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The effects of ibuprofen and vitamin E on delayed onset muscle soreness and performance

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THE EFFECTS OF IBUPROFEN AND VITAMIN E ON DELAYED ONSET MUSCLE SORENESS AND PERFORMANCE

by

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An Abstract

of a thesis submitted in partial fulfillment

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ABSTRACT

Through independent but unconfirmed physiological mechanisms, ibuprofen (IB) and vitamin E (E) are each proposed to potentially attenuate delayed onset muscular soreness (DOMS) that often accompanies novel eccentric exercise. The purpose of this study was to determine whether or not the use of IB or E reduces the rating of soreness (RS) associated with DOMS or affects the decline in muscular performance that usually accompanies DOMS. A secondary purpose was to determine the accuracy of each group's perception of performance (PP) following the onset of DOMS. It may be hypothesized that if IB or E alter perception during DOMS, then susceptibility to muscle injury upon subsequent performance could also be impacted. Twenty-nine college-aged females were randomly assigned in double-blind fashion to either an E (800 IU/d), IB (1200 mg/d), or placebo (P) group. Administration of E or P began 14 days prior to and continued throughout the 5-day testing period, while IB began 2 days prior to and continued throughout the testing period. Baseline measurements consisted of maximal isometric, eccentric, and concentric contractions of the quadriceps and hamstrings on a Biodex isokinetic dynamometer. Immediately after baseline testing,

subjects performed a series of intense squats to induce muscular soreness in the upper legs. RS and PP were assessed daily with Likert-type scales. The four subsequent testing days were used for comparison to the baseline, and to examine differences between conditions. Multivariate mixed models were used to compare the three groups on selected rise time (RT), peak torque (PT), and time to fatigue (TTF) measurements across the five testing days. RS and PP measures were analyzed with a mixed model ANOVA. Analysis of RT was not significant indicating that all groups achieved PT in a similar fashion on each day. The group x day interaction for PT was significant ($p<.05$), however, the analyses of simple group effects were not significant for each day. The time main effect was significant between Days 2 and 5, indicating a drop in muscular performance following the occurrance of DOMS. Analysis of TTF revealed both a significant interaction and time main effect ($p<.05$). Simple effects for groups at days showed significant differences between the groups only at Day 1, however, follow up contrasts showed no significant differences between the groups at Day 1. Bonferroni contrasts for time main effect also showed no significant differences between mean scores on Days 2 and 3, 2 and 4, or 2 and 5.

A mixed model ANOVA for PP showed a significant time main effect ($p<.05$), and follow-up contrasts showed significant differences between mean scores on Days 2 and 4, and Days 2 and 5 ($p<.05$). The IB group's PP tended to be consistently lower than E or P, however, this trend did not reach statistical significance ($p=.083$). Analysis of RS showed no group differences, but a time main effect revealed that soreness peaked by 48 h postexercise and began to recede thereafter. The use of IB or E does not appear to enhance the perception of performance nor does it appear to have an ergogenic effect during DOMS.

Ithaca College

Division of Health, Physical Education, and Recreation

Ithaca, New York

CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that the Master of Science Thesis of

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submitted in partial fulfillment of the requirements for the degree of Master of Science in the Division of Health, Physical Education, and Recreation at Ithaca College has been approved.

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DEDICATION

This thesis is dedicated to my family-

Theodore, the late Janet Kofod,

Lauren and Karen

for their constant support

throughout my life

in the pursuit of my goals.

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Chapter 1

INTRODUCTION

Delayed onset muscle soreness (DOMS) is defined as a dull, aching pain in the skeletal muscles that develops 24 to 48 hours following unaccustomed activity, and can last from 5 to 7 days (Armstrong, 1984). The mechanisms responsible for the perception of pain related to the soreness have not been clearly identified, although the concept, etiology, and mechanisms were first introduced nearly a century ago (Hough, 1902).

Almost everyone has experienced muscle soreness at some time, especially after the first or second day of a new exercise program. Through independent though unconfirmed physiological mechanisms, ibuprofen and vitamin E are each proposed to potentially diminish DOMS that often accompanies novel eccentric exercise. The questions remain, are ibuprofen and vitamin E effective in reducing DOMS? And, will these substances reduce the perception of pain and help to maintain normal muscular performance in a person experiencing DOMS?

This study was designed to investigate if a nonsteroidal anti-inflammatory drug (NSAID), such as ibuprofen, would decrease pain associated with DOMS or reverse the decline in muscular performance that usually accompanies DOMS. In addition, the recent suggestion that vitamin E may serve as an antioxidant protecting against free radical induced damage to the muscle cell membrane, and possibly against soreness, was also investigated (Jenkins, 1983). A secondary purpose of the study was to determine the ibuprofen, vitamin E, and placebo groups' perception of performance ability following the onset of DOMS.

Scope of the Problem

This study was conducted to investigate whether the use of ibuprofen (IB) or vitamin E (E) has a positive effect on reducing muscle soreness and/or the associated decline in muscular performance that occurs with unaccustomed resistive exercises. Twenty-nine female undergraduates at Ithaca College volunteered to serve as subjects. They were divided into 3 groups, an IB, an E, and a placebo (P) group. The E and P groups began ingestion of the designated dosage 2 weeks prior to testing, while the IB group began 2 days before

testing. All three groups continued ingestion throughout the five testing days. On the first day of testing, all groups performed a series of maximal resistive exercises on the Biodex (to gather baseline data), followed by a routine of near maximal squat exercises designed to induce DOMS. For the following 4 days, muscular performance was determined by repeating the maximal resistive exercises on the Biodex. Ratings of soreness and perception of performance scales were given prior to initial testing, and before muscular performance testing on each subsequent day.

Statement of the Problem

This study was conducted to determine whether use of IB or E prior to and following unaccustomed exercise reduced the perception of pain or the decline in muscular performance related to DOMS. It was also determined whether a relationship existed between measures of perceived performance and perceived sensations of soreness.

Hypotheses

The null hypotheses of the study were as follows:

1. Ho: Muscular performances on the Biodex do not differ significantly among E, IB, and P subjects

following unaccustomed squat exercises.

2. Ho: Perception of soreness and performance scales do not differ significantly among E, IB, and P subjects in response to unaccustomed squat exercises.

Assumptions of Study

The following were assumptions of the study:

1. The subjects used maximum effort during the maximal resistive exercises.

2. The protocol used in this study was sufficient to induce muscle soreness in the target muscle groups.

3. The placebo group was representative of both the E and IB groups.

4. The subjects used IB, E, or P as prescribed throughout the study.

Definition of Terms

The following terms are defined for the purpose of this study:

1. Isokinetic: The speed of limb rotation is performed at a constant value throughout the range of motion during the exercise (Kreighbaum & Barthels, 1985).

2. One repetition maximum (1 RM): the weight of the load that can be lifted one time only.

3. Maximal resistive exercise: All out effort during contraction of a muscle.

4. Untrained subjects: Women who have not regularly strength trained their lower body for 3 months prior to the study.

Delimitations of Study

The delimitations of the study were as follows:

1. Twenty-nine female undergraduates from Ithaca College were recruited as subjects.

2. Only non-smoking, untrained subjects were selected for participation in the study.

3. Following a brief warm-up, five sets of 10 repetition squats at 80% of 1 RM were the only exercises used to induce soreness.

4. The perception of performance and soreness scales were the only subjective measures used in the study.

5. Muscular performances on the Biodex were the only objective measures used in the study.

Limitations of Study

The limitations of the study were as follows:

1. The subjects used may not be representative of the total female undergraduate population.

2. Results may only be generalized to quadricep and hamstring soreness induced by universal squat exercises at 80% of 1 RM for five sets of 10 repetitions.

3. Subjective measures may only be generalizable to the perception of performance and soreness scales.

4. Objective measures may only be generalizable to muscular performances on the Biodex.

Chapter 2

REVIEW OF LITERATURE

DOMS is a condition recognized by most people. Because of the multitude of people it afflicts, explanations for this phenomenon, whether factually based or not, are frequently proposed. In turn, one would think that research in this area would be relatively concrete with few remaining questions. On the contrary, little is known about the basic etiologies and mechanisms involved in DOMS (Armstrong, 1984). The present study examines the effects of vitamin E and ibuprofen on muscular performance in the presence of DOMS. This review of literature addresses the following topics: (a) model of DOMS, (b) pain mechanisms, (c) consequences of DOMS, and (d) treatments.

Model of DOMS

There is general agreement among researchers that the degree of DOMS one experiences is related to both the intensity of muscular contractions and duration of the activity. Intensity seems to be the more critical of the two factors. However, there is disagreement about which of the many factors associated with increased force production is specifically responsible for DOMS (Armstrong, 1984). Mechanisms involved in

DOMS that have been proposed by Armstrong are: structural damage, metabolic waste accumulation, temperature, and altered neural control. Armstrong devised a model that describes the sequence of DOMS events based on available data: (a) high tensions, (b) cell membrane disruption, and (c) macrophage activity.

High Tensions

Eccentric exercises, as compared to concentric and isometric, are known to produce greater tension per fiber and as a result, greater muscle soreness. To produce a given muscle force, fewer motor units for eccentric exercises are activated, and thus force is distributed over a smaller cross-sectional (recruited) area of the muscle. This action causes a disruption of structural proteins in muscle fibers and connective tissue between active cross-bridges and the bony attachments (Armstrong, 1984). In support of this theory, Tiidus and Ianuzzo (1983) found that high intensity, short duration eccentric exercises produced DOMS and changes in creatine kinase (CK), a serum enzyme marker for tissue damage. The different intensities and durations that were tested indicated that a relationship exists between work performed, 24-

hour post-exercise serum CK activity, and magnitude of DOMS in untrained individuals.

A study by Friden, Sjostrom, and Ekblom (1981, 1983) showed myofibrillar disturbances following eccentric exercises in mainly fast-contracting, type II fibers immediately postexercise and up to 3 days later. Friden et al. (1983) speculated that the type IIb fibers with narrow Z-bands may also have a significant metabolic demand placed on them, which may further exacerbate the risk of damage to the inherently weak Z-disks. In opposition to this finding, Armstrong, Warren, and Warren (1991) found predominantly slow-twitch fibers were most affected in eccentric downhill running by rats, although no ultrastructural fiber typing was performed in the study.

