Effects of warm-up before eccentric exercise on indirect markers of muscle damage

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ABSTRACT

EVANS, R. K., K. L. KNIGHT, D. O. DRAPER, and A. C. PARCELL. Effects of warm-up before eccentric exercise on indirect markers of muscle damage. *Med. Sci. Sports Exerc.*, Vol. 34, No. 12, pp. 1892–1899, 2002. **Purpose:** To test whether active and passive warm-up conducted before eccentric exercise attenuates clinical markers of muscle damage. **Methods:** Untrained subjects were exposed to one of five conditions: low-heat passive warm-up ($N = 10$), high-heat passive warm-up ($N = 4$), or active warm-up ($N = 9$), preceding eccentric exercise; eccentric exercise without warm-up ($N = 10$); or high-heat passive warm-up without eccentric exercise ($N = 10$). Passive warm-up of the elbow flexors was achieved using pulsed short-wave diathermy, and active warm-up was achieved by concentric contraction. Creatine kinase (CK) activity, strength, range of motion, swelling, and muscle soreness were observed before treatment (baseline) and 24, 48, 72, and 168 h after treatment. **Results:** High-heat passive warm-up without eccentric exercise did not affect any marker of muscle damage and was used as our control group. Markers of muscle damage were not different between groups that did or did not conduct warm-up before eccentric exercise. The active warm-up and eccentric groups exhibited a greater circumferential increase than controls ($P < 0.0002$), however, that was not observed after passive warm-up. Additionally, the active warm-up group exhibited a greater CK response than controls at 72 h ($P < 0.05$). The high-heat passive warm-up before eccentric exercise group exhibited significant change from controls at the least number of time points, but due to a small sample size ($N = 4$), these data should be viewed as preliminary. **Conclusion:** Our observations suggest that passive warm-up performed before eccentric exercise may be more beneficial than active warm-up or no warm-up in attenuating swelling but does not prevent, attenuate, or resolve more quickly the other clinical symptoms of eccentric muscle damage as produced in this study. **Key Words:** ACTIVE WARM-UP, PASSIVE WARM-UP, DIATHERMY, MUSCLE TEMPERATURE, MUSCLE SORENESS

Stretch-induced injury to muscle, or muscle strain, is one of the most common injuries experienced by a recreationally active population (19). Muscle strain can occur when muscle is stretched while in a passive state, or when the muscle is actively contracting (7). The latter is a lengthening, or eccentric contraction, and has been well established as the most damaging type of muscle action (2,20).

During unaccustomed eccentric exercise, damage results when strain exceeds the limits of the connections between noncontractile elements in the cytoskeletal and extracellular matrices, such as collagen and other structural proteins (18,29). This structural damage results in microscopic disruption of muscle fiber elements (3,14,17,20), the extracellular connective tissue matrix (2,9,29), or a combination of these structures and initiates a series of reactions that lead to loss of cellular integrity, disrupted calcium homeostasis, and cell necrosis (1). Ultimately, this process results in clinical manifestations of damage that include high plasma creatine kinase (CK) activity, strength loss, range of motion loss, swelling, and delayed onset muscle soreness (27). Although most individuals have experienced this type of injury, there is no evidence that any specific intervention consistently prevents, attenuates, or treats the clinical symptoms of eccentric strain injury.

Warm-up of muscle is routinely recommended as a way to prevent strain injury (26,28), despite a lack of scientific evidence to support the clinical efficacy of warm-up in injury prevention. There is, however, evidence to suggest that elevating muscle temperature might decrease strain injury through changes in muscle tissue extensibility, by allowing increased stretch to occur before the onset of damage in both tendon (13,31) and muscle tissue (21,26,30).

Increased muscle temperature can be achieved actively, through exercise, or passively, through the use of heat modalities. Temperature elevations observed after 15 s to 15 min of active muscle contraction range from 1.0 to 3.3°C (24,26,32). Modalities used in sports medicine clinics to passively raise tissue temperature include ultrasound, short-wave diathermy, and warm-water immersion, all of which have shown to raise deep muscle temperature between 3.4 and 3.8°C after 10- to 30-min treatments (4–6). It is unclear, however, what degree of temperature elevation is necessary to increase tissue extensibility to levels that may prevent injury. An increase in muscle extensibility has been observed after a 1°C elevation (26) and in tendon after a 3–4°C elevation (12). Other researchers have recommended that

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tissue reach a specific temperature for extensibility to occur (13,32). Most studies have looked at the degree of extensibility that occurs prior to tissue rupture, however, rather than onset of strain injury.

Recently, Nosaka and Clarkson (22) reported that an active warm-up of 100 concentric contractions performed just before an eccentric exercise bout decreased indicators of muscle damage. They concluded that exercise before the eccentric bout may have warmed up the muscle, minimizing damage. We are not aware of any studies that have assessed the effects of passive warm-up before eccentric exercise. In addition, studies are lacking that have compared the effects of passive and active warm-up and their role in preventing strain injury to muscle.

This study was designed, therefore, to test the effects of warm-up on muscle damage resulting from eccentric exercise and to compare the effects of passive versus active warm-up. Additionally, we explored the effects of a high-heat passive warm-up before eccentric exercise. We hypothesized that warm-up before eccentric exercise would attenuate indirect markers of eccentric muscle damage and that active and passive warm-up methods designed to elevate muscle temperature by similar levels would have similar effects. We also hypothesized that passive warm-up of 3–4°C would be more effective than passive warm-up of 1°C in attenuating damage.

