

Muscle Soreness Following Resistance Exercise With and Without Eccentric Contractions

WILLIAM C. BYRNES
PRISCILLA M. CLARKSON
FRANK I. KATCH
University of Massachusetts

Muscle soreness is often experienced following unaccustomed exercise. This common phenomenon known as delayed onset muscle soreness (DOMS) occurs following such unfamiliar tasks as downhill running (Schwane, Johnson, Vandenakker, and Armstrong, 1983a; Schwane, Watrous, Johnson, & Armstrong, 1983b) and eccentric exercise regimens (Friden, Sjostrom, & Ekblom, 1983). The mechanisms behind DOMS have been evaluated by examining markers of muscle damage (creatine kinase and lactate dehydrogenase (Newham, Jones, & Edwards, 1983; Schwane et al., 1983a; Tiidus & Ianuzzo, 1983)), or connective tissue damage (hydroxyproline [Abraham, 1977]). Muscle damage has also been evaluated by ultrastructure observation of muscle biopsy samples obtained from the affected muscles (Friden et al., 1983). Although still the subject of debate, it appears that DOMS results from either mechanical or ischemic damage to the muscle fibers (Clarkson, Kroll, Graves, & Record, 1982; Friden et al., 1983; Mayer & Clarkson, 1984; Schwane et al., 1983a; Tiidus & Ianuzzo, 1983).

Recently, several investigators have provided anecdotal evidence that DOMS does not occur following an initial session of circuit weight training which utilizes isokinetic exercise equipment (Gettmann, Ayers, Pollock, Durstine, & Grantham, 1979; Gettman, Culter, Strathman, 1980). The lack of DOMS with isokinetic exercise may be related to the type of muscular contraction exerted during the exercise. Most forms of exercise have an eccentric component (muscle lengthening against resistance) whereas the isokinetic resistive equipment involves only a concentric component (muscle shortening against resistance). In the present study we evaluated the development of DOMS following the initial bout of two different circuit exercise regimens differing in their eccentric component. Because DOMS has been associated with muscle damage (Friden et al., 1983), serum creatine kinase (CK) activity was also assessed.

Method

Sixteen women volunteers who had no prior resistive exercise experience participated in the study and signed an informed consent document in accord with the university guidelines for the protection of human subjects. Previous research from our laboratory has demonstrated that a single bout of exercise significantly reduces DOMS and serum enzyme changes on subsequent exercise bouts performed up to six weeks later (Byrnes et al., 1985). Therefore, because one group of subjects could not be tested on both exercise tasks, they were randomly assigned to one of two groups.

Group C completed an exercise regimen which utilized hydraulic resistive exercise devices (Hydra-Fitness, Inc., Belton, TX). These devices require concentric contractions with no eccentric phase. Subjects performed an eight-station exercise circuit designed to alternately stress the major muscle groups of the upper and lower body. Three sets were performed at each station. One set was equal to 20 s of maximal effort work followed by 40 s of rest. Group CE completed an exercise regimen using the same work-to-rest ratio except that the eight stations were performed on an exercise machine (Kidde, Inc., Cedar Rapids, Iowa) requiring both concentric and eccentric contractions. Subjects exercised at 60% of their own repetition maximum, which was previously determined at each station.

Blood samples and perceived muscle soreness ratings were obtained prior to exercise and at 5, 10, and 25 hr after exercise. Blood samples were taken from a superficial arm vein using standard phlebotomy techniques, allowed to clot, and then centrifuged at 2600 rpm for 10 min. After storage at -20°C , the serum was analyzed for creatine kinase using a Boehringer test kit according to the method of Szasz, Gruber, and Bernt (1976). Perceived soreness was assessed by a questionnaire by which subjects rated their soreness level for 1 (normal) to 10 (very, very sore) for 17 different muscle areas.

Table 1
Perceived Soreness Ratings 25 Hrs Following the
Acute Exercise Bouts

Site	Group C		Group CE	
	\bar{X}	SD	\bar{X}	SD
Chest	2.3	1.7	5.1	1.8
Back (upper)	2.6	1.9	2.8	2.2
Shoulders (front)	2.2	1.3	3.6	2.4
Shoulders (back)	1.9	1.5	3.6	2.0
Biceps (mid)	1.9	1.4	4.3	2.0
Biceps (lower)	1.8	1.4	3.5	1.8
Triceps (mid)	1.9	1.4	3.4	1.8
Triceps (lower)	1.9	1.4	3.0	1.1
Forearm (front)	1.7	1.4	3.4	1.8
Forearm (back)	1.7	1.4	2.9	1.4
Back (lower)	1.7	1.4	2.9	2.2
Buttocks	1.8	1.4	2.5	1.3
Quadriceps (mid)	2.0	1.6	4.1	2.6
Quadriceps (lower)	2.1	1.8	3.8	2.1
Hamstrings (mid)	2.1	1.5	3.5	2.6
Hamstrings (lower)	2.1	1.8	3.0	2.7

