J Appl Physiol 109: 669–676, 2010.
First published June 24, 2010; doi:10.1152/japplphysiol.01254.2009.

Delayed recovery of velocity-dependent power loss following eccentric actions of the ankle dorsiflexors

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Submitted 5 November 2009; accepted in final form 20 June 2010

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. First published June 24, 2010; doi:10.1152/japplphysiol.01254.2009.—Unaccustomed eccentric exercise has been shown to impair muscle function, although little is known regarding this impairment on muscle power. The purpose of this study was to investigate changes in neuromuscular properties of the ankle dorsiflexors during and after an eccentric contraction task and throughout recovery in 21 (10 men, 11 women) recreationally active young adults (25.8 ± 2.3 yr). All subjects performed 5 sets of 30 eccentric contractions at 80% of maximum isometric voluntary contraction (MVC) torque. Data were recorded at baseline, during the fatigue task, and for 30 min of recovery. There were no significant sex differences for all fatigue measures; thus data were pooled. After the fatigue task, MVC torque declined by 28% ($P < 0.05$) and did not recover fully, and voluntary activation of the dorsiflexors, as assessed by the interpolated twitch technique, was near maximal (>99%) during and after the fatigue task ($P > 0.05$). Peak twitch torque was reduced by 21% at 2 min of recovery and progressively decreased to 35% by 30 min ($P < 0.05$). Low-frequency torque depression (10-to-50 Hz ratio) was present at 30 s of recovery, increased to 51% by 10 min, and did not recover fully ($P < 0.05$). Velocity-dependent concentric power was reduced by 8% immediately after task termination and did not recover fully within 30 min ($P < 0.05$). The main findings of an incomplete recovery of MVC torque, low-frequency torque depression, and shortening velocity indicate the presence of muscle damage, which may have altered excitation-contraction coupling and cross-bridge kinetics and reduced the number of functional sarcomeres in series, ultimately leading to velocity-dependent power loss.

isotonic power; muscle fatigue; sex differences; electromyography; muscle damage

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 (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 excitation-contraction (EC) 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 approximately four times 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 on 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, the muscle group involved, and the 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, after 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 [maximum isometric voluntary contraction (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 better than isometric torque. However, only isokinetic power has been reported after eccentric con-

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tractions (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, is 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 more greatly affect 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 less 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 that there will be a modest reduction in shortening velocity resulting in velocity-dependent power loss that will remain blunted 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.

MATERIALS AND METHODS

Participants. Ten young men (25.6 ± 2.9 yr) and eleven young women (26.0 ± 1.7 yr) 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 before testing.

Participants visited the laboratory on two occasions separated by 7 days. All participants were recreationally active with no known neurological or cardiovascular diseases. The first session was familiarization to the testing procedures, and data were collected during the second session. Participants were asked to refrain from strenuous exercise 1 day before testing and not to consume caffeine on the day of testing.

Experimental setup. A Biodex multijoint dynamometer (System 3, Biodex Medical Systems, Shirley, NY) 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 ~45° 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 with nonelastic shoulder, waist, and thigh straps. Participants sat in a slightly reclined position with hip, knee, and ankle angles at ~110°, ~140°, and ~30° plantar flexion, respectively. All isometric dorsiflexion contractions were performed at 30° of plantar flexion. Concentric contractions began from the plantar flexed position of 30° and ended at the neutral ankle angle (0°). The eccentric contractions started at the neutral ankle angle and ended at 30° plantar flexion, thus moving through a 30° 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 preprogrammed 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 article we refer to these contractions as velocity dependent.

Surface electromyography (EMG) was collected from the tibialis anterior and soleus muscles with self-adhering Ag-AgCl electrodes (1.5 × 1 cm; Kendall, Mansfield, MA). The skin was rubbed vigorously with alcohol before application of the electrodes. A monopolar electrode setup 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 was placed over the calcaneal tendon.

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

Experimental procedures. Peak twitch torque (P_t) 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 (2 pulses at 10-ms interpulse interval) to assess voluntary activation. Next, 100 Hz peak torque (P_{100}) was determined by increasing the current until there was a plateau in P_{100} (20–65 mA). A torque-frequency relationship was constructed by 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 P_{100} with 1 s between trains.

