

THE TRAINING EFFECT ON MUSCLE SORENESS
FOLLOWING DOWNHILL RUNNING
OF VARYING DURATIONS

by

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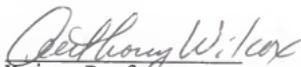
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DEDICATION

This thesis is dedicated to my parents, D.G. and Sylvia Miller, who have given me their love and their support in my return to school. It is also dedicated to my sister, Denise, my brother, Stan, their spouses, Larry and Joni, and my niece, Kelsey, who have given me encouragement and love when I needed it.

CHAPTER 1

Introduction

Physical activity to which an individual is unaccustomed is likely to result in muscle soreness 24 to 48 hours after the exercise period ends. This phenomenon is termed delayed onset muscle soreness (DOMS). Overuse of the muscle by an activity producing higher forces or forces extended over a longer period of time than the muscle is accustomed to can produce DOMS. The soreness is only a temporary discomfort and medical attention is rarely sought. The soreness will subside in most cases within five to seven days.

During dynamic exercise, a concentric contraction occurs when the active muscle shortens and performs positive work. In contrast, an eccentric contraction is when the active muscle is lengthened during dynamic exercise and performs negative work. Fewer motor units are activated to produce a given muscular force in eccentric contractions than in concentric contractions (Asmussen, 1956). The force is thus distributed over a smaller cross-sectional area of muscle. Komi and Buskirk (1972), Schwane, Johnson, Vandenakker, and Armstrong (1983), and Talag (1973) all have observed that DOMS is more likely to occur following eccentric contractions than concentric contractions. The increased tension per unit area could cause mechanical disruption of the structural elements of the contractile or

elastic elements, thus resulting in DOMS.

Exercise producing DOMS will cause morphological changes in the muscle fibers. These changes are believed to be responsible for the soreness. Friden (1984) used a bicycle ergometer, modified for use in eccentric work, to induce DOMS. The most prominent finding from morphological observation in the study was evidence of disruption of contractile material, particularly the myofibrillar Z-band. The high tension placed on the muscles during eccentric exercise caused uneven mechanical stresses on the contractile apparatus, and produced myofibrillar damage. Z-band disruption leads to the formation of degraded protein components and the release of protein-bound ions, resulting in the edema which gives the characteristic firmness and stiffness of sore muscles.

Another response to DOMS is an efflux of intracellular enzymes into the circulatory system. This occurs when skeletal or cardiac muscle tissue is damaged. The two enzymes principally studied in DOMS are creatine phosphokinase (CK) and lactic dehydrogenase (LDH). The presence of myoglobin in the urine and blood is also evident when muscle tissue has been damaged.

Several studies (Hunter & Critz, 1971; Maxwell & Bloor, 1981; & Nuttall & Jones, 1968) have reported reduced serum CK responses following an exercise training program. The

fact that both muscle pain and elevated serum enzyme levels can be eliminated with training indicates that normal healthy muscle is able to adapt to the mechanical stress that occurs during eccentric contractions (Newham, Mills, Quigley, & Edwards, 1982).

Byrnes, Clarkson, White, Hsieh, Frykman, and Maughan (1985) observed the effect of a single bout of exercise on the development of muscle soreness and CK levels in subsequent exercise. Groups with three and six weeks of recovery between exercise bouts demonstrated a decrease in muscle soreness ratings and CK levels after the second bout of exercise.

Armstrong, Ogilvie, and Schwane (1983) suggested that a pool of fragile or stress-susceptible muscle fibers are damaged after a bout of novel exercise. This would result in a large serum CK response. When the workout is repeated, fewer stress-susceptible fibers would be present due to the dynamic process of degeneration and regeneration. With a smaller pool of stress-susceptible fibers present, a smaller increase in serum CK activity and muscle soreness should be evident on subsequent bouts.

Research has documented that repeating the same bout of exercise will result in decreased responses of CK activity and muscle soreness for a three to six week interval between bouts. A prophylactic effect is seen with training,

protecting the muscles from damage on subsequent exercise bouts. How much protective effect is seen when the second bout of exercise is of a different intensity and duration has not been documented.

Purpose of Study

The purpose of this investigation was to determine the effect changing the duration of an exercise bout has on developing DOMS. The proposed investigation observed differences in the level of muscle soreness and changes in CK concentrations in the blood when the duration of the second exercise bout is extended to twice that of the first.

Hypotheses

To study this effect, three groups of subjects were used. Group I ran downhill for 20 minutes for both of their exercise bouts. Group II ran downhill for 20 minutes during their first exercise bout and 40 minutes for their second exercise bout. Group III reversed group II's protocol. They ran downhill for 40 minutes in the first exercise session and then decreased to 20 minutes in the second exercise bout.

The total work being done by the subjects in group II increased from the initial exercise bout to the second bout. The stress of the longer bout of exercise exceeded the adaptation and resistance to DOMS brought about by the first exercise session. Three separate hypotheses were proposed

for the study:

1. The control group, exercising for 20 minutes at both sessions, will experience a decrease in DOMS and serum CK activity when they repeat their initial, eccentrically-biased session.

2. The second bout of exercise will be twice as long as the first bout for one experimental group, and they will experience greater DOMS and serum CK activity following their second exercise session compared to the second run for the group running the same duration for both runs.

3. One experimental group, performing the second bout of exercise at one-half the duration as the first exercise session, will experience less muscle soreness and serum CK activity following the second exercise session compared to the second downhill run for the group running the same duration for both runs.

The present investigation examined the level of protection against muscle soreness produced by a prior bout of exercise, when a second bout of exercise was twice as long as the first. The study helped to describe the extent of the prophylactic effect of the first exercise session on the development of DOMS in subsequent exercise.

CHAPTER 2

REVIEW OF THE LITERATURE

This chapter will review the literature related to the following areas: Descriptors of sore muscles, causes of DOMS, Identifiers of DOMS, tissue examination, and training effects.

Descriptors of Sore Muscles

Onset, Duration, and Quality of Pain

Nearly everyone who has participated in a physical activity has experienced muscle soreness. Soreness arises when a muscle participates in an activity to which it is unaccustomed or when the activity is of sufficiently high intensity. Nearly everyone who has participated in a physical activity has experienced muscle soreness. The working muscle may become sore during exercise, immediately following exercise, or at 24 to 48 hours after the cessation of the activity. The term coined for the soreness appearing during or immediately following exercise is acute soreness. Delayed onset muscle soreness (DOMS) is when pain appears following exercise.

Acute Soreness

Dorpat and Holmes (1955) demonstrated that the cause of acute soreness is related to ischemia developing in muscle tissue. Ischemia develops when the work by the muscle is at an intensity where anaerobic metabolism is the primary energy pathway for the activity. Lactic acid builds up,

causing pain, possibly by increasing the acidity in the tissue. Pain will continue until the exercise intensity decreases or stops completely, thereby decreasing lactic acid production. When work is stopped, the blood flow removes the by-products and pain ceases.

Causes of DOMS

DOMS has been postulated to be caused by one of several possible mechanisms: 1) Increased metabolism resulting in accumulation of by-products toxic to the tissues; 2) Altered neural control of the muscle, producing spasms that elicit pain; or 3) Increased tension in contractile and elastic elements that cause physical damage to the structural components (Armstrong, 1984).

Metabolic Demands of Muscle Contractions

Abbott, Bigland, and Ritchie (1952) demonstrated metabolic and mechanical differences between eccentric and concentric contractions. Metabolically, less oxygen is consumed during eccentric than concentric contractions for the same amount of force production (Abbott et al., 1952 and Asmussen, 1952). Less lactic acid is produced during eccentrically-biased exercise than exercise involving primarily concentric contractions (Bonde-Petersen, Knuttgen, & Henriksson, 1972; Davies & Barnes, 1972; & Schwane, Watrous, Johnson, & Armstrong, 1983).

Helweg (cited in Asmussen, 1956) suggested DOMS might arise from lactic acid and other metabolic by-products

accumulating during exercise. This theory was contradicted by Schwane, Watrous, Johnson, and Armstrong (1983). They ran subjects on the treadmill on both level and downhill inclines and compared the metabolic demands for the different contractions and the subsequent development of muscle soreness. The subjects ran at 80% of their VO₂ max on the level and then used that same speed when running downhill. VO₂ and blood lactic acid was measured before, during, and after exercising. Blood lactic acid was increased in level running for the period of 2.5 minutes of exercising through the fifth minute of recovery. Running downhill did not elevate the blood lactate above that of the preexercise level at any time during the run or the recovery stage. The VO₂ during exercise, as predicted, was higher in the level running.

Subjects indicated they had greater soreness following downhill running compared to the more metabolically demanding level running. These findings demonstrate that the mechanism for DOMS was not the accumulation lactic acid or other metabolic by-product. The mode of exercise resulting in the greatest amount of soreness was the less metabolically demanding eccentric contractions, so little or no lactic acid accumulation appeared from these exercises.

Eccentric Contractions Causing DOMS

The number of motor units activated is greater when shortening the muscle than in lengthening the muscle.

Electromyographic studies concluded that an eccentric contraction uses fewer motor units to produce a given tension than concentric contractions (Abbott et al., 1952; Katz, 1939; & Komi, 1973). The force generated in eccentric contractions is distributed over a smaller cross-sectional area of muscle than concentric contractions. This results in greater muscle tension per unit area for eccentric contractions, despite the greater metabolic demands for concentric exercise (Bigland-Ritchie & Woods, 1976).

Activities using eccentric contractions include exercises lengthening the muscle, such as in lowering a weight in a bicep curl, running downhill, or walking downstairs. Exercises using principally concentric contractions include activities such as moving uphill or upstairs. Running on the level makes use of both eccentric and concentric contractions. In both level and downhill running, movements occur at the hip, knee, and ankle joints during each stride cycle. The body's center of mass is negatively accelerated in the vertical plane, resulting in eccentric contractions. This would affect muscles of the buttocks, quadriceps, hamstrings, and anterior and posterior muscles of the lower leg. Eccentric contractions are emphasized in downhill running because of relatively more lowering than raising of the body's center of mass.

Hough (1902) studied delayed onset muscular soreness.

According to his research, pain was associated with mechanical tension in muscle and connective tissue.

Talag (1973) studied arm exercises using eccentric, concentric, and isometric contractions in an attempt to classify the potential for soreness. The results indicated eccentric contractions elicited greater delayed onset muscular soreness than other contractions. No difference in muscular soreness was observed between static and concentric contractions.

Several studies have produced evidence to confirm that DOMS is produced from eccentric contractions (Asmussen, 1956; Edwards, Mills, & Newham, 1981; Komi & Buskirk, 1972; Newham, Mills, Quigley, & Edwards, 1983; & Schwane, Johnson, Vandenaeker, & Armstrong, 1983). The increased tension in the contractile and elastic elements during the activity could damage the muscle fibers, myofibrils, myofilaments, and proteins in the skeletal muscle, contributing to the soreness. Studies have shown that eccentric contractions resulted in a greater disruption or injury to muscle tissue than concentric contractions (Armstrong, Ogilvie, & Schwane, 1983; & Edwards, Mills, & Newham, 1981) as observed by microscopic examination of muscle tissue and measurement of intracellular enzymes in serum.

Electromyographic Studies

deVries (1966) theorized that DOMS was caused by increased electrical activity in sore muscles at rest. He

proposed that the increased resting EMG levels indicated the existence of tonic localized spasms of motor units in the affected muscles. The spasms in the muscle would then cause a compression of blood vessels. Ischemia would develop from this compression and cause soreness, similar to pain arising when muscle is exercised with the circulation arrested. This series of events is referred to as deVries' spasm theory. The hypothesis is dependent on an increase in the resting EMG for the sore muscles as compared to normal muscles. In his study, the elevated level of electrical activity was proportional to the level of pain experienced by the subjects.

Abraham (1977, 1979) duplicated deVries' study, but did not find an elevation in resting EMG levels following eccentric exercise. One discrepancy between the studies was that Abraham used bipolar electrodes in measuring electrical activity and deVries used unipolar electrodes. Bipolar electrodes are up to three times more sensitive to changes in electrical activity in the muscle than unipolar electrodes. If electrical activity is indeed increased as deVries reported, the bipolar electrodes should have detected a rise in EMG levels.