A study comparing the effects of eccentric, concentric, and isometric exercises on ratings of soreness (RS) and serum CK activity showed both eccentric and isometric exercises increased the perceived soreness, with eccentric producing greater perceived soreness. Both types of exercise also caused a significant increase in absolute and relative CK but there was not a significant difference between the two

exercises (Clarkson, Byrnes, McCormick, Turcotte, & White, 1985). In their study on rats, Appell, Soares, and Duarte (1992) considered both eccentric and concentric prolonged exercises to compare the metabolic and mechanical origins of muscle damage. As expected, the concentric prolonged exercise group (1 h of level running) showed changes in the striation pattern, predominantly in fibers which were also glycogen depleted. In addition, an increase in the concentration of lysosomal enzymes was found, suggesting a primarily metabolic origin of contractile material breakdown. The eccentric downhill running group exhibited higher incidence of damaged fibers and at the same time, an absence of glycogen-depleted fibers, lending more support to the mechanical origin of damage.

Cell Membrane Disruption

Structural damage to the sarcolemma, or disruptions in the permeability of the cell membrane as a result of high tension forces, is accompanied by an influx of calcium (Ca++) from the interstitial tissue. This abnormal influx of Ca++ inhibits cellular respiration, which lowers the cell's ability to produce

ATP and therefore, slows oxidative phosphorylation. This event can activate certain calcium-dependent enzymes that degrade Z-discs, troponin, and tropomyosin filaments (Armstrong, 1984).

In agreement with Armstrong's model of DOMS, Friden et al. (1983) found similar conclusions in their own investigation to further validate the "mechanical disruption theory". The authors hypothesized that in addition to mechanical overload (e.g., eccentric exercise), excessive Ca++, lysosomal enzymes produced in damaged fibers, and alfa-actinin or Z-line proteinase (activated by Ca++) could also be responsible for Z-band disruption.

Byrd (1992) hypothesized that the involvement and alterations in the sarcoplasmic reticulum (SR) might be a link to exercise-induced muscle damage. Possible causes of altered SR function following strenuous exercise include a decrease in muscle pH, an increase in muscle temperature, altered metabolism, an increase in oxygen free radicals, or fluid and ion shifts. Byrd hypothesized that a combination of these effects may occur since no one mechanism is present in all types of exercise. The sequence of events eventually leading to

fiber necrosis (death) may be alterations in the SR structure and function, followed by an increase in cell Ca++ concentration, stimulation of muscle degradation by calcium-sensitive proteases (acting on Z-lines) and calcium activated phospholipases (acting on proteins in cell membranes) and finally, muscle damage.

Another study by O'Reilly et al. (1987) investigated impairment of glycogen repletion following eccentric exercise. They attributed the delay in glycogen repletion and myofibrillar damage to alterations in the sarcolemma based on the appearance of large proteins such as CK, lactate dehydrogenase, and myoglobin in the interstitial space. Because glucose transport into the cell may be the rate-limiting step in glucose utilization in the resting muscle (post-exercise), the effect of the altered membrane permeability could have resulted in less glucose available in the cell for glycogen resynthesis (O'Reilly et al., 1987).

Macrophage Activity

Following the progressive deterioration of the sarcolemma in the postexercise period, comes the accumulation of intracellular components into the

interstitial fluid. These substances attract monocytes that convert to macrophages, and in turn, activate histocytes in the injured area. Active phagocytosis, cellular necrosis, inflammatory edema (swelling), an increase in local temperature, and accumulation of analgesic agents (histamine, kinins, potassium), then stimulate the nociceptors in the free nerve endings of the muscle resulting in the sensation of DOMS (Armstrong, 1984).

In strong support of acute inflammation as the underlying mechanism in DOMS, Smith (1991) emphasized the similarities between the sensation of soreness and the acute inflammatory response. In her brief review, she noted that both events exhibit markers of pain, swelling, and loss of function, heat and redness, evidence of cellular infiltrates (i.e., macrophages) at 24 h and 48 h after initial tissue disruption, biochemical markers such as increased lysosomal activity and increased circulation of proteins, and signs of histological (tissue) healing at approximately 72 hours. Although not all time frames associated with DOMS coincide with those described for acute inflammation, the majority did. Smith concluded that

because the body responds to all forms of acute tissue injury by initiating the inflammatory response, there is no need to believe a separate response has evolved to combat an injury brought about by unaccustomed eccentric exercise.

Pain Mechanisms

The sensation of pain in skeletal muscle is transmitted to the conscious level by myelinated group III and unmyelinated group IV afferent fibers (Armstrong, 1984; Byrnes & Clarkson, 1986). Both group III and group IV sensory neurons terminate in free nerve endings around the muscle fibers and are distributed primarily in the regions of capillaries, arterioles, and at the musculotendinous junctions (Byrnes & Clarkson, 1986). The group IV fibers, classified as nociceptors and metaboceptors, are two times greater in number than the group III fibers and are known to carry dull, diffuse pain. It is likely that the sensation of DOMS is carried primarily by group IV neurons.

The nociceptors respond to noxious stimuli while the metaboceptors respond to mechanical and/or chemical changes produced by contracting muscle. Acutely

damaged cells cause the pain sensation by producing noxious stimuli. Swelling, inflammatory products associated with elevated local temperatures, release of endogenous chemicals, or a combination of the three could be considered noxious agents. The chemical substances are shown to activate nociceptors and appear to have their own separate receptor sites on the free nerve endings (Armstrong, 1984).

According to Berne and Levy (1983), bradykinin (associated with the sensation of dull pain) and other short-chain polypeptides are presumed to be cleaved by enzymes released by necrotic cells circulating in the blood. The time delay between the occurrance of injury and sensation of pain, may be due to the time it takes for cells to die and noxious agents to accumulate.

Pain is also thought to be transmitted via central nervous system pathways. The spinal cord, brain stem and thalamus, and the sensory cortex all contain various receptors and pain modulating abilities which may explain the large intersubject variability found in the perception of soreness postexercise (Byrnes & Clarkson, 1986; Clarkson et al., 1985).

The biochemical mechanisms underlying skeletal

muscle soreness and damage with unaccustomed exercise remain unclear. However, evidence is accumulating that oxygen free radicals play an important part as mediators of skeletal muscle damage and inflammation (Sjodin, Westing & Apple, 1990). During exhaustive exercise, the muscle's oxygen uptake can increase up to more than 100 times normal (Amelink, van der Wal, Wokke, van Asbeck & Bar, 1991). At the same time, the rate of ATP utilization exceeds the rate of ATP production and creates a metabolic stress. The metabolic stress within the cell results in a marked increase in the production of oxygen free radicals (Sjodin et al., 1990). These free radicals attack polyunsaturated fatty acids and initiate lipid peroxidation which in turn, damages membranes and may thus play a part in enzyme release and (focal) muscle necrosis (Amelink et al., 1991).

Vitamin E, or alpha-tocopherol is a fat-soluble compound that exists in minute quantities in the cell membrane and helps to stabilize the cell by interaction with polyunsaturated phospholipids. Vitamin E also exhibits antioxidant properties as it serves as a chain-breaker and helps to prevent the propogation of

lipid peroxidation (Jenkins, 1988).

Performance Consequences

In 1902, Hough became the first to note that the force of a maximal contraction is reduced in sore muscles. He suggested that the decrease in performance resulted both from reduced voluntary effort due to perceived muscle soreness, and to an inherently lowered capacity of the muscle to produce force (Hough, 1902).

Two subsequent studies support Hough's observations that performance is reduced in sore muscles. Both experiments, using direct electrical stimulation, found the ability of muscles to produce force was lowered (Davies & White, 1981; Newham, Mills, Quigley, & Edwards, 1983). However, Newham and coworkers also found that muscle force returned to normal by 24 h postexercise which preceded the time when sensations of soreness reached maximum intensity.

In agreement with this finding, Friden et al. (1983) found discrepencies in the amount of time between the development of soreness and the decreases in strength. Results indicated that strength losses did not occur at peak soreness, therefore it could not be concluded that pain alone affected strength.

In another study, the effects of eccentric exercise on motor performance in young and older women were examined. One point that was noted was a reduction in isometric force which occurred in both older and younger women. Secondly, the rate of recovery to baseline strength was significantly slower in older than younger women, which was expected. However, the researchers attributed the slow strength recovery not to pain associated with soreness, but possibly to caution against injury exhibited by the older subjects exerting maximal force (Dedrick & Clarkson, 1989).

Similarly, Clarkson and Tremblay (1988) also ruled out apprehension and pain tolerance as causes for the reduced ability by college-age women to produce force. Instead, they concluded that at lower frequencies of stimulation, the reduced ability to generate force was due to a damaged sarcoplasmic reticulum shown by an influx of Ca++ and delayed appearance of CK in the blood.

In yet another study, Friden et al. (1983) suggested from their findings that muscle weakness during isometric and dynamic contractions following

eccentric exercise might rely on the amount of motor unit activity involved in force generation, which in turn, might depend on damage to contractile fibers. In particular, they found that the type II fibers were more extensively damaged at higher angular velocities than type I, which explained the slower recovery rate for type II at the faster speeds. They also pointed out the fact that 3 days after eccentric exercise, subjects experienced considerable pain on movement (especially fast movements) which might have limited the maximal contraction strength they could reach voluntarily.

Treatments

Early experiments by Hough suggested that performance of the specific motor task that induced DOMS would result in alleviation of the discomfort felt afterward and possibly have a prophylactic effect (Hough, 1902). Subsequent research has supported this hypothesis on many occasions, though some differ in their conclusions on the duration of the "protective" effect.

Similar results were found in three investigations that included a single bout of exercise. During

subsequent activity, one study found a decrease in serum protein responses (an indirect assessment of muscle damage) and an increase in DOMS following eccentric contractions. These effects lasted up to 6 weeks (Byrnes et al., 1985). Two other studies found similar results from isometric contractions with prophylactic effects lasting only 3 weeks (Triffletti, Clarkson, & Byrnes, 1985; Triffletti, Litchfield, Clarkson, & Byrnes, 1988). Due to the difference in results, Triffletti et al. (1985, 1988) suggested that future studies first examine the time course of the rapid adaptation effect for the specific exercise model being used.

