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ARTICLE

Peak power is reduced following lengthening contractions despite a maintenance of shortening velocity

Geoffrey A. Power, Brian H. Dalton, Charles L. Rice, and Anthony A. Vandervoort

Abstract: Following repetitive lengthening contractions, power (the product of torque and velocity) is impaired during shortening contractions. However, the relative contribution of each component to power loss and the underlying factors are unclear. We investigated neuromuscular properties of the dorsiflexors in 8 males (27 ± 3 years) and 8 females (26 ± 4 years) for a potential sex-related difference before, during, and after 150 unaccustomed maximal lengthening actions. Velocity-dependent power was determined from shortening contractions at 8 levels (1 N·m to 70% of maximum voluntary isometric contraction (MVC)) before, after, and throughout recovery assessed at 0–30 min, 24 h, and 48 h. Immediately following task termination, both sexes displayed similar impairments of 30%, 4%, and 10% in MVC torque, shortening velocity, and overall peak power, respectively ($P < 0.05$). Peak rate of isometric torque development (RTD) was reduced by 10% in males, but females exhibited a 35% reduction ($P < 0.05$). Rate of torque development for the MVC remained depressed in both sexes throughout the 30 min recovery period; however, the RTD returned to normal by 24 h in males but did not recover by 48 h in females. Power was reduced preferentially at higher loads (i.e., 60% MVC), with a greater loss in females (65%) than males (45%). For lower loads (<20% MVC), power was impaired minimally (4%–8%; $P < 0.05$) and recovered within 30 min in both groups. The reduction in maximal angular velocity persisted until 30 min of recovery, and peak power did not recover until 24 h for both sexes. Unaccustomed lengthening contractions decreased power preferentially at higher loads, whereas peak power was reduced minimally owing to maintenance of maximal shortening velocity.

Key words: muscle damage, sex, eccentric, human, dorsiflexors, torque–velocity.

Résumé : Une altération de la puissance générée (moment de force × vélocité) est observée pendant des contractions miométriques effectuées à la suite de contractions pliométriques répétées. Toutefois, la contribution relative de ces facteurs à la diminution de puissance n'est pas bien établie. Avant, pendant et après 150 contractions pliométriques maximales inhabituelles, on examine les propriétés neuromusculaires des fléchisseurs dorsaux chez 8 hommes (27 ± 3 ans) et 8 femmes (26 ± 4 ans) pour vérifier la présence possible d'une différence liée au sexe. On évalue la puissance selon la vélocité au cours de contractions miométriques de 8 intensités (de 1 N·m jusqu'à 70 % de la tension isométrique maximale (MVC)), et ce, avant, à la suite et tout au long de la récupération aux moments suivants : 0–30 min, 24 h, et 48 h. Immédiatement après la fin de la tâche, les deux groupes présentent une diminution similaire, soit 30, 4 et 10 % du moment de force MVC, de la vélocité de raccourcissement et de la puissance de crête globale, respectivement ($P < 0,05$). Le taux de pointe du développement de la tension isométrique (RTD) diminue de 10 % chez les hommes et de 35 % chez les femmes ($P < 0,05$). Le RTD pour atteindre la MVC reste bas dans les deux groupes tout au long des 30 min de récupération; le RTD revient à la normale en 24 h chez les hommes, mais n'a pas retrouvé sa valeur initiale en 48 h chez les femmes. La puissance diminue surtout aux intensités plus élevées (c.-à-d. 60% MVC), la diminution étant plus élevée chez les femmes (65 %) que chez les hommes (45 %); aux intensités plus faibles (<20 % MVC), la puissance diminue légèrement (4–8 %; $P < 0,05$) et revient dans les deux groupes à sa valeur normale en moins de 30 min. La diminution de la vélocité angulaire maximale persiste jusqu'à la récupération de 30 min et la puissance de pointe ne revient pas à la normale avant 24 h dans les deux groupes. Des contractions pliométriques inhabituelles suscitent une diminution de la puissance surtout aux intensités élevées et la puissance de pointe diminue légèrement à cause du maintien de la vélocité maximale de raccourcissement. [Traduit par la Rédaction]

Mots-clés : lésion musculaire, sexe, pliométrique, humain, dorsifléchisseurs, moment de force–vélocité.

Introduction

Optimal power generation is based on the finely tuned relationship between torque and angular velocity and is expressed as the torque–velocity (*T*–*V*) relationship. As velocity increases, less torque can be generated owing to fewer cross-bridge attachments, and thus an optimal combination of submaximal torques and velocities is required to achieve peak power (Abbott and Wilkie 1953; Lieber and Ward 2010). Following repetitive voluntary lengthening contractions, neuromuscular function can be diminished severely (Barker et al. 2012; Byrne et al. 2004; Power et al. 2010; Prasartwuth et al. 2005; Sargeant and Dolan 1987) due to such impairments as cytoskeletal damage, excitation contraction uncoupling (Allen et al. 2005; Ingalls et al. 1998; Proske and Allen

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2005; Warren et al. 2001), and increased series compliance (Gregory et al. 2007), as observed in reduced preparations. Recently, we demonstrated in the ankle dorsiflexors that maximal voluntary isometric torque (MVC) and velocity-dependent power (isotonic load: 20% MVC) were reduced by 28% and 8%, respectively (Power et al. 2010). Because maximal torque generating capacity was attenuated to a greater extent than angular velocity at a moderate load, it is conceivable that power will be reduced preferentially at higher rather than lower loads, but this has not been tested systematically. As a consequence of the presumed muscle damage and associated muscle weakness following lengthening contractions, peak power may shift and occur at lower loads, thereby relying more on the velocity component for peak power generation. This hypothesis is partially supported by studies involving isovelocity (i.e., constant speed or isokinetic) actions (for review, see Byrne et al. 2004). To determine the extent of concentric strength loss following lengthening contractions, an isovelocity model relies specifically on testing the torque component of power when angular velocity is fixed, but the conclusions are equivocal. Some report no velocity dependence (Byrne et al. 2001), whereas others report greater impairments at slow (i.e., impaired torque generation) (Deschenes et al. 2000; Gibala et al. 1995; Michaut et al. 2002) or fast (i.e., velocity impairment) (Eston et al. 1996; Friden et al. 1983; Golden and Dudley 1992) angular velocities. Unfortunately, the isovelocity contraction mode constrains angular velocity artificially and therefore is a modality that poorly reflects natural in vivo contractile function. As well, the determination of power is underestimated when evaluated isokinetically owing to a limited velocity range and highly restricted limb acceleration (Aagaard et al. 1994). When torque is held relatively constant and angular velocity can vary freely, the muscle functions more closely to in vivo conditions (Barker et al. 2012; Power et al. 2011) and alterations in the *T*–*V* relationship can be explored to offer insight into key factors responsible for power loss.