Bobbert, Hollander, and Huijing (1986), in trying to substantiate deVries' results, observed no difference in resting EMG activity in sore muscles. They followed the

subjects for 72 hours after exercise and observed no correlation between muscle soreness and EMG levels. They concluded that there is a large interindividual variation in the resting EMG levels of sore muscles. Presently deVries' spasm theory is not considered a valid explanation for the mechanism of DOMS.

In summary, three causal mechanisms have been postulated to explain DOMS. Metabolically, eccentric contractions are less demanding than concentric contractions. Less lactic acid accumulates in eccentrically-biased activities versus concentrically-biased activities, yet they cause greater soreness. DOMS, thus is not related to metabolic factors. EMG studies have shown conflicting results, but it is generally accepted that sore muscles do not have elevated resting electrical activity, disproving deVries' spasm theory. Eccentric contractions develop more tension per unit area because fewer motor units are recruited to produce a given amount of force. Therefore, the cause of DOMS seems most likely to be due to increased tension in contractile and elastic elements, causing physical damage to the structural components in the muscle.

Diminished Force Production

Muscular performance was shown to be inhibited by delayed onset muscle soreness (Hough, 1902). Hough postulated that the diminished performance resulted from a

reduced voluntary effort by the subject due to the pain felt and also from a lowered inherent capacity of the muscle to produce the force. Many investigations have found that, following eccentric contractions, there was a decrease in the force the muscle was able to produce, either electrically stimulated or as a maximal volitional contraction (Friden, Sjostrom, & Ekblom, 1983; Griffiths, 1966; Komi & Buskirk, 1972; Newham, Mills, Quigley, & Edwards, 1983; & Talag, 1973).

A comparison between eccentric, concentric and static contractions was made concerning the ability of the working muscle to generate muscular strength at several time periods following an exercise session (Talag, 1973). Muscular soreness was greater following eccentric contractions than concentric and static contractions. Muscular strength decreased immediately following eccentric contractions and remained depressed throughout the soreness period. The pretest strength values were not reattained during the 72 hour postexercise data collection period. With static and concentric contractions, a drop in strength was seen immediately following exercise, but pretest strength values were reattained by 72 hours after exercise. Talag suggested that the soreness may have inhibited the subjects' effort, contributing to the inability of the muscle to exert full force.

Weight-training programs often induce muscle soreness

from lengthening the muscle, as in slowly lowering the weight in a bicepbrachii curl. In a study by Tiidus and Ianazzo (1983), there was a temporary reduction in maximum voluntary contraction following exercises involving weights. Subjects indicated that the inability to regain their maximal strength was not due to the soreness felt, as hypothesized by Talag. Tiidus and Ianazzo suggested that the strength reduction was caused by physical damage to the exercised muscle.

Using negative work involving lowering the body from a step, Davies and White (1981) noticed that maximal twitch tension was decreased in the involved leg following exercise. The electrically stimulated and voluntary forces were not fully recovered to pretest values in the negative leg within 20 hours after exercising. In attempting to explain the loss in force, Davies and White examined the concentration of ATP and lactate accumulation in the working muscle. They found that ATP concentration remained unchanged and lactate did not accumulate to an exceptional level in the muscle during or following negative work. Davies and White suggested that due to the nature of the eccentric exercise, the contracting muscle is repeatedly stretched during the lengthening phase. The repetition of lengthening the muscle causes damage, resulting in the loss of force production.

Newham et al. (1983) found a decrease in maximum

voluntary isometric contraction at two and ten minutes following eccentric contractions. By 24 hours postexercise, maximum strength of the muscle was regained. Results of Friden, Sjostrom, and Ekblom (1983) and Newham et al. (1983) found no relationship between the decrements in strength and the muscular soreness following eccentric contractions. These studies conflict with Tiidus and Ianazzo and Davies and White, making it difficult to make a conclusive statement on how much strength, if any, is lost following eccentrically-biased exercises. If strength is lost, it is unclear whether it is related to the development of muscle soreness.

Physiological Identifiers of DOMS

Intracellular Enzymes

Damage to cardiac and skeletal muscle tissue results in an efflux of intracellular enzymes into the blood. (Fowler, Gardner, Kazerunian, & Lauvstad, 1968; Griffiths, 1966; & Shumate, Brooke, Carroll, & Davis, 1979). Creatine phosphokinase (CK) and lactic dehydrogenase (LDH) are the primary enzymes released. Damage to skeletal muscle may result from several different activities. Among them are eccentrically-biased exercises, treadmill running, and cycling on a bicycle ergometer (Fowler et al, 1968).

The onset and level of activity of enzymes can be useful in determining the extent of cardiac tissue damage. Damage to skeletal muscle results in a similar enzymatic

release and is proportional to the intensity and duration of the damaging bout of exercise (Apple, 1981; Brooke, Carroll, & Davis, 1977; & Fowler et al., 1968).

Serum enzyme levels were measured in untrained rats following running bouts on uphill, level, and downhill treadmills (Armstrong, Ogilvie, & Schwane, 1983). Immediately after exercise, CK and LDH values were elevated in level and downhill running. Enzyme levels were increased to a greater extent following downhill running. Uphill running did not increase serum levels above preexercise values.

Eccentric contractions cause high mechanical stresses resulting in cellular changes and enzyme efflux. The time course for peak enzyme levels were reported by Fowler et al. (1968) and Halonen and Knottmen (1962) as being 15 to 60 minutes following exercise. Enzyme levels returned to preexercise activity within one to two hours after exercise is completed.

In contrast, Tiidus and Ianazzo (1983), Schwane, Johnson, Vandenakker, and Armstrong (1983), and Armstrong, et al. (1983) reported peak levels for CK and LDH 24 hours postexercise. Values were still elevated at 48 hours, but they were declining towards preexercise levels.

Armstrong et al. (1983) observed a biphasic peak in postexercise enzyme elevations in rats. Immediately after exercise, CK and LDH values began to rise. By 6 to 12

hours, enzyme levels were comparable to non-exercised animals. At 36 to 48 hours, both enzymes rose above preexercise levels. Enzyme levels rose to a peak, declined, and increased to a second peak value before returning to baseline values. Monophasic elevations in CK and LDH enzyme levels are usually seen in human subjects following exercise. Enzyme levels increase to a peak and then decrease to preexercise levels. Newham et al. (1983) observed humans displaying a biphasic rise in enzyme levels. A double peak was observed in approximately one-half of Newham and coworkers subjects, while the remaining subjects had a single elevation. CK levels in the secondary peak were one to two orders of magnitude greater 48 hours postexercise than 24 hours.

Armstrong et al. (1983) suggested late phase elevations in plasma enzymes resulted from frank necrosis of fibers in the injured muscle tissue. Plasma enzyme levels noted immediately after exercise, are derived from mechanical stresses to tissue. These are two mechanisms possibly responsible for the biphasic peaks observed in rats and some humans.

Different theories have been proposed relating the enzyme efflux from the muscle cells and the damage received by the tissue during exercise. Fowler et al. (1968) and Garbus, Highman, and Altland (1964) found that excessive enzyme elevations were not always necessarily associated

with overt histological changes. Garbus and coworkers (1964) examined muscle fibers histologically following exercise, and necrotic fibers constituted only a minute fraction of the total fibers. Necrotic fibers were not apparent in many of the animals showing marked serum enzyme elevations, suggesting elevated enzymes are derived chiefly from non-necrotic cells. These studies suggest elevations in serum enzymes was due to increased membrane permeability, allowing leakage of enzymes from the cells.

Garbus and coworkers (1964) histologically examined muscle fibers following exercise, and necrotic fibers constituted only a minute fraction of the total fibers. Necrotic fibers were not apparent in many of the animals showing marked serum enzyme elevations, suggesting that elevated enzymes are derived chiefly from non-necrotic cells.

Conversely, studies have shown elevations in enzyme values when exercise-induced necrosis of skeletal muscle fibers are present (Armstrong et al., 1983; & Schwane, Johnson, Vandenakker, & Armstrong, 1983). Damaged tissue releases enzymes into blood, implying that more than permeability changes are responsible for the enzyme efflux. This damage was time correlated with late plasma increase in CK. Enzyme efflux may be due to necrotic fibers and altered membrane permeability.

In summary, intracellular enzyme levels are increased

in serum when damage to muscle occurs. Release of skeletal muscle enzymes was proportional to intensity and duration of the exercise performed. CK is increased following both eccentric and concentric exercise, but it is increased to a greater extent following eccentric exercise. Peak levels arise 8 to 24 hours postexercise. In humans, serum levels usually peak a single time and then return to preexercise levels within seven days. However, after animals have been exercised, two peaks of CK are observed before returning to normal levels.

Myoglobin

In addition to intracellular enzymes, myoglobin is also released when muscle tissue is damaged. The released myoglobin then is excreted in the urine. Abraham (1979) observed the appearance of myoglobin in the urine six hours after weight-lifting exercise. Myoglobin was present in the urine in seven of eight subjects following eccentric exercise while all eight subjects experienced delayed onset muscle soreness. A greater incidence of soreness and higher levels of myoglobinuria was evident 24 hours after exercise. Abraham then had the subjects perform a strenuous work bout. None of the subjects experienced muscle soreness but all of them had significant amounts of myoglobin in their urine. It becomes evident from the results of Abraham and Newham et al. (1983) that myoglobin's release following exercise is not specifically correlated with the development of muscle

soreness. Thus, myoglobin is not an appropriate marker to examine the correlation between muscle damage and delayed muscle soreness.

Hydroxyproline

Damaged connective tissue can be studied by the detection of components of the tissue in the subject's serum or urine following exercise. Hydroxyproline is a specific breakdown product of connective tissue. It is excreted in the urine and is measured as free and peptide-bound hydroxyproline (Steffen, 1949). The rate of collagen breakdown influences the amount of hydroxyproline excreted (Prockop & Sjoerdsma, 1961).

Abraham (1977) induced muscle soreness by having subjects perform stepping exercise. The level of hydroxyproline in the urine was measured following the exercise. Urinary hydroxyproline was not elevated immediately after the exercise. By 48 hours following exercise, the excretion of hydroxyproline was significantly higher than control values. This elevation correlated high with subject's soreness, suggesting that the connective tissue may be damaged from exercise inducing muscle soreness.

Myoglobin, hydroxyproline, and intracellular enzymes have all been examined in the search for physiological markers for delayed onset muscle soreness. Two of the markers, myoglobin and intracellular enzymes also are

present without soreness.

Tissue Changes

Morphological Examination Following

Eccentric Contractions

Studies examining morphological changes following eccentric exercise have only recently begun to appear in the literature. Friden, Sjostrom, and Ekblom (1981) induced delayed onset muscle soreness in subjects with stair-stepping exercise. No evidence of ischemic tissue injury or mechanical fiber disruption following morphological examination of the muscle tissue was found. Muscle fibers before and after exercise were tightly packed in well-organized groups.

Examination of muscle fibers at the cellular level showed no evidence of ischemic fiber necrosis or fiber rupture in sore muscles. Focal or diffuse fiber abnormalities were not apparent following examination of the muscle fibers. Focal disturbances refer to areas of disruption affecting one or two adjacent myofibrils and one or two adjacent sarcomeres. When the disruption affects more than two adjacent sarcomeres and more than two adjacent myofibrils, the damage is termed extensive. The highest level of damage is very extensive. This occurs when the damaged fibers are contained in more than one extensive area (Newham et al., 1983). Minimal, if any, tissue damage occurs at the cellular level following exercise.

Subcellular abnormalities were seen in muscle biopsies taken two days after stair-stepping exercise (Friden et al. 1981). Frequent focal disturbances of cross-striated band patterns were seen in the biopsy. Newham et al. (1983) induced muscle soreness using step testing. Subjects performed the task using the same leg to lift them up the step. The other leg was used as the descending leg. By performing the exercise in this manner, one leg is performing concentric contractions and the other eccentric contractions for the duration of the activity. Morphological examination prior to exercise revealed that muscles of both legs were free of abnormalities. Following exercise, the leg performing concentric contractions remained devoid of any signs indicating morphological damage. The eccentrically contracting leg muscles showed damage immediately after exercise, however damage was much greater 24 to 48 hours after exercise than immediately following exercise.

