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The effects of ibuprofen on exercise-induced muscle soreness and performance

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THE EFFECTS OF IBUPROFEN ON EXERCISE-INDUCED
MUSCLE SORENESS AND PERFORMANCE

An Abstract of a Thesis
Submitted
In Partial Fulfillment
of the Requirements for the Degree
Master of Arts

Atta Kofi Osei
University of Northern Iowa
December 1996

ABSTRACT

This study was designed to investigate whether the ingestion of ibuprofen prior to or after a 30-min bout of downhill (-10%) treadmill running would result in reduced sensation of soreness. A secondary purpose was to investigate whether ibuprofen would augment the diminished muscle performance following downhill running. Subject consisted of 21 males (age = 20.3 ± 1.7 years; weight = 77.4 ± 7.8 kg). Each subject was randomly assigned to either a control group, a prophylactic group which received 600 mg of ibuprofen 1 hour prior to the 30-min treadmill run and (200 mg every 3 hr postexercise up to 24 hours), or a therapeutic group which ingested 600 mg of ibuprofen at 24-hours postexercise and (200 mg every 3 hr till 48-hr postexercise). Multivariate ANOVA with a repeated measure revealed no significant difference in muscle soreness measures at preexercise, 0-hr, 24-hr, and 48-hr postexercise between the control, prophylactic, or therapeutic groups. Also, there were no differences between groups for vertical jump, fatigue index, maximal anaerobic power, or work capacity at any of the post-exercise testing times. Based on these results, the effectiveness of ibuprofen in regressing exercise-induced soreness or diminished muscle performance is doubtful.

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Titled: The Effects of ibuprofen on Exercise-Induced Muscle
Soreness and Performance

has been approved as meeting the thesis requirements for the
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CHAPTER I

INTRODUCTION

Soft tissue injuries, like all other tissue injuries, have plagued physical performance since early human time. The concept of muscle soreness after strenuous activity is a universal problem that confronts athletes and other occupational personnel alike. Sports participants are perhaps the worst victims since the problem has its highest incidence in sports participation (Garrett, 1990). Because of its inhibitory effects on performance, exercise physiologists, athletic coaches, clinicians, trainers, and athletes have attempted to find antidotes.

Exercise-generated pain presents itself in two basic forms namely, temporary soreness and residual soreness. Temporary soreness occurs in the course of exercise and terminates as activity stops (Friden, 1984). In residual soreness, the pain is not felt until approximately 8 hours after activity and peaks at 24 to 48-hours postexercise (Abraham, 1977). Because of the time lapse between termination of activity and its occurrence, residual soreness has been dubbed "delayed-onset muscle soreness" (DOMS) (Abraham, 1977). Postexercise soreness is the symptomatic manifestation of an injury caused to a muscle tissue (Friden, 1984). It is the aftermath of strenuous activity and characteristic of people who routinely do not perform such activity (Friden, 1984). Additionally,

activities that are intensive and extend over a long duration are also capable of inflicting severe injury (Armstrong, 1984; Newham, 1988). About 3 to 4 hours after a damage-inducing exercise, a chain of inflammatory events involving autogenic destructive processes are believed to commence in the myofiber (Almekinders & Gilbert, 1986). Phagocytic blood-borne mononuclear cells begin to invade the damaged site, and by Days 1 and 2 after exercise, the inflammatory reaction becomes more pronounced (Armstrong, 1990). Other important changes may also occur during the pre-invasion period (Armstrong, 1990) however, there is still much to be understood (Garrett, 1990).

Promoting the inflammatory process are identifiable mediators such as kinin, histamine, and prostaglandins (Weissmann, 1982). Free oxygen radicals have also been noted as important mediators in the inflammatory process (Armstrong, 1990; Brune, 1982). Even though several mediators are present, considering the critical role of the cyclooxygenase pathway in the inflammatory reaction, the resultant prostaglandins may be a primary intermediate in the inflammatory process (Onyanaugui, 1978).

Inflammation, per se, might not be directly related to DOMS however, prostaglandins, a mediator of inflammation, has been shown to be a contributor to DOMS (Ferreira, 1972; Weissmann, 1982). No specific factor has been identified as the primary cause of DOMS but potential physical and

chemical factors that have been investigated are rupture to muscle and connective tissues (Hough, 1902), tonic spasm (deVries, 1966), and edema (Bobbert, Hollander, & Huijing, 1986; Brendstrup, 1962; Friden, 1984). The period of DOMS is accompanied with a drastic muscle weakness that leads to diminished muscle performance (Newham, Jones, & Clarkson, 1987). Presumably, subjects in pain have little voluntary will to activate the sore muscle which eventually causes decrements in muscle performance.

In spite of the concurrence of DOMS and diminished muscle performance, biochemical markers of muscle damage such as serum creatine kinase and lactate dehydrogenase do not seem to have the same time course as DOMS. This suggests that DOMS may not be responsible for the reduced functional ability of the injured muscle. Studies have indicated that whereas DOMS peaks at 24 to 48-hr postexercise, diminished muscle performance persists for over 7 days postexercise. Results of studies by Herring (1990) and Newham et al. (1987) indicated that at the time of diminished DOMS, functional deficits in sore muscles existed. The failure of traumatized muscle to exert maximal force has been viewed as the result of disorganized muscle structure (Newham, McPhail, Mills, & Edwards, 1983). Diminished muscle performance that follows the injury process has also been attributed to an overextended resting length of muscle fibers (Friden, 1984).

The negative impact that mechanical and inflammatory injuries inflict on subjects have raised concerns in the area of human performance. To counteract the ensuing pain and diminished muscle performance, traditional and modern preventive and curative modalities have evolved (VanHelder, 1992). Identifiable classical clinical approaches are heat therapy; hydrocollator packs and hot immersion; electrical or ultrasonic devices; cold compress and gallium arsenide laser. Some studies have also investigated such alleviative approaches as exercise (Appell, Soares, & Duarte, 1992; iontophoresis (Hasson, Wibble, Reich, Barnes, & Williams, 1992), and trolamine salicylate (Cicccone, Leggin, & Callamaro, 1991). Another modality that has frequently been prescribed in recent times is designated as nonsteroidal anti-inflammatory drugs (NSAIDs) which include ibuprofen, aspirin, and naproxen as well as diclofenac, piroxicam, flurprofen, etodolac, diflunisal, oxaprozin, nabumetone, azapropazone, fenoprofen, fenbufen, tolmetin, and sulindac (McCormack & Brune, 1991).

The mechanism by which ibuprofen exerts its effect on DOMS is still unknown however, three mechanistic modes have been proposed namely: (a) analgesia, the shielding of pain nerve endings from pain stimuli; (b) anti-nociception, chemically blocking muscle excitation in injured muscles; and (c) anti-inflammation which involves the blocking of cyclooxygenase enzyme which catalyzes the biotransformation

of arachidonic acid. Although not universally accepted, current thought is that NSAIDs inhibit the synthesis of prostaglandins to achieve the analgesic efficacy (Brune et al., 1991). McCormack and Brune (1991) have suggested that the anti-nociceptive action of NSAIDs could be tied to the inhibition of accumulation of prostaglandins and other mediators of inflammation. They further argue that the two modes of action, analgesia and nociception differ only in terminology, but that the chemical processes are basically the same.

Studies that attempt to establish a link between anti-inflammation and analgesia of NSAIDs have suggested that the two mechanisms are inseparable (Cicccone et al., 1991; Hasson et al., 1993). Some NSAIDs are also believed to be scavengers of the hydroxyl radical (OH⁻) (Famaey, 1982). The scavenging action hastens the transformation of PGG₂ (inflammatory) to PGH₂ (less inflammatory) (Onyanaugui, 1978). There seems to be a clear association among DOMS, diminished muscle performance and muscle damage. The damage itself can be attributed to mechanical and biochemical processes. Using NSAIDs may thus provide reliable intervention to DOMS through such mechanistic modes as analgesia, antinociception, and anti-inflammation.

Statement of the Problem

The purpose of this study was to investigate the effects of ibuprofen ingested either prophylactically or therapeutically on delayed-onset muscle soreness and on muscle performance following an eccentric-biased running bout.

Significance of the Study

Previous studies have investigated the effectiveness of ibuprofen in reducing DOMS and diminished muscle performance but produced conflicting results. Most studies have focused on subjective assessment of DOMS to evaluate the efficacy of drugs disregarding the deleterious effects of the injury on muscular performance. Due to the inconclusiveness of such investigations the present study is warranted.

Research Hypotheses

The research hypotheses are as follows:

1. Ibuprofen would diminish DOMS.
2. Ibuprofen would help restore muscle performance more quickly than without.
3. Prophylactic treatment with ibuprofen would be more effective than therapeutic in reducing DOMS and restoring muscular performance.

Assumptions

The following assumptions were made:

1. Subjects exerted maximal effort on the cycle ergometer and in the vertical jump test.

2. Subjects abstained from ingesting NSAIDs or additional dosages of ibuprofen or other NSAIDs before and after the exercise treatment or employing any intervention mode to alleviate soreness.

3. Motivation was equally high in all subjects.

4. Subjects honestly indicated their true extent of soreness perception on the analogue scale.

5. Instruments and tests used in the study were accurate, reliable, and valid.

6. Pre-testing did not influence post-test scores.

7. There was consistency and reliability in the soreness measuring scale, the Wingate and vertical jump tests.

Delimitation of the Study

This study was delimited to the following:

1. Twenty-one volunteer male students of the University of Northern Iowa.

2. Students who were not participating in any intercollegiate sport at the time of the study.

3. Students between 18 and 33 years of age.

4. Healthy individuals (subjects who 3 months prior to the study, had not been referred to a physician for any muscle injury treatment).

