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Proprioceptive and Muscle Activation Changes in Triceps Surae Associated with Exercise Induced Muscle Damage

By

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USE OF THESIS

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ABSTRACT

The aim of this thesis was to examine proprioceptive and activation changes that occur in triceps surae in response to exercise induced muscle damage (EIMD). While proprioceptive changes have previously been demonstrated in association with EIMD, the present investigation examined the role of a number of potential contributing factors to these changes, following both single and repeated bouts of eccentric exercise.

As the volume of literature relating to EIMD in triceps surae was found to be limited when compared with that in the biceps and quadriceps, the need to validate a model for EIMD in triceps surae was highlighted. Since downhill backward walking had been used in the past as a method for eccentric contractions of triceps surae, this exercise was examined for its effectiveness to produce the common indicators of EIMD of soreness, tenderness, plasma creatine kinase and voluntary strength declines following both a single and a repeated exercise bout. Twenty subjects (28.0 ± 1.6 years) completed a single exercise protocol and 17 subjects (26.6 ± 1.6 years) completed two exercise protocols (separated by 14 days) consisting of 60-minutes of downhill backward walking (-15%) at a stepping rate of 30 - 35 strides per minute. The step together action of the exercise model allowing for one limb to be eccentrically exercised (exercised limb) and the other to act as a control (unexercised limb). Significant increases in soreness, tenderness and plasma creatine kinase (24 – 96 hours), and significant decreases in strength (0.5 – 96 hours) were recorded in both groups following a single exercise protocol. The group completing the repeated exercise protocol demonstrated significant increases in soreness and tenderness (24 – 96 hours), and significant decreases in strength (0.5 – 24 hours) post-walk, with significant differences between the two bouts recorded for soreness (24 – 96 hours) and plasma creatine kinase (24 hours) post-walk. These changes were shown to being consistent with previous investigation for EIMD and it was therefore concluded that downhill backward walking was an effective model for EIMD in triceps surae The attenuated responses for soreness, plasma creatine kinase and strength with the repeated exercise bout was consistent with the repeated bout effect demonstrated in other muscle groups, further validating downhill backward walking as an effective model for EIMD in triceps surae.

Subsequent to validation of downhill backward walking as a model for EIMD in triceps surae, measurements of maximal voluntary strength (torque and avEMG), Hoffmann and Achilles tendon reflex responses, muscle compound action potentials, contractile properties, vertical jump, joint position perception, torque perception, ankle range of movement, and relaxed angle were performed pre-walk, and 0.5, 24, 48, 72 and 96 hours post-walk in 12 subjects following a single bout of downhill backward walking. The non-exercised limb showed no significant changes from baseline except when acting as the matching limb during the torque and position perception tasks. For the exercised limb, significant differences from baseline ($p<0.05$) were recorded at one or more time interval post-walk for the variables of maximal voluntary strength (torque: 0.5 – 96 hours, and avEMG: 0.5 & 48 hours), contractile properties (0.5 hours), vertical jump (0.5 – 72 hours), and joint position (24 & 48 hours) and torque perception (0.5 – 24 hours). While no significant differences from baseline were recorded for the remaining variables, significant correlations ($p<0.05$) were recorded between the Hoffmann reflex and matching angle error ($r = -.355$ matching to non-exercised), and voluntary avEMG and matching torque error ($r = -.420$ for soleus; $r = -.458$ for gastrocnemius). The results showed that errors produced during performance of proprioceptive tasks following a downhill backward walking were more likely the result of changes in muscle activation and control rather than disruption to muscle spindle or Golgi tendon organ function.

In order to examine the influence of the repeated bout effect on proprioception 12 subjects (27.5 ± 2.1 years) completed two exercise bouts of downhill backward walking (separated by two weeks). The same variables were recorded as in the previous study with results focusing on those changes in the exercised limb only, except where the non-exercised limb had been shown to be influenced previously using this model. Significant differences from baseline were recorded for the exercised limb following both exercise bouts for maximal voluntary strength (torque only: 0.5 – 96 hours), vertical jump (0.5 hours), and torque perception (0.5 hours). A protective effect between bouts ($p<0.05$) occurred for avEMG during the maximal voluntary strength protocol only (48 – 96 hours). Again, significant correlations ($p<0.05$) were recorded between the Hoffmann reflex and matching error during the angle perception task ($r=-.463$), and avEMG and matching error measured

during performance of the torque perception task. The results suggested that the repeated bout effect, which has been described for parameters of strength, soreness, and plasma creatine kinase levels following eccentric exercise, might not extend to proprioceptive function. Or, that conferral of protection may occur only after exposure to multiple bouts of eccentric exercise rather than just a repeated bout.

The findings indicate that the response of skeletal muscle to eccentric exercise may be dependant on the intensity at which the exercise is performed, and that not all responses of exercise induced muscle damage are protected in a repeated exercise bout following an initial bout of 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

(iii) contain any defamatory material.

Signed: .....

Date: 05-08-2004.....

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DEFINITION OF TERMS

Antidromic	Movement in the opposite direction to normal (Dirckx, 1997, p. 55)
bpm	Beats per minute
CNS	Central nervous system
Concentric	Contraction during which the muscle shortens
DOMS	Delayed onset muscle soreness
Dorsi	Refers to the back, upper or posterior of any part of the body (Dirckx, 1997, p. 255)
Dorsiflexion	The action of flexion of the ankle resulting in elevation of the sole of the foot (Martini, 1998, p. 261)
Dynamometer	Instrument for measuring the degree of muscular power (Dirckx, 1997, p. 261)
Eccentric	Contraction during which the muscle lengthens
EIMD	Exercise induced muscle damage
EMG	Electromyogram
Extrafusal	Contractile muscle fibres (Martini, 1998,p. G-12)
Force sensation	Perception of the effort required to produce a given tension in a muscle (McCloskey, Gandevia, Potter & Colebatch, 1983)
Fusiform	Tapering at both ends (Dirckx, 1997, p. 349)
Intrafusal	Muscle spindle fibres (Martini, 1998, p. G-17)
MVC	Maximal voluntary contraction
Myotatic	Meaning muscle stretch (Cohen & Sherman, 1988, p. 206)
Plantar	Referring to the sole of the foot (Dirckx, 1997, p. 686)
Plantarflexion	Action that extends the ankle and elevates the heel (Martini, 1998, p. 261)
Proprioception	Subconscious level sense of perception of the position or movement of the body, independent of vision (Dirckx, 1997,

	p. 719)
Proprioceptive	Capable of receiving stimulus originating in muscles, tendons and internal tissues (Dirckx, 1997, p. 719)
Proprioceptive reflex	Any reflex response to stimulation of proprioceptors (Dirckx, 1997, p. 719)
Reflex	Involuntary reaction in response to a periphery stimulus; contraction of a muscle resulting from a stimulus applied to its proprioceptors (Dirckx, 1997, p. 751)
SD	Standard deviation
SEM	Standard error of the mean
Triceps surae	The soleus, and the medial and lateral bellies of the gastrocnemius considered as one muscle (Dirckx, 1997, p, 568)

CHAPTER I INTRODUCTION

1.1 Background to Study

Most individuals have sustained exercise induced muscle damage (EIMD), and experienced its consequences following either sport or strenuous physical work. EIMD is most common following activity of an unaccustomed nature (Armstrong, 1990), or which involve repeated eccentric contractions (Newham, McPhail, Mills & Edwards, 1983). Indicators of EIMD include myofibril damage, muscle weakness, stiffness, soreness, and tenderness (Howell, Chleboun & Conaster, 1993; Smith, Keating et al., 1994). Functionally, the consequences of EIMD include reduced strength and power (Sargeant & Dolan, 1987), impaired performance of skilled tasks (Pearce, Sacco, Byrnes, Thickbroom & Mastaglia, 1998), and altered joint proprioception (Saxton et al., 1995).

The exact mechanism(s) responsible for the ultrastuctural disruption in EIMD remains unclear. However, a high level of mechanical stress on the exercised muscle is the most frequently cited explanation according to reviewing authors (Armstrong, Warren & Warren, 1991; Ebbeling & Clarkson, 1989). While it was first suggested that the performance declines documented with EIMD result from decreased motivation associated with stiffness and soreness (Hough, 1902), subsequent investigators have surmised otherwise. For example, strength declines have been shown to occur in association with EIMD in the absence of pain (Byrnes et al., 1985; McGlynn, Laughlin & Rowe, 1979). Studies investigating proprioceptive and motor performance variables have concluded that the effects were a result of disruption to nervous system feedback from muscle spindles and cutaneous afferents, and / or from a decline in the force generating capacity of the damaged muscle (Brockett, Warren, Gregory, Morgan & Proske, 1997; Saxton et al., 1995).

An additional characteristic of EIMD is the observation of a reduction in the extent of muscle injury when exercise of a similar nature is replicated. Termed the 'repeated bout effect', investigators have shown that the common indicators of damage including increased levels of plasma creatine kinase (CK), delayed onset muscle soreness (DOMS) and tenderness are reduced or absent after exercise is repeated within 4 days to

6 weeks of the initial exercise (Clarkson & Tremblay, 1988; Mair et al., 1995). The time course of strength recovery observed with EIMD is also affected with a repeat bout of exercise (Newham, Jones & Clarkson, 1987). The adaptations responsible for conferral of this protective effect are not known. Furthermore, little information exists regarding the nature of the repeated bout effect as it pertains to functional variables of force perception and limb position awareness.

1.2 Significance of Study

Proprioception plays an essential role in everyday movements and activities (Guyton & Hall, 1996, p. 319 – 321; Jones, 1994). Altered proprioception can place individuals at an increased risk of injury, particularly from falls (Maeda, Nakamura, Otomo, Higuchi & Motohashi, 1998). Brockett et al. (1997), Howell et al. (1993), and Saxton et al. (1995) all noted that a disruption in joint proprioception following eccentric exercise was accompanied not only by an increase in stiffness and swelling, but also by changes in resting joint angle. Primarily controlled via muscle tone, resting joint angle is dependent both on the sensitivity of the muscle spindles and the functioning of the alpha motor neurons involved in the stretch reflex (Marieb, 1998, p. 486). Alterations in muscle tone may result in a change in resting joint angle and altered proprioceptive feedback, leading to inappropriate action(s) and movements and increased risk of injury. While the relationship between muscle swelling and stiffness have been examined following eccentric exercise (Chleboun, Howell, Conaster & Giesey, 1998), to date, the relationships between changes in relaxed joint angle, muscle swelling and altered joint proprioception in association with EIMD are not known.

Much of the work relating to EIMD has focused on changes within the muscle-tendon complex as the mechanism for strength loss and performance declines during skilled tasks, rather than the role of the central nervous system (CNS). Furthermore, possible CNS adaptations involved in the conferral of protection with a repeated bout of exercise have not been systematically investigated. Golden and Dudley (1992) found that following a second bout of damaging exercise there was a decrease in muscle activation during a muscular contraction without a decrease in the exercise intensity or the resultant torque output. The authors proposed that this resulted from an increase in the efficiency of muscle activation thus reducing mechanical stress and damage during the subsequent exercise bouts. To what extent these changes reflect structural adaptations within the muscle-tendon unit as opposed to alterations in the pattern of activation is not

known. A further point of issue is that, although it is known that proprioception is affected by eccentric exercise, whether this parameter exhibits the repeated bout effect has not been investigated.

1.3 Purpose of Study

The main objective of this thesis was to examine the functional changes that occur in triceps surae in association with EIMD; in particular alterations in proprioception and activation.

The research examined three main questions relating to triceps surae and EIMD.

(1) Do proprioceptive and activation changes occur in tricpes surae in response to a bout of downhill backward walking?

(2) Do relationships exist between proprioceptive and activation changes, and alterations in strength and contractile properties of triceps surae following a bout of downhill backward walking?

(3) Do any changes in proprioception and activation of triceps surae exhibit the repeated bout effect following a second bout of downhill backward walking?

CHAPTER 2 LITERATURE REVIEW

2.1 Introduction

This chapter aims to provide the relevant background information necessary to understand the outlined research questions. The literature reviewed covers the areas of proprioception and reflex functioning, electromyography, muscle activation and contraction, and EIMD.

2.2 Proprioception

Proprioception is the term used to describe sensations of static position, velocity and direction of movement, and force or heaviness (McCloskey, 1978). Proprioception involves afferent feedback signalled from peripheral receptors in the muscle, skin, and joints to the CNS with a combination of these receptors providing information relating to movement and body position (Table 1).

Table 1
Neural Receptors Involved in the Proprioceptive Variables of Movement, Position and Force

Proprioception variable	Proposed neural receptors
Limb movement	Muscle spindle receptors
	Cutaneous mechanoreceptors
	Joint receptors
Limb position	Muscle spindle receptors
	Cutaneous mechanoreceptors
Force or heaviness	Golgi tendon organs

From: Jones, L. L. (1994). Peripheral mechanisms of touch and proprioception. *Canadian Journal of Physiology and Pharmacology, 72*, 484 – 487.

2.2.1 *Muscle Spindle Anatomy and Function*

The muscle spindle is a fusiform neuromuscular receptor located in skeletal muscle. Linked to the CNS by both afferent and efferent nervous fibres, muscle spindles are responsible for providing information relating to muscle stretch (Marieb, 1998, p. 485-486). A modified muscle fibre, the muscle spindle differs from extrafusal fibres in that it has a non-striated region near its centre, with contractile tissue at the ends of the fibre (Figure 1). Each muscle spindle consists of up to ten intrafusal fibres enclosed in a

connective sheath lying parallel to the surrounding extrafusal fibres. Each intrafusal fibre is innervated by both sensory and motor neurons with the end of each capsule of fibres attaching either to the tendon of the muscle or the sides of the surrounding extrafusal fibres (Ganong, 1993, p. 112; Shier, Bulter & Lewis, 1996, p. 447).

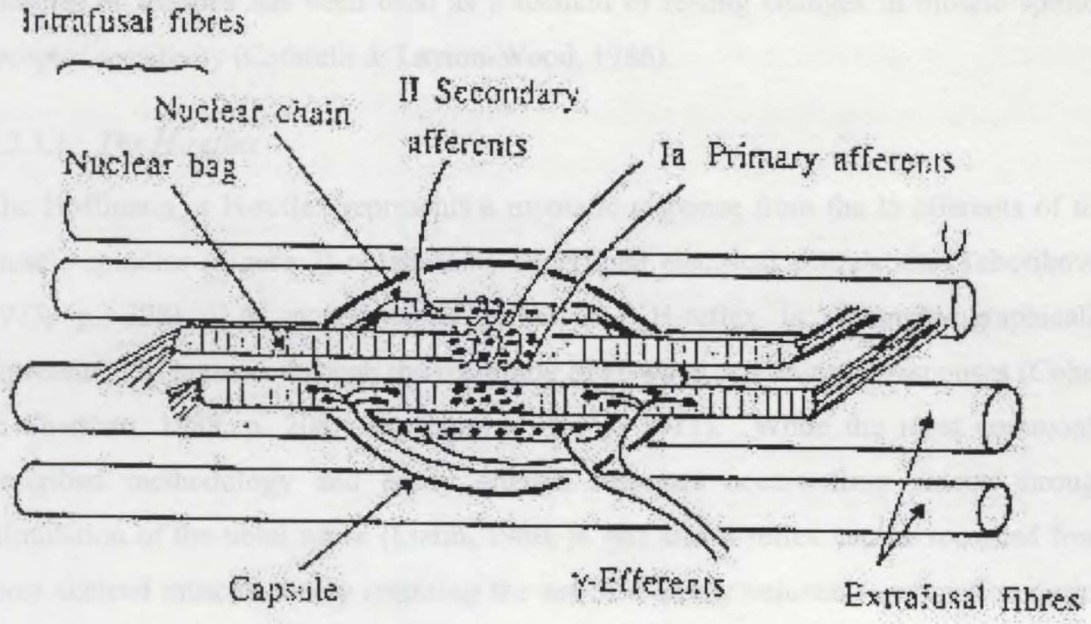


Figure 1. The main features of the muscle spindle including the afferent and efferent fibres.

From: Jones, D. A., & Round, J. M. (1990). *Skeletal muscle in health and disease.* Great Britain : Manchester University Press.

The dilated central area of the intrafusal fibres, referred to as the nuclear bag, acts as the sensory region of the spindle innervated by both type Ia afferent (responsive to both rate and degree of stretch) and type II afferent fibres (responsive only to the degree of stretch). The contractile ends of the spindles are innervated by both gamma efferent fibres, innervating only the intrafusal fibres, and alpha efferent fibres, innervating both the intra and extrafusal fibres (Marieb, 1998, 485 – 486; Martini, 1998, p. 434).

When a muscle is stretched, the nuclear bag fibres of the spindle are stretched, increasing the action potential frequency from the sensory endings in proportion to the rate and degree of stretch. The increase in firing rate from the spindle initiates a reflex contraction in the extrafusal fibres, thus increasing muscle tone. When the nuclear bag is compressed, the decreased stimulus from the intrafusal fibres results in a decrease in

muscle tone (Martini, 1998, p. 434; Shier et al., 1996, p. 447). While no direct test exists for quantifying muscle spindle function in humans (van Deursen, Sanchez, Ulbrecht & Cavanagh, 1998), muscle spindle sensitivity and resting spindle discharge can be evaluated using the Hoffman or H-reflex, and the tendon or jerk reflex (Enoka, Hutton & Eldred, 1980). Additionally, the application of vibration to surrounding muscles or tendons has been used as a method of testing changes in muscle spindle receptor sensitivity (Cafarelli & Layton-Wood, 1986).

2.2.1.1 *The H-reflex*

The Hoffmann or H-reflex represents a myotatic response from the Ia afferents of the muscle spindles (Figure 2) obtained by superficial electrical stimulation (Taborikova, 1973, p. 328). A monosynaptic reflex, the H-reflex is electromyographically represented in humans through the recording of H-wave and M-wave responses (Cohen & Sherman, 1988, p. 208; Windhorst, 1996, p. 1011). While the most commonly described methodology and easily elicited response occurs from soleus through stimulation of the tibial nerve (Ludin, 1980, p. 46), the H-reflex can be recorded from most skeletal muscles, many requiring the use of a steady voluntary contraction during stimulation (Aminoff, 1998, p. 178).

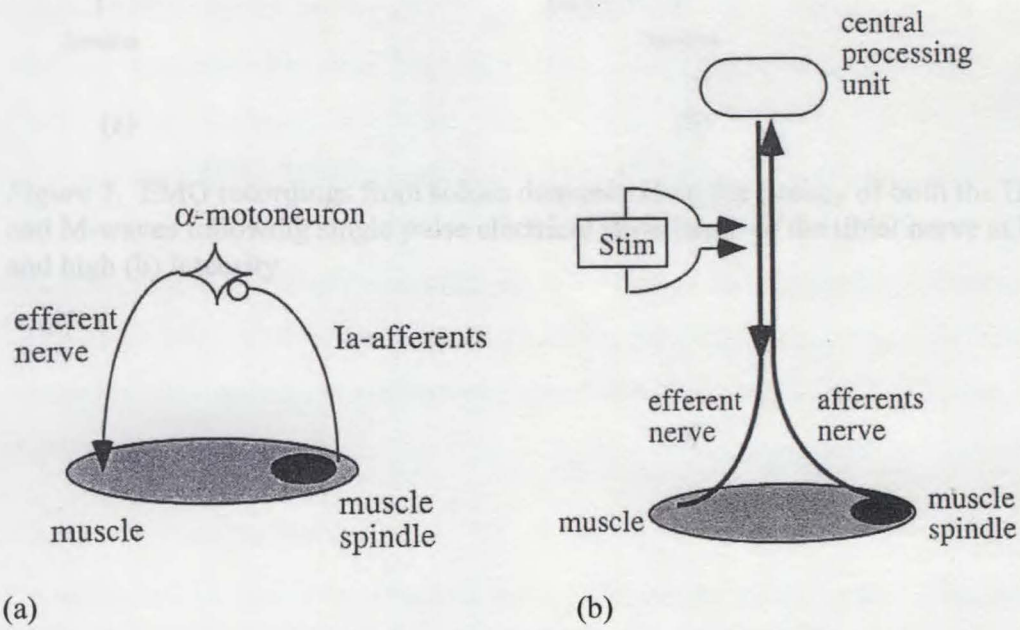


Figure 2. Representation of the pathway of a monosynaptic reflex (a) and the monosynaptic Hoffman reflex (b).

From: Latash, M. (1998). *Neurophysiological basis of movement.* Champaign, IL : Human Kinetics.

Low intensity stimulation of the tibial nerve produces an EMG response referred to as an H-wave in triceps surae with a latency of approximately 30 – 35 ms while increasing the intensity of the stimulus produces both the H-wave and a second EMG response with a 5 – 10 ms latency referred to as an M-wave (Figure 3a). As stimulus intensity, and the amplitude of the M-wave is increased, the H-reflex is suppressed due to direct stimulation of the α-motoneurons producing both orthodromic and antidromic action potentials (Figure 4). At high stimulation intensities all efferent axons are directly stimulated which causes a collision effect from the antidromic action potentials and results in the complete disappearance of the H-wave, while the M-wave (Figure 3b) reaches a peak (Latash, 1998, pp. 66 – 68; Schmidt, 1983, pp. 77 – 78).

