low percentage of one's maximal strength, the critical threshold for RTD to move the load quickly is not stressed and performance is unimpeded by muscle damage (Figure 27). Therefore, muscle weakness contributes minimally to power production for lighter loads whereas at higher loads torque loss impedes power production severely.

6.1 Limitations

The markers of muscle damage I used in my experiments were self-reported muscle soreness, optimal angle of torque production and maximal isometric voluntary strength. These are indirect markers of muscle damage and can be influenced by central factors and voluntary effort. Ideally muscle biopsies could have been obtained, but were not practical for these experiments. We were not interested in investigating the extent of muscle damage per se, but rather,

neuromuscular function following lengthening contractions. Additionally, the disruption to the muscle from the biopsy and additional intimation and pain from the procedure could have altered neuromuscular properties. Despite the limitations of these measures in identifying structural muscle damage, they do reflect impairments in function, which is ultimately the primary concern following muscle damage.

Surface electromyographic (EMG) recordings during a dynamic task are known to be affected due to movement of the muscle fibers recorded in relation to the surface detected potentials as the muscle shortens or lengthens under the skin (7). In an attempt to minimize this limitation, I employed a mono-polar electrode configuration to record from a large global area of the tibialis anterior. Further, to provide improved assessment of EMG in Chapter 5 and to correct for this limitation I inserted a fine-wire electrode into the muscle to 'move with' the muscle during dynamic contractions. Central neural influences were ruled out as factors responsible for power loss. However, measuring voluntary activation is restricted to isometric or slow isokinetic contractions. Thus I do not have a measure of voluntary activation during fast shortening contractions, and therefore it is unclear if deficits in voluntary activation were indeed present. Voluntary activation was high and consistent throughout all studies; this measure along with EMG may not have been sensitive enough to detect central deficits brought about by lengthening contractions.

The isotonic mode of the Biodex dynamometer was used. However, due to inherent mechanical limitations of the dynamometer (i.e., unable to maintain an

exact constant external load throughout an entire range of motion), these contractions are neither purely isotonic, nor are they iso-inertial as the load is fixed (mechanically) and velocity of contraction is determined by the effort of the participant. Therefore we define and refer to these contractions first in Chapter 3 as "velocity-dependent" (5, 15). The distinction between an isotonic movement true to the theoretical term and practical sense is secondary to the fact that these movements are more representative than isovelocity for replicating movements performed in everyday life, and emphasize the unconstrained velocity component of power.

Many indirect measurements were obtained using electrical stimulation to infer fatigue/muscle damage induced changes in neuromuscular function, such as, central drive to the muscle (Voluntary activation, EMG), neuromuscular propagation (M-wave), and excitation-contraction coupling / Ca^{2+} handling (electrically evoked twitch and tetanus measures). Even though the specific mechanisms of failure cannot be confirmed in these experiments, this does not impair interpretation of these results based on the available literature, and also emphasizes the need for future studies.

6.2 Future Directions

A second bout of lengthening contractions within days or weeks following exercise-induced muscle damage provides a prophylactic effect on muscle function compared with those of the initial insult. This phenomenon is known as the repeated bout effect (RBE). In my thesis I suggest torque loss - specifically impaired

rate of torque development - is a key factor in power loss following muscle damage. Thus, it would be interesting to explore whether power loss is less impaired following a second bout of lengthening contractions, and if so, attempt to elucidate further those mechanisms responsible. Presumably, there would be less of a leftward shift in the force-velocity curve owing to maintenance of torque generation. Currently, it is unknown how the force-velocity relationship is altered following muscle damage in older adults and also what effects would be seen in other muscle models such as the elbow flexors.

In my thesis, there were no indications of central impairments following lengthening contractions. Thus, more sensitive measures may be required to identify central limitations following muscle damage such as transcranial magnetic stimulation (TMS), cervicomedullary (CM) stimulation and single motor unit recordings. The silent period following stimulation would provide insight into cortical (TMS) and spinal inhibition (CM) following muscle damage. Single motor unit recordings would provide information on recruitment thresholds and firing rates; the precursors of motor output.

