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## The effects of movement based interventions on DOMS and muscle damage following eccentric exercise

Zainal A. Zainuddin  
Edith Cowan University

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# **The Effects of Movement Based Interventions on DOMS and Muscle Damage Following Eccentric Exercise**



By

**Zainal A. Zainuddin MSc (Sports Science)**

Principal Supervisor: Associate Professor Dr Kazunori Nosaka

Associate Supervisor: Dr Paul Sacco

Associate Supervisor: Mr. Mike Newton

This thesis is presented for the award of Doctor of Philosophy (Sports Science) at the School of Biomedical and Sports Science, Faculty of Computing, Health and Science, Edith Cowan University, Perth, Western Australia

Date of Submission: 16/12/2004

## ABSTRACT

Delayed onset muscle soreness (DOMS) is a common symptom experienced by most adults following unaccustomed exercise. It is known that DOMS is peculiar to eccentric exercise that results in muscle damage which is characterized by prolonged loss of muscle function. DOMS and subsequent impaired muscle function, reduces the ability to maximize performance of daily living tasks, and has negative effects on sports adherence and activity based healthy lifestyle. Thus, treatments to ameliorate DOMS and enhance recovery of muscle function are important.

The purpose of this study was to investigate the effect of three therapeutic interventions on DOMS, recovery of muscle function and symptoms related to muscle damage. The three interventions were immobilisation (limited movement); massage (passive movement); and light concentric exercise (active movement). The effects of each treatment were investigated separately and compared to a control condition. The underlying research question throughout the three studies was to determine whether one of the following movement treatments, active, passive, or limited movement was better at ameliorating DOMS, restoring muscle function, and facilitating recovery from the symptoms of muscle damage. An arm to arm comparison model was used in each study such that both arms performed the same exercise separated by at least two weeks, with one randomly assigned arm received treatment while no treatment for the contralateral arm (control). A total of 34 subjects were recruited for the study with age, weight and height being  $24.2 \pm 2.4$  yrs,  $67.2 \pm 13.7$  kg,  $164.5 \pm 11.8$  cm, respectively and placed randomly into intervention study group of : immobilisation ( $n = 10$ ), massage ( $n = 10$ ) and light concentric exercise ( $n = 14$ ).

All subjects performed 10 sets of six maximum voluntary eccentric actions of the elbow flexors against the lever arm of an isokinetic dynamometer (Cybex 6000, Ronkonkoma, NY, U.S.A.) moving at a constant velocity of  $90^\circ \cdot s^{-1}$  where the elbow joint was forcibly extended from a flexed ( $90^\circ$ ) to an extended position ( $180^\circ$ ) in one second while the subject was required to maximally resist against the motion.

For the immobilisation study, the intervention arm was placed in a sling to secure the elbow joint angle at 90°, 30 minutes after eccentric exercise for four days. In the massage study, the intervention arm received 10 minutes of sports massage three hours after eccentric exercise. Subjects in the light concentric exercise study performed light exercise (10 sets of 60 elbow flexions and extensions with minimal force generation) on days 1, 2, 3 and 4 days following eccentric exercise.

Changes in muscle soreness and tenderness, maximal voluntary strength, ROM, upper arm circumference, and plasma creatine kinase (CK) activity before and for 7 days following eccentric exercise were compared between the treatment and control arms using two-way repeated measured ANOVA for each study. When the ANOVA found a significance effect, Tukey's Post Hoc analyses were used to locate the significant difference.

Eccentric exercise resulted in large strength losses ( $\approx 40\%$ ), reduced ROM ( $\approx 15^\circ$ ), increased arm circumference ( $\approx 10\text{mm}$ ), elevated plasma CK activity ( $\approx 2,000 \text{ IU}\cdot\text{L}^{-1}$ ), and development of DOMS and increased tenderness. Immobilisation produced no significant differences between treatment and control arms for all of the measures with the exception of upper arm circumference. Upper arm circumference for immobilisation on day 7 post exercise recorded significantly smaller (4 mm) measured compared to the control condition (11mm). Massage was effective in reducing DOMS significantly ( $p < 0.05$ ) approximately 36%. Increase in plasma CK activity was significantly smaller for the arm received massage than the control arm. However, muscle functions and swelling were not affected by massage. Light concentric exercise produced a significant reduction of DOMS by 50%, decreases in muscle strength by 10%, and increases in CIR by 5mm immediately after exercise bouts. However, no significant differences were found with light concentric exercise for changes in any criterion measures over time.

Comparisons between the three interventions were also conducted. No significant differences between immobilisation, massage, and light concentric exercise were evident for the recovery of muscle strength and ROM. DOMS was affected by

massage, but the effect was limited to days 3 and 4 post exercise. Increases in plasma CK activity and swelling were also attenuated by massage.

These results demonstrate that light immobilisation does not enhance recovery of muscle function following exercise-induced muscle damage. It would appear that the immobilisation protocol needs to be very strict in order to produce substantive beneficial effects. Massage intervention was found effective in alleviating DOMS but has minimal effects on muscle function and swelling. Light concentric exercise on the other hand has only temporary palliative effect on DOMS, but no effect on the recovery of muscle function. It is concluded that immobilisation and light concentric exercise following eccentric exercise do not affect the occurrence of DOMS and other symptoms of muscle damage. The therapeutic interventions investigated in this study do not seem to result in alleviating DOMS with an exception to massage or enhancing recovery of muscle function. Emphasis should be shifted to prophylactic treatments to prevent and treat DOMS and muscle damage.

## DECLARATION

I certify that this thesis does not, to the best of my knowledge and belief:

- (i) incorporate without acknowledgment any material previously submitted for a degree or diploma in any institution of higher education;
- (ii) contain any material previously published or written by another person except where due reference is made in the text; or
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# CHAPTER 1

## INTRODUCTION

### 1.1 Background of Study

Following unaccustomed or strenuous exercise, discomfort may be felt in the muscles that have been worked (Clarkson, Byrnes, McCormick, Turcotte, & White, 1986; Jones & Round, 1990; Stauber, 1989). The discomfort exacerbates gradually and usually peaks one or two days after exercise (MacIntyre, Reid, & McKenzie, 1995). Muscle soreness consists of a dull, aching pain combined with tenderness and often stiffness (Armstrong, 1984; Byrnes & Clarkson, 1986) and is referred to as a delayed onset muscle soreness (DOMS) (Armstrong, 1984; Miles & Clarkson, 1994). Virtually all healthy adults have experienced DOMS following exercise, however the exact cause of DOMS is still not clearly understood (Armstrong, 1984; MacIntyre et al., 1995). DOMS was first described by Hough (1902) more than a century ago. In his classical experiment of ergographic tracing from the flexors of the middle finger, he observed 'no discomfort accompanied this tracing, nor was any noticed or some hours afterward. Eight or ten hours later, however, the muscle began to show signs of soreness, and this soreness increased to its height twelve or more hours after the tracing.' Hough concluded that this kind of soreness was "fundamentally the result of ruptures within the muscle."

Following unaccustomed eccentric exercise, which consists of lengthening actions of contracted muscles, produces greater DOMS than those in which mainly concentric (shortening) or isometric (static) muscle actions are performed (Clarkson et al., 1986; Talaq, 1973). Although isometric contractions at a long muscle length induce some DOMS (Jones, Newham, & Torgan, 1989), concentric muscle actions alone do not appear to induce DOMS. Therefore, to understand DOMS, it is important to understand the characteristics of eccentric exercise.

In addition to DOMS, a number of functional, structural and biochemical changes occur after eccentric exercise. These include declines in muscular strength and power (Armstrong, 1984; Miles, Clarkson, Keller, & Hackney, 1994; Sargeant & Dolan, 1987), swelling (Franklin, Currier, & Franklin, 1991), decreased flexibility and range of motion (Saxton & Donnelly, 1995), efflux of intramuscular proteins (Schwane, Johnson, Vandenakker, & Armstrong, 1983) and Z myofibrillar band and sarcomere disruption (Friden & Lieber, 1992). These phenomena are often used as indicators of muscle damage (Clarkson, 1997; Proske & Morgan, 2001). It is not clearly understood why eccentric exercise induces muscle damage. However, it seems likely that the mechanical strain caused by lengthening of active muscles is a factor involving in the process (Clarkson, 1997; Lieber & Friden, 1993). since motor units recruited during lengthening actions are than those in shortening actions, resulting in higher mechanical stress per fibre (Kuipers, 1994).

Since eccentric exercise causes both DOMS and muscle damage, DOMS and muscle damage are often used interchangeably. However, some symptoms of muscle damage are not necessarily accompanied by DOMS (Rodenburg, Bar, & De Boer, 1993; Warren, Lowe, & Armstrong, 1999), moreover DOMS is not necessarily indicative of the extent of injury (Nosaka, Newton, & Sacco, 2002a). Nosaka et al. (2002a) reported poor correlation between DOMS and other indicators of muscle damage as well as different time course of recovery from DOMS and other variables. Thus, although both DOMS and indicators of muscle damage occur commonly after eccentric exercise, it is important to distinguish between them, when investigating the effects of an intervention. It may be that some interventions may be effective for DOMS but not other indicators of muscle damage.

Athletic performance may be impaired when muscle damage and DOMS are present (Connolly, Sayers, & McHugh, 2003). Any practice that limits the extent of muscle damage and DOMS will be of interest to athletes, coaches, trainers and therapists. The effects of muscle discomfort are ignored by athletes who are accustomed to its presence; however, because impairment of muscle function can impact athletic performance, expeditious recovery of function after damaging exercise is essential. (Cheung, Hume, & Maxwell, 2003; Connolly et al., 2003). Damaging eccentric



exercise also results in reduced fine motor skills (Pearce, Sacco, Byrnes, Thickbroom, & Mastaglia, 1998). Therefore, treatments of DOMS and muscle damage are an important issue for athletes and their performance capabilities.

DOMS and muscle damage are also of interest to the general population, since they may interfere with activities of daily living or present barriers to the adoption or maintenance of physical activity performance. Although transient in nature, DOMS may be severe enough to cause concern in those previously unaccustomed to such responses. In sedentary or untrained individuals, the effects of DOMS could lead to negative attitudes in regard to exercise. Thus, the aftermath of exercise, such as difficulty in getting out of bed or walking down stairs because of sore muscles may be a factor for avoidance of involvement in sports and physical activity. Therefore, interventions to reduce DOMS would also prove beneficial for these individuals.

Many interventions have been trialled under the pretext of preventing or attenuating DOMS and muscle damage, or enhancing recovery from muscle damage. Such interventions can be categorised into one of two groups, based on when they are applied; prophylactic or therapeutic. In the case of exercise likely to induce DOMS and muscle damage, prophylactic interventions are applied prior to exercise, whereas therapeutic interventions are performed after exercise.

Prophylactic interventions include stretching and warm up (Johansson, Lindstrom, Sundelin, & Lidstrom, 1999; Rodenburg, Steenbeek, Schiereck, & Bar, 1994; Safran, Seaber, & Garrett, 1989; Shellock & Prentice, 1985), non-steroidal anti inflammatory drugs (NSAIDs) (Giamberardino, Dragani, Valente, Di Lisa, & Vecchiet, 1996), exercise (Brown, Child, Day, & Donnelly, 1997; Clarkson, Nosaka, & Braun, 1992; Costill, Coyle, Fink, Lesmes, & Witzmann, 1979; Gleeson, Eston, Marginson, & McHugh, 2003) and nutritional supplements (Dangott, Schultz, & Mozdziak, 2000; Nissen et al., 1996; Panton, Rathmacher, Baier, & Nissen, 2000; Volek et al., 1997).

Therapeutic interventions include massage (Farr, Nottle, Nosaka, & Sacco, 2002; Smith, Keating et al., 1994; Tiidus, 1997), immobilisation (Sayers, Clarkson, & Lee, 2000a; Zarzhevsky et al., 2001), compression (Thorsson, Lilja, Nilsson, & Westlin, 1997), stretching (Rodenburg et al., 1994), exercise (Saxton et al., 1995), ice therapy (Yackzan,

Adams, & Taunton, 1984), NSAIDs (Cooper, 1981), laser therapy (Craig, Barron, Walsh, & Baxter, 1999), monochromatic infrared therapy (Craig, Barron et al., 1999), ultrasound (Roebroeck, Dekker, & Oosterndrop, 1998), transcutaneous electrical nerve stimulation (TENS) (Denegar, Perrin, Rogol, & Rutt, 1989), hyperbaric oxygen therapy (Delaney & Montgomery, 2001; Harrison et al., 2001) and acupuncture (Barlas, Robinson, Allen, & Baxter, 2000).

Although many prophylactic and therapeutic interventions have been investigated for their effect on DOMS and symptoms related to muscle damage after eccentric or eccentrically biased exercises, none have been found to prevent or eliminate DOMS completely, or enhance recovery of muscle function after exercise. However, some studies reported 'significant' effects on DOMS or markers of muscle damage. It should be noted that the 'significant' effect does not necessarily mean 'physiological' or 'practical' significance. For example, a reduced increase in plasma creatine kinase (CK) activity, which is often used as a marker of muscle damage, after an intervention may not be meaningful if the intervention does not affect DOMS or muscle function. On the other hand, if an intervention is effective in ameliorating DOMS substantially without any effects on muscle function, it may still be important for those people whose main concern is soreness.

Controversy exists concerning the effects of most interventions on DOMS and muscle damage, and no consensus exists regarding the most appropriate intervention. For example, several studies (Farr et al., 2002; Tiidus, 1999; Weber, Servedio, & Woodall, 1994) have examined the effect of massage on DOMS and muscle damage, with inconsistent results. Further studies are necessary to clarify whether massage is effective for DOMS and/or enhancing recovery of muscle function.

Previous studies of interventions for DOMS and muscle damage used various exercise modalities in terms of load, intensity and type of exercise. To date, no research has systematically used the same protocol in inducing DOMS and muscle damage to compare the effects of different interventions on DOMS and muscle damage using the same population of subjects in a controlled condition. Therefore it is important to standardise the intensity and the amount of exercise, to set a randomised trial, and to have a reasonable number of subjects. The standardised protocol inducing DOMS is a

major feature of the present study whereas previous works has focused on a specific intervention, this study investigates the effects of three different interventions on DOMS and indicators of muscle damage in a controlled condition.

In this thesis, three therapeutic treatments namely massage, immobilisation, and exercise were chosen to investigate their effects on DOMS and indicators of muscle damage. Based on previous studies (Saxton & Donnelly, 1995; Sayers et al., 2003), it was thought that 'movement' is an important influencing factor for DOMS and for recovery of muscle function after eccentric exercise. In fact, pain sensation has been shown to be influenced by exercise (Koltyn, 2000; Koltyn & Arbogast, 1998), and mobilisation of injured soft tissue has been shown to be beneficial for functional recovery (Jarvinen & Lehto, 1993; Sayers et al., 2000a). On the other hand, complete rest, such as occurs with immobilisation, has been reported to be beneficial for recovery from muscle damage (Jarvinen & Lehto, 1993; Sayers et al., 2003). If DOMS is a sign not to use or move a sore muscle, complete rest would be the most obvious choice. However, exercise of sore muscles is also known to palliate DOMS to some degree (Nosaka & Newton, 2002d; Saxton & Donnelly, 1995). Since movement based interventions are still in their infancy, it is important to compare the influence of immobilisation and active mobilisation (exercise) on DOMS and other symptoms of muscle damage. Massage treatment may be described as a passive movement of muscle. It is also important to mention that these interventions are practical in daily life as they can be applied easily and economically.

There are many exercise models to investigate effects of treatment interventions on DOMS and other symptoms of muscle damage. A major issue in monitoring the effects is a large variability of responses among subjects to eccentric exercise (Clarkson et al., 1986; Clarkson & Hubal, 2002; Nosaka & Clarkson, 1996b). The inconsistency among subjects in response to eccentric exercise is likely to act as a confounding factor that affects the reliability of the method of investigating the effect of an intervention on DOMS and indicators of muscle damage. One solution to this problem is to utilize a 'limb to limb' comparison model in which the treatment limb is compared with the contralateral (untreated) limb of the same subject.

Thus, this study applied the 'limb to limb' comparison model, to the elbow flexors, and compared the treatment and control arms for changes in DOMS and some indicators of

muscle damage after eccentric exercise of the elbow flexors. The specific modalities tested in this study were massage, immobilisation and exercise, and the treatments were applied after a bout of eccentric exercise. The effect of each treatment was tested separately by using a different group of subjects, however, all subjects were recruited from the same population. Comparisons between the treatment and control arms for DOMS and indicators of muscle damage were performed within each study, then comparisons between the three treatments made.

## 1.2 Significance of Study

DOMS represents a potential deterrent to the attainment of optimal physiological fitness and performance, especially when high levels of performance are required for athletic competition. The treatments and mechanism of DOMS and muscle damage are still poorly understood. This research will contribute to the body of knowledge regarding the most appropriate interventions to ameliorate DOMS. The chosen interventions may also be important for reducing the recovery time of impaired muscle function associated with muscle damage. Basic understanding of the proposed mechanism of DOMS and eccentric exercise-induced muscle damage, and the effects of treatments in this study will aid recreational and elite athletes, coaches and practitioners in the design and practice of programs which allow minimal damage and optimum productivity over the recovery and training period and to avoid the negative effects in regard to the exercise adherence. Information gained on muscle strength and function of elbow flexors and responses to potentially damaging exercise may provide optimal strategies for recovery, responses to high intensity exercise and periodisation strategies for training following competition. The findings may also enhance the understanding of possible benefits linked with physical therapies to alleviate DOMS and other symptoms of muscle damage. The findings of this study will improve the understanding of methods for reducing DOMS involving movement of the affected area.

### 1.3 Purpose of Study

The study aimed to answer the following questions.

- 1) Will four-days of partial immobilisation of the limb alleviate DOMS and improve recovery of other parameters associated with muscle damage following eccentric exercise?
- 2) Will 10-minutes of sports massage applied three hours post exercise to the limb alleviate DOMS and improve recovery of other parameters associated with muscle damage following eccentric exercise?
- 3) Will four-days of daily light concentric exercise of the affected limb alleviate DOMS and improve recovery of other parameters associated with muscle damage following eccentric exercise?
- 4) Which of the three chosen interventions will be the most effective at alleviating DOMS and improve recovery of other parameters associated with muscle damage following eccentric exercise?

### 1.4 Hypothesis

It was hypothesized that:

- 1) Short term immobilisation would be beneficial for recovery from eccentric exercise-induced muscle damage.
- 2) Massage would alleviate DOMS, and enhance recovery of muscle function after eccentric exercise.
- 3) A bout of light concentric exercise undertaken daily following exercise would alleviate DOMS and enhance recovery of muscle function following eccentric exercise.

## CHAPTER 2

### LITERATURE REVIEW

#### 2.1 Introduction

This chapter reviews the current body of knowledge on eccentric exercise-induced muscle damage (EIMD) and delayed onset muscle soreness (DOMS), and introduces exercise models and indicators of EIMD. This chapter describes briefly prophylactic treatments and focuses more on therapeutic treatments for EIMD and DOMS.

#### 2.2 Eccentric Exercise

During locomotion, skeletal muscles shorten and lengthen. The lengthening of active muscles is referred to as eccentric action (Armstrong, Warren, & Warren, 1991; Clarkson & Sayers, 1999; McCully & Faulkner, 1985). Eccentric muscle actions occur when the external load exceeds muscle force (Cleak & Eston, 1992; McHugh, Connolly, Eston, & Gleim, 1999; Stauber, 1989). A typical eccentric muscle action involves forced lengthening of maximally contracted muscles by applying a higher external force than the muscles are producing. Eccentric muscle actions also occur when muscles are used as a brake and absorb shock (Fox, 1979). Eccentric exercise refers to exercise consisting of predominantly eccentric muscle actions. For example, the quadriceps or knee extensors perform eccentric actions when walking downhill, skiing and horse riding (Fox, 1979). An example of an eccentric action of the elbow flexors is lowering a heavy weight slowly.

#### 2.3 Exercise Induced Muscle Damage (EIMD)

EIMD is generally defined as a loss due to injury (Armstrong et al., 1991). Safran et al. (1989) have stated that muscle injury can be divided into three major types based on clinical presentation. Type I injury is muscle soreness that occurs 24 to 48 hours after unaccustomed exercise, so called DOMS. Type II injury is characterised by an acute disabling pain from a muscle tear, ranging from a tear of a few fibres with fascia remaining intact to a complete tear of the muscle and fascia. Type III injury includes muscle soreness or cramp that occurs during or immediately after exercise.

It has been well documented that the Type I injury is peculiar to eccentric exercise (Proske & Morgan, 2001). Unaccustomed eccentric exercise results in muscle damage characterised by Z-line streaming and myofibrillar disruption (Friden & Lieber, 1992). Other indications of muscle damage include prolonged loss of muscle strength, reduced range of motion and swelling and an increase in muscle protein in the blood (Clarkson, 1997). DOMS is often accompanied by varying degrees of muscle function impairment such as decreases in maximal voluntary strength (Brown, Child, & Donnelly, 1997; Nosaka & Clarkson, 1996a) and range of motion (Howell, Chila, Ford, David, & Gates, 1985). These phenomena in combination are often referred to as exercise-induced muscle damage or EIMD (Clarkson & Tremblay, 1988; Warren, Lowe et al., 1999).

Proske and Morgan (2001) suggested that the primary damage originates from either disrupted sarcomeres in myofibrils or damage to excitation-contraction (E-C) coupling. Mechanical factors such as high force or strain appear to initiate the damage process by disrupting intracellular calcium homeostasis or mechanically disrupting the integrity of sarcomeres (Proske & Morgan, 2001). Previous researchers (Allen, 2001; Balnave & Allen, 1995; Warren, Ingalls, Lowe, & Armstrong, 2001; Warren et al., 1993) support damage to E-C coupling as a primary event in muscle damage.

Armstrong (1990) described four stages in EIMD. The initial event produces micro injury to the muscle. This phase is considered to be the trigger and may be mechanical or metabolic in origin. This event also leads to a second stage, the autogenetic phase where a loss of  $\text{Ca}^{2+}$  homeostasis in the muscle fibre takes place. Elevated intracellular  $\text{Ca}^{2+}$  levels in the cell initiate several intrinsic degradative pathways, when the calcium level exceeds a limit, triggered by calcium activated neutral and phospholipase  $\text{A}_2$ . This process leads to muscle fibre necrosis followed by regeneration. The inflammatory processes are important in the removal of the damaged proteins and seem essential for stimulating regeneration (Armstrong, 1984; Armstrong et al., 1991; Clarkson & Hubal, 2002). Muscle swelling usually accompanies this phase (Armstrong et al., 1991; Pyne, 1994).

## 2.4 Delayed Onset Muscle Soreness

DOMS is the sensation of discomfort and pain in the muscles involved in exercise which increases in intensity for the first 24 hours after exercise, peaks between 24 and 72 hours after exercise and then gradually declines (Armstrong, 1984; Miles & Clarkson, 1994). Nearly all healthy adults have experienced DOMS through involvement in unaccustomed physical activity (Armstrong, 1984). DOMS is most prevalent at the beginning of the sport season when athletes usually begin their training and also when a new type of training, to which the athlete is unaccustomed are introduced (Cheung et al., 2003). DOMS is different from temporary soreness. Temporary soreness is only felt during the final stages of fatiguing exercise and can be attributed to the accumulation of metabolic waste products, whereas DOMS is unrelated to fatigue and is described as a dull, aching pain combined with tenderness and stiffness (Armstrong, 1984; Byrnes & Clarkson, 1986).

Eccentric exercise causes greater DOMS than isometric or concentric exercise (Talaq, 1973). The exact cause of DOMS is unclear. DOMS was originally thought to be a result of the accumulation of lactic acid and other noxious waste products (Armstrong, 1984) or associated with local muscular spasm (Friden, Sjostrom, & Ekblom, 1981). However, the involvement of lactic acid in DOMS has been disproved based on the findings that eccentric exercise produces less lactate compared to concentric or isometric exercise (Schwane, Watrous, Johnson, & Armstrong, 1983). Muscle and/or connective damage theory (Friden et al., 1981; Jones & Round, 1990) and inflammation theory (Smith & Roberts, 1991; Stauber, Clarkson, Fritz, & Evans, 1990) are widely accepted as causative. The sensitisation of free nerve endings in response to inflammation and efflux of substances from muscle fibers into the extracellular space have been implicated as possible contributing factors of DOMS (Kendall & Eston, 2002; Miles & Clarkson, 1994).



The presence of DOMS may prevent the athlete from performing at their optimum levels and also may interfere with the performance of activities of daily living for the untrained individual. Furthermore, the decrease in force generating capability associated with DOMS will further reduce performance in high intensity exercise (Clarkson et al., 1992).

## 2.5 Differences between EIMD and DOMS

It is important to note that EIMD and DOMS do not necessarily reflect the same phenomenon. As described previously, EIMD is characterised by morphological alternations of normal muscle and/or connective tissue (Armstrong et al., 1991; Clarkson, 1997). EIMD is also associated by loss of muscle function (Warren, Ingalls, Shah, & Armstrong, 1999; Warren, Lowe et al., 1999). Other symptoms of EIMD include decreased range of motion and swelling, and abnormality in magnetic resonance or ultrasound images, and increases in muscle proteins in the blood (Byrne, Eston, & Edwards, 2001; Clarkson, Byrnes, Gillis, & Harper, 1987; Clarkson et al., 1992; Nosaka & Clarkson, 1995; Nosaka & Clarkson, 1996b; Nosaka et al., 2002a). DOMS is also often included in the symptoms of muscle damage (Byrnes et al., 1985; Close, Ashton, Cable, Doran, & MacLaren, 2004). However, DOMS does not necessarily accompany muscle damage. Furthermore, the level of DOMS is not indicative of the extent of tissue injury (Nosaka et al., 2002a). It should be also noted that the time course of DOMS is different from changes in muscle strength and ROM, upper arm circumference, and plasma CK activity (Nosaka et al., 2002a). Previous studies (Malm et al., 2000; Nosaka & Clarkson, 1996b; Nosaka et al., 2002a; Rodenburg et al., 1993; Rodenburg et al., 1994; Vincent & Vincent, 1997) reported either poor correlation between DOMS and other indicators of muscle damage such as strength loss, ROM and CK response, thus, it is better to differentiate between DOMS and EIMD.

## 2.6 Models of EIMD and DOMS

Many exercise models have been used to investigate EIMD and DOMS such as long distance running (Chosa et al., 2003; Vickers, 2001), downhill running (Donnelly, McCormick, Maughan, Whiting, & Clarkson, 1988; Eston, Finney, Baker, & Baltzopoulos, 1996), downhill walking or hiking (Farr et al., 2002), downhill backward walking (Weerakkody, Whitehead, Canny, Gregory, & Proske, 2001), stepping exercise (Allen et al., 2003; Hasson, Barnes, Hunter, & Williams, 1989), chest press (Smith, Fulmer et al., 1994), drop jumps (Horita, Komi, Nicol, & Kyrolainen, 1999; Semark, Noakes, Gibson, & Lambert, 1999), stretch shortening cycling (SSC) exercise (Nicol, Komi, Horita, Kyrolainen, & Takala, 1996), and cycling exercise (Byrne, Twist, & Eston, 2004; Gleeson, Blannin, Walsh, Field, & Pritchard, 1998).

Different muscle groups have also been examined in different models. Two muscle groups examined frequently are knee extensors (Lund, Vestergaard-Poulsen, Kanstrup, & Sejrsen, 1998; Saxton, Donnelly, & Roper, 1994; Soricther et al., 1995; Tiidus & Shoemaker, 1995) and elbow flexors (Brown, Child, Day et al., 1997; Brown, Child, & Donnelly, 1997; Croisier et al., 1996; Donnelly, Clarkson, & Maughan, 1992; Eston et al., 1996; Gregory, Morgan, & Proske, 2003; Smith, Keating et al., 1994). Other muscle groups that have been investigated include triceps surae or calf (Klass, Guissard, & Duchateau, 2004; Webster, Syrotuik, Bell, Jones, & Hanstock, 2002) hamstrings (Hilbert, Sforzo, & Swensen, 2003) and wrist (Miles, Clarkson, Bean et al., 1994).

Maximal eccentric exercise of the elbow flexors is the most widely used model to study EIMD and DOMS. Use of the same muscle groups, the intensity of the exercise, the number of repetitions, time, sets and rest, range of motion, and subjects used in the study are important factors to consider (Cleak & Eston, 1992; Farthing & Chilibeck, 2003; Paddon-Jones, Leveritt, Lonergan, & Abernethy, 2001). In the case of the elbow flexors, many protocols have been used (Brockett, Warren, Gregory, Morgan, & Proske, 1997; Clarkson & Tremblay, 1988; Newham, Jones, Ghosh, & Aurora, 1988; Nosaka & Newton, 2002b; Nosaka, Newton, & Sacco, 2002b, 2002c; Nosaka & Sakamoto, 2001; Saxton et al., 1995) such as 24 eccentric contraction (Gulbin & Gaffney, 2002; Nosaka & Newton, 2002a, 2002c), 50 maximal isokinetic eccentric contractions (Philippou, Bogdanis, Nevil, & Marikadi, 2004), 5 sets of 10 maximal isokinetic eccentric

contractions (Gleeson et al., 2003), 2 sets of 50 arm curls (Kraemer, Bush, Wickham, Denegar, Gomez, Gotshalk, Duncan, Volek, Newton et al., 2001), and 70 maximal voluntary eccentric muscle action (Saxton & Donnelly, 1995).

## 2.7 Markers of EIMD

Several direct and indirect indicators of EIMD have been described (Clarkson & Hubal, 2002; Foley, Jayaraman, Prior, Pivarnik, & Meyer, 1999; Hather, Mason, & Dudley, 1991; Nosaka & Newton, 2002b; Warren, Lowe et al., 1999). Direct assessment of damage in human muscle is only possible by muscle biopsies. Indirect measures such as magnetic resonance imaging (MRI) or ultrasound imaging can be used to visualise the sites of muscle damage (Newham, McPhail, Mills, & Edwards, 1983; Nosaka et al., 2002a). More commonly, EIMD is indirectly examined by using measures such as maximal voluntary strength, ROM, swelling and muscle protein levels in the blood (Clarkson et al., 1992; Warren, Lowe et al., 1999). The choice of indicators of muscle damage is dependent on the exercise model. Some indicators are suitable for upper extremity model only such as arm circumference, relaxed and flexed joint angles. For the eccentric exercise of the elbow flexors, most of previous studies included the measurements of muscle strength, relaxed and flexed elbow joint angles and range of motion, upper arm circumference and plasma CK activity (Nosaka & Clarkson, 1995; Nosaka & Clarkson, 1996a; Nosaka & Newton, 2002b, 2002c). In this review, most indicators discussed are predominately used for the elbow flexor models.

### 2.7.1 *Histological Changes*

Ultrastructural changes in muscles following eccentric exercise can be detected by electron microscopy, which shows myofibrillar disturbance of the contractile proteins characterised by Z-line streaming (Friden et al, 1981). Other noted changes include loss of thick myofilaments, swollen mitochondria or loss of mitochondria in abnormal injured area, disturbed arrangements of filaments at the A band (Clarkson & Hubal, 2002) and disruption of the plasma membrane (McNeil & Khakee, 1992). Eccentric exercise has been found to disrupt the arrangements of t-tubules and the disposition of triads such as increased number of longitudinal segments of the t-tubular network (Takekura & Yoshioka, 1990). At a light microscopic (histological) level, invasions of

mononuclear cells are observed both between and within degenerating fibres following eccentric exercise (Jones, Newham, Round, & Tolfree, 1986). Although biopsy is a direct measurement, the small size of tissue sample makes it difficult to quantify the magnitude of damage because the sample may miss, or over represent the damage of the whole muscle especially in human studies (Warren, Lowe et al., 1999).

### 2.7.2 *Magnetic Resonance Imaging (MRI)*

MRI is considered a powerful tool to understand what is happening in the entire muscle (Clarkson & Hubal, 2002). Muscle activation increases the MR (T2) signal intensity, which provides the recruitment images (Patten, et al. 2003). It has been documented that MRI can detect localisation of damage and is a promising alternative to evaluate muscle tissue, and changes in MRI T2 relaxation time is correlated with histological changes in muscle (Bosboom, et al. 2003). It has been shown that T2 relaxation time of muscle increases in damaged muscles for several days following eccentric exercise (Foley et al., 1999; Newham et al., 1987). The increase in T2 appears to arise from increases in extracellular fluid (Ploutz-Snyder et al., 1997) or increases in intracellular water and intracellular acidification (Patten et al., 2003). Nosaka & Clarkson (1996a) documented that the increase in T2 relaxation time was likely due to oedema. T2 relaxation time was reported to peak at 6 days following eccentric exercise, and coincided with CK responses (Clarkson & Hubal, 2002). Study by LeBlanc, Jaweed and Evans (1993) found high correlation (0.94) between peak T2 relaxation and serum CK activity following eccentric exercise. Thus, it appears that MRI is a powerful tool to investigate muscle damage; however, the cost involving in MRI is high.

### 2.7.3 *Strength Loss*

Following unaccustomed high intensity eccentric exercise, a marked reduction in strength is evident. Strength loss is generally observed immediately after exercise with significant decreases still evident in some cases for over two weeks (Newham, Jones, & Clarkson, 1987). The magnitude of strength loss immediately after high force eccentric exercise of the elbow flexors has been shown to be 50% to 70% (Clarkson, 1992; Nosaka & Clarkson, 1996a; Nosaka et al., 2002b; Saxton et al., 1995). The ability to produce maximal voluntary torque usually returns to baseline levels by two weeks or longer after eccentric exercise (Howell, Chleboun, & Conatser, 1993; Pearce et al., 1998).

Strength loss occurs for a number of reasons. One of the major contributors to force impairment is excitation-contraction (E-C) coupling failure. E-C coupling refers to the cascade of events starting with acetylcholine being released at the neuromuscular junction and ending with release of  $\text{Ca}^{2+}$  ions from the sarcoplasmic reticulum (SR). Reduced  $\text{Ca}^{2+}$  release by the SR may contribute to force reduction after eccentric exercise since  $\text{Ca}^{2+}$  plays a vital role in the coupling action between myosin and actin to produce muscle contraction (Warren et al., 2001).

Novel eccentric exercise may disrupt the contractile components of the muscle by producing higher forces and recruit fewer motor units (Astrand & Rodahl, 1997). Under these circumstances, force is spread over a small cross sectional area, which puts the active fibres under excessive strain (Jones & Round, 1990). This condition may have an effect on the fibres especially the Z-line (Friden et al., 1981) and lead to non-uniform overstretching of sarcomeres, resulting in A-band disruption, Z-band streaming, misalignment of myofibrils, and disintegration of the intermediate filament system (Armstrong, 1984; Stauber, 1989). Sarcomeres, which are not uniform in terms of fibre length, can result in shorter sarcomeres towards the end. If some central sarcomeres are pulled relatively further apart by the lengthening action, the overlap between actin and myosin filaments at this point would be reduced and may negatively affect the ability of the muscles to produce force (Clarkson et al., 1992)

#### *2.7.4 Range of Motion (ROM)*

ROM of the elbow joint can be determined by the difference between the flexed (FANG) and relaxed or stretched (SANG) elbow joint angle and has been shown to decrease immediately after novel eccentric exercise of the elbow flexor muscles, reaching the nadir around three days post exercise, slowly recovering over the next several days (Clarkson, 1992). Relaxed arm angle (RANG), determined by the angle at the elbow while the arm is hanging by the side of the body, is found to be at its most acute three days post exercise and also slowly recovering to baseline level over the following several days (Clarkson, 1992). Nosaka and Clarkson (1996a) reported a 30° decrease in ROM after maximal eccentric exercise of the elbow flexors. One of the

reasons in the decrease in ROM after unaccustomed eccentric exercise is likely due to the accumulation of fluid in the muscle (Howell et al., 1985). This in turn could affect the extensibility of connective tissue structures, connective tissue shortening and muscle contractures consequential to the loss of calcium homeostasis in damaged cells (Saxton & Donnelly, 1995).

