CHAPTER 1

INTRODUCTION

Delayed Onset Muscle Soreness (DOMS) is a common phenomenon experienced by individuals who perform unaccustomed exercise that typically involves an eccentric component. The soreness usually begins to occur at approximately 8 - 24 hours post-exercise and peaks at approximately 48 hours post exercise. Perception of soreness is generally reduced at 72 hours with residual soreness remaining beyond that time frame. The symptoms may begin to appear from a few hours to a day, reach a maximum between a period of 1 and 3 days and would subside in 5 to 7 days. (Clarkson and Hubal, 2002). This cycle of symptoms is commonly referred to as “Delayed Onset Muscle Soreness (DOMS)”.

DOMS is usually experienced by athletes especially when they are not training continuously for a period of 6 to 8 weeks during which time they lose their ability to adapt themselves to rigor. DOMS may also be experienced by athletes when they change their training regime to include new and unaccustomed modules to it. Individuals who are beginning a training schedule are also at risk of developing DOMS.

Negative implications of DOMS include minimal to severe soreness, inability to continue safe and effective training or performance, biomechanical alterations, predisposition to injury, decreases in strength and power, interruption of Activities of Daily Living (ADL’s) and a decreased motivation and willingness to continue training due to the negative experiences of soreness (Weber et al., 1994). Evidence suggests that a single bout of eccentric exercise will result in some adaptation in the exercised
muscle. Eccentric exercise has a protective effect during subsequent bouts of eccentric exercise (Ebbeling and Clarkson, 1990; Clarkson et al., 1992; Stauber, 1996) in that DOMS and other markers of microscopic muscle damage are significantly reduced (Smith et al., 1993). It is suggested that during the repair process muscle and connective tissue are strengthened and become more resistant to subsequent microscopic muscle damage (Smith, 1992; Kuipers, 1994).

There are many theories describing the reasons for the onset of delayed muscle soreness. In an attempt to prevent and alleviate DOMS it is important to have an understanding of the underlying mechanisms which contribute to DOMS. These mechanisms include damage to the contractile elements of muscle and associated connective tissue (Hough, 1902), as well as the inflammatory process associated with micro injury to both the contractile and non contractile components of muscle (Smith, 1991). Many clinical and therapeutic interventions have been used in an attempt to minimize DOMS and its negative impact on performance. Massage, various exercises, cryotherapy, ultrasound and anti-inflammatory drugs have all been used to alleviate symptoms of DOMS. Many of these scientific trials have attempted to provide evidence substantiating the widely accepted theories for their effectiveness, without success.

Use of unproven treatment techniques is common in the field of health care, more specifically Physiotherapy, Athletic Therapy and Massage Therapy (Weber et al., 1994). Intervention effectiveness appears to be based more on prior utilization and acceptance than on scientific evidence.

Although DOMS has been demonstrated by many researchers to cause negative impacts on performance, (Davies and White, 1981; Smith, 1992; Saxton and
Donnelly, 1995; Macintyre et al., 1996; Paddon-Jones and Quigley, 1997), until today there has been inconclusive research in the area of effective treatment interventions or DOMS prevention. In order to minimize negative experiences associated with DOMS and potential detrimental effects on performance, it is necessary to identify a successful treatment intervention. Ideally it would be beneficial to employ a treatment intervention that is readily available to the athlete/participant. In other words, the objective would be to determine a treatment intervention that is simple to use by considering both cost and time effectiveness. If an efficacious intervention can be developed the negative effects of DOMS may be substantially reduced.

DOMS may leave individuals in a condition of mild to extreme soreness depending on the intensity of their activity and their level of fitness. This may discourage the general population from participating in such activities. DOMS may also decrease the effectiveness of performance for those individuals who participate at high levels of competition. Regardless of the population, it is necessary to determine whether or not there is a method that may be able to reduce DOMS.