5. Running on a Quinton 18-60 or a Marquette 2000 T motor-driven treadmill in the Human Performance Laboratory of the University of Northern Iowa.

6. The visual analogue scale used to measure soreness.
7. The vertical jump and Wingate cycle ergometer tests.

Limitations of the Study

The following were limitations of the study:

1. The inability to ensure equal efficacy of the drug in all subjects based on varying individual body composition and metabolism.
2. The possibility that subjects may take extra dosages of ibuprofen or other NSAIDs.
3. The uncertainty of honesty or otherwise with which subjects disclosed muscle soreness.
4. The validity and reliability of the testing instrument, soreness measurement scale, vertical jump, and the Wingate tests.

Definition of Terms

1. Prophylactic--the administration of ibuprofen before activity.
2. Therapeutic--the ingestion of ibuprofen 24 hours after exercise when soreness begins to reach its peak.
3. Concentric work--movement in which the muscle shortens as it produces force.
4. Eccentric work--movement in which the muscle lengthens as it produces force.

7. Antioxidants--substances that help reduce the degree of oxygen stress either by forming a lesser active radical or inhibiting the reaction.

8. Nociceptor--a drug that has the potential to shield pain receptors.

CHAPTER II

REVIEW OF LITERATURE

To date, the use of ibuprofen and other pharmacological modalities as an antidote to exercise-induced muscular soreness has not exhaustively addressed the issue of dosage, timing, efficacy in performance restoration, and, most importantly, its mode of action. The present study was therefore designed to investigate whether the oral ingestion of ibuprofen before or after treadmill running would reduce pain sensation and more quickly restore muscular performance.

Of the soft tissues injuries associated with sports, overload injuries to muscle fibers seem to be the most prevalent (Glick, 1980; Ryan, 1969). Victims to such damage show such symptomatic signs as pain (Newham, 1988), swelling, anatomical deformity, and athletic dysfunction (Kibler, 1990; Newham et al., 1987). The cost of muscle damage to athletes is enormous and in instances of relatively high performance, the cost is expressed in terms of high energy expenditure leading to further injuries (Kibler, 1990).

Muscle soreness and occasional diminished muscular performance that follow tissue damage are attributable primarily to mechanical damage and secondly, to inflammatory responses as part of biochemical reactions to the initial fiber damage (Almekinders & Gilbert 1986).

Muscle Damage

There is very little clarity about the mechanism that leads to DOMS. Ebbeling and Clarkson (1989) however, suggest that mechanical and biochemical factors as discussed below, could be causative.

Mechanical Disruption of Cells

Mechanical stress on muscle fiber is believed to be very instrumental in generating structural disruptions which later subjects the muscle to further damage through biochemical reactions. Arguments in favor of mechanical processes as potential cause of muscle damage has been investigated and is tied to eccentric motion. In a study by McCully and Faulkner (1985), eccentric motion resulted in greater injury to muscle fibers than did isometric or concentric activities. Muscles of female albino mice were stimulated through isometric contraction and lengthening activity. Tissue preparations following the eccentric protocol revealed greater damage to the fibers.

Almekinders (1993) further suggested that during long distance running, each running cycle involves a shortening and lengthening process of the muscle. The singular impact of this process is less than enough to cause injury to the muscle tendon unit, but the summative effect could be enormous.

Faulkner, Brooks, and Opittek (1993) suggested that injury from eccentric motion is attributable to the increased tension per individual cross bridge. Force development by a fiber during eccentric motion is approximately two times that developed during isometric movements, yet there is only a 10% increase in the number of cross bridges in eccentric motion (Faulkner et al., 1993). With a higher fiber to force ratio, mechanical disruption may occur to the ultrastructural elements in the muscle fibers such as the Z-line and contractile filaments and associated connective tissues (Armstrong, 1984). Duration and intensity of activity have also been shown to play an important role in fiber damage (Armstrong, 1984; Newham, 1988). Although Tiidus and Ianazzo (1983) have suggested that intensity of activity has greater potency in causing muscle soreness than the duration of exercise, tendency for a negatively moving muscle fiber to be damaged is still great.

Muscle types which have greater vulnerability to damage are: (a) two-joint muscles, (b) Type II fibers, and (c) muscles involved in eccentric motion (Garrett, 1990). His views are suggestive that activities which require burst, speed, and high tension recruit a greater percentage of Type II fibers and, due to the higher speed of contraction, the Type II muscles are likely to be predisposed to injury. As evidence, Friden (1984), had subjects perform eccentric

movement on a modified cycle ergometer. Micrographs from prepared tissues showed 4:1 ratio of Z-band anomalies for Type II to Type I fibers. Histological and ultrastructural analyses of muscle fibers indicate that whereas the Z-disk appears as a square lattice (Hoppeler, 1986), under damaged conditions the Z-disk shows streaming, broadening, and disruption in electron micrographs (Friden, 1984).

Myofibrilla and sarcolemmal disruptions and widening of A band and I bands were also seen as signs of damaged muscle fibers (Armstrong, Ogilvie, & Schwane, 1983).

Biochemical Damage

The principal response involved in biochemical damage is inflammation at the site of the initial mechanical damage. Inflammation is in itself characterized by movement of fluid and migration of neutrophils and monocytes to the damaged area. Biochemical response help to promote clearance of damaged tissues, eliminate microbial invaders, and also promote tissue repair (MacIntyre, Darlene, & McKenzie 1995). Little is known as to when the injury process starts, but the inflammatory response is believed to follow the injury approximately between 3 and 4 after the damage (Armstrong, 1990). The associated pain sensation appears about 8 hours post-activity (Newham, 1988).

Inflammation is associated with an initial vasoconstriction that lasts for 5-10 min followed by vasodilation, increased vascular permeability, and extrusion

of blood products into the surrounding tissues causing edema (Abramson & Weissmann, 1985). This is followed by recruitment of inflammatory cells such as leukocytes and macrophages which attract neutrophils to the injured site (Armstrong, 1990). Neutrophils have the ability to release agents that assist in the digestion of surrounding dead tissue cells. Since neutrophils have difficulty distinguishing between damaged and healthy tissues, healthy tissues may be mistaken for damaged or foreign bodies (Weiss, 1989). Under such circumstance, toxic agents are released by the neutrophils to attack healthy tissues, culminating in oxidative damage and further inflammation (Weiss, 1989). Neutrophils are also capable of causing tissue injury through the release of oxygen radicals. Oxygen radicals are highly reactive chemical species that have one or more unpaired electrons in the outer shell. In creating lipid peroxidation, free oxygen radicals decrease the barrier function of cell membranes. Free oxygen radicals are believed to be associated with necrosis and enzyme release following damaging exercise (Ebbeling & Clarkson, 1989).

Prostaglandins, Mediator of Inflammation

The origin of prostaglandins and the associated inflammatory symptoms including pain, redness, and swelling. Whereas some authors hypothesize prostaglandins as able to

sensitize pain endings to specific mediators (Brune, Menzel-Soglowak, & Zeilhfer, 1992), others consider prostaglandins as modulators of inflammation (Willoughby & Sedgwick 1982). Onyanagui (1978) yet proposed that prostaglandins can potentiate the increased vascular permeability under the influence of other mediators namely, histamine, serotonin and kinin, which will likely cause edema. Prostaglandins are classified into stable such as PGE2 and PGF alpha and unstable example, PGE1 (Wiessmann, 1982). PGE1 is a vasodilator which, when injected into normal human skin, produces erythmia and pain. PGE1 has also been shown to act with bradykinin and histamine to potentiate edema. The presence of prostaglandins in damaged muscles was observed in a study by Bansil, Wilson, and Stone (1986). After having 32 males perform squats, aspirin and placebo were administered. Blood drawn at preexercise and every 12 hours postexercise up to 72 hours was analyzed by radioimmunoassay. The results showed a significantly increased plasma levels of PGE1 and PGF2 alpha in the placebo group.

Prostaglandins and the Cyclooxygenase Pathway

The association between prostaglandins and the cyclooxygenase pathway during inflammation is seen in the biotransformation of arachidonic acid in which cyclooxygenase plays a catalytic role. Arachidonic acid is a natural component of phospholipids in cell membrane and is

released from phospholipids in the presence of phagocytic cells (Weissmann, 1982). In the oxidation of arachidonic acid, cyclooxygenase and other enzymes act as catalysts to produce thromboxanes A₂ and prostalandins. Thus, the extent of inflammation caused by mediating prostaglandins can be speculated to depend on the amount of cyclooxygenase available to catalyze the oxygenation of arachidonic acid. The same reaction produces an unstable endoperoxide, PGG which after oxidation produces hydroperoxy endoperoxide. In the end, free oxygen radicals are liberated to cause further damage (Allessio, 1993; Grisham & McCord, 1986; Jenkins & Goldfarb, 1993).

The presence of tissue and cellular artifacts have variously been used as markers of tissue damage. After having subjects perform eccentric work, Clarkson, Litchfield, Graves, Kirwan, and Byrnes (1985) observed significantly greater muscular soreness and elevated muscle enzymes in the blood. Similar findings were made by Kuipers, Keizer, Verstappen, and Costill (1985) in a study in which three subjects performed an eccentric work bout for 45 min and one for 60 min at 80% of the individual's maximal work load on a cycle ergometer. In all the 4 subjects used, moderate increases of muscle enzymes were observed. In addition, plasma concentration of creatine phosphokinase peaked at 24-hours postexercise.

