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This article was originally published in: Journal of Strength and Conditioning Research (2011), 25 (1) 211-219 Original article available here
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Effect of Slow-Velocity Lengthening Contractions on Muscle Damage Induced by Fast-Velocity Lengthening Contractions

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Abstract

Chapman, DW, Newton, MJ, McGuigan, MR, and Nosaka, K. Effect of slow-velocity lengthening contractions on muscle damage induced by fast-velocity lengthening contractions. *J Strength Cond Res* 25(1): 211–219, 2011—This study tested the hypothesis that the first exercise bout consisting of slow-velocity ($30°\cdot s^{-1}$) maximal lengthening contractions would not affect muscle damage in a subsequent bout consisting of fast-velocity ($210°\cdot s^{-1}$) lengthening contractions. Eighteen men were randomly assigned into either a repeated bout group ($n = 10$) or control group ($n = 8$). The repeated bout group performed 2 bouts of exercise consisting of 210 maximal lengthening contractions of the elbow flexors separated by 14 days at a velocity of $30°\cdot s^{-1}$ for the first and $210°\cdot s^{-1}$ for the second bout. The control group performed a single bout of the fast-velocity exercise. Changes in maximal isometric strength, range of motion (ROM), upper-arm circumference, muscle thickness, muscle soreness, serum creatine kinase, and lactate dehydrogenase activities were measured before, immediately after, and 24 to 96 hours after exercise. The repeated bout group showed significantly ($p < 0.05$) smaller changes in all criterion measures except for muscle soreness after the fast-velocity exercise compared with the control group. A significant ($p < 0.05$) difference was evident only for ROM between the slow- and fast-velocity bouts of the repeated bout group. These results suggest that slow-velocity exercise reduced muscle damage induced by fast-velocity exercise, although the reduction was not large.

Key Words eccentric exercise, repeated bout effect, contraction velocity, muscle strength, muscle soreness, creatine kinase

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25(1)/211–219

Journal of Strength and Conditioning Research
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Introduction

A bout of eccentric exercise reduces muscle damage in a subsequent bout of the same or similar eccentric exercise (10,21), and this adaptation is known as the "repeated bout effect" (22). Previous investigations have identified factors influencing the repeated bout effect including intensity (25,28), number of contractions (3,8), muscle length (20,26), mode of exercise of the initial bout (23,28), and time period between bouts (4,24,30). However, no previous study has investigated the effect of lengthening contraction velocity on the repeated bout effect.

It has been shown that lengthening contraction velocity affects the magnitude of muscle damage. For example, Chapman et al. (6) reported that changes in isometric strength, range of motion (ROM), upper-arm circumference, muscle soreness, and plasma creatine kinase (CK) activity were significantly greater after a fast-velocity ($210°\cdot s^{-1}$) than a slow-velocity ($30°\cdot s^{-1}$) eccentric exercise of the elbow flexors when the total muscle contraction time was matched. This was confirmed in the subsequent study (5) in which the number of lengthening contractions was matched between the fast- ($210°\cdot s^{-1}$) and slow- ($30°\cdot s^{-1}$) velocity lengthening contractions of the elbow flexors.

If a slow-velocity eccentric exercise bout that does not induce as severe muscle damage as a fast-velocity eccentric exercise can still reduce potential severe muscle damage induced by a fast-velocity eccentric exercise, this strategy could be used to minimize muscle damage. Conversely, it is conceivable that only a fast-velocity eccentric exercise provides protection against muscle damage induced by a fast-velocity eccentric exercise because the population of muscle fibers affected by eccentric exercise may be velocity dependent (33). As such, it is important for strength and conditioning practitioners to understand the effect of lengthening contraction velocity on repeated bout effect.

No previous study has investigated the effect of slow-velocity eccentric exercise on fast-velocity eccentric exercise. Therefore, this study tested the null hypothesis that a bout of maximal eccentric exercise performed at a slow ($30°\cdot s^{-1}$) velocity would not affect the magnitude of muscle damage induced by a subsequent bout of maximal eccentric exercise performed at a fast ($210°\cdot s^{-1}$) velocity.