Of all the treatment interventions attempting to reduce DOMS that has reviewed to date, the single one that best meets the previously outlined objectives is exercise. Several investigators have been successful in reducing performance deficits on exercise induced damaged muscle using exercise as an intervention. Both Hasson et al. (1989) and Donnelly et al. (1992) using exercise as an intervention, have demonstrated positive effects by reducing perception of pain and reducing performance deficits. Exercise has been accepted by the majority of the athletic community as a temporary or transient relief from DOMS. To date there has been limited research to support the effectiveness of this treatment. Tiidus (1995) and
Ernst. (1998) concluded that massage was not an effective treatment modality for enhancing restoration of post-exercise muscle strength. Both authors agree that the positive effect of massage on exercise induced damaged muscle has not been demonstrated convincingly,

Using ultrasound as an intervention method that has been attempted by Ciccone et al. (1991) and Craig et al. (1999), and they failed to provide conclusive evidence of beneficial effects. When investigating the effects of cryotherapy on DOMS most investigators agree that Cryotherapy is not effective in reducing the symptoms associated with DOMS (Isabell et al., 1992; Gulick et al., 1996; Paddon-Jones and Quigley, 1997). However, Denegar and Perrin. (1992) were able to provide evidence that suggested that ice was in fact effective in treatment of the pain associated with DOMS and did not report positive effects of cryotherapy on performance measures.

Phonophoresis is routinely used for the management of soft tissues injuries, and clinically its effect is better as compared to other modality, but evidence is limited and inconclusive.

Therefore, to find out its effectiveness in DOMS, we have selected this modality. Regarding Phonophoresis, following key words (Phonophoresis, ultrasound and delayed onset muscle soreness, sport injury, recreational athlete) and with following search engine we have reviewed the literature. (CINHAL, PUBMED, MEDLINE, EMBASE, INMED, PEDRO’S, GOOGLE SEARCH) between 1980 to 2011. Only 10 studies involved in investigating the effect of ultrasound on DOMS and out of these seven studies have shown positive results and three studies have shown negative results.
So far there has been only one study done on phonophoresis and this study found that ultrasound used alone increased the symptoms associated with DOMS. These increases were not observed when ultrasound was administered through a topically applied anti-inflammatory-analgesic agent (ie, trolamine salicylate). These results suggest that the ability of ultrasound to enhance the mechanisms underlying DOMS may be offset by the pharmacologic activity of trolamine salicylate (Ciccone et al., 1991).

For cryotherapy intervention, studies have investigated the effect of cold water immersion, ice massage and cryotherapy techniques on DOMS and out of these studies, Cold water immersion does not enhanced recovery from a bout of damaging eccentric contractions (Goodall, 2008). Ice massage is ineffective in reducing the indirect markers associated with exercise induced muscle damage and enhancing recovery of muscle function in male exercisers unaccustomed to eccentric biased exercise (Howatson, et al., 2005). Sellwood, et al., (2007) found that the protocol of ice-water immersion used in this study was ineffectual in minimizing markers of DOMS in untrained individuals. Day and Ploen, (2010) found that use of ice in the treatment of DOMS is effective in minimizing perceived pain 24-96 hours after activity and four more studies have found positive findings on cryotherapy effects on DOMS.

Exercise as an intervention most of the studies have shown that repeated bouts may have lesser damage on muscle following eccentric contractions and only one study have shown that light concentric exercise has a temporarily analgesic effect on delayed-onset muscle soreness, but no effect on recovery from eccentric exercise (Kazunori, et al., 2006 ). Nosaka, (2001) found that a lower level of soreness, a faster recovery of maximal isometric force generation, a smaller decrease in relaxed elbow
joint angle and a smaller increase in upper arm circumference and creatine kinase activity were evident after the concentric-eccentric condition compared to the eccentric condition and that muscle damage was attenuated by performance of previous concentric exercise. So far no single randomized control trial has done to find out the efficacy of alternative concentric and eccentric exercise in biochemical parameters, clinical and subjective measures on DOMS.

Therefore in India no studies have done to find out the efficacy of the electrophysical agents and as well as exercise intervention in the management of DOMS.

There is conflicting evidence with regard to the use of non-steroidal anti inflammatory drugs (NSAIDs) and DOMS. Some success has been reported by Hasson et al. (1993), while Donnelly et al. (1990) and Bourgeois et al. (1999) reported non-significant findings.

Based on the reviews, Studies with varying dosages of ibuprofen (Donnelly, 1990; Hasson, 1993) revealed contradictory results. Hasson et al. (1990) administered 400 mg of ibuprofen 3 times per day, whereas this investigator examined the effect of a load dose of 1,800 mg and daily doses of 1,200 mg of oxaprozin. Perhaps the inflammatory process is a necessary component of the healing process, and interference in this process impairs the recovery of muscle function. NSAIDs administered in greater dosage may in fact impede the production of myofibrillar protein (Hasson et al., 1992). However, our objective of the study is to find out the various electro physical agents and exercise in the recovery of the DOMS.

