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The Effects of Massage on Delayed Muscle Soreness

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THE EFFECTS OF MASSAGE ON DELAYED ONSET MUSCLE SORENESS

by

James E. Hilbert

An Abstract

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ABSTRACT

The purpose of this study was to investigate the physiological and psychological effects of massage on delayed onset muscle soreness (DOMS). To that end, 18 subjects were randomly assigned to a massage or control group. Prior to inducing DOMS, the following baseline measures were made: range of motion (ROM), peak hamstring torque, neutrophil count, and mood (POMS). DOMS was induced with 6 sets of 8 maximal eccentric contractions of the right hamstring. Two hours later, subjects received 20 min of massage or sham massage (control). Peak torque, POMS, neutrophil count, and intensity and unpleasantness of soreness were assessed. at 2, 6, 24, and 48 h post-exercise. A two-factor ANOVA (treatment vs. time) with repeated measures on the second factor showed no significant treatment differences for peak hamstring torque, ROM, unpleasantness of soreness, POMS, and neutrophil count ($p > 0.05$). However, the massage group indicated significantly lower intensity of soreness at 48 h post-exercise than the control ($p < 0.05$). In conclusion, massage administered after DOMS-inducing exercise does not appear to reduce inflammation or improve hamstring function. Massage treatments may still be practical because our data revealed it lowered the intensity of soreness. The mechanisms for such psychological benefits require further investigation.

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the School of Health Sciences and

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In Partial Fulfillment of the

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by

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CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that the Thesis of

James E. Hilbert

submitted in partial fulfillment of the requirements for the

degree of Master of Science in the School of Health Sciences and Human Performance at

Ithaca College has been approved.

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DEDICATION

This thesis is dedicated to my family members – who have guided me, believed in my cartoon-childlike graphs, and provided me with countless chapters about love.

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INTRODUCTION

Delayed onset muscle soreness (DOMS) refers to skeletal muscle pain that follows unaccustomed exercises, primarily those involving eccentric contractions (Armstrong, 1984; Powers & Howley, 1996; Smith, 1991). The intensity of soreness usually increases during the first 24 h following exercise, peaks around 24-48 h, and subsides at 5-7 d post-exercise (Armstrong, 1984; Powers & Howley, 1996). The sore muscles are described as being stiff, tender, and aching especially after palpatation or movement (Armstrong, 1984; Smith, 1991). The sensation of DOMS is common and often constrains the quality of daily living, interferes with exercise adherence, and limits athletic performance. Researchers have proposed many therapies to alleviate DOMS, but little scientific evidence exits for consistent success (Ernst, 1998; Powers & Howley, 1996; Smith, 1991).

Current hypotheses indicate that DOMS may be related to muscle structural damage, ion imbalance, inflammation, and pain mechanisms (Armstrong, 1984; Clarkson & Sayers, 1999; Powers & Howley, 1996). These mechanisms also produce the bimodal changes in strength reported with DOMS. Mechanical disruptions are hypothesized to cause the first decline in strength immediately after exercise because of sarcoplasmic reticulum (SR) injury, alterations in the length-tension relationship within the sarcomeres, and decreases in contractile proteins (Clarkson & Sayers, 1999; Faulkner, Brooks, & Opiteck, 1993; MacIntyre, Reid, Lyster, Szasz, & McKenzie, 1996). After a slight recovery in muscle strength at 8 h post-exercise, there is another decrease at 24 h post-exercise that is said to be linked to the inflammatory response (MacIntyre et al.).

Inflammation is considered an important component of DOMS and efforts to reduce its response may prove therapeutic (Armstrong, 1986; MacIntyre, Reid, & McKenzie, 1995; Pizza et al., 1995). Inflammation includes the movement of fluid, plasma proteins, and leukocytes into injured tissue with neutrophils, the most abundant of the leukocytes, being the first to arrive (Sherwood, 1997). The neutrophils detect chemical signals from the damaged tissue and migrate to the injured sites. The neutrophils then adhere to the endothelial wall (margination) and squeeze through the endothelial cells in a process called diapedesis (Edwards, 1994; Sherwood, 1997). Once inside the muscle cell, the neutrophils engulf the damaged cell fragments, degranulate, and activate the respiratory burst (Edwards, 1994; Sherwood, 1997). Degranulation refers to the emptying of the granules inside the neutrophil, which contain a host of antimicrobial proteins that become activated when released. Also, the granules house several enzymes used during the respiratory burst, a form of nonmitochondrial oxidative metabolism. The respiratory burst produces toxic reactive oxygen species that assist in the degradation of engulfed fragments (Edwards, 1994; Pyne, 1994). If tight control of this immune response is altered, host tissue can be destroyed (Clarkson & Sayers, 1999; Edwards, 1994; Pyne, 1994; Weiss, 1989).

To offset the negative consequences of DOMS, several researchers tested the effectiveness of non-steroidal anti-inflammatory drugs (NSAIDs). The results of these studies, however, are equivocal (Bougie, 1997). Administration of NSAIDs had no effect on DOMS in some studies (Bourgeois, MacDougall, MacDonald, & Tarnopolsky, 1998; Donnelly, Maughan, & Whiting, 1990; Gulick, Kimura, Sitler, Paolone, & Kelly, 1996; Pizza, Cavender, Stockard, Baylies, & Beighle, 1999), but produced positive

results in others (Dudley et al., 1997; Hasson et al., 1993). Several factors may account for these conflicting results including various training levels of subjects, and types, dosages, and timing of medications. Moreover, high dosages of NSAIDs may have long term detrimental effects. Researchers administered high doses of NSAIDs to rabbits and observed negative alterations in muscle structure and function up to 28 d after initial muscle damage (Mishra, Friden, Schmitz, & Lieber, 1995). NSAIDs have also been linked to ulcer formation (Sherwood, 1997). In light of these dangerous side effects and conflicting results for NSAIDs, a safer therapy for DOMS is warranted.

Muscle massage may provide an alternative therapy that is both safe and effective. Despite the ancient origins of massage and its current popularity, there is limited data about the ability of massage to reduce DOMS (Goats, 1994; Rodriques & Whiddon, 1997). In theory, massage may reduce DOMS by altering the inflammatory response. A 30-min athletic massage to the forearm muscles was rendered 2 h post-exercise in order to test this hypothesis (Smith et al., 1994). In the massage group, researchers indicated a trend for lower analog soreness ratings at 24, 72, and 96 h post-exercise and a trend for higher circulating neutrophil counts at 8 and 24 h post-exercise. It was argued that massage reduced neutrophil accumulation in the cell by inhibiting margination, evident by higher circulating neutrophil levels (Smith et al., 1994). It is hypothesized that this reduction of neutrophil adhesion (thus, infiltration) will limit the toxic effects of the neutrophil on host tissue during the first 24 h after exercise and reduce DOMS (MacIntyre et al., 1995; Smith et al., 1994; Tiidus, 1999). A review indicated that the neutrophil-endothelial adhesion is a reversible process with mild insult to the area, which supports the claim that massage can alter margination (MacIntyre et al.).

Some researchers have focused on the effects of massage on muscle function, which may be the most accurate marker for muscle injury (Warren, Lowe, Armstrong, 1999). The conflicting results are difficult to reconcile because the massage treatments were of various lengths, intensities, and administration times (Cafarelli, Sim, Carolan, Lieberman, 1990; Gulick et al., 1996; Rodenburg, Steenbeck, Shiereck, & Bar, 1994; Tiidus & Shoemaker, 1995; Viitasalo et al., 1995).

Therefore, the proposed experiment focuses on whether or not vigorous massage rendered 2 h after novel eccentric exercise of the hamstrings will reduce perceptions of soreness, increase peak eccentric torque values, range of motion (ROM), and circulating neutrophil levels. The potential decline in neutrophil infiltration is proposed to limit the toxic destruction of host tissue and reduce various negative aspects of DOMS.

Massage may also provide psychological benefits to individuals experiencing DOMS. One study found that massage and exercise both enhance mood (Weinberg & Kolodny, 1988). In this experiment, subjects from physical education classes were randomly assigned to either swimming, jogging, tennis or racquetball, rest, and massage groups. Only jogging and massage enhanced mood as they reduced tension, confusion, fatigue, anxiety, depression, and anger while maintaining vigor (Weinberg & Kolodny, 1988). In another study, researchers reported that a massage intervention significantly increased boxers' perceptions of recovery without altering heart rate, blood lactate, glucose, and boxing performance measures (Hemmings, Graydon, & Dyson, 2000). The authors concluded that the benefits of massage might be more psychological than physiological. Thus, a second purpose of this experiment will be to analyze the effects of massage on perceptions of soreness and mood states after exercise-induced muscle injury.

REVIEW OF LITERATURE

Introduction

Establishing treatments for DOMS is difficult because the mechanisms behind it are not clearly understood. Therefore, the initial focus of this chapter will review the current theories regarding the etiology of DOMS: muscular structural damage, ion imbalance, inflammation, and pain mechanisms. Several therapeutic interventions have been based on these mechanisms, but the results are equivocal. The review was narrowed to include pharmacological agents and massage because the list of treatments found in the literature is too extensive and less relevant to the proposed study.

It is believed that therapies administered at time frames associated with the mechanisms of DOMS will reveal successful results. In particular, massage may provide an effective strategy when targeted to interfere with the early stages of inflammation. The final component of this review will focus on the physiological and psychological effects of massage.

Etiology of DOMS

DOMS refers to skeletal muscle pain that follows unaccustomed exercises, primarily those involving eccentric contractions (Armstrong, 1984; Powers & Howley, 1996; Smith, 1991). The soreness increases in intensity during the first 24 h following exercise, peaks around 24-48 h, and subsides at 5-7 d post-exercise (Armstrong, 1984; Powers & Howley, 1996). Sore muscles are described as being stiff, tender, and aching especially after palpatation or movement (Armstrong, 1984; Clarkson & Sayers, 1999; Smith, 1991). The condition is benign and rarely requires medical attention.

In terms of muscle function, intense eccentric exercise results in a prolonged loss in muscle strength (Clarkson & Sayers, 1999). Researchers observed strength declines of 50 to 60% immediately after exercise, with full restoration occurring 10 or more days later (Clarkson & Sayers, 1999). Some authors report two declines in muscle strength after novel eccentric activity, with the latter decline aligning with the onset of muscle soreness (Faulkner et al., 1993; MacIntyre et al., 1996).

Different hypotheses have been formulated to explain the mechanisms of muscle damage after eccentric exercise, but a complete understanding of DOMS remains elusive. In the following pages, however, a proposed sequence of events will be described for DOMS that includes mechanical injury, calcium ion imbalance, inflammation, and the sensation of pain (Armstrong, 1984; Clarkson & Sayers, 1999; Powers & Howley, 1996).

