Acute adaptation to low volume eccentric exercise

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ABSTRACT

PADDON-JONES, D., and P. J. ABERNETHY. Acute adaptation to low volume eccentric exercise. *Med. Sci. Sports Exerc.*, Vol. 33, No. 7, 2001, pp. 1213–1219. **Purpose:** Many symptoms of eccentric muscle damage can be substantially reduced if a similar eccentric bout is repeated within several weeks of the initial bout. The purpose of this study was to determine whether a nondamaging, low repetition, low volume eccentric exercise bout could also provide a protective/adaptive effect. **Methods:** Subjects were assigned to a control (CON), eccentric exercise (ECC), or low volume familiarized eccentric exercise group (LV+ECC). Before the study, the LV+ECC group performed six maximal eccentric contractions during two familiarization sessions. The main eccentric bout targeted the elbow flexor muscle group and consisted of 36 maximal eccentric contractions. Muscle soreness, upper arm girth, elbow angle, creatine kinase activity, isometric torque, and concentric and eccentric torque at 0.52 and 3.14 rad·s^{-1} were assessed 0, 1, 2, 3, 4, 7, and 10 d postexercise. **Results:** No evidence of muscle damage was observed as a result of the low volume eccentric bouts. Nevertheless, with the exception of muscle soreness and concentric torque, all variables recovered more rapidly in the LV+ECC group ($P < 0.05$). **Conclusion:** Adaptation to eccentric exercise can occur in the absence of significant muscle damage. Exposure to a small number of nondamaging eccentric contractions can significantly improve recovery after a subsequent damaging eccentric bout. Furthermore, this adaptation appears to be mode-specific and not applicable to concentric contractions. **Key Words:** DOMS, CREATINE KINASE, ISOKINETIC, STRENGTH, REPEATED-BOUT

Despite the often debilitating effects of unaccustomed eccentric exercise, research has consistently shown that when the same eccentric bout is repeated 1–4 wk after the first bout, little or no structural or functional impairment occurs (3,18,19). Two studies have reported similar adaptive effects when a normally damaging eccentric exercise bout was preceded by a lower volume eccentric bout (2,4). Clarkson et al. (4) demonstrated that an initial bout of 24 maximal eccentric repetitions reduced plasma creatine kinase (CK) activity, the magnitude of the strength loss and delayed onset muscle soreness (DOMS) when a 70-repetition bout was performed 2 wk later. However, this initial 24-repetition eccentric bout was sufficient to cause significant muscle soreness and a 15–20% decrement in isometric torque 24 h post exercise. In a similar study, 10 maximal eccentric repetitions facilitated a reduction in plasma CK activity and reduced the magnitude of isometric force loss (48–72 h postexercise) when a 50-repetition eccentric bout was performed 3 wk later (2). However, this initial eccentric bout also produced significant muscle soreness and a 15–20% reduction in peak isometric force 24–48 h post exercise.

Many of the potential mechanisms responsible for the repeated-bout effect have been reviewed in detail (1,6,18,19). One early, yet prominent, hypothesis contends that exposure to eccentric contractions damages a susceptible pool of high threshold motor units resulting in a loss of contractile force, increased muscle protein release, swelling, and DOMS (9,13,17,20). In a repeated-bout model, if the initial eccentric insult does induce a greater stress or strain on a selective group of muscle fibers (e.g., Type II), motor unit activation and recruitment patterns may be subsequently altered, thereby resulting in less muscle damage and functional impairment after a repeated eccentric exercise bout.

Although it is clear that previous exposure to eccentric exercise reduces the severity of many common markers of muscle damage, the initial eccentric bouts in all repeated-bout models to date have adversely affected one or more markers of muscle damage (2,3,4,18,19). Consequently, it is not clear whether initial exposure to eccentric contractions must induce muscle soreness and/or cause a significant reduction in functional capacity (e.g., $\geq$ 15–20% reduction in strength parameters) in order to initiate the adaptive process. Similarly, despite the wide range of eccentric contraction velocities that can be used to induce muscle damage and measure functional impairment/recovery (e.g., fast: downhill running, slow: isokinetic dynamometry), it remains uncertain whether the ability to generate force after an eccentric muscle injury is dependent on the contraction mode and/or velocity of the posttest strength measure (15).

The primary purpose of this study was to determine whether a small number of noninjurious eccentric contractions (i.e., low volume bout) could influence the recovery of several common markers of eccentric muscle damage after a higher volume eccentric exercise bout performed 7 d later. A secondary purpose was to determine whether the resultant changes in muscle torque were mode (concentric vs

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isometric vs eccentric) and/or velocity (0.52 rad·s^{-1} vs 3.14 rad·s^{-1}) specific.

