Intensity of eccentric exercise, shift of optimum angle, and the magnitude of repeated-bout effect

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Chen TC, Nosaka K, Sacco P. Intensity of eccentric exercise, shift of optimum angle, and the magnitude of repeated-bout effect. J Appl Physiol 102: 992–999, 2007. First published November 30, 2006; doi:10.1152/japplphysiol.00425.2006.—This study compared the effect of four different intensities of initial eccentric exercise (ECC1) on optimum angle shift and extent of muscle damage induced by subsequent maximal eccentric exercise. Fifty-two male students were placed into 100%, 80%, 60%, or 40% groups (n = 13 per group), performing 30 eccentric actions of the elbow flexors of 100%, 80%, 60%, or 40% of maximal isometric strength [maximal voluntary contraction (MVC)] for ECC1, followed 2–3 wk later by a similar exercise (ECC2) that used 100% MVC load. MVC at six elbow joint angles, range of motion, upper arm circumference, serum creatine kinase activity, myoglobin concentration, and muscle soreness were measured before and for 5 days following ECC1 and ECC2. A rightward shift of optimum angle following ECC1 was significantly (P < 0.05) greater for the 100% and 80% than for the 60% and 40% groups, and it decreased significantly (P < 0.05) from immediately to 5 days postexercise. By the time ECC2 was performed, only the 100% group kept a significant shift (4°). Changes in most of the criterion measures following ECC1 were significantly greater for the 100% and 80% groups compared with the 60% and 40% groups. Changes in the criterion measures following ECC2 were significantly (P < 0.05) greater for the 40% group compared with other groups. Although the magnitude of repeated bout effect following ECC2 was significantly (P < 0.05) smaller for the 40% and 60% groups, all groups showed significantly (P < 0.05) reduced changes in criterion measures following ECC2 compared with the ECC1 100% bout. We conclude that the repeated-bout effect was not dependent on the shift of optimum angle.

Recently, a shift in the angle-force curve toward longer muscle lengths has been shown following eccentric exercise of the knee flexors (24), knee extensors (1, 15), and elbow flexors (23). The resulting rightward shift in the optimum joint angle for voluntary strength (such that higher muscle torque output occurs at a more lengthened muscle position) has been proposed as a simple and reliable indicator of muscle damage (24). This effect was observed immediately after eccentric exercise and appears to result from disrupted sarcomeres increasing the muscle’s series compliance, but a sustained shift is likely to indicate an increased number of sarcomeres in series (24). It has been documented that the rightward shift in the optimum joint angle plays a role in the repeated-bout effect (1, 2, 12). However, no relationships have been identified between alterations in the muscle length-tension relationship and magnitude of muscle damage or level of protection afforded by an initial exercise bout.

Nosaka et al. (22) reported that two maximal eccentric muscle actions of the elbow flexors, which resulted in minor changes in indirect markers of muscle damage, attenuated muscle damage induced by 24 maximal eccentric muscle actions. However, the protective effect conferred by two maximal eccentric actions was not as great as that seen following 24 maximal eccentric actions. From a practical perspective, the use of submaximal eccentric loads rather than high-intensity eccentric training in the early stages of resistance programming could be beneficial for curtailing the negative consequences of exercise-induced muscle damage. Although Nosaka and Newton (19) found that a period of submaximal eccentric training of the elbow extensors did not confer a strong protective effect against a subsequent maximal eccentric exercise bout, no study has systematically investigated whether submaximal eccentric exercise confers such a protective effect.

Therefore, the present study was designed to investigate the effect of initial eccentric exercise intensity (40, 60, 80, and 100%) on the joint angle-strength relationship and, second, to determine the extent of the protective effect conferred against maximal eccentric exercise (100%) performed after recovery from the initial bout. We hypothesized that any changes in optimal elbow joint angle for maximum voluntary isometric strength [maximal voluntary contraction (MVC)] would correspond to the intensity of the initial exercise bout. Furthermore, we postulated that the extent of protection against a maximal eccentric exercise bout would vary according to the intensity of the initial intervention, as indicated by changes muscle damage; maximal isometric strength; length-tension relationship; delayed-onset muscle soreness; elbow flexors

A BOUT OF ECCENTRIC EXERCISE confers protection against skeletal muscle damage following a subsequent bout of the same or similar exercise performed several weeks to several months later (7, 16, 21). The protective effect is characterized by a faster recovery of muscle strength and range of motion (ROM), reduced increases in muscle proteins in the blood, and less significant development of swelling and muscle soreness (7, 16). The underlying mechanisms of this protective effect, often referred to as the “repeated-bout effect,” are not fully understood; however, a reduction in the number of stress-susceptible fibers, remodeling of existing myofibrils, changes in connective tissue, and neural adaptation have been proposed to explain this phenomenon (13, 16, 24).