Mechanical Injury

It is well established that eccentric exercise results in greater muscle cell damage and perceptions of soreness than concentric exercises (Armstrong, 1984; Clarkson & Sayers, 1999; Powers & Howley, 1996; Smith, 1991). Researchers have observed alterations in the sarcolemma, T-tubules, cytoskeleton, myofibrils, and Z-line proteins (Clarkson & Sayers, 1999). This increased damage with eccentric effort is a product of greater force generation per muscle fiber, a result of fewer motor units being recruited at any give load compared to concentric work (Armstrong, 1994; Clarkson & Sayers, 1999). Hence, the magnitude of damage increases during eccentric contractions because smaller areas of muscle generate the same amount of force. Studies indicate that this high strain may cause sarcomeres to elongate and become disrupted (Child, Saxton, & Donnelly, 1998; Newman, Jones, Ghosh, & Aurora, 1988).

During eccentric contractions, the elongation of sarcomeres is irregular, as some sarcomeres maintain their length while others are stretched beyond their limit (McHugh, Connolly, Eston, & Gleim, 1999). When there is no longer filament overlap, the sarcomere is said to have "popped". As sarcomeres "pop", a greater dependence is placed on the passive structures of the muscle cell, such as the structural proteins desmin, vimentin, and synemin. These proteins maintain the structural integrity of the cell and can transmit force when sarcomeres are stretched beyond their limit (McHugh et al., 1999). Muscle damage does not occur because of "popping", but by the damage to structural proteins during sarcomere failure. The amount of muscle damage is directly related to muscle length, the longer the muscle length, the greater the muscle cell destruction. Researchers report that DOMS is more dependent on muscle length rather than velocity or tension (Child et al., 1998; Newman et al., 1988; Talbot & Morgan, 1998).

Aside from sarcomere popping and subsequent disruption of structural proteins during eccentric activity, sarcoplasmic reticulum (SR) damage also contributes to DOMS. The injured SR leads to an increase in cytoplasmic Ca++ concentration, which in turn, further influences protein degradation.

Calcium Ion Imbalance

Damage occurs to the SR during eccentric contractions, the extent of which remains controversial (Clarkson & Sayers, 1999). The damage allows Ca++ to leak into the cell and this triggers calcium-sensitive degenerative pathways. One calcium-activated protease is calpain, which is closely associated with the I- and Z-band regions of the sarcomere. When activated, calpain selectively destroys various contractile and

structural elements of the cell and cleaves cytoskeleton proteins (desmin, α actin, and vimentin) and receptor proteins in vitro (Belcastro, Shewchuk, & Raj, 1998; Clarkson & Sayers, 1999).

During exhaustive exercise, an increase in calpain activity has indeed been reported (Belcastro, 1993), and this activity may account for the predominance of ultrastructural damage in the Z-disk region of the sarcomere after eccentric contractions (Belcastro et al., 1998; Clarkson & Sayers, 1999). Calpain, however, is not exclusively responsible for the initial decline in muscular strength immediately after intense exercise. Proposed mechanisms for decreased muscular strength include failure of excitation-coupling due to SR injury, alterations of the length-tension relationship because of sarcomere damage, and decreases in contractile proteins (Clarkson & Sayers, 1999). More research is needed to clarify these hypotheses and the time needed for muscle recovery.

Moreover, these mechanisms do not account for the sensation of DOMS that appears hours after exercise or the appearance of a second decline in strength. Research indicates that inflammation could be a secondary mechanism for cellular injury (Clarkson & Sayers, 1999; MacIntyre et al., 1995; Smith, 1991).

Inflammation

Inflammation refers to the movement of fluid, plasma proteins, and leukocytes into tissue in response to injury (Edwards, 1994; MacIntyre et al., 1995). Leukocytes or white blood cells are defined as the mobile units of the immune system that defend the body against invading microbes, destroy cancer cells, and remove debris from dead or injured cells (Sherwood, 1997). There are five different types of leukocytes (Sherwood,

1997): neutrophils, esoinophils, and basophils are classified as polymorphonuclear (many-shaped nucleus) granulocytes (granule-containing cells), while monocytes and lymphocytes are classified as mononuclear (single nucleus) agranulocytes (cells without granules).

Neutrophils are the most abundant leukocyte, accounting for 60-70% of total count. These cells are guided by certain chemical mediators termed chemotaxins and are the first to arrive at the site of injury. Next, the neutrophils adhere to the inner endothelial lining of capillaries, a process termed margination (Edwards, 1994; MacIntyre et al., 1995). Cell adhesion molecules are special plasma proteins that extend from the outer surface of neutrophils and allow them to slow down and adhere to the endothelial wall. Then, the neutrophils infiltrate the host cell (diapedesis) and move to the damaged sites.

Inside the cell, the neutrophils continue to be guided by chemotaxins. It is suggested that peptide fragments of calpain (previously discussed under ion imbalance) act as an attractant for neutrophils (Belcastro et al., 1998; Clarkson & Sayers, 1999). When chemotaxins bind to the neutrophils, cellular structures are turned on that result in amoeba-like crawling of the neutrophil to the injured debris (Edwards, 1994). Here, the leukocytes are involved in phagocytosis, which is the engulfment and intracellular degradation of cell debris and foreign particles (Sherwood, 1997). After this first wave of neutrophils arrive 8-12 hr after injury, these cells release products that signal further leukocyte attraction to the injured region.

In response to exercise-induced muscle damage, researchers observed increases in circulating neutrophils and accumulation of these cells in skeletal muscles (Clarkson &

Sayers, 1999; Pizza et al., 1995; Sherwood, 1997). Researchers argue that documenting changes in the number of circulating leukocytes is a better indicator of muscle damage than muscle biopsy because the latter samples are usually low in number and restricted to small areas of the muscle (MacIntyre et al., 1995). To counter the limitations of biopsies, circulating white blood cells were radioactively labeled and significant increases were seen after subjects performed eccentric exercise (MacIntyre et al., 1996). This study also demonstrated a bimodal pattern to maximum eccentric torque, which was seen for the first time in humans. The study revealed that the first decline in strength occurred immediately after DOMS inducing exercise, slight recovery occurred at 8 h, and a second decline occurred 20-24 h post exercise. Previously, this pattern was only observed in animals (Faulkner, Brooks, & Opiteck, 1993).

An inflammatory mechanism for damage may occur after initial mechanical stress because all leukocytes subsets are capable of producing oxygen free radicals and cytoxic enzymes (Clarkson & Sayers, 1999; MacIntyre et al., 1995). After neutrophils adhere to the damage tissue, phagocytosis occurs because of the respiratory burst and degranulation of cytoplasmic granules. As will be explained below, if these processes are not tightly controlled, normal host cell structures may be destroyed inadvertently.

Important to the destruction of normal tissues is a process termed the respiratory burst. This refers to a sudden, stimulus-induced increase in the oxidative metabolism of the neutrophil that does not involve mitochondria (Pyne, 1994). The production of reactive oxygen species occurs via the nicotinamide adenine dinucleotide phospate (NADPH) oxidase complex located at the plasma membrane. Briefly summarized (Pyne, 1994; Weiss, 1989), the formation of these oxygen species include the transfer and

shuffling of electrons from NADPH, oxygen, and hydrogen ion to a superoxide anion, to hydrogen peroxide, and finally to hypochlorous acid (HOCL).

The enzyme myeloperoxidase (MPO) is abundant in the granules of neutrophils and is responsible for the final conversion to HOCL in the respiratory burst. The release of enzymes from the granules is termed degranulation. Primary granules contain MPO and another antimicrobial enzyme named elastase (Edwards, 1994). These enzymes serve as markers for the activation of neutrophils in many experiments because they are influential in the destruction of healthy tissue (Camus et al., 1992; Pyne, 1994; Weiss, 1989). Host tissue can be destroyed because elastase is a proteinase that cleaves peptide bonds in the body of amino acid chains (Weiss, 1989). Normally, an antiproteinase shield guards against the deleterious effects of these enzymes. The destruction of normal cell fragments can occur because HOCL is a powerful inhibitor of this antiproteinase shield (Weiss, 1989). Thus, elastase is free to destroy muscular protein.

In summary, neutrophils have the potential to alter normal cellular structures. During activation of the neutrophils, a respiratory burst produces large quantities of reactive oxygen species and proteinases are released from the granules. An antiproteinase protective shield exists in the host cell, but HOCL inhibits this shield and allows for the unchecked destruction of host fragments. These inflammatory events have been proposed to align with the time frame for a second decline in muscular strength observed after DOMS inducing exercise.

In addition to tissue destruction, leukocytes also secrete chemical mediators that induce a broad range of other activities. In particular, some secretions trigger the release of histamine that induces local vasodilation. This vascular change serves to increase

blood flow to the region, which accounts for the redness commonly associated with inflammation (Vander, Sherman, & Luciano, 1994). Also, the vasodilation permits plasma proteins to enter the injured site. Other leukocytic secretions result in kinin production, which may activate pain receptors (Sherwood, 1997).

Pain mechanisms

Pain is defined as the "unpleasant sensory and emotional experience associated with actual or potential tissue damage" (O'Conner & Cook, 1999). Pain is a complex phenomenon that relies on somatosensory perception and emotional components such as fear, anxiety, unpleasantness, and memory. Somatosensory perception involves the two primary sensory fibers that detect noxious stimuli, which are the Type III (A-delta), and Type IV (C-delta) afferent nerves (O'Conner & Cook, 1999). These neurons contain receptors termed nociceptors that have high thresholds for activation and respond to intense mechanical, thermal, and chemical irritants (Vander et al., 1994). Nociceptors are located primarily in the connective tissue among muscle fibers, arterioles, and capillaries – ideal locations for receiving algesic substances from injured or active skeletal muscles (Armstrong, 1984; O'Conner & Cook, 1999).

Bradykinin, potassium, serotonin, and histamine act directly on Type III and IV nociceptors (O'Conner & Cook, 1999; Sherwood, 1997). These substances increase spontaneous nerve activity, lower the threshold for activation, and prolong the firing response to a supra-threshold stimulus (O'Conner & Cook, 1999). Other biochemicals such as hydrogen ions and prostaglandins work indirectly to stimulate the nociceptors. Upon activation, the fibers transmit dull-aching and cramping pain familiar to DOMS. Similarly, elevated pressures due to edema (caused by vasodilation substances) and

elevated temperatures (caused indirectly by inflammation) could activate the nociceptors in muscle and add to sensations of DOMS (Armstrong, 1984). Thus, it appears that the perception of soreness is due to a combination of chemical, mechanical, and thermal factors.

The central nervous pathways associated with pain are extremely complex, and these networks consist of many coordinated systems that may override the sensation of pain. Little research exists regarding the possible effects of exercise and other interventions on stimulating these inhibitory spinal and supraspinal neural circuits involved in pain modulation (O'Conner & Cook, 1999). One proposed mechanisms is the gate theory, which suggests that pain can be alleviated by pressure or cold stimuli because the sensory neurons that respond to such stimuli are less myleniated and shorter than neurons involved with the sensation of pain (O'Conner & Cook, 1999). Thus, the pressure receptors may become stimulated while those for pain are "closed".

It is important to note that implied in the definition of pain are the concepts that it is a sensory and subjective experience (O'Conner & Cook, 1999). These ideas are important when analyzing data from experiments measuring the sensation of pain after application of treatments for DOMS.

