



# IAAF @-Letter

## for CECS Level II Coaches

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SPECIFIC THEME: Avoidance and treatment of muscle soreness

GENERAL THEME: Physiological basis of muscle soreness

### Specific Theme

#### AVOIDANCE AND TREATMENT OF MUSCLE SORENESS

##### 1 Introduction

Muscle soreness is an unavoidable side effect of hard training. The only way to avoid post-exercise muscle soreness completely is to avoid exercise.

However, according to the available advisory literature, there are several things one can do to minimize muscle soreness – caused mainly by damage to muscle proteins – without sacrificing fitness. Common tips are the following (Quinn, 2004):

- Warm up thoroughly before activity.
- Cool down completely after exercise.

- Perform easy stretching before training.
- Perform thorough flexibility exercises after training, while the muscles are warm.
- Start with easy to moderate activity and build up your intensity over time.
- Avoid making sudden major changes in the type of exercise you do and in the amount of time that you exercise.

Other typical recommendations for treatment include topical application of athletic balms, creams, and/or ice, submersion in hot baths, exposure to a sauna, and the use of aspirin or other anti-inflammatory drugs (Clarkson, 1996).

A lot of research has been done to examine the effects of these measures.

## **2 Frequently recommended forms of treatment for DOMS and their effects**

### *2.1 Exercise and Stretching*

Exercise is often considered to reduce the effects of muscle soreness. Donnelly, Clarkson and Maughan (1988) investigated the effect of a light bout of eccentric exercise 1 day after heavy eccentric exercise consisting of 70 maximum eccentric contractions of the forearm flexor and extensor muscles of the non-dominant arm. The experimental group of nine subjects performed 25 submaximal contractions with the same arm. Although there was no difference in muscle soreness between the two groups 19 days after exercise, there was a significant reduction in creatine kinase enzyme outflow on days 2-6 for the experimental group (creatine kinase is a commonly used marker of muscle damage).

It is not clear why eccentric training has this protective effect but it is possible that an eccentric workout 'teaches' the nervous system to redistribute potentially damaging forces over a larger number of fibers within a muscle, lessening the stress and strain on individual muscle cells.

A study by Hasson, Barnes, Hunter and Williams (1989) found that a high-velocity concentric isokinetic exercise (6 x 20 maximal voluntary contractions of the knee flexors and extensors at 5.23 rad/sec, with 3 min recovery between each set) performed 24 hours after soreness-producing stepping exercise, significantly reduced muscle soreness and facilitated the return of strength after 48

hours in five subjects when compared with a control group. Nevertheless, the results showed that the soreness and strength loss were still significantly above baseline levels at 48 hours.

Stretching is often advocated for the prevention of post-exercise soreness. The widespread belief that stretching alleviates muscle soreness is based on studies as that conducted by de Vries (1961), in which stretching exercises are reported to have helped to relieve the pain in seven of nine subjects. Abraham (1977) investigated the acute effects of stretching, too, and demonstrated immediate relief from soreness. However, later studies could not confirm these findings. In a study conducted by Buroker and Schwane (1989), 23 subjects performed a 30-minute step test to induce delayed-onset muscle soreness (DOMS) in the eccentrically contracting thigh and calf muscles. A regimen of static stretching performed after the test did not alleviate DOMS; there was neither temporary relief of pain immediately after stretching nor a general reduction in pain during the three-day post-exercise period. Serum creatine kinase level – a commonly used marker of muscle damage, as already mentioned above – was elevated after exercise and the strength of the sore thigh muscles was reduced; stretching did not effect these responses. These results contradict claims of the benefits of static stretching for relief of exercise-induced DOMS.

The results of a similar study conducted by Wessel and Wan (1994) also indicated that neither stretching before nor stretching after pain-

producing exercise had any effect on the post-exercise pain (see also the studies by High & Howley, 1989, and Lund, Vestergaard-Poulsen, Kanstrup & Sejrsen, 1998).

However, these results do not prove that stretching is never effective in alleviating DOMS. They only indicate that stretching does not *always* reduce soreness. It is clear that, even if stretching has limited effects on DOMS, it is still important in achieving and maintaining flexibility and appropriate lengths of muscle-tendon units.