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strength, ROM, upper arm circumference (Cir), muscle proteins in the blood, and muscle soreness.

METHODS

Subjects

Fifty-two male students who were physically active but had not performed regular resistance training gave informed consent to participate after the study was approved by the Institutional Ethics Committee, in conformity with the Declaration of Helsinki. Subjects mean (± SD) age, height, and body weight were 20.7 ± 1.9 yr, 173.8 ± 5.0 cm, and 67.6 ± 7.2 kg, respectively. Sample size was estimated using the data from a previous study in which a similar dumbbell eccentric exercise was performed by 41 men (6). Based on the effect size of 1, alpha level of 0.05, and a power of 0.80, with an expected difference of 10% for the MVC at 5 days postexercise between two groups, it was shown that 12 subjects per group were necessary. Subjects were placed into one of four groups (n = 13 per group) based on baseline maximal voluntary isometric strength (MVC) of the elbow flexors at an elbow joint angle of 90° (1.57 rad). Groups (designated 100%, 80%, 60%, and 40%) had similar mean MVCs, with no significant differences in age, height, or body mass evident across groups. All subjects were asked to refrain from unaccustomed exercise or vigorous physical activity and to not take any anti-inflammatory drugs or nutritional supplements during the experimental period.

Eccentric Exercise

Subjects performed the first eccentric exercise of the elbow flexors (ECC1) with the nondominant arm using a dumbbell adjusted to 100%, 80%, 60%, and 40% of each individual’s MVC at an elbow angle of 90° (1.57 rad). Subjects were instructed to lower the dumbbell from an elbow flexed (50°, 0.87 rad) to an elbow extended position (170°, 2.97 rad) in 4–5 s, keeping the velocity as constant as possible by following the examiner’s counting “0” for the beginning and “1, 2, 3, 4, and 5” for the movement. After each eccentric action, the examiner removed the load, and the arm was returned to the start position. Because the elbow flexors can generate 10–15% greater torque eccentrically than isometrically (5), subjects were able to lower the “100%” weight in a controlled fashion. The movement was repeated every 45 s for 30 repetitions, and this long interval between repetitions minimized the effect of fatigue. Subjects were verbally guided to lower the dumbbell at a consistent velocity (~30°/s: 0.52 rad/s) for the whole range of motion.

All subjects performed the second bout of eccentric exercise (ECC2) with the same arm using a dumbbell equivalent to 100% MVC 2–3 wk after ECC1, when MVC returned to the pre-ECC level (2 wk for most of subjects, 3 wk for some subjects who showed a slower recovery of MVC). The protocol for ECC2 was the identical to that of ECC1, except that the dumbbell weight for the 40%, 60%, and 80% groups, whereas the same load was used in both trials for the 100% group.

Criterion Measures

MVCs at six different elbow joint angles, active ROM about the elbow joint, and Cir before, immediately after, and over 5 consecutive days following ECC1 and ECC2 were measured. Serum creatine kinase (CK) activity, myoglobin (Mb) concentration, and muscle soreness were assessed before and for 5 consecutive days after both exercise bouts. The test-retest reliability of the criterion measures was established before the data collection of this study by an intraclass correlation coefficient (R) using measurements taken 1 wk apart. R value for MVC at 6 different angles, ROM, Cir, CK, and Mb was 0.77–0.97 (50°: 0.86, 70°: 0.77, 90°: 0.97, 110°: 0.92, 140°: 0.89, 160°: 0.82), 0.90, 0.99, 0.81, and 0.83, respectively.