Treatments

Even though the complete mechanisms for DOMS are not fully understood, many strategies have been proposed to alleviate it, such as pre- and post-exercise stretching, light exercise, ultrasound, topical analgesics, massage, and pharmacological agents. None of these treatments are completely effective in attenuating DOMS (Bougie, 1997;

Gulick et al., 1996). To assess the effects of various treatments, the first part of this section will address certain methodological flaws that appear in the literature. In particular, the assessment of creatine kinase (CK) as an injury marker and the scales used to measure soreness will be questioned. The second part of this section only addresses pharmacological agents and massage as treatments for DOMS. A longer list of treatments can be found in the literature (Bougie, 1997; Gulick et al., 1996), but it is too extensive and less relevant to the proposed study.

Methodological errors

Blood levels of myofibril proteins are commonly used in the muscle injury literature. CK is a key enzyme in converting ADP to ATP and is often used as a measuring tool for exercise-induced muscle injury. It is a large molecule that does not enter the blood stream directly (Hortobagyi & Denahan, 1989). Previous theories state that the mechanical damage of muscle cells during eccentric contractions allows CK to be released into the bloodstream, and this leakage can serve as an indirect marker for injury. The authors of a recent review, however, argued against such rationale and indicated that changes in plasma levels of CK correlate poorly with decrements in muscle function (Warren et al., 1999). Moreover, extreme variations in CK levels are observed despite similar reductions in contractile function after eccentric contractions (Hortobagyi & Denahan, 1989; Warren et al.). Factors influencing such variables are quite diverse and include: age, gender, ethnicity, time of blood sampling, and intensity and duration of exercise (Hortobagyi & Denahan, 1989).

Because of the extreme variability of CK as an indirect marker for cell damage, it does not seem prudent to state that one treatment has a significant effect on muscle

function based solely on CK levels. Some researchers suggest that future investigators refrain from measuring plasma levels of myofibril proteins (Warren et al.,1999). Instead, they recommend that investigators assess torque and range of motion as indices of muscle injury. They also argued against the use of soreness scales.

The arguments against the use of soreness scales stem from reports that indicate soreness correlates poorly with muscle function (Warren et. al, 1999). However, soreness scales are essential when considering the previously discussed definition of pain, which states that pain is a sensory and an emotional experience. Soreness scales can provide vital information on both factors. Despite this necessity, the common measurements found in the literature, visual and analog scales, contain several methodological flaws.

Visual analog and pain intensity scales treat pain as a one-dimensional sensation, varying only by intensity (Gracely & Kwilosz, 1988). These scales rely on spatial judgment or a specific response to a list of choices. Consequently, the subjects repeatedly use the same category description or part of a line and rely more heavily on past experiences of pain (Gracely & Kwilosz, 1988; MacIntyre et al., 1996). Because of this inclination to repeat categories, the sensitivity of the pain scale measurements needs to be questioned.

To combat these problems, several tools are available to assess pain as a multidimensional concept. One such scale is the Descriptor Differential Scale (DDS). The DDS consists of 12 descriptor items each for measuring the intensity and unpleasantness of soreness, and it enables the collection of multiple responses to minimize the tendency to rate an item based on memory (Gracely & Kwilosz, 1988; Warren et al., 1999). The features of the DDS suggest that it may be less subjective to

the biases associated with simple analog and visual scales. Authors recommended that future research include multidimensional assessments of pain such as the DDS (Gracely & Kwilosz, 1988). In light of these methodological considerations, the focus of this review now turns to techniques designed to attenuate DOMS.

Pharmacological Agents

Attempts have been made to reduce the inflammatory response associated with DOMS. In particular, NSAIDs are often studied as they block the production of prostaglandins. Prostaglandins are local chemical mediators that can be triggered during inflammation and the loss of calcium homeostasis in cells (Sherwood, 1997). Also, prostaglandins are important intermediates for the stimulation of nociceptors that transmit pain signals. It is theorized that NSAIDs may reduce DOMS through the blocking effect they have on prostaglandins.

To date, reports on the effects of NSAIDs on DOMS are equivocal (Bougie, 1997). Conflicting results may be due to the various training levels of individuals and types, dosages, and timing of the medications. In one study, naproxen sodium was administered 4 h before exercise and twice daily for 48 h during recovery (Bourgeois et al., 1998). The ingestion of this NSAID did not alter CK, analog soreness ratings, or inflammatory cell infiltrates of the quadriceps muscle. Several factors may account for these findings. For instance, the muscle biopsies may have missed the greatest areas of inflammation. Also, the subjects were moderately trained and the repeat bout effect may have provided them with a protective advantage. The repeat bout effect describes how a repeated bout of similar eccentric exercises reduces DOMS because of neural, connective tissue, and excitation-contraction coupling adaptations (McHugh et al.,1999). When

looking at muscle force data, subjects who ingested naproxen sodium had a more rapid return to baseline levels of maximum voluntary contractions (MVC) compared to a placebo group (Bourgeois et al., 1998). However, the exact mechanism by which NSAIDs may have altered MVC is not clear because data taken from electrically stimulated muscles do not show a similar recovery rate (Bourgeois et al., 1998). The authors attributed the quicker restoration time to possible lowering of edema by NSAIDs.

Improvements of muscular function, however, were not observed when researchers administered ibuprofen. Subjects were given two ibuprofen tablets (600 mg) 30 min before a 45 min downhill run and one tablet every 6 h until 72 h after the exercise (Donnelly et al., 1990). No significant difference was observed for analog soreness ratings, quadriceps isometric strength and endurance, and serum protein levels. Pizza et al. (1999) questioned these observations and argued that dosages of ibuprofen at low levels (< 1600 mg/d) result in an algesic effect, while higher dosages produce anti-inflammatory effects (1600-2400 mg/d). Also, they stated that effective blood levels of ibuprofen are achieved only after days of ingesting high dosages of the drug.

To assess these claims, subjects were given 2400 mg of ibuprofen 5 d prior to eccentric arm exercise and during 10 d of recovery (Pizza et al., 1999). However, isometric strength, analog soreness ratings, and arm angles were not significantly altered by the high doses of ibuprofen. Additionally, there were no differences in the number of circulating neutrophils and production of oxygen radical species between placebo and drug treatment. In contrast, researchers reported that 1200 mg of ibuprofen administered 4 h before eccentric bench stepping and during recovery lessened muscle soreness and the loss of isometric strength (Hasson et al., 1993). Along with various dosages of

NSAIDs, different modes and intensities of the exercises may explain these conflicting results (Pizza et al.). The exact mechanisms of prostaglandin synthesis and its relationship with neutrophil activation are still unknown, which further confounds the research.

One problem with high dosages of NSAIDs is the potential for long term detrimental effects. These effects include myofibril protein loss and force reductions 28 d after initial muscle damage in rats (Mishra et al., 1995). NSAIDs have also been linked to ulcer formation (Sherwood, 1997; Vander, Sherman, & Luciano, 1994). Further research involving various dosages and timing of NSAIDs is needed to give a more adequate representation of their effectiveness. In light of the potentially dangerous side effects and the apparent lack of evidence suggesting a role of prostaglandins in DOMS, alternative methods are needed to ease DOMS symptoms. Muscle massage may be a safer therapy, but the literature is unclear about the benefits of massage.

Massage

Massage refers to the "mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being" (Cafarelli & Flint, 1992). Massage has been a therapeutic tool used since early civilizations and was included in ancient Chinese, Indian, and Greek texts (Goats, Part 1, 1994). A recent survey of elite athletes and coaches indicated that they have a strong preference for massage therapy in the design of future Olympic training centers (Rodriques & Whiddon, 1997). Despite the ancient origins of massage and continued requests for therapy by elite athletes and lay persons, the scientific data regarding massage and its effects on muscle damage are scarce.

Muscle or athletic massage represents the classical techniques referred to as Swedish massage, which is favored by most physiotherapists (Callaghan, 1993). The five basic techniques that make up Swedish massage are effleurage, petrissage, friction, tapotement, and vibration. *Effleurage* is cited as a light stroking motion usually in the direction of venous or lymph flow that serves as an introductory procedure to the other techniques (Callaghan, 1993). *Petrissage* refers to kneading of deep tissue by grasping, lifting, and proximal movement of the muscles. Variations can exist regarding the extent of pressure and the amount of muscle tissue grasped. *Friction* is penetrating pressure applied by the fingertips and performed in a slow circular movement. *Tapotement* refers to a series of blows from the base of the hand and caution should be taken to avoid extremely vigorous application of this technique (Callaghan, 1993). Finally, *vibration* occurs when the tissue is shaken and elevated by the therapist.

These techniques are often administered in an attempt to reduce the symptoms of exercise-induced muscle damage and promote healing. Proposed effects of massage on circulation (blood flow and edema), inflammation, muscle recovery, and psychological states have been studied and will be reviewed.

Circulation

A common belief is that massage increases blood flow, but recent data contradict this notion. A ten minute massage (effleurage only) applied to the quadriceps muscle of subjects who completed an intense bout of eccentric exercise did not significantly elevate arterial and venous blood velocity (Tiidus & Shoemaker, 1995). Leg blood flow was measured via pulsed Doppler ultrasound velocimetry, which is more sensitive than the techniques used to measure blood flow in previous studies, such as radioactive clearance

rates. These data correspond with other studies that show little or no effect of massage on blood flow (Tiidus, 1999). Possible explanations for this null effect include variations in micro-circulation, muscle mass size, and the length and techniques of the massage sessions.

To assess the latter two critiques, effluerage, petrissage, and tapotment were applied to quadriceps and forearm muscles, and blood velocity was measured via Doppler ultrasound (Shoemaker, Tiidus, & Mader, 1997). Neither massage technique nor muscle group size altered blood flow. To verify these effects of massage on blood flow, more experiments are needed with larger sample sizes, more rigorous massage therapies, and analysis of micro-circulation. If massage is able to promote healing of damaged muscles, another mechanism seems likely to be involved.

An interesting point that received little attention by authors assessing blood flow (Tiidus & Shoemaker, 1995) was the reduced muscle soreness at 48 h post exercise. One proposed mechanism for this reduction is that massage alters muscle lymph channels, thereby attenuating the edema associated with DOMS (Goats, Part 2, 1994; Tiidus, 1999). A small amount of empirical evidence exists in regard to lymph flow because of the difficulty in measuring this movement in muscles. Despite this difficulty, effleurage and petrissage did increase lymph flow in anesthetized dogs (Goats, Part 2, 1994; Tiidus, 1999). No studies have directly measured the influence of massage on lymph flow in human models, and the effects of massage on reducing edema are still debatable (Tiidus, 1999). If massage can alter lymph flow and if edema is a factor in DOMS, massage may be a successful therapy.

Inflammation

Another way in which massage may reduce soreness is by altering the proposed inflammatory response of DOMS. A 30 min athletic massage to forearm muscles was rendered 2 h post exercise to test this hypothesis (Smith et al., 1994). During recovery, lower analog soreness ratings and higher circulating neutrophil counts were observed for subjects in the massage group.