Three MVCs of 3- to 5-s duration were then performed. 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 with 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 after the MVC. Percent voluntary activation was calculated as voluntary activation (%) = [1 − (interpolated twitch doublet/resting twitch doublet)] × 100. Values from the peak MVC were used for data analysis. Once MVC torque was determined, the dynamometer was switched from the isometric to the 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 can 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 ~60% 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. After practice, two contractions were performed to establish values for peak shortening velocity at baseline.

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 and 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 footplate 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 after each of the five sets, and throughout the recovery period at 0.5 min, 2 min, 5 min, 10 min, 15 min, 20 min, and 30 min (Fig. 1). Measures after the fatigue protocol included the following, performed in the following order: *1*) maximum evoked twitch properties, *2*) assessment of MVC and voluntary activation, *3*) postactivation twitch and twitch doublet, *4*) a measure of low-frequency torque depression (10-to-50 Hz ratio), and *5*) velocity-dependent concentric power.

Data reduction and analysis. Torque, position, and velocity data were sampled at a rate of 100 Hz. All data were converted to digital format with a 12-bit analog-to-digital converter (model 1401 Plus, Cambridge Electronic Design, Cambridge, UK). Surface EMG signals were preamplified (×100), amplified (×2), band-pass filtered (10–1,000 Hz), and sampled online at 2,500 Hz with Spike 2 software (version 6.10, Cambridge Electronic Design). 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 were calculated during the lowering phase through the 30° range of motion and then normalized to the M wave. Postactivation potentiation was calculated by comparing the twitch following the MVC to the baseline twitch. Power was calculated as the product of torque (Nm) 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, P_t, peak doublet torque (D_t), 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 by using a ratio of peak 10 Hz- 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.

Statistics. With SPSS software (version 16, SPSS Chicago, IL) a two-way (sex × 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 honestly significant difference (HSD) post hoc test was performed to identify where significant differences existed. Values are presented as means ± SD in Table 1 and as means ± SE in Figs. 3, 5, and 6.

RESULTS

Baseline measures. As expected, because of differences in anthropometrics, men had higher values for absolute measures of P_t, MVC torque, velocity, and power than women, ~49%, 30%, 16%, and 38%, respectively (Table 1). When absolute values were compared men were stronger than women ($P < 0.05$) at every stimulation frequency, but when the torque frequency curves (Fig. 2) were normalized to 100 Hz torque there were no differences in the relationship between men and women ($P > 0.05$). Evoked torque corresponded to ~62% and 50% of MVC torque for 50 Hz and 64% and 52% of MVC torque for 100 Hz for men and women, respectively.

Fatigue and recovery measures. All participants were capable of completing the 5 sets of 30 eccentric contractions, although some subjects had difficulty in lowering the footplate at a constant velocity for the last few contractions of each set. This failure to maintain a constant velocity resulted in increased eccentric velocities that 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

