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Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage

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This study investigated the relationship between delayed-onset muscle soreness and other indicators of muscle damage following eccentric exercise. Male students ($n = 110$) performed 12 (12ECC), 24 (24ECC), or 60 maximal eccentric actions of the elbow flexors (60ECC). Maximal isometric force, relaxed and flexed elbow joint angles, upper arm circumference, and plasma creatine kinase activity were assessed immediately before and after, and for 4 days after exercise. Muscle soreness (SOR) was evaluated by a visual analog scale (a 50-mm line, 0: no pain, 50: extremely painful) when the elbow flexors were palpated (SOR–Pal), flexed (SOR–Flx) and stretched (SOR–Ext). Although 24ECC and 60ECC resulted in significantly ($P < 0.05$) larger changes in all indicators and slower recovery compared to 12ECC, no significant differences were evident for SOR–Pal and SOR–Flx between 12ECC and 24ECC, or 12ECC and 60ECC. In contrast, SOR–Ext was significantly ($P < 0.05$) lower for 12ECC compared to 24ECC and 60ECC. A Pearson product-moment correlation showed SOR–Pal did not correlate significantly with any indicators, however, SOR–Ext and SOR–Flx showed weak ($r < 0.32$) but significant ($P < 0.05$) correlations with other indicators. Because of generally poor correlations between DOMS and other indicators, we conclude that use of DOMS is a poor reflector of eccentric exercise-induced muscle damage and inflammation, and changes in indirect markers of muscle damage and inflammation are not necessarily accompanied with DOMS.

Delayed onset muscle soreness (DOMS) is a sensation of dull, aching pain, usually felt during movement or palpation of the affected muscle, combined with tenderness and stiffness, that develops several to 24 h after unaccustomed exercise, peaks 1–3 days post-exercise, and disappears by 7–10 days (Armstrong, 1984; Ebbeling & Clarkson, 1989; Jones & Round, 1990; Miles & Clarkson, 1994; MacIntyre, Reid, McKenzie, 1995). It is well documented that DOMS is one of the outcomes of eccentric exercise (Armstrong, 1984; Newham, 1998; Ebbeling & Clarkson, 1989; Clarkson, Nosaka, Braun, 1992; Lieber & Fridén, 2002), and generally accepted that DOMS is associated with muscle and/or connective tissue damage, and/or subsequent inflammatory responses induced by eccentric exercise (Armstrong, 1984; Smith, 1991; Clarkson et al., 1992; Howell, Chleboun, Conatser, 1993; Pyne, 1994; MacIntyre et al., 1995; Lieber & Fridén, 2002). However, it is still debatable whether either muscle or connective tissue damage has a direct causal link to DOMS (Cleak & Eston, 1992; Miles & Clarkson, 1994), nor has it been confirmed that inflammation subsequent to damage is associated with DOMS (Newham, 1998; Jones & Round, 1990; Malm et al., 2000).

Whilst the exact cause of DOMS remains a mystery, numerous studies have been directed at preventative measures and treatments of DOMS and/or muscle damage (Cleak & Eston, 1992; Rodenburg, Steenbeek, Schiereck, Bär, 1994; Tiidus, 1997; Pizza, Cavender, Stockard, Baylies, Beighle, 1999; Barlas, Craig, Robinson, Walsh, Baxter, Allen, 2000). Such measures have been reported to reduce the magnitude of changes in muscle damage indicators, but not for DOMS (Rodenburg et al., 1994; Tiidus, 1997; Pizza et al., 1999). Studies often place muscle soreness and muscle damage in the same category, and it is generally considered that the greater the magnitude of muscle damage, the larger the degree of DOMS. Although both DOMS and changes in indirect markers of muscle damage, such as prolonged loss of muscle strength and range of motion, swelling, increases in muscle proteins in the blood, are induced by eccentric exercise (Clarkson et al., 1992), it may be possible that they are not closely related. To investigate any preventative or treatment measures for DOMS and decreases in

muscle function following eccentric exercise, it is necessary to understand the extent to which the degree of DOMS reflects the magnitude of muscle damage and inflammation, and ultimately whether DOMS is related to muscle damage and/or inflammation.

Previous studies have investigated associations between DOMS and other indices of muscle damage such as plasma creatine kinase (CK) levels (Croisier et al., 1996; Dierking, Bemben, Bemben, Anderson, 2000), changes in magnetic resonance images (Nurenberg, Giddings, Stray-Gundersen, Fleckenstein, Gonyea, Peshock, 1992; Evans, Haller, Wyrick, Parkey, Fleckenstein, 1998) or ultrasound (Dierking et al., 2000), morphological changes in muscle fibers (Fridén, 1984; Nurenberg et al., 1992), or changes in indicators of inflammation (Bobbert, Hollander, Huijing, 1986; Smith, 1991; Howell, Chleboun, Conatser, 1993; Crenshaw, Thornell, Fridén, 1994; MacIntyre et al., 1995; Croisier et al., 1996). However, other investigations (Rodenburg et al., 1994; Nosaka & Clarkson, 1996; Vincent & Vincent, 1997; Malm et al., 2000) found little or no relationship between DOMS and other indicators of muscle damage and inflammation. Further studies are necessary to answer the question whether DOMS is associated with muscle damage and inflammation following eccentric exercise.

Rodenburg, Bär and De Boer (1993) reported that the magnitude of DOMS showed few and weak correlations with biochemical and functional indicators of muscle damage. However, the sample size was relatively small, and the verbal rating scale used to assess DOMS may be relatively insensitive compared to a visual analog scale (VAS) (Ohnhaus & Adler, 1975). It was hypothesized that the magnitude of DOMS assessed by using a VAS scale would show a significant correlations with the magnitude of changes in indirect markers of muscle damage, if a larger sample size ($n \geq 100$) is used.

Therefore, to scrutinize the relationship between DOMS and muscle damage and inflammation following eccentric exercise, the present study compared the effect of different exercise protocols that resulted in various degrees of muscle damage on muscle soreness, and correlated the degree of muscle soreness with changes in indirect markers of muscle damage.