The higher circulating neutrophil count may have been due to the ability of massage to shear marginated cells from the endothelial wall (Smith et al., 1994). Such shearing would reduce the accumulation of neutrophils within the muscle cell and limit the potential destruction of healthy tissue (Smith et al.). Thus, the massage may have altered the inflammatory response and reduced DOMS. Reviewers further support this claim by noting that mild insults, such as massage, can alter the neutrophil-endothelial adhesion process (MacIntyre et al., 1995). However, more research is needed to study the effects of massage on inflammation and DOMS. Future research with larger muscle groups and sample sizes are advised to assess neutrophil activation, soreness perceptions, and muscle function during recovery from exercise.

Muscle function

Other experiments have examined massage's ability to alter muscle function following DOMS, but results from these studies are difficult to compare because various massage treatments and muscle groups were used (Bougie, 1997). In addition, these experiments utilized an analog scale for the assessment of soreness, and the validity of such responses has already been noted. Nonetheless, ice massage with a circulating motion was performed on forearm flexor muscles for 20 min, and no significant effects

on soreness and muscle function were observed (Gulick et al., 1996). This treatment is markedly different from classical massage techniques because it lacks any mechanical insult to the muscle tissue. To alter inflammation and edema, the mechanical stress of massage may be vital and not present in general ice rotation.

Mechanical stress was employed in an experiment that used 15 min of warm-up and a stretching routine before exercise of the forearm flexor muscles, followed by 15 min of muscle massage 10 min after exercise (Rodenburg et al., 1994). This combination of treatments increased isotonic force and flexion of the elbow, but failed to alter soreness ratings. However, one can not distinguish between treatment effects because all modalities were applied to the same experimental group. A similar improvement in muscle function was observed for water massage. In this experiment, subjects who underwent 20 min of warm underwater jet massage after training sessions had an increased recovery of jumping ability compared to controls (Viitasalo et al., 1995). It is difficult to determine if the warm water or the mechanical action of the massage affected jumping ability. An interesting follow up experiment would be to assess various temperatures of the water and include subjects who underwent a similar massage without water intervention.

In a study designed to assess the effects of massage on short term recovery from muscle fatigue, a 4 min percussive vibratory massage was applied in between three sets of static contractions of the quadriceps muscle (Cafarelli et al., 1990). No significant differences between control and massage groups for the rate of muscular fatigue were observed. This null effect could be the result of the length and improper timing of the massage session. The same rationale can be used to potentially explain the results of

another experiment that indicated no significant difference between massage and control groups for dynamic and isometric peak torque values of the quadriceps (Tiidus & Shoemaker, 1995). In this experiment, therapists administered 10 min massage treatments (effleurage stokes only) within 1 h of exercise and during 3 days of recovery. To shear marginated inflammatory cells or limit edema, the massage sessions may need to be longer and perhaps, more vigorously applied. Follow-up experiments manipulating time and intensity of massage are recommended.

Finally, a new type of massage, augmented soft tissue mobilization (ASTM), has also been employed to alleviate the symptoms of chronic muscle damage. This type of massage utilizes specifically designed instruments to apply a considerable amount of frictional pressure to connective tissue (Gehlsen, Ganion, & Helfst, 1999). One experiment applied various degrees of ASTM to harvested rat Achilles tendons with tendinitis, and the application of the heaviest pressure promoted a higher extent of healing (Gehlsen et al., 1999). Fibroblast ("fiber" forming cells that play a key role in the tendon healing process) recruitment was higher in the heavy pressure group, and ASTM provides an exciting new technique for treatment of cumulative trauma disorders (Gehlsen et al.). An attractive area of research would be to study the application of ASTM in relation to exercise-induced muscle damage.

Psychological

One reviewer noted that across studies, decreases in anxiety, depression, stress, and catecholamines were observed following massage therapies used in a wide range of clinical conditions such as preterm infancy, chronic pain, pregnancy, migraines, asthma,

and attention deficits (Field, 1998). Surprisingly, only one study has explained its impact on sport and exercise (Cafarelli & Flint, 1992; Field, 1998).

In this study, massage and exercise both enhanced mood (Weinberg & Kolodny, 1988). In this experiment, subjects from physical education classes were randomly assigned to either swimming, jogging, tennis or racquetball, rest, and massage groups. All activities lasted 30 min and the massage consisted of Swedish techniques. Subjects completed the Profile of Mood States (POMS), state anxiety, and an activation checklist before and immediately after the experimental activity. Only jogging and massage produced positive mood enhancement as they reduced tension, confusion, fatigue, anxiety, depression, and anger while maintaining vigor in a physical education setting (Weinberg & Kolodny, 1988).

The original POMS test is a 65-item questionnaire, but many abbreviated versions have been developed to ease time constraints for researchers and subjects. Most of these versions consist of 30-37 items and the reliabilities and validities are high (Grove & Prapavessis, 1992). However, both long and short questionnaires can be highly situation specific. Authors noted that the subscales might reveal different results for various sports, competition levels, and populations (Grove & Prapavessis, 1992). Future research should focus on replicating experimental results and refining the scales used for measuring mood states.

In terms of massage, future research is needed to support or refute the comments by athletes that endorse the efficacy of such therapy. In case massage works via systemic effects, future research should avoid the use of contralateral limbs (Ernst, 1998). For example, many researchers may be tempted to use one limb for exercise and massage

while the other limb is used as a control. This is often done to boost sample size, but assessment of psychological and physiological parameters may be blurred by this practice.

Summary

DOMS refers to the dull and aching pain in skeletal muscles that usually occurs 24-72 h after novel eccentric exercise. A proposed sequence of events for DOMS includes mechanical injury, ion imbalance, inflammation, and the sensation of pain. Especially after eccentric contractions at long muscle lengths, mechanical injury disrupts the sarcolemma, T-tubules, myofibrils, and cytoskeleton proteins of the cells. Damage also occurs to the SR and causes a disturbance in Ca++ homeostasis. This rise in Ca++ concentration activates an enzyme called calpain that further degrades the protein integrity of the cell. Leukocytes are also attracted to the injured area via calpain and other chemoattractants, with neutrophils being the first cells to arrive within 8-12 h after exercise to phagocytosize damaged tissue. If neutrophil function is not tightly controlled, normal host structure may be destroyed inadvertently due to the production of reactive oxygen species. This damage may relate to the observation of the second decline in muscle strength seen within the first 24 h after initial injury. Also, inflammation and structural damage in the cells result in chemical, thermal, and mechanical alterations that trigger Type III and IV pain receptors.

Due to the complexity of DOMS, elucidating treatments is difficult. The use of NSAIDs has produced conflicting results, perhaps because of the training status of subjects, and the types, dosages, and timing of the medications. Moreover, these drugs may have long term detrimental effects such as further muscle injury and ulcer

development. Future research is needed to assess the ability of NSAIDs to alleviate the symptoms of DOMS and to analyze their side effects.

Massage has been proposed as a safe treatment for DOMS and its popularity remains high while scientific knowledge of its effects remains low. Alterations in muscle blood flow are debatable and additional research is needed with larger sample sizes, rigorous massage techniques, and assessment of micro-circulation. Massage rendered 2 h after exercise may disrupt the initial phases of inflammation, and future research should assess neutrophil counts and muscle function. Also, various intensities, psychological components, and time frames for massage sessions should be investigated. Future experimental designs should use multi-dimensional assessments of pain, maximum voluntary contraction torque, range of motion, and vigorous massage techniques similar to classical Swedish designs. Also, researchers should guard against the use of contralateral limbs and measurement of myofibre proteins such as CK. All in all, DOMS remains an intriguing syndrome, and effective treatments would be beneficial to nearly everyone.

METHODS

Subjects

Twenty-four male and female subjects will be recruited to participate in the study. Subjects will be excluded if they are weight training, participating in a competitive sport, pregnant, recovering from a recent knee injury, or taking anti-inflammatory medication. The Ithaca College Review Board for Human Subjects Research approved this project.

Pretests

After signing an informed consent sheet (Appendix A), subjects will report to the lab one to two days prior to the experiment. During this visit, they will complete a psychological profile to assess mood using a POMS test, which measures six identifiable mood states: Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigor-Activity, Fatigue-Inertia, and Confusion-Bewilderment (McNair, Lorr, & Droppleman, 1981).

Next, the subjects will complete the DDS to assess soreness levels. The DDS is superior to analog scales for assessing pain because it enables collection of multiple responses, and minimizes the tendency to rate an item based on memory (Gracely & Kwilosz, 1988). Another problem of analog scales is that they treat pain as a one-dimensional sensation, varying by intensity only (Gracely & Kwilosz, 1988). Pain, however, is defined as both a sensory and emotional experience (O'Conner & Cook, 1999). To combat this one-dimensional limitation of analog scales, the DDS consists of 12 descriptor items each for measuring the intensity and unpleasantness of soreness (Appendix B).

After completing the DDS, hamstring ROM will be measured with a standard uni-level inclinometer during a straight leg raise. The subjects will then be familiarized with

an isokinetic testing device (Cybex Norm, Version 2.01, Henly Healthcare,USA). As a warm-up, they will complete eight submaximal and two maximal eccentric contractions with their right hamstring muscles (0-70° of extension) at a slow velocity (30° per second). Subjects will resist the eccentric movement of the lever arm and observe visual feedback of torque to encourage maximal effort. After two minutes of rest, the subjects will complete five maximal eccentric contractions of the hamstrings and the highest peak torque will be recorded.

After completing the lab measurements, a blood sample will be drawn at the Ithaca College Health Center and used to measure neutrophil levels. A standard protocol for blood withdrawal and counting neutrophils will be performed. After the baseline measures, subjects will be randomly assigned to receive one of two treatments: a non-therapeutic lotion to serve as a control (C), or a vigorous massage (M).

Experimental Design

Each trial will begin between 8-9:30 am with the POMS questionnaire, followed by the previously described warm-up protocol. Subjects will then perform repeated eccentric contractions of the hamstrings to induce muscle damage. The exercise will consist of 6 sets of 10 repetitions of eccentric contractions at a slow speed (30°/s) using the passive mode of an isokinetic dynamometer (MacIntyre et al., 1996). Subjects will be asked to maximally resist the eccentric movement of the lever arm from 70 - 0° of flexion (MacIntyre et al.). This degree of flexion was chosen because longer muscle lengths increase the magnitude of damage (Child, Saxton, & Donnelly, 1998; Newman, Jones, Ghosh, & Aurora, 1988; Talbot & Morgan, 1998). One minute of rest will be given between sets and strength and ROM will be assessed immediately after the exercise.

At 2 h post-exercise, subjects will receive 20 min of control or massage treatment. The massage will consist of effleurage (stroking), petrissage (kneading), tapotement (percussion), and vibrational strokes (Cafarelli & Flint, 1992). These techniques represent classical Swedish massage, which is preferred by most physiotherapists (Callaghan, 1993). The time spent on each stroke will be standardized by an audiotape (Smith et al., 1994). After the massage session, the subjects will complete a POMS test. The subjects will return at 6 h and 24 h post-exercise and their mood state, soreness, peak torque, ROM, and neutrophil levels will again be measured. Further data collection will be performed 48 h post-exercise without neutrophil assessment.