Following a comparison of uphill, level, and downhill training methods, Schwane and Armstrong (1983) also supported Hough's hypothesis. They found that a single bout of eccentric downhill running helped in decreasing the appearance of plasma enzymes (indicating muscle fiber injury) in subsequent bouts of prolonged downhill running. Clarkson and Tremblay (1988) continued investigating the repeated bout effect and rapid adaptation to eccentric exercise in humans. Their protocol included eight female subjects

performing 70 maximal (max) eccentric forearm contractions with one arm, and 24 max followed by 70 max forearm contractions (2 weeks later) with the other arm. The results suggested that the minor intensity bout of eccentric exercise (24 max) was sufficient to produce an adaptation such that the strength of the surrounding connective tissue protected the membrane against further damage and possibly a loss of sarcolemmal integrity.

Although evidence of its worthiness to treat DOMS is incomplete, the recreational use of anti-inflammatory drugs is increasing at a rapid pace. Janssen, Kuipers, Verstappen, and Costill (1983) conducted a study on the effects of flurbiprofen (a prostaglandine antagonist) on muscle soreness and serum CK changes. They concluded that the drug had no effect on soreness or enzyme release. One aspect to consider was the design of their study and the fact that using repeated bouts of exercise may have masked the actual effect of the drug (Byrnes & Clarkson, 1986).

In another study, Muckle (1974) compared ibuprofen and aspirin in terms of their analgesic effects on soft tissue injuries and effectiveness. Using a double-

blind trial, the author suggested that ibuprofen significantly reduced the period of pain, the time to return to soccer training, and the time to become match fit as compared to aspirin. The only stipulation the researchers pointed out was that the drug would have to be administered within a short period after injury, so as to coincide with the occurrance of local biochemical changes.

Conversely, in a double-blind crossover study, the anti-inflammatory drug Diclofenac was found to have no influence on exercise-induced muscle damage (serum enzyme changes) nor did it reduce overall soreness. However, it did relieve some specific individual soreness during the first period of the study. The reasons for these findings were unclear to the researchers as they raised the possibility that an interaction occurred between the action of the drug and the repeated bout effect (Donnelly, McCormick, Maughan, Whiting, & Clarkson, 1988). A review by Evans (1987) suggested that since prostaglandins increased muscle protein synthesis, inhibiting their production would prevent or possibly slow muscle repair, making the muscle more susceptible to further damage. But, the

results showed otherwise and the damage may have actually protected against further soreness.

Recently, the possibility that vitamin E plays a role in the maintenance of cell continuity during exercise has been studied. In 1984, Packer conducted an experiment manipulating dietary vitamin E and exercise to look at the relationship between metabolic rate and tissue oxidative damage. One group of rats was endurance trained for 8 weeks on a treadmill and another group remained sedentary with a diet totally deficient in vitamin E. Results showed the level of damage was greater in vitamin E deficient animals before exercise. The other important findings were: ATP production was markedly decreased in skeletal muscle mitochondria of exercised rats and even more so in vitamin E deficient rats; prolonged exhaustive exercise decreased latency of lysosomal enzymes which might contribute to tissue damage; rate of lipid peroxidation increased, and the amount of stable free radicals increased.

Quintanilha (1984) conducted a similar experiment controlling dietary vitamin E among sedentary and endurance trained rats. During the 9 weeks, two groups

out of four were fed 40 IU dl-alpha-tocopherol per kg/body weight. The other two groups were fed 15 IU per kg/body weight for the first 5 weeks, then no dl-alpha-tocopherol for the remaining 4 weeks. Ten animals from each of the two dietary groups were endurance trained and the following results were the found: red cell hemolysis increased in only the vitamin E deficient group (15 IU to no vitamin E) and between trained and sedentary groups, hemolysis occurred 1 week later in the endurance trained group. Quintanilha concluded there was a need for increased vitamin E during endurance training and also a possibility that training produces a protective effect against red cell hemolysis despite vitamin E deficiency.

Another study utilizing rats, focused on the possible effects that increased extracellular vitamin E would have on the response of skeletal muscles to damage induced by elevated intracellular Ca++. The soleus muscles of the rats were analyzed after the addition of Ca2+ ionophore (20 um) at 30 min intervals. Results showed an expected release of intracellular CK and, following the addition of 230 um of alpha-tocopherol, a total inhibition of the rise in CK efflux

(as well as a maintainence of low level release) throughout the 3 h period of the study (Phoenix, Edwards & Jackson, 1989). These results may be helpful for the case of vitamin E supplementation and its "protective effect".

Summary

DOMS still remains a controversial issue. Ever since 1902 when Hough first cleared the path in the recognition of this phenomenon, the etiologies and mechanisms, performances consequences and most beneficial treatments for it are still being questioned. Because of the nature of DOMS and the many variables involved, there may never be one right solution to the problem. Future study should focus not only on how muscle soreness/damage occurs, but also how it can be prevented or alleviated. The use of vitamin supplements and anti-inflammatory agents is still being argued, as many individuals may be spending needlessly on such substances. Therefore, continuous research must be emphasized to better understand all the mechanisms and hopefully find reliable treatments for DOMS.

Chapter 3

METHODS AND PROCEDURES

This study was designed to investigate if IB and/or E would decrease the sensation of pain and thereby maintain muscular performance associated with the presence of DOMS. A secondary purpose was to determine the relationship between perceived performance and the perception of soreness following the onset of DOMS. This chapter is divided into the following sections: (a) selection of subjects, (b) testing procedures and instrumentation, and (c) treatment of data.

Selection of Subjects

Data collection for this study was conducted during the Spring of 1992. Subjects were recruited by announcements to classes in the School of Health Science and Human Performance at Ithaca College and through personal communication. Twenty-nine females ranging in age from 18 to 28 years volunteered, and were accepted for participation. Prior to beginning the study, each subject filled out a medical history questionnaire (Appendix A), and read and signed an

informed consent form describing the experimental procedures (Appendix B). If a candidate had no contraindications for exercise testing as outlined by the American College of Sports Medicine (1991) and had not strength trained her lower body for 3 months prior to testing, she was accepted for participation. Thirty-five subjects began the study, however 6 dropped out. Two subjects became ill, and four subjects did not comply with all testing procedures during the study.

Testing Procedures and Instrumentation

All subjects completed five sessions of data collection. The first was a baseline measure of isometric, eccentric, and concentric maximal resistive exercises. The next four sessions were used to follow-up on these measures after muscle soreness was induced. Explanations and directions were given prior to each test in addition to a warm-up/practice period. The data collection sessions are subsequently described in greater detail.

Through a random, double-blind procedure, each subject was assigned to one of three groups. One group took 800 IU of d-alpha tocopherol (Henkel Corporation,

#5 Oval Softgel, LaGrange, Ill) (vitamin E) daily for 2 weeks prior to and throughout the 5 days of testing. The second group took a placebo tablet twice a day, for the first 12 days, and then 1200 mg/d of ibuprofen (Fays Drugs, 2 x 200 mg each, Ithaca, NY 3x/day) for the next 7 days of the testing period. The third group served as a control and took two placebo tablets/day (B.C Cowley Co., 5.0 grain, Shrewsbury, MA) for 2 weeks prior to and throughout testing. To ensure compliance, each subject was periodically asked whether or not they were following their schedule of ingestion and if there were any concerns regarding it.

Baseline Testing Session (Day 1)

Upon entering the lab, each subject completed a 24-hour health history (Appendix C) followed by a pretesting overall soreness scale (Appendix D). The 24-hour health history questionnaire was completed each day prior to testing to assess overall feeling, sleep patterns, and activity done in the previous 24 h. The subject was then seated on the Biodex with securely fastened belts across the chest, lap, over the dominant leg, and around the lower shin. The proper height and position of the chair were found by lining up the

lateral condyle of the femur to the center of the attachment shaft, as recommended by the manufacturer (Biodex Corp. Shirley, NY, 1988). Once the subject was in the correct position, biographical information (e.g. name, i.d. number, sex, birth date, height, weight, etc.) was entered into the Biodex computer and saved. Before the test trials, an explanation of the test was given followed by a warm-up period of 15 submaximal concentric (dynamic) contractions. Baseline strength measurements were then determined by the performance of maximal resistive exercise tests on the Biodex. The sequence of tests were as follows: (a) isometric, (b) eccentric, and (c) isokinetic.

Isometric Testing (Day 1)

An angle of +60 deg was used for all isometric tests performed. Direction 1 was defined as a maximal contraction of the quadriceps and was performed for a duration of 10 s with 1 min of rest between each set for a total of three sets. Direction 2 was defined as a maximal contraction of the hamstrings, and was performed for the same duration and total sets following the performance of the Direction 1 tests.

Eccentric Testing (Day 1)

Two minutes after the completion of the isometric tests, a test of maximal eccentric contractions of the upper leg was performed by each subject. In the "passive" mode, a speed of 60 deg/s was set for the performance of three sets of eight contractions with 1 min of rest between each set. To perform eccentric contractions of the quadriceps and hamstrings, the subject contracted her muscles in opposition to the robotic motion of the Biodex.

Isokinetic Testing (Day 1)

Two minutes after the completion of the eccentric tests, the isokinetic test was conducted. At a fixed speed of 120 deg/s, the subject was told to perform eight repetitions consisting of leg flexion and extensions as hard and as fast as possible. A total of two sets were performed separated by 1 min of rest. The sets were followed by a "time to fatigue" (TTF) test. The subject was given the same instructions as previously stated, but was told to continue performing repetitions until her peak torque diminished to half of maximum on three consecutive contractions as noted by the tester. After a 2 min rest period, a fixed speed

of 60 deg/s was set for the subject to perform another three sets of eight maximal leg flexion and extensions. The entire testing sequence was repeated over the next 4 days for comparison to the baseline measures.

