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THE EFFECTS OF STATIC STRETCHING AND WARM-UP ON PREVENTION OF DELAYED-ONSET MUSCLE SORENESS

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

David M. High

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DEDICATION

To my parents,

Katharine and Thomas,

whose unbounded love and support

shall never be forgotten.

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ABSTRACT

It has been suggested in the lay literature that static stretching and/or warm-up will prevent the occurrence of Delayed-Onset Muscle Soreness (DOMS). Therefore, the primary purpose of this study was to systematically determine to what extent static stretching and/or warm-up affects the level of pain associated with DOMS. Sixty-two healthy male and female volunteers were divided randomly into four groups. The four groups were (1) Ss who static stretched the quadricep muscle group before a step test, (2) Ss who only performed a stepping warm-up, (3) Ss who both stretched and performed a stepping warm-up prior to a step test, and (4) Ss who only performed a step test. The step test required subjects to do positive work with their right leg and negative work with their left leg. The step test followed procedures used by Asmussen (1956). Ss rated their muscle soreness on a ratio scale from 0 to 6 at 24 hour intervals for five days following the step test. An 4x2x2 ANOVA and Duncan's new multiple range post hoc test found no difference in peak muscle soreness among the stepping groups, or for gender (alpha > 0.05). There was a significant difference in peak muscle soreness between eccentrically and concentrically worked legs, with the eccentrically worked leg having more muscle soreness. The conclusion of this study is that static stretching and/or warm-up does not prevent DOMS resulting from heavy exercise.

CHAPTER I

INTRODUCTION

Muscle soreness is a common experience following heavy, unaccustomed exercise. Since this delayed-onset muscle soreness (DOMS) is such a common experience, one might think that the physiological causes of DOMS are known and that preventive measures exist. Unfortunately, that is not the case. Little is known about the basic cellular mechanisms which cause DOMS and there is an apparent lack of research to systematically determine what actions might be taken to avoid its occurrence. Unfortunately, many people starting an exercise program will quit because of the muscle soreness that develops after one bout of exercise.

However, some facts are known about DOMS. DOMS is most commonly associated with eccentric work of muscles (31) and is most commonly found in muscles following work to which the muscle has not been conditioned. Soreness usually develops around 8 hours after exercise and may last up to 7 days (23). The discomfort of DOMS usually reaches its peak within 48 hours following the exercise. The soreness is characterized by a reduction in performance which lasts the duration of the soreness. The severity of the pain ranges from little discomfort to disabling pain. After one has experienced soreness, there seems to be a "prophylactic" effect which lasts up to 6 weeks (32).

Although some facts are known about DOMS, research to

systematically determine what actions can be taken to prevent DOMS are still lacking. In spite of this, the American Medical Association (AMA), has made recommendations to the lay population at large that there is support for the notion that a warm-up period prior to strenuous exercise will limit the occurrence of DOMS. Scientific evidence to support this claim is lacking. The AMA (125) recommended two methods for relieving DOMS and five ways for preventing DOMS. The two methods for relieving DOMS were "mild exercise" and holding the muscle "in an extended position for a brief period." The five ways for preventing DOMS were: (1) warm-up before exercising, (2) use static sretching when warming up, (3) progressively increase workloads over time, (4) avoid bouncing and jerky motions and (5) warm down after the exercise (125, p. 42). Although, Asmussen (14) mentions that exercise will relieve the pain associated with DOMS, he states that exercise will only relieve that pain for the duration of that exercise. As soon as the exercise ceases, the soreness will return. Likewise, deVries (45) reports that static stretching relieves the pain associated with DOMS but only for a short period of time following the bout of stretching. Therefore, these activities are only temporary measures for relieving DOMS and should not be prescribed as long term methods for the relief of DOMS. Only two of the recommendations for preventing DOMS have been researched or discussed in the literature. These are that jerky movements might aid in developing muscle soreness and that a slow progression in the intensity of the exercise will lessen the pain associated with DOMS (81, 83, 103, 127). These unproven preventive measures along with

recommendations for the use of aspirin, analgesic muscle rubs, heat and cold, massages, and dosages of vitamin C are all present in the popular press as proven or possible steps in combating DOMS (57, 77, 125). These recommendations are based on intuitive grounds, rather than empirical evidence and underline the need for futher research into the physiological mechanisms that cause DOMS.

Purpose

The primary purpose of this study was to determine to what extent pre-exercise static stretching and warm-up affect the level of pain associated with DOMS following exhausting exercise.

Hypotheses

The following hypotheses were be tested in this study: (1) stretching or a combination of stretching and warm-up, prior to a soreness inducing task, will not diminish the magnitude of DOMS experienced as a result of exhausting bench step exercise, (2) there will be no difference in the perception of soreness between genders, and (3) there will be a significant difference between the perception of muscle soreness in the right leg (which does concentric work) and in the left leg (which does eccentric work), with the left leg having the higher perceived pain.

Definitions

Terms related to Delayed-onset Muscle Soreness are defined below:

Concentric contraction. A muscle contraction in which the muscle fibers are shortened while under tension (positive work).

Connective tissue. A basic tissue group of the body which is responsible for binding and supporting.

Creatine kinase. An intramuscular enzyme which is present in the blood following exercise.

Cytoskeletal. A another way of refering to the skeletal muscle cell.

Dantrolene sodium. A drug which inhibits the release of calcium by the sarcoplasmic reticulum.

Dehydrogenase. An enzyme that catalyzes the oxidation of a substance which causes that substance to give up its hydrogens.

Double-blind study. A study designed so that both the experimenter and subjects do not know what group is receiving the independent variable or a placebo.

Eccentric contraction. A muscle contraction in which the muscle fibers are lengthened while under tension (negative work). This type of contraction seems to be responsible for the occurrence of DOMS most of the time.

Electromyogram (EMG). A graph of the electrical activity of a muscle.

Erythrocytes. A mature red blood cell.

Extensor muscles. Muscles that restore a body part to its anatomical position after flexion.

Fascial sheath. Membrane that covers, supports, and separates muscle fibers.

Fiber necrosis. Death of muscle cells.

Hydroxyproline. Special amino acids that are associated with connective tissue.

I-bands. Name given to the light region of the muscle cell and contains mostly actin filaments, with the z-line bisecting the band.

Lactate (Lactic acid). The end-product of the anaerobic metabolism of glucose.

Leukocyte. A White blood cell.

Macrophage. A large phagocytic "cell eating" cell which is common in loose connective tissue.

Mitochondrion. The cellular structure responsible for most of the ATP production in the cell.

Muscle biopsy. Dissecting a muscle tissue in order to have microscopic examinations for structural damage.

Muscle Fiber Types. There are two major muscle fiber types: fast twitch fibers and slow twitch fibers.

Muscle nociceptors. Pain receptors found in the muscles.

Mylinated. The white lipid which covers certain nerve cells.

Myoglobin. A protein usually found in skeletal muscle and released into the blood when muscle fibers are damaged.

Myofibril. A threadlike structure found in muscle cells which contains actin and myosin.

Osmotic pressure. The pressure which develops when two solutions of different concentrations are separated by a semipermeable membrane.

Phagocytic cell. A cell that "eats" or engulfs substances.

Sarcolemma. The cell membrane of the muscle cell.

Sarcomere. The portion of a striated myofibril lying between two adjacent z-line.

Sarcoplasmic reticulum. A network of canals in the muscle cell.

Sliding filament theory. A theory that states that the contraction of muscle fibers is due to filaments sliding over one another.

Z-line (z-band). The border of the basic unit of muscle contraction, the sarcomere.

CHAPTER II

REVIEW OF THE LITERATURE

This chapter will present a review of the literature on delayed-onset muscle soreness. Static stretching and warm-up will be reviewed only as they relate to the prevention or relief of delayed-onset muscle soreness.

There are two types of muscle soreness which occur as the result of exercise. These two types of muscle soreness are referred to as "immediate muscle soreness" and "delayed-onset muscle soreness" (DOMS). A distinction is being made here so that the reader will have a clear understanding of the differences between these two types of muscle soreness. Immediate muscle soreness is pain that occurs during or at the end of an exercise. This type of soreness may last up to 2 or 3 hours following the exercise. However, DOMS differs from immediate muscle soreness in that the sensation of soreness does not start to occur until after 8 hours following exercise (23).

What is Delayed-onset Muscle Soreness (DOMS)?

Delayed-onset muscle soreness is the perceived pain and stiffness which occurs in the skeletal muscles of a person who has performed unaccustomed muscular activity. It is a localized soreness which appears in the exercised muscle between 8 to 48 hours after the activity has ended (23, 73, 127). The greatest preceived intensity of

the pain has been reported to occur from 24 to 72 hours, post exercise (8). The soreness may last up to 7 days before dissipating. The effects of DOMS range from severe pain and stiffness which interferes with and limits the range of movement of muscles involved, to only a slight sensation of tenderness or tightness felt only when touched (60). External pressure (128, 177) or movement (83) of a sore muscle makes the soreness or pain more noticable. DOMS is temporary in nature and medical attention is usually not sought by individuals suffering its effects. Yet, it has been reported that muscle soreness can be so intense as to prohibit normal standing or walking (14).

Who can suffer from DOMS?

Every healthy adult has experienced DOMS at some time or another in his/her lifetime. DOMS is experienced by athletes, but is usually associated with those individuals who engage in exercise after a long deconditioning phase (2, 8, 14, 23, 31, 32, 60, 83, 107, 127). A large number of people who start exercise programs quit because of the discomforts of DOMS. Hill (81) reported that anyone who engages in any physical activity to which the muscle being used is not conditioned can suffer from DOMS. This phenomenon has been reported to be especially true when the exercised muscle performs eccentric contractions (14, 32, 33, 53, 59, 103, 105, 128, 154, 155). Athough every adult has experienced the pain and discomfort of DOMS, it has been a common, and yet, unexplained observation that pre-adolescent children do not suffer from DOMS. It may be that they have greater

elasticity or flexibility of muscle or connective tissue compared to adults. Futher research needs to be done in this area.

What Physiological Changes Accompany DOMS?

Hough (83) reported a reduction in muscular performance with the occurrence of DOMS. Two possible explanations for this reduction in maximal muscular force were given. One explanation was that the subjects voluntarily reduced their effort due to the pain they were experiencing. The other explanation was that the muscle inherently could not function at the same level of intensity as before because of DOMS. Since Hough reported a decrease in the perceived pain soon after the onset of a bout of exercise without an increase in the maximal force output of the muscle, he concluded that the reduction was probably more inherent than voluntary in nature. This explanation has found support in later research (41, 103, 128, 177). Davies and White (41), using electrical stimulation, showed that the intrinsic ability of a muscle to produce force was lower with DOMS. Newham et al. (128) reproduced these findings, except they found a reduction in force production in both eccentrically and concentrically worked muscles, with the eccentrically worked muscles exhibiting a greater fall in force production.