Electron microscope examination of muscle biopsies immediately after exercise indicated mostly focal damage, with Z-line streaming and sarcomeres having undergone disruption (Newham et al., 1983; Friden, Kjorell, & Thornell, 1984; Friden, Seger, Sjostrom, & Ekblom, 1983; Friden et al., 1981; & Friden, Sjostrom, & Ekblom, 1983). In biopsies taken one to two days after eccentric exercise, damage was similar to that seen immediately after exercise,

but involved more sarcomeres.

Vihko, Salminen, and Rantamaki (1978, 1979) observed increased lysosomal enzyme activity five to seven days following heavy exercise in mice. Friden et al. (1984) reported an increase in lipofuscin granules, indigestible residues from lysosomal degradation, in sore muscles three days after exercise. Friden, Sjostrom, and Ekblom (1983) postulated that following the primary rupturing of Z-bands from activity, the Z-band proteins are exposed to proteolytic enzymes, resulting in the greater disorganization seen three days after exercising. In contrast to Friden et al. (1983), Kuipers, Drukker, Frederik, Geurten, and Kranenburg (1983) do not believe that lysosomal activity was responsible for producing protein degradation. Kuiper's group noticed muscle fiber degeneration a few hours following exercise while the hydrolytic enzyme activity was not evident until 24 hours after exercise.

Relationship to Pain

Since the Z-line streaming is observed in muscles after performing eccentric exercise and in muscles that become sore, Friden et al. (1984) related the fiber damage with DOMS. Following an exercise bout, there is a disruption of myofibrils, leading to the formation of protein components, globular proteins, and degraded Z-proteins. Subsequently, a

release of protein-bound ions causes edema. Edema causes an increase in muscle pressure giving rise to delayed onset muscle soreness. According to Friden, structural disturbances are secondary to activation of lysosomal enzymes leading to inflammation and pain.

Training and DOMS

Soreness, CK, and LDH

DOMS occurs in highly trained athletes, if they engage in an activity to which they are unaccustomed. Training regimens result in tissues being stressed. Normal healthy muscle is well able to adapt to the mechanical stress induced. As a result of muscle adaptations, delayed onset muscle pain can be reduced and eliminated through training (Abraham, 1977; Davies and Barnes, 1972; Hill, 1951; & Komi & Buskirk, 1972)

Davies and Barnes (1972) observed that there was extreme muscle soreness in the postexercise period after performing a single bout of eccentric exercise. However, succeeding experimental sessions for the same subjects did not produce the same degree of soreness. Several studies have demonstrated the effect of training programs on serum enzymes (Hunter & Critz, 1971; Maxwell & Bloor, 1981; Noakes & Carter, 1982; & Nuttal & Jones, 1968). All research groups confirmed that eccentric training reduces delayed muscle pain in addition to decreasing CK elevations following an eccentrically-biased exercise.

Morphological Effects

Microscopic examination following a single eccentric exercise trial showed damage to muscle tissue. However, following an eccentric training program, there was an absence or decrease in widespread damage to myofibrils (Friden, Seger, Sjostrom, & Ekblom, 1983). The Z-disks adapt successfully to repeated powerful tension. Alteration of muscle tissue to training include adaptation of Z-disks to repeated powerful tension and an increase in coordination and efficiency of the movement (Friden et al., 1983).

Protection from Training

Schwane and Armstrong (1983) trained rats on the treadmill at uphill (+16 degrees), level (0 degrees), and downhill (-16 degrees) slopes. One group trained for five days and a second group for one day. Three days following training, the groups ran downhill. At 48 hours after the downhill run, the soleus, vastus intermedius, and triceps brachii were all evaluated for tissue damage. Training effects were observed in all three muscles following five days of training on level or downhill. One day of downhill or level training prevented exercise-induced injury in the vastus intermedius. Injury to rat skeletal muscle resulting from prolonged downhill running was prevented by relatively little downhill training.

Eccentric stress from training eliminates necrotic or

susceptible fibers. The remaining fibers are able to withstand an acute bout of downhill running without sustaining injury. Evidence of injury is present following a single training bout, thereby clearing the muscle of susceptible fibers (Schwane and Armstrong, 1983). This adaptation is termed the stress-susceptible hypothesis (Armstrong, Ogilvie, & Schwane, 1983).

Data from Schwane and Armstrong (1983) do not support the stress-susceptible theory. A single 30 minute downhill run did not result in observable injury to susceptible fibers, but a training effect was seen for a subsequent 90 minute downhill run. The training bout did not eliminate stress-susceptible fibers, but training did provide sufficient stimulus to muscle fibers to increase resistance to injury without undergoing degradation.

Byrnes et al. (1985) observed less muscle soreness following single training bouts on subsequent runs. Subjects ran 30 minutes downhill at a -10 degrees decline. The exercise bout was repeated at either three, six, or nine weeks after the initial bout. Muscular soreness and CK levels were measured. For a single exercise training bout, there was a decrease in CK levels and soreness perception when the subsequent run was three or six weeks following the training run. Differences in CK levels and DOMS were not apparent when the second run was nine weeks following the training run.

Summary

Delayed onset muscle soreness appears in exercised muscle between 8 to 48 hours after exercising. The pain will increase in intensity until a peak is reached between 24 to 72 hours postexercise. The involved muscles become stiff and tender with a decrease in flexibility and mobility. The soreness is usually gone in 5 to 7 days. Sore muscles are more likely to arise from eccentric contractions than from concentric contractions. The number of motor units involved in shortening a muscle is greater than in lengthening the muscle for a given tension. This results in greater muscle tension per unit area leading to tissue damage and muscle soreness in eccentric contractions.

Intracellular enzymes, CK and LDH, are used as markers to identify muscle damage. The release of these enzymes follows exercise involving eccentric and concentric contractions, but they are increased to a greater extent in eccentric contractions. The enzyme release may be from increased membrane permeability or from exercise-induced necrosis of the skeletal muscle fibers.

Another marker proven useful in identifying sore muscles is hydroxyproline. The detection of this breakdown product of connective tissue in the urine has been well correlated with the appearance of muscle soreness following exercise.

Tissue examination at the cellular level produced no

signs of ischemic tissue injury or mechanical fiber disruption in muscles which became sore following an eccentrically-biased exercise. However, at the subcellular level, abnormalities were seen in biopsies taken two days after stair-stepping exercise. Z-band disturbances are most frequently reported with a marked broadening, streaming, and total disruption in some places. The damage to the myofibrils following exercise leads to the formation of protein components, globular proteins, and degraded Z-proteins. Subsequently, there is a release of protein-bound ions, causing edema. Edema then causes an increase in muscle pressure giving rise to the delayed muscle soreness.

DOMS, rise in intracellular enzymes, and the morphological changes observed are all decreased with an eccentric training program. Relatively little training is necessary. One day of downhill running shows significant decreases in CK levels and tissue damage. These are decreased to an appreciable extent when the initial bout is repeated. This protection is present for at least six weeks following the training bout.

CHAPTER 3

METHODS

This chapter outlines procedures followed in this experiment. Included is information related to recruitment of subjects, variables tested, informed consent document, and the equipment and tests used to evaluate the hypotheses. Each subject's exercise protocol for the study is explained. Finally, the statistical methods used in analyzing the data are outlined.

Subjects

The subjects for the study were volunteers from the Manhattan, Kansas community and from Kansas State University activity classes. Well-conditioned runners were used to evaluate the hypotheses.

Equipment

Treadmill. The running was done on a motor-driven Quinton treadmill, with adjustable speed. In order to simulate downhill running, the rear of the treadmill was raised, resulting in a -10 degrees slope (Appendix B).

Procedures

The subjects were randomly divided into three groups of five subjects each. There were three stages to the experimental protocol. All three groups participated in each stage. There was a one to seven day recovery period between the first and second stages and a four week interval between stages II and III. During the first recovery

period, the subjects were asked to continue their normal exercise training program. Following their downhill running episodes, each subject was asked to refrain from exercising for 48 hours.

Stage I. On the first visit to the laboratory, each subject completed the informed consent document (Appendix A). Experimental procedures were outlined at this session. Stage I was used to find the speed which would elicit 80% of the subject's maximal oxygen uptake. In order to identify this speed, a maximal oxygen uptake test was completed in such a way as to measure the oxygen uptake at several speeds of level running. Oxygen consumption was measured throughout the test, in 30 second intervals.

The subjects warmed up by running at a relatively easy pace on the treadmill. Treadmill speed was increased to 6 mph and measurement of oxygen consumption began. The speed of the treadmill was increased in one mph increments every two minutes until 10mph was reached, or when it was judged that the subject was beyond 80% $\dot{V}O_2$ max. Once this part of the stage was completed, the $\dot{V}O_2$ max test began. As the subject continued to run on the treadmill, its slope was increased 2.5 degrees every minute until the test was complete. The $\dot{V}O_2$ max test was termed complete when the subject could not continue running. The peak value of oxygen consumption was considered the $\dot{V}O_2$ max if a plateau in $\dot{V}O_2$ was observed and if the respiratory equivalent ratio

was greater than 1.10. Using the above protocol, the speed which elicited 75% of the subject's VO₂ max could be calculated. This speed was used for stages II and III of the study.

Stage II. The second visit to the lab was seven days later. The slope of the treadmill was changed to a -10 degrees. Subjects in all three groups began running on the treadmill at the speed requiring 75% of their VO₂ max on the level as calculated in stage I.

Running bouts were split into ten minute segments with a rest period of two to five minutes between each segment. Subjects in groups I and II ran for two 10 minute segments, for a total running time of 20 minutes. Subjects in group III ran for four 10 minute segments, for a total running time of 40 minutes. Oxygen consumption was measured for the final two minutes of the first two 10 minute segments. Blood samples and muscle soreness ratings were taken before exercise began and 24 and 48 hours postexercise.

Stage III. A second bout of downhill running constituted the third stage. Running bouts were performed on the treadmill set at -10 degrees and they were split into ten minute segments, with a rest period of two to five minutes between each segment.

Group I subjects ran for two 10 minute segments, duplicating their stage II exercise. Group II subjects ran four 10 minute intervals. Group III subjects ran two 10

minute sessions. Oxygen consumption was measured for the final two minutes of the first two 10 minute segments. For each group, the speed at which the subject ran was that speed determined in stage I which elicited 80% of the VO₂ max when running on the level. Blood samples and muscle soreness ratings were taken before running downhill and at 24 and 48 hours after exercising.

Tests

Muscle Soreness. A questionnaire was administered to evaluate muscle soreness (Appendix C). General muscle soreness for several muscle groups, including the buttocks and the front and back of the upper and lower leg, was rated by each subject, using a scale with 1 (normal) to 10 (very very sore) as lower and upper limits, respectively (Byrnes et al. 1985). Subjects were asked to evaluate the soreness at the proximal, medial, and distal portions of the aforementioned muscle groups. In addition, overall soreness of the entire muscle group was rated.

Blood Sample. A blood sample was drawn to evaluate creatine phosphokinase (CK) levels. Blood was sampled using standard blood collection techniques. The blood was allowed to clot and then was centrifuged in an Aloe Conical Centrifuge to obtain serum for the test. The serum was stored at -20 degrees Celsius until analyzed. Analysis of the serum was done spectrophotometrically (Sigma 520C). The analytical procedures for determination of serum CK are in

Appendix D.

Analysis of Data

Dependent variables measured were muscle soreness ratings and CK levels. Independent variables were duration of exercise, treatment groups, and collection times. General linear models procedures(SAS) were performed. From these programs, least square means and ANOVAs were obtained. Least square means compared means of soreness and CK values for significant differences between treatment groups, collection times, and stages. ANOVAs were calculated for analysis of CK and soreness between test groups collection times and bout of exercise performed. Descriptive characteristics were analyzed for significance differences between groups by ANOVAs. Small sample t-tests were used to compare oxygen consumptions between the downhill runs. The required level of significance for all statistical tests was $p < 0.05$.