Statistical analyses

A two-factor ANOVA (treatment x time) with repeated measures on the second factor will be used for each dependent variable – peak torque, ROM, DDS intensity and unpleasantness of soreness, neutrophil levels, and POMS score. Tukey and Bonferroni post-hoc tests will be used if a significant main effect or interaction are observed, respectively. Statistical significance will be set at $p < 0.05$ for all tests.

Budget

A senior physical therapy student at Ithaca College will perform the massage therapy under the supervision of the Clinical Director of the Physical Therapy program. Internal requests were submitted to obtain funding for the blood withdrawal and analysis, which will be performed at the Ithaca College Health Center.

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Research Manuscript

THE EFFECTS OF MASSAGE ON DELAYED ONSET MUSCLE SORENESS

Publication Guidelines: British Journal of Sports Medicine (Appendix C)

The effects of massage on delayed onset muscle soreness.

Abstract

Objectives – Despite the ancient origins of massage and its current popularity, there is limited data about the ability of massage to reduce delayed onset muscle soreness (DOMS). Thus, the purpose of this study was to investigate the physiological and psychological effects of massage on DOMS.

Methods – Eighteen subjects volunteered for this study and were randomly assigned to either a massage or control group. DOMS was induced with 6 sets of 8 maximal eccentric contractions of the right hamstring. Two hours later, subjects received 20 min of massage or sham massage (control). Peak torque, mood (POMS), neutrophil count, and intensity and unpleasantness of soreness were assessed at 2, 6, 24, and 48 h post-exercise.

Results – A two-factor ANOVA (treatment vs. time) with repeated measures on the second factor showed no significant treatment differences for peak torque, range of motion, neutrophils, unpleasantness of soreness, and mood score ($p > 0.05$). A two-factor ANCOVA (treatment vs. time) for neutrophils also indicated no significant treatment differences ($p < 0.05$). However, a significant interaction was found for intensity of soreness ($p < 0.05$). Post-hoc testing revealed that the massage group reported significantly lower intensity of soreness at 48 h post-exercise compared to the control group.

Conclusions – Massage administered after exercise-induced muscle injury does not improve hamstring function but may reduce the intensity of soreness. The mechanisms for such psychological benefits require further investigation.

Delayed onset muscle soreness (DOMS) refers to skeletal muscle pain that follows novel eccentric exercises (1,2,3,4). The intensity of soreness increases during the first 24 h, peaks at 24-48 h, and subsides around 5-7 d post-exercise (1,3). The sore muscles are described as being stiff, tender, and aching especially after palpatation or movement (1,3,5). The condition is common, benign, and rarely requires medical attention. Currently, researchers hypothesize that DOMS is related to muscle structural damage that is followed by ion imbalance, inflammation, and pain (1,2,3,5). Muscle damage includes disrupted sarcolemma, T-tubules, myofibrils, cytoskeleton proteins, and sarcoplasmic reticulum (SR) (1,5,6,7). Damage to the SR is particularly problematic because it causes an ion imbalance that activates calpain, an enzyme that further degrades muscle proteins (5,8,9). Within 8 h of the initial injury, neutrophils are attracted to the damaged area by various chemoattractants and adhere to the endothelium wall of nearby blood vessels (2,4,5). After adhesion or margination, the neutrophils infiltrate the muscle cell and phagocytosize the damaged tissue. If neutrophil function is not tightly controlled, normal host tissue is destroyed inadvertently (5,13,14). Ultimately, these mechanical and inflammatory responses trigger Type III and IV pain receptors (15). These negative responses are also associated with bimodal changes in strength seen with DOMS. Researchers report a decline in strength immediately after initial insult, which is linked to the mechanical disruptions in the muscle, followed by a slight recovery at 4 h post-exercise, and ultimately another decrease in strength at 24 h post-exercise that is linked to the inflammatory response (5,10,11,12).

Some strategies proposed to alleviate DOMS include pre- and post-exercise stretching, light exercise, ultrasound, topical analgesics, and pharmacological agents.

None of these treatments, however, are completely effective in attenuating DOMS (16,17). Another possible intervention to lessen DOMS is massage, which may reduce the inflammatory response by decreasing neutrophil margination (18,19). Decreased margination would attenuate infiltration, thereby lowering the toxic effect of neutrophils and potentially reducing DOMS (19,20). Accordingly, the secondary decrease in muscle strength that follows the inflammatory response may also be attenuated, but this has not been documented. Ultimately, measuring alterations in muscle function, such as maximal voluntary contraction torque and range of motion (ROM), would provide simple and accurate markers for muscle injury (21). Some studies have examined the effects of massage on muscle function, but the findings are equivocal and difficult to reconcile because the massage treatments were of various lengths, intensities, and administration times (17,22,23,24,25).

Therefore, this study sought to determine if massage rendered 2 h after exercise will alter peak eccentric torque values, ROM, and circulating neutrophil levels. This experiment also analyzed the effects of massage on perceptions of soreness and mood states after exercise-induced muscle injury. Massage has been shown to enhance mood and perceptions of recovery following exercise (26,27), and quite possibly, massage may reduce DOMS via a psychological rather than a physiological mechanism.

Methods

SUBJECTS

Eighteen male and female subjects aged 20.4 (1.0) yr, weighing 72.6 (14.1) kg, volunteered and gave their informed consent (mean (SD)). None of the subjects were pregnant, participating in a competitive sport, recovering from a recent knee injury,

taking anti-inflammatory medication, or had resistance trained the hamstrings in the previous four months. The Ithaca College Review Board for Human Subjects Research approved this project.

BASELINE TESTS

Subjects reported to the lab one to two days prior to the experimental treatments. In this visit, they completed the Profile of Mood States (POMS) questionnaire to assess six identifiable mood states: Tension-Anxiety, Depression-Dejection, Anger-Hostility, Vigor-Activity, Fatigue-Inertia, and Confusion-Bewilderment (28). The sum for each mood state was recorded, with higher scores representing greater mood disturbance. After the POMS, hamstring ROM was measured with a standard uni-level inclinometer during a straight leg raise. An average over three trials was recorded as ROM (29).

The subjects were then familiarized with the isokinetic testing device (Cybex Norm, Version 2.01, Henly Healthcare,USA). As a warm-up, they completed eight submaximal and two maximal eccentric contractions with their right hamstring muscles (0-70° of extension) at a slow velocity (30° per second). Subjects resisted the eccentric movement of the lever arm and observed visual feedback of torque to encourage maximal effort. After two minutes of rest, the subjects completed five maximal eccentric contractions of the hamstrings and the highest peak torque was recorded.

After five maximal efforts, subjects completed a Descriptor Differential Scale (DDS) to assess muscle soreness levels. The descriptor items of this scale are randomly placed unlike visual-analog, numerical, and verbal scales, which are vulnerable to biases because subjects tend to repeatedly use the same category or part of a line (30). Simple

scales also measure pain as a one-dimensional sensation, varying by intensity only. Pain, however, is defined as both a sensory and emotional experience (15). To combat this one-dimensional limitation of simple pain scales, the DDS contains two sets of 12 descriptor items that measure the intensity and unpleasantness of soreness. The descriptors were randomly arranged for each session and located above a line with 21 points. The DDS intensity and unpleasantness of soreness were scored by assigning values of 0-21 for each point along the respective line of descriptors (12). The values were added together and averaged over the 12 descriptors to obtain the final scores.

After completing the DDS, a 5-ml blood sample was taken by venipuncture from each subject. Differential slides were created, dried, and stained for 7 min with a Hematek 100 (Miles and Company, USA). Then, a trained technician counted 100 cells and recorded the percentage of neutrophils. After these baseline measures were completed, the subjects were randomly assigned to one of two groups: massage intervention (n=9) or control (n=9).

EXPERIMENTAL TREATMENT

Each trial started between 8-9:30 a.m. with the POMS questionnaire, followed by the previously described warm-up protocol. Subjects then performed repeated eccentric isokinetic contractions of their right hamstrings to induce muscle damage. The exercise consisted of six sets of 10 maximal eccentric contractions with 1 min rest between sets. Immediately after the exercise, the subjects performed an additional five maximal contractions and peak torque was recorded. The POMS and torque data from this session were labeled as 0 h post-exercise.

The subjects returned at 2 h post-exercise and peak torque was measured again. Next, the subjects received 20 minutes of massage or placebo lotion treatment. The muscle massage consisted of 5 minutes of effleurage (stroking), 1 minute of tapotement (percussion), 12 minutes of petrissage (kneading), and 2 additional minutes of effleurage (31). These techniques represent classical Swedish massage preferred by most physiotherapists (32). A senior Physical Therapy student performed all treatments, and the time spent on each stroke was standardized by an audiotape. A placebo lotion was applied to the subjects in the control group, and they were instructed to rest for 20 min while listening to the same audiotape. All subjects were told that their treatment might reduce inflammation. After the treatment, the subjects completed a POMS test. At 6 and 24 h post-exercise, the subjects returned and mood state, ROM, peak torque, soreness, and neutrophil levels were measured. Final data collection without blood assessment occurred at 48 h post-exercise.

STATISTICAL ANALYSIS

A two-factor ANOVA (treatment x time) with repeated measures on the second factor was used for each dependent variable: peak torque, ROM, DDS intensity and unpleasantness of soreness, neutrophil levels, and POMS score. Tukey and Bonferroni post-hoc tests were used when a significant time effect or interaction was observed, respectively. Statistical significance was set at $p < 0.05$ for all tests.

Results

The raw data for each variable (peak torque, ROM, DDS intensity and unpleasantness of soreness, neutrophil levels, and POMS score) can be found in Appendix D. ANOVA and ANCOVA summary tables are recorded in Appendix E.

Tables 1-3 (Appendix F) indicate the functional alterations associated with DOMS. The peak torque analysis revealed a significant main effect for time ($F(5,80) = 24.63$, $p < 0.000$), but no main effect for treatment ($F(1,80) = 0.32$, $p > 0.05$), or interaction ($F(5,80) = 0.429$, $p > 0.05$). Additionally, no gender and time interaction was found ($p > 0.05$). Tukey post-hoc analysis showed significant decreases in strength at all post-exercise times compared to baseline (Table 1).

The alterations in hamstring ROM are presented in Table 2 (Appendix F). The analysis again showed a significant main effect for time ($F(3,48) = 20.28$, $p < 0.000$), but no main effect for treatment ($F(1,48) = 1.70$, $p > 0.05$) or interaction ($F(3,48) = 1.49$, $p > 0.05$). Tukey post-hoc analysis revealed significantly lower hamstring ROM at 24 and 48 h post-exercise for both groups.