Much of the literature suggests the possibility that with strenuous, unaccustomed exercise, there is a physical change (implied to be damage) to the muscle fibers or connective tissue (2, 14, 21, 53). Many investigators have evaluated DOMS by looking for the

appearance of markers that would signify injury or damage to the muscle fibers or connective tissues. It has been thought that the appearance of intramuscular enzymes in plasma following exercise is an indication of such physical damage (4). Increases in creatine kinase (CK), lactate dehydrogenase, myoglobin, hydroxyproline, and acid hydrolase have all been understood to be suitable markers by which injury to muscle or connective tissue can be evaluated. Extensive research has been done looking at the effects of exercise on plasma creatine kinase. Examples include studies on exercise of long duration (6, 7, 29, 43, 50, 73, 130, 131, 145, 153, 164), short duration (16, 18, 28, 31, 35, 38, 70, 71, 100, 105, 109, 128, 134, 146, 163, 179), with different types of training (55, 56, 76, 84, 133, 139, 143, 148, 182) with different types of injury (66, 72, 98, 112, 115, 152, 172, 173), and with animals (4, 13, 62, 79, 80, 94, 111, 113, 156, 183, 184). Likewise, extensive research has also been done in measuring levels of lactate dehydrogenase (34, 44, 74, 75, 137, 154, 166, 177), myoglobin (2, 66, 101, 135, 167, 173), hydroxyproline (2, 67, 138), and acid hydrolase (150) in relationship to muscle damage following exercise. Some of the literature suggest that the rate of development of DOMS is very similar to the rate of increase in plasma enzymes. Likewise, intensity of exercise has a direct correlation to the level of plasma enzymes following exercise (37, 154, 177). A majority of these studies support the claim that the increase in serum enzyme "activity following exercise results from mechanical disruption of the muscle" (37, p.370). However, Byrnes et al. (32, p.285) in a recent article on DOMS, points out that

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

Muscle biopsies of animals and humans have been performed following heavy exercise and during the occurrence of DOMS to see if any relationship exists between changes taking place in the ultrastructure of muscle and DOMS (60, 61, 128). It has been suggested that minute tears in the muscle fibers or in surrounding connective tissue are responsible for DOMS (14, 81, 83, 171). Muscle biopsies in animals have revealed damage to be generally located in deep extensor muscles (13, 107, 184). Following eccentric work in rats, Armstrong et al. (12) found the degeneration of muscle fibers. These fibers were being invaded by "macrophages" and had "widened I-bands." Friden et al. (60) and Friden et al. (61) found "z-band disorganization" and disruption of the "contractile machinery" in human skeletal muscles following eccentric work. They (61, p. 15) concluded that, "muscle subjected to unusual exercise contains myofibrillar and cytoskeletal disturbances accompanied by increased lysosomal activity."

Hagerman et al. (73) found in extracellular and extravascular spaces of marathon runners' muscles many "mitochondria," "erythrocytes," "leukocytes," or other "phagocytic cells." They reported "z-line streaming and degeneration," "contracture knots," "disrupted sarcolemmas," and "empty basal lamina tubes." They suggested that the inflammatory reaction that occurs with these abnormal activities could be responsible for DOMS. Their findings are similar to those reported by other investigators (60, 128, 129, 164). Kasperek and Snider (94, p. 32) found support for their own hypothesis "that the increase in the rate of protein degradation observed after exercise was due to repair of muscle tissue that was damaged during the exercise bout." Similar reports of myofibrillar protein degradation following exercise in rats has been reported (48, 168). If the reader is interested in the regeneration of skeletal muscle fibers following injury, please see a review by Carlson (33).

When does DOMS Usually Occur?

Hill (81) reported that DOMS will develop in any muscle which has been exercised at an intensity or duration that exceeds that to which the muscle is conditioned. However, muscles performing eccentric contractions have been shown to be more susceptible to DOMS (2, 14, 27, 40, 45, 53, 103, 105, 116, 123, 127, 128, 176). The metabolic cost of eccentric exercise is less than concentric exercise (1, 14, 20, 39, 40, 93, 102, 114, 127) at the same power output. Perceived exertion (78) is also less with eccentric exercise than with

concentric exercise. However, despite a lower metabolic cost, it has been demonstrated that greater tension can be developed in a muscle fiber that is working eccentrically than a muscle fiber that is working concentrically (1, 95, 102, 186).

Studies using electromyographic techniques have shown that the total muscle electrical activity during eccentric contractions is nearly half of what it is during concentric contractions, even though the same force was generated (17, 20, 21, 22, 25). Therefore, eccentric work requires the activation of fewer motor units having a much greater tension per cross sectional area of each motor unit. With a greater tension being developed by fewer muscle fibers, the likelihood of injury occurring to the muscle or connective tissue is increased. It is therefore plausible to say that most occurrences of DOMS in athletics are due to eccentric contractions.

What is the General Anatomical Location of DOMS?

DOMS can be located in any skeletal muscle which performs an eccentric exercise at an intensity to which it is not accustomed. Experimentally, DOMS has been produced by using step tests, weight lifing (14, 46), finger resistance exercises (83), and downhill running (31, 153). Newham et al. (128) and Tiidus & Ianuzzo (177) reported that with acute DOMS, pain is located throughout the muscle's belly. However, the tenderness associated with DOMS has most often been reported to be localized in areas of the muscle which contain the greatest amount of connective tissue (14, 53, 103, 128). These

reports raise the question of whether or not the elasticity of the muscle is responsible for DOMS. These areas, where the muscles and tendons join, are thought to be the most inelastic regions of the muscle. Thus, excessive tension placed on these areas would produce the highest degree of muscle trauma (14). Newham et al. (128, p. 130) reported from unpublished work of D. A. Jones that "the angle between the muscle fibers and the line of pull of the tendon is greatest in these areas," therefore providing another reason for injury to the connective tissue. This concentration of tenderness can also be explained by the fact that most of the muscle nociceptors are located in these regions (108, 170). Therefore, perceived pain might be located in these areas because of the nociceptors and not because of actual injury to the area.

Exercise-induced injury has also been reported to occur more often in deep red muscle tissue than white muscle tissue of animals (13, 149, 183, 184). This increased injury to deep red (predominantly slow twitch) muscle fibers is thought to be caused by the fact that these fibers are recruited more often during exercise (4, 9, 63, 175, 186). Although it is presumed that the same pattern of injury would occur in humans, this fact has not yet been documented (8).

What Preventive Measures are there for DOMS?

One might get the impression from the lay literature that extensive research has been done in this area. However, this is not the case. The lay literature and The American Medical Association

have reported the following possible ways to preventing DOMS, when starting an exercise program. These preventive measures include: static stretching before exercising, warm-up before and cool-down after exercise, taking aspirin while training, and using alternate treatments of heat and cold following a workout (57, 77, 125). In general, there is little scientific evidence to support these claims (8, 161). Warm-up will increase the body temperature and therefore may prove benefical to performance (58, 159, 160). Yet, there is no scientic evidence to suggest that warm-up will prevent DOMS. Likewise, static stretching may enhance performance and prevent injuries (136, 141, 159) but has not been shown to prevent DOMS. However, one possible way of preventing muscle soreness that does have evidence to support it is to exercise with only concentric contractions (32, 64, 65). However, most exercises require some eccentric work and therefore this is not always a plausible preventive measure. Likewise, there remains a question as what extent, if any, concentric contractions are responsible for DOMS. Since the intensity of pain associated with DOMS is directly related to the intensity and duration of an exercise, another possible way of lowering the pain level might be to begin slowly and gradually increase intensity and duration over time. Probably the best prevention of DOMS occurs with training. There is an adaptation which takes place with training which provides protection aganist reoccurring bouts of DOMS with exercise at the same intensity (157). Brynes et al. (31) reported that one bout of DOMS prevents the development of another bout of DOMS in the same muscle from the same exercise up to six weeks.

What Type of Relief is there for DOMS?

Hough (83) found that DOMS disappeared in a muscle when it was exercised in the manner that had caused the muscle soreness. This relief for DOMS was only temporary and the pain soon returned after the cessation of work. Armstrong noted that this is probably the best known way of effectively reducing soreness. DeVries (45, 46) reported that static stretching significantly reduced the pain associated with DOMS. However, McGlynn et al. (116) found no difference in perceived pain when trying to reproduce deVries' work. Claims have been made that massage can decrease muscle soreness (5). An explanation for this can be found in the Gate Control Theory of Pain (117, 118). This theory proposes that neural impulses are controlled by a mechamism (at the nerve synapses) which acts like a gate in order to increase or decrease the flow of nerve impulses. "The theory suggests that large-fiber inputs tend to close the gate while small-fiber inputs generally open it" (119, p. 149). When a muscle is massaged, the larger nerves which carry the touch sensation from the skin to the spinal cord would close the gate and thus lower the pain sensation which is carried over the smaller nerves. More research is needed in this area before any strong conclusion can be made. However, Politino et al. (140) used a double-blind clinical study to show that a topical analgesic containing 10% triethanolamine salicylcte in a cream base reduced muscle soreness. The cream probably worked in reducing the perception of pain by either blocking pain receptor neurons or inhibiting pain by the above mentioned "gate" theory, instead of

actually affecting the underlying mechanisms which cause DOMS. Different types of drugs have been used to see what effect they have on DOMS. Anti-inflammatory drugs have not proven to be effective in diminishing the perception of soreness (89). With two subjects who were experiencing dull painful aching, Walton (187) reported a complete recovery of the patients by 60 mg. of verapamil three times daily. Verapamil blocks the release of Ca++ ion from the sarcoplasmic reticulum. It was suggested, in this case, that the muscle fiber necrosis might be calcium induced. It was hypothesied that "excessive calcium load upon the muscle mitrochondria accounted for the pain and that this has been reduced by the verapamil" (187, p. 993). Bertorni et al. (19) reported a similar case of muscle pain in a subject. They used dantrolene sodium (a drug which inhibits the release of calcium by the sarcoplasmic reticulum) to achieve the same results. This drug would prevent the muscle's ability to contract.