Reports addressing sex-related differences in response to lengthening contractions in humans are inconclusive (for review, see Clarkson and Hubal 2002). Following lengthening contractions in a large sample of males ($n = 98$) and females ($n = 94$), Sayers and Clarkson (2001) reported a disproportionately higher number of females than males demonstrating greater initial force loss. Similarly, Sewright et al. (2008) reported that immediate force loss was more prominent in females than males. Thus, it is possible that when tested at heavier isotonic loads (i.e., representing a greater percentage of maximal strength), angular velocity and velocity-dependent power production will be more impaired in females than males. A potential key component that may contribute to the impaired power production following lengthening contractions is the rate of torque development (RTD) (Andersen and Aagaard 2006). The reduction in MVC following fatigue or lengthening contractions is associated with impaired RTD (Behrens et al. 2012; Crameri et al. 2007; Hannah et al. 2012), and this would be manifested during dynamic contractions such that the ensuing angular velocity and power production will be reduced when the contractile torque equals and then exceeds the specific isotonic load.

The purpose of this study was to investigate the effect of lengthening contractions on velocity-dependent power loss and to determine whether a sex difference exists when assessed across multiple isotonic loads. Because torque production and RTD are impaired substantially following lengthening contractions (Behrens et al. 2012; Crameri et al. 2007; Hannah et al. 2012), we hypothesized that there would be a greater loss of power at higher loads during dynamic shortening actions. However, due to less reliance on RTD to achieve the requisite isotonic load at a lower percentage of MVC, shortening velocity will be less impaired than at higher loads. Finally, to further highlight the role of strength loss, which we suspect is the larger contributor (compared with velocity) to power loss following lengthening contractions, we tested females. Females typically experience greater strength loss following lengthening contractions (Clarkson and Hubal 2002), and thus we expected the females to rely more on the velocity component of power and have a greater loss of power at heavier loads than males.

Materials and methods

Ethical approval

Following oral and written explanation of procedures, risks, and benefits of the study, written informed consent was obtained from all participants prior to testing. This study was approved by the University of Western Ontario's Health Science Research Ethics Board for research involving human subjects and conformed to the Declaration of Helsinki.

Participants

Eight male (27 ± 3 years, 178.1 ± 7.3 cm, 81.4 ± 10.1 kg) and 8 female (26 ± 4 years, 170.6 ± 6.8 cm, 63.9 ± 6.8 kg) volunteers, who were recreationally active but not systematically trained and were free from musculoskeletal disorders, were recruited for the study. All participants were asked to refrain from strenuous exercise 1 day before and 2 days after baseline testing and not consume caffeine 4 h before testing.

Experimental arrangement

All testing was conducted on a Biodex multi-joint dynamometer (System 3; Biodex Medical Systems, Shirley, New York, USA). The right foot was fastened tightly to the ankle attachment footplate with nonelastic straps, aligning the lateral malleolus of the ankle with the rotational axis of the dynamometer. Extraneous movements were minimized using nonelastic 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 (Maganaris 2001; Power et al. 2012*b*) 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, while minimizing possible range of motion failure, the lengthening contractions occurred from the neutral ankle angle until 30° of plantar flexion; thus, all dynamic actions moved through a 30° range of motion.

Electromyography (EMG)

To focus the recording from the tibialis anterior, electromyography was collected using a custom-made insulated stainless-steel fine wire electrode (50 μm; California Fine Wire Company, Grover Beach, California, USA) with a relatively large (~5 mm) piece of insulation removed from the tip to record from multiple motor units. The active electrode was inserted using a 30G sterilized hypodermic needle (B-D PrecisionGlide; Becton Dickinson and Company, Franklin Lakes, New Jersey, USA) in the proximal portion of the tibialis anterior near the innervation zone (7 cm distal to the tibial tuberosity and 2 cm lateral to the tibial anterior border). A monopolar electrode configuration was used to provide a more global recording area, and therefore a reference surface electrode was placed over the distal tendinous portion of the tibialis anterior at the malleoli. For the antagonist EMG, self-adhering Ag-AgCl surface electrodes (1.5 × 1 cm; Kendall, Mansfield, Mass., USA) were placed over the soleus to collect a global surface EMG. The surface 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 at the malleoli. The ground electrode for both tibialis anterior and soleus EMG configurations was positioned over the patella. Prior

to electrode placement and insertion, the skin was cleansed with presoaked alcohol swabs.

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) (two pulses at a 10 ms interpulse interval) to assess voluntary activation. Finally, 10 Hz (5 pulses over 0.5 s) and 50 Hz (25 pulses over 0.5 s) stimuli were delivered to assess peak tetanic torque by increasing the current until there was a plateau in evoked 50 Hz torque.