CHAPTER 4

RESULTS AND DISCUSSION

This chapter includes results of data analysis, interpretation of analysis, and a discussion of significance of results.

Results

Descriptive Characteristics

These abbreviations are used to describe results of groups, downhill runs, and sampling periods: Groups are referenced according to duration of their two downhill bouts. Group 1 is 20-20, group 2, 20-40, and group 3, 40-20. The initial downhill run is Bout A and Bout B corresponds to the second downhill run. Data obtained before exercise are preex. For the 24 hour sampling period, post 24 is used, and post 48 for 48 hours following exercise.

Data for 15 subjects (11 male, 4 female) were collected and analyzed. Means for age and weight appear in Table 1. Their VO₂ max, percent of VO₂ max used when running on level slope at a speed corresponding to the downhill running speed, and speed used for downhill running, are presented in Table 1. One-way ANOVA were used to test these variables. Table 2 contains one-way ANOVA for variables in Table 1. Significant differences were not apparent in mean age between groups ($p = 0.4451$). Overall mean age for subjects was 25.5 years with a range of 19 to 35 years. Differences

Table 1

Mean age, weight, VO2 max, % VO2 max for downhill run, and downhill running speed for groups (+ standard error)

| | GROUPS | | | |
|---|-----------------|-----------------|-----------------|-----------------|
| | 20 - 20 | 20 - 40 | 40 - 20 | MEANS |
| Number | 5 | 5 | 5 | 5 |
| males | 3 | 4 | 4 | |
| females | 2 | 1 | 1 | |
| Age (years) | 23.8 \pm 2.08 | 27.6 \pm 3.03 | 26.4 \pm 2.60 | 25.9 \pm 2.57 |
| Weight (kg) | 64.8 \pm 5.46 | 71.9 \pm 4.94 | 66.8 \pm 3.29 | 67.8 \pm 4.56 |
| VO2 Max (ml/kg/min) | 60.4 \pm 4.58 | 61.1 \pm 4.67 | 64.6 \pm 4.33 | 62.0 \pm 4.53 |
| Downhill Speed (mph) | 7.9 \pm 0.40 | 8.3 \pm 0.43 | 8.4 \pm 0.28 | 8.2 \pm 0.37 |
| %VO2 Max for Downhill Speed for level running | 75.8 \pm 3.29 | 77.6 \pm 2.40 | 75.6 \pm 2.69 | 76.3 \pm 2.79 |

Table 2

One-way ANOVA for age of subjects for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 163.33 | 32.67 | 1.05 | 0.4451 |
| Error | 9 | 279.60 | 31.07 | | |
| Corrected Total | 14 | 442.93 | | | |

One-way ANOVA for weight of subjects for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 400.23 | 80.05 | 0.70 | 0.6397 |
| Error | 9 | 1034.85 | 114.98 | | |
| Corrected Total | 14 | 1435.09 | | | |

One-way ANOVA for VO2 max of subjects for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 316.50 | 63.30 | 0.59 | 0.7089 |
| Error | 9 | 965.90 | 107.32 | | |
| Corrected Total | 14 | 1282.40 | | | |

Table 2 (cont.)

One-way ANOVA for percent of VO2 max for level running for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 101.13 | 20.23 | 0.47 | 0.7907 |
| Error | 9 | 388.20 | 43.13 | | |
| Corrected Total | 14 | 489.33 | | | |

One-way ANOVA for downhill running speed for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 2.12 | 0.42 | 0.54 | 0.7445 |
| Error | 9 | 7.12 | 0.79 | | |
| Corrected Total | 14 | 9.24 | | | |

in weight for groups were not significant ($p = 0.6397$). VO_2 max values were not significantly different ($p = 0.7089$). Oxygen consumption for subjects running on the level treadmill at the speed used for the downhill running trials was approximately 75% of VO_2 max. The analysis demonstrated no significant difference in percent of VO_2 max for level running at the downhill running speed among the three groups ($p = 0.7907$). Differences among groups for downhill running speed were not significant ($p = 0.7445$).

Metabolic Cost for Downhill Runs

Table 3 contains means for volume of oxygen consumed and the percentage of VO_2 max used on downhill runs. Oxygen collection times were for the final two minutes of the first and second ten minute periods. The two 1-minute sample measurements were averaged to obtain oxygen consumption per minute of exercise. All three groups had similar oxygen consumption levels, expressed as VO_2 and percent of VO_2 max, for Bouts A and B (Table 4). During Bout A, VO_2 was not significantly different among the groups, for either sampling period (Table 4) ($p = 0.3441$ and 0.2157 for first and second ten minutes of Bout A, respectively). Percents of VO_2 max were not significantly different during Bout A among the groups ($p = 0.7940$ and 0.2288 for first and second ten minutes of bout A, respectively). For Bout B, no significant difference among the three groups for VO_2 was found ($p = 0.7066$ and 0.7224 for first and second ten

Table 3

Mean oxygen consumption during downhill running (+ standard error)

| | GROUPS | | |
|-----------------|------------|-----------|-----------|
| | 20 - 20 | 20 - 40 | 40 - 20 |
| Bout A | | | |
| VO2 (ml/kg/min) | | | |
| First 10 min | 33.1±2.94 | 32.0±1.74 | 31.6±1.65 |
| Second 10 min | 40.8±7.22* | 36.8±1.42 | 33.2±1.98 |
| % VO2 Max | | | |
| First 10 min | 51.2±3.52 | 54.0±2.44 | 49.4±2.94 |
| Second 10 min | 61.7±6.81 | 61.2±4.07 | 51.6±2.36 |
| Bout B | | | |
| VO2 (ml/kg/min) | | | |
| First 10 min | 28.1±3.27 | 31.1±1.48 | 28.5±2.33 |
| Second 10 min | 30.7±3.31* | 33.6±1.79 | 31.0±2.35 |
| % VO2 Max | | | |
| First 10 min | 46.0±2.49 | 51.6±3.33 | 44.1±2.14 |
| Second 10 min | 50.6±2.59 | 56.2±5.23 | 48.2±2.45 |

* significantly different between Bout A and Bout B for the same time period ($p < 0.05$)

Table 4

One-way ANOVA for VO2 at first ten minute period of bout A for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 123.57 | 24.71 | 1.30 | 0.3441 |
| Error | 9 | 171.01 | 19.00 | | |
| Corrected Total | 14 | 294.58 | | | |

Table 4 (cont.)

One-way ANOVA for VO₂ at second ten minute period of bout A for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 648.17 | 129.63 | 1.77 | 0.2157 |
| Error | 9 | 660.08 | 73.34 | | |
| Corrected Total | 14 | 1308.25 | | | |

One-way ANOVA for percent of VO₂ max at first ten minute period of bout A for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 128.98 | 25.80 | 0.50 | 0.7744 |
| Error | 9 | 465.35 | 51.71 | | |
| Corrected Total | 14 | 594.33 | | | |

One-way ANOVA for percent of VO₂ max at second ten minute period of bout A for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 823.00 | 164.60 | 1.71 | 0.2288 |
| Error | 9 | 867.89 | 96.43 | | |
| Corrected Total | 14 | 1690.89 | | | |

Table 4 (cont.)

One-way ANOVA for VO2 at first ten minute period of bout B for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 97.47 | 19.49 | 0.59 | 0.7066 |
| Error | 9 | 295.73 | 32.86 | | |
| Corrected Total | 14 | 393.20 | | | |

One-way ANOVA for VO2 of second ten minute period of bout B for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 100.39 | 20.08 | 0.57 | .7224 |
| Error | 9 | 317.21 | 35.25 | | |
| Corrected Total | 14 | 417.60 | | | |

One-way ANOVA for percent of VO2 max at first ten minute period of bout B for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 334.58 | 66.92 | 2.35 | 0.1254 |
| Error | 9 | 256.18 | 28.46 | | |
| Corrected Total | 14 | 590.76 | | | |

Table 4 (cont.)

One-way ANOVA for percent of VO₂ max at second ten minute period of bout B for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 5 | 573.71 | 114.74 | 2.60 | 0.1008 |
| Error | 9 | 379.07 | 44.12 | | |
| Corrected Total | 14 | 970.78 | | | |

Table 5

Statistical comparisons between bout A and bout B for VO₂ and %VO₂ max

| GROUP | MEASUREMENT | t-Value | P-Value |
|-------|----------------------|---------|---------|
| 20-20 | VO ₂ | | |
| | First 10 minutes | 2.103 | 0.0166 |
| | Second 10 minutes | 1.138* | 0.0938* |
| | %VO ₂ max | | |
| | First 10 minutes | 1.077 | 0.3557 |
| 20-40 | Second 10 minutes | 1.363 | 0.0869 |
| | VO ₂ | | |
| | First 10 minutes | 0.370 | 0.3557 |
| | Second 10 minutes | 1.240 | 0.1075 |
| | %VO ₂ max | | |
| 40-20 | First 10 minutes | 0.517 | 0.3050 |
| | Second 10 minutes | 0.668 | 0.2546 |
| | VO ₂ | | |
| | First 10 minutes | 0.979 | 0.1635 |
| | Second 10 minutes | 0.622 | 0.2676 |
| 40-20 | %VO ₂ max | | |
| | First 10 minutes | 1.318 | 0.0934 |
| | Second 10 minutes | 0.912 | 0.1814 |

* significantly different ($p < 0.05$)

minutes, respectively). Percent of VO₂ max was not significantly different during Bout A among groups ($p = 0.1254$ and 0.1008 for first and second ten minutes, respectively).

Determinations of differences in oxygen consumption for Bout A and Bout B were made using a small sample t-test (Table 5). Significant differences for oxygen consumption, expressed as VO₂ and percent VO₂ max, were apparent only for group 20-20, during the second ten minute period. During Bout B, group 20-20 subjects consumed significantly less oxygen than in Bout A ($p = 0.0166$). Careful examination of Table 3 reveals a trend towards less oxygen consumption for Bout B, but the differences were not significant.

Muscle Soreness

Muscle soreness was evaluated using subjective questionnaires (Appendix C). Muscle groups evaluated were quadriceps, hamstrings, anterior lower leg, posterior lower leg, and buttocks. Individual scores for each muscle group are listed in Appendix E. Appendix E also includes sums of the soreness ratings for the five muscle groups.

Sums of the five muscle soreness values for each test group are presented in Table 6. Sums of the soreness ratings for the five muscle groups will be the only variable used in statistical comparisons of muscle soreness.

Time Differences. Soreness ratings, grouped by time, are presented in Table 7. Before engaging in downhill

Table 6

Sum of soreness ratings
 (+ standard error)

| GROUP | BOUT | TIME | SUM |
|-------|------|--------|-----------|
| 20-20 | A | preex | 5.1+0.10 |
| | | post24 | 18.2+3.29 |
| | | post48 | 22.6+4.46 |
| | B | preex | 5.2+0.20 |
| | | post24 | 10.2+2.25 |
| | | post48 | 11.4+2.50 |
| 20-40 | A | preex | 5.0+0.00 |
| | | post24 | 15.9+2.95 |
| | | post48 | 16.4+3.60 |
| | B | preex | 5.0+0.00 |
| | | post24 | 11.8+3.60 |
| | | post48 | 13.8+3.31 |
| 40-20 | A | preex | 5.2+0.20 |
| | | post24 | 19.6+3.90 |
| | | post48 | 17.5+2.95 |
| | B | preex | 5.2+0.20 |
| | | post24 | 8.4+0.68 |
| | | post48 | 7.2+0.49 |

Table 7

Least Square Means for sum of soreness ratings for
difference between times

| Time | Sum LSMean | Std Err LSMean | Time | P-Values |
|--------|---------------|-------------------|--------|------------------|
| | | | | post24 post48 |
| preex | 5.12 | 0.80 | preex | 0.0001 0.0001 |
| post24 | 14.02 | 0.80 | post24 | 0.6609 |
| post48 | 14.51 | 0.80 | | |

running, the average mean soreness level for the three sampling periods was 5.12. Twenty-four hours after exercising, the soreness state had increased to 14.02. Soreness values increased to 14.51 by 48 hours following exercise. Values for the combined groups indicates running downhill significantly increased soreness values above baseline levels. The differences in means for 24 and 48 hours was not significant ($p = 0.6609$).

