

# The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase

L. L. Smith PhD, M. G. Fulmer MA, D. Holbert\* PHD, M. R. McCammon MA, J. A. Houmard PhD, D. D. Frazer<sup>†</sup> MD, E. Nsien<sup>‡</sup> MD and R. G. Israel EdD

Human Performance Laboratory, \*Department of Biostatistics, <sup>†</sup>Department of Rheumatology and <sup>‡</sup>Department of Internal Medicine, East Carolina University, Greenville, USA

The purpose of this study was to determine if there were any beneficial or detrimental effects regarding delayed onset muscle soreness (DOMS), serum creatine kinase (CK), and maximum concentric strength at 80% of 1-RM<sub>conc</sub>, if a bout of eccentric exercise was repeated at 48 h after an initial bout. A secondary purpose was to determine whether unaccustomed eccentrics might affect plasma cholesterol (TC). Twenty-six men were randomly assigned to a control (Group 1) or experimental group (Group 2). Both groups performed three sets (12 repetitions per set) of the eccentric phase of a chest press, at 80% of one repetition maximum (1-RM<sub>conc</sub>); Group 2 repeated this exercise 48 h later. DOMS and CK were measured before, and every 24 h for 8 days after; TC was measured before, and every 24 h for 4 days. Maximum strength during the concentric phase of a chest press (1-RM<sub>conc</sub>) was measured before and at 48-h intervals after. A repeated measures analysis of variance revealed a significant time effect ( $P < 0.05$ ) for DOMS, CK and strength, but no significant difference between groups ( $P < 0.05$ ). An interesting finding was the significant ( $P < 0.05$ ) reduction in TC at 24, 48 and 72 h, after exercise in both groups, which we hypothesized was associated with cellular repair. From these results we concluded that when a bout of eccentrics is repeated 48 h after an initial bout, there is no change in the characteristic time-course and/or intensity of DOMS, CK or 1-RM<sub>conc</sub>.

**Keywords:** Eccentric muscle action, delayed onset muscle soreness, strength, total cholesterol

Delayed onset muscle soreness (DOMS) is a sensation of discomfort or pain that occurs in response to unaccustomed exercise, or in response to large increases in the volume of exercise. It is first felt between 8–24 h after exercise, peaks in intensity between 24 and 72 h and usually disappears by 5 days<sup>1–4</sup>. DOMS is associated with connective<sup>5–8</sup> and contractile<sup>3,4</sup> tissue microtrauma, resulting from high tensions generated during the eccentric phase of a movement<sup>1</sup>. Complete healing does occur, although little is known about this aspect<sup>2</sup>.

The general recommendation concerning exercise during the period of muscle soreness<sup>2</sup> is to 'ignore the sensations and work through the pain'. In addition, anecdotal reports suggest that exercise during this period might hasten dissipation of the soreness. However, there is no scientific evidence to substantiate or refute these claims.

Friden and colleagues<sup>9</sup> reported that in the days following eccentrically biased exercise, traumatized muscle fibres are swollen and presumably weaker and more vulnerable to injury. Leadbetter<sup>10</sup> suggested that after any sports injury, there is a susceptible period during which there is a greater risk of reinjury. Therefore, exercise during this early stage might be detrimental to recovery either because of reinjury or interference with healing<sup>10,11</sup>. Therefore, the main objective of this study was to determine whether repeating a bout of eccentrics at 48 h would exacerbate, alleviate or have no effect on DOMS, creatine kinase (CK) and strength.

Increases in serum levels of CK are used as an indirect marker of the microtrauma associated with DOMS<sup>3,4</sup>. However, there appear to be no markers related to the process of healing. Since cholesterol is a component of cell membrane<sup>12</sup>, and since blood cholesterol levels are temporarily reduced in response to trauma<sup>13</sup>, and after surgery<sup>14</sup>, we were interested in whether there would be a reduction in total cholesterol levels in response to microtrauma induced by eccentric activity. Therefore a second objective of this study was to examine changes in plasma cholesterol levels in response to one and two bouts of eccentric exercise.