Methods

Subjects

Subjects were derived from our several previous studies that were conducted in last three years using the same exercise and measurement protocols and 8–35 subjects in each study. The total number of subjects was 110 in the present study. All subjects were healthy male students who had not been involved in resistance training programs participated in this study. Subjects were informed of the procedures of the experiment before involvement in the study, which was in accordance with Declaration of Helsinki, and a written informed consent document was obtained from all subjects. Their mean ($\pm$SD) age, height, weight are 20.3 $\pm$ 2.3 years, 170.8 $\pm$ 5.0 cm, and 61.1 $\pm$ 6.4 kg, respectively. They were not allowed to take any anti-inflammatory drugs during the experiment period, and instructed not to stretch, massage, or do anything for the sore muscles. Subjects were placed into one of the two groups; 12ECC ($n = 50$) and 24ECC ($n = 60$), based on the number of eccentric actions performed. There was no significant ($P > 0.8$) difference in age, height, and weight between the groups.

Exercise

Subjects ($N = 110$) performed either 12 (12ECC, $n = 50$) or 24 maximal eccentric actions of the elbow flexors (24ECC, $n = 60$) with their non-dominant arm. Fourteen subjects, who had performed 12ECC and showed relatively small changes in the indirect markers of muscle damage such as plasma creatine kinase activity (< 750 IU/l), performed 60 maximal eccentric actions of the elbow flexors (60ECC) approximately one year later. During the eccentric actions of 12, 24, or 60ECC, 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 that was attached to a modified arm curl machine (Nosaka & Clarkson, 1996). After 1 s of maximal isometric contraction, the forearm was forcibly extended from an elbow half-flexed (90°, 1.57 rad) to an elbow extended (180°, 3.14 rad) position in 3 s by an investigator who manually moved the lever arm to the extended position. Subjects were verbally encouraged to generate maximal isometric force at the flexed position and to maximally resist against the action throughout the range of motion. This action was repeated every 15 s for 12, 24 or 60 contractions.

Muscle soreness

Ohnhaus and Adler (1975) reported that a visual analog scale (VAS), which consists of a line (50–200 mm) with "no pain" at the left end and "unbearable pain" at the right end, reflected more precisely what a subject actually felt for pain associated with muscle compared to a verbal rating scale (VRS). In the present study, the intensity of muscle soreness was assessed by an examiner using a modified VAS with a 50-mm line with "no pain" on one end (0) and "unbearably painful" on the other end (50), when palpating over the elbow flexors and flexing and extending the elbow joint (Nosaka & Clarkson, 1996). In the palpation assessment of muscle soreness (SOR–Pal), an investigator placed his four fingers against the upper arm and applied digital pressure with the tips of the fingers toward the deeper tissues for approximately 3 s (Ohbach & Gale, 1989) while the subject was placing the forearm on an armrest of a chair at an elbow joint angle of approximately 90° (1.57 rad). The pressure given to the muscles was similar over days (approximately 1 kg/cm), and subjects were asked to report the soreness of three sites (proximal, middle, and distal of the biceps brachii) while the investigator was palpating the muscle. Since the investigator was experienced in this procedure, this palpation soreness assessment was considered to be as reliable as other two procedures described next. For the flexion (SOR–Flx) and extension (SOR–Ext) assessment, the examiner held the subject's arm and slowly moved the forearm to a maximal flexed position or a possible full-extended position while asking the subjects to relax. Subjects were asked to mark their subjective scale of soreness on the line during or immediately after flexion and extension under the supervision of the examiner. The length of the line from 0 to the marked point provided a numerical measure of soreness. Although using 100-mm line is a standard for a VAS scale, the 50-mm line method has been also established (Ohnhaus & Adler, 1975) and used in a previous study (Nosaka & Clarkson, 1996). To ensure the reliability, subjects

were occasionally asked to mark their soreness value on a different line without seeing the previous marking. The test–retest reliability was determined by an intraclass correlation coefficient (R), and the R-value for the palpation, extension, and flexion soreness assessment was 0.95, 0.92, and 0.93, respectively.

Indirect markers of muscle damage

Several indirect markers of muscle damage shown below were measured before, immediately after, and 1, 2, 3, and 4 days after exercise. The reliability of the measurements has been well established, and these markers have been used in many studies (Jones, Newham, Clarkson, 1987; Clarkson et al., 1992; Howell et al., 1993; Rodenburg et al., 1993; Rodenburg et al., 1994; Nosaka & Clarkson, 1996) on the assumption that the amount of the change reflects the magnitude of muscle damage.

Maximal isometric force (MIF) was determined at an elbow joint angle of 90° (1.57 rad) and 90° (1.57 rad) of shoulder flexion for 3 s. The measurement was taken twice (1 min between the measurements) using a loadcell (Model 1269, Takei Scientific Instruments Co. Ltd, Niigata, Japan) positioned in between two cables between the wrist and the frame of the measurement device. The load cell was connected to a computer (Macintosh Performer 5410, Apple Computer, Inc., Cupertino, USA) with a software program (LabVIEW, National Instruments, Austin, Texas, USA). The mean value of the two measurements was used for analysis.

Relaxed (RANG) and flexed elbow joint angle (FANG) were measured twice for each measurement by a Goniometer. Relative changes in RANG and FANG from the pre-exercise values were obtained.

Upper arm circumference (CIR) was assessed at 3, 5, 7, 9, and 11 cm from the elbow joint by a tape measure with the arm hanging relaxed by the side of the body, and the mean value of the five measurements was used for the further analysis. Relative changes in CIR from the pre-exercise value were obtained.

Approximately 5-ml of blood was drawn from the antecubital vein at all measurement time points except immediately after exercise, and centrifuged for 10 min to obtain plasma. The plasma samples were stored at −20 °C until analysis for CK activity. Plasma CK activity was determined spectrophotometrically by the VP-Super (Dainabot Co. Ltd, Tokyo, Japan) using a test kit (Dainabot Co. Ltd, Tokyo, Japan), and the normal reference ranges for male adults with this method was 45–135 IU/l.

