CHAPTER 3
REVIEW OF LITERATURE

This chapter will include a review of books and articles that are relevant to the mechanisms of Delayed Onset Muscle Soreness, previously employed treatment interventions, physiological adaptation to training and impacts on performance, and soreness inducing exercise. The literature review will contain information on the following topics (1) Damage to the sarcomere and connective tissue, swelling and inflammation with an emphasis on the underlying mechanism of DOMS, (2) Creatine kinase, (3) Lactate dehydrogenase, (4) Eccentric muscle actions, (5) Exercise, massage, cryotherapy, ultrasound, and anti-inflammatory drug therapy as methods of treatment intervention, (6) Adaptation regarding both the neurological and musculoskeletal systems, (7) Performance deficits, (8) Soreness inducing exercise, and (9) Other relevant material as it is related to the proposed study.

3.1 UNDERLYING MECHANISMS OF DOMS

Damage to the sarcomere and connective tissue

The most supported theory to date describing the cause of DOMS was developed by Hough in the early 1900's. This theory is based on the supposed structural damage to the sarcomere and surrounding connective tissue during high intensity eccentric loading of the muscle. The muscle activity which causes the most soreness and damage at the structural level is eccentric activity. The initiating event may be related to high specific tension produced by the muscle during eccentric actions which results in shearing of the myofibrils (Macintyre et al., 1995). Armstrong, (1984), in an earlier study also stated that it seems probable that this increased tension per unit area could
cause mechanical disruption of structural elements in the muscle fibers themselves or in the connective tissue that is in series with the contractile elements. One reason eccentric actions cause more damage to muscle than concentric actions is because fewer motor units are recruited during eccentric exercise, and therefore a smaller cross-sectioned area of muscle is activated to handle the same load as would be handled in a concentric action (Clarkson and Sayers, 1999). Thus, in eccentric actions the force is distributed over a smaller cross-sectional area and therefore, the tension per active cross-sectional area is greater (Armstrong, 1984). The reason tissue disruption occurs appears to be related to the fact that fewer motor units are activated during an eccentric, compared to a concentric action for a comparable amount of work. Since the weight is the same, more tension is placed on fewer muscle fibers resulting in disruption of the involved tissue.

Clarkson and Sayers. (1999) hypothesized that certain sarcomeres may become overextended and pull apart due to the stress placed on them by the lengthening actions of the muscle. Because some sarcomeres may be stronger than others, weaker sarcomeres are unable to maintain tension as the fiber lengthens, thus possible structures are left to provide support. Although a considerable amount of information exists on the possible underlying mechanisms of DOMS, the true mechanism underlying this phenomenon remains unclear (Smith, 1991; Kuipers, 1994; Macintyre et al., 1995). Hough first described in 1902, "When an untrained muscle makes a series of actions against a strong spring, soreness frequently results which cannot be regarded as a phenomenon of pure fatigue " and indicated that DOMS has its origin in some sort of rupture within the muscle itself.
Stauber. (1996) suggested that DOMS is due to a complex set of reactions involving disruption of the muscle fiber and connective tissue. There may be two aspects of muscle tissue damage that need to be differentiated from each other: (a) direct myofiber damage, and (b) connective tissue or fascial damage. Direct myofiber damage occurs during activity or can be observed immediately after the activity is completed. Connective tissue damage is less well defined but certainly involves collagen and other extracellular matrix components as well as the interconnections between adjacent muscle cells (Stauber, 1989). Kuipers. (1994) indicated that muscular overuse is associated with structural damage of the contractile elements and is reflected in DOMS. Unaccustomed eccentric exercise has previously been shown to induce disruption within the myofibrillar and connective tissue structures of skeletal muscle (Newham et al., 1983).

The mechanical micro trauma after eccentric muscle action results in myofiber damage as well as alterations to the extracellular matrix (Stauber et al., 1990), both of which may lead to inflammation and pain. The theory of intrinsic muscle damage associated with eccentric muscle actions has been supported by many studies using muscle biopsies to document both myofiber damage and connective tissue damage. The myofiber damage consisted of hyper contracted sarcomeres, Z line streaming, and refractory fibers that could be observed immediately after exercise when no pain was present (Stauber, 1996). The mechanism of injury from eccentric exercise is due to the increased tension per individual cross bridge causing mechanical disruption of the ultra structural elements within the muscle fibers such as the Z line and contractile filaments (Macintyre et al., 1995). During eccentric activity, the force developed is approximately twice that developed during isometric actions, but the total number of strongly bound cross bridges during eccentric activity is only about 10% greater than
during an isometric action. Macintyre. (1995) suggests that this high tension may result in streaming of the Z lines. Streaming and smearing of Z lines, focal loss of Z lines and extension of the Z line into the A band have been observed immediately following eccentric exercise (Lieber et al., 1991).

It has been suggested that eccentric exercise preferentially damages sarcomeres that are nearing the end of the cycle of growth and replacement (Newham et al., 1987). As these sarcomeres are replaced with newer, stronger fibers, the eventual outcome may be stronger muscle fibers.

### 3.2 DIRECT EVIDENCE FOR ECCENTRIC CONTRACTION-INDUCED MUSCLE DAMAGE

Among the three types of contraction (isometric, concentric and eccentric contraction), it has been shown that the probability of contraction induced muscle damage is greater during an eccentric contraction. Baker et al. (2006) found that, in rats, exercise involving eccentric contractions (15 sets of 10 stretch-shortening-cycles) can induce significantly more skeletal muscle damage compared to isometric contractions exercise of the same stimulation duration.

One of the earliest studies to show direct evidence of skeletal muscle damage, in humans, following eccentric exercise was performed by Friden et al. (1983). In that study, 12 subjects performed a bout of eccentric bicycle (60 rpm for 30min), Open muscle biopsies were obtained from 5 subjects prior the exercise, immediately after, 3 days, and 6 days following the eccentric exercise bout. In ultra structural level, disturbances of the cross-striated pattern were observed originating from the myofibrillar Z-band which showed marked streaming, broadening and in some cases total disruption. The disturbances were found in every skeletal muscle or myofibril up
to 3 days after exercise and in one tenth of the fibers 6 days following the exercise. It is important to note here that the disturbances were observed only in ultra structural level because the overall fiber morphology as seen with light microscope was absolutely normal with no morphological fiber abnormality in any of the sections. The ultra structural disturbances, however, as seen with electron microscope were predominantly localized in type 2 fibers. The study concluded that eccentric exercise with regard to muscle fiber type, selectively influenced the fine structure of the contractile apparatus.

In agreement with Friden et al. (1983), ultra structural disturbances were observed by Hortobagyi et al. (1998). Twelve subjects performed 100 eccentric contractions with the quadriceps muscle and repeated the same exercise 2-weeks after the first exercise bout. Needle muscle biopsies were obtained from the distal portion of vastus lateralis before each exercise bout; two and seven days post the exercise bouts. Two days following the first exercise bout, electron microscopy revealed substantial disorganization of the myofilaments, widening of Z-lines, and Z-line streaming whereas seven days later no abnormality was observed. The same disturbances were observed two days after the second exercise bout (two-weeks later) while no disturbances were observed seven days after the second exercise bout.