A two-factor ANCOVA (treatment x time) was used to account for baseline differences in the neutrophil data, and the adjusted means are recorded in Table 3 (Appendix F). The analysis indicated no significant main effect for time ($F(1,15) = 0.101$, $p > 0.05$), main effect for treatment ($F(1,15) = 1.774$, $p > 0.05$), or interaction ($F(1,15) = 0.001$, $p > 0.05$).

Figures 1-3 (Appendix G) represent the psychological changes measured with DOMS. Analysis of the DDS intensity data indicated a significant interaction ($F(3,45) = 3.02$, $p < 0.05$) and main effect for time ($F(3,45) = 45.57$, $p < 0.05$). Inspection of the interaction using post-hoc Boferroni testing revealed significantly higher intensities of soreness at 6, 24, and 48 h post-exercise for both groups. Also, the control subjects reported significantly greater soreness intensity at 48 h post-exercise compared to the massage group (Figure 1).

The DDS unpleasantness data are represented in Figure 2 (Appendix G). The analysis showed a significant main effect for time ($F(3,42) = 34.12$, $p < 0.05$), but no main effect for treatment ($F(1,42) = 1.50$, $p > 0.05$) or interaction ($F(3,42) = 1.97$, $p > 0.05$). Tukey post-hoc testing indicated higher feelings of unpleasantness at 24 and 48 h post-exercise for both groups.

The results for the POMS scores are represented in Figure 3 (Appendix G). The analysis indicated a main effect for time ($F(4,56) = 3.144$, $p < 0.05$), but no main effect for treatment ($F(1,56) = 1.28$, $p > 0.05$) or interaction ($F(4,56) = .36$, $p > 0.05$). Tukey post-hoc testing revealed significantly larger mood disturbances at 24 h compared to baseline for both groups.

Discussion

The purpose of the study was to assess the physiological and psychological effects of massage on DOMS. A major finding in this study is that massage rendered 2 h after strenuous eccentric exercise did not alter neutrophil levels, peak torque, or ROM. The inability of the massage treatment to alter margination contrasts with previous work (19). In that study, the sample size was small and reductions in margination were inferred from changes in circulating neutrophil levels. Our ability to fully quantify the changes in neutrophil activity following massage was limited by our protocol, as we did not measure the total and differential white blood cell counts. Instead, we estimated neutrophil levels by measuring the percentage of neutrophils in a sample of 100 leukocytes. Given the limitations of this methodology and others (19), future experiments may want to assess the distribution and functional activity of neutrophils such as migration, adherence, expression of various plasma membrane receptors, phagocytosis, and the release of

enzymes (14,33). However, the inability of the massage to alter neutrophil activity corresponds to our peak torque and ROM data, which indicated no treatment effects as well. Collectively, these data make it appear that there were no differences in muscle function following massage compared to a sham control.

Previous researchers observed a bimodal effect on eccentric torque in female quadriceps muscles following exercise-induced muscle injury (11,12). This pattern was not observed in our study, as the peak torque values at 6 h post-exercise were not significantly higher compared to 2 or 24 h post-exercise. Several factors may account for these conflicting results. For example, the present study included males and females. Our peak torque analysis, however, revealed the same decline pattern regardless of gender and highlights the need for further testing on this matter. Another factor is the susceptibility of different muscles to injury because the hamstrings are more prone to damage than the quadriceps (34). Additionally, the subjects in the previous experiments had shorter recovery times because measurements were taken at 4 h post-exercise compared to 6 h in our experiment (11,12). It may be necessary for future researchers to do a more complete time course analysis, such as 4,6,8, and 10 h post-exercise, to better describe the potential peak and secondary declines in strength.

The effects of massage may be more psychological than physiological (26,27,31). Our data support these claims by indicating that subjects in the massage group had significantly lower DDS intensity of soreness at 48 h post-exercise. A significant difference between treatments is not indicated by the DDS unpleasantness data, but previous authors suggested that this dimension of the DDS scale is not a prominent experience in DOMS (11). Also, the POMS score analysis indicated no significant

difference between groups. Inspection of individual data, however, revealed that 13 of the 18 subjects showed improved mood states. All subjects were verbally encouraged to relax at the onset of the massage or sham condition and listened to music during its duration, which may have masked treatment differences. To combat this limitation, future researchers should employ a less relaxing control and better address the potential mechanisms by which massage may improve mood states. A few authors have suggested that massage may increase endorphin and serotonin levels, activate pressure instead of pain receptors, and enhance sleep (31,35,36). It remains unclear which mechanisms lowered the intensity of soreness in our study, and this provides an exciting area for future research.

In conclusion, massage administered after DOMS-inducing exercise does not appear to reduce inflammation or improve hamstring function. Additionally, a bimodal effect on eccentric torque was not observed in this experiment. Massage, however, may still be of practical value because it lowered the intensity of soreness. The mechanisms for such psychological benefits require further investigation.

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APPENDIX A: INFORMED CONSENT FORM

Purpose of the study

The study is being conducted to determine the effects of muscle massage and a therapeutic lotion on factors involved with delayed onset muscle soreness.

Benefits

The potential for a safe and effective treatment of muscle soreness is great. Almost all individuals have experienced soreness and it often interferes with daily living activities, adherence to exercise programs, and athletic performance. Large amounts of money are spent on various types of soreness therapies for athletes and the general population, but little scientific evidence exists regarding their effects. The results of this project may give credence to such expenditures and provide evidence for some positive effects of massage and lotion therapies. Alternatively, we may find these therapies are not effective and not worth such expenditures.

What you will be asked to do

Testing will occur during five visits to the Exercise Physiology Lab. Each visit will take approximately 30 min. You will be asked to attend an orientation meeting to fully explain the details of the study, introduce you to the testing procedures, answer your questions, and collect baseline data. Strength, blood, psychological, soreness perceptions and range of motion (ROM) will be measured as follows:

1.) You will be asked to perform maximum contractions of the upper leg (hamstrings muscle group) to assess strength. 2.) Technicians at the Ithaca College Health Center will draw a small sample of blood from an antecubital vein, and the blood will be stored for later analysis. The size of the sample is approximately the same as the amount drawn for routine health screening procedures. A total of 3 blood samples will be drawn during the entire experiment. 3.) Psychological testing will involve filling out simple forms that assess mood fluctuations. 4.) A scale that allows you to indicate your perceptions of soreness from 12 descriptor items will measure the intensity and unpleasantness of muscle soreness. Your muscles will be palpated and you will move the leg through a range of motion prior to assessing soreness 5.) Range of motion (ROM) will be measured during a straight leg raise and flexion of the knee.

Day One

Exercise Session: You will be asked to perform a series of maximal contractions of the upper leg (hamstrings muscle group located in the back of the thigh). To serve as warm-up, you will be seated on a weight-training machine and asked to extend and flex your knee against a submaximal resistance. During the exercise protocol, you will be asked to resist the machine during knee flexion. The computer screen will provide visual feedback to encourage maximal effort. You will be asked to perform 6 sets of 10 repetitions, with 1 min of rest between each set.

Initial here_____

Two hours post-exercise: You will be randomly assigned to a massage or lotion therapy group: one group will receive 20 min of muscle massage and the other will undergo 20 min of rest with an applied therapeutic lotion. A Senior Physical Therapy student from Ithaca College will perform the massage and lotion treatments. After the 20 min, you will be given the psychological, and soreness questionnaires.

Six hours post-exercise: You will be asked to return six hours post-exercise to be given the strength, psychological, ROM, and soreness measurement tests. Blood will be drawn immediately after these tests at the Health Center.

Day Two

Twenty-four hours post-exercise: You will be asked to return 24 hours post-exercise to be given the strength, psychological, ROM, and soreness measurement tests. Blood will be drawn immediately after these tests at the Health Center.

Day Three and Four

Forty-eight and seventy-two hours post-exercise: You will be asked to return 2 and 3 days after the exercise to undergo strength, psychological, ROM, and soreness measurements.

Risk associated with participation

Exercise always involves some risk of injury to the participants. In the proposed study, the exercise session will only be significant enough to induce muscular soreness. The perceptions of soreness have been experienced nearly by everyone and many have experienced the sensation numerous times. The soreness normally increases in intensity in the first 24 h after exercise, peaks around 24-48 h, and finally subsides around 4-6 days post exercise. The sensation of pain has been described as being "stiff" or "tender". You can expect to have reduced flexibility, and the muscles will be particularly sensitive upon movement and palpation.

Delayed onset muscle soreness is accepted as a temporary discomfort and usually does not require medical attention. In extreme cases, the muscle soreness can progress to exertional rhabdomyolyis. This symptom is characterized by elevated swelling, tenderness, and stiffness of muscles, fever, nausea, and vomiting. The condition is rare and is usually enhanced by extreme, unaccustomed exercise, especially in heat.

There is always a small risk of infection when having blood drawn. Maintaining sterile conditions and being at the Health Center will minimize the risks greatly.

Also, well-trained technicians will be used in all testing procedures and a complete emergency plan is in place in the Exercise Physiology Lab.

Initial here_____

More information

If you have any further questions, please feel free to call either Jim Hilbert (273-2341), Dr. G. A. Sforzo (274-3359), or Dr. Tom Swensen (274-3114).

Withdrawal from the study

Participation in this study is voluntary and you may withdraw at any time if you so choose.

Confidentially

Information gathered during this study will be maintained in complete confidence. Only Mr. Hilbert, Dr. Sforzo, and Dr. Swensen will have access to this information. All reporting to outside parties will be done in group form. You and your name will never be associated with this information in any future disclosures.

Please sign and date below.

I have read the above consent form and understand its contents. I acknowledge that I am at least 18 years of age or older and agree to participate in this study.