Imaging techniques would further identify muscle damage. Ultrasound imaging could be used to measure real time fascicle length changes and tendon compliance. This would help to decipher whether tendon and musculotendinous stiffness is affected during damaging lengthening contractions and the extent to which the fascicles of the tibialis anterior actually lengthen. As well, advances in magnetic resonance imaging (MRI) can be used to identify muscle damage less invasively than the biopsy technique.

6.3 Summary

Many natural movements are comprised of not only shortening actions but lengthening phases which act to lower the body and brake/decelerate movements, which can result in muscle damage. My thesis provides support for the importance of measuring the velocity-component of power to provide a complete description of muscle function and mechanics. Indeed, my unique results indicate that isometric torque loss per se is of lesser importance in explaining functional impairment following dynamic lengthening actions than factors that impede dynamic contractile kinetics (i.e., RTD). Additionally, my results offer further evidence that fatigue is task-specific and that experimental designs need to extend beyond isometric models to provide a greater understanding of muscle fatigue and damage. The compromised neuromuscular system of older adults, and weaker system of women has helped highlight this feature. Finally, I propose that because shortening velocity is related to the number of sarcomeres working in series whilst torque production is related to those sarcomeres in parallel (10), muscle damage preferentially affects torque production, and lead to my observations of a subsequent greater loss of power at heavier rather than lighter loads following muscle damage.

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Appendix A

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Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. A.A. Vandervoort
Review Number: 16118 **Review Level:** Full Board
Review Date: May 19, 2009
Protocol Title: Short-Term Muscle Fatigue in Older Adults and The Potential Related Benefits on Increasing Force-Producing Capacity on Fatigue Resistance
Department and Institution: Physical Therapy, University of Western Ontario
Sponsor: NSERC-NATURAL SCIENCES ENGINEERING RSRCH COU
Ethics Approval Date: July 29, 2009 **Expiry Date:** September 30, 2013
Documents Reviewed and Approved: UWO Protocol and Letter of Information and Consent Form dated June 22, 2009, and Poster
Documents Received for Information:

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (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 REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

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Investigators must promptly also report to the HSREB:

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If these changes/adverse events require a change to the information/consent documentation, and/or recruitment advertisement, the newly revised information/consent documentation, and/or advertisement, must be submitted to this office for approval.

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Chair of HSREB: Dr. Joseph Gilbert

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Appendix B

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Geoffrey A. Power

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- Muscle architecture plasticity
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- Age-related alterations to muscle mechanics

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2012	Canadian Centre for Activity and Aging – Research to Action Graduate Student Winner
2012	Ontario Graduate Scholarship – $15,000
2012	The Queen Elizabeth II Graduate Scholarship in Science and Technology (declined) – $15,000
2011	Canadian Institute for Health Research **Age+ Prize** *Paper:* Motor unit number estimates in masters runners: use it or lose it?
2011	Ontario Graduate Scholarship – $15,000
2010	Canadian Society for Exercise Physiology Student Poster Award Winner
2010	Western Research Forum Oral Presentation Award – The University of Western Ontario
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Articles Submitted to Refereed Journals

1. **Power GA,** Dalton BH, Booth WJ, Rice CL, Vandervoort AA. (2012). Peak power is reduced following muscle damage despite a maintenance of shortening velocity in men and women. Submitted to Experimental Physiology (EXPPHYSIOL/2012/069427)

2. Klein CS, **Power GA**, Rice CL, Brooks D. (2012). Neural and muscular determinants of dorsiflexor weakness in chronic stroke survivors. Submitted to Motor Control (MC_2012_0098)

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1. Dalton BH, **Power GA**, Harwood B, Rice CL. (2012). Motor unit properties in the three heads of the triceps surae. *International Motoneuron Meeting: Motoneurons and Beyond*, Sydney, Australia. July 23-26.

2. **Power GA,** Rice CL, Vandervoort AA. (2012). Increased force production in old age is not a far stretch. *Appl. Physiol. Nutr and Metab* 37:(S1). CSEP, Regina, SK. October 10-13.