1.1 DOMS PHYSIOLOGICAL CAUSES

In the mid 1980’s, many observations related to lactic acid build up as a cause of DOMS were negated. While in the 1980’s and 1990’s, it was widely believed that DOMS is due to damage in the muscle cells and muscle fibers(where pain signal

It is suggested that a sequence of events starting with exercise causes muscle damage and then muscle protein break down, resulting in cell inflammation and increased local muscle temperature. Due to this the pain receptors in the body are triggered and results in Delayed onset muscle soreness (DOMS). Exercise-Induced Muscle Damage (EIMD) has been hypothesized to be initiated by disruption of the force generating and / or transmitting structures and loss of sarcolemma integrity followed by a calcium overload phase resulting in an influx of extra cellular calcium (Ca$^{2+}$) that activates several intrinsic degradative pathways. The specific event that serves to initiate exercise induced muscle fiber injury is not known. It is generally recognized that this type of injury is associated with eccentric contractions. However, there are 2 possible initial events responsible for the subsequent damage: Damage to the excitation- contraction coupling system and disruption at the level of sarcomeres. Other changes seen after eccentric exercise which include a fall in active tension, shift in optimum length for active tension and rise in passive tension to favour sarcomere disruption as the starting point for the damage.

In the initial event, sarcomeres in myofibrils are disrupted and damage to the excitation-contraction (E-C) coupling system takes place. In a review, Warren et al. (2001) summarized their position by declaring that 75% or more of the decline in tension after eccentric exercise was attributable to a failure of the E-C coupling process. The rest of the damage that is evident during the initial days after the exercise is because of the damage of the elements in the muscle that are responsible for withstanding tension.
In 1902, Hough published the first report on muscle soreness. He found out that soreness experienced in the finger flexor muscles, 8-10 hours after performing rhythmical exercise, was most likely due to “some sort of rupture within the muscle”. In the early part of 1980’s myofibrillar disruption was observed when there were occurrences of morphological alterations, (Friden et al., 1983; Newham et al., 1983) these findings have kindled new interest in DOMS and in the preceding two decades from 1990s, a lot of information has been obtained on this topic. The hypothesis prevailing is that the myofibril structure gets ruptured in DOMS. However the mechanism underlying DOMS still remains unclear (Warren et al., 1999; Clarkson and Hubal, 2002; Lieber and Friden, 2002), but from the previous reviews it was found that mainly the eccentric contractions leading to the symptoms of DOMS. An eccentric contraction occurs when a muscle is contracting, and an external force is trying to lengthen the muscle. Experiments have shown that for the same work load eccentric exercises use fewer fibers and require less energy. Hence a greater amount of force per unit muscle fiber is applied thus increasing the likelihood of muscle fiber
damage. Also during eccentric exercise the body recruits more type II fibers which are
more prone to muscle fiber damage due to their wider Z lines. In addition, during
eccentric action the muscle lengthens under tension thus stretching the connective
tissue components associated with tendons/muscle fibers, causing their damage and
thus eccentric exercises causes more pain.

1.2 SYMPTOMS OF MUSCLE SORENESS

Sore muscles occurring after exercises are observed to be firm, tender and painful.
The stiffness associated with DOMS is not a function of antagonistic muscular action
but is probably caused by edema occurring in the perimuscular connective tissue
(Howell, 1993). Symptoms of DOMS develop during the first 24 to 48 hours, peak
between 24 and 72 hours, and disappear within 5 to 7 days, (Ebbeling, 1989) usually
without intervention. Passive stretching and increased activity increase the pain
further irrespective of the exact location of palpable region of soreness occurs. The
relationship between maximum voluntary force and symptoms of soreness is highly
debatable.