Maximal voluntary isometric contraction (MVC)

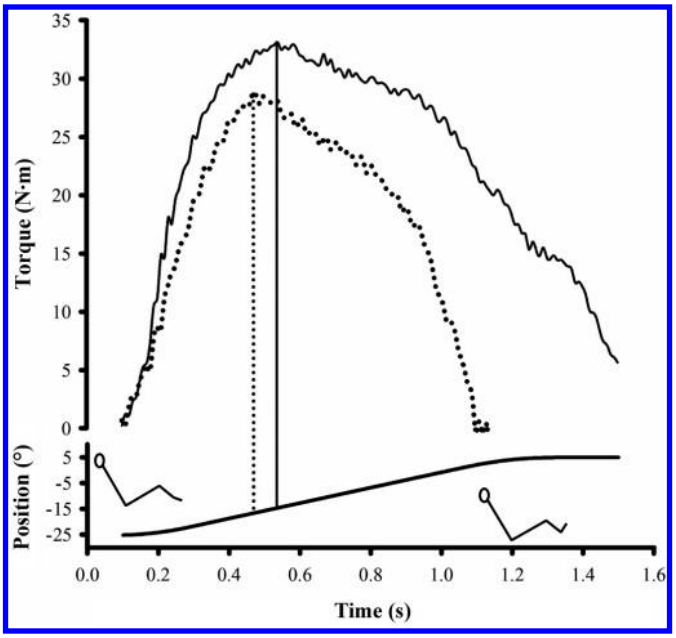
Three MVCs were performed, each of 3 to 5 s in duration. A rest of 3 min was given between all contractions. To ensure that MVC attempts were maximal, participants were provided visual feedback of the torque tracing on a computer monitor and exhorted verbally during all voluntary efforts. The coefficient of variation of the MVC for males and females was 0.24 and 0.13, respectively. Voluntary activation was assessed using the modified interpolated twitch technique (Behm et al. 2002). An electrically evoked doublet was triggered manually by the investigator when the dorsiflexion MVC torque reached a plateau. The amplitude of the interpolated torque during 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 [1 – (interpolated P_d/resting P_d)] × 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 10 to 50 Hz relationship using the current required to evoke peak 50 Hz torque.

Dynamic contractions

Once MVC torque was determined, the dynamometer was switched from isometric mode 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 isoinertial 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" (for further details, see Power et al. 2011).

The dynamometer was programmed to allow the footplate to return passively to 30° 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 angular 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 were 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 predetermined loads (1 N·m, 10%, 20%, 30%, 40%, 50%, 60%, and 70% MVC) in a randomized order with 30 s of rest between attempts. The peak value of the two contractions performed at each load was used to establish baseline values for angular velocity at each of the 8 loads, with the peak angular velocity value obtained at 1 N·m considered maximal shortening velocity, and peak power was determined as the highest power across the 8 loads tested. Two attempts at each load were performed to ensure that maximal velocity was achieved for all loads. To investigate changes in the series compliance of the muscle, optimal angle of torque production during a 30°·s^{-1} isokinetic contraction was performed (Fig. 1). Participants first practiced 2 of these contractions and, after 3 min of rest, performed 2 more for baseline values. The attempt with the greater torque value was used for analysis.

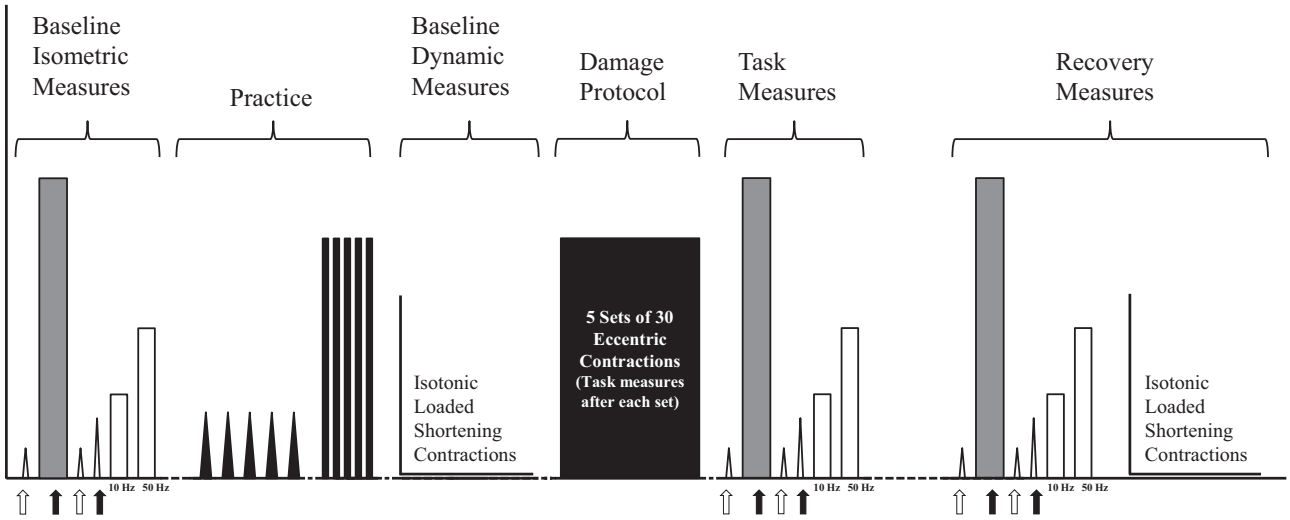
Fig. 1. Raw data depicting the change in optimal angle of torque production during a 30°·s^{-1} isokinetic contraction from baseline (solid line) and following (dotted line) the lengthening contraction protocol at the 30 min time point for a representative male participant.



Lengthening contraction task and recovery protocol

Participants performed 5 sets of 30 eccentric isokinetic dorsiflexion contractions at 60°·s^{-1} with each set separated by 30 s 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 isometric 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, 5, 10, 15, 20, and 30 min and 24 and 48 h. Neuromuscular measures before and after the protocol included the following (performed in order): (1) maximum evoked twitch properties, (2) assessment of MVC and voluntary activation, (3) postactivation twitch and twitch doublet, (4) measure of low-frequency torque depression (10:50 Hz ratio), (5) isotonic loaded shortening contractions (1 N·m to70% MVC), and (6) determination of optimal angle of torque production (30°·s^{-1} isokinetic contraction) (for experimental protocol, see Fig. 2). Muscle soreness was assessed during a voluntary contraction at baseline and 30 min, 24 h, and 48 h after task termination. Participants evaluated their muscle soreness subjectively 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.

Fig. 2. Schematic diagram of experimental protocol. Grey bars are isometric maximum voluntary contractions (MVC). Open torque profiles are electrically evoked contractions (twitches, doublet, 10 Hz, 50 Hz). Solid profiles are dynamic contractions; concentric at 20% MVC (triangles), and dynamic maximal eccentric contractions (rectangles). Isotonic loaded dynamic shortening contractions were performed under the following loads: 1 N·m, 10%, 20%, 30%, 40%, 50%, 60%, and 70% of MVC. Open arrows are electrically evoked twitches, and solid arrows are electrically evoked doublets. Recovery time points: immediately upon task termination, 2.5, 5, 10, 15, 20, and 30 min, and 24 and 48 h.