METHODS

Subjects. Twenty-three nonresistance trained male (N = 12) and female (N = 11) subjects participated in this study that complied with the requirements of The University of Queensland Medical Research Ethics Committee. Written consent was obtained from all participants. Mean (± SD) age, height, and mass were 23.9 yr (± 4.9), 172.6 cm (± 8.2), and 67.4 kg (± 11.6), respectively. Subjects were counterbalanced on pretest torque values and randomly assigned to an eccentric (ECC, N = 7), low volume plus eccentric (LV+ECC, N = 8), or control (CON, N = 8) group.

Experimental design. All subjects completed two familiarization sessions 10 and 7 d before the main eccentric bout. During each familiarization session, all subjects performed three maximal isometric contractions of the elbow flexors and three maximal concentric contractions at both 0.52 and 3.14 rad·s^{-1}. In addition, on each occasion the LV+ECC group performed three maximal eccentric elbow flexor contractions at 0.52 and 3.14 rad·s^{-1}. To minimize the number of repetitions performed before the main eccentric bout, potential changes in muscle torque attributable to the low volume bouts were investigated using posttest CON group data (see below).

Seven days after the second familiarization session, the LV+ECC and ECC groups completed the main eccentric bout. This eccentric bout was preceded (pretest) and followed (posttesting) by identical testing sessions that included the measurement of plasma CK activity, muscle soreness, upper arm girth, relaxed elbow angle, and eccentric, concentric, and isometric torque.

Subjects in the CON group did not perform the initial low volume eccentric familiarization sessions or the main eccentric bout. However, during the pretest and each posttesting session the CON group completed the same low volume eccentric protocol initially performed by the LV+ECC group. Thus, compared with the initial low volume eccentric familiarization sessions (LV+ECC group), the CON group actually performed a greater total number of the low volume bouts during the posttesting period. Consequently, although CON group data provide only an indirect indication of the damaging effects of the two low volume eccentric bouts, it is also likely that any error in this approach would be manifest as an overestimation of the muscle damage experienced.

Dependent variable testing. The choice of which dependent variables to assess after an eccentric exercise intervention may influence data interpretation (24). Nevertheless, the majority of previous studies have exclusively examined postexercise changes in isometric torque, whereas very few have examined changes in muscle torque during concentric and eccentric contraction modes. Furthermore, there are very few data on the consequences of using different contraction velocities to quantify posteccentric exercise strength loss (9). These gaps in our knowledge led us to select a broader range of dependent variables to examine in the present study, including novel measures of mode and velocity specificity.

The order of dependent variable pre- and post-testing was standardized to avoid potential confounding interactions between the various tests (21,25). Muscle soreness was measured first, followed by upper arm girth, elbow angle, plasma CK activity, and finally strength parameters. Pretesting was completed 15–20 min before the main eccentric bout. Posttesting of all variables occurred at 15 min (time 0) and at 1, 2, 3, 4, 7, and 10 d with the exception of plasma CK activity, which was not measured immediately after the main eccentric bout. For each individual, all pre- and posttesting sessions were performed at the same time of day (range: 0800–1400).

Muscle soreness was assessed via pressure algometry (8,14). With the subject in a supine position, arm comfortably extended and relaxed, the algometer (Pain Diagnostics & Thermography, Inc., Great Neck. NY) was applied vertically with increasing force to the middle of the biceps brachii, 4 cm proximal to the elbow fold. Subjects indicated when the algometer elicited a pain, rather than a pressure sensation. The corresponding force gauge measurement was recorded and averaged over two trials. The reliability and reproducibility of pressure algometry has been described as excellent (8).

Upper arm girth was measured mid-way between the lateral aspect of the acromial process and the lateral epicondyle of the elbow. Relaxed elbow angle was defined as the acute angle between the capitate depression of the wrist, the lateral epicondyle, and the 2 cm anterior to the lateral acromial process of the shoulder. Measurements were obtained using a metal goniometer with the subjects in a relaxed upright stance, palms facing medially.

Plasma CK activity was measured spectrophotometrically (Cobas Mira, Roche Diagnostics, Basel, Switzerland) from venous blood samples by means of an N-acetylcysteine activated, optimized ultraviolet test kit (Roche Diagnostics, Sydney, Australia).