Results

The perceived soreness ratings for the two exercise regimens at the 24 hr sampling time are presented in Table 1. A repeated measures ANOVA was calculated for each sample time (pre, 5, 10 and 25 hr post) to assess the between groups and between site main effects. No significant difference between groups or sites was found for the pre-, 5-, or 10-hr sample times. The mean soreness rating for these times rarely exceeded 2.0 and never more than 2.5. However, for the 25-hr sample time, a significant difference was found between groups, $F(1,15) = 6.85$, $p < .05$. Note from Table 1 that Group CE had mean soreness ratings that ranged from 2.5 to 5.1 compared with a range of 1.7 to 2.6 for Group C. No significant difference in soreness ratings were found among sites for either group ($p > .05$).

When serum CK levels were compared between groups (Table 2), only a significant sample time effect was observed, $F(3,45) = 6.49$, $p < .01$; this indicated that both groups responded in the same manner to the

Table 2
Creatine Kinase Activity (mU/ml) Following Acute
Exercise Bouts

	Group C		Group CE	
	\bar{X}	SD	\bar{X}	SD
Pre	86.7	27.3	126.9	58.1
Five hr post	344.8	426.3	232.0	134.2
Ten hr post	394.3	473.1	368.5	203.9
Twenty-five hr post	288.0	305.5	482.2	411.2
Absolute increase	319.3	474.6	399.9	381.1
Relative increase	435.6	732.6	355.4	437.9

exercise tasks. Post hoc analysis revealed that serum CK levels were elevated for both groups at the 5-hr sample time and remained elevated through the 25-hr sample time.

Discussion

Anecdotal evidence of others (Gettman et al., 1979; Gettman et al., 1980) has indicated that circuit weight training using exercise devices requiring only concentric contractions does not result in DOMS. The present study indicates that individuals who participate in an initial session of concentric weight training exercise do not develop significant DOMS. In contrast, however, individuals who participate in an initial session of weight training consisting of both concentric and eccentric muscle contractions reported significant increases in DOMS. These results confirm the work of others who have reported significant muscle soreness following eccentric exercise (Friden et al., 1983; Newham et al., 1983; Schwane et al., 1983a, 1983b; Talag, 1973). It should be noted that some subjects in Group C reported minimal muscle soreness which may relate to the isometric component involved in stabilizing body segments. Talag (1973) reported muscle soreness following isometric work. A recent study from our laboratory (Clarkson, Byrnes, McCormick, Turcotte, & White, 1985) examined muscle soreness following arm flexion concentric and arm flexion eccentric exercise designed to minimize the need for isometric stabilization. Following the concentric exercise, the maximal soreness rating for the biceps muscle was 1.6 (based upon the same 10-point scale used in this study). Following the eccentric arm flexion exercise, however, the maximal soreness rating was 4.8. Based upon the preceding information, it appears that the degree of isometric stabilization for a concentric exercise may determine the magnitude of DOMS. However, data from this study suggest that even when isometric contractions are part of an exercise movement, the perception of soreness is greater following a task involving both eccentric and concentric contractions.

Substantial increases in serum CK activity have been reported following eccentric and isometric exercise (Abraham, 1977; Clarkson et al., 1982; Newham et al., 1983; Schwane et al., 1983a), while a smaller CK response has been reported following level running (Schwane et al., 1983a). Smaller CK responses following level running have been attributed to a lower eccentric component. The elevated serum CK activity following the concentric resistive exercise in the present study may relate to the isometric component of each exercise. The magnitude of the CK changes following isometric exercise are quite large even when a small muscle mass is involved. Clarkson and associates

(1982) and Graves, Clarkson, Kirwan, and Litchfield (1984) have reported CK changes of 143% and 288% for one-arm and one-leg exercise, respectively.