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Methods

Experimental Approach to the Problem

This study compared 2 groups (control group, repeated bout group) for the changes in indirect markers of muscle damage after fast-velocity eccentric exercise of the elbow flexors. Comparisons were also made between bouts for the repeated bout group and between the fast-exercise bout of the control group and the slow-exercise bout of the repeated bout group. With the exception of blood sampling, all criterion measures were taken from the exercised arm. Strength measures, ROM, upper-arm circumference, and ultrasound images were taken during a familiarization session, which was scheduled 5 days before exercise, immediately before and after exercise, and 24, 48, 72, and 96 hours after exercise. Blood samples and muscle soreness measures were taken immediately before exercise and 24, 48, 72, and 96 hours after exercise. The measurement order was standardized as follows: blood sample, muscle soreness, ultrasound images, ROM, upper-arm circumference, and the strength measures. The test-retest reliability was determined from the measured taken in the familiarization session and the pre-exercise measures by an intraclass correlation coefficient (ICC).

Subjects

Eighteen recreationally active men were recruited for this study from the student population. Their mean ($\pm SD$) age, body mass, and height were 26.3 ± 4.2 years; 79.8 ± 8.2 kg, and 1.8 ± 0.1 m, respectively. The subjects were randomly placed into 2 groups: the repeated bout group ($n = 10$) and control group ($n = 8$). The number of subjects was determined using the difference in the changes in maximal voluntary isometric strength between the fast- and slow-velocity exercises in our previous study (6), with an alpha level of 0.05 and a power (1-β) of 0.80. The repeated bout group performed 2 bouts of maximal voluntary eccentric exercise of the elbow flexors, a slow-velocity exercise first followed by a fast-velocity exercise with the same arm, separated by 14 days. The control group ($n = 8$) performed a single bout of the fast-velocity eccentric exercise of the elbow flexors. All subjects had no upper-body resistance training experience for at least 6 months before participating in this study and were requested not to perform any strenuous exercise in the immediate 48 hours before the exercise intervention. They were instructed not to alter their diet, consume dietary supplements, or use anti-inflammatory medications or any therapeutic treatments (e.g., massage) during the experimental period. All subjects provided written informed consent, which was approved by the institutional human research ethics committee, and the study conformed to the Declaration of Helsinki for medical research involving human subjects.

Eccentric Exercise

All subjects performed maximal isokinetic eccentric exercise using their nondominant arm on an isokinetic dynamometer (Cybex 6000 Isokinetic Dynamometer, Ronkonkoma, NY, USA) at an angular velocity of $210°\cdot s^{-1}$. The subjects in the repeated bout group performed maximal isokinetic eccentric exercise at an angular velocity of $30°\cdot s^{-1}$ 2 weeks before the $210°\cdot s^{-1}$ bout with the same arm. The velocities and the eccentric exercise protocol were based on our previous studies (5,6). Each eccentric exercise bout consisted of 35 sets of 6 maximal lengthening contractions (total 210 contractions) through a range of 120° from an elbow flexed (60°) to a fully extended (180°) position. The rest between contractions was 10 seconds, during which the elbow joint was moved passively from the extended position to the start position at $12°\cdot s^{-1}$, and the rest between sets was 90 seconds. Subjects were individually positioned on the isokinetic dynamometer with their arm supported at 45° of shoulder flexion on an arm preacher curl bench. The subjects were encouraged by the investigator throughout the exercise to resist maximally against the lever arm of the isokinetic dynamometer that forcibly extended the elbow joint, and visual feedback of torque generated during each contraction was given. Torque and work data of each contraction were displayed on a screen by the dynamometer operating system (HUMAC 6000 interface software, Computer Sports Medicine Inc, Stoughton, MA, USA), and the torque and lever arm position data were sampled by way of an 8-channel amplification rack (Powerlab, ADInstruments, Bella Vista, Australia) at 200 Hz and recorded in real time using an IBM desktop computer operating ChartPro (ADInstruments, Australia) data acquisition software. The average peak torque of each set was used for further analysis. The total work was recorded from the Cybex operating software.

Strength Measures

Maximal voluntary isometric strength of the elbow flexors was measured at 5 joint angles of 70°, 90°, 110°, 130°, and 150°. Subjects were provided with strong verbal encouragement to perform 3 maximal contractions, holding each contraction for 4 seconds, and were allowed 30 seconds of rest between each effort and 60 seconds of rest between different angles. Torque and lever arm position data were accessed from the dynamometer and sampled by way of an 8-channel amplification rack (Powerlab, ADInstruments, Bella Vista, Australia) at 200 Hz. An average peak torque from the 3 contractions performed at each joint angle was used for further analysis. The ICC (r value) for the strength measures at 5 joint angles ranged between 0.80 and 0.98.