The main eccentric exercise bout and all familiarization, pre- and post-test strength measures targeted the elbow flexors of the nondominant arm and were performed on a preacher curl bench (Force Fitness Systems: 42SBP-U, Brisbane, Australia) located along side an isokinetic dynamometer (Cybex 6000, Lumex Inc., Ronkonkoma, NY). With the subject in a stable seated position, the nondominant arm was placed on the sloping front incline (0.78 rad) of the preacher curl bench with the elbow adjacent to the axis of rotation of the dynamometer. Mechanical stops were engaged to prevent excessive flexion or extension and standardize elbow range of motion at 2.27 rad for concentric and eccentric contractions. Peak isometric elbow flexor torque was assessed at an elbow angle of 1.57 rad. Similarly, angle-specific concentric and eccentric torque values were obtained at an elbow angle of 1.57 rad. During each pre- and post-test session, peak torque values were recorded during three maximal isometric contractions and three maximal

concentric and eccentric contractions at 0.52 and 3.14 rad·s^{-1}. There was a 2-min rest period between contraction modes and a 2.7-s interval between concentric and eccentric repetitions while the dynamometer returned to the starting position.

Eccentric exercise. Fifteen to twenty minutes after the pretest, subjects in the LV+ECC and ECC groups performed the main eccentric bout, which consisted of 36 maximal voluntary eccentric contractions. Repetitions were performed in sets of six (6 reps × 6 sets) at an angular velocity of 0.52 rad·s^{-1} with a 60-s rest period between sets. Each repetition was performed using the same criteria employed during pre- and post-testing. Specifically, subjects maximally resisted the dynamometer as it forced their arm from full flexion (0.70 rad) to near full elbow extension (2.90 rad). On completion of each eccentric repetition, the dynamometer passively returned the subject's arm to full flexion at 0.26 rad·s^{-1} for the start of the next repetition.

Statistical analysis. Data were assessed for normality using a D'Agostino-Pearson omnibus test. A logarithmic transformation (Log_{10}) was applied to nonnormally distributed data (girth, CK, strength parameters) before analysis. *t*-tests were used to identify potential differences between male and female subjects. Planned comparisons of group means were performed to investigate differences between concentric and eccentric contraction velocities and LV+ECC, ECC, and CON groups at each testing session (16). Significance was accepted at an alpha of $P < 0.05$. CON group data were assessed using a one-way ANOVA with repeated measures. To reduce the likelihood of a type II error in CON group analysis, significance was accepted at an alpha of $P < 0.1$ (23). For consistency of units and comparison, strength, girth, and elbow angle graphical data were expressed as mean (±SEM) percentage values, whereas absolute values were reported in the text.

RESULTS

Low volume eccentric exercise. During the low volume familiarization sessions, LV+ECC peak (± SEM) eccentric torque values at 0.52 and 3.14 rad·s^{-1} were 57.1 ± 10.6 Nm and 54.1 ± 9.7 Nm, respectively. These values were not significantly different from pretest values ($P > 0.05$). During each posttesting session, the CON group completed the same low volume eccentric protocol performed by LV+ECC during the initial familiarization sessions. Despite performing a total of six low volume bouts of eccentric exercise during the posttesting period, CON group data (0–10 d) did not differ significantly from initial pretest values for any dependent variable ($P > 0.1$).

Acute eccentric exercise bout. During the 36 repetition eccentric protocol, LV+ECC and ECC groups performed a similar amount of eccentric work, with mean (± SEM) values of 2532 ± 560 J and 2524 ± 404 J, respectively ($P > 0.05$). Total work per set in the LV+ECC group decreased from 544.7 ± 111.3 J to 340.0 ± 78.0 J, whereas the ECC group decreased from 501.7 ± 65.5 J in the first set to 376.5 ± 70.2 J in the final set. Similar reductions in peak eccentric torque were observed over the six sets, with values in the LV+ECC group decreasing from 60.6 ± 9.3 Nm (set 1) to 43.6 ± 7.7 Nm (set 6) and ECC group values decreasing from 60.8 ± 10.8 Nm (set 1) to 41.0 ± 6.6 Nm (set 6).