Muscle damage, as a result of an unaccustomed eccentric exercise, is a process involving an initial and a secondary damage. The initial damage is predominantly a mechanical damage to individual sarcomeres while the secondary damage involves an acute inflammatory response and is a consequence of the initial injury. One of the earliest indices of muscle damage is the cytoskeleton disruption and particularly the loss of desmin intermediate filaments. Lieber et al. (1996), found that the desmin...
intermediate filament is lost within 15 minutes after a single bout of eccentric contraction (5, 15, or 30 minutes resulting 150-900 eccentric contractions), in Zealand White Rabbits. The results showed that control animals (passively stretch or isometrically exercised) had normal fiber morphology while in the eccentrically exercised group the fibers appeared large, rounded, partially or totally negative for desmin and unable to exclude plasma fibronectin.

In agreement with Lieber et al. (1996) results, Friden et al. (1998) also found that the earliest manifestation of muscle damage is the loss of desmin intermediate filament, in New Zealand White rabbits that were exercised eccentrically (900 eccentric contractions in 30 minute period).

Beyond the loss of desmin, another very early index of muscle damage is the loss of sarcolemma integrity. Lovering et al. (2004) found that 15 minutes after a single eccentric contraction the sarcolemma integrity was lost in 55% of the fibers. A single eccentric contraction was performed on the Tibialis Anterior muscle of Male Sprague–Dawley rats at 900°/s throughout a 90°-arc of motion. To evaluate sarcolemma integrity Evans Blue Dye was injected intra peritoneally 24 hours before the animal were scarified. An interesting finding was that all fibers positive for Evans Blue Dye had discontinued or lost dystrophin organization. The study concluded the occurrence of selective dystrophin vulnerability after a single eccentric contraction.

The earliest abnormality observed by Komulainen et al. (1998), was discontinuous dystrophin staining while at the same time no alterations occurred in the sarcolemma integrity and the staining of desmin and actin of Male Wistar rats that were subjected to 240 forced lengthening contractions.
Even though, the above mentioned studies, it is not clear which is the earliest event; it is most probably the loss of sarcolemma integrity. This is based on the fact that sarcolemma disruption will result in the alteration of intracellular calcium. Loss of $\text{Ca}^{++}$ homeostasis and particularly, intracellular elevation of free cytosolic $\text{Ca}^{++}$ will activate a number of $\text{Ca}^{++}$-depended proteolytic and phospholipolytic pathways which respectively degrade contractile and membrane phospholipids leading to myofiber damage and necrosis (Armstrong et al., 1991).

After the initial damage to myofibers, phagocytes and macrophages infiltrate the damaged myofibers and initiate an acute inflammatory response. The inflammatory response, that usually commences 1 to 3 days after the initial injury, is the primary reason for the secondary injury. The infiltrated phagocytic cells remove the damaged myofibrils, cytosolic organelles, and sarcolemma leading to total muscle fiber degeneration and thereby the initial damage is aggravated.

### 3.3 DIRECT EVIDENCE AGAINST ECCENTRIC CONTRACTION - INDUCED MUSCLE DAMAGE

The above mentioned studies clearly documents the existence of the eccentric contraction induced- muscle damage, in animals. In addition, no contradictory findings have been published considering the effectiveness of eccentric exercise to induce muscle damage. In humans, however, there is significantly less direct evidence supporting the contraction-induced muscle damage. Available evidence supports that the human’s skeletal muscles are not prone to eccentric contraction induced-muscle damage; or at least the severity of damage is less than that observed in animals.

Yu et al. (2002) found no loss of desmin, no sarcolemma damage; and any myofiber degeneration, inflammation or necrosis after a single bout of downstairs running,
eccentric bicycling or downhill treadmill running. In that study the subjects were exercised eccentrically by either downstairs running (from the tenth floor to the ground floor for 15 times), eccentric bicycling (60 rpm for 30 minutes), or downhill treadmill running (8° decline for 45 minutes). The results showed that all muscle biopsies taken before and after each exercise type at various time points (up to 8 days after the exercise) showed normal fiber appearance with no loss of desmin, no sarcolemmal damage; and any myofiber degeneration, inflammation or necrosis. The authors concluded that, in humans, eccentric exercise does not cause loss of desmin nor myofibre necrosis.

Crameri et al. (2004), in agreement with Yu et al. (2002), showed no muscle damage in seven of eight subjects that performed a single bout of 210 maximum voluntary eccentric contractions. Needle muscle biopsies were collected before, 2, 4, and 8 days after the exercise bout and stained with hematoxylin and eosin, desmin, fibronectin, and CD68 (macrophages marker). In seven of eight subjects the result showed, no evidence of gross myofiber damage, no myofiber necrosis, no positive staining for CD68, no loss of desmin staining and no fibronectin staining inside the muscle fibers. Only one subject showed myofiber necrosis, fibronectin positive myofibers and loss of desmin staining, all of which commenced at day 2 and were not fully recovered at day 8.

3.4 THE INFLAMMATORY RESPONSE

Inflammation is a generalized response of the body to any kind of tissue injury. This injury may be the result of chemical, thermal or mechanical stimuli. Clinical studies have attempted to find evidence that supports the theory that tissue inflammation is
the underlying mechanism of DOMS (Armstrong et al., 1983 and Schwane et al., 1983).

The sensation of muscle soreness that is evident between 24 and 48 hours post exercise can be associated with an acute inflammatory response as suggested by Smith. (1991). It is hypothesized that morphological injury occurs after the initial exercise bout. During the first few hours after the onset of injury, white blood cells (WBC), specifically neutrophils are attracted to the injured site. Similarly, it was found out that neutrophils are speculated to be the first cells to infiltrate damaged muscle fibers. Damage to muscle fibers results in an inflammatory response that causes entry of fluid and cells into the damaged tissue. Increased fluid produces the characteristic swelling after injury (Clarkson and Sayers, 1999). However the role of neutrophils in the damage and repair process is unknown (Macintyre et al., 1995). The inflammatory response plays a key role in removal of damaged proteins before regeneration ensues. Following degradative processes, some macrophages may then play a role in muscle repair (Tidball, 1995). Macrophages are the predominant type of inflammatory leukocyte after the first 12 hours post-exercise and are the principal removers of cellular debris. Macrophages act as phagocytes and remove cellular debris in damaged tissue. Macrophages also regulate the consequent repair process and appear when muscle regeneration begins (Tidball, 1995).