Range of Motion

The measurements of the elbow joint angles were made using a plastic goniometer while the subject was standing with the arm initially relaxed by his side. The elbow joint angles were measured when the subject attempted to fully extend his elbow joint (extended angle) and when the subject fully flexed his elbow joint in an attempt to touch his shoulder with the palm (flexed angle). Two measurements were taken for each angle, and the ROM was determined by deducting the flexed

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angle from the extended angle using the mean value of the 2 measures, and the ICC (*r* value) for this method was 0.94. Landmarks used to measure the elbow joint angles were the lateral epicondyle of the humerus, the palpated distal end of the deltoid muscle, the mid-point of between the styloid processes of the ulna and radius, and the styloid process of the radius. These sites were marked on the skin with a semipermanent ink marker to obtain consistent measures.

Upper-Arm Circumference

Circumference of the upper arm was assessed using a constant tension tape while the arm was relaxed and hanging by the subject's side in a standing position. Measurements were taken twice from sites 3, 5, 7, 9, and 11 cm above the crease line of the elbow of the exercised arm. Each site was marked with a semipermanent ink marker to obtain consistent measures. An average of the 5 sites (using the average of the 2 measures for each site) was used for further analysis, and the ICC (*r* value) was 0.98.

B-Mode Ultrasound Images

Transverse B-mode ultrasound images were obtained using an SSD-1000 (Aloka Co., Ltd., Tokyo, Japan) with a 7.5 MHz, 6 cm linear probe from a position 9 cm above the elbow crease at the same site used for the upper-arm circumference measurement. Subjects were seated, and their arm was supported in a relaxed supinated position, palm facing upward and their forearm was supported on a padded cushion forming a joint angle of approximately 135° on a table. The same examiner placed the probe on the same marked site on the upper arm at all time points. All images were obtained using the same references, gains, and contrasts during the experimental period. Muscle thickness was quantified using the software Image J (version 0.0, National Institute of Health, Bethesda, MD, USA) by measuring the distance from the subcutaneous adipose layer to the most proximal surface of the humerus bone. A known distance (1 cm) within the image was used to calibrate within the parameters and guidelines of the software program. The ICC (*r* value) for this method was 0.77.

Serum Creatine Kinase and Lactate Dehydrogenase Activity

An 8.5 mL blood sample was drawn from the antecubital vein using a standard venipuncture technique into an SST vacutainer (Becton, Dickson and Company, Franklin Lakes, NJ, USA). The sample was allowed to clot at room temperature for 30 minutes before being centrifuged for 10 minutes at 3,000 rpm and 4°C. The serum was separated by a pipette into 0.5 mL aliquots and stored at −80°C for later analysis. Spectrophotometric analysis was conducted by a Roche-Hitachi Modular PT (Mannheim, Germany) for serum CK and LDH activities using a test kit (Roche Diagnostics, Mannheim, Germany; 12132672 for CK and 11876961 for LDH, respectively). The normal reference ranges for CK and LDH activity using this method are less than 200 $U \cdot L^{-1}$ and less than 350 $U \cdot L^{-1}$, respectively, according to the kits' manuals.

Muscle Soreness

Muscle soreness was assessed using a 100-mm visual analogue scale where the subject was instructed that 0 indicated no pain and 100 mm was an indication of "unbearable" pain. Subjects rated the soreness on the 100-mm line when the investigator palpated 4 sites of the elbow flexors (biceps brachii at 3–5 cm and 9–11 cm above the elbow crease, brachialis, and brachioradialis). The same investigator conducted the muscle soreness assessment throughout the study, and the protocol was standardized such that the same pressure was applied to the sites using the tips of 3 fingers with a uniform circular movement while the subject's arm was placed on a table. The total value of the 4 sites (maximum value of 400) was used for further analysis.

Statistical Analyses

The baseline values of each criterion measure and the total work performed in the exercise were compared among the first and second bouts of the repeated bout group and the control group by a one-way analysis of variance (ANOVA). Changes in the average torque during each set of exercise were compared by a two-way repeated measure ANOVA among the first and second bouts of the repeated bout group and the control group. A two-way repeated measure ANOVA was used to compare the changes in criterion measures between the slow eccentric exercise bout of the repeated bout group and the fast eccentric exercise of the control group, between the slow (first) and fast (second) eccentric exercise bouts of the repeated bout group, and between the fast (second) eccentric exercise bout of the repeated bout group and the fast eccentric exercise of the control group. If the ANOVA identified a significant main or interaction effect, a Tukey's HSD post hoc analysis followed. A rate of recovery of muscle strength and ROM was determined as the change between immediately and 96 hours postexercise, and the rate was compared between the first and second bouts of the repeated bout group, the second bout of the repeated bout group and control group, and the first bout of the repeated bout group and control group by Student *t*-test. The significance was set at $p < 0.05$. All data are presented as mean ± *SEM* unless otherwise stated.