B-mode ultrasound pictures of the elbow flexors were taken from the mid-belly of the biceps brachii by using 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 pre-exercise image. The thickness of the elbow flexors was assessed on the ultrasound images by measuring the distance between the subcutaneous fat layer and the edge of the humerus (Nosaka & Clarkson, 1996).

Statistical analyzes

Changes in all measures with time were compared between 12ECC and 24ECC, and 12ECC and 60ECC using two way repeated measures ANOVA. A Tukey's post hoc test was used to detect differences in the measures at different time points. Pearson's product moment correlation coefficient was used for analyzing the relationship between DOMS and other criterion measures. A statistical significance was set at $P < 0.05$. The values shown are means ± SEM.

Results

12ECC vs. 24ECC

Maximal isometric force

There was no significant difference in the pre-exercise MIF between 12ECC (191.1 ± 8.4 N) and 24ECC (196.6 ± 7.7 N). MIF dropped to 58.1 ± 1.7% (12ECC) and 47.1 ± 1.2% (24ECC) of the pre-exercise level immediately after exercise, and the force deficit was significantly ($P < 0.01$) larger for 24ECC compared to 12ECC (Fig. 1a). By 4 days after exercise, MIF had recovered to 73.1 ± 2.4% of the pre-exercise value for 12ECC, which was significantly ($P < 0.01$) larger than 24ECC (52.5 ± 1.9%) at the same time point (Fig. 1a).

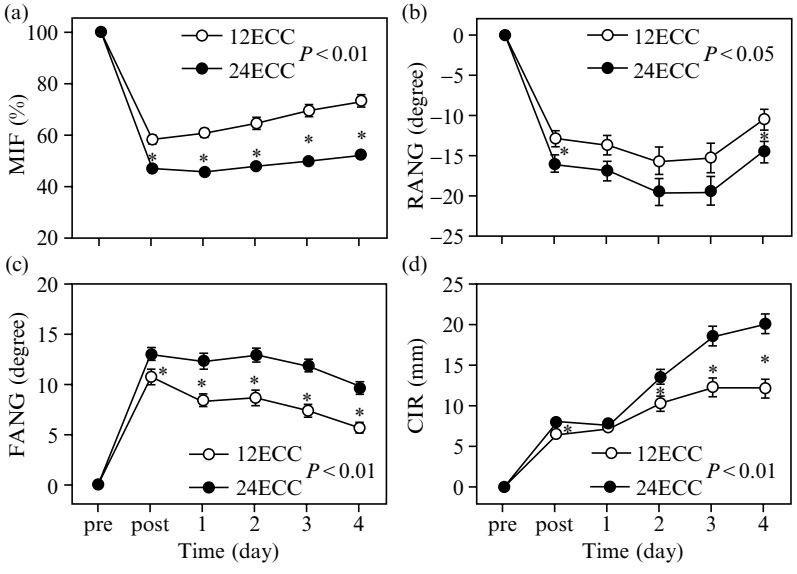

Fig. 1. Comparison between 12 (12ECC) and 24 maximal eccentric actions of elbow flexors (24ECC) for changes in maximal isometric force (a), relaxed (b) and flexed elbow joint angles (c), and upper arm circumference (d) from the baseline (pre) to immediately (post) and 1–4 days after exercise. *$P < 0.01$–0.05.

Elbow joint angles

The mean decrease in RANG immediately following 24ECC was significantly ($P < 0.05$) larger than that of 12ECC (Fig. 1b). At 4 days post-exercise, RANG was still significantly ($P < 0.05$) smaller than the pre-exercise level for both 12ECC and 24ECC, but the amount of decrease from the baseline was significantly ($P < 0.05$) larger for 24ECC ($-14.4 \pm 1.2°$) compared to 12ECC ($-10.4 \pm 1.3°$). The mean increase in FANG following 24ECC was significantly ($P < 0.01$) larger compared to 12ECC (Fig. 1c). At 4 days post-exercise, FANG was still significantly ($P < 0.05$) larger than the pre-exercise level for both 12ECC and 24ECC, but the amount of increase from the baseline was significantly ($P < 0.05$) larger for 24ECC ($9.6 \pm 0.6°$) compared to 12ECC ($5.6 \pm 0.5°$).

Upper arm circumference

CIR increased significantly ($P < 0.01$) immediately after exercise for both 12ECC (6.5 ± 0.4 mm) and 24ECC (8.0 ± 0.5 mm), however, no significant difference was evident between the exercises at this time (Fig. 1d). CIR continued to increase and peaked 3–4 days post-exercise for both exercises; however, the greatest value for 24ECC (20.0 ± 1.2 mm) was significantly ($P < 0.01$) larger than 12ECC (12.0 ± 1.2 mm).

Plasma CK activity

As shown in Fig. 2, pre-exercise plasma CK activities were similar for 12ECC (122 ± 16 IU/l) and 24ECC (142 ± 10 IU/l). CK activity increased significantly ($P < 0.01$) and peaked 3–4 days post-exercise for both protocols, but was significantly ($P < 0.01$) larger for 24ECC (peak: $15\,284 \pm 1188$ IU/l) compared to 12ECC (5549 ± 850 IU/l).

Muscle soreness

Figure 3 shows changes in SOR–Pal, SOR–Flx, and SOR–Ext following 12ECC and 24ECC. SOR developed 1 day after exercise and peaked 2–3 days after exercise for both exercises. Although the time course of changes in soreness was similar among SOR–Pal, SOR–Flx, and SOR–Ext, the peak soreness values were significantly ($P < 0.01$) smaller for SOR–Flx compared to SOR–Pal or SOR–Ext. There were no significant differences between 12ECC and 24ECC for SOR–Pal or SOR–Flx, but 24ECC showed significantly ($P < 0.05$) higher SOR–Ext values 3 and 4 days after exercise compared to 12ECC. Peak soreness values were similar for 12ECC (40.8 ± 1.4 mm) and 24ECC (40.9 ± 1.2 mm) for SOR–Pal, as well as for SOR–Flx (12ECC: 23.3 ± 1.7 mm, 24ECC: 22.2 ± 1.2 mm); however, 24ECC showed significantly ($P < 0.05$) higher peak SOR–Ext values (41.4 ± 1.3 mm) compared to 12ECC (37.0 ± 1.8 mm).