Approximately 8-12 hours after exercise a second shift of WBC (macrophages) begin to infiltrate the damaged area. These cells further penetrate the damaged tissue and also synthesize chemical substances extremely important in the healing process (Smith, 1992). It is believed that one important substance - prostaglandin E produced
by the macrophage is central in orchestrating the inflammatory process and also a potent pain producing agent. Prostaglandin E also plays a major role in healing.

The synthesis is inhibited by aspirin and other nonsteroidal anti-inflammatory drugs, suggesting that the use of these drugs might reduce DOMS. The wide use of anti-inflammatory drugs may actually have an adverse effect on muscle healing (Stauber, 1996). Although prostaglandins have been documented to be involved in tissue degradation following injury, they are also involved in tissue growth (Stauber, 1996). Macintyre. (1996) have demonstrated that there is a greater presence of white blood cells in exercised muscle in the first 24 hours after eccentric exercise, indicating that acute inflammation is one of the underlying reactions of exercise-induced muscle injury. They support the hypothesis that there is more than one mechanism underlying exercise-induced muscle injury, firstly that of mechanical injury and fatigue and then the subsequent events of the inflammatory response. It has been shown that exercise and exercise-induced muscle injury can trigger mobilization of some aspects of the inflammatory response but the specific events initiating this are not known. Different mechanisms have been suggested to be associated with the soreness perception. Since the cellular response does not parallel the symptoms, other factors must contribute to the soreness perception. This may explain the findings that anti-inflammatory drugs fail to alleviate the soreness rating (Kuipers, 1994). Friden, et al. (1986) suggested that increased tissue pressure from tissue swelling may be associated with the soreness perception.

It is hypothesized that prostaglandins may increase the sensitivity of free nerve endings and that movement causes a sudden increase in the already elevated tissue pressure, leading to pain (Smith, 1991). Edema associated with DOMS does not
produce a significant increase in intramuscular pressure at rest in a compliant compartment. However, movement or palpation may exaggerate even small increases in pressure and thus provides a mechanical stimulus for “pain” receptors already sensitized by prostaglandins.

Thus, the combination of increased pressure and hyper sensitization produces the sensation of DOMS indicating that acute inflammation is the generalized response of the body to acute tissue injury. The main purpose of this response is to promote healing, an event critical to survival. Since the body responds to all forms of acute tissue injury by activating the inflammatory response, there is no reason to believe that a separate response has evolved to deal with injury incurred during unaccustomed eccentrically based exercise. However, if inflammation is not present in exercised damaged muscle, then agents and modalities that have been demonstrated to assist in the process of pain reduction and tissue healing in inflammatory conditions might not be effective in the treatment or prevention of DOMS (Stauber et al., 1990).

### 3.5 ECCENTRIC MUSCLE ACTION

A muscle's ability to produce tension throughout all or part of a joint's range of motion is known as a dynamic action. A muscle can produce dynamic tension by either shortening or lengthening. If the joint motion is in a direction opposite the normal (gravitational) force and the tension produced by the muscle exceeds the external resistance encountered, the action is shortening (or concentric) in nature. If the joint motion is in the direction of the normal force and the external resistance encountered exceeds the muscle's ability to generate tension, the action is lengthening (or eccentric) in nature (Perrin, 1993). Eccentric muscle actions involve the lengthening of a muscle while the muscle produces tension (Smith, 1992).
Generally, eccentrics or negatives are involved in lowering, braking and shock absorption movements usually in the direction of gravity (Stauber, 1989). When a muscle lengthens as it is being stimulated to develop tension, the action is eccentric. The eccentric tension acts as a braking mechanism. For example, eccentric tension occurs in the elbow flexors during the elbow extension or weight-lowering phase of a curl exercise. Without the presence of eccentric tension in the muscles, the weight would drop uncontrolled because of the force of gravity (Hall, 1991). Most movements involve a negative component, but it is not always easily identifiable. In fact, there is no standard scientific method for identifying which muscles perform an eccentric action in any given skill. Researchers, therefore, merely examine how the muscle behaves during a particular movement. Also, it should be noted that in some movements such as landing from a jump, the negative action is more accentuated than in others.

3.6 SORENESS INDUCING EXERCISE

Many studies have been successful in inducing DOMS using an exercise session consisting of strictly eccentric actions (Hasson et al., 1989; Donnelly et al., 1992; Hasson et al., 1993; Smith et al., 1994; Weber et al., 1994; Tiidus and Shoemaker, 1995; Craig et al., 1996; Giamberardino et al., 1996; Gulick, et al., 1996; Paddon Jones and Quigley, 1997), or a combination of concentric actions and eccentric actions (Donnelly et al., 1990; Dennegar and Perin, 1992; Isabell et al., 1992; Rodenburg et al., 1994; Bourgeois et al., 1999). These studies support the theory that unaccustomed eccentric exercise if applied at a sufficient intensity will elicit DOMS.

Successful methods to induce DOMS using standard exercise equipment have also been previously reported. Using the weight of a concentric one repetition maximum
(IRM), Weber et al. (1994) were successful in inducing soreness in the elbow flexors by having the subject perform 10 repetitions of lowering the weight (eccentric action) over a five-second count. The weight was passively returned to the starting (flexed elbow position) by the investigator. Once the subject was no longer able to control the descent of the weight the set was considered finished, if the subject was able to successfully perform the 10 repetitions it was considered a complete set. At this point the weight was lowered by one half of a plate, and the regimen continued. Statistical analyses showed significant increases of perceived soreness when baseline measurements were compared with the 24 and 48 hour measures.

Using a similar protocol, Craig et al. (1996) using each subjects concentric 1RM exhausted the elbow flexors by three bouts of eccentric exercise to exhaustion. The subjects clearly showed increases in pain levels and tenderness in all groups as a result of DOMS induction. Using 110% of concentric 1RM, subjects performed 8 sets of 8 eccentric seated dumbbell curls using the elbow flexors. Each eccentric action was performed over a 3-second period with an assistant returning the weight to the starting position. Perceived soreness peaked for all subjects at 48 hours and was significantly different than the baseline measures. By 120 hours, no significant soreness remained relative to the pre-test. In another study, the exercise session used to induce soreness consisted of 6 sets of 10 repetitions of unilateral knee extension (concentric and eccentric) at an intensity of 80-85% of the baseline concentric 1 RM (Bourgeois et al., 1999). Significant DOMS was present at 24 and 48 hours as compared with baseline.
3.7 INTERVENTIONS

3.7.1 Exercise

There have been few studies to date that employ exercise as a possible intervention in an attempt to prevent or delay the negative impacts of DOMS. Gentle exercise involving the affected muscles can be useful as a rehabilitative tool (Macintyre et al., 1995). The use of physical activity has long been a standard suggestion to aid in recovery from intense exercise bouts (Weber et al., 1994). This recommendation was initially based on observations by Hough (1902), who noted a decrease in soreness with continued actions of the sore muscle. The above statement is supported by Hasson (1989) who stated that the success of an exercise intervention was related to a reduction in intramuscular pressure through the muscle pump action.