METHODS

Experimental Design

A 5 × 5 factorial with repeated measures on one factor guided this study. The two independent variables were time (repeated measures at baseline, 24, 48, 72, and 168 h) and treatment group, the latter of which included high-heat passive warm-up without eccentric exercise (CONTROL), eccentric exercise without warm-up (ECC), low-heat passive warm-up preceding eccentric exercise (PW−LO+ECC), high-heat passive warm-up preceding eccentric exercise (PW−HI+ECC), or active warm-up preceding eccentric exercise (AW−LO+ECC). Pilot work determined that passive warm-up, achieved by applying pulsed short-wave diathermy to the elbow flexors, resulted in temperature elevation of the biceps brachii by approximately 1°C (PW−LO) or 3.5°C (PW−HI), depending on the setting used. Active warm-up was achieved by performing 100 concentric contractions of the elbow flexors and was found to elevate temperature of the biceps brachii by approximately 1°C (AW−LO). Treatment effects were assessed by observing changes in CK activity, isometric strength (MVC), resting elbow extension, active elbow flexion, circumference at the biceps muscle belly and distal biceps tendon region, and muscle soreness with extension and flexion.

Subjects

Forty-three healthy male (N = 16) and female (N = 27) college students volunteered to participate in this study (age 22.4 ± 5.9 yr). Subjects were excluded if they had experienced recent illness, were on medications that might interfere with study results, or had participated in any resistance-training program during the 3-month period before the study. Written informed consent was obtained from all subjects in accordance with the Institutional Review Board for Research with Human Participants. Volunteers agreed to refrain from any new or strenuous physical activity while participating in the study.

Thirty-nine subjects were randomly assigned to one of four treatment groups (CONTROL (N = 10; 3 male, 7 female), ECC (N = 10; 2 male, 8 female), PW−LO+ECC (N = 10; 4 male, 6 female)), or AW−LO+ECC (N = 9; 4 male, 5 female). Due to the remote possibility that elevated muscle temperature would significantly increase eccentric muscle damage (33), we limited our PW−HI+ECC treatment to a small group of four volunteers (3 male, 1 female). Each subject reported to the research lab on six separate days. On day 1, subjects were familiarized with the study methods, and baseline measurements of the dependent variables were obtained. On day 2, subjects were treated with their assigned experimental condition. Subjects then reported to the lab at 24, 48, 72, and 168 h (days 3–6) after treatment, where postexercise measurements were taken, identical to those taken at baseline. The dominant arm was tested in all cases.

Experimental Procedure

Passive warm-up. Passive warm-up was achieved utilizing a Magnatherm SSP (1000 SS) pulsed short-wave diathermy unit (International Medical Electronics, Ltd., Kansas City, MO) with a 24-cm diameter induction applicator operating at 27.12 MHz. Heat delivery is controlled using 12 independently adjustable power (pulse train width) and rate (pulse·s^{-1}) settings, where 12 represents 100% of maximal capability, and 6 represents 50% of maximal capability. To passively heat the elbow flexors, the subject was placed supine, the shoulder abducted approximately 40°, and the forearm supported at the wrist to place the elbow in slight flexion. The applicator head was then positioned directly over the muscle belly of the biceps brachii. For the PW−LO+ECC condition, heat was applied to the muscle for 10 min at a power and rate setting of 5 and 5 (5/5), respectively. For the PW−HI+ECC group, heat was applied for a period of 10 min at a 12/12 setting. The high-heat passive warm-up alone group (CONTROL) was also treated for 10 min at a 12/12 setting. Pilot work conducted in our laboratory estimated that the temperature rise of the elbow flexor muscles after 10-min treatments at settings of 5/5 and 12/12, as determined by thermocouple needle microprobes placed in the biceps brachii to a depth of 2 cm, was approximately 1° and 3.5°C, respectively. These muscle temperature increases are similar to those found in a previous investigation assessing change in deep muscle temperature at a depth of 3 cm (5).

Active warm-up. The BIODEX dynamometer (Biodex Medical Systems, Shirley, NY) was used to actively

warm-up the elbow flexors, without inducing fatigue, using a previously established method (22). After being secured in the test position, the subject moved the elbow joint smoothly from an extended (170°) to a flexed (50°) position and back 100 times, completing each repetition in 2 s. The BIODEX was set at $120°\cdot s^{-1}$ to minimize the load and to ensure that fatigue-provoking forces were not generated. We determined that that this active warm-up protocol elevates muscle temperature of the elbow flexor muscle group, as determined by pilot work identical to that used to assess changes in deep muscle temperature of the biceps brachii after passive warm-up, by approximately 1°C.

Eccentric exercise. The passive mode of a computer-interfaced dynamometer (BIODEX System 3) was used to induce muscle damage (29), and was conducted immediately (within 10–15 s) after any warm-up procedure. Subjects were seated with the arm supported and were stabilized at the waist and chest. Starting with the elbow flexed to 50° and ending at an angle of 170°, each subject performed 50 maximal eccentric movements of the elbow flexors at $120°\cdot s^{-1}$. During each movement, subjects were verbally encouraged to produce a maximal effort to resist the ability of the dynamometer to extend the elbow. Subjects were given a 10-s rest between each contraction, during which time the dynamometer arm returned passively to the starting position.