Inducing Soreness (Day 1)

Immediately after the baseline testing was completed, each subject was escorted to the weight room. The Universal squat machine was the apparatus used to induce soreness (Universal Gym Equipment Co., Cedar Rapids, IA). Through trial and error, each subject's 1 RM was determined. Subsequently, five sets of 10 repetitions using 80% of 1 RM were used in the soreness induction protocol. Each set was separated by 15 to 20 seconds of rest. Following the last set, an eccentric set of ten repetitions was performed at 80% of 1 RM. Two testers aided the subject by lifting the weight, but then allowed the weight to be lowered eccentrically by the subject. If a subject could not successfully complete a squat or showed other signs of extreme fatigue, the soreness protocol was terminated. Following the soreness induction protocol, each subject completed a post-test soreness scale based on sensations in both the exercised (dominant) leg and the

nonexercised leg (Appendix D).

Follow-up Testing (Days 2-5)

In the four days following the initial Biodex measurements and soreness induction, testing was performed and the results were compared to baseline measurements and used to examine differences among treatments. Upon arriving each day, the subjects completed a 24-hour history, a soreness scale, and a perception of performance scale prior to Biodex performance. The perception of performance scale (Appendix E) was a prediction by the subject of how they thought they would perform compared to their previous day's performance.

Treatment of Data

Descriptive statistics were determined for the following variables: Rise time (RT), peak torque (PT), and TTF. A multivariate mixed model analysis of variance (Schutz & Gessaroli, 1987) was used to determine whether any differences existed in RT among groups across the 5 days. The level of significance was set at .05. The three dependent variables used were eccentric rise time at 60 deg/s (RTECC60), isokinetic rise time at 60 deg/s (RTISO60), and

isokinetic rise time at 120 deg/s (RTISO120). The between-subjects variable, treatment group, consisted of the E, IB, and P groups. The within subject variable, day, included the repeated measures over the 5 days.

A multivariate mixed model analysis was also used to determine whether any differences existed in PT among groups across the 5 days. The three dependent variables used were isometric peak torque for direction 2 (PTIDIR2), eccentric peak torque at 60 deg/s (PTECC60), and isokinetic peak torque at 120 deg/s (PTISO120). The between-subjects variable, treatment group, consisted of the E, IB, and P groups. The within subject variable, day, included the repeated measures obtained over the 5 days.

Two mixed model analysis of variances were used to determine whether any differences existed in Perception of Performance (PP) and RS. The between-subjects variable for both analyses, treatment group, consisted of the E, IB, and P groups. The within-subject variable, day, included repeated measures obtained over the 4 days for PP and 5 days for RS. The SPSS MANOVA procedure was used to perform all of the analyses.

Chapter 4

ANALYSIS OF DATA

This study was conducted to determine whether or not the use of IB or E reduces the perception of pain associated with DOMS and/or affects the decline in muscular performance that usually accompanies DOMS. In addition, each group's perception of their ability to perform was assessed following the onset of DOMS. Statistical analyses of these data are described in this chapter.

Description of Subjects

Twenty-nine college-aged females were recruited for this study. Acceptance into the study was gained if the individual met the following criteria: (1) had not done lower body strength training within the last 3 months, (2) had no known allergies to ibuprofen or aspirin, and (3) had no current knee or leg ailments. Subjects ranged from recreational exercisers to varsity level athletes, and were recruited for participation by verbal announcements.

Rise Time

Descriptive statistics for RT are shown in Tables

1, 2, and 3. RT was equal to the amount of time it took to reach peak torque. A multivariate mixed model ANOVA was used to determine whether any differences existed among the treatment groups across the five testing days. The three dependent variables used were eccentric rise time at 60 deg/s (RTECC60), isokinetic rise time at 120 deg/s (RTISO120), and isokinetic rise time at 60 deg/s (RTISO60). The level of significance for the tests of the interaction and main effects was .05. The interaction $F(24,276)=.984$, group main effect $F(6,48)=1.14$, and day main effect $F(12,251)=1.59$ for these RT variables were not significant (See Table 4).

Peak Torque

The highest torque output, or the PT, obtained from the Biodex data, was equal to the single greatest amount of force applied on a repetition. A multivariate mixed model ANOVA was utilized to analyze PT data. The three dependent variables used were eccentric peak torque at 60 deg/s (PTECC60), isometric peak torque for direction 2 (PTIDIR2) (flexion), and isokinetic peak torque at 120 deg/s (PTISO120). The means and standard deviations for each variable are shown in Tables 5, 6, and 7. The level of significance

Table 1

Eccentric Rise Time 60 deg/s (RTECC60)

Day	Group	Mean*	SD	n
1	Vitamin E	1049	432	11
	Placebo	1003	227	10
	Ibuprofen	1020	167	7
2	Vitamin E	1167	179	10
	Placebo	1023	221	10
	Ibuprofen	1143	232	8
3	Vitamin E	1192	232	10
	Placebo	1085	238	10
	Ibuprofen	1043	246	8
4	Vitamin E	1093	212	11
	Placebo	985	204	10
	Ibuprofen	1067	218	8
5	Vitamin E	1096	207	10
	Placebo	1223	254	10
	Ibuprofen	1174	261	8

* msec

Table 2

Isokinetic Rise Time 120 deg/s (RTISO120)

Day	Group	Mean*	SD	n
1	Vitamin E	242	39.1	11
	Placebo	223	32.9	10
	Ibuprofen	260	23.8	8
2	Vitamin E	227	53.2	11
	Placebo	247	45.4	9
	Ibuprofen	259	50.4	8
3	Vitamin E	225	61.5	11
	Placebo	233	24.5	10
	Ibuprofen	271	55.2	8
4	Vitamin E	226	49.8	11
	Placebo	238	44.2	10
	Ibuprofen	247	43.9	7
5	Vitamin E	224	31.7	10
	Placebo	234	31.4	10
	Ibuprofen	222	33.4	8

* msec

Table 3

Isokinetic Rise Time 60 deg/s (RTISO60)

Day	Group	Mean*	SD	n
1	Vitamin E	354	53.9	11
	Placebo	346	55.3	10
	Ibuprofen	366	100.0	8
2	Vitamin E	333	87.8	11
	Placebo	346	97.6	10
	Ibuprofen	337	74.2	8
3	Vitamin E	355	115.4	11
	Placebo	316	60.5	10
	Ibuprofen	370	91.0	8
4	Vitamin E	322	60.9	11
	Placebo	331	16.5	10
	Ibuprofen	339	59.6	8
5	Vitamin E	306	41.2	11
	Placebo	322	33.9	10
	Ibuprofen	318	52.5	8

* msec

Table 4

ANOVA Table for Rise Time

SOURCE	Sums of Squares RTECC[a]	RT120[b]	RT60[c]	df	WILK'S LAMBDA	F	p
Between Subjects							
Groups	47555	20850	47512	48	.765	1.14	.352
Error	4430509	99120	277660				
Within Subjects							
Days	251575	5517	36972	252	.823	1.59	.093
Days x Group	375246	10849	19602	276	.788	.984	.487
Error	317339	131932	352042				

[a]Eccentric rise time 60 deg/s
[b]Isokinetic rise time 120 deg/s
[c]Isokinetic rise time 60 deg/s

Table 5

Eccentric Peak Torque 60 deg/s (PTECC60)

Day	Group	Mean*	SD	n
1	Vitamin E	80.3	18.07	11
	Placebo	76.8	26.85	10
	Ibuprofen	66.8	12.24	8
2	Vitamin E	75.9	37.64	11
	Placebo	69.3	22.63	10
	Ibuprofen	64.6	11.73	8
3	Vitamin E	76.4	48.42	11
	Placebo	71.6	27.27	10
	Ibuprofen	60.5	8.28	8
4	Vitamin E	75.1	36.47	11
	Placebo	73.1	22.41	10
	Ibuprofen	64.1	7.77	8
5	Vitamin E	80.3	40.61	11
	Placebo	87.0	32.16	10
	Ibuprofen	70.4	18.66	8

* ft/lbs

Table 6

Descriptive Statistics for Isometric Peak Torque Direction 2 (PTIDIR2)*

Day	Group	Mean**	SD	n
1	Vitamin E	56.9	4.02	6
	Control	61.5	10.54	9
	Ibuprofen	54.0	10.48	7
2	Vitamin E	43.9	12.38	11
	Control	47.8	12.90	10
	Ibuprofen	49.4	6.84	8
3	Vitamin E	43.0	13.35	11
	Control	43.9	15.40	10
	Ibuprofen	47.3	9.13	8
4	Vitamin E	45.8	13.46	11
	Control	51.1	12.77	10
	Ibuprofen	48.1	5.70	8
5	Vitamin E	46.6	11.68	11
	Control	55.7	12.51	10
	Ibuprofen	49.7	7.78	8

* values for isometric contraction of hamstrings
** ft/lbs

Table 7

Isokinetic Peak Torque 120 deg/s (PTISO120)

Day	Group	Mean*	SD	n
1	Vitamin E	75.3	12.74	11
	Placebo	81.8	10.88	10
	Ibuprofen	80.2	11.44	8
2	Vitamin E	71.5	11.96	11
	Placebo	89.6	14.40	9
	Ibuprofen	78.4	12.87	8
3	Vitamin E	73.5	7.97	11
	Placebo	87.5	17.18	10
	Ibuprofen	82.3	9.64	8
4	Vitamin E	77.3	12.28	11
	Placebo	87.8	15.84	10
	Ibuprofen	82.7	11.89	7
5	Vitamin E	77.2	10.60	10
	Placebo	90.9	13.21	10
	Ibuprofen	80.1	9.96	8

* ft/lbs

for the tests of the interaction and main effects was .05. As shown in Table 8, significant group x day interaction $F(24,267)=1.57$, and day main effect $F(12,243)=8.31$ were found. However, subsequent simple group effects analyses for each day were non-significant, indicating the three groups did not differ from each other on any of the 5 days. Follow-up tests for time main effect were performed with three one-way multivariate ANOVA's to determine if Day 2 differed from Days 3, 4, and 5. A significant difference was found between Days 2 and 5 ($F=.022$, $p<.05$) indicating that on Day 5, the subjects' ability to generate force was significantly greater than that obtained on Day 2. The group main effect was not significant $F(6,48)=.829$.