Consequences of Muscle Fiber Damage

Consequent to mechanical and biochemical damage to the muscle fiber, delayed-onset muscle soreness results along with a reduction in muscular force in the affected areas. Nonetheless, the time course of the two phenomena do not seem to be related.

DOMS and Theories of Origin

Opinions are divided on the chemical or physical agents that stimulate DOMS (Armstrong, 1984) which is felt approximately, 8 hours postexercise and peaking at 24 to 48 hours after exercise (Abraham, 1977; Tiidus & Ianazzo, 1983). DOMS is the aftermath of a strenuous activity and is characteristic of people who are not accustomed to such activities (Friden, 1984). Three causes of DOMS have been proposed namely, torn tissue (Hough, 1902), tonic spasm (deVries, 1966), and edema (Brendstrup, 1962).

The Torn Tissue Theory

From electromyographic (EMG) studies conducted by Hough (1902), muscle soreness observed in untrained individuals after heavy resistance work added to loss of contraction strength. Hough suggested the cause of the pain as due to rupture within the muscle itself or the connective tissue that transmits the pull of the fibers to the tendons. Tenderness is restricted to musculotendinous attachments, so that in the process of contraction, muscles pull on the

injured tendons to cause pain (Newham, Mills, Quigley, & Edwards, 1983). The Torn Tissue model however, has not received substantial support over the years.

Tonic Spasm Theory

deVries (1966) explained DOMS from an EMG point of view. The results of his investigations revealed a greater resting motor unit activity in sore muscles than in control subjects. He proposed the possibility that spasms within motor units can be the cause of DOMS. He also suggested that severity of the tonic activity is a factor of the number of motor units involved. A report by Cobb, deVries, Urban, and Leukens (1975) indicated that increases in EMG activity may be accompanied by muscle pain perception. Supporting this view, Bobbert et al. (1986) concluded that if tonic muscular spasm of motor units is present and is severe enough to partially occlude blood vessels, ischemia could result causing pain. Bobbert et al. (1986) had 11 subjects perform plantar-dorsiflexion with one leg on a 20-cm thick board while the other leg was held off the floor. Results of this study showed tonic spasm in some of the subjects. Even if all the subjects experienced pain or otherwise it is difficult to speculate if the discriminatory spasm is the actual cause of the pain. In a study conducted by Komi and Viitalaso (1977) however, pain was inferred as a probable cause for increased neural activation since more central activation, at a given tension, is required for sore

muscle compared to healthy ones. Komi and Viitalaso (1977) had four subjects perform concentric and eccentric exercises by exerting 40 maximal contractions, each lasting for 3.5 sec with a 11.5 sec break. Knee angle was set at 107 degrees and subject instructed to exert maximal force on an electromechanical dynamometer. EMG was recorded continuously before, during, immediately, and 2 days after the work. Muscle tension averaged for each contraction decreased substantially more in eccentric than in concentric fatigue conditions. An integrated EMG analysis showed that substantially more neural energy was needed for the production of certain muscle tension after fatigue loading than before.

Edema and Intramuscular Pressure

In a study conducted by Brendstrup (1962) using rabbits, an increase in water and chloride concentration were observed in the exercised muscles 24-48 hours after exercise but disappeared by 6 days. Since the time courses for postexercise pain and increased fluid content were similar, he attributed the pain to edema. Talag (1973) supported the edema model after observing increased limb volume at 24-, 48-, and 72-hours post-exercise which was accompanied with subsequent soreness. Fritz and Stauber (1988) used histological procedures to observe changes in the extracellular matrix of rat muscle 24 hours after

eccentric exercise. They suggested that disruption of proteoglycan components result in movement of water to the extracellular matrix through increased osmotic force.

Diminished Muscle Performance and Causes

There seems to be very little known about the duration of diminished muscle performance following muscle injury. Clarkson and Tremblay (1988) observed loss of performance up to 1-hour postexercise. Sergeant and Dolan (1987) had four subjects perform repeated eccentric contraction with leg extensor in a prolonged downhill (-25% grade) walking. Muscle function, expressed in terms of maximum voluntary contraction and short-term power output remained diminished for 96-hour postexercise.

There is little in the literature on what causes loss in strength (MacIntyre, Darlene, & McKenzie, 1995). Studies have however speculated that the loss in muscle performance is attributed to biochemical and mechanical media namely, DOMS, edema, disorganized muscle morphology, or a combination.

DOMS

Several studies have supported the notion that the time of soreness sensation is occasioned with severely reduced muscle strength. The reasoning behind this is that during the periods of DOMS, victims feel very reluctant to voluntarily activate a muscle to move a limb. Newham et al. (1983) suggested that tenderness was restricted to

musculotendinous attachments, and as the muscle contracts, a pulling action is made on the injured tendons to cause pain. Because of the voluntary nature of movement, the existence of pain will offer victims little voluntary will to contract the sore muscles to generate maximal force.

Muscle Morphology

Muscle damage is associated with disorganization of the contractile material within the muscle fibers (Friden, 1984). The immediate loss of function may be due to overstretched sarcomeres. As a result, actin and myosin filaments are separated due to the high tension developed from the initial eccentric work which impairs force production. Faulkner et al. (1993) suggested that some sarcomeres stretch beyond a physiological limit during eccentric activity and become injured. Although there may be some metabolic involvement, Faulkner et al. (1993) believe that the initial decline in force may be a function of fatigue and mechanical injury which results in myofibrilla disruption at the Z-line level.

Biochemical Factors (Calcium Extrusion and ATP Loss)

Two substances considered to initiate biochemical damage are ATP and calcium. When actin and myosin are pulled apart, the sarcolemma is damaged allowing entry of extracellular calcium. Increased intracellular calcium in muscle cells from a damaged muscle sarcolemma or sarcoplasmic reticulum can induce biochemical damage to an

already mechanically damaged muscle fiber (Armstrong, 1984; Armstrong, 1990). A principal enzyme that has been identified as the underlining factor in the calcium pathway is phospholipase A2 (Duncan & Jackson, 1987). One action of phospholipase A2 is the breakdown of fatty acids and lysophospholipids in the sarcolemma (Armstrong, 1990). Fatty acids may in turn produce prostaglandins which will eventually mediate inflammatory reactions.

Fiber Cross-sectional Area Model

Friden (1984) explained the loss of contractile strength as due to the reduction in cross-sectional area of the injured muscle fiber. This is in accordance with the reasoning that a muscle fiber exert force directly proportional to its cross sectional area (Garrett, 1990). In a study by McCully and Faulkner (1985), the left extensor digitorum longus of female albino mice were stimulated and performed isometric, shortening, or lengthening contractions. Similar muscles of the contralateral leg were used as a control. The results showed decrements in force at Day 3 in muscle which underwent lengthening contraction and also a strong correlation between loss in force and shrinkage in the total cross-sectional area.

Physiological reactions in the body following biochemical and physical damage give rise to pain and reduced muscle performance. Whereas tonic spasm, edema and torn tissue have been speculated to mediate the pain,

diminished muscle performance is essentially believed to arise from pain, loss of calcium from the sarcolemma, overextended fiber length and emaciated fiber cross sectional area.

Soreness Alleviating Procedures

Athletes who sustain muscle injuries show several clinical signs including pain, swelling and such functional deficiencies as anatomical deformity, and athletic dysfunction (Kibler, 1990). He suggested that treatment of muscle injury must be geared towards reestablishing function rather than relief of symptoms. Traditional procedures of alleviating DOMS have constituted thermal therapy, cryotherapy, and physical therapy. In recent times, supplementary sources have been found in the form of pharmacological resources, predominantly non-steroidal anti-inflammatory drugs (NSAIDs). NSAIDs are medicinal agents which are believed to inhibit the process of biochemical cellular degradation in soft tissues involved in inflammation. The mode of action of NSAIDs is thought to be inhibiting the synthesis and release of prostaglandins and inhibition of neutrophils (Abramson, Edelson, Kaplan, Ludewig, & Weissmann, 1984). Common examples of NSAIDs easily available for use currently are aspirin and ibuprofen.

Mechanism Of Action Of NSAIDs

NSAIDs have been noted for their ability to act as an inhibitor to prostaglandins synthetase (Boots Lab leaflet, 1994; Brune, 1982). In spite of the confusion regarding their pharmacology, researchers believe that NSAIDs act in the following three related ways.

Analgesia

The analgesic action of NSAIDs is observable from its ability to inhibit prostaglandin synthesis (McCormack & Brune, 1991; Brune et al., 1991). A study conducted by Brune et al. (1992) looked at changes in pain perception that result from tissue damage. The researchers suggested that tissue damage induced by prostaglandins can transform fine nerve endings of high threshold mechanoreceptor into a nociceptor. This transformation combines with increased reflex activity in the dorsal horn of the spinal cord to generate hyperalgesia associated with tissue damage. By inhibiting prostaglandin activities, NSAIDs reduce the nociceptive process.

Anti-Nociception

Braga, Biella, and Tiengo (1987) investigated the anti-nociceptive action of aspirin and tenoxicam on adjuvant-induced arthritis in rats. They reported that intravenously administered aspirin (54 mg/kg) produced a marked anti-nociceptive effect within 8 minutes. McCormick and Brune (1991) reported that NSAIDs possess antinociceptic

features which make them capable of shielding the injured site from pain sensation. This shielding mechanism could be critical in protecting pain nerve endings of the injured muscles. Presumably it serves as chemical blocks to excitations that could otherwise generate pain sensation in the affected sites.