12ECC vs 60ECC

As shown in Table 1, significantly ($P < 0.01$) larger decreases in MIF and RANG, and increases in

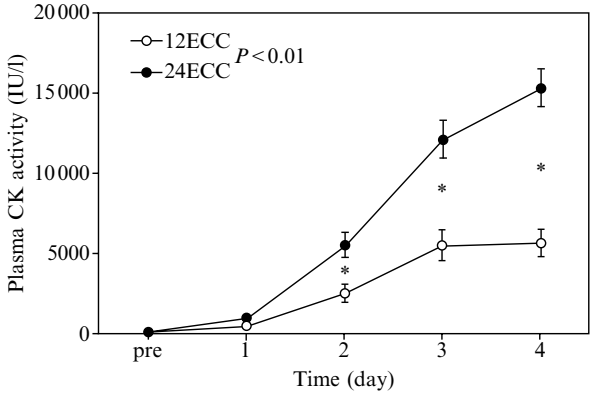

Fig. 2. Comparison between 12 (12ECC) and 24 maximal eccentric actions of elbow flexors (24ECC) for changes in plasma CK activity before (pre) and for 4 days (1–4 d) after exercise. $*P < 0.05$.

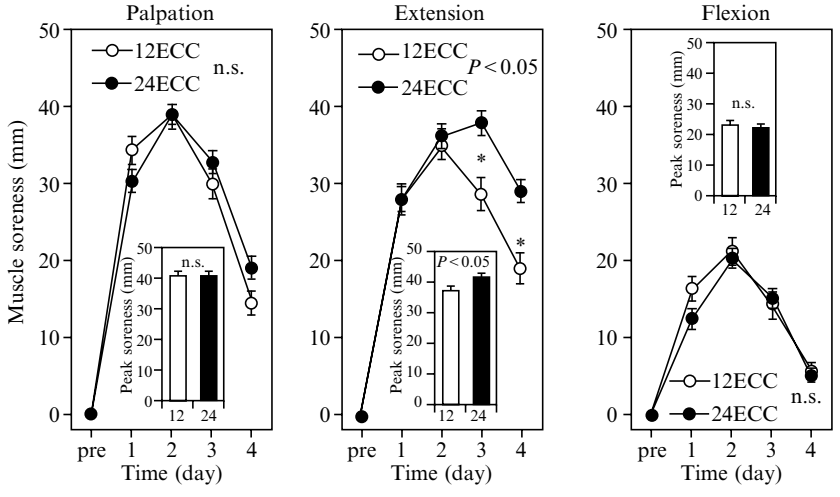

Fig. 3. Comparison between 12 (12ECC) and 24 maximal eccentric actions of elbow flexors (24ECC) for changes in soreness with palpation, extension, and flexion. In the inserted graph, peak soreness value after exercise is compared between 12ECC and 24ECC for each soreness assessment. $*P < 0.01$–0.05.

Table 1. Comparison between 12 (12ECC) and 60 maximal eccentric actions of elbow flexors (60ECC) for changes in maximal isometric force (MIF), relaxed (RANG) and flexed elbow joint angles (FANG), and upper arm circumference (CIR), plasma CK activity (CK), and soreness with palpation (SOR–Pal), extension (SOR–Ext), and flexion (SOR–Flx) before (pre), immediately (post) and 1–4 days after exercise (D1–D4)

		Pre	Post	D1	D2	D3	D4
MIF (N)	12ECC	190.0 (4.2)	132.6 (3.5)	138.9 (6.2)	154.0 (6.3)	163.8 (5.7)	173.6 (5.5)
	60ECC	185.8 (4.8)	44.5 (5.0)	83.6 (7.3)	89.5 (6.9)	90.3 (8.4)	98.8 (9.9)
	**	ns	**	**	**	**	**
RANG (°)	12ECC	160.5 (0.9)	152.1 (1.1)	152.7 (1.7)	153.3 (1.3)	154.0 (1.2)	155.4 (1.5)
	60ECC	162.4 (1.3)	148.3 (2.2)	141.9 (2.6)	142.6 (3.2)	140.6 (3.8)	142.6 (4.0)
	**	ns*	*	**	**	**	**
FANG (°)	12ECC	35.1 (1.0)	43.2 (0.9)	41.4 (0.5)	40.6 (0.6)	40.1 (0.6)	38.8 (0.6)
	60ECC	36.2 (0.7)	57.1 (1.6)	46.6 (1.3)	45.8 (1.1)	46.1 (1.1)	45.4 (1.3)
	**	ns	**	*	*	*	*
CIR (mm)	12ECC	256.7 (3.7)	262.4 (4.0)	262.4 (3.8)	262.1 (3.5)	262.6 (3.3)	261.7 (3.8)
	60ECC	259.4 (4.6)	269.9 (4.5)	268.9 (4.4)	272.8 (4.4)	277.2 (4.3)	281.4 (4.2)
	**	ns	**	*	**	**	**
CK (Iu/l)	12ECC	164 (46)	–	168 (32)	197 (28)	163 (19)	258 (54)
	60ECC	194 (33)	–	1963 (904)	3890 (1461)	10 546 (3179)	15 573 (4482)
	**	ns		**	**	**	**
SOR–Pal (mm)	12ECC	0	–	37.2 (2.8)	34.4 (3.6)	24.6 (3.4)	13.1 (2.8)
	60ECC	0	–	32.9 (2.2)	33.9 (3.3)	28.9 (3.2)	18.6 (2.9)
	ns	ns		ns	ns	ns	ns
SOR–Ext (mm)	12ECC	0	–	19.4 (3.2)	24.9 (3.5)	15.4 (3.4)	7.4 (2.2)
	60ECC	0	–	32.1 (4.1)	36.5 (3.9)	33.4 (4.3)	30.9 (4.6)
	*	ns		*	*	**	**
SOR–Flx (mm)	12ECC	0	–	13.5 (2.0)	16.5 (2.5)	10.4 (2.6)	3.7 (1.9)
	60ECC	0	–	16.9 (3.6)	22.4 (3.8)	19.6 (3.7)	11.9 (3.2)
	ns	ns		ns	ns	ns	ns

*$P < 0.05$, **$P < 0.01$, ns: not significant.