Results

Baseline Measures

A significant difference in the baseline isometric strength was evident for all angles except for 70°, and the second bout of the repeated bout group showed significantly lower strength at angles of 90°, 110°, 130°, and 150° compared with control (Table 1). A significant difference was also found for ROM and CK such that ROM was significantly smaller and serum CK activity was significantly greater for the control group compared with the first bout of the repeated bout group (Table 2).

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Table 1. Baseline values (mean ± *SEM*) and ranges among subjects (min – max) for maximal isometric strength (Nm) at 70°, 90°, 110°, 130°, and 150° elbow joint angles for first and second bouts of repeated bout group and control group.

Group	70°	90°	110°	130°	150°
Repeated bout					
1st	57.0 ± 2.8 (41.2–70.0)	56.7 ± 2.7 (41.7–69.4)	51.7 ± 2.0 (40.8–57.1)	47.8 ± 3.5 (35.5–60.2)	46.8 ± 4.2 (33.0–62.6)
2nd	50.1 ± 3.2 (36.4–71.2)	49.3 ± 3.9* (33.5–68.0)	45.1 ± 3.5* (32.2–59.3)	41.2 ± 2.8* (28.2–51.7)	38.7 ± 3.4* (25.9–54.9)
Control	61.0 ± 5.1 (42.4–78.1)	63.5 ± 4.7 (44.0–80.5)	59.9 ± 4.6 (40.3–77.8)	53.9 ± 4.1 (40.9–73.2)	52.3 ± 4.4 (42.1–75.2)

*Significantly ($p < 0.05$) different from control group.

Table 2. Baseline values (mean ± *SEM*) and ranges among subjects (min – max) for range of motion (ROM), upper arm circumference (CIR), muscle thickness (MT), serum creatine kinase (CK) , and lactate dehydrogenase (LDH) activities for first and second bouts of repeated bout and control group.

Group	ROM (°)	CIR (mm)	MT (mm)	CK ($IU \cdot L^{-1}$)	LDH ($IU \cdot L^{-1}$)
Repeated bout					
1st	134 ± 2.1*(120 – 145)	281.1 ± 9.1 (234–321)	25.9 ± 0.1 (19–33)	150.2 ± 26.2*(37–320)	292.8 ± 21.1 (139–346)
2nd	133 ± 1.9*(119–139)	281.0 ± 8.8 (234–317)	25.7 ± 0.1 (18–31)	179.3 ± 28.1 (52–371)	300.1 ± 19.5 (149–349)
Control	125 ± 2.1 (115–132)	284.5 ± 4.7 (269–314)	25.0 ± 0.1 (19–32)	348.0 ± 73.5 (167–893)	317.1 ± 10.6 (282–364)

*Significantly ($p < 0.05$) different from control group.

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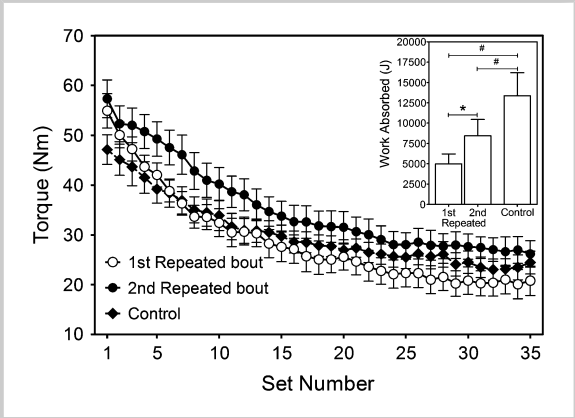

Figure 1. Changes in average peak torque generated in each set during exercise for first (slow) and second (fast) bouts of repeated bout group and control group. Inset shows total work performed during exercise for first (slow) and second (fast) bouts of repeated bout group and control group. Mean (±*SEM*) values shown. *Significantly different between bouts of repeated bout group; #significantly different from control group.

Torque and Work During Exercise

All subjects completed the exercise as instructed. As shown in Figure 1, a significant decrease in average peak torque from the first to the last set was observed for all exercise bouts. Changes in torque were not significantly different between the 2 bouts of the repeated bout group and between the first (slow) or the second (fast) bout of the repeated group and control group. The total work performed during the exercise (Figure 1 inset) was significantly greater for the control group compared with both exercise bouts of the repeated bout group. In the repeated bout group, the total work performed was significantly greater for the second bout (fast-velocity exercise) compared with the first bout (slow-velocity exercise).