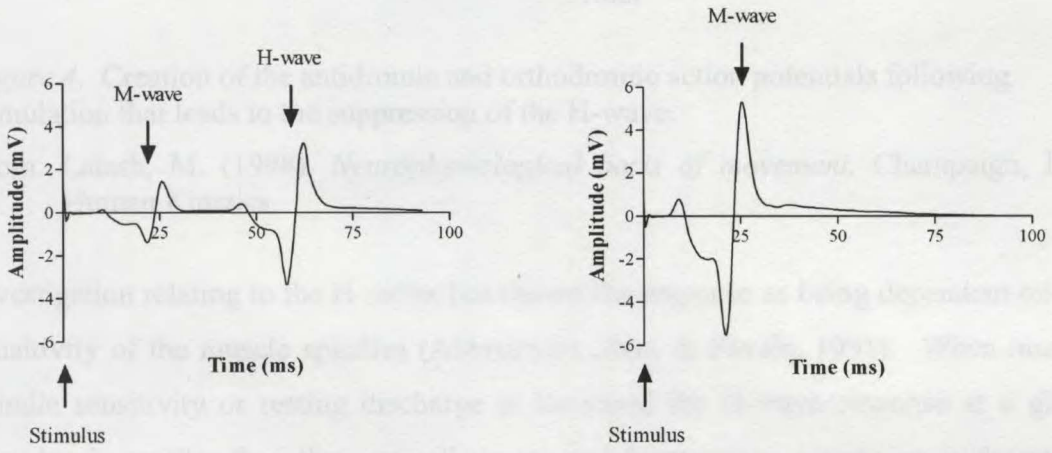


Figure 3. EMG recordings from solues demonstrating the latency of both the H-wave and M-waves following single pulse electrical stimulation of the tibial nerve at low (a) and high (b) intensity.

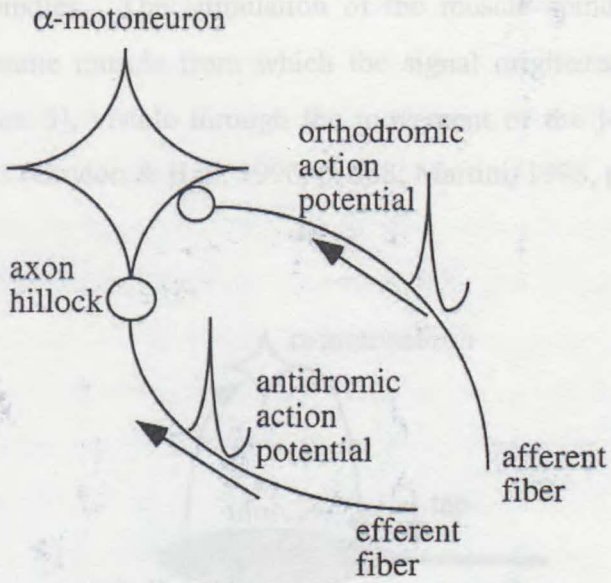


Figure 4. Creation of the antidromic and orthodromic action potentials following stimulation that leads to the suppression of the H-wave.

From: Latash, M. (1998). *Neurophysiological basis of movement.* Champaign, IL : Human Kinetics.

Investigation relating to the H-reflex has shown the response as being dependent on the sensitivity of the muscle spindles (Abbruzzese, Reni & Favale, 1991). When muscle spindle sensitivity or resting discharge is increased the H-wave response at a given stimulus is smaller than that normally seen, and larger when sensitivity is decreased (Polus, Patak, Gregory & Proske, 1991; Woods, Gregory & Proske, 1996). The recovery curve of the H-reflex, and a comparison of the maximum H-wave and M-wave response (H/M ratio) has been shown to produce characteristic findings in subjects with spasticity where the phasic and tonic stretch reflexes are exaggerated (Ludin, 1980, p. 122). There is a lack of information however, relating to the use of the H-reflex for investigation of spindle sensitivity changes associated with EIMD and other forms of muscle injury.

2.2.1.2 *The Stretch Reflex*

Considered to be one of the simplest spinal reflexes, the stretch reflex initiates a muscle contraction in the stretched muscle to prevent the intrinsic fibres from being damaged (Pritchard & Alloway, 1999, p. 108). Clinically examined by a tendon tap response, the stretch reflex has most commonly been described in the elbow through a triceps or biceps tendon tap, the knee through a patella tendon tap, and the ankle via an Achilles tendon tap (Martini & Welch, 1998, p. 78). Tapping on the tendon results in a slight stretching of the attached muscle and thus excitation of the Ia afferent fibres of the

enclosed muscle spindles. The stimulation of the muscle spindles causes a reflex contraction of the same muscle from which the signal originated via the alpha (α) motoneurones (Figure 5), visible through the movement of the joint over which the stretched muscle acts (Guyton & Hall, 1996, p. 688; Martini, 1998, p. 435).

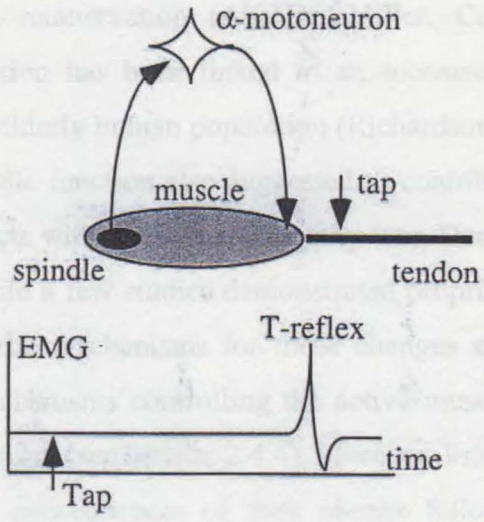


Figure 5. Reflex pathway of the tendon tap stretch reflex.

From: Latash, M. (1998). *Neurophysiological basis of movement.* Champaign, IL : Human Kinetics.

While both the stretch and the H-reflex are mediated over the Ia afferent pathways, electrical stimulation of the H-reflex bypasses the muscle spindle, allowing changes in spindle sensitivity to be indirectly assessed by comparing the responses of the two reflex methods together (Aminoff, 1998, p. 179).

2.2.2 *Mechanisms and Consequences of Proprioceptive Alterations*

Alterations in proprioception have been demonstrated to occur in association with an array of conditions and pathologies, with significant impact on functional tasks such as walking. Altered position sense of the knee has been demonstrated following anterior cruciate ligament (ACL) rupture and repair (Birmingham et al., 2001), with increased reflex latencies of the hamstring in ACL deficient individuals linked to knee instability and 'giving way' (Beard, Kyberd, Fergusson & Dodd, 1993). Similarly, functional ankle instability and postural sway patterns have been established to occur in individuals with a history of recurrent ankle strains as a consequence of declines in joint proprioception (Baier & Hopf, 1998).

Postural sway increases, changes in position and force sensations, and alterations in reflex latencies have been verified in relation to local muscle fatigue (Asmussen & Mazin, 1978; Duchateau & Hainaut, 1993; Jones & Hunter, 1982; Lundin, Feuerbach & Grabiner, 1993). The disruption of interjoint coordination and locomotion also shown to occur as a result of local loss in proprioception and spindle function in cats undergoing experimental reinnervation (Abelew, Miller, Cope & Nichols, 2000). Peripheral nerve dysfunction has been linked to an increased instance of falls and postural instability in the elderly human population (Richardson & Hurvitz, 1995), with disruption of muscle spindle function also suggested to contribute to impaired balance and unsteadiness in subjects with diabetic neuropathy (van Deursen, Sanchez, Ulbrecht & Cavanagh, 1998). While a few studies demonstrated proprioceptive disturbances in association with EIMD, the mechanisms for these changes are not well understood. Damage to the reflex mechanisms controlling the active muscle have been cited as a possible source of these errors (see section 2.4.4), however, limited literature relating to reflex responses and the consequences of their change following eccentric exercise exists.

2.3 Electromyography

The electromyogram (EMG) is defined as being "a graphic representation of the electric currents associated with muscular action" (Direkx, 1997, p. 276), with electromyography the recording of such electrical activity. Electromyographic recordings are typically obtained from either surface or needle electrode recordings depending on the nature of the study being conducted. Needle EMG electrodes are used to record activity from single motor units or from small muscles with little interference from neighbouring muscles (Ludin, 1980, p. 15). While different types of needle electrodes exist they typically consist of a fine wire forming a stigmatic electrode within a cannula that is inserted directly into the muscle from which the recordings are required (Aminoff, 1998, pp. 48 – 49).

In contrast, non-invasive surface electrodes are routinely used for investigation of conduction velocities and neuromuscular transmission, with the recording a representation of the sum of individual potentials produced by all of the nerve/muscle fibres activated within the recording scope of the electrode (Loeb & Gans, 1986, p. 51). As with needle electrodes, a variety of different types of surface electrodes exist,

although all consist of a small metal disk (sometimes suspended in an electrolytic medium) placed on the skin overlaying the active muscle. This then forms a metal – electrode interface from which a charge gradient is developed (Cram, Kasman & Holtz, 1998, pp. 81 – 84). When two electrodes are used together, mechanical disturbances (i.e. movement or contraction) created by electrical potentials, alter the potential difference between the two electrodes, a difference that when measured gives rise to the recordable EMG (Aminoff, 1998, pp. 49 – 51). The recorded signal that can be obtained using surface EMG electrodes is dependent on both the size and placement of the electrodes.

2.3.1 *Skeletal Muscle Anatomy, Mechanics and Voluntary EMG*

At a macro level skeletal muscle is comprised of connective tissue, blood vessels, nerves and skeletal muscle tissue. Each skeletal muscle contains multiple muscle fascicles, which in turn comprise numerous skeletal muscle fibres. Individual muscle fibres are composed of multiple myofibrils that consist of bundles of myofilaments, which can actively shorten and are therefore responsible for skeletal muscle contraction (Figure 6). Surrounding the myofibrils is the sarcolemma that, due to an unequal distribution of positively and negatively charged ions, has the ability to conduct nervous system impulses. Skeletal muscle contraction occurs under the control of the central nervous system, the link between the nervous system and the muscle fibre occurring at the neuromuscular junction (NMJ). Each motoneuron leaving the spinal cord innervates a number of different muscle fibres that collectively are known as a motor unit. The number of motor units acting in a single muscle is dependant on the degree of control and speed with which that muscle is required to act. Nervous impulses from a motoneuron (referred to as action potentials) arriving at the NMJ stimulate the release of acetylcholine (ACh) that binds to receptors on the muscle fibre plasma membrane. This ACh binding changes the permeability of the sarcolemma to sodium ions resulting in an action potential being transmitted across the surface of the sarcolemma. This action potential then triggers the process of excitation-contraction (EC) coupling (Guyton & Hall, pp. 73 – 74; Martini, 1998, pp. 278 – 281; Spence & Mason, 1992, pp. 252 – 253).

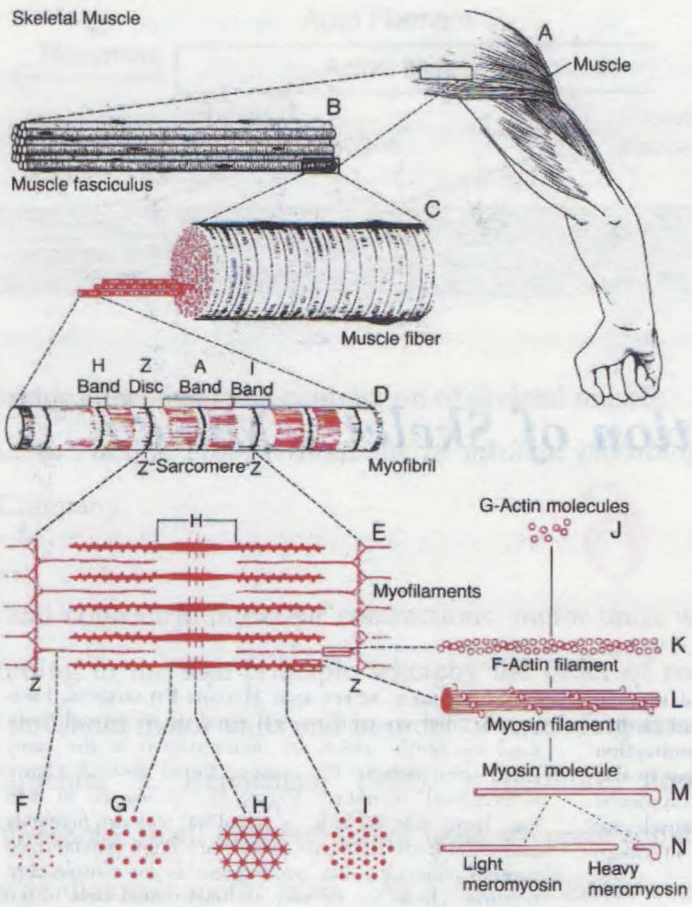


Figure 6. Macro and micro level organisation of skeletal muscle.

From: Guyton, A., & Hall, J. (1996). *Textbook of medical physiology*. USA : W.B. Saunders Company.

EC coupling begins with the temporary (0.03 seconds) release of calcium (Ca^{2+}) from the sarcoplasmic reticulum that binds to troponin, weakening the bond between the troponin complex and actin. This allows cross bridge binding between the actin and myosin and the release of stored energy causing the myosin head to pivot or 'cock', sliding the two filaments (actin and myosin) across each other in what is termed the *power stroke* (Figure 7). When this sliding occurs adenosine diphosphate (ADP) and phosphate are released, allowing the formation of adenosine triphosphate (ATP) that results in the breaking of the cross bridge attachment, the reactivation of ADP and P, and the recocking of the myosin head. This cycle is repeated as long as Ca^{2+} concentrations are increased and ATP reserves are sufficient. It is this sliding action of the filaments that is responsible for the shortening of the sarcomere and myofibril. The combined process occurring in multiple filaments resulting in the shortening, or contraction, of the muscle (Gordon, Regnier & Homsher, 2001; Pollack, 1983; Sieck & Regnier, 2001; Irving & Piazzesi, 1997).

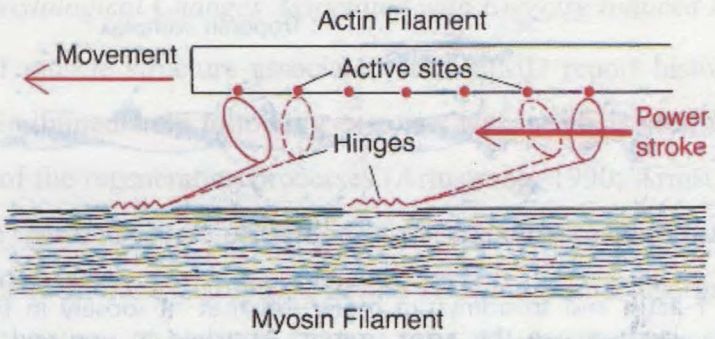


Figure 7. Cross bridge attachment and contraction of skeletal muscle.
From: Guyton, A., & Hall, J. (1996). *Textbook of medical physiology*. USA : W.B. Saunders Company.

During isometric and concentric muscular contractions, motor units within the muscle are recruited according to the size principle whereby the order of recruitment occurs from low to high threshold motor units and in order of ascending size (Cope & Pinter, 1995; Olson, Carpenter & Henneman, 1968). Therefore, during submaximal contractions low threshold, small diameter motor units are preferentially recruited over high threshold, large diameter motor units. As a result a direct correlation between EMG amplitude and force production has been demonstrated during muscular contractions of increasing intensity (Solomonow, Baratta, Shoji & D'Ambrosia, 1990). During eccentric contractions however it has been proposed that this order is reversed, thus high threshold motor units are preferentially recruited at low intensity contractions (Enoka, 1996). The reversed order of recruitment resulting in both a decreased EMG output during eccentric contractions for the same force generation during concentric contractions (Enoka, 1996), and increased torque output during electrical stimulation during maximal voluntary eccentric actions (Seger & Thorstensson, 2000). The extent to which this reversal of recruitment does occur with eccentric contractions however remains to be elucidated.

2.4 Exercise Induced Muscle Damage (EIMD)

EIMD commonly refers to muscle damage noted after exercise of a long duration, exercise of a strenuous unaccustomed nature, or exercise with a substantial eccentric component (Armstrong, 1990). The exact mechanism(s) by which the damage occurs is unknown, although it is thought to be initiated by mechanical stress within the muscle (McNeil & Khakee, 1992; Newham, Jones & Edwards, 1983) with the degree of damage increased as a result of metabolic factors and inflammation (Duncan, 1987).

2.4.1 *Histological Changes Associated with Exercise Induced Damage*

Investigations of muscle structure associated with EIMD report histological changes within the muscle immediately following exercise, with necrosis seen 1 – 2 days post-exercise as part of the regeneration processes (Armstrong, 1990; Armstrong, Ogilvie & Schwane, 1983). Mild to severe disruption of muscle ultrastructure has been reported, including plasma membrane disruption, Z-line streaming, and disorganisation of the myofilaments (Crenshaw, Thornell & Friden, 1994; Flitney & Hirst, 1978; McNeil & Khakee, 1992). The distribution and patterning of damage throughout the muscle being focal and inconsistent in nature (Newham, McPhail et al., 1983).

2.4.2 *Biochemical Changes Associated with Exercise Induced Muscle Damage*

Various investigators have reported the efflux of intramuscular enzymes into the blood following damaging exercise (Byrd, 1992; Child, Saxton & Donnelly, 1998; Ebbeling & Clarkson, 1989; Warren, Lowe & Armstrong, 1999). Creatine kinase (CK) efflux is a commonly reported observation (Eston, Finney, Baker & Baltzopoulos, 1996; Schwane, Johnson, Vandenakker & Armstrong, 1983), although poor correlations between the time course, degree of efflux, and degree of damage have been demonstrated (Newham, Jones et al., 1983). Found in the brain, skeletal and cardiac muscle, CK is a controlling enzyme for the production of anaerobic adenosine triphosphate (ATP) from the phosphagen or ATP-PC system, and is responsible for the replenishment of ATP during strenuous or short-term high intensity exercise (Eston, Mickleborough & Baltzopoulos, 1995). Variation in the magnitude of CK responses following eccentric exercise has been shown to be gender related and very individualistic (Clarkson & Ebbeling, 1988). In general, the peak CK response is seen 24 hours following exercise involving submaximal contractions and downhill running, with a more delayed peak occurring 4 – 5 days post-exercise where high force eccentric exercise is performed (Clarkson, Nosaka & Braun, 1992). Increases in concentrations of plasma myosin heavy chain fragments, circulating leukocyte and lymphocyte subsets, aspartate aminotransferase and lactate dehydrogenase have also been associated with EIMD, however, as with CK the time course and degree of efflux is not indicative of the magnitude of damage (Mair, Koller et al., 1992; Nosaka & Clarkson, 1996; Pizza et al, 1995).

2.4.3 *Delayed Onset Muscle Soreness*

The muscle pain that occurs 24 – 48 hours after strenuous unaccustomed exercise is referred to as delayed onset muscle soreness (DOMS) (Miles & Clarkson, 1994), with

the sensation closely associated with EIMD (Schwane et al., 1983). A characteristic of DOMS is that pain is absent at rest and is experienced only upon active movement or contraction of the affected muscle, or when direct pressure is placed on the muscle (Gulick & Kimura, 1996). Originally suggested as resulting from the accumulation of lactic acid and ischemia initiated by local muscular spasm (Travell, Rinzler & Herman, 1942), later investigators have largely rejected these ideas (Abraham, 1977; Asmussen, 1956; Newham, Mills, Quigley & Edwards, 1983; Thomas, Londeree, Ziogas & Cox, 1994). Opposition for the lactic acid theory is largely based on the poor relationship between lactate levels during eccentric exercise and the magnitude of DOMS experienced (Nottle, 1998; Schwane, Watrous, Johnson & Armstrong, 1983). It is now proposed that the pain experienced is the result of stimulation of free nerve endings caused by an inflammatory response to microtrauma within the muscle (Tiidus & Ianuzzo, 1983). The delay in the appearance of pain postulated as resulting from the time taken for noxious substances to enter the extracellular space and for the inflammatory response to occur (Stauber, Clarkson, Fritz & Evans, 1990).

2.4.4 *Performance Changes Associated with Exercise Induced Muscle Damage*

Numerous authors have reported declines in performance associated with EIMD. Davies and White (1981) and Smith, Fulmer et al. (1994) demonstrated a reduction in maximal voluntary isometric strength following damaging exercise. Reductions in maximal voluntary isokinetic strength (Eston et al., 1996) and power production (Sargeant & Dolan, 1987) have also been shown. The greatest reductions in performance reported to occur immediately post-exercise; with complete recovery occurring 5 – 28 days post-exercise (depending upon the extent of maximum strength loss).

While the mechanism(s) responsible for the reductions in strength and power have not been completely identified, it is likely that muscle contractility, and therefore force production, are reduced as a result of ultrastructural damage and impairment of the calcium pump within the muscle (Byrd, McCutcheon, Hodgson & Gollnick, 1989; Clarkson et al., 1992). EC coupling disruption has also been suggested as a mechanism contributing to initial levels of strength loss post-exercise (Warren, Ingalls, Lowe & Armstrong, 2001). The pain commonly associated with EIMD has been suggested to result in reduced subject motivation, explaining the observed reduction in voluntary strength. However, the findings that the strength loss typically occurs before the onset

of pain (Clarkson et al, 1992; Hortobagyi et al., 1998), and similar force declines occur with supramaximal transcutaneous electrical stimulation following eccentric exercise (Davies & White, 1981) point to other causes.

With many of the suggested mechanisms for strength loss focusing on the peripheral mechanisms of ultrastructural damage, inflammation and necrosis (Asmussen, 1956; Armstrong, 1990; Smith, 1991), little consideration has been given to centrally mediated control of the active muscle. The findings of EMG investigations have been conflicting with increases (Kroon & Naeiji, 1991), decreases (Komi & Rusko, 1974) and no change (Deschenes et al., 2000) in EMG reported following eccentric contractions. Furthmore, Saxton et al. (1995) concluded that no loss in voluntary muscle activation occurred following eccentric exercise, while Gibala, MacDougall, Tarnopolsky, Stauber and Elloriaga (1995) reported a 6% decrease in voluntary activation using twitch interpolation following eccentric exercise. Sayers et al. (2003) also reporting a reduction in compound muscle action potentials of the elbow flexors following eccentric contractions, although the authors did not report examining voluntary EMG or the central activation ratio. The low volume of published findings in this area appears to be the main cause for conflicting results, with too few studies of similar methodology to demonstrate a pattern in the results.