## Subjects and methods

Twenty-six healthy, untrained men volunteered for this study. None had performed any weight training for at least 3 months before the study. Subjects were screened using a medical history form and were required to complete an informed consent. Subjects were then randomly assigned to Group 1 (performed one bout of eccentric exercise,  $n = 13$ ) or Group 2 (performed two bouts of eccentric exercise,  $n = 13$ ). For means and standard errors of physical characteristics for groups 1 and 2, see *Table 1*. A standard *t* test

Address for correspondence: Dr Lucille L. Smith PhD, Human Performance Laboratory, 371 Sports Medicine Building, Greenville, North Carolina 27858, USA

© 1994 Butterworth-Heinemann Ltd  
0306-3674/94/040267-05

Table 1. Subject demographics

	Group 1 (n = 13)	Group 2 (n = 13)
Age (years)	20.6(0.6)	21.6(0.6)
Height (cm)	178.8(1.5)	179.3(2.3)
Weight (kg)	77.8(3.6)	79.1(4.2)
Body fat (%) (five-site skinfold)	14.6(1.3)	14.7(1.8)
1 Repetition max (kg) (concentric)	68.4(3.9)	68.0(4.2)

Values are mean(s.e.)

was used to test for group differences. No significant differences ( $P > 0.05$ ) were found between the groups for any of the demographic variables.

### Strength assessments

The Cybex Eagle Chest Press Machine (Cybex, Division of Lumex, Ronkonkoma, New York, USA) was used for all strength assessments. One concentric repetition maximum (1-RM<sub>conc</sub>) was determined during a preliminary visit. Subjects warmed up by performing ten repetitions of the concentric phase of a chest press at a standard resistance of 20 kg. A 2-min rest followed the warm-up. The subject then estimated the maximum amount of weight that he could push (concentric action) through a full range of motion. The 1-RM<sub>conc</sub> was determined in three to five trials for all subjects; this was considered the baseline for subsequent strength measures.

1-RM<sub>conc</sub> was assessed at 48, 96 and 192 h after exercise. At each time the subject was required to perform a warm-up at a load equal to 30% of their 1-RM<sub>conc</sub>. After a 2-min rest the resistance was adjusted to the level recorded on the previous visit; if necessary, the resistance was increased or decreased.

### Eccentric exercise protocol

Exercise was performed on the Cybex Eagle Chest Press Machine. The eccentric exercise protocol involved performance of the eccentric, lowering phase of the chest press with two safety spotters performing the positive, lifting phase. On the day of the eccentric exercise, subjects warmed up as described in the previous section. Subjects then performed three sets of 12 repetitions of the eccentric phase of a chest press at an intensity equal to 80% of their previously determined 1-RM<sub>conc</sub>; there was a 2-min rest period between each set. This number of repetitions and intensity were selected since they represent what is commonly prescribed when an individual initiates a weight-training programme<sup>15</sup>. Group 1 performed one bout of exercise; Group 2 repeated the exercise 48 h after the initial bout. For Group 2, assessments of plasma creatine kinase, strength and muscle soreness were made before subjects performed the second bout of exercise. Subjects were informed of which group they were in, after the 48-h assessments.

### Delayed onset muscle soreness (DOMS) ratings

DOMS measurements were made before, and every 24 h following, the bout of eccentric exercise, for 8 days. Subjects were shown a soreness scale with a range of 1–10 (1 = no soreness, 10 = very sore<sup>16,17</sup>). They were instructed to palpate muscles of the chest and upper arm and assign a number between 1 and 10 that best represented their overall rating of soreness.

### Creatine kinase (CK)

CK was assessed before the initial bout of exercise, and then every 24 h for 8 days. On each occasion, upon arriving in the laboratory, subjects sat quietly for 5 min. Blood was then drawn from an antecubital vein into nontreated vacutainers. The blood (3 ml) was allowed to clot at room temperature for 10 min and centrifuged for 15 min. Serum was separated and frozen at  $-20^{\circ}\text{C}$  for subsequent analysis. Total CK was determined spectrophotometrically, in duplicate, at  $25^{\circ}\text{C}$ , using a commercially available kit (Sigma Diagnostics, St. Louis, Missouri, USA).

### Total cholesterol (TC)

TC was assessed for 13 subjects in Group 1 and 13 subjects in Group 2. A 10-ml venous blood sample was drawn into Vacutainer serum separator tubes (Becton Dickinson, Rutherford, New Jersey, USA), allowed to sit for 10 min, then centrifuged at 5000 r.p.m. for 15 min. Plasma total cholesterol was analysed using an Abbott Spectrum Chemistry Analyser (Abbott Laboratories, Abbott Park, Illinois, USA).

### Statistical analysis

All dependent variables were analysed using a repeated measures analysis of variance factorial design. Where significance was found, the least significant difference (LSD) *post-hoc* test was used. The level of significance was set at  $P < 0.05$ .