3. McNeil CJ, **Power GA,** Gandevia SC, Taylor JL, Rice CL. (2012). The decrease of motor evoked potential size with strong contractions is not attenuated with age. *Appl. Physiol. Nutr and Metab* 37:(S1) CSEP. Regina, SK. October 10-13.

4. Paturel JP, **Power GA,** Rice CL. (2012). Repetitive Isotonic contractions are more fatigable compared with isokinetic when matched for peak power. *Appl. Physiol. Nutr and Metab* 37:(S1). CSEP, Regina, SK. October 10-13.

5. Booth WJ, Allen MD, **Power GA,** Marsh GD, Rice CL. (2012). Assessment of skeletal muscle quality and motor unit loss in leg muscle of old and very old men. Proceedings of the International Conference on Sarcopenia Research, Orlando, FA. December 6-7.

6. Harwood B, Dalton BH, **Power GA**, Rice CL. (2012). Muscle-dependent motor unit recruitment behavior of the elbow extensors. Society for Neuroscience, New Orleans, LA. October 13-17.

7. **Power GA,** Paturel JR, Rice CL, Vandervoort AA. (2012). Increased residual force enhancement following damage. Proceedings of the 17th Biannual Conference of the Canadian Society for Biomechanics, Burnaby, BC. June 6-9.

8. **Power GA,** Dalton BH, Booth WJ, Rice CL, Vandervoort AA. (2012). Muscle damage and sex: Implications of rate of torque development on velocity-dependent power loss. *Med Sci Sports and Ex* 44: S5. ACSM, San Francisco, CA. May 31-June 4.

9. Allen MD, Dalton BH, **Power GA**, Rice CL. (2012). Effect of aging on the relationship between knee angle and triceps surae power output. *Med Sci Sports and Ex* 44: S5. ACSM, San Francisco, CA. May 31-June 4.

10. **Power GA,** Harwood B, Dalton BH, Vandervoort AA, Rice CL. (2011). Motor unit recruitment and initial discharge rate of the elbow extensors: The triceps brachii and anconeus. Program no. 920.14 Abstract Viewer/Itinerary Planner. CD-ROM. Society for Neuroscience, Washington, DC. November 12-16.

11. Dalton BH, Harwood B, **Power GA,** Vandervoort AA, Rice CL. (2011). Motor unit properties of the triceps surae during a sustained sub-maximal plantar flexion task. Program no. 920.16 Abstract Viewer/Itinerary Planner. CD-ROM. Society for Neuroscience, Washington, DC. November 12-16.

12. Harwood B, **Power GA**, Allen MD, Rice CL. (2011). The relationship between elbow extension velocity and motor unit recruitment thresholds of anconeus motor units. Program no. 920.15 Abstract Viewer/Itinerary Planner. CD-ROM. Society for Neuroscience, Washington, DC. November 12-16.

13. Allen MD, Dalton BH, **Power GA**, Rice CL. (2011). Effect of knee angle on velocity-dependent power production of the triceps surae in young men. *Appl. Physiol. Nutr and Metab* 36:(S2)S299-S360, 2011. CSEP, Quebec City, QC. October 19-22.

14. **Power GA,** Dalton BH, Booth WJ, Rice CL, Vandervoort AA. (2011). Preferential loss of velocity-dependent power at high loads following muscle damage. *Appl Physiol. Nutr. and Metab* 36: (S2)S299-S360, 2011. CSEP, Quebec City, QC. October 19-22.

15. **Power GA,** Dalton BH, Rice CL, Vandervoort AA. (2011). Rate of Activation and Torque Development of Velocity-Dependent Contractions Following Muscle Damage *Exercise Neuroscience Group*, Waterloo, ON. June 18-19. http://www.wix.com/oeng2011/oeng2011.

16. **Power GA**, Dalton BH, Behm DG, Rice CL, Vandervoort AA, Doherty TJ. (2011). Preservation of motor unit number estimates (MUNEs) in masters runners is muscle dependent. *Med. Sci. Sports and Ex.* 43: S5. ACSM, Denver, CO. May 31-June 4.

