Muscle damage and soreness after endurance exercise of the elbow flexors

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ABSTRACT
NOSAKA, K., M. NEWTON, and P. SACCO. Muscle damage and soreness after endurance exercise of the elbow flexors. Med. Sci. Sports Exerc., Vol. 34, No. 6, pp. 920 –927, 2002. Purpose: This study investigated changes in indirect markers of muscle damage after endurance exercise of the elbow flexors and compared the changes with those after maximal eccentric actions (Max-ECC) of the elbow flexors. Methods: Eighteen male students rhythmically lifted (1 s) and lowered (1 s) a light dumbbell (1.1–1.8 kg: 9% of MIF) in 60–180° of elbow joint angle for 2 h (2-h Ex). Maximal isometric force (MIF), relaxed (RANG) and flexed elbow joint angles (FANG), upper-arm circumference (CIR), muscle soreness (SOR), B-mode ultrasound (US), and plasma creatine kinase (CK) activity were assessed before and immediately after, and up to 96 h after exercise. Results: All measures were altered significantly \((P < 0.05)\) after 2-h Ex in a similar time course to Max-ECC; however, changes in RANG, FANG, CIR, US, and CK (peak: 356 ± 121 IU·L\(^{-1}\)) were significantly \((P < 0.05)\) smaller compared with those after Max-ECC. SOR developed immediately after 2-h Ex and peaked 24 – 48 h after exercise. MIF dropped to 44.1% of the preexercise level, which was significantly \((P < 0.05)\) lower than that after Max-ECC (58.1%), immediately postexercise. MIF recovered to 79.8% at 24 h, and 97.8% at 96 h postexercise, which was a significantly \((P < 0.05)\) faster recovery compared with that of Max-ECC (73.1% at 96 h). Conclusion: These results showed low-intensity continuous muscle contractions (3600 times) resulted in muscle damage; however, the magnitude of the muscle damage was less severe, and the recovery was faster compared with 12 maximal eccentric muscle actions. Key Words: MAXIMAL ISOMETRIC FORCE, UPPER-ARM CIRCUMFERENCE, PLASMA CK ACTIVITY, FATIGUE, ECCENTRIC EXERCISE

A n eccentric muscle action refers to a lengthening of an active muscle, a common example being the forced lengthening of a muscle generating maximal force by imposing higher load (1,32). It has been reported that only a few maximal eccentric actions of the elbow flexors induce delayed onset muscle soreness (DOMS) and muscle damage (7,29). This maximal eccentric action model has been frequently used to investigate DOMS and muscle damage (5,27,29); however, submaximal eccentric actions are more often performed in resistance training, for example, when lowering a dumbbell in a controlled manner. Submaximal eccentric exercise models have been also used for the study of DOMS and muscle damage (11,28). Muscle damage after submaximal exercise was significantly reduced compared with that obtained by maximal exercise.

In addition to these intentional eccentric muscle actions, sports or daily activities are comprised of involuntary eccentric muscle actions in many phases (1). We are not usually conscious of performing eccentric actions in running or throwing; however, eccentric actions are repeatedly performed when absorbing shocks and during deceleration (1), or in stretch-shortening cycle (SSC) movements (5,16). It has been reported that muscle soreness develops during and after endurance events such as marathon running (2,3,5). Endurance exercises also induced histological damage (12,14,36), increases in muscle proteins in the blood (12,17,24,34), and abnormalities in magnetic resonance images (8). These symptoms are similar to those observed after maximal eccentric exercise (6,27–29); however, it is not clear whether the causes of the symptoms are the same between maximal eccentric exercise and endurance exercises.