Time to Fatigue

Descriptive statistics for TTF are shown in Table 9. The means and standard deviations for each group across the 5 days are indicative of the amount of time it took each subject's torque reading to be reduced by one half, as measured by a combination of isokinetic leg flexion and extensions. A mixed model ANOVA was used to determine if the groups differed over the testing period. The level of significance for the

Table 8

ANOVA Table for Peak Torque

SOURCE	Sums of Squares PTIDIR2[a]	PTECC[b]	PTISO120[c]	df	WILK'S LAMBDA	F	p
Between Subjects							
Groups	431	1983	2460	48	.821	.829	.553
Error	13716	80224	14998				
Within Subjects							
Days	2251	1963	352	249	.403	8.31	.000
Days x Group	489	874	347	267	.682	1.57	.047
Error	2425	24683	2899				

[a]Isometric peak torque direction 2 (hamstrings/flexors)
[b]Eccentric peak torque 60 deg/s
[c]Isokinetic peak torque 120 deg/s

Table 9

Time To Fatigue

Day	Group	Mean*	SD	n
1	Vitamin E	40.0	9.27	10
	Placebo	45.2	8.01	10
	Ibuprofen	44.3	7.21	8
2	Vitamin E	44.0	7.75	9
	Placebo	41.6	9.15	9
	Ibuprofen	45.2	6.25	8
3	Vitamin E	45.3	7.13	10
	Placebo	40.6	9.31	10
	Ibuprofen	47.5	6.15	8
4	Vitamin E	45.2	7.06	10
	Placebo	45.1	9.12	10
	Ibuprofen	44.2	7.37	7
5	Vitamin E	44.4	7.07	9
	Placebo	47.9	10.03	10
	Ibuprofen	48.7	7.75	8

* seconds

tests of the interaction and main effects was .05. The group x day interaction was significant $F(8,84)=2.23$, indicating that the trend of the three groups' mean TTF scores differed over the 5 days (See Table 10). The day main effect was also significant $F(4,84)=3.27$. The group main effect was not significant $F(2,21)=.19$. Simple effects for groups at days showed significant differences between the groups only at Day 1 ($F=2.31$, $p<.05$). The groups' scores were not significantly different on Days 2, 3, 4, and 5. Follow-up Bonferroni pairwise contrasts showed no significant differences between the groups at Day 1 (1 vs. 2 $t=2.14$; 2 vs. 3 $t=.575$; 1 vs. 3 $t=1.45$, $p>.05$). In addition, the Bonferroni contrasts for day main effect showed no significant differences between mean scores on Days 2 and 3 ($t=.811$, $p>.05$), 2 and 4 ($t=.956$, $p>.05$), or 2 and 5 ($t=2.40$, $p>.05$), indicating that the subjects' scores did not differ between the specific days of interest.

Perception of Performance

In Table 11, the means and standard deviations for each group's PP for 4 testing days are shown. A mixed model ANOVA was used to determine if the groups

Table 10

ANOVA Table for Time To Fatigue

Source	SS	df	MS	F	p
Between Subjects					
Group	83.86	2	41.93	.19	.831
Error	4726.81	21	225.09		
Within Subjects					
Days	318.70	4	79.68	3.27	.015
Days x Group	434.82	8	54.35	2.23	.033
Error	2049.05	84	24.39		

Table 11

Perception of Performance

Day	Group	Mean*	SD	n
2	Vitamin E	9.33	1.97	12
	Placebo	9.10	1.29	10
	Ibuprofen	8.12	1.73	8
3	Vitamin E	9.25	3.39	12
	Placebo	8.70	3.37	10
	Ibuprofen	8.12	2.75	8
4	Vitamin E	12.3	3.65	12
	Placebo	13.2	3.39	10
	Ibuprofen	10.6	3.25	8
5	Vitamin E	15.0	3.63	12
	Placebo	15.7	2.58	10
	Ibuprofen	12.0	2.93	8

*sum of scores based on scale of 1-7

differed across the 4 days. The level of significance for the interaction and main effects was .05. The group x day interaction was not significant $F(6,50)=.75$ while the day main effect was significant $F(3,81)=31.40$ (See Table 12). The group main effect was not significant, however, it nearly attained significance $F(2,27)=2.73$. The day main effect was followed up with Bonferroni pairwise contrasts and showed a significant difference between mean scores on Days 2 ($M=8.93$) and 4 ($M=12.17$) ($t=4.78$, $p<.05$), and Days 2 ($M=8.93$) and 5 ($M=14.47$) ($t=8.18$, $p<.05$). However, no difference was found between Days 2 and 3 ($t=.245$, $p>.05$) . These results indicate that subjects perceived they could produce greater force on the fourth and fifth days, compared to the first day following soreness induction.

Soreness

The means and standard deviations for RS in the exercised (dominant) legs for each group over the 5 days are displayed in Table 13. A mixed model ANOVA was used to determine if any differences existed in RS among the groups and/or across the days. The level of significance for the tests of the interaction and main effects was .05. The group x day interaction

Table 12

ANOVA Table for Perception of Performance

Source	SS	df	MS	F	p
Between Subjects					
Group	81.92	2	40.96	2.73	.083
Error	404.72	27	14.99		
Within Subjects					
Days	632.74	2.69*	210.91	31.40	.000
Days x Group	30.12	6	5.02	.75	.613
Error	544.08	72.7*	6.72		

* Huynh-Feldt adjusted

Table 13

Ratings of Soreness

Day	Group	Mean*	SD	n
	Vitamin E	1.82	1.02	10
1	Placebo	2.22	1.06	10
	Ibuprofen	1.98	1.35	8
	Vitamin E	3.52	.801	10
2	Placebo	2.98	1.010	10
	Ibuprofen	3.05	.715	8
	Vitamin E	3.64	.974	10
3	Placebo	3.88	.839	10
	Ibuprofen	3.15	.521	8
	Vitamin E	2.66	.900	10
4	Placebo	2.32	1.080	10
	Ibuprofen	2.20	.849	8
	Vitamin E	1.28	.880	10
5	Placebo	1.14	.985	10
	Ibuprofen	1.52	.555	8

*mean scores based on scale of 0-6

$F(8,44)=1.25$, and the group main effect $F(2,25)=.23$ were not significant, while a significant day main effect $F(4,22)=39.44$ was found (See Table 14). Bonferroni contrasts for day main effect showed no significant differences between Days 2 and 3 ($t=1.94$, $p>.05$), but did show significant differences between mean scores on Days 2 ($M=3.19$) and 4 ($M=2.41$) ($t=3.9$, $p<.05$), and Days 2 ($M=3.19$) and 5 ($M=1.30$) ($t=9.3$, $p<.05$). These results indicate that between Days 2 and 3, the subjects' soreness was reaching its peak, so a significant difference was not found. However, by Days 4 and 5, sensations of soreness were on the decline leading to the significant differences between the early and later days.

Table 14

ANOVA Table for Ratings of Soreness

Source	SS	df	MS	F	p
Between Subjects					
Group	.93	2	.47	.23	.799
Error	51.46	25	2.06		
Within Subjects					
Days	89.93	3.65	22.46	39.44	.000
Days x Group	5.70	8	.71	1.25	.277
Error	56.93	94.0	.57		

*Huynh-Feldt adjusted

Chapter 5

DISCUSSION OF RESULTS

The findings from this study demonstrate that the use of IB or E did not provide relief from DOMS, nor did they have a significant effect on the decline in muscular performance that accompanies DOMS. However, individual perception of performance ratings (regardless of group) did increase as days passed and soreness subsided. A discussion of these results are presented in this chapter under the following subtopics: (a) muscular performance variables, (b) perception of performance and soreness, (c) implications of findings, and (d) summary.

Muscular Performance Variables

In the present study, muscular performance as represented by RT, PT, and TTF were the variables selected from the Biodex measurements for analysis. RT (i.e., the time to reach peak torque) did not differ between the three groups over the course of testing. Peak soreness occurred for all groups at Day 3 (48 hours postexercise) but did not correspond with a significant change in RT. The fact that RT was not notably altered may be due to the time frame in which

it was analyzed. According to Clarkson and Tremblay (1988), the greatest force reduction occurs immediately following eccentric exercise, which is before the onset of DOMS. The present study analyzed RT at 24-hour intervals postexercise, which may have missed the greatest reduction in RT.

According to Smith (1991), inability of a sore muscle to generate force occurs from acute inflammation following eccentric or negative contractions. Although not clearly understood, the loss of function is thought to be due to the presence of a mechanical barrier from swelling and/or due to a reflex inhibition of the muscles experiencing pain. These decrements in performance have been seen immediately postexercise and can return to baseline by 24 h or take up to 14 days later to normalize (Smith, 1991).

Analyses of PT (i.e., force) indicated the groups were performing similarly after Day 1. PT decreased at 24 h postexercise for all groups and had increased by the fourth day after inducing soreness.

In the present study, all three groups' PT did decline at 24 h postexercise (Day 2) and began to improve by 48 h (Day 3). Interestingly, this

improvement occurred at the same time as PP was lowest and soreness sensations at their peak. These findings may indicate that PP was not an accurate reflection of performance, but rather an accurate reflection of soreness sensations.

Following a stepping exercise, Newham et al. (1983) found that ultrastructural changes were limited to only the muscles worked eccentrically and not to those worked concentrically or isometrically. It may be that soreness reported in the present study was limited to sensations in eccentrically damaged muscle fibers, which should have been enough to produce a marked decrease in strength performance variables (i.e., RT and PT). Also, it was assumed that maximal effort was being exerted by each subject for all testing purposes. During the soreness induction protocol on Day 1, subjects may have been unfamiliar and possibly apprehensive when trying to give a "maximal" effort and consequently, less damage and soreness occurred.

A plausible explanation for this reported lack of significance may involve the combination of contractions used in the soreness protocol. There may

not have been a sufficient amount of adequately intense eccentric contractions performed to assure enough damage and swelling to elicit extreme soreness. The squat exercise performed at 80% of 1 RM (for five sets of 10 repetitions plus an eccentric set) utilized both concentric and eccentric contractions of the quadriceps and hamstring muscle groups, while the Biodex protocol used a combination of concentric, eccentric, and isometric contractions. According to the soreness scale that was administered, the maximal mean score reached was only 3.88 out of a possible 6 points at peak soreness. This number corresponded to "more than slight pain" or "painful" which may not have been enough to significantly inhibit muscular performance.