#### *2.7.5 Limb Circumference and Swelling*

Following high intensity eccentric activity of the elbow flexors, circumference of the upper arm increases by 1 to 3 cm and remains elevated for up to three days post exercise (Clarkson, 1992; Howell et al., 1993). The reason behind this increase is not exactly known, but it has been suggested to be due to swelling within the affected muscle fibres (Crenshaw, Thornell, & Friden, 1994), swelling of the connective tissue (Clarkson, 1992) and an increase in both the intravascular and extravascular fluid contents as a result of cellular accumulation of metabolites or increased synthesis of connective tissue (Smith & Roberts, 1991). Smith (1991) has suggested that the swelling following eccentric exercise may result from increased permeability of small blood vessels which allow the protein rich fluid, known as exudate, to escape into the damaged area. Increases in upper arm circumference from 5 mm to 40 mm have been reported in previous studies (Kraemer, Bush, Wickham, Denegar, Gomez, Gotshalk, Duncan, Volek, Newton et al., 2001; Nosaka & Clarkson, 1996a; Nosaka et al., 2002b).

#### *2.7.6 Intracellular Protein Release*

A number of intracellular muscle proteins such as CK and lactate dehydrogenase (LDH) have been employed to indicate the presence of muscle injury (Schwane, Johnson et al., 1983). Because such intracellular protein molecules are too large to escape from muscle fibres unless the cell membrane is damaged, their increases in the blood are thought to be due to plasma membrane damage (Evans et al., 1986; Kuipers, 1994; Nosaka & Clarkson, 1996b; Sayers, Clarkson, & Lee, 2000b). CK is the most commonly employed intracellular protein used as a marker muscle damage (Armstrong, 1984; Thorsson et al., 1997). CK release often continues for several days after eccentric exercise (Stauber, 1989), peaking between three to seven days post exercise and slowly

returning to baseline levels thereafter (Newham, Jones, & Edwards, 1986; Nosaka, Clarkson, & Apple, 1992). Because CK displays a delayed increase (24 to 48 hour) in the blood, it has been suggested that CK gets into the central circulation from the lymphatic system (Nosaka & Clarkson, 1992). Activity of CK in the blood following unaccustomed eccentric activity has a large variability among individuals (Clarkson & Ebbeling, 1988). Some individuals show small increases after strenuous exercise while others produce an increase many thousands of times above resting level (Sayers et al., 2000b). In one study, intersubject variability in peak plasma CK activity ranged from 236 IU to 25,244 IU (Nosaka & Clarkson, 1996b).

### *2.7.7 Soreness and Tenderness*

Muscle soreness is commonly referred to as discomfort and unpleasantness experienced by the subjects after exercise (Armstrong, 1984). Soreness occurs upon contraction or palpation approximately 24 hours after exercise, peaks 24 to 48 hours after exercise and slowly dissipates but may not fully subside up to 8 to 10 days following exercise (Clarkson, 1992).

Because of its subjective nature, it is difficult to quantify soreness. Several scales to assess muscle soreness include visual analogue scale (VAS) (Ohnhaus & Adler, 1975), numerical rating scale (Strong, Unruh, Wright, Baxter, & Wall, 2002), verbal rating scale (VRS) (Strong et al., 2002), descriptors differential scale (DDS) (MacIntyre, Sorichter, Mair, Berg, & McKenzie, 2001; Wall & Melzack, 1994), and McGill pain questionnaire (MPQ) (MacIntyre et al., 1995; Perry, Heller, & Levine, 1991). Among these, VAS has been shown to be most suitable for assessing muscle soreness (Ohnhaus & Adler, 1975). Subjects using VAS are asked to place a single vertical mark on a horizontal line 50 or 100mm in length with 'no pain' at one end, and 'unbearably sore' at the other end, at a point corresponding to their current pain intensity (Perry et al., 1991). Studies on elbow flexor soreness following eccentric exercise have demonstrated soreness values 30 -40 mm on a scale 0 to 50mm (Nosaka & Newton, 2002b, 2002c, 2002d; Nosaka et al., 2002c).

Tenderness is the unpleasant sensation felt when pressure is applied to a damaged muscle (Eston et al., 1996). It is measured using a myometer being applied sequentially at the specific muscle sites (Newham, Mills, Quigley, & Edwards, 1983). As with soreness, tenderness was reported to increase from baseline to day 2 post exercise, decrease from day 2 to day 4 post exercise and return to baseline by day 7 post exercise (Eston et al., 1996). The pain threshold usually records the pressure applied in either Kilopascal (kPa) or newton (N).

## 2.8 Treatment of EIMD and DOMS

Treatments of DOMS and EIMD can be described as prophylactic or therapeutic categories depending on when the treatment is administered. Prophylactic treatments refer to all those modalities employed before exercise or development of symptoms of muscle damage. Basically, the purpose of the prophylactic interventions is to prevent or minimize the occurrence of DOMS and EIMD.

Therapeutic modalities refer to treatments employed after exercise or development of symptoms of EIMD and DOMS. Since study designs, including exercise protocols to induce EIMD and DOMS and variables to assess the efficacy vary, it is difficult to compare the effects of various therapeutic modalities among studies. Studies comparing different therapeutic interventions on an identical damage protocol are yet to be undertaken. Some interventions have been reported to have effects on DOMS and/or recovery of muscle function, however, contradictory results have often been found for the same interventions. It seems that no specific intervention has been shown to be very effective in reducing DOMS and symptoms related to EIMD.

## 2.9 Effect of Prophylactic Modalities on DOMS and EIMD

The prophylactic interventions that have been examined in relation to DOMS and EIMD include stretching (High & Howley, 1989; Johansson et al., 1999), warming-up (Rodenburg et al., 1994), taking anti-inflammatory drugs (Semark et al., 1999), nutritional supplements (Kaminski & Boal, 1992) and exercise (Costill et al., 1979). Since this study focuses on therapeutic treatments, only several key studies are introduced in the following sections.



### 2.9.1 *Stretching and Warming-up*

Stretching is performed widely before exercise and sports (De Weijer, Gorniak, & Shamus, 2003). Stretching exercises increase flexibility, thus decreasing the likelihood of musculoskeletal injuries (Safran et al., 1989). Several studies (Evans, Knight, Draper, & Parcell, 2002; High & Howley, 1989; Johansson et al., 1999) have investigated the effects of stretching performed before exercise on DOMS and EIMD. Various stretching protocols have been used in these studies. Johansson et al. (1999) reported that 4 sets of 20 second static stretching had no preventive effects on DOMS, tenderness, and force loss. High and Howley (1989) had four groups; control, warm-up only, stretching only and warm-up and stretching, perform a step test until exhaustion. No significant differences were identified for the development of DOMS between groups. Evans et al. (2002) in comparing several types of warm-up, including active warm-up, low and high heat passive warm-up before eccentric exercise of elbow flexors reported that passive warm-up was more beneficial than active warm-up or no warm-up in attenuating swelling. However, their results suggested that stretching was not as effective for other clinical symptoms of EIMD. No beneficial results of warm-up were reported for strength, ROM and DOMS.

Increasing muscle temperature through warming-up exercise speeds up metabolic process and produces faster contractions (Shellock & Prentice, 1985). Nosaka & Clarkson (1997) showed that warm-up exercise before eccentric exercise reduced the negative aspect of eccentric exercise. Zarins and Ciullo (1983) suggest that a 'cold' muscle make its elastic component more susceptible to injury. However, Nosaka et al. (2004) recently reported that altered muscle temperature, either 5°C colder or 3°C hotter than normal, did not affect the magnitude of EIMD and DOMS.

### 2.9.2 *Non-steroidal Anti Inflammatory Drugs (NSAIDs)*

NSAIDs are medications which have pain-relieving (analgesic) effects and reduce inflammation (Connolly et al., 2003). NSAIDs work by inhibiting the cyclo-oxygenase (COX) prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) synthesis (Connolly et al., 2003). NSAIDs such as aspirin, naproxen, flurbiprofen and ibuprofen are COX inhibitors, and others such as diclofenac and ketoprofen block both COX and lipooxygenase (LOX) pathways (Connolly et al., 2003). NSAIDs are among the most widely prescribed medication in

the field of physical medicine and rehabilitation (Lapointe, Fremont, & Cote, 2003). NSAIDs are commonly used to treat acute and chronic sports related injuries that occur in competitive as well as recreational athletes (Clayman, 1986; Weiler, 1992).

Jacobs et al. (1996) reported that intake of predonisone (50mg/kg body weight per day) was effective in reducing CK levels possibly by stabilizing the muscle fibre membranes. However, this result was obtained through study on rabbits and the effect cannot be generalised to human subjects. Semark et al. (1999) monitored the effect of flurbiprofen taken 12 hours before exercise on DOMS and sprinting performance after drop jumps exercise, and did not find any beneficial effects on DOMS, EIMD and sprinting performance. Other kinds of NSAIDs have been also investigated (Weiler, 1992; Lapointe et al., 2003); however, it appears that the effect of NSAIDs on DOMS and EIMD is minimal, if any

### *2.9.3 Nutritional Supplements*

There are many nutritional supplements available, but antioxidants are the most tested substance in the study of muscle damage. Antioxidant is a classification of several organic substances, including vitamins C and E, vitamin A (which is converted from beta-carotene), and a group known as the carotenoids. The supplements of antioxidant was believed to detoxify the peroxides produced during exercise and therefore may prevent muscle damage. Supplements of 3g vitamin C three times a day was found effective in reducing soreness (Kaminski & Boal, 1992). Supplement of Vitamin E was reported to attenuate CK activity in the blood (Cannon et al., 1990). However, other studies of supplementation of vitamin E found no significant effects on strength, CK (Warren, Jenkins, Packer, Witt, & Armstrong, 1992) and muscle function (Jakeman & Maxwell, 1993).

A study on L-carnitine conducted by Giamberardino et al. (1996) found that eccentric exercise performed after prolonged L-carnitine supplementation 3 weeks before the exercise significantly reduced pain and tenderness. Intake of either  $\beta$ -hydroxy or  $\beta$ -methylbutyrate (HMB) was found to significantly decrease the proteolysis during exercise (Nissen et al., 1996), reduced membrane damage during periods of high stress activity (Nissen & Abumrad, 1997) and blunted CK response (Panton et al., 2000). Although there was a number of studies (Giamberardino et al., 1996; Nissen &

Abumrad, 1997) claiming the beneficial effect of creatine supplementation on DOMS, the findings were not always consistent.

#### 2.9.4 *Exercise*

It is well documented that muscles adapt rapidly to eccentric exercise and become less susceptible to EIMD. This effect is often referred to as repeated bout effect (Nosaka & Newton, 2002d; Nosaka, Sakamoto, Newton, & Sacco, 2001). It is also known that the repeated bout effect is produced without significant damage, simply through exposure to a small number of eccentric contractions (Costill et al., 1979). Nosaka et al. (2001) revealed that only two maximal eccentric actions performed in the first bout elicited repeated bout effect in a subsequent bout of 24 maximal eccentric actions performed 4 weeks later.

Recent evidence suggests that prior concentric training increases the susceptibility of muscle to EIMD. Whitehead et al. (1998) showed that training the triceps surae muscle with 30 minutes of concentric exercise per day for 5 days increased the susceptibility of muscle to changes associated with subsequent eccentric exercise. This was supported by Gleeson et al. (2003) who found that concentric exercise prior to damage protocol produced greater reductions in isometric strength, arm circumference, soreness and relaxed arm angle (RANG). However Nosaka and Newton (2002) showed that 8 weeks of concentric training of the elbow flexors did not make the muscles more susceptible to muscle damage

#### 2.10 Effect of Therapeutic Modalities on DOMS and EIMD

Many therapeutic modalities have been used in previous studies, these interventions include taking pharmacological agents such as anti-inflammatory drugs (Almekinders, 1990; Donnelly et al., 1988), various therapies such as athletic massage (Smith, Keating et al., 1994) and ice massage (Isabell, Durrant, Myrer, & Anderson, 1992), stretching (Lund et al., 1998), immobilisation (Sayers et al., 2003), compression (Kraemer, Bush, Wickham, Denegar, Gomez, Gotshalk, Duncan, Volek, Putukian et al., 2001), exercise (Saxton & Donnelly, 1995), cryotherapy (Paddon-Jones & Quigley, 1997), acupuncture (Barlas, Robinson et al., 2000), electrical therapy (Denegar et al., 1989; Glasgow, Hill,

McKevitt, Lowe, & Baxter, 2001), ultrasound (Plaskett, Tiidus, & Livingston, 1999) and hyperbaric oxygen therapy (Webster et al., 2002). This section introduces several therapeutic treatments that have been investigated for their effects on DOMS, muscle function, and other markers of EIMD.

### *2.10.1 Massage*

Massage increases blood circulation, cellular permeability and elimination of lactate (Gupta, Goswami, Sadhukhan, & Mathur, 1996) and is likely to have psychological effects (Hemmings, Smith, Graydon, & Dyson, 2000). Massage has been one of the most popular interventions in sports physiotherapy and is used for the treatment of DOMS and EIMD (Robertson, Watt, & Galloway, 2004). Kresge (1988) stated that a post-exercise massage enhanced strength recovery from damage following an intense bout of eccentric exercise. Smith et al. (1994) found that athletic massage performed 2 hours post exercise was effective in reducing DOMS. A study by Farr et al. (2002) reported that 30 minutes of massage performed 2 hours post eccentric exercise on the quadriceps was effective in the attenuation of DOMS but not effective in the prevention of muscular strength and functional loss after downhill walking. On the other hand, Weber et al. (1994) applied 8 minutes of massage immediately and 24 hours post high intensity eccentric exercise and reported no beneficial effects on DOMS. Similarly Tiidus and Shoemaker (1995), who examined the effects of effleurage massage on leg muscles following an intense bout of eccentric exercise, found no effect on DOMS and strength recovery. Hilbert et al. (2003) reported massage performed 2 hours after leg exercise did not alter muscle function. Howatson and Van Someren (2003) also found no positive effects of massage performed immediately, 24 and 48 hours after eccentric exercise of the arm on DOMS. Ice massage was found to attenuate CK appearance but was not beneficial for DOMS, ROM, arm circumference and strength recovery. Rodenburg et al. (1994) showed that 15 minutes of massage after exercise with warm up and stretching prior to eccentric exercise reduced DOMS and produced positive effects on ROM and CK, however, it is not clear how much massage alone contributed to the effects.

The positive effects of massage could be explained by increased circulation and lymph flow as well as the relaxing of the involved muscles (Hemmings et al., 2000). Massage is believed to improve microcirculation (Gupta et al., 1996; Tiidus, 1999) which in turn helps to reduce oedema and accumulation of 'noxious substances' and enhance the regenerative process through increasing blood flow and oxygen delivery (Callaghan, 1993; Horsham, 2001; Lehn & Prentice, 1994; Lowdon, Mourad, & Warne, 1984)

During acute inflammation, blood flow slows as the vessels dilate in an area of injury (Smith, 1991). When this occurs, white blood cells, including neutrophils, are displaced from the central axial zone of blood flow to the peripheral zone, and subsequently margined along vessel walls (Smith, 1991). Since massage appears to increase blood flow through the vascular bed, it could affect the typical outward displacement of neutrophils and hinder the migration of the cells from the circulation into tissue spaces (Smith, Keating et al., 1994). However, a recent study of neutrophil count following massage by Hilbert et al. (2003) found that 20 minutes massage intervention following maximal eccentric exercise of hamstrings did not alter the circulating neutrophil count. Although massage has some potential in reducing DOMS (Farr et al., 2002) and other symptoms of muscle damage (Howatson & Van Someren, 2003), its effectiveness has not been demonstrated convincingly (Ernst, 1998). Based on previous studies, it can be concluded that massage as a therapeutic intervention demonstrates inconsistent results. These different results could be due to different muscles involved between studies. It is also possible that type, time of application and duration of the massage may influence the effects of massage as a therapeutic modality on DOMS and other symptoms of muscle damage.

### *2.10.2 Stretching*

It is generally believed that stretching after exercise as a cooling down exercise is beneficial for preventing DOMS and enhancing recovery. However, the effects of stretching following eccentric exercise has not been widely investigated, and only two studies have been published. Lund et al. (1998) investigated the effectiveness of passive stretching (3 times for 30s duration) of the quadriceps following eccentric exercise of knee extensors and found that the stretching exercise did not have any significant

influence on muscle pain. A recent study by Jayaraman et al. (2004) investigated topical heat and static stretching as therapeutic interventions on muscle damage. In their study subjects were assigned to four groups which were control, heat, stretching and combined heat and stretching. All subjects received the treatments 36 hours after completing intense eccentric knee extension exercise. No significant difference between groups was recorded for MRI, DOMS or swelling and muscle strength.

### *2.10.3 Immobilisation*

Complete rest is a popular recommendation among sports practitioners and athletes when injury occurs. This is due to a belief that further involvement in activities will induce further damage and retard recovery. Furthermore, it is the standard prescription for most musculo-skeletal injuries (Jarvinen & Lehto, 1993). Immobilisation is a treatment to minimise movements of the injured area. A few studies have been carried out to investigate the effect of immobilisation on DOMS and EIMD.

Sayers et al. (2003) examined the effects of short-term immobilisation treatment following eccentric exercise of the elbow flexors. Following eccentric exercise subjects had their elbow joint immobilized in a secured sling for four days. They reported that immobilisation improved the recovery of isometric strength, but not relaxed or flexed arm angle. Sayers and Clarkson (2003) also found that increases in plasma CK activity were blunted during immobilisation and speculated that overall lower peak CK in immobilisation conditions compared to a control condition could be due to inactivation of CK activity before entering the circulation.

From the same laboratory, another two studies concerning immobilisation treatments following eccentric exercise were conducted (Sayers et al., 2000a, 2000b). In these studies one group of subjects was assigned to immobilize the exercise arm in a cast and secure sling at 90° for four days post exercise. Interestingly, they found that recovery of maximal isometric force was facilitated by light exercise and immobilisation, and immobilisation delayed the recovery of soreness (Sayers et al., 2000a). They also found that immobilisation treatments blunted CK activity. Reduced CK activity in the immobilized arm could be due to a decrease in lymphatic transport with inactivity (Sayers et al., 2000b).

It has been also suggested that immobilisation should be applied immediately after damaging exercise (Clanton & Coupe, 1998). It is thought that this may help prevent the injury becoming more severe as early immobilisation promotes an increase in the connective tissue volume and number of muscle fibres (Kannus, Jozsa, Kvist, Jarvinen, & Jarvinen, 1998). A decrease in activity between 3 to 5 days following damaging eccentric exercise is beneficial to the recovery of muscle function (Sayers et al., 2000b). It was also suggested that it is effective to have early post immobilisation rehabilitation for patients with musculoskeletal injury (Kannus et al., 1998).

#### *2.10.4 Compression*

Compression is a common therapy commonly used to reduce tissue swelling following injury since it effectively reduces intramuscular blood flow (Thorsson et al., 1997). Kraemer et al. (2001) reported that compression was effective in reducing DOMS and facilitating recovery of muscle strength after eccentric exercise of the elbow flexors. In this study a compression garment was worn by subject for 24 hours a day for three days following exercise except the time for taking a shower. A different study from the same group (Kraemer, Bush, Wickham, Denegar, Gomez, Gotshalk, Duncan, Volek, Putukian et al., 2001) also reported that compression wore for five days after eccentric exercise of elbow flexors significantly reduced DOMS and swelling, attenuated the loss of motion and enhanced the recovery of muscle strength.

#### *2.10.5 Exercise*

Anecdotal evidence suggests that the feeling or sensation of pain and soreness is reduced when sore muscles are used, although more pain is induced at the beginning of activity. As stated earlier, exercise is one of the most effective strategies for alleviating DOMS (Armstrong, 1984) However, pain relief is also temporary and rapidly resumes again following exercise cessation (Smith & Roberts, 1991). This may be associated with the phenomenon of 'exercise-induced analgesia' (Koltyn 2000), however few studies have systematically investigated this phenomenon in relation to DOMS after eccentric exercise. Donnelly et al. (1992) reported that a light eccentric exercise performed one day after eccentric exercise of the elbow flexors did not affect DOMS.

In contrast, Saxton and Donnelly (1995) used sub-maximal (50%) concentric actions (5 sets of 10) performed daily following a bout of maximal eccentric exercise of the elbow flexors, and found that the light exercise caused a significant reduction in DOMS after two days. Nosaka and Newton (2002d) also reported a significant decrease in DOMS immediately after the repeated eccentric exercise bouts. However, the analgesic effect seems temporary and it is not clear whether exercise performed in the recovery days facilitates the recovery of muscle function and DOMS.

Few studies have evaluated the therapeutic effects of exercise on DOMS the recovery from eccentric exercise. Hasson et al. (1989) reported that a high speed voluntary contraction of quadriceps was effective in alleviating DOMS and facilitating recovery of muscle function after 120 voluntary maximum knee extensions at  $300^{\circ}\cdot\text{s}^{-1}$ . In contrast, Saxton and Donnelly (1995) showed no effect on indicators of muscle damage other than DOMS. Donnelly et al. (1992) reported that light eccentric exercise did not alter DOMS and muscle function following a bout of damaging exercise, but reduced or delayed increases in serum CK activity. Similarly, Weber et al. (1994) found no significant effect of 8 to 10 minutes upper arm ergometry on DOMS and muscle function applied immediately and 24 hours after eccentric exercise of the elbow flexors. These contrasting findings may be related to the different protocols, types of exercise, timing and intensity of exercise used. Activities undertaken during the recovery period from a damaging exercise bout do not seem to have a negative effect on the recuperative process. However, whether such exercise is beneficial in reducing DOMS and enhancing recovery of muscle function needs to be confirmed. Gurevich et al. (1994) in evaluating the influence of exercise in pain perception and pain tolerance stated that moderate exercise consisting of 12 minutes climbing a double step at an average intensity of 63%  $\text{VO}_2$  max results in beneficial effects in pain perception and pain tolerance.

Sayers et al. (2000a) reported that exercise with 50 biceps curls using a 5 lb dumbbell was effective for recovery of maximal isometric force and DOMS following eccentric exercise of the elbow flexors. They suggest that an increase in blood flow may be responsible for the effects.



Sorichter et al. (1995) found that additional concentric contractions during the recovery period after a bout of heavy eccentric exercise had no apparent effect on the degree of EIMD and breakdown of connective tissue but resulted in a five-fold, statistically significant increase in serum CK levels in comparison to the concentric group.

Dannecker et al. (2002) investigated the influence of endurance exercise of 20 minutes cycling at approximately 60 RPM on a cycle ergometer on DOMS. In their study DOMS was induced through eccentric exercise on the non dominant arm elbow flexors. Cycling exercise introduced in this study as an endurance exercise as a therapeutic treatment showed no significant effects on DOMS compared to the control condition.

#### *2.10.6 Non-steroidal Anti Inflammatory Drugs (NSAIDs)*

As described previously, NSAIDs are effective for reducing pain and inflammation (Connolly et al., 2003). If DOMS is associated with inflammatory responses, it seems reasonable to assume that NSAIDs attenuate DOMS and enhance recovery from EIMD. Several studies have examined the effect of NSAID on DOMS and other parameters associated with muscle damage after eccentric exercise.

One animal study reported that diclofenac sodium was beneficial for recovery of maximal force after eccentric exercise (Lapointe et al., 2003). In this study two groups of rats received diclofenac sodium either before and after 450 eccentric contractions of the ankle dorsiflexors. This effect does not appear to occur in humans. Donnelly et al. (1988) reported that diclofenac tablets did not affect overall soreness following 45 minutes downhill running, when 50mg of the diclofenac tablet was taken one and half hours before and every eight hour interval until 72 hours post exercise. Howell et al. (1998) also found no significant effects of ibuprofen and flurbiprofen intake (1600 mg and 3200 mg per day for 6 days) on DOMS, swelling and stiffness following eccentric exercise of the elbow flexors.

The effect of aspirin has also been examined. Francis and Hoobler (1987) reported significant decrease in soreness in subjects who took 10 grains (approximately 0.65grams) of aspirin four times a day compared to control group following eccentric exercise of the elbow flexors, however, maximal elbow flexors torque and ROM were not significantly affected. Barlas et al. (2000) investigated the effectiveness of oral supplementation of aspirin, codeine and paracetamol on DOMS after eccentric exercise of the elbow flexors, and found no effects.

Iontophoresis is a process that uses bipolar electric fields to propel molecules across intact skin and into underlying tissue. Hasson et al. (1992) evaluated the effect of dexamethasone iontophoresis on DOMS produced by bench stepping eccentric exercise. They reported that dexamethasone iontophoresis was not beneficial on DOMS and recover of muscle function of the knee extensors

Lecomte et al. (1998) showed that taking 1000 mg naproxen on the day of exercise to 7 days post exercise enhanced recovery of force and reduced DOMS after repeated bout of eccentric exercise of the knee extensor. Ibuprofen was taken every 8 hours for 48 hours after isotonic eccentric leg curl exercise was found to decrease DOMS and blunt CK responses, but did not affect strength, ROM and vertical jump performance (Tokmakidis, Kokkinidis, Smilios, & Douda, 2003).

#### *2.10.7 Cryotherapy*

Cryotherapy, or cold therapy, is a technique to reduce pain and swelling after injury by the use of ice or cold temperature. Applying ice to cure the injured soft tissue is a treatment method that has been extensively used for managing a wide variety of injuries such as strains, sprains, contusions, fractures and inflammatory conditions (McMaster, 1977) and is considered effective in ameliorating soft tissue injury (Gulick & Kimura, 1996), pain (Isabell et al., 1992), and reduce swelling (Connolly et al., 2003; Swenson, Sward, & Karlsson, 1996).

In a study by Yackzan et al. (1984), ice massage was applied for 15 minutes immediately after exercise, 24 and 48 hours following eccentric biased exercise of the elbow flexors. The results of the study indicated that the ice massage was not effective in decreasing DOMS. Isabell et al. (1992) reported that ice massage applied to subjects who performed 300 concentric/eccentric contractions of the elbow flexors did not have positive effects on strength, ROM, and DOMS. This finding was also supported by Paddon-Jones and Quigley (1997) who found no significant effect of 20 minutes ice immersion/bath of the exercised arm on muscle soreness and strength after following eccentric exercise of the elbow flexors. Eston and Peter (1999) had their subjects immerse their exercise arm in cold water (15°C) for 15 minutes immediately after eccentric exercise of the elbow flexors and reported positive effects on ROM but no effects on tenderness and strength.

#### *2.10.8 Accupuncture*

Acupuncture is defined as a penetration of the skin with sharp objects, usually stainless steel needles, which are used to stimulate the tissue either manually, electrically or by heat (Knardahl, Elam, Olausson, & Wallin, 1998). Several clinical trials have shown the effectiveness of acupuncture to relieve pain (Pomeranz, 1996). It is believed that the acupuncture needle stimulates the high threshold, small diameter nerves in the muscle, which send messages to the spinal cord and activate the spinal cord, midbrain and pituitary gland to release neurochemicals (endorphins and monoamines) to block the sensation of pain. Application of acupuncture on DOMS and EIMD is limited and only two studies (Barlas, Robinson et al., 2000; Lin & Yang, 1999) are available to date. Barlas et al. (2000) found little effect on DOMS when acupuncture was applied to the traditional acupuncture points using stainless steel disposable needles after eccentric exercise using dumb-bell and free weights. Lin and Yang (1999) applied acupuncture treatments to subjects with muscle soreness following eccentric exercise and found that acupuncture was effective in decreasing muscle soreness but did not affect CK release in the blood. Further studies are necessary to investigate the effects of acupuncture on DOMS and recovery of muscle function after EIMD.

#### *2.10.9 Transcutaneous Electrical Nerve Stimulation (TENS) and Others*

Electrical stimulation is commonly used in pain management (Holcomb, 1997). It was suggested that electrical stimulation releases the body's natural pain killers such as  $\beta$ -endorphin and methionine enkephaline into the cerebrospinal fluid which have an effect similar to morphine (Holcomb, 1997). Two studies have examined the effect of TENS on DOMS and EIMD. Denegar et al. (1989) examined the effect of low frequency 300 second pulse with TENS on EIMD following eccentric exercise of the elbow flexors and reported that TENS reduced pain and improved ROM. Weber et al. (1994) failed to find any beneficial effects of microcurrent electrical stimulation at 30 Hz, 30 $\mu$ A gentle wave slope (0.5 s) on DOMS and maximal voluntary isometric strength. An electro-membrane microcurrent therapy has been used to treat postoperative pain and soft tissue injury. Lambert et al. (2002) investigated the effects of this therapy on symptoms of muscle damage. In this study non-dominant arm muscle was damaged using eccentric protocol consisted of 5 sets of 25 eccentric actions and the arm was covered by electrostatically charged membrane for 96 hours. The therapy was found to reduce peak plasma CK and to improve ROM, however did not provide significant effects on DOMS, swelling and force recovery.

#### *2.10.10 Ultrasound*

Ultrasound has been used for soft tissue injuries, scar tissue, musculoskeletal pain, arthritic conditions, and chronic oedema based on the belief that it decreases symptoms of inflammation (pain and oedema) and increases the rate of healing (Craig, Bradley, Walsh, Baxter, & Allen, 1999; Roebroek et al., 1998).

The ultrasound treatment has been reported to influence a number of physiological responses that might accelerate healing of muscle and connective tissues after EIMD (Tiidus, Cort, Woodruff, & Bryden, 2002). Craig et al. (1999) reported both low and high dosage pulsed ultrasound applied to elbow flexors following eccentric exercise was effective in improving ROM but not beneficial for DOMS. Studies conducted by Tiidus et al. (2002) and Plasket et al. (1999) also showed no significant effects.

Positive findings of ultrasound however were reported by Hasson et al. (1990). They found that ultrasound treatments applied 24 hour following leg eccentric exercise accelerated recovery of muscle strength and decreased DOMS.

#### *2.10.11 Laser Therapy and Infrared Therapy*

Laser therapy has been promoted as an effective treatment for a myriad of conditions including acceleration of wound healing and the relief of pain (Craig, Barron et al., 1999). However, Craig et al. (1999) examined the effect of low intensity laser therapy/phototherapy on symptoms of EIMD over 11 days following 'preacher bench' eccentric exercise and found no significant effects.

Monochromatic infrared therapy is a treatment where subjects received active irradiation (Glasgow et al., 2001). Glasgow et al. (2001) examined the effect of the infrared therapy on DOMS and muscle function following eccentric exercise of the elbow flexors. They reported no significant effect of 5 minutes irradiation on DOMS and recovery of isometric strength and resting arm angle (Glasgow et al., 2001).

#### *2.10.12 Hyperbaric Oxygen Therapy (HBOT)*

HBOT is used in a sports medicine setting to reduce hypoxia and oedema, and appears to be particularly effective for treating crush injuries, and facilitating soft tissue injuries (Delaney & Montgomery, 2001). During HBOT, patients breath 95% to 100% oxygen at pressures above 1.0 atmosphere absolute (ATA) (Delaney & Montgomery, 2001). Normally 97% of the oxygen delivered to body tissue is bound to haemoglobin, while only 3% is dissolved in the plasma. During HBOT, barometric pressure is usually limited to 3 ATA or lower. The combination of increase ATA and increased oxygen concentration dissolves enough oxygen in the plasma alone to sustain life in a resting state. Increased oxygen delivery to the tissue with HBOT may prevent tissue damage by decreasing the tissue lactic acid level and by helping maintain the ATP level. HBOT is thought to facilitate the soft tissue healing following injury.

Effects of HBOT on DOMS and muscle damage have been investigated by several researchers (Babul, Rhodes, Taunton, & Lepawsky, 2003; Harrison et al., 2001; Mekjavic, Exner, Tesch, & Eiken, 2000; Staples, Clement, Taunton, & McKenzie, 1999; Webster et al., 2002). Harrison et al. (2001) induced muscle damage to the elbow flexors by repeated eccentric muscle actions and examined the effect of HBOT treatment performed at 2 or 24 hours post exercise for 4 consecutive days. They showed that HBOT was not effective for DOMS, CK and recovery of isometric strength. Webster et al. (2002) examined the effect of HBOT following strenuous eccentric exercise of calf muscle, and found faster recovery of isometric peak torque and reduced pain sensation. Babul et al. (2003) reported that intermittent HBOT had no effect on DOMS, strength, circumference, CK, malondialdehyde and MRI. Mekjavic et al. (2000) also failed to find significant effects of HBOT on recovery of maximal isometric strength, swelling and muscle soreness. Another study (Staples et al., 1999) reported positive findings of HBOT on EIMD, in which they showed a faster recovery of eccentric torque of the quadriceps.

#### *2.10.13 Combination Treatments*

Combination of treatments may be more effective than each treatment alone. This is because some interventions are beneficial to a certain symptom of EIMD or DOMS and other interventions may simply be superior to or potentiate the effects of the first intervention. The combination of warm up, stretching and massage resulted in reduced DOMS, positive effects on CK, ROM and strength recovery (Rodenburg et al., 1994). However combination of exercise and ultrasound (Van Der Windt et al., 1999) did not produce any significant effects. It may be also possible that any effect is reduced by combining two or more treatments.

### 2.11. Summary of Therapeutic Effects on DOMS and EIMD

As summarised in Table 1, controversy exists concerning the efficacy of a therapeutic treatment on DOMS and/or indicators of EIMD, and the findings are inconsistent. In the total of 48 studies reviewed on Table 1, 31 studies included both DOMS and muscle function measurements. Among 44 studies examining DOMS, approximately half of the studies reported that DOMS was reduced significantly by treatment. For the recovery of muscle strength, 21 out of 33 studies in which included a muscle strength measurement reported no significant effect, and 11 studies reported faster recovery of strength. Only 14 studies investigated changes in ROM, and 9 studies found no effects. CK was included in the measurements by 20 studies, and 10 studies found smaller increases in the treatment condition. Limb circumference was measured by 8 studies, and 2 studies reported that the increase in limb circumference was attenuated by treatment. It is important to investigate effectiveness of therapeutic intervention on DOMS and muscle function, since some of them may only work for DOMS or recovery of muscle function. It should be noted that no treatment has been proven to be effective for ameliorating DOMS and enhancing recovery of muscle function.