The Role of Calcium Ions in the Breakdown of Muscle Tissue

Calcium ions (Ca++) play an important role in the activation and deactivation of muscle tissue in the sliding filament theory of muscle contraction (85,86). However, as stated above, Ca++ ions seem to play an important role in muscle pains (19, 187), as well as muscle diseases (30, 42). It seems that abnormally high concentrations of Ca++ ions in muscle cells cause a reduction in the energy capacity of that cell due to the accumulation of Ca++ ions in the mitrochondria. Therefore, the cell is limited in its performance of its ordinary

function (189, 190). It has been shown that high concentrations of Ca++ ions activate an enzyme within the cell that removes the z-discs and damages the cell membrane (30, 42, 87). Exactly when and why this occurs in relation to exercise is not understood. Yet, this suggests that Ca++ ions play an important role in the leakage of enzymes from the muscle cell by a change in the muscle membrane as well as a breakdown of the muscle membrane (169). Enzyme leakage from the cell has been well documented (see review above) in relationship to muscle injury. Therefore, Ca++ ions could play a significant role in the underlying mechanisms associated with DOMS. More research needs to be done in this area.

What Pain Mechanisms are Responsible for DOMS?

The pain mechanisms are not well understood in human beings and the nociceptors (pain receptors) that would sense the type of pain associated with DOMS have not been identified (8). Pain sensation in muscle tissue is known to be conducted by mylinated group III and unmyelinated group IV afferent nerve fibers (69, 121, 122). Unmyelinated group IV fibers carry dull pain sensations which travel at slow speeds and are therefore thought to be more closely associated with the pain of DOMS (69, 97). These fibers end in the muscle and connective tissue (170). Exactly what agent would stimulate the nociceptors during the occurrence of DOMS can only be hypothesized at this time. The literature suggests that mechanial stimuli (69, 108), elevated pressure (88, 106), thermal stimuli (69, 108), chemical

substances (79, 108), and electrical stimuli (45, 46, 69) could all be responsible. If the reader would like to review the basic mechanisms and physiology of pain, the following reviews are suggested (69, 97, 120).

Proposed Hypotheses of the Mechanisms Responsible for DOMS

Six major hypotheses have been proposed with regard to the phenomenon of DOMS. These hypotheses include: (1) accumulation of waste products (such as lactic acid) in muscle tissue following exercise, (2) tearing of muscle tissue, (3) connective tissue damage, (4) cycles of spasms in the muscle tissue, (5) elevated temperature causing structural damage to muscle tissue, and (6) increase in intracellular metabolites which increase osmotic pressure within the cell and results in fluid retention. These hypotheses will be presented separately, even though they may be found compatible in a working model of the underlying mechanism for DOMS.

Waste Products Accumulation

The first hypothesis suggests that lactic acid accumulation in exercised muscle causes DOMS (49). The accumulation of lactic acid has been, in recent years, the most popular hypothesis for the underlying mechanism for DOMS. Asmussen (14) discussed Helweg's review of the origin of this hypothesis in the Scandinavian literature. The hypothesis does seem logical in that the intensity of

DOMS is directly related to the intensity of the exercise. However, research over the past ten years has brought much evidence in opposition to this hypothesis. The hypothesis is weak because there is no significant amount of lactic acid left in the muscles at the time DOMS is experienced (155). Also, eccentric contractions produce significantly lower quantities of lactic acid than do concentric contractions because of the lower metabolic cost of eccentric work. Yet, eccentric contractions are more effective in causing DOMS (1, 12, 14, 21, 24, 39, 155).

Torn Tissue Hypothesis

The second hypothesis, developed by Hough (83), suggests that microscopic tears in the muscle fibers following heavy work are responsible for DOMS. Hough reported that there are two types of muscle soreness. The first type is associated with the mechanisms that cause fatigue in a working muscle and the second type of soreness is the result of ruptures within the muscle fibers. This second type of soreness appears around eight hours after the exercise has ended and remains noticeable for up to four or more days. He reported that DOMS is more prevalent with slow rhythmic contractions. Although Hough presents no direct evidence for muscle fiber damage, more recent evidence suggests that with DOMS, muscle injury does occur. Structual damage in muscle has been suggested by elevations in plasma enzymes (12, 15, 51, 84, 92, 99, 110, 124, 144, 147, 151, 158, 174) and verified by changes in the ultrastructure of muscle under an electron

microscope (13, 26, 60, 61, 62, 68, 73, 107, 127, 128, 129).

Friden et al. (60) found abnormalities in the "filament system" of muscles with DOMS. Friden et al. (64, p.18) reported finding a considerable increase in amount of "lipofuscin granules" in sore muscles. They stated two possible explanations for this finding; "it may be due to either an acutely increased protein degradation after exercise or a gradually enhanced turnover of contractile protein in muscles usually subjected to physical activity." Even though this hypothesis does have very strong support, muscle cell injury has not been proven to be the underlying causative factor in DOMS.

Although much of the literature has been in general support of this hypothesis, there are critics. DeVries (45) raises the question, that if the torn tissue hypothesis is correct, why does the pain not occur immediately when the injury takes place as with other injuries (142). As stated earlier, the pain mechanisms are not well understood in man and the nociceptors that would sense the type of pain associated with DOMS has not been identified (8). The delay in pain could be due to the time needed for injuried muscle cells to die and repair of the tissue to begin.

Connective Tissue Damage Hypothesis

Another hypothesis which is closely related to the previous one is the connective tissue damage theory. In fact, it could be argued that to separate connective tissue damage from muscle fiber damage is impossible. Nonetheless, this hypothesis is presented in the

literature as a distinct hypothesis and will be addressed as such. This hypothesis states that DOMS is caused by the overstretching of elastic elements of the muscle or connective tissue (2, 14, 103, 181). It has been frequently reported that the most severe soreness develops in areas of the muscle which contain the greatest amount of connective tissue (14, 53, 103, 128). These areas are where the muscle and tendon join and are thought to be the most inelastic regions of the muscle. Since these regions are inelastic regions of the muscle, it is thought that the highest degree of muscle trauma from exercise would be localized there. Newham et al. (128, p. 130) report from unpublished work of D.A. Jones that "the angle between the muscle fibers and the line of pull of the tendon is greatest in these areas," thus providing another reason why injury may occur in these localized areas. However, this localization of tenderness could also be explained by the fact that most of the muscle nociceptors are located in the area of the fascial sheaths, and tendons (108, 170). DeVries (45), Jessup (90) and McGlynn et al. (116) report increased EMG activity in sore muscle. Although there is much support for the two previously mentioned hypotheses, injury to connective tissue or muscle fibers still remain to be proven as the underlying mechanism for DOMS.

The Muscle Spasm Hypothesis

A fourth hypothesis on the possible underlying mechanisms of DOMS is the muscle spasm theory (38, 45, 178). Cobb et al. (38, p. 119) reviewed the historical origin of this hypothesis. DeVries, a strong

supporter of this hypothesis, believes that "the delayed, localized soreness which occurs following unaccustomed exercise is caused by tonic localized spasms of motor units." He, along with his colleagues (45, 46), reported elevated electromyographic (EMG) activity in sore muscles. They suggested that the soreness was caused by a vicious cycle of reflex induced muscle contractions which caused ischemia and pain. Proof of this was the elevated EMG activity with DOMS. DeVries (45) claimed the pain could be reduced by static stretching and showed reduced EMG activity in sore muscles following stretching. He also reported that unipolar electrodes placed on the skin was the only kind of lead system sensitive enough to record elevated EMG activity of a sore muscle. Jessup (90) found support for the muscle spasm hypothesis when looking at local blood flow in exercised muscles. He reported that a reduction in local muscular blood flow appeared at the same time higher EMG activity levels were present, which were associated with DOMS. However, deVries' hypothesis has it critics. For if deVries' hypothesis is correct, elevated EMG activity would always have to be present with sore muscles. Abraham (2, p. 73) using bipolar electrodes found there to be "no relationship between the development of muscle pain and the EMG activity" in sore muscles. However, since deVries (45) had already reported that unipolar electrodes were the only kind of lead system sensitive enough to record elevated EMG activity of sore muscles, this variation in technique could explain why Abraham could not repeat deVries' findings. On the other hand, Newham et al. (128) using unipolar electrodes, found no elevated EMG activity during post exercise pain.

McGlynn et al. (116) did report elevated EMG activity following eccentric contractions, but reported finding no correlation between the elevated EMG activity and perception of pain. Because of this evidence against the spasm theory, this hypothesis is generally not viewed in the literature as being a probable mechanism for DOMS.

Elevated Temperatures

A fifth hypothesis for the underlying mechanisms which are responsible for DOMS is elevated temperature associated with exercise. Temperatures of 38 to 48 degrees C are known to trigger pain responses from type III and IV nerve endings (108). It has been hypothesized that elevated temperatures during exercise could conceivably result in damage and/or inflammation to the muscular structure. Eccentric contractions, which are more effective in causing DOMS, produce higher, local, muscular temperatures than do concentric contractions (40, 126). However, you would then expect the pain to be experienced during the exercise and not following it. But, if muscle damage occurred due to increased temperatures, inflammation of this damaged tissue would also be associated with higher temperatures, which would stimulate the type III and IV pain receptors (108).

Increased Intracellular Metabolites and Osmotic Pressure

A sixth proposed hypothesis is that increases in intracellular metabolites and osmotic pressure cause muscle soreness (52, 162, 176).

This hypothesis states that due to the increase in intracellular metabolites with exercise, an increase in osmotic pressure and fluid retention in the muscle cell occurs (88, 106). This inflammation activates the nociceptors which results in the pain sensation. Several studies have reported the same type of pain that occurs with DOMS following the injections of saline solution or sodium chloride into human skeletal muscle (38, 96, 188). Asmussen (14, p. 114) found that his data "tentatively suggested that the soreness is caused by a swelling of or in the connective tissue, causing it to be distended at right angles to its natural direction of traction." Korge & Viru (106) discuss the shifts of Na+, K+, and water in the skeletal muscle of rats. They reported that after a short treadmill run in untrained rats, water content of the muscle increased. But, with trained rats, there was the opposite effect. Several studies have looked for increases in the white blood cell count in humans following exhausting exercise as an indication of inflammation. Smith et al. (165) reported finding an increase while Schwane et al. (156) and Bobbert et al, (23) did not. Gollnick and King (68) in comparing swimming and running reported a swelling in the muscle fibers and mitrochondria only after exhaustive running. There is a close relationship between muscle inflammation and soreness, as reported by Armstrong et al. (10), Asmussen (14), and Kihlstrom et al. (99). However, anti-inflammatory drugs have had no effect of DOMS (89). An increase in muscle fluid could possibly influence blood flow and cause edema and muscular soreness.