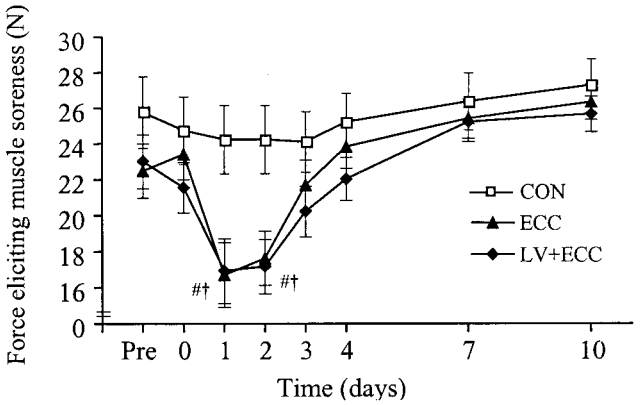

FIGURE 1—Force required to produce muscle soreness in the elbow flexor muscle group; # denotes a significant difference between ECC and CON; † denotes a significant difference between LV+ECC and CON ($P < 0.05$).

Muscle soreness. Muscle soreness values peaked 24–48 h postexercise in both LV+ECC and ECC groups ($P < 0.05$) but were not significantly different from the CON group by 72 h postexercise. There were no differences between LV+ECC and ECC groups at any time during the study ($P > 0.05$) (Fig. 1).

Girth. Immediately after the eccentric exercise bout, upper arm girth in the ECC and LV+ECC groups had increased by 3.2% and 2.6%, respectively ($P < 0.05$). ECC group girth values remained significantly greater than CON and LV+ECC through days 1–10. LV+ECC group values were not significantly different from CON group values from 72 h postexercise (Fig. 2).

Elbow angle. No significant differences in pretest resting elbow angle were observed between groups. Throughout posttesting, the ECC group experienced significant reduction in resting elbow angle. Elbow angle values in the LV+ECC group ranged from 2.72 ± 0.02 rad (pretest) to 2.60 ± 0.05 rad (post 48 h) but were not significantly

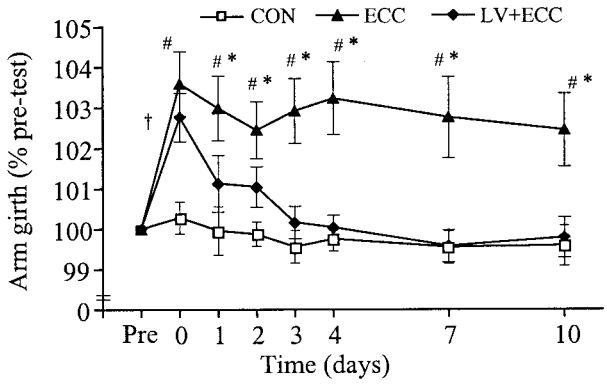

FIGURE 2—Relative change in upper arm girth (%); * denotes a significant difference between LV+ECC and ECC; # denotes a significant difference between ECC and CON; † denotes a significant difference between LV+ECC and CON ($P < 0.05$).

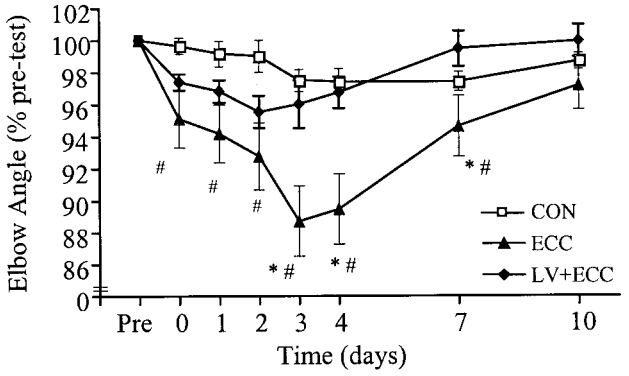

FIGURE 3—Relative change in resting elbow angle (%); * denotes a significant difference between LV+ECC and ECC; # denotes a significant difference between ECC and CON ($P < 0.05$).

different from CON group values. In comparison, significant reductions in elbow angle were observed in the ECC group with values ranging from 2.65 ± 0.04 rad (pretest) to 2.35 ± 0.08 (post 72 h) (Fig. 3).

Plasma CK activity. Plasma CK activity in the CON group did not change significantly during the study (CON peak: 114.7 ± 21.1 $U \cdot L^{-1}$). Similarly, despite the large functional impairment caused by the major eccentric bout, no significant increases in plasma CK in the LV+ECC group were observed (LV+ECC peak: 182.1 ± 64.1 $U \cdot L^{-1}$). In contrast, plasma CK values in the ECC group increased dramatically after the eccentric exercise bout, reaching peak concentrations of 4030.4 ± 1029 $U \cdot L^{-1}$ on day 4 (Fig. 4).