Many studies have examined changes in these indicators of muscle damage after endurance exercise with a strong eccentric component such as downhill walking (22,30), downhill running (31), shuttle running (34), cycling exercise (10), and stepping (21,23) or sitting (33) exercises. It is generally accepted that eccentric muscle actions are responsible for DOMS and/or muscle damage in these exercises (3); however, a large number of repeated eccentric muscle actions are necessary to induce muscle damage in these exercises. It seems reasonable to assume that mechanism of muscle damage induced by a small number of maximal eccentric muscle actions is different from that by a large number of muscle contractions containing involuntary eccentric muscle actions. In fact, the magnitude and the time...
course of changes in markers of muscle damage such as plasma creatine kinase (CK) activity is different between downhill walking or running and maximal eccentric exercise of the elbow flexors (6,27,29). It has been reported that large (>10,000 IU·L⁻¹) increases in plasma CK activity were observed 72–120 h after maximal eccentric exercise of the elbow flexors (27,28); however, downhill walking (22,30) or running (31) showed an earlier (0–12 h after exercise) and smaller (<1000 IU·L⁻¹) peak.

The comparisons between maximal eccentric exercise and endurance types of exercise should be done with caution, because the studies used different muscle groups. It could be that leg muscles respond differently than arm muscles to eccentric exercise. No study has compared the two types of exercise using the same muscle groups with similar movements. To shed light on the mechanisms underlying muscle soreness and damage after exercise, it is important to understand the differences in the time course of changes in indirect markers of muscle damage and the magnitude of the changes between maximal eccentric muscle actions and endurance exercise in which a large number of involuntary eccentric actions are performed.

This study investigated the differences in markers of muscle damage after a typical muscle damage-inducing protocol versus an exercise bout more typical of everyday muscle actions. The present study developed an endurance exercise model of the elbow flexors and compared the changes in several markers of muscle damage with those after maximal eccentric exercise of the same muscles. It was hypothesized that the magnitude and time course of muscle damage and recovery after exercise would be different between the exercises, with the intensity of eccentric load being the primary factor determining the magnitude of muscle damage.

METHODS

Approach to the Problem and Experimental Design

There is an abundance of data available for the changes in indirect markers of muscle damage after eccentric exercise of the elbow flexors. Thus, the present study developed an endurance exercise model employing the elbow flexors. For developing the model of endurance exercise, it was considered that 1) the elbow flexors were the prime mover of movement, 2) the movement was simple and rhythmical, 3) all subjects could continue the exercise for at least 1 h, 4) the exercise contains a large number of eccentric muscle actions, and 5) muscle fatigue is evident at the end of exercise. From our pilot testing, an endurance exercise of the elbow flexors to fulfill the aforementioned conditions was established. For comparison with this exercise, the number of eccentric actions and the duration of the exercise time were minimized for the maximal eccentric exercise of the elbow flexors. Prolonged loss in muscle force, range of motion of the joint, swelling, increased muscle proteins in the blood, muscle soreness, and abnormalities on magnetic resonance or ultrasound images have been used to indirectly evaluate the magnitude of muscle damage induced by maximal eccentric exercise of the elbow flexors (6,27,28,29). The present study compared the changes in these indirect markers of muscle damage between the endurance exercise of the elbow flexors and maximal eccentric exercise of the same muscles.

Subjects

Subjects comprised 18 male students who were nonathletes and had not been involved in a resistance-training program for at least 1 yr before the study. The number of subjects was considered to be adequate for statistical power. Their mean ± SD (range) age, height, and body mass was 20.4 ± 2.0 (18–25) yr, 172.7 ± 5.7 (160–182) cm, and 61.7 ± 6.8 (48–74) kg, respectively. Before involvement in the study, subjects were informed of the experimental procedures, which were in accordance with the ethical standards of the American College of Sports Medicine and required to complete a written informed consent document. Subjects were requested to avoid any vigorous physical activities or unaccustomed exercises, other than that required for the study, during the experimental periods. All subjects were free from any musculoskeletal disorders and were instructed not to take any medicine or dietary supplements during the experimental periods. No restrictions of diet, other than the supplementation, were imposed. Subjects were allowed to take their preferred soft drinks such as sports drink, juice, or carbonated drinks during the prolonged exercise ad libitum, and the amounts of fluid volume and energy intake from the drinks were recorded. All subjects had a meal (1500–3000 kJ, 360–720 kcal) within 2 h after exercise.