Conclusion

Although these hypotheses have been presented separately, in some cases they can be tied together in a proposed mechanism for DOMS. For example, Armstrong (8, p. 535) proposed a model of DOMS, that in his opinion, "best fits the available data." His model included (1) injury to both connective and muscle fiber tissue due to high mechanical forces, (2) damage to the sarcolemma due to high mechanical forces, (3) damage to the sarcolemma due to high influx of Ca++ which accompany high mechanical forces, (4) diffusion of intracellular components (such as deterioration of the sarcolemma) into the plasma following exercise, and (5) the accumulation of potassium, kinins and histamine around group IV free nerve endings causing "elevated pressure from tissue edema and increased local temperature" and resulting in the activation of nociceptors and percieved pain of DOMS. However, there is still much research needed on the basic cellular mechanisms of DOMS before a proven model of DOMS can be presented.

CHAPTER III

METHODS AND PROCEDURES

In this chapter the proposed methods and procedures of the present study will be presented in order to address the hypothesis mentioned earlier.

Purpose

The primary purpose of this study was to determine to what extent pre-exercise static stretching and warm-up affect the level of pain associated with DOMS at twenty-four hours following exhausting exercise.

Description of Subjects

Sixty-two healthy volunteers, with no known health problems, were used in this study. The sixty-two subjects (Ss) were divided into four groups randomly. Each of groups 2,3, and 4 had eight males and eight females (18-39 years), drawn from students taking physical education classes at the University of Tennessee during the Fall of 1985. Group 1 had seven males and seven females (18-38 years), drawn from the same population.

Step Test

The method for inducing DOMS was a step test given according to the procedures of Asmussen (14). Groups 1,3, and 4 performed the step test. The step's height was adjusted according to gender in the following manner: 50 cm for males and 40 cm for females. The Ss were asked to use their right leg for positive work (ascent) and their left leg for negative work (descent). By using each leg for either positive or negative work, an internal control was set up because the left leg was the only leg expected to get sore. The pace of the step test was kept by an electronic metronome at sixteen steps per minute. In most cases, work continued until the Ss could no longer maintain the workrate. However, if a subject reached the one hour and twenty minute mark into the test and had not stopped for his/her first 3 minute rest period, the subject was then allowed to stop. The reason for this was that a continuation over this time was not likely to increase the soreness rating of the subject. If a subject did fatigue before the hour and twenty minute mark, he/she was then allowed to rest for three minutes. The resting period was followed by 3 more bouts of the same fatiguing exercise with a three minute rest period between each.

Procedures

The study was conducted over a one-week period at the Applied Physiology Lab at the University of Tennessee, Knoxville. On the

first day the Ss were randomly assigned to a group. The Ss were divided into four groups: Group 1 performed stretching exercise immediately prior to taking the step test, Group 2 performed only a warm-up with no step test (control group), Group 3 performed a warm-up and static stretch before the step test, and Group 4 performed only the step test (control group). Each day (every 24 hours) after the step test, the Ss rated their muscle soreness and record their scores.

Stretching

Groups 1, and 3 performed the following static stretching of the quadricep muscle group in the following order: (1) behind the body (see Figure 5, Appendix D), (2) over the ankle (see Figure 6, Appendix E), (3) hurdler stretch (see Figure 7, Appendix F), (4) lying on the side (see Figure 8, Appendix G), and (5) sitting on the legs (see Figure 9, Appendix H).

Warm-up

Groups 2, and 3 performed a warm-up which consisted of stepping on a bench 10 cm high for ten minutes. The Ss were asked to use their right leg for positive work (ascent) and their left leg for negative work (descent). The pace was kept by an electric metronome at sixteen step per minute. A ten-second heart rate count was taken within the first ten seconds of completion of the warm-up exercise.

All Groups

Each group met for differing amounts of times before the step test depending on what they were being asked to perform. After the Ss arrived at the lab, each subject was asked to sign a consent form and list their social security number and phone number on a sheet of paper in case they needed to be contracted. The Ss were then instructed on how to fill out the muscle soreness rating chart and told to make sure to rate their muscle soreness at 24 hour intervals following the step test. This was stressed again upon the completion of the step test or warm-up (Group 2) and before the subject was allowed to leave the lab. The Ss then rated their soreness for that day. Ss that were sore on the first day were dropped from the study.

Group 1

Group 1 met approximately twenty-five minutes before the step test in order to perform the series of static stretches of the quadricep muscle group. After signing the consent form, the Ss were then told that they would be stretching for eighteen minutes using the previous mentioned exercises. Each exercise was demonstrated and all Ss were asked if they had any questions. They were then told that they would have one minute following the stretching before they would start stepping. The step test was then explained in detail (which leg to step up with, which leg to step down with, rate they would be keeping, and three minutes of resting between each bout of stepping).

The stretching exercises were then performed in the previous stated sequence (each one of the five exercises being performed for fifty seconds with ten seconds of rest between each). Each exercise, except the fifth exercise (Sitting on the Legs), were performed alternating between the right and left legs. Each of these exercises were performed four times during the bout of stretching (twice on each leg). The fifth exercise was only performed twice during the bout of stretching because it required both legs to be stretched at once. Overall, each quadricep muscle group on each leg received 100 seconds of stretching from each of the five exercises or 500 seconds of stretching overall. It took eighteen minutes to complete the stretching routine. The stretches were performed on mats with the project director leading each exercise. Ss were told to take each stretch to a point where they started to feel some discomfort. They were told not to bounce but to hold the static stretch. After completing the stretching exercises, the Ss then rested for one minute before starting the step test. During this time, they were reminded of the rate and procedure for the step test. At this time, each subject was asked to keep track of the amount of time he/she was stepping as well as the three minutes of rest between each bout of stepping. Ss then underwent the step test, continuing until they perceived their right leg to be fatigued and could no longer keep up the pace, or until they reached the time cut off point of one hour and twenty minutes without stopping. Those Ss who did not stop, did four bouts of this fatiguing exercise. Once a subject stopped for a three minute rest, he/she was asked how long he/she had be stepping. This

time was recorded each time a subject stopped. Upon completion of the test, all time of stepping was added up to reveal how long each subject had stepped. The Ss rested for at least five minutes in the lab after the completion of the test in order to insure that they did not have any physical problems. During this time, they were reminded about keeping the muscle soreness chart and making sure they performed this task at 24 hour intervals.

Group 2

Group 2 performed the warm-up exercise. After signing the consent form, the Ss were instructed on how they would be stepping during the warm-up session and how to take heart rate from the radial artery pulse. The Ss then performed the warm-up session that has been explained previously. After completing the warm-up they took the their heart rate by palpation of the radial artery. This heart rate was recorded in the right top hand corner of their muscle soreness chart. The Ss rested for at least five minutes in the lab following the completion of the test in order to insure that they did not have any physical problems. During this time, they were reminded about filling out their muscle soreness chart at 24 hour intervals.

Group 3

Group 3 met thirty-five minutes before the step test in order to perform the stretching sequence and warm-up. After each subject

signed a consent form, the Ss were told that they would be stretching for eighteen minutes using the previous mentioned exercise, followed by ten minutes of warm-up. Each exercise was demonstrated and all Ss were asked if they had any questions. They were then told that they would have one minute following the stretching before they would start the warm-up and one minute between the warm-up and the starting of the step test. The warm-up and step test were then described in full detail. The stretching exercises were then performed in the previous stated sequence, as for Group 1 (see Group 1). After completing the stretching, the Ss then rested for one minute before starting the warm-up. During this time, they were reminded of the rate and procedure for the warm-up as well as the procedure for taking heart rate from the radial artery pulse. The Ss then underwent the ten minutes of warm-up. They took their heart rate for ten seconds following the warm-up and recorded the number on the upper right hand corner of their muscle soreness chart. At this time, the Ss were asked to keep up with the amount of time they were stepping as well as keep track of their three minutes of rest between each bout of stepping. After the one minute rest, the Ss then began the step test. Ss performed four bouts of stepping with three minute rest intervals between each bout. Ss continued each bout until they preceived their right leg to be fatigued and could no longer keep up with the step pace or reached the cut off time of one hour and twenty minutes without having stopped. Once a subject stopped for a three minute rest, he/she was asked how long he/she had be stepping. This time was recorded each time a subject stopped. Upon completion of the test,

all time of stepping was added up to reveal how long each subject had stepped. The Ss rested for at least five minutes in the lab after the completion of the test in order to insure that they did not have any physcial problems. During this time, they were reminded about filling out the muscle soreness chart at 24 hour intervals.

Group 4

This group was one of the control groups. Group 4 met five minutes before they underwent the step test in order to receive instructions on how the step test was to be performed. After signing a consent form, the Ss underwent the step test, continuing until they preceived their right leg to be fatigued and could no longer keep up with the pace, or achieved the one hour and twenty minute cut off time. The Ss who had to stop, preformed four bouts of this fatiguing exercise with a three minute rest period between each bout. The time was recorded each time a subject stopped and upon completion of the step test, all time of stepping was added up for each Ss to reveal how long each had stepped. The Ss rested for at least five minutes in the lab after completing the step test in order to insure that they did not have any physical problems. During this time, each subject was reminded about filling out the muscle soreness chart at 24 hour intervals.

Soreness Evaluation

The soreness rating scale describe by Tagal (176) was modified for this study (see Appendix A). Soreness evaluation took place before the step test and at 24, 48, 72, 96, and 120 hours after the step test. Ss were asked to rate their muscle soreness on a scale between zero to six. The data were determined to be parametric in nature because the subjects were asked to rate their muscle soreness on a ratio scale of zero to six with the intergers of the scale being clearly defined in order to insure validity. Other scales such as the Borg Category-Ratio scale utilize the same principles as this one (132).

Statistical Evaluation

Comparisons of soreness ratings between groups were made by a 4x2x2 analysis of variance (ANOVA) and Duncan's New Multiple Range Post Hoc Test. The design used was a 4x2x2 model with the three independant variables being group (1,2,3, or 4), gender (male or female) and leg (right or left). Peak muscle soreness was determined to be at twenty-four hours by comparison of the mean muscle soreness values of each group. Group 2 was the only group that did not peak at the twenty-four hour mark. However, since Group 2 was a control group that had not undergone the step test, and the pain reported by this group was questionable in nature as being DOMS, it was decided that the twenty-four time mark was the peak of perceived muscle

soreness for the study. Peak muscle soreness was the dependent variable. Groups, gender, and leg (the independent variables) were all class variables. The data were determined to be parametric in nature because the subjects were asked to rate their muscle soreness on a ratio scale, as stated earlier. Significant differents were determined with a 95% confidence level, therefore an alpha risk of .05 was used on all tests.