Ebbeling and Clarkson. (1989), suggested that there is very little or no relationship
between the development of soreness and a decrease in muscle strength. Newham et
al. (1983) demonstrated that the peak pre-exercise level of strength in quadriceps
occurs within 24 hours after step exercise and there are others who have indicated that
more than two weeks is required to recover peak isometric strength. Prolonged
weakness, reduced range of motion and higher levels of serum creatine occur along
with softness and palpitation. DOMS is commonly seen in patients performing new
exercises and in athletes involved in weight-lifting or other eccentric activities.
DOMS results from muscle damage (Clarkson and Nosaka, 1992) following eccentric
exercise. The onset of DOMS is characterized as a dull, aching pain usually beginning 12 to 48 hours after exercise (Tiidus and Ianuzzo, 1983). Clarkson et al. (1992) found that soreness peaks 2 to 3 days following eccentric exercise and subsides linearly within 10 days. In addition to pain, other symptoms include decreased motion and decreased force production (Nikolaou, 1987). Newham. (1987), however, reported a decreased force production with electrical stimulation of these muscles, indicating that the soreness itself does not inhibit force production.

Muscles adapt to a single bout of eccentric exercise. This is evidenced by less damage to the muscle after the same exercise months later (Clarkson, 1992). The muscle is repaired without any residual dysfunction or scarring and the muscle is often able to resist even greater forces (Byrnes et al, 1985).

1.3 PREDISPOSING FACTORS:

Armstrong. (1990) suggested two possible causative factors during the initial events of DOMS (Stauber, 1991): high tensions and metabolic changes.

High Tension: High tension produced during eccentric exercise is more apt to produce myofiber injury than isometric or concentric contractions.

Metabolic Changes: Increased temperature, decreased aerobic capacity, and decreased pH of the muscle may have a role in causing DOMS.

1.4 PATHOPHYSIOLOGY OF DELAYED ONSET MUSCLE SORENESS (DOMS)

Transmission of pain sensation is carried out by myelinated group III (A-delta fiber) and unmyelinated group IV (C-fiber) afferent fibers. Group III and IV sensory neurons terminate in free nerve endings. These free nerve endings are scattered
mainly in the muscle connective tissue between fibers (especially in the regions of arterioles and capillaries) and at the musculotendinous junctions. Quick, sharp and highly localized pain is transmitted by the myelinated group III fibers. Slightly lesser, duller and gentler pains are carried by group IV fibers.

DOMS sensation is transmitted mainly by the group IV afferent fibers. The free nerve endings of group IV afferent fibers in muscles are poly modal i.e., they have the ability to respond to different stimuli, including chemical, mechanical, and thermal. Chemical substances that initiate action in muscle group IV fibers are bradykinin, 5-hydroxytryptamine (serotonin), histamine, and potassium, in order of effectiveness.

DOMS occurs because of overuse of the muscles. Activities that require higher force in the muscle than normal or those which produce force over an elongated period of time can cause DOMS. According to Tiidus and Ianuzzo. (1983), the degree of muscle soreness is related to the intensity of the muscle contractions and to the duration of the exercise. Intensity is more important than determination.

The pathophysiology of DOMS can be explained using five hypotheses

**Structural damage from high tension**

This hypothesis was originally proposed by Hough and is the most scientifically accepted theory. The delayed pain is related directly to the development of peak forces during exercise and to the rate of force development in rhythmic contractions. The state of fatigue of the muscle and DOMS are not related.

The rhythmic and tetanic contractions that cause the greatest acute fatigue and discomfort in the muscles during exercise results in the least delayed pain following
the exertion. When the muscles are not trained for a particular exercise, structural damage is more likely to develop.

Hough. (1902) concluded that DOMS was “fundamentally the result of ruptures within the muscle.” Although “ruptures” of muscle fibres are not associated with DOMS, ultra structural disruptions of myofilaments, especially at the Z disc, characterized by broadening, streaming, or smearing of the Z disc structure as observed under electron microscope, have been reported to accompany DOMS (Friden et al., 1981, 1984; Yu and Thornell, 2002). Therefore the ‘Damage Theory’ proposed by Hough is still valid with some modification, and it is most likely that muscle or connective tissue damage (or both) and subsequent inflammatory responses are associated with DOMS (Cheueng et al., 2003). It may be that changes in connective tissue (endomysium or perimysium), rather than damage to muscle fibres relate directly to DOMS. Jones et al. (1997) suggested that damage and shortening of muscle connective tissue would increase the mechanical sensitivity of muscle nociceptors and cause pain with stretching or palpation. One postulate is that the inflammatory response process leading to sensitization of muscle nociceptors takes time, and this would explain the delay (Smith, 1991).