Table 1:

*Summary of the Effect of Therapeutic Interventions of DOMS and EIMD*

Interventions	Authors (year)	Exercise Model	DOMS	Muscle function		Other measures	
				ST	ROM	CK	CR
Massage							
	Weber et al. (1994)	EF	no	no	x	x	x
	Smith et al.(1994)	EFE	*	x	x	*	x
	Tiidus & Shoemaker (1995)	KE	*	no	x	x	x
	Farr et al. (2002)	DW	*	no	x	no	x
	Howatson & Someren (2003)	EF	no	no	no	*	no
	Hilbert et al. (2003)	Hams	*	no	no	x	x
Stretching							
	Lund et al. (1998)	KE	no	no	x	no	x
	Jayaraman et al. (2004)	KE	no	no	x	x	x
Immobilisation							
	Sayers et al. (2000)	EF	#	*	x	x	x
	Sayers et al. (2000)	EF	x	x	x	x	x
	Sayers et al. (2003)	EF	x	*	*	x	x
	Sayers & Clarkson (2003)	EF	x	x	x	*	x
Compression							
	Kraemer et al. (2001a)	EF	*	*	#	*	*
	Kraemer et al. (2001b)	EF	*	*	#	no	*
Exercise							
	Hasson et al. (1989)	Bench S	*	*	x	x	x
	Donnelly et al. (1992)	EF	no	no	no	*	x
	Weber et al. (1994)	EF	no	no	x	x	x
	Sorichter et al. (1995)	KE	x	x	x	#	x
	Saxton & Donnelly(1995)	EF	*	*	no	*	x
	Dannecker et al. (2002)	EF	no	x	x	x	x
NSAIDs							
	Francis & Hoobler (1987)	EF	*	x	x	x	x
	Hasson et al.(1992)	BenchS	no	no	x	x	x
	Howell et al. (1998)	EF	no	#	x	x	x
	Lecomte et al. (1998)	KE	no	*	x	x	x
	Donnelly et al. (1998)	DR	*	x	x	no	x
	Barlas et al. (2000)	EF	no	x	x	x	x
	Tokmakiddis et al. (2003)	KE	*	no	no	*	x
Cryotherapy							
	Yackzan (1984)	EF	no	x	x	x	x
	Isabell et al.(1992)	EF	no	no	no	no	no
	Paddon jones & Quigley (1997)	EF	no	x	x	x	x
Acupuncture							
	Barlas et al.(2000)	EF	*	x	x	x	x
	Lin & Yang (1999)	EF	*	x	x	no	x
TENS							
	Denegar et al. (1989)	EF	*	x	#	x	x



	Weber et al. (1994)	EF	no	no	x	x	x
	Lambert et al. (2002)	EF	no	no	*	*	no
	Glasgow et al.(2001)	EF	no	x	x	x	x
Ultrasound							
	Plasket et al. (1999)	KE	no	no	x	x	x
	Hasson et al. (1990)	KE	*	*	x	x	x
	Craig et al. (1999)	EF	no	no	x	x	x
	Tiidus et al. (2002)	EF	no	no	no	x	x
Laser therapy							
	Craig et al. (1999)	EF	no	x	x	x	x
HBO							
	Harrison et al. (2001)	EF	no	no	x	no	x
	Webster et al. (2002)	Gastr	*	*	x	x	x
	Babul et al. (2003)	KE	no	no	x	no	no
	Mekjavic et al. (2000)	EF	no	no	x	x	no
	Staples et al. (1999)	KE	no	*	x	x	x
Combination							
	Rodenburg et al. (1994)	FF	*	*	*	*	x
	Howatson and Van Someren (2003)	EF	no	no	no	*	no

MF = Muscle Function, ST = Strength, RM = ROM, CK = Creatine Kinase, CR = Circumference

EF = Eccentric exercise of the elbow flexors, EFE = Eccentric exercise of the elbow flexors and extensors, KE = Eccentric exercise of the knee extensors, Hams = Eccentric exercise of hamstring, Gastr = Eccentric exercise of gastrocnemius, BenchS = Bench Stepping, DW = Downhill walking, DR = Downhill running

\*: positive effect, #: negative effect, no: no effect, x: not included in the study

## 2.12 Conclusion

EIMD prevents athletes from competing and performing optimally, and stimulating muscles optimally in training (Clarkson et al., 1992). DOMS and EIMD also interferes with activities of daily living and may discourage people from exercising on a regular basis. Therefore, effective therapeutic treatments for DOMS and/or recovery of muscle function are important.

Many prophylactic (Gleeson et al., 2003; Nissen et al., 1996) and therapeutic treatments (Hasson et al., 1989; Howel et al., 1998) have been examined for their effects on DOMS and EIMD, however, most treatments have not had their efficacy confirmed. It appears that prophylactic treatments are generally more effective than therapeutic approaches for preventing and reducing DOMS and EIMD, however, therapeutic treatments are of importance for people who want to minimize the adverse effects of unaccustomed exercise. Although some studies have shown beneficial effects of therapeutic treatments on DOMS and/or recovery of muscle function, no treatments have been proven to be effective because of contradictory findings. Therefore, all therapeutic treatments warrant further studies to confirm their effects on DOMS and EIMD.

Studies especially designed to examine the effect of immobilisation and exercise is limited. It is still not clear whether muscle suffering from DOMS and EIMD should be rested or actively utilized to reduce DOMS and facilitate recovery. Although several studies have investigated the effect of massage (Weber et al., 1994; Rodenburg et al., 1994), controversy exists about the effects of massage on DOMS and recovery of muscle function. These need to be investigated systematically. Since most of the physical therapies such as ultrasound, infrared, electrical stimulation, and hyperbolic therapies require expensive equipments that are usually found in special facilities, the accessibility may not be good for people who have only DOMS and minor symptoms of EIMD. On the other hand, massage can be performed relatively easily. Immobilisation, especially without use of a cast, can be done more easily. Exercise, if it is effective, is not difficult to perform without requiring expensive equipment. Therefore, for practical reasons, it seems reasonable to choose massage, immobilisation, and exercise in this study to investigate their effects on DOMS and EIMD.

## **CHAPTER 3**

### **METHODS**

#### **3.1 Study Design**

The studies described in this thesis investigated the effects of three therapeutic interventions, massage, immobilisation, and light concentric exercise, on DOMS and indicators of muscle damage responses to a bout of maximal eccentric exercise. Each intervention was examined separately using a 'limb to limb' comparison model in which one arm received a treatment and the contralateral arm was used as a control. A different group of subjects was used for each study, however, the subjects were recruited from the same population and had similar characteristics. The same eccentric exercise protocol and criterion measures were used in all three studies. Therefore, the methods are presented in a common chapter.

The arm to arm model required subjects to perform two bouts of maximal eccentric exercise of the elbow flexors (one for each arm). This exercise model was chosen because it has been used in previous studies and has been shown to induce appreciable muscle soreness and large decreases in muscle function (Clarkson et al., 1992; Donnelly et al., 1992; Nosaka et al., 2002a; Nosaka & Sakamoto, 2001). With this model, maximal isometric strength of the elbow flexors, range of motion (ROM) at the elbow joint, upper arm circumference, muscle proteins in the blood, and subjective measures of muscle soreness and muscle tenderness have been used as indicators of muscle damage. Within group changes in static and dynamic elbow flexor strength, elbow joint ROM, upper arm circumference, plasma creatine kinase (CK) activity, and muscle soreness and tenderness following exercise were compared between treatment and control arms for each intervention. The same criterion measures were used for comparisons between groups and among interventions.

Study 1 (Chapter 4) investigated the effect of immobilisation of the elbow joint for four consecutive days initiated 30 minutes post exercise. In Study 2 (Chapter 5), the effect of a single session of sport massage performed three hours after exercise, was investigated. Lastly, the effect of light concentric exercise performed on each of four

consecutive days after eccentric exercise was examined in Study 3 (Chapter 6). The timing of the interventions was chosen based on previous studies (Sayers, et al. 2003; Saxton & Donnelly, 1995 and Tiidus, et al. 1997). Muscle soreness peaks 1-3 days post exercise (MacIntyre, et al. 1995), therefore it is more practical to apply the interventions during this period.

It is well known that responses to eccentric exercise vary widely between subjects (Clarkson et al., 1986; Clarkson & Ebbeling, 1988; Foley et al., 1999; Whitehead et al., 1998). The arm to arm comparison model reduces the potentially large between-subject variability, since the control and experimental data are derived from the same subject. This model is also advantageous as it allows comparisons in a relatively small number of subjects. Both arms underwent identical eccentric exercise, so any differences observed between them were likely to be due to the effect of the treatment.

A previous study (Sayers et al., 2003) considered that a 10% difference between control and treatment for strength recovery was physiologically significant. Using this information, a power calculation of the estimated sample size was carried out using a two tailed design, an alpha of 0.05, and power level of 0.8. This indicated an effective sample size of >9 for each study. Also, previous studies of this type found significant effects with similar subject numbers used in this study (Farr et al., 2002; Hasson et al., 1989; Hemmings et al., 2000; Robertson et al., 2004; Saxton & Donnelly, 1995). Using the same power calculation, it was shown that the estimated sample size for ROM, plasma CK activity, and muscle soreness was also less than 10. However, larger sample size will be beneficial for future studies to investigate the effects of other interventions.

The experimental period consisted of two blocks of testing over 8 days, with at least one familiarisation session. Subjects underwent the exercise one day after the familiarisation session. Identical eccentric exercise was performed for both arms with at least 2 weeks between conditions. Measurements were taken prior to and immediately after eccentric exercise, and on days 1, 2, 3, 4 and 7 post-exercise.

### 3.2 Subjects

Volunteers were recruited for each study via advertisements using notice board flyers (Appendix A) from students and staff of Edith Cowan University, and from the general community around the university. Ethical approval was obtained from Edith Cowan University Human Research Ethics Committee. Subject age ranged between 18 and 35 years old and any individuals with prior or current upper arm injury were excluded. Subjects had not been involved in resistance training for at least previous six months. A total of 34 subjects (20 males and 14 females) were recruited for the three studies. Although there may be sex-based differences in response to eccentric exercise induced muscle damage (Clarkson & Hubal, 2002), controversies exist concerning the effects of sex on the magnitude of muscle damage. For generalisation, both genders were used as subjects for the studies. The mean  $\pm$  SD for age, weight and height were  $24.2 \pm 2.4$  yrs,  $67.2 \pm 13.7$  kg, and  $164.5 \pm 11.8$  cm, respectively. Subjects gave written informed consent (Appendix B) before participating in the study, and were screened via a medical questionnaire (Appendix C).

All subjects were free from any musculoskeletal disorders and in good health during the experimental period. Subjects were requested not to change their pattern of nutrition and not to take any medication, non-steroidal anti-inflammatory drugs (NSAIDs), and nutritional supplements. They were also asked to refrain from any recreational exercise other than that prescribed by the researcher for the eight days following each exercise intervention.

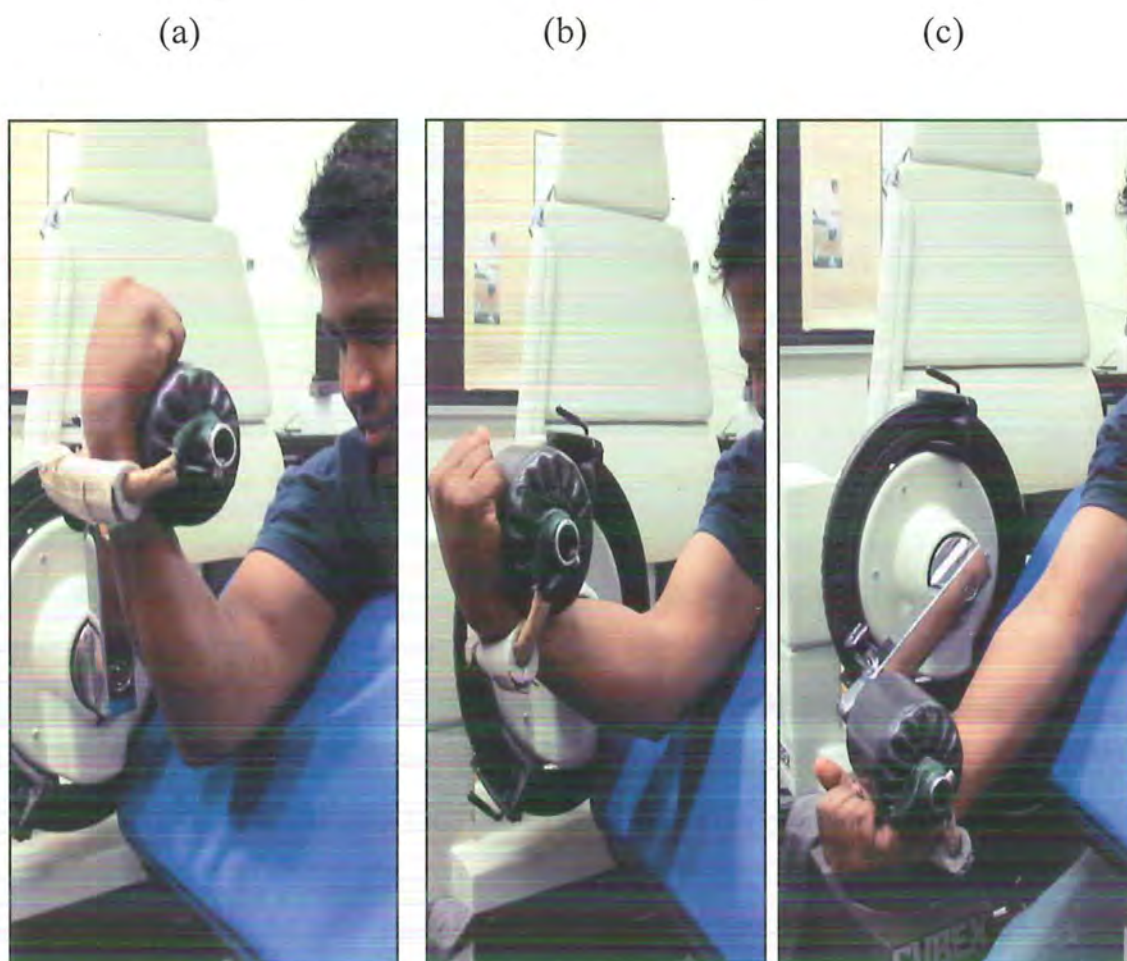
### 3.3 Pre-exercise Test Familiarization

Two sessions of a pre-exercise test familiarization were conducted, where subjects were familiarised with the testing protocols. Static and dynamic elbow strength was assessed and range of motion (ROM), upper arm circumference, plasma creatine kinase activity (CK), and soreness and tenderness were measured (detailed in later sections).

### 3.4 Exercise Protocol

The eccentric exercise protocol consisted of 10 sets of 6 maximum voluntary lengthening actions of the elbow flexors against the lever arm of the Cybex isokinetic dynamometer (Cybex 6000, Ronkonkoma, NY, U.S.A.) moving at a constant velocity of  $90^{\circ}\cdot\text{s}^{-1}$ . Subjects were seated on an arm curl bench with the forearm in a supinated position and the elbow aligned with the axis of rotation of the dynamometer lever arm (Figure 1). The movement began at an elbow joint angle of  $90^{\circ}$  (where full elbow extension was considered as  $0^{\circ}$ ). The elbow joint was forcibly extended from the flexed position ( $90^{\circ}$ ) to the extended position ( $0^{\circ}$ ) in one second while the subject was asked to maximally resist against the motion. After each eccentric action, the lever arm of the isokinetic dynamometer returned to the starting point at a velocity of  $9^{\circ}\cdot\text{s}^{-1}$  while subjects relaxed the arm, so that 10 seconds of passive recovery period was allowed between eccentric repetitions. Sixty actions were carried out in total divided into 10 sets, with 3 min rest between each set. Torque outputs were recorded and displayed in real time for each eccentric action and the data was saved on an IBM desktop computer operating AMLAB (version II, Lewisham, Australia) data acquisition software.





*Figure 1: Eccentric exercise protocol. Each eccentric action started from the position (a) followed by (b) and finished at (c) position.*

### 3.5 Criterion Measures

The aforementioned criterion measures (see Chapter 2) have been widely used as markers of muscle damage in many studies eg. (Clarkson et al., 1992; Nosaka & Clarkson, 1996a; Nosaka et al., 2002b). However, it should be noted that the measures used in this study were indirect markers of muscle damage. Warren, Lowe and Armstrong (1999) suggested that strength was the best measure of muscle injury resulting from eccentric exercise. ROM can be also used as a marker of muscle function. Increases in arm circumference represent swelling in response to inflammation (Chleboun, Howell, Conatser, & Giesey, 1998) and CK activity was used as indirect evidence of muscle membrane damage. This study also included muscle tenderness assessment. Tenderness differs from soreness in that muscle soreness

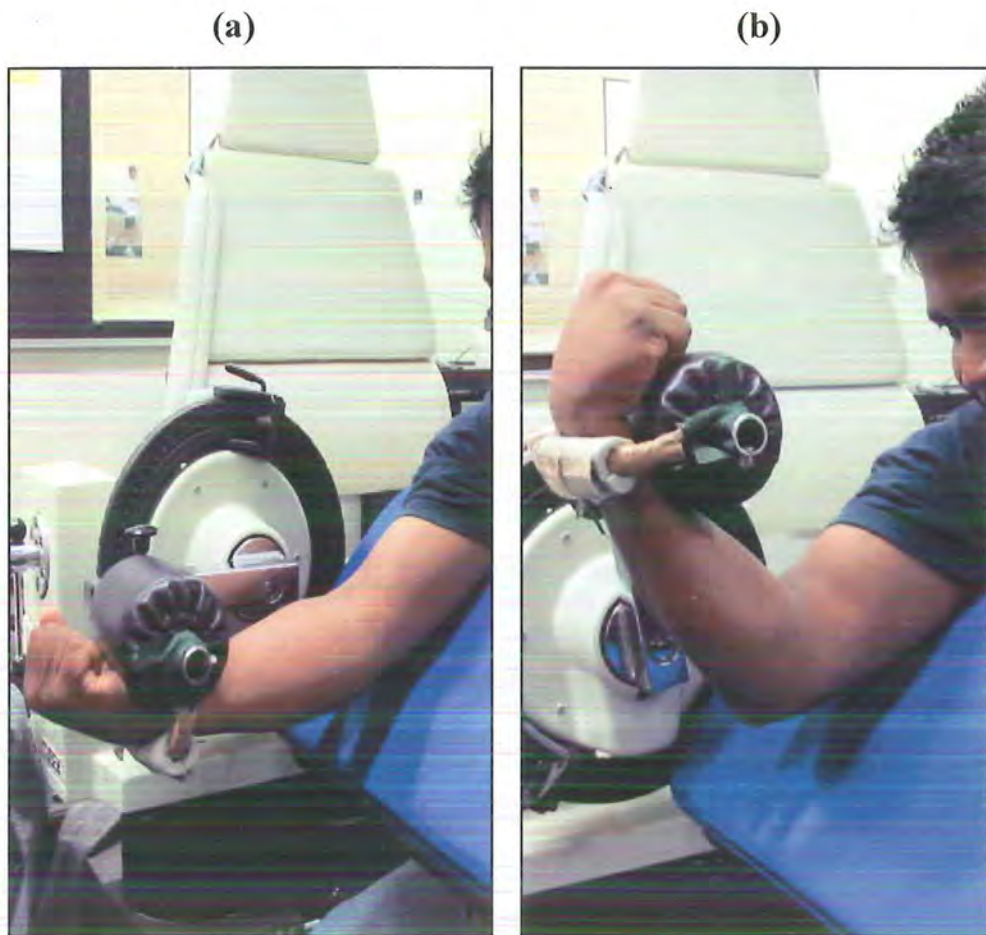
measures the response to palpation or movement, while muscle tenderness represents the pain threshold as pressure is applied to a specific area.

### 3.5.1 Muscular Strength

An isokinetic dynamometer (Cybex 6000, Ronkonkoma, NY, U.S.A.) was used to measure isometric and isokinetic concentric torque output during maximal voluntary contractions of the elbow flexors. Verbal encouragement was given during all recordings. For the isometric contractions, subjects were asked to sustain maximal effort for four seconds at a fixed elbow joint angle of 30° (Figure 2a) and 90° (Figure 2b) where a full extension of the elbow joint was considered 0°. Two measurements were performed at each joint angle. Torque outputs were recorded and displayed in real time for each contraction in the AMLAB and the highest peak torque value was used for subsequent analysis. The rest between each maximal isometric contraction was 30 seconds, and a one minute recovery period was allowed between testing at different joint angles.

Concentric maximal voluntary torque of the elbow flexors was assessed isokinetically at five different velocities; 30°·s<sup>-1</sup>, 90°·s<sup>-1</sup>, 150°·s<sup>-1</sup>, 210°·s<sup>-1</sup>, and 300°·s<sup>-1</sup> with the same positioning as the isometric assessment and a 90° range of motion from an elbow extended (0°) to a flexed (90°) position. The isokinetic strength testing was carried out in order of increasing velocity from 30°·s<sup>-1</sup> to 300°·s<sup>-1</sup> with the highest peak torque value of two trials used for subsequent data analysis. A five second period was provided between attempts at a given velocity and a 1-minute recovery period between each different velocity. The average peak torque of the two contractions at each velocity was used for analysis. Comparison between baseline pre-exercise data showed no learning effects to the criterion measures.





*Figure 2 : Maximal isometric torque measurement at two different angles, 30° (a) 90° (b).*

### 3.5.2 Muscle Soreness and Tenderness

Muscle soreness was rated by subjects on a visual analogue scale (VAS) with the researcher palpating, extending and flexing the subject's arm. The VAS incorporates a 100 mm line marked with 0 indicating no soreness, and 100 at the other end representing very painful. The subject marked on the line along the scale that coincides with their perceived level of soreness. The distance from 0 in millimetres was measured and the numerical result recorded. The palpation was applied by index and middle fingers of the same investigator each time, and the pressure on the marked sites was kept as consistent as possible between trials (Figure 3a). Muscle soreness was evaluated by palpating four marked sites (between 3-5 cm above elbow crease on the biceps brachii, lateral site of brachialis and brachioradialis) (Figure 4), and when extending and flexing the subject's elbow joint passively (Figure 5).

(a)



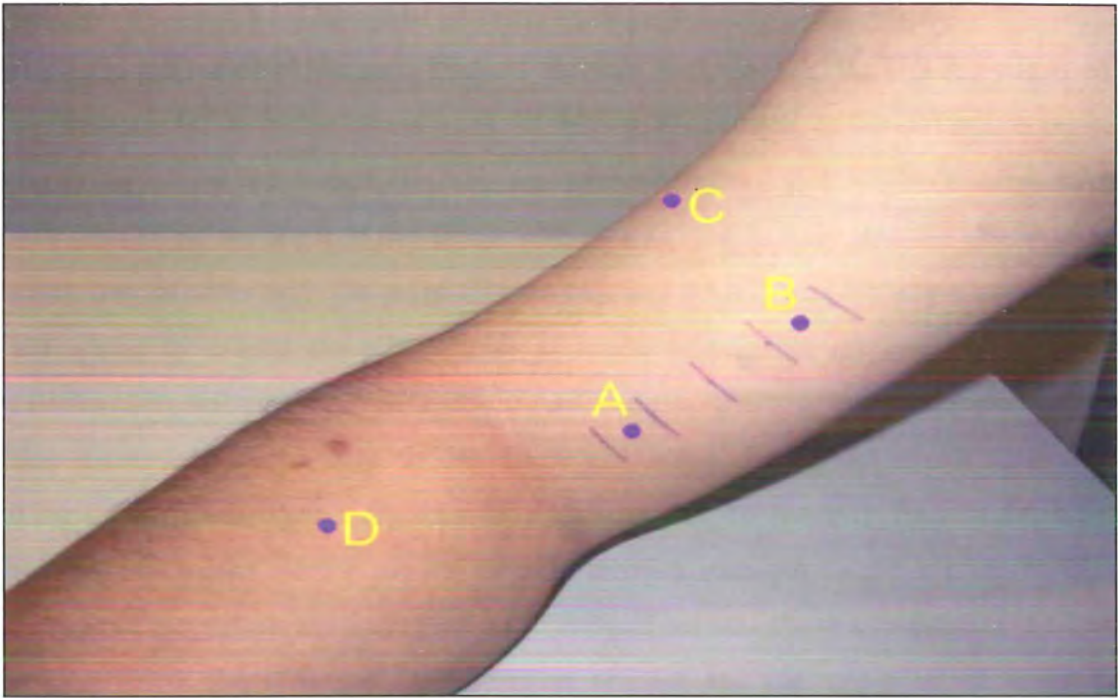
(b)



*Figure 3: Measurements of palpation soreness (a) and tenderness (b) of the biceps brachii.*

Muscle tenderness was measured using a Dobros myometer (Figure 3b) at 4 sites by palpating pre-placed marks on the upper arm between 3-5cm and 9-11 cm above the elbow crease as marked for circumference measurements and on both the brachialis and brachioradialis (Figure 4). Semi-permanent marks were used to identify the location for subsequent measurements. The spring-loaded shaft myometer was applied at each of the four sites with increasing pressure administered to the 1.5 cm diameter rubber tip and subjects were asked to report the moment pain was perceived. The pressure reading coinciding with the onset of pain was then recorded as the tenderness score. This allowed recordings of the pressure at which subjects perceive pain in kPa to be used as a tenderness score.





*Figure 4:* The palpation sites used for measurements of palpation soreness and tenderness. Between 3 to 5cm (A), between 9 to 11cm (B), Brachialis (C) and Brachioradialis (D).

(a)

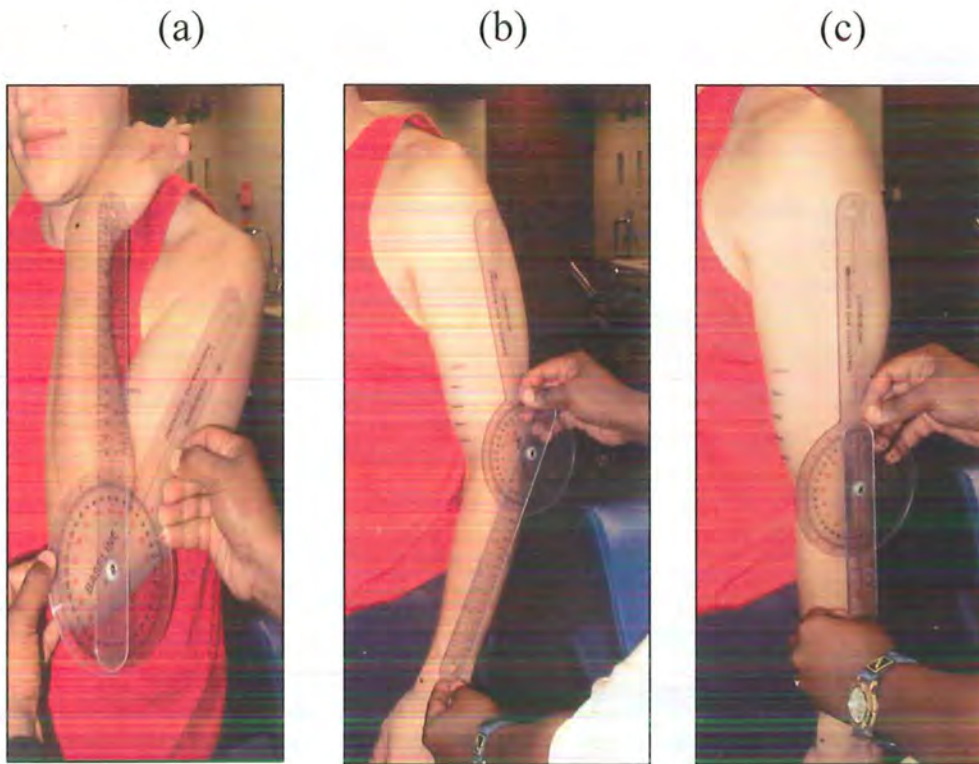
(b)



*Figure 5:* Measurements of soreness when arm was moved. Soreness measurement upon flexion (a), soreness measurement upon and extension (b).

### 3.5.3 Range of Motion (ROM) and Elbow Joint Angle

A plastic goniometer (Sammon Preston, Rolyan. Bolingbrook. IL, U.S.A.) was used to measure the ROM of the elbow joint. ROM was determined as the difference between the flexed elbow joint angle (FANG) and stretched elbow joint angle (SANG). FANG was defined as the angle at the elbow when attempting to fully flex the elbow joint to touch the shoulder with the palm (Figure 6a), and SANG was the angle created when attempting to extend the elbow joint as much as possible (Figure 6c). ROM was calculated by subtracting FANG from SANG. Relaxed elbow joint angle (RANG) was also determined as the angle at the elbow joint when the subject allows their arm to hang in a relaxed manner by their side (Figure 6b). To measure FANG, SANG and RANG, landmarks were made on the skin using a semi-permanent ink pen to obtain consistent measurements during the experimental period. These consisted of the lateral epicondyle of the humerus, the acromion process and the mid-point of the styloid process of ulna and radius. Two measurements were taken for each elbow joint angle, and a mean value of the two measurements used for analysis. All of the measurements were taken by the same investigator. Intraclass correlation coefficient was used to assess the reliability of the measurements by using the two pre-exercise measurements that were taken during the familiarization session and immediately before exercise.

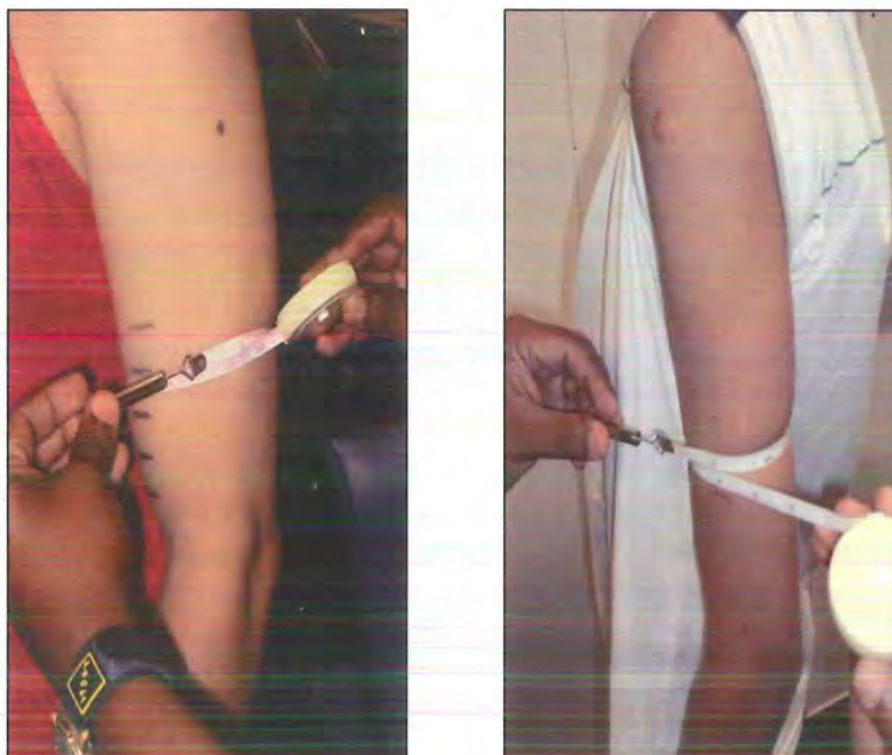


*Figure 6: FANG (a), RANG (b) SANG (c) and positioning during measurements of elbow joint angles.*

#### 3.5.4 Upper Arm Circumference

A constant tension tape (Lafayette, U.S.A) measure was used to measure upper arm circumference of five marked sites; 3, 5, 7, 9 and 11 cm distance from the elbow crease (Figure 7). The marks were maintained using a semi-permanent ink marker during the experimental period. Measurements were taken while the subject's arm was relaxed and hanging at their side. Two measurements were taken from each marked site and the mean value of the five sites was used for analysis.

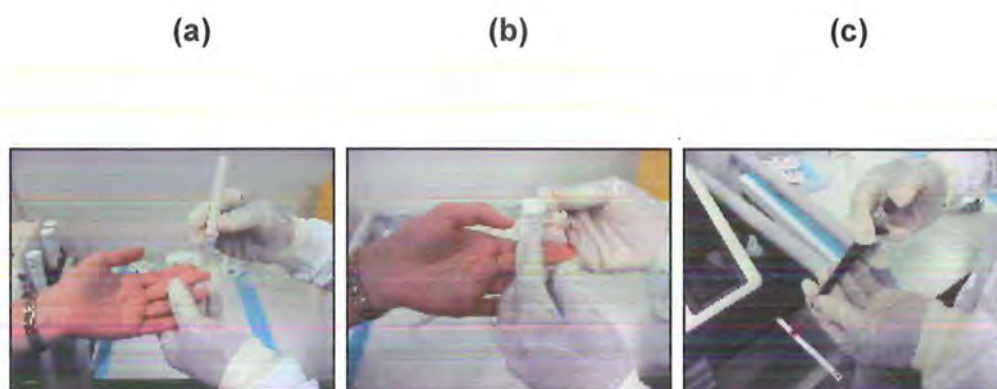




*Figure 7: Upper arm circumference measurement of the elbow.*

### 3.5.5 Plasma Creatine Kinase (CK) Activity

Personal protection equipments (clinic gown, eyeglass and disposable gloves) were worn when taking blood samples from subjects. Approximately 50 $\mu$ l of blood were collected in a heparinised capillary tube (Roche Diagnostic, Indiana, U.S.A) from a cleaned finger-prick made with a spring-loaded disposable lancet. The blood sample was immediately analysed for plasma CK activity using a Reflotron spectrophotometer (Boehringer-Manheim, Pöde, Czech Republic) (Figure 8). The normal reference range for CK using this method is 50 to 220 IU $\cdot$ L<sup>-1</sup>. When the CK activity exceeded the detection limit of the test apparatus (approximately 1500 IU $\cdot$ L<sup>-1</sup>). In this case, another blood sample was taken and diluted with saline solution until the recording value reached within the detection limit (2, 3, 5, or 10 times) and reanalysed.



*Figure 8: Measurement of plasma creatine kinase (CK) activity. Blood drawn from fingerprick (a), Blood filled into capillary pipette (b) and blood put onto CK strip (c)*

### 3.6 Reliability of Criterion Measures

All of the measurements were taken by the same investigator who was familiar with the measurements. Intraclass correlation was used to analyse the reliability of the measurements using the pre-exercise measurements. Data collected for familiarization one and two were compared to find the intraclass correlations using SPSS (Version 11) statistical package. Correlation coefficient values ( $r$ ) for isometric and isokinetic strength, ROM, upper arm circumference, plasma CK activity were 0.91, 0.89, 0.98, 0.94, and 0.95 respectively. Moreover, coefficient of variations (CV) for the corresponding measures shown previously were 6.6%, 7.1%, 4.2%, 7.9% and 9.5%, respectively. These measurements have been established in previous studies (Rothstein, Miller, & Roettger, 1983; Sforzo & Lamb, 1985; Sorichter et al., 2001; Triffletti, Litchfield, Clarkson, & Byrnes, 1988; Warren, Lowe et al., 1999).

### 3.7 Time Course

Muscle function measures (isometric and isokinetic strength, ROM) and upper arm circumference were measured before, immediately after, 30 minutes after exercise, and at 1, 2, 3, 4 and 7 days after exercise for all studies. Plasma CK activity and muscle soreness and tenderness were measured at the same time points except immediately and 30 min post exercise. Previous studies have reported that DOMS and other indicators of muscle damage peak within the first few days and dissipate slowly back to near pre-exercise level 5 to 7 days post exercise. However, for the Study 1 (massage study), additional measurements were taken 10 and 14 days after exercise to ensure the time course of recovery after eccentric exercise

### 3.8 Analysis of Results

Changes in criterion measures over time were compared between control and intervention conditions using two way repeated measures ANOVA. Isometric and isokinetic strength data were also analysed using the normalised data (% change from baseline), and analyses for ROM and circumference data were also performed using the changes from baseline. Statistical significance was set at  $P < 0.05$  for all analyses, and Tukey's post hoc tests were applied to the data to locate any significant main effects. For some occasions (i.e. comparison of pre and post effects of exercise), A student t-test was also used to detect differences between two measures. Detailed of the measurements will be discussed in each chapter of interventions

### 3.9 Ethical Considerations

Subjects for each study were asked to provide written informed consent and were free to withdraw from that particular study at any stage for any reason without prejudice. The handling of sample and dealing with human subjects also complied with Declaration of Helsinki (1964).



### 3.10 Limitations and Delimitations

Subjects were between 18 to 35 years old, therefore results may not be representative of the entire population. Both males and females subjects were included, but the number of subjects was not enough to compare between the genders. This study used a maximal eccentric exercise model of the elbow flexors assuming that this model could be generalized to the response of other muscle groups. The arm to arm comparison model has a potential of creating placebo effects since subjects know both the treatment and control conditions. If a subject thinks that a treatment is better than a control condition, a psychological effect may be involved. It is also possible that a carry-over effect, which is generated by the experience from the first arm, may occur. To minimize these effects, subjects were randomly grouped by test order; control-treatment or treatment-control, and dominant and non-dominant arms were equally balanced over the two conditions.

It should be noted that the measures used were indirect markers of muscle damage since direct assessment of damage requires muscle biopsies, which is not suitable for this study. This study was undertaken to determine the effects of therapeutic treatments on muscle soreness and recovery of muscle function (mainly strength) after eccentric exercise. Thus the criterion measures were considered to be appropriate for the study and considered as the best measure of muscle injury resulting from eccentric exercise (Warren, Lowe et al., 1999). Measurements of soreness and tenderness were also used in this study and, although these represent subjective responses, they have been widely used to quantify soreness in similar studies.