In addition to reporting strength declines, Saxton et al. (1995) found that, following eccentric contractions of the elbow flexors, both joint position sense, and the perception of force (as determined by a contralateral limb matching task) were altered. When matching force post-exercise, subjects overestimated the force, i.e. they felt that the exercised (reference) arm was producing a stronger contraction than actually produced. During the position matching task subjects perceived the exercised (matching) elbow angle to be in a more flexed position post-exercise, thus placing the arm in a more extended position than the matching arm. Brockett et al. (1997) reported similar disruption in relation to position sensation following eccentric exercise. However, in the latter study subjects perceived their elbow to be in a more extended position post-exercise, with the exercised (reference) arm placed in a more flexed position than the matching arm. Despite this difference between findings both groups proposed that the altered joint sense occurred as a result of damage to, or dysfunction of, the muscle spindles resulting in alteration of proprioceptive feedback during the task performance.

Both groups also cited the observed reduction in relaxed elbow angle following the eccentric exercise as a possible contributor to changes in joint position perceptions.

Proske et al. (2003) suggested that following eccentric exercise of the elbow flexors, the initial over estimation of matching forces was likely the result of changes in motor unit recruitment patterns, with sustained errors possibly resulting from the delayed onset muscle soreness experienced. The view that errors were prolonged as a result of soreness occurring due to a similar pattern of errors being observed during a matching task where soreness was induced by injection of hypertonic saline into the biceps of the matching arm. A study by Peace et al. (1998) examined the influence of eccentric exercise on performance of a visuomotor tracking task of the elbow flexors/extensors. They found that immediately following eccentric exercise tracking error increased by an average of 14%, further increasing to 27% at 24 hours, with recovery at approximately 14 days. Disruption of proprioceptive feedback from the muscle spindles and cutaneous afferents being suggested as contributing factors to the observed changes in tracking performance. It was also suggested that the changes in performance of the tracking task might be related to reductions in the force generating capacity of the damaged muscle since a significant correlation was reported between strength losses and tracking error.

2.4.5 *Protocols for inducing EIMD*

Although the phenomena of EIMD and DOMS are not limited to specific muscle groups, throughout the literature, the elbow flexors and knee extensors have been by far the most commonly studied. In a methodical review of 30 muscle damage and muscle soreness studies from 1983 – 2001 (Table 2), 15 (50%) of the protocols exercised the elbow flexors, 7 (23%) exercised the knee extensors, 5 (17%) involved downhill walking or running (exercising predominantly the gluteals and quadriceps), and 3 (10%) performed downhill backward walking (exercising triceps surae). The most obvious link between all protocols being an eccentrically biased exercise protocol.

Of the studies on the elbow flexors 14 of the 15 (93%) exercise protocols employed maximal eccentric contractions (>70% concentric one repetition maximum). Twelve (86%) involved a predetermined number of sets and repetitions (references 1-12), with the remaining two protocols (14%) consisting of eccentric actions to exhaustion or fatigue (references 13-14). Only one study (7%) was conducted using a submaximal exercise protocol, with a minimum number of contractions being set and subjects then

exercising until fatigue (reference 15). Similarly, all 7 of the 30 (23%) papers using protocols performed on the knee extensors involved maximal eccentric contractions, with 5 (71%) protocol using a predetermined number of sets and repetitions (references 16-20), and 2 (29%) protocols performed to fatigue (references 21-22). An additional 5 studies examining changes in the knee extensors employed downhill walking or running, of which 4 (80%) consisted of a set exercise time (references 23-26), and 1 (20%) was conducted until exhaustion (reference 27). Finally, 3 of the 30 (10%) papers employed a downhill backward walking protocol to examine exercise induced muscle damage in triceps surae (references 28-30). All protocols were of a fixed length of time although this varied from 60 – 120 minutes, with two studies using a step – together walking action such that only one limb was eccentrically exercised.

The strength losses reported following eccentric exercise of the elbow flexors were typically greater than those involving either the knee extensors or triceps surae. Additionally, the strength losses are maintained for a longer period in the elbow flexors, probably reflective of the greater initial strength losses. For the elbow flexors, peak strength losses of approximately 40% of their pre-exercise values were common, with reductions of up to 50% occurring. Significant strength loss was usually present for 3 or more days post-exercise with the time course of recovery largely dependent on the levels of initial strength loss. The exception to this being the protocol involving submaximal eccentric contractions of the elbow flexors where only a 10% reduction in strength was observed immediately following exercise with recovery to baseline occurring by 1 day post-exercise. Exercise protocols specifically targeting the knee extensors tended to report strength losses of approximately 20 – 40%, with the higher repetition protocols generally reporting the larger strength losses. While the downhill walking/running protocols reported strength losses in the knee extensors of approximately 20 – 30%.

All studies regardless of the exercise protocol employed or the muscle group tested reported soreness following eccentric exercise (where measured). Likewise, all studies reported an increase in plasma CK levels (where measured) following the exercise protocol with the time course and concentration of the peak reading varying across the protocols. While a large volume of literature with similar protocols exists for the elbow flexors and to a lesser extent the knee extensors, examination of EIMD in the lower limb or in any muscle group following submaximal exercise is comparatively limited.

Table 2
Reference Details for Papers Inducing Muscle Soreness and Damage

	Authors	Year	Sets	Reps
1	Behm, Baker, Kellard & Lomond	2001	7	10
2	Jones, Newham & Torgan	1989	1	80
3	Murayama, Nosaka, Yoneda & Minamitani	2000	1	24
4	Nosaka & Sakamoto	2000	1	24
5	Nosaka & Sakamoto	2001	1	24
6	Paddon-Jones & Quigley	1997	8	8
7	Paddon-Jones, Muthalib & Jenkins	2000	1	36
8	Pearce et al.	1998	7	5
9	Saxton & Donnelly	1995	1	70
10	Saxton & Donnelly	1996	1	70
11	Smith, Keating et al.	1994	4-5	35
12	Stauber et al.	1990	1	70
13	Weber, Servedio & Woodall	1994	To fatigue	
14	Yackzan, Adams & Francis	1984	To fatigue	
15	Brockett et al.	1997	To fatigue	
16	Bourgeois, MacDougall, MacDonald & Tarnopolsky	1999	6	10
17	Child et al.	1998	1	75
18	Deschenes et al.	2000	4	25
19	MacIntyre, Reid, Lyster, Szasz & McKenzie	1996	3	10
20	Tiidus & Shoemaker	1995	7	20
21	Crenshaw et al.	1994	To fatigue	
22	Golden & Dudley	1992	To fatigue	
23	Byrnes et al.	1985	30 minutes	
24	Eston et al.	1996	40 minutes	
25	Maughan et al.	1989	45 minutes	
26	Schwane et al.	1983	45 minutes	
27	Sargeant & Dolan	1987	To fatigue	
28	Jones, Allen, Talbot, Morgan & Proske	1997	120 minutes	
29	Whitehead, Allen, Morgan & Proske	1998	60 minutes	
30	Whitehead, Weerakkody, Gregory, Morgan & Proske	2001	60 mintues	

2.4.6 *The Repeated Bout Effect*

The repeated bout effect refers to the observations that a single bout of eccentric exercise produces an adaptation that reduces muscle damage during subsequent bouts of eccentric exercise (Nosaka, Sakamoto, Newton & Sacco, 2001). A number of studies investigating the repeated bout effect have demonstrated little or no increase in plasma CK or DOMS with a second bout of exercise performed 4 days to 6 weeks after the initial bout (Clarkson & Tremblay, 1988; Mair, Mayr et al., 1995). Studies investigating the effect of a repeated bout of exercise on strength decline have shown that while the same degree of strength loss occurs post-exercise, the rate of strength recovery is more rapid following the second exercise bout (Eston et al., 1996; Newham et al., 1987).

Three main mechanisms have been proposed to explain the protection associated with the repeated bout effect (McHugh, 2003). Firstly, it has been suggested that connective tissue and membranes within the muscles are strengthened following the initial bout of damage providing protection against the same forces during the repeat bout (Clarkson & Tremblay, 1988; Newham et al., 1987). Secondly, it has been proposed that the initial bout of exercise may result in a neural adaptation, so as to increase the efficiency by which muscle is activated during the repeated bout. More efficient recruitment and increased synchrony of motor unit firing resulting in a 'learnt' protective mechanism against damage rather than a structural adaptation. (Golden & Dudley, 1992; Nosaka & Clarkson, 1995). Finally, adaptations in the inflammatory response (Pizza et al., 1996), longitudinal addition of sarcomeres (Brockett, Morgan & Proske, 2001; Lynn & Morgan, 1994), and adaptations in E-C coupling (Warren et al, 2001), also providing possible mechanisms for a reduction in the severity of strength loss and other indicators of muscle damage with a repeated bout of eccentric exercise. All of the above concepts however, are based on the model that the damage associated with EIMD is initiated by mechanical stress resulting in peripheral changes in the muscle rather than centrally mediated alterations in muscle function and control. Little consideration therefore has been given to the possible role CNS adaptations may have in the conferral of protection with a repeated bout of eccentric exercise.

2.5 Summary

To date much of the literature relating to EIMD focuses on the common indicators of soreness, plasma CK, and strength declines, with little consideration given to functional

changes in proprioception, and the activation changes that may influence control and performance during skilled tasks. While proprioceptive and activation changes are readily studied in patient populations, it appears the transient and non-permanent nature of EIMD has limited the extent to which this mode of damage has been investigated. Only in recent years have investigators begun to consider the greater implications of EIMD in regards to being a possible stimulus for the occurrence of secondary injuries. The lack of understanding of the mechanisms responsible for EIMD also highlighting the need for multivariable investigations to allow for causational relationships to be better developed.

CHAPTER 3 METHODS AND EQUIPMENT

3.1 Experimental Design

Data collection was completed over two main studies; the first examined the effectiveness of a downhill backward walking protocol for inducing muscle damage in triceps surae (Chapter 4). While a downhill backward walking protocol had been used previously, the number of studies and the results relating to triceps surae are sparse compared to those relating to the elbow flexors or knee extensors. This study was also designed to establish whether triceps surae responded to a second bout of eccentric exercise according to the 'repeated bout effect' (Chapter 5), as this had yet to be determined. The second phase of testing focused on the proprioceptive and activation changes in triceps surae following a single bout of submaximal eccentric exercise using the downhill backward walking model (Chapter 6). This study also included examination of the protective mechanism in relation to proprioceptive and activation responses of triceps surae following a repeated bout of submaximal eccentric exercise (Chapter 7).

3.2 Subjects

Subjects for each study were recruited from associates of the investigator, staff and student populations of the Edith Cowan University Joondalup Campus. Methodology for all studies receiving approval from the Edith Cowan University Human Ethics Committee. All subjects volunteered to participate and provided informed written consent (Appendix A) and completed a medical questionnaire (Appendix B) prior to testing. Anthropometric measures of height (cm) and weight (kg) were taken for each subject prior to testing. Additionally, the following standard selection criteria was applied to all studies:

- Healthy adult (18 – 45 years);
- Had not participated in resistance training of the plantarflexors in past 6 months;
- No known neurological or neuromuscular disorders;
- Not currently taking any medications that may affect the central nervous system or muscle function;
- No injury to the ankle or surrounding muscle, tissue or tendons in the past 6 months;
- An ankle range of motion of 10° of dorsiflexion to 30° of plantarflexion.

3.2.1 *Sample Size*

While it is recommended that a test sample should consist of at least 30 subjects in order to reduce the likelihood of a type I error (Burns & Grove, 1993, p. 247), previously conducted work in areas relating to the current research, have shown that significant results can be obtained with a sample size as small as five subjects (Brockett et al., 1997; Clarkson & Tremblay, 1988; Davies & White, 1981; Jones et al., 1997; Saxton et al., 1995). For this reason, subject numbers throughout the current studies were selected based on previous research rather than the power calculation method. A minimum sample size of 10 subjects being set for all studies with 15 – 20 subjects the preferred sample size.

3.3 Equipment and Measurement

3.3.1 *Data Acquisition*

The AmLab windows–based software (version 2.0, Amlab : AUS) and hardware (single digital signal processor, mini-rack interface, and 18 channel isolated ground card) computer base application package was used to record, store and analyse data. Additionally, data contained in AmLab replay files was exported and analysed using the Microsoft Excel (version 1997, Microsoft, USA) application program. Specific setting and schematic applications relating to AmLab are further discussed within each experimental methodology.

3.3.2 *Apparatus*

To simultaneously measure torque or angular displacement of the ankle, a purpose built (Ribuck Industries, Western Australia : AUS) Dual Ankle Dynamometer (DAD) was developed (Figure 8). The DAD consisted of a base frame and a variable height seat, mounted with two footplates that could be adjusted for both plate height, and distance between the two plates.

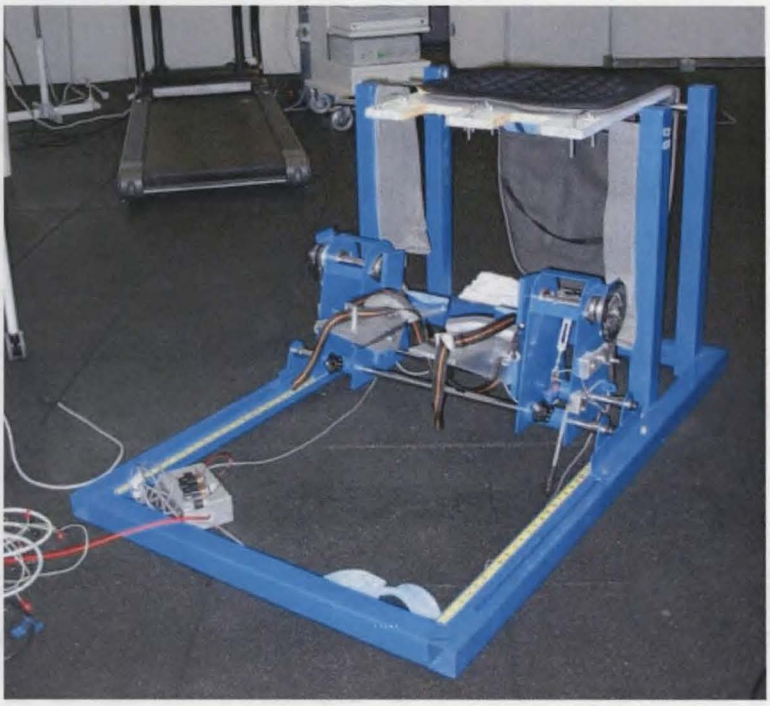


Figure 8. The Dual Ankle Ergometer (DAD) designed specifically to measure torque and angular displacement in the ankle plantarflexors.

Each footplate was attached to a rotating rod connected via a belt pulley system to a displacement transducer to determine angular displacement around the ankle axis of rotation. Using a force transducer fixed to the rotating rod via a 5 mm Zenith turnbuckle, each footplate could also be locked in position to give plantarflexion torque at variable ankle angles (Figure 9).

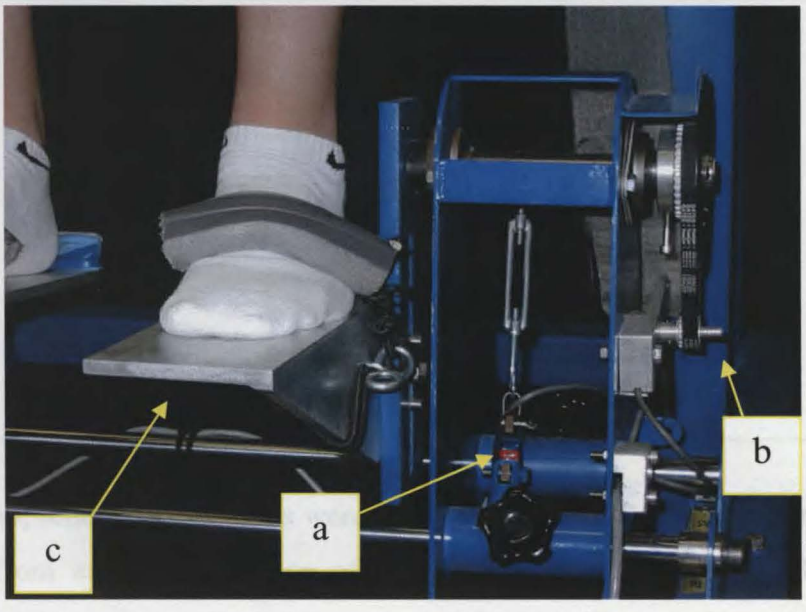


Figure 9. Location of force (a) and displacement (b) transducers in relation to the footplates (c) of the DAD.

The force transducers (Radio Spares model 021-300) used during experimentation were foil (copper-nickel alloy) uni-axial strain gauges (resistance 120 Ω, Wheatstone bridge connection) receiving a constant DC input from a 9 V battery. The displacement transducers were 5 kOhm linear rotary potentiometers (Dick Smith Electronics: part number R6805). A 4:1 gearing reduction was achieved via the belt pulley system with each potentiometer receiving a constant input voltage from mains AC power supply (± 3.3 V swing). All output signals from the strain gauges and potentiometers were relayed to AmLab via shielded cabling.

Signals from the DAD were sampled and viewed in real time as a voltage change using AmLab. Conversions to angular degrees (°) and torque (Nm) units were conducted post – test based on calculations determined via calibration procedures (see calibration section 3.3.3). The sampling rates, channel gain, and scaling factors for each signal-input device from the DAD are provided in Table 3.

Table 3.
Sampling Rates, Amplifier Gains and Scaling Factors For Each Input Device From The DAD

Protocols	Input	Sampling Rate (Hz)	Scaling Factor	Channel Gain	Storage Decimation Factor
H-reflex & tendon reflex	Left strain gauge	4000	-245	100	5
	Right strain gauge	4000	-295	175	5
Strength & torque matching	Left strain gauge	1000	-245	100	5
	Right strain gauge	1000	-295	175	5
Angle matching	Left and right potentiometer	250	1	8.918	1

Using AmLab, schematic programs were designed to detail the exact sequence of signal processing from each input device of the DAD, in addition to signal input from electromyographic sources. Each schematic developed is illustrated in Appendix C.

3.3.3 *Calibration of the DAD*

Calibration of the signal output for the strain gauges and potentiometers of the DAD was conducted at regular intervals throughout the testing period, and whenever the battery powering the strain gauges was replaced. Calibration of the strain gauges involved loading each footplate fixed at –10° with a known weight (45.36 kg maximum - using 11.35 kg increments) and recording the subsequent voltage displayed through AmLab. Readings were taken during both the loading and unloading of the weights to ensure linearity was maintained. From the calibration procedure involving the strain gauges and using the same method of weight application on the Cybex 6000 isokinetic dynamometer (Cybex, NY : USA), the following calculations were used to convert voltage recordings to torque values in Nm:

From Cybex	From DAD	∴
11.35 kg = 17.43 Nm	11.35 kg = 63.44 V	1Nm = 3.64 V

Calibration of the signal output from each potentiometer was conducted using protractors mounted on the side of the DAD. Each footplate was moved at 5° intervals from -20° to +30° with the subsequent voltage displayed and recorded through AmLab. From this procedure it was determined that 1° was equal to 0.052 V. Calibration records for each strain gauge and potentiometer are given in Appendix D and E respectively.

3.3.4 *Subject Positioning on the DAD*

For all testing on the DAD, subjects were seated with the knees and hips at 90° flexion and the ankles at 10° dorsiflexion. The distance between the footplates was adjusted so that the line from the knee to the ankle of both limbs was parallel to each other and therefore, was perpendicular to the axis of rotation of the ankle. The height of the footplate was also adjusted so that the axis of rotation of the plate was aligned with the lateral malleolus (Figure 10).

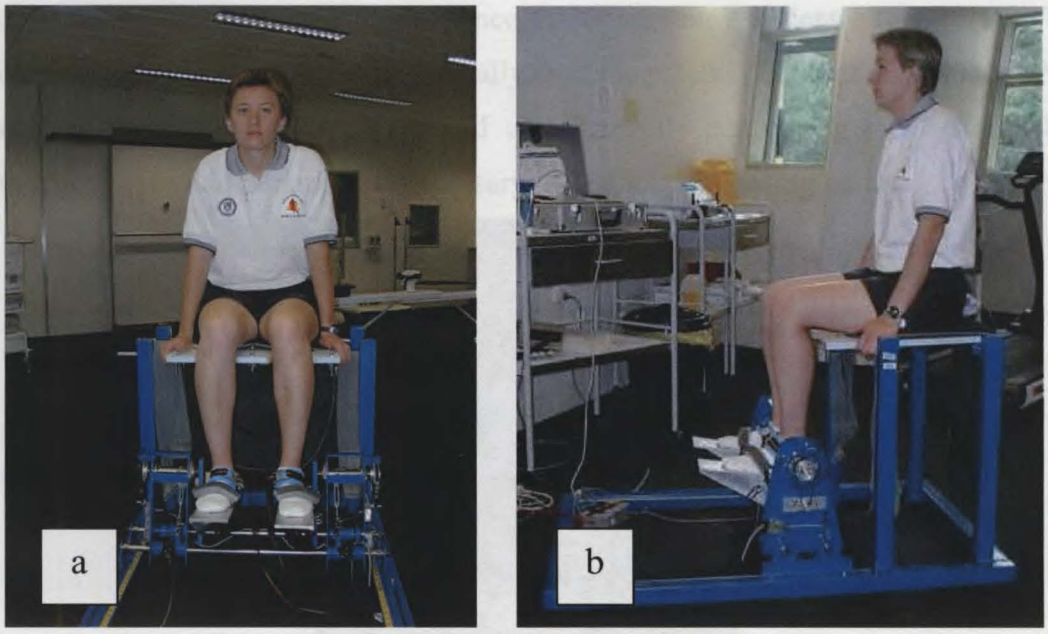


Figure 10. Front (a) and lateral (b) view of subject positioning during testing on the DAD.

Each foot was secured in place using strapping from below the footplate over the region of the inferior and superior extensor retinaculum (Figure 11). The straps were set as tight as possible with the aim of minimising heel lift during plantarflexion, but without causing discomfort to the subject.