FANG and CIR were evident after 60ECC compared to 12ECC. Plasma CK activity was significantly ($P < 0.01$) larger following 60ECC compared to 12ECC, and the peak activity after 60ECC (16 231 $\pm$ 4582 IU/l) was significantly ($P < 0.01$) greater than that after 12ECC (221 $\pm$ 22 IU/l). However, levels of muscle soreness were not significantly different between 12ECC and 60ECC for SOR–Pal and SOR–Flx, and only SOR–Ext showed a significant ($P < 0.05$) difference between the exercise protocols (Table 1).

Correlation between muscle soreness and other measures

For the correlation analyzes, subjects from the 12ECC and 24ECC groups were combined, and the relationships between different muscle soreness measurements (SOR–Pal vs SOR–Ext vs SOR–Flx), as well as muscle soreness and other criterion measures at each time point or their peak values were obtained. Significant ($P < 0.01$) correlations were evident between SOR–Pal and SOR–Ext ($r = 0.60$), SOR–Ext and SOR–Flx ($r = 0.33$), and SOR–Pal and SOR–Flx ($r = 0.44$), however, a larger scatter was evident for the correlations, and the r-values were not high. Similar correlation coefficient values were found when the peak values of each soreness assessment were used. When comparing the different soreness ratings of each subject, most individuals showed lower SOR–Flx than SOR–Pal and SOR–Ext, and the rating between SOR–Pal and SOR–Ext was similar.

Table 2 shows the correlations between the mean soreness values 1–4 days after exercise for SOR–Pal, SOR–Ext, and SOR–Flx and changes in maximal isometric force, relaxed or flexed elbow joint angle, upper arm circumference after exercise. SOR–Pal did not show any significant correlations for all measures. However, significant ($P < 0.01$–0.05) correlations were found between SOR–Ext or SOR–Flx and maximal isometric force, relaxed joint angle, and upper arm circumference. No significant correlation was found between soreness value and peak plasma CK activity for SOR–Pal, but a significant ($P < 0.01$) correlation with CK was found for the SOR–Ext ($r = 0.23$) and SOR–Flx ($r = 0.22$). Although significant correlations were observed for SOR–Ext or SOR–Flx and criterion measures, weakness of the relationships was apparent.

Figure 4 shows correlations between peak SOR–Ext and changes in maximal isometric force (1 and 4 days post-exercise), RANG (3 days post-exercise) and FANG (immediately post-exercise), CIR (4 days post-exercise), and peak plasma CK activity. The time points for RANG, FANG, and CIR represent peak changes and correlation coefficients were similar to those shown in Table 1. Although SOR–Ext was significantly ($P < 0.01$) correlated with MIF of both 1 and 4 days post-exercise values, the regression lines indicate that the higher the SOR values, the larger the recovery

Table 2. Correlation matrix between muscle soreness and other indicators of muscle damage. SOR–Pal: mean soreness with palpation of the elbow flexors 1–4 days post-exercise, SOR–Ext: mean soreness when extending the elbow joint 1–4 days post-exercise, SOR–Flx: mean soreness when flexing the elbow joint 1–4 days post-exercise, MIF: maximal isometric force, RANG: relaxed elbow joint angle, FANG: flexed elbow joint angle, CIR: upper arm circumference, CK: plasma CK activity, post: immediately post-exercise, D1–4: mean value of 1–4 days post exercise $n = 110$

	Mif post	MIF D1–4	RANG post	RANG D1–4	FANG post	FANG D1–4	CIR post	CIR D1–4	CK peak	CK D1–4
Sor–Pal	−0.14 ns	−0.14 ns	−0.07 ns	−0.11 ns	0.03 ns	0.14 ns	0.04 ns	0.17 ns	0.06 ns	0.01 ns
Sor–Ext	−0.22 *	−0.27 **	−0.13 ns	−0.19 *	0.12 ns	0.22 *	0.08 ns	0.21 *	0.23 *	0.19 *
Sor–Flx	−0.31 **	−0.26 **	−0.08 ns	−0.21 *	0.07 ns	0.29 **	−0.15 ns	0.29 *	0.22 *	0.19 *

*$P < 0.05$, **$P < 0.01$, ns: not significant.