## CHAPTER 4

### EFFECTS OF IMMOBILISATION

#### 4.1 Introduction

Rest is a common prescription for most musculo-skeletal injuries, especially in the early stages of recovery (Jarvinen & Lehto, 1993). Minimising use of the injured tissue is believed to prevent further damage and promote the processes of repair and regeneration (Jarvinen & Lehto, 1993). In this context, immobilisation treatment is effective for injuries such as lacerations, contusions, or strains that are common consequences of sporting activities (Jarvinen, Kaariainen, Jarvinen, & Kalimo, 2000). Short-term immobilisation has been documented to accelerate the formation of granulation tissue matrix at the injury site and enhance recovery of muscle function (Jarvinen et al., 2000).

It is well known that repeated eccentric actions (particularly if they are unaccustomed) induce muscle injury (Armstrong, 1990). Muscle injury may be characterised by sustained loss of muscle function (Clarkson et al., 1992), histological disturbance of muscle and connective tissue (Clarkson & Sayers, 1999), development of delayed onset muscle soreness (DOMS) (Byrnes & Clarkson, 1986), and swelling (Chleboun et al., 1998). Many of these phenomena resemble those observed after traumatic muscle strain injuries (Jarvinen et al., 2000). It is reasonable to assume that short-term immobilisation would be beneficial for recovery from eccentric exercise-induced muscle damage, and this has been confirmed in a number of recent studies. Thus, Sayers et al. (2003a; 2000a; 2000b; 2003b) and Sayers & Clarkson, 2003, examined the effects of 4-days of strict arm immobilisation on recovery of the elbow flexors from a bout of eccentric exercise. They found that immobilisation facilitated recovery of muscle strength and range of motion (ROM), and attenuated increases in plasma creatine kinase (CK) activity. It should be noted that the previous studies involved fixating the arm in a cast at all times over the duration of immobilisation. Whilst this maximises the effect of inactivity, it is a procedure which subjects find uncomfortable and therefore is of questionable practical benefit as a therapeutic modality. Furthermore, prior studies have provided no information regarding the time-course of any effects occurring over the

immobilisation period (with the exception of blood markers of muscle damage such as CK activity and myoglobin concentration). If an immobilisation treatment involving use of a simple sling to secure the elbow joint at 90° could be shown to have positive effects in enhancing recovery from injury, it would have important practical implications. Moreover, such a “light” immobilisation method would allow the subject to periodically release the arm for functional activities or to aid in sleeping.

Thus, the present study aimed to investigate whether a light immobilisation regimen would be beneficial for recovery of muscle strength, ROM, upper arm circumference, and other measures that are used as indirect markers of muscle damage (Howell et al., 1993). In order to further refine the investigation, this study used an arm-to-arm comparison model in which subjects acted as their own control. This is advantageous since it is well known that responses to eccentric exercise vary widely between individuals (Clarkson & Hubal, 2002; Nosaka & Clarkson, 1996b).

## 4.2 Methods

### 4.2.1 Experimental Design

The control arm received no treatment, while the experimental arm was immobilized for 4 days after eccentric exercise. Dominant and non-dominant arms were randomly chosen for the control and immobilisation conditions, and the order in which the conditions were carried out was counter-balanced amongst the subjects.

The experimental period consisted of two blocks of 8 days each with one familiarization session. Measurements were taken before and immediately after exercise, 4 consecutive days following exercise, and 7 days post-exercise. Immobilisation started approximately 30 minutes after the exercise bout for 4 days; however, the sling was removed when taking measurements at 1-4 days after exercise. Subjects were also allowed to remove the sling when taking a shower and sleeping. Criterion measures used in this study were maximal voluntary isometric and isokinetic elbow flexor strength, ROM, upper arm circumference, muscle soreness and tenderness, and plasma creatine kinase (CK) activity.

#### 4.2.2 Subjects

Ten healthy male ( $n = 5$ ) and female ( $n = 5$ ) subjects with no history of upper arm injury gave informed consent for the study which was approved by the Research Ethics Committee of the institute. Subject were aged between 19 to 32 yrs, and mean ( $\pm$  SD) age, height, and weight were  $23 \pm 4.2$  yrs,  $163.2 \pm 15.2$  cm, and  $63.7 \pm 11.9$  kg, respectively. Prior to, and during the experimental period, subjects were requested not to take any medications or undergo any interventions other than that given by the investigators, and asked not to perform any recreational exercise.

#### 4.2.3 Eccentric Exercise Bout

The exercise protocol consisted of 10 sets of 6 maximum voluntary eccentric contractions of the elbow flexors against the lever arm of the isokinetic dynamometer (Cybex 6000, Ronkonkoma, NY, U.S.A.) moving at constant velocity of  $90^\circ\text{s}^{-1}$ . The exercise protocol consisted of 10 sets of 6 maximum voluntary eccentric contractions of the elbow flexors against the lever arm of the isokinetic dynamometer (Cybex 6000, Ronkonkoma, NY, U.S.A.) moving at constant velocity of  $90^\circ\text{s}^{-1}$ . Protocol and position for this exercise was explained earlier in chapter 3.

#### 4.2.4 Immobilisation Protocol

The arm was immobilized after the immediately post measurements (approximately 20 minutes post-exercise) using a Montreal Sling (Bodyworks Orthopaedic Supports, New Zealand) consisting of two support pads attached to adjustable straps allowing the elbow joint to be secured at the required angle ( $90^\circ$ ) as shown in Figure 9. The sling made it impossible to extend the elbow joint, but flexing the elbow joint was still possible. Subjects were asked to record periods of the day and night which they removed the sling over the 4 day period (for showering, sleeping, and during the daily laboratory testing).



*Figure 9: Immobilisation method.*

#### 4.2.5 Activity Monitoring

The extent of immobilisation was assessed using an activity monitor (Actigraph©Model7164, Version 2.2, Manufacturing Technology Inc., Florida, U.S.A.) fitted on the wrist of the immobilized arm. It had been confirmed that the activity of elbow flexors was reflected by the frequency and magnitude of movements of the wrist. The monitor was 50 x 36 x 15 mm in size and weighed 45 grams. The product of frequency and magnitude of wrist movements was recorded for 2 days prior to exercise and for 4 days afterwards. Activity data was stored in the monitor and downloaded through an infrared reader interface unit to a computer providing a measure of activity in units of counts per minute ( $\text{cnt.min}^{-1}$ ). A summation of daytime activity ( $\text{cnt.min}^{-1}$ ) pre and post treatment were then averaged to provide an indication of mean daily activity; these were compared in terms of relative immobilisation for the purposes of analysis. Subjects were not allowed to remove the activity monitor except for when showering and sleeping at night, but were asked to remove it when they came to the measurements on 1-4 days post exercise.



(a)



(b)



*Figure 10: Activity monitor (a) and activity monitor position on the arm (b)*

#### 4.2.6 Criterion Measures

All of the measurements were taken twice during the familiarization session, before and immediately after exercise, 30 minutes and 1, 2, 3, 4 and 7 days following exercise. Plasma CK activity and muscle soreness and tenderness were not measured immediately and 30 min post exercise. The order in which measurements were taken was consistent throughout the testing period starting with muscle soreness and followed by CK, ROM, upper arm circumference and finished with strength measurements. During the immobilisation period, the sling was removed for approximately 30 minutes for testing on days 1-4. All criterion measures including muscular strength, ROM, upper arm circumference, plasma CK activity and soreness and tenderness measurements were conducted as described in methods section in chapter 3.

#### 4.2.7 Data Analysis

Changes in all criterion measures over time (pre, post, 30 min, D1-D4, and D7) were compared between the immobilisation and control arms using a two-way repeated measures analysis of variance (ANOVA). Where the ANOVA showed a significant difference between conditions, Tukey's post hoc test was applied to locate any significant interactions. Paired *t*-test was also used to examine differences between conditions for peak plasma CK activity. Data for isometric and isokinetic strength, circumference and range of motion were normalised and presented in percentage. Data analysis was performed using a statistical software package SPSS (version 11.0). A statistical significance was set at  $P < 0.05$  for all analyses. Data are presented as means  $\pm$  SEM, unless otherwise stated. In most cases there were no difference between pre and 30 minutes post exercise. Therefore, data for 30 minutes post exercise are not reported

### 4.3 Results

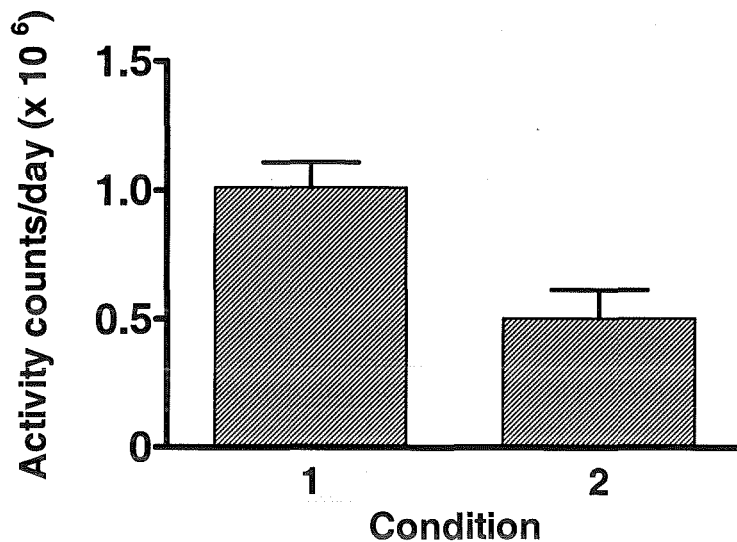
#### 4.3.1 Exercise

Before exercise, there were no significant differences in the pre-exercise values for all criterion measures between the conditions. Mean peak torque value for isometric contraction at 90° before exercise was  $38.0 \pm 5.10$  N for the control and  $39.0 \pm 5.84$  N for the immobilisation. All subjects were able to complete the two bouts of maximal eccentric exercise without undue difficulty. The average peak torque and total work during the exercise was  $29.0 \text{ N} \pm 1.96 \text{ N}$ , and  $1739.5 \pm 151.7 \text{ J}$  respectively. No significant differences in the peak torque and total work were evident between the conditions.

#### 4.3.2 Activity Monitoring

The small size and minimal weight of this activity monitor does not impede the subjects in daily activities. When attached to the wrist with a velcro strap the monitor weighed only slightly more than a large watch, was relatively unobtrusive and meant minimal disruption to the regular activities of the subject. As expected, immobilisation resulted in a large reduction in arm movement (Figure 11), with average activity levels decreasing by approximately 50% during the 4-day period compared to baseline levels

( $p < 0.05$ ). It is worth pointing out that some of the activity recorded represents that associated with whole body movement, as opposed to that specific to the arm. There was a comparatively small variation in the activity decreases between subjects, ranging from 40%-50% of baseline values.

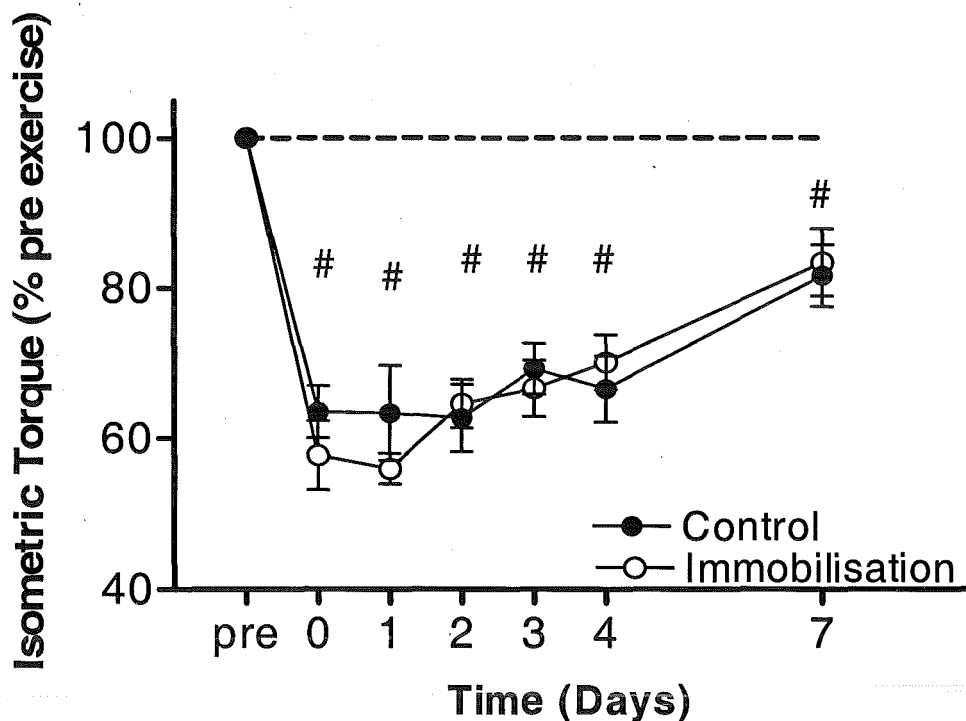


*Figure 11:* Average activity counts 1-2 days before exercise (1) and 1-3 days of immobilisation (2). Results are in counts/day where a count is the product of movements per minute produced by subjects.

#### 4.3.3 Muscular Strength

Maximal voluntary isometric torque at both angles of elbow flexion ( $30^\circ$  and  $90^\circ$ ) decreased to approximately 50% of the pre-exercise level immediately after exercise, showed no significant change at 30 min after exercise, little change over the next 2 days, and started to recover appreciably after 4 days post exercise. Recovery was not complete by 7 days after exercise for either condition (Figure 12). No significant differences in either strength loss or rate of recovery were evident between conditions.





*Figure 12:* Changes in isometric torque (as % of baseline values) at 90° from the baseline (pre), immediately after (0), and 1-7 days post exercise for immobilisation and control arm. # represents a significant difference from the baseline.

The magnitude of decrease in isokinetic torque (35%) was similar among the five test velocities (Table 2), but was smaller than that shown in the isometric values (Figure 12). The two conditions showed generally similar patterns of isokinetic strength loss and recovery following eccentric exercise with no significant differences identified between groups. At 7 days after exercise, isokinetic torque was still significantly lower than the pre-exercise values.

#### 4.3.4 ROM and Elbow Joint Angles

Significant changes in RANG, SANG, FANG and ROM were observed following exercise (Table 3). ROM decreased by approximately 15° immediately and 30 minutes after exercise, showing further reductions over next 2 days, and started to recover 4 days post-exercise. ROM was still some 8° lower than baseline at 7 days post-exercise. There was a trend for the changes in arm angle and ROM to be greater following

immobilisation, but the differences did not prove significant for any of the variables between arm conditions.

Table 2.  
*Normalized Changes in Isokinetic Torque at Five Different Velocities from the Baseline (100%) Immediately after (Post) and 1-7days (D1-D7) after Exercise for The Immobilisation and Control Condition. Mean and Standard Error of the Mean (SEM) of 10 Subjects are Shown.*

Velocity	Condition		Percentage of pre exercise (%)					
			Time (Days)					
			Post	D1	D2	D3	D4	D7
30°·s <sup>-1</sup>	Control	Mean	65.0	56.3	62.1	74.0	76.0	85.8
		SEM	3.9	5.8	5.7	5.5	5.2	7.3
	Immobilisation	Mean	67.6	67.3	66.7	68.9	77.6	85.7
		SEM	4.7	2.9	4.4	2.9	5.0	4.9
90°·s <sup>-1</sup>	Control	Mean	63.6	49.5	60.2	67.0	73.2	81.8
		SEM	5.5	6.1	5.6	6.9	9.1	7.1
	Immobilisation	Mean	66.8	62.5	64.4	71.1	76.6	86.6
		SEM	3.9	2.0	4.9	3.7	5.6	3.6
150°·s <sup>-1</sup>	Control	Mean	66.7	53.0	63.0	73.7	73.6	80.7
		SEM	6.5	6.8	4.8	4.8	6.4	6.1
	Immobilisation	Mean	66.0	65.5	63.2	69.5	68.8	84.2
		SEM	3.5	3.7	3.7	3.5	3.6	4.5
210°·s <sup>-1</sup>	Control	Mean	59.9	60.7	59.4	63.5	73.4	83.1
		SEM	7.7	9.0	5.3	4.9	8.7	6.7
	Immobilisation	Mean	68.7	63.3	56.2	76.1	67.1	77.7
		SEM	3.7	4.3	3.4	5.5	4.6	6.8
300°·s <sup>-1</sup>	Control	Mean	68.0	74.5	76.4	74.0	86.9	95.9
		SEM	7.6	10.8	8.7	11.0	17.4	13.6
	Immobilisation	Mean	66.8	72.2	65.7	66.6	72.4	75.8
		SEM	4.0	8.1	6.5	4.9	5.9	7.8

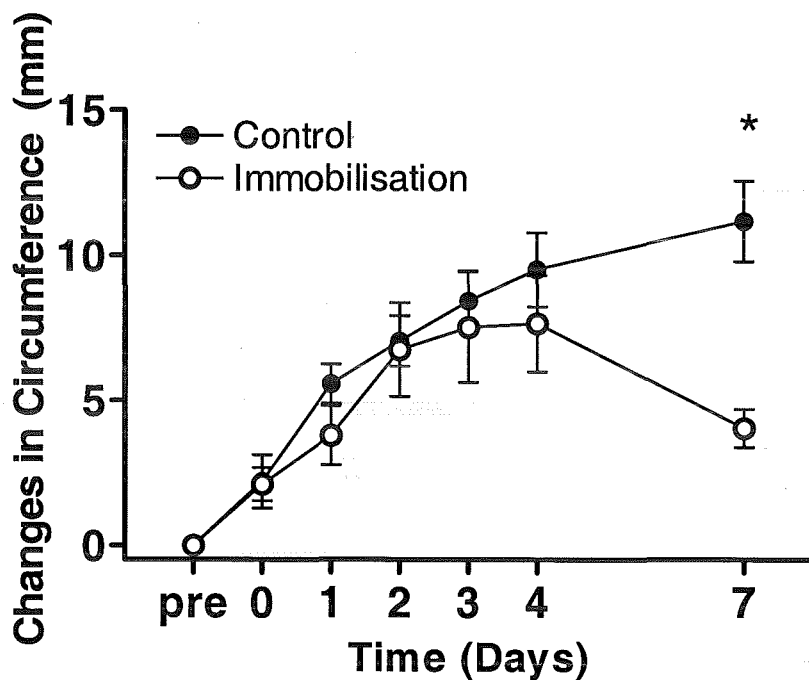
Table 3.

*Changes in Relaxed (RANG), Stretched (SANG), and Flexed Elbow Joint Angles (FANG) and Range of Motion (ROM) From the Pre-Exercise Level Immediately after (Post) and 1-7 Days (D1-D7) after Exercise for the Control and immobilisation Condition. Mean and Standard Error of the Mean (SEM) of 10 Subjects are Shown.*

Variable	Condition		Changes from pre exercise (degree)					
			Time (Days)					
			Post	D1	D2	D3	D4	D7
RANG	Control	Mean	-3.7	-8.3	-7.9	-7.4	-9.1	-6.6
		SEM	1.25	1.96	1.51	1.77	2.31	3.53
	Immobilisation	Mean	-4.3	-8.1	-10.7	-12.5	-10.5	-5.1
		SEM	1.81	2.64	1.97	2.76	3.23	2.4
SANG	Control	Mean	-3.2	-8.0	-6.8	-9.9	-10.6	-6.6
		SEM	1.34	2.19	1.46	2.78	2.06	2.36
	Immobilisation	Mean	-3.0	-8.4	-11.5	-12.9	-10.2	-4.2
		SEM	1.34	2.02	2.53	3.48	2.81	1.67
FANG	Control	Mean	14.7	11.5	10.3	10.5	7.4	2.0
		SEM	1.97	2.01	2.62	3.91	2.41	2.23
	Immobilisation	Mean	15.2	12.0	11.6	9.6	9.1	3.5
		SEM	1.69	1.7	1.93	1.77	1.46	1.13
ROM	Control	Mean	-18.4	-19.8	-18.2	-17.9	-16.5	-8.6
		SEM	1.68	2.99	3.32	4.06	3.94	5.32
	Immobilisation	Mean	-19.5	-20.1	-22.3	-22.1	-19.6	-8.6
		SEM	1.63	3.15	2.56	3.17	4.07	3.05

#### 4.3.5 Upper Arm Circumference

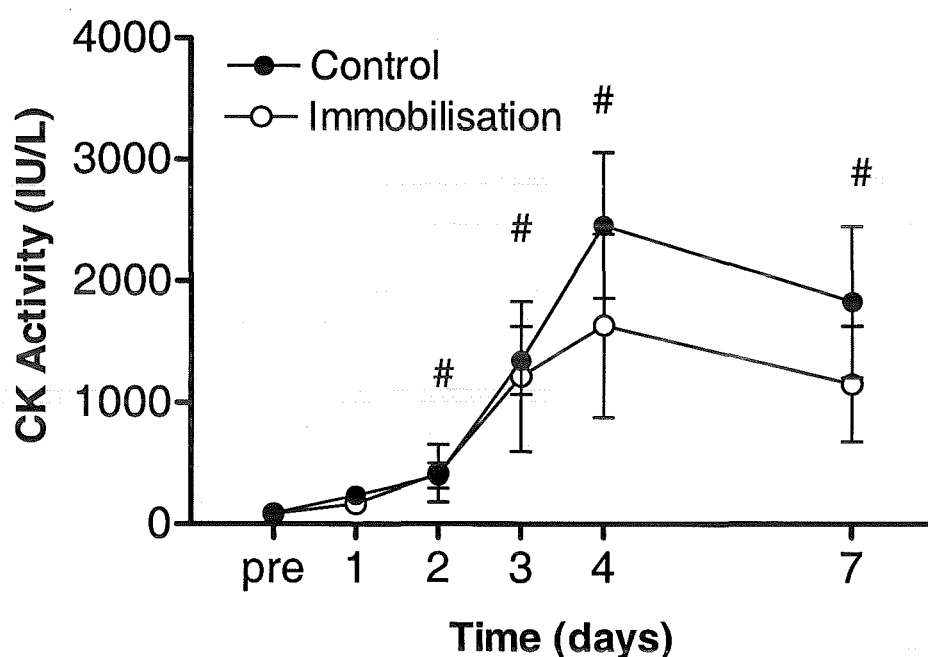
Significant increases in circumference were measured at all five sites after exercise for both conditions, with the five site average peaking 4 days after exercise, being approximately 7 mm larger than the pre-exercise value (Figure 13). The increase in arm circumference was smaller for the immobilized arms, with a significant condition  $\times$  time effect found. Post-hoc analysis demonstrating a significant difference between groups at day 7 following exercise ( $p < 0.05$ ).



*Figure 13:* Changes in upper arm circumference from pre exercise value (pre), immediately after (0), and 1-7 days (1-7) after exercise for immobilisation and control arm. \* Indicates significant different between groups.

#### 4.3.6 Plasma CK Activity

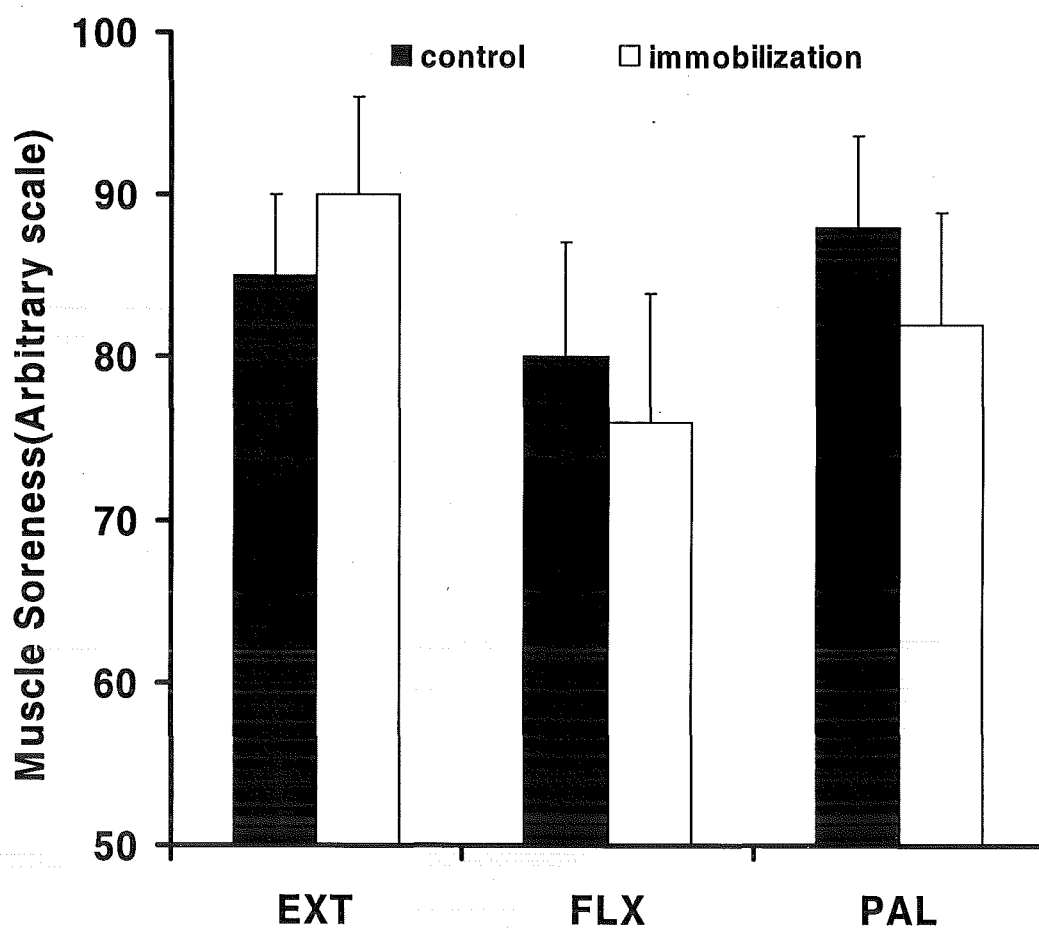
Figure 14 shows plasma CK activity following exercise for each condition. Significant increases occurred 2 days after exercise, peaking at 4 days post-exercise for both the immobilisation ( $1,631 \pm 755 \text{ IU}\cdot\text{L}^{-1}$ ) and control ( $2,455 \pm 596 \text{ IU}\cdot\text{L}^{-1}$ ) groups, and remaining elevated at 7 days post-exercise. Although CK values were lower following exercise in the immobilized arm, no significant differences between the conditions were evident.



*Figure 14:* Changes in plasma creatine kinase activity before (pre) and 1-7 days after exercise for immobilisation and control arm. # represents a significant difference from the baseline.

#### 4.3.7 Muscle Soreness

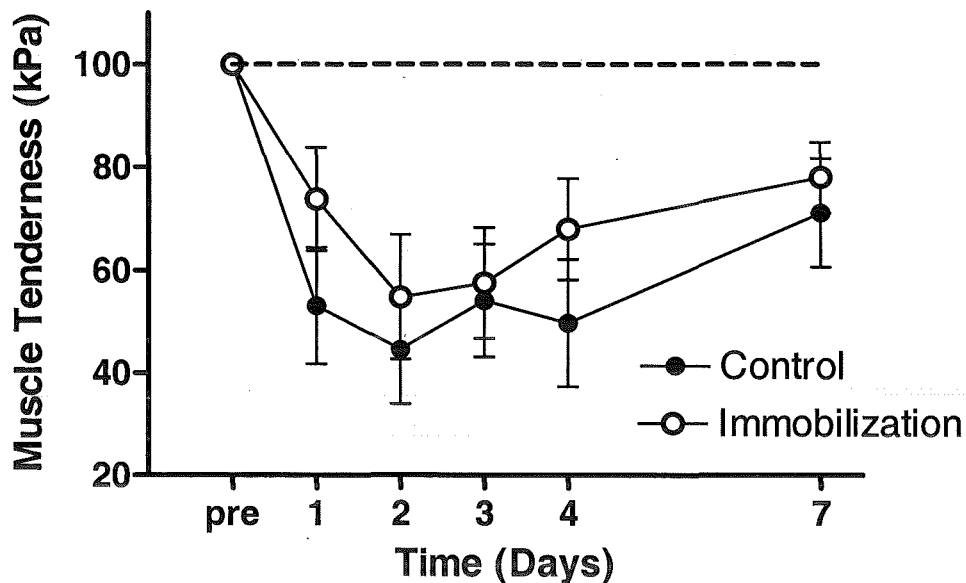
Muscle soreness developed after exercise in both conditions for palpation, extension, and flexion measures, peaked 2-4 days after exercise, and subsided by 7 days post-exercise (Figure 15). No significant differences between the arms were observed for the development of palpation, extension, and flexion soreness. When comparing the immobilisation and control condition for the peak soreness values, no differences were evident.



*Figure 15: Peak soreness with extension (EXT), flexion (FLX), and upon palpation (PAL) following exercise for immobilisation and control arm.*

#### 4.3.8 Muscle Tenderness

Muscle tenderness decreased following exercise for both control and immobilisation conditions (Figure 16). No significant differences between the arms were observed for the changes in muscle tenderness.



*Figure 16:* Changes in muscle tenderness before (pre) and 1-7 days after exercise for immobilisation and control arm.

#### 4.4 Discussion

This study examined the effect of light immobilisation on eccentric exercise-induced muscle damage. The use of an arm-to-arm comparison model, and assessment of changes in markers of muscle damage during the immobilisation period were also novel aspects of this investigation. The results showed that, with the exception of upper arm circumference, light immobilisation had no significant effects on changes in criterion measures following exercise, and indicate that previous findings regarding the beneficial effects of immobilisation may not be applicable to less severe modes of limb restriction.

The magnitude of decrease in maximal isometric strength in the days following exercise (approximately 60% of baseline) was similar to that shown in the arm immobilisation studies of Sayers et al. who found significantly enhanced recovery of muscle strength and ROM (Sayers et al., 2000a; Sayers et al., 2003), as well as lower plasma CK activity (Nosaka & Clarkson, 1996b) with immobilisation. Moreover, the other criterion measures such as RANG and FANG, and ROM, upper arm circumference, and plasma CK activity of the present study showed similar alterations to the non-immobilized arm of the previously described reports. This indicates that the exercise protocol of the present study resulted in a comparable degree of muscle damage as those of Sayers et al. (2003a; 2000a; 2000b; 2003b), and Sayers & Clarkson, (2003) although there were differences in the exercise protocols (6 sets of 10 reps for the present study vs. 2 sets of 25 reps in the former studies). The most likely explanation for the absence of a beneficial effect of immobilisation is the nature of the immobilisation protocol used. Sayers et al. (2003a; 2000a; 2000b; 2003b) fixed the elbow joint angle at 90° permanently in a cast and a sling during the 4-day immobilisation period. In contrast, the present study used a milder form of elbow extension movement restriction which allowed very limited flexion actions. Also, subjects were allowed to remove the sling for sleeping, showering, and during laboratory testing. Even so, subjects reported that their arm remained in a restricted position for an average of 13 to 15 hours a day, and this was confirmed by the 50% decrease in activity during the immobilisation period (Figure 11). This was not dissimilar to the arm activity movement decrements reported by Sayers et al. during the more severe immobilisation (approximately 40% of baseline) (Sayers et al., 2000b). A further confounding factor in comparing the present results with previous findings is that, in order to test muscle strength, we asked subjects to perform 14 brief maximal voluntary isometric and isokinetic contractions per day in the recovery period. It is conceivable that these brief periods of intense activity may have eliminated the possible beneficial effects associated with activity restriction. It is also possible that removing the sling when sleeping or during bathing affected the results. If this was the case, immobilisation needs to be very strict to provide beneficial effects on the recovery of muscle function.

Animal studies have demonstrated significant benefits of immobilisation on muscle regeneration following both traumatic and activity-induced injury (Jarvinen et al., 2000;



Lehto, Duance, & Restall, 1985). Sayers et al. (2003) found benefits of immobilisation in terms of recovery of muscle strength and ROM following exercise, which could not be explained by altered activation or electrically evoked contractile properties. On this basis, restricted activity could provide the basis for optimising recovery from injurious sporting activity. However, from a practical perspective, such a modality would have to be acceptable by the individuals. Continuous complete immobilisation for 4 days, as carried out by Sayers et al. (2003; 2000a; 2000b; 2003) is uncomfortable and imposes difficulties on the ability of subjects to perform everyday activities such as showering, as well as causing sleep disruption. For this reason the experiment was designed to establish whether a milder form of restriction of movement would maintain any of the beneficial effects of immobilisation. In this context, only arm circumference showed significant positive effects with immobilisation, with the extent of swelling being only 35% of control values 7 days after exercise. It is not exactly known why there was a rapid drop of arm circumference in immobilisation group on day 7 of the exercise. It should be noted that immobilisation treatment was applied until day 4 post exercise. Remobilization of the arm after immobilisation could be the contributing factors of this condition assuming the accumulated fluid was drained by the increased blood and fluid flow through mobilization the arm.

The potential confounding effects of the performance of the test procedures over the 4 days of immobilisation cannot be ruled out. It may be that the same outcomes reported in the studies by Sayers et al. (2000a; 2003) would have been found had the sling had been applied for 4-days after exercise without muscle strength and elbow angle measurements in these days. Increase in upper arm circumference reflects the degree of tissue swelling of upper arm (Brondstrup & Denmark, 1962; Howell et al., 1993; Nosaka & Clarkson, 1996a). Swelling is always present to some degree in acute inflammation, and is a result of increased permeability of small blood vessels that allow protein-rich fluid, known as exudate, to escape into the tissue of the damaged area (Smith, 1991). It is possible that the increase in arm movements after remobilization resulted in enhanced blood and lymph flow and accelerated the removal of exudate from the injured area. However, this is unlikely since the control arm would presumably have had a similar pattern of movement in the 4 to 7 days after exercise. Indeed, Sayers et al. (2000b) reported no difference between the immobilized and control arm activity for 5

days after cessation of immobilisation. The present study did not measure the arm activity after removing the sling, but it seems unlikely that significantly large increases in arm activity would have been found for the immobilized arm between 4 and 7 days post-exercise in the present study. Sayers et al. (2003; 2000b) reported attenuated increases in plasma CK activity for the immobilisation condition. They speculated that this was due to reduced lymph flow, since CK is reported to enter the blood from the injured muscles through lymph flow (Clarkson & Hubal, 2002). The present study did not find such large differences in CK responses for the immobilized arm (Figure 14). It is possible that muscle contractions during strength measurements were enough to equalize the CK response to the level of control arm. If the CK response reflects the lymph flow level, the light immobilisation method in the present study did not seem to affect the lymph flow. Furthermore, a notable finding of the previous study was that following remobilization of the arm, there was a large, sustained rise in plasma CK, which was explained by activity-related efflux of accumulated muscle exudates from the lymphatic vessels. Interestingly, it appears that no such rise occurred in the days following removal of immobilisation following exercise. This suggests that the periods of activity during the immobilisation period were sufficient to maintain lymphatic flow in the upper arm.

This study also found that muscle soreness was not affected by the light immobilisation. Sayers et al. (2000a) reported that residual muscle soreness was sustained longer for the immobilized arm compared to the control, and attributed this to a delay in the removal of pain-generating inflammatory products. The present study did not find such an effect. Since the immobilisation protocol in the present study was not as strict as that of the studies by Sayers and Clarkson (2003) and Sayers et al. (2003) it may be that, under the conditions of this study, sustained lymph flow did not allow the pain-generating products to remain within the muscles.

Short-term immobilisation after injury is believed to allow newly formed granulation tissue to achieve a more rapid increase in tensile strength, allowing it to better withstand the forces created by contracting muscle (Jarvinen et al., 2000). It may be that remobilization at some optimal point in the recovery period acts to accelerate formation of new granulation tissue, allowing a more rapid reduction of tissue swelling. In this

context, further studies will be required in order to determine the optimal period of immobilisation, as well as whether the pattern of activity following remobilization plays a role in the time course of recovery to complete function after injury.