Bout by Time Differences. Table 8 lists soreness means for Bouts A and B at preex, post 24, and post 48. The Satterthwaite Approximation was used to make comparisons between Bouts A and B at various time periods. Following this procedure, comparison of Bout A with Bout B for each measurement period was performed. Differences in soreness means for Bout A and B before exercise were non-significant ($p > 0.05$). However, 24 hours following the downhill run, soreness ratings were significantly less for Bout B ($p < 0.05$). Results 48 hours following exercise were similar to those obtained 24 hours after exercise, Bout B was significantly less than Bout A ($p < 0.05$).

Bout Differences. Least-square-means analysis for differences in soreness ratings between bouts is presented in Table 9. Combining ratings in all three groups for the three times across both bouts reveals a significantly higher mean ($p = 0.0002$) for Bout A (13.74) than Bout B (8.64).

Mean soreness value for the three groups and the two

Table 8

Least Square Means Analysis for sum of soreness ratings for differences between bouts and time

| Bout | Time | Sum LSMean | Std Err LSMean | Time | P-Value | |
|------|--------|---------------|-------------------|--------|---------|--------|
| | | | | | post24 | post48 |
| A | preex | 5.10 | 1.13 | preex | 0.0001 | 0.0001 |
| A | post24 | 17.90 | 1.13 | post24 | | 0.8361 |
| A | post48 | 18.23 | 1.13 | | post24 | post48 |
| B | preex | 5.13 | 1.13 | preex | 0.0031 | 0.0009 |
| B | post24 | 10.13 | 1.13 | post24 | | 0.6792 |
| B | post48 | 10.80 | 1.13 | | | |

Table 9

Least Squares Means Analysis for sum of soreness ratings differences between bouts

| Bout | Sum LSMean | Std Err LSMean | Bout | P-Values |
|------|---------------|-------------------|------|----------|
| | | | | B |
| A | 13.74 | 0.68 | A | 0.0002 |
| B | 8.69 | 0.68 | | |

Table 10

Least Square Means Analysis for sum of soreness ratings differences between groups and bouts

| Group | Bout | Sum LSMean | Std Err LSMean | Bout | P-Values |
|-------|------|---------------|-------------------|------|----------|
| | | | | | B |
| 20-20 | A | 14.63 | 1.18 | A | 0.0052 |
| 20-20 | B | 8.93 | 1.18 | | |
| | | | | | B |
| 20-40 | A | 12.43 | 1.18 | A | 0.2070 |
| 20-40 | B | 10.20 | 1.18 | | |
| | | | | | B |
| 40-20 | A | 14.17 | 1.18 | A | 0.0010 |
| 40-20 | B | 6.93 | 1.18 | | |

different bouts are listed in Table 10. These measurements represent the mean soreness rating for the three measurement periods. For group 20-20, Bout A averaged 14.63, Bout B was 8.93. Soreness was significantly less during Bout B compared to Bout A ($p = 0.0052$). Group 20-40 measurements were 12.43 for Bout A, and 10.20 for Bout B. A p -value of 0.2070 demonstrates that differences were non-significant for the two groups. A significant difference was observed for group 40-20 ($p = 0.0010$). Bout A means (14.17) were higher than Bout B (6.93).

Group Differences. Performing a downhill run resulted in muscle soreness. A 20 or 40 minute run in Bout A did not affect the degree of soreness experienced. A 20 or 40 minute run did not result in significant differences across the three groups in mean soreness ratings (Table 11) ($p = 0.8739$).

As indicated in Table 10, performing an exercise bout decreases soreness on a subsequent bout if the second bout was not longer than the first. However, the analysis in Table 11 gave conflicting results. According to Group by Bout comparisons, no significant differences were observed among the three groups at either bout ($p = 0.1386$). Even though group by bout interaction was not significant, least square means analysis was performed since this was a preplanned comparison desired. Examination of group means for the three times reveals that the pattern was similar for

Table 11

One-way ANOVA for sum of soreness ratings for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-------------------------|----|----------------|-------------|---------|---------|
| Model | 41 | 4043.53 | 98.62 | 5.12 | 0.0001 |
| Error | 48 | 924.00 | 19.25 | | |
| Corrected Total | 89 | 4967.53 | | | |
| Group | 2 | 23.27 | | 0.14 | 0.8739 |
| Subject (Group) | 12 | 1024.30 | | | |
| Bout | 1 | 575.07 | | 27.35 | 0.0002 |
| Group by Bout | 1 | 98.42 | | 2.34 | 0.1386 |
| Subject by Bout (Group) | 12 | 252.30 | | | |
| Time | 2 | 1678.20 | | 43.59 | 0.0001 |
| Group by Time | 4 | 45.68 | | 0.59 | 0.6692 |
| Bout by Time | 2 | 291.76 | | 7.58 | 0.0014 |
| Group by Bout by Time | 2 | 54.53 | | 0.71 | 0.5903 |

groups. Preex was the lowest value, then 24 hours following the run, soreness increased. Post 48 values were similar to post 24. From these qualifications, the least square means analysis was performed to observe differences between bouts for groups.

Exercising for 20 or 40 minutes in Bout A did not affect the amount of reduction in soreness during Bout B. Soreness levels were not significantly different among the groups, whether analyzed by group interaction ($p = 0.8739$), group by bout interaction ($p = 0.1366$), group by time interaction ($p = 0.6692$) or group by bout by time interaction ($p = 0.5903$).

Summary. Muscular soreness appeared 24 hours after the downhill run. Soreness ratings remained elevated through 48 hours. Post 24 and Post 48 values were significantly less for Bout B than for Bout A. The mean soreness ratings across the three measuring periods were significantly less for Bout B than Bout A. Comparisons of differences between groups reveal group 20-20 and group 40-20 had significantly less soreness following Bout B than that experienced from Bout A. No significant differences were observed for group 20-40. Soreness levels were not significantly different among the groups.

Creatine Phosphokinase

CK values are listed in Appendix F for each subject. Group mean CK values are in Table 12. One-way ANOVA assumes

Table 12

Mean group CK Values (+ standard error)

| GROUP | BOUT | TIME | CK | LOG CK |
|-------|------|--------|-------------|-----------|
| 20-20 | A | preex | 8.75+ 1.75 | 2.19+0.22 |
| | | post24 | 28.70+17.92 | 2.87+0.45 |
| | | post48 | 15.70+ 9.48 | 2.35+0.43 |
| | B | preex | 9.65+ 3.08 | 2.19+0.29 |
| | | post24 | 12.00+ 3.48 | 2.42+0.27 |
| | | post48 | 11.35+ 3.26 | 2.30+0.36 |
| 20-40 | A | preex | 10.55+ 4.47 | 1.96+0.58 |
| | | post24 | 18.50+ 6.28 | 2.77+0.32 |
| | | post48 | 21.95+ 7.83 | 2.90+0.34 |
| | B | preex | 21.40+10.47 | 2.27+0.76 |
| | | post24 | 22.30+ 6.60 | 2.96+0.33 |
| | | post48 | 17.10+ 7.19 | 2.65+0.32 |
| 40-20 | A | preex | 14.55+ 2.04 | 2.70+0.16 |
| | | post24 | 47.65+10.16 | 3.81+0.19 |
| | | post48 | 25.55+ 3.17 | 3.25+0.12 |
| | B | preex | 10.80+ 2.81 | 2.31+0.30 |
| | | post24 | 15.50+ 3.61 | 2.71+0.21 |
| | | post48 | 9.85+ 1.48 | 2.34+0.15 |

Table 13

One-way ANOVA for CK analysis of preexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 17 | 6125.41 | 360.32 | 3.72 | 0.0126 |
| Error | 12 | 1161.03 | 96.75 | | |
| Corrected Total | 29 | 7286.44 | | | |

Table 13 (cont.)

One-way ANOVA for CK analysis of 24 hours postexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 17 | 22518.12 | 1324.60 | 2.24 | 0.0803 |
| Error | 12 | 7103.05 | 591.92 | | |
| Corrected Total | 29 | 29621.17 | | | |

One-way ANOVA for CK analysis of 48 hours postexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 17 | 9274.01 | 545.53 | 4.14 | 0.0081 |
| Error | 12 | 1581.83 | 131.82 | | |
| Corrected Total | 29 | 10855.84 | | | |

Table 14

One-way ANOVA for Log CK analysis of preexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Squares | F-Value | P-Value |
|-----------------|----|----------------|--------------|---------|---------|
| Model | 17 | 46.36 | 2.73 | 10.74 | 0.0001 |
| Error | 12 | 3.05 | 0.25 | | |
| Corrected Total | 29 | 49.41 | | | |

Table 14 (cont.)

One-way ANOVA for Log CK analysis of 24 hours postexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 17 | 27.92 | 1.64 | 3.56 | 0.0151 |
| Error | 12 | 5.53 | 0.46 | | |
| Corrected Total | 29 | 33.45 | | | |

One-way ANOVA for Log CK analysis of 48 hours postexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|-----------------|----|----------------|-------------|---------|---------|
| Model | 17 | 24.71 | 1.45 | 3.36 | 0.1090 |
| Error | 12 | 5.19 | 0.43 | | |
| Corrected Total | 29 | 29.90 | | | |

Table 15

Least Square Means Analysis for Log CK differences between times

| Time | Log CK LSMean | Std Err LSMean | Time | P-Values | |
|--------|---------------|----------------|--------|----------|--------|
| | | | | post24 | post48 |
| preex | 2.27 | 0.09 | preex | 0.0001 | 0.0094 |
| post24 | 2.92 | 0.09 | post24 | | 0.0351 |
| post48 | 2.63 | 0.09 | | | |

equal variances exist for the groups being analyzed. Analysis of group CK values revealed that variances for each time period were not equal. Table 13 lists mean square errors for the three times. The mean square error of subjects' preexercise values is 96.75. Their mean square error for 24 hours postexercise is 591.92. At 48 hours postexercise, the mean square error for CK is 131.82. Large differences in variances at the three times prevent further analysis on CK values.

CK values were adjusted to decrease the large differences in mean square error at the three time periods. CK values were transformed logarithmically. Since zero was a possible value for CK serum level, $\text{Log}(\text{CK} + 1)$ was used. For simplicity, this variable was designated Log CK. Log CK values for each subject are listed in Appendix F. Group Log CK means are listed in Table 12. Mean square errors for Log CK are presented in Table 14. At preexercise, the mean square error was 0.25, for post 24 it was 0.46, and at post 48, it was 0.43. Transforming CK values to Log CK resulted in decreased variances between preex and post 24, and post 24 to post 48.

Time Differences. Similar to muscle soreness data, downhill running increased Log CK measurements above preex at post 24 and post 48 sampling periods (Table 15). Log CK means for preex were 2.27, 2.92 for post 24, and 2.63 for post 48. Significant differences were observed between all

three time periods. Preex was significantly lower than post 24 ($p = 0.0001$) and post 48 ($p = 0.0094$). Post 24 achieved a significantly higher level than post 48 ($p = 0.0351$).

Preex, Groups by Bouts Differences. Least square means analysis for averages of Log CK values of preex, post 24, and post 48 samples are presented in Table 16. For preex, group 20-20 had Log CK means of 2.18 before either downhill run. Group 20-40 Log CK means were 1.96 and 2.27 for Bouts A and B, respectively. These values were not significantly different ($p = 0.1874$). Group 40-20 preex Log CK means for Bout A was 2.69, and Bout B was 2.31. This difference was not statistically significant ($p = 0.1101$). None of the groups demonstrated significant changes in their baseline (preex) Log CK value between Bouts A and B. Table 17 also confirms this finding. The group by bout interaction had a p -Value of 0.1284. The duration of the previous bout of downhill running, whether 20 or 40 minutes, did not have an effect on preexercise Log CK values for the second bout of exercise.