**Metabolic waste product accumulation**

One of the most popular concepts in the lay exercise community is that delayed soreness is a result of lactic acid accumulation in the muscles. An apparent relationship exists between exercise intensity and the extent of soreness. It must be noted that there is opposing observations for metabolic hypothesis. Two hypotheses, metabolic overload and mechanical strain, have been suggested as causative mechanisms for muscle damage. As eccentric activation causes most muscle damage, it suggests that high local muscle tensions are in some way more important than
extreme metabolic demand in the aetiology of DOMS injury (Armstrong, 1986). Appell, et al. (1992) presented evidence to suggest that the mechanisms producing muscle damage after level endurance running and downhill running were not the same. A comparison was made of the histological structure of the rat soleus muscle, subjected either to a level endurance run or a downhill run. The losses of striation pattern seen in 15% of fibres at 48 h were predominantly in fibres which were glycogen depleted. This suggests a metabolic aetiology for the damage. Glycogen depletion was present in 25% of the fibres immediately after level running and increased to 33%, 48 hours later. There were similar levels of lysosomes in the muscle fibres. This, combined with an immediate 16% loss of sarcomere organization, suggested an autophagic response, due to metabolic exhaustion and enzyme leakage. In contrast, however, the downhill running muscle had higher disruption levels (33% of fibres with immediate loss of sarcomere organization) but no glycogen depletion or lysosome occurrence in the fibres. This lack of glycogen depletion and absence of autophagic response supports a mechanical origin of muscle damage (Armstrong, 1981).

The lactic acid theory is based on the assumption that lactic acid continues to be produced following exercise cessation. For the lay public, the accumulation of toxic metabolic waste product is thought to cause a noxious stimulus and the perception of pain at a delayed stage (Armstrong, 1984; Gulick, 1996; Isabell, 1992). However, this theory has largely been rejected as the higher degree of metabolism associated with concentric muscle contractions have failed to result in similar sensations of delayed soreness (Asmussen, 1956). In addition, lactic acid levels return to pre-exercise levels within 1 hour following exercise and blood lactate levels measured before, during and sporadically up to 72 hours after level and downhill running have failed to show a
relationship between lactic acid levels and soreness ratings (Schwane, et al., 1983). Therefore, lactic acid may contribute to the acute pain associated with fatigue following intense exercise, however, it cannot be attributed to the delayed pain that is experienced 24–48 hours post exercise (Cazorla, et al., 2001). Relatively low energy is expended in the case of muscle contractions that cause higher soreness. Lesser oxygen consumption and lower lactate production are observed in the case of eccentric exercises than in concentric contractions at the same power output. Lesser energy use per unit area is observed in the case of eccentric exercise than in concentric exercises. Schwane et al. (1983) tested the metabolic hypothesis. Their results indicated that downhill running requires significantly lower oxygen uptake (VO₂) and produces less lactic acid than does level running but that it nonetheless results in greater DOMS.

**Increased temperature**

Type III and IV nerve endings are sensitive to temperatures of 38-48°C. Higher temperatures can cause damage to the structural elements in the muscle. This could lead to necrosis of muscle fibers and eventually tissue breakdown. Atypical exercises of muscles generate higher local temperatures than that produced by the concentric contractions. Rhabdomyolysis (severe form of DOMS) is seen more in untrained objects while exercising in hot conditions.
Spastic contracture

Travell et al. (1942) and a later series of experiments by Cobb. (1975), demonstrated elevated electromyographic activity in sore muscles (Hedayatpour, 2008). Altered nerve control and vasoconstriction lead to decreased blood flow and ischemia, which in turn initiate a pain-spasm-pain cycle (Davis, 2008). The impact of the pain directly depends on the number of motor units involved. Enhanced electrical activity in the sore muscles has not been detected by other investigators.

Myofibrillar remodeling

The literature (Yu, 2004) suggests that myofibrillar and cytoskeletal alterations are the hallmarks of DOMS and that they reflect adaptive remodeling of the myofibrils.