MVC. Elbow flexor MVCs were measured at six different elbow joint angles (50°: 0.87 rad, 70°: 1.22 rad, 90°: 1.57 rad, 110°: 1.92 rad, 140°: 2.44 rad, and 160°: 2.79 rad; where full elbow extension angle equated to 180°: 3.14 rad) in random order using a modified arm curl machine that included a force gauge (model DFG51, Omega Engineering, Stamford, CT) connected to a digital recorder (Biopac Systems, Goleta, CA). The force gauge was placed between a pole of the machine and a metal cord that was connected to a wristband worn by the subject. Subjects were seated on a chair of the arm curl bench, which ensured their trunk position, and the upper arm was supported on an armrest so that the shoulder and elbow were positioned at 45° (0.79 rad) flexion with 0° abduction. Elbow angles were determined using a goniometer (Creative Health Products, Plymouth, MI), and the six different angles were set carefully. Subjects maintained each MVC for ~3 s, with 45-s rests between attempts (3 for each angle), and 2-min rests between angles. Strong verbal encouragement was provided during MVC measurements. The greatest 1-s average of each contraction was measured, and the average of the three trials for each angle was used for subsequent analysis. The voluntary elbow flexor forces were not corrected for the effect of gravity.

The optimum angle for maximal voluntary torque was calculated from the MVC values obtained at six elbow angles. This method was modified from that described by Philippou et al. (23), and it included an additional angle of 110°, because the optimum angle was expected to lie between 90° and 140°. The optimal angle was calculated using the fitted quadratic polynomial equation for the six MVCs of each subject for each time point: Force = a + bα + cα2 (where A refers to elbow joint angle, and a, b, and c are the fitted polynomial parameters), and MVC at the optimum angle was also obtained.

Elbow joint angles and ROM. Flexed elbow joint angle (FANG) was determined when subjects were asked to fully flex the elbow joint by touching their palm to shoulder without raising the elbow. Relaxed elbow joint angle (RANG) represented the angle with the arm relaxed along the side of the body during standing. A semipermanent marker was used to identify the lateral middle point of the humerus, the lateral axis point of elbow joint, and the lateral middle point between radius and ulna for the goniometer placements. FANG and RANG were measured three times for each time point using the goniometer, and ROM was defined as the angle subtracting the mean FANG from the mean RANG.

Cir. Cir was measured at 8 cm above the elbow joint with a Gulick tape measure while allowing the arm to hang down by the side of the body. The measurement point was marked on the subject’s arm to ensure consistent placement of the tape measure. The mean values of three measurements were used for the analysis.

Blood sampling, CK, and Mb. A 10-ml sample of venous blood was collected by venipuncture from the cubital fossa region of the dominant arm into a serum separation tube. The blood was allowed to clot for 30 min at room temperature and centrifuged for 10 min to obtain serum. After separation, all serum samples were stored at −20°C until analysis for CK activity and Mb concentration. Serum CK activity was determined spectrophotometrically by a Genstar chemistry analyzer (Electro-Nucleonics, Fairfield, NJ) using a test kit (Sigma Diagnostics, St. Louis, MO). Serum Mb concentration was assayed by a biochemical analyzer (model ADVIA-Centaur, Bayer, Leverkusen, Germany) using a test kit (Denka-Seiken, Tokyo, Japan). Samples were analyzed in duplicate, the means of which were used for subsequent statistical analysis. The normal reference ranges for CK and Mb are 38–174 IU/l, and <110 µg/l, respectively, based on the manufacturer’s information.

Muscle soreness. Muscle soreness was evaluated using a visual analog scale consisting of a 100-mm continuous line representing “no pain” at one end (0 mm) and “very, very painful” at the other (100 mm). Subjects were asked to indicate the soreness level on the line when an investigator extended the elbow joint.
Statistical Analyses

Changes in the criterion measures over time were compared between groups by an ANOVA with repeated measures. Initially, the four groups (100%, 80%, 60%, and 40%) were compared for changes over time following the first and second bouts by a three-way ANOVA. A significant \( P < 0.05 \) group \( \times \) time \( \times \) bout interaction was found for all criterion measures. A two-way ANOVA was also conducted to compare the four groups for changes following the first and second bout separately. This also resulted in a significant group \( \times \) time interaction effect for all measures. Therefore, several multiple pairwise two-way ANOVAs were performed to examine 1) how the changes in criterion measures after ECC1 or ECC2 were different between groups (100% vs. 80%, 100% vs. 60%, 100% vs. 40%, 80% vs. 60%, 80% vs. 40%, and 60% vs. 40%), and 2) how the changes in criterion measures were different between ECC1 and ECC2 for each group. When a significant interaction (group \( \times \) time, bout \( \times \) time) effect was evident, a Scheffe’s post hoc test was conducted to specify the time points.