A comparison group was provided from our previous study (unpublished) in which 50 students performed 12 maximal eccentric actions of the elbow flexors. It was shown that the sample size (N = 50) was large enough to make comparisons with the group of subjects who performed the 2-h arm-curl exercise in statistical analyses. Subjects in the comparison group were recruited from the same large group of students as the present study by employing the same selection criteria. Their mean ± SD (range) age, height, and body mass was 20.3 ± 2.4 (18–32) yr, 171.2 ± 4.5 (163–182) cm, and 61.4 ± 5.4 (53–78) kg, respectively, and these values were not significantly different from those in the present study. During the experimental period, subjects were given the same instructions as those provided to the subjects who performed the prolonged exercise.

Exercise

Endurance exercise (2-h Ex). Subjects performed 2 h of arm-curl exercise (2-h Ex) with their nondominant arm on an arm curl bench by using a light dumbbell (1.1–1.8 kg) that was set at 9% of their maximal isometric force determined at an elbow joint of 90° (1.57 rad). This load was similar to approximately 10% of 1RM concentric movement of the elbow joint from an extended position (180°, 3.14 rad).
to a flexed position (60°, 1.05 rad). Subjects sat on a bench, the arm was positioned in front of the body on a padded support adjusted to 45° (0.79 rad) of shoulder flexion, and the forearm was kept supinated during exercise. In this exercise, subjects were asked to flex and extend the elbow rhythmically at 30 cycles per minute for 3600 cycles with the tempo of metronome. The range of motion was approximately 120° (2.09 rad) from a flexed (90°, 1.57 rad) to an extended elbow position (180°, 3.14 rad). Therefore, the movement distance in a cycle was approximately 0.6 m (forearm length: 0.3 m, ROM: 120°; 0.6 × 3.14 × 0.33 = 0.62 m). During the elbow-extending actions, subjects were asked to use the triceps brachii muscle as little as possible and to lower the dumbbell in a slow, controlled manner. If the subjects were unable to maintain the cycle rate of the actions, the load was gradually reduced. Subjects performed the exercise in the morning between the hours of 07:00 and 11:00 a.m. Although the circadian rhythm of cortisol concentration may affect the extent of muscle damage (7), the time frame (07:00–11:00 a.m.) was considered to be adequate to minimize any possible influence of cortisol.

Maximal eccentric exercise (Max-ECC). The comparison group performed 12 maximal eccentric actions of the elbow flexors (Max-ECC) where the elbow joint was forcibly extended from a flexed (90°, 1.57 rad) to an extended elbow position (180°, 3.14 rad) in 3 s after 1 s of maximal isometric contraction (29). Subjects sat on a bench, the arm was positioned in front of the body on a padded support adjusted to 45° (0.79 rad) of shoulder flexion, and the forearm was kept supinated with the wrist placed against a lever arm connected to the bench (29). Subjects were verbally encouraged to generate maximal isometric force at the flexed position and to maximally resist when the lever arm extended the elbow joint. The action was repeated every 15 s for 12 contractions with the total exercise time approximately 3 min. This 12-action protocol was chosen to minimize fatigue. Force generated during eccentric phase of motion was measured by a load transducer (9E01-L43, NEC San-ei Co., Tokyo, Japan) installed in a specially designed wrist attachment and monitored and recorded by a digital indicator (F360A, UNIPULSE, Saitama, Japan) and a computer (Macintosh Performer 5410, Cupertino, CA). The peak and average force during each eccentric action was recorded from the digital indicator at a sampling rate of 100 Hz, and the work during each eccentric action was estimated by the average force for 3 s and the distance of the movement (forearm length: 0.3 m, ROM: 90°; 0.6 × 3.14 × 0.25 = 0.47 m).

Criterion Measures

Several indirect markers of muscle damage, such as maximal isometric force (MIF), relaxed (RANG) and flexed elbow joint angles (FANG), upper-arm circumference (CIR), muscle soreness, and B-mode ultrasound images (US) for echo-intensity and muscle thickness, that have been used in previous studies (6,27–29) were assessed before (pre), immediately after (post), and 24, 48, 72, and 96 h after both exercises. For 2-h Ex, measurements were also taken 1, 3, 6, and 10 h after exercise. Blood samples were taken from the antecubital vein at the same time points, and plasma CK activity was determined. Blood lactate concentration was determined before, immediately after, and 1 h after the exercise.