Post 24, Groups by Bouts Differences. For group 20-20, Log CK post 24 means for Bout A and Bout B were 2.88 and 2.42, respectively, with $p = 0.1570$, indicating no significant differences between bouts (Table 18). Group 20-40 means at post 24 were also not significantly different ($p = 0.5429$). For Bout A, the mean was 2.77, and 2.96 for

Table 16

Least Square Means Analysis at preexercise for Log CK differences between groups and bouts

| Group | Bout | Log CK LSMean | Std Err LSMean | Bout | P-Values |
|-------|------|------------------|-------------------|------|----------|
| | | | | | B |
| 20-20 | A | 2.18 | 0.16 | A | 0.9998 |
| 20-20 | B | 2.18 | 0.16 | | |
| | | | | | B |
| 20-40 | A | 1.96 | 0.16 | A | 0.1874 |
| 20-40 | B | 2.27 | 0.16 | | |
| | | | | | B |
| 40-20 | A | 2.69 | 0.16 | A | 0.1101 |
| 40-20 | B | 2.31 | 0.16 | | |

Table 17

One-way ANOVA for Log CK analysis of preexercise samples for groups 20-20, 20-40, and 40-20

| Source | DF | Sum of Squares | Mean Square | F-Value | P-Value |
|--------------------|----|-------------------|----------------|---------|---------|
| Model | 17 | 46.36 | 2.73 | 10.74 | 0.0001 |
| Error | 12 | 3.05 | 0.25 | | |
| Corrected Total | 29 | 49.41 | | | |
| Group | 2 | 1.68 | | 0.23 | 0.7963 |
| Group*Bout | 2 | 1.24 | | 2.43 | 0.1284 |

Table 18

Least Square Means Analysis at 24 hours postexercise for Log CK differences between groups and bouts

| Group | Bout | Log CK LSMean | Std Err LSMean | Bout | P-Values |
|-------|------|------------------|-------------------|------|----------|
| | | | | | B |
| 20-20 | A | 2.88 | 0.21 | A | 0.1570 |
| 20-20 | B | 2.42 | 0.21 | | |
| | | | | | B |
| 20-40 | A | 2.77 | 0.21 | A | 0.5429 |
| 20-40 | B | 2.96 | 0.21 | | |
| | | | | | B |
| 40-20 | A | 3.81 | 0.21 | A | 0.0035 |
| 40-20 | B | 2.71 | 0.21 | | |

Table 19

Least Square Means Analysis at 48 hours postexercise for Log CK differences between groups and bouts

| Group | Bout | Log CK LSMean | Std Err LSMean | Bout | P-Values |
|-------|------|------------------|-------------------|------|----------|
| | | | | | B |
| 20-20 | A | 2.35 | 0.21 | A | 0.8709 |
| 20-20 | B | 2.30 | 0.21 | | |
| | | | | | B |
| 20-40 | A | 2.90 | 0.21 | A | 0.4048 |
| 20-40 | B | 2.64 | 0.21 | | |
| | | | | | B |
| 40-20 | A | 3.25 | 0.21 | A | 0.0094 |
| 40-20 | B | 2.34 | 0.21 | | |

Bout B. Group 40-20 demonstrated a significant difference between means at post 24, which were 3.81 and 2.71 for Bouts A and B, respectively ($p = 0.0035$).

Post 48, Groups by Bouts Differences. Log CK analysis for post 48 are presented in Table 19, and they demonstrated a pattern similar to that found 24 hours postexercise. Group 20-20 and group 20-40 did not demonstrate significant differences between Bouts A and B ($p = 0.8709$ and 0.4048 , respectively). The means for group 20-20 were 2.35 and 2.30 for Bouts A and B, respectively. Group 20-40 means indicated a trend towards being less for Bout B, with Log CK means of 2.90 for Bout A and 2.64 for Bout B, but, this difference was not significant. The means for group 40-20 revealed a significant lowering in Log CK response following the second bout ($p = 0.0094$). Log CK values were 3.24 for Bout A and 2.34 for Bout B.

Sample Means Groups by Bouts Differences. Averages from the three sample periods were analyzed for differences between groups and bouts (Table 20). Mean Log CK values for group 20-20, Bout A and B were 2.47 and 2.31, respectively. The difference between the two bouts was not significant ($p = 0.4593$). For group 20-40, the mean value for Bout A was 2.54 and Bout B was 2.63. No significant difference was observed between the bouts ($p = 0.7119$). Group 40-20 demonstrated less than Bout A ($p = 0.0036$). Log CK values were 3.25 for Bout A and 2.45 for Bout B. This was the only group

Table 20

Least Square Means Analysis for Log CK differences between groups and bouts

| Group | Bout | Log CK LSMean | Std Err LSMean | Bout | P-Values |
|-------|------|------------------|-------------------|------|----------|
| | | | | | B |
| 20-20 | A | 2.47 | 0.16 | A | 0.4593 |
| 20-20 | B | 2.31 | 0.16 | | B |
| 20-40 | A | 2.54 | 0.16 | A | 0.7119 |
| 20-40 | B | 2.63 | 0.16 | | B |
| 40-20 | A | 3.25 | 0.16 | A | 0.0036 |
| 40-20 | B | 2.45 | 0.16 | | |

demonstrating a significant difference between Bouts A and B.

Summary. CK values obtained from assaying blood samples were transposed into Log (CK + 1). Downhill running increased measurements above preexercise levels at 24 and 48 hours postexercise. The preex Log CK values were not significantly different between any of the groups at either bouts. Group 40-20 was the only group demonstrating a significant decrease in post 24 or post 48 measurements between Bouts A and B. The mean of the sampling periods was decreased only between Bouts A and B for group 40-20. The other two groups did not reveal a significant decrease in Log CK response for the two bouts.

Discussion

Descriptive Characteristics

Table 1 presents physical characteristics for the groups. The three groups were similar in number, age, weight, VO₂ max, and energy cost, expressed in terms of relative percent of VO₂ max. Thus, all groups exercised at the same relative degree of intensity for the downhill runs. Observed workload similarities between groups decreases the possibility varied intensities influenced the significant differences observed.

Metabolic Demands for Downhill Runs

Results of the study showed decreased energy cost for negative work, consistent with other research (Abbott et

al., 1952; Asmussen, 1952; and Byrnes et al., 1985).

Oxygen consumption data showed significant decreases between bouts of downhill running for group 20-20 (Table 5). All three groups' mean oxygen values were greater for Bout A than Bout B. A similar response was observed by Byrnes et al. (1985). Their results indicated oxygen consumption was significantly less during the second bout than the first bout. The three groups listed in this investigation were not statistically different. There was a trend towards significance but $p < 0.05$ level was not reached. Friden et al. (1983) observed an increase in efficiency of movement as a result of downhill training. Increased efficiency results in a decreased energy cost for the same workload, which was consistent with the results obtained.

Muscle Soreness and Creatine Phosphokinase

Results of the study indicated a bout of downhill running produced delayed onset muscle soreness (Table 7) and elevated serum levels of Log CK (Table 15). Before exercise, soreness was minimal. One and two days later, however, soreness and Log CK had significantly increased above preexercise levels, consistent with other investigations (Talag, 1973; Byrnes et al. 1985; Schwane, Johnson, Vandenakker, & Armstrong, 1983; & Friden et al., 1983).

Research has demonstrated peak CK and soreness levels in humans normally occur between 24 and 72 hours following

exercise (Abraham, 1977; & Schwane, Jonhson, Vandenakker, & Armstrong, 1983). Soreness at post 24 was not different than post 48, suggesting a plateau had occurred and soreness would not increase past the collecting period. The assumption was made that no significant increase in pain past 48 hours appeared.

No significant differences were found in analysis of group comparisons of Log CK or muscle soreness. All three groups experienced the same level of response for Log CK and muscle soreness. A lack of observed differences may be due to an internal threshold level preventing further damage. Once the threshold is reached, additional stress to the system will not necessarily cause any further changes in measurements. The current study indicates that a 20 minute run exceeds the minimum threshold. No indication for a subthreshold was given here. Continuing with the run an additional 20 minutes did not produce any added changes, although the trend was for the mean Log CK and soreness values to be higher. A second threshold, if apparent, was not reached in the investigation. The question not answered in the current study is whether there are a series of thresholds for a range of workloads or if there is just one threshold, and that once it is reached, no further elevated responses occurs, even by increasing the running duration.

Combining all subjects into one group revealed a training effect following Bout A for muscle soreness and Log

CK, supporting Byrnes et al. results. The control group (20-20), virtually duplicated the protocol for the group exercising three weeks apart in Byrnes et al. study. Group 20-20 developed significantly less soreness following Bout B. Log CK did not show a significant decrease for Bout B, conflicting with the results of Byrnes et al. An explanation for this may be due to large variances in CK levels between subjects. Some subjects demonstrated a large CK response following downhill runs, while the magnitude of increase in others was much less.

An indication of training, expressed as a decrease in soreness, was apparent for group 20-20. Hypothesis one indicated soreness and CK levels would decrease following a downhill running session when it was preceded by a downhill run of identical duration. The hypothesis was partially verified in that soreness decreased, while Log CK levels remained constant.

Group 20-40 did not show significant reductions in soreness and Log CK following Bout B. Having performed the training bout for one-half the duration of the second bout. Expectations were that subjects for group 20-40 would experience more pain and a greater efflux of CK following Bout B. Results indicate soreness and Log CK levels were not elevated above values obtained from Bout A. The protection observed for soreness in group 20-20 was not evident for group 20-40, indicating the protection provided

by the training was applicable for runs of equal duration, but not when the running time was doubled.

Group 40-20 demonstrated significantly greater Log CK and muscle soreness values for Bout A compared to Bout B. This was an expected finding, since the duration of Bout B was shorter. Beneficial effects of soreness derived from the first run could have been reached during the first 20 minutes of the initial run, consistent with of group 20-20. Examination of the data reveals that the lowest means for soreness were present for the 20 minute run in group 40-20. Although differences in soreness means for Bout B among the three groups were not significant, a trend was developing for less soreness for group 40-20 than for group 20-20. This would indicate a greater training affect occurred, as indicated by less soreness, when preceeding the 20 minute run by a run of 40 minutes, rather than 20 minutes.

Findings from other studies indicated that CK levels decreased when a given exercise was repeated (Byrnes et al., 1985; Hunter and Critz, 1971; & Nuttal & Jones, 1968). Results from the present study indicated that a longer run was necessary before adaptations occurred resultiing in a smaller efflux of CK. The protective affect, observed in past studies with activities of similar duration, was not evident unless the duration of the second run was decreased by one-half.

The second hypothesis postulated muscle soreness

ratings and Log CK levels during Bout B for group 20-40 would be elevated above those obtained from the second bout of group 20-20. Results indicated no significant differences were apparent between the two groups. Running for 20 or 40 minutes resulted in the same degree of soreness and Log CK release when the event was preceded by a 20 minute run. It might be that the 40 minute run did not reach the next threshold level which would increase the measured responses from the downhill run.

Support for the second hypothesis may be generated by examining the training effects obtained with muscle soreness between bouts for the three groups. Group 20-20 demonstrated a training effect between bouts with regard to muscle soreness. Group 20-40 did not have a training effect, suggesting that the 40 minute run elevated soreness levels more than the 20 minute run. Although group means were not different for soreness, the fact that group 20-20 had significant differences in the runs and group 20-40 did not, lends support to an observable difference in responses.

Hypothesis three stated Bout B's soreness and Log CK levels for group 40-20 would be significantly less than soreness and Log CK developed during Bout B for group 20-20. Data indicated rejection of the hypothesis. The only support for this hypothesis comes from the fact that the 40-20 group was the only one to produce a significant reduction in Log CK between Bouts A and B. However, the muscle

soreness and Log CK values in Bout B were not significantly different between group 40-20 and group 20-20.

Log CK was higher for group 40-20 at Bout B than group 20-20. This may be due to the large variation in subjects' CK response from the run. Soreness levels decreased significantly for both group 20-20 and 40-20 between Bout A and B. No significant differences were observed between the means, but the mean value is less for group 40-20, possibly indicating a trend towards extra benefit from exercising for a longer period.

Summary. Peak values for Log CK and soreness were obtained at 24 hours for Log Ck and at 24 to 48 hours for muscle soreness. No significant differences were observed for Log CK or soreness ratings between groups. The duration of the run did not seem to affect the response. The possibility of working within thresholds may explain this lack of a group difference. When looking at all subjects as one group, a decrease in soreness ratings was evident for Bout B, indicating a training effect had occurred. No such findings were seen for Log CK. Only group 40-20 saw a significant difference in their Log CK values between Bout A and B.