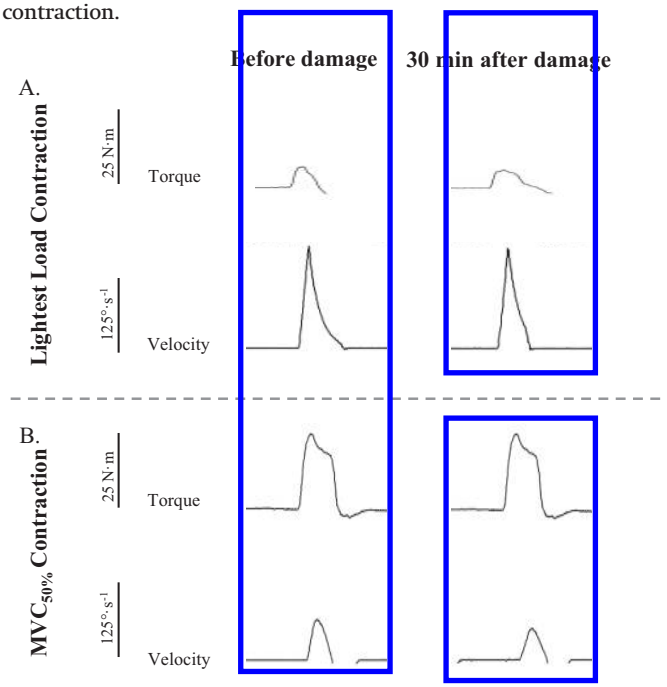
Data reduction and analysis

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 Ltd., Cambridge, UK). Electromyographic signals were pre-amplified (×100), amplified (×2), and band-pass filtered (10–1000 Hz) and sampled online at 2500 Hz using Spike 2 software (version 7.07; Cambridge Electronic Design Ltd.). Rate of torque development was calculated for the MVC as the peak tangential slope using a moving mean method of the torque–time curve (Δtorque/Δtime) over the first 400 ms from onset of contraction. Dorsiflexor EMG from the MVC 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 × 100%. All subsequent MVC RMS values were normalized to the level obtained during baseline. To estimate the mean magnitude of neuromuscular activity during the dynamic tasks, 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 mean magnitude of neuromuscular activity (mV) (Del Balso and Cafarelli 2007; Suetta et al. 2004). Ankle dorsiflexion power (W) was calculated as the product of torque (N·m) and the peak shortening velocity (rad·s^{-1}) for each of the 8 predetermined loads (Fig. 3). Postactivation potentiation was determined by calculating the ratio between the amplitude of the peak twitch torque recorded before and after 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 (DTPT), doublet half relaxation time (DHRT), 50 Hz HRT, MVC torque, rate of torque development (RTD), and optimal angle of torque production during a slow (30°·s^{-1}) isokinetic shortening contraction. Low-frequency torque depression was calculated using a ratio of peak 10 Hz to peak 50 Hz evoked torques (10 Hz : 50 Hz).

Statistical analysis

Using SPSS software (version 16; SPSS Inc. Chicago, Illinois, USA), a two-way (sex × 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 em-

Fig. 3. Unprocessed data from a representative male participant depicting (A) lightest load contraction and (B) 50% MVC velocity-dependent contraction. MVC, maximum voluntary isometric contraction.



ployed. 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 *t* tests was performed with a modified Bonferroni correction factor to determine where significant differences existed. A power calculation was performed on velocity-dependent power to ensure that there was sufficient power ($1 - \beta = 0.88$) to detect significant differences. 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 (before testing).

Table 1. Voluntary and electrically evoked neuromuscular properties of the dorsiflexors.

	Electrically evoked isometric properties								
Group	P_d (N·m)	10 Hz (N·m)	50 Hz (N·m)	50 Hz_{HRT} (ms)	MVC (N·m)	MVC_{RTD} ((N·m)·s^{-1})	V_{MAX} (°·s^{-1})	PP (W)	%MVC_{PP} (%)
Male (8)	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 (8)	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

Note: Females had lower absolute evoked peak doublet twitch torque (P_d; $P < 0.05$), 10 Hz peak torque ($P < 0.05$), and 50 Hz peak torque ($P < 0.05$) compared with males, but 50 Hz 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 the MVC (MVC_{RTD}; $P < 0.01$), maximum shortening velocity determined at 1 N·m (V_{MAX}; $P < 0.01$), and peak power (PP (the greatest power value achieved from across the various loads); $P < 0.01$) were less in females than males. The relative load at which peak power (%MVC_{PP}) was achieved was lower for females than males ($P < 0.01$). Asterisk (*) denotes significant sex difference. Values are means ± SD.

Results

Baseline measures

As shown in Table 1, females were 34% weaker than males for MVC torque ($P < 0.01$), despite similar values for voluntary activation (~95%, $P = 0.68$) and optimal angle of torque production ($P = 0.78$). Females had a 31% lower P_d than males ($P < 0.05$), whereas groups did not differ for all other doublet contractile properties (DTPT, $P = 0.58$; DHRT, $P = 0.86$; Table 1), and both sexes had a similar capacity for twitch potentiation (124% ± 16%) ($P = 0.98$). Females had a 20% lower absolute isometric rate of torque development for the MVC (MVC_{RTD}) ($P < 0.01$) than males, but when normalized to MVC, both groups were similar ($P = 0.15$). Maximal (1 N·m) angular velocity was 13% slower ($P < 0.01$), and peak power (Tables 1 and 2), which was 49% lower in females ($P < 0.01$), was reached at a 19% lighter load. Both groups had a similar mean magnitude of neuromuscular activity ($P = 0.54$).