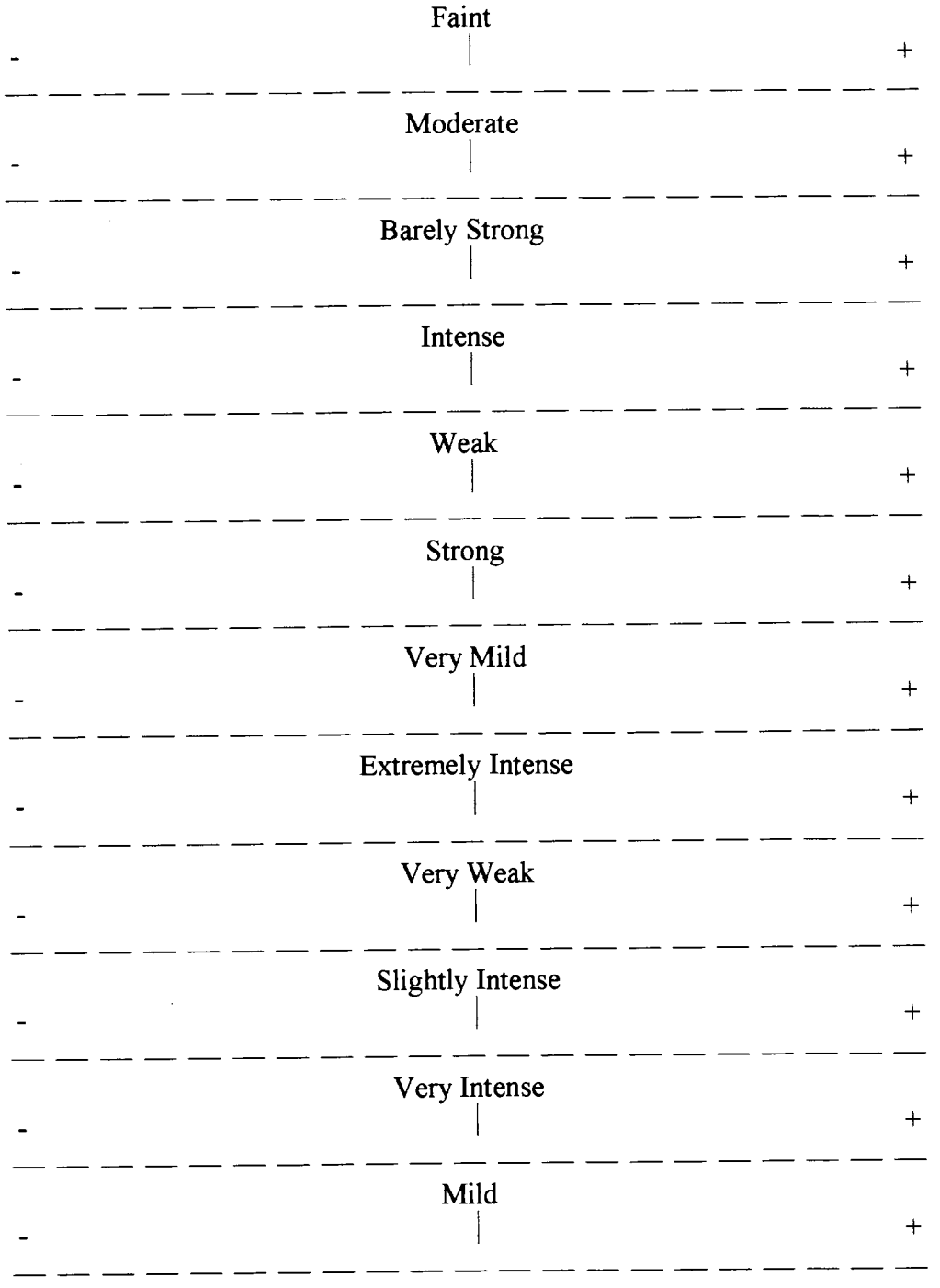
_____

(Signature) (Date)

APPENDIX B: VERSIONS OF THE DDS

Each word represents an amount of sensation.

Rate your sensation in relation to each word with a check mark.



Each word represents an amount of unpleasantness.

Rate your unpleasantness in relation to each word with a check mark.

Slightly unpleasant

\- | +

Slightly annoying

\- | +

Unpleasant

\- | +

Annoying

\- | +

Slightly distressing

\- | +

Very unpleasant

\- | +

Distressing

\- | +

Very annoying

\- | +

Slightly intolerable

\- | +

Very distressing

\- | +

Intolerable

\- | +

Very intolerable

\- | +

APPENDIX C: MANUSCRIPT GUIDELINES

Abbreviated guidelines taken from the British Journal of Sports Medicine.

Scope

The *British Journal of Sports Medicine* covers all aspects of sports medicine and science – the management of sports injuries; all clinical aspects of exercise, health, and sport; exercise physiology and biophysical investigation of sports performance; sports psychology, physiotherapy and rehabilitation in sport; and medical and scientific support of the sports coach.

Style

The format must be as described in this section, although for reviews and letters to the Editor the use of subsections is flexible. Abbreviations should be kept to a minimum and must always be explained. Drugs should be referred to by their approved names.

1. *Title page:* should include the title (not more than 120 characters), a short title for running head, names, and affiliations of authors (only one affiliation per author), and the name, address, telephone number and fax number of the corresponding author.
2. *Abstract:* must not exceed 300 words and should be subdivided into four sections: Objectives; Methods; Results; Conclusions. Sections should not be combined. Statistical values should be given (confidence intervals preferred). The abstract should be followed by five key terms.
3. *Introduction:* outline of the background and rationale of the study.
4. *Methods:* this section should be sufficiently detailed to permit the reader to replicate the study. Published methods should be described in brief, with appropriate citation.
5. *Results:* should be concise and should not repetition of the methods. Data in the text should not be replicated in tables or figures or vice versa. SI units should be used, except for fluid pressure, which should be in mm HG.
6. *Discussion:* a clear distinction should be made between deduction and speculation.
7. *Acknowledgements,* where appropriate.
8. *References:* the Vancouver style must be used (see papers in issues) with citations numbered consecutively in the order in which they appear in the text, tables, and figures. List all authors when there are three or fewer; if there are more than three, list the first three followed by et al. Except in the case of review articles, the total number of references should not exceed 40. Responsibility for completeness and accuracy of references rests entirely with the authors. References will not be checked in detail by the Editor, but papers in which error are detected in the references are unlikely to be accepted. Submitted work or work in preparation cannot be cited in the reference list.
9. *Figures and tables:* Figures will generally be redrawn or re-lettered to conform with journal style, so they should be checked carefully by the authors. Figure legends must always be supplied and must be typed on a separate sheet. Color illustrations may only be used if monochrome cannot show what is desired. Authors are responsible for part of the cost of color figures. Use journal style in tables (see any issue). Do not divide tables with rules. Place brief explanatory legend at the top of the table (not on a separate sheet) and any further necessary clarification at the foot. All tables and figures must be referred to in the text.

APPENDIX D: RAW DATA

Table 1D

Raw data for hamstring peak eccentric torque (Nm).

Subject	Baseline	Oh	2h	6h	24h	48h
m	150.4	128.7	112.4	123.3	111.1	115.2
m	201.9	161.2	142.2	154.4	146.3	134.1
m	113.8	97.5	74.5	90.8	84.0	90.8
m	161.2	117.9	124.6	104.3	119.2	130.1
m	89.4	78.6	61.0	67.7	55.5	71.8
m	227.6	163.9	142.2	149.0	154.4	168.0
m	105.7	86.7	101.6	103.0	96.2	93.5
m	162.6	172.1	162.6	172.1	154.4	170.7
m	89.4	75.9	73.2	74.5	70.4	67.7
c	203.7	174.4	152.3	167.0	150.5	156.0
c	273.5	218.4	192.7	209.2	198.2	181.7
c	154.2	132.1	100.9	123.0	113.8	123.0
c	218.4	159.7	168.9	141.3	161.5	176.2
c	121.1	106.5	82.6	91.8	75.2	97.3
c	308.3	222.1	192.7	201.9	209.2	227.6
c	143.2	117.5	137.7	139.5	130.3	126.6
c	220.2	233.1	220.2	233.1	209.2	231.3
c	121.1	102.8	99.1	100.9	95.4	91.8

Note. m = massage subject; c = control subject.

Table 2D

Raw data for hamstring ROM (degrees).

Subject	Baseline	6h	24h	48h
m	96	91	81	85
m	85	81	78	75
m	68	66	66	65
m	115	103	89	93
m	117	103	99	104
m	83	82	80	76
m	74	70	65	59
m	91	85	83	87
m	71	82	73	73
c	65	57	59	57
c	83	77	74	60
c	85	73	73	68
c	87	91	81	89
c	87	78	68	63
c	95	91	84	83
c	86	83	83	63
c	75	75	71	67
c	79	80	83	71

Note. m = massage subject; c = control subject.

Table 3D

Raw data for neutrophil levels (neutrophils/100 cells).

Subject	Baseline	6h	24h
m	36	51	49
m	61	55	60
m	52	73	53
m	58	73	62
m	53	45	50
m	53	72	56
m	54	36	34
m	41	40	53
m	58	70	60
c	73	72	59
c	54	42	36
c	71	67	64
c	63	61	57
c	76	55	61
c	62	59	66
c	52	58	39
c	68	63	61
c	68	48	49

Note. m = massage subject; c = control subject.

Table 4D

Raw data for DDS intensity of soreness.

Subject	Baseline	6h	24h	48h
m	1.00	10.92	13.92	13.33
m		9.00	9.66	10.42
m	1.17	6.50	13.92	7.33
m	5.67	12.16	13.83	15.08
m	1.00	9.75	9.67	9.83
m	1.00	1.83	8.00	9.83
m	1.75	9.33	11.83	11.17
m	1.00	3.17	6.67	4.83
m	1.00	2.83	13.75	12.08
c	1.00	4.75	12.17	17.75
c	1.00	1.00	6.75	18.58
c	1.00	10.33	16.25	14.33
c	2.00	11.75	11.33	10.75
c	1.00	11.27	15.40	15.08
c	1.42	3.17	15.42	10.75
c	1.00	4.67	11.25	18.08
c	1.08	4.67	6.83	9.83
c	1.00	1.25	1.00	17.83

Note. m = massage subject; c = control subject.

Table 5D

Raw data for DDS unpleasantness of soreness.

Subject	Baseline	6h	24h	48h
m	1.00		7.08	9.42
m		7.80	9.00	12.17
m	1.08	2.17	9.83	6.42
m	4.25	7.33	11.58	10.08
m	1.00	4.33	10.08	8.03
m	1.00	1.17	2.33	8.92
m	1.00	1.00	1.00	1.25
m	1.00	1.00	5.17	6.33
m	1.00	1.50	11.08	12.00
c	1.00	5.83	10.17	15.33
c	1.00	3.75	2.17	10.92
c	1.00	8.33	14.08	12.41
c	3.00	12.17	14.41	15.00
c	1.00	7.25	13.00	12.42
c	1.33	2.83	6.92	10.33
c	1.58	4.25	5.42	12.00
c	1.00	4.25	4.42	8.00
c	1.00	1.00	1.00	1.92

Note. m = massage subject; c = control subject.

Table 6D

Raw data for POMS scores.

Subject	Baseline	Oh	2h	24h	48h
m	-15	-14	-11	-5	-7
m	2	11	2	15	16
m	-3	2	-8	4	4
m	-7	-9	-8	-9	-11
m	-8	3	2	0	-4
m	-7	1	5	5	
m	3	7	9	6	-6
m	-6	-2	0	-3	-3
m	-7	3	-7	2	-3
c	-11	0	-13	8	11
c	1	-8	-8	-6	-5
c	-5	0	-4	1	-3
c	20	23	19		7
c	10	15	16	17	12
c	-4	1	-2	-1	-4
c	-3	5	5	15	13
c	-3	5	5	10	-2
c	15	-5	-2	0	0

Note. m = massage subject; c = control subject.

APPENDIX E: STATISTICAL ANALYSIS

Table E1
ANOVA summary table for peak torque data (treatment x time).

Source	ss	dF	MS	F	p
Treatment	3099.80	1	3099.80	0.32	0.582
Error	156751	16	9796.96		
Time	14953.70	5	2990.74	24.63	0.000
Time x Treatment	260.52	5	52.10	0.43	0.827
Residual	9713.18	80	121.42		

Table E2
ANOVA summary table for peak torque data (gender x time).