## Results

### Delayed onset muscle soreness (DOMS) ratings

No significant treatment effect ( $P = 0.548$ ) or significant treatment by time interaction ( $P = 0.962$ ) was found. A significant time effect was evident ( $P = 0.0001$ ). The LSD *post-hoc* test revealed that DOMS ratings were significantly elevated ( $P < 0.0001$ ) over baseline levels between 24 (mean(s.e.) 4.15(0.24)) and 96 h (mean(s.e.) 1.8(0.24)). Both groups showed an increase followed by a steady decrease with peak soreness occurring at 48 h after exercise (mean(s.e.) 4.7(0.24)). (See Figure 1.)

### Creatine kinase (CK)

The statistical analysis revealed no significant treatment effect between Groups 1 and 2 ( $P = 0.295$ ), or a significant treatment by time interaction ( $P = 0.074$ ).

However, there was a significant time effect ( $P = 0.0001$ ). The LSD *post-hoc* test revealed that CK was significantly elevated over baseline (mean(s.e.)  $95.16(9.90) \text{ U l}^{-1}$ ) at 48 h (mean(s.e.)  $1410.01(335.13) \text{ U l}^{-1}$ ) and 72 h (mean(s.e.)  $2361.01(339.71) \text{ U l}^{-1}$ ) after exercise, at which time CK peaked. CK remained significantly elevated through 144 h (mean(s.e.)  $1063.32(335.13) \text{ U l}^{-1}$ ). At 192 h CK was still somewhat elevated over baseline (mean(s.e.)  $348.22(335.13) \text{ U l}^{-1}$ ), but this difference was not statistically significant. (See Figure 2.)

**Strength (1-RM<sub>conc</sub>)**

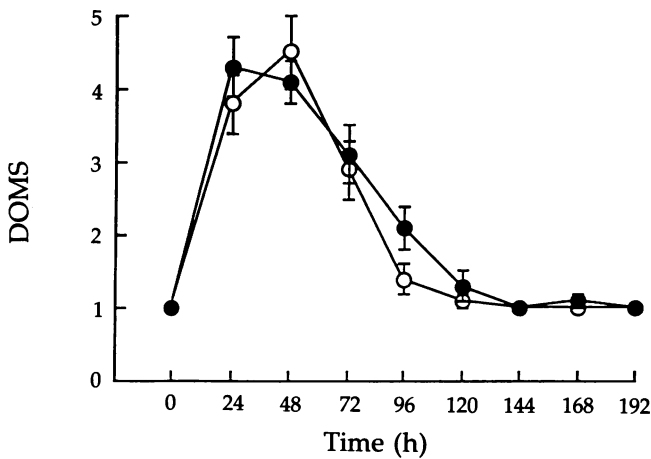
There was no significant treatment effect ( $P = 0.509$ ) or significant interaction effect ( $P = 0.106$ ) but there was a significant time effect ( $P = 0.0001$ ). The greatest reduction in 1-RM<sub>conc</sub> strength compared with baseline values occurred at 48 h after exercise in both groups ( $P = 0.0001$ ); this represented a 9% decrease in strength. Strength remained significantly depressed at 96 h after exercise by 5.1% ( $P = 0.003$ ) and at 192 h after exercise by 3.4% ( $P = 0.037$ ). (See Figure 3.)

**Total cholesterol (TC)**

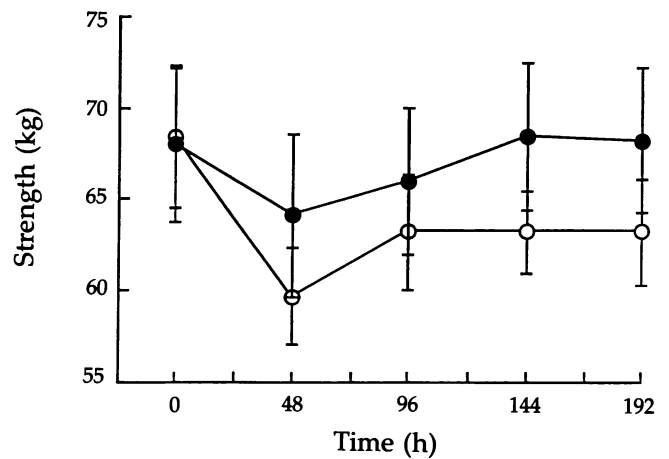
There was no significant group or interaction ( $P > 0.05$ ) effect for TC, suggesting that the two bouts of exercise did not alter the response when compared with one bout of eccentrics. However, there was a significant time effect ( $P = 0.0001$ ). The combined group mean(s.e.) values were  $171.500(5.822)$ ,  $161.231(6.015)$ ,  $158.962(5.485)$ ,  $156.600(5.922)$  and  $160.269(5.232) \text{ mg dl}^{-1}$  before exercise, and at 24, 48, 72 and 96 h after exercise, respectively. The LSD *post-hoc* test revealed that TC values were significantly lower than baseline values at 24 ( $P = 0.005$ ), 48 ( $P = 0.0001$ ), 72 ( $P = 0.0001$ ) and 96 h ( $P = 0.0001$ ) after exercise. (See Figure 4.)