### 2.2 Cold application (cryotherapy)

Cryotherapy, the application of cold, is widely regarded as an effective, easy to use, and inexpensive treatment modality for traumatic soft-tissue injury. Benefits attributed to cryotherapy use following injury include a diminution of the inflammatory response, pain reduction, reduced edema formation and a decrease in secondary hypoxic cell death.

If cryotherapy can reduce the severity of the initial inflammatory response and promote recovery following traumatic injury, it may also facilitate recovery of muscle tissue experiencing DOMS, i. e. damage and inflammatory changes associated with eccentric exercise. However, this theory has not been supported by the limited number of studies conducted to date (Paddon-Jones & Quigley, 1997, p. 588).

For example, Isabell, Durant, Myrer and Anderson (1992) investigated the effects of ice massage, ice massage with exercise, and exercise on the prevention and treatment of DOMS.

The authors arrived at the conclusion that the therapeutic use of ice and exercise, combined or used separately, was not effective in reducing the symptoms of DOMS. Though not statistically significant, the patterns in the data suggest that ice application may even be contraindicated in the treatment of DOMS.

### 2.3 Hydrotherapy

Hydrotherapy, the external application of water to the body for therapeutic purposes, is an example of superficial heating or cooling. During whirlpool therapy, heating and cooling of tissues occurs through conduction. Warm and cold whirlpools, administered at temperatures between 35.0°C and 43.3°C and 12.8°C and 18.3°C, respectively, for 20 to 30 minutes have been believed to be especially useful to decrease swelling, muscle spasm, and pain. Additionally, contrast therapy, the cyclical alternation of hot and cold whirlpool immersions, has been cited as decreasing symptoms of the inflammatory process.

Against this background, Kuligowski, Lephart, Giannantonio and Blanc (1998) measured the effects of warm whirlpool, cold whirlpool, and contrast therapy on the elbow flexors of subjects experiencing DOMS, as defined by the dependent variables of resting elbow flexion (REF), active elbow flexion, perceived soreness, and maximal voluntary isometric contraction. The results of the investigation indicate that both cold whirlpool and contrast therapy were effective in treating DOMS across time for dependent variables REF and pain. Cold

whirlpool and contrast therapy returned REF values closer to pre-exercise values than warm whirlpool or no treatment. Additionally, warm whirlpool was found to significantly return REF values to pre-exercise values over no treatment.

The rationale for using contrast therapy in the inflammatory process is based upon a theory that alternative cycles of warm and cold whirlpool immersions cause vasoconstriction and vasodilatation, commonly referred to as a 'pumping action'.

Based on the results of their study, Kuligowski et al. (1998, p. 228) conclude that the application of cold through the use of cold whirlpool or contrast therapy is the best treatment for DOMS.

#### 2.4 Post-exercise massage treatment

Massage has also been used in an attempt to reduce DOMS. However, Wenos, Brilla and Morrison (1990) did not find any significant difference in soreness or in strength loss, post-exercise, in the quadriceps muscles of the treatment leg when compared with the control leg in a group of nine subjects (Cleak & Eston, 1992, p. 336). In a systematic review of the available literature dealing with the effects of massage on DOMS, Ernst (1998, p. 214) arrives at the following conclusion:

Even though massage has some potential in reducing the symptoms of DOMS, its effectiveness has not been demonstrated convincingly.

#### 2.5 Effects of aspirin and ibuprofen

Aspirin is a known anti-inflammatory agent that suppresses the synthesis of prostaglandins. Prostaglandins act as potentiators of inflammatory substances like histamine and bradykinin. As inflammation may be a contributing factor in DOMS, Francis and Hoobler (1987) investigated the effects of aspirin on DOMS. Immediately prior to 24 and 48 hours following fatiguing eccentric exercise of the elbow flexor muscles, the perception of soreness, elbow extension, and maximal flexor force was determined in 20 subjects who had been equally divided into two groups. The groups consisted of a control group and a group that received aspirin. There were no differences in soreness 'between' the two groups at 24 hours whereas the mean soreness score of the aspirin group was approximately 25% less than the mean control group's score at 48 hours. Whereas both groups exhibited a significant decrease in elbow extension at both 24 and 48 hours in comparison to pre-exercise values, the group receiving aspirin exhibited approximately 50% less change in extension at each time period than did the control group. Francis and Hoobler (1987) arrive at the following conclusion:

The soreness reduction in the group receiving aspirin may be related to the known function of aspirin inhibiting prostaglandin synthesis and release.