Isometric torque. Pretest isometric torque values were 62.1 ± 10.2 Nm (LV+ECC), 60.9 ± 8.9 Nm (ECC), and 57.9 ± 9.8 Nm (CON) ($P > 0.05$). Both the ECC and LV+ECC groups experienced a 45% reduction in torque immediately postexercise ($P < 0.05$). However, the LV+ECC group demonstrated a greater rate of recovery, producing significantly higher isometric torque values than the ECC group from 48 h postexercise. Isometric torque values in both groups remained significantly lower than CON on day 10 (LV+ECC: 55.5 ± 8.2 Nm, ECC: 48.0 ± 9.8 Nm) (Fig. 5).

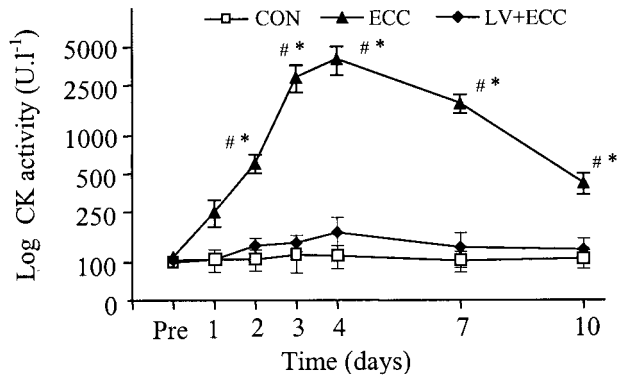

FIGURE 4—Change in plasma creatine kinase activity; * denotes a significant difference between LV+ECC and ECC; # denotes a significant difference between ECC and CON ($P < 0.05$).

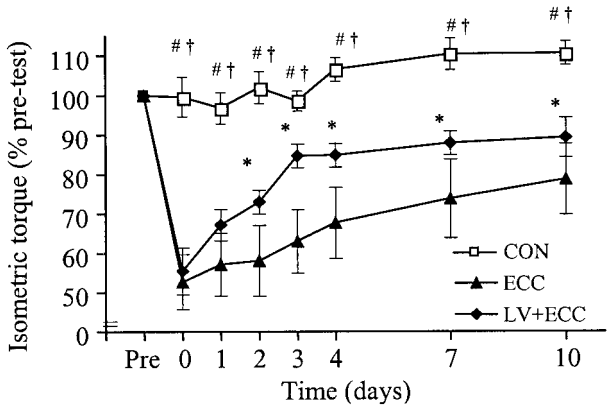

FIGURE 5—Relative change in isometric torque (%);* denotes a significant difference between LV+ECC and ECC; # denotes a significant difference between ECC and CON; † denotes a significant difference between LV+ECC and CON ($P < 0.05$).

Concentric torque. Throughout the posttesting period, the LV+ECC and ECC groups produced significantly lower concentric torque values than the CON group at both contraction velocities (Fig. 6, a and b). Pretest concentric torque values at 0.52 $rad \cdot s^{-1}$ were 46.8 ± 8.1 Nm (LV+ECC), 44.4 ± 6.1 Nm (ECC), and 39.9 ± 6.6 Nm (CON), respectively ($P > 0.05$). By 24 h postexercise, torque had decreased to 30.5 ± 4.8 Nm (LV+ECC) and 24.5 ± 5.3 Nm (ECC). Concentric torque values at 0.52 $rad \cdot s^{-1}$ in the ECC group were significantly lower than the LV+ECC group between 48 and 96 h postexercise but were not significantly different at any other time.

No significant differences between ECC and LV+ECC were observed when concentric torque was assessed at 3.14 $rad \cdot s^{-1}$. Pretest values were 38.3 ± 6.8 Nm (LV+ECC), 39.9 ± 5.8 Nm (ECC), and 34.3 ± 7.3 Nm (CON), respectively ($P > 0.05$), with values decreasing to 20.9 ± 2.7 Nm (LV+ECC) and 28.1 ± 4.7 Nm (ECC) 24 h postexercise. At 15 min and 24 h postexercise, LV+ECC concentric torque values at 3.14 $rad \cdot s^{-1}$ were lower than values obtained at 0.52 $rad \cdot s^{-1}$ ($P < 0.05$).

Eccentric torque. Pretest eccentric torque values measured at 0.52 $rad \cdot s^{-1}$ were 58.8 ± 9.8 Nm (LV+ECC), 57.3 ± 9.3 Nm (ECC), and 57.4 ± 9.4 Nm (CON), respectively ($P > 0.05$). The lowest torque values for the LV+ECC (37.4 ± 5.8Nm) and ECC groups (31.9 ± 6.2 Nm) were recorded 24–48 h postexercise. Torque values at 0.52 $rad \cdot s^{-1}$ in the ECC group were significantly lower than CON throughout posttesting and were also significantly lower than LV+ECC on days 7 and 10 (Fig. 7a).