The third muscle function test used to assess the impact of DOMS on performance was TTF. In the present study, all three groups' scores were similar, except prior to DOMS induction when the E group displayed significantly longer TTF than the IB or P groups. However, during the 4 days following soreness induction, all groups demonstrated a similar trend in TTF performance in that there was no significant change across the 4 days for any of the groups. In their

investigation, Davies and White (1981) found no significant differences in relative strength decrement during a 2-minute muscle fatigue test 20 h after eccentric exercise was performed. The authors suggested the possibility that the relative endurance capabilities of the muscle (i.e., slow twitch fibers) were not affected by DOMS. This may have been the case in the present study.

Perception of Performance and Soreness

An individual's perception of his/her ability to perform when soreness is present is critical. If the damage is severe enough to hinder muscular performance, an individual should be able to recognize this through sensations of soreness and therefore, be able to protect themselves against further injury. In the present study, subjects were asked to rank themselves prior to testing each day, compared to their performance on each previous day. Perception scales were completed on each day following the induction of soreness. It was shown that PP changed over the 5-day testing period. Significant changes were seen between 24 and 72 h and between 24 and 96 h postexercise, which coincided with the rise (24 h post) and then decline

(72 h post) in sensations of soreness. At 24 h postexercise, PP was low, but by 72 h when soreness was subsiding, PP was increasing. These changes in PP and RS were reported while performance scores were essentially unaffected by DOMS occurrance. Although there was no significant difference between the three groups, it appeared as though the IB group had a consistently lower perception of their daily performance than the E or P groups ($p=.083$). While possibly reducing inflammation during the healing process, IB seemed to also lessen subjects' perception of performance ability. The mechanism that accounted for this perception is unknown, but this phenomenon may serve to "protect" damaged muscle fibers by preventing overexertion and further injury.

Soreness perception scales were completed at the beginning of all five testing sessions. Once again, results indicated that the subjects' soreness ratings were changing over time. Similar to PP, RS was significantly changing from 24 to 72 and 96 h postexercise. By 24 h postexercise, all three group's soreness ratings were beginning to peak, but by 72 to 96 h, soreness was on the decline. This observation

may be evidence that there is a strong relationship between RS and PP regardless of treatment used. The congruency of these two measures points to an accuracy of human perception that may serve to protect the organism against further exercise-induced damage. In this regard, it is fortunate that IB and E administration do not confound the sensitivity of that perception.

Human perceptions of soreness continue to be a confusing problem today. The variability among individuals makes it even more difficult to point to any one mechanism leading to DOMS. Byrnes and Clarkson (1986) and Clarkson et al. (1985) explain their large intersubject variability in soreness perception as due to different nervous system pathways. They claim the reason for such fluctuation is due to the various receptor types (for the Reticular Activating System and cortex) and pain regulating abilities. In other studies, it has been suggested that exercise increases the release of endorphins which could potentially provide an analgesic effect, minimizing the sensation of DOMS during the exercise period. Another mechanism for alleviation of soreness may result from the subject

focusing attention toward the activity and away from the pain at the cortical level in the brain (Armstrong, 1984).

Jones, Newham, Round, & Tolfree (1986) noted considerable variability among subjects in the severity of soreness responses to damaged muscle fibers. They attributed this result to differences in training and activity levels, which have been found to protect muscle against such damage. However, the researchers hypothesized that this could not be the full explanation as no obvious correlation has been seen between susceptibility to damage and factors such as age, sex or general activity level.

Implications of Findings

Because there were no obvious differences among the three groups in muscular performance, PP, or RS, the effectiveness of both IB and E use in prevention and/or treatment of DOMS must be questioned. Studies on E are inconclusive regarding it's antioxidant abilities during physical exercise. Amelink et al. (1991) found that after 5 weeks on an E deficient diet, rats were more susceptible to exercise-induced muscle damage following a 2 h endurance

treadmill run. These results indicated a possible but not definite, increase in lipid peroxidation. Similarly, Quintanilha (1984) conducted a 9-week experiment where an E deficient diet was fed to both sedentary and endurance trained rats. The author found that endurance trained rats exhibited less red blood cell hemolysis, which may have indicated a lowered rate of lipid peroxidation.

In a study by Jenkins (1983), it was found that unaccustomed, acute bouts of exercise increased lipid peroxidation in untrained rats. The author hypothesized that endurance training would reduce lipid peroxidation by decreasing circulating catecholamines.

The present study utilized muscular contractions to produce muscle fiber damage and induce soreness. Several reasons for lack of differences among the groups following 19 days of E use may be: (1) subjects had various aerobic fitness levels, (2) the exercises used were anaerobic in nature and did not produce a substantial amount of oxygen-mediated free radicals, and (3) the dosage and supplementation time may not have been enough to produce a "protective" effect on cell membranes.

In the present study IB, like E, lacked any noteworthy effect on muscular performance and soreness. Donnelly et al. (1988) administered Diclofenac, an anti-inflammatory drug, to untrained males prior to and 72 h following a 45-minute eccentric treadmill run. They found that the drug had no influence on muscle damage, but may have slightly reduced soreness perceptions in certain individuals. Donnelly et al. also noted the only evidence for the effectiveness of anti-inflammatory drugs for DOMS in humans, was one in which aspirin reduced soreness and prevented changes in prostaglandin E and F2 alpha levels observed in a control group (Bansil, Wilson, & Stone, 1985). In 1974, Muckle compared IB and aspirin in terms of their analgesic effects on soft tissue injuries and effectiveness for return to soccer playing. He found that the 1200 mg dose of IB (daily for 5 days) decreased the duration and severity of pain and helped players to return earlier to training and match play than did aspirin. Muckle also stressed that to utilize IB's effectiveness, the drug would have to be administered within a short period after injury, when the local biochemical changes are beginning.

In this study, a 1200 mg dose of IB was taken 2 days prior to and throughout the 5 days of testing. Although the dose given was similar to that of Muckle's study, the present study dealt with muscle soreness, not soft tissue injuries (i.e., groin strains, muscle hematomas, subcutaneous bruising). The inflammation accompanying these types of soft tissue injuries might be much more pronounced than the muscle fiber damage induced, therefore, a significant effect of the treatment was not seen in this study.

Another important consideration in the identification of DOMS and its response to treatment is the design of a study. Janssen et al. (1983) examined the effect of an anti-inflammatory drug on muscle soreness and serum CK changes. They had subjects perform the same exercise protocol on two separate days using a crossover design, and although they concluded the drug had no significant effects, the repeated bout of exercise may have masked the actual effect. In the present study, the same exercises were repeated over five consecutive days. Because the repeated bout effect is so large, any treatment effects from IB would have to have been of great magnitude in order to be

detected.

However, as mentioned previously, the IB group did have a consistently lower PP compared to the other two groups. This observation may warrant further research to look at IB's potential to have a "protective" effect in humans during exercise in the presence of DOMS.

Summary

It is well known that unaccustomed activity produces DOMS, the sensation of pain and stiffness in the muscles that occurs anywhere from 24 to 48 h postexercise and can last up to 5 days (Armstrong, 1984). IB, with it's anti-inflammatory properties, may be able to reduce the swelling and/or inflammation which is thought to be a mechanism following muscle fiber damage (Smith, 1991). The anti-oxidant properties of E may be able to maintain the integrity of the cell by preventing the propagation of lipid peroxidation (Amelink et al., 1991). In this study, it was proposed that each would have an influence on preventing muscle fiber damage, attenuating the DOMS sensation, and possibly improving performance.

The results of the present study indicate that all three groups had similar muscular performances over the

course of testing regardless of the treatment used. Following soreness induction, PT did decline by 24 h, but there was no change in RT and TTF measures. A parallel between PP and RS was noted, however, and must be viewed as an important initial finding. More research should be conducted regarding IB's effect on perception and soreness so as to understand more fully its potential to combat overuse injuries in athletes experiencing DOMS.

Chapter 6

SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS

Summary

This study was designed to determine whether or not the use of IB or E reduces the perception of soreness or affects the decline in muscular performance that usually accompanies DOMS. A secondary purpose was to determine each group's ablility to perceive their performance following the onset of DOMS.

E and P were taken 2 weeks prior to and throughout testing, while IB was taken 2 days prior to and throughout the 5-day testing period. Baseline measurements on the Biodex consisted of maximal isometric, eccentric, and concentric contractions of the quadriceps and hamstring muscles. Immediately after baseline testing, subjects performed a series of near maximal squats to induce soreness in the upper legs. RS and PP scales were completed daily. Multivariate mixed models were used to compare the IB vs. E vs. P groups on selected RT, PT, and TTF measurements over the 5 days. RS and PP measures were analyzed with a mixed model ANOVA. Overall results indicated that RT did not change between the groups

over the testing period. Analysis for PT revealed that IB, E, and P groups all performed similarly throughout testing, and that PT declined initially following soreness, but then recovered between 24 and 96 h postexercise. Analysis of TTF indicated that the groups performed differently at baseline testing on Day 1, but then performed in a similar fashion for the remainder of the testing period. The IB group's PP tended to be consistently lower than E or P, however, this trend did not reach significance. All subjects' PP tended to increase from 24 to 72 and 96 h postexercise, but not from 24 to 48 h postexercise. Analysis of RS showed no differences between IB, E, and P groups over the testing period, and that all subjects peaked in soreness by 48 h postexercise and began to recover thereafter. PP followed a similar trend and therefore, tended to reflect RS rather than performance.

Conclusions

The results of this study led to the following conclusions regarding the effect of IB and E on DOMS and performance:

1. Muscular performance was not significantly

altered by the use of IB or E in the presence of DOMS.

2. RS was not significantly different between IB, E, and P groups. However, soreness sensations peaked for all groups by 48 h postexercise and began to recede thereafter.

3. PP did not significantly differ among the three groups, although the IB group was consistently lower than E or P groups for the 5 days of testing.