Basically there are four types of changes Amorphous widened Z-disks, Amorphous sarcomeres, Double Z-disks, and Supernumerary sarcomeres

1.5 BIOCHEMICAL CHANGES IN DELAYED ONSET MUSCLE SORENESS

Damage to the sarcolemma and extra cellular matrix (ECM) creates an altered chemical environment within the muscle. Release of proteins and ions into the plasma as a result of inflammation is similar to that found in acute strains (Stauber, 1991). Increases in these levels indicate damage to the sarcolemma. Elevations of intracellular molecules such as CK, LDH, protein metabolites, and myoglobin have been found in plasma up to 48 hours following eccentric exercise (Clarkson et al, 1986). Liberation of these biochemical substances occur from the muscle cells and begin approximately 24 hours post exercise (Clarkson, 1992), before phagocytic cells enter the injury site (Armstrong, 1990). Time-specific clinical events (such as peak soreness at 2 to 3 days) may correspond to the time of increased enzyme levels (such
as CK increase at 2 days). While Tiidus. (1983), reported such a correlation between soreness and enzyme levels, Clarkson et al. (1992) cautioned against claiming a cause-and-effect relationship based on limited research. Structural disruption leads to the normal inflammatory response: an increase in chemical mediators such as histamine, bradykinin, prostaglandin, and serotonin (Arnheim, 1989) causing pain and swelling. The products of the inflammatory response sensitize free nerve endings in muscle (Smith, 1991), thus increasing soreness. Stauber et al. (1991) concluded that the DOMS after repeated eccentric muscle action is not because of actual myofiber damage, but more likely results from inflammation.

### 1.6 WHY DOMS NEED TO BE TREATED:

Temporary muscle performance decline is observed with DOMS. This is because of the sensation of soreness coupled with muscle’s inherent capacity to produce force that creates reduced voluntary effort. Eventually, there is a huge decline in the range of motion, lesser power in muscles and swelling in the affected area due to DOMS. Negative implications of DOMS include minimal to severe soreness, inability to continue safe and effective training or performance, biomechanical alterations predisposing individuals to injury, decrease in strength and power, interruption of activities of daily living (ADL’S) and decreased motivation and willingness to continue training due to negative experiences of the soreness (Weber et al., 1994). In order to minimize negative experiences associated with DOMS and potential detrimental effects on performance, it is necessary to identify a successful treatment intervention.
1.7 HOW DOMS CAN BE TREATED:

DOMS can be treated either by advising complete rest from the participation, or by medical management and various physiotherapy approaches to reduce the soreness symptoms.

MEDICAL MANAGEMENT: General analgesics and nonsteroidal anti-inflammatory medications have not been consistently effective against post exercise muscle soreness (McAnulty, 2007). Cannavino. (2003) showed that transdermal 10% ketoprofen cream was effective in alleviating self-reported DOMS in isolated quadriceps muscles of patients following repetitive muscle contraction, particularly after 48 hours. The apparent relief is secondary to the effects of medication as no other medication was used in the study. DOMS can be treated with Oral ascorbic acid (vitamin C) and other antioxidants albeit with mixed results. In a study by Connolly and coauthors it was observed that a vitamin-C supplementation protocol of 1000 mg taken 3 times a day for 8 days is not effective in protecting against selected markers for DOMS (Connolly, 2006).

1.8 VARIOUS PHYSIOTHERAPY APPROACHES

Physiotherapy Interventions play a major role both in prevention and treatment for the negative effects associated with DOMS. Many treatments have been investigated including massage, electrical stimulation, external counter pulsation (Catanese and Louis, 2008) ultrasound, acupuncture (Barlas, 2000), cryotherapy, repeat bout exercise, stretching (Johansson, 1999), Low intensity Laser therapy (Craig, 1999), Iontophoresis (Hasson, 1992), Transcutaneous Electrical Nerve Stimulation (TENS) (Craig, 1996), preventative training, and even hyperbaric oxygen therapy (Mekjavic, 2000). But none of these have been proved to benefit DOMS treatment.
1.9 PROBLEM STATEMENT

Some of the important factors in performing sports and exercise are strength, range of motion, and a sense of competence. Unaccustomed eccentric exercise causes adverse effects on these factors (Bourgeois and MacDougall, 1999). Exercises that are eccentric may result in a significant decline in performance both during training and competition while further increasing the risk of injury. If such eccentric exercises are carried on by athletes during training, the efficiency of such exercises on the subsequent day can be affected. No efficient and consistent treatment for DOMS has been made available till date. Even though there are many practices for treating DOMS, they lack scientific backing. DOMS is being treated through numerous ways which include herbal products, pharmaceuticals, stretching, nutrition supplements, massage and many more. As DOMS has been shown to alter biomechanics of movement (Ebbeling and Clarkson, 1989) and predispose participants to injury, it is important to determine an effective treatment intervention that will reduce the negative impacts of DOMS. The present research is done on recreational athletes as they usually begin a new exercise programme, increase their exercise intensity and commonly change their sports activity.