Comparison Between Slow-Velocity Eccentric Exercise of Repeated Bout Group and Fast Eccentric Exercise of Control Group

In regard to the recovery of isometric strength, the fast-velocity eccentric exercise had significantly slower recovery rate than the slow-velocity exercise for all angles (Figure 2). As shown in Figure 3, recovery of ROM was also significantly slower for the fast-velocity than the slow-velocity exercise. The increases in upper-arm circumference, muscle thickness, and serum CK activity were significantly greater for the fast-velocity exercise compared with the slow-velocity exercise. No significant difference between the exercises was evident for the changes in muscle soreness (Figure 3D) and plasma LDH activity (Figure 3F).

Comparison Between Slow and Fast Eccentric Exercise Bouts for Repeated Bout Group

Changes in isometric strength were significantly different between the first and second bouts for 110°, 130°, and 150° but not for 70° and 90°. No significant differences in the rate of recovery were evident between bouts for all angles (Figure 2). A significant difference was also found for the changes in ROM, and the recovery of ROM was significantly faster for the second than the first exercise bout (Figure 3A). No significant differences between bouts were evident for changes in upper-arm circumference (Figure 3B), muscle thickness (Figure 3C), muscle soreness (Figure 3D), and serum CK (Figure 3E) and LDH (Figure 3F) activities.

Comparison Between Groups for Responses to Fast-Velocity Eccentric Exercise

Changes in isometric strength were significantly different between groups for all angles (Figure 2). The magnitude of the decrease in maximal isometric strength immediately after fast eccentric exercise was significantly different between groups for 90°, 130°, and 150°. The rate of recovery of

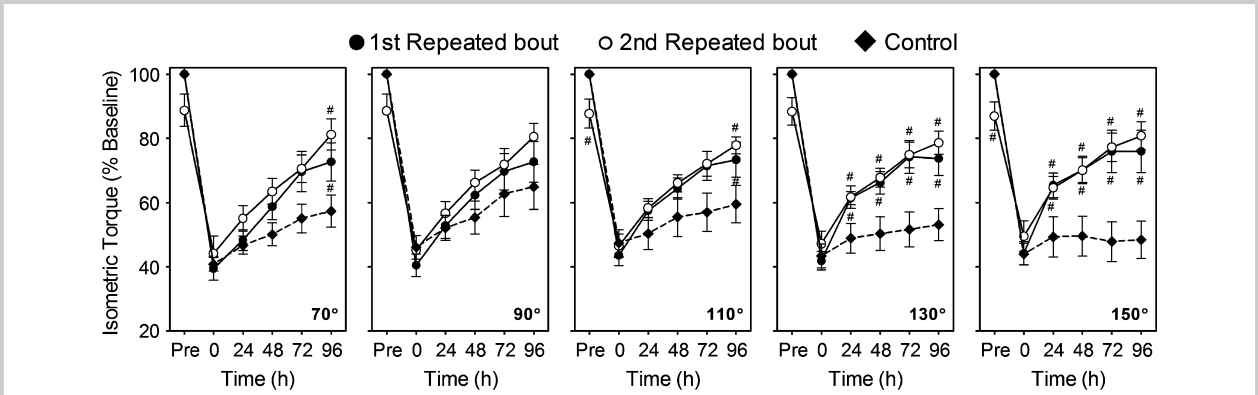

Figure 2. Percent changes in maximal voluntary isometric torque at 70°, 90°, 110°, 130°, and 150° elbow joint angles from baseline (pre: 100%) immediately (0), 24, 48, 72, and 96 hours after exercise for first and second bouts of repeated bout group and for control group. For repeated bout group, pre-exercise value of second bout is expressed as percentage of torque before first bout. Mean (±*SEM*) values shown. #Significantly different from control group.