Recommendations

The following recommendations for further study were made after the completion of this investigation:

1. Future studies should use a soreness protocol with only eccentric contractions, as these types of contractions tend to elicit the most damage, soreness responses, and drop in performance.

2. Future studies should use variable doses and supplementation times to determine which treatment may be beneficial.

3. A similar study should include a practice session far enough in advance that subjects gain familiarity with the apparatus and testing protocol so as to avoid a learning effect and possible interference in the soreness protocol.

4. Future studies should examine more closely the relationship between IB and PP and the possibility of a protective effect in humans.

5. Future studies should pre-match subjects for activity levels before placing into groups.

6. PP and RS should be scaled based on percentages rather than the Likert-type scale for more accurate analyses.

Appendix A

ITHACA COLLEGE FITNESS PROGRAM

MEDICAL HISTORY/HEALTH HABIT QUESTIONNAIRE

Name _____ Age _____ Birthdate _____

School Address _____ Phone _____

Home Address _____ Phone _____

Present Physician _____

FAMILY HISTORY - Check if any blood relatives (parents, siblings, etc.) had?

Heart Disease ( ) Stroke ( ) Diabetes ( )
High Blood Pressure ( ) High Cholesterol ( )

Other conditions/comments:

MEDICAL/HEALTH HISTORY - Check if you have ever had?

Heart Disease/Stroke	( )	Lung Disease	( )
High Blood Pressure	( )	Diabetes	( )
Heart Murmur	( )	High Cholesterol	( )
Skipped, rapid beats or irregular rhythms	( )	Epilepsy	( )
Rheumatic fever	( )	Injuries to back, knees, or ankles	( )
Cancer	( )	stomach ulcer	( )
Pregnant	( )		

Other conditions/comments:

** If you have reason to believe you are pregnant, please state symptoms here _____

PRESENT SYMPTOMS - Have you recently had?

Chest pain	( )	Illness, surgery, or hospitalization	( )
Shortness of breath	( )	Ankle/leg swelling	( )
Lightheadedness	( )	Joint/muscle pain	( )
Heart palpitations	( )	Allergies	( )
Loss of consciousness	( )	Allergic to aspirin? Y/N	
		Tylenol? Y/N	
		Advil (ibuprofen)? Y/N	

Other conditions/comments:

LIST ALL MEDICATIONS PRESENTLY TAKING:

HEALTH HABITS

1. SMOKING HISTORY
   Do you smoke? Yes ( ) No ( ) Quit ( )
   How much did(do) you smoke a day? _____
   How long have(had) you been smoking? _____
   If quit, when? _____-

2. EXERCISE HABITS
   Are you presently active? Yes ( ) No ( )
   What type of activity? _____
   How hard? Light ( ) Moderate ( ) Strenuous ( )
   How often? 1-2d/wk ( ) 2-4d/wk ( ) 5+d/wk ( )

   Did your past exercise habits differ from what you are doing now? Yes ( ) No ( )
   What kind of exercise did you do in the past?_____
   How hard? Light ( ) Moderate ( ) Strenuous ( )
   How often? 1-2d/wk ( ) 2-4d/wk ( ) 5+d/wk ( )

   Is your present occupation- Sedentary ( ) Active ( )
   Heavy work ( ) Explain:_____

   Do you have discomfort, shortness of breath, or pain with exercise? Yes ( ) No ( )
   If Yes, what type(s) of exercise?_____

3. NUTRITIONAL BEHAVIOR
   Do you consider yourself overweight? Yes ( ) No ( )
   If so, how long have you been overweight? _____

   How many meals do you eat on a typical day? _____
   How often do you eat meals outside of the home? _____

   Do you presently consume alcohol? Yes ( ) No ( )
   If Yes, what? _____ Number of drinks/wk _____

4. STRESS
   Do you consider your day stressful? Yes ( ) No ( )
   What is the nature of your stress? _____
   How many hours (average) do you sleep at night? _____
   Is your sleep sound? Yes ( ) No ( )

ADDITIONAL PERTINENT INFORMATION:

SIGNATURE _____ DATE _____

Appendix B

INFORMED CONSENT FORM

I. **Purpose of the study**

This study has been designed to investigate the possibility that ibuprofen and/or vitamin E will decrease pain and improve performance in a muscle group experiencing muscle soreness.

II. **Benefits of the study**

The results from this study will help sports medicine related professionals to better understand the effects of ibuprofen and vitamin E when they are administered to individuals before and during episodes of muscle soreness. This will provide insight into how muscle soreness may be treated for athletic injuries or unaccustomed exercise (e.g., stair climbing). Another benefit would be the possibility that performance may be affected by the treatments which could be very important information for athletes as well as regular exercisers.

III. **Subject participation**

Amount of time needed: The amount of time commitment involved will be approximately 1 hour the first day, and approximately 30 minutes for each of the subsequent days. The total amount of sessions are on 5 consecutive days.

Tasks and procedures: Prior to administration of medications, all subjects will be asked to give a urine sample. Then, two days preceding the initial testing, you will be asked to take (2) 200 mg doses of ibuprofen three times a day, every four to six hours, with food or milk. Or, 2 weeks prior to initial testing, you will be asked to take (2) 400 IU gelcaps of vitamin E with food or milk. You will continue to follow this procedure until the final day of testing, which will be a total of 7 days (if ibuprofen) or 19 days (if vitamin E). On the first day and each subsequent day, you will be asked to complete 2 questionnaires; a soreness scale, and a perception of performance scale. These two forms will aid in the interpretation of the data collected from the exercise tests. You will then be asked to perform several bouts of exercises requiring maximum effort involving the upper leg muscles. These tests will be performed on the Biodex, a computerized device used to interpret and assess power and strength of muscles. Following that, you will be asked to exert maximal efforts on a Universal squat machine. After the first day of exercise testing, you will experience some muscle soreness and discomfort. This is to be expected and should subside within a few days during testing. You will be asked to perform the same initial exercises on the Biodex for 5 consecutive days, and to continue to take the ibuprofen or vitamin E until the final day of testing. In addition, blood samples will also be taken following exercise on several occasions. This will aid in the analysis of the actions of

the supplements given. All testing will take place in the Exercise Physiology lab in Hill Center (Room #46).

Also included is an exerpt from the United States Pharmacopeial Convention regarding various pieces of information on ibuprofen. All subjects must read this exerpt before the start of testing.

IV. **Risks associated with participation:**

This study will cause you to experience muscle soreness. This soreness should only last a few days, and should not prevent you from carrying on your normal daily activities.

Maximum effort exercises cannot be performed without some minor risk of injury. All precautions will be taken to minimize this risk and assure your safety. The Biodex and the Universal squat machine are very safe and effective exercise devices and the researchers are well trained on their use. Exercise testing will be supervised at all times by at least one of the researchers.

Ibuprofen is an over the counter drug that has been approved by the FDA and is considered safe for general consumption. Ibuprofen should not be taken if you have a known allergic reaction to aspirin or non-aspirin pain reliever, such as Tylenol, or ibuprofen itself. Some of the possible side effects of ibuprofen include gastrointestinal (stomach) problems, dizziness, rash, weight gain, and retention of fluids. These side effects are not common among the majority of people and are highly unlikely to occur with the small dosage and short duration of this study. Ibuprofen should be taken with meals or a glass of milk.

Vitamin E is a fat soluble substance that is found in the cell membranes within our bodies. It is known as an antioxidant because of its ability to protect the membrane from damaging effects of free radicals that can be produced with exercise. Toxicity is very rare with this vitamin and has only been cited in animals after administration of extremely high dosages. Adverse effects that may occur include nausea and gastrointestinal (stomach) problems.

In summary, this study involves only safe experimental protocols that are common in exercise physiology research. If you are allergic to aspirin, Tylenol (acetaminophen), or ibuprofen, have had a previous ulcer, are pregnant, currently smoke cigarettes, or have an injury contraindicating exercise, you will be excluded from the study. Hopefully this study will provide information of great interest to you as well as sports medicine experts, coaches, athletes, and all those that exercise.

V. **Need more information?**

If you would like more information or would like to know of the results of the study, please feel free to contact Susan Kofod at 256-2346. Dr. G.A. Sforzo, Department of Exercise Sport and Science, will also be able to answer your questions. Dr. Sforzo may be contacted in his office Room 41, Hill Center at 277-3359.

VI. **Withdrawel from the study:**

Participation in this study is voluntary, and you are free to withdraw at any time. If you have any questions about the study, risks, or procedures, we will be happy to answer them before or after you agree to participate. If you choose to withdraw from the study, you will not suffer any penalty of any kind.

VII. **Will the data be maintained in confidence?**

All of the participants in this study will be given a number code that will be used whenever related data is analyzed or presented. All data, questionnaire answers, and results will be kept completely confidential.

Thank you for your time in considering this study and especially for your participation.

I have read the above information and understand its contents. I have also read the exerpt on ibuprofen and have had the opportunity to ask questions if I need to.

_____ _____

Signature Date

Appendix C

24-Hour History

NAME: _____ DATE: _____

TIME: _____

HOW MUCH SLEEP DID YOU GET LAST NIGHT? (Please circle one)
1 2 3 4 5 6 7 8 9 10 (hours)

HOW MUCH SLEEP DO YOU NORMALLY GET? (Please circle one)
1 2 3 4 5 6 7 8 9 10 (hours)

HOW LONG HAS IT BEEN SINCE YOUR LAST MEAL OR SNACK?
1 2 3 4 5 6 7 8 9 10 11 12 13 14 (hours)

LIST THE ITEM(S) EATEN BELOW:

_____

WHEN DID YOU LAST:

Have a cup of coffee or tea _____

Smoke a cigarette, cigar, or pipe _____

Take drugs (including aspirin) _____

Drink alcohol _____

Give blood _____

Have an illness _____

Suffer from respiratory problems _____

WHAT SORT OF PHYSICAL EXERCISE DID YOU PERFORM YESTERDAY?

_____

WHAT SORT OF PHYSICAL EXERCISE DID YOU PERFORM TODAY?