Another possibility that may play a role in the reduction of muscle soreness with exercise is the endorphin release. These endogenous opiates (endorphins and encephalin), are secreted by neurons in the brain and spinal cord with the overall effect of inhibiting the transmission of pain (Armstrong, 1984; Starkey, 2004). It has been suggested that endorphin release is increased during exercise, so exercise-enhanced endorphin secretion could potentially provide an analgesic effect, minimizing the effect of DOMS.

Hasson et al. (1989), while examining the effects of a high velocity therapeutic exercise regimen on DOMS and muscular performance found that high speed maximal concentric muscle actions were effective in decreasing muscle soreness and facilitating return of normal muscular performance. Dependent variables (performance measures) included Maximum Voluntary Action (MVC) by the quadriceps, Peak Torque (PT) by the quadriceps at high resistance, and Total Work (TW) by the
quadriceps. These variables had significantly less decrease from baseline for the experimental group when compared to the control group. Following therapeutic Exercise intervention (TE), the TE group was significantly higher than the control group for all muscle performance variables measured at 48 hours. At 48 hours post muscle soreness exercise bout, the Soreness perception index (SPI) of quadriceps was also significantly less for the experimental versus control. These results suggest that high-speed voluntary muscle actions are effective in decreasing DOMS and facilitating return of normal muscle performance. Factors affecting DOMS are presently believed to be related to the processes of inflammation and muscle edema, which follow tissue injury. The tissue disruption caused by eccentric lengthening actions cannot be reversed instantaneously, but the production of prostaglandins can be affected (Hasson et al., 1989).

This is the strategy utilized when patients are given non steroidal anti-inflammatory agents. If prostaglandin production is retarded prior to large amounts of fluid accumulation in the injured area, muscle soreness should be minimal. Hasson. (1989) believed that the end process of tissue damage is inflammation and fluid accumulation, which are the major causes for the development of the soreness. Research has demonstrated (Friden, 1986) that concentric actions resulted in much lower intramuscular pressures than eccentric actions. The mechanism for decreasing muscle soreness following high speed muscle actions has been proposed to be related to decreased inflammation, or decreased fluid compartmental pressures, or both. Further research to examine the effects of high speed voluntary muscle actions on the inflammatory process and intramuscular compartmental fluid pressures after a bout of eccentric exercise is recommended.
Isabell et al. (1992) indicated that the pattern of change for the exercise group appeared favorable with respect to soreness levels as compared to the control group. The patterns of change that their subjects demonstrated somewhat support the argument of exercise as an effective method of reducing DOMS. The exercise group had the smallest decreases in ROM and strength and the smallest increases in soreness and Creatine Kinase (CK) levels. The individuals in the exercise group performed mild full ROM elbow flexion and extension exercises, with only the gravitational pull on the hand and arm providing resistance. The repetitions were performed continuously during a 20-second period and then rested for 40 seconds. This exercise/rest interval was continued for a total treatment time of 15 minutes. Treatment was applied at 0, 2, 4, 60, 24, 72, and 96 hours post-exercise.

Donnelly. (1992) and Weber. (1994), in contrast to Hasson. (1989), conducted studies and found that after a heavy bout of unaccustomed eccentric exercise, exercise employed immediately and 24 hours after, did not significantly reduce/alter muscle soreness, strength or force generation. After inducing soreness of the non-dominant elbow flexors, upper body ergometry was performed at 60 rpm for a workload of 400 kg. M/min in a counterclockwise direction using the upper extremity as the point of reference. Although the intervention did not produce any statistically significant differences between groups, the author did indicate that having an athlete perform light concentric actions post eccentric exercise bout may prove to be the most effective method of diminishing the effects of DOMS (Weber et al., 1994). The exercise intervention that was employed by Donnelly et al. (1992) was at 50% of the maximum torque produced during the initial heavy eccentric bout. The exercise intervention was performed on a Biodex isokinetic dynamometer and was set to cease movement if the torque exceeded the 50% pre set point so it was clearly sub maximal.
in nature. The exercise intervention was employed 24 hours after the initial heavy eccentric bout. The intervention did not appear to alter muscle soreness strength or flexibility (Donnelly et al., 1992). Alternatively, rest would appear to be less effective. As there is no soreness involved with rest, this is what most people choosing and allowing the DOMS to produce maximum effects on performance.

3.7.2 Massage

Another treatment intervention that has been studied in an attempt to decrease the negative effects of DOMS is therapeutic massage. Massage has been used to assist recovery from muscle fatigue in the sports medicine field for many years but uncertainly exists about its effectiveness (Callaghan, 1993; Smith, 1994; Tiidus 1997). The mechanisms suggested for the apparent efficacy of massage include increase in circulation and lymph flow and decrease in muscle tension. Deep friction or vigorous massage can evoke vascular changes similar to those of inflammation. The treated area is marked by increased blood flow, histamine release and an increased temperature. When performed properly, massage can increase venous and lymphatic flow that assists in the removal of edema. Massage increases lymphatic flow and movement of fluid depends on forces outside of the system. Such factors as gravity, muscle action and massage can affect the flow of lymph which assists in the reduction of edema (Prentice, 1994). If edema, swelling and inflammation are significant factors in muscle soreness sensation, massage may be able to affect soreness by reducing their presence in affected muscles (Tiidus, 1997). It is not immediately apparent how massage may be able to physiologically affect the time course or severity of the post-exercise muscle damage/repair process.
In a study conducted by Tiidus et al. (1995), subjects, one hour after undergoing a heavy eccentric bout of exercise, had one leg manually massaged for 10 minutes by a Registered Massage Therapist (RMT). The RMT used both superficial and deep effleurage strokes beginning at the knee and moving proximally covering approximately 75% of the thigh area of the treatment leg. This treatment was repeated at 24 and 72 hours post heavy eccentric bout, the perceived level of soreness tended to be reduced in the massaged leg 48-96 hours post-exercise although it was not significant. However, it was concluded that massage was not an effective treatment modality for enhancing long-term restoration of post-exercise muscle strength.

A systematic review conducted by Ernst. (1998) found that many studies conducted with respect to DOMS and manual massage formed positive associations and suggested that post-exercise massage may alleviate symptoms of DOMS and also massage therapy may be a promising treatment for DOMS and definitive studies are warranted. Although massage may be a promising intervention for the reduction of DOMS, its effectiveness has not been demonstrated convincingly.

Rodenburg et al. (1994) attempted to combine three interventions to study the effects on subjective DOMS pain scores. After participating in a 15-minute warm-up and a stretching session subjects underwent an eccentric exercise session. Fifteen minutes following the exercise session, the intervention group underwent a massage that was performed by a physiotherapist. This combination did prove to reduce some of the negative effects of eccentric exercise. However, the subjective scores in the treatment group were lower than the control group. The treatments used in this study may be useful to reduce DOMS and functional restrictions due to sports activities, but also may be useful during normal daily activities in which performance may be hindered
by DOMS and large decreases in maximal force and range of motion after exercise. However, since the effects are only small, it has to be questioned how much effort should be taken to do a warm-up, stretching exercises and massage, to reduce DOMS.