Lengthening contraction task

Participants reported no muscle soreness immediately preceding the lengthening contraction task (0 ± 0 mm) and mild to no muscle soreness within the first 30 min of recovery (13.1 ± 7.1 mm). Soreness peaked 24 h after task termination (27.6 ± 10.3 mm) and returned to mild to no soreness (9.4 ± 9.2 mm) within 48 h, with no detectable differences between groups ($P = 0.42$). For dorsiflexor MVC torque, there was a significant time × sex interaction ($P < 0.05$), indicating that although isometric MVC torque decreased similarly in the males and females during the task (25%–30%), females had a more depressed recovery from 10 min to 24 h. Females recovered to 81.3% ± 5.5% of baseline, whereas males recovered to 89.2% ± 8.1% by 30 min after task termination (Fig. 4), indicating that females incurred more overall neuromuscular dysfunction from the lengthening contractions. By 48 h, both males and females recovered fully. Voluntary activation was well maintained throughout the task (>95%, $P = 0.91$) and recovery. The optimal angle of torque production showed a main effect for time ($P < 0.01$), with a change between 2° and 8° in both males and females, and neither was recovered by 24 h.

Dynamic responses

All participants were able to complete the lengthening contraction task, with eccentric torque for both males and females decreased similarly by 27.8% ± 10.9% throughout the protocol ($P = 0.27$). Neuromuscular measures were analyzed and compared with baseline and reported as a relative change over time. For maximum angular shortening velocity (Fig. 5A) and peak power (Fig. 5B), there were main effects for time ($P < 0.01$ and $P < 0.01$, respectively) and a trend for a main effect for sex during recovery of angular velocity ($P = 0.07$) and peak power ($P = 0.06$), respectively. At task termination, both males and females had a similar 4% loss of maximal shortening velocity but a 10% loss of peak power ($P < 0.05$). The reduction in maximal angular velocity persisted until 30 min into recovery, and peak power did not recover until 24 h.

For velocity-dependent power loss across loads (Figs. 6A–6H), there was a time × sex × load interaction ($P < 0.01$), with main effects for each factor time ($P < 0.01$), sex ($P < 0.01$), and load ($P < 0.01$). Although both sexes showed a preferential loss of power at the heavier loads, females had a greater loss than males. Both sexes recovered similarly when tested at lighter loads (<30% MVC), but females had a slower recovery than males for heavier loads ($P < 0.01$), recovering by 48 h, whereas males recovered by 24 h. The tibialis anterior mean magnitude of neuromuscular activation remained similar to baseline (before, 1.8–3.5 mV; after, 1.7–3.9 mV) throughout recovery ($P = 0.76$) and did not differ between males and females ($P = 0.66$), indicating that impairments in neuromuscular function likely occurred distal to the neuromuscular junction.

Table 2. Angular velocity reached at each relative load for males and females.

Load	Male	Female
1 N·m	167.5±12.4	145.6±10.3*
10% MVC	150.0±15.5	130.3±12.0*
20% MVC	134.2±13.6	116.4±11.7*
30% MVC	122.6±13.4	102.9±12.5*
40% MVC	103.5±12.9	83.6±17.2*
50% MVC	83.4±17.5	59.5±18.9*
60% MVC	56.9±15.9	35.9±19.9*
70% MVC	31.0±10.3	11.0±17.3*

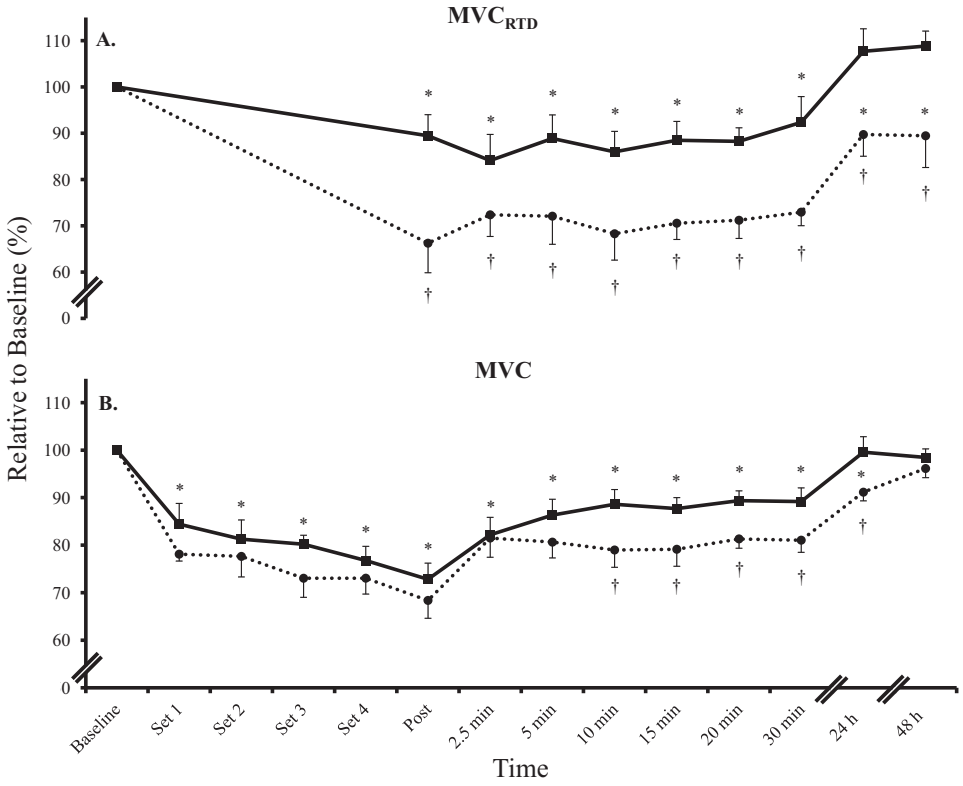
Note: Females had lower absolute angular velocities (°·s^{-1}) across all loads ($P < 0.01$). Values are means ± SD. Asterisk (*) denotes significant sex difference.