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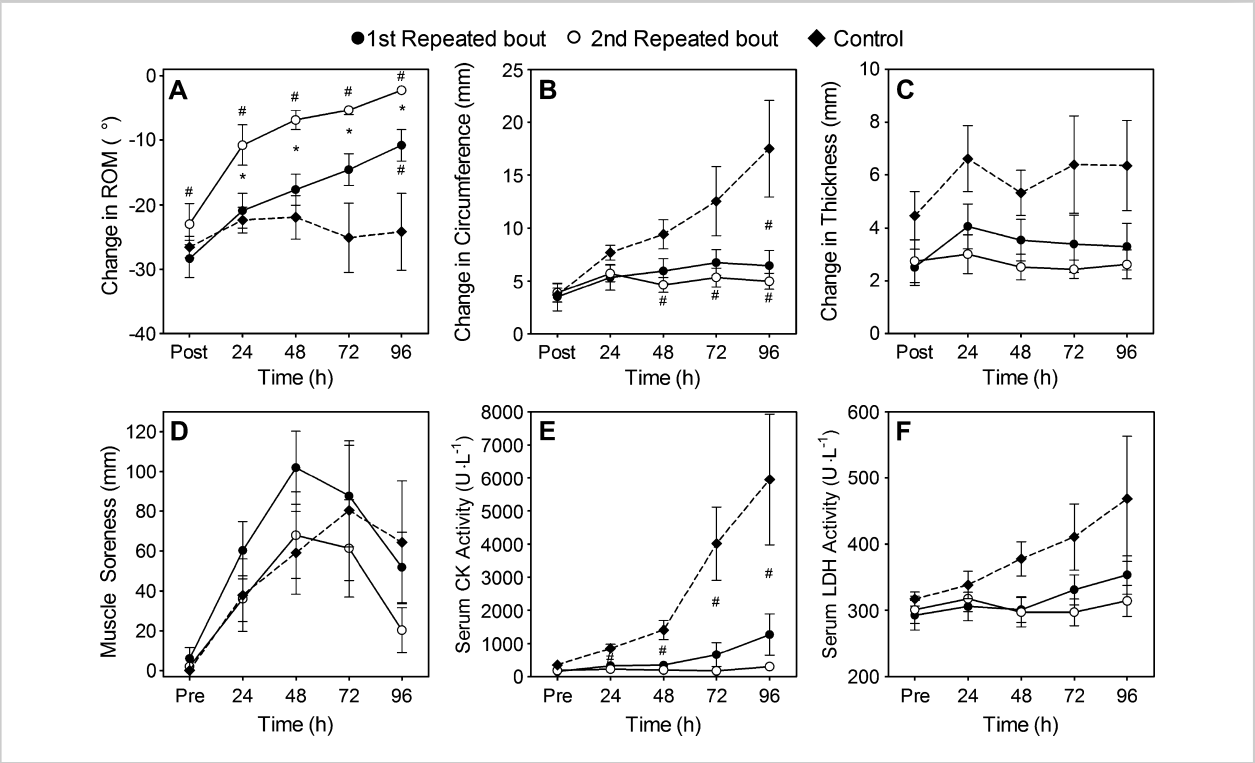

Figure 3. Absolute changes in range of motion (A), upper arm circumference (B), and upper arm muscle thickness (C) from baseline at immediately (post), and 24, 48, 72, and 96 hours after exercise and muscle soreness (D), serum creatine kinase (E) and lactate dehydrogenase (F) activities before (pre), and 24, 48, 72, and 96 hours after exercise for first and second bouts of repeated bout group and for control group. Mean (±*SEM*) values shown. *Significantly different between bouts of repeated bout group; #significantly different from control group.

strength was significantly faster for the repeated group compared with the control group for all angles. As shown in Figure 3A, changes in ROM were significantly smaller for the repeated bout group than the control group. Changes in upper-arm circumference were also significantly smaller for the repeated bout group than the control group (Figure 3B). Changes in serum CK (Figure 3E) activity were significantly greater for the control group compared with the repeated bout group. No significant difference between groups was found for changes in muscle thickness (Figure 3C), muscle soreness (Figure 3D), and serum LDH activity (Figure 3F).

Discussion

The novelty of this study was that a different velocity of eccentric exercise was used for the first and second bout to test the null hypothesis that muscle damage induced by a fast-velocity eccentric exercise would not be prevented by a slow-velocity eccentric exercise. The main findings of this study were a) the comparison between the slow-velocity eccentric exercise of the repeated bout group and fast-velocity eccentric exercise of the control group demonstrated that muscle damage was less and recovery of muscle function was faster for the slow- than fast-velocity eccentric exercise, b) the repeated bout group showed significantly faster recovery of isometric strength and ROM after the second bout (fast-velocity eccentric exercise) compared with the first bout (slow-velocity eccentric exercise), and c) the comparison between groups showed that recovery of muscle strength and ROM was faster, and increases in upper-arm circumference and serum CK activity were smaller, when the high-velocity eccentric exercise was preceded by the slow-velocity eccentric exercise bout. These findings did not support the null hypothesis.