CHAPTER IV

RESULTS

Fifty out of the sixty-two subjects in this study experienced some delayed-onset muscle soreness. All of the forty-six subjects, except one, who performed the step test, reported experiencing some delayed-onset muscle soreness. Out of the fifty subjects that experienced soreness, fourteen reported that their soreness had not returned to pre-testing values by the fifth day (120 hours). The overall mean muscle soreness was 1.96 with a standard deviation of 2.16. Muscle soreness scores ranged from zero to six. There were 124 (N = 124) observations in the study, but only 62 subjects. The 124 observations were due to perceived soreness scores being obtained in the right and left leg's quadricep muscle group of each subject. These observations were treated as independent measures because each of the two muscle groups (right quadricep, and left quardricep) underwent a different stimulus due to the specifications made by the step test protocol. Data from the hamstrings were not included in the analysis of the data because this muscle group was not stretched and therefore could not add any support for the hypotheses of this study. However, if the reader is interested in these scores, Table 1 reports the peak mean muscle soreness scores for both quadriceps and hamstrings muscle groups.

Table 1. Perceived Muscle Soreness Values.

Group	Test Day	24 hours	48 hours	72 hours	96 hours	120 hours
			Group 1 (Stretched and Stepped)			
(M,Q,L)*	0.111	3.890	3.111	1.778	1.111	0.222
(M,Q,R)	0.111	1.220	1.000	0.556	0.111	0.111
(M,H,L)	0.111	2.890	2.333	1.333	0.778	0.222
(M,H,R)	0.111	1.890	1.444	0.667	0.111	0.111
(F,Q,L)	0.000	3.857	3.571	2.571	1.571	0.714
(F,Q,R)	0.000	2.428	2.143	1.428	2.333	0.143
(F,H,L)	0.000	4.000	3.714	3.000	1.714	0.857
(F,H,R)	0.000	2.285	1.857	1.143	0.571	0.143
			Group 2 (Warmed-up only)			
(M,Q,L)*	0.000	0.000	0.125	0.000	0.000	0.000
(M,Q,R)	0.000	0.000	0.000	0.000	0.125	0.125
(M,H,L)	0.000	0.125	0.125	0.000	0.125	0.125
(M,H,R)	0.000	0.000	0.125	0.000	0.000	0.000
(F,Q,L)	0.125	0.500	0.750	0.000	0.000	0.000
(F,Q,R)	0.125	0.500	0.750	0.000	0.000	0.000
(F,H,L)	0.000	0.500	0.000	0.000	0.000	0.000
(F,H,R)	0.000	0.500	0.000	0.000	0.000	0.000
			Group 3 (Stretched, Warmed-up, and Stepped)			
(M,Q,L)*	0.000	4.250	4.125	3.000	1.125	0.375
(M,Q,R)	0.000	0.125	0.000	0.000	0.000	0.000
(M,H,L)	0.000	1.125	1.125	1.000	0.250	0.000
(M,H,R)	0.000	0.625	0.375	0.000	0.000	0.000
(F,Q,L)	0.000	3.375	3.375	2.750	1.625	0.625
(F,Q,R)	0.000	1.250	0.875	0.750	0.750	0.000
(F,H,L)	0.000	1.875	2.250	1.750	0.875	0.375
(F,H,R)	0.000	0.875	0.875	0.750	0.000	0.000
			Group 4 (Stepped only)			
(M,Q,L)*	0.000	3.875	3.625	2.750	1.500	0.875
(M,Q,R)	0.125	0.875	0.750	0.375	0.125	0.000
(M,H,L)	0.000	1.875	2.125	1.750	1.125	0.500
(M,H,R)	0.000	0.750	0.500	0.250	0.000	0.000
(F,Q,L)	0.000	4.125	4.000	2.250	1.125	0.125
(F,Q,R)	0.125	1.125	0.750	0.250	0.125	0.000
(F,H,L)	0.000	3.125	2.875	2.125	1.250	0.125
(F,H,R)	0.125	2.125	1.625	0.875	0.375	0.000

* M/F = Male/Female, Q/H = Quadricep/Hamstring, L/R = Left/Right

Peak Soreness Determination

The highest peak of the reported muscle soreness was determined to be at the twenty-four time period by comparison of the mean muscle soreness for each group over time (see Figure 1). Group 2's pain (Ss who performed only a warm-up) peaked at 48 hours post exercise, but because this was a control group and there was some question about the soreness being DOMS, peak muscle soreness scores (dependent variable for ANOVA) were compared at twenty-four hours, post exercise.

ANOVA Results

Comparisons of peak soreness perceptions between groups, legs, gender, and all possible interactions of these variables were made by a 4x2x2 analysis of variance (ANOVA) and Duncan's New Multiple Range Post Hoc Test. The ANOVA had 123 degrees of freedom and produced an overall F-value of 5.02. This was significant at the 0.0001 alpha level (see Appendix B). Therefore, the overall null hypothesis (Ho) of the ANOVA (which was that all means of the variables or interactions were of equal value compared to the overall mean) would be rejected and the alternative hypothesis (Hl) of the ANOVA (which was that at least one of these means differed from the overall mean) would be accepted with 99.99% level of confidence. However, in order to determine where the significant differences existed, the main effects and interactions were examined.

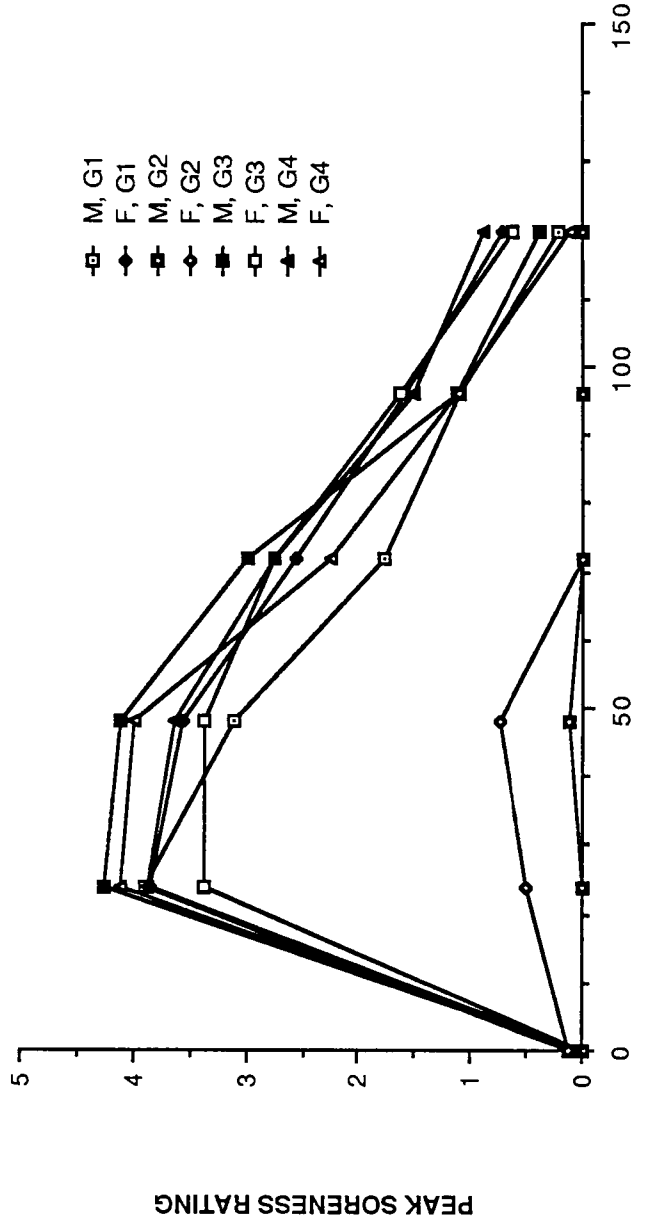


Figure 1. Group's Mean of Perceived Muscle Soreness over Time for the Quadricep Muscle Group. (M,G1) represents males from group 1, (F,G1) represents females from group 1, (M,G2) represents males from group 2, (F,G2) represents females from group 2, (M,G3) represents males from group 3, (F,G3) represents females from group 3, (M,G4) represents males from group 4, and (F,G4) represents females from group 4.

Main Effects

The main independent variables for the ANOVA were group, leg, and gender while the dependent variable was peak muscle soreness. The independent variable, group, showed a significant effect on the dependent variable (peak muscle soreness) at an alpha level of 0.0001 with a F-value of 16.12. Post hoc testing using Duncan's New Multiple Range Test showed that all three groups that underwent the stepping test differed significantly ($p < 0.05$) from the control group that only performed a warm-up exercise. Group 1 had the highest mean soreness preception of 2.71, followed by Group 3 with 2.5, and Group 4 with 2.28. Group 2, of course, had the lowest mean score with a soreness preception of 0.25. With respect to the three groups that did stepping exercise, no significant differences were found between the two experimental groups (Groups 1 and 3) and the control group (Group 4).

The differnce in maximal perceived muscle soreness between legs was significant. The independent variable, leg, had a significant effect on the dependent variable (peak muscle soreness) with a F-value of 43.0. This F-value was significant at an alpha level of 0.0001. Post hoc testing revealed that the average mean score for maximal perceived muscle soreness was 2.85 for the eccentrically worked left leg and 0.97 for the concentrically worked right leg (see Table 2).

TABLE 2. Differences in Peak Muscle Soreness in Eccentrically and Concentrically Worked Quadricep Muscle Group.

Variable	N	Mean	Standard Deviation
		Left leg	
Peak Soreness	62	2.855*	2.253
		Right leg	
Peak Soreness	62	0.968	1.568

* Difference is significant ($p < 0.0001$)

Sixteen of the forty-six subjects who underwent the stepping test reported soreness in their right calf. This soreness ranged from very mild to severe. Almost all subjects who performed the step test complained of a weakness in the eccentrically worked leg immediately following the test. Soreness was concentrated in the left quadricep muscle group (see Figure 2) as had been expected (14, 128). However, pain was reported in the right quadricep muscle group by 24 subjects.