Blood sampling and analysis. Approximately 5 mL of blood were taken from an antecubital vein using sterile venipuncture techniques. Blood was collected in EDTA-coated tubes (Becton Dickinson, Franklin Lakes, NJ) and immediately centrifuged for 10 min. Plasma samples were separated and stored at −80°C until analysis. Plasma CK activity was measured in duplicate using an enzymatic assay kit (Sigma 47UV, St. Louis, MO).

Strength. Maximal voluntary isometric contraction of the elbow flexors was measured using the BIODEX dynamometer. After seating the subject in the test position with the elbow flexed to an angle of 90°, two 3-s maximal isometric contractions of the elbow flexor muscle group were performed, with a 1-min rest between trials. The highest value of the two trials (in N·m) was used to represent MVC.

Range of motion. Measurements of elbow range of motion were taken using a 30-cm clear plastic goniometer (Bissell Healthcare Corp., Brookfield, IL). The anatomic reference points for goniometer placement were the deltoid insertion, the lateral humeral epicondyle, and the ulnar styloid process. These points were marked with semipermanent ink when baseline measurements were taken and were maintained during the study. The relaxed angle of the elbow joint reflects changes in muscle stiffness of the elbow flexors and was measured with the subject standing with the arm relaxed. Flexion was measured after asking the subject to bend the elbow as much as possible while reaching for the ipsilateral shoulder.

Circumference. Swelling was assessed by circumference measurements taken at the biceps tendon region and the biceps muscle belly (1 cm and 6 cm proximal to the humeral epicondyles, respectively) by using a standard tape measure. Measurement sites were kept consistent by repeatedly marking the arm with semipermanent ink. With the subject standing and the upper extremity in a neutral, relaxed position, three measurements were taken at each point and the average recorded as the score (in cm).

Muscle soreness. Subjects rated muscle soreness on a 100-mm visual analog scale (VAS), with the far-left end point representing no pain and the far-right end point representing very sore muscles. Standing with the upper limb relaxed and the forearm fully supinated, subjects actively extended the elbow. They then placed a mark on the VAS that represented the soreness experienced in the elbow flexor region of the arm during the motion. In the same manner, they rated the soreness experienced during full flexion of the elbow.

Statistical Analyses

The data were analyzed using SAS statistical software (Statistical Analysis System Institute, Inc., Cary, NC) to conduct a repeated measures analysis of variance (ANOVA) between the five treatment groups for each dependent variable at five time points. If significant main effects or interactions were found, Tukey *post hoc* tests were used to determine where differences existed. Statistical significance was set at $P < 0.05$.

RESULTS

There were no differences in baseline measurements between groups for any dependent variable (Table 1). High-heat passive warm-up without eccentric exercise (CONTROL) had no significant effect on any dependent variable at any time (Table 1) and was used as the control group. Although significant differences were observed in how the experimental groups differed from the control group, we observed no significant difference in markers of muscle damage between the groups that conducted warm-up before eccentric exercise and the eccentric exercise without warm-up group.

Plasma CK activity. Creatine kinase exhibited large intersubject variability (Table 1). For all groups combined, CK levels exceeded baseline values only at 72 h ($F_{4,152} = 7.86$, $P < 0.001$, Table 1), at which point values were higher than at 24, 48, and 168 h. There was no overall treatment effect ($F_{4,38} = 1.90$, $P = 0.13$); however, *post hoc* analysis of a significant time-by-treatment interaction ($F_{16,152} = 1.88$, $P = 0.03$) revealed that CK activity was greater in the AW−LO+ECC than in the CONTROL group at 72 h (Fig. 1).

Strength. Strength was analyzed as the percent change from baseline MVC to account for gender differences observed at baseline. Strength changed significantly over time ($F_{4,152} = 44.79$, $P < 0.001$) and was significantly less than control values at 24, 48, 72, and 168 h for all experimental groups (Table 1). Experimental groups were not different from each other but were collectively different than the control group ($F_{4,38} = 10.82$, $P < 0.001$) and exhibited a

TABLE 1. Measurement of indirect markers of muscle damage (mean ± SD).