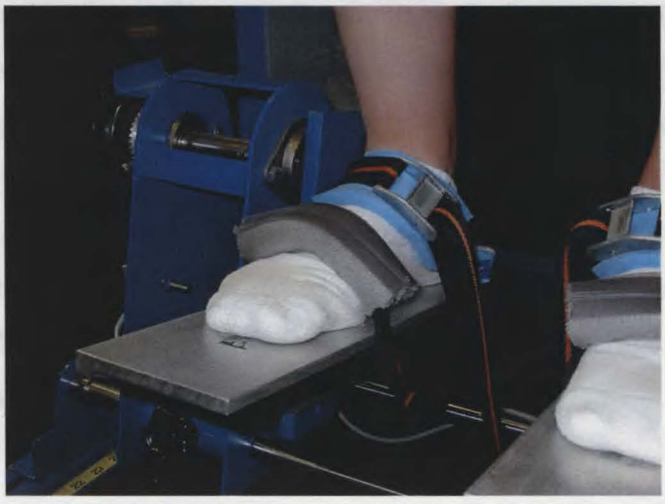


Figure 11. Strapping used to secure the foot during the maximal voluntary strength task performed on the DAD.

3.3.5 *Electromyographical Analysis*

Electromyographical (EMG) analysis of muscular activity was conducted during the maximal voluntary strength protocol of the plantarflexors (using the DAD only), the force perception task, and the reflex protocols. EMG responses were recorded with Meditrace 200 Ag/AgCl surface electrodes placed over the medial head of the

gastrocnemius and the soleus at a distance of 30mm centre to centre (Figure 12). The reference electrode was positioned centrally over the patella. In order to replicate EMG signalling, electrode sites were marked with indelible pen during the first baseline testing occasions and remarked as necessary throughout the testing period.

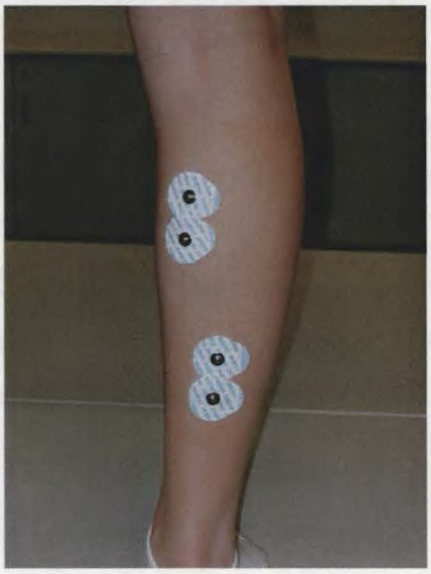


Figure 12. Electrode placement for the gastrocnemius and soleus muscle used during EMG analysis

Table 4 outlines the filter settings, sampling rates, scaling factors and channel gains for each protocol. The band-pass filter consisted of a second order quasi-Butterworth low-pass filter in series with a second order quasi-Butterworth high pass filter. The points of reference defining the band-pass were –3 dB points. EMG signals collected during the reflex protocols (reflex EMG) were filtered, displayed, stored, and analysed in raw format. EMG signals collected during the maximal voluntary strength and force perception protocols were filtered, rectified, displayed, and stored (using AmLab). The EMG data were then exported to Microsoft Excel where an average of the values was calculated for data analysis (avEMG).

Table 4
Filter Settings, Sampling Rates, Scaling Factors, Channel Gains and Storage Factors for EMG Data Collection with AmLab

Protocols	Filtering		Sampling Rate (Hz)	Scaling Factor	Channel Gain	Storage Decimation Factor
	Low Pass	High Pass				
Reflex EMG	3.528	1025.16	4000	2	2000	1
avEMG	5.746	478.98	1000	2	4000	1

3.4 Limitations

1) As all subjects were adult (18 – 45 years) volunteers recruited predominantly from a university environment, the results obtained may not be a true representation of the general population.
2) Changes in motivation across the testing time intervals may have influenced the subjective ratings for soreness and concentration levels of the subjects during matching tasks.
3) All subjects were tested with the left limb acting as the exercised limb regardless of limb dominance. It is unknown if limb dominance would have influenced the results obtained had this been randomised.

CHAPTER 4 EVALUATION OF DOWNHILL BACKWARD WALKING AS A MODEL FOR EXERCISE-INDUCED MUSCLE DAMAGE OF TRICEPS SURAE

4.1 Introduction

Delayed onset muscle soreness or DOMS can be described as the "sensation of discomfort or pain in the skeletal muscle that occurs following unaccustomed muscular exertion" (Armstrong, 1984, p. 529). DOMS is typically quantified subjectively through the use of visual analogue or numerical scales (Mattacola, Perrin, Gansneder, Allen & Mickey, 1997). Subjects are required to rate pain while performing a movement such as walking or elbow flexion, or on palpation of the exercised muscles (Franklin, Currier & Franklin, 1991; Howell et al., 1993). Some studies have attempted to refine the technique by using the force applied to a muscle at the pain threshold as an index of soreness, often referred to as tenderness (Newham, Mills et al., 1983; Weerakkody, et al., 2003).

While the exact etiology of DOMS remains unknown, it is presumed to be associated with the inflammatory response that occurs within muscle (Gulick & Kimura, 1996). The sensitisation of polymodal nociceptors following the release of noxious substances from damaged fibres results in a reduced threshold for activation of the sensation of pain during normally non-painful movements and palpation (Weerakkody, Whitehead, Gregory & Proske, 2000). Histological examination of myofibre injury from muscle biopsy samples is the most definitive method of quantifying EIMD following eccentric exercise, however its use in the study of eccentric-contraction induced injury is low compared to other methods due to the inconsistent nature of the damage, and the risk of further damage by the biopsy procedures. A review paper conducted by Warren, Lowe et al. (1999) found that only 6 out of 52 papers (12%) examined conducted quantitative histological analysis of the muscle following eccentric exercise. Instead the assessment of maximal voluntary torque (26/52: 50%), blood proteins (27/52: 52%), and soreness (39/52: 75%) proved more common measures. The papers reviewed by Warren published in the period 1981-1995. A methodical examination of literature conducted on articles published after 1995 demonstrating a similar trend with histology (H), maximal voluntary torque (T), blood proteins (B), and soreness (S), used in 16, 69, 65 and 83% of the 51 articles reviewed (Table 5).

Table 5.
Reviewed literature published post 1995 and their measurement tools for EIMD

Authors	Year	Measurement Tool			
		H	T	B	S
Barlas, Walsh, Baxter & Allen	2000				√
Beaton, Allan, Tarnopolsky, Tiidus & Phillips	2002	√	√	√	√
Bourgeois et al.	1999	√	√	√	√
Brockett et al	2001		√		√
Brown, Child, Day & Donnelly	1997		√	√	√
Byrne & Eston	2002		√	√	
Chen & Hsieh	2000		√	√	√
Chen & Hsieh	2001		√	√	√
Child et al.	1998		√	√	√
Connolly, Reed & McHugh	2002		√	√	√
Craig, Barlas, Baxter, Walsh & Allen	1996				√
Craig, Bradley, Walsh, Baxter & Allen	1999				√
Dannecker, Koltyn, Riley III & Robinson	2002				√
Dannecker, Koltyn, Riley III & Robinson	2003				√
Dolezal, Potteiger, Jacobsen & Benedict	2000			√	√
Eston et al.	1996		√	√	√
Evans, Knight, Draper & Parcell	2002		√	√	√
Foley, Jayaraman, Prior, Pivarnik, & Meyer	1999			√	√
Gibala et al.	2000	√			
Gleeson, Eston, Marginson & McHugh	2003		√	√	√
Gulbin & Gaffney	2002		√	√	
Harrison et al.	2001		√	√	√
Hortobagyi et al.	1996	√	√	√	√
Hortobagyi et al.	1998	√	√	√	√
Jamurtas et al.	2000			√	√
Jones et al.	1997	√	√		√
Kauranen, Siira & Vanharanta	2001		√	√	√

(table continues)

Table 5 (continued)

Authors	Year	Measurement Tool H	T	B	S
Komulainen, Koskinen, Kalliokoski, Takala & Vihko	1999	√		√	
Koskinen et al.	2001			√	
Kraemer et al.	2002				√
Lambert, Marcus, Burgess & Noakes	2002		√	√	√
Lee et al.	2002		√	√	√
McHugh, Connolly, Eston, Gartman & Gleim	2001		√	√	
McHugh, Connolly, Eston, Kremenic et al.	1999		√	√	√
Michaut, Pousson, Babault & Van Hoecke	2002		√		
Nosaka & Newton	2002		√	√	√
Nosaka, Newton & Sacco	2002		√	√	√
Nosaka et al.	2001		√	√	√
O'Connor, Poudevigne & Pasley	2002				√
Paddon-Jones et al.	2000		√	√	√
Patel, Cuizon, Mathieu-Costello, Friden & Lieber	1998	√	√		
Pearce et al.	1998		√	√	√
Rindard, Clarkson, Smith & Grossman	2000		√		√
Roth, Gajdosik & Ruby	2001			√	√
Sayers, Clarkson & Lee	2000a		√	√	√
Sayers, Clarkson & Lee	2000b			√	
Sayers, Knight, Clarkson, Van Wegen & Kamen	2001		√		√
Sbriccoli et al.	2001		√	√	
Weerakkody, Whitehead, Canny, Gregory & Proske	2001				√
Whitehead et al.	1998		√		√
Whitehead et al.	2001		√		√
Number of reviewed articles using measurement tool	(n)	8	35	33	42
Percentage of reviewed articles using measurement tool	(%)	16	69	65	83

Where H = histology, T = maximal voluntary torque, B = blood proteins, S = soreness

Elevation in plasma levels of a number of different myofibre proteins have also been examined following damaging exercise (eg. creatine kinase) and are generally accepted

as indicators of skeletal muscle damage (Clarkson & Ebbeling, 1988). Previous investigators have demonstrated large increases in serum CK occur following a novel or eccentric exercise protocol (Maughan et al., 1989; Saxton & Donnelly, 1995). Whether any cause-and-effect relationship exists between the observed CK increases and DOMS is unclear, although the intensity and duration of the exercise protocol has been suggested to influence both variables (Tiidus & Ianuzzo, 1983).

Evaluation of DOMS and the measurement of CK efflux following eccentric exercise are the most widely used representations of the occurrence of muscle damage (Nosaka, Newton & Sacco, 2000). Additionally, a reduction in maximal voluntary strength is an accepted indicator of muscle damage, although the exact mechanism(s) of the torque reduction are unknown (Byrne & Eston, 2002). Few studies have examined the responses of triceps surae following a bout of eccentric exercise. As it was intended to examine activation and proprioceptive changes in triceps surae associated with muscle damage, it was necessary to characterise the EIMD responses following downhill backward walking. Therefore, the aim of the current investigation was to establish the effectiveness of a downhill backward walking protocol for inducing the common symptoms associated with EIMD in triceps surae.

4.2 Methods

4.2.1 *Subjects*

Twenty subjects were recruited for the study with all subjects tested for soreness, tenderness and plasma CK across time. Seventeen of the 20 subjects completed all time intervals for maximal voluntary isokinetic strength, with 15 of the 20 subjects completing all time intervals for maximal voluntary isometric strength. The group consisted of male and female subjects for whom mean values for age, height and weight are reported in Table 6.

Table 6
Subject Characteristics for Age, Height and Weight

	Mean ± SEM		
	Group (N=20)	Males (n=11)	Females (n=9)
Age (years)	28.0 ± 1.6	31.3 ± 2.3	24.1 ± 1.6
Height (cm)	170.9 ± 1.9	175.6 ± 1.9	165.1 ± 2.6
Weight (kg)	74.5 ± 2.8	78.9 ± 3.2	69.2 ± 4.4

4.2.2 *Testing Schedule*

Testing was conducted on seven occasions over a 10-day period with the exercise protocol performed once by each subject during this period. Criterion testing was conducted 72 and 48 hours pre-walk, and 0.5, 24, 48, 72 and 96 hours post-walk for all subjects.

4.2.3 *Exercise Protocol*

The exercise protocol consisted of 60 minutes of downhill backward walking (grade – 15%) on a modified Trackmaster (TM500) motor driven treadmill (JAS manufacturing, TX, USA). Based on a protocol described by Jones et al. (1997), subjects were asked to concentrate on stepping backward with a toe-to-heel action with the left limb (Figure 13), whereby the plantarflexors were contracted eccentrically (exercised limb). The right leg was then brought together with the left causing minimal stretch of the plantarflexors of the right (non-exercised) limb, with speed individualised in order to maintain a stepping rate of 30-35 strides per minute. Treadmill speed was kept constant throughout the 60-minute protocol. As the task was considered submaximal no specific warm-up or stretching was performed prior to the exercise protocol.

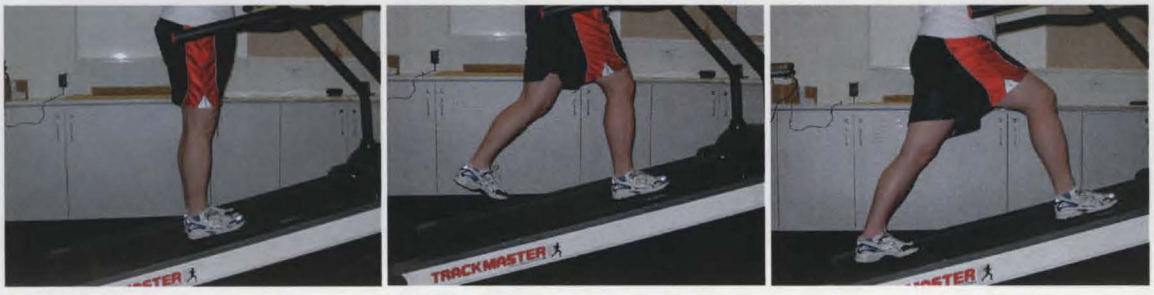


Figure 13. Downhill backward walking protocol with a step-together action allowing for eccentrically biased loading of the left limb and minimal loading of the right limb

To determine the intensity of the exercise protocol, heart rate was monitored for six individuals at rest, and over the course of the 60-minute protocol using a Polar A_3 heart rate monitor (Polar Electro Oy, Kempele, Finland).

4.2.4 *Creatine Kinase (CK)*

Plasma creatine kinase activity was determined from a blood sample collected from the fingertip. Following puncture of the cleaned finger using the Unistik 2 autolancing

device (Owen Mumford, Oxford, UK), a 30 μL blood sample was collected via a heparinised capillary tube (Bohringer-Mannheim, Indiana, USA). This was pipetted immediately on to a CK test strip and analysed using a Reflotron analyser (Bohringer-Mannheim, Indiana, USA).

4.2.5 *Soreness and Tenderness*

Muscle soreness was evaluated using a 1 (normal) to 10 (very very sore) scale (Smith, Fulmer et al., 1994). Subjects were asked to report soreness values while walking at a comfortable pace on a level and stable surface for five sites on the lower leg (Figure 14). The proximal sites were marked 5cm below the popliteal fossa over the belly of the medial and lateral gastrocnemius. The distal sites were marked 5 cm above the insertion of medial and lateral heads of gastrocnemius over the belly of the muscle. The soleus site marked 5 cm below the insertion of the medial and lateral heads of gastrocnemius along the midline of the limb. Subjects were asked to score the general area around each site as specifically as possible.

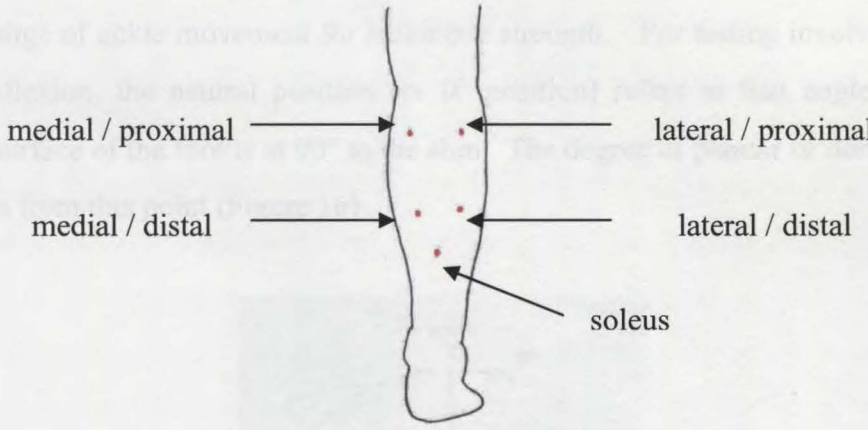


Figure 14. Standard sites used during recording for muscle soreness and tenderness.

Tenderness was determined using a myometer (Dobros, supplier unknown) with a 1.5 cm rubber tip (Figure 15). The myometer was applied at each marked site with increasing pressure (to a ceiling value of 100 kPa/14.5 psi), with subjects asked to report the moment pain was perceived (Eston et al., 1996). The pressure required to elicit that pain was then recorded, with increased tenderness represented by a decrease in the pressure recorded. For testing consistency, each site was marked on the initial day of testing with a semi permanent surgical marker and remarked throughout testing as required.

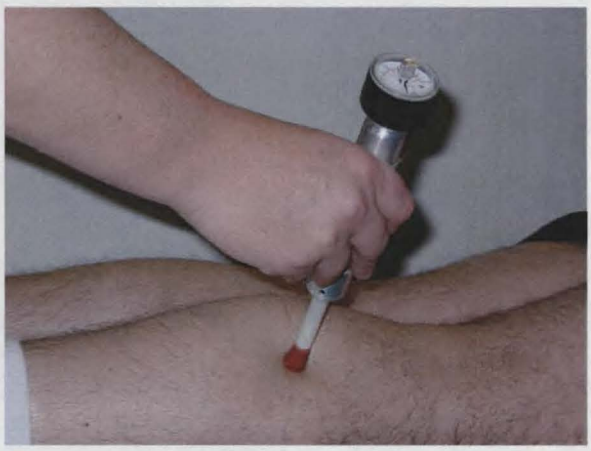


Figure 15. Application of the myometer to determine pain pressure threshold.

4.2.6 *Maximal Voluntary Strength*

Maximal voluntary isometric and isokinetic torque of the ankle plantar and dorsiflexors was determined using the Cybex 6000 isokinetic dynamometer (Cybex, NY, USA). Subjects were secured in a reclined position with the knee angled at 90° flexion (Figure 17) in accordance with the manufacturers users guide, with torque measured throughout the full range of ankle movement for isokinetic strength. For testing involving plantar and dorsiflexion, the neutral position (or 0° position) refers to that angle when the superior surface of the foot is at 90° to the shin. The degree of plantar or dorsiflexion is then taken from this point (Figure 16).

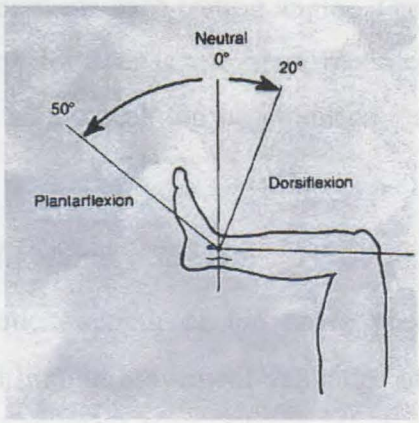


Figure 16. Neutral position and range of movements for the ankle.

From: Lumex. (1991). *Cybex 6000 extremity testing and rehabilitation system user guide*. Ronkonkoma, New York : Author.

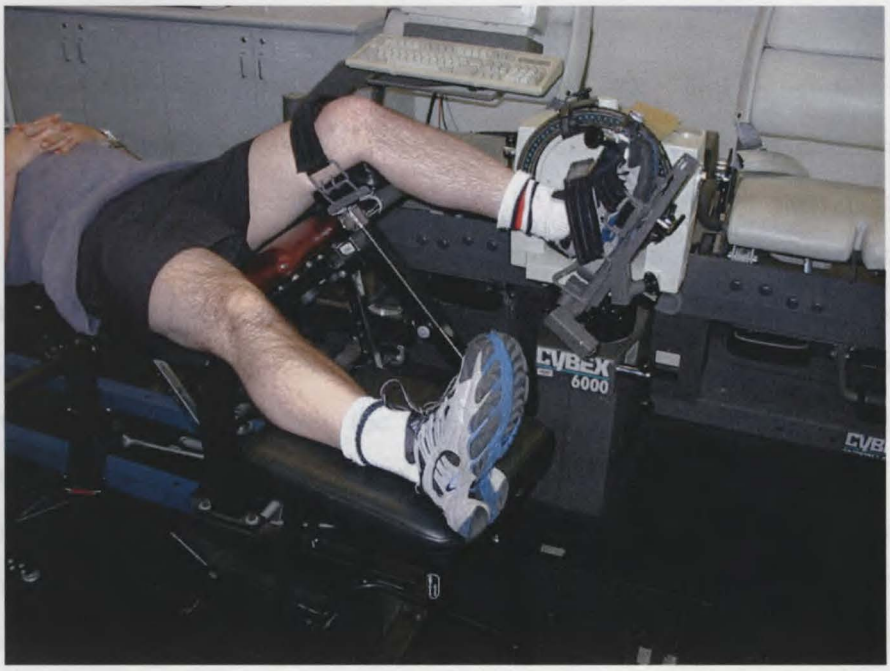


Figure 17. Subject positioning for strength testing on the Cybex of the plantarflexors and dorsiflexors with the knee secured at 90° flexion.

4.2.6.1 *Isometric Maximal Voluntary Strength*

Maximal voluntary isometric strength of the ankle plantarflexors and dorsiflexors was measured at three angles for the exercised limb. Dorsiflexion strength was measured as a control variable to ensure the protocol produced changes in the plantarflexors only. Three sets of three 5-second maximal efforts with the foot in a neutral (0°), dorsiflexed and plantarflexed (10°) position were performed with 20 seconds rest between each effort, and 60 seconds rest between each testing angle. The peak torques from the best of the three maximal efforts for each angle were recorded for analysis across time. Baseline values were taken as the peak torque produced during the 72 or 48 hour pre-exercise testing occasions.

4.2.6.2 *Isokinetic Maximal Voluntary Strength*

Maximal voluntary isokinetic strength of the ankle plantar and dorsiflexors were determined in the exercised limb at movement velocities of 30°, 60° and 120°/second. Each subject performed three maximal repetitions at each movement velocity with the peak torque value for each velocity recorded for data analysis. A rest interval of 60 seconds was allowed between each testing velocity with the presentation of the sets always ordered 60°, 120° then 30°/second. The peak value from the 72 and 48 hours pre-exercise values were taken as baseline.