It was assumed that changes in the criterion measures following ECC1 for the 100% group indicated a normal response. Therefore, changes in all criterion measures following ECC1 for the 100% group and following ECC2 for other groups were compared using a two-way repeated-measures ANOVA (100% ECC1 vs. ECC2 of the 40% group, 100% ECC1 vs. ECC2 of the 60% group, 100% ECC1 vs. ECC2 of the 80% group, 100% ECC1 vs. ECC2 of the 100% group). This allowed us to determine whether the initial exercise bout conferred a protective effect. Statistical significance was set at \( P < 0.05 \). Data are presented as means \( \pm SE \), unless otherwise stated.

RESULTS

All subjects were able to complete the exercise in the expected manner for both ECC1 and ECC2, although some subjects in the 100% and 80% groups had difficulty lowering the dumbbell at a constant velocity for the last few contractions.

Comparison Between Groups for Changes in Criterion Measures After ECC1 and ECC2

MVC and optimum angle. Each group had similar force-angle characteristics of the elbow flexors before performing eccentric exercise, with strength significantly \( P < 0.01 \) reduced at 50 and 70° of elbow extension compared with other angles. No significant differences existed between groups for preexercise MVC at any angles, and the baseline force-angle curves before ECC2 were similar to those before ECC1 for all groups.

Figure 1 shows changes in optimum angle for voluntary isometric strength following two eccentric exercise bouts. The preexercise optimum angle (\( \sim 117° \)) was not significantly \( (P = 0.78) \) different among the groups. All groups showed significant \( (P < 0.01) \) changes in optimum angle following exercise, with the greatest shift seen 1–2 days postexercise, remaining increased for a further 5 days after ECC1. The magnitude of the shift in optimum angle was significantly \( (P < 0.01) \) larger for the 100% and 80% groups compared with the 60% and 40% groups, but no significant differences were evident between the 100% and 80% groups \( (P = 0.12) \) or between the 60% and 40% groups \( (P = 0.14) \). The optimum angle immediately before ECC2 remained significantly \( (P = 0.02) \) larger (by some 4°, Fig. 1) than the pre-ECC1 value for the 100% group only (ECC1: 116.8 \( \pm 3.1° \), ECC2: 120.8 \( \pm 3.4° \)). Significant \( (P < 0.01) \) increases in optimum angle following ECC2 was observed for all groups, but these were significantly \( (P < 0.01) \) greater for the 40% and 60% groups compared with the 100% group, and they were significantly \( (P < 0.01) \) greater for the 40% group than the 80% group. No significant differences existed between the 100% and 80% \( (P = 0.15) \), 80% and 60% \( (P = 0.22) \), and 60% and 40% \( (P = 0.90) \) groups. At 5 days post-ECC2, no significant \( (P = 0.89) \) differences in optimum angle were evident between groups.

Joint angle was a significant factor in MVC loss following exercise, with strength losses in the 160° position being significantly \( (P < 0.01) \) smaller than other angles. No significant \( (P = 0.38) \) differences in the magnitude of strength losses were evident for the MVC at 70, 90, and 110°. The elbow joint angle to show the highest MVC changed over time, but the highest MVC was recorded at either 90, 110, or 140° for all time points. In Fig. 2 the normalized changes in MVC at the optimum angle are illustrated. All groups showed significant \((P < 0.01)\) decreases in MVC compared with baseline following ECC1, but significant differences were found between the 100% and 60% \((P = 0.01)\), 100% and 40% \((P < 0.01)\), 80% and 60% \((P < 0.01)\), and 80% and 40% \((P < 0.01)\) groups. No significant differences were evident for 100% and 80% \((P = 0.10)\). MVC remained below baseline at 5 days after ECC1 for all groups, but it had returned to baseline before ECC2, with no significant \((P = 0.79)\) differences between groups for the pre-ECC2 values. All groups showed significant \((P < 0.01)\) changes in MVC following ECC2, but significant interaction effects were found between the 100% and other groups \((P < 0.01)\), the 80% and 60% or 40% groups \((P < 0.02)\), and the 60% and 40% groups \((P < 0.01)\). Changes in MVC at each angle were similar to those shown in Fig. 2.