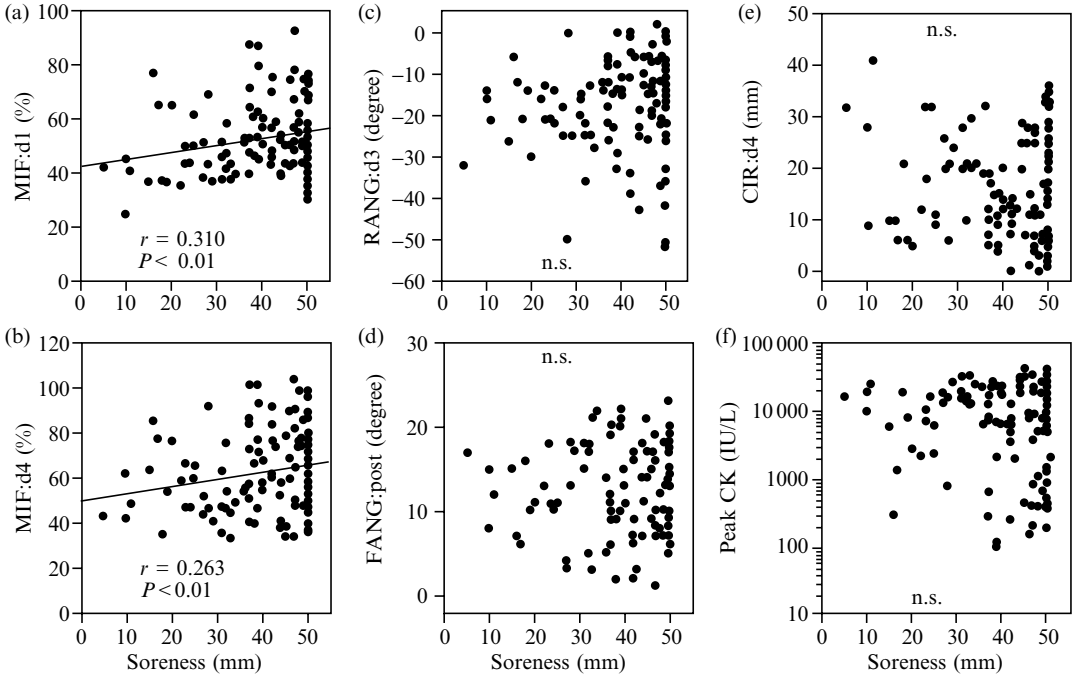

Fig. 4. Correlations between peak muscle soreness when extending the elbow joint and other indicators of muscle damage (a, b: maximal isometric force, c: relaxed elbow joint angle, d: flexed elbow joint angle, e: upper arm circumference, f: peak plasma CK activity). post: immediately post-exercise, d3: 3 days post-exercise, d4: 4 days post-exercise, ns: not significant, $n = 110$.

of MIF. This is against what it should be; the higher the SOR values, the smaller the recovery of MIF shown by smaller MIF values compared to the pre-exercise level. It was apparent that subjects who had similar soreness showed a large variability in other measures, and subjects who had severe soreness did not necessarily show retarded recovery of MIF, large changes in RANG and FANG, conspicuous swelling (increases in CIR), and high CK activity (Fig. 4). It is interesting to note that subjects whose muscle damage seemed to be severe because of the large decreases in MIF (Figs 4a, b), large increases in CIR (Fig. 4e) and plasma CK activity (Fig. 4f) showed a large variety of the soreness.

As shown in Fig. 5, three examples of ultrasound images from before, immediately after, and 4 days after exercise demonstrate that the peak muscle soreness levels were hardly different although there were large differences in peak CK activity, isometric force recovery, upper arm circumference changes, and changes in images among three subjects. Although typical examples are shown in the figure, this kind of cases were common in the present study, and there were also many opposite cases such that muscle soreness levels were largely different among subjects whose ultrasound images and changes in other criterion measures were similar.

Discussion

The present study showed that individuals did not necessarily experience more soreness when performing

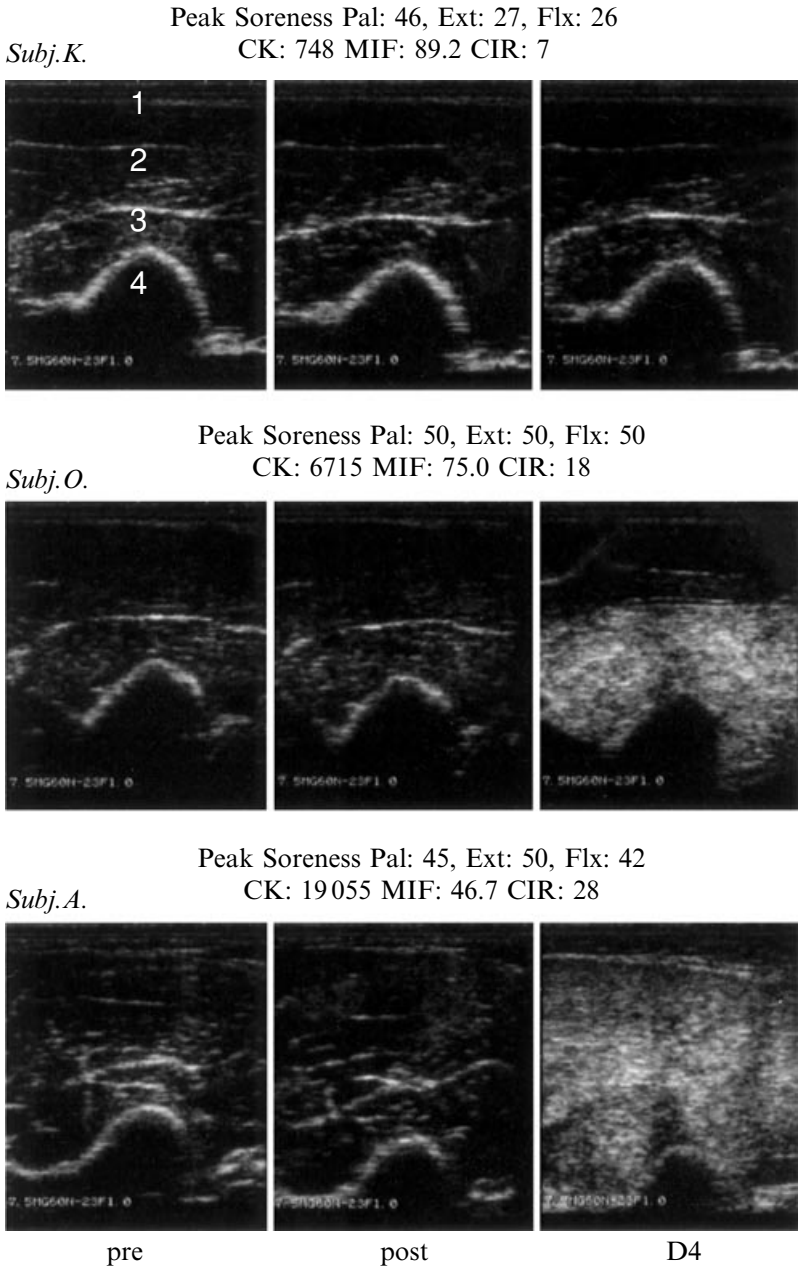

Fig. 5. Examples of ultrasound pictures of three subjects (Subj. K, O, A) taken before (pre), immediately after (post), and 4 days post-exercise (D4). For each subject, peak muscle soreness values (mm) with palpation (Pal), extension (Ext), and flexion (Flx), as well as maximal isometric force level (% pre) at 4 days post-exercise (MIF), and the amount of maximal increase in upper arm circumference (CIR; mm), and peak CK activity (CK; IU/l) are shown. In the top-left ultrasound picture, 1: subcutaneous fat layer, 2: biceps brachii, 3: brachialis, 4: humerus.