Voluntary isometric and electrically evoked responses

Rate of torque development for the isometric MVC following lengthening contractions showed a significant time × sex interaction ($P < 0.05$) and was reduced more in females (66.2% ± 18.1% of baseline) than males (89.5% ± 12.9% of baseline) (Fig. 4A). Rate of torque development for the MVC remained depressed in both sexes throughout the short-term 30 min recovery period, but males recovered by 24 h, whereas females did not show any significant recovery (Fig. 4A). There was a significant effect for time ($P < 0.01$) but not sex ($P = 0.48$) for low-frequency torque depression (10 Hz : 50 Hz). Males and females had a similar decrease to 83.6% ± 16.7% of baseline by 30 min and recovered fully within 48 h. Alterations in 10 Hz : 50 Hz were manifested by the greater reduction in 10 Hz evoked torque compared with the 50 Hz (Figs. 7A–7C). During the task, 50 Hz torque decreased ($P < 0.05$) similarly in females and males and recovered in both by 24 h. The 10 Hz torque was significantly depressed throughout recovery in both males and females ($P < 0.05$). Females did not recover fully ($P < 0.01$) by 48 h, but males recovered within 24 h (Fig. 7B). Reductions in 10 Hz : 50 Hz indicated that there was significant low-frequency torque depression following the lengthening contractions for both males and females but a greater impairment in females throughout recovery.

For twitch torque, there was a main effect for time ($P < 0.01$). Peak twitch torque was potentiated to 127.0% ± 17.7% immediately after task equally in both males and females. By 30 min of recovery, the potentiating effects decayed and twitch torque was

Fig. 4. (A) Maximal rate of torque development and (B) maximal voluntary isometric contraction (MVC) before and following the lengthening contraction intervention. Maximal rate of torque development was impaired more in females than males, with the males recovering by 24 h while females did not recover fully by 48 h. Maximal voluntary strength was reduced similarly in males (solid line; square) and females (dotted line; circle), with a depressed recovery in females. MVC_{RTD}, rate of torque development for the MVC. Values are means ± SE. Asterisk (*) denotes significant effect of time; dagger (†) denotes significant effect of sex.



reduced to 82.4% ± 17.5% of baseline. Electrically evoked contractile speeds had main effects of time for DTPT ($P < 0.01$) and DHRT ($P < 0.01$), slowing similarly in both groups by 15%–20% compared with the baseline.

Discussion

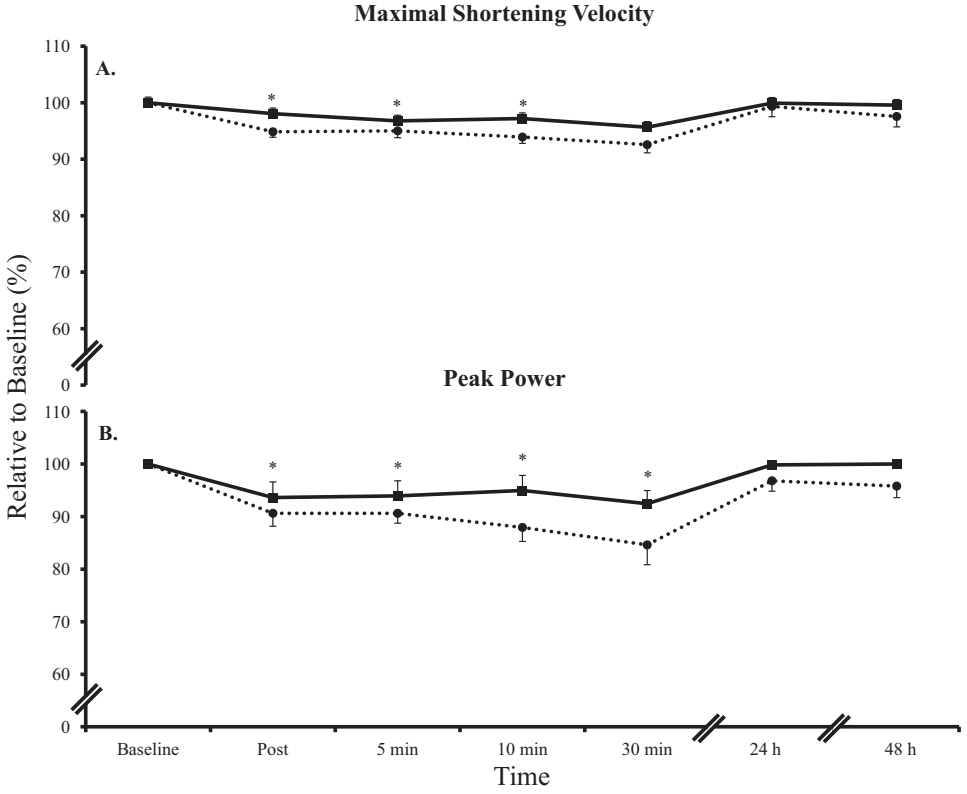
We investigated the effects of repeated high-intensity lengthening contractions on velocity-dependent power loss over a range of isotonic loads. Results indicated that there was a greater loss of power at higher loads (Fig. 6), mainly due to impaired torque-generating capacity; however, at lower loads (<20% MVC), angular velocity was impaired only up to and including 10 min of the recovery period. The initial impairment in angular velocity at lower loads seemed to be related to the transient effects of fatigue, which recovered fully within 30 min. Furthermore, a sex difference was observed in which females had a greater loss of power than males when tested at loads >50% MVC, and this effect persisted throughout recovery (Fig. 6). Achievement of peak power shifted to lighter loads and was reduced similarly (10%) in both sexes following the protocol but recovered fully within 24 h. The greater loss of power at heavier loads in females than males appears to be driven by those factors affecting strength loss and not shortening velocity, as reflected fundamentally by the inability to generate torque rapidly (i.e., rate of torque development) and a shift in the torque–velocity–power relationship to lighter loads and faster relative angular velocities. Irrespective of the mechanism, alterations in neuromuscular function as a result of unaccustomed lengthening contractions seem to differ with sex, with greater impairments in neuromuscular function in women when tested at heavier loads.