	Baseline	24 h	48 h	72 h	168 h
Creatine kinase ($U \cdot L^{-1}$)					
CONTROL	86.0 ± 96.5	126.8 ± 185.3	100.2 ± 121.2	102.7 ± 108.9	63.1 ± 29.0
ECC	66.0 ± 60.4	124.9 ± 132.3	569.4 ± 1250.7	2109.1 ± 3331.3	1245.3 ± 1304.1
PW−LO+ECC	50.2 ± 37.1	116.6 ± 145.2	179.0 ± 215.6	970.9 ± 503.7	654.6 ± 607.6
PW−HI+ECC	89.5 ± 37.4	213.5 ± 86.2	449.7 ± 757.5	2096.7 ± 983.8	780.3 ± 1032.2
AW−LO+ECC	74.7 ± 100.9	415.8 ± 901.4	1262.2 ± 1822.1	5199.4 ± 8183.3	1074.2 ± 918.8
MVC (N·m)					
CONTROL	47.2 ± 20.1	48.7 ± 21.3	48.4 ± 21.3	47.9 ± 20.8	48.0 ± 21.2
ECC	41.2 ± 18.6	24.2 ± 11.9	24.5 ± 11.5	24.5 ± 12.3	30.2 ± 15.3
PW−LO+ECC	50.2 ± 17.1	32.0 ± 19.0	38.2 ± 24.2	37.7 ± 22.4	42.3 ± 20.9
PW−HI+ECC	58.9 ± 14.2	37.7 ± 17.1	37.7 ± 11.6	41.3 ± 19.1	47.1 ± 17.3
AW−LO+ECC	52.0 ± 25.4	33.4 ± 15.9	33.7 ± 13.0	33.4 ± 12.3	39.1 ± 18.0
Resting extension (deg)					
CONTROL	19.5 ± 6.6	19.1 ± 7.0	18.6 ± 6.4	19.3 ± 6.9	19.3 ± 7.4
ECC	21.3 ± 8.6	30.3 ± 7.0	34.1 ± 9.9	33.6 ± 10.8	25.0 ± 5.4
PW−LO+ECC	17.5 ± 8.5	23.5 ± 7.8	24.5 ± 8.0	25.7 ± 10.0	20.1 ± 7.2
PW−HI+ECC	21.8 ± 3.5	27.3 ± 4.0	27.8 ± 2.5	25.3 ± 1.9	22.8 ± 4.0
AW−LO+ECC	26.0 ± 4.8	35.1 ± 13.3	35.2 ± 14.0	33.3 ± 12.8	27.3 ± 10.0
Active flexion (deg)					
CONTROL	142.7 ± 5.2	143.3 ± 6.1	143.6 ± 7.2	143.7 ± 6.7	143.7 ± 5.5
ECC	145.5 ± 3.6	140.8 ± 4.1	141.5 ± 5.4	140.3 ± 4.9	143.9 ± 4.2
PW−LO+ECC	143.5 ± 8.4	135.4 ± 9.6	137.0 ± 8.1	136.2 ± 9.7	138.8 ± 7.7
PW−HI+ECC	144.8 ± 4.5	138.5 ± 12.2	140.3 ± 7.6	140.0 ± 12.1	143.5 ± 7.1
AW−LO+ECC	144.7 ± 3.9	140.2 ± 5.1	138.7 ± 6.6	139.6 ± 7.4	142.6 ± 5.0
Prox circumference (cm)					
CONTROL	29.1 ± 3.4	29.0 ± 3.5	29.0 ± 3.2	29.0 ± 3.3	29.1 ± 3.4
ECC	27.3 ± 2.3	27.8 ± 2.1	28.1 ± 2.4	28.4 ± 2.9	28.1 ± 2.5
PW−LO+ECC	27.3 ± 2.5	27.5 ± 2.4	27.7 ± 2.3	28.0 ± 2.2	27.8 ± 2.1
PW−HI+ECC	28.9 ± 4.4	29.5 ± 4.7	29.4 ± 4.8	30.0 ± 5.7	29.9 ± 5.5
AW−LO+ECC	27.9 ± 2.6	28.7 ± 2.7	28.8 ± 2.9	29.2 ± 3.0	28.9 ± 2.9
Distal circumference (cm)					
CONTROL	26.6 ± 2.5	26.5 ± 2.5	26.4 ± 2.5	26.5 ± 2.5	26.5 ± 2.6
ECC	25.1 ± 1.8	25.4 ± 1.7	25.6 ± 1.8	26.1 ± 2.4	25.9 ± 2.0
PW−LO+ECC	24.9 ± 2.0	25.0 ± 1.9	25.2 ± 1.8	25.5 ± 1.7	25.4 ± 1.5
PW−HI+ECC	25.9 ± 3.1	26.3 ± 3.0	26.2 ± 3.2	26.6 ± 4.1	26.8 ± 4.3
AW−LO+ECC	25.3 ± 2.7	25.7 ± 2.8	26.2 ± 3.2	26.6 ± 3.5	26.3 ± 3.0
Extension soreness (mm)					
CONTROL	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
ECC	0.0 ± 0.0	28.6 ± 13.4	44.0 ± 23.2	44.5 ± 32.1	1.4 ± 2.4
PW−LO+ECC	0.0 ± 0.0	35.3 ± 25.5	32.9 ± 22.4	41.2 ± 33.0	1.1 ± 1.9
PW−HI+ECC	0.0 ± 0.0	24.8 ± 3.7	31.8 ± 27.3	19.5 ± 25.9	3.5 ± 7.0
AW−LO+ECC	0.0 ± 0.0	40.1 ± 21.7	41.9 ± 22.0	28.3 ± 19.7	7.1 ± 13.5
Flexion soreness (mm)					
CONTROL	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
ECC	0.0 ± 0.0	31.6 ± 18.2	48.8 ± 21.4	44.4 ± 28.9	0.8 ± 1.6
PW−LO+ECC	0.0 ± 0.0	43.0 ± 32.0	44.1 ± 24.2	40.3 ± 30.2	0.5 ± 1.1
PW−HI+ECC	0.0 ± 0.0	19.8 ± 9.5	36.3 ± 15.2	21.5 ± 17.2	1.8 ± 3.5
AW−LO+ECC	0.0 ± 0.0	29.3 ± 23.4	38.8 ± 25.6	28.2 ± 17.9	1.3 ± 2.1

CONTROL, passive warm-up (high) without exercise; ECC, eccentric exercise without warm-up; PW−LO+ECC, passive warm-up (low) preceding eccentric exercise; PW−HI+ECC, passive warm-up (high) preceding eccentric exercise; AW−LO+ECC, active warm-up preceding eccentric exercise.