Hypothesis one stated that group 20-20 soreness and CK values would be less for Bout B than Bout A. This was supported because soreness ratings decreased for Bout B. No difference in Log CK values were observed, however. The

second hypothesis stated the second run for group 20-40 would have a greater increase of Log CK and soreness values than group 20-20's second run. This was supported because soreness ratings were significantly less for group 20-20 following their second run, whereas the second run for group 20-40 did not show significant differences. No difference in Log CK values were observed between the two groups for Bout B, however. Hypothesis three stated the second run for group 40-20 would result in decreased soreness and Log CK values, compared to group 20-20's second run. This hypothesis was rejected in that no significant difference was observed in mean values of Log CK and soreness, but a trend may have been developing for soreness ratings since the decrease in group 40-20 was of greater magnitude than group 20-20.

CHAPTER 5
SUMMARY, CONCLUSIONS, AND
RECOMMENDATIONS FOR FUTURE STUDIES

This chapter includes a summary of the procedures and results of the study, conclusions based on the results, and recommendations for future research related to the study.

Summary

Previous research demonstrated that a single bout of downhill running results in a significant decrease in muscle soreness and blood concentrations of CK when the run was preceded by a run of similar duration and intensity. This study investigated altering the duration of the initial training run, and its affect on soreness and CK levels in subsequent exercise bouts, which also were of differing durations.

Fifteen well-conditioned runners were randomly divided into three experimental groups. Group 20-20 had two bouts of downhill running of 20 minutes each. Group 20-40 ran for 20 minutes on their initial downhill run and 40 minutes for their second bout. Group 40-20 had an initial run of 40 minutes and a second run of 20 minutes. Blood samples were taken and subjective questionnaires were administered before exercise, and at 24 and 48 hours postexercise. During the downhill runs, subjects ran at an intensity which elicited 75% of their VO₂ max on a level treadmill.

Soreness ratings and Log CK were significantly

increased above preexercise samples 24 and 48 hours following each downhill bout. Statistical analyses showed that soreness ratings decreased following the second run for groups 20-20 and 40-20. No decrease in soreness was observed for group 20-40 following the second run. Log CK levels were not significantly less for groups 20-20 or 20-40. Group 40-20 showed a significant decrease of Log CK following their second downhill run, however.

Examination of soreness ratings and Log CK values indicated neither variable was affected by the duration of Bout A or B. Means of Log CK and soreness ratings showed no significant differences between the test groups. A 40 minute run did not increase soreness or Log CK levels above the values found in the groups exercising for 20 minutes.

The three hypotheses formulated for the study were as follows:

1. Group 20-20 would exercise for 20 minutes at both sessions and would experience a decrease in DOMS and serum CK activity when they repeated their downhill run.
2. Group 20-40 would exercise twice as long for their second bout as for their first bout, and they would experience greater DOMS and serum CK activity than the 20-20 group following their second exercise session.
3. Group 40-20 would exercise at one-half the duration of the first exercise session during their second session and they would experience less muscle soreness and serum CK

activity than the 20-20 group's second downhill session.

The first hypothesis was partially supported since soreness ratings were decreased. CK ratings did not decrease following the second run, however. Hypotheses two and three were both rejected. No treatment differences existed between groups for either downhill running bout.

Conclusions

Downhill running induces soreness and increases serum CK activity above preexercise values. The stress induced during the run results in increased tension development and tissue abnormalities as evident by an efflux of CK into the blood. The mechanism responsible for the increased levels was beyond the scope of the study.

A training effect for DOMS was apparent following a single bout of downhill running, provided the initial bout was of equal or longer duration than the subsequent run. The initial run lead to a protective effect for the second run. Protection was not apparent when the second run was extended to twice the duration of the first run. CK activity did not show evidence of a training effect following the second bout of downhill running when the subsequent run was of equal or greater duration than the first run. A training effect for CK was evident when the second bout was one-half the duration of the first.

Recommendations

Recommendations for future studies include:

1. A similar study examining the same variables, serum CK activity and soreness rating, while altering the intensity of the two bouts.
2. A study determining the quantity of training necessary until soreness levels, following eccentrically-biased exercises, are not significantly different than baseline levels.
3. A study examining the effect of anabolic-steroids on recovery or prevention of delayed onset muscle soreness.

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APPENDIX A

INFORMED CONSENT PART I

Title of project: Muscle soreness following downhill running

Investigators: Gary Miller, Dr. Anthony Wilcox

I _____ voluntarily consent to participate in this study examining the effect of training on muscle soreness. As a participant, I will be randomly placed into one of three groups: one that performs two 20 minute downhill runs, one that performs a 20 minute and, later, a 40 minute downhill run, or one that performs a 40 minute and, later, a 20 minute downhill run. Three to four weeks will separate the first run from the second. Both runs will be at the same speed, which can be characterized as being a moderately-fast pace.

The downhill runs will likely result in sore muscles in my legs. The soreness experienced may range between some soreness to very sore and may last for two to four days. The stiffness/soreness is not unusual for a person who exercises, being the type of soreness that can follow a hard workout or competitive event. Medical attention is not called for, since the soreness disappears after a few days.

With each downhill run, three blood samples will be taken from a forearm vein by standard blood withdrawal techniques. One will be just prior to exercise while the second and third samples will be 24 and 48 hours following exercise, respectively. The blood withdrawal may cause some temporary discomfort and a bruise might occur at the site of needle insertion.

A maximal oxygen uptake test will be conducted. I will begin the test running at a slow, easy pace on the treadmill. The speed and/or inclination will be increased at two minute intervals, continuing until I am too tired to run any longer, at which point I will signal for the test to stop. Overall, the test takes 10-14 minutes with only the last few minutes being at a high intensity. The effort is comparable to the effort involved in running a half-mile race. The fatigue upon completion persists for only a few minutes.

I understand that I am not to change my exercising routine for the time period between the downhill runs.

The benefits of my participation in this study, in part, will be that of contributing in a scientific research project that will contribute to understanding the effects of training on developing muscle soreness. I will also acquire knowledge having to do with my aerobic condition.

I understand that the data derived from any participation in the project will remain confidential. I will be informed to the results of my tests, but I will not be identified in any way in any subsequent presentation or publication of results of the study.

I have been completely informed and understand the nature and purpose of the study. I understand that if any questions arise concerning the procedures or purpose of the study, the researchers will answer them. I understand that I will be able to withdraw from the study at any time.

I understand that the regulations of the state prohibit Kansas State University from carrying insurance for financial compensation in the event of physical injury resulting from the testing. I understand the procedures and potential risks involved.

Signature _____

Date _____

INFORMED CONSENT
PART II
MEDICAL AND CONDITIONING HISTORY

NAME _____ SEX _____ AGE _____

HEALTH RISKS

| | |
|---------------------------|--------------------------------|
| Have you ever had: | Are you taking medication for: |
| High blood pressure _____ | High blood pressure _____ |
| Low blood pressure _____ | Low blood pressure _____ |
| Heart disease _____ | Heart disease _____ |
| Diabetes _____ | Diabetes _____ |

Do you have a family history of coronary or other atherosclerotic disease prior to age 50? _____

MEDICAL QUESTIONS

Have you ever been advised by a physician to avoid exercise? _____

Do you ever have difficulty breathing? _____

Have you ever experienced fainting or dizzy spells? _____

Do you smoke? _____ If yes, how much? _____

Is there any other health conditions that might limit your participation in the study? (i.e. bone and joint disabilities, pregnancy, etc.) _____

Do you have abnormal blood cholesterol levels? _____

Do you have abnormal resting ECG readings? _____

TRAINING HISTORY

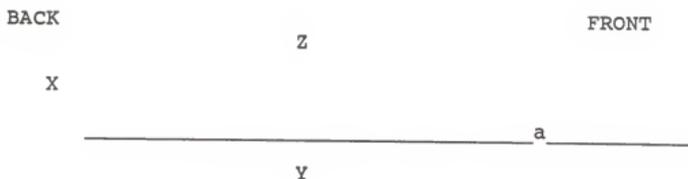
For the past 6-9 months, what has been your average weekly mileage? 5-10 miles/week, 10-20 m/wk, 20-30 m/wk, over 30 m/wk

Do you participate in fun runs or races? _____ If yes, what are some of your best times? _____

Have you had your fitness status checked in the past 6 months? _____ If yes what was your assessment? _____

APPENDIX B
DOWNHILL SLOPE

The steps employed to ensure that the treadmill used in downhill running was at a -10 degree slope are outlined below.



X = HEIGHT TREADMILL RAISED
Y = FLOOR UNDER TREADMILL
Z = TREADMILL
a = ANGLE DESIRED (10 DEGREES)

KNOWN VARIABLES:
a = 10 degrees

Z = 150.00CM

solving for X:

$\text{SIN } a = X / Z$

$\text{SIN } 10 = .1736$

X = 26.04CM

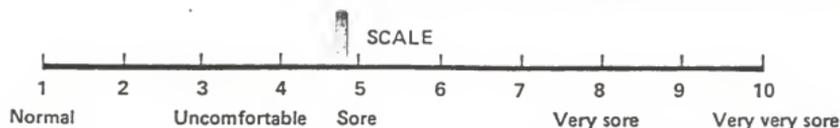
UNKNOWN VARIABLES:
X = ??????
Y = ??????

APPENDIX C
MUSCLE SORENESS

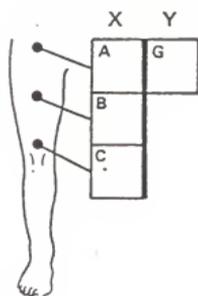
The purpose of this questionnaire is to evaluate the degree of muscle soreness after exercise

In column X please palpate (press in lightly) your muscle in the areas indicated on the diagram and then rate the degree of soreness

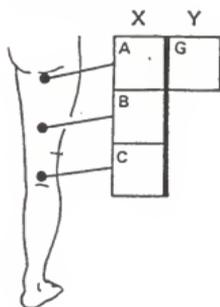
In column Y please indicate the general degree of soreness of the entire muscle when using or moving it (i.e. without pressing in on it).



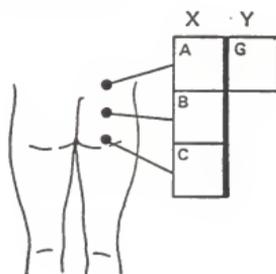
FRONT OF THIGH



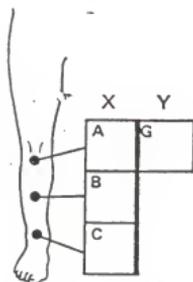
BACK OF THIGH



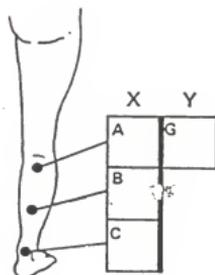
BUTTOCKS



FRONT OF LOWER LEG



BACK OF LOWER LEG



Abdomen general

APPENDIX D

CK ASSAY

Instruments

Spectrophotometer set at 540nm

Reagents

Phosphocreatine Solution
ADP-Glutathione Solution
p-Hydroxymercuribenzoate Solution
alpha-Naphthol Solution
Deacetyl Solution
Creatine Standard

Procedures

1. Two test tubes were prepared for each sample of serum.
2. .5ml of Phosphocreatine Solution and .1ml of a 10-fold dilution of serum in water was pipeted into each tube.
3. To start the reaction, .2ml of ADP-Glutathione solution was added.
4. The tubes were then incubated for exactly 30 minutes in a 37 degree Celsius water bath.
5. To stop the reaction, .2ml of p-Hydroxymercuribenzoate Solution was added.
6. To each tube 1.0ml of alpha-Naphthol Solution, 1.0ml Diacetyl Solution, and 7.0ml of water was added.
7. To allow for color development, each tube was incubated for 15-20 minutes in a 37 degree Celsius water bath.
8. At this time, the tubes were centrifuged for 15 minutes.
9. With the use of the spectrophotometer, the color development in each tube was read.
10. CK activity was determined from a previously constructed calibration curve. The values are reported as Sigma Units/ml.