Anti-Inflammation

The anti-inflammatory effect of aspirin and indomethacin may be attributed to its ability to inhibit the cyclooxygenase enzyme, a catalyst to the metabolism of arachidonic acid. By inhibiting the catalytic activities of cyclooxygenase, the biotransformation of arachidonic acid to thromboxanes and endoperoxides is prevented (Weissmann, 1982). As a result, the production of endoperoxides and inflammation mediators are blocked (Onyanaugui, 1978). Since the quantity of thromboxanes, a mediator of inflammation is reduced, less inflammatory reaction is likely to occur. NSAIDs are also believed to inhibit oxygen radicals and are capable of scavenging OH⁻, thus transforming PGE₂ (inflammatory) to PGH₂ (less inflammatory) (Onyanaugui, 1978).

In regressing inflammation, Almekinders (1993) recommended treatment to be aimed at initial symptoms of inflammation whereas subsequent treatment should be geared towards repair of the damaged muscle fibers. He further suggested the use of NSAIDs after injury but for a

relatively shorter period of time so as not to interfere with the subsequent muscle repair and remodeling process.

This assertion was substantiated in a study by Almekinders and Gilbert, (1986) using three groups of Sprague-Dawley rats. Group 1 underwent muscle strain of the tibialis anterior muscle. They then rested in the post-injury period. In Group 2 similar muscle was strained but piroxicam (10 mg/kg) was administered. Group 3 had no strain, immobilization, nor drug treatment. Histological evaluation showed hemorrhage and muscle fiber disruption along the strain. At Day 2, the group that underwent muscle strain showed extensive invasion of macrophages. There was but slight inflammation in the piroxicam treatment group with little monocytic response. Muscle tissues in the third group had normal microscopic appearance and also showed evidence of muscle regeneration. The delay in muscle regeneration of muscle fibers in the treatment group was attributed to the inhibition by piroxicam to inflammation.

Ibuprofen has been studied on a limited scale in terms of its effect on DOMS. The few investigations conducted using ibuprofen and/or related NSAIDs have had conflicting conclusions regarding its efficacy in reducing DOMS and restoring muscle performance.

In a study by Donnelly, Maughan, and Whiting (1990), ibuprofen did not reduce muscle soreness nor restore muscle strength after 32 subjects performed two bouts of downhill treadmill running separated by ten weeks. Group 1 ($N = 16$) received ibuprofen at first period and placebo at the second. Group 2 ($N = 16$) received placebo at first, but ibuprofen at the second period. Subjects ingested 600-mg ibuprofen or placebo 30 minutes before each exercise bout and took another 600 mg every 6 hours up to 72 hours post-exercise for a total of 8,400 mg. Preexercise, 16-hr, 24-hr, 48-hr, and 72-hr postexercise measures of muscle soreness indicated significant increases in total muscle soreness after each exercise bout. However, no difference in soreness response existed between the two treatments nor the first or second downhill run. Measurements of maximal voluntary force or isometric contraction of the legs decreased about 10% from initial leg strength. The group that received ibuprofen for the first bout and placebo for the second bout demonstrated the greatest decline in strength following exercise irrespective of treatment. Based on the results of this study, the researchers concluded that at a dosage of 600 mg/6 hour, ibuprofen is not effective in reducing DOMS nor muscle strength loss. They further suggested that DOMS may not be the result of inflammation.

In a similar study by Hasson et al. (1993), prophylactic or therapeutic use of ibuprofen were both found to be effective in reducing DOMS and improving muscle performance compared to placebo and control conditions. The 20 subjects involved in this study were randomly assigned to a prophylactic group who ingested 400 mg of ibuprofen 4 hours prior to the exercise or additional 800 mg over the next 24 hours; a therapeutic group which took 400 mg of ibuprofen at 24 hours postexercise and 800 mg over the following 24 hr; placebo group; and a control group. The exercise protocol involved step-ups on a bench that was 110% in height of the lower leg length while carrying an additional load that was 10% of subjects body weight. Pre-exercise, 24-hr, and 48-hr postexercise muscular performance, creatine kinase and soreness perception were evaluated. The prophylactic ibuprofen group reported significantly less soreness than the therapeutic, placebo or control group. At 48-hr postexercise, soreness was significantly less for prophylactic ibuprofen group than for the placebo and control groups. Maximal isometric contraction by an EMG as a percentage of change from baseline measures indicated that in all groups, maximum voluntary contraction was significantly reduced at 24 hours relative to baseline measures. The prophylactic group significantly decreased less in isometric force production than the therapeutic, placebo or control group. At 48

hours, maximum voluntary contraction declination from baseline value was significantly less than that of the placebo and control groups. They suggested that if the development of muscle soreness is an indication of inflammatory response, and a relationship exists between the magnitude of DOMS and inflammation, then the reduced soreness exhibited by the treatment groups will show markedly attenuate inflammation.

Summary

There is evidence in the literature which supports the view that DOMS arises from an initial mechanical damage. Following the trauma, biochemical reactions autogenic to the muscle fiber ensue. Evidently, eccentric motion is a plausible cause for the initial muscle injury. Later, the injury manifests itself in the form of inflammatory reaction and DOMS. An interplay of DOMS, disorganized muscle morphology, calcium loss, and ATP shortage then generate diminished muscle performance. Traditional and modern procedures ranging from cryotherapy, thermal therapy, physical therapy and pharmacological modalities have been prescribed to alleviate DOMS. Clinicians and physical therapists have resorted to and frequently prescribed the use of such pharmacological sources as aspirin, piroxicam, indomethacin and ibuprofen, generally referred to as NSAIDs. In spite of the popularity among users, ibuprofen does not seem to reduce DOMS or diminished muscle performance.

CHAPTER III

METHODS

The primary purpose of this study was to determine whether the oral ingestion of ibuprofen would cause a reduction in the sensation of muscle soreness that results from physical activity. The secondary purpose was to determine if ibuprofen can restore muscle performance more quickly than nontreatment group. A third purpose was to determine whether pre-exercise or post-exercise treatment of ibuprofen would generate differential effects on muscle soreness and degraded muscle performance.

Subjects

Twenty-nine male non-athletes from the University of Northern Iowa between the ages of 18 and 33 years volunteered to participate in this study. Eight subjects were unable to complete all phases of the study for various reasons, leaving 21 subjects that participated in the research. The subjects were not engaged in any regular sports training requiring leg power nor had sustained any lower limb injury in 3 months prior to participation.

All were fully informed of the nature and risk involved in the procedures to be used, and they provided informed consent. Subjects were instructed to abstain from exercise, ingesting alcohol, smoking, massage, using anti-inflammatory drugs, analgesics or thermal modalities during their involvement in the study.

Experimental Procedure

All testing was conducted in the University of Northern Iowa's Human Performance Laboratory at the same times of day for each subject. Subjects reported to the laboratory three times throughout the study. On the first visit to the laboratory, each subject was randomly assigned to one of the following three groups:

1. Control (Con): (\underline{n} = 7)-no ibuprofen administered.

2. Prophylactic ibuprofen (PI): (\underline{n} = 7)-initial 600 mg ibuprofen taken 1 hr before exercise followed by 200 mg every 3-hr postexercise through 24-hr postexercise for a total of 2,200 mg of ibuprofen.

3. Therapeutic ibuprofen, (TI): (\underline{n} = 12)-600 mg of ibuprofen at 24 hr and 200 mg every 3 hr up to 48 hr post-exercise for a total of 2,200 mg.

Baseline measures were made of each subject on the following dependent variables: perceived muscle soreness, anaerobic explosive power, fatigue index, maximal power, and work capacity.

Soreness-Inducing Exercise Bout

The exercise bout consisted of downhill running on the treadmill. All running was performed on either a Quinton Model 18-60 (Quinton Instruments, Seattle, Washington, USA) or Marquette 2000 T (SensorMedics Corporation, Yorba Linda,

California, USA) motorized treadmill. Heart rates were measured during exercise by telemetry using a Polar heart rate monitor (Polar CIC Inc. Fort Washington, NY, USA).

The treadmill running begun with a 3-min warm-up. Subjects begun 1 min of walking. Afterwards, treadmill speed was increased progressively for subjects to start jogging for a 3-min warm-up. Treadmill speed was adjusted to attain a heart rate equivalent to 80% of estimated age adjusted ($220 - \text{Age}$) maximal heart rate of subject. The treadmill was then declined to -10% for running for another 30 minutes. Following the 30 minutes of downhill running, the treadmill speed was reduced to 2 k.hr⁻¹ for a 2-min walk for recovery.

Measurements

Prior to the baseline measures, subjects received tutorials on the tests which were repeated at 0-hr, 24-hr, and 48-hr post-exercise in the order presented below. This order of presentation was adopted to avert the chances of fatigue from a more intensive activity influencing the results of subsequent tests.

Muscular Soreness

A visual analogue scale which had presumably been tested for reliability and validity, as used by Redenburg, Bar, and De Boer (1993), ranged from 0 (no soreness) to 6 (intolerable soreness) was employed, see Appendix E. The scale was shown to subjects and explained as a self-

reporting estimate of their soreness by picking the most appropriate adjective that describes muscle soreness on the scale. Soreness was measured in the following manner: from an erect position with hands held in front, the subject bent at the knees and hips until reaching 90 degrees at both the hip and knee joints. This position was held for 5 sec and repeated three times separated by a 10-sec rest. The subject then perceptually assessed their feelings of pain for all three trials and chose a number on the scale that was representative of the extent of his soreness.