38.2% loss of strength at 24 h compared with baseline values ($P < 0.05$, Fig. 2). Significant strength loss was evident throughout the course of the study and, although significantly improved at 168 h when compared with the 24-, 48-, and 72-h measures ($P < 0.05$), was only 78 ± 16% of control values.

Range of motion. Resting extension and active flexion measurements are presented in Table 1. Extension and flexion loss was significant at 24, 48, and 72 h after eccentric exercise with or without warm-up ($F_{4,152} = 20.57$ and 19.33, $P \geq 0.0001$) and continued to be evident for flexion at 168 h. All groups conducting eccentric exercise lost a significant degree of extension when compared with controls ($F_{4,38} = 2.97$, $P < 0.03$), whereas only the PW−LO+ECC and AW−LO+ECC groups lost more flexion than controls ($F_{4,38} = 5.02$, $P = 0.002$). *Post hoc* analysis of significant time-by-treatment interactions ($F_{16,152} = 2.59$ and 2.55, $P = 0.001$) for both extension and flexion revealed significant differences from control values for all experimental groups at one or more time points. The ECC group exhibited significant extension loss at the greatest number of time points; the PW−LO+ECC exhibited significant flexion loss at the greatest number of time points; and the PW−HI+ECC group exhibited significant change at the least number of time points for both extension and flexion (Fig. 3).

Circumference. Circumferential measurements of both the proximal and distal arm are presented in Table 1. Due to significant differences in baseline girth measurements between male and female subjects, we analyzed the percent change in circumference compared to baseline. A significant increase over control values was observed beginning at 24 h proximally ($F_{4,152} = 27.79$, $P < 0.0001$) and 48 h distally ($F_{4,152} = 16.25$, $P < 0.0001$), and remained elevated at 168 h for both measures. Overall, the ECC and AW−LO+ECC groups exhibited more swelling than the

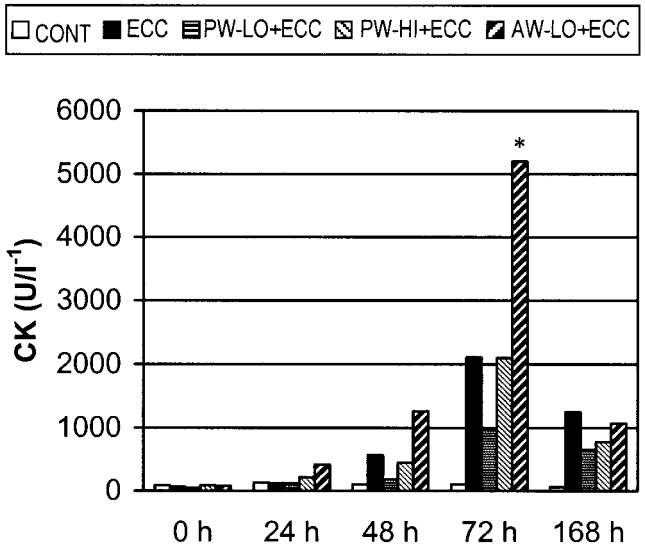

FIGURE 1—Creatine kinase activity: Mean CK activity for all treatment groups at baseline (0 h) and 24, 48, 72, and 168 h after treatment. *$P < 0.05$ compared with control group at same time point.

control group for both proximal ($F_{4,38} = 7.09$, $P < 0.0002$) and distal ($F_{4,38} = 4.12$, $P < 0.007$) measures. Analysis of a significant time-by-treatment interaction for both proximal ($F_{16,152} = 3.30$, $P < 0.0001$) and distal ($F_{16,152} = 1.96$, $P < 0.019$) measures revealed that all experimental groups exhibited a significant increase in circumference for at least one time point (Fig. 4). Additionally, a difference between experimental groups was observed at 24 h and 48 h, when the AW−LO+ECC group exhibited a greater increase in proximal swelling than the PW−LO+ECC group ($P < 0.05$).

Muscle soreness. Peak soreness was noted at 48 h for both extension and flexion, with no significant difference in perception of soreness between the two measures (Table 1). Soreness was elevated from baseline at 24, 48, and 72 h for both flexion ($F_{4,152} = 51.71$, $P < 0.001$) and extension ($F_{4,152} = 46.61$, $P < 0.0001$), and all treatment groups experienced significantly more soreness than the CONTROL group ($F_{4,38} = 10.33$ and 7.71, $P < 0.0001$ for flexion and extension, respectively). *Post hoc* analyses of significant time-by-treatment interactions for both flexion and extension ($F_{16,152} = 4.74$ and 4.72, $P < 0.0001$) showed that the high-heat passive warm-up before exercise group exhibited significant change at the least number of time points (Fig. 5).

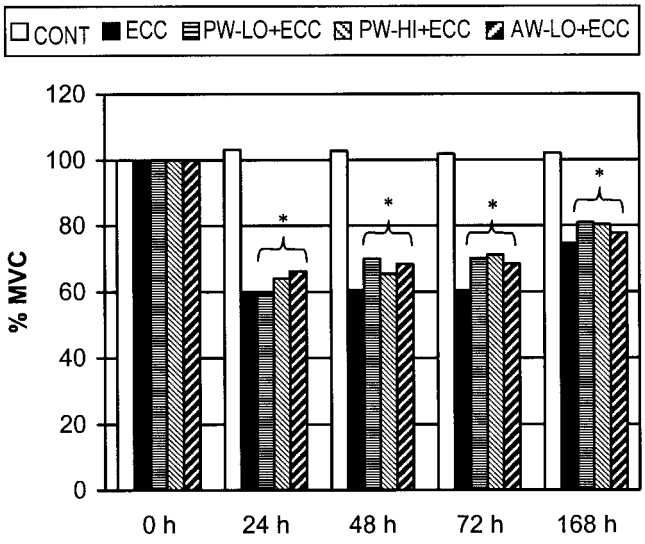

FIGURE 2—Percent of maximal voluntary contraction: Mean percent of MVC for all treatment groups at baseline (0 h) and 24, 48, 72, and 168 h after treatment. *$P < 0.05$ compared with control values.