Pretest eccentric torque values measured at 3.14 $rad \cdot s^{-1}$ were 54.8 ± 8.7 Nm, 53.6 ± 9.0 Nm, and 54.1 ± 8.4 Nm for LV+ECC, ECC, and CON groups respectively ($P > 0.05$). ECC group torque values were significantly lower than the CON group throughout posttesting and were lower than LV+ECC values from 48 h to day 10. In comparison, LV+ECC group eccentric torque values (3.14 $rad \cdot s^{-1}$) were not significantly different from the CON group from 72 h postexercise (Fig. 7b). At 72 h postexercise, LV+ECC

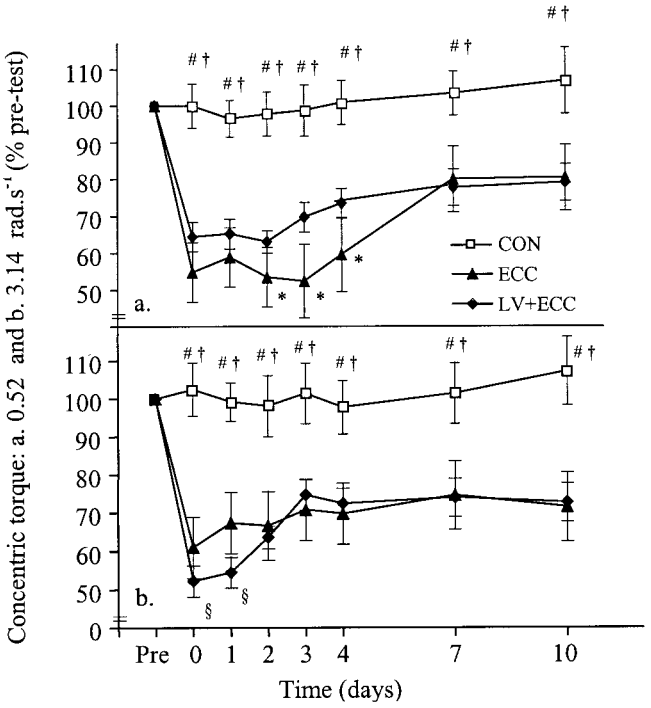

FIGURE 6—Relative change in concentric torque at (a) 0.52 rad·s^{-1} and (b) 3.14 rad·s^{-1}; * denotes a significant difference between LV+ECC and ECC; # denotes a significant difference between ECC and CON; † denotes a significant difference between LV+ECC and CON; § denotes significant difference between LV+ECC at 0.52 and 3.14 rad·s^{-1} ($P < 0.05$).

eccentric torque at 3.14 rad·s^{-1} was greater than eccentric torque at 0.52 rad·s^{-1} ($P < 0.05$).

DISCUSSION

The results of this study suggest that a small number of eccentric contractions can facilitate an adaptive process, which improves the rate of recovery of upper arm girth, elbow angle, CK activity, isometric torque, and eccentric torque after a second larger eccentric exercise bout. Furthermore, unlike previous research (2,4), this adaptive response occurred despite the fact that repeated daily bouts of low volume eccentric contractions (i.e., CON group) did not provoke significant changes in several common markers of muscle damage, including muscle soreness, upper arm girth, elbow angle, CK activity, isometric torque, and concentric and eccentric torque. These data appear to refute an earlier hypothesis (9,13,17,20) and suggest that initial symptomatic muscle fiber damage and/or destruction of a susceptible muscle fiber pool may not be a necessary prerequisite or stimulus for the eccentric adaptation process.

The magnitude of the immediate (0–24 h) posteccentric exercise torque loss during different contraction modes and velocities ranged from 35 to 50% and was consistent with previous research using both lower (10) and upper limb models (20). Functional impairment and strength loss after eccentric contractions has been strongly linked to structural damage to the contractile apparatus with concomitant morphological changes including Z-band disruption, myofibrillar damage, and disruption of sarcolemmal integrity (5,6,9,18). However, it appears that after repeated eccentric bouts, substantial ultrastructural damage may occur without a corresponding reduction in force producing capacity. In a recent study (11), subjects repeated a bout of 100 eccentric quadriceps contractions 2 wk after an initial bout. Forty-eight hours after the repeated bout there was no increase in plasma CK concentration or strength impairment despite visual evidence of morphological damage to 23 ± 4% of regions (cf. 65 ± 12% in bout 1) examined in micrographed biopsy samples.