Another powerful anti-inflammatory drug is ibuprofen. Ibuprofen interferes with the metabolism of arachidonic acid (an unsaturated fatty acid, usually essential in human nutrition, which is the biological precursor of

the prostaglandins) by inhibiting the enzyme cyclooxygenase (synonymous with prostaglandin endoperoxide synthase). This blocks the production of endoperoxides and the inflammation mediating prostaglandins PGE<sub>2</sub> and PGF<sub>2A</sub>. Ibuprofen also has an effect on the kinin and histamine systems mediating inflammation. The effect of ibuprofen on these systems is similar to that on inhibition of prostaglandin formation.

Ibuprofen has become one drug of choice for muscle and joint pain because of the powerful effect on inhibiting inflammatory precursors.

In a study by Hasson, Daniels, Divine, Niebuhr, Richmond, Stein and Williams (1993), 20 subjects were randomly assigned to (1) prophylactic ibuprofen (400 mg three times a day initiated 4 hours before collection of baseline data and a strenuous eccentric exercise bout), (2) therapeutic ibuprofen (400 mg three times a day initiated 24 hours after baseline), (3) placebo, or (4) control. Muscle soreness perception, plasma creatine kinase, knee extensor torque, and EMG of the quadriceps were evaluated at baseline, 24, and 48 hours. The prophylactic ibuprofen group had between 40 and 50% less muscle soreness perception and significantly less decline in isometric, concentric, and eccentric torque at 24 hours compared with the other three groups. At 48 hours both prophylactic and therapeutic ibuprofen had significantly less muscle soreness perception and decline in torque than the placebo and control groups. There was no difference between the amount of muscle damage between the four groups at 24 and 48 hours. Vastus

medialis and lateralis EMG magnitude decreased across time. Vastus lateralis EMG magnitude had significantly less decline from baseline for prophylactic ibuprofen compared with the other three treatments at 24 hours, while both prophylactic and therapeutic ibuprofen had significantly less decline at 48 hours. These data indicate that a prophylactic dosage of ibuprofen does not prevent creatine kinase release from muscle, but does decrease muscle soreness perception and may assist in restoring muscle function (Hasson et al. 1993, p. 9).

Starting from the suggestion that muscle soreness is related to an inflammatory response and that prostaglandins play a role in the inflammatory process, Kuipers, Keizer, Verstappen and Costill (1985) investigated the influence of a cyclooxygenase-inhibiting drug (flurbiprofen) on the subjective symptoms of soreness and eventual structural changes in six male subjects. The subjects performed one concentric and two eccentric work bouts of 30 min at 80% of the individual maximal workload on the bicycle ergometer. Muscle biopsy taken before, immediately after, and 24 hours after work were used to examine structural and ultrastructural changes as well as for assessment of glycogen content. Plasma levels of muscle enzymes and subjective soreness were determined at regular intervals. Eccentric work elicited muscle soreness in all subjects. However, the soreness was consistently less in the second eccentric trial. No significant enzyme release was noticed in any of the subjects, whereas ultrastructural changes were restricted to the mitochondria.

No influence of flurbiprofen on subjective soreness was noticed. After both eccentric trials muscle glycogen was lower 24 hours after work compared to the content immediately after work. According to Kuipers et al. (1985), these results suggest that eccentric exercise interferes with glycogen synthesis and that prostaglandins do not play a major role in exercise-induced muscle soreness.