_____

DESCRIBE YOUR GENERAL FEELINGS BY CHECKING ONE OF THE FOLLOWING:

_____ Excellent	_____ Bad
_____ Very, very good	_____ Very, very bad
_____ Very good	_____ Very bad
_____ Neither good nor bad	_____ Terrible

HAVE YOU HAD ANY STOMACH CRAMPS/PAINS IN THE LAST 24 HOURS?
_____ Yes _____ No

Appendix D

Perception of Performance Scale

Subject Number: _____

Complete the following statements with the answer that best suits the way you feel today. Please take your time and read the statements carefully.

Exercised Leg

Today, in comparison to my previous test(s), I feel that my isometric strength performance will be: (please circle one)

1	2	3	4	5	6	7
much weaker	somewhat weaker	slightly weaker	the same	slightly better	somewhat better	much better

Today, in comparison to my previous test(s), I feel that my dynamic strength performance will be: (please circle one)

1	2	3	4	5	6	7
much weaker	somewhat weaker	slightly weaker	the same	slightly better	somewhat better	much better

Today, in comparison to my previous test(s), I feel that my range of motion will be: (please circle one)

1	2	3	4	5	6	7
much weaker	somewhat weaker	slightly weaker	the same	slightly better	somewhat better	much better

Subject Number: _____

Unexercised Leg

Today, in comparison to my previous test(s), I feel that my isometric strength performance will be: (please circle one)

1	2	3	4	5	6	7
much weaker	somewhat weaker	slightly weaker	the same	slightly better	somewhat better	much better

Today, in comparison to my previous test(s), I feel that my dynamic strength performance will be: (please circle one)

1	2	3	4	5	6	7
much weaker	somewhat weaker	slightly weaker	the same	slightly better	somewhat better	much better

Today, in comparison to my previous test(s), I feel that my range of motion will be: (please circle one)

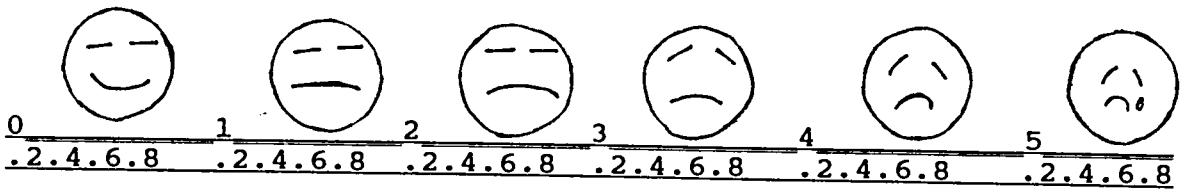
1	2	3	4	5	6	7
much weaker	somewhat weaker	slightly weaker	the same	slightly better	somewhat better	much better

Appendix E

SORENESS RATING SCALE

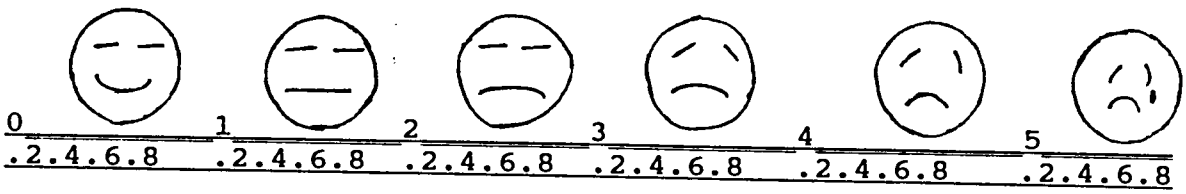
Initials _____
Date _____

EXERCISED LEG _____



Pain: None Vague Slight Slight + Painful Painful +

UNEXERCISED LEG _____



Pain: None Vague Slight Slight + Painful Painful +

None - Mild, bearly perceptible symptoms of pain

Vague - Dull ache upon palpation

Slight - Persistent discomfort, but does not interfere with movement

Slight + - Sorness which hampers complex movement

Painful - Constant pain and stiffness which interferes with most daily tasks

Painful + - Continual pain without movement

REFERENCES

Amelink, G. J., van der Wal, W. A. A., Wokke, J. H. J., van Asbeck, B. S., & Bar, P. R. (1991). Exercise-induced muscle damage in the rat: The effect of vitamin E deficiency. European Journal of Physiology, 419, 304-309.

American College of Sports Medicine. (1991). Guidelines for exercise testing and exercise prescription (4th ed.). Philadelphia: Lea & Febiger.

Appell, H. J., Soares, J. M. C., & Duarte, J. A. R. (1992). Exercise, muscle damage and fatigue. Sports Medicine, 13, 108-115.

Armstrong, R. B. (1984). Mechanisms of exercise-induced delayed onset muscle soreness: A brief review. Medicine and Science in Sports and Exercise, 16, 529-538.

Armstrong, R. B., Warren, G. L., & Warren, J. A. (1991). Mechanisms of exercise-induced muscle fibre injury. Sports Medicine, 12, 184-203.

Bansil, C. K., Wilson, G. D., & Stone, N. H. (1985). Role of prostaglandins E and F2 alpha in exercise induced delayed muscle soreness. Medicine and

Science in Sports and Exercise, 17, 276.

Berne, R. M., & Levy, M. N. (1983). Physiology. St. Louis: Mosby.

Biodex Corporation, (1988). Shirley, NY.

Byrd, S. K. (1992). Alterations in the sarcoplasmic reticulum: A possible link to exercise-induced muscle damage. Medicine and Science in Sports and Exercise, 24, 531-536.

Byrnes, W. C., & Clarkson, P. M. (1986). Delayed onset muscle soreness and training. Clinics in Sports Medicine, 5, 605-614.

Byrnes, W. C., Clarkson, P. M., White, J. S., Hsien, S. S., Frykman, P. N., & Maughan, R. J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. Medicine and Science in Sports and Exercise, 17, 276-282.

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 (Abstract). Medicine and Science in Sports and Exercise, 17, 277.

Clarkson, P. M., & Tremblay, I. (1988). Exercise-

induced muscle damage, repair, and adaptation in humans. Journal of Applied Physiology, 5, 1-6.

Davies, C. T. M., & White, M. J. (1981). Muscle weakness following eccentric work in man. Pflugers Archives, 392, 168-171.

Dedrick, M. E., & Clarkson, P. M. (1989). The effects of eccentric exercise on motor performance in young and older women. European Journal of Applied Physiology, 60, 183-188.

Donnelly, A. E., McCormick, K., Maughan, R. J., Whiting, P. H., & Clarkson, P. M. (1988). Effects of a non-steroidal anti-inflammatory drug on delayed onset muscle soreness and indices of damage. British Journal of Sports Medicine, 22, 35-38.

Evans, W. J. (1987). Exercise-induced skeletal muscle damage. Physician and Sportsmedicine, 15, 81-100.

Friden, J., Sjostrom, M., & Ekblom, B. (1981). A morphological study on delayed muscle soreness. Experientia, 37, 506-507.

Friden, J., Sjostrom, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric

exercise in man. International Journal of Sports Medicine, 4, 170-176.

Hough, T. (1902). Ergographic studies in muscular soreness. American Journal of Physiology, 7, 76-92.

Janssen, E., Kuipers, H., Verstappen, F., & Costill, D. (1983). Influence of an anti-inflammatory drug on muscle soreness (Abstract). Medicine and Science in Sports and Exercise, 15, 165.

Jenkins, R. R. (1983). The role of superoxide dismutase and catalase in muscle fatigue. Biochemistry of exercise, 13, 467-471.

Jones, D. A., Newham, D. J., Round, J. M., & Tolfree, S. E. J. (1986). Experimental human muscle damage: Morphological changes in relation to other indices of damage. Journal of Physiology, 375, 435-448.

Kreighbaum, E. & Barthels, K. M. (1985). Biomechanics: A qualitative approach for studying human movement (2nd ed.). Minneapolis: Burgess.

Muckle, D. S. (1974). Comparative study of ibuprofen and aspirin in soft tissue injuries. Rheumatology and Rehabilitation, 13, 141-147.

Newham, D. J., Mills, K. R., Quigley, B. M., & Edwards, R. H. T. (1983). Pain and fatigue after concentric and eccentric contractions. Clinical Science, 64, 55-62.

O'Reilly, K. P., Warhol, M. J., Fielding, R. A., Frontera, W. R., Meredith, C. N., & Evans, W. J. (1987). Eccentric exercise-induced muscle damage impairs muscle glycogen repletion. Journal of Applied Physiology, 63, 252-256.

Packer, L. (1984). Vitamin E, physical exercise and tissue damage in animals. Medical Biology, 62, 105-109.

Phoenix, J., Edwards, R. H. T., & Jackson, M. J. (1989). Inhibition of Ca2+-induced cytosolic enzyme efflux from skeletal muscle by vitamin E and related compounds. Biochemistry Journal, 257, 207-213.

Quintanilha, A. T. (1984). Effects of physical exercise and/or vitamin E on tissue oxidative metabolism. Biochemical Society Transactions, 12, 403-404.

Schutz, R. W., & Gessaroli, M. E. (1987). The analysis of repeated measures designs involving multiple

dependent variables. Research Quarterly for Exercise and Sport, 58, 132-149.

Schwane, J. A., & Armstrong, R. B. (1983). Effect of training on skeletal muscle injury from downhill running in rats (Abstract). Medicine and Science in Sports and Exercise, 15, 165.

Sjodin, B., Westing, Y. H., & Apple, F. S. (1990). Biochemical mechanisms for oxygen free radical formation during exercise. Sports Medicine, 10, 236-254.

Smith, L. L. (1991). Acute inflammation: The underlying mechanism in delayed onset muscle soreness? Medicine and Science in Sports and Exercise, 23, 542-549.

Tiidus, P. M., and 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.

Triffletti, P., Clarkson, P. M., Byrnes, W. C. (1985). Effect of repeated local isometric exercise bouts on serum creatine kinase activity and muscle soreness (Abstract). Medicine and Science in

Sports and Exercise, 17, 277.

Triffletti, P., Litchfield, P. E., Clarkson, P. M., & Byrnes, W. C. (1988). Creatine kinase and muscle soreness after repeated isometric exercise. Medicine and Science in Sports and Exercise, 20, 242-248.