exercise protocols that lead to greater changes in indirect markers of muscle damage. Warren, Lowe and Armstrong (1999) suggested that decreases in muscle strength best indicate muscle damage. Changes in MIF and other measures (Figs 1 and 2) were shown to be greater following 24ECC compared to 12ECC. Therefore, it seems reasonable to assume that the magnitude of muscle damage was greater following 24ECC compared to 12ECC. However, SOR–Pal and SOR–Flx were not significantly different between 12ECC and 24ECC (Fig. 3). Since different subjects were used for the comparison of the two protocols, it could be argued that subjective responses to soreness may have been different between groups. However, this is unlikely, since the subjects were recruited from the same large pool of subjects, and physical characteristics were similar between groups.

Since pain sensation is subjective and individual, quantification of pain is difficult (Ohnhaus & Adler, 1975; Revill, Robinson, Rosen, Hogg, 1976). There are several methods to evaluate pain including pressure pain threshold, tenderness, and McGill Pain Questionnaire (Ohbach & Gale, 1989; MacIntyre et al., 1995; Bajaj, Graven-Nielsen, Arendt-Nielsen, 2001), and a VAS has been often used to quantify pain (Ohnhaus & Adler, 1975; Revill et al., 1976; Ohbach & Gale, 1989). A number of difficulties exist in the use of the VAS to quantify soreness, although the

scale has been used in many studies (Bobbert et al., 1986; Vincent & Vincent, 1997; Barlas et al., 2000; Malm et al., 2000; Bajaj et al., 2001). These include questions regarding the sensitivity of the instrument, for example, many subjects marked "50", or "unbearably painful" for the peak DOMS level (Fig. 4). Since the "50" was maximal for the scale, subjects could not indicate a greater pain level, even if they experienced greater soreness the following day for instance. This is a disadvantage of using a close-ended scale, and a better way of recording pain may be to use an open-ended scale in which subjects can choose any number to represent the pain associated with some intermediate level of stimulation and then scale all subsequent tests in relation to the initial reference stimulus (Jones & Round, 1990). It is also important to note that the pain sensation could vary among subjects. For example, some subjects might mark "40" even if their soreness level is medium, or others might mark "10" even if the pain level is very severe. Because of the subjective and individual nature of the pain sensation, the question arises whether it is possible to compare levels of soreness between subjects, or even changes in DOMS over days in the same subjects. It has been documented that perception of a noxious stimulus may differ greatly between individuals and may also vary with the person's mood, health or hormonal status (Jones & Round, 1990). Melzack (1982) stated "pain is not simply a function of the amount of bodily damage alone, but is influenced by attention, anxiety, suggestion, and other psychological variables." For these reasons, it is important for researchers to be cognizant of the limitations in quantitatively assessing DOMS in experimental studies.

In the present study, no differences in SOR–Pal and SOR–Flx were found between 12ECC and 24ECC (Fig. 3) and even when the same subjects were used for the 12ECC and 60ECC bouts that resulted in significantly different magnitude of changes in the criterion measures (Table 1). The differences between 12ECC and 60ECC for the changes in the criterion measures were greater than the differences in those between 12ECC and 24ECC (Table 1, Figs 1 and 2), however, SOR–Pal and SOR–Flx did not distinguish between 12ECC, 24ECC, and 60ECC (Table 1, Fig. 3). Moreover, many studies have used similar methods to quantify the level of DOMS, employing either VAS (Bobbert et al., 1986; Vincent & Vincent, 1997; Barlas et al., 2000; Malm et al., 2000; Bajaj et al., 2001) or VRS (Nurenberg et al., 1992; Howell et al., 1993; Rodenburg et al., 1993; Rodenburg et al., 1994; Croisier et al., 1996; Evans et al., 1998; Pizza et al., 1999; Dierking et al., 2000) and its response to various preventative or therapeutic measures. Therefore, it is reasonable to conclude that muscle soreness, at least the palpation or flexion soreness assessment, is a poor reflection of the magnitude of muscle damage.

It should be noted that SOR–Ext showed a significant difference between 12ECC and 24 ECC (Fig. 3) as well as 12ECC and 60ECC (Table 1). The correlations between the three different soreness assessments (SOR–Pal vs SOR–Ext, SOR–Ext vs SOR–Flx, SOR–Flx vs SOR–Pal) were statistically significant, however, the correlation coefficients were not high ($r = 0.33$–0.60), and SOR–Ext was of a greater magnitude and longer lasting than SOR–Flx (Fig. 3, Table 1). This suggests that the causes of pain are not the same between the three assessments. SOR–Pal may be similar to muscle tenderness or allodynia, a feeling of discomfort elicited by pressure (Jones & Round, 1990; Bajaj et al., 2001). Increased internal fluid pressure due to swelling appears to be associated with SOR–Pal and SOR–Flx (Crenshaw et al., 1994). On the other hand, SOR–Ext is probably also influenced by increases in muscle stiffness. Subjects had difficulty in straightening the elbow following exercise, and this was shown in the decreases in RANG (Fig. 1b, Table 1). Many studies (Jones et al., 1987; Clarkson et al., 1992; Howell et al., 1993; Rodenburg et al., 1993; Nosaka & Clarkson, 1996) have shown that muscle stiffness increases following eccentric exercise. Howell et al. (1993) suggested that swelling was related to an increased muscle stiffness associated with shortening of the connective tissue arranged in parallel with the muscle fibers (Jones et al., 1987). Weerakkody, Whitehead, Canny, Gregory and Proske (2001) showed that muscle mechanoreceptors, including muscle spindles, contribute to muscle soreness after eccentric exercise. It is possible that SOR–Pal is more related to inflammatory factors, but reflex-mediated pain contributed to SOR–Ext (Lieber & Fridén, 2002).