The present study provides further support to the findings of our previous studies (5,6) showing that changes in indirect markers of muscle damage were significantly greater after a fast-velocity eccentric exercise than a slow-velocity eccentric exercise. The previous studies used an arm-to-arm comparison model in which one arm of the same subject performed the fast-velocity eccentric exercise and the other arm performed the slow-velocity eccentric exercise. However, the present study used 2 groups of subjects to compare the 2 different velocity exercises but still provided similar results to those reported in the arm-to-arm studies. The reason why fast-velocity lengthening contractions induce greater muscle damage than slow-velocity lengthening contractions has not been confirmed as yet, but it is possible that fast-velocity lengthening contractions place greater

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mechanical stress onto contractile elements because fewer strongly bound cross-bridges are formed during fast-velocity contractions (2).

It was hypothesized that the slow-velocity eccentric exercise bout would not reduce muscle damage induced by the fast-velocity eccentric exercise because the population of muscle fibers affected by eccentric exercise might be velocity dependent. Shepstone et al. (33) showed a greater increase in the area of type II muscle fibers after 8 weeks of fast-velocity ($210°\cdot s^{-1}$) than slow-velocity ($20°\cdot s^{-1}$) eccentric training. They speculated that the hypertrophy of type II muscle fibers was associated with greater damage to these fibers because it was found that an acute bout of the fast-velocity eccentric exercise induced greater Z-line streaming compared with the slow-velocity bout. It has been shown that type II muscle fibers are more susceptible than type I fibers to muscle damage induced by lengthening contractions (14,15,19). It is possible that more type II muscle fibers were recruited during fast-velocity than slow-velocity eccentric exercise. Beltmen et al. (1) reported that 79% of muscle fibers are recruited during slow- ($60°\cdot s^{-1}$) velocity lengthening contractions of the knee extensor, which was significantly lower than shortening (93%) or isometric (92%) contractions. It is unknown whether this recruitment level is the same for the elbow flexors and whether the contraction velocity affects the proportions of muscle fibers contributing to the torque generated. However, considering the high percentage (~80%) of muscle fibers recruited in the slow-velocity lengthening contractions (1) and the small difference between type I and type II muscle fiber distribution percentages in the elbow flexors (32), it is unlikely that a large difference exists between the slow- and fast-velocity eccentric exercises for the number of muscle fibers recruited or the proportion of type I and II muscle fibers contributing to force generation. Thus, the assumption that the population of muscle fibers recruited by eccentric exercise is velocity dependent does not appear justified.

It should be noted that the work performed during exercise was significantly different between groups and between bouts (Figure 1 inset). The greater work of the control group appears to be associated with greater pre-exercise strength (Table 1). Because no significant difference between the first bout of the repeated bout group and the control group existed for the maximal isometric strength, it appears likely that the difference between the groups before the fast-velocity eccentric exercise bout was caused by the delayed recovery from the initial exercise bout. Thus, it is possible that the reduced muscle damage after the first exercise bout for the repeated bout group was caused by less stress induced by the exercise. We have previously reported a lack of association between the total work performed during eccentric exercise and the magnitude of change in indirect markers of muscle injury (7). Therefore, it appears unlikely that the lower work performed by the repeated bout group was the main reason for the reduced muscle damage, and the lack of a difference in the average peak torque produced per exercise set does not appear to support the notion that less stress was induced by the second exercise bout. In fact, the changes in muscle strength and ROM from pre- to immediately postexercise were similar between the groups (Figures 2 and 3A). This indicates that the mechanical stress to the muscle was similar between the 2 fast-velocity eccentric exercises.

The findings of the present study appear to be in line with the previous studies (3,8–10,18,26) showing that an initial bout of eccentric exercise resulting in less damage can still reduce muscle damage in a more demanding eccentric exercise performed later. Recent studies (9,18) have demonstrated that even very low-intensity eccentric exercise, which results in little or no changes in indirect markers of muscle damage, can still reduce muscle damage in a subsequent bout of more damaging eccentric exercise. It appears that any form of eccentric exercise can confer some protection for subsequent bouts of eccentric exercise; however, the magnitude of protection is affected by the magnitude of muscle damage induced by the initial exercise bout.