There is very little evidence (Tiidus, 1997) that manual massage has any significant impact on the recovery of muscle function following exercise or on any of the physiological factors associated with the recovery process. In addition, the types and durations of massage employed by therapists varies based on athlete and therapist preference and not on scientific data. The time and money spent by sports teams on the provision of sports massage may be misplaced. Because little evidence exists which supports manual massage as an effective therapeutic modality in affecting recovery of muscle strength and performance following damage and DOMS reduction, the use of massage for these purposes was questioned.

3.7.3 Ultrasound

The effectiveness of electrotherapeutic modalities has been investigated as an effective treatment intervention to decrease the negative symptoms associated with DOMS. Ultrasound is an electrotherapeutic modality that has been used to decrease the symptoms of inflammation, pain and edema, and to increase the rate of healing of damaged tissues. A localized warming of the tissues may occur that can lead to an increase in the extensibility of tissues such as scar tissue (Starkey, 2004; Prentice, 1994). These effects may contribute to the reported analgesic action of ultrasound and the reduction of edema (Craig, 1999) and ultrasound might be expected to accelerate the inflammation and healing processes while reducing the pain associated with DOMS. Little conclusive evidence supporting the positive effects of ultrasound has been reported.
Subjects in a study conducted by Craig. (1999) were randomly divided into four separate treatment groups: control, placebo, low-dosage pulsed ultrasound, or high-dosage pulsed ultrasound. DOMS was induced in the elbow flexors through repeated eccentric exercise until exhaustion. There were no significant differences in the groups when compared for elbow flexion strength and resting angles or pain. The study provided no convincing evidence to support the use of pulsed ultrasound therapy in the management of DOMS within the parameters of this study. Ultrasound demonstrated no significant benefits in terms of subjective pain relief or range of movement.

Similarly, (Ciccone et al., 1991) attempted to determine the effect of salicylate phonophoresis as compared with ultrasound used alone on DOMS. Phonophoresis is a technique in which ultrasound is used to drive a topical application of a selected medication into the tissues (Prentice, 1994). Medications commonly applied through phonophoresis most often are either anti-inflammatory or analgesics. This comparison was conducted so that any ultrasound effects could be distinguished from the pharmacologic effects of trolamine salicylate (an anti-inflammatory-analgesic cream) when both are used together in the form of salicylate phonophoresis. Salicylates are compounds that evoke a number of pharmacologic effects, including analgesia and decreased inflammation caused by a reduction in prostaglandins (Prentice, 1994). In a group of 10 subjects, findings suggested that the use of ultrasound alone increased the symptoms associated with DOMS. When ultrasound was applied to a group of 10 subjects with the trolamine salicylate, the same increases were not observed. These results suggest that the ability of ultrasound to increase the mechanisms underlying the DOMS may be offset by the pharmacologic activity of the trolamine salicylate.
3.7.4 Cryotherapy

Cryotherapy, the application of a cold modality to the human body (Starkey, 2004) is widely regarded as an effective, easy to use, and inexpensive treatment modality for traumatic soft-tissue injury (Paddon-Jones and Quigley, 1997). If one considered the body's inflammatory response to tissue injury, it may be expected that the use of cryotherapy would be effective in decreasing the symptoms associated with DOMS. The local effects of cold application include vasoconstriction and a decrease in metabolic rate and pain transmission. The most beneficial effect of cold application during an acute injury is to decrease the need for oxygen in the area being treated. A cold environment decreases cellular metabolic rate, consequently decreasing the amount of oxygen required by the cells to survive. By reducing the number of cells killed by a lack of oxygen, the degree of secondary hypoxic injury is limited. Since fewer cells are damaged from secondary hypoxic injury, smaller amounts of inflammatory substances are released into the area (Prentice, 1994).

Several investigators (Isabell et al., 1992; Gulick et al., 1996; Paddon-Jones and Quigley, 1997) have provided evidence to support the theory that cryotherapy is not effective in reducing the symptoms associated with DOMS. During one such study, after muscle soreness was induced, the subject using ice ball applied ice massage. An ice ball is formed by freezing water in a plastic or Styrofoam cup. During application of the ice ball, the ice melts and the edges of the cup can be peeled away to expose more ice. This application process allows the therapist/subject to massage the body part with the ice while holding the bottom of the cup. Subjects massaged the entire length of their biceps using circular and stroking motions. The treatment was applied at 0, 2, 4, 6, 24, 72, and 96 hours post-exercise. Each treatment continued for 15
minutes (Isabell et al., 1992). The therapeutic use of ice was not effective in reducing the symptoms of DOMS. Though not statistically significant, the author suggested that the patterns in the data may indicate that ice application may be contraindicated in the treatment of DOMS. They noted that the ice group had the highest peak soreness at rest scores, the highest serum CK levels, and the lowest low peak total ROM of all the groups.

Similar to the previous study described, results from a study performed by Paddon-Jones and Quigley. (1997) indicate that cryotherapy does not facilitate recovery of strength or reduce the seventy of DOMS following eccentric exercise. Following the eccentric exercise protocol, subjects had the eccentrically exercised arm placed in an ice-water immersion bath. A total of five, 20-minute immersions were performed; each separated by 60-minute rest intervals. The first ice-water immersion occurred immediately following the completion of the eccentric exercise session. It was concluded that muscular soreness and strength loss occur in spite of attempts to use cryotherapy.

Gulick et al. (1996) employed a treatment intervention of ice massage following the soreness inducing exercise session. They received an ice massage for 20 minutes. The ice cup was moved in circular motions dong the length of the exercised muscle group. The ice group generated less isometric force after treatment and provided transient relief from acute muscle soreness but was not successful in abating DOMS. It was concluded that ice massage was not effective in abating signs and symptoms of DOMS.

In contrast to the above-mentioned studies with regard to cryotherapy as an intervention method, Denegar and Perrin. (1992) produced positive results. Forty-
eight hours post exercise bout, subjects underwent a 20-minute ice application using a plastic bag filled with crushed ice. Immediately following the ice application perceived pain scores and concentric and eccentric average torques were recorded. These values were compared to the baseline values taken 48 hours earlier and to values taken immediately prior to the ice application. Results indicated that ice was effective in treatment of the pain associated with DOMS. Although these results appear favorable, it would be expected that perceived soreness would decrease immediately following an application of ice over a 20-minute period. The analgesic effect of the ice application disappears within hours (Starkey, 2004), and after this time the soreness returns. The effectiveness of the ice therapy should have been tested 3-4 hours after the cryotherapy to determine the long-term effect.