Strength loss following lengthening contractions

Although baseline MVC torque was 34% greater in males, 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. Females did not recover until 48 h, whereas males recovered fully within 24 h following lengthening contractions (Fig. 4B). These results support previous findings in which sex-related differences in response to damaging lengthening contractions were observed (Sayers and Clarkson 2001; Sewright et al. 2008). The initial and similar decline in MVC amplitude for both females and males in the present study may have occurred via different mechanisms, as indicated by their divergent recovery profiles. By 30 min, the females recovered to only 81% of baseline, whereas males recovered to 89%. It has been shown that females are more fatigue-resistant during repetitive intermittent tasks (Hicks et al. 2001; Hunter 2009), and in the present study, females recovered to a lesser extent than males by 30 min. Thus, it is likely that the decline in MVC for the females can be attributed to greater damage-induced impairments rather than the transient effects of fatigue as suggested for males. The different recovery profiles indicate that females experienced a longer lasting impairment in muscle function, whereas the impairment in males was transient and may be explained by a greater initial fatigue response compared with the females.

Previously, we reported no change in neuromuscular (EMG_{RMS}) or voluntary activation following lengthening contractions of the ankle dorsiflexors (Power et al. 2010, 2011, 2012*a*), and these results are in line with our current study in which the mean magnitude of neuromuscular activity did not differ from baseline for either sex. No changes in EMG amplitude indicate that peripheral mecha-

Fig. 5. (A) Maximal shortening velocity and (B) peak power. Maximal shortening velocity determined at 1 N·m was impaired minimally in both males (solid line; square) and females (dotted line; circle) and recovered fully by 30 min. Peak power was reduced similarly in males and females and recovered fully by 24 h. Values are means ± SE. Asterisk (*) denotes significant effect of time.



nisms are responsible fundamentally for decrements in performance following lengthening contractions in this model. Both sexes had a similar increase in perceived muscle soreness and increase in muscle series compliance, as indicated by the shift in optimal angle for torque production to longer lengths. Although females and males seem to have experienced a similar level of structural impairment, there remained a greater degree of strength loss in the females. To account for this disparity, it is reasonable to suggest that excitation–contraction (E–C) coupling was more impaired in females than males, which is supported by a 15% greater reduction in the 10 Hz torque for the females by 24 h of recovery (Fig. 7B). The main contributor to E–C uncoupling following muscle damage is impaired Ca^{2+} release (Edwards et al. 1977; Ingalls et al. 1998). Thus, sex-related differences in E–C uncoupling may also be related to a greater impairment in Ca^{2+} channel regulation following lengthening contractions in females than males in response to differing sex hormone levels, as suggested for cardiac myocytes in mice (Farrell et al. 2010). Although a direct link between cardiac myocytes and skeletal muscle fibers has not been made, it is conceivable that the sex difference in E–C uncoupling following the lengthening contractions could be attributed to less Ca^{2+} released from the female sarcoplasmic reticulum owing to a smaller safety factor than males. This could indicate the greater impairment in females than males possibly was due to an impaired ability to engage a sufficient quantity of viable force generators.

Velocity and power

Strength loss and the reductions in angular velocity at each relative isotonic load altered the *T*–*V* relationship and thus power production following the lengthening contractions. Due to muscle weakness immediately following the task, the imposed isotonic loads represented a relative higher percentage of initial MVC and required a greater relative reliance on torque generation compared with the baseline from all subjects to accomplish the task successfully. Peak power was reduced similarly in females and males, and power assessed at heavier loads (i.e., >50% MVC) was attenuated to a greater extent than when assessed at lighter loads in both sexes. Following the lengthening contractions, power did not recover at heavier loads for either sex, but for the lighter loads (<20% MVC), power recovered within 30 min equally for both sexes. The shift in peak power to lighter loads allowed the muscle to compensate for the associated muscle weakness and impaired velocity-dependent power at higher loads by relying more on the velocity component for power generation with less dependence on strength and RTD.

Dynamic performance, specifically power production, is highly dependent on ballistic force generation and may be related to the ability to generate torque rapidly (Andersen et al. 2005). Following lengthening contractions, in our study, females had a 35% reduction in RTD, whereas males exhibited a 10% decrement. This greater reduction in RTD for females compared with males may be a key factor responsible for the observed power loss following the lengthening contractions, particularly at higher loads. 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 (Abbott and Wilkie 1953; Lieber and Ward 2010). The increase in muscle series compliance likely reflected by the change in optimal angle of torque production to longer muscle lengths (Fig. 1) could have contributed to power loss, particularly the ability to generate torque at higher loads. Females reached peak power at a lower %MVC than males; therefore, the additional loss of strength fol-

Fig. 6. Power loss across multiple loads following lengthening contractions. Each panel represents a recovery time point as indicated. Males (solid line; square) and females (dotted line; circle). Values are means ± SE. Asterisk (*) denotes significant effect of time; dagger (†) denotes significant effect of sex.