4.2.7 *Data Analysis*

Dependent variables (soreness, tenderness, plasma CK, and maximal voluntary isometric and isokinetic torques) were analysed using a repeated measures (time) ANOVA. Where a significant p value ($p<0.05$) was obtained, post hoc analysis consisted of a simple contrast (first) to determine those time intervals that were significantly different from baseline. Analysis of muscle soreness and tenderness across time was conducted by calculation of a mean soreness/tenderness score for the five testing sites. Differences between the five sites for determination of a patterning of soreness/tenderness tested using paired sample t-tests with a Bonferroni adjusted significance level of $p<0.0083$. Gender differences in each dependent variable were assessed using a t-test at each time interval with significance set at $p<0.05$. To assess the existence of relationships between the testing variables a Pearson Product moment correlation coefficient was calculated. Results for reliability of all testing protocol are reported separately in Chapter 8.

4.3 Results

4.3.1 *Exercise Data*

The 6 subjects monitored for heart rate during the exercise protocol ranged in age from 21 to 42 years, with both males (n=4) and females (n=2) tested, and sedentary and recreationally active subjects (as indicated by pre-test health screening) represented. The mean heart rates for these individuals at rest before the start of exercise, and following 5, 30 and 60 minutes of walking were 69.8 ± 2.5, 82.0 ± 3.3, 88.2 ± 2.8, and 88.3 ± 4.2 bpm respectively (mean $\pm$ SEM). The greatest variation recorded in heart rate from rest to end of exercise being 24 bpm.

4.3.2 *Creatine Kinase*

Significant increases in plasma CK occurred at all time intervals from 0.5 to 96 hours post-walk with the mean value peaking 96 hours post-walk at 504 IU (Figure 18). Subjects demonstrating a CK response (n=12; CK peak > 200 IU) showed significant increases ($p<0.05$) from baseline at all time intervals 24 to 96 hours post-walk with a mean CK peak of 763 IU at 96 hours. In contrast, CK non responders (n=8; CK peak < 200 IU) showed a significant increase from baseline at 24 and 96 hours post-walk only with a mean CK of 139 and 114 IU respectively. No gender bias was observed for the low CK responders, while 7 of the high CK responders were males compared with 5 females. A significant difference between the genders for CK was observed 0.5 and 24

hours post-walk (Figure 19), with males recording higher CK responses compared with females at all time intervals with the exception of 96 hours post-walk at which time the female participants recorded their peak mean CK response.

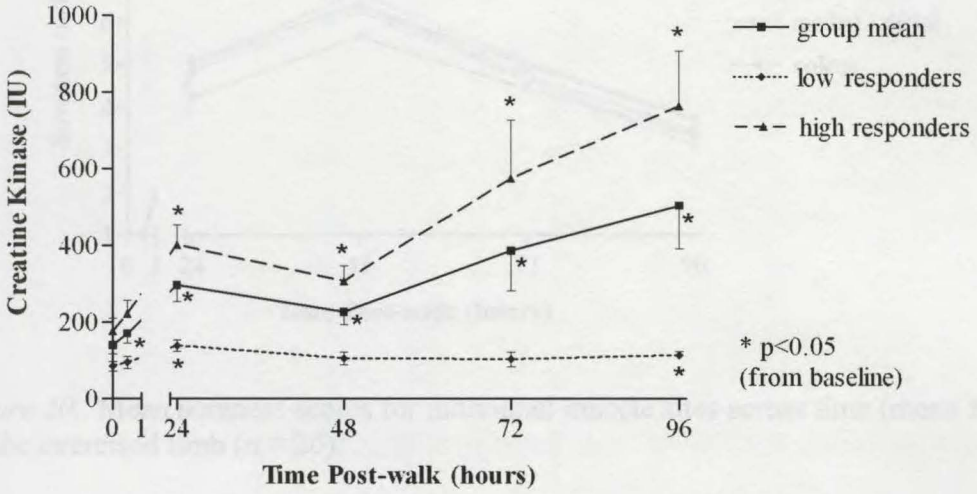


Figure 18. Plasma CK activity (n = 20) across time (mean ± SEM) for the group (n=20), CK non-responders (n=8) and CK responders (n = 12).

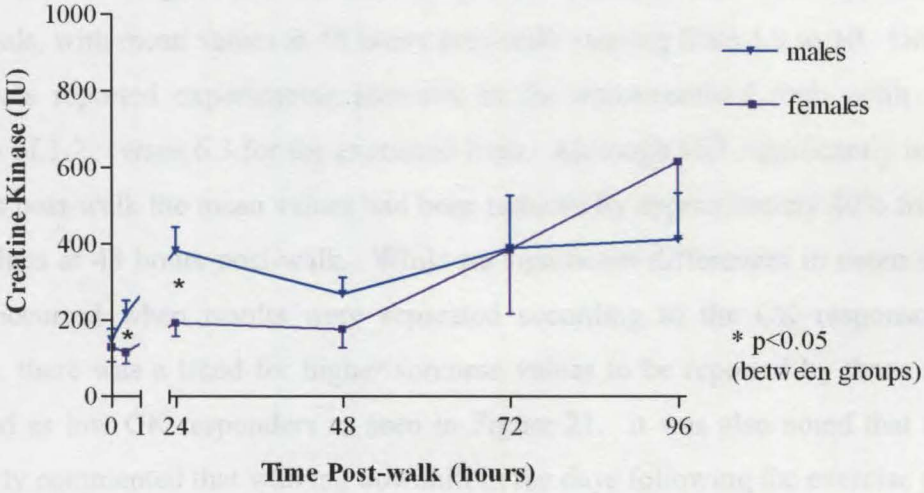


Figure 19. Plasma CK activity across time (mean ± SEM) for the male (n = 12) and female (n = 8) participants.

4.3.3 *Soreness and Tenderness*

Similar soreness levels were reported across the limb sites tested for each of the post-walk time intervals, with no significant differences between any of the sites recorded post-walk. There was however a tendency for the values reported for the three distal sites to be higher than those of the two proximal sites (Figure 20).

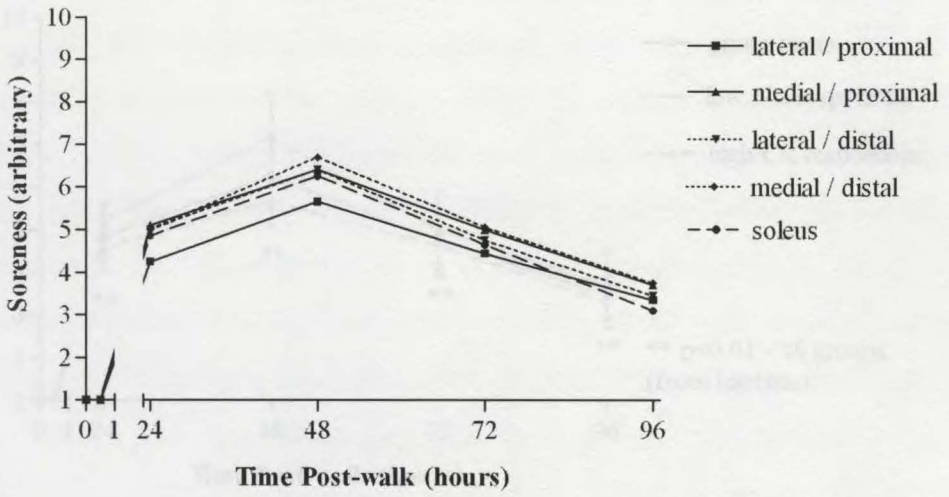


Figure 20. Mean soreness scores for individual muscle sites across time (mean ± SEM) for the exercised limb (n = 20).

Significant increases ($p<0.01$) in site mean soreness were recorded at all time intervals post-walk with the exception of 0.5 hours, with the highest site mean occurring at 48 hours post-walk (Figure 21). Soreness of the exercised limb was reported by all individuals, with mean values at 48 hours post-walk ranging from 1.9 to 10. Only three individuals reported experiencing soreness in the non-exercised limb, with a mean soreness of 1.2, versus 6.3 for the exercised limb. Although still significantly increased 96 hours post-walk the mean values had been reduced by approximately 40% from their peak values at 48 hours post-walk. While no significant differences in mean soreness scores occurred when results were separated according to the CK response of the subjects, there was a trend for higher soreness values to be reported by those subjects classified as low CK responders as seen in Figure 21. It was also noted that subjects frequently commented that walking downhill in the days following the exercise protocol was particularly painful, and that while some pain was present during the strength tasks, all felt that it was not a factor limiting their ability to perform a maximal voluntary contraction.

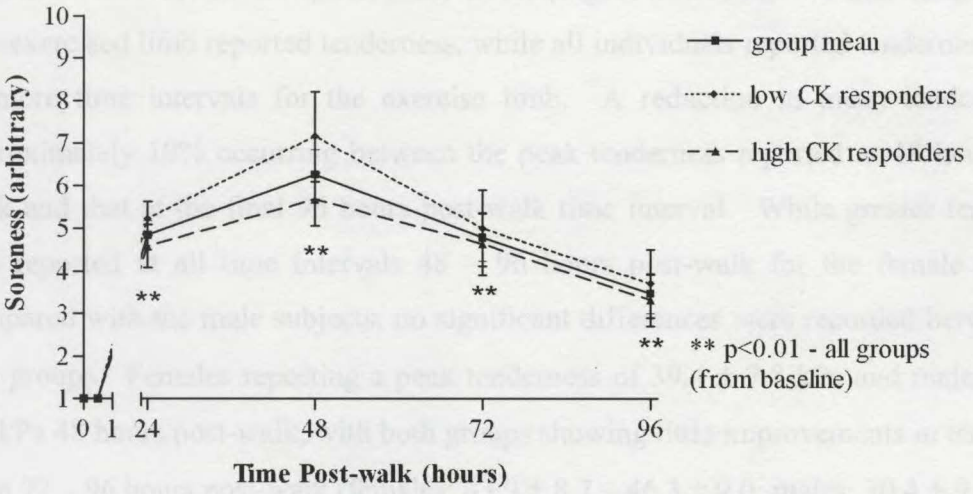


Figure 21. Mean soreness values recorded across time for the exercised limb (mean ± SEM) of the group (n = 20), CK non-responders (n = 8) and CK responders (n = 12).

A significant gender response was observed for soreness reported following the exercise protocol, with females reporting greater soreness at all time intervals 24-96 hours post-walk (Figure 22). The soreness reported by the females approximately 2 points higher on the 1 – 10 pain scale at each of these time intervals when compared with the males, with the two groups reporting a peak (mean ± SEM) soreness of 5.2 ± 07 (males) and 7.6 ± 2.2 (females) at the 48 hour post-walk time interval.

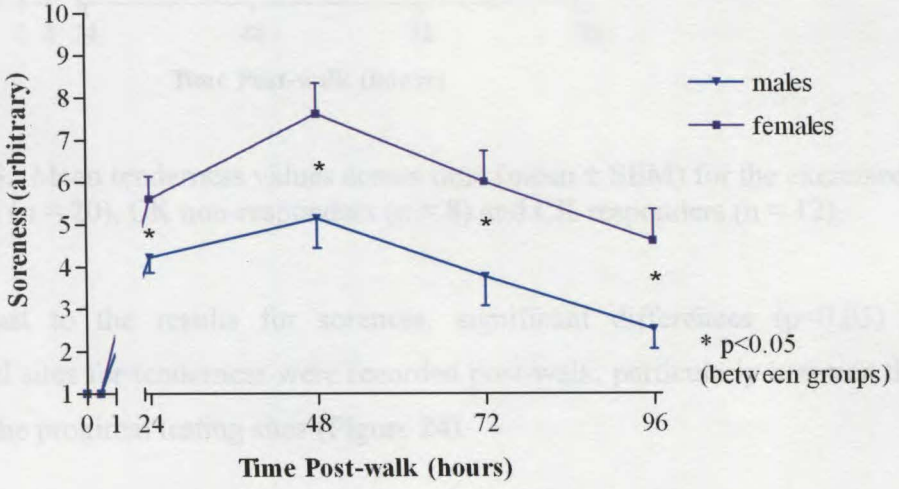


Figure 22. Mean soreness scores across time (mean ± SEM) for the male (n = 11) and female (n = 9) participants.

As with soreness, the greatest values for mean tenderness in the exercised limb were recorded 48 hours post-walk with significant increases ($p<0.01$) recorded at all post-

walk intervals with the exception of 0.5 hours (Figure 23). Only two individuals for the non-exercised limb reported tenderness, while all individuals reported tenderness at one or more time intervals for the exercise limb. A reduction in mean tenderness of approximately 10% occurring between the peak tenderness reported at 48 hours post-walk and that at the final 96 hours post-walk time interval. While greater tenderness was reported at all time intervals 48 – 96 hours post-walk for the female subjects compared with the male subjects, no significant differences were recorded between the two groups. Females reporting a peak tenderness of 39.4 ± 7.8 kPa and males 62.6 ± 9.2 kPa 48 hours post-walk, with both groups showing little improvements in tenderness from 72 – 96 hours post-walk (females: 43.9 ± 8.7 – 46.3 ± 9.0; males: 70.4 ± 9.1 – 71.6 ± 9.6). As with soreness no differences were noted for tenderness between groups when subjects were separated into CK responders and non-responders.

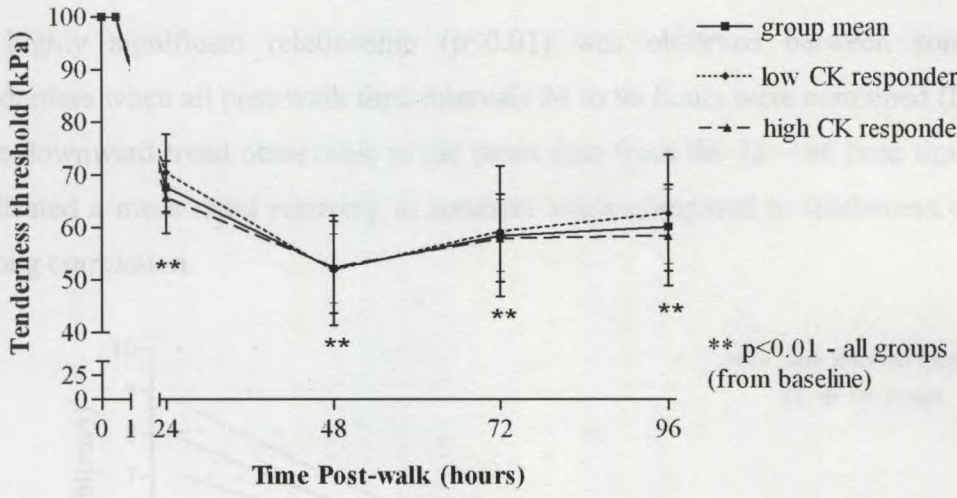


Figure 23. Mean tenderness values across time (mean ± SEM) for the exercised limb of the group (n = 20), CK non-responders (n = 8) and CK responders (n = 12).

In contrast to the results for soreness, significant differences ($p<0.05$) between individual sites for tenderness were recorded post-walk, particularly between the soleus site and the proximal testing sites (Figure 24).

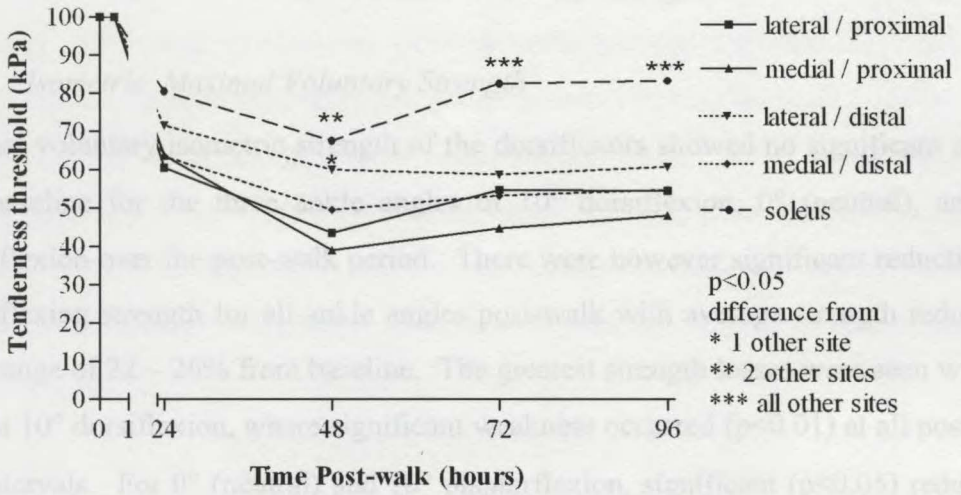


Figure 24. Mean tenderness scores for individual muscle sites across time for the exercised limb (n=20).

A highly significant relationship ($p<0.01$) was observed between soreness and tenderness when all post-walk time intervals 24 to 96 hours were combined (Figure 25). The downward trend observable in the mean data from the 48 – 96 hour time intervals indicated a more rapid recovery in soreness when compared to tenderness despite the strong correlation.

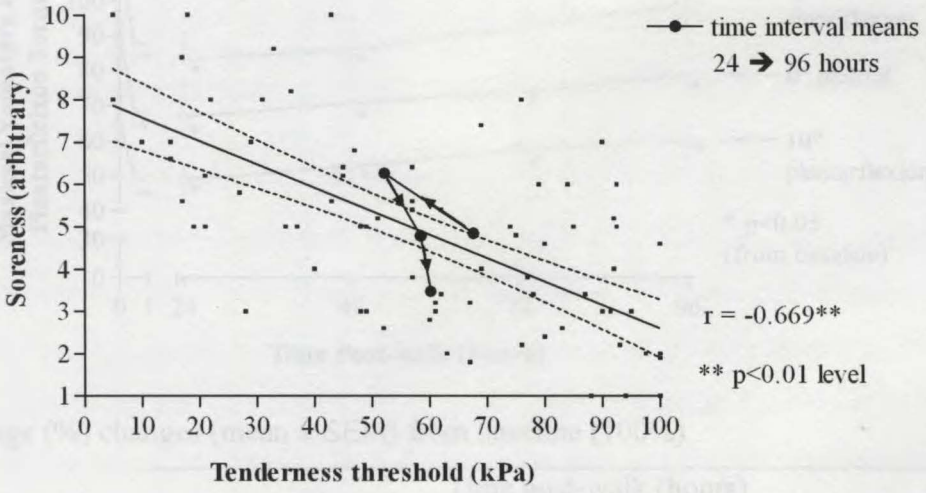
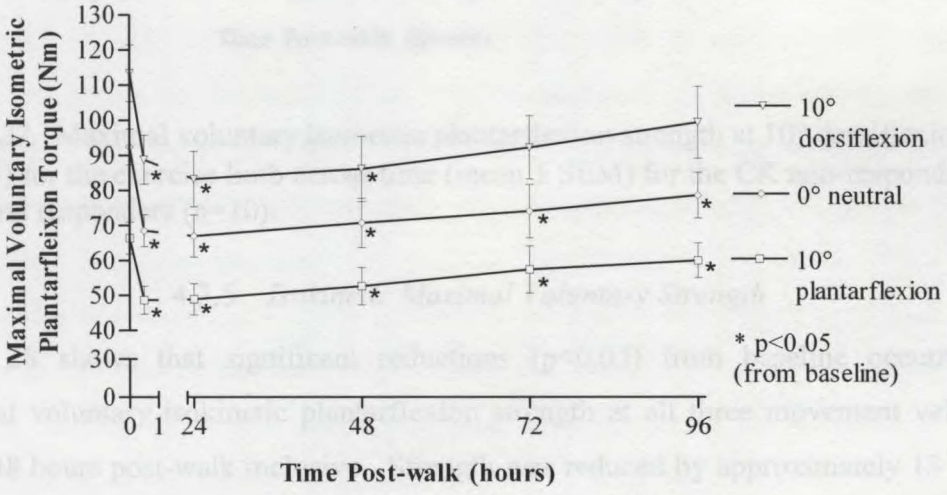


Figure 25. Relationship between soreness and tenderness of the exercised limb for all time intervals 24 to 96 hours post-walk inclusive (n=80).

4.3.4 *Maximal Voluntary Strength*

4.3.4.1 *Isometric Maximal Voluntary Strength*

Maximal voluntary isometric strength of the dorsiflexors showed no significant change from baseline for the three ankle angles of 10° dorsiflexion, 0° (neutral), and 10° plantarflexion over the post-walk period. There were however significant reductions in plantarflexion strength for all ankle angles post-walk with average strength reductions in the range of 22 – 26% from baseline. The greatest strength losses were seen with the ankle at 10° dorsiflexion, where significant weakness occurred ($p<0.01$) at all post-walk time intervals. For 0° (neutral) and 10° plantarflexion, significant ($p<0.05$) reductions from baseline were recorded at 0.5 to 96 hours, and 0.5 to 24 hours post-walk respectively (Figure 26). A relatively linear recovery in strength at each ankle angle occurring between the 24 and 96-hour time intervals. None of the angles tested for isometric strength had recovered to baseline values by the final testing time interval (96 hours post-walk), with mean reductions of 9-15% still recorded. Gender comparisons showed no significant differences in the percentage strength loss post-walk.



Percentage (%) changes (mean ± SEM) from baseline (100%)

	Time post-walk (hours)				
	0.5	24	48	72	96
10° dorsiflexion	78.3±4.4	73.4±7.4	76.1±5.0	81.4±4.6	87.6±4.82
0° neutral	73.9±6.1	72.8±7.6	76.1±5.2	80.4±3.8	84.8±3.4
10° plantarflexion	72.7±6.1	73.5±7.6	78.8±5.7	86.3±5.3	90.9±4.8

Figure 26. Maximal voluntary isometric plantarflexion strength at 10° dorsiflexion, 0° (neutral) and 10° plantarflexion for the exercised limb (mean ± SEM) across time (n = 15).

Significant differences ($p<0.05$) in isometric strength loss (% baseline) were seen between the CK responders (n=10) and non-responders (n=5) post-walk with the ankle positioned at 10° dorsiflexion and 0° neutral (Figure 27). The CK non-responders showing greater strength deficits across all time intervals, with recovery to baseline occurring in the CK responders and not the non-responders. A similar trend was observed with the limb positioned at 10° plantarflexion, although these differences were not significant.