ROM. ROMs were similar for all groups before ECC1, averaging 126 \( \pm 1.5° \). ROM decreased significantly \((P < 0.01)\) from preexercise values (Fig. 3), and it had not recovered by 5 days postexercise for any of the groups. Decreases in ROM were significantly smaller for the 40% group compared with the 80% \((P < 0.01)\) and 100% \((P < 0.01)\) groups, with a significant difference also present between the 60% and 100%
groups ($P < 0.01$). No significant differences between the 40% and 60% groups ($P = 0.91$), the 60% and 80% groups ($P = 0.98$), and the 80% and 100% groups ($P = 0.34$) were evident. ROM before ECC2 was similar to that before ECC1, and it did not differ significantly ($P = 0.79$) between groups. Significant ($P < 0.01$) changes in ROM following ECC2 were evident for all groups; however, decreases in ROM were significantly larger for the 40% group compared with the 100% ($P < 0.01$) and 80% groups ($P < 0.01$). No significant differences were found between 40% and 60% ($P = 0.77$), 60% and 80% ($P = 0.42$), and 80% and 100% ($P = 0.26$).

**Cir.** No significant ($P = 0.98$) difference in Cir was observed before ECC1 among groups, and the average Cir before ECC1 was $267.6 \pm 3.4$ mm. Changes in Cir from the pre-exercise value are shown in Fig. 4. Cir increased significantly ($P < 0.01$) following ECC1 for all groups, and peaked 4–5 days postexercise. The 40% group showed significantly smaller increases compared with 80% ($P = 0.02$) and 100% ($P < 0.01$) groups, but no significant differences were found between the 40% and 60% groups ($P = 0.64$), the 60% and 80% groups ($P = 0.55$), and the 80% and 100% ($P = 0.26$).

The pre-ECC2 Cir was similar to that before ECC1, and no significant ($P = 0.92$) differences between groups were observed. All groups showed significant ($P < 0.01$) increases in Cir following ECC2; however, the increases were significantly larger for the 40% ($P < 0.01$) and 60% ($P = 0.03$) groups compared with the 100% group. No significant differences were found between the other groups following ECC2.

**CK and Mb.** Baseline values of CK activity ($P = 0.54$) and Mb concentration ($P = 0.71$) were similar across groups. All groups showed significant increases in CK ($P < 0.01$) and Mb concentration ($P < 0.01$), but a significant ($P < 0.01$) interaction effect was found for the changes over time among groups (Fig. 5). Increases in serum CK activities and Mb concentrations were significantly smaller for the 40% group than 80% ($P < 0.01$) and 100% ($P < 0.01$) groups, but no significant differences were evident between 40% and 60% ($P = 0.06$ for CK, $P = 0.06$ for Mb).

Fig. 2. Changes in MVC corresponding to the optimum angle from preexercise value (100%) before (pre), immediately after (post), and 1–5 days following ECC1 and ECC2 for the 100%, 80%, 60%, and 40% groups. Pairs of groups showing a significant ($P < 0.05$) interaction (group × time) effect are indicated.

Fig. 3. Changes in range of motion (ROM) from preexercise value (0) immediately after (post) and 1–5 days following ECC1 and ECC2 for the 100%, 80%, 60%, and 40% groups. Pairs of groups showing a significant ($P < 0.05$) interaction (group × time) effect are indicated.

Fig. 4. Changes in upper arm circumference (Cir) from preexercise (pre) value (0) immediately after (post) and 1–5 days following ECC1 and ECC2 for the 100%, 80%, 60%, and 40% groups. Pairs of groups showing a significant ($P < 0.05$) interaction (group × time) effect are indicated.

Fig. 5. Changes in serum creatine kinase (CK) activity (A) and myoglobin (Mb) concentration (B) before (pre), and 1–5 days following ECC1 and ECC2 for the 100%, 80%, 60%, and 40% groups. Pairs of groups showing a significant ($P < 0.05$) interaction (group × time) effect are indicated.
0.08 for Mb) groups. Also, no significant differences in pre-ECC2 were seen for the baseline CK (P = 0.63) and Mb (P = 0.79) among groups. Significant increases in CK and Mb were observed following ECC2 for the 40% and 60% groups (P < 0.01) but not for the 80% and 100% groups (P = 0.11). No significant difference existed between the 40% and 60% groups (P = 0.16 for CK, P = 0.19 for Mb, respectively).