It is speculated that the muscle fibers not affected by the slow-velocity eccentric exercise were damaged in the subsequent bout of the fast-velocity eccentric exercise, but the muscle fibers that were affected by the slow-velocity eccentric exercise were not damaged in the fast-velocity eccentric exercise. Nosaka et al. (29) reported that eccentric exercise of the elbow flexors performed at short muscle lengths reduced muscle damage induced by the eccentric exercise at long muscle lengths, and the magnitude of the attenuated responses of indirect muscle damage markers after the short muscle length eccentric exercise was approximately half of that observed after a prior bout of long muscle length eccentric exercise. This is similar to the magnitude of the effect produced by the slow-velocity eccentric exercise against the fast-velocity eccentric exercise shown in the present study. It is important to note that the slow-velocity eccentric exercise did not reduce the magnitude of changes in the markers of muscle damage after the fast-velocity eccentric exercise in the same magnitude as has been demonstrated in previous studies (10,21) in which the same exercise was performed for both bouts. It appears that the magnitude of the repeated bout effect conferred by the slow-velocity eccentric exercise against the fast-velocity eccentric exercise would not have been as strong as that conferred by fast-velocity eccentric exercise. However, this should be confirmed in future studies because the present study did not include a group that performed 2 bouts of the fast-velocity eccentric exercise.

The present study demonstrates that the magnitude of strength loss immediately after exercise was similar among the angles (Figure 2), but the rate of recovery was significantly more protracted for a long (e.g., 150°) than a short (e.g., 70°) muscle length. Previous studies (22,29) also showed that the strength loss immediately after eccentric exercise

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was not attenuated in the second bout, although the recovery was enhanced. It has been shown that inhibition at the motor cortex or the spinal level limits voluntary drive to the muscle in the first 24 hours after eccentric exercise (31). It is also documented that the strength loss after eccentric exercise, especially within a 5-day period after exercise, is primarily caused by the excitation-contraction coupling failure in animal studies (34,35). It appears possible that central or peripheral fatigue is associated with the decrease in strength immediately after exercise, and this is not attenuated by the initial eccentric exercise. Muscle soreness is one of the symptoms of muscle damage but does not necessarily reflect the magnitude of muscle damage (27). The results of the present study showed that, despite the significant effects of the slow-velocity eccentric exercise on muscle strength, ROM, upper-arm circumference, muscle thickness, and CK activity, no significant effect on muscle soreness was evident (Figures 1 and 2). This would suggest that damage to muscle fibers is disassociated from the mechanisms causing muscle soreness. It may be that the muscle soreness resulting from eccentric exercise is associated with an inflammatory response originating in connective tissue damage rather than muscle fiber damage (12). If this is the case, it appears possible that the connective tissue damage-inflammation was similar regardless of the eccentric exercise velocity and the protective effect conferred by the slow velocity eccentric exercise.

There is a wealth of literature demonstrating the repeated bout effect showing that the changes in indirect markers of muscle damage are significantly smaller and the recovery of muscle function is significantly faster after the second bout compared with the first bout. The present study has demonstrated that voluntary eccentric exercise of the elbow flexors performed with maximal intensity at a slow lengthening velocity reduces muscle damage in the subsequent bout of voluntary eccentric exercise performed with maximal intensity at a fast lengthening velocity. It appears likely that this is also the case for other muscles such as the knee extensors; however, this should be investigated in future studies. It is concluded that if muscle damage needs to be minimized, slow-velocity eccentric exercise should be performed before fast-velocity eccentric exercise.

Practical Applications

The findings of the present study should be placed in context for the sports injury rehabilitation specialists and the strength and conditioning professionals. It is known that the inclusion of lengthening contractions in resistance training produces greater strength gains (11) and greater type II fiber hypertrophy (16,17) than concentric training. It has been shown that fast-velocity lengthening contractions are more effective in inducing a hypertrophic response in type II fibers than slow-velocity lengthening contractions (13,33). However, it should be noted that fast-velocity lengthening contractions can induce greater muscle damage than slow-velocity lengthening contractions, as shown in the present study and previous studies (6). Thus, caution is required when introducing fast-velocity lengthening contractions into a resistance training program. It appears possible to prevent severe muscle damage by preconditioning muscles using submaximal eccentric exercise (9,25). In addition to this strategy, the present study shows that performing a slow-velocity eccentric exercise bout can be used as a precursor to fast-velocity eccentric exercise when it is necessary to avoid potential severe muscle damage induced by the fast-velocity eccentric exercise. Furthermore, the outcomes of this research support the process of periodization and progressive overload that are required to achieve a positive adaptation response. For example, when a practitioner is programming for maximal eccentric exercise at a sport-specific movement velocity, it is recommended to use a progression that is from slow- to fast-velocity lengthening contractions at a maximal intensity. It is proposed that this would achieve greater athlete compliance because of a reduction in the debilitating muscle damage while still achieving the programmed volume of work required.

Acknowledgments

This research was supported in part by the Gatorade Sports Science Institute. Graduate research grants awarded to D. W. Chapman.

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