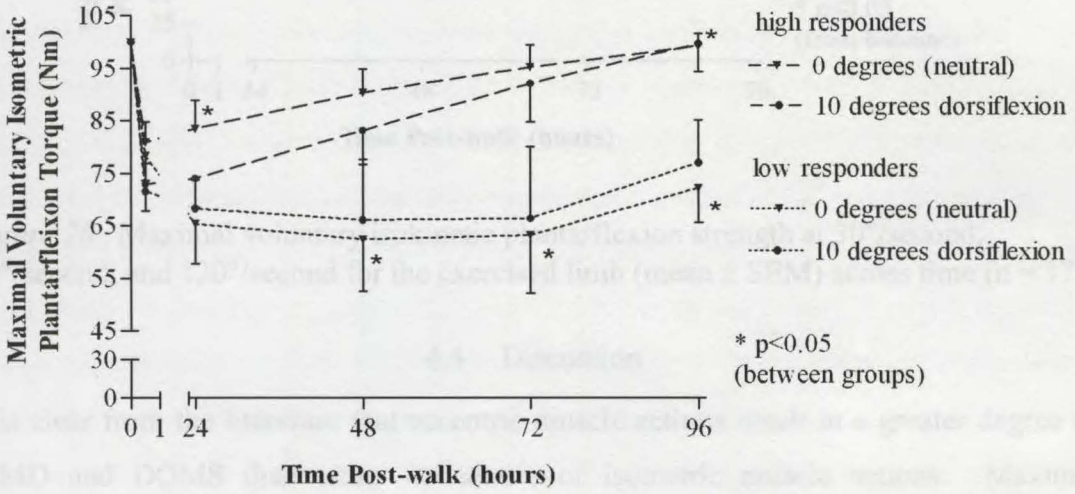


Figure 27. Maximal voluntary isometric plantarflexion strength at 10° dorsiflexion, 0° (neutral) for the exercise limb across time (mean ± SEM) for the CK non-responders (n=5) and responders (n=10).

4.3.5 *Isokinetic Maximal Voluntary Strength*

Figure 28 shows that significant reductions ($p<0.05$) from baseline occurred for maximal voluntary isokinetic plantarflexion strength at all three movement velocities 0.5 to 48 hours post-walk inclusive. Strength was reduced by approximately 13 – 15% for each movement velocity with a deficit of 8% still evident at the slower velocity of 30°/second 96 hours post-walk. In contrast, there was a trend for maximal voluntary isokinetic dorsiflexion strength to be increased from baseline post-walk, with strength gains of between 7% and 13% recorded at 60°/second and 120°/second. Smaller gains occurring at 30°/second (approximately 3%). As with isometric strength, no gender differences were recorded for any of the isokinetic testing velocities in relation to the percentage strength lost post-walk, with no obvious trends between the genders in the degree of strength lost. Nor were any differences seen in percentage strength loss post-walk when individuals were grouped according to their CK response. While non-

responders tended to show greater strength losses, with an increased recovery time, these variations were not significantly different between the two groups.

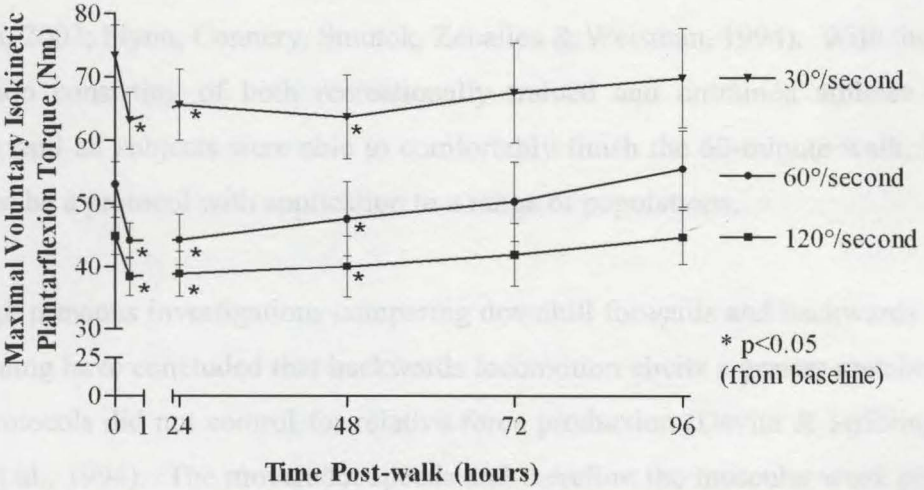


Figure 28. Maximal voluntary isokinetic plantarflexion strength at 30°/second, 60°/second, and 120°/second for the exercised limb (mean ± SEM) across time (n = 17).

4.4 Discussion

It is clear from the literature that eccentric muscle actions result in a greater degree of EIMD and DOMS than either concentric or isometric muscle actions. Maximal eccentric contractions are most often used in experiments investigating these two phenomena. The degree to which maximal effort and/or maximal voluntary eccentric contractions are involved in both sporting and non-sporting settings however is not well documented. It was therefore the purpose of this investigation to examine the effectiveness of a downhill walking protocol to submaximally eccentrically load triceps surae, with the aim of inducing both EIMD and DOMS. The degree to which the eccentric loading of triceps surae consisted of submaximal or maximal eccentric contractions however can only be speculated.

As it has been reported that the metabolic cost of muscle contraction is proportional to the magnitude and frequency of the external force produced (Sih & Stuhmiller, 2003), it is unlikely that the present exercise protocol involved maximal eccentric actions. It has been demonstrated that the oxygen consumption for positive work is greater than negative work for the same muscles exerting the same force (Abbott, Bigland & Ritchie, 1952), and that a direct relationship exists between workload, heart rate and oxygen consumption (Byrne & Hills, 2002). As the heart rates during the walking protocol showed the exercise to be a submaximal effort, it is suggested that the contractions

produced were submaximal in nature. Had the exercise consisted of maximal effort it would be expected that a greater increase in the exercise heart rate would have been observed due to increases in the energetic cost of the exercise (Derrick, Dereu & McLean, 2002; Flynn, Connery, Smutok, Zeballos & Weisman, 1994). With the subject population consisting of both recreationally trained and untrained athletes of both genders, and all subjects were able to comfortably finish the 60-minute walk, it would appear to be a protocol with application to a range of populations.

Although previous investigations comparing downhill forwards and backwards walking and running have concluded that backwards locomotion elicits a greater metabolic cost, these protocols did not control for relative force production (Devita & Stribling, 1991; Flynn et al., 1994). The movement speeds and therefore the muscular work performed during the backwards locomotion were far greater than that in the present investigation, 4 mph and 6 mph compared with 1.5 mph. Using a running speed of 6.71 mph, Devita et al (1991) reported that peak ankle plantarflexion torque during backwards running was approximately 50% that of forwards running, with a lower net energy generation of approximately 30%. From this it can only be suggested that backwards walking at 1.5 mph would have a lower peak torque and lower energy generation than forwards walking at the same speed, and would therefore consist of relatively mild submaximal muscular contractions. A biomechanical analysis of the joint kinetics during the exercise protocol would be necessary to determine the exact loading of triceps surae during downhill backward walking at the given speed and declination.

4.4.1 *Plasma Creatine Kinase*

Similar to previous investigations (Newham, Jones et al., 1983; Shumate, Brooke, Carroll & Davis, 1979), a large inter-subject variability was seen in CK efflux following the eccentric exercise protocol, with subjects being clearly defined as either low or high CK responders. Whilst the reason for this variation is not known, it has been shown that it is unlikely the result of differences in the release of CK inhibitors in the sera from subjects following eccentric exercise (Clarkson & Ebbeling, 1988). A dialysable inhibitor of CK has been observed in the sera in some individuals with muscle disease, resulting in a reduction in total CK activity of those patients with significant inhibition occurring in approximately 20% of the subjects (Kagen & Aram, 1987). These inhibitors however do not appear to be evident in individuals experiencing signs and symptoms of EIMD (Clarkson et al., 1988). Various authors (Roth et al., 2001; Tiidus,

2003) have also suggested that the CK efflux may be reduced in females due to a protective effect of estrogen, with elevated estrogen acting to maintain muscle cell membrane stability and therefore reduce CK leakage (Kendall & Eston, 2002; Tiidus, 2000). While the current results did demonstrate a gender difference in CK efflux in the period following the exercise protocol consistent with such an idea, the non-responders group consisted of an even number of males and females suggesting that factors other than gender must also influence CK efflux following eccentric exercise. Additionally, although testing was not scheduled in the female subjects according to their menstrual cycle, it is known from the pre-participation medical questionnaires that 3 of the CK responders, and 2 of the non-responders were taking regular hormonal contraceptive medication at the time of testing. This further highlights that it unlikely that gender alone influenced the gender differences in CK efflux post-exercise reported in the present investigation.

Despite the large variability in subject responses, elevated CK activity remains a common indicator of exercise-induced muscle damage, with exercise modes that consist largely of high levels of eccentric loading typically resulting in a single peak CK efflux between 24 and 96 hours post-exercise (Paddon-Jones et al., 2000; Nosaka, Clarkson & Apple, 1992). In contrast, eccentric exercise consisting of submaximal repetitions typically results in a bimodal response with the greatest CK peak occurring at 72 hours (or later) post-exercise (Schwane et al., 1983). The results of the present study support this finding with a bimodal response occurring following the submaximal eccentric loading of the downhill walking protocol. The reason for such a response however is not clear.

None of the previous studies using a downhill backward walking protocol investigated CK efflux following exercise. As the swelling measured in these two studies was not the primary focus of investigation, little explanation was offered as to the cause of the observation. Generally, the CK efflux reported in the present investigation was lower than those reported following maximal eccentric exercise of the biceps. Group means following eccentric exercise in excess of 4000 IU have been reported (Nosaka & Clarkson, 1995; Nosaka et al., 1992). In contrast, lower mean peaks in the range of 500 to 1000 IU have been reported for the quadriceps (Maughan et al., 1989; Sargeant & Dolan, 1987), with a mean peak of 1237 IU reported following submaximal eccentric exercise of the elbow flexors (Nosaka & Newton, 2002). While it appears that the

degree of efflux is related to the type and number of eccentric contractions being performed, the reasoning for the large differences in efflux between muscle groups is also not known (Nosaka et al., 2002). Further investigations across different muscle groups using the same subject population would be required to attempt to investigate the differences that occur between muscles as opposed to individual subject variations.

Analysis of the correlation between parameters of EIMD showed that, as in previous work, no relationships existed between plasma CK and soreness, or plasma CK and tenderness (Clarkson, Byrne, McCormick, Turcotte & White, 1986). Those subjects classified as CK non-responders, still demonstrated an increase in soreness and tenderness following the exercise protocol to a greater degree than the CK responders. Additionally, at those time intervals (0.5 and 24 hours post-walk) where CK efflux was significantly greater in the male subjects, either no soreness was reported, or females reported significantly greater soreness than males. Based on these results it appears likely that the mechanism for CK efflux differed from that responsible for the sensation of DOMS following the downhill backward walking protocol.

Although plasma CK efflux is the most commonly examined intracellular enzyme released following eccentric exercise, investigators have also examined the release of serum phosphokinase, glutamic oxaloacetic transaminase, lactate dehydrogenase, calcium and aspartate aminotransferase (Nosaka & Clarkson, 1996; Tiidus & Ianuzzo, 1983). Additionally, extensive investigations of the inflammatory response following eccentric exercise have generally concluded that inflammation has a major influence on the necrosis and soreness that occurs following eccentric exercise (MacIntyre, Reid & McKenzie, 1995; Warren, Ingalls, Shah & Armstrong, 1999). More recently, a relationship between the elevated myosin heavy chains in the presence of elevated serum CK following eccentric exercise and abnormal magnetic resonance imaging has been demonstrated (Nosaka & Clarkson, 1996; Sorichter et al., 2001). Investigation of leakage of intracellular compounds other than CK following eccentric exercise would appear to be a key factor in understanding the cause of soreness and tenderness following such exercise.

4.4.2 *Soreness and Tenderness*

Newham, Mills et al. (1983) investigated tenderness of the quadriceps muscles of subjects following repeated stepping. They reported greater tenderness values in the

distal, medial, and lateral portions of the exercised muscle compared to central and proximal regions. The localisation of the tenderness however was more diffuse when tenderness was at its peak intensity, being absent by 4 days post-exercise. Weerakkody et al. (2001) also reporting an uneven distribution of soreness following eccentric exercise. While soreness measures were not specifically included in the post-exercise testing or results, the authors reported that subjective soreness was noted by all subjects in the exercised quadriceps. The soreness being more uncomfortable while descending stairs or with the muscle contracting at a short length, compared to a mid length. While it is difficult to compare the present pattern of damage with that described by Newham et al. (1983) due to differing protocols and active muscle groups, the current results demonstrated greater soreness in proximal compared with distal sites, and medial rather than lateral sites, with both soreness and tenderness still present at significant levels 4 days post-walk. The tendency for greater soreness with contraction of the muscle at a short length compared with long lengths was also common between the two studies, with subjects in the present study also commenting on the heightened pain when walking downhill.

As muscular contraction of the lower limb with the knee in a straightened position employs predominantly the gastrocnemius muscle (Signorile, Applegate, Duque, Cole & Zink, 2002), it would seem reasonable that the walking protocol used in the present study would preferentially load gastrocnemius over soleus. This in turn may explain the greater tenderness experienced proximally, with little tenderness reported for soleus. A similar observation was also reported by Weerakkody et al. (2001) following backwards walking.

One of the more prominent findings of the current study was that soreness and tenderness, while commonly reported synonymously in the literature, might represent distinct phenomenon. While a significant relationship was observed between the two variables, the recovery of soreness following the exercise protocol appeared shorter (faster) than that of tenderness. Soreness scores 96 hours post-exercise were well below those reported 24 hours post-exercise, while tenderness levels were only slightly reduced from those reported 48 hours post-exercise at their peak levels. Furthermore, while no difference was recorded between the five muscle sites tested in relation to soreness, differences between sites were observed for tenderness post-walk. This suggests that while subjects may be able to localise tenderness to different intensities

within an exercise muscle, soreness scores may be more generalised and not specific to individual areas of the muscle. In order to accurately determine a patterning of damage following a particular eccentric exercise protocol, it is therefore advisable for testing protocols to evaluate both soreness and tenderness. Soreness representing pain during normal and otherwise pain free movements, and tenderness representing the pain experience when a muscle is touched or palpated at specific sites.

Pathophysiologically, pain can be classified as either 'slow' or 'fast' depending on the conduction rate of the fibre from which the stimuli is received, and can be elicited by mechanically, thermally and chemically originated stimuli (Kingsley, 2000, p. 152). Fast pain occurs due to stimulation of the small type Aδ myelinated fibres (6 – 20 m/second), elicited by either mechanical or thermal stimuli. Slow pain can be elicited by all three types of stimuli and is transmitted by the larger type C non-myelinated fibres (1 m/sec), the pain from which can occur after a noticeable delay (Carlsson & Pellettieri, 1982; Kerr, & Wilson, 1978). The pain associated with DOMS is thought to occur due to the sensitisation of the nociceptors by noxious intracellular substances released extracellular due to muscle fibre damage (Armstrong, 1984; Gulick & Kimura, 1996). Such stimuli would therefore be classified as a slow pain response occurring due to the presentation of chemical stimuli. However, as pain is absent at rest and occurs only upon muscle contraction or compression it would seem reasonable to conclude that the pain is also elicited by a mechanical stimulus and therefore a fast pain sensation.

Slow pain elicited along the paleospinothalamic pathway is considered to be poorly localised and can usually only be assigned to a general area of the body (Goodman, 1983). In relation to the current study, sensitisation of this slow pathway may have caused soreness ratings in the calf to be general and non-specific to the five different sites being tested. In contrast however, fast pain can often be localised to within 10 centimetres of a stimulated area when pain receptors are stimulated by muscle contraction, and localisation is nearly exact when pain and tactile receptors exciting the dorsal column-medial lemniscal are stimulated (Guyton & Hall, 1996, p. 612). It is likely that muscle contraction caused by walking would result in more widely localised fast pain, while the tactile receptors excited by touch, pressure and vibration (Pritchard & Alloway, 1999, p. 208-210; Kingsley, 2000, p. 151-152), would be stimulated during the examination of tenderness. Once this occurs subjects are more accurately able to distinguish or localise pain, resulting in the observed differences in the five muscle sites

examined, and possibly resulting in the prolonged sensation of tenderness compared with soreness.

In response to experimentally induced noxious substances, females will generally report higher intensities of pain more frequently than males, however as pain is individual and subjective, sample sizes in excess of 40 subjects may be required to demonstrate true statistical significance (Riley III, Robinson, Wise, Myers & Fillingim, 1998). The literature specifically relating to gender differences in DOMS and tenderness following eccentric exercise is scarce and not fully in agreement with other pain studies. The present observation of no gender differences in tenderness supports the findings of Dannecker et al. (2003), who reported no significant sex differences in pressure threshold for pain over the biceps musculotendonous junction. The same authors however reporting that females rated DOMS significantly lower than the male participants using a 10 cm visual analogue scale with the anchored extremes of "no pain" and "worst pain possible". A similar study by Rindard et al. (2000) reported no significant differences between men and women for soreness upon palpation or lifting. The palpation component most alike a tenderness reading, while soreness upon lifting is similar to the soreness ratings in the current study and that used by Dannecker et al., (2003). The potential impact of sex hormones in modulation of pain has been largely inconclusive (Kendall & Eston, 2002), with the current study supporting typical pain studies, with females reporting significantly higher levels of DOMS post-walk.

4.4.3 *Maximal Voluntary Strength*

Declines in maximal voluntary strength are characteristic following eccentric exercise with the results of the current study demonstrating reductions in both maximal voluntary isometric and isokinetic strength following 60 minutes of downhill backward walking. Typically, the greatest declines in strength are observed immediately following completion of the exercise protocol, although greater reductions at 24 hours post-exercise, such as that observed during the current study, have previously been reported in the quadriceps (Sargeant & Dolan, 1987). The absence of gender differences in strength loss from the results is also in agreement with the previous findings of Rinard et al. (2000), who reported no significant sex differences in strength loss or recovery rate of the elbow flexors following a maximal eccentric exercise protocol. While gender differences have been noted in membrane damage following eccentric exercise (Kendall & Eston, 2002; Komulainen et al., 1999), it would appear

that this apparent protection is not transferred to strength losses post-exercise. This observation is consistent with the idea that fibre damage is not the sole reason for strength loss following eccentric exercise, with other potential mechanisms for the strength loss including fatigue and EC coupling disruption (Warren et al. 2001). The degrees to which these mechanisms may be responsible for the strength loss observed following downhill backward walking are discussed in further detail in Chapters 6 & 7.

Three previous investigations of EIMD have used a downhill backward walking protocol. Of these, one reported a reduction of approximately 30% in peak strength (N=8) following 60 minutes of walking (-13° and ~3.5km/hr) using a two limbed walking action (Whitehead et al., 1998). The treadmill speed was adjusted to allow for a stepping rate of approximately 30 paces per minute per leg, thus each leg completed approximately 1800 contractions during the protocol. A maximal voluntary strength reduction of approximately 40% post-walk for peak strength (N=13) was recorded by investigators using a 60 minute single limb protocol similar to that used in the current study (-13° and 2.2km/hr) however, subjects were weighted with an additional 5-10kg load (Whitehead et al., 2001). This protocol also allowing for approximately 1800 paces to be completed for the eccentrically exercised limb. Following a 2 hour single limb protocol (-13° and 1.3km/hr), Jones et al (1997) reported a reduction in peak strength of approximately 25%. The reductions of 20 – 25% (isometric) and 10 – 15% (isokinetic) reported in the current study are similar to the latter investigation. While the protocol completed by Jones (1997) was a 2-hour protocol, the treadmill speed was approximately half that of the present study, 1.3km/hr versus 2.4km/hr. Thus overall subjects would have completed a similar number of contractions throughout the course of the exercise (approximately 1800 paces).

Overall, the strength reductions reported for the triceps surae are similar to those for the elbow and knee flexors. It should be noted however, that in both the elbow flexors and knee extensors, protocols utilising submaximal contractions generally demonstrate strength losses of approximately 10% (Behm et al., 2001; Farr, et al., 2002). These declines are considerably smaller than the 30-40% deficit commonly reported following maximal contractions (Child et al., 1998; Nosaka & Sakamoto, 2001; Paddon-Jones et al., 2000; Tiidus & Shoemaker, 1995). While reporting greater strength losses following submaximal eccentric contractions (compared with those previously mentioned) of the biceps (approximately 40%), Nosaka and Newton (2002) also

demonstrated greater strength loss in the same subjects following maximal eccentric exercise (approximately 50%). Future investigation comparing strength losses across muscle groups in the same subjects may increase the understanding of the degree to which contraction intensity, muscle fibre composition and muscle structural differences influences strength loss and other variables following eccentric exercise.

4.5 Conclusions

Following an eccentrically biased downhill backward walking protocol, the presence of significant ratings in DOMS and muscle tenderness, significant increases in plasma creatine kinase, and significant decreases in maximal voluntary strength indicate the presence of EIMD in triceps surae post-exercise. These changes being consistent in degree and time course, with previous investigations of EIMD for triceps surae and other muscle groups. Therefore, it was concluded that such a protocol is an effective model for EIMD of triceps surae.

CHAPTER 5 THE PROTECTIVE RESPONSES IN TRICEPS SURAE TO A REPEAT BOUT OF DOWNHILL BACKWARD WALKING

5.1 Introduction

In the previous chapter, it was demonstrated that a downhill backward walking protocol was an effective model for EIMD in triceps surae. The model producing results typical of those seen with EIMD in muscle groups such as the biceps and quadriceps. Downhill backward walking thus proved to be an effective model for EIMD, as it incorporates a more natural submaximal contraction condition compared with the more artificial models involving repeated maximal contractions. However, the extent to which this model is effective in providing protection against damage in accordance with the repeated bout effect is unknown.