Muscle soreness. As shown in Fig. 6, no muscle soreness existed before ECC1, but it was evident (P < 0.01) by 1 day and peaked 1–2 days postexercise. The 40% group showed significantly lower soreness values compared with the 80% (P < 0.01) and 100% (P < 0.01) groups, with no significant differences observed between the 40% and 60% groups (P = 0.71), the 60% and 80% groups (P = 0.11), or the 80% and 100% groups (P = 0.29). Following ECC2, changes in muscle soreness were significantly different between the 40% and 80% (P = 0.02), 40% and 100% (P < 0.01), 60% and 100% (P < 0.01) groups, but no significant differences were evident between the 40% and 60% (P = 0.12), 60% and 80% (P = 0.86), and 80% and 100% (P = 0.42) groups.

Comparison Between ECC1 and ECC2 for Each Group

MVC and optimum angle. Compared with ECC1, significantly greater increases in optimum angle were seen following ECC2 for the 40% (P < 0.01) and 60% (P < 0.01) groups but not for 80% (P = 0.11) or 100% (P = 0.15) groups (Fig. 1). Decreases in MVC following ECC2 were significantly smaller than those following ECC1 for the 80% (P < 0.01) and 100% (P < 0.01) groups, but they were significantly greater for the 40% (P < 0.01) and 60% (P = 0.04) groups (Fig. 2).

ROM. Significantly smaller decreases in ROM after ECC2 than ECC1 were evident for the 80% (P = 0.04) and 100% (P < 0.01) groups, but significantly greater decreases were seen for the 40% (P = 0.03) and 60% (P = 0.04) groups (Fig. 3).

Cir. As shown in Fig. 4, increases in Cir were significantly smaller after ECC2 compared with ECC1 for the 80% (P = 0.05) and 100% (P = 0.04) groups, but there was no significant difference between bouts was evident for the 60% group and a significantly (P = 0.04) greater increase after ECC2 than ECC1 was observed for the 40% group.

CK and Mb. Compared with ECC1, changes in plasma CK activity and Mb concentration following ECC2 were significantly smaller for the 80% (P = 0.03 for CK, P = 0.02 for Mb) and 100% (P < 0.01 for CK, P < 0.01 for Mb) groups, but they were significantly greater for the 40% (P = 0.04 for CK, P = 0.03 for Mb), and there was no significant (P = 0.12 for CK, P = 0.08 for Mb) difference between bouts for the 60% group (Fig. 5).

Muscle soreness. Subjects reported less soreness following ECC2 than ECC1 for the 80% (P < 0.01) and 100% (P < 0.01) groups, with no significant difference between bouts for the 60% group (P = 0.12), and significantly greater soreness after ECC2 for the 40% group (P = 0.03).

Comparison Among Groups for the Magnitude of Repeated-Bout Effect

To compare the extent of protective effect conferred by the initial bout against the subsequent 100% bouts between groups, we compared the first bout of the 100% group and the second bout of each group, giving an “index of protection” based on the mean values of each group (Fig. 7). Thus, if the same responses occurred between first and second bouts it would represent “no protection” with an index of “0,” whereas if the responses to the second bout were smaller than that of the first bout it would represent a % protective effect. Because protection is better represented by the recovery for MVC and ROM, maximal changes for Cir, and peak value for serum CK activity, Mb concentration, and muscle soreness, the calculation of the index used the decrease in MVC and ROM at 5 days postexercise from baseline, the increase in Cir at 5 days postexercise from baseline, and the increase from preexercise to the peak for serum CK activity, Mb concentration, and muscle soreness. The index of protection shows that the larger the intensity of ECC1, the greater the magnitude of the protective effect. Although the magnitude of protective effect for the 40% group was the most discrete, this intervention was still effective in reducing the changes in criterion measures following ECC2 on ROM (65% protection), CK and Mb (~50%), Cir (25%), and muscle soreness (20%). However, it was notable that the 40% group conferred no protection for strength loss following ECC2, despite a significant effect on MVC following the initial bout (Fig. 2).
DISCUSSION

The present study tested the hypothesis that the higher the intensity of the initial bout, the larger the shift of the optimum angle, and the greater the magnitude of protective effect against a subsequent bout of maximal eccentric exercise. Although the differences between groups were not necessarily significant for all criterion measures following ECC1 and/or ECC2, the magnitude of the repeated-bout effect increased in line with the intensity of ECC1, such that the higher the intensity, the greater the level of protection conferred. However, the shift of optimum angle did not appear to explain the repeated-bout effect conferred by the 80%, 60%, and 40% groups, because no significant optimum angle shift was evident from ECC1 to ECC2 (Fig. 1), but these groups still demonstrated a protective effect for most criterion measures following ECC2 (Figs. 2–6).