Maximal isometric force. MIF was measured twice (1 min between the measurements) at an elbow joint of 90° (1.57 rad) for 3 s, by way of a transducer (Model 100, Takei Scientific Instrument Co. Ltd., Tokyo, Japan) connected to a digital recorder. The peak force of each 3-s value was determined, and the mean of the two measurements was used for analysis.

Relaxed and flexed elbow joint angles. RANG and FANG were each measured twice by a goniometer, and the mean of the two values was used for analysis. Subjects were instructed to hold the arm by their side in a relaxed manner for the RANG measurement, and FANG was determined when subjects maximally flexed the elbow joint voluntarily.

Upper-arm circumference. CIR was assessed at 3, 5, 7, 9, and 11 cm from the elbow joint by a tape measure while letting the arm hang down by the side, and the mean value of the five measurements was used for analysis.

Muscle soreness. Muscle soreness during palpation on the upper-arm (SOR-pal) as well as flexion (SOR-flx) and extension of the forearm (SOR-ext) were evaluated by a visual analog scale (VAS) that had a 50-mm line with “no pain” on one end and “extremely sore” on the other.

Plasma CK activity and lactate concentration. Approximately 5 mL of blood was drawn from the antecubital vein by a standard venipuncture technique using heparin lithium coated tubes. After obtaining three 20-µL blood samples for lactate analysis, blood was centrifuged for 10 min to obtain plasma. The plasma samples were stored at −20°C until being analyzed for CK. Plasma CK activity was determined spectrophotometrically by the VP-Super (Dinabot Co., Ltd., Tokyo, Japan) using a test kit (Dinabot Co. Ltd.). The normal reference ranges for male adults by this method are 45–135 IU·L⁻¹. Blood lactate concentration was determined by a Biosen 5010 (EKF Industrie, Elektronik GmbH, Barleben, Germany). Inter- and intrainjectivity were 2.4% and 2.8%, respectively for CK, and 1.7%, and 1.8%, respectively for lactate. Minimum sensitivity for the measurement was 20 IU·L⁻¹ for CK and 0.2 mM·L⁻¹ for lactate according to the method sheet provided by the manufacture.

Ultrasound images. B-mode ultrasound images of the elbow flexors were taken from the mid-belly of the biceps brachii at the same sites as the CIR measurements by using an SSD-500 (Aloka Co. Ltd., Tokyo, Japan) with a 7.5-MHz linear probe. To obtain the ultrasound images, the examiner placed the probe on the marked site on the upper arm while subjects were sitting on a chair with the forearm on an armrest and found the transverse images by using same references as that of the preexercise image. Echo intensity of the biceps brachii and brachialis was assessed by software (Mac SCOPE, Mitani Co., Fukui, Japan).
Statistical Analyses

Changes in the criterion measures over time were analyzed by a one-way ANOVA with repeated measures for 2-h Ex and Max-ECC, separately. When the ANOVA found a significant main effect, Tukey’s post hoc test was used to detect the differences between each time point. Comparisons between 2-h ECC and Max-ECC for each matched time point (pre, post, 24, 48, 72, and 96 h) were analyzed by a one-way ANOVA with repeated measures. The significant level was set at $P < 0.05$. The values shown are means ± SEM.

RESULTS

Endurance Exercise

Reduction of the load had to be made during the exercise for all subjects, and the dumbbell weight was 0.2–0.8 kg lighter at the end compared with the beginning of exercise. The amount and the timing of reduction varied among subjects; however, all subjects completed at least one-fourth of the exercise (30 min) without changing the load. All subjects completed the 2-h Ex and performed 3600 flexion/extension actions. The total amount of work of the elbow flexors was estimated roughly 60,000 Nm (average force: 14 N, distance: 0.6 m, concentric and eccentric actions: 7200; $14 \times 0.6 \times 7200 = 60,480$) for the 2-h exercise and 1000 Nm (average force: 150 N, distance: 0.6 m, eccentric actions: 12; $150 \times 0.5 \times 12 = 900$) for the Max-ECC.