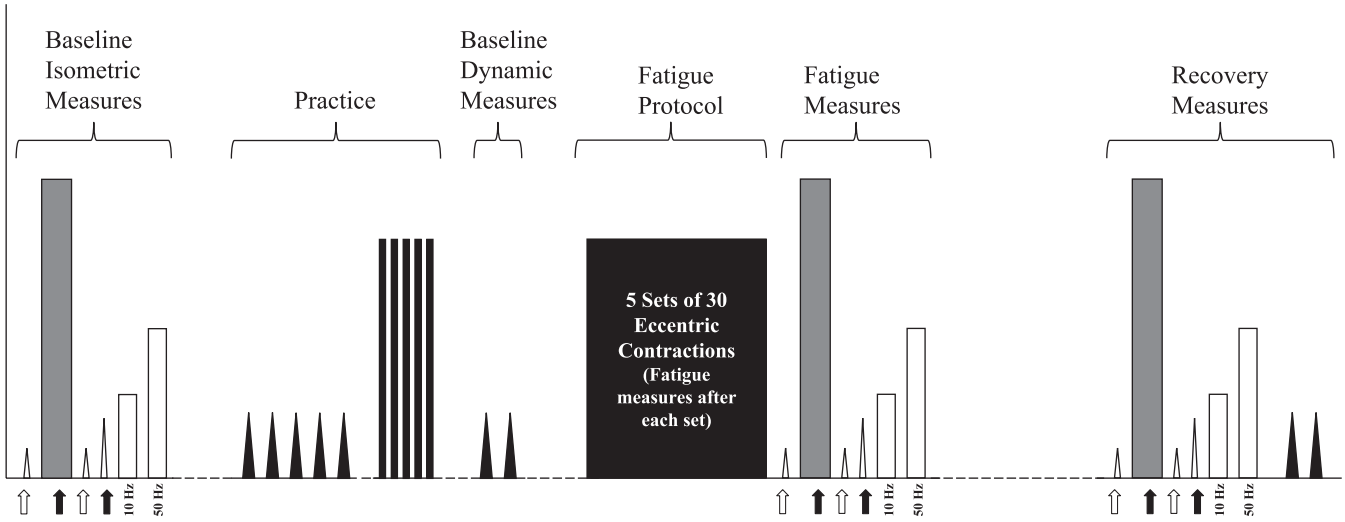

Fig. 1. Schematic diagram of experimental protocol. Gray bars are maximum isometric voluntary contractions (MVC). Open profiles are electrically evoked contractions [twitches (small triangles), doublet (large triangles), 10 Hz and 50 Hz (bars)]. Filled profiles are dynamic concentric contractions at 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 0.5, 2, 5, 10, 15, 20, and 30 min.

Table 1. *Baseline contractile data*

Sex	Twitch Properties				MVC, Nm	VA, %	Velocity, °/s	Power, W
	Torque, Nm	TPT, ms	HRT, ms	CD, ms				
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

Values are means ± SD. 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.

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 ($P < 0.05$) after the first set of 30 eccentric contractions (Fig. 3). The MVC torque progressively decreased after each successive set to 72% of baseline immediately after task termination and did not recover fully. There were no significant changes from baseline ($P > 0.05$) in RMS EMG of the agonist tibialis anterior during MVCs, and voluntary activation was >99% at baseline and did not change ($P > 0.05$; Fig. 4) throughout fatigue and recovery. Conversely, soleus RMS EMG during MVCs increased ($P < 0.05$) to 111 ± 21% of baseline after 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 of 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 tibialis anterior normalized to M wave did not differ significantly ($P > 0.05$) among sets.

Twitch potentiation increased to 130 ± 16% from baseline after the first set of 30 contractions and 140 ± 28% of baseline immediately after 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 of 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 after the third set of contractions and was further reduced to 63 ± 11% of baseline by 30 min. Twitch doublet contractile property 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 10 Hz was 13.9 ± 5.7 Nm at baseline, was reduced to 64 ± 24% of baseline immediately after the eccentric exercise ($P < 0.05$), and did not recover fully. As well, peak torque of 50 Hz (baseline 24.0 ± 10.2 Nm) was reduced only to 85 ± 16% of baseline after the second set of eccentric contractions ($P < 0.05$) and to 79 ± 15% of baseline immediately after task termination and did not recover fully. The change in 10:50 Hz was manifested by the greater reduction in 10 Hz-evoked torque compared with 50 Hz.

10:50 Hz decreased to 28% of baseline immediately after task termination, continued to decrease to 47% of baseline ($P < 0.05$) at 10 min of recovery (Fig. 5), and remained blunted. This indicated that there was significant low-frequency torque depression after the last set of eccentric contractions.

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 1. Maximum shortening velocity and subsequently velocity-dependent power were reduced to 92% of baseline immediately after the fatigue protocol ($P < 0.05$; Fig. 6), and neither recovered fully.