The repeated bout effect refers to the rapid adaptation which occurs when the same or similar eccentric exercise is repeated following an initial exercise bout (Newham et al., 1987). This adaptation results in substantially reduced, and in some instances, absent symptoms of soreness and plasma CK efflux, and a more rapid recovery of strength loss (Rowlands, Eston & Tilzey, 2001). While EIMD and the repeated bout effect are both widely studied and published phenomena, the elbow flexors and knee extensors are by far the most commonly examined models, typically using maximal contractions as opposed to submaximal contractions. A review of the literature revealed no previous studies examining responses of a repeated bout of eccentric exercise on triceps surae.

As future studies were premeditated to examine the protective effect of repeated eccentric exercise on muscle activation and proprioceptive responses of the triceps surae, it was first necessary to demonstrate the existence of the repeated bout effect in this muscle group for the commonly accepted indicators of EIMD. Therefore, the aim of the current investigation was to establish if the common indicators of EIMD in triceps surae (soreness, tenderness, plasma CK and maximal voluntary strength) demonstrate a repeated bout effect following a second bout of downhill backward walking.

5.2 Methods

5.2.1 *Subjects*

A total of 17 subjects (9 females and 8 males) were recruited for the study with all subjects completing the full testing schedule for soreness, tenderness and plasma CK. Twelve subjects completed all time intervals for maximal voluntary isometric and isokinetic strength. Subject details are reported in Table 7.

Table 7
Subject Characteristics for Age, Height and Weight

	Mean ± SEM		
	Group (N=17)	Females (n=9)	Males (n=8)
Age (years)	26.6 ± 1.6	24.0 ± 1.6	29.6 ± 2.5
Height (cm)	169.6 ± 2.2	165.3 ± 2.5	174.4 ± 2.9
Weight (kg)	72.2 ± 3.5	68.7 ± 4.3	76.3 ± 5.6

5.2.2 *Testing Schedule*

Criterion measures were tested on 13 occasions over a 26-day period with an exercise protocol performed twice by each subject. Criterion testing was conducted 72 and 48 hours pre-walk, and 0.5, 24, 48, 72 and 96 hours post-walk for the initial exercise bout (bout 1). For the repeated bout, testing occurred 48 hours pre-walk, and 0.5, 24, 48, 72 and 96 hours post-walk (bout 2). A period of 7 days elapsed between the completion of testing for the initial exercise bout and baseline testing for commencement of the repeat exercise bout. This inter – bout period was chosen to allow for recovery of all measures back to baseline prior to the second bout. All exercise and criterion testing protocols and procedures were carried out as previously outlined in Chapter 4, for measures of plasma CK, soreness, tenderness, and maximal voluntary isometric and isokinetic strength.

5.2.3 *Data Analysis*

Each dependent variable was statistically analysed using a two-way (limb) repeated measures (time) ANOVA. Where a significant p value ($p<0.05$) was obtained, simple contrasts to baseline were conducted to determine those time intervals that were significantly different from baseline for each individual bout. To determine any significant differences between bouts, paired sample t-tests with a Bonferroni adjusted significance level ($p<0.0083$) were conducted between time intervals for the two

exercise bouts. Differences in the gender responses were assessed using a t-test at each testing time interval with significance set at $p<0.05$. Any relationships between variables was determined by calculation of Pearson product moment correlation coefficients with a significant correlation level of $p<0.05$. All values are reported as the mean ± SEM.

5.3 Results

5.3.1 *Creatine Kinase (CK)*

Significant differences from baseline for serum CK were recorded following only the initial exercise bout at all time intervals 24 to 96 hours post-walk inclusive (Figure 29). Reduced responses in the second exercise bout resulted in a significant difference between the two exercise bouts at 24 and 96 hours post-walk with values of 300.9 ± 46.5 and 497.6 ± 115.8 IU (bout 1), and 176.9 ± 28.6 and 135.5 ± 20.6 IU respectively (bout 2). As with the previous study (Chapter 4), subjects could clearly be identified as either responders (CK peak < 200 IU) or non-responders (CK peak > 200 IU), with the responders consisting of 5 females and 6 males, and the non-responders 4 females and 2 males. Following the repeat exercise bout however, all subjects demonstrated a low CK response (non-responders) with the exception of 2 males and 1 female, who could still be classified as responders. All three subjects however demonstrated a blunted CK response following the repeat exercise bout with peak responses of 405, 605 and 1626 IU reduced to 360, 415 and 285 IU respectively.

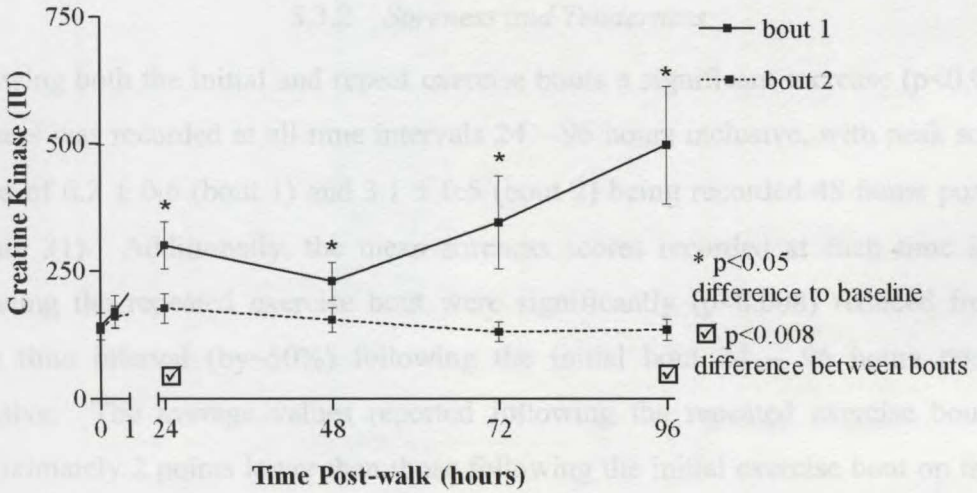


Figure 29. Plasma CK (n = 17) responses across time (mean ± SEM) following two separate exercise bouts.

Gender differences in the CK responses of the subjects were seen following both exercise bouts, with male subjects typically showing a higher CK response post-walk compared with females (Figure 30). Following the initial exercise bout, significant differences between the two groups were seen at baseline, and 24 hours post-walk, with mean responses of 205.9 ± 43.2 and 422.1 ± 70.1 IU for males and 85.1 ± 16.0 and 193.1 ± 35.6 IU for females. Following the repeat exercise bout gender differences were recorded at 0.5 and 24 hours post-walk with the male and female groups recording 215.2 ± 32.6, 238.8 ± 48.7, 112.6 ± 14.5 and 121.9 ± 21.0 IU respectively at these time intervals.

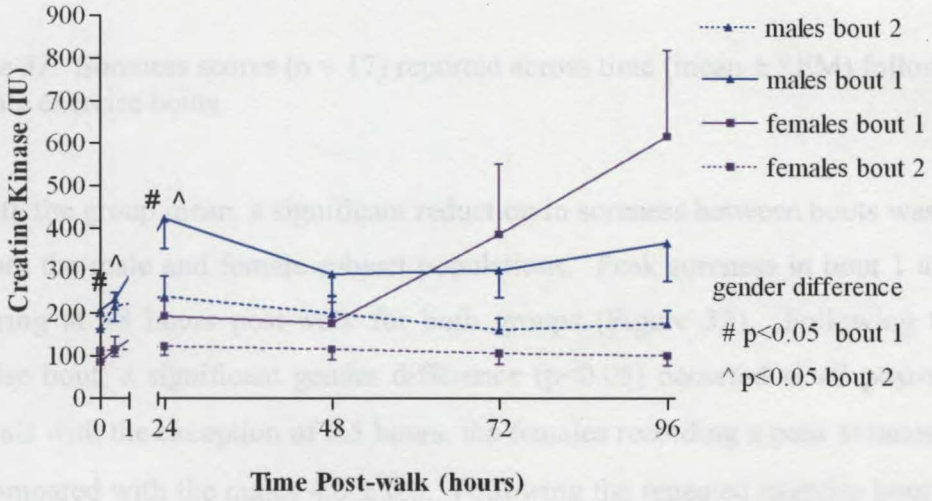


Figure 30. Plasma CK responses across time (mean ± SEM) following two separate exercise bouts for the male (n = 8) and female (n = 9) subject populations.

5.3.2 *Soreness and Tenderness*

Following both the initial and repeat exercise bouts a significant increase ($p<0.001$) in soreness was recorded at all time intervals 24 – 96 hours inclusive, with peak soreness scores of 6.2 ± 0.6 (bout 1) and 3.1 ± 0.5 (bout 2) being recorded 48 hours post-walk (Figure 31). Additionally, the mean soreness scores recorded at each time interval following the repeated exercise bout were significantly ($p<0.008$) reduced from the same time interval (by~50%) following the initial bout 24 – 96 hours post-walk inclusive. The average values reported following the repeated exercise bout were approximately 2 points lower than those following the initial exercise bout on the 1-10 scale. The same trend for a protective effect between exercise bouts occurred for each of the 5 individual sites tested for soreness. Soreness was significantly ($p<0.008$) reduced 24 – 96 hours post-exercise for all sites with the exception of lateral / proximal (24 hours post-exercise only) and soleus (24 – 72 hours post-exercise).

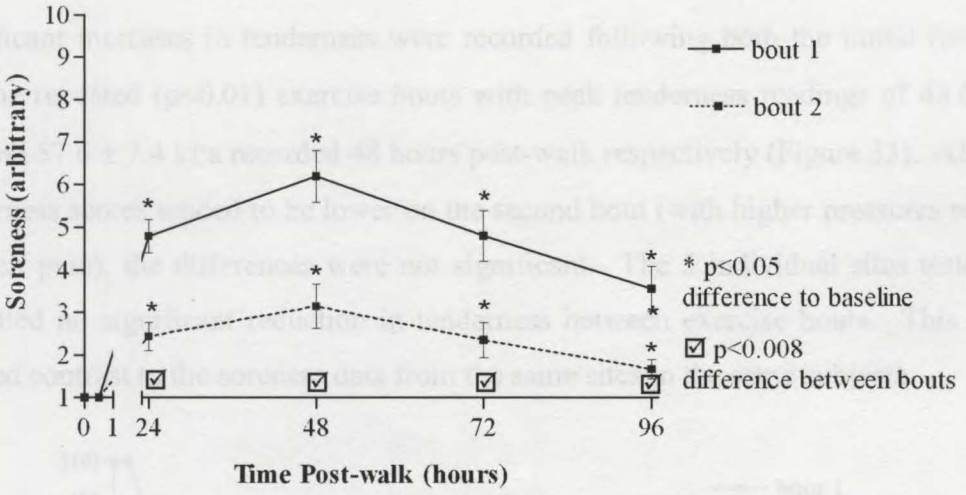


Figure 31. Soreness scores (n = 17) reported across time (mean ± SEM) following two separate exercise bouts.

As with the group mean, a significant reduction in soreness between bouts was recorded for both the male and female subject populations. Peak soreness in bout 1 and bout 2 occurring at 48 hours post-walk for both groups (Figure 32). Following the initial exercise bout, a significant gender difference ($p<0.05$) occurred at all post-walk time intervals with the exception of 0.5 hours, the females recording a peak soreness of 7.6 ± 0.7 compared with the males 4.6 ± 0.6. Following the repeated exercise bout however, a significant gender difference was recorded only for the 24-hour post-walk time interval, although the mean soreness scores for the male subjects were always lower than those of the females.

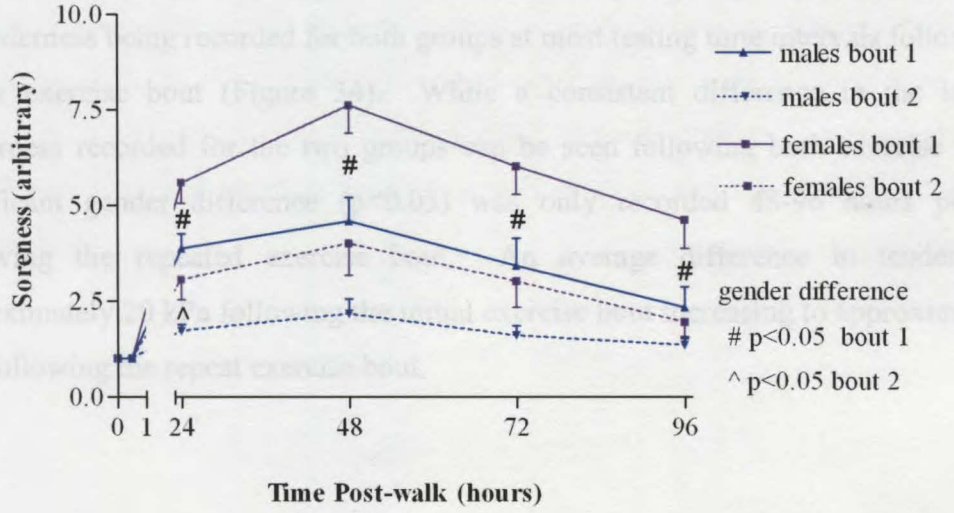


Figure 32. Soreness scores reported across time (mean ± SEM) following two separate exercise bouts for the male (n = 8) and female (n = 9) subject populations.

Significant increases in tenderness were recorded following both the initial ($p<0.001$) and the repeated ($p<0.01$) exercise bouts with peak tenderness readings of 48.0 ± 7.0 kPa and 57.6 ± 7.4 kPa recorded 48 hours post-walk respectively (Figure 33). Although tenderness scores tended to be lower on the second bout (with higher pressures required to elicit pain), the differences were not significant. The 5 individual sites tested also exhibited no significant reduction in tenderness between exercise bouts. This was in marked contrast to the soreness data from the same sites in the same subjects.

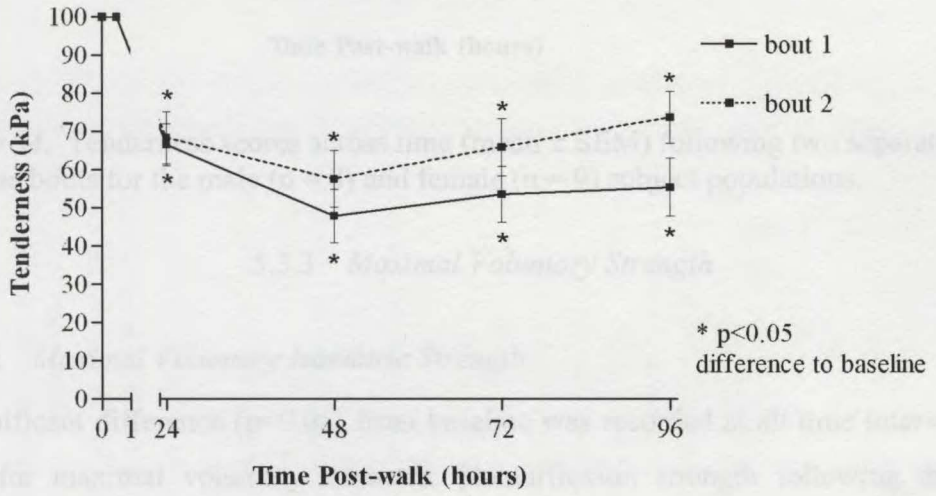


Figure 33. Mean tenderness (n = 17) reported across time (mean ± SEM) following two separate exercise bouts.

As with the group mean tenderness, no significant differences were recorded between bouts for tenderness of either the male or female subject groups, despite lower degrees of tenderness being recorded for both groups at most testing time intervals following the repeat exercise bout (Figure 34). While a consistent difference in the levels of tenderness recorded for the two groups can be seen following both exercise bouts, a significant gender difference ($p<0.05$) was only recorded 48-96 hours post-walk following the repeated exercise bout. An average difference in tenderness of approximately 20 kPa following the initial exercise bout increasing to approximately 30 kPa following the repeat exercise bout.

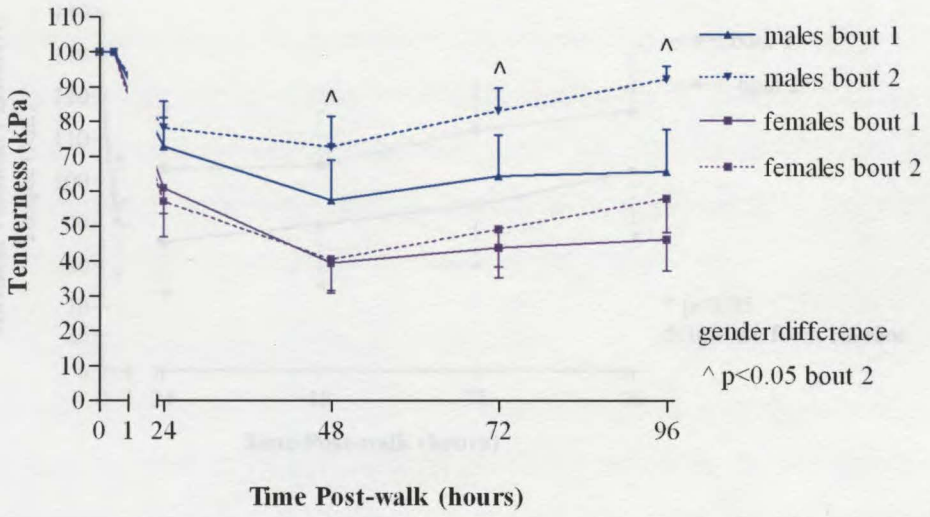


Figure 34. Tenderness scores across time (mean ± SEM) following two separate exercise bouts for the male (n = 8) and female (n = 9) subject populations.

5.3.3 *Maximal Voluntary Strength*

5.3.3.1 *Maximal Voluntary Isometric Strength*

A significant difference ($p<0.05$) from baseline was recorded at all time intervals post-walk for maximal voluntary isometric plantarflexion strength following the initial exercise bout at an angle of 10° dorsiflexion (Figure 35), and 0.5 and 24 hours post-walk for 0° (neutral) and 10° plantarflexion (Figures 36 and 37). An average strength reduction of approximately 25% was recorded across the three ankle angles with the greatest reductions recorded 24 hours post-walk for all testing angles. Following the repeated exercise bout, a significant difference ($p<0.05$) from baseline was recorded 0.5 and 24 hours post-walk for all testing angles, with an average strength reduction of approximately 23%. Unlike strength reductions following bout 1, the greatest strength losses following bout 2 were all recorded at the first post-walk testing time interval (0.5 hours) with a gradual return to baseline occurring thereafter over the 96 hour post-walk time period.

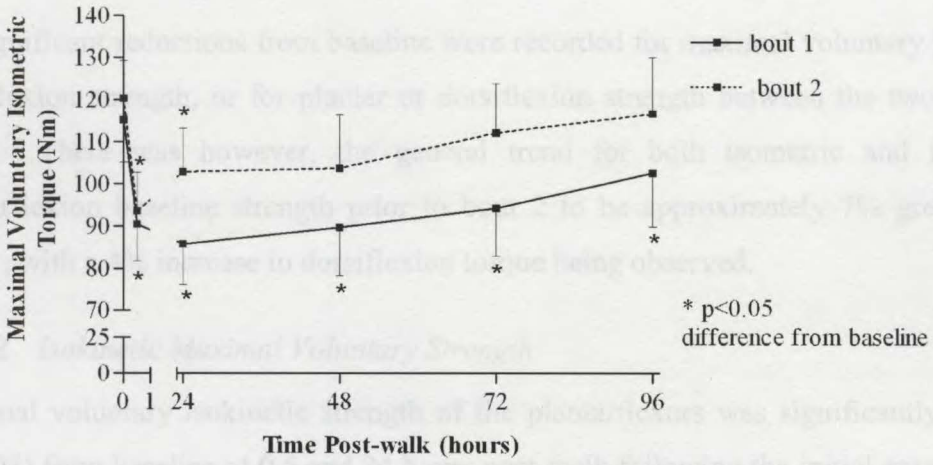


Figure 35. Maximal voluntary isometric plantarflexion strength (n = 12) at 10° dorsiflexion across time (mean ± SEM) following two separate exercise bouts.

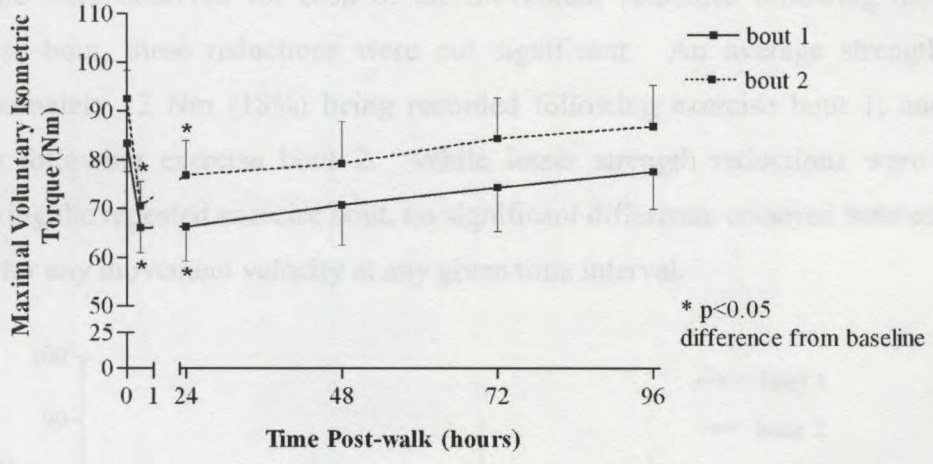


Figure 36. Maximal voluntary isometric plantarflexion strength (n = 12) at 0° (neutral) across time (mean ± SEM) following two separate exercise bouts.