Source	ss	dF	MS	F	p
Gender	102892.8	1	1571401	441.41	0.000
Error	56958.28	16	3559.89		
Time	14079.40	5	2815.88	24.38	0.000
Time x Gender	734.03	5	146.81	1.27	0.285
Residual	9239.68	80	115.50		

Table E3
ANOVA summary table for ROM data.

Source	ss	dF	MS	F	p
Treatment	868.06	1	868.06	1.69	0.212
Error	8208.72	16	513.05		
Time	1331.72	3	443.91	20.28	0.000
Time x Treatment	97.95	3	32.65	1.49	0.229
Residual	1050.83	48	21.90		

Table E4
ANOVA summary data for DDS intensity data.

Source	ss	dF	MS	F	p
Treatment	3.76	1	3.76	0.20	0.665
Error	288.63	15	19.24		
Time	1289.70	3	429.90	45.57	0.000
Time x Treatment	85.52	3	28.51	3.02	0.039
Residual	424.55	45	9.43		

Table E5
ANOVA summary table for DDS unpleasantness data.

Source	ss	dF	MS	F	p
Treatment	44.60	1	44.60	1.48	0.245
Error	423.45	14	30.25		
Time	588.75	3	196.25	34.12	0.000
Time x Treatment	33.93	3	11.31	1.97	0.134
Residual	241.59	42	5.75		

Table E6
ANCOVA summary table for neutrophil data.

Source	ss	dF	MS	F	p
Covariate	913.02	1	913.02	5.82	0.029
Treatment	278.05	1	278.05	1.77	0.203
S w Treatment	2350.76	15	156.72		
Time	5.08	1	5.08	0.10	0.755
Time x Covariate	0.50	1	0.50	0.01	0.922
Time x Treatment	0.03	1	0.03	0.001	0.982
Residual	751.28	15	50.09		

Table E7
ANOVA summary table for POMS score data.

Source	ss	dF	MS	F	p
Group	248.51	1	248.51	1.28	0.277
Error	2718.58	14	194.18		
Time	338.05	4	84.51	3.14	0.021
Time x Group	39.05	4	9.76	0.36	0.843
Residual	1505.30	56	26.88		

APPENDIX F: MANUSCRIPT TABLES

Table 1
Changes in hamstring peak eccentric torque (Nm).

		Baseline	O h *	2 h *	6 h *	24 h *	48 h *
Massage (n=9)	Mean	144.66	120.27	110.48	115.46	110.19	115.77
	SD	49.19	38.23	35.61	36.67	36.66	38.16
Control (n=9)	Mean	154.44	134.27	123.59	128.70	119.22	120.88
	SD	42.35	43.11	41.23	44.82	46.29	44.84

* Indicates that values for both groups were significantly different from baseline ($p < 0.000$). No treatment differences were observed.

Table 2
Changes in hamstring ROM (degrees)

		Baseline	6 h	24 h *	48 h *
Massage (n=9)	Mean	88.89	84.78	79.33	79.67
	SD	17.87	12.77	10.74	14.03
Control (n=9)	Mean	82.44	78.33	75.11	69.00
	SD	8.59	10.26	8.45	10.62

* Indicates that values for both groups were significantly different from baseline ($p < 0.000$). No treatment differences were observed.

Table 3
Changes in neutrophil levels (neutrophils/100 cells)

		6 h	24 h
Massage (n=9)	Mean	61.50 [a]	57.50 [a]
	SD	4.56	3.26
Control (n=9)	Mean	54.06 [a]	50.19 [a]
	SD	4.59	3.26

[a] Adjusted means; baseline = 58.50. No significant changes were observed.

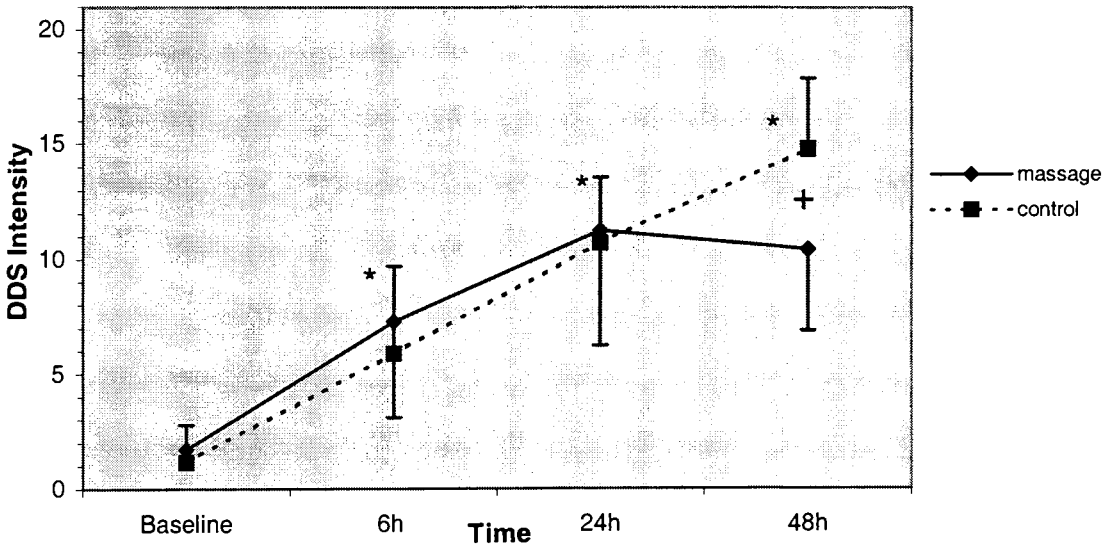


Figure 1. DDS intensity of soreness in massage and control subjects. Data are presented as means (SD), and higher scores represent greater intensities of soreness. Both groups indicated significantly higher (* $p < 0.000$) intensities of soreness at 6, 24, and 48 h post-exercise compared to baseline. Also, the control group reported significantly greater (+ $p < 0.000$) intensity of soreness at 48 h post-exercise compared to the massage group.

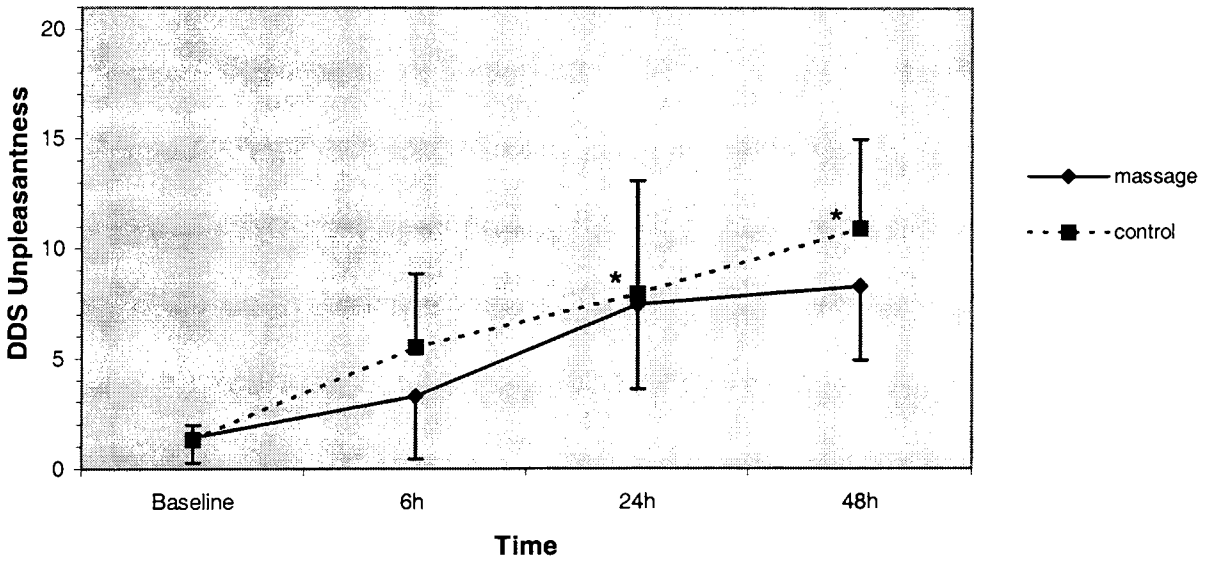


Figure 2. DDS unpleasantness of soreness in massage and control subjects. Data are presented as means (SD), and higher scores represent greater perceptions of unpleasantness. Both groups indicated significant increases (* $p < 0.000$) in unpleasantness at 24 h and 48 h post-exercise compared to baseline.

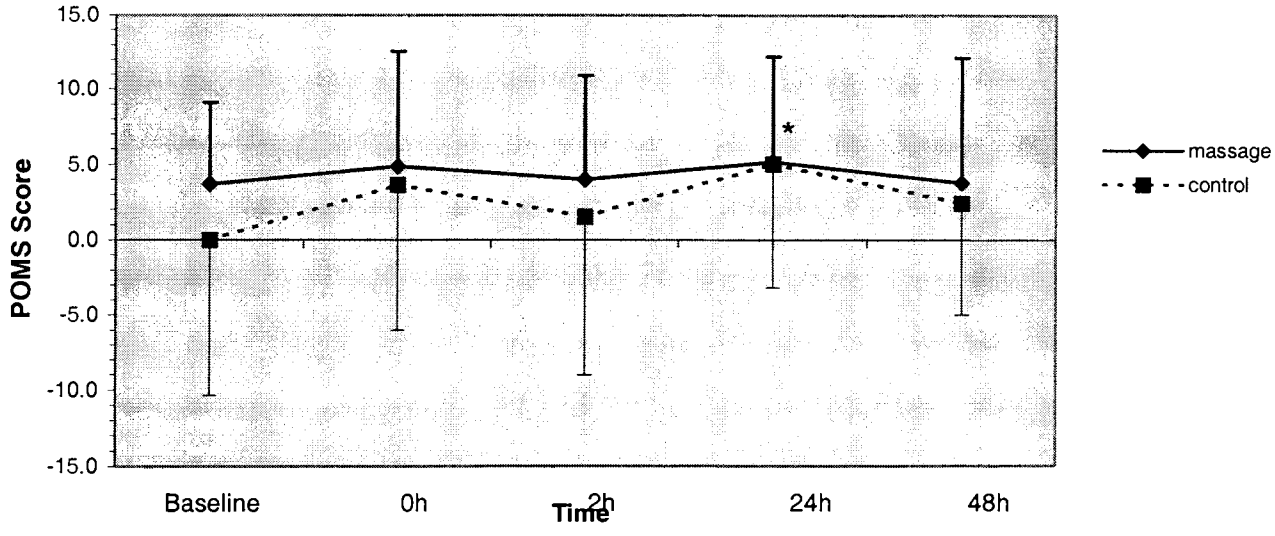


Figure 3. POMS scores in massage and control subjects. Data are presented as means (SD), and higher scores represent greater mood disturbances. Both groups indicated significantly higher (* $p < 0.000$) POMS scores at 24 h post-exercise compared to baseline.