While creatine kinase is accepted as a serum marker for muscle damage (Newham et al., 1983), a clear association has not been established between the extent of muscle damage and the magnitude of the CK release. Likewise, serum CK levels following exercise have not been shown to relate to perceived muscle soreness. In a recent study, Byrnes et al. (1985) found no significant relationship between DOMS following downhill running and serum CK or myoglobin changes, even though there was a substantial increase of 339.7% in serum CK activity and of 432.3% in serum myoglobin levels. Because serum CK activity represents a net value of CK release from tissue and CK clearance from the blood, CK levels may not provide a sensitive enough indicator of the magnitude of muscle damage.

In summary, the initial bout of a weight training program with or without eccentric contractions produced marked increases in serum CK activity. However, group CE members, who performed exercises that required eccentric and concentric contractions, developed significantly more soreness than Group C members, who performed exercises requiring only concentric contractions. These data suggest that both forms of exercises certainly produced cellular modifications which resulted in elevated serum CK activity. However, only the exercise involving concentric muscle contractions did not result in delayed onset muscle soreness.

The authors would like to acknowledge the expert technical assistance of J. Spencer White. This study was supported by a grant from Hydra-Fitness Industries (Belton, Texas) as part of the Jerry D. Brentham Research Professorship in Muscular Dynamics in the Department of Exercise Science at the University of Massachusetts, Amherst, MA.

References

- Abraham, W. M. (1977). Factors in delayed muscle soreness. *Medicine and Science in Sports*, 9, 11-22.
- Byrnes, W. C., Clarkson, P. M., White, J. S., Hsieh, S. S., Frykman, P. N., & Maughan, R. J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. *Journal of Applied Physiology*, in press.
- Clarkson, P. M., Byrnes, W. C., McCormick, K. M., Turcotte, L. P., & White, J. S. (1985). Muscle soreness and serum creatine kinase activity following isometric, eccentric and concentric exercise. *International Journal of Sport Medicine*, in press.
- Clarkson, P. M., Kroll, W., Graves, J., & Record, W. A. (1982). The relationship of serum creatine kinase, fiber type, and isometric exercise. *International Journal of Sports Medicine*, 3, 145-148.
- Friden, J., Sjoström, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. *International Journal of Sports Medicine*, 4, 170-176.
- Gettman, L. R., Ayers, J. J., Pollock, M. L., Durstine, J. L., & Grantham, W. (1979). Physiologic effects on adult men of circuit strength training and jogging. *Archives of Physical Medicine and Rehabilitation*, 60, 115-120.
- Gettman, L. R., Culter, L. A., & Strathman, T. A. (1980). Physiologic changes after 20 weeks of isotonic vs isokinetic circuit training. *Journal of Sports Medicine and Physical Fitness*, 20, 265-274.
- Graves, J., Clarkson, P. M., Kirwan, J., & Litchfield, P. (1984). Serum creatine kinase levels following three different isometric exercise regimens. *Medicine and Science in Sports and Exercise*, 16, 186.
- Mayer, S. J., & Clarkson, P. M. (1984). Serum creatine kinase levels following isometric exercise. *Research Quarterly for Exercise and Sport*, 55, 191-194.
- Newham, D. J., Jones, D. A., & Edwards, R. H. T. (1983). Large delayed plasma creatine kinase changes after stepping exercise. *Muscle and Nerve*, 6, 380-385.
- Schwane, J. A., Johnson, S. R., Vandenakker, C. K., & Armstrong, R. B. (1983a). Delayed onset muscular soreness and plasma CPK and LDH activities after downhill running. *Medicine and Science in Sports and Exercise*, 15, 51-56.
- Schwane, J. A., Watrous, B. G., Johnson, S. R., and Armstrong, R. B. (1983b). Is lactic acid related to delayed onset muscle soreness? *Physician and Sportsmedicine*, 11, 124-131.
- Ssasz, G., Gruber, W., & Bernt, E. (1976). Creatine kinase in serum 1. Determination of optimum reaction conditions. *Clinical Chemistry*, 22, 650-656.
- Talag, T. (1973). Residual muscular soreness as influenced by concentric, eccentric, and static contractions. *Research Quarterly*, 44, 458-469.
- Tiidus, P. M., & Ianuzzo, C. D. (1983). Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. *Medicine and Science in Sports and Exercise*, 15, 461-465.

Submitted: December 19, 1984

Accepted: April 1, 1985

William C. Byrnes is currently an assistant professor with the Department of Physical Education, University of Colorado, Boulder, CO 80309. Priscilla Clarkson is an associate professor and Frank I. Katch is a professor with the Department of Exercise Science, University of Massachusetts, Amherst, MA 01003.