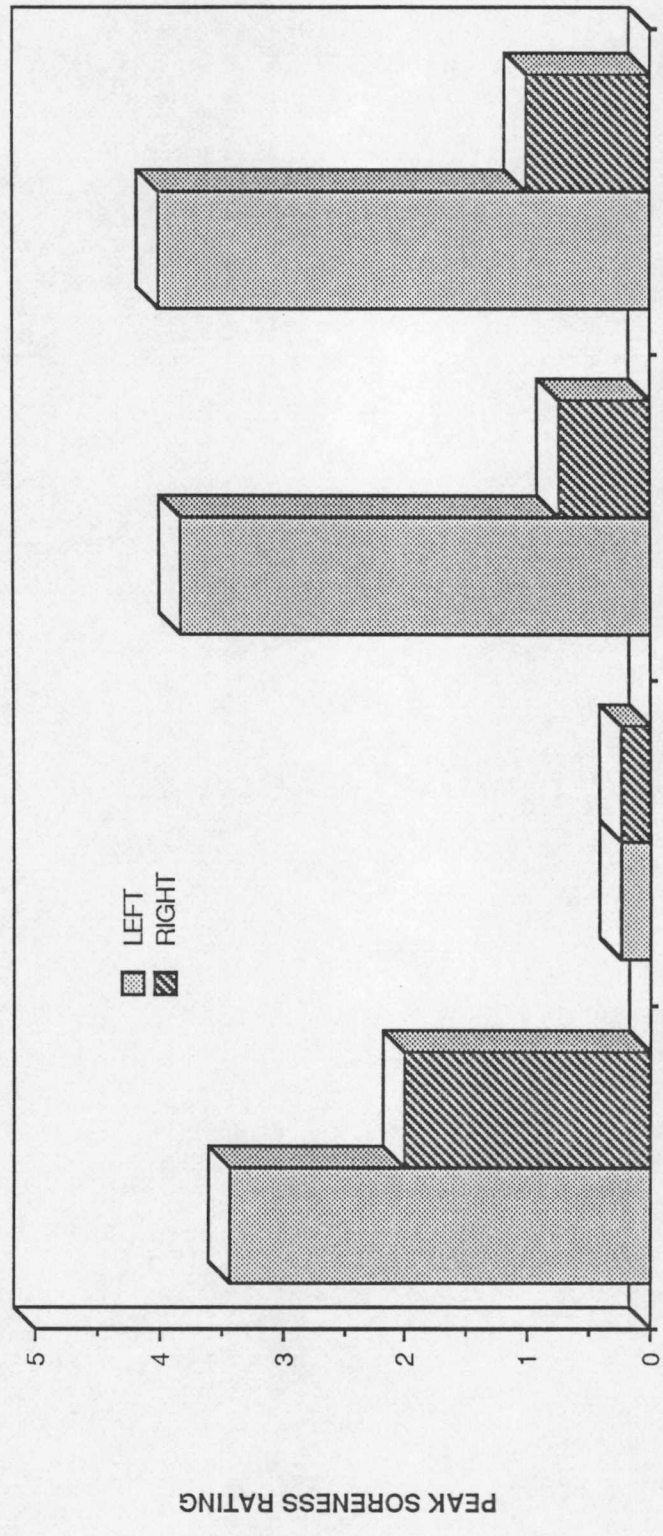


Figure 2. Mean Muscle Soreness for the 24 Hour Time Period of the Left Eccentrically Worked Leg and Right Concentrically Worked Leg.

The independent variable, gender, did not show a significant effect on the dependent variable (peak muscle soreness) with a F-value of 2.14 and a p-value of 0.1468. The mean maximal perceived soreness was 1.71 for males and 2.11 for females. Table 3 presents the mean perceived peak muscle soreness according to males and females overall and shows that males and females did not differ in their perception. However, there was trend for the females to rate their muscle soreness higher than males as shown by Figure 3.

Therefore the overall main effects were as follows; the main independent variables, group and leg, showed a significant effect on the dependent variable (peak muscle soreness), while the variable gender did not.

Table 3. Differences of Perceived Muscle Soreness between Males and Females.

Variable	N	Mean	Standard Deviation
		Males	
Peak Soreness	62	1.710*	2.026
		Females	
Peak Soreness	62	2.113	2.269

* Difference is not significant ($p > 0.05$)

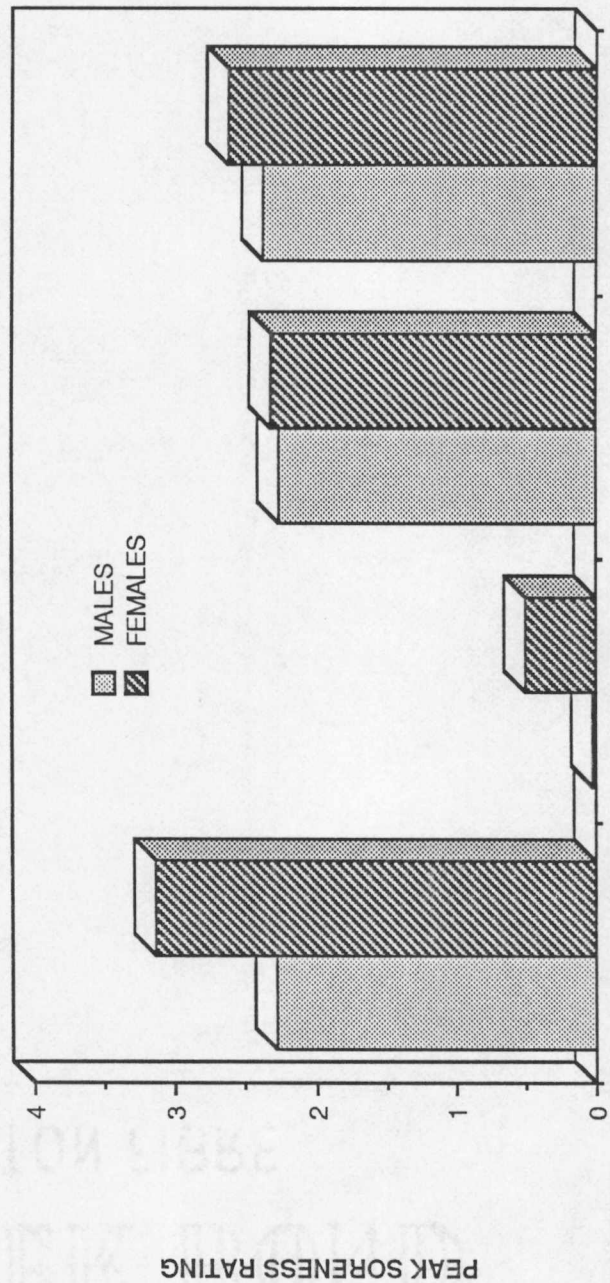


Figure 3. Perceived Mean Muscle Soreness According to Gender.

Interactions

There were no significant interactions between the independent variables of group times gender ($F = 2.63$), gender times leg ($F = 1.69$), and group times gender times leg ($F = 5.22$). However, the interaction among the independent variables group times leg ($F = 50.92$) was significant at an alpha level of 0.0004. Figure 4 shows that this interaction was due to the low muscle soreness perception scores in Group 2. As was expected from this control group, both left and right legs had the same mean maximal muscle soreness perception because of the short and light intensity of the warming-up stepping exercise. Therefore the interaction among these two independent variables was expected. The ANOVA was repeated leaving out Group 2 to make sure that this was the case. This interaction was not significant once Group 2 had been removed.

Results from the Subjects who Performed the Step Test

Out of the 46 subjects who underwent the step test, 24 experienced soreness of some intensity in both legs and 21 experienced soreness in only the left leg. One subject did not get sore following the step test. The total time that subjects stepped ranged from 17 to 80 minutes.

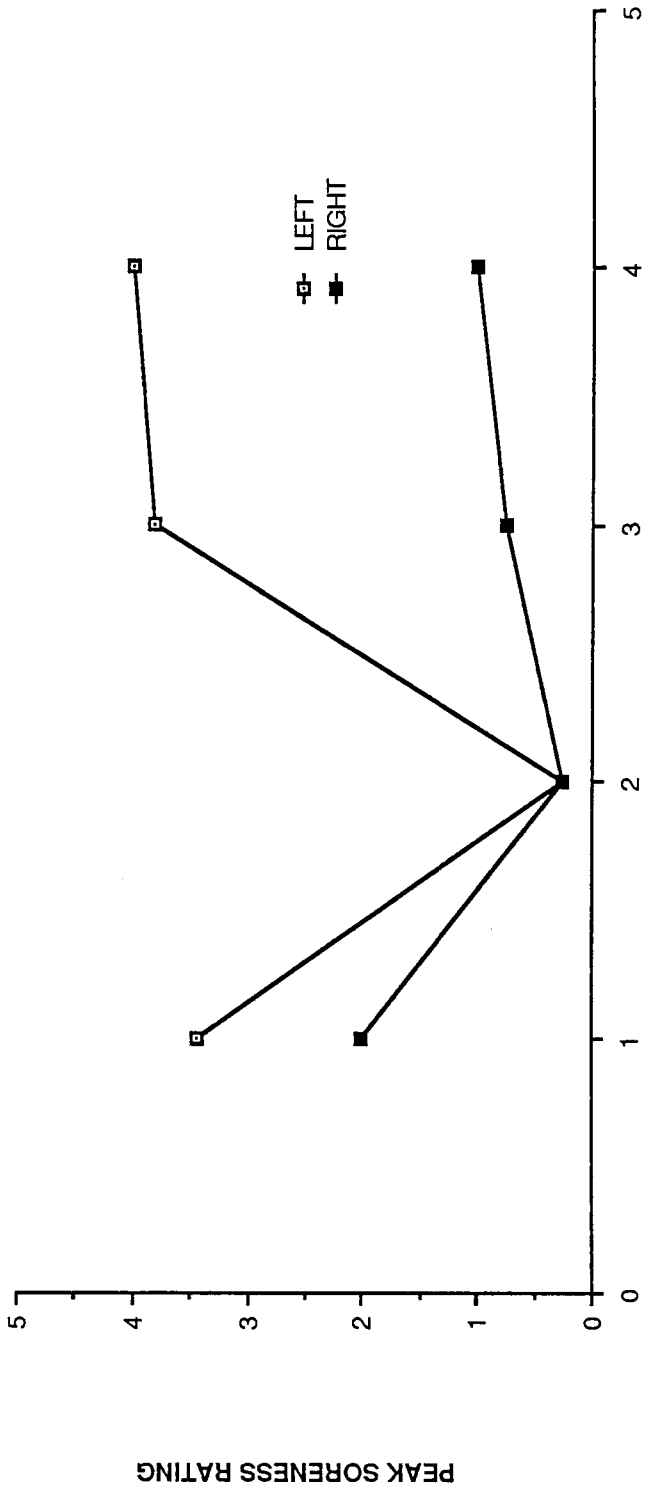


Figure 4. Interaction between Group and Leg.