APPENDIX E

RAW DATA
MUSCLE SORENESS RATINGS ($\bar{X} \pm$ S.E.)

| SUBJECT | GROUP | BOUT | TIME | M1 | M2 | M3 | M4 | M5 | SUM |
|---------|-------|------|------|-----|-----|-----|-----|------|------|
| 1 | 20-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 2.0 | 1.0 | 4.0 | 1.0 | 2.0 | 10.0 |
| | | | 3 | 2.0 | 1.0 | 3.0 | 1.0 | 1.0 | 8.0 |
| | B | 1 | 1.0 | 1.0 | 2.0 | 1.0 | 1.0 | 6.0 | |
| | | 2 | 1.0 | 1.0 | 2.0 | 1.0 | 1.0 | 6.0 | |
| | | 3 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| 2 | 20-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 5.0 | 2.0 | 3.0 | 3.0 | 1.0 | 14.0 |
| | | | 3 | 5.0 | 2.0 | 3.0 | 2.0 | 1.0 | 23.0 |
| | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| | | 2 | 2.0 | 1.0 | 1.0 | 1.0 | 1.0 | 6.0 | |
| | | 3 | 3.0 | 2.0 | 2.0 | 1.0 | 1.0 | 9.0 | |
| 3 | 20-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 8.0 | 2.0 | 1.0 | 7.0 | 8.0 | 26.0 |
| | | | 3 | 8.0 | 2.0 | 6.0 | 8.0 | 7.0 | 31.0 |
| | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| | | 2 | 2.0 | 1.0 | 1.0 | 1.0 | 4.0 | 9.0 | |
| | | 3 | 2.0 | 1.0 | 2.0 | 1.0 | 4.0 | 10.0 | |
| 4 | 20-20 | A | 1 | 1.5 | 1.0 | 1.0 | 1.0 | 1.0 | 5.5 |
| | | | 2 | 1.0 | 1.0 | 8.0 | 9.0 | 7.0 | 26.0 |
| | | | 3 | 2.0 | 1.0 | 5.0 | 8.0 | 6.0 | 22.0 |
| | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| | | 2 | 2.0 | 1.0 | 5.0 | 5.0 | 5.0 | 18.0 | |
| | | 3 | 3.0 | 1.0 | 6.0 | 6.0 | 4.0 | 20.0 | |
| 5 | 20-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 3.0 | 3.0 | 2.0 | 3.0 | 4.0 | 15.0 |
| | | | 3 | 6.5 | 6.0 | 5.0 | 6.5 | 5.0 | 29.0 |
| | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| | | 2 | 4.0 | 2.0 | 1.0 | 1.0 | 4.0 | 12.0 | |
| | | 3 | 3.0 | 2.0 | 1.0 | 4.0 | 3.0 | 13.0 | |
| 6 | 20-40 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 7.0 | 1.0 | 3.0 | 1.0 | 5.5 | 17.5 |
| | | | 3 | 9.0 | 1.0 | 5.0 | 1.0 | 1.0 | 17.0 |
| | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| | | 2 | 3.0 | 1.0 | 1.0 | 1.0 | 1.0 | 7.0 | |
| | | 3 | 4.0 | 1.0 | 3.0 | 1.0 | 1.0 | 10.0 | |
| 7 | 20-40 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 4.0 | 2.0 | 2.0 | 1.0 | 1.0 | 10.0 |
| | | | 3 | 3.0 | 1.0 | 1.0 | 1.0 | 1.0 | 7.0 |
| | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 | |
| | | 2 | 3.0 | 1.0 | 2.0 | 2.0 | 1.0 | 9.0 | |
| | | 3 | 4.0 | 1.0 | 1.0 | 3.0 | 1.0 | 10.0 | |

| SUBJECT | GROUP | BOUT | TIME | M1 | M2 | M3 | M4 | M5 | SUM |
|---------|-------|------|------|-----|-----|-----|-----|-----|------|
| 8 | 20-40 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 4.0 | 4.0 | 7.0 | 4.0 | 7.0 | 26.0 |
| | | | 3 | 4.0 | 3.0 | 6.0 | 5.0 | 8.0 | 26.0 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 5.0 | 6.0 | 5.0 | 3.0 | 7.0 | 26.0 |
| | | | 3 | 5.0 | 4.0 | 6.0 | 5.0 | 7.0 | 27.0 |
| 9 | 20-40 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 5.0 | 1.0 | 2.0 | 1.0 | 1.0 | 10.0 |
| | | | 3 | 6.0 | 1.0 | 3.0 | 1.0 | 1.0 | 12.0 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 3.0 | 1.0 | 1.0 | 1.0 | 1.0 | 7.0 |
| | | | 3 | 5.0 | 1.0 | 3.0 | 1.0 | 1.0 | 11.0 |
| 10 | 20-40 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 2.0 | 1.0 | 3.0 | 5.0 | 5.0 | 16.0 |
| | | | 3 | 4.0 | 4.0 | 3.0 | 5.0 | 4.0 | 20.0 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 2.0 | 2.0 | 1.0 | 2.0 | 3.0 | 10.0 |
| | | | 3 | 1.0 | 4.0 | 1.0 | 1.0 | 4.0 | 11.0 |
| 11 | 40-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 3.0 | 1.0 | 2.0 | 3.0 | 5.0 | 14.0 |
| | | | 3 | 4.0 | 2.0 | 3.0 | 3.5 | 6.0 | 18.5 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 2.0 | 1.0 | 2.0 | 2.0 | 3.0 | 10.0 |
| | | | 3 | 2.0 | 1.0 | 1.0 | 1.0 | 2.0 | 7.0 |
| 12 | 40-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 8.0 | 3.0 | 6.0 | 9.0 | 5.0 | 31.0 |
| | | | 3 | 4.0 | 1.0 | 4.0 | 5.0 | 1.0 | 15.0 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 2.0 | 1.0 | 1.0 | 2.0 | 2.0 | 8.0 |
| | | | 3 | 2.0 | 2.0 | 1.0 | 1.0 | 1.0 | 7.0 |
| 13 | 40-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 2.0 | 1.0 | 1.0 | 5.0 | 3.0 | 12.0 |
| | | | 3 | 1.0 | 1.0 | 1.0 | 6.0 | 1.0 | 10.0 |
| | | B | 1 | 1.0 | 2.0 | 1.0 | 1.0 | 1.0 | 6.0 |
| | | | 2 | 1.0 | 1.0 | 1.0 | 1.0 | 2.0 | 6.0 |
| | | | 3 | 2.0 | 1.0 | 1.0 | 1.0 | 1.0 | 6.0 |
| 14 | 40-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 2.0 | 6.0 |
| | | | 2 | 2.0 | 1.0 | 2.0 | 5.0 | 4.0 | 14.0 |
| | | | 3 | 2.0 | 2.0 | 4.0 | 4.0 | 5.0 | 17.0 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 4.0 | 1.0 | 1.0 | 1.0 | 2.0 | 9.0 |
| | | | 3 | 2.0 | 1.0 | 1.0 | 1.0 | 2.0 | 7.0 |
| 15 | 40-20 | A | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 8.0 | 1.0 | 4.0 | 7.0 | 7.0 | 27.0 |
| | | | 3 | 9.0 | 2.0 | 4.0 | 7.0 | 7.0 | 27.0 |
| | | B | 1 | 1.0 | 1.0 | 1.0 | 1.0 | 1.0 | 5.0 |
| | | | 2 | 3.0 | 1.0 | 1.0 | 1.0 | 3.0 | 9.0 |
| | | | 3 | 4.0 | 1.0 | 1.0 | 1.0 | 2.0 | 9.0 |

Time 1 = Preexercise Time 2 = 24 hours postexercise
Time 3 = 48 postexercise M1 = Quadriceps M2 = Hamstrings
M3 = Shins M4 = Calves M5 = Buttocks

APPENDIX E

GROUP MUSCLE SORENESS RATINGS ($\bar{X} \pm S.E.$)

| GROUP | BOUT | TIME | M1 | M2 | M3 | M4 | M5 |
|-------|------|------|----------|----------|----------|----------|----------|
| 20-20 | A | 1 | 1.1±0.10 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 |
| | | 2 | 3.8±1.24 | 1.8±0.37 | 3.6±1.21 | 4.6±1.47 | 4.4±1.36 |
| | | 3 | 4.7±1.20 | 2.4±0.93 | 4.4±0.60 | 5.1±1.50 | 4.0±1.26 |
| | B | 1 | 1.0±0.00 | 1.0±0.00 | 1.2±0.20 | 1.0±0.00 | 1.0±0.00 |
| | | 2 | 2.2±0.49 | 1.2±0.20 | 2.0±0.77 | 1.8±0.80 | 3.0±0.68 |
| | | 3 | 2.4±0.40 | 1.4±0.25 | 2.4±0.93 | 2.6±1.03 | 2.6±0.68 |
| 20-40 | A | 1 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 |
| | | 2 | 4.4±0.81 | 1.8±0.58 | 3.4±0.93 | 2.4±0.87 | 3.9±1.23 |
| | | 3 | 5.2±1.07 | 2.0±0.63 | 3.6±0.87 | 2.6±0.98 | 3.0±1.38 |
| | B | 1 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 |
| | | 2 | 3.2±0.49 | 2.2±0.97 | 2.0±0.77 | 1.8±0.37 | 2.6±1.17 |
| | | 3 | 3.8±0.73 | 2.2±0.73 | 2.8±0.92 | 2.2±0.80 | 2.8±1.20 |
| 40-20 | A | 1 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 | 1.2±0.20 |
| | | 2 | 4.6±1.40 | 1.4±0.40 | 3.0±0.89 | 3.0±1.02 | 4.8±0.66 |
| | | 3 | 4.0±1.38 | 1.6±0.24 | 3.8±0.55 | 5.1±0.64 | 4.0±1.26 |
| | B | 1 | 1.0±0.00 | 1.2±0.20 | 1.0±0.00 | 1.0±0.00 | 1.0±0.00 |
| | | 2 | 2.4±0.51 | 1.0±0.00 | 1.2±0.20 | 1.4±0.24 | 2.4±0.24 |
| | | 3 | 2.4±0.40 | 1.2±0.20 | 1.0±0.00 | 1.2±0.40 | 1.8±0.24 |

Time 1 = preexercise Time 2 = 24 hours postexercise

Time 3 = 48 hours postexercise

M1 = Quadriceps M2 = Hamstrings M3 = Shins M4 = Calves

M5 = Buttocks

THE TRAINING EFFECT ON MUSCLE SORENESS
FOLLOWING DOWNHILL RUNNING
OF VARYING DURATIONS

by

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Delayed Onset Muscle Soreness (DOMS) refers to pain appearing 8 to 48 hours following an unaccustomed exercise. The causal mechanism of DOMS is believed to be due to increased tension in myofibrils from eccentric contractions. Eccentric contractions recruit fewer motor units in generating the same amount of force than concentric contractions. The tension per area of muscle is thus greater in eccentric contractions. The weak link in the contractile chain appears to be Z-bands. The damage to the myofibrils following exercise leads to the formation of protein components, globular proteins, and degraded Z-proteins. DOMS can be prevented or decreased following training for the eccentrically-biased exercise. The present study investigated the training effect a single bout of downhill running has on developing soreness following a subsequent run of varying durations. Previous research has demonstrated that two identical runs decreased soreness following the second exercise for a period of three to six weeks.

Results from the current study revealed soreness levels decreased following the second downhill run when the duration was identical or one-half the duration of the initial run ($p = 0.0052$ and 0.0010 , respectively). A second

downhill run of twice the duration as the first, did not result in significant reduction in soreness for the second run ($p = 0.2070$). No differences in degree of soreness was apparent following runs of 20 or 40 minutes.

The intracellular enzyme, creatine phosphokinase (CK) was measured in the study. It is present when damage to muscle tissue has incurred. No significant difference in CK levels were apparent following the second downhill run, when both runs were of identical duration or the second run was twice the duration of the first ($p = 0.4593$ and 0.7119 , respectively). However, a significant decrease was observed when the second run was of one-half the duration of the first ($p = 0.0036$).