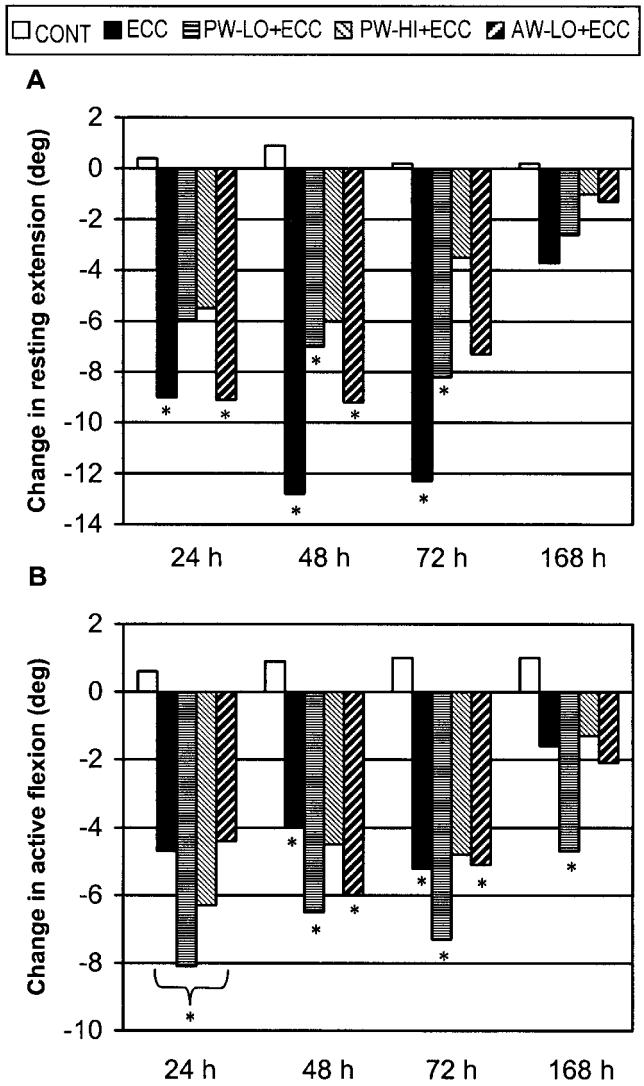

FIGURE 3—Changes in range of motion: Mean change in resting extension (A) and active flexion (B) compared with baseline for all treatment groups at 24, 48, 72, and 168 h after treatment. *$P < 0.05$ compared with control values.

DISCUSSION

It was our goal to assess the effects of warm-up on damage resulting from high-load eccentric muscle contraction. Although we observed differences with respect to how our experimental groups differed from control values, the evidence of this study does not support the hypothesis that warm-up immediately preceding eccentric exercise results

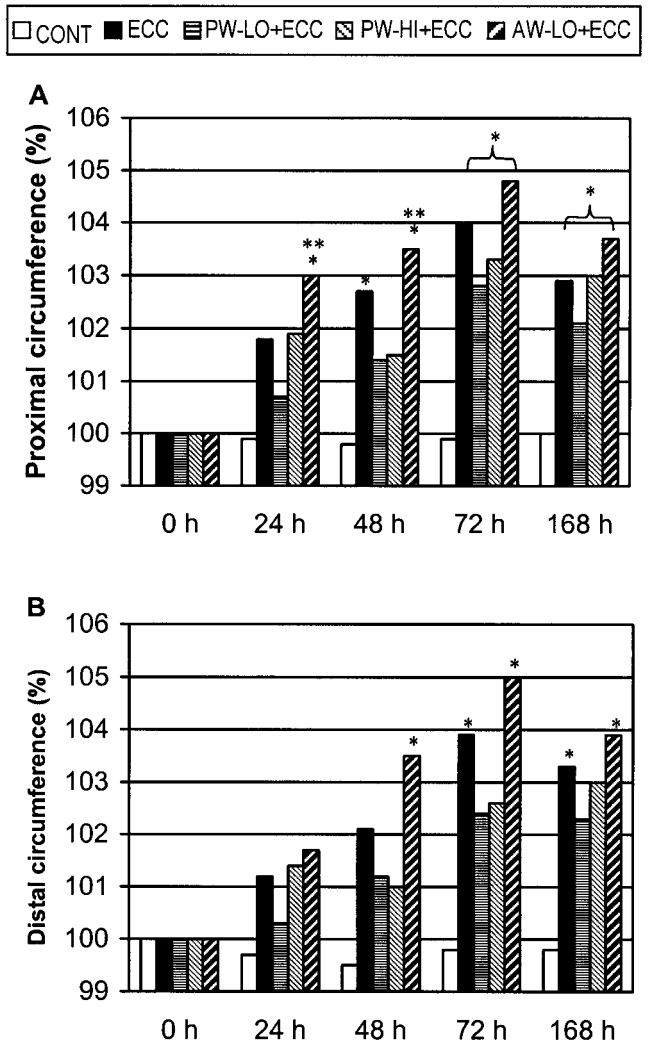

FIGURE 4—Arm circumference: Percent of proximal (A) and distal (B) arm circumference compared with baseline for all treatment groups at baseline (0 h) and 24, 48, 72, and 168 h after treatment. *$P < 0.05$ compared with control values, **$P < 0.05$ compared with PW−LO+ECC.

in fewer clinical signs of muscle damage than eccentric exercise alone.