Vertical Jump

Assessment of each subject's maximal reach was made initially. Standing 10 cm from and facing a wall, subjects rocked onto their toes and, with a powdered middle finger made a spot on the wall. The distance between the floor and the spot was measured and taken as subject's reach. Subjects then did five bound-offs and 1 min of stretching activities as warm-up. Subjects then dipped the tip of middle finger of dominant hand into powdered chalk. Standing perpendicular to and about 30 cm away from a wall, subjects lowered their body by bending at the hip and knee and jumped three times with 10 sec rest between jumps. This was performed maximally and placed a chalk mark on the wall at the apex of the jumps. Deviations of the height of the chalk spots from subjects' reach were measured and the mean value for the three repetitions was used.

Anaerobic Work Capacity

Muscle work capacity was measured by using a 30-sec Wingate test (Patton & Duggan, 1983). A Monark 814 cycle ergometer (Ergomedics Sweden) with a weighted pan was used. A photocell (Sports Medicine Industries) interfaced with a personal computer was used to count flywheel revolutions. Before each test, the ergometer seat height was adjusted such that at the lowest pedal propulsion the legs are fully extended. Subjects' feet were slotted into stirrups of the pedal and warmed up on the cycle ergometer by gently pedalling for 3 min. Thereafter, subjects were instructed to increase pedalling rate steadily. On reaching maximal pedal rate within 5-10 sec, a weight equivalent to 7.5% of the subject's weight was applied. Subjects were verbally encouraged to maintain maximal effort during the 30-sec test. The weight was then removed while subjects pedalled at low resistance for 3 min to recover.

Three variables were calculated by the computer: (a) maximal power-highest power output within any 5-sec period, (b) work output, and (c) rate of fatigue, the percentage of power drop off. To ensure compliance to the requirements of the study, subjects were directed to complete a general log which asked questions pertaining to the timely ingestion of ibuprofen, abstention from smoking, alcohol, exercise and ingestion of extra NSAIDs. These forms were reviewed for any indicators which might exclude a

given subject from the study. Subjects made second and third visits to the laboratory to go through all but the treadmill protocol at the same times with an interval of approximately 24 hours.

Analysis of Data

A multivariate ANOVA with repeated measure on one factor was used to determine the effects of the treatment on muscle soreness and muscle performance and any interaction effect. Level of significance was established at .05. Data were entered into the mainframe computer at the University of Northern Iowa and were analyzed using the Statistical Package for the Social Sciences (SPSS).

CHAPTER IV

RESULTS AND DISCUSSION

The purpose of the study was to investigate whether ingestion of ibuprofen prior to or after a muscle-damaging exercise would reduce muscle soreness. A secondary purpose was to find out whether ingestion of ibuprofen would restore diminished muscle performance more quickly than without ibuprofen. The primary research hypothesis was that use of ibuprofen before or after the damage-inducing exercise would reduce muscle soreness and diminished muscle performance. Furthermore, prophylactic administration of ibuprofen was hypothesized to cause a greater reduction in soreness and diminished muscle performance.

Results

Twenty-one subjects completed all parts of the study. The mean age and weight of the subjects completing the study were 20.3 ± 1.7 yr and 77.4 ± 7.8 kg, respectively. Subjects' physical characteristics and raw data are presented in Appendix F. In Table 1 descriptive statistics of subjects at preexercise, 0-hr, 24-hr, and 48-hr postexercise of muscle soreness, fatigue, vertical jump, maximal power, and work capacity are presented.

Table 1

Descriptive Statistics of Soreness, Vertical Jump, Maximal Power, and Work Capacity (N = 21)

	<u>Control</u>	<u>Prophylactic</u>	<u>Therapeutic</u>
Muscle Soreness (V/A Scale)			
PreEx	0.43 ± 0.79	0.29 ± 0.49	0.14 ± 0.38
0 hr	2.29 ± 0.76*	2.43 ± 1.51*	2.15 ± 1.21*
24 hr	3.00 ± 1.41*	2.29 ± 1.50*	3.29 ± 0.95*
48 hr	3.43 ± 1.62*	2.14 ± 1.35*	2.71 ± 1.38*
Vertical Jump (cm)			
PreEx	43.33 ± 2.51	42.40 ± 3.73	45.90 ± 4.74
0 hr	40.90 ± 2.26	40.15 ± 3.72	42.40 ± 5.25
24 hr	41.24 ± 2.32	40.36 ± 3.68	40.06 ± 4.90
48 hr	41.40 ± 2.21	41.07 ± 3.91	41.46 ± 4.03
Fatigue Index (%)			
PreEx	27.09 ± 12.00	24.13 ± 9.50	27.67 ± 7.67
0 hr	18.37 ± 10.36	24.54 ± 18.89	31.57 ± 9.88
24 hr	35.09 ± 15.38	29.19 ± 7.79	36.99 ± 7.50
48 hr	26.66 ± 13.55	36.61 ± 14.68	30.04 ± 5.66

(table continued)

	<u>Control</u>	<u>Prophylactic</u>	<u>Therapeutic</u>
Maximal Power (W)			
PreEx	908.14 ± 89.32	813.43 ± 53.23	857.86 ± 120.28
0 hr	834.43 ± 72.68	833.71 ± 62.22	844.14 ± 161.88
24 hr	919.57 ± 111.75	882.00 ± 68.75	873.43 ± 162.19
48 hr	920.43 ± 97.70	815.29 ± 103.97	894.71 ± 180.75
Work Capacity (kJ)			
PreEx	23.57 ± 17.53	21.56 ± 16.44	22.53 ± 36.62
0 hr	22.84 ± 18.30	22.55 ± 18.73	20.37 ± 21.64
24 hr	23.43 ± 34.39	22.62 ± 17.34	21.57 ± 47.64
48 hr	24.34 ± 24.45	20.59 ± 28.73	22.86 ± 52.09

Note. There were no significant main or time effects between the treatment and control groups ($p > .05$).

* = Significantly different from preexercise values ($p < .05$).

PreEx = Preexercise.

Muscle Soreness

In all groups, muscle soreness significantly increased after the exercise protocol for all postexercise periods compared to preexercise values. However, there were no

significant differences at any of the preexercise and postexercise measuring times between treatment and control groups.

Fatigue Index

Preexercise and postexercise changes in fatigue index did not show any significant differences between treatment and control groups.

Vertical Jump

At all measuring times, vertical jump values between treatment and control groups were not significantly different.

Maximal Power

At preexercise and all postexercise measuring times, there were no significant differences between treatment and control groups.

Work Capacity

Postexercise changes in work capacity from baseline values were highly variable. Work capacity at 0 hr and 24-hr postexercise, relative to preexercise, decreased in the control and therapeutic groups. In the prophylactic group however, work capacity increased above preexercise value. At 48-hr postexercise, a reverse of the situation was observed. In spite of these inconsistencies and variabilities, there were no significant differences in work capacity of the treatment and control groups at all measuring times.

Discussion

The present study was designed to measure two conditions of ibuprofen administration to test its effectiveness in reducing muscle soreness and restoration of diminished muscle performance following a damage-inducing exercise. A 2,200-mg prophylactic administration of ibuprofen ingested over 24 hr was intended to ensure peak plasma level of ibuprofen before exercise (Pyne, 1994). The therapeutic administration was given 24-hr postexercise and was hypothesized to prevent further damage from occurring.

The present study failed to demonstrate the effectiveness of ibuprofen in reducing muscle soreness and diminished muscle performance. The findings are in agreement with the conclusions of other studies. Donnelly Maughan, and Whiting (1990) found ibuprofen ineffective in the treatment of DOMS or muscle strength loss at a total dosage of 8,400 mg over 24 hr. Also, Kuipers et al. (1985) concluded that flurbiprofen, an ibuprofen-related drug, was ineffective in the treatment of DOMS. The lack of treatment effect by ibuprofen could be attributed to a combination of at least two factors.

First, Gracely and Kwilosz (1988) have suggested that subjective assessment of pain is associated with a flaw termed as scaling error. From their view, repetitive use of the same soreness scale has the potential to provide subjects with the same category or part of a line or to

remember past specific response. They did not, however, indicate any likelihood of differences in pain measurement due to the flaw. They further argue that since pain sensation has both sensory and physiologic components, a more versatile instrument should be designed to measure DOMS. Contrary to expectations, all three groups in this study had some level of soreness at preexercise time. This likely implies that some bodily discomfort other than soreness was being measured.

Second, the period of soreness evaluation may not have been long enough for any distinct intergroup differences to be observed in subjects in the postexercise period. It is possible that, beyond 48-hr postexercise, the rate of muscle soreness decrease in the treatment group could be faster than in the control group. Even though the prophylactic group ingested an initial 600 mg of ibuprofen 1 hour prior to the treadmill run, no marked effect was observed on muscle soreness and muscle performance.

Whereas dosage has a potential influence on the level of muscle soreness in human subjects, McCormack and Brune (1991) suggested that when administering NSAIDs, the target pain must be intense enough to significantly distinguish perceived soreness of the treatment group from placebo group. To date, studies which tested ibuprofen for its effectiveness in regressing DOMS are inconclusive in their findings. At 500 mg, diclofenac, an ibuprofen-related

NSAID, was effective in reducing soreness at specific spots in humans (Donnelly, McCormick, Maughan, Whiting, & Clarkson, 1988). Hasson et al. (1993) administered a total of 1,200 mg/day of ibuprofen in a study and found it effective in reducing both muscle soreness and diminished muscle performance.