In summary, the present study examined the effect of light immobilisation in which only a sling was used to secure the elbow joint angle at approximately 90° for 4 days following eccentric exercise of the elbow flexors, and compared the results of the present study with the previous studies by Sayers and Clarkson (2003) and Sayers et al. (2003) who reported that immobilisation was effective in enhancing recovery of muscle function. However, the effects of immobilisation reported in the previous studies (Sayers & Clarkson, 2003; Sayers et al., 2000a, 2000b; Sayers et al., 2003) were not as convincing, probably due to the fact that the immobilisation protocol used in this study was less severe. It can be concluded that light immobilisation does not enhance recovery of muscle function following EIMD. It would appear that the immobilisation protocol needs to be very strict in order to produce substantive beneficial effects.

## CHAPTER 5

### EFFECTS OF MASSAGE

#### 5.1 Introduction

Massage is widely used as a therapeutic modality for recovery from muscle fatigue and injury (Robertson et al., 2004). Although physiological theory to support how massage works for eccentric EIMD is obscure, massage is often recommended by coaches and therapists to alleviate or prevent DOMS following sporting activity (Tiidus, 1997), maybe due to clinical experience. Several studies have examined the effects of massage on DOMS and other markers of muscle damage such as impairment of muscle function, swelling, and increases in muscle proteins in the blood. Tiidus (1997) in his review of the literature on the effects of massage therapy, casts doubt on its effectiveness in aiding DOMS and other indicators of muscle damage. Weber et al. (1994) who examined the effect of 8-minutes of massage immediately after exercise on DOMS, failed to find a positive effect of massage. In contrast, Smith et al. (1994) reported that a 30-min massage, applied 2 hours after eccentric exercise of the elbow flexors and extensors, reduced DOMS and lowered plasma creatine kinase (CK) activity compared to a non-massage group. More recently, Farr et al. (2002) reported that a 30-min therapeutic massage of one leg 2 hours after downhill running was effective in the attenuation of DOMS compared to the contralateral limb with no treatment. Other researchers (Hilbert et al., 2003; Tiidus & Shoemaker, 1995) have also found positive effects of massage on DOMS to varying degrees.

Although a number of studies have investigated the effect of massage on DOMS and/or markers of muscle damage, the findings have been inconclusive or contradictory in nature, possibly reflecting methodological differences. One of the reasons for the controversy seems to stem from the different eccentric exercise models utilized in the studies, which result in different magnitude of muscle damage to different muscles. A second possible confounding factor in such studies is the fact that individuals show a wide variation in their responses to the same exercise protocol (Nosaka & Clarkson, 1996b). The large variability in responses between individuals to the effects of eccentric exercise has made comparison with control conditions difficult. Most studies (Farr et

al., 2002; Hilbert et al., 2003; Rodenburg et al., 1994; Smith, Keating et al., 1994) have compared massage and control groups composed of different populations of subjects. The inconsistency between subjects in response to eccentric exercise is likely to act as a confounding factor, reducing the likelihood of exposing any positive effects the massage therapy may have incurred. One solution to this problem is to utilize an "arm to arm comparison model" in which a treatment arm is compared with responses from the contralateral (untreated) arm of the same subject. No previous researchers have used this model to investigate the effects of massage on DOMS and other markers of muscle damage following eccentric exercise of the elbow flexors. It was expected that the arm to arm comparison model would give a better picture of whether massage is effective for alleviating DOMS and enhancing recovery of muscle function after eccentric exercise.

When evaluating the effect of massage, it is important to differentiate between the symptoms of DOMS from other indicators of muscle damage, and to clarify whether it enhances recovery of muscle function, which seems to be the most important factor to be considered by athletic trainers or physical therapist. It should be noted that most of the aforementioned studies (Clarkson & Tremblay, 1988; Nosaka et al., 2002a) reported a positive effect of massage on DOMS, but not necessarily on recovery of muscle function and other indicators of muscle damage. Because of the subjective nature of pain sensation, comparing massage and control condition in the same subject would be preferable. It was hypothesized that massage would alleviate DOMS to some extent, but would not affect the recovery of muscle function.

Therefore, the purpose of the present study was to examine the effects of a sport massage on DOMS, muscle function, and other parameters associated with muscle damage such as impairment of muscle strength and range of motion, swelling, a biochemical marker of muscle damage in the blood, using the "arm to arm comparison model."

## 5.2 Methods

### 5.2.1 Subjects

Ten healthy subjects (5 males and 5 females ) with no history of upper arm injury and no experience in resistance training were recruited after approval from the Institutional Ethics Committee. Subjects were aged between 19 to 32 years (mean  $\pm$  SD:  $23.0 \pm 4.2$  years), and their mean height and weight was  $163.2 \pm 15.2$  cm,  $63.7 \pm 11.9$  kg, respectively. During the experimental period, subjects were requested not to take any medication, change their diet or perform any strenuous exercise.

### 5.2.2 Experimental Design

An arm to arm comparison model was used in this study. One arm was used as a control and the other arm was used for a treatment condition. Subjects reported to the laboratory on several occasions including at least one familiarization session prior to the baseline measurements. For each condition, measurements were taken before, immediately and 30 min after exercise, followed by 1, 2, 3, 4, 7, 10, and 14 days following exercise. An identical maximal eccentric exercise protocol of the elbow flexors was performed by each arm for each condition. For the treatment condition, subjects received 10-minute massage on the exercised arm 3 hours following exercise. Criterion variables consisted of maximal isometric voluntary strength, range of motion (ROM), upper arm circumference, plasma creatine kinase (CK) activity, and muscle soreness. Changes in these variables over time were compared between the control and experimental arms.

### 5.2.3 Exercise

A similar exercise protocol for immobilisation intervention involving eccentric contractions of the elbow flexors against the lever arm of the isokinetic dynamometer was used in this. The protocol and position of the arm for this exercise was explained earlier in chapter 3.



#### 5.2.4 Massage

A standardised 10-minute sports massage was applied to the exercised arm by a qualified massage therapist, who had been working for a football club for several years as a professional masseuse, 3 hours after exercise for the massage condition. The 3-hour time point was chosen based on a previous study (Ohnhaus & Adler, 1975). The massage protocol used deeply applied clearing techniques using palmar and finger stroking to the muscles. Massage was applied as the subject laid on her/his back on a massage table (Figure 17). It consisted of effleurage (stroking) of the hand (30 s), wrist to elbow (1 min), and elbow to shoulder (1 min), petrissage (kneading) of the wrist to elbow (30 s) and elbow to shoulder (30 s), cross fibre massage to the forearm (1 min), biceps, triceps, and deltoids (1 min), thumb petrissage of the wrist to elbow (1 min) as well as elbow to shoulder (1 min), repeat effleurage of the hand (30 s), wrist to elbow (1 min), and elbow to shoulder (1 min). The massage protocol was performed by the same therapist throughout under verbal instruction recorded on an audiocassette. The therapist was requested to keep the depth and rate of massage as consistent as possible throughout the massage period.



*Figure 17: Massage performed on the exercised arm*

### 5.2.5 Criterion Measures

As explained in the Chapter 3, the criterion measures included isometric and isokinetic muscular strength, ROM, upper arm circumference, plasma CK activity and muscle soreness and tenderness. All of the measurements were taken twice during familiarization session. For the exercise day, these measurements were taken before, immediately and 30 min after exercise, and repeated 1, 2, 3, 4, 7, 10, and 14 days following exercise. Plasma CK activity and muscle soreness were measured at the same time points as those described previously except immediately and 30 min post exercise.

### 5.2.6 Data Analysis

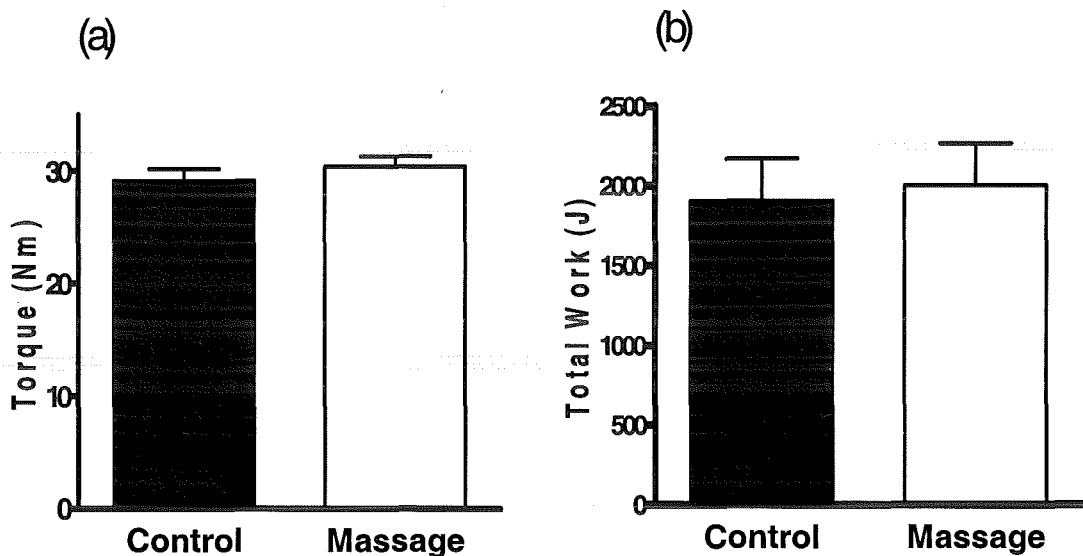
Changes in muscle strength, ROM, circumference, and plasma CK activity over time were compared between massage and control conditions using a two-way repeated measures analysis of variance (ANOVA). When the ANOVA showed a significant difference between conditions, Tukey's post hoc test was applied to find the location of the significance. Peak soreness (extension, flexion and palpation) was compared between conditions by paired *t*-test. Paired *t*-tests were also used to examine differences between conditions for peak plasma CK activity, and change in arm circumference. Data analysis was performed using a statistical software package SPSS (version 11.0). A statistical significance was set at  $P < 0.05$  for all analyses. Data are presented as means  $\pm$  SEM, unless otherwise stated.



## 5.3 Results

### 5.3.1 Exercise

Baseline values for the maximal isometric and isokinetic strength showed no significant differences ( $P=0.93$  and  $P=0.95$ , respectively) between massage and control arms. Also, peak torque and total work values recorded over the course of the eccentric exercise protocol were quite similar for the two conditions, and no significant differences between the arms were evident (Figure 18).

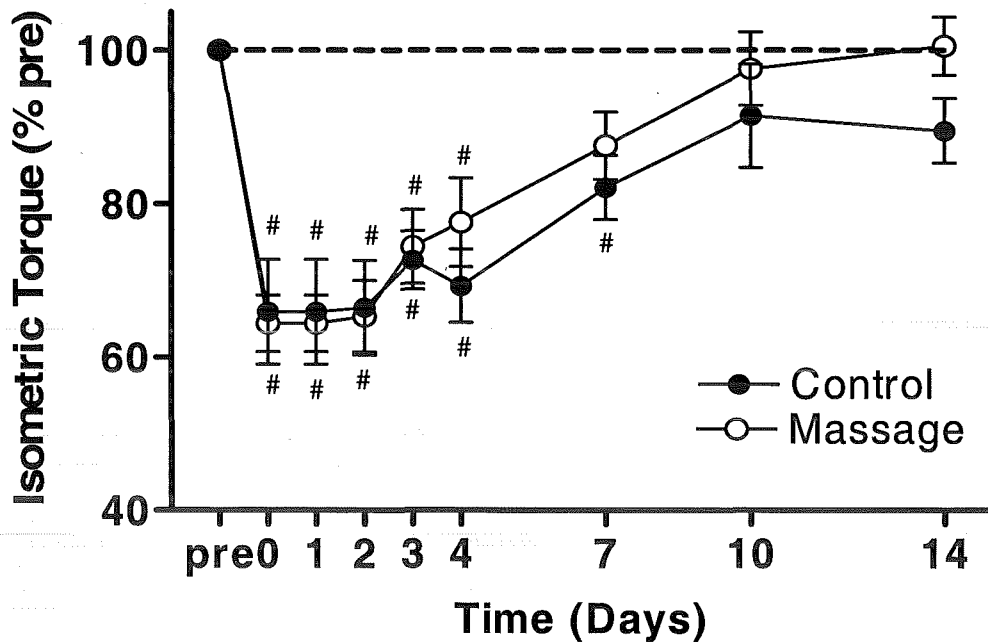


*Figure 18:* Comparison of baseline value of torque (a) and total work performed during exercise (b) between control and massage conditions.

### 5.3.2 Muscular Strength

Maximal isometric torque was significantly larger at an elbow angle of  $90^\circ$  ( $37.2 \pm 6.6$  Nm) than at  $150^\circ$  ( $27.3 \pm 4.6$  Nm) before exercise, and throughout the measurements, however, the magnitude of decrease in torque post-exercise was similar between the two angles. No significant differences ( $P=0.64$ ) in either maximal isometric torque at either angle or maximal isokinetic torque at the five velocities were observed between massage and control arms. As shown in Figure 19, isometric torque decreased to approximately 40% of pre-exercise values immediately and 30 minute after exercise,

and remained at this level for a further 2 days, after which there was a return to the pre-exercise level by 10-day post-exercise. The treatment and control arms displayed similar degrees of strength loss and time course of recovery, although strength recovery was slightly faster in the massaged arm but not statistically ( $P=0.64$ ) different (Figure 19).



*Figure 19:* Changes in maximal voluntary isometric torque from baseline (pre), immediately after (0), and 1-14 days post exercise for massage and control arm expressed as percentage of baseline. # represents a significant difference from the baseline.

Changes in maximal voluntary isokinetic torque were similar to the isometric torque over the course of the post-exercise period. Furthermore, no significant differences ( $P=0.82$ ) were evident between treatment and control arms for any of the velocities tested (Table 4). The isokinetic torque recovered to the pre-exercise level by 10-days after exercise for both conditions.

Table 4.

*Changes in Peak Isokinetic Torque at  $30^{\circ}\cdot s^{-1}$  and  $300^{\circ}\cdot s^{-1}$  before (pre), Immediately After (post) and 1-14 Days after Exercise for the Control and Massage Condition. Mean and Standard Error of the Mean (SEM) of 10 Subjects are Shown.*

		Time post exercise (days)									
			pre	post	1	2	3	4	7	10	14
Torque 30°·s <sup>-1</sup> (Nm)	CONTROL	Mean	25.8	17.3	14.8	16.0	19.0	20.2	21.6	22.2	23.3
		SEM	4.8	3.2	2.8	2.5	3.9	4.2	4.1	4.1	4.5
	MASSAGE	Mean	25.6	17.7	18.9	19.5	21.0	23.0	23.1	25.7	25.4
		SEM	4.4	2.9	4.4	3.9	4.5	4.3	3.9	4.2	4.7
Torque 300°·s <sup>-1</sup> (Nm)	CONTROL	Mean	19.8	14.8	14.5	15.0	14.2	14.8	16.2	19.2	18.1
		SEM	4.2	3.8	2.9	3.4	3.2	3.3	3.6	3.5	3.7
	MASSAGE	Mean	19.3	13.2	13.9	15.2	17.2	16.7	17.0	19.4	18.3
		SEM	4.2	3.2	3.9	3.7	3.7	3.9	4.1	4.1	3.6

### 5.3.3 ROM

No significant difference ( $P=0.70$ ) in the pre-exercise ROM values was evident between the control and massage arms. ROM values decreased significantly ( $P=0.04$ ) immediately and 30 min after exercise by approximately 30% from baseline, and did not recover for the next 4 days. Changes in ROM after exercise were similar for both conditions (Table 5).

Table 5.

*Changes in Range of Motion (ROM) and Upper Arm Circumference (CIR) from the Pre-Exercise Level (Baseline) Immediately After (Post) and 1-14 Days after Exercise for the Control and Massage Condition. Mean and Standard Error of the Mean (SEM) of 10 Subjects are Shown. # Represents a Significant Difference from Control.*

		Time post exercise (days)									
		post	1	2	3	4	7	10	14		
ROM (degree)	CONTROL	Mean	-15.2	-16.4	-15.1	-17.4	-19	-10.3	-2.8	0.8	
		SEM	1.9	3.2	3.6	4.6	4.1	3.7	3.4	2.1	
	MASSAGE	Mean	-16.6	-14.3	-11.8	-10.2	-7.8	-1.6	-0.5	0.0	
		SEM	4.3	3.8	3.4	2.2	1.9	2.3	1.7	1.7	
	CIR (mm)	CONTROL	Mean	2.3	5.2	5.9	7.8	10.4	10.9	6.5	4.8
			SEM	1.3	1.6	1.5	1.4	2.0	2.1	1.8	2.0
MASSAGE		Mean	1	1.1	4.1	2.5 #	3.3 #	6.8	2.8	0.7	
		SEM	1.2	1.6	2.1	1.2	1.3	1.8	1.8	1.0	

#### 5.3.4 Upper Arm Circumference

The baseline upper arm circumference was not significantly ( $P=0.74$ ) different between the arms. Upper arm circumference increased significantly ( $P=0.04$ ) after exercise in both conditions, and the massaged arm showed a significantly ( $P=0.04$ ) smaller increase compared to the control arm (Table 5). Post-hoc analysis revealed significant differences in circumference between massage and control arm were recorded at 3 ( $p=0.04$ ) and 4 days ( $p=0.03$ ) following exercise.

### 5.3.5 Plasma Creatine Kinase Activity

There was no significant difference ( $P=0.90$ ) in plasma CK activity between the arms before exercise. Massage had a significant ( $P=0.03$ ) effect on plasma CK activity following exercise. Figure 20 shows that, although plasma CK activity increased significantly ( $P=0.01$ ) following both exercise bouts, significantly ( $P=0.02$ ) smaller CK efflux occurred for the massaged arm, which had peak values of approximately 36% of those following no massage condition ( $982 \pm 356 \text{ IU}\cdot\text{L}^{-1}$  vs  $2,704 \pm 637 \text{ IU}\cdot\text{L}^{-1}$ ). The time of peak value and recovery of plasma CK activity were similar for the two conditions.

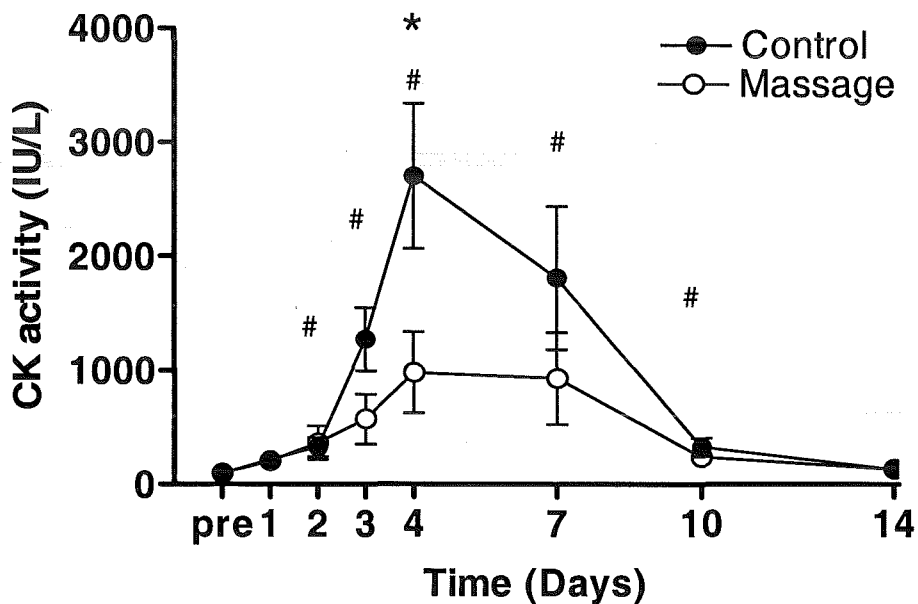


Figure 20: Plasma creatine kinase activity before (pre) and 1-14 days after exercise for massage and control arm. \* represents a significant difference between arms; # represents a significant difference from the baseline.

### 5.3.6 Muscle Soreness

Muscle soreness developed after both exercise bouts. The time course of development of soreness differed depending on the type of measurement. Peak soreness for palpation of the brachioradialis and brachialis, and flexing the elbow joint was reported 1-3 days after exercise, whereas soreness on extension of the elbow joint occurred 4 days after exercise (Table 6). The highest peak soreness score was observed for the extension, followed by palpation of the brachioradialis. All reports of soreness had resolved by 10 days post-exercise.

Table 6.

*Peak Muscle Soreness with Palpating the Brachialis and Brachioradialis (B/radialis), and Flexing (FLX) and Extending (EXT) the Elbow Joint After Exercise for the Control and Massage Condition. Mean and Standard Error of the Mean (SEM) of 10 Subjects are Shown. P Values of the Paired t test are Shown.*

		Peak soreness (mm)			
		Brachialis	B/radialis	FLX	EXT
<b>CONTROL</b> (0-100 scale)	Mean	46.7	51.6	42.1	52.8
	SEM	6.58	6.93	6.45	7.02
<b>MASSAGE</b> (0-100 scale)	Mean	35	33	25.1	42.9
	SEM	7.87	8.05	7.46	5.57
<b>Significance level</b>		<b><math>p = 0.06</math></b>	<b><math>p = 0.01</math></b>	<b><math>p = 0.07</math></b>	<b><math>p = 0.02</math></b>

As shown in Table 6, significant differences ( $P=0.01-0.02$ ) between massage and control conditions were found for peak soreness with palpation of the brachioradialis and extending the elbow joint, with peak values for the other two soreness variables showing borderline significance ( $P=0.06-0.07$ ). The most profound difference occurred for palpation of the brachioradialis, with massage resulting in a 36% decrease in the severity of soreness compared to no treatment in the same individuals.

#### 5.4 Discussion

This study investigated the effects of a 10-min massage performed 3 hours after an eccentric exercise protocol on DOMS and other indicators of muscle damage. In the present study, a self-reported visual analogue scale was used to quantify the magnitude of muscle soreness in response to standardized levels of palpation, extension and flexion of the elbow flexors. This technique has been widely used in previous studies and has been reported to represent the most satisfactory means of assessing pain sensation (Ohnhaus & Adler, 1975). Since the perception of pain is highly subjective in nature, and varies widely between individuals (Clarkson, 1992), the use of soreness as a quantifier of muscle injury is problematic. Yet it is the most widely experienced negative consequence of exercise, making it an important variable to consider. In order to minimize the confounding effects associated with difference in individual responses, the present study used the “arm to arm comparison” model to compare massage and control conditions. The results showed that massage was effective in reducing the magnitude of DOMS, muscles swelling, and plasma CK activity. In contrast, no positive effects of massage were found for muscle strength or range of motion.

DOMS is a symptom of eccentric exercise-induced muscle damage, occurs 8-12 hours after exercise when the affected muscle contracts, stretches or is palpated, peaks at 2-3 days, and slowly dissipates by 8-10 days post exercise (Nosaka et al., 2002a). It is important to note that the time course of muscle soreness development is very different from changes in muscle strength and ROM, upper arm circumference, and plasma CK activity (Gupta et al., 1996; Stauber et al., 1990). Although the underlying mechanism of DOMS remains uncertain, it is generally accepted that DOMS is caused by inflammation of the damaged muscle and/or connective tissue, and efflux of substances from damaged tissue to extracellular space sensitising the free nerve endings (Armstrong, 1984). It has been described that DOMS is the result of activation of group IV pain receptors, which are responsible for the transmission of dull aching pain signals (Armstrong, 1984). These receptors can respond to pressure and shear stress and/or chemical substances such as bradykinin, serotonin and histamine that accumulate in the interstitium (Farr et al., 2002; Hilbert et al., 2003; Robertson et al., 2004; Smith, Keating et al., 1994). The responses of Type IV receptors to any one stimulus may be sensitised and/or potentiated if the chemical environment of the interstitium is altered,



and this is a possible mechanism for the development of DOMS following eccentric exercise.

Findings of this study support those previously made regarding the positive effects of massage on DOMS. In addition, this study also found significant effects of massage on muscle swelling and CK response. The massage protocols used in previous studies have varied widely in terms of the timing, duration and frequency of treatment. Most have used one session of massage 2-4 hours after exercise (Tiidus & Shoemaker, 1995). Only one study by Tiidus and Shoemaker (Tiidus & Shoemaker, 1995) repeated the massage 10min, 2 days, and 4 days after exercise. Massage duration has been 8 (Gupta et al., 1996; Tiidus & Shoemaker, 1995), 10 (Rodenburg et al., 1994), 15 (Hemmings et al., 2000; Hilbert et al., 2003; Robertson et al., 2004), 20 (Farr et al., 2002; Smith, Keating et al., 1994), or 30 minutes (Weber et al., 1994). It is interesting to note that all studies except that of Weber et al. (1994) reported that massage had a positive effect on DOMS. This suggests that a massage performed following exercise, but prior to the development of DOMS can alleviate soreness, no matter how the massage is performed. In this study massage was found to be effective in reducing muscle soreness when muscles were palpated or extended, resulting in a 36% reduction of soreness compared to the control condition.

It is difficult to explain how massage reduces DOMS, since no studies have investigated the effect of massage on cellular events or pathophysiological changes in the muscle and/or connective tissue following eccentric exercise. Smith et al. (Smith, Keating et al., 1994) suggested that the increase in tissue blood flow associated with massage 2-3 hours after exercise results in a disruption of margination and subsequent emigration of neutrophils to the affected muscle area. However, no study has yet shown whether massage can increase migration of neutrophils, macrophages, or other leucocytes to the injured sites. It may be also possible that increases in blood and lymph flow enhance removal of pain substrates that start to accumulate in the injured area (Smith, Keating et al., 1994; Tiidus, 1997). However, it should be noted that the massage in the present study was performed before soreness occurred. It is not known how the massage performed 3 hours after exercise affects the DOMS and plasma CK activity in later events (day 3 and day 4 post exercise).

It is interesting that increases in plasma CK activity were significantly smaller for the massage condition compared to the control condition (Figure 2). The blunted CK response for the massaged arm compared to the control arm could be explained either by smaller CK efflux from the damaged muscle or increased clearance of CK from the circulation. It may be that massage enhanced the transport of CK from the damaged muscle to the circulation via lymph and increased CK clearance from the blood by increasing blood and lymph flow (Smith, Fulmer et al., 1994). It is also possible to assume that massage assists in flushing neutrophils and macrophages from the injured area, thus avoiding fibre necrosis and CK efflux (Cannon et al., 1990; Smith, 1991). However, no concrete evidence to support these speculations are available at this stage. Cardinal signs of acute inflammation include redness, heat, swelling, pain, and impairment of function, and swelling, pain, and impairment of muscle function appear in eccentric exercise-induced muscle damage (Smith, 1991). Reduced muscle swelling in the massaged condition may support the concept of an ameliorated inflammatory response following treatment, as does the smaller CK efflux observed. Since no direct indicators of inflammation were measured in the present study, it is not possible to state that the severity of DOMS is linked to the processes of inflammation and/or subsequent muscle oedema. Further study is necessary to investigate how massage affects the inflammatory responses in the damaged muscles.

Given the fact that massage had such positive effects in reducing the size of the aforementioned markers of muscle damage, it is surprising that no significant protective effects occurred against losses in muscle strength and range of motion. However, this is consistent with previous studies (Farr et al., 2002; Hilbert et al., 2003) which did not find beneficial effects of massage on either loss of, or recovery of, muscle function. It might be more important for athletes and coaches to enhance recovery of muscle function after eccentric exercise than reducing symptoms of muscle damage. If this is the case, it should be noted that massage is not beneficial for this purpose. However there are some sports that rely on techniques rather than strength to perform better. If this is the case, it may be beneficial to attenuate muscle soreness. Findings of this study thus support the idea that DOMS should be treated with caution as an indicator of muscle damage and may be more associated with individual responses to the sensations eliciting pain than the mechanisms responsible for muscle injury per se. This makes it

all the more important to consider such variation in the design and interpretation of studies such as this one.

In summary, using an arm to arm comparison model to quantify the effects of a therapeutic massage following high intensity eccentric exercise, we found reductions in muscle soreness, muscle swelling and lowered CK efflux compared to responses in the contralateral arm. However, massage had no protective effect on muscle strength. The findings of this study suggest that massage, used appropriately, is useful in reducing DOMS and swelling associated with high intensity eccentric exercise. However, those recreational athletes and sports professionals who utilise massage should be cognisant of the fact that no positive effects of massage on recovery of muscle function can be expected.

## CHAPTER 6

### EFFECTS OF LIGHT CONCENTRIC EXERCISE

#### 6.1 Introduction

Eccentric exercise induces greater muscle damage and delayed onset muscle soreness (DOMS) than activities mainly consisting of concentric or isometric actions (Armstrong, 1990; Clarkson, 1997). A peculiar aspect of DOMS is that pain is felt when the affected muscles are moved or palpated (Jones, Newham, & Clarkson, 1987). DOMS generally develops 8 to 12 hours after exercise, peaks between 24 and 72 hours, and subsides by a week following eccentric exercise (Clarkson, 1992; Clarkson, 1997; Clarkson & Hubal, 2002). DOMS may represent a protective mechanism, acting as a sign to reduce muscle activity and prevent further injury, however, many athletes and exercise enthusiasts continue to train despite the presence of muscle soreness. Interestingly, when performing exercise with sore muscles, although more pain is induced at the beginning of activity, it is anecdotally reported that DOMS is alleviated. This may be associated with the phenomenon of “exercise-induced analgesia” which is described as an increase in pain thresholds and pain tolerance following exercise (Koltyn 2000). However few studies have systematically investigated the nature of exercise-induced analgesia during DOMS after eccentric exercise in a controlled experimental setting.

Donnelly et al. (1992) reported that light eccentric exercise performed one day after a heavy eccentric exercise bout did not affect DOMS. In contrast, Saxton and Donnelly (1995) investigating the effect of sub-maximal (50%) concentric actions (5 sets of 10) performed daily following a bout of maximal eccentric exercise of the elbow flexors, found that the light exercise caused a significant reduction in DOMS after 2 days. In an investigation of the effect of repeated bouts of eccentric exercise on the recovery from eccentric exercise, Nosaka and Newton (2002d) reported a significant decrease in DOMS immediately after the repeated eccentric exercise bouts. No study has investigated the effect of light exercise, equivalent to that used in regular warm-up routines, on DOMS developing after eccentric exercise.

It is also important to investigate whether the analgesic effect of exercise, if any, is beneficial for alleviating DOMS and enhancing recovery from muscle damage over time. Several studies have evaluated the therapeutic effects of exercise on the recovery from damaging exercise with conflicting findings. Hasson et al. (1989) reported that a high speed voluntary contraction of quadriceps was effective in alleviating DOMS and facilitating recovery of muscle function after 120 voluntary maximum knee extension at  $300^{\circ}\text{s}^{-1}$ , whilst the aforementioned study by Saxton and Donnelly, (1995) showed no effect on indicators of muscle damage other than DOMS. Conversely, Donnelly et al. (1992) reported that light eccentric exercise did not alter DOMS and muscle function following a bout of damaging exercise, but reduced or delayed increases in serum CK activity. Similarly, Weber et al. (1994) found no significant effect of upper arm ergometry on DOMS and muscle function applied immediately and 24 hours after eccentric exercise of the elbow flexors. These contrasting findings may be related to the different protocols, types of exercise, timing and intensity of exercise used. Activities undertaken during the recovery period from a damaging exercise bout do not seem to have a negative effect on the recuperative process, however, whether such exercise is beneficial in reducing DOMS and enhancing recovery of muscle function needs to be confirmed.

Therefore, the purpose of this study was to test the hypothesis that a bout of light concentric exercise undertaken daily following eccentric exercise would alleviate DOMS and enhance recovery of muscle function. The concentric type of exercise was utilized because it has been found that it does not induce muscle damage (Nosaka & Newton, 2002a) and therefore will not exacerbate the already incurred muscle damage or retard the recovery process.

## 6.2 Methods

### 6.2.1 Experimental Design

Both arms performed maximal eccentric exercise of the elbow flexors, but only the treatment arm performed a bout of light concentric exercise (LCE) for 4 days after maximal eccentric exercise (Max-ECC). Dominant and non-dominant arms were randomly chosen for the control and light exercise conditions, and the order in which the conditions were carried out was counter-balanced amongst subjects.

The experimental period consisted of two blocks of testing over 8 days, with one familiarization session. Criterion measures used included maximal voluntary isometric and isokinetic elbow flexor strength, range of motion (ROM), upper arm circumference, muscle soreness and tenderness, and plasma CK activity. Measurements were taken prior to and immediately after Max-ECC, and on days 1, 2, 3, 4 and 7 post-exercise. The LCE was performed initially 1-day post exercise and repeated 2, 3, and 4 days following Max-ECC. In addition to the above measures, maximum voluntary isometric and isokinetic strength, ROM, upper arm circumference, and muscle soreness and tenderness were also assessed directly after each LCE bout.

### 6.2.2 Subjects

Ten males and four females gave written, informed consent to participate in this study, after receiving ethical approval from the Research Ethics Committee of the Institute. Subjects age ranged between 22 and 32 yrs, and the mean ( $\pm$  SD) age, height, and weight were  $24.4 \pm 2.4$  yrs,  $165 \pm 10.9$  cm, and  $69.9 \pm 14.1$  kg, respectively. All subjects were screened via a medical questionnaire and were free from any musculoskeletal disorders. Subjects were requested not to take any medications or undergo any interventions other than that given by the investigators, and were asked not to perform any recreational sports activities over the duration of the study.

### 6.2.3 Maximal Eccentric Exercise Protocol (Max-ECC)

Similar exercise protocol involving eccentric contractions of the elbow flexors against the lever arm of the isokinetic dynamometer was used in this. Protocol and position for this exercise was explained earlier in chapter 3.

### 6.2.4 Light Concentric Exercise

The light concentric exercise (LCE) consisted of 600 (10 sets of 60) continuous elbow flexion and extension movements on the isokinetic dynamometer with a 30 s rest period between sets. This exercise took approximately 25 minutes to complete, but the actual muscle contraction time was approximately 20 minutes. Since the purpose of this exercise was to use the muscles with DOMS whilst minimizing fatigue, subjects were asked to perform this exercise as comfortably as possible (ie. with minimal effort). This exercise protocol was based on a regular warming-up exercise that would enhance blood and lymph flow, and the length was chosen according to a recommended warming-up protocol (Rodenburg et al., 1994). To minimize the force generation during the exercise, the velocity of the isokinetic dynamometer was set at  $240^{\circ}\text{s}^{-1}$  for the flexion and  $210^{\circ}\text{s}^{-1}$  for the extension, based on a pilot study showing that subjects were able to flex and extend elbow joint rhythmically with minimal effort at these settings with minimal fatigue. For the elbow extensions, subjects were asked to relax the elbow flexors to let the elbow joint extend with gravity. The torque produced during exercise was less than 20% of maximal isometric torque for the elbow flexions, and negligible for the elbow extensions. During the interval between sets, subject's arms were in a relaxed position for 30 s. The torque data during exercise were transferred to an IBM desktop computer operating AMLAB data acquisition software (version II, Lewisham, Australia). Feedback of torque generation was provided to subjects and experimenters via a PC monitor.



### 6.2.5 Criterion Measures

Maximal voluntary isometric and isokinetic elbow flexor strength, ROM, and upper arm circumference, muscle soreness and tenderness, and plasma CK activity were recorded prior to Max-ECC for the baseline data. On the day of exercise, all measurements except plasma CK activity were measured pre, immediately after and 30 minutes after Max-ECC. On 1, 2, 3, and 4 days following Max-ECC, all dependent variables except CK were measured before and after LCE. Plasma CK activity was measured 1, 2, 3, 4, and 7 days after Max-ECC. The order of the measurements was consistent during the experiment for pre and post exercise, starting with muscle soreness followed by CK, range of motion, arm circumference and lastly strength measures. Details of criterion measures including muscle strength, ROM, CIR, plasma CK activity, soreness and tenderness were discussed earlier in the method section of chapter 3.