**Discussion**

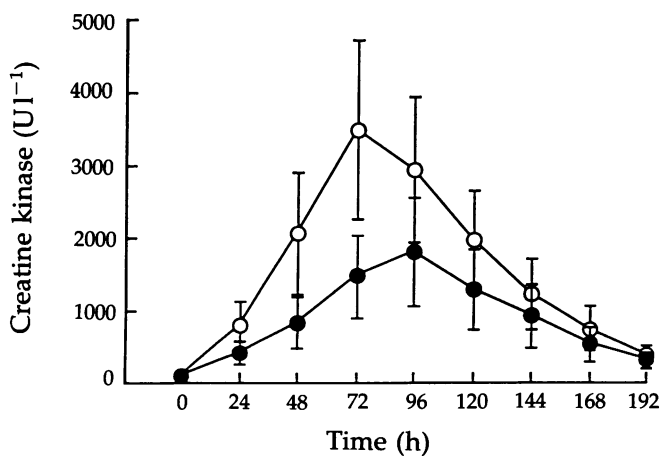
The present study investigated whether a second bout of eccentric muscle action performed 48 h after an initial bout would alter the course of delayed muscle soreness (DOMS), serum creatine kinase (CK) and maximum concentric strength (1-RM<sub>conc</sub>). The



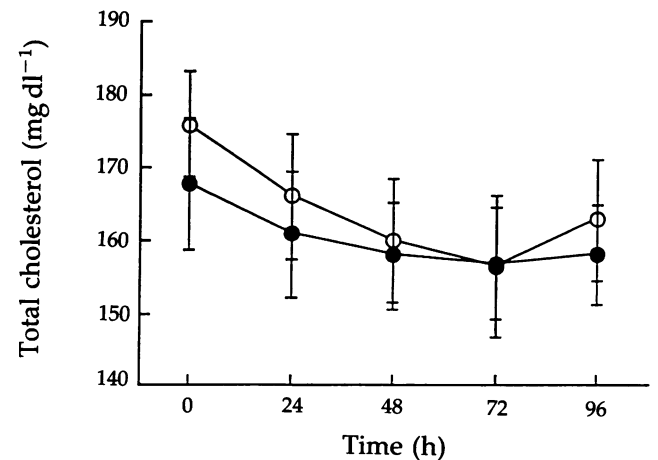
**Figure 1.** Mean(s.e.) ratings for delayed onset muscle soreness (DOMS) for Group 1 (○) and Group 2 (●), across all time periods



**Figure 3.** Mean(s.e.) of strength measurements (1-RM<sub>conc</sub>) for Group 1 (○) and Group 2 (●), across all time periods



**Figure 2.** Mean(s.e.) serum creatine kinase (CK) levels for Group 1 (○) and Group 2 (●), across all time periods



**Figure 4.** Mean(s.e.) of total cholesterol (TC) for Group 1 (○) and Group 2 (●)

results revealed no significant differences in the rating of soreness between Group 1 and Group 2, suggesting that an equivalent bout of eccentrics performed 48 h later does not increase or prolong DOMS (Figure 1). On the other hand, the second bout did not reduce the time course for DOMS, implying that an earlier resolution does not occur in response to this additional bout of eccentric exercise.