Maximal Isometric Force (MIF)

There was no significant difference in preexercise MIF between 2-h Ex (187.1 ± 4.3 N) and Max-ECC (191.1 ± 8.4 N). As shown in Figure 1, MIF changed significantly ($P < 0.05$) after both exercises. MIF dropped to 44.1 ± 5.0% of the preexercise value immediately after 2-h Ex, recovered to 79.8 ± 3.4% at 24 h, and approximated the preexercise level (97.8 ± 1.9%) by 96 h postexercise. The decline in MIF immediately postexercise was significantly smaller for Max-ECC (58.1 ± 1.7%); however, the recovery over the next 4 d was minimal resulting in values of 60.7 ± 1.8% at 24 h and 73.1 ± 2.4% at 96 h, which were significantly ($P < 0.05$) smaller than 2-h Ex.

Relaxed Elbow Joint Angle (RANG)

Significant ($P < 0.05$) changes in RANG were found after both exercises as shown in Figure 2. Small decreases in RANG were observed immediately after 2-h Ex (approximately 5°); however, no further decrease was seen after this time point. Max-ECC showed a significantly ($P < 0.05$) larger decrease in RANG immediately after exercise (approximately 13°) and decreased further over the ensuing days, resulting in more than 15° decrease 48–72 h after exercise (Fig. 2).

Flexed Elbow Joint Angle (FANG)

Both exercises resulted in significant ($P > 0.01$) changes in FANG as shown in Figure 3. The initial change in FANG was significantly ($P < 0.01$) larger for 2-h Ex (18°) compared with Max-ECC (11°); however, the recovery was significantly ($P < 0.01$) faster for 2-h Ex compared with Max-ECC (Fig. 3).

Upper-Arm Circumference (CIR)

Although CIR increased significantly ($P < 0.05$) after 2-h Ex, the amount of increase was significantly ($P < 0.05$) smaller for 2-h Ex (3 mm) compared with Max-ECC (12 mm) (Fig. 4).

Muscle Soreness

As shown in Figure 5, SOR-pal developed immediately after exercise, peaked 24 h postexercise, and decreased...
rapidly after 2-h Ex. After Max-ECC, no soreness was evident immediately after exercise but developed at 24 h, peaked at 48 h, and still remained at 96 h after exercise. There was no significant difference in the peak SOR-pal value between 2-h Ex and Max-ECC. SOR-ext also developed immediately after, and peaked 24 h after 2-h Ex; however, the magnitude of extension soreness was significantly \( (P < 0.05) \) smaller than that after Max-ECC.

**Plasma CK Activity and Blood Lactate**

Although plasma CK activity increased significantly \( (P < 0.05) \) after 2-h Ex (peak: 356 IU·L\(^{-1}\)), the increases were significantly \( (P < 0.01) \) smaller compared with Max-ECC (peak: 5549 IU·L\(^{-1}\)). The peak value was observed at 96 h postexercise for both exercises.

**DISCUSSION**

Both 2-h Ex and Max-ECC resulted in significant changes in all criterion measures after exercise (Figs. 1–6). Although changes in MIF and FANG immediately after exercise were significantly larger after 2-h Ex compared with Max-ECC, the recovery was significantly faster after 2-h Ex than Max-ECC (Figs. 1 and 3). Changes in RANG...
It has been documented that specific tension in the active muscle fibers is greater during eccentric actions than concentric muscle actions (3,4,7,9). Armstrong (3) proposed that muscle damage in endurance events was associated with both “metabolic overload” and “mechanical strains,” but high local tensions in fibers during eccentric muscle actions might be more important than metabolic considerations in the etiology of the damage. Although a light dumbbell (9% of MIF) was used for the 2-h Ex in the present study, and some reduction of the load had to be made during exercise, the relative load of the dumbbell would have gradually increased as MIF decreased due to the effects of fatigue, failure to maintain the required or expected power output. However, the muscle forces generated during the dumbbell exercise would still have been lower than that during Max-ECC. Despite the fact that the duration of Max-ECC was less than 3 min, and the number of muscle actions and the total work was only one-300th and one-60th, respectively, of the 2-h Ex, 12 maximal eccentric actions induced significantly larger and longer-lasting damage than 2-h Ex (Figs. 1–6). The results of present study support the proposition that mechanical factors rather than metabolic factors are responsible for determining the magnitude of eccentric exercise-induced muscle damage (4,7,9,23).