Additional factors that could possibly cause lack of treatment effect are the exercise protocol employed, duration of exercise bout and dosage/duration interaction of drug administration. As evidence, Hasson et al. (1993) found ibuprofen effective in reducing both muscle soreness and diminished muscle performance. Subjects performed step-ups in which lowering the body was done on one leg to induce muscle soreness. Because this movement is not as common in human motion as running exercise, it may have caused greater damage than from the 30-min downhill running utilized in the present study. This was supported from the lack of difference between preexercise and postexercise values of the fatigue index and work capacity.

Exercise duration poses a critical factor in determining extent of damage. Kuipers et al. (1985) had subjects perform eccentric cycling for 30 min at 80% of maximal oxygen uptake. Later, subjects performed the same activity for 45 and 60 min. In subjects who performed for 30 min, histological examination did not show any inflammation. Subjects in the 45-min and 60-min exercise

bouts groups however, showed inflammation. Similarly, Schwane and Armstrong (1983) noticed pronounced enzyme release, a marker of sarcolemma damage, in subjects who performed downhill running for 45 min but not after 30 min. Since the present protocol lasted only 30 min, it is possible that the duration and/or intensity were not enough to induce sufficient tissue damage.

Dosage/duration interaction of drug administration seems a more reasonable factor to consider in attempting to establish lack of treatment effect of a drug. Considering dosage or duration of treatment alone there is seemingly little certainty about what impact each factor could exert on DOMS and diminished muscle performance. It is therefore reasonable to concede that a more versatile factor would be a combination of the two. Credence to this view is given in a study by McLatchie et al. (1985). The study was designed to justify the benefits for the use of NSAIDs in injuries which when left untreated would eventually heal. In the 3-day study, 144 sport persons who had sustained Grade 1 (mild stretching of the ankle ligament with no instability) or Grade 2 injury (moderate sprain with incomplete tear and mild instability) were randomly assigned to ibuprofen groups, 600 mg/4 daily; 1200 mg/2 daily and placebo ibuprofen group. Subjects were assessed at Day 1, Day 3, and Day 7 for joint tenderness, muscle movement, overall severity, and match fitness. Even though the results

indicated significant treatment effect in both active treatment groups, the 4 times daily group was superior. Relating to the findings, this suggest that the dosage utilized in the current study was insufficient to reduce soreness.

Results from the vertical jump, fatigue index, maximal power, and work capacity tests fluctuated considerably from preexercise values. This is very misleading, yet, does not conceal probable reasons underlying the lack of treatment effect on performance. In the present study, vertical jump in the respective groups declined from preexercise values at all postexercise times which implies that the exercise bout had affected performance. Supportive of this, two subjects randomly chosen had increased thigh circumferences at 24-hr postexercise. The fatigue indexes for the control group at 0-hr and 48-hr postexercise were not significantly lower than preexercise levels.

An interesting phenomenon that this study did not observe is a second loss in force in mice as reported by Faulkner et al. (1993). They reported that the second loss of force in the postexercise period might be related to DOMS. Faulkner et al. (1993) believed that the initial loss in force could be a factor of mechanical injury and fatigue. They also suggested that a second biochemical injury occurs to the muscle as a result of the phagocytic activity at the

site of original damage possibly causing deterioration in the muscle fiber resulting in loss of force.

Perhaps the most surprising findings of this study is a seemingly lack of relationship between the fatigue index and maximal power. In all groups, fatigue index was greater at all postexercise times except for the control group at 0-hr and 48-hr. However, the postexercise increases of the fatigue index did not correspond to the postexercise decrements in maximal power. The correlation coefficients for fatigue and maximal power for the four measuring times were as follows: preexercise ($r = .291$), 0 hr ($r = .959$), 24 hr ($r = .248$), and 48 hr ($r = .138$).

If the exercise protocol in the present study did not cause sufficient damage, then a possible speculation that can be made is that damage which results in ultrastructural disorganization in fiber may be a more potent factor in determining muscle function. The speculation is based on the assumption that the intensity and duration of the exercise protocol were not enough to cause sufficient damage to the muscle fibers even though some level of soreness was experienced, postexercise levels of muscle performance remained higher. It is possible that if the exercise protocol had caused maximal structural damage to the muscles, force production could have been impaired. In

summary, the findings of this study failed to support the capability of ibuprofen in reducing muscle soreness and diminished muscle performance at a dosage of 2,200 mg/24 hr.

CHAPTER V
CONCLUSIONS, IMPLICATIONS, AND RECOMMENDATIONS
FOR FURTHER STUDIES

The purpose of this study was to investigate whether the ingestion of ibuprofen before and after a muscle-damaging activity would reduce exercise-induced muscle soreness. A second purpose was to investigate whether the ibuprofen would greatly cause decrements in diminished muscle performance. Thirdly, the study investigated which of the two modes of drug administration, prophylactic and therapeutic, is superior in reducing soreness and diminished muscle performance.

Conclusions

Based on the results of this study, the researcher concluded that:

1. Ibuprofen at a total dosage of 2,200 mg/day does not diminish DOMS up to 48-hr postexercise.
2. Ibuprofen does not help restore muscle performance more quickly in treatment group than in control group following a 30-min downhill (-10% grade) treadmill run.
3. Prophylactic treatment was not superior to either therapeutic or control in reducing DOMS and restoring muscle performance.

Implications

From the results of this study, it could be suggested that the use of ibuprofen, a common practice among athletes

may not, after all, be beneficial for purpose of reducing DOMS and restoring muscle performance.

Recommendations for Further Study

Based on the results of this study, the following recommendations for repeated studies are made:

1. The use of a larger sample size so as to attain a threshold point where group differences in treatment if any, would be distinct.
2. The employment of a more standardized and objective soreness measuring instrument in assessing muscle soreness.
3. Use of an additional questionnaire that would incorporate both the affective and sensory components to pain (Gracely & Kwilosz, 1988).
4. Use of homogeneous subjects. Test for similarities in physical characteristics in terms of age of age, sex athletic level and skill should be conducted prior to participation (Bar-Or, 1987).
5. Increased dosage and longer period of ibuprofen administration.
6. Extension of postexercise period of testing and data collection must be made to ensure that a point is reached where group difference in response to treatment become distinct.
7. Employment of a more intense and longer duration exercise is recommended.

8. Assessment of muscle performance should be done side-by-side with muscle biopsy in order to measure changes in performance against any possible morphological irregularities and changes in blood variables like creatine kinase. In this way, questionable trends in performance and soreness can be matched against possible instrumental and sampling errors.

REFERENCES

- Abraham, W. M. (1977). Factors in delayed muscle soreness. Medicine and Science in Sports, 9(1), 11-20.
- Abramson, S., Edelson, H., Kaplan, H., Ludewig, R., & Weissmann, G. (1984). Inhibition of neutrophil activation by nonsteroidal anti-inflammatory drugs. American Journal of Medicine, 7(4B), 3-6.
- Abramson, S., & Weissmann, G. (1989). The mechanism of action of nonsteroidal anti-inflammatory drugs: Review. Clinical and Experimental Rheumatology, 7 Supplementary (3), 9163-9170.
- Allessio, H. M. (1993). Exercise-induced oxidative stress. Medicine and Science in Sports and Exercise, 25(2), 218-224.
- Almekinders, L. C. (1993). Anti-inflammatory treatment of muscular injuries in sports. Sports Medicine, 15(3), 139-145.
- Almekinders, L. C., & Gilbert, J. A. (1986). Healing of experimental muscle strains and the effects of nonsteroidal anti-inflammatory medication. American Journal of Sports Medicine, 14(4), 303-308.
- Appell, H. J., Soares, F. M., & Duarte, J. A. R. (1992). Exercise, muscle damage and fatigue. Sports Medicine, 13(2), 108-115.
- Armstrong, R. B. (1984). Mechanism of exercise-induced delayed onset muscular soreness: A brief review. Medicine and Science in Sports and Exercise, 16(6), 529-538.
- Armstrong, R. B. (1990). Initial events in exercise-induced muscle injury. Medicine and Science in Sports and Exercise, 22(4), 429-435.
- Armstrong, R. B., Ogilvie, R. W., & Schwane, J. A. (1983). Eccentric exercise-induced injury to rats skeletal muscle. Journal of Applied Physiology: Respiration, Environment, and Exercise Physiology, 54(1), 80-93.
- Bansil, C. K., Wilson, G. D., & Stone, M. H. (1986). Role of prostaglandins E and F₂ alpha in exercise induced delayed muscle soreness. Medicine and Science in Sport and Exercise Supp.

- Bar-Or, O. (1987). The Wingate anaerobic test: An update on methodology, reliability and validity. Sports Medicine, 4, 381-394.
- Bobbert, M. F., Hollander, A. P., & Huijing, P. A. (1986). Factors in delayed onset muscular soreness of man. Medicine and Science in Sports and Exercise, 18(1), 75-81.
- Boots Laboratories Leaflet. (1994, February 23). IBU Ibuprofen tablets, USP. (Available from Boots Pharmaceutical, Inc. Lincolnshire, IL 60069 USA).
- Brendstrup, P. (1962). Late edema after muscular exercise. Archives of Physical Medicine, 43, 401-409.
- Braga, P. C., Biella, G., & Tiengo, M. (1987). Effects of tenoxicam on nociceptive thalamic neuronal firing in arthritic rats. Drugs Under Experimental Clinical Research, XIII, 389-398.
- Brune, K. (1982). Prostaglandins, inflammation and anti-inflammatory drugs. European Journal of Rheumatology and Inflammation, 5, 335-349.
- Brune, K., Beck W. S., Geisslinger, G., Menzel-Soglowek, S., Peskar, B. M., & Peskar, B. A. (1991). Aspirin-like drugs may block pain independently of prostaglandin synthesis inhibition. Experientia, 47(3), 257-261.
- Brune, K., Menzel-Soglowak, S., & Zeilhofer, H. U. (1992). Differential analgesic effects of aspirin-like drugs: Review. Drugs, 44, 52-58. Abstract.
- Cicccone, C. D., Leggin, B. G., & Callamaro, J. J. (1991). Effect of ultrasound and trolamine salicylate phonophoresis delayed-onset muscle soreness. Physical Therapy, 71(9), 666-678.
- Clarkson, P. M., Litchfield, P., Graves, J. Kirwan, J., & Byrnes, W. C. (1985). Serum creatine kinase activity following forearm flexion isometric exercise. European Journal of Applied Physiology, Occupational Physiology, 53, 368-371.
- Clarkson, P. M., & Trembley, I. (1988). Exercise-induced damage, repair, and adaptation in humans. Journal of Applied Physiology, 65(1), 1-6.