The significant time effect for CK seen in this study is similar to what has been reported previously<sup>3,4,16,17</sup>, with CK peaking several days after the eccentric bout of exercise and then gradually returning to baseline. Although there was no significant difference in CK between the two groups, it was interesting to note that Group 1 consistently had higher CK values compared with Group 2. There is considerable individual variability in CK response to eccentric exercise, with some individuals demonstrating an exaggerated response, 'high responders', and others demonstrating a reduced response, 'nonresponders', for the same bout of exercise<sup>18</sup>. Examination of the individual data in the present study did not suggest that Group 1 had a disproportionate number of high CK responders. A possible explanation for this difference is that Group 1 produced a 'truer maximum' effort during the testing for their 1-RM and consequently performed the exercise at a greater relative percentage of their maximum compared with Group 2. Group 1 also showed a consistently greater loss of strength (although not significantly greater), again suggesting that this group worked harder. Since an increase in CK after eccentrically biased exercise is taken as indirect evidence of disruption of muscle cell membrane<sup>3,4</sup>, and since there were no significant group differences, the results of this study suggest that repeating the exercise during the period of soreness produces no additional damage. However, such an interpretation should be made with caution since there is not a good correlation between serum CK and the extent of tissue injury<sup>19</sup>.

The present study revealed no significant treatment effect for strength, suggesting that a repeated bout of eccentrics performed 48 h after the initial bout, is not deleterious to 1-RM<sub>conc</sub>. There was, however, a significant time effect with the largest decrement occurring for both groups at 48 h after exercise. The literature concurs<sup>4,16,20,21</sup> that after a bout of unaccustomed eccentrics there is a reduction in strength most likely due to a decline in the inherent capacity of the muscle to produce force<sup>1,4</sup>.

Although a limited amount of research has focused on repeating eccentric exercise 48 h later, a number of researchers have studied repeated bouts spaced at longer intervals such as 5 and 14 days<sup>17</sup> and 3, 6 and 9 weeks apart<sup>22,23</sup>. All studies concur that changes in DOMS, CK and strength measures are significantly reduced after the second bout of exercise compared with changes after the first bout. Apparently, some 'adaptation' occurs in response to the initial micro-trauma and subsequent healing, which then acts to 'protect' the musculature<sup>4,16,17</sup>. It is clear that the adaptation lasts for a considerable amount of time<sup>4</sup>, but it is not known how soon after the initial bout this adaptation occurs. In the present study, if the second

bout had resulted in an earlier resolution of DOMS, CK and strength, we could have surmised that an adaptation had occurred; this was not the case. However, the fact that DOMS, CK and strength responses were not exacerbated after Group 2 repeated the exercise, suggests that the 'protective effect' might be present as early as 48 h after the initial eccentric bout.

An interesting finding of this study was the significant decrease in TC seen for both groups at 24, 48 and 72 h after exercise (Figure 4). Increased levels of blood cholesterol have been linked with a substantial increase in risk for coronary artery disease (CAD). Although cardiovascular exercise might have some beneficial lowering effects on blood lipids, there is little conclusive evidence about the relationship between muscular strengthening exercise and lipid levels<sup>24</sup>. To the best of our knowledge no strength training studies have investigated acute changes in TC in response to the eccentric component of weight training. In view of the fact that cholesterol may constitute 13% of a cell membrane<sup>12</sup>, and that signs of healing have been observed in human subjects as early as 36 h after eccentric exercise<sup>25</sup>, we suggest that the acute decrease in TC in the present study represents the diversion of cholesterol for synthesis of new cell membranes. An alternative or supplementary explanation for the acute reduction in TC could be related to exudative changes which involve the loss of plasma proteins<sup>26</sup>, since swelling, and presumably an increase in exudate, has been reported in association with DOMS<sup>9</sup>.

In conclusion, the results of this study suggest that repeating a bout of exercise during the time of DOMS will not influence the time course of DOMS, serum CK, or strength decrements. Whether or not it might be beneficial or detrimental in terms of other variables, such as factors related to the healing process<sup>13,14</sup> is presently not known.