3.7.5 NSAIDS (Non-steroidal anti-inflammatory drugs)

Anti-inflammatory drugs have been studied as a possible successful treatment intervention with regard to DOMS. Ibuprofen and Naproxen are two anti-inflammatory drugs commonly used in the treatment of soft-tissue injuries (Donnelly et al., 1990). One study appears to have been successful in using these NSAIDs (Hasson et al., 1993) while others (Donnelly et al., 1990; Bourgeois et al., 1999) were not.

Results from a study conducted by Donnelly et al. (1990) suggest that ibuprofen is not an appropriate treatment for delayed onset muscle soreness and a specified dosage was administered to the participants 30 minutes prior to the exercise bout of downhill running and every 6 hours up to 72 hours post-exercise. There were no significant differences between the drug group and the placebo group with respect to subjective soreness or isometric strength.
Using similar outcome measures Bourgeois et al. (1999) indicates that NSAID administration did not alter muscle force deficit, nor perceived muscle pain post-exercise. Naproxen was administered at a specified dose both before and after resistance exercise yielding no significant results.

In contrast to the above, Hasson et al. (1993) conducted a study which considered the effect of ibuprofen on muscle soreness, damage, and performance. Ibuprofen was once again administered prophylactically and therapeutically at specified dosages. Outcome measures were taken at 24 hours and 48 hours and compared to baseline. Results from the study indicate that a prophylactic dosage of ibuprofen does decrease muscle soreness perception and may assist in restoring muscle function.

3.8 ADAPTATION

It has been well documented that after one bout of eccentric exercise the muscle becomes more resistant to damage for a time period of up to six weeks (Ebbeling and Clarkson, 1990; Clarkson et al., 1992; Stauber, 1996). It should be emphasized that the best prevention for DOMS is regular exercise. It is recognized that repetition of an activity that includes eccentric muscle actions leads to protection from repeated injury. Eccentric effort is known to produce rapid training effects on muscles which last for a long time, 4-6 weeks even after a single exercise (Giamberardino, 1996).

In a study conducted by Newham, et al., (1987), the authors state that one interesting feature is the rapidity with which the pain and muscle damage are reduced or abolished with repeated exercise. In their previous work, release of creatine kinase (CK), an indirect marker of muscle damage, and muscle tenderness were measured after one bout of exercise involving actions at 50% maximum force. When the
exercise was repeated one week later both pain and CK release were much reduced. This training effect was found to last approximately six weeks, indicating a considerable and long lasting adaptation of the muscles to eccentric exercise.

It is widely accepted that DOMS will only occur after the first few bouts of an exercise program and therefore training acts in a preventive fashion to reduce muscle damage and soreness (Macintyre et al., 1995). It has been shown (Friden et al., 1983), that there is less muscle fiber damage after training implying that there is a protective effect associated with regular physical exercise. There is evidence that the pain and stiffness experienced after eccentric actions are a consequence of shortening of the non contractile material that is arranged in parallel with the contractile material. This may be a response to some form of damage to the connective tissue, and if so the training could have caused some adaptation of this tissue. It is suggested that during the healing process muscle and connective tissue are strengthened and thus more resistant to subsequent damage.

Possible explanations for the adaptation may include that there may be a change in the pattern of motor unit recruitment. Training, and thus, adaptation may cause a change in the order of motor unit recruitment such that either susceptible fibers are spared on the second and subsequent occasions or more fibers are recruited and the force-fiber ratio is reduced (Newham et al., 1987). There may also be some adaptation in the muscle fibers such that they become more resistant to the fatiguing and damaging effects of eccentric exercise. This might be seen as a change in the strength and contractile properties of the muscle. Kuipers, (1994) is in agreement and stated that the adaptation can probably be attributed to a change in recruitment as well as to an increase in connective tissue thickness and strength.
Ebbeling et al. (1990) demonstrated that an adaptation response had taken place within the affected muscle prior to full recovery and restoration of muscle function following the initial eccentric exercise bout. It can be concluded that complete recovery and restoration of muscle function is not a prerequisite for adaptation following eccentric exercise. Further research in this area is required to provide more conclusive evidence regarding the mechanisms of adaptation.

3.9 MUSCLE SORENESS PERCEPTION-VISUAL ANALOG SCALE (VAS)

An investigator needs to score one or more aspects (features) of pain in order to constrict a profile of a pain state. The VAS is perhaps the most familiar approach (Turk and Melzack, 1992). VAS is primarily used to gather information about internal feelings, perceptions, or sensations that are difficult to measure on scales with predetermined intervals (Lee and Kieckhefer, 1989). Their use is common in determining perceived pain levels of individuals experiencing pain related to DOMS. The VAS that most clearly delineate extremes (ie., the worst imaginable pain, the most intense pain imaginable) are 10-15 cm in length, and have been shown to have the greatest sensitivity and are the least vulnerable to distortions or biases in rating. Another advantage of VAS is the potential increase in sensitivity of subject responses. Since respondents are not restricted to arbitrary, previously quantified intervals, they may make as fine discrimination as they wish. This also has the potential for enhancing respondent satisfaction (Lee and Kieckhefer, 1989).

The VAS provides a simple, efficient and minimally intrusive measure of pain intensity that has been used widely in clinical and research settings where a quick index of pain is required and to which a numerical value can be assessed. The VAS
are relatively simple so that the majority of patients as well as experimental subjects can easily respond to these scales (Price et al., 1983).

The patient is required to place a mark on the 10 cm line at a point that corresponds to the level of pain intensity he or she presently feels. Lee et al. (1989) agrees that a typical scale is composed of a horizontal line with end anchor. For ease of calculation, 100-mm lines are most common. Horizontal lines are less subject to respondent error attributable to the angle at which the scale is viewed.

The distance in centimeters from the low end of the VAS to the patients mark is used as a numerical index of the severity of pain. VAS in which line length is the response continuum, have been reported as valid and reliable measures for the intensity of pain (Price et al., 1983). Also, the verbal anchor points on VAS cm are modified to delineate the different dimensions of pain so that although subjects use the same type of scale, they could respond differentially to multiple dimensions of the pain.

There is much evidence supporting the validity of the VAS for pain intensity. Such scales demonstrate possible relations to other self-report measures of pain intensity. They are sensitive to treatment effects and are distinct from measures of other subjective components of pain. The lack of bias or distortion in VAS ratings may be partly the result of the fact that, in the studies described, subjects were instructed carefully about how to use the VAS and the entire range of stimulus intensities to be used was gradually presented beforehand. Directions to subjects should be clear, concise, and specific. Directions should be followed immediately with an example of how to use VAS, so that misunderstandings can be promptly identified and corrected (Lee and Kieckhefer, 1989).
The visual analog scale (VAS) has been used successfully by several investigators to assess DOMS. It is a commonly used assessment tool when pain levels are reported. Gulick et al. (1996) used a VAS that consisted of a 10 cm line with descriptors at each end. At the left end there was the number zero with the descriptor "no soreness at all", and at the right end there was the number ten with the descriptor "soreness as bad as it could be". Each subject placed an x dong a 10 cm line to describe the amount of muscle soreness he/she was presently experiencing. Data was collected pre induced DOMS, 24, 48, and 72 hours after treatment. Peak soreness at 48 hours was reported with a mean value of approximately 3.5 cms. The present study is similar to Gulick et al. (1996), with the exception of an added assessment time of 96 hours post DOMS induction.