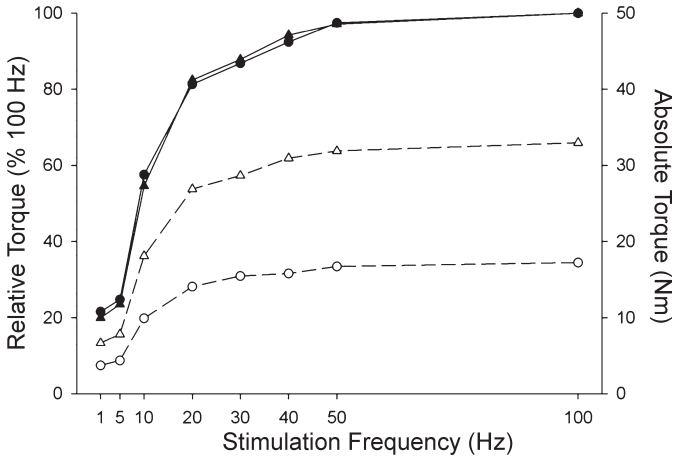

Fig. 2. Torque-frequency relationship. △, men absolute torque; ▲, men relative torque; ○, women absolute torque; ●, women relative torque. Men had higher absolute torques at all frequencies (1–100 Hz) compared with women ($P < 0.05$). Relative torques were similar at all stimulation frequencies ($P > 0.05$).

DISCUSSION

We tested the hypothesis that after 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 blunted throughout recovery. The main findings indicate that velocity-dependent power loss occurred immediately after 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 that after a bout of eccentric muscle contractions there is a reduction in velocity-dependent power driven by impairment in maximum shortening velocity.

With normalization to prefatigue 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 (Fig. 2) and twitch contractile speeds (time to peak twitch, half-relaxation time, and CD) (Table 1), were not different between the men and women. Thus both groups may have similar muscle properties

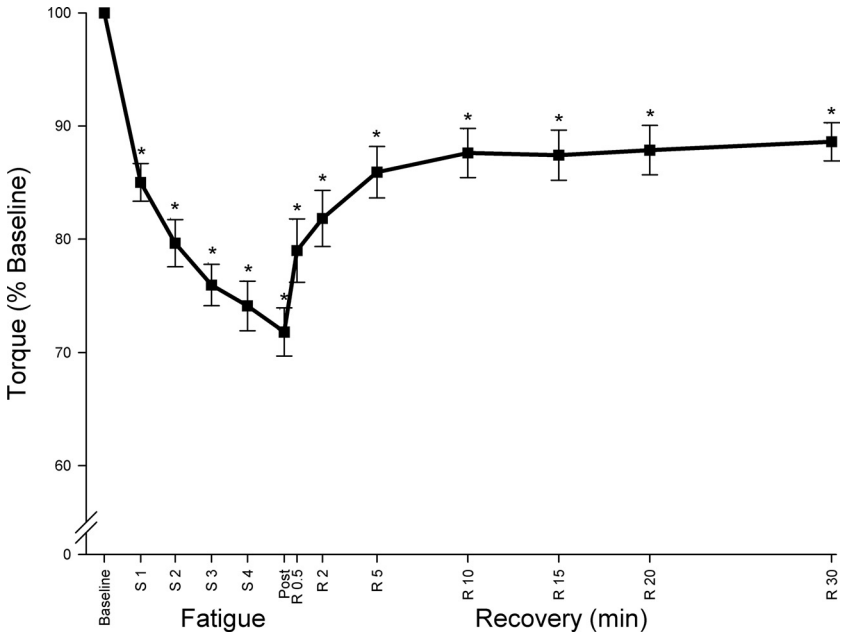

Fig. 3. MVC. Maximal voluntary strength was reduced after the first set (S1) of eccentric contractions and continued to decline to ~70% of baseline at Post (task termination), and it did not recover fully (*$P < 0.05$) within 30 min. S, set; R, minutes of recovery. Values are means ± SE.

(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% after 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% after eccentric exercise (25, 40). Thus the ability to fully activate the dorsiflexors, even when the muscle is stressed severely or in this case has undergone damaging lengthening contractions, is unique.