In the present study, the prolonged strength decrement and grossly elevated plasma CK concentrations observed in the ECC group suggest that substantial ultrastructural muscle fiber damage occurred after the main eccentric bout. Similarly, previous research supports our contention that the LV+ECC group also experienced muscle fiber damage despite the improved rate of recovery of several variables (11). The failure of the eccentric bout to increase plasma CK activity in the LV+ECC group was consistent with a number of previous studies employing a repeated-bout design (3,4,5,6). However, a novel finding of this study was that the CK response after a damaging bout of intense eccentric exercise can be abolished by previous exposure to a small number of eccentric contractions that, when performed in the absence of a subsequent larger eccentric bout, do not adversely effect any of the markers of muscle damage used in this study design. The mechanism/s responsible for the

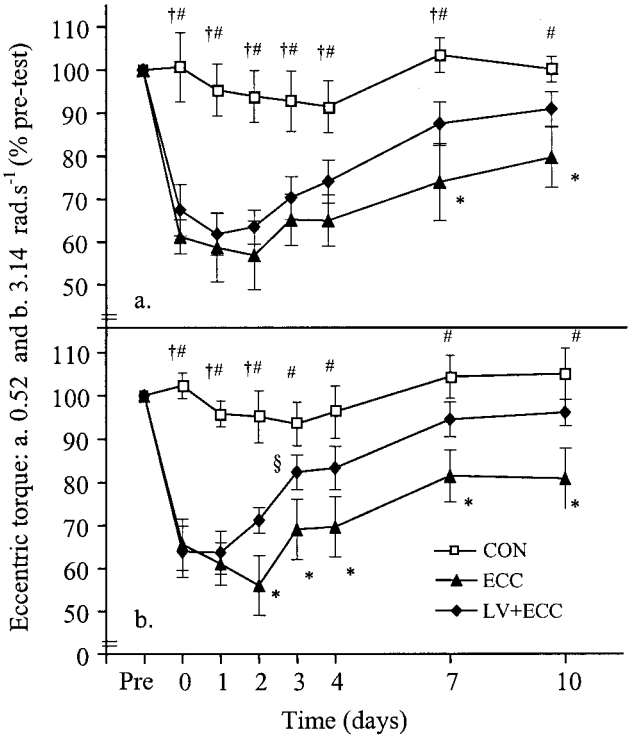

FIGURE 7—Relative change in eccentric torque at (a) 0.52 rad·s^{-1} and (b) 3.14 rad·s^{-1}; * denotes a significant difference between LV+ECC and ECC; # denotes a significant difference between ECC and CON; † denotes a significant difference between LV+ECC and CON; § denotes significant difference between LV+ECC at 0.52 and 3.14 rad·s^{-1} ($P < 0.05$).

muted CK response in the LV+ECC group are unclear. It is possible that strengthening of the sarcolemmal membrane occurred after the low volume eccentric bouts (2,5,6). However, if the LV+ECC group experienced sufficient sarcolemmal and myofibrillar disruption to provide the potential for muscle protein release after the main eccentric bout (11), then the failure to elicit an increase in plasma CK activity may have simply been a consequence of a down-regulation of CK production and/or an increased plasma CK clearance (6). Clearly, further research is necessary.

Although the initial postexercise increase in girth and reduction in elbow angle were most likely related to a transient increase in peripheral blood flow, the changes observed from 24 h postexercise were most likely due to edema formation and swelling (13,24). The greater subsequent recovery of these variables in the LV+ECC group is also suggestive of a peripheral adaptation facilitated by the initial low volume eccentric bouts. As previously described, a recent study demonstrated a 40% reduction in visual evidence of morphological damage after repeated bouts of eccentric exercise (11). This raises the possibility that one or more peripheral structures (e.g., myofibrils, sarcolemma, extracellular matrix) may become more resistant to eccentric injury as a result of an initial low volume eccentric bout and that this adaptation may contribute to the faster rate of recovery observed after a subsequent damaging eccentric bout (7). Alternatively, although the initial low volume eccentric bouts may not result in observable symptoms of muscle damage, they may prime the body's injury repair mechanisms, resulting in an earlier, more efficient mobilization of inflammatory mediators, such as neutrophils, macrophages, and associated cytokines; and interleukin-1, interleukin-6, and tumor necrosis factor (TNF) (1,6). Nevertheless, the existence or extent of peripheral muscular adaptation after only a small number of eccentric contractions has yet to be directly examined. Further, the determination that DOMS was not influenced by the low volume eccentric bouts provides further evidence of the tenuous relationship between markers of DOMS, inflammation, muscle fiber damage, and swelling (1,6,13).