Results from Subjects who Performed the Warm-up

Of the 16 subjects in the control group that performed only a warm-up, 3 reported experiencing some mild soreness and one, moderate soreness. This soreness displayed no specific pattern in respect to a particular muscle group. Likewise, the reported soreness displayed no specific relationship to time, nor displayed a pattern of soreness that would be expected with DOMS. Because of the reported pattern of pain, it is believed that the warm-up did not result in DOMS. Those subjects who performed the warm-up had a heart rate between 68 and 118 beats per minute at the end of ten minutes of warm-up.

CHAPTER V

DISCUSSION

The lay literature as well as the American Medical Association has stated that static stretching and warm-up exercises will help prevent Delayed-Onset Muscle Soreness. Possible explanations of why these measures would offer any help in the prevention of DOMS was not given and the claim for such must rest upon intuition and not experimental findings.

Differences among Groups

The first hypothesis of this thesis was that static stretching and/or warm-up before heavy exercise has no significant effect on the perception of delayed muscle soreness following the exercise. Because the two experimental groups (1 and 3) in this study did not differ significantly from the control group (4), this hypothesis is supported. In fact, the means of the three groups do not reflect any kind of trend to lend support for statements in the lay literature that static stretching and/or warm-up before exercise tends to prevent DOMS. This is exemplified by the fact that Group 1 (Ss who stretched before stepping) and Group 3 (Ss who stretched and warmed-up before stepping) as well as the control Group 4 (Ss who just stepped) had mean perceived peak muscle soreness of 2.71, 2.5, and 2.28, respectively. Therefore, the recommendation put forth by any exercise

specialist that one should static stretch and/or warm-up prior to fatiguing exercise in order to prevent the occurrence of DOMS must be questioned. These exercises may be benefical in regard to other aspects of physical exercise (see review of literature under static stretching) but not in their ability to prevent DOMS. There are three possible reasons why these exercises offer no protection against DOMS.

One possible reason is that intensity of the exercise was too great for any positive effects of the static stretching and/or warm-up to be seen. It is interesting to note that Newham (128) (in preforming a step test very similar to the one in this study) reported no soreness in the right (or concentrically worked) leg. However, 24 out of the 50 subjects that developed DOMS in this study reported having some soreness in the concentrically exercised leg. This difference could be due to the fact that Newham had his subject step from 15 to 20 minutes. All subjects in this study, except one, stepped longer than 20 minutes. It is known that DOMS is directly related to intensity and duration. Therefore, the intensity and duration of the stepping test in this study might have been too great for the benefits of the stretching and/or warm-up to be shown. But, the exercise was deliberately heavy in order to see whether or nor stretching and/or warm-up would prevent DOMS. This "problem" will be discussed in more detail under the limitations of the study and recommendations for futher research.

A second possible reason for why there was no difference is that the intensity and/or duration of the static strecthing and warm-up was inadequate. However, the duration of these exercises for this study

was based upon what was expected to be the average recommendation to the public at large when exercise specialists give their exercise prescription. Likewise, the intensity of the warm-up exercises was again based upon what is recommended to the pubic at large. Therefore, if these are the recommendations given to the public at large, then the issue of whether or not intensities or durations were adequate is not valid.

The last possible explanation of why there was no difference in groups could be that static stretching and/or warm-up have no effects on the underlying mechanisms responsible for DOMS. This could be said to be true for this study, only when one is speaking of a very intense exercise.

Differences between Legs

The significant difference between the right and left legs of the subjects was expected, as stated in the second hypothesis of this study. This hypothesis was based on the findings of Asmussen (14) and Newham et al. (128) which found that the eccentrically work leg in the step test always had the highest degree of soreness. Therefore the findings of this study agree with the literature which suggests that eccentric contractions are primarily responsible for DOMS (14, 19, 128, 155, 156, 176).

No Differences between Genders

The third hypothesis of this thesis, which stated that no significant differences in muscle soreness perception were expected between males and females, was supported. This means that men and women have similar reactions and perceptions to exercise and that muscle soreness is not a sex-linked phenomenon.

Interactions

The interaction of the independent variables of group and leg was due to the fact that the subjects in Group 2 did not have a higher perceived pain in the left leg, like the other three groups did. This was expected since Group 2 performed only a stepping warm-up for 10 minutes. The intensity and duration of this exercise was not thought to be high enough to produce DOMS. As stated in chapter 4, when the data from Group 2 were removed from the ANOVA, the significance of the interaction disappeared.

Questions that are Raised by the Findings of this Study

There is really no good answer to the question of why one subject who performed the step test did not get sore. One possible explanation is that the individal had previously experienced soreness in the quadricep muscle group within the previous 6 weeks, due to some type of exercise that is similar to eccentric stepping exercise.

However, all subjects were asked at the beginning of the study if they had done any training on stairs (or any exercise similar in nature) in order to prevent any possible interference or profolactic effect from previous bouts of DOMS (32).

Probably the most interesting question which is raised by this study is, can concentric work cause muscle soreness? Some investigators have reported that circuit weight training requiring isokinetic contractions does not cause DOMS (31, 64, 65). Likewise, almost all of the literature suggests that eccentric contractions are responsible for delayed muscle soreness (59, 128, 155, 156, 176). Talag reported in 1973 that isometric work did result in soreness. Whether or not the soreness experienced in the right leg of the subjects of this study was due to concentric contractions can not be said for sure. For the soreness could very well be due to the isometric contractions needed to stabilize the leg during such exercise, as reported by Byrnes et al. (32). Yet, over half of the subjects who got sore in this study experienced soreness in the concentrically worked leg. And one subject reported experiencing a maximal amount of soreness (level 6 in the right concentrically worked leg), at twenty-four hour post exercise. Twelve subjects reported perceived soreness, between light pain when walking up and down stairs (level 3) and moderate pain, stiffness or weakness when walking (level 5). All but two of these subjects experienced higher levels of pain in their right leg. It seems very unlikely that this high level of soreness would be due to isometric contractions needed for stabilization of the leg during work. Related to this,

Byrnes et al. (32) reported findings from unpublished work (by Clarkson, Byrnes, McCormick, Turcotte, & While, 1985) of a study designed to minimize the need for isometric stabilization. The maximal soreness rating for the concentrically worked group was 1.6 based upon a ten point scale verus a 4.8 for the eccentric group. Byrnes did not explain this minute level of soreness from the concentrically worked muscle except in light of isometric stabilization. He did not report the intensity of the exercise which might be very important in determining whether concentric contractions can cause DOMS.

The reason why it is important to find out if concentric work causes DOMS or not is that the torn tissue hypothesis is based on the assumption that the high tension developed during eccentric work is causing the DOMS. If concentric work can cause DOMS, the torn tissue hypothesis losses some of its power as the leading hypothesis and other possible explanations, such as the role of Ca++ ions, take on a much greater signficance.

The Limitations of the Study

One limitation of the study was that all the Ss were already in some type of training program. There was no way to control for possible benefits gained from pervious exercise that might carry over and prevent DOMS. Since the population was male and female college students between the ages of 18 and 39, any interpretation of the data must be limited to the above population. Another limitation was that

perception of soreness may not be a sensitive enough tool with which to measure the differences between groups in this study. It should be noted that the static streching was only a "one-time" performance and recommendations from this study can only be made to static stretching and to the duration and frequency used in this study. Likewise, any recommendations from this study cannot be made to a lighter intensity of work than a maximal effort. Lastly, any recommendations based on this study cannot include cool-down since cool-down was not included in the study. It is interesting to note at this point that all the hypotheses put forth by this study have found support in the data.

Recommendations for Futher Research

If this study were to be repeated, one recommendation would be to have a population of sedentary individuals of all ages, working at an intensity similiar to that found at the beginning of an exercise program. Likewise, cool-down should be included in further reseach. Futher reseach needs to be carried out on the effects of long term stretching on muscle soreness, to see if this offers any benefits. Different types of stretching and their possible benefits in preventing soreness ought to be investigated as well. The question of why children do not suffer from the effects of DOMS should be investigated in that it might lead to a better understanding of the underlying mechanisms of DOMS. Likewise, the role of Ca++ ions as a possible chemical stimulant for DOMS needs futher research.

And lastly, the question of whether or not concentric contractions cause soreness should be readdressed.

Conclusions

From the data of this study, it can be said that (1) a one time static streching of up to 18 minutes on a college age population does not prevent DOMS resulting from a maximal exercise, (2) Static Stretching combinded with a 10 minute warm-up does not prevent DOMS for the above population and intensity of work, (3) the question as to what extent concentric contractions can cause soreness is still not solved, but eccentric work does cause greater amounts of perceived soreness and (4) there is no evidence from the study that there is a difference in the peak perception of DOMS by males or females following a step test.

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APPENDIXES

APPENDIX A

MUSCLE SORENESS RATING FORM

Ss ID. Num.:
Group Num.:

SORENESS SCALE
FOR ALL SUBJECTS

You might feel some pain or discomfort in the days following the first exercise bout. I would like you to keep track of this for us and the scale outlined below is to be used as a guide. Please fill in the appropriate number for each muscle group. If you feel pain or discomfort elsewhere, please indicate the site.

RATING YOUR PAIN

0-complete absence of soreness
1-light pain felt only when touched
2-moderate pain felt only when touched
3-light pain when walking up or down stairs
4-light pain when walking on a flat surface
5-moderate pain, stiffness or weakness when walking
6-severe pain that limits your ability to move

The following chart outlines the major muscle groups in the legs which we will be concerned with in this study. Please fill in one of the above numbers.

	Left leg		Right leg	
	Upper front of leg (Quadriceps)	Upper back of leg (Hamstrings)	Upper front of leg (Quadriceps)	Upper back of leg (Hamstrings)
Test Day	_____	_____	_____	_____
Day One	_____	_____	_____	_____
Day Two	_____	_____	_____	_____
Day Three	_____	_____	_____	_____
Day Four	_____	_____	_____	_____
Day Five	_____	_____	_____	_____

APPENDIX B

ANOVA TABLE

Table 4. Summary Table for Analysis of Variance of Mean Scores for Maximal Soreness.