A recent investigation (30) found that motor unit conduction velocity in the quadriceps was decreased after eccentric exercise because of sarcolemmal damage. However, excitation failure of the sarcolemma cannot account for the torque and power depression in the ankle dorsiflexors because, similar to other reports (48), M-wave properties (area, duration, amplitude) did not change during and after task termination. This was further corroborated with the findings from the electrically evoked contractions. For example, P_t declined by 21% 2 min after task termination. Concomitantly, twitch potentiation, which could offset the initial fatigue response in P_t, was no longer measurable at that time, and P_t remained depressed. Similarly, the 10 Hz- and 50 Hz-evoked torques were reduced after the eccentric exercise and did not recover fully. As previously observed (9, 48) after eccentric exercise of the ankle dorsiflexors, the contractile speeds (time to peak twitch, half-relaxation time, and CD) 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).

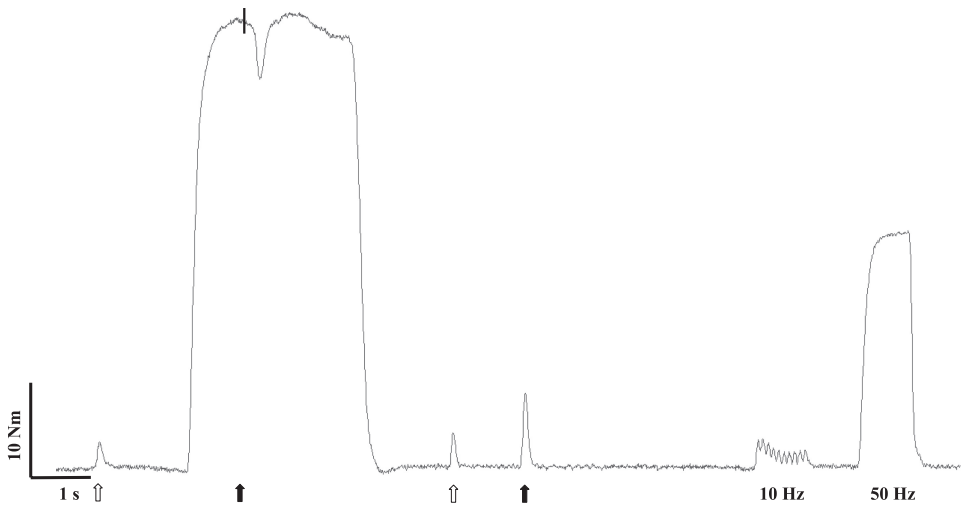

Fig. 4. Torque output and activation for a representative subject at 30 min of recovery. The vertical bar on the torque tracing represents the evoked doublet. Open arrows indicate electrically evoked twitches, and filled arrows indicate electrically evoked doublets.

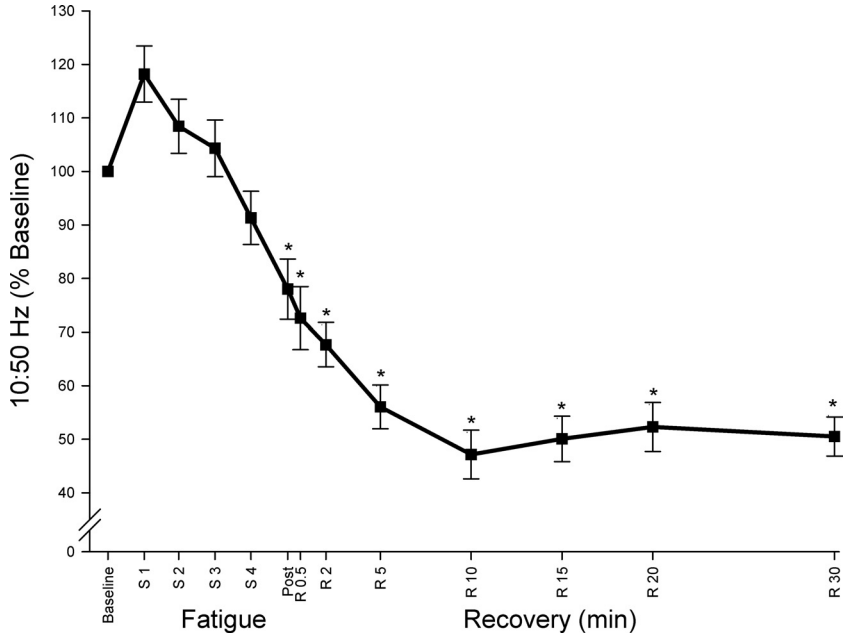

Fig. 5. Low-frequency torque depression [10-to-50 Hz ratio (10:50 Hz)]. A significant increase in low-frequency torque depression as shown by the decreased 10:50 Hz was present at Post (task termination) with a continued decrease in 10:50 Hz until 10 min and remained depressed for 30 min (*$P < 0.05$). Values are means ± SE.