It is well known that the response to eccentric exercise varies widely among individuals (27). Although CK activity is no exception and exhibits large intersubject variability, it is a sensitive marker of muscle damage (8). Mean CK values at 72 h for the AW−LO+ECC and ECC conditions in our study (5199.4 ± 8183.3 $IU \cdot L^{-1}$ and 2109.1 ± 3331.3 $IU \cdot L^{-1}$, respectively) were greater than the values observed by Nosaka and Clarkson at 72 h (22) (approximately 400 ± 200 $IU \cdot L^{-1}$ and 1800 ± 1400 $IU \cdot L^{-1}$), who tested the effects of active warm-up preceding eccentric exercise. Although this may represent increased damage, it may also reflect normal individual variability.

Significant decreases in force production resulted from our eccentric damage protocol, and the pattern of strength loss and recovery was similar to that described in other studies (22,25). Our warm-up conditions, however, had no significant effect on force production. All experimental groups were below control values throughout the course of the study, and a similar recovery pattern was evident between groups. Nosaka and Clarkson (22), using a similar active warm-up protocol, observed no significant differences in strength loss at 24 h for those conducting eccentric exercise with or without warm-up. Contrary to our results, however, they did note that strength recovery was significantly faster in the warm-up condition. Rodenburg et al. (25) reported no difference in strength loss or recovery when warm-up preceded eccentric exercise, but they combined warm-up with other treatments, making comparison with our study difficult.

We also observed no significant differences in range of motion, circumferential change, and subjective pain levels between those groups that conducted warm-up and those that did not. Again, Nosaka and Clarkson noted that pain decreased and range of motion was improved when active warm-up preceded eccentric exercise (22). They did, however, use a crossover design that allowed for comparison between like extremities, which may have created better control for variability in individual response, increasing their ability to see statistical differences resulting from treatment.

As our active warm-up was similar to that used by Nosaka and Clarkson (22), our differing results may also reflect the use of different damage protocols. In their study, subjects performed 12 low-velocity eccentric contractions ($40° \cdot s^{-1}$)

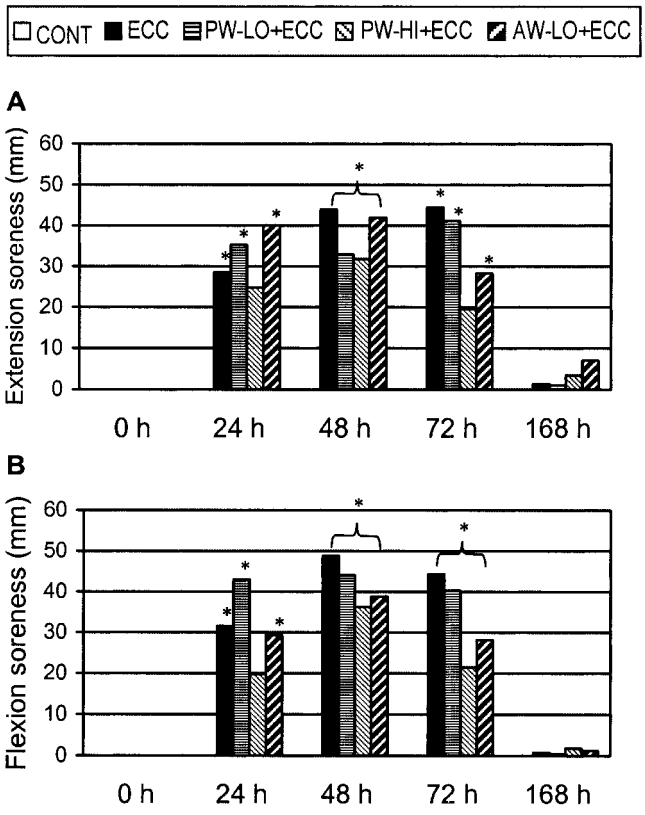

FIGURE 5—Muscle soreness: Changes in subjective muscle soreness during extension (A) and flexion (B) as noted on a 100-mm visual analog scale at baseline (0 h) and 24, 48, 72, and 168 h after treatment. * $P < 0.05$ compared with control values.

and exhibited an average force loss of 28% at 24 h. Our subjects performed 50 maximal effort high-velocity eccentric contractions ($120°\cdot s^{-1}$) and exhibited an average force loss of 39% at 24 h, similar to other studies using more severe damage protocols (23,25). As injury has been observed to increase with the duration and velocity of eccentric contraction (16), our protocol may have produced considerably more damage than the protocol used by Nosaka and Clarkson, and may have worsened the clinical indicators of muscle damage.