- Cobb, C. R., deVries, H. A., Urban, R. T., Luekens, C. A., & Bagg, R. J. (1975). Electrical activity in muscle pain. American Journal of Physical Medicine, 54, 80-87.
- deVries, H. A. (1966). Quantitative electromyographic investigation of the spasm theory of muscle pain. American Journal of Physical Medicine, 45, 119-134
- Donnelly, A. E., Maughan, R. J., & Whiting, P. H. (1990). Effect of Ibuprofen on exercise-induced muscle soreness and indexes of muscle damage. British Journal of Sports Medicine, 24(3), 191-195.
- Donnelly, A. E., McCormick, K., Maughan, R. J., Whiting, P. H., & Clarkson, P. M. (1988). Effects of a non-steroidal anti-inflammatory drug on delayed onset muscle soreness and indices of damage. British Journal of Sports Medicine, 22, 35-38.
- Duncan, C. J., & Jackson, M. J. (1987). Different mechanisms mediate structural changes and intracellular enzyme afflux following damage to skeletal muscle. Journal of Cell Science, 87, 183-188.
- Ebbeling, C. B., & Clarkson, P. M. (1989). Exercise-induced muscle damage and adaptation. Sports Medicine, 7, 207-234.
- Famaey, J. P. (1982). Free radicals and activated oxygen. European Journal of Rhomboidal Inflammation, 5(4), 350-359.
- Faulkner, J. A., Brooks, S. V., & Opiteck, J. A. (1993). Injury to skeletal muscle fibers during contractions: Conditions of occurrence and prevention. Physical Therapy, 73(12), 911-921.
- Ferreira, S. H. (1972). Prostaglandins, aspirin-like drugs and analgesia. Nature: New Biology, 240, 200-203.
- Friden, J. (1984). Muscle soreness after exercise: Implication of morphological changes. International Journal of Sports Medicine, 5, 57-66.
- Fritz, V. K., & Stauber, W. T. (1988). Characterization of muscle injured by forced lengthening. II. Proteoglycans. Medicine and Science in Sports and Exercise, 20(4), 354-361.

- Garret, Jr., W. E. (1990). Muscle strain injuries: Clinical and basic aspects. Medicine and Science in Sports and Exercise, 22(4), 436-443.
- Glick, J. M. (1980). Muscle strains. Prevention and treatment. Physician Sports Medicine, 8, 73-77.
- Gracely, R. H., & Kwilosz, D. M. (1988). The descriptor differential scale applying psychophysical principles to clinical pain assessment. Pain, 35, 279-288.
- Grisham, M. B., & McCord, J. M. (1986). Chemistry and cytotoxicity of reactive oxygen metabolites. In A. E. Taylor, S. Matalon, & P. A. Ward (Eds.), Physiology of oxygen radicals (pp. 1-18). Bethesda, MD: American Physiological Society.
- Hasson, S. M., Daniels, J. C., Divine, J. G., Niebuhr, B. R., Richmond, S., Stein, P. G., & Williams, J. H. (1993). Effect of ibuprofen use on muscle soreness, damage, and performance: A preliminary investigation. Medicine and Science in Sports and Exercise, 25(1), 9-17.
- Hasson, S. M., Wible, C. L., Reich, M., Barnes, W. S., & William, J. H. (1992). Dexamethasone iontophoresis: Effect on delayed muscle soreness and muscle function. Canadian Journal of Sports Science, 17(1), 8-13.
- Herring, S. A. (1990). Rehabilitation of muscle injuries. Medicine and Science in Sports and Exercise, 22(4), 435-456.
- Hoppeler, H. (1986). Exercise-induced ultrastructural changes in skeletal muscle. International Journal of Sports Medicine, 7, 187-204.
- Hough, T. (1992). Ergographic studies in muscular soreness. American Journal of Physiology, 7, 76-92.
- Jenkins, R. R., & Goldfarb, A. (1993). Introduction: Oxidant stress, aging and exercise. Medicine and Science in Sports and Exercise, 25(2), 210-212.
- Kibler, W. B. (1990). Clinical aspects of muscle injury. Medicine and Science in Sports and Exercise, 22(4), 450-452.

- Komi, P. V., & Viitasalo, J. T. (1977). Changes in motor unit activity and metabolism in human skeletal muscle during and after repeated eccentric and concentric contractions. Acta Physiological Scandinavia, 100, 246-254.
- Kuipers, H., Keizer, H. A., Verstappen, F. T. J., & Costill, D. L. (1985). Influence of a prostaglandin-inhibiting drug on muscle soreness after eccentric work. International Journal of Sport Medicine, 6(6), 336-339.
- McLatchie, G. R., Allister, C., MacEwen, C., Hamilton, G., McGregor, H., Colquhoun, I., & Pickvance, N. J. (1985). Variable schedules of ibuprofen for ankle sprains. British Journal of Sports Medicine, 19, 203-206.
- McCully, K. K., & Faulkner, J. A. (1985). Injury to skeletal muscle fibres of mice following lengthening contractions. Journal of Applied Physiology, 9, 119-126.
- McCormick, K., & Brune, K. (1991). Dissociation between the anti-nociceptive and anti-inflammatory effects of the nonsteroidal anti-inflammatory drugs: A survey of the anagelsi efficacy. Drugs, 41(4), 533-547.
- MacIntyre, D. L., Darlene, R., & McKenzie, D. C. (1995). Delayed muscle soreness the inflammatory response of muscle injury and its clinical implications. Sports Medicine, 2(1), 24-40.
- Newham, D. J. (1988). Consequences of eccentric contraction and the relationship to delayed onset muscle pain. European Journal of Applied Physiology, 57, 353-354.
- Newham, D. J., Mills, K. R., Quigley, B. M., & Edwards, R. H. T. (1983). Pain and fatigue after concentric and eccentric muscle contractions. Clinical Science, 64, 55-62.
- Newham, D. J., Jones, D. A., & Clarkson P. M. (1987). Repeated high force eccentric exercise: Effects on muscle pain and damage. Journal of Applied Physiology, 3, 1381-1386.
- Newham, D. J., McPhail, G., Mills, K. R., & Edwards, R. H. T. (1983). Ultrastructural changes after concentric and eccentric contractions of human muscle. Journal of Neurological Science, 61, 109-122.

- Onyanagui, Y. (1978). Inhibition of superoxide anion production in non-stimulated guinea pig peritoneal exudate cells by anti-inflammatory drugs. Biochemical Pharmacology, 27, 777-782.
- Patton, J. R., & Duggan, A. (1987). An evaluation of tests of anaerobic power. Aviation and Space Environmental Medicine, 58, 237-242.
- Pyne, D. (1994). Regulation of neutrophil function during exercise. Sports Medicine, 17(4), 245-258.
- Rodenburg, J. B., Bar, P. R., & De Boer, R. W. (1993). Relationship between muscle soreness and biochemical and functional outcome of eccentric exercise. Journal of Applied Physiology, 7(6), 2976-2983.
- Ryan, A. J. (1969). Quadriceps strain, rupture, and charlie horse. Medicine and Science In Sports, 1, 106-111.
- Sargeant, A. J., & Dolan, P. (1987). Human muscle function following prolonged eccentric exercise. European Journal of Applied Physiology, 56, 704-711.
- Schwane, J. A., & Armstrong, R. B. (1983). Effect of training on skeletal muscle injury from downhill running in rats. Journal of Applied Physiology, 55(1), 969-1983.
- Talag, T. S. (1973). Residual muscle soreness as influenced by concentric, eccentric, static contraction. Res. Quarterly, 44, 458-469.
- Tiidus, P. M., & Ianazzo, C. D. (1983). Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. Medicine and Science in Sports and Exercise, 15(6), 461-465.
- VanHelder, W. P. (1992). Medical treatment of muscle soreness. Canadian Journal of Sports Sciences, 17(1), 74.
- Weiss, S. J. (1989). Tissue destruction by neutrophils. New England Journal of Medicine, 320, 365-376.
- Weissmann, G. (1982). The biochemistry of inflammation: Rheumatoid arthritis and anti-inflammatory drugs. European Journal of Rheumatology & Inflammation, 5(4), 366-381.

Willoughby, D. A., & Sedgwick, A. (1982). Mediators of inflammation. European Journal of Rheumatology & Inflammation, 5(4), 360-365.

APPENDIX A
SUBJECTS INFORMED CONSENT

SUBJECTS INFORMED CONSENT

I hereby consent to take part in a research directed by Atta Kofi Osei, School of Physical Education and Leisure Services, University of Northern Iowa. I understand that other persons may assist this individual or be associated with him.