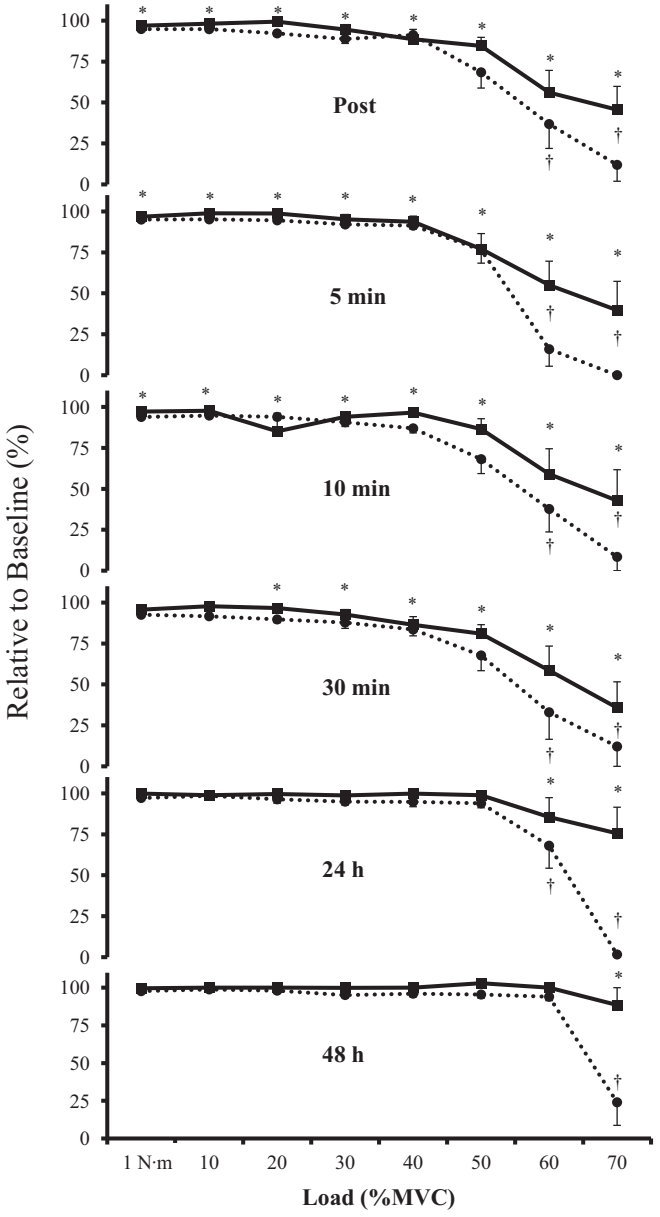
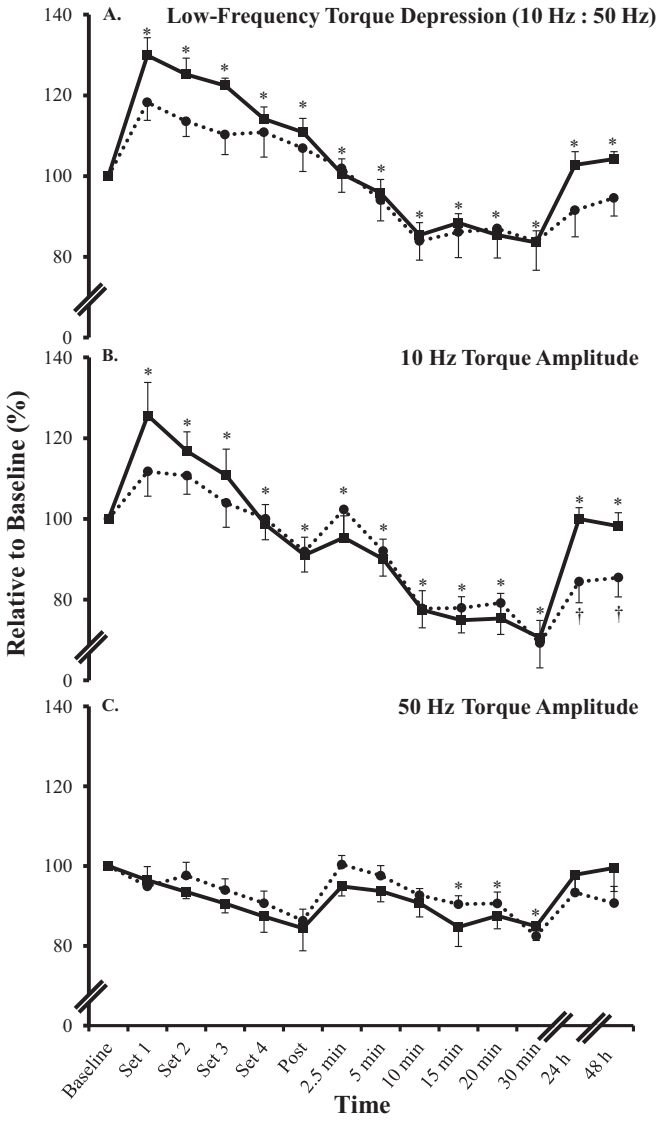


Fig. 7. (A) Low-frequency torque depression as a combined consequence of impaired (B) 10 Hz torque and (C) 50 Hz torque. A significant increase in low-frequency torque depression in males (solid line; square) and females (dotted line; circle), as shown by the decreased 10 Hz : 50 Hz, was present throughout recovery, with a trend towards greater low-frequency torque depression in females ($P = 0.07$). The decreased ratio was driven by the 10 Hz component, which also presented a sex difference throughout recovery. Values are means ± SE. Asterisk (*) denotes significant effect of time; dagger (†) denotes significant effect of sex.



lowing lengthening contractions created an even greater loss of power at heavier loads in females compared with males.

Power loss at lighter loads (i.e., <20% MVC) recovered quickly, likely due to transient fatigue-related impairment in shortening velocity. Muscle damage, however, leads to longer lasting impairments (Choi and Widrick 2009; Morgan et al. 2004) in torque generation, and therefore, power production capacity is unable to recover at higher loads. However, we acknowledge that it is difficult to determine the relative influences of fatigue processes versus muscle damage in their contributions to impaired short-term neuromuscular performance. The change in angular velocity reached at each load following lengthening contractions is consistent with velocity-specific alterations in the *T*–*V* relationship. As reported previously (Power et al. 2010), when velocity-dependent power is tested at a single moderate load (20% MVC) following lengthening contractions, the compromised torque production of the muscle is not stressed as the velocity of contraction was fast with little need for high torque generation to achieve peak power. The results of our present study seem to indicate that the impairment in torque generation capacity and velocity-dependent power production are related. Velocity-specific alterations in power are observed during isovelocity (i.e., isokinetic) movements in which the constrained angular velocity is high enough to not impede torque production (Gibala et al. 1995). However, when tested at fast velocities, the isovelocity model does not permit the relative impact of torque and angular velocity on power production to be discriminated. Therefore, this important aspect of maintained angular velocity was not previously recognized when assessing muscle power using a single moderate load (Power et al.

2010). From a practical aspect, it seems that men and women will respond differently to exercises involving unaccustomed eccentric exercises.

Repetitive lengthening contractions fatigued and temporarily weakened the dorsiflexors, thus impairing their ability to generate torque maximally and rapidly. This impairment in torque generation performance 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, females displayed a depressed recovery relating to a greater and longer lasting failure in E–C coupling than males, which was presumably attenuated by Ca^{2+} release from the sarcoplasmic reticulum. Thus power generation was further impaired at higher loads in the females than the males, while maximal shortening velocity (i.e., near unloaded) was well maintained in both sexes.

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