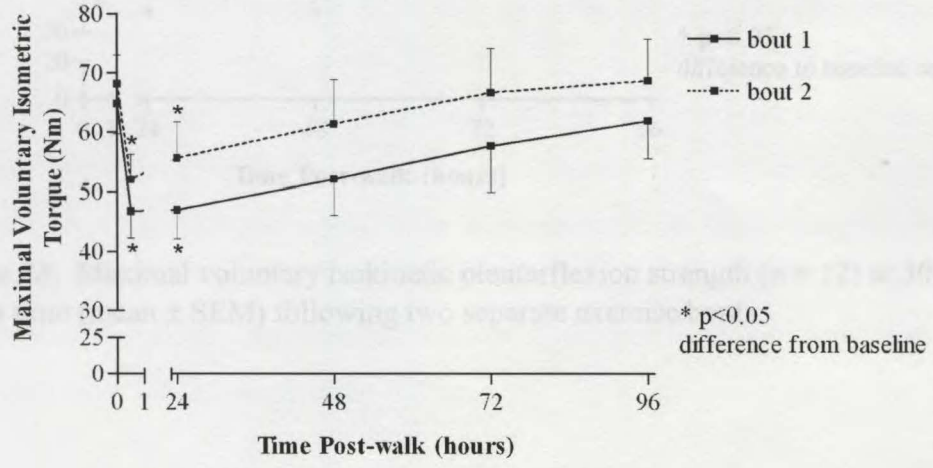


Figure 37. Maximal voluntary isometric plantarflexion strength (n = 12) at 10° plantarflexion across time (mean ± SEM) following two separate exercise bouts.

No significant reductions from baseline were recorded for maximal voluntary isometric dorsiflexion strength, or for plantar or dorsiflexion strength between the two exercise bouts. There was however, the general trend for both isometric and isokinetic plantarflexion baseline strength prior to bout 2 to be approximately 7% greater than bout 1, with a 4% increase in dorsiflexion torque being observed.

5.3.3.2 *Isokinetic Maximal Voluntary Strength*

Maximal voluntary isokinetic strength of the plantarflexors was significantly reduced ($p<0.05$) from baseline at 0.5 and 24 hours post-walk following the initial exercise bout for the movement velocity of 120°/second (Figure 40), and 0.5 to 48 hours post-walk inclusive for 30°/second and 60°/second (Figure 38 and 39). While reductions from baseline were observed for each of the movement velocities following the repeated exercise bout, these reductions were not significant. An average strength loss of approximately 12 Nm (18%) being recorded following exercise bout 1, and 10 Nm (14%) following exercise bout 2. While lesser strength reductions were recorded following the repeated exercise bout, no significant difference occurred between the two bouts for any movement velocity at any given time interval.

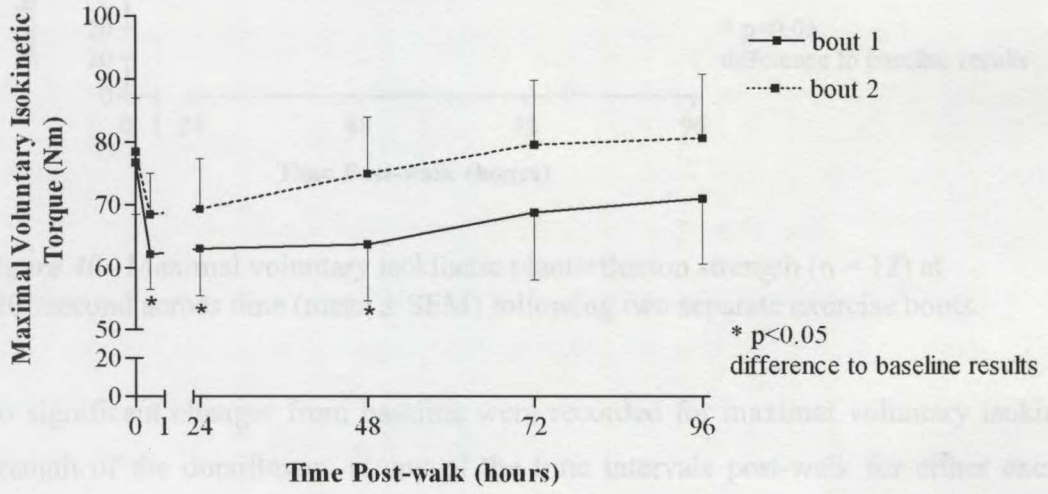


Figure 38. Maximal voluntary isokinetic plantarflexion strength (n = 12) at 30°/second across time (mean ± SEM) following two separate exercise bouts.

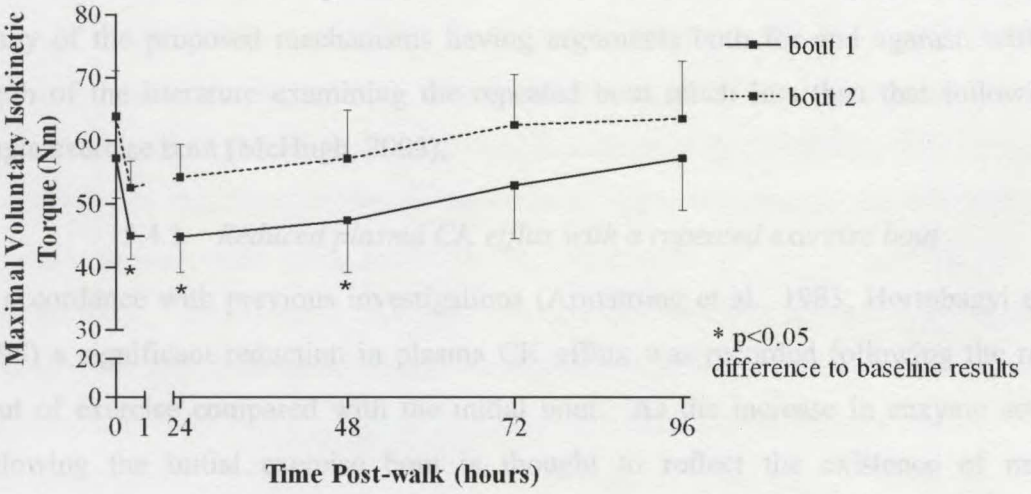


Figure 39. Maximal voluntary isokinetic plantarflexion strength (n = 12) at 60°/second across time (mean ± SEM) following two separate exercise bouts.

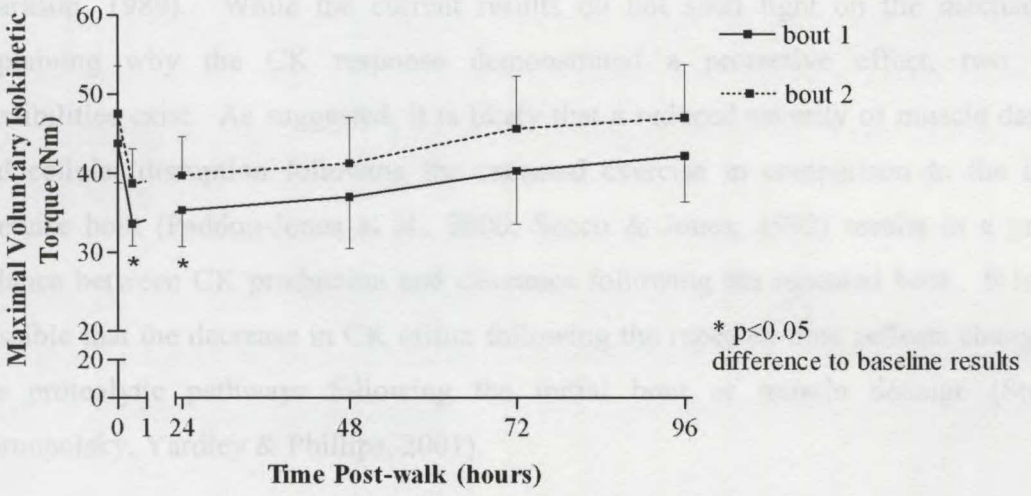


Figure 40. Maximal voluntary isokinetic plantarflexion strength (n = 12) at 120°/second across time (mean ± SEM) following two separate exercise bouts.

No significant changes from baseline were recorded for maximal voluntary isokinetic strength of the dorsiflexors at any of the time intervals post-walk for either exercise bout. As with strength production of the plantarflexors, the peak strengths produced at 30°/second was approximately twice that of the strength produced at 120°/second of the dorsiflexors, although dorsiflexion strength was considerably less than that of plantarflexion (26 Nm compared to 78 Nm).

5.4 Discussion

While advances have been made into understanding the mechanisms responsible for the changes seen following a single bout of eccentric exercise, there is still little understand

of the mechanisms for the protection observed in relation to the repeated bout effect. Many of the proposed mechanisms having arguments both for and against, with the depth of the literature examining the repeated bout much less than that following a single exercise bout (McHugh, 2003).

5.4.1 *Reduced plasma CK efflux with a repeated exercise bout*

In accordance with previous investigations (Armstrong et al., 1983; Hortobagyi et al., 1998) a significant reduction in plasma CK efflux was recorded following the repeat bout of exercise compared with the initial bout. As the increase in enzyme activity following the initial exercise bout is thought to reflect the existence of muscle membrane damage (Armstrong, 1986), the results suggest a significant amelioration of fibre damage. The results of previous studies that directly quantified the histological damage following repeated eccentric exercise bouts support this idea (Ebbeling & Clarkson, 1989). While the current results do not shed light on the mechanisms explaining why the CK response demonstrated a protective effect, two main possibilities exist. As suggested, it is likely that a reduced severity of muscle damage and cellular disruption following the repeated exercise in comparison to the initial exercise bout (Paddon-Jones et al., 2000; Sacco & Jones, 1992) results in a greater balance between CK production and clearance following the repeated bout. It is also possible that the decrease in CK efflux following the repeated bout reflects changes in the proteolytic pathways following the initial bout of muscle damage (Stupka, Tarnopolsky, Yardley & Phillips, 2001).

5.4.2 *Reduction of soreness and tenderness*

Results of the present study demonstrated a significant decrease in DOMS responses of triceps surae with active movement between the two eccentric exercise bouts. This is consistent with a number of studies which also evaluated soreness during active movement (McHugh et al., 2001; Rowlands et al., 2001). In contrast to the findings of Newham et al. (1987), the present investigation showed no significant difference (or protection) for muscle tenderness between the two eccentric exercise bouts. Newham (1987) demonstrated a progressive reduction in tenderness over biceps and brachioradialis following repeated maximal eccentric exercise of the elbow flexors where three exercise bouts were performed at 2-week intervals. It should however be noted that increased tenderness was reported following all three-exercise bouts, the authors however, did not state whether the tenderness reached significant levels following each exercise bout.

McHugh et al. (2001) reported a clear protective effect for the mean tenderness recording from biceps femoris, semimembranosus and semitendinosus following repeated (2 weeks apart) 60% maximum contractions of the hamstrings (10 repetitions of 6 sets). In this study however, tenderness values represented the mean of three values, with tenderness being recorded over the muscle belly of each of the specified muscles only. Since there is a suggestion that tenderness is greater at the musculo-tendinous junction (Edwards, Mills & Newham, 1981), the selected sites for tenderness recording may have underestimated the existence of tenderness within the entire muscle. The differences in tenderness across different sites of the triceps surae have been previously highlighted (Chapter 4). This possibility is further supported by the noticeable difference between the peak tenderness values reported in the two previous investigations following the initial exercise bout. The present study reported a peak tenderness of 48.0 ± 7.0 kPa where 100 kPa represented no tenderness and 0 kPa was maximal tenderness. McHugh (2001) demonstrating only minimal tenderness with a peak recording of just 6.4 ± 7.4 N where 0 N represented no tenderness and 40 N maximal tenderness.

The reduced tenderness reported by the above authors with a repeat bout is similar to that reported during palpation of the elbow flexors (Nosaka et al., 2001) following two maximal eccentric exercise bouts separated by a period of six months. Using the same model, Nosaka & Newton (2002) also reported a significant reduction in palpation soreness following two bouts of maximal eccentric exercise performed 7 days apart. A comment regarding these results is that it must be assumed that the pressure applied during palpation of the sore muscle remained consistent across the testing intervals. Caution is therefore warranted when comparing this methodology of soreness with that of tenderness, although it would appear to be a comparable measure based on the stimulation of tactile receptors as discussed in the previous chapter.

Throughout the literature, little explanation is given as to why an attenuation of soreness and/or tenderness occurs with a repeated exercise bout. Where an explanation is given for the repeated bout effect, it is most often offered in relation to the differences seen between bouts for strength declines rather than soreness. Neural adaptation is a frequently proposed mechanism in relation to 'protection' of strength losses (McHugh, Connolly, Eston & Gleim, 1999), with an improved ability to repair injury (Brown et

al., 1997), and a decreased inflammatory response (Pierrynowski, Tiidus & Plyley, 1987) all cited as possible mechanisms for a reduction in soreness. Given that the mechanisms responsible for DOMS following an initial bout of eccentric exercise are poorly understood (Gulick & Kimura, 1996), it is unsurprising that the mechanisms responsible for a reduction in this variable following a repeated exercise bout are also unclear.

In the previous chapter it was suggested that the soreness and tenderness measurements might represent the responses of different pathways of pain perception. The differences in the repeated bout effect for the two variables can therefore be best explained as a variation in the level of analgesia (pain suppression) and allodynia (sensitivity to innocuous stimuli) activity of the two pain pathways (Pritchard & Alloway, 1999, p. 217). Further investigations expanding the recent work of Weerakkody et al. (2001) who demonstrated that muscle mechanoreceptors contribute to soreness following eccentric exercise, may offer additional perspectives on both the cause of DOMS and the mechanisms of the repeated bout effect. These authors demonstrated pain to be more intense with vibration of a sore muscle or the application of nerve compression resulting in muscle weakness. This led to the conclusion that the sensation of DOMS was the result of a combination of input from nociceptors and large mechanoreceptor afferents.

5.4.3 *Strength declines following a repeated exercise bout*

The present study demonstrated significant reductions from baseline for maximal voluntary isometric plantarflexion strength, with reductions occurring following both exercise bouts. While there were no significant differences between bouts, a trend for faster recovery of strength was observed following the repeated exercise bout, particularly in relation to strength production with the limb in a dorsiflexed position. The failure of the between bout results to reach the adjusted level of significance ($p<0.008$) however, may have been limited by the small sample size. The results for each isometric angle falling below the standard level of $p<0.05$ for at least one testing time interval post-walk. More subjects would therefore be needed to confirm the trend for protection between bouts in relation to strength decline. This observation for a greater strength recovery however, is in agreement with previously reported data (Newham et al., 1987; Nosaka et al., 2001).

The protective effect on muscle strength with a repeated bout of exercise is somewhat different to that seen for plasma CK and soreness. While CK and soreness responses are attenuated with a repeat bout, the strength declines are usually of the same magnitude as losses following the initial exercise bout. The protective effect in this instance relates to the observations of a more rapid recovery of strength following the repeat exercise (Nosaka & Clarkson, 1995; Nosaka & Newton, 2002)

Similar to the investigations of Hortobagyi et al. (1998), a significant reduction in maximal voluntary isokinetic plantarflexion strength was also recorded but following the initial exercise bout only in the present study. While similar percentage reductions from baseline were recorded following the repeated bout, the results did not prove significantly different from baseline, nor were there any differences recorded between the two bouts at any post-walk time period. While it is not understood why a greater protective effect was seen for isokinetic strength over that for isometric strength in the present study, it may be related to suggestions of the greater preservation of elastic energy for concentric than isometric actions, when the force generating capacity of a muscle is otherwise reduced following eccentric exercise (Bobbert, Gerritsen, Litjens & van Soest, 1996). The dynamic action of an isokinetic movement coupled with co-activation of the antagonist muscle (Psek & Cafarelli, 1993), allows for greater strength maintenance than for isometric contractions. Neural, mechanical, and cellular adaptations have also been proposed for the repeated bout effect in relation to strength decline.

It has been previously shown that eccentric or negative contractions have a unique activation pattern compared to that of isometric and concentric actions, with less motor unit activation for a similar level of force production. Therefore, it may not be the forced lengthening that results in the exercise-induced damage, but the greater muscular forces that can be produced (Enoka, 1996; Kellis & Baltzopoulos, 1998). It is this lower level of motor unit activation which has been suggested to provide a 'learnt' effect, allowing for more efficient recruitment during the repeated bout of eccentric exercise. More efficient recruitment meaning subjects may have 'learned' to activate motor units that would otherwise not have been recruited according to the size principle during the repeated eccentric exercise (Golden & Dudley, 1992). This learned pattern of recruitment reducing the stress on the active muscle during the repeated bout decreases the amount of damage, and therefore the degree of strength loss (Mair et al,

1995; Nosaka & Clarkson, 1995). While the pattern of muscle activation of triceps surae was not recorded in the present study following the exercise protocol, this will be examined in later investigations (see Chapters 6 & 7).

It has also been suggested that an adaptive response during repair from the initial bout of damage may result in structural reorganisation allowing the muscle fibres to become more resistive to damage in the repeated bouts. Results from both animal (Leiber & Friden, 1993) and human (Rowlands et al., 2001) models have shown that the further beyond resting length the muscle is strained, the greater the symptoms of muscle damage. The stretching caused by such contractions resulting in damage to the sarcomeres within the muscle, referred to as sarcomere 'popping' (Flitney & Hirst, 1978; Morgan, 1990). The 'popping' refers to the idea that stretching of the sarcomere causes disruption to the titin filament, the anchor point of the myosin filament to the Z-disc. The disruption to the filament inhibiting the protein interaction necessary for force generation between sarcomeres and therefore limits force generation of the affected muscle (Allen, 2001; Lindstedt, LaStayo & Reich, 2001). Following this 'popping', remodelling of the sarcomeres and other intermediate filaments within the muscle may protect the muscle against the repeated bout of eccentric exercise. The proposed remodelling includes removal of weakened sarcomeres (Byrnes et al., 1985), longitudinal addition of sarcomeres (Friden, Seger, Sjostrom & Ekblom, 1983; Lynn & Morgan, 1994), and a strengthening of the cell membrane (Clarkson & Tremblay, 1998)

An adaptation or reduction in the inflammatory response to eccentric contractions has also been linked to the reduced degree of muscle damage, and enhanced recovery from a repeated bout of eccentric exercise (Mair et al, 1995). Lapointe, Fremont and Cote (2002) concluded that the inflammation process following the initial exercise bout was a mediating factor in the adaptation seen following lengthening contractions. Strengthening of the muscular structures and cellular elements, dependent on the inflammatory process within the muscle, rather than neural components/changes alone. As no measure of the inflammatory response was performed it is not possible to comment on the degree to which inflammation occurred following either the initial or repeated exercise bout, and therefore the degree to which the response may have contributed to the observed repeated bout effect.

5.5 Conclusions

The current study demonstrated attenuated responses in the triceps surae for DOMS, plasma CK efflux, and maximal voluntary strength declines following a repeated bout of downhill backward walking. Such reductions in indicators of EIMD following the repeated bout effect are consistent with those previously demonstrated in other muscle groups (Eston et al., 1996; Nosaka et al., 2001; Rowlands et al., 2001). The repeated bout effect demonstrated in the present investigation further validates downhill backward walking as an effective model for EIMD in triceps surae. While tenderness in the present investigation did not exhibit the same protected effect as expected based on previous results (Newham et al., 1987), this may reflect differences in the exercise protocols, or a difference between tenderness measurement techniques.

CHAPTER 6 CHANGES IN PROPRIOCEPTIVE AND ACTIVATION RESPONSES OF TRICEPS SURAE ASSOCIATED WITH EXERCISE INDUCED MUSCLE DAMAGE

6.1 Introduction

Although a reduction in maximal voluntary strength is an important indicator of EIMD following eccentric exercise, it has been well demonstrated that the time courses of recovery of strength and fibre damage are different. Voluntary strength loss is usually greatest immediately following exercise, with recovery generally linear dependent on the level of initial strength loss (Clarkson et al., 1992; Hortobagyi et al., 1998). Muscle fibre damage however is delayed, with alterations in fibre structure due to the process of necrosis and regeneration (Jones, Newham, Round & Tolfree, 1986), complete recovery can take up to 30 days (Faulkner, Brooks & Opitek, 1993). Immediately following eccentric exercise muscle biopsies reveal mild myofiber disruption including mast cell degranulation, and separations of the extracellular matrix from myofibers (Newham, McPhail et al., 1983; Stauber et al., 1990). In the days following exercise more marked fibre disruption is observed with myofibrillar necrosis and inflammatory cell infiltration evident (O'Reilly et al., 1987).

Although numerous investigators have attempted to establish the underlying causes of EIMD and strength loss following eccentric exercise, the exact mechanisms remain largely undetermined (Leiber & Friden, 1999; Malm, 2001; McHugh, Commolly, Eston & Gleim, 1999). Recently, disruption of the excitation-contraction coupling process has been identified as a possible factor contributing to the strength losses observed immediately following eccentric exercise (Ingalls, Warren, Williams, Ward & Armstrong, 1998). Additionally, it has been shown that the strength losses observed cannot be explained by inadequate voluntary muscle activation associated with muscle soreness, since poor correlations have been reported between strength loss and soreness (MacIntyre et al., 1995). Furthermore, significant reductions in maximal voluntary strength, stimulated maximal twitch tension, and tetanic tension at varying frequencies have been demonstrated following eccentric exercise (Davies & White, 1981). Previous work has also demonstrated alterations in the pattern of the voluntary electromyographic (EMG) signal following eccentric exercise, but this was unrelated to post-exercise soreness perception (McGlynn et al., 1979). Moreover, results

demonstrating the effects eccentric contractions have on muscle EMG following an exercise protocol are inconsistent.

Deschenes and co-workers (2000) found no overall increase in EMG activity of the quadriceps following eccentric exercise, but a localised increase in rectus femoris EMG occurred, although this was unexplained. This finding in part supports the results of Kroon and Naeije (1991) and Weerakkody, Percival, Morgan, Gregory and Proske (2003) who reported an increase in the EMG of the elbow flexors following submaximal eccentric exercise. In the case of Weerakkody et al (2003), the authors also reported that while submaximal EMG was increased following exercise, no increases in EMG during a post-exericse maximal contraction were recorded. In contrast studies by Day, Donnelly, Brown and Child (1998), and Pearce et al. (1998) reported no significant changes in EMG following maximal eccentric contractions of the knee extensors and biceps brachii respectively. They suggested that the unchanged EMG suggested no alteration in the sarcolemmal depolarization of the muscle, and/or