It has been documented that the optimum joint angle for muscle torque output changes to a longer muscle length following eccentric exercise (23, 24), and this rightward shift in the muscle length-tension relationship has been attributed to an increase in the number of sarcomeres in series (24). Philippou et al. (23) reported that optimum angle increased 16–18° for 3 days following 50 maximal eccentric actions of the elbow flexors, and it was still 12° greater than baseline at 4 days postexercise. We found similar alterations in the optimum angle of elbow flexors for the 100% group, with other groups showing smaller increases after ECC1 (Fig. 1). The fact that these changes occurred immediately after exercise implies that sarcomere disruption is responsible, rather than the addition of new sarcomeres, although it cannot be discounted that sarcomere addition could have contributed to the changes present after several days of recovery. The significantly greater increases in optimum angle immediately after exercise for the 80% and 100% than the 40% and 60% groups may indicate that muscle fibers were more lengthened in the 80% and 100% exercises compared with the 40% and 60% groups. Butterfield and Herzog (3) reported a greater reduction of torque production in rabbit hindlimb muscles at short muscle lengths than long muscle lengths, resulting in an altered shape of the force-angle curve, and that the shift of optimum angle was caused by a combination of muscle damage and fatigue. The present study also found that the magnitude of changes in MVC at 160° after ECC1 was significantly smaller than that of other angles.

It is possible that an increase in passive tension of the elbow flexors at longer muscle lengths contributed to the smaller decrease in MVC at 160° because Howell et al. (8) reported that eccentric exercise increased passive tension of this muscle group. Although passive muscle tension was not measured in the present study, the decrease in ROM (Fig. 3), resulting from an increase in flexed elbow joint angle and a decrease in relaxed elbow joint angle, was likely to be associated with a rise in muscle passive tension. Whitehead et al. (26) reported that the increases in whole muscle passive tension after eccentric exercise occur as a result of developing injury contractures. Because MVC values were not gravity corrected and changes in muscle passive tension were not taken into account in the present study, some of the significant shifts in angle of peak torque may have been overestimated. It is important to note that the optimum angle was estimated from MVC values at different joint angles, and the smaller decreases in MVC at elbow-extended angles than elbow-flexed angles could explain the rightward shift in optimum angle.

In the present study, a significant shift of the optimum angle (4°) was still evident 2–3 wk following ECC1 for the 100% group (Fig. 1). Shifts in optimum angle of 6.5–10.9° for 10 days following eccentric exercise have been reported for the hamstrings (2) and 3.4–15.6° for 8 days following a step exercise for the quadriceps (1). The present study is the first demonstrate a shift of optimum angle lasting for at least 2–3 wk after eccentric exercise of the elbow flexors. It is important to note that only the 100% group showed a significantly larger optimum angle immediately before ECC2 compared with pre-ECC1 (Fig. 1). This is in contrast to the findings of Jones et al. (9), who found that the increase in optimal angle of the ankle plantar flexors following backward downhill walking returned to control values by 2 days postexercise. The most likely explanation for our finding of a long-lasting shift in the joint angle-force relationship is that the exercise stimulus used in the present study induced greater muscle damage than the backward walking model. Butterfield and Herzog (4) proposed that the larger the intramuscular stress and strain associated with an exercise bout, the greater the subsequent sarcomerogenesis. Our finding of larger shifts in optimum angle for the 100% and 80% groups compared with 60% and 40% groups (Fig. 1) supports this concept. Whitehead et al. (26) found that eccentric contractions at long muscle lengths resulted in a larger shift of optimum angle compared with those at short lengths. It may be that greater forces generated at long muscle lengths during the 100 or 80% eccentric exercise were responsible for the larger shift of the optimum angle.