I understand that:

1. This research is to investigate the effect ibuprofen may have on exercise-induced muscle pain.
2. This will be my part in the research: to have soreness perception, muscle power and muscle capacity assessed daily for three days; complete a 30-minute running on a treadmill declined to -10%, on the first visit; orally ingest the required dosage of ibuprofen as stipulated by the study; stay away from massaging or ingestion of additional dosage of pain killers. These procedures would be conducted in the Human Performance Laboratory, 207 West Gymnasium, UNI.
3. Participation in the research will require approximately a total 3 hours of my time.
4. My participation is voluntary. I am free to stop participating at any time. If I do not volunteer or if my participation is ended for any reason by the researcher or me, this will involve no penalty or loss of any of the benefits to which I am entitled.

5. I will be told of any significant new information that might affect my willingness to take part in this research.

6. These benefits can be expected from the research: I will receive useful information about my age adjusted (220-Age) maximum heart rate; muscle power and anaerobic capacity, extent to which my sore muscle could sustain itself under resistance, rate of my body's response to NSAIDs and extent of treadmill running at -10% declination.

7. My participation will expose me to the following risks: Leg instability to moderate intensity exercise for 30 minutes can cause leg fatigue and general tiredness. Dizziness and loss of consciousness, although rare, are likely results of explosive cycle ergometer pedalling. Standard safety procedures will be used, and qualified personnel will be present for assistance during exercise testing.

8. There are no other satisfactory procedures to obtain information needed for this research.

9. The following steps will be taken to protect the confidentiality of my identity and the data I have contributed: All data will be filed in locked rooms. Only averages will be reported and the data will not be linked with any individual in published or oral reports. The investigator will keep an identifier key, but the subjects may request the identity link to his data to be destroyed.

Otherwise, the data will be maintained in storage for an indefinite period.

10. I will receive no monetary or other compensation for my time.

11. I further allow Atta K. Osei and his assistants or associates to perform the procedures referred to above, report their findings to government agencies, funding agencies, manufacturers, or scientific bodies, and to publish their findings. My questions about this research have been answered. If I do have any further questions, I am to contact Atta K. Osei at the school office in West Gymnasium, 273-2141 or 277-4629, 2113 Main Street, Cedar Falls, IA 50613 or Dr. Fred Kolkhorst, 207B West Gymnasium, 273- 5921. Questions regarding this research project and the rights of research subjects also may be directed to the Human Subjects Coordinator, University of Northern Iowa, 273-2748. I am fully aware of the nature and extent of my participation in this project as stated above and the possible risks involved. I hereby agree to participate in this project. I acknowledge receipt of a copy of this consent statement.

Signature of Volunteer

Date

Printed Name of Volunteer

APPENDIX B
SUBJECTS NOTATION SHEET

SUBJECTS NOTATION SHEET

NAME:

CODE:

NOTE IF YOU DID ANY OF THE FOLLOWING:

1. Did you involve in any vigorous physical activity ?

Yes No

If yes, what type and how long ?

2. Did you take additional pain killers or any drug to
suppress pain within the study period ? Yes NoIf yes, please indicate the type of medication dosage, and
day1 (testing day)

2 (recovery day 1)

3 (recovery day 2)

APPENDIX C
SUBJECTS PRETEST QUESTIONNAIRE

SUBJECTS PRETEST QUESTIONNAIRE

NAME:

CODE:

(Circle one)

1. Do you have any known heart diseases? Yes No

2. Do you have asthma? Yes No

3. Do you have any physiological/physical impairment that will make it difficult to:

*run on the treadmill? Yes No

*pedal the ergometer? Yes No

*jump the wall? Yes No

If yes, describe: _____

4. Are you currently taking medications? Yes No

If yes, please list all medications and why you are taking them. _____

5. Please describe any regular physical activity you are involved in.

*jogging (mileage)

*sport (name) duration and number of times per week.

*aerobic exercises/dancing; duration and number of times per week.

6. Have you ever taken any pain killers? (i.e. ibuprofen, advil, tylenol, anacin, aspirin). Yes No

If yes, when was the last time?

What dosage?

How often do/did you take the said drug?

2*2 daily 1*2 daily 2*1 daily other (explain)

7. Have you ever taken any substance in order to regress pain in physical performance? Yes No

If yes, please explain. _____

APPENDIX D
SUBJECTS PERSONAL DATA

SUBJECTS PERSONAL DATA SHEET

Name: _____ Code #: _____

Age: _____ Reach: _____ Weight: _____

	Soreness	Vet. Jump	Fatigue	Max. Power	Work Output
PreEx	_____	_____	_____	_____	_____
0 hr	_____	_____	_____	_____	_____
24 hrs	_____	_____	_____	_____	_____
48 hrs	_____	_____	_____	_____	_____

APPENDIX E
VISUAL ANALOGUE SCALE

VISUAL ANALOGUE SCALE

<u>Numerical Rating</u>	<u>Description of Feelings</u>
0	No Soreness
1	Dull feeling of soreness
2	Light continuous soreness
3	More than light soreness
4	Annoying soreness
5	Severe soreness
6	Intolerable soreness

APPENDIX F
RAW DATA

RAW DATA
PRE-EXERCISE

	Grp	Age	Wgt (Kg)	Snss	A'bic Vert.	Power Jump (Inch)	An'bic Ftg (%)	Wrk Max.Pwr (W)	Capacity Work (Kj)
Subj 1	1	20	85	0	13	31	922	25152	
2	1	20	81	0	18	26	937	24937	
3	1	20	77	2	17	45	936	21270	
4	2	22	76	1	13	31	867	22723	
5	3	19	74	0	14	16	1050	29572	
6	3	19	86	0	22	30	978	24775	
7	3	22	81	0	25	30	863	22372	
8	2	24	78	0	14	34	845	20540	
9	1	19	77	0	15	19	815	22225	
10	1	19	69	0	21	10	768	21666	
11	2	22	76	0	13	21	791	20926	
12	2	18	74	0	17	20	751	20248	
13	3	18	75	0	21	23	720	20030	
14	3	21	85	1	12	29	776	19769	
15	1	21	98	0	17	38	1037	24684	
16	1	22	81	1	18	20	942	25024	
17	2	20	67	0	22	34	767	19551	
18	2	22	62	1	16	07	784	22992	
19	3	19	68	0	16	26	820	22101	
20	3	20	80	0	19	41	789	19104	
21	2	18	74	0	21	22	889	23929	

Snss = Soreness
A'bic = Aerobic
An'bic = Anaerobic
Grp = Group
Wgt = Weight
Ftg = Fatigue
Pwr = Power
Jmp = Jump
Vert = Vertical

RAW DATA
IMMEDIATE POST-EXERCISE

Subj	Snss.	A'bic Power		Anaerobic Work Capacity		
		Vert. Jmp (Inch)	Ftg (%)	Mx. Power (W)	Work (Kj)	
1	2	13	34	875	23067	
2	3	17	15	780	21468	
3	3	17	26	806	21128	
4	4	13	26	885	23719	
5	2	13	26	1167	21157	
6	4	22	13	853	23825	
7	1	23	32	895	21666	
8	1	14	18	788	21800	
9	2	16	08	750	22201	
10	1	20	24	880	22227	
11	2	11	20	826	22812	
12	4	16	13	742	21038	
13	3	20	42	839	20681	
14	3	11	39	675	17597	
15	2	15	18	958	26631	
16	3	15	05	792	23127	
17	4	21	66	840	19779	
18	2	16	15	933	25497	
19	1	11	37	741	19683	
20	1	18	32	739	18003	
21	1	20	13	822	23179	

RAW DATA
24-HOUR POST EXERCISE

		Snss	A, bic Power Vet. Jump (inch)	Anaerobic Work Ftg (%)	Work Power (W)	Capacity Work (Kj)
Subj	1	4	13	28	943	25266
	2	4	15	13	907	25534
	3	5	16	45	954	21522
	4	3	13	37	985	24175
	5	2	12	31	1197	30783
	6	4	21	34	929	23719
	7	2	23	29	676	16593
	8	2	14	35	793	19689
	9	1	17	31	945	23807
	10	2	20	41	946	24405
	11	0	12	19	794	21671
	12	5	16	27	885	22662
	13	4	19	37	869	21905
	14	4	12	34	815	20701
	15	2	18	61	688	16594
	16	3	15	27	1054	26853
	17	2	21	37	910	22466
	18	2	16	19	890	25407
	19	4	12	43	831	20541
	20	3	18	51	797	17226
	21	2	20	31	917	23283

RAW DATA
48-HOUR POS-EXERCISE

	Snss	A,bic Power Vet Jump (inch)	Anaerobic Work Ftg (%)	Capacity Power (W)	Work Work (Kj)	
Subj	1	3	13	10	875	24973
	2	3	15	07	898	26060
	3	6	16	41	955	22521
	4	3	14	34	987	24479
	5	2	11	28	1213	31850
	6	3	21	25	943	24702
	7	1	23	30	945	23800
	8	2	14	26	861	22412
	9	3	16	26	893	23340
	10	3	20	36	943	24270
	11	0	12	20	749	20365
	12	4	15	28	874	22606
	13	1	20	32	935	23876
	14	4	10	22	810	21775
	15	1	18	39	779	20852
	16	5	17	28	1100	28342
	17	3	22	36	830	20319
	18	1	16	62	705	16452
	19	4	10	37	632	15163
	20	4	20	37	785	18824
	21	2	21	50	701	17464