It should be noted that the decrease in MIF immediately after exercise was larger for 2-h Ex (44% of the preexercise level) compared with Max-ECC but recovered to 80% of the preexercise level by 24-h after exercise and returned to the preexercise level by 4 d after exercise (Fig. 1). It seems reasonable to assume that the force loss immediately after exercise was caused by two components, fatigue and damage. Westerblad et al. (38) suggested that a reduction of sarcoplasmic reticulum (SR) Ca$^{2+}$ release was associated with reduction of isometric force in the late fatigue phase and that this reduction of Ca$^{2+}$ release correlated with a decline of ATP. Fatigue during repetitive low-intensity muscle actions has been shown to be associated with glycogen depletion (35). However, this was unlikely for 2-h Ex, because carbohydrate ingestion occurred during the exercise with fluid intake, and the exercise did not seem to be intense enough to deplete muscle glycogen. It was also unlikely that muscles became ischemic and led to disruption of normal steady cellular respiration during 2-h Ex, as indicated by only modest increases in blood lactate. Metabolic byproducts such as ADP, AMP, IMP, P$_i$, H$^+$, and NH$_3$ could potentially result in a feedback inhibition of metabolic pathways involved in ATP regeneration or by direct inhibition of the excitation or contraction process (13). It is possible that larger increases in the metabolic by-products were attributed to the larger decrease in MIF immediately after 2-h Ex compared with Max-ECC.

In contrast, the long-lasting decrease in MIF after Max-ECC (Fig. 1) was more likely to have been the result of E-C coupling failure as a result of disruption to the contractile elements (15). It has been reported that eccentric exercise induces damage to sarcotubular system, cytoskeletons, and contractile proteins (9,15). Ingalls et al. (15) have reported that the prolonged loss of MIF after eccentric exercise is caused mostly by E-C coupling failure for the first 3 d after exercise followed by disruption of contractile elements for the long lasting force deficit. It seems that E-C coupling failure was also associated with the acute large decreases in MIF after 2-h Ex (Fig. 1), but other factors such as reduced Na$^+$/K$^+$-pump function (25) might also be involved. It has been hypothesized that loss of Ca$^{2+}$ homeostasis is a key event leading to myofibrillar and membrane degradation (4,7,9). The rapid recovery of MIF in the first 24 h after 2-h Ex (Fig. 1) and smaller increases in plasma CK activity (Fig. 6) would suggest that structural damage to contractile elements was minimal, and membrane damage was not fatal for the affected muscle fibers to become degenerated (4).

Muscle soreness after 2-h Ex was apparent immediately after exercise (Fig. 5), this being a rare observation immediately after Max-ECC (6,27,32). After maximal eccentric exercise or resistance training containing eccentric loading, soreness tends to have an onset delay of some 8–12 h, and peaks 1–3 d after exercise (6,7). On the other hand, it is known that muscle soreness often starts to develop during endurance events such as marathon running and peaks 1–3 d later (8). It may be that the cause(s) of soreness experienced in the first 6–10 h after 2-h Ex differ(s) from that developed 24–72 h after exercise. Factors related to repetitive muscle contractions or changes caused by exercise such as accumulation of metabolites or increased intramuscular pressure may be associated with the soreness in the early phase after exercise (2,34). Soreness in the later stage may be more closely associated with factors related to muscle and/or connective tissue damage and inflammatory responses (3,7).