Further evidence that our protocol was too severe is the number of "high responders" we observed. High responders have been defined as individuals who present with pronounced swelling, extended decrements in muscle function, and greatly elevated CK levels after eccentric exercise (27). Sayers et al. (27) reported a 3% incidence of high responders (6/204) in subjects who performed 50 low-velocity eccentric contractions. Using the same loosely defined criteria, we observed a 14% incidence of subjects who exhibited pronounced swelling (2.3- to 3.9-cm increase in arm circumference), significant strength loss (34.4–58.2% of baseline), or both. All exhibited greatly elevated CK levels (4,832–10,483 $IU\cdot L^{-1}$) and were noted to have strength decrements well beyond the 7-d study period, during which time they were followed to assure swelling and strength loss resolved. At least one subject exhibiting this high response to eccentric exercise was observed in each treatment group.

Using an eccentric protocol that results in such severe symptoms may not be appropriate in studying the efficacy of clinical interventions for eccentric muscle damage. The symptoms exhibited by high responders characterize rhabdomyolysis, a condition of muscle breakdown that can be caused by excessive exercise (8). A less intense damage-producing protocol of 12 eccentric contractions (22) versus the 50–70 eccentric contractions used in this study and others (29) may be more representative of what is seen clinically when untrained individuals perform eccentric exercise. Further studies should be encouraged to define the appropriate damage protocol to be used to produce a clinical presentation of eccentric muscle damage. As well, it is clinically important to define the criteria for a diagnosis of rhabdomyolysis, a condition that could potentially lead to adverse systemic response if large muscle groups are involved (8). Further research is warranted that can elucidate the reasons certain individuals exhibit this exceptionally high response.

Although there is ample evidence to suggest that muscle warm-up of as little as 1°C might decrease strain injury (13,21,26,28,30,31), our active and low-heat passive warm-up treatments, which elevated tissue temperature by approximately 1°C, may not have increased tissue extensibility by amounts great enough to observe clinically significant changes. Additionally, warm-up may increase extensibility when noncontracted muscle is stretched (21,30) but might not affect extensibility when stretch is imposed on contracted muscle. Indeed, warmed, noncontracted muscle achieved increased elongation when warmed from 25° to 40°C, but an eccentrically lengthened muscle warmed by the same degree did not achieve greater elongation at the point of failure (21). The increased elongation noted in warmed, noncontracted muscle during stretch may have occurred within the contractile element, rather than in the collagenous connective tissue (15). During eccentric lengthening, contracting sarcomeres may prevent elongation from occurring within the contractile apparatus.

Further, it has been reported that tissue temperature elevation of at least 3–4°C is required to increase connective tissue extensibility (13). Interestingly, elevated tissue temperature has also been hypothesized to increase eccentric muscle damage by enhancing tissue degradation (1). Indeed, increasing mouse muscle temperature from 25 to 35°C resulted in greater damage during eccentric contraction (33). Risk factors for exercise-induced rhabdomyolysis include high ambient temperature, high humidity, and lack of proper hydration, all of which may result in elevated body temperature (8). For this reason, we limited our high-heat treatment group (PW−HI+ECC) to a small number of volunteers ($N = 4$).

We observed no evidence that passively elevating muscle temperature by 3–4°C before eccentric contractions caused more damage than passive warm-up of ≈1°C or eccentric exercise alone. Although this may appear to contradict the results of Zerba and Faulkner (33), there were major differences in our methods; they induced a 10°C rise in temperature of mouse muscle (from 25 to 35°C), whereas we heated human muscle by 3–4°C (from approximately 37 to 41°C). To provide more information, three of the four subjects were treated with the eccentric exercise without warm-up condition 3 months later using the contralateral arm. We observed no differences in indicators of muscle damage. Although we cannot conclude that passively heating muscle tissue by 3–4°C prevents clinical presentation of muscle damage, it does appear that local heating of muscle tissue may not exacerbate muscle damage in human subjects. In fact, our PW−HI+ECC group exhibited significant change from control values at the least number of time points for measures of range of motion and pain. Due to the small sample size of our PW−HI+ECC group, however, we can only draw preliminary conclusions as to the effect of localized heat on eccentric muscle damage. Further study with more subjects is necessary before definitive conclusions can be drawn.

The ECC and AW−LO+ECC groups exhibited a greater percent change in circumference than controls. Additionally, we observed significant differences in response between active and passive warm-up with the proximal arm circumference measure, which was significantly greater at 24 h and 48 h in the ECC+AW−LO group than the ECC+PW−LO group. Although studies have observed increased tissue extensibility after both active and passive warm-up (21,26), it has been suggested that contractile activity during active warm-up may result in higher myotatic feedback loop activation and increased stiffness, and may impose limitations on fiber elongation (10), increasing the chance of strain damage. Given an identical lengthening stimulus, elevating tissue temperature passively before stretch is thought to decrease strain damage by decreasing the sensitivity of the muscle spindles to stretch (11). This hypothesized increase in tissue stiffness resulting from

active warm-up may have resulted in the increased swelling we observed in our active warm-up group. This is not conclusive, however, as other indicators of damage were not statistically different between those who performed active warm-up and those who performed passive warm-up before eccentric exercise.

CONCLUSION

Our observations suggest that passive warm-up performed before eccentric exercise may be more beneficial than active warm-up or no warm-up in attenuating swelling but does not support the use of warm-up to prevent, attenuate, or resolve more quickly the strength loss, loss of motion, or soreness resulting from eccentric muscle damage as produced in this study. Additional research on the use of warm-up techniques is encouraged to provide scientific rationale for those who regularly use warm-up techniques to achieve specific performance or injury-prevention goals.

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