The influence of neural factors on functional parameters after eccentric exercise has attracted recent attention. Researchers have suggested that the development of maximal voluntary eccentric torque in novice subjects is incomplete and may be limited by an inhibitory or tension limiting neural mechanism (12,22,25). This submaximal muscle activation could represent a greater reserve for improvements in eccentric strength and may contribute to enhanced neural strength gains following initial exposure and adaptation to eccentric exercise (12). Although it may be argued that, in the absence of any peripheral musculotendinous adaptation, a neurally mediated increase in eccentric force production may place more stress on the contractile apparatus thereby increasing the severity of muscle damage, previous research has suggested that the opportunity to learn a more efficient motor unit recruitment pattern may favorably alter motor unit recruitment and reduce the stress placed on individual myofibrils during a repeated eccentric bout (10,11). Neural adaptation to eccentric exercise has been clearly demonstrated in a study reporting substantial centrally mediated strength gains in an untrained contralateral limb after ipsilateral eccentric training (12). Furthermore, these neural strength gains appear to be mode specific, producing significant ipsilateral increases in eccentric and isometric torque but not concentric torque.

In the present study, the initial reduction in the ability to generate muscle torque after the eccentric bout (0–48 h postexercise) was similar in both the LV+ECC and ECC groups. However, during later posttesting sessions, it became apparent that not only did the LV+ECC group recover faster than the ECC group but the improved recovery rate was largely mode-specific, improving the recovery of eccentric and isometric but not concentric torque. This result was consistent with an earlier study which found that concentric but not eccentric or isometric strength was impaired when a bout of 100 eccentric leg extensions was repeated 3 wk after bout 1 (10). As a possible explanation for this phenomenon, we suggest that although the magnitude of muscle damage and functional impairment immediately after the major eccentric bout were similar in both experimental groups, low volume eccentric familiarization provided a sufficient central stimulus to partially offset the neural inhibitory mechanism, thereby improving eccentric and isometric torque production during later posttest sessions.

In terms of velocity specificity, data from the present study indicate that at 0 and 24 h postexercise, the LV+ECC group experienced a greater concentric torque impairment at 3.14 $rad \cdot s^{-1}$ than 0.52 $rad \cdot s^{-1}$. Given that the damaging eccentric bout was performed at 0.52 $rad \cdot s^{-1}$, this result suggests that in a repeated-bout design, the initial relative impairment in concentric torque may be greater when examined using a concentric contraction velocity (i.e., 3.14 $rad \cdot s^{-1}$) that is greater than (or perhaps just different from) the velocity of the damaging eccentric bout (i.e., 0.52 $rad \cdot s^{-1}$). This result was consistent with an early study which reported that concentric knee extensor torque recovered more slowly at higher contraction velocities after and eccentric cycling task (9). An experimental design that examines the recovery of torque at contraction velocities both faster and slower than the velocity of the damaging bout would be useful to further explore this topic.

Posttest eccentric torque values in the LV+ECC group showed some indication of a more direct relationship with the velocity of the damaging eccentric bout. Specifically, a greater relative impairment in eccentric torque was observed on day 3 ($P < 0.05$) when eccentric torque was posttested at 0.52 $rad \cdot s^{-1}$, the same velocity as the damaging eccentric bout. The reason for this apparent discrepancy between concentric and eccentric contraction modes is unclear but may be related to preferential or selective Type II muscle fiber recruitment and/or damage after eccentric exercise (9,17). Again, further research is needed to clarify the relationship between muscle damage, contraction mode, and contraction velocity.

In conclusion, this study demonstrates that initial exposure to a small number of eccentric contractions can facilitate faster

recovery of some but not all strength and anthropometric variables after a larger subsequent eccentric exercise bout. Improved recovery of eccentric and isometric torque, elbow range of motion, limb girth, and plasma CK concentration occurred despite the fact that the low volume eccentric exercise bouts (CON group) did not significantly influence any marker of muscle damage. We suggest that the inducement of symptomatic muscle fiber damage may not be necessary to initiate the eccentric adaptation process. However, given the fact that muscle soreness was not influenced by the low volume eccentric familiarization, we suggest that a greater volume and/or an increased number of sessions of eccentric familiarization may be required to initiate an adaptive effect that improves subject comfort as well as functional ability.

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