## References

- 1 Newham DJ, Mills KR, Quigley, BM, Edwards RHT. Pain and fatigue after concentric and eccentric muscle contractions. *Clin Sci* 1983; **64**: 55–62.
- 2 Armstrong RB. Mechanisms of exercise induced delayed onset muscular soreness: a brief review. *Med Sci Sports Exerc* 1984; **16**: 529–38.
- 3 Armstrong RB. Muscle damage and endurance events. *Sports Med* 1986; **3**: 370–381.
- 4 Ebbeling CB, Clarkson PM. Exercise induced muscle damage and adaptation. *Sports Med* 1989; **7**: 207–34.
- 5 Clarkson PM, Fritz VK, Stauber WT. Extracellular matrix disruption in human muscle resulting from eccentric muscle action. (Abstract) *Med Sci Sports Exerc* 1989; **21**(Suppl): S80.
- 6 Newham DJ, Jones DA, Tolfree SEJ, Edwards RHT. Skeletal muscle damage: a study of isotope uptake, enzyme efflux and pain after stepping. *Eur J Appl Physiol* 1986; **55**: 106–12.
- 7 Fritz VK, Stauber WT. Characterization of muscles injured by forced lengthening. II. Proteoglycans. *Med Sci Sports Exerc* 1988; **20**: 354–61.
- 8 Newham DJ, McPhail G, Mills KR, Edwards RHT. Ultrastructural changes after concentric and eccentric contractions of human muscle. *J Neur Sci* 1983; **61**: 109–22.
- 9 Friden J, Sfikianos PN, Hargens AR, Akeson WH. Residual muscular swelling after repetitive eccentric contractions. *J Orthopaedic Res* 1988; **6**: 493–8.
- 10 Leadbetter WB. Cell matrix response in tendon injury. *Clinics Sports Med* 1992; **11**: 533–78.

- 11 Peacock EE. *Wound Repair*. 3rd ed. Philadelphia, USA: W. B. Saunders, 1984: 1–14.
- 12 Guyton AC. *Text Book of Medical Physiology*. 6th ed. Philadelphia, USA: W. B. Saunders, 1981: 50.
- 13 Carlson LA, Holmquist L, Lindholm M. Plasma lipid metabolism and trauma: appearance of characteristic apolipoproteins in high density lipoproteins. *Acta Chir Scand* 1985; **522**: 87–106.
- 14 Man EB, Bettcher PG, Cameron CM, Peters JP. Plasma amino acids, nitrogen and serum lipids of surgical patients. *Clin Invest* 1946; **25**: 701–8.
- 15 Dons V, Bollerup K, Bonde-Peterson F, Hancke S. The effect of weight-lifting exercise related to muscle fiber composition and muscle cross-sectional area in humans. *Eur J Appl Physiol* 1979; **40**: 95–106.
- 16 Clarkson PM, Tremblay I. Exercise-induced muscle damage, repair, and adaptation in humans. *J Appl Physiol* 1988; **65**: 1–6.
- 17 Ebbeling CB, Clarkson PM. Muscle adaptation prior to recovery following eccentric exercise. *Eur J Appl Physiol* 1990; **60**: 26–31.
- 18 Newham DJ, Jones DA, Edwards RHT. Large delayed plasma creatine kinase changes after stepping exercise. *Muscle Nerve* 1983; **6**: 380–5.
- 19 Van Der Meulen JH, Kuipers H, Drukker J. Relationship between exercise-induced muscle damage and enzyme release in rats. *J Appl Physiol* 1991; **71**: 999–1004.
- 20 Francis K, Hoobler T. Delayed onset muscle soreness and decreased isokinetic strength. *J Appl Sports Sci Res* 1988; **2**: 20–3.
- 21 Talag TS. Residual muscular soreness as influenced by concentric, eccentric and static contractions. *Research Quarterly* 1972; **44**: 458–69.
- 22 Byrnes WC, Clarkson PM, White JS et al. Delayed onset muscle soreness following repeated bouts of downhill running. *J Appl Physiol* 1985; **59**: 710–15.
- 23 Triffletti P, Litchfield PE, Clarkson PM, Byrnes WC. Creatine kinase and muscle soreness after repeated isometric exercise. *Med Sci Sports Exerc* 1988; **20**: 242–8.
- 24 Kokkinos PF, Hurley BF. Strength training and lipoprotein–lipid profiles. *Sports Med* 1990; **9**: 266–72.
- 25 Friden J, Sjostrom M, Ekblom B. Myofibrillar damage following intense eccentric exercise in man. *Int J Sports Med* 1983; **4**: 170–6.
- 26 Keele KD, Stern PRS. Serum lipid changes in relation to pain. *J R Coll Physicians Lond* 1973; **7**: 319–29.

## Announcement

---

### Relocation of Journal Publishing Office

As from 1st October 1994, the publishing, editorial, production and reprint offices are moving to:

Elsevier Science Ltd  
The Boulevard, Langford Lane,  
Kidlington, Oxford, OX5 1GB, UK  
Main switchboard:  
Tel: +44 (0)1865 843000  
Fax: +44 (0)1865 843010

All correspondence and queries relating to the journal should be sent to this address

Newly submitted manuscripts should be mailed to the Editor

---