17. **Power GA**, Dalton BH, Vandervoort AA, Rice CL, Doherty TJ. (2010) Motor unit number estimates in a proximal human upper limb muscle: An age-related reduction. Program no. 180.13 Abstract Viewer/Itinerary Planner. CD-ROM. Society for Neuroscience, San Diego, CA. November 13-17.

18. Dalton BH, Harwood B, **Power GA,** Rice CL. (2010). Motor unit properties of the triceps surae. Program no. 180.5 Abstract Viewer/Itinerary Planner. CD-ROM. Society for Neuroscience, San Diego, CA. November 13-17.

19. **Power GA,** Dalton BH, Rice CL, Vandervoort AA. (2010). Velocity-dependent power loss following lengthening contractions in young and old women. *Appl. Physiol. Nutr. and Metab.*, 35, S1. CSEP, Toronto, ON. November 3-6. <u>*CSEP student poster award*</u>

20. **Power GA,** Dalton BH, Rice CL, Vandervoort AA. (2010). Velocity-dependent power loss in the knee extensors of young and old men. *Med. Sci. Sports and Ex.,* 42 (5). ACSM, Baltimore, MD. June 2-5.

21. Dalton BH, **Power GA,** Vandervoort AA, Rice CL (2010). Recovery from fatigue of velocity-dependent power in young and old men. *Med. Sci. Sports and Ex.,* 42 (5). ACSM, Baltimore, MD. June 2-5.

22. **Power GA,** Dalton BH, Behm DG, Doherty TJ, Vandervoort AA, Rice CL. (2010). Motor unit number estimates in masters runners: use it or lose it? *23rd Annual Western Research Forum*. London, ON. February 27. *Oral presentation winner*

23. **Power GA,** Dalton BH, Rice CL, Vandervoort AA. (2009). Power loss following velocity-dependent eccentric contractions of the ankle dorsiflexors. *Appl. Physiol. Nutr. and Metab.*, 34, S74. CSEP, Vancouver, BC. November 11-14.

24. Dalton BH, **Power GA,** Rice CL. (2009). Velocity-dependent fatigability of the plantar flexors in young and old men. *Appl. Physiol. Nutr. and Metab.*, 34, S22. CSEP, Vancouver, BC. November 11-14.

25. **Power GA,** Dalton BH, Rice CL, Vandervoort AA. (2009). Isotonic power loss following lengthening contractions of the dorsiflexors. *Exercise Neuroscience Group*, London, ON. June 18-19. http://www.wix.com/bradharwood/OENG-2009

26. **Power GA,** Dalton BH, Rice CL, Vandervoort AA. (2009). Muscle fatigue: blame it on the pain! an investigation of varying contraction types on perceived effort and neuromuscular fatigue. *22nd Annual Western Research Forum*. London, ON. February 28.

27. **Power GA,** Handrigan GA, Evely K, Gilliam D, Basset FA (2007). Breathing patterns at isometabolic intensities between cycle and treadmill ergometers: a training specificity effect? *Appl. Physiol. Nutr. and Metab.*, 32, S22. CSEP, London, ON. November 14-17.

28. Billaut F, **Power GA**, Handrigan GA, Basset FA. (2007). Muscle coordination during multiple-sprint exercise with varied recovery patterns. *Appl. Physiol. Nutr. and Metab.*, 32, S22. CSEP, London, ON. November 14-17.

29. Handrigan GA, **Power GA,** Mackinnon S, Basset FA. (2007). A comparison of the oxygen cost in three standardized pulling tasks. Sixth International Scientific Conference on Prevention of Work-Related Musculoskeletal Disorders (PREMUS), Boston, MA.

30. **Power GA,** Handrigan GA, Basset FA. Boulay M. (2006). Modulation of breathing parameters between treadmill and cycle ergometer tests. *Atlantic Provinces Exercise Scientists Conference,* Halifax, NS. March 24-25.

Academic and Professional Experience:

Per-Course Appointments

HKR 4310: Evaluation and Measurement: *Laboratory Instructor* (Summer 2008)
School of Human Kinetics and Recreation, Memorial University
I was given the freedom to develop the laboratory component of this course which focused on basic statistical and data management processes.