That ibuprofen is not an appropriate treatment for DOMS is indicated by the results of a study conducted by Donnelly, Maughan and Whiting (1990). In this study, 32 volunteers participated in a two-period crossover study in which ibuprofen was tested against an identical placebo for its effectiveness in reducing muscle soreness and damage after two bouts of downhill running. Subjective soreness, quadriceps isometric strength and isometric endurance time at 50% of maximum strength, serum activities of creatine kinase, lactate dehydrogenase and aspartate transaminase and serum levels of creatinine and urea were recorded at intervals up to 72 hours after exercise. Each downhill run produced muscle soreness, and a decline in muscle strength and 50% endurance time, although these parameters were unaffected by ibuprofen treatment. All serum parameters measured increased after both runs, but for the three enzymes this increase was smaller after the second run. Serum creatine kinase and urea levels were higher in the ibuprofen group after both runs.

### 2.5 Summary

Although some success has been reported by a few authors using light exercise, hydrotherapy, or topical anti-inflammatory drugs to alleviate DOMS, the majority of studies indicate that no convincingly effective way has yet been found to reduce the soreness once it has occurred. Therefore, in general, prevention seems to be better than cure (Cleak & Eston, 1992, p. 337).

## 3 The prevention of muscle soreness

The prevention of muscle soreness is important for maximizing training gains. The eccentric component of muscle action should be minimized during early training, but this is not possible for athletes in most sports.

An alternative approach is to start training at a very low intensity and progress slowly through the first few weeks. Yet another approach is to initiate the training program with a high-intensity, exhaustive training bout. Muscle soreness would be great for the first few days, but some evidence suggests that subsequent training bouts would cause considerably less muscle soreness (Ebbeling & Clarkson, 1989; Wilmore & Costill, 2004, p. 103).

## **Classification of Muscle Soreness**

(according to Noakes, 2003, p. 502)

<b>Grading</b>	<b>Symptoms</b>	<b>Indication</b>
<b>0</b>	<b>No discomfort</b>	<b>Continue training</b>
<b>1</b>	<b>Some discomfort on feeling muscle</b>	<b>Reduce training for 7 days</b>
		<b>No racing for 2 weeks</b>
<b>2</b>	<b>Discomfort on walking</b>	<b>Reduce training for 14 days</b>
	<b>Unable to squat without discomfort</b>	<b>No racing for 1 month</b>
<b>3</b>	<b>Severe pain</b>	<b>Reduce training for at least 1 month</b>
	<b>Walking with difficulty</b>	<b>No racing for 2 months</b>

## General Theme

### PHYSIOLOGICAL BASIS OF MUSCLE SORENESS

#### 1 Introduction

Athletes in hard training often report considerable soreness in their lower limbs, usually a day after intense, fast-paced running sessions or following back-to-back very hard training days or after a hard race. The longer and more intense the effort, the greater the chance for such discomfort. However, the same soreness can occur if athletes suddenly begin a hard training cycle without adequate transition from moderately intense training (Martin & Coe, 1997, p. 394).

According to Wilmore and Costill (2004, p. 99), muscle soreness can generally be present

- during the latter stages of an exercise bout and the immediate recovery period,
- between 12 and 48 hours after a strenuous bout of exercise, or
- at both times.

#### 2 Acute muscle soreness

Pain felt during and immediately after exercise can result from accumulation of the end products of exercise, such as lactate, and tissue edema, that is caused by a fluid shifting from the blood plasma into the tissues. This is the pumped-up feeling that the athlete is conscious of following heavy endurance or strength training. This pain and soreness usually disappears within a few minutes to several hours

after the exercise. Thus it is often referred to as *acute muscle soreness*.

Acute muscle soreness occurs late in an exercise bout and during the immediate recovery period.

#### 3 Delayed-onset muscle soreness

Muscle soreness which does not occur immediately but is felt a day or two after a heavy bout of exercise is referred to as *delayed-onset muscle soreness* (DOMS). None of the theories that attempt to explain this form of muscle soreness yet has universal support.

Almost all current theories acknowledge that eccentric action is the primary initiator of this form of muscle soreness. Examples of eccentric tension include running downhill, stepping down, or reverse bicycle pedaling. Eccentric tension is an integral part of running even on level ground, occurring with every stride as the gastrosoleus, anterior and posterior tibialis, and quadriceps absorb much of the impact forces placed on the knee joint and foot at footstrike (Martin & Coe, 1997, p. 396).