1.10 RECENT TRENDS IN THE SCIENTIFIC STUDY OF THE IMPORTANCE OF PHYSIOTHERAPY MODALITIES IN AMELIORATING THE DELAYED ONSET MUSCLE SORENESS (DOMS) OF RECREATIONAL ATHLETES

While gentle stretching was considered as one of the best ways to reduce the impact of soreness in the muscles due to exercises, it was proven ineffective through a study by Australian research in a 2007 publication (Herbert, 2007). Recently study was done on better attenuation effects of compression in DOMS, compression (and compression suits) have been studied over the past 8 years. In another recent study
Lau and Nosaka. (2011) investigated the effect of Vibration treatment on symptoms associated with Eccentric Exercise-Induced Muscle Damage and the results showed that the vibration treatment was effective for attenuation of delayed-onset muscle soreness and recovery of range of motion after strenuous eccentric exercise but did not affect swelling, recovery of muscle strength, and serum creatine kinase activity. Many studies have been done to find out the significance effects of modalities on DOMS, but none of the studies so far have been shown much significant results in the biochemical markers of DOMS and in relieving the symptoms of muscle soreness.

1.11 NEED FOR THE STUDY

The extensive reviews of the literature reveal that, there is paucity of studies on role of various Physiotherapy modalities in ameliorating the DOMS. Coaches, athletes, and medical practitionerers are well aware of the symptoms of muscle damage after eccentric exercise because it clearly affects subsequent exercise or performance (i.e., strength, power, range of motion and probably exercise economy). Muscle weakness requires a longer recovery and might have more impact on performance than soreness sensation. Therefore the present study is needed to help for the athletes to be safe from the above problems arising out of muscle damage.

1.12 SIGNIFICANCE OF THE PROPOSED STUDY IN THE CONTEXT OF CURRENT STATUS

International status

Developed countries with their modern “Know how” are progressing rapidly in exploring the possibilities of various physiotherapy modalities to supplement the medical management thereby to facilitate early recovery.
National status

The wealth of knowledge that has been provided by our researchers regarding the use of various physiotherapy modalities for DOMS has to be collaborated with detailed well organized study. Such a scientific study will facilitate Indian researchers and clinicians to use these modalities to treat DOMS effectively.

1.13 SIGNIFICANCE OF THE PROBLEM

Contraction-induced skeletal muscle damage, especially as a result of unaccustomed eccentric contractions, is a phenomenon that commonly excruciates athletes/individuals. Although in the last decades a remarkable effort has been made to understand this particular phenomenon a lot of issues remain to be elucidated. Particularly, there is little information regarding the continuation of exercise with damaged muscles. Evidence suggests that exercise continuation prior recovery do not exacerbate muscle damage and neither affects the ability of skeletal muscle to recover. This evidence, however, have been based on indirect markers of muscle damage the reliability of which have been challenged by several studies. Consequently, this issue to be resolved requires morphological evidence and direct quantification of muscle damage.

It is well known that once skeletal muscle is damaged, as a result of an unaccustomed eccentric exercise it requires approximately 10 days or more to recover. In practical situations, however, the resting interval needed for the skeletal muscle to recover is not always available because most of the training plans are performed more than once per week. Consequently, there is an emergent need to determine the effects and subsequently the appropriateness of exercise continuation with damaged muscles.
In order to provide a more complete picture, regarding the effects and appropriateness of exercise continuation prior recovery, we have designed an integrated study investigating several physiological aspects including mechanical, morphological, and cellular-molecular.

1.14 OPERATIONAL DEFINITIONS

RECREATIONAL ATHLETE: “Recreational athlete” means an individual participating in fitness training and conditioning, sports or other athletic competition, practices or events requiring physical strength, agility, flexibility, range of motion, speed or stamina and who is not affiliated with an amateur, educational or professional athletic organization or any association that sponsors athletic programs or events in the state.

ECCENTRIC EXERCISE: A voluntary muscle activity in which there is an overall lengthening of the muscle in response to external resistance.