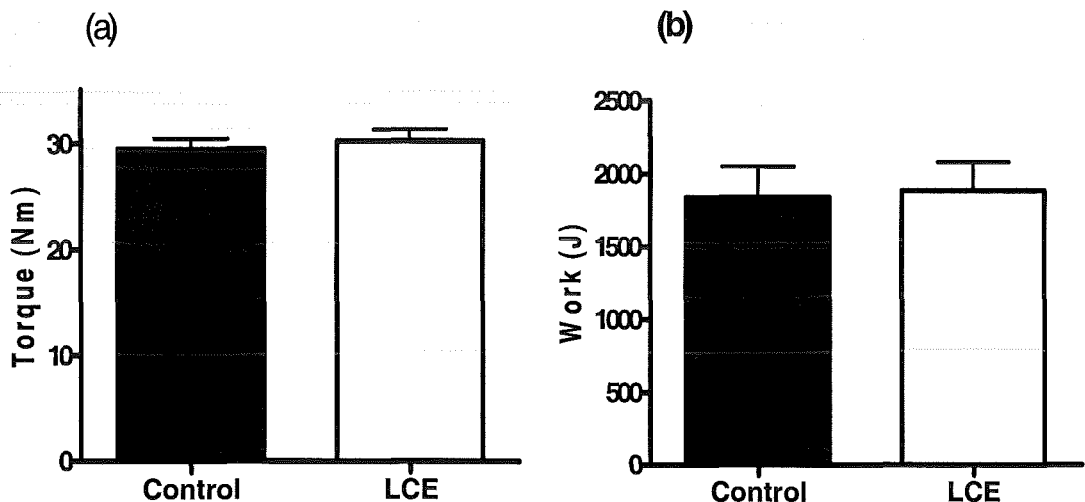
### 6.2.6 Data Analysis

Acute effects of the light concentric exercise on the measures were analysed using a one way repeated measures analysis of variance (ANOVA) by comparing the pre and post values on days 1-4. Changes in isometric and isokinetic muscle strength, ROM, upper arm circumference, and plasma CK activity over time were compared between control and exercise conditions using a two-way repeated measures ANOVA. When the ANOVA showed a significant difference between conditions, Tukey's post hoc test was applied to find the location of the significance. Data analysis was performed using a statistical software package (SPSS, version 11.0). A statistical significance was set at  $P < 0.05$  for all analyses. Data are presented as means  $\pm$  SEM unless otherwise stated.

## 6.3 Results

### 6.3.1 Exercise

All subjects completed the two bouts of Max-ECC similarly with no significant differences between bouts for work absorbed or peak torque generated during the exercise between arms (Figure 21). The mean peak torque of 60 contractions for the control and the LCE arms were  $29.5 \pm 1.0$  N, and  $30.3 \pm 1.1$  N, respectively, with no significant difference between the conditions. The total work during the exercise was  $1840.5 \pm 210.9$  J for the control and  $1881.9 \pm 197.0$  for the LCE arms, and no significant difference between arms was evident. Before Max-ECC, there were no significant differences in the pre exercise values for any of the criterion measures between conditions. All criterion measures changed significantly following exercise for both arm groups.



*Figure 21:* Comparison of peak torque (a) and total work (b) between control and light concentric exercise (LCE) condition for eccentric exercise.

### 6.3.2 Acute Effects of Light Concentric Exercise (LCE)

Subjects often reported some discomfort at the beginning of the LCE (early first sets), however, at no time did this prove too severe for subjects to continue, and all individuals were able to complete the 10 sets of 60 repetitions. Subjects also reported some level of fatigue towards the end of LCE protocol. Acute effects of LCE were evident for muscle strength, upper arm circumference, and muscle soreness and tenderness, but not for elbow joint angles and ROM.

#### 6.3.2.1 Muscle Strength

Table 7 shows changes in isometric torque before and after LCE. Isometric torque at 90° decreased from  $25.1 \pm 3.6$  Nm to  $19.9 \pm 3.2$  Nm (21%) after the first LCE performed 1 day post Max-ECC. A similar decrease in isometric torque ( $\approx 20\%$ ) was seen after subsequent bouts of LCE however, the difference between pre and post for day 3 was not significant. No significant decreases in isometric torque at an elbow joint angle of 30° from full extension were evident between pre and post on any of the test days.

Isokinetic torque decreased in a similar fashion to the isometric torque following LCE, and the magnitude of decrease was consistent for the 5 different velocities. For example, mean peak isokinetic torque at  $90^\circ\text{s}^{-1}$  decreased significantly from pre to post LCE on days 1 (10%) and 2 (10%), and on days 2 (13%) and 3 (9%).

Table 7.

*Maximal Isometric Torque at 90° And 30° Of Elbow Joint Angle Before (Pre) And Immediately After (Post) The Light Concentric Exercise On 1-4 Days (D1-D4) After Maximal Eccentric Exercise. Data are the Means And SEM Of 14 Subjects Expressed as N·M. \* Indicates Significant Difference From the Pre Value.*

			Maximal isometric torque (N·m)			
Angle			D1	D2	D3	D4
90°	Pre	Mean	25.1	26.9	26.9	29.3
		SEM	3.6	4.2	3.4	4.5
	Post	Mean	19.9*	23.1*	24.0	25.5*
		SEM	3.2	3.5	3.4	4.2
30°	Pre	Mean	18.7	18.4	17.9	20.1
		SEM	2.4	2.7	2.3	2.9
	Post	Mean	17.2	19.4	19.5	20.3
		SEM	2.4	2.9	2.9	3.2

#### 6.3.2.2 ROM and Elbow Joint Angles

No significant changes occurred for elbow joint angles (RANG, SANG and FANG) and ROM after LCE.

#### 6.3.2.3 Upper Arm Circumference

Significant increases in upper arm circumference were observed from pre to post LCE for the 5 measurement sites, and the amount of increase was similar across sites. All sites showed 2-3 mm increases on circumference immediately after LCE on days 1-4.

#### 6.3.2.4 Muscle Soreness and Tenderness

Muscle soreness decreased significantly immediately after LCE. Figure 22a illustrates the soreness reported with elbow extension pre and post LCE. As a result of LCE,

soreness scores decreased approximately 50% on day 1, and 46%, 31% and 39% on days 2, 3 and 4, respectively. The amount of change was not significantly different between days. Similar decreases in soreness score to the extension were observed after LCE for palpation (19-44%) and flexion (16-22%)

Figure 22b shows changes in the thresholds for muscle tenderness of the biceps brachii before and after LCE. In this case, lower values represent increases in muscle tenderness. A significant increase in the score was evident after LCE on days 1 (26%), 2 (35%), 3 (75%) and 4 (51%), indicating a decrease in muscle sensitivity to localised pressure of some 47% as a result of LCE.

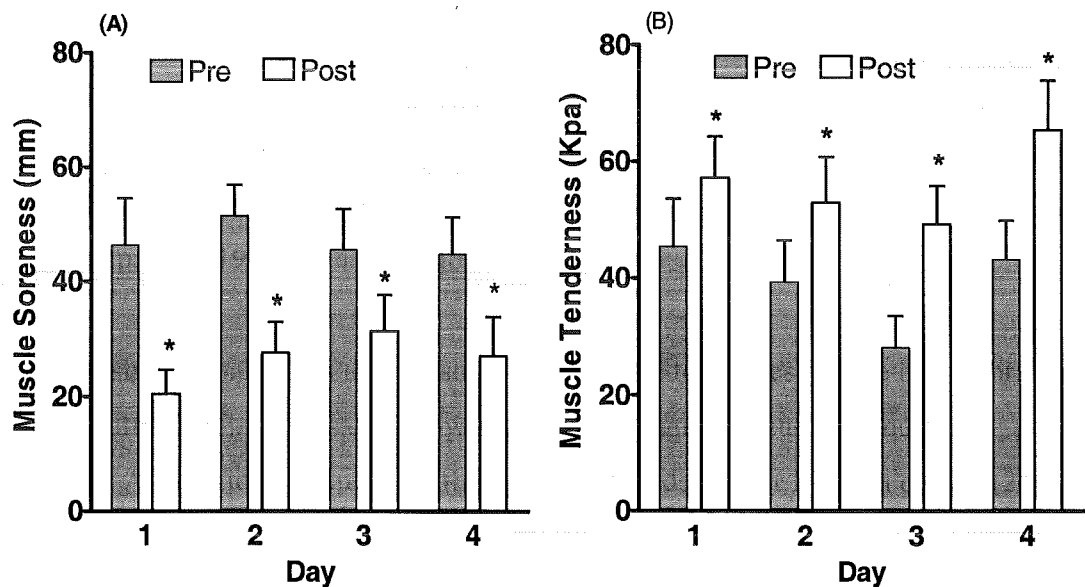


Figure 22: Changes in muscle soreness with extension (a) and muscle tenderness (b) before (Pre) and immediately after (Post) light concentric exercise on days 1 to 4 after maximal eccentric exercise. Mean  $\pm$  SEM of 14 subjects are shown. \* indicates significant difference from the pre-value.

### 6.3.3 Comparison between Arms for Changes in Criterion Measures after Max-ECC

In order to evaluate any longer-term effects of LCE, changes in criterion measures before, and for 7 days following Max-ECC were compared between the control and light exercise arms. For the latter group, measurements taken prior to performing LCE on each day were used. All measures changed significantly after Max-ECC, however, no significant differences between arm groups were evident for any of the variables.

#### 6.3.3.1 Muscle Strength

Maximal isometric torque at 90° decreased significantly immediately after Max-ECC ( $\approx 40\%$ ) for both control and exercise arms from baseline, remained below baseline for the next 4 days, and started to recover after 4 days post-exercise (Figure 23). However, isometric torque was still approximately 20% below baseline 7 days after Max-ECC. The findings were similar for the maximal isometric torque measured at an elbow flexion angle of 30°. No significant differences between the control and light exercise arms were evident for the changes in maximal isometric torque following Max-ECC.

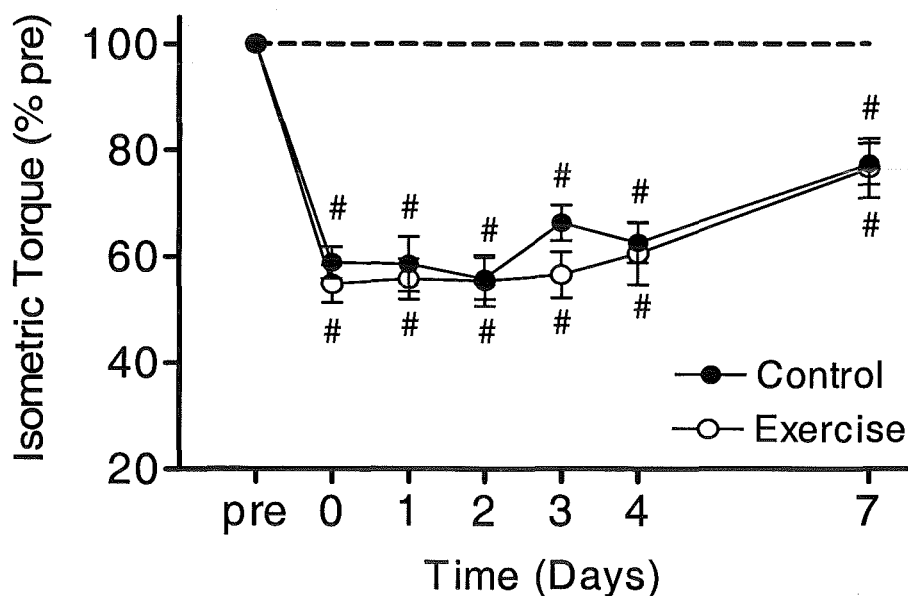


Figure 23: Normalized maximal isometric torque at 90° for control (Control) and light exercise (Exercise) arm before (pre), immediately after (0), and 1–7 days after maximal eccentric exercise. Mean  $\pm$  SEM of 14 subjects are shown. # represent significant different from the baseline

Maximal isokinetic torque at the five different velocities decreased following Max-ECC, and the magnitude of the decrease was similar between the five velocities. The time course of changes in isokinetic torque was similar to that of isometric torque, eg.  $90^{\circ}\text{s}^{-1}$  showed a decrease of approximately 35% from the pre-exercise value immediately after Max-ECC, had little recovery over the next 4 days, and remained diminished by 20-25% at 7 days post-exercise. No significant differences between the control and exercise arms were evident for the changes in any of the isokinetic torque variables following Max-ECC. For example, isokinetic torque at  $90^{\circ}\text{s}^{-1}$  before exercise was  $30.4 \pm 3.6$  Nm for the control, and  $28.3 \pm 3.3$  Nm for the exercise arm, and decreased to  $18.9 \pm 2.8$  Nm, and  $17.6 \pm 1.9$  Nm, respectively immediately after Max-ECC. Little recovery of the isokinetic torque was observed for the next 4 days, and the torque was still significantly lower than the baseline at 7 days after Max-ECC for the control ( $22.4 \pm 3.2$  Nm) and exercise ( $24.0 \pm 3.0$  Nm) arm.

#### *6.3.3.2 ROM and Elbow Joint Angles*

Figure 24 shows changes in ROM following Max-ECC. Both control and exercise arms showed similar changes in ROM after MAX-ECC. Mean ROM decreased from  $133.3 \pm 2.2^{\circ}$  to  $116.4 \pm 2.5^{\circ}$  immediately after exercise for the control arm and from  $133.8 \pm 2.6^{\circ}$  to  $108 \pm 4.8^{\circ}$  for the exercise arm, no significant changes occurred between 1-4 days post exercise, and were still approximately  $10^{\circ}$  smaller than the baseline 7 days after Max-ECC. No significant differences in elbow joint positions were observed between the conditions for RANG, SANG and FANG. The time course of changes in RANG and SANG was similar to that of ROM. FANG showed a large increase immediately to 1 day after exercise ( $\approx 18^{\circ}$ ) and a gradual recovery from day 2 to day 7, but was still  $3-4^{\circ}$  smaller at 7 days after MAX-ECC.



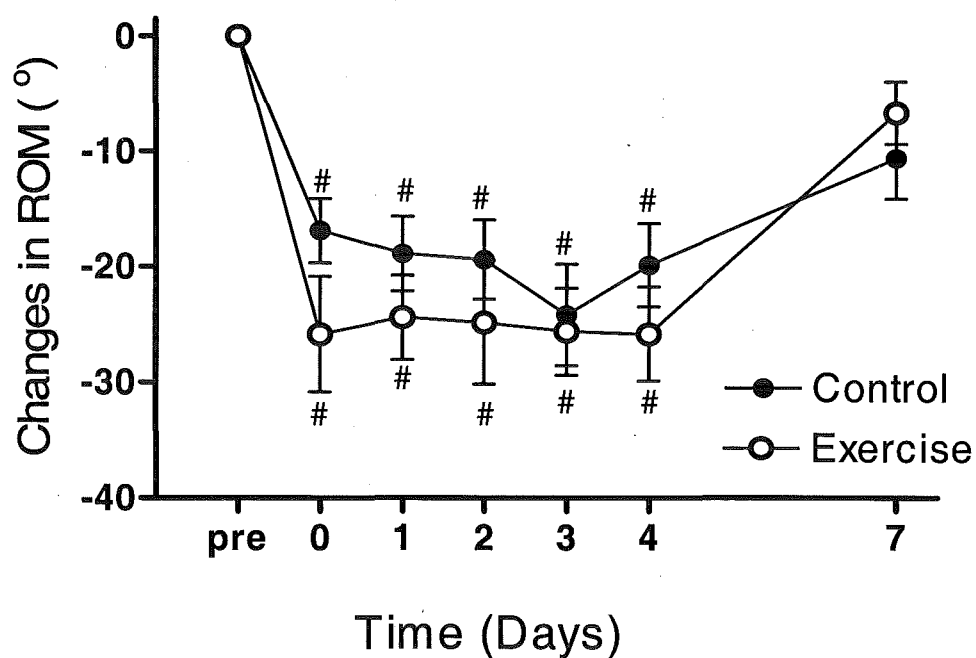
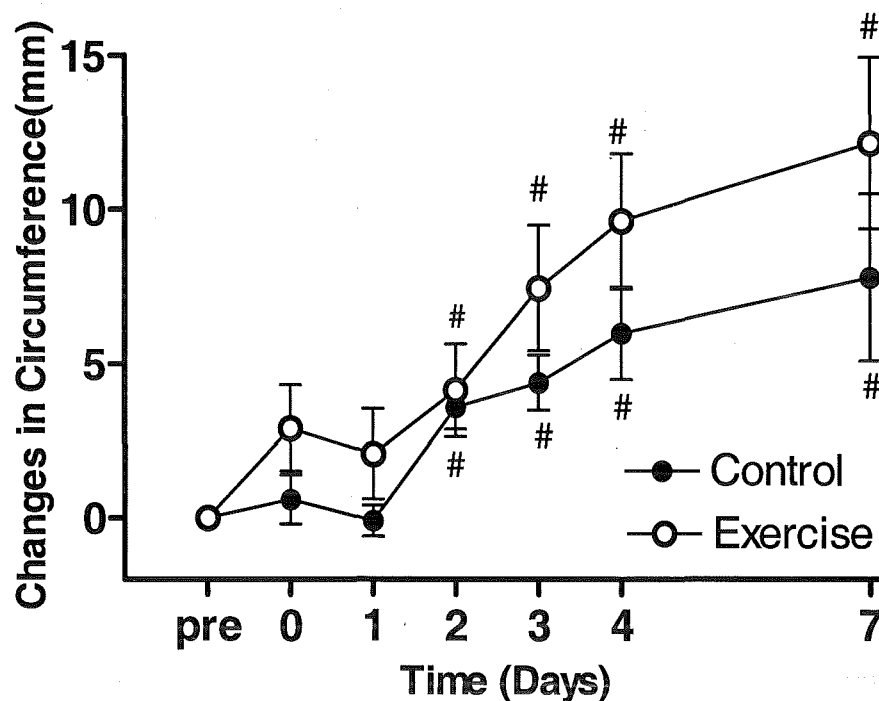


Figure 24: Changes in ROM from baseline for control (Control) and light exercise (Exercise) arm before (pre), immediately after (0), and 1–7 days after maximal eccentric exercise. Mean  $\pm$  SEM of 14 subjects are shown. # indicates significant different from the baseline.

### 6.3.3.3 Upper Arm Circumference

Upper arm circumference increased significantly, peaking 7 days after Max-ECC. The increases in circumference were similar between the five measured sites. Figure 25 shows the changes in upper arm circumference 5 cm proximal to the elbow joint. The amount of increase in the circumference from pre to 7 days post exercise was  $7.8 \pm 2.7$  mm for the control arm and  $12.7 \pm 2.8$  mm for the exercise arm. Although there was a tendency for the exercise arms to show larger increases, no significant differences between the arm conditions were evident.



*Figure 25:* Changes in upper arm circumference from baseline for control (Control) and light exercise (Exercise) arm before (pre), immediately after (0), and 1–7 days after maximal eccentric exercise. Mean  $\pm$  SEM of 14 subjects are shown. # indicates significant different from the baseline.

#### 6.3.3.4 Muscle Soreness and Tenderness

Muscle soreness developed 1 day after Max-ECC, peaked 1–4 days, and subsided by 7 days after exercise for palpation, flexion, and extension measures. Soreness score was highest with extension followed by palpation, and flexion. Figure 26a shows changes in muscle soreness with extension after Max-ECC. Both control and LCE arm groups showed a similar pattern, and no significant differences between the groups were evident. This was also the case for other soreness assessments.

As shown in Figure 26b, the pressure to elicit pain sensation in the biceps brachii decreased significantly 1–4 days after Max-ECC, and returned to the baseline at 7 days post exercise. This was similar for tenderness at 9 to 11 cm above the elbow crease, brachialis and brachioradialis, indicating that muscles became more sensitive to

pressure stimuli after Max-ECC. No significant differences in muscle tenderness between the exercise and control arms were observed.

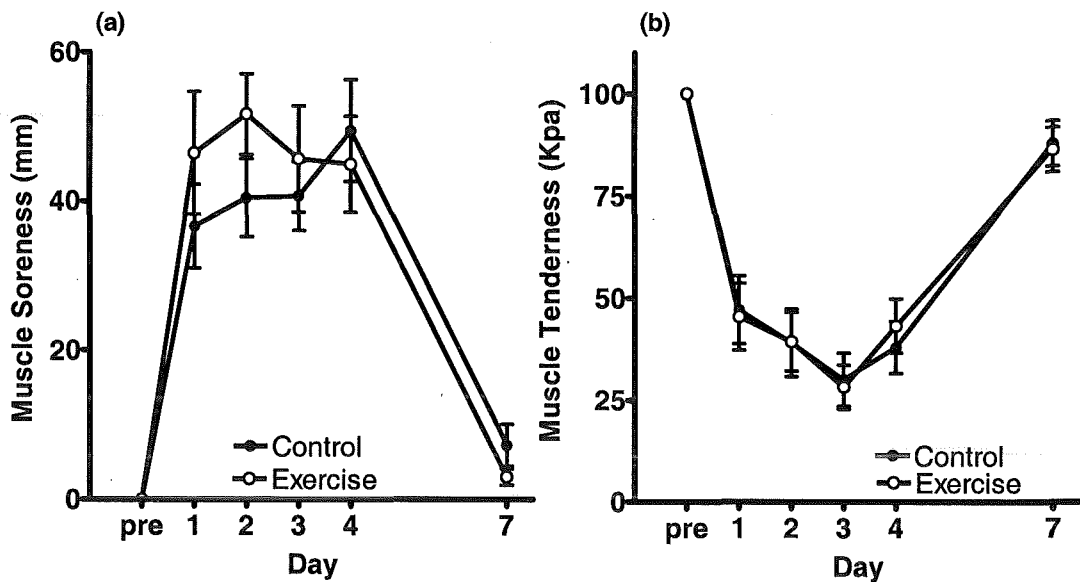
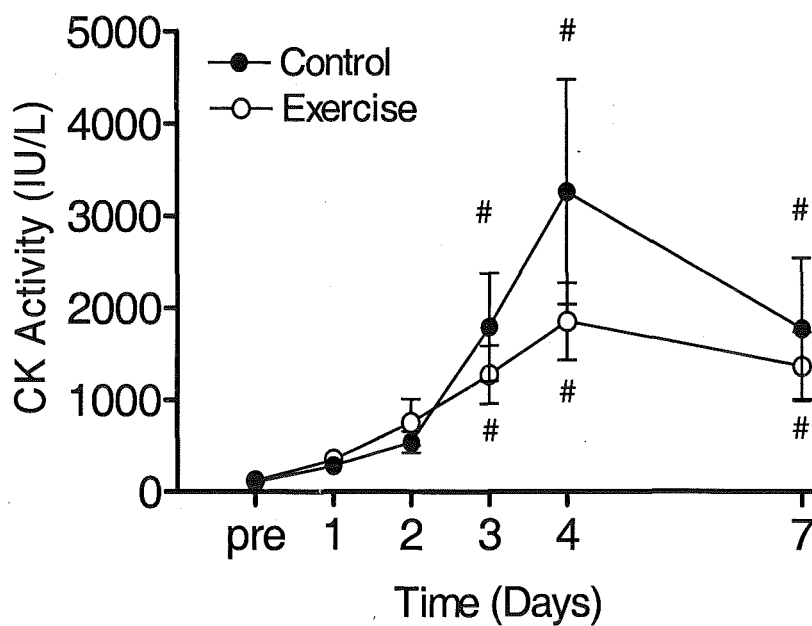


Figure 26: Changes in muscle soreness (a) and muscle tenderness (b) for control (Control) and for control (Control) and light exercise (Exercise) arm before (pre) and 1–7 days after maximal eccentric exercise. Mean  $\pm$  SEM of 14 subjects are shown.

#### 6.3.3.5 Plasma CK Activity

Figure 27 shows changes in plasma CK activity before and after Max-ECC. Plasma CK activity increased significantly, and peaked 4 days after Max-ECC. Although the peak CK values were, on average, some 45% lower for the LCE group, because of the large inter-subject variability in CK responses the changes in plasma CK activity were not significantly different between the two arm conditions.



*Figure 27:* Changes in plasma CK activity for control (Control) and light exercise (Exercise) arm before (pre) and 1–7 days after maximal eccentric exercise. Mean  $\pm$  SEM of 14 subjects are shown. # indicates significant difference from the pre value.

## 6.4 Discussion

Changes in maximal isometric and isokinetic torque, ROM, upper arm circumference, and plasma CK activity following Max-ECC were similar to those reported in previous studies in which a similar eccentric exercise protocol was used (Nosaka & Newton, 2002d). Similar changes in work and strength occurred during the maximal eccentric exercise of the elbow flexors (Max-ECC) between the control and exercise arms. Moreover, the changes in muscle strength (Figure 23), ROM (Figure 24) and upper arm circumference (Figure 25) were comparable between conditions immediately and 1 day after Max-ECC (i.e. prior to the first light concentric exercise). Thus, Max-ECC appeared to induce a similar degree of muscle damage to both arms, and any subsequent differences can be attributed to the effects of LCE.

This study investigated the hypothesis that a LCE performed 1-4 days after Max-ECC would alleviate DOMS and enhance recovery from muscle damage. The results showed that LCE had a short-lived palliative effect on DOMS, but no sustained therapeutic effect on either DOMS or other indicators of muscle damage. The transitory effects will be discussed first, and comparison between the control and LCE arm groups will then be addressed separately.

### 6.4.1 Acute Effects of Light Concentric Exercise (LCE)

More than 100 years ago, Hough (1902) reported that performing a second bout of exercise on the day after an activity that induced muscle soreness caused excessive pain for the first 2-3 minutes, which disappeared over the course of 5-10 minutes of exercise. It has been also anecdotally reported that DOMS is alleviated when affected muscles are exercised. However, only a few studies (Donnelly et al., 1992; Nosaka & Newton, 2002a, 2002d; Saxton & Donnelly, 1995) have investigated the palliative effect of exercise on DOMS, with somewhat conflicting findings. Furthermore, to the authors knowledge only four studies (Donnelly et al., 1992; Nosaka & Newton, 2002a, 2002d; Saxton & Donnelly, 1995) have reported changes in soreness level immediately after performing activity in the recovery days following damaging eccentric exercise.

The exercise protocol used in the present study was different from that used in previous reports. Saxton & Donnelly (1995) had subjects perform 50 concentric actions of the elbow flexors at 50% of maximum concentric force. 1-4 days after maximal eccentric exercise. An eccentric exercise protocol was performed in the other two studies, Donnelly et al. (1992) used 25 submaximal (< 50%) eccentric actions, whilst Nosaka & Newton (2002b) employed 3 sets of 10 eccentric actions using a dumbbell set at 50% of each subject's maximal voluntary isometric force. In the present study, the LCE was specifically designed to maximise blood and lymph flow with minimal fatigue, equating to a traditional warm-up exercise and resulting in a lower intensity intervention than that used in other studies. Although isometric torque at 90° and isokinetic torque decreased 10-20% immediately after LCE, this is more likely related to the effects of fatigue rather than muscle damage given the large number of contractions carried out. This is confirmed by the absence of any significant changes in elbow joint angle or ROM following LCE. Upper arm circumference did show a small (2-3 mm) but significant increase immediately after LCE which is likely to be associated with increased muscle perfusion (Sayers et al., 2000a).

The major finding of this study was that both muscle soreness and tenderness were reduced immediately after LCE (Figure 22). The palliative effect of LCE on muscle soreness and tenderness did not change over the course of the recovery period following eccentric exercise, and the magnitude of this effect was not influenced by the degree of muscle soreness and tenderness. Saxton and Donnelly (1995) reported that DOMS decreased by some 40% immediately after concentric exercise was performed 2 days after eccentric exercise, but no significant effect was evident on days 1, 3, and 4. However, the present study found the palliative effect of LCE on DOMS whenever the LCE was performed between 1 and 4 days after Max-ECC. Nosaka & Newton (2002d) reported that DOMS decreased 10-20% immediately after bouts of high intensity eccentric exercise bouts were performed at 2 and 4 days after the initial bout. This suggests that the extent of the analgesic effect of activity is not related to the intensity of the exercise performed as a much lighter exercise was used in the present study, resulting in a more pronounced reduction in muscle soreness and tenderness.

Although information regarding the analgesic effect of exercise on DOMS is limited, it has been well documented that both pain threshold and pain tolerance are raised following exercise, an occurrence referred to as exercise-induced analgesia (Koltyn, 2000). Most studies have investigated this phenomenon using endurance exercise protocols, and a few studies have reported an analgesic effect following resistance exercise. For example, Koltyn and Arbogast (1998) showed that pain threshold was significantly elevated for 5 minutes following a resistance exercise consisted of 45 min of weight lifting. Kosek and Ekholm (1995) reported that the pressure pain threshold of the quadriceps increased significantly during and after isometric exercise. The mechanism(s) of the analgesic effect of exercise, however, are poorly understood. Some suggestions include activation of the endogenous opioid system, release of endorphins, increases in blood pressure leading to elevated pain threshold, and a "gate control theory" (Goldfarb & Jamurtas, 1997; Hoffman & Thoren, 1988; O'Connor & Cook, 1999; Randich & Maixner, 1984).

Since the mechanism of DOMS is not clearly understood, it is difficult to explain how, in the present study, light concentric exercise reduced muscle soreness and tenderness. It is known that the sensation of pain in skeletal muscle is transmitted by myelinated group III and unmyelinated group IV afferent nerve fibres, with the former believed to elicit the sensation of sharp pain, whereas the latter are responsible for the perception of the dull aching pain commonly associated with exercise-induced muscle damage (Miles & Clarkson, 1994). Both types of pain afferents are sensitised and/or stimulated by chemicals such as the prostaglandin bradykinin, serotonin, histamine and potassium released from damaged muscles (Kendall & Eston, 2002; Keskula, 1996). It has also been shown that perception of the activity of pain afferents can be inhibited by endogenous opioids (Koltyn, 2000; O'Connor & Cook, 1999). The exact mechanism for this effect is uncertain but may involve direct action on the proximal terminals of small diameter primary afferents via selective inhibition of nociceptive input to dorsal horn neurons, or through processes occurring at higher levels of the CNS. The dorsal horn of the spinal cord is the location for the local interneurons containing enkephalins, serotonin, noradrenalin and associated opioid receptors which control excitatory and inhibitory effects on nociceptive projection neurons (O'Connor & Cook, 1999; Zimmermann, 1987). Armstrong (1984) cited a "close the gate" theory that relates to



interference of pain via increase of afferent input from large, low-threshold sensory muscle receptor units. This could explain the short-term effect on soreness and tenderness following LCE, since the light exercise will result in activity of other afferents (ie. groups Ia, Ib, II fibres) which may modify the pain sensations associated with group III and IV afferent activity.

Muscle swelling is a well known characteristic of exercise-induced muscle damage and has been implicated in the process of DOMS through increases in intra-muscular pressure resulting in the sensitisation of muscle pain afferents (Howell et al., 1993). It is interesting to note that upper arm circumference increased significantly immediately after LCE at the same time as muscle soreness and tenderness decreased (Figure 22). This would suggest that the 2-3 mm increase of the arm circumference did not stimulate the pain afferents nor sensitize them. In fact, it has been shown that swelling of muscle immediately after exercise does not cause pain (Howell et al., 1993). It would appear that the increases in upper arm circumference indicate increases in muscle blood or lymph flow that may contribute to removal of pain-eliciting substances from the damaged muscles. Thus, a further possibility to explain the reduction in DOMS following LCE is via the activity-related removal of noxious substances from the muscle tissue. Further research into these possibilities is clearly required to elucidate the mechanism for the acute analgesic effect of exercise on DOMS.

#### 6.4.2 Effect of Light Concentric Exercise on Recovery from Max-ECC

Although muscle soreness and tenderness were alleviated immediately after LCE, no difference between the control and LCE arms for the changes in muscle soreness and tenderness were found following Max-ECC (Figure 26). This suggests that the analgesic effect of the exercise was temporary. Since no measurements were taken between immediately after LCE and the next day, it is not known how long the analgesic effect lasted after LCE. Koltyn and Arbogast (1998) reported that the decreased pain threshold returned to baseline level within 15 minutes after resistance exercise.

No significant differences were found between the control and LCE arms for changes in muscle strength (Figure 23), ROM (Figure 24), upper arm circumference (Figure 25) and plasma CK activity (Figure 27) following Max-ECC. These results indicate that recovery of muscle function was not affected by the LCE.

Using a similar exercise protocol to the present study, Saxton and Donnelly (1995) showed that a light concentric exercise did not affect changes in CK, strength and ROM after exercise. Weber et al. (1994) also reported no significant effect of upper arm ergometry applied immediately and 24 hours after eccentric exercise of the elbow flexors on DOMS and muscle function. The results of the present study are consistent with their findings. In contrast, Donnelly et al. (1992) reported that a light eccentric exercise reduced or delayed increases in serum CK activity approximately 30%. This was not the case for the present study, which may be due to the different type of exercise used.

On the other hand, some studies showed a positive effect of exercise on recovery from muscle damage. Hasson et al. (1989) reported that a high speed voluntary contraction of quadriceps was effective in alleviating DOMS and facilitating recovery of muscle function after 120 voluntary maximum knee extension at  $300^{\circ}\text{s}^{-1}$ . Light exercise consisting of 50 biceps curls of a 5 lb dumbbell was found facilitate recovery of maximal isometric force by approximately 30% compared to control condition and to reduce muscle soreness by a similar amount (Sayers et al., 2000a). It is not yet clear whether performing exercise in the early phase following eccentric exercise-induced muscle damage is beneficial for the recovery. More studies are necessary to specify the type, intensity, and duration of activity, if it is to be used as a therapeutic modality.

It should be noted that no adverse effect of LCE on recovery from muscle damage was evident. It has been reported that muscle adapts rapidly after eccentric exercise and becomes less susceptible to the same exercise already in an early recovery phase (Chen, 2003; Ebbeling & Clarkson, 1990; Nosaka & Newton, 2002d). Since low intensity concentric exercise does not induce muscle damage, it seems unlikely that any exercises consisting of mainly concentric muscle actions performed in the recovery phase from

eccentric exercise induce further damage and retard recovery. However, this does not necessarily mean that no additional injury occurs by performing an exercise with sore muscles. It should be cautioned that the risk of injury may increase when an exercise is performed with sore and weak muscles (Nosaka and Newton, 2002b).

In conclusion, light eccentric exercise performed daily after maximal eccentric exercise inducing muscle damage has a temporarily analgesic effect on muscle soreness and tenderness, however, no beneficial effects on alleviating DOMS and enhancing recovery of muscle function were found. Further study is necessary to investigate how the analgesic effect is produced, and how long it lasts. It is also important to determine the optimum strategies for rest and/or activity in aiding recovery from eccentric exercise-induced muscle damage.

## CHAPTER 7

### COMPARISON BETWEEN IMMOBILISATION, MASSAGE, AND LIGHT CONCENTRIC EXERCISE

#### 7.1 Introduction

The purpose of this chapter is to compare the effects of three therapeutic treatments, immobilisation, massage and light concentric exercise, investigated separately in previous chapters (4 to 6), on DOMS and indicators of muscle damage. Since the same exercise protocol and criterion measures were used in the studies shown in Chapter 4 to 6, and subjects were recruited from the same population, the three treatments are comparable. Moreover, the control arm in the three studies showed similar changes in all criterion measures, when the control data of the three studies were compared by two-way repeated measures ANOVA. The comparisons between the three treatments will answer the question which of the three interventions is the best for DOMS and recovery of muscle function after eccentric exercise, and whether a sore muscle should be rested or moved.

If DOMS is a warning signal not to use or not to move the sore muscle, complete rest would be the best choice however, active mobilization of muscles recovering from injury is also known to enhance recovery and reduced soreness (Sayers et al., 2000a). Comparison between immobilisation and light concentric exercise will provide an answer to the question whether sore muscles need rest or not. In this context, massage treatment is not necessarily associated with movement. However, massage treatment is one of the most widely used treatments for soft tissue injuries (Robertson et al., 2004; Tiidus, 1997), and it can be considered as a 'passive' movement treatment. In fact, the increases in blood and lymph flow that occur during massage are similar to those which take place during exercise (Smith, Keating et al., 1994; Tiidus, 1997). For this reason, comparison of the effects of massage on DOMS and recovery of muscle function in comparison to immobilisation and light exercise treatments is of interest.