Scource of Variation	Sum of Squares	df	Mean Square	F
Model	176.82	11	16.08	*5.02
Error	256.16	80	3.20	
Total	432.98	91		

*Significant at .0001.

Table 5. Type III Sums of Squares of the ANOVA Table for Maximal Perceived Soreness Scores.

Source	df	Type III SS	F Value	P Value
		(MAIN EFFECTS)		
Group	3	121.84	16.12	0.0001
Gender	1	5.38	2.14	0.1468
Leg	1	108.36	43.00	0.0001
		(INTERACTION EFFECTS)		
Group*Gender	3	2.63	0.35	0.7929
Group*Leg	3	50.92	6.74	0.0004
Gender*Leg	1	1.70	0.67	0.4136
Group*Gender*Leg	3	5.22	0.69	0.5636

APPENDIX C

CONSENT FORM

CONSENT FORM

PURPOSE OF STUDY

The purpose of this study is to gain a better understanding of muscle soreness. Since the causative factors of delayed muscle soreness are not known, this study will contribute to the body of knowledge on the factors that lead to delayed muscle soreness.

DESCRIPTION OF THE STUDY

This study will take one week to complete. On the first day you will be assigned to a group. Once assigned to a group you will be instructed on which treatment you will be receiving. One or more of the treatments may require you to step on a box or bench which is 50 cm (males) or 40 cm (females) in height. If you are assigned to one of these groups, there will be four bouts of exercise, each continuing until you can no longer keep up with the step rate. Between each of these bouts of exercise you will have three minutes of rest. You will be asked to use your right leg to step up on the box and your left leg to step down from the box. You will be given a form with which to rate your muscle soreness during the following days.

POTENTIAL RISKS AND DISCOMFORTS

It is presumed that the stepping test will result in muscular soreness for 12 to 48 hours after the exercise (Asmussen, 1956). However, no injuries are expected in a study of this nature.

I have read the statement above and understand my role in the experiment and the risks involved. In addition, I am aware that:

(1) My name and scores will remain confidential;
(2) I am entitled to have any further inquiries answered regarding the procedures;
(3) I may withdraw my consent and discontinue my participation at any time without penalty or prejudice toward me.

Date:_____ Signature:_____

Date:_____ Witness:_____

Edward T. Howley	Tim Lightfoot	David M. High
336 HPER Building	347 HPER Building	347 HPER Building
974-1294	974-5111	974-5111

APPENDIX D

FIGURE 5 -- Behind the body

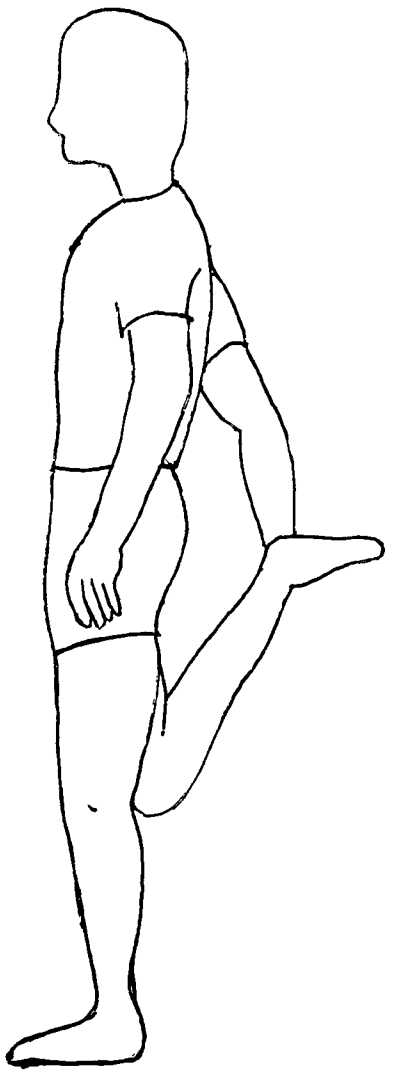


FIGURE 5. Behind the Body -- While you are standing, find some support with one hand. Raise the leg on the same side and catch it behind you with the opposite hand. Pull gently back until tension is felt. Hold in this position for 50 seconds.

APPENDIX E

FIGURE 6 -- Over the Ankle

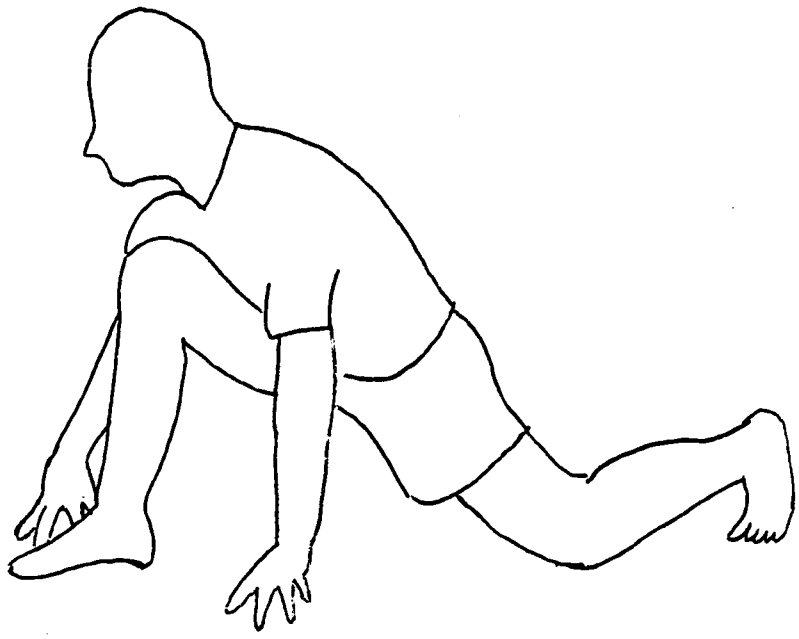


Figure 6. Over the Ankle -- Move forward with one leg until the knee is directly over the ankle. With the knee of the other leg on the ground, move the body slightly forward. Hold this position for 50 seconds.

APPENDIX F

FIGURE 7 -- Hurdler Stretch

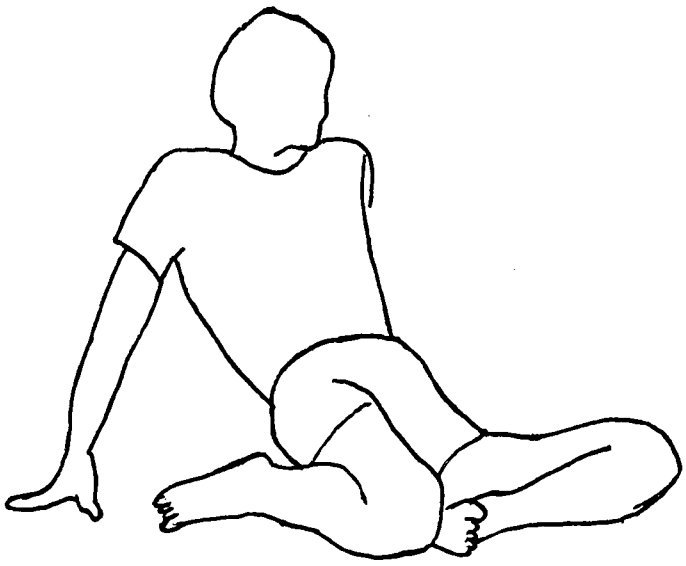


Figure 7. Hurdler Stretch -- Sit with one leg bent in front of you and the other leg tucked under you with the heel of your foot next to your hip. Slowly lean back until tension is felt. Hold this position for 50 seconds.

APPENDIX G

FIGURE 8 -- Lying on the Side

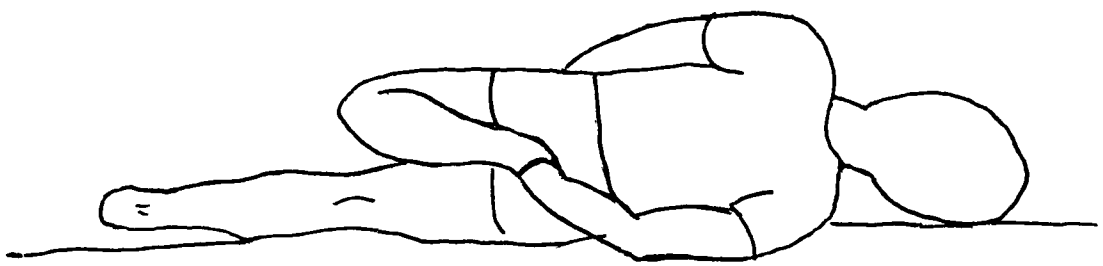


Figure 8. Lying on the Side -- While lying on one side on the floor, move the top leg back behind you and catch it with your top arm. Pull back until tension is experienced. Hold this position for 50 seconds.

APPENDIX H

FIGURE 9 -- Sitting on the Legs

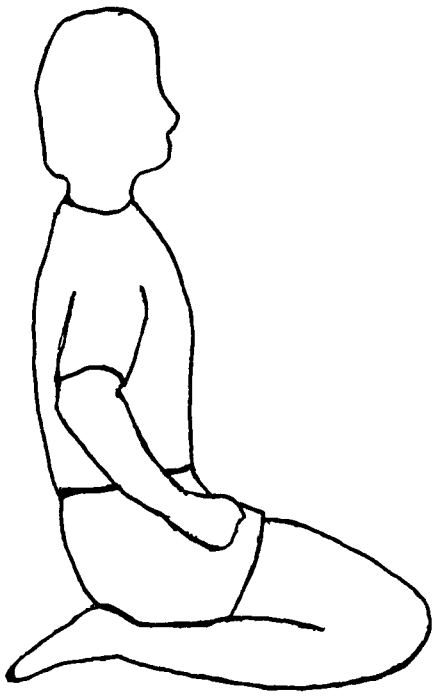


Figure 9. Sitting on the Legs -- With both legs tucked under you, lean back until tension is experienced in the quadricep muscle group. Then hold this position for 50 seconds.

VITA

David Marshall High was born in Ogbomosho, Nigeria. He attended several schools in the United States and Nigeria before graduating from Woodruff High School in June 1980. He then entered Furman University in Greenville, South Carolina and in June of 1984 received a Bachelor of Arts in Physical Education with an emphasis in Adult Fitness.

In the fall of 1984 he accepted a research assistantship at The University of Tennessee, Knoxville and began study toward a Master's degree. In the fall of 1985, he accepted a teaching assistantship at The University of Tennessee, Knoxville and was graduated with a M.S. degree in June 1986.