Similar to the above study (Bourgeois et al., 1999) used a 100 mm VAS using descriptor terms of "no discomfort whatsoever" (0 mm) to "maximal discomfort (100 mm). The VAS was given to each of the subjects at 24 and 48 hours post exercise. This scale was used to determine the degree of discomfort in the quadriceps muscle group after the exercise stimulus.

Another study which used the VAS to assess DOMS was performed by (Ciccone et al., 1991). The VAS consisted of a continuous horizontal line 150 mm in length, with anchor points of "no soreness" and "worst possible soreness" at the left and right ends respectively. Subjects indicated the amount of soreness by placing a slash somewhere on the VAS. Relative soreness was then calculated by measuring the distance of the slash from the left end of the VAS.

Although the length of the Line used was 150 mm, as opposed to the 100 mm lines in the above studies, the descriptors remain similar. Soreness increased from negligible
levels on day one to appreciable levels by day two. Groups reported peak levels of soreness between days two and three, reaching levels that ranged between 29% and 45% of the maximum possible rating (150 mm). Soreness began to decline toward baseline values (0 mm) by day four of the study.

3.10 PERFORMANCE DEFICITS

The functional outcome of DOMS, as demonstrated by Hough. (1902) was reduction in muscular force output immediately after the exercise and lasting several days. The performance deficit preceded the onset of muscle soreness, which began the day after the exercise session. The decrease in muscle performance is due to a reduction in the muscle's intrinsic ability to produce force (Stauber, 1996). Researchers have shown that performance deficits can last for more than 5 days following a bout of eccentric exercise (Kowell et al., 1993). Muscular performance impairment has previously been described in terms of loss of maximum voluntary force production (Newham et al., 1987). Indirect evidence of exercise-induced muscle damage is associated with the development of muscle soreness and a prolonged loss of strength and range of motion (Saxton et al., 1995).

Assessing the relationship between development of soreness and the loss of muscle strength suggests that there is little or no relationship between the two (Ebbeling and Clarkson, 1989). Exercised muscles exhibited a dramatic 35% loss of strength, on the day following the exercise. Even on the tenth post exercise day the muscles had recovered only to about 70% of their control strength. At this time, soreness had fully disappeared for most of the subjects, confirming our impressions that the decrease in contractile strength was not simply an artifact of pain limited effort by the subjects during the force measurements (Howell et al., 1993). It has taken as long as a week
for eccentric torque at high velocities to recover. After fatiguing eccentric exercise, there is a decrease in maximal force production observed as early as one hour after the exercise (Macintyre, et al., 1996). As the loss of force production of muscles is observed almost immediately following an eccentric exercise session, the onset of perceived soreness would appear to have little effect on this performance deficit as it is not normally observed until 24 to 48 hour post-exercise session.

Individuals who experience severe DOMS after performing unaccustomed eccentric exercise show significant reductions in eccentric strength, as well as concentric and isometric strength (Ebbeling and Clarkson, 1989; Smith, 1992). This reduction in strength is most pronounced immediately after exercise, with little restoration at 34 and 48 hours and recovery may be slow, lasting from eight to ten days. The loss of strength/power impacts performance. On days two, three and four after beginning football practice, an inverse relationship exists between the height of a vertical jump and the intensity of DOMS induced through a variety of activities (Smith, 1992).

3.11 THE EFFECT OF VARIOUS MODES OF EXERCISE ON DOMS AND SERUM MARKERS OF SKELETAL MUSCLE DAMAGE

Schwane et al. (1983) were the first researchers to study how running at different grades influence muscle soreness. They hypothesized that running down on incline, in which muscles are experiencing eccentric contractions, caused greater DOMS than running on level ground. Seven male subjects, aged 19 to 21 years, volunteered for this study. All were physically active on a regular basis, but for the duration of the study subjects were asked to abstain from activity other than that performed during the experiment. Each subject performed three treadmill tests: a maximal oxygen
consumption (VO2 max) test, a run on level ground (0% grade), and a run at a 10% grade downhill, both for a 45 minute duration.

DOMS was assessed via plasma levels of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) before exercise, 5 minutes, 24, 48, and 72 hours after the run at each grade. Six of the subjects waited six to seven days, and the seventh subject waited twenty-three days between test one and two, and fourteen days between test two and three. Muscle soreness was also assessed using the Abraham scale. There was significant muscle soreness in the gluteal, quadriceps, anterior leg, and posterior leg muscles, but not the hamstrings after the downhill run, while there was no significant muscle soreness after the run on level ground. There was no significant change in LDH levels, but there was a 351% increase of CPK within 24 hours of running downhill. The researchers also wanted to determine if there was a relationship between DOMS and inflammation, identifiable by the abundance of white blood cells found in the blood, but no significant relationship was found.

According to their results, it has been found that running downhill perpetuated muscle soreness and caused CPK levels to significantly increase. They suggested that DOMS and increased levels of CPK were results of changes in muscle tissue structure due to the eccentric contractions performed. They also proposed that DOMS could have been the result of muscles being used in an unaccustomed way. Koskinen, et al (2001) conducted a study assessing proteins released into the bloodstream from the breakdown of extracellular matrix components, after exercise-induced muscle damage. The researchers induced muscle damage in fourteen healthy, physically active male volunteers, aged 20-32 years. The men were separated into two groups, with both running at a 10% grade downhill on a treadmill for 45 minutes, but one group
performed the run at room temperature (22 degrees Celsius) and the other in the cold (5 degrees Celsius). Each subject had venous blood samples taken pre-run, immediately post-run, and one, four, and seven days after the run.

The results showed that CK increased for both groups, each peaking after one day, some reaching four to six times the normal levels, although the participants who ran in the cold had significantly higher levels of CK. Therefore, those who performed in the cold temperature experienced more muscle damage. It was presumed that running in a cold environment decreased work capacity and therefore CK levels increased because it took increased work and involvement of additional muscle fibers.

Kyrolainen et al. (2000) examined metabolic measurements of seven triathletes including one woman and six men, aged 29 ± 5 years, who had volunteered to run a marathon. Blood samples were taken from each subject’s ulnar vein before, during, and after the marathon to evaluate levels of serum creatine kinase (S-CK). Finger pricks were taken to determine blood lactate (B-La) levels. The presence of CK in the blood reached its peak two to four days following intense exercise. By day six, CK had returned back to normal levels. Muscle damage may be experienced if there is an increase in the levels of metabolites in the skeletal muscles or a mechanical disturbance of the muscle cell.