## 7.2 Methods

Since no significant differences in control data were evident between the three studies, all data from the control arm were pooled and used as a control condition. Therefore, the control data represent the mean values of 34 subjects. Data from the treatment arm that underwent either immobilization ( $n = 10$ ), massage ( $n = 10$ ) or light concentric exercise ( $n = 14$ ) were compared. It is important to note that the timing of the interventions was not the same among three studies. In this study, the massage was applied to the exercised arm three hours after exercise, the immobilisation was started 30 minutes after exercise and maintained for four days, and the light concentric exercise was performed intermittently at 1, 2, 3, and 4 days after exercise. Changes in all criterion measures before, immediately after and 1 to 7 days following exercise were compared between immobilisation, massage, and light concentric exercise treatments using a two way repeated measures ANOVA. When any significant main effects were established by the ANOVA, Tukey's Post Hoc analysis was used to locate the significant difference. Data was analysed using the statistical software package (SPSS, version 11.0) with significance set at  $p < 0.05$ . Data are presented as mean  $\pm$  SEM unless otherwise stated.

## 7.3 Results

Similar baseline values for the criterion measures were evident and no significant differences in the changes of the measures immediately after exercise for the four conditions (Table 8). Soreness with palpation, extension and flexion were recorded '0' for all subjects before exercise, and no differences between the conditions were seen. Muscle tenderness values recorded as 100kPa before exercise for all subjects with no differences between conditions. These results suggest that exercise was performed equally, and no differences between the conditions were evident before the treatments were applied.

Table 8.

*Before (PRE) and Immediately after Exercise (POST) Values for Isokinetic Strength at 90°s<sup>-1</sup> (STRENGTH), Range of Motions (ROM) and Arm Circumference (CIR) for Control (CON), Immobilization (IMMO), Massage (MSG), and Light Concentric Exercise (LCE).*

			CONDITIONS			
			CON	IMMO	MSG	LCE
STRENGTH (Nm)	PRE	Mean	30.4	25.5	26.3	28.3
		SEM	3.7	4.2	5.7	3.3
	POST	Mean	18.8	16.8	15.3	17.6
		SEM	2.86	3.1	3.11	1.94
ROM (°)	PRE	Mean	133.3	127.0	130.3	133.9
		SEM	2.2	3.6	2.6	2.7
	POST	Mean	116.4	110.5	114.8	108.0
		SEM	2.58	3.8	4.7	4.8
CIR (mm)	PRE	Mean	251.4	247.4	246.5	259.2
		SEM	7.7	9.3	8.0	7.4
	POST	Mean	251.8	249.4	248.5	263.1
		SEM	7.9	9.5	7.2	7.2

### 7.3.1 Muscle Soreness and Tenderness

Figure 28 shows changes in muscle soreness upon palpation. No significant differences were evident between immobilization, massage, and exercise conditions and soreness subsided by 7 days for all conditions. Similar results were obtained for soreness while extending and flexing the elbow joint.

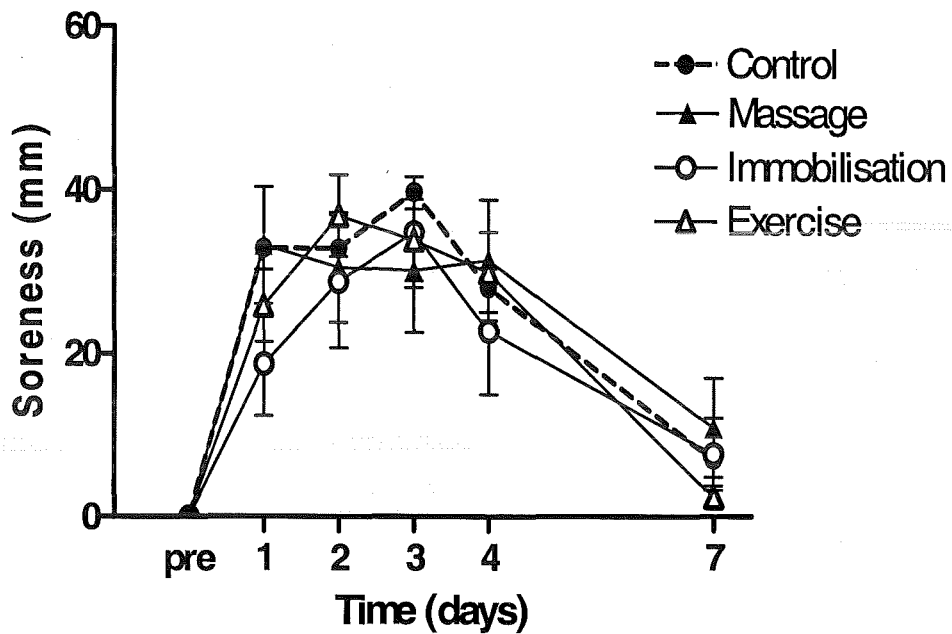


Figure 28: Changes in soreness upon palpation before (pre) and 1 to 7 days post exercise for control, massage, immobilization and exercise conditions.



There were no significant differences between conditions recorded for tenderness at 10 cm above the elbow crease as shown in Figure 29. Similar results were also obtained for tenderness at 4 cm above elbow crease for brachialis and brachioradialis.

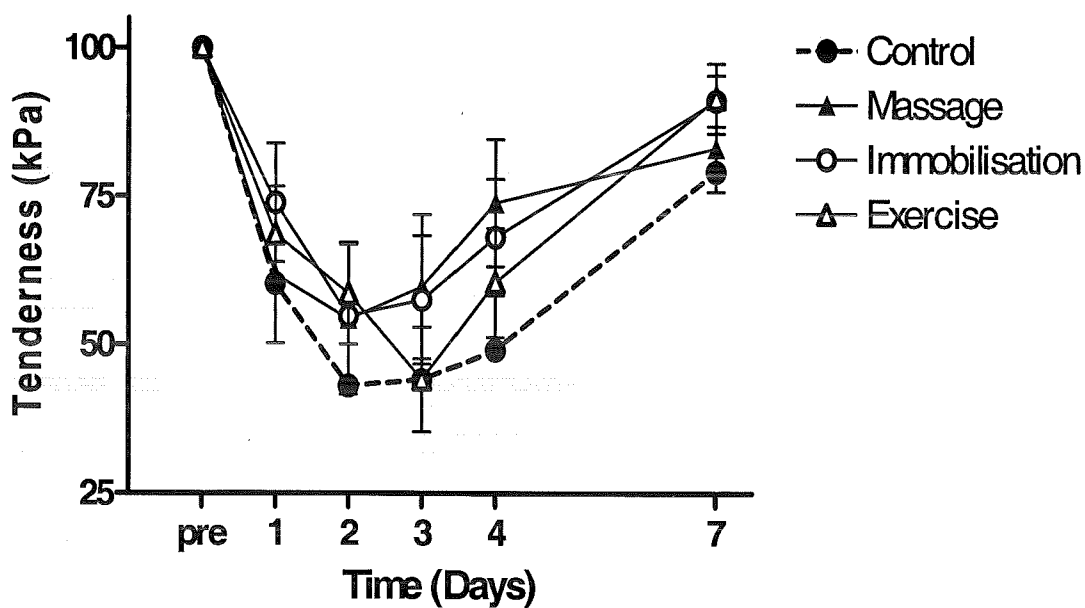
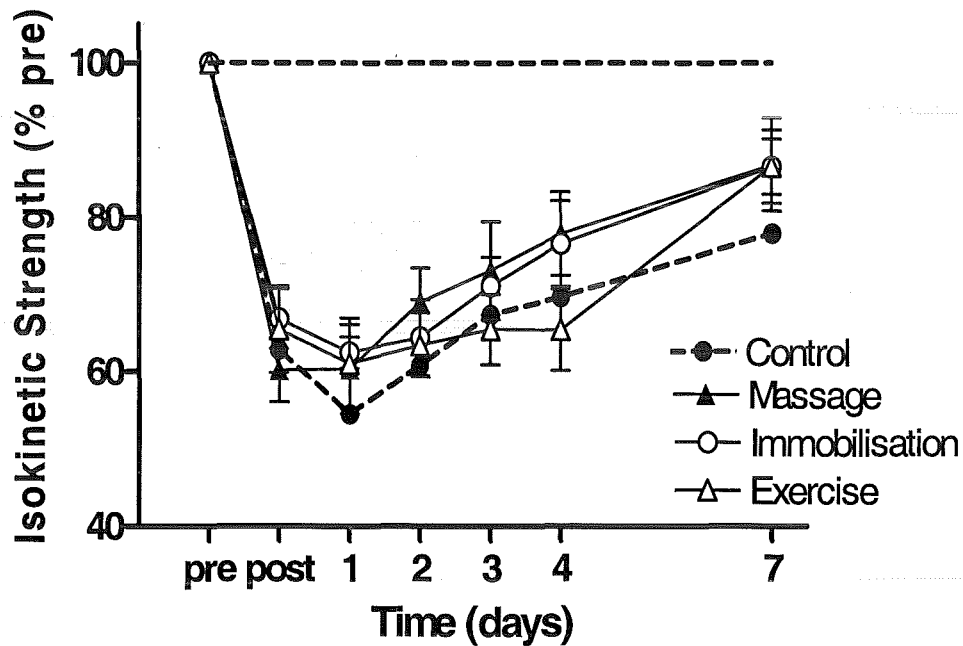


Figure 29: Changes in tenderness before (pre) and 1 to 7 days post exercise for control, massage, immobilization and exercise conditions.

### 7.3.2 Muscle Strength

Figure 30 shows changes in isometric strength at 90° of the elbow flexion before and 1 to 7 days following eccentric exercise. No significant differences between the conditions were evident for the changes. This was also the case for isometric strength at 30° and isokinetic strength at different velocities ( $30^{\circ}\text{s}^{-1}$ ,  $90^{\circ}\text{s}^{-1}$ ,  $150^{\circ}\text{s}^{-1}$ ,  $210^{\circ}\text{s}^{-1}$  and  $300^{\circ}\text{s}^{-1}$ ).



*Figure 30:* Changes in isometric strength following eccentric exercise from pre exercise (pre), immediately after exercise (post) and day 1 to day 7 (1 to 7) post exercise for control, massage, immobilization and exercise conditions.

### 7.3.3 ROM

Changes in ROM were not significantly different between the immobilisation and exercise, and massage conditions (Figure 31). However, significantly smaller changes in ROM were evident for the massage compared to the exercise condition on day 3 and 4 post exercise.

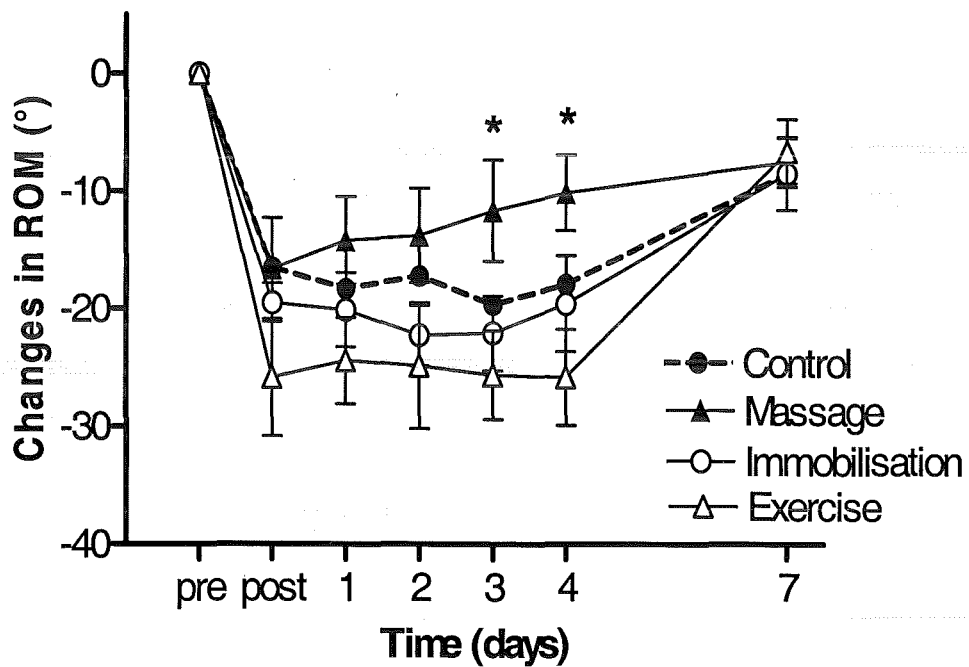


Figure 31: Changes in range of motion (ROM) following exercise from pre exercise (pre), immediately after exercise (post) and day 1 to day 7 (1 to 7) post exercise for control, massage, immobilization and exercise conditions.

\* indicates significant different ( $P < 0.05$ ) between massage and exercise condition.

### 7.3.4 Upper Arm Circumference

No significant differences between conditions were observed until 4 days post-exercise, however, at day 7, upper arm circumference was significantly small for the immobilisation condition compared to the exercise condition (Figure 32). Other than these, no significant differences were evident between conditions.

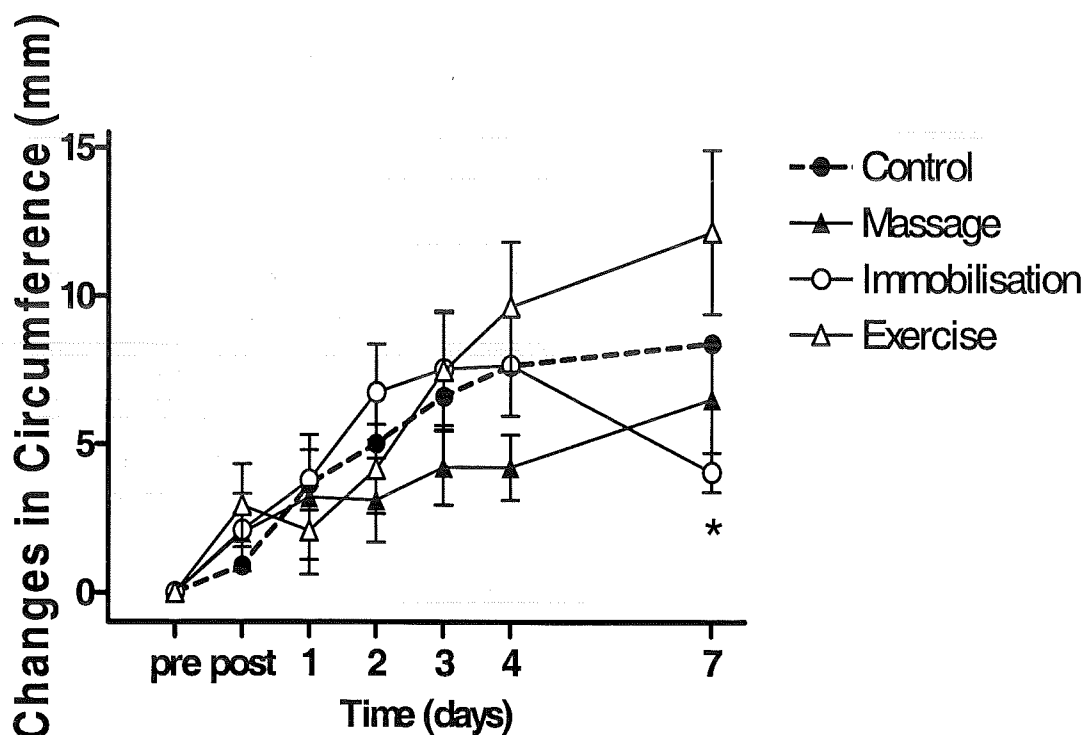


Figure 32: Changes in arm circumference following exercise from pre exercise (pre), immediately after exercise (post) and 1 to 7 days post exercise for control, massage, immobilization and exercise conditions.

\* indicates significant difference between immobilisation and exercise condition

### 7.3.5 Plasma CK Activity

No significant differences between the conditions were evident for changes in plasma CK activity except between massage and control conditions (Figure 33).

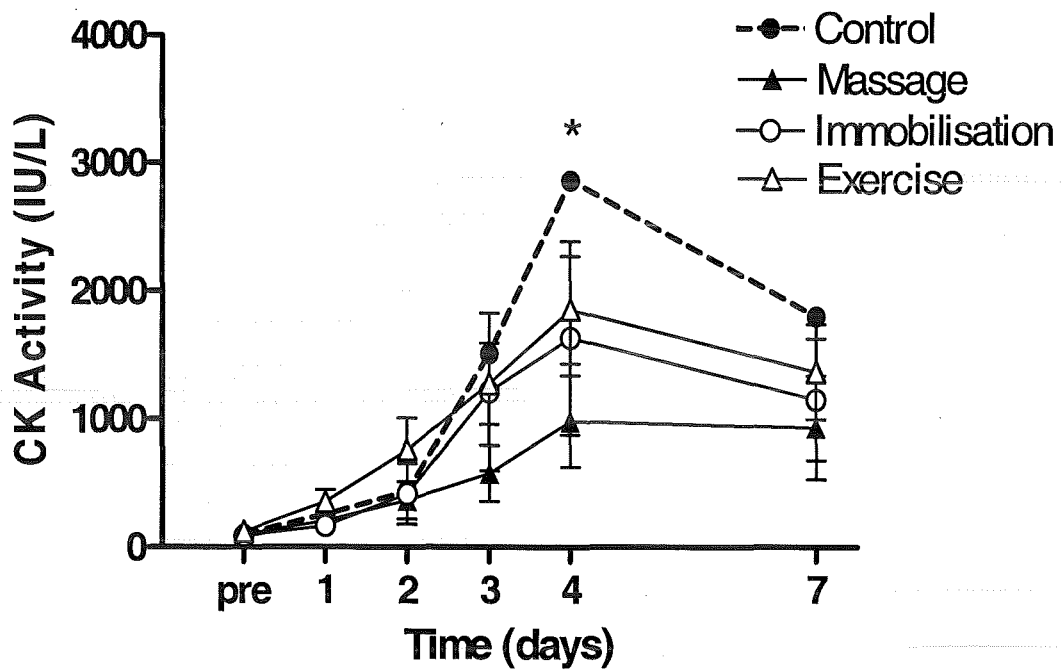


Figure 33: Changes in plasma CK activity CK following exercise from before (pre), and 1 to 7day post exercise for control, massage, immobilization and exercise conditions.

\* indicates a significant difference between massage and control condition.

## 7.4 Discussion

### 7.4.1 Effects on Muscle Soreness and Tenderness

No significant differences between the immobilization, massage, and light exercise conditions were evident for soreness (Figure 28) and tenderness following eccentric exercise. A small, but significant difference from the control condition was evident for massage when muscles were palpated or flexed as shown in chapter 5 (Table 6). It is difficult to explain how massage reduces DOMS but it is possible that enhanced local blood and lymph flow which in turn reduced oedema, or accumulation of substances causing pain (Ernst, 1998). It seems reasonable to assume that light massage is also capable of increasing local blood and lymph flow to a larger extent than massage. If the reduced DOMS and swelling in the massage condition is associated with the enhanced circulation, similar effects on muscle soreness and upper arm circumference should have been seen for the exercise condition. However, the light concentric exercise was only found to reduce muscle soreness temporarily. It is important to note that the massage treatment was performed 3 hours after exercise, when muscle soreness had not yet developed, but the first light concentric exercise was performed 1 day after exercise, when muscle soreness already existed. This would suggest that the time when applying the intervention may be a contributing factor for this result. However, such difference was not observed for the muscle tenderness and the reduction of muscle soreness by massage was small and seen only at one time point. Therefore, it seems reasonable to conclude that the effect of massage on muscle soreness and tenderness is limited.

Since no significant differences between the immobilisation and exercise conditions were found in this study, it seems reasonable to conclude that muscle soreness is not affected by movement. This suggests that it makes minimal difference on DOMS whether sore muscles are rested or used. Muscle soreness subsided by 7 days after exercise for all conditions. Once muscle soreness develops, no treatments seem effective in reducing muscle soreness significantly, although a temporary effect may be induced by some treatments including light concentric exercise, which showed a palliative effect in this study. To reduce DOMS, it may be necessary to do something before muscle soreness develops. In fact, several prophylactic treatments have been shown to prevent or reduce a potential development of DOMS (Donnelly et al., 1988; Kaminski & Boal, 1992; Miller, Bailey, Barnes, Derr, & Hall, 2004; Nosaka & Newton,

2002a). It would appear that the best strategy to prevent or reduce DOMS is a prophylactic approach. DOMS disappears naturally in a week or so without any treatment, and physical interventions do not affect this process.

#### 7.4.2 Effects on Muscle Strength and ROM

According to Warren et al. (1999), maximal torque and ROM provide the best measure of muscle injury resulting from eccentric exercise. Muscle function is important for examining the effect of a treatment, since one of the purposes of therapeutic treatment is to enhance recovery of muscle function such as muscle strength and ROM. It is interesting to note that none of the interventions in this study produced significant effects on recovery of strength (Figure 30) and ROM (Figure 31) following exercise compared to control condition. It seems reasonable to conclude that nothing could be done for the recovery of muscle function after eccentric exercise. It is possible that a certain amount of time is necessary for the recovery process after eccentric exercise, and the time required for recovery cannot be shortened by therapeutic treatments.

It has been reported that additional eccentric exercise bouts in an early recovery period from initial eccentric exercise do not induce additional muscle damage nor retard the recovery process (Chen, 2003; Nosaka & Newton, 2002c). However, a certain period of complete rest is necessary for most soft tissue injuries (Jarvinen & Lehto, 1993). This might suggest that eccentric exercise-induced muscle damage is different from other soft tissue injuries. It should be also noted that an early mobilization of injured soft tissue enhance recovery by preventing scar tissue formation and promoting regeneration (Jarvinen & Lehto, 1993; Jarvinen et al., 2000). However, it seems unlikely that this is the case for eccentric exercise-induced muscle damage. None of the therapeutic treatments investigated in this series of studies, have succeeded in enhancing recovery of muscle function after eccentric exercise. Like DOMS, once eccentric exercise impaired muscle function, it seems that only a time can solve the problem. Therefore, a prophylactic approach rather than therapeutic approach is necessary to reduce the magnitude of decrease in muscle function following eccentric exercise.



#### 7.4.3 Effects on Swelling

Changes in arm circumference represent the amount of swelling in response to inflammation (Chleboun et al., 1998). Swelling was less for massage treatment on day 3 and 4 post exercise compared to the control condition as shown in chapter 5 (Table 5). This indicates that among the three interventions observed, massage was found effective in reducing swelling. Since swelling is one of cardinal signs of acute inflammation (Smith, 1991), it seems that massage was effective in ameliorating inflammatory response. Immobilisation was found to reduce swelling at 7 days post exercise in comparison to control condition as shown in chapter 4 (Figure 13). It is interesting to note that the immobilisation intervention was applied for four days post exercise. It is possible to speculate that the significant decrease in arm circumference on day 7 observed in the immobilisation condition could be due to a rapid 'flush-out' of accumulated fluid and other substances in the injured area provided by movements after immobilization treatment, however, this assumption is unlikely since daily movement of the injured arm provides in the light exercise condition does not seem to be effective in reducing swelling.

Swelling itself may not be a problem if it is not accompanied with functional impairment or pain. In fact, the time course of swelling does not match with changes in muscle soreness, strength, and ROM. Even if a treatment is effective in reducing swelling, if it does not affect muscle soreness and muscle function, from a practical point of view, its relevance is open to question.

#### 7.4.4 Effects on Plasma CK Activity

No significant differences in CK response were evident between immobilisation, massage, and light concentric exercise control conditions (Figure 33). The large increases in plasma CK activity indicate that muscle cell damage occurred following eccentric exercise (Clarkson, Litchfield, Graves, Kirwan, & Byrnes, 1985; Nosaka & Clarkson, 1996b). This would suggest that neither treatments used in this study provide beneficial effect in reducing muscle cell damage following eccentric exercise. Indeed, it is again strengthening the idea that once muscle cell damage occurred, there is little one can do to stop or reduce it. Although increases in plasma CK activity were smaller for the massage condition compared to the control on day four post-exercise as shown in

chapter 5 (Figure 20), it does not seem to indicate that the membrane damage was attenuated by massage. Since no significant differences in muscle function were observed between massage and control condition, the difference shown in plasma CK activity between them does not appear to have practical significance.

## 7.5 Conclusion

A different level of mobility used in this study as therapeutic intervention provides no significant beneficial effect on DOMS and other symptoms of muscle damage. Only small differences were observed between the massage and control conditions on soreness, swelling, and plasma CK activity, and immobilisation and control on upper arm circumference. According to the comparisons between immobilisation and light concentric exercise treatments, it can be concluded that muscle soreness and recovery of muscle function are not dependent on movement. At the moment, no therapeutic treatments appear to work effectively for reducing DOMS and enhancing recovery of muscle function action after exercise. At the moment, the normal practice is to wait until the symptoms dissipate by itself. It should be cautioned that the risk of injury may increase when an exercise is performed with sore and weak muscles (Nosaka and Newton, 2002b).

It is unfortunate that no effective therapeutic treatments on DOMS and recovery of muscle function are available at this moment. Investigation of the mechanism of DOMS and eccentric exercise-induced muscle damage is still in progress. Understanding the mechanism better may provide a breakthrough therapeutic treatment. At this moment, the mechanism of DOMS and muscle damage are still mystery. Our understanding of DOMS has not been progressed dramatically since Hough (1902) described DOMS more than a century ago. More research is needed to understand the mechanism of DOMS to provide a beneficial treatment to prevent or palliate DOMS and other symptoms of muscle damage either therapeutic or prophylactic.

It should be noted that limitations always exist for this kind of research. The more we try to separate the effect of an intervention on something, the further it will become from reality. For example, when someone has massage after exercise, he or she usually

expects to have some benefit from the treatment. It may be that the expectation itself can make a difference. However, in a study that especially focuses on physiologic parameters, we usually ignore the psychological aspects. To reduce the psychological effect, placebo treatment is often used. However, having a placebo treatment may not effectively control for the psychological effects.

In this study, the arm-to-arm comparison model was used. This model appears to be advantageous when comparing two conditions in relatively small number of subjects; however, it also has some disadvantages. The possibility of carryover effects cannot be denied, and experiencing muscle soreness and other symptoms induced from one bout may affect the perception of pain and other measures in another bout.

It is very difficult to control all factors that may influence the results of the study. It is assumed that the difference between the control and intervention conditions was due to the treatment, but other factors, such as food intake, sleeping time, and activities during the experimental periods, may influence results. Often little control of subjects outside of the laboratory is possible, and we must trust that subjects follow the instructions we give.

Statistical significance alone does not convey clinical importance; in fact, the statistical analysis may mask "evidence." For example, a 0.1-s difference is enormous for the 100-m sprint, but it is tricky to show the difference as statistically significant, even if an intervention consistently and effectively improves the time by 0.1-s. It was found that muscle soreness was reduced about 30% by massage and upper arm circumference was decreased, although the latter difference was small. The pre-exercise upper arm

circumference was approximately 240 mm for both arms, so the 5-mm difference was only 2%. A reduction in soreness of 30% is likely clinically important, but a 2% reduction in arm circumference likely has less clinical importance. It may be that the non-significant differences are important. When considering the pre-exercise maximal voluntary contraction (approximately 37 N·m), the difference of 4.1 N·m is more than 10%. For the athlete who resumes training or competition, a 10% deficit may affect performance.

Based on the findings from this study, the best treatment to ameliorate DOMS and muscle damage is yet to be determined. It is obvious from the findings that the light concentric exercise is the most beneficial treatment for DOMS, however only temporary analgesic effect was observed. Immobilization intervention on the other hand may positively reducing swelling while massage was effective for reducing DOMS. Since different interventions produced different results related to DOMS and muscle damage following EIMD, future research might consider of combining interventions to investigate its response to DOMS and muscle damage. It is also important to have a larger number of subjects to confirm the results of the present study.

## CHAPTER 8

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## APPENDIX A – FLYERS

# SUBJECTS WANTED FOR SPORTS SCIENCE RESEARCH



A research project is underway to comparing left and right upper arm responses towards different therapeutic interventions following a single bout of exercise

Healthy person aged between 18 – 45 years old without any history of upper arm injury are required to be subjects.

Exercise testing (14 visits) will be conducted at the Exercise Physiology laboratory of the School of Biomedical and Sports Science on the Joondalup Campus of Edith Cowan University.

To register your interest, please contact

**ZAINAL ZAINUDDIN** on

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**APPENDIX B – INFORMED CONSENT FORM**

## **INFORMED CONSENT FORM**

**Project Title:** The Effect Of Massage, Immobilisation and Light Concentric exercise During Recovery On Parameters Associated With Muscle Damage Following Eccentric Exercise

### **Purpose of Investigation**

The research will look at the effect of three interventions during recovery after muscle damage. The interventions consist of massage, immobilisation and light concentric exercise. Parameters to be measured are muscular strength, delayed onset muscle soreness (DOMS), range of motion, upper arm circumference and plasma creatine kinase (CK) activity. The study aims to evaluate the effectiveness of different strategies to reduce the temporary muscle soreness and loss in strength associated with high intensity muscle contractions. The major findings serve to indicate efficacy of various interventions in aiding muscle recovery subsequent to eccentric loading

### **Procedure of Study**

This investigation will be conducted over seven-day period at Edith Cowan University, Joondalup campus. Subjects participating in the investigation will be required to answer a medical questionnaire as outlined by American College of Sports Medicine (ACSM) to exclude any underlying injury or illness which may influence the findings. If you agree to participate, you will be asked to perform eccentric exercise with one arm. Several measurements described below will be taken immediately before and after exercise, and 1,2,3,4 and 7 days following exercise.

The following measurements will be taken from the exercised arm.

#### **Maximal Isometric Strength**

You will be asked to generate maximal voluntary force of the elbow flexors for 3 second against an immovable lever arm of Cybex 6000 at fixed elbow joint. Two efforts will be allowed at each joint angle and the highest torque production of the two will be recorded.

#### **Maximal Isokinetic Strength**

You will be asked to generate maximal voluntary force of the elbow flexors against an immovable lever arm of Cybex 6000 at 5 different velocities which are  $30^{\circ}\text{s}^{-1}$ ,  $90^{\circ}\text{s}^{-1}$ ,  $150^{\circ}\text{s}^{-1}$ ,  $210^{\circ}\text{s}^{-1}$  and  $300^{\circ}\text{s}^{-1}$ . Two efforts will be allowed at each velocities and the highest torque production of the two will be recorded

### **Range of Movement**

Goniometer will be used to measure the Range of movement (ROM) of the elbow joint. ROM will be assessed in three ways. Relaxed, stretched and flexed arm angle will be taken. Four marks will be placed on the arm to obtain consistent measurements positions.

### **1 Arm Circumference**

Five sites will be marked on exercise arm to indicate 3, 5, 7, 9 and 11 centimeters from the elbow crease. Circumference of the upper arm marked point will be measured using a special tape measure.

### **Muscle soreness and Tenderness**

Muscle soreness will be rated by extending and flexing the arm by holding the elbow. You will be asked to mark your soreness rating on a visual Analog Scale (VAS) which incorporates 100 mm line (0 indicating no tenderness, and at 100 indicating very painful). Muscle tenderness of 4 sites will be access by palpating the marked on forearm between 3-5 cm, 9-5 cm, brachialis and brachioradialis using a Dobros myometer (Eston 1996). The myometer will be applied at each side with increasing pressure and you will be asked to report the moment pain is perceived. The pressure reading coinciding with the onset of pain will be recorded as the tenderness score.

### **Creatine Kinase**

About 30 $\mu$ l (i.e. One drop) of blood will be collected in a heparinised tube following the piercing of your finger-prick with a spring-loaded lancet. Blood collection will occur prior to the eccentric exercise task and at 1, 2, 3, 4, and 7 days post exercise. The blood will be immediately assessed by spectrophotometer for serum creatine kinase activity.

### **Eccentric Exercise**

The exercise session, and determination of Maximal Isokinetic Torque, will consist of contraction of the elbow flexor muscles using the isokinetic dynamometer.

The exercise will consists of performing 10 sets of 6 maximal voluntary eccentric contractions of the elbow flexors against the lever arm of the isokinetic dynamometer moving at a constant velocity of 90° per second.

### **Testing**

At least one session of pre exercise test familiarization will be conduct. During this test you will be tested in strength test on an isokinetic dynamometer (Cybex 6000) including isometric an isokinetic test where you need to flex your arm to the full tension at 90° and 150 °.

## **Interventions**

After exercise, one of three different interventions will be applied to your upper arm after eccentric exercise. These interventions include

Massage. Consisting of 15-20 minutes sports massage to the exercised arm will be applied 2 hours after eccentric exercise.

Immobilisation. 90° sling will be applied to an exercised arm immediately after exercise for the period of seven days.

Light concentric exercise: 10 minutes of light concentric exercise of the elbow flexors will be performed using a Cybex isokinetic dynamometer involving 30% of maximal isometric contraction.

## **Risks and discomforts**

You are likely to experience some soreness, tenderness and stiffness in the bicep and triceps brachii area of the exercised arm, in the days following the eccentric exercise protocol. This will be similar to the feelings you would normally experience after a hard session of unaccustomed activity. However, any discomfort will dissipate within 5-7 days post exercise. Every effort will be made to minimize the risk of abnormal blood pressure, fainting, heart attack, stroke or death by evaluation of preliminary information relating to your health and fitness and by observation during testing. Emergency equipment and trained personal are available to deal with unusual situations that may arise. You also expect some temporary weakness in the arm, but this should not prevent you undertaking normal daily activities. All parameters will be carefully monitored on a daily basis by the investigators. This type of exercise protocol has been used many times before in this laboratory and others, and no subjects have reported any long term effects of participation.

All of the interventions have been shown to cause any additional muscle damage.

The investigators(s) ask that you don't make any changes to your daily activities, eating plan or take any medications, anabolic steroids, anti-inflammation drug(s) or similar as this may influence the results during experiment. Since the study involved exercise protocol, it is required for you to be healthy at exercise time and also required to complete the medical questionnaires provided.

## **Responsibilities of the subject**

The information you, the subject, possess about your health status or previous experiences of unusual feelings with physical effort may affect the safety and value of your exercise test. Your prompt reporting of feelings with effort during exercise test itself is also a great importance. You are responsible for fully disclosing such information when requested by the researcher(s)

## **Benefits to the subjects**

The results and experience obtained from the exercise test and participation in the present investigation may assist you in understanding recovery methods and recovery times following exercise-induced muscle damage and the effects of massage,

compressions and immobilisation on muscle damage recovery. Information gained may provide the subject with periodization strategies for training following competition(s).

### **Inquiries**

Questions concerning this investigation may be directed to

Zainal Abidin Zainuddin	Researcher PhD candidate	email: <a href="mailto:z.abidin@ecu.edu.au">z.abidin@ecu.edu.au</a> 63045152 (o) or 92423631 (h)
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Assoc. Professor Kazunori Nosaka	Supervisor	email: <a href="mailto:k.nosaka@ecu.edu.au">k.nosaka@ecu.edu.au</a> 63045655
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### **Address**

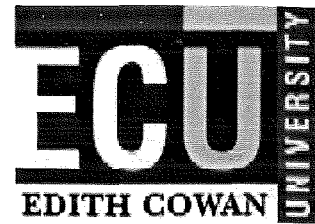
School of Biomedical and sports Science  
Faculty of Computing Health and Science  
Edith Cowan University  
Joondalup Drive  
Joondalup WA 6027

If you have any concerns or complaints about the research project and wish to talk to an independent person, you may contact:

Assoc. Prof. Barry Gibson  
The Head of School,  
School of Biomedical and Sports Science  
Edith Cowan University  
Joondalup Drive  
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Email address : [b.gibson@ecu.edu.au](mailto:b.gibson@ecu.edu.au)  
Phone Number : 6304 5037  
Fax : 6304 5106





**Project Title:** The Effect Of Massage, Immobilisation and Light Concentric exercise  
During Recovery On Parameters Associated With Muscle Damage  
Following Eccentric Exercise

**Freedom of Consent:**

Your permission to perform this exercise test is voluntary. You are free to stop the test at any point, if you so desire, and you may withdraw at any time for any reason.