It has been postulated that the addition of sarcomeres in series allows myofibrils to operate at longer lengths, thus avoiding the descending limb of the length-tension curve (4, 12, 17), and this may explain the underlying mechanism of the repeated-bout effect (17, 24). Further support for this theory comes from the fact that eccentric knee extensor exercise performed over the shortened part of the length-tension curve did not protect against damage when the same exercise was performed over longer lengths (46). In contrast, Nosaka et al. (20), in the elbow flexors, showed that eccentric exercise over short muscle lengths conferred some protection (~50–70% attenuation) against a repeated bout over a lengthened ROM. On the basis of the results from the present study, these apparently contradictory findings may reflect the level of damage seen following the initial bout. It should be noted that the optimum angle immediately before ECC2 was not significantly different from pre-ECC1 for the 40%, 60%, and 80% groups. Despite this, all groups showed a repeated-bout effect for most of the criterion measures (Figs. 2–6). Moreover, significant differences in the magnitude of the repeated-bout effect were observed between the 40%, 60%, and 80% groups, despite similar optimum angles before ECC2. Additionally, the differences between the 80% and 100% groups for changes in the criterion measures following ECC1 and ECC2, with the exception of strength, were negligible (Fig. 7), but a significant difference between the groups was evident for the pre-ECC2 optimum angle (Fig. 1). Taken together, these findings suggest that a sustained rightward shift in optimum joint angle is not...
necessarily a prerequisite for conferral of the protective effect. It is not known how the small shift of the optimum angle (4°) observed before ECC2 for the 100% group contributed to the attenuation of muscle damage after the second eccentric exercise bout. Further studies are required to examine the relationship between the shift of optimum angle and the changes in sarcomeres number in series.

When making comparisons between the responses of the different eccentric exercise groups, it should be borne in mind that the motor unit populations responsible for torque production may well differ between conditions. Our study was based on the assumption that gradation of force production of the elbow flexors is a result of a greater number of motor units recruited (10). Also, the 40% MVC paradigm will probably have had a greater proportion of slower units active than that of the 100% MVC group during the eccentric exercise protocol. This issue is further complicated by the fact that there is evidence that the motor unit activation patterns of the elbow flexors during eccentric actions differ from concentric or isometric actions (11). Moreover, it is known that the length-tension properties of motor units are affected by the rate of firing, such that there is a shift in the length-tension curve to the right (i.e., longer lengths) during submaximal activation (18). However, these differences would also exist when individuals perform submaximal eccentric actions in real-life activities, and so the studies described here represent a valid model for studying the repeated bout effect from an applied exercise context. Another issue to be taken into account is the velocity of eccentric actions. It has been reported from findings comparing two distinct velocities (i.e., 20°/s vs. 210°/s) that eccentric action velocity was a factor in determining the magnitude of muscle damage (25). We were careful to observe any inconsistency in our subjects while they were performing the eccentric actions, and some subjects had difficulty lowering the dumbbell at a constant velocity for the last few contractions, we do not believe this was sufficient to affect the magnitude of muscle damage.

The present study clearly showed that the protective effect against maximal eccentric exercise was conferred by submaximal eccentric exercise, and even a low (40%) intensity eccentric exercise conferred protection of 20–60% on the indexes of muscle damage following a subsequent 100% exercise bout, with the notable exception of strength loss (where no protection was conferred). It would be interesting to examine whether an initial eccentric exercise bout of <40% would confer some protective effect. Our findings suggest that preconditioning by submaximal eccentric exercise can be used as a strategy to reduce muscle damage and soreness, but the magnitude of the effect varies between the criterion measures. Thus the protective effect is strong for ROM and plasma protein efflux from muscle, in which even the initial 40% bout afforded a protective effect of over 50% against a maximal bout. In contrast, Cir and muscle soreness resulted in a smaller attenuation to ECC2, and no repeated bout effect was conferred by the 40% bout for strength loss.

In summary, we have shown that the magnitude of the repeated-bout effect is dependent on the exercise intensity of the initial bout, and the higher the intensity of the initial bout, the greater the protective effect conferred. The rightward shift of optimum angle also shows a relationship with the degree of muscle damage associated with the initial bout, but it does not appear to be directly related to the mechanisms responsible for the repeated bout effect. Further studies are necessary to delineate the mechanisms of the repeated-bout effect, but the fact that submaximal eccentric exercise, resulting in minor damage, can still confer a protective effect against maximal eccentric exercise, provides practical implications for procedures aimed at ameliorating the symptoms of exercise-induced muscle damage.

REFERENCES