The present study found no correlations between SOR–Pal and other criterion measures, and only weak correlations were shown between SOR–Ext or SOR–Flx and other measures (Fig. 4, Table 2). The regression line in the Fig. 4(b) showed that the higher the soreness score, the larger the recovery of MIF, however, it should have been the higher the soreness score, the smaller the recovery of MIF. Therefore, it seems that most of the significant correlations shown in Fig. 4 and Table 2 happened by chance, or at least the physiological significance of them is low. This is consistent with the results shown by Rodenburg et al. (1993) who reported few and low correlations between DOMS (mean value of palpation and extension soreness) and other parameters including most of the measures used in the present study. Although the present study used a larger number of subjects, and a VAS was used to quantify the intensity of DOMS, correlations were still absent or weak (Fig. 4, Table 2). It is evident from Fig. 4 that individual difference in sensation of muscle soreness is diverse. Figure 5 also demonstrates that the degree of DOMS does not indicate the magnitude of muscle damage shown by ultrasound images.

These findings further support the concept that the magnitude of DOMS does not reflect the magnitude of muscle damage (Jones & Round, 1990; Warren et al., 1999).

If the degree of DOMS does not reflect the magnitude of muscle damage, what does DOMS indicate and what determines the degree of DOMS? It has been proposed that mechanical, rather than chemical factors are more associated with the induction of DOMS (Armstrong, 1984; Bobbert et al., 1986; Cleak & Eston, 1992; Howell et al., 1993). Of the former, it is unlikely that swelling is a main contributor to soreness, since DOMS subsides when upper arm circumference reaches its peak (Figs 1d and 3). Pain receptors are free nerve endings within the tissue that are excited by chemical, mechanical, and thermal stimuli (Jones & Round, 1990; Miles & Clarkson, 1994). Of the two types of pain receptors; large, myelinated type III (A delta) fibers relay sharp, localized pain sensations to the brain very rapidly, whilst the small non-myelinated type IV (C) fibers conduct dull, diffuse, achy pain sensations to the brain relatively slowly (Jones & Round, 1990; Miles & Clarkson, 1994). C fibers are found in connective tissue, between extrafusal and intrafusal muscle fibers, near arterioles and venules, in Golgi tendon organ capsules, in tendinous tissue at the muscle tendon junction, and associated with fat cells (Jones & Round, 1990; Miles & Clarkson, 1994). It is no doubt that DOMS is an outcome of eccentric or eccentric biased exercise, because pure concentric or isometric muscle actions seldom induce DOMS (Armstrong, 1984; Newham, 1998). However, DOMS may not be directly related to muscle damage and subsequent inflammation. Malm et al. (2000) have shown that DOMS is not related to muscle inflammation. In this context, it is noteworthy that primary muscle diseases (e.g., Duchenne muscular dystrophy) do not cause muscle pain in spite of major disruptions of the myofibrillar and sarcotubular apparatus (Marchettini, 1993; Lieber & Fridén, 2002). More likely, muscle pain sensation may be associated with changes in the chemical environment surrounding muscle tissue, or by stimulation of the fascia.

Jones & Round (1990) have suggested that DOMS is due to an inflammation of the connective tissue in or around the muscle, and the inflammation appears to sensitize mechanoreceptors, probably situated in the connective tissue sheaths, so that they respond excessively when the muscle is stretched or massaged. It seems possible that connective tissue damage rather than muscle damage is more closely related to the induction of DOMS (Jones & Round, 1990). This would account for the poor relationships observed between soreness, plasma CK activity and decrement of muscle function (Fig. 4, Table 2). As shown by Weerakkody et al. (2001), it is also possible that afferents from muscle spindles contribute to the sensation of DOMS. Future studies should investigate relationships between muscle spindle and nociceptors. As the gate control theory proposed, pain signals from the body are modulated by other, concurrent somatic inputs as well as by descending influence of brain (Melzack, 1982). Therefore, it should be recognized that the degree of DOMS does not necessarily reflect the degree of muscle and/or connective tissue damage.

In conclusion, the results of the present study did not support the hypothesis that the magnitude of DOMS assessed by using a VAS scale would be significantly correlated with the magnitude of changes in indirect markers of muscle damage. It would appear that the degree of DOMS associated with eccentric exercise is not affected by the severity of the resulting muscle damage. Furthermore, muscle soreness levels have little or no correlation with other commonly used indicators of muscle damage. In this regard, it should be noted that preventative or treatment measures for DOMS are not necessarily the same as those for muscle degeneration and regeneration. It is also important to delineate what DOMS reflects and how it should be treated in the future studies.

Perspectives

The results of the present study, together with previous studies (Rodenburg et al., 1993; Nosaka & Clarkson, 1996), suggest that finding preventative or treatment measures to reduce strength loss or plasma CK response, for instance, do not necessarily mean that the measures are effective for the treatment of DOMS, and vice versa. It is also possible that muscle damage and loss of muscle function persist in the absence of DOMS. Therefore, the use of DOMS to judge the magnitude of muscle damage should be avoided, although in conjunction with other muscle damage indicators it provides supplementary evidence of changes in the muscle environment. The physiological significance of DOMS remains a mystery.

Key words: delayed onset muscle soreness; maximal isometric force; upper arm circumference; plasma CK activity; elbow joint angle; visual analog scale.

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