Kobayashi et al. (2005) was interested in the long term effects of marathon running performed by recreational runners. They wanted to determine how muscle damage affected muscle enzymes creatine kinase and lactate dehydrogenase. Fifteen healthy male volunteers took part in this study (mean age 43.5 years). All had participated in endurance training for an average of 50 km/week for 9 years. The study was performed at a temperature of between 8 and 10 degrees Celsius. The participants
were not allowed to eat or drink the morning of the race. Before beginning the marathon (flat terrain), 10 ml venous blood samples of CK and LDH were taken from an ante cubital vein to determine baseline levels. During the race the subjects had fluids available at all times. Ten milliliter blood samples were taken 5 minutes post-race. The baseline levels of serum CK and LDH were within the normal range, 163 U/L and 323 U/L, respectively. Subsequent samples were taken on days 1, 2, 3, 7, and 14. This method of sampling did not allow for maximal and minimal values of CK and LDH values during the recovery period. CK levels tripled, on average. One day post-run the values had increased fifteen times baseline, then decreased, taking about one week to return to baseline. LDH levels doubled post-race and from there on decreased. It took about two weeks for LDH to return to normal levels. The subjects participated in very little running during the first week post-run due to muscle soreness. Kobayashi et al. (2005) concluded that CK and LDH levels increased in recreational runners after running a marathon, therefore inferring that the run caused the muscle damage. Within two weeks the runners were back to baseline and normal training intensity.

Pachalis et al. (2005) examined differences in muscle damage between high and low intensity eccentric exercise. The method of exercise was isokinetic quadriceps eccentric exercise. Twelve male subjects, aged 20 to 22 years, participated, all who had no prior resistance training experience. The extent of muscle damage experienced by each subject was assessed pre- and post-exercise, 24, 48, 72, and 96 hours after the exercise. Each subject performed two isokinetic quadriceps eccentric exercise sessions on each leg, with a 2 week rest period between sessions. This time period had previously been shown to recover muscle damage markers back to baseline. Each subject participated in high intensity (HI) and low intensity (LI) eccentric exercise,
with equal work performed doing both HI and LI. Plasma CK and DOMS increased for both HI and LI eccentric exercise, however, there was only a significant difference between CK levels 24 hours post-exercise. The results indicated that HI and LI exercise had similar effects on muscle damage suggesting that muscle damage is caused more by the volume of exercise as opposed to the intensity of exercise.

Clarkson et al. (2006) had 203 subjects aged 18 to 40 years perform two sets of 25 maximal eccentric contractions of the elbow flexor on a modified preacher curl bench. There was a five minute rest period between each set. Each contraction lasted for three seconds with a subsequent twelve second rest period. The subjects had to maximally contract their elbow flexors to resist the downward movement of a lever controlled by a tester. Subjects were encouraged to remain hydrated pre- and mid-exercise. Prior to the exercise and 4, 7, and 10 days post-exercise, blood samples were drawn to assess renal function and muscle damage until blood markers had returned to baseline levels.

Peak CK levels were averaged and reported to be 6420 UL-1 on day 4, 2100 UL-1 on day 7, and 311% above baseline on day 10. The normal CK range was reported to be 24-195 U/L. One-hundred eleven subjects had CK levels above 2000 UL-1 by day 4 which has been used to diagnose myositis (an inflammatory muscle disease due to a viral, bacterial, or parasitic infection). Fifty-one subjects (25%) had greater than 10,000 UL-1 which has been associated with rhabdomyolysis (a condition in which there is excessive amount of myoglobin in the urine—due to muscle damage) (Huether and McCance, 2004) The normal range for LDH was reported to be 118-273 U/L with the greatest range being 86 - 1608 ± 292 U/L.
LDH levels increased significantly with a strong correlation existing between CK and LDH levels ($r = .95$). Renal function was not impaired as a result of the elevated blood markers. It was concluded that elevated CK levels, as a result of exercise induced muscle damage, did not influence renal function.

The purpose of Sayers et al. (2000) study was to determine if activity affected the recovery of muscle of the elbow flexors after eccentric exercise. Twenty-six college aged men aged 20.8 + 0.9 years who were not weight trained participated in this study. Each subject was randomly assigned to one of three groups: (1) immobilization—in which the subjects’ non-dominant arm was placed in a cast immediately post-exercise for the duration of the treatment, (2) control, and (3) light exercise—which consisted of 50 bicep curls with 5 lb weights, therefore increasing activity level without causing additional muscle damage. The duration of the study was fifteen days. For three days baseline measurements of relaxed arm angle (RANG), flexed arm angle (FANG), maximal isometric force (MIF), and perceived muscle soreness (SOR), were taken. For four days subjects performed 50 maximal eccentric contractions of their non-dominant elbow flexor using a modified preacher curl. The purpose of this exercise was to induce muscle damage. As the subject applied maximal resistance to perform a curl, an investigator used a lever to cause the elbow to extend. The subjects were asked to resist the lever, therefore resisting the action to extend their elbow. The fifty contractions were split into two sets with five minute rest periods, each contraction lasting approximately three seconds with a twelve second rest period between contractions. Treatment was followed by eight days of recovery measurements.
No significant difference was found among groups over time for baseline RANG, FANG, or MIF, though there was a significant main effect for time in RANG, FANG, and MIF from before to immediately after eccentric exercise. There was no significant group by time interaction in pre- and post-exercise RANG and MIF, but there was in exercise FANG. The immobilization group had a significantly greater average FANG than the control group immediately post exercise. Subjects from all groups experienced an extended decrease in RANG, an increase in FANG and SOR, and a decrease in MIF in the days subsequent to exercise. Throughout the eight day recovery period no significant group by time interaction was found in RANG or FANG, though there was a significant group by time interaction for MIF and SOR. Activity or inactivity did not influence the recovery of the joint angle.

No significant difference was found in MIF among the groups until the fourth day of recovery, in which the light exercise group had a significantly greater MIF than the control group. From the fifth to the eighth day of recovery the immobilization and light exercise group experienced significantly greater MIF than the control group. No significant difference was found between the immobilization and light exercise group throughout the duration of recovery. Immediately post treatment (day 5) the control group had the greatest recovery of baseline MIF of 76%, as opposed to the light exercise group (71%), and the immobilization group (68%). Within the next seven days the control group experienced a plateau in recovery, whereas the immobilization and light exercise group improved considerably. No group returned to baseline levels for MIF, but 90% improvement was experienced by the immobilization group, 88% for the light exercise group, and 82% for the control group.
A significant group main effect, time main effect, and group by time interaction was found in SOR throughout the eight day recovery period. The immobilization group had significantly greater SOR the last day of treatment and the first day of recovery than either the control or light exercise group. After day two there was no longer a significantly greater SOR. All groups had practically returned to baseline levels of SOR by the fourth day of recovery. The researchers determined that activity level did influence muscle recovery after eccentric exercise of the elbow flexor, with immobilization and light exercise resulting in improved muscle function post exercise.