Delayed-onset muscle soreness (DOMS) occurs a day or two after the exercise bout. DOMS results primarily from eccentric action and is associated with actual muscle damage.

The fact that lengthening (eccentric) tension generation seems more prone to causing DOMS than shortening (concentric) tension generation – although both can do it (Ebbeling & Clarkson, 1989; Schwane, Watrous, Johnson & Armstrong, 1983) – was initially observed in a study that inves-

tigated the relationship of muscle soreness to eccentric, concentric, and static actions. It found that a group training solely with eccentric actions experienced extreme muscle soreness, while the static and concentric action groups experienced little (Tallag, 1973). This was further explored in studies where subjects were asked to run on a treadmill for 45 min on two separate days, one day on a level grade and the other day on a 10% downhill grade (Schwane, Johnson, Vandenakker & Armstrong, 1983; Schwane, Watrous, Johnson & Armstrong, 1983). No muscle soreness was associated with the level running. But the downhill running, which required extensive eccentric action, resulted in considerable soreness within 24-48 hours, even though the blood lactate levels, previously thought to cause muscle soreness, were much higher with level running.

A number of questions obtrude themselves in this context:

- Is the painful combination of tenderness and stiffness, which is characteristic of DOMS indicative of actual tissue injury?
- If so, are the muscles or the connective tissues (or both) affected?
- Is inflammation present?
- Because such soreness may be nearly unavoidable after racing efforts, should training that produces such soreness be avoided at all costs?

These and other questions have characterized quite active recent investigation of the problem, although scientists have studied the phenomenon for a long time (Martin & Coe, p. 394).

#### **4 Explanations for exercise-induced delayed-onset muscle damage**

##### *4.1 Lactic acid theory*

As already indicated above, it was first thought that lactic acid accumulation with severe exercise was responsible for the muscle soreness experienced acutely with exercise as well as the delayed soreness experienced 2-3 days following exercise (Asmussen, 1956). This is still widely believed among lay persons in the exercise community, even though a number of observations indirectly indicate that this is not the case.

In the studies conducted by Watrous, Armstrong and Schwane (1981), Schwane, Johnson, Vandenakker and Armstrong (1981), and Schwane, Watrous, Johnson and Armstrong (1983), the investigators ran subjects 45 minutes on a treadmill at both 0 and -10° incline at 78 and 58%  $VO_2$ max, respectively, to study the correlation of delayed soreness and plasma lactate dehydrogenase (LHD) activity. Blood lactic acid concentration and subjective sensations of muscular soreness were assessed at intervals for 72 hours after the runs. Results:

Lactic acid concentration was significantly increased during running on the level, but subjects experienced no significant post-exercise muscular soreness. Lactic acid was never elevated in downhill runners, but subjects experienced significant delayed-onset soreness. These results show that lactic acid is not related to exercise-induced delayed-onset muscle soreness (Schwane, Watrous, Johnson & Armstrong, 1983, p. 124; Francis, 1983, p. 11).

#### 4.2 Torn tissue theory

More than 90 years ago, the notion was advanced by Hough (1902) that hard work in muscle unadapted to such loads caused a microscopic tearing or rupturing of the cells. This could, of course, involve damage to the muscle cells, to their associated connective tissues, or to both. The soreness may occur all along the involved muscles and is often greatest near the muscle-tendon junctions. Newham, Mills, Quigley and Edwards (1982) have suggested that at these junctions the long axes of muscle fibers are at least parallel to the long axis of the entire muscle. Also, pain receptors are very common in the tendons and connective tissue. In several of the long lower-limb muscles, the tendons, instead of being restricted to the ends of the muscle, extend a considerable distance along the muscle to which they connect. During soreness the tension-generating abilities of the muscle cells are reduced (Francis & Hoobler, 1988). According to Newham, Mills, Quigley and Edwards (1983) more muscle cells than previously (i.e., be-

fore the onset of muscle soreness) will need recruiting to achieve a given level of force output. This may explain why racing effectiveness (or continuous fast-paced work) is so difficult during such a period. Clearly, tissue breakdown has been extensive, involving both skeletal muscle cells and connective tissue (Martin & Coe, 1997, p. 395).