It is also important to note that the time course of changes in plasma CK activity after 2-h ECC was similar to Max-ECC, although the magnitude of increase after 2-h Ex was significantly smaller than that of Max-ECC (Fig. 6). It has been reported that plasma CK activity increases immediately after and peaks 12–24 h after a marathon run (12,17,26), downhill running (31), or shuttle running (34). Because of the endurance-like nature of the 2-h Ex, it was hypothesized that there would be an immediate increase in plasma CK activity after exercise. However, no significant increases in CK were found for the first 48 h after 2-h Ex, and small increases occurred 3–4 d after exercise (Fig. 6). The smaller increase in CK is consistent with the notion of minimal muscle damage induced by 2-h Ex. Nosaka and Newton (28) showed that increase in CK after eccentric exercise using a submaximal load of 50% of MIF was approximately one-sixth of Max-ECC. The amount of
increase in CK after 2-h Ex was much smaller than that observed after eccentric exercise using 50% MIF. Therefore, it appears that membrane damage or degeneration of muscle fibers was modest in 2-h Ex.

Although the magnitude of muscle damage was less with 2-h Ex compared with Max-ECC, it seems likely that eccentric muscle actions were still responsible for the muscle damage induced in both exercises. Because the relative load progressively increased during 2-h Ex as a result of fatigue, it is possible that the concentric component of the exercise contributed to the postexercise soreness and weakness. No subjects had problems flexing and extending the elbow joint at least for the first 30 min in the 2-h Ex; however, subjects had difficulty in lifting the dumbbell and controlling the slow elbow-extending motions toward the end of 2-h Ex. According to the material fatigue theory, the relationship between stress and the number of cycles to failure is exponential for most ductile materials, and as stress increases, the number of cycles to failure decreases (4). This theory can be applied to the 2-h Ex model in the present study. Mair et al. (18) showed that fatigued muscles absorbed less energy applied to the 2-h Ex model in the present study. Mair et al. number of cycles to failure decreases (4). This theory can be applied to the 2-h Ex model in the present study. Mair et al. (18) showed that fatigued muscles absorbed less energy before reaching the degree of stretch that causes injuries. It seems possible that, at a certain point during 2-h Ex, cumulative stress caused by the repetitive muscle actions exceeds the threshold where muscles can tolerate eccentric loading.

It has been reported that slowing of relaxation occurs with muscle fatigue (37). Increases in resting muscle [Ca\(^{2+}\)] associated with fatigue increases resting tension, and the slowing of myoplasm [Ca\(^{2+}\)] uptake during relaxation will further increase muscle stiffness (38). McHugh et al. (19) reported that more severe symptoms of muscle damage were observed in subjects who had stiffer hamstring muscles. They proposed that the strain imposed by active lengthening of stiff muscles resulted in myofibrillar strain. Stauber (32) stated that inability of SR to relax a muscle at the appropriate speed could result in a rigor-type state, which would increase a shear force between adjacent units producing rupture and myofibrillament damage. If such is the case, muscle fibers with increased stiffness have higher susceptibility to eccentric exercise-induced muscle damage, and this might have happened during the 2-h Ex. It may be also possible that repetitive muscle actions cause decreases in muscle coordination and sarcomere uniformity. There is some evidence that when sarcomere length within muscle fibers loses uniformity because of incomplete recovery of the interdigitating pattern of the filaments after lengthening actions, some sarcomeres “pop” to the point where there is no overlap of thick and thin filament arrays (20). It seems reasonable to assume that a greater incidence of sarcomere inhomogeneity will occur during prolonged exercise. Further study is needed to confirm these possibilities.

In conclusion, the results of this study showed that the extent of muscle damage induced by 2-h Ex was less than that by Max-ECC. It seems that the eccentric component is associated with muscle damage in both exercise models; however, it appears that fatigue also played a role in the damage process during 2-h Ex. It seems likely that the eccentric loading increased with repetitive muscle actions; however, mechanism(s) linking fatigue with muscle damage warrants further study.

REFERENCES