HKR 4320: Fitness Leadership: *Lecturer* (Fall 2007)
School of Human Kinetics and Recreation, Memorial University
I instructed this 4th year course to over 50 students which serves as the basis for CSEP certification in the HKR department. In addition to the basic course material I expanded the material to include many exercise training principles.

HKR 1000: Fitness and Wellness: *Lecturer* (Summer 2007)
School of Human Kinetics and Recreation, Memorial University
I co-instructed this course which is a very popular university course outside of the HKR department, and effectively communicated many difficult physiological concepts to students who did not necessarily have a science background.

HKR 3310: Exercise Physiology: *Laboratory Instructor* (Fall 2004 – Fall 2006)
School of Human Kinetics and Recreation, Memorial University
I was originally hired as a work term student to instruct the exercise physiology labs. In my 2nd year I overhauled the existing labs and developed new ones to broaden the scope of the laboratories.

HKR 2310: Human Anatomy: *Laboratory Instructor* (Fall 2004)
School of Human Kinetics and Recreation, Memorial University
I co-instructed the anatomy laboratories.

Teaching Assistantship

KIN3337A: Physiology of Fitness Appraisal (Fall 2008)
School of Kinesiology, The University of Western Ontario
Duties: Laboratory facilitator

HKR 2000: Introduction to Kinesiology, Physical Education and Recreation (Winter; 2006, 2007, 2008) School of Human Kinetics and Recreation, Memorial University
Duties: marking

Memorial University of Newfoundland and Labrador: Instructional Development Office
Workshop Instructor: Teaching Opportunities for Graduate Students (Winter 2007)

ED 2001: Physical Education in the Primary and Elementary Grades (Fall 2005)
School of Human Kinetics and Recreation, Memorial University
Duties: marking

HKR 1000: Introduction to Fitness and Wellness (Fall 2006, 2007)
School of Human Kinetics and Recreation, Memorial University
Duties: marking

Professional Membership

American College of Sports Medicine (ACSM): Graduate student member (2008-present)

Canadian Society for Exercise Physiology (CSEP): Graduate student member (2008-present)

Society for Neuroscience (SFN): Graduate student member (2010-2012)

Canadian Society for Biomechanics: Graduate student member (2012-present)

Invited Lecturer/Presentations

Canadian Centre for Activity and Aging: Research to Action, Grad student Award presentation The University of Western Ontario (July 2012)
Topic: Use it or lose it holds true in old age.

Herzog Biomechanics Group Seminar: The University of Calgary (June 2012)
Topic: Aging, lifelong exercise and motor unit loss: Use it or lose it!

KIN 4474b: The University of Western Ontario (Winter 2012)
Topic: Aging and resistance training

The University of Western Ontario Kinesiology Bioscience Seminar (Winter 2012)
Topic: Neuroprotective effects of exercise on motor unit survival

SHAD Valley Seminar presentation, Memorial University of Newfoundland
"An Introduction to Kinesiology and Exercise Physiology" St. John's, NL 2006

SHAD Valley Seminar presentation, Memorial University of Newfoundland
"An Introduction to Kinesiology and Exercise Physiology" St. John's, NL 2007

HKR 2300: Memorial University of Newfoundland (Winter 2007)
Topic: The aging neuromuscular system

Research Assistantship

Research Assistantship, Kinesiology, The University Of Western Ontario, London, ON. Fall 2009/2010/2011

Research assistant, School of Human Kinetics and Recreation, Memorial University of Newfoundland, St. John's, NL. Summer 2008
Supervisor: Dr. David Behm

Duties:
- Ethical approval application
- Participant recruitment
- Data collection
- Supervision of visiting undergraduate students
- Supervision of an undergraduate honors thesis

Research technician, National Research Council, Memorial University of Newfoundland, St. John's, NL. Winter 2007. *Supervisor: Dr. Fabien Basset*

Duties:
- Subject preparation
- Calibration of metabolic cart
- Collecting and exporting data
- Coordinating efforts with the rest of the team