Subsequently, mechanical disruption of the link between the t tubule and the sarcoplasmic reticulum leads to EC uncoupling, which remains as the likely peripheral impairment responsible for the immediate torque and power loss (2, 35, 59). The most plausible stage of EC coupling that was impaired after 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 10:50 Hz, and MVC. 10:50 Hz decreased immediately after 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 10:50 Hz was manifested by the greater reduction in 10 Hz- than 50 Hz-evoked torque. This further supports an impairment in EC 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 h 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 after 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 after eccentric contrac-

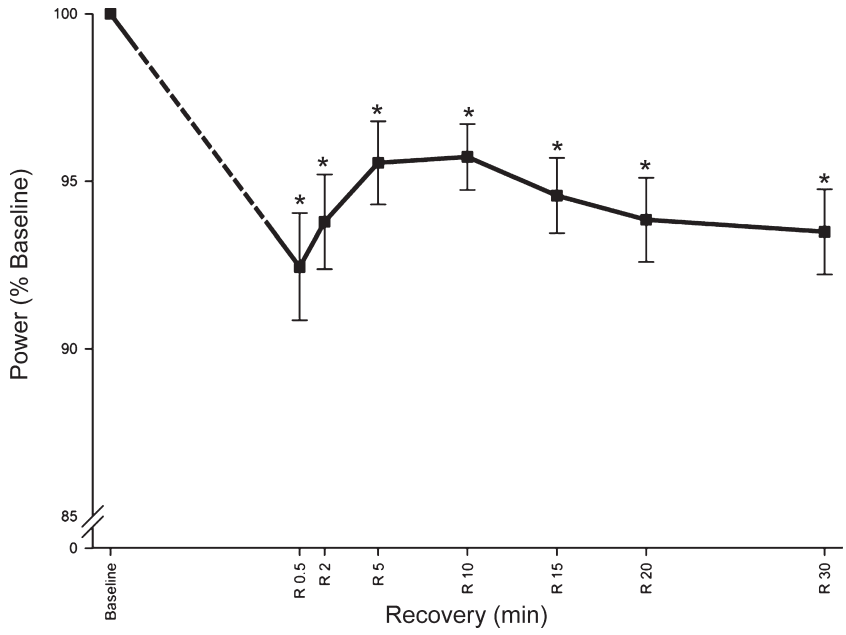

Fig. 6. Velocity-dependent power. Velocity-dependent concentric power was reduced by 8% at Post (task termination) compared with baseline and did not recover fully within 30 min (*$P < 0.05$). Values are means ± SE.

tions 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% after eccentric exercise and did not recover fully. These observations are unlike previous reports that 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 after concentric contractions. For example, Cheng and 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 by using the isotonic mode of the Biodex to evaluate eccentric fatigue-induced reductions in shortening velocity that 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 that 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 (~15° 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) can account for the initial decrease in shortening velocity, where excessive ADP surrounding the contractile proteins actin and myosin results in slower cross-bridge cycling. However, because of 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" is reduced (45, 46), resulting in a reduced shortening velocity, as well as 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 blunted recovery following eccentric exercise.

Although the present study cannot determine the specific mechanisms of reduced power, we found significant EC 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 after task termination. In summary, when velocity-dependent contractions are used as the criterion measure to calculate power, we demonstrated that after 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.

ACKNOWLEDGMENTS

We thank all those who participated in the study and give special thanks to Dr. Arthur Cheng for technical assistance and insightful discussions during the preparation of this manuscript.

GRANTS

This research is supported by funding from the Newfoundland and Labrador Center for Applied Health Research (NLCAHR) and the Natural Sciences and Engineering Research Council of Canada (NSERC).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

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