The presence of muscle enzymes in blood after intense exercise suggests that some structural damage may occur in the muscle membranes. It has been reported that these enzymes increase from 2-10 times their normal levels following bouts of heavy training. Recent studies support the idea that these changes might indicate various degrees of muscle tissue breakdown. Examination of tissue from the leg muscles of marathon runners has revealed remarkable damage to the muscle fibers after both training and marathon competition (Hagerman, Hikida, Staron, Sherman & Costill, 1984). The onset and timing of these muscle changes parallels the degree of muscle soreness experienced by the runners.

Although the effects of muscle damage on performance are not fully understood, experts generally agree that this damage is, in part, responsible for the localized muscle pain, tenderness, and swelling associated with DOMS.

However, blood enzyme levels might rise and muscle fibers might be damaged frequently during daily exercise that produces no muscle soreness.

#### 4.3 Inflammatory reaction

White blood cells serve as a defense against foreign materials that enter the body or against conditions that threaten the normal function of its tissues. The white blood cell count tends to rise following activities that induce muscle soreness. This led some investigators to suggest that soreness results from inflammatory reactions in the muscle. But the link between these reactions and muscle soreness has been difficult to establish.

Researchers have tried to use drugs to block the inflammatory reaction, but the success of these efforts in reducing either the amount of muscle soreness or the degree of inflammation has been doubtful (see pp. 4-6 of this *@-Letter*). Because both remain, conclusions about the role of inflammation in muscle soreness cannot be drawn from this research. More recent studies, however, are beginning to establish a link between muscle soreness and inflammation.

Proposed causes of DOMS include structural damage to muscle cells and inflammatory reactions within the muscles.

#### 5 Sequence of events in DOMS

In 1984, Armstrong conducted a review of possible mechanisms for exercise-induced DOMS. He has surmised that the debilitation of muscular force in connection with DOMS occurs because of an increased  $\text{Ca}^{2+}$  ion level in the muscle cells. Cell membrane damage from intense activity permits more  $\text{Ca}^{2+}$  ions to diffuse inward (because their concentration

outside these cells is greater than that inside). An elevated cell  $\text{Ca}^{2+}$  level inhibits the rate at which Krebs cycle enzymes permit fuel breakdown. The ionic disruption is entirely transitory, thanks to homeostatic process that permit complete regeneration of membrane integrity during the recovery period. Calcium entry is restored to an acceptable rate, and fuel metabolism proceeds normally (Martin & Coe, 1997, p. 395).

Armstrong concluded that DOMS is associated with

- elevations in plasma enzymes,
- presence of myoglobin in the blood (myoglobinemia), and
- abnormal muscle histology and ultrastructure.

He developed a model of DOMS that proposed the following sequence of events:

- (1) High tension in the contractile-elastic system of muscle results in structural damage to the muscle and its cell membrane.
- (2) The cell membrane damage disturbs calcium homeostasis in the injured fiber, resulting in necrosis (cell death) that peaks about 48 hours after exercise.
- (3) The products of macrophage activity and intracellular contents (such as histamine, kinins, and  $\text{K}^+$ ) accumulate outside the cells. These substances then stimulate the free nerve endings in the muscle. This process appears to be accentuated in eccentric exercise, in which large forces are distributed over relatively small

cross-sectional areas of the muscle.

Recent comprehensive reviews have provided much greater insight into the cause of muscle soreness.

Researchers now are confident that muscle soreness results from injury or damage to the muscle itself, generally the muscle fiber, and possibly the sarcolemma (Armstrong, Warren & Warren, 1991).

This damage sets up a chain of events that includes the release of

intracellular proteins and an increase in muscle protein turnover. The damage and repair process involves calcium ions, lysosomes, connective tissue, free radicals, energy sources, inflammatory reactions, and intracellular and myofibrillar proteins. But the precise cause of skeletal muscle damage and the mechanisms of repair are not well understood. Some evidence suggests that this process is an important step in muscle hypertrophy (Wilmore & Costill, 2004, p. 102).

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