All information will be secured under lock and key by the researchers. Data entry and analysis of that data will not include any name or information that may identify or link an individual as a subject.

I agree that the research data gathered for this study may be published provided I am not identifiable

I \_\_\_\_\_ (the participant) have read this form, and I understand the best procedures that I will perform and the associated risks and discomforts have had any questions answered to my satisfaction. I agree to participate in this research project.

Signature of participant: \_\_\_\_\_ Date \_\_\_\_:\_\_\_\_:\_\_\_\_

Signature of Investigator:  
Zainal A. Zainuddin : \_\_\_\_\_ Date \_\_\_\_:\_\_\_\_:\_\_\_\_

Signature of Supervisor:  
Assoc. Prof Kazunori Nosaka : \_\_\_\_\_ Date \_\_\_\_:\_\_\_\_:\_\_\_\_

If you have any additional question please contact :

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**APPENDIX C – MEDICAL QUESTIONNAIRE**

# Medical Questionnaire

The following questionnaire is designed to establish a background of your medical history, and identify any injury and/or illness that may influence your testing and performance. Please answer all questions as accurately as possible, and if you are unsure about any thing please ask for clarification. All information provided is strictly confidential. If you answer “yes” to any non-exercise related question that may contraindicate you from completing this study a clearance from a qualified medical practitioner will be required prior to commencement of any exercise or testing.

## Personal detail

Name:\_\_\_\_\_ ID number : \_\_\_\_\_

Date of Birth (D/M/Y) : \_\_\_\_\_ Gender : Female/Male

## Medical History

Have you ever had, or do you currently have any of the following?

If YES, Please provide details

High or abnormal blood pressure	Yes No	_____
High Cholesterol	Yes No	_____
Rheumatic fever	Yes No	_____
Heart abnormalities	Yes No	_____
Asthma	Yes No	_____
Diabetes	Yes No	_____
Epilepsy	Yes No	_____
Recurring back pain	Yes No	_____
Recurring neck pain	Yes No	_____
Severe allergies	Yes No	_____
Any infectious disease	Yes No	_____
Any neurological disorders	Yes No	_____
Any neuromuscular disorders	Yes No	_____
Are you currently on medications?	Yes No	_____
Have you had a flu in the last 2 weeks?	Yes No	_____
Have you recently injured yourself?	Yes No	_____
Do you have any recurring muscle or joint injuries?	Yes No	_____

Have you had any elbow or shoulder problems in the past?

Yes No \_\_\_\_\_

Have you participated in resistance Training in the last 12 months?

Yes No \_\_\_\_\_

Is there any other condition not previously mentioned which may effect your upper arm exercise?

Yes No \_\_\_\_\_

## LIFESTYLE HABITS

Do you exercise regularly?

Yes No

If YES, what do you do?

\_\_\_\_\_

How many hours per week?

\_\_\_\_\_

Do you smoke tobacco?

Yes No

If YES, how much per day?

\_\_\_\_\_

Do you consume alcohol?

Yes No

If YES, how much per week?

\_\_\_\_\_

Do you consume tea or coffee?

Yes No

If YES, how many cups per day?

\_\_\_\_\_

Do you take any recreational drug?

Yes No

If YES, what, and how much or how often a week?

\_\_\_\_\_

## DECLARATION

I acknowledge that the information provided on this form, is to the best of my knowledge, a true and accurate indication of my current state of health

## PARTICIPANT

Name : \_\_\_\_\_ Date \_\_/\_\_/\_\_ Signature: \_\_\_\_\_

## PRACTITIONER (Only if applicable)

I, Dr \_\_\_\_\_ have read the medical questionnaire and information/ consent form provided in the study entitled : The Effect Of Massage, Compression and Immobilisation During Recovery On Parameters Associated With Muscle Damage Following Eccentric Exercise

Date \_\_/\_\_/\_\_

Signature: \_\_\_\_\_

APPENDIX D – SORENESS RATE

Name \_\_\_\_\_ Bout

Exercised arm L R STUDY \_\_\_\_\_

D1 D2 D3 D4 D7 D10 D14

Upper arm

Palpation 1 ( 3-5) 0 \_\_\_\_\_ 100

Palpation 2 (9-11) 0 \_\_\_\_\_ 100

Palpation 3 0 \_\_\_\_\_ 100

Palpation 4 0 \_\_\_\_\_ 100

Extension 0 \_\_\_\_\_ 100

Flexion 0 \_\_\_\_\_ 100

D1 D2 D3 D4 D7 D10 D14

Upper arm

Palpation 1 ( 3-5) 0 \_\_\_\_\_ 100

Palpation 2 (9-11) 0 \_\_\_\_\_ 100

Palpation 3 0 \_\_\_\_\_ 100

Palpation 4 0 \_\_\_\_\_ 100

Extension 0 \_\_\_\_\_ 100

Flexion 0 \_\_\_\_\_ 100

APPENDIX E – RAW DATA

# APPENDIX E1 - RAW DATA FOR IMMOBILIZATION STUDY

			pre	post	30min	day 1	day 2	day3	day 4	day 7	Anova
ISO 90	Control	Mean	33.98	20.39	20.16	19.72	19.51	22.18	21.01	26.41	Not Sig
		SD	16.14	6.82	7.44	5.70	4.79	7.04	6.29	9.22	
	IMM	Mean	40.02	22.61	22.01	22.40	25.47	26.54	28.30	33.32	
		SD	18.48	10.20	10.24	10.31	10.58	11.82	13.94	17.71	
ISO 150	Control	Mean	24.25	14.97	15.74	12.45	13.06	14.12	14.77	18.41	Not Sig
		SD	12.24	7.95	7.78	5.01	4.07	6.03	4.76	5.48	
	IMM	Mean	27.06	18.32	18.48	17.25	18.87	19.04	19.96	22.99	
		SD	12.95	10.76	9.78	7.07	5.26	6.70	10.31	12.73	
FV30	Control	Mean	22.55	14.30	14.30	12.48	13.50	15.77	16.38	18.06	Not Sig
		SD	10.37	5.51	5.75	5.35	5.11	5.07	6.14	5.52	
	IMM	Mean	26.23	18.33	18.11	17.26	16.71	17.23	19.47	22.02	
		SD	12.32	12.12	11.45	7.86	6.86	6.03	8.49	11.05	
FV90	Control	Mean	23.30	14.03	13.96	11.38	12.75	14.03	15.17	17.59	Not Sig
		SD	11.91	5.81	7.62	5.89	4.12	5.04	4.98	6.72	
	IMM	Mean	25.48	16.76	15.10	15.95	15.81	16.98	18.04	21.73	
		SD	13.30	9.59	9.13	8.79	7.70	6.19	6.71	11.64	
FV150	Control	Mean	20.00	13.02	13.42	10.20	11.75	13.69	13.90	15.17	Not Sig
		SD	10.19	6.34	6.91	4.48	4.30	4.65	6.30	6.06	
	IMM	Mean	23.75	15.62	15.24	15.03	14.44	16.06	16.26	19.55	
		SD	12.00	9.03	9.53	6.32	6.41	7.65	8.76	10.66	
FV210	Control	Mean	19.73	11.41	11.95	11.08	11.08	11.28	13.34	15.57	Not Sig
		SD	11.19	6.85	6.94	5.86	5.54	3.68	6.84	7.72	
	IMM	Mean	21.75	14.98	14.90	13.79	12.72	16.55	14.64	16.77	
		SD	11.76	9.05	10.26	8.04	8.50	8.47	9.26	11.14	
FV300	Control	Mean	16.38	11.08	12.22	11.34	11.28	10.34	11.68	13.83	Not Sig
		SD	10.61	9.33	7.89	6.38	5.83	5.22	6.08	8.78	
	IMM	Mean	20.67	13.60	13.41	13.96	12.75	13.68	15.63	16.01	
		SD	12.50	8.76	9.98	7.65	7.89	9.44	11.60	12.12	



			pre	post	30min	day 1	day 2	day3	day 4	day 7	Anova
RANG	Control	Mean	156.20	152.50	152.70	147.90	148.30	148.80	147.10	149.60	Not Sig
		SD	6.80	8.61	8.79	8.49	6.02	6.34	5.00	8.88	
	IMM	Mean	153.50	149.20	147.80	145.40	142.80	141.00	143.00	148.40	
		SD	9.20	10.16	9.27	8.29	10.46	14.08	13.02	9.66	
SANG	Control	Mean	169.70	166.50	166.40	161.70	162.90	159.80	159.10	163.10	Not Sig
		SD	7.24	6.45	6.42	5.27	5.93	6.91	7.17	9.96	
	IMM	Mean	167.40	164.40	163.80	159.00	155.90	154.50	157.20	163.20	
		SD	10.81	9.16	9.62	10.64	11.84	15.23	15.55	10.93	
FANG	Control	Mean	35.80	50.50	46.80	47.30	46.10	46.30	43.20	37.80	Not Sig
		SD	5.71	8.96	8.70	8.51	9.02	11.67	7.67	7.08	
	IMM	Mean	32.50	47.70	45.40	44.50	44.10	42.10	41.60	36.00	
		SD	5.93	9.64	7.18	9.55	8.23	8.25	8.62	5.54	

			pre	post	30min	day 1	day 2	day3	day 4	day 7	Anova
CK	Control	Mean	86.78	/	/	234.24	394.48	1343.07	2454.91	1824.21	Not Sig
		SD	36.37	/	/	161.21	327.80	880.20	1896.61	1963.57	
	IMM	Mean	83.98	/	/	165.85	414.28	1210.98	1631.39	1149.95	
		SD	17.64	/	/	126.49	750.61	1945.25	2387.27	1505.14	
CIR 3	Control	Mean	23.24	23.30	23.30	23.62	23.83	23.96	24.09	24.15	0.01
		SD	2.25	2.14	2.08	2.20	2.31	2.43	2.33	2.55	
	IMM	Mean	23.71	23.76	23.97	24.05	24.24	24.40	24.41	24.12	
		SD	2.67	2.60	2.97	2.70	3.03	3.06	3.05	2.64	
CIR 5	Control	Mean	23.94	24.12	23.96	24.35	24.57	24.79	24.84	25.00	0.01
		SD	2.53	2.65	2.46	2.40	2.43	2.62	2.69	2.51	
	IMM	Mean	24.74	24.94	25.00	25.06	25.47	25.48	25.48	25.25	
		SD	2.94	3.02	3.15	2.94	3.26	3.30	3.36	3.08	
CIR 7	Control	Mean	24.62	24.87	24.84	25.23	25.33	25.53	25.70	25.87	0.02
		SD	2.75	2.60	2.63	2.61	2.51	2.76	2.49	2.64	
	IMM	Mean	25.45	25.66	25.74	25.90	26.19	26.37	26.41	25.85	
		SD	3.00	3.08	3.23	3.18	3.38	3.60	3.66	2.89	
CIR 9	Control	Mean	25.32	25.60	25.48	25.94	26.13	26.19	26.26	26.59	0.02
		SD	2.65	2.78	2.71	2.63	2.69	2.78	2.63	2.58	
	IMM	Mean	26.04	26.27	26.31	26.37	26.73	26.76	26.86	26.47	
		SD	3.00	3.21	3.25	3.23	3.48	3.41	3.36	2.96	
CIR 11	Control	Mean	25.92	26.25	26.33	26.68	26.70	26.78	26.90	27.01	0.01
		SD	2.72	2.85	2.97	2.78	2.69	2.87	2.67	2.64	
	IMM	Mean	26.60	26.96	26.88	27.05	27.28	27.29	27.20	26.87	
		SD	3.20	3.42	3.39	3.34	3.56	3.67	3.38	3.13	

			pre	post	30min	day 1	day 2	day3	day 4	day 7	Anova
TEN UPP	Control	Mean	257.50	/	/	125.10	94.30	96.10	98.50	204.70	Not Sig
		SD	50.62	/	/	78.01	87.31	79.07	85.64	92.32	
	IMM	Mean	257.50	/	/	136.80	127.80	108.00	171.50	230.00	
		SD	50.62	/	/	73.89	72.82	80.94	76.81	73.48	
TEND FORE	Control	Mean	100.00	/	/	53.10	44.60	54.10	49.70	71.20	Not Sig
		SD	0.00	/	/	35.86	33.98	34.93	39.16	33.27	
	IMM	Mean	100.00	/	/	73.80	54.80	57.50	68.00	91.00	
		SD	0.00	/	/	31.74	38.62	34.18	30.84	13.70	

			pre	post	30min	day 1	day 2	day3	day 4	day 7	Anova
SOR UPP	Control	Mean	0.00	/	/	112.80	109.70	112.10	95.20	49.30	Not Sig
		SD	0.00	/	/	65.78	55.32	48.97	40.01	52.99	
	IMM	Mean	0.00	/	/	95.00	118.00	107.10	72.40	40.60	
		SD	0.00	/	/	72.83	78.72	59.50	56.50	60.61	
SOR FORE	Control	Mean	0.00	/	/	43.50	27.40	29.00	22.70	13.40	Not Sig
		SD	0.00	/	/	26.27	15.56	16.80	16.92	23.77	
	IMM	Mean	0.00	/	/	18.70	28.80	34.80	22.70	7.70	
		SD	0.00	/	/	20.02	25.68	21.48	24.45	14.05	
SOR EXT	Control	Mean	0.00	/	/	37.90	39.50	43.00	45.80	6.60	Not Sig
		SD	0.00	/	/	25.80	21.93	21.05	15.75	12.69	
	IMM	Mean	0.00	/	/	31.60	50.00	49.30	27.30	9.20	
		SD	0.00	/	/	28.71	30.29	25.60	25.45	16.18	
SOR FLEX	Control	Mean	0.00	/	/	38.60	37.50	34.90	35.00	3.50	Not Sig
		SD	0.00	/	/	23.79	22.76	22.36	24.40	8.21	
	IMM	Mean	0.00	/	/	29.40	37.40	31.90	18.60	4.20	
		SD	0.00	/	/	21.39	25.49	21.46	24.86	13.28	

			pre	post	30min	day 1	day 2	day3	day 4	day 7	Anova
ROM	Control	Mean	120.40	102.00	105.90	100.60	102.20	102.50	103.90	111.80	Not Sig
		SD	6.64	6.46	7.78	10.84	7.13	8.81	8.76	12.73	
	IMM	Mean	121.00	101.50	102.40	100.90	98.70	98.90	101.40	112.40	
		SD	11.38	11.90	10.98	14.70	12.19	17.87	17.79	12.38	

# APPENDIX E2 - RAW DATA FOR MASSAGE STUDY

			pre	post	30min	D1	D2	D3	D4	D7	D10	D14	Anova
ISO 90	CON	Mean	37.17	23.58	23.63	22.29	22.14	25.81	24.87	29.88	32.24	33.14	Not Sig
		SD	21.89	12.62	12.83	10.66	9.10	14.44	16.23	18.80	17.10	20.12	
	MSG	Mean	37.33	25.14	24.02	24.92	25.87	29.22	30.85	33.47	36.22	37.47	
		SD	19.93	14.83	12.59	15.85	17.45	20.25	21.69	20.68	19.98	20.18	
ISO 150	CON	Mean	27.99	18.93	19.77	17.65	16.36	19.21	18.79	22.49	23.85	25.42	Not Sig
		SD	17.79	12.41	13.34	10.84	7.85	13.87	11.34	12.01	12.39	13.72	
	MSG	Mean	26.58	16.19	17.48	16.30	20.76	22.12	23.46	25.53	27.21	27.26	
		SD	11.32	7.42	9.29	9.81	12.30	13.93	11.73	11.57	13.34	12.61	
FV30	CON	Mean	25.84	17.32	16.71	14.83	15.98	19.00	20.21	21.61	22.18	23.29	Not Sig
		SD	15.04	10.19	10.27	8.75	7.90	12.53	13.20	13.07	12.96	14.26	
	MSG	Mean	25.59	17.70	17.85	18.86	19.55	21.00	23.02	23.09	25.70	25.43	
		SD	13.75	9.34	8.84	13.85	12.28	14.20	13.58	12.53	13.14	14.75	
FV90	CON	Mean	26.45	16.65	16.51	14.83	15.50	16.45	18.80	20.14	21.71	22.55	Not Sig
		SD	15.79	11.21	12.11	10.69	8.26	10.10	12.71	12.33	12.89	14.05	
	MSG	Mean	26.26	15.30	16.51	16.65	17.79	18.79	20.27	22.08	24.74	23.43	
		SD	18.05	9.84	11.69	13.97	12.40	14.23	14.37	14.36	13.69	14.86	
FV150	CON	Mean	23.76	15.71	15.84	13.16	14.63	17.12	17.53	18.32	20.68	22.15	Not Sig
		SD	13.93	9.90	10.26	10.09	9.13	11.14	11.77	11.45	12.10	13.26	
	MSG	Mean	23.83	15.57	14.83	15.64	17.25	17.86	17.92	19.80	21.14	21.65	
		SD	15.39	9.55	9.51	12.42	13.17	13.75	13.14	14.04	13.78	13.92	
FV210	CON	Mean	22.69	14.97	15.17	14.57	14.70	14.84	17.50	18.32	19.47	19.87	Not Sig
		SD	14.39	10.06	10.89	9.21	10.24	10.17	13.19	13.02	10.54	13.23	
	MSG	Mean	21.63	14.16	14.30	14.83	16.04	17.72	17.85	19.00	19.94	18.93	
		SD	14.36	10.15	10.39	11.00	12.42	13.18	12.51	12.79	13.72	11.86	
FV300	CON	Mean	19.80	14.84	16.04	14.50	15.04	14.17	14.77	16.24	19.20	18.06	Not Sig
		SD	13.13	11.91	11.53	9.22	10.77	10.25	10.45	11.32	11.19	11.81	
	MSG	Mean	19.33	13.22	14.23	13.96	15.24	17.25	16.71	17.05	19.40	18.26	
		SD	13.40	10.28	11.08	12.26	11.74	11.80	12.45	13.04	13.07	11.46	

			pre	post	30min	D1	D2	D3	D4	D7	D10	D14	Anova
RANG	CON	Mean	154.9	152.5	151.8	147.1	148.5	149.4	145.9	147.9	151.2	151.3	Not Sig
		SD	8.43	9.80	9.92	9.46	6.50	6.52	4.72	9.13	8.73	11.72	
	MSG	Mean	153.5	152.4	152.7	149.9	149.0	147.4	148.5	153.8	153.5	153.5	
		SD	4.60	6.72	6.40	6.81	8.15	7.50	7.20	6.16	4.67	4.84	
SANG	CON	Mean	166.1	165.1	165.4	161.5	161.4	159.0	155.9	159.5	162.6	166.2	Not Sig
		SD	7.37	7.72	8.06	6.93	5.72	5.98	5.80	9.36	8.85	8.72	
	MSG	Mean	163.2	163.5	164.1	162.8	160.2	160.4	161.2	165.7	164.4	164.2	
		SD	4.94	7.58	7.32	8.59	7.05	8.91	9.45	8.21	7.37	7.25	
FANG	CON	Mean	34.80	49.00	45.60	46.60	45.20	45.10	43.60	38.50	34.10	34.10	Not Sig
		SD	6.49	9.55	8.10	8.75	9.22	12.52	7.38	5.38	3.57	4.41	
	MSG	Mean	32.90	49.40	46.80	46.10	41.50	39.30	37.50	35.80	33.90	33.70	
		SD	7.77	12.34	10.11	7.98	6.88	4.16	4.20	5.22	4.09	3.47	

			pre	post	30min	D1	D2	D3	D4	D7	D10	D14	Anova
CK	CON	Mean	98.04	/	/	217.5	328.3	1274.	2704.	1812	325.0	130.3	0.03
		SD	41.29	/	/	117.2	264.3	873.2	2014.	1977.	251.0	43.26	
	MSG	Mean	99.25	/	/	206.6	365.0	573.9	981.9	930.3	237.6	134.0	
		SD	35.15	/	/	141.5	465.0	689.1	1125.	1283.	215.2	85.74	
CIR 3	CON	Mean	23.77	23.82	23.86	24.10	24.23	24.36	24.47	24.56	24.07	23.98	Not Sig
		SD	2.18	2.09	2.18	2.08	2.07	2.20	2.05	2.18	2.01	2.11	
	MSG	Mean	24.05	24.08	23.92	24.10	24.20	24.33	24.47	24.47	24.17	24.11	
		SD	2.26	2.13	2.02	2.11	2.06	2.32	2.36	2.33	2.11	2.15	
CIR 5	CON	Mean	24.54	24.63	24.57	24.88	25.09	25.25	25.32	25.54	24.99	24.87	Not Sig
		SD	2.49	2.54	2.62	2.39	2.43	2.41	2.51	2.37	2.37	2.50	
	MSG	Mean	24.65	24.85	24.71	24.97	24.96	25.07	25.07	25.30	25.04	24.88	
		SD	2.53	2.29	2.31	2.55	2.51	2.72	2.58	2.53	2.43	2.39	
CIR 7	CON	Mean	25.42	25.65	25.58	25.94	26.01	26.20	26.46	26.51	26.07	25.90	Not Sig
		SD	3.04	2.89	2.96	2.83	2.77	2.86	2.79	2.71	2.71	2.76	
	MSG	Mean	25.60	25.70	25.57	25.71	26.01	25.85	25.93	26.28	25.88	25.67	
		SD	2.72	2.55	2.60	2.71	2.90	2.83	2.80	3.02	2.49	2.52	
CIR 9	CON	Mean	26.00	26.27	26.19	26.73	26.86	26.82	27.10	27.17	26.56	26.38	Not Sig
		SD	2.87	2.93	2.95	2.95	3.05	2.93	2.90	2.55	2.60	2.68	
	MSG	Mean	26.21	26.30	26.19	26.45	26.56	26.58	26.80	26.76	26.46	26.26	
		SD	2.77	2.64	2.87	2.77	3.02	3.09	3.12	2.92	2.63	2.64	
CIR 11	CON	Mean	26.65	26.91	26.98	27.49	27.35	27.47	27.61	27.64	27.23	27.06	Not Sig
		SD	2.99	2.93	3.16	3.11	2.97	3.07	2.88	2.66	2.71	2.78	
	MSG	Mean	26.53	26.78	26.87	27.05	27.10	27.10	27.16	27.21	26.92	26.87	
		SD	2.66	2.61	2.85	2.75	3.08	3.16	2.99	2.88	2.67	2.70	

			PRE			D1	D2	D3	D4	D7	D10	D14	Anova
TEN UPP	CON	Mean	257.5			116.6	105.8	111.1	110.6	207.7	228.3	256.5	Not Sig
		SD	50.62			80.20	86.32	75.35	84.90	94.06	92.22	52.18	
	MSG	Mean	278.5			156.1	115.9	133.2	153.2	234.2	252.5	278.5	
		SD	38.16			91.24	90.63	72.83	77.45	63.01	65.54	36.52	
TEND FORE	CON	Mean	91.00			54.30	43.10	46.90	45.20	77.20	79.00	91.00	Not Sig
		SD	13.08			38.96	35.72	36.42	33.41	32.41	29.23	13.70	
	MSG	Mean	95.50			61.80	54.20	59.70	73.80	83.00	85.00	95.50	
		SD	10.12			36.73	39.97	38.13	33.78	23.12	20.14	10.12	

			PRE			D1	D2	D3	D4	D7	D10	D14	Anova
SOR UPP	CON	Mean	0.00			96.70	95.90	105.8	83.00	45.70	20.40	4.70	Not Sig
		SD	0.00			65.70	63.13	58.33	48.77	55.02	51.77	9.93	
	MSG	Mean	0.00			68.00	65.10	69.00	79.00	25.70	10.20	0.00	
		SD	0.00			36.14	33.33	47.48	55.44	33.20	22.31	0.00	
SOR FORE	CON	Mean	0.00			36.20	23.90	32.30	24.40	9.90	1.80	0.00	0.01
		SD	0.00			26.90	13.46	25.79	16.66	22.28	3.36	0.00	
	MSG	Mean	0.00			20.60	20.30	18.50	17.50	7.00	2.90	0.00	
		SD	0.00			17.58	22.48	16.55	20.47	18.12	9.17	0.00	
SOR EXT	CON	Mean	0.00			38.00	37.10	38.80	46.90	6.60	1.40	0.00	0.02
		SD	0.00			22.93	22.94	21.45	26.90	12.69	4.43	0.00	
	MSG	Mean	0.00			20.40	27.80	19.30	21.70	0.80	0.00	0.00	
		SD	0.00			17.07	22.82	24.28	24.23	2.53	0.00	0.00	
SOR FLEX	CON	Mean	0.00			30.20	35.60	28.60	28.60	3.50	0.00	0.00	Not Sig
		SD	0.00			21.85	22.06	24.83	25.79	8.21	0.00	0.00	
	MSG	Mean	0.00			16.70	18.00	16.00	12.20	0.00	0.00	0.00	
		SD	0.00			13.73	16.70	24.70	21.25	0.00	0.00	0.00	

# **APPENDIX E3 -RAW DATA FOR LIGHT CONCENTRIC EXERCISE**

test			pre	post	30min	D1	D2	D3	D4	D7	Anova
ISO 90	Control	Mean	46.90	27.16	26.60	25.64	25.40	30.60	29.87	36.26	Not Sig
		SD	21.70	13.51	14.61	11.13	13.11	15.39	17.97	19.94	
	LCE	Mean	49.42	24.98	24.18	25.12	26.89	26.88	29.31	36.80	
		SD	21.71	12.69	11.08	13.32	15.52	12.81	16.83	18.54	
ISO 150	Control	Mean	31.48	21.13	22.23	18.57	17.61	20.97	19.49	25.92	Not Sig
		SD	15.00	12.68	12.57	9.29	8.77	14.20	11.74	12.53	
	LCE	Mean	31.28	18.59	18.58	18.66	18.43	17.90	20.08	26.49	
		SD	12.60	8.59	8.68	8.93	10.08	8.75	11.09	14.22	
FV30	Control	Mean	31.39	19.84	20.40	18.60	19.71	22.07	21.43	24.24	Not Sig
		SD	13.52	9.32	9.09	7.52	8.84	12.49	13.26	13.83	
	LCE	Mean	30.96	18.37	17.93	18.37	18.85	20.48	20.06	24.17	
		SD	12.81	8.58	7.94	8.61	7.42	8.75	10.20	11.47	
FV90	Control	Mean	30.38	18.85	21.07	17.45	17.85	20.18	19.75	22.35	Not Sig
		SD	13.77	10.71	11.01	9.06	8.99	10.83	13.31	12.20	
	LCE	Mean	28.29	17.61	16.65	17.54	17.42	18.26	17.06	24.02	
		SD	12.37	7.25	8.54	9.68	7.49	9.05	9.35	11.34	
FV150	Control	Mean	27.71	17.21	20.13	16.29	17.20	19.10	18.71	20.62	Not Sig
		SD	13.18	9.33	10.21	8.88	8.93	10.83	11.85	12.46	
	LCE	Mean	25.59	16.18	16.51	15.45	17.27	17.86	17.53	22.77	
		SD	11.12	7.83	8.32	7.65	7.46	8.74	9.73	10.43	
FV210	Control	Mean	25.00	16.76	18.27	15.64	16.99	15.91	17.67	19.06	Not Sig
		SD	13.65	9.95	10.09	8.50	9.32	9.43	12.58	13.10	
	LCE	Mean	23.57	14.07	15.27	12.85	15.33	16.16	15.12	19.37	
		SD	10.89	6.68	7.77	6.38	6.94	8.11	6.40	10.73	
FV300	Control	Mean	20.74	14.35	16.73	14.72	15.02	13.88	21.68	16.28	Not Sig
		SD	12.70	11.04	10.25	7.71	10.08	9.59	29.22	10.25	
	LCE	Mean	21.00	13.63	13.37	11.93	14.99	14.61	13.28	17.30	
		SD	11.48	6.62	8.11	5.88	7.84	6.70	8.99	9.32	

test			pre	post	30min	D1	D2	D3	D4	D7	Anova
RANG	Control	Mean	152.14	148.43	148.00	143.64	143.57	143.86	142.79	146.50	Not Sig
		SD	7.15	7.64	8.28	10.80	9.62	9.20	5.75	8.17	
	LCE	Mean	148.14	143.50	143.14	141.43	140.86	139.00	138.64	143.50	
		SD	4.93	6.35	6.31	9.48	6.32	6.73	8.93	6.69	
SANG	Control	Mean	167.64	164.07	163.57	159.57	160.43	156.36	156.86	161.43	Not Sig
		SD	7.93	7.65	8.03	7.02	7.32	7.08	7.74	9.59	
	LCE	Mean	164.64	159.14	159.64	156.29	156.29	153.93	153.43	160.79	
		SD	7.38	7.41	7.34	9.52	8.76	8.41	10.42	8.72	
FANG	Control	Mean	34.36	47.71	46.21	45.21	46.57	47.29	43.50	38.79	Not Sig
		SD	5.31	8.11	7.63	6.12	9.38	10.02	5.50	4.87	
	LCE	Mean	30.79	51.14	51.57	46.86	47.29	45.71	45.43	33.64	
		SD	4.66	12.43	13.97	11.48	13.36	11.30	13.31	4.86	
ROM	Control	Mean	133.29	116.36	117.36	114.36	113.86	109.07	113.36	122.64	Not Sig
		SD	8.22	9.66	9.92	9.25	11.90	10.91	9.13	11.14	
	LCE	Mean	133.86	108.00	108.07	109.43	109.00	108.21	108.00	127.14	
		SD	9.96	17.94	18.46	17.18	19.93	15.93	18.61	11.12	

test			pre	post	30min	D1	D2	D3	D4	D7	Anova
CIR 3	Control	Mean	24.03	24.09	24.11	24.34	24.57	24.64	24.79	24.72	Not Sig
		SD	2.50	2.44	2.43	2.45	2.38	2.46	2.41	2.55	
	LCE	Mean	24.49	24.99	24.93	24.99	25.31	25.48	25.66	25.54	
		SD	2.73	2.74	2.71	2.68	2.68	2.79	2.72	2.85	
CIR 5	Control	Mean	25.14	25.18	25.11	25.47	25.53	25.68	25.85	25.77	Not Sig
		SD	2.88	2.97	2.85	2.81	2.74	2.72	2.65	2.60	
	LCE	Mean	25.92	26.31	26.22	26.36	26.76	26.97	27.21	27.01	
		SD	2.77	2.68	2.68	3.00	2.92	2.99	2.98	2.90	
CIR 7	Control	Mean	26.11	26.20	26.19	26.50	26.54	26.67	26.93	26.82	Not Sig
		SD	3.34	3.20	3.23	3.16	3.08	3.17	3.05	3.08	
	LCE	Mean	26.56	27.23	27.09	27.46	27.67	27.88	27.96	27.64	
		SD	2.96	3.00	2.94	2.98	3.12	3.12	3.07	2.94	
CIR 9	Control	Mean	26.74	26.69	26.91	27.30	27.39	27.38	27.52	27.53	Not Sig
		SD	3.32	3.36	3.34	3.30	3.19	3.26	3.05	3.04	
	LCE	Mean	27.42	28.00	28.00	28.09	28.35	28.49	28.94	28.33	
		SD	2.99	3.00	2.98	3.09	3.15	3.37	3.17	3.38	
CIR 11	Control	Mean	27.34	27.62	27.66	28.09	27.97	28.04	28.09	28.10	Not Sig
		SD	3.28	3.50	3.55	3.53	3.32	3.39	3.05	3.06	
	LCE	Mean	28.21	28.68	28.63	28.83	28.98	29.10	29.23	29.02	
		SD	3.11	3.18	3.13	3.26	3.24	3.31	3.45	3.42	

			pre	d1	d2	d3	d4	d7	Anova
TEN 3-5	Control	Mean	98.57	61.43	38.86	39.50	45.71	95.00	Not Sig
		SD	5.35	27.63	29.00	26.91	21.47	8.55	
	LCE	Mean	98.57	53.57	45.36	40.36	51.43	93.57	
		SD	5.35	24.29	19.66	19.66	20.70	9.29	
TEND 9-11	Control	Mean	100.00	47.14	39.00	30.00	37.86	87.86	Not Sig
		SD	0.00	30.99	30.99	24.18	24.00	20.45	
	LCE	Mean	100.00	45.36	39.29	28.21	43.21	86.43	
		SD	0.00	30.79	26.81	20.06	24.78	20.23	
TEN BRAC	Control	Mean	100.00	57.50	55.00	40.36	40.00	99.29	Not Sig
		SD	0.00	29.27	25.34	21.16	27.10	2.67	
	LCE	Mean	100.00	59.64	56.43	44.29	51.07	99.29	
		SD	0.00	27.07	24.99	17.85	26.32	2.67	
TEND FORE	Control	Mean	100.00	55.21	38.43	32.57	39.43	77.29	Not Sig
		SD	0.00	35.90	33.00	31.89	31.27	34.56	
	LCE	Mean	100.00	68.57	58.57	44.07	60.36	91.43	
		SD	0.00	30.09	32.31	32.82	34.33	22.14	
SOR 3-5	Control	Mean	0.00	24.14	26.00	34.36	22.57	4.64	Not Sig
		SD	0.00	15.19	10.44	13.36	17.12	9.32	
	LCE	Mean	0.00	28.21	36.29	37.00	30.64	1.21	
		SD	0.00	12.93	20.89	11.49	19.14	2.89	
SOR 9-11	Control	Mean	0.00	33.64	37.43	41.93	28.07	3.29	Not Sig
		SD	0.00	16.87	19.98	18.65	17.83	8.15	
	LCE	Mean	0.00	36.86	46.50	41.43	38.57	2.36	
		SD	0.00	17.60	20.07	20.09	19.20	5.44	
SOR BRA	Control	Mean	0.00	22.36	29.29	31.71	21.93	3.29	Not Sig
		SD	0.00	15.98	18.68	18.04	20.24	7.35	
	LCE	Mean	0.00	20.43	33.00	29.79	30.00	0.36	
		SD	0.00	14.15	18.40	19.51	17.08	0.93	
SOR BRACH I	Control	Mean	0.00	30.71	27.93	28.86	20.29	5.21	Not Sig
		SD	0.00	21.80	15.00	15.52	18.42	11.18	
	LCE	Mean	0.00	17.86	31.57	27.14	20.29	5.50	
		SD	0.00	21.08	15.38	23.19	17.51	12.09	
SOR EXT	Control	Mean	0.00	36.64	40.36	40.57	49.43	7.29	Not Sig
		SD	0.00	20.95	19.58	17.19	25.39	10.65	
	LCE	Mean	0.00	46.36	51.57	45.57	44.86	3.00	
		SD	0.00	30.60	20.06	26.69	24.03	4.10	
SOR FLEX	Control	Mean	0.00	34.86	40.50	38.64	37.21	4.57	Not Sig
		SD	0.00	20.79	17.64	21.15	25.13	7.18	
	LCE	Mean	0.00	38.36	42.57	33.50	27.21	1.14	
		SD	0.00	20.62	19.62	20.63	13.96	1.75	
CK	Control	Mean	108.07	285.22	539.79	1792.79	3258.43	1772.86	Not Sig
		SD	41.92	162.72	430.61	2190.06	4564.27	2861.87	
	LCE	Mean	120.91	350.30	757.98	1273.46	1851.78	1365.84	
		SD	63.11	355.75	939.39	1185.14	1574.86	1388.73	



# APPENDIX E4 - RAW DATA FOR ECCENTRIC EXERCISE

## RAW DATA FOR EXERCISE

### IMMOBILIZATION

	Torque
sum	1739.49
Average	30.97895
SD	6.319517
SEM	1.998407

### CONTROL

	Torque
sum	1571.88
Average	26.24883
SD	4.901921
SEM	1.550124

### MASSAGE

	Torque
sum	1801.249
MEAN	30.02082
SD	6.3543
SEM	1.8365

### CONTROL

	Torque
sum	1748.795
MEAN	29.19741
SD	5.25461
SEM	1.7452

### LIGHT EXERCISE

	Torque
SUM	1817.975
mean	30.29958
SD	4.016242
SEM	1.073386

### CONTROL

	Torque
SUM	1771.046
mean	29.51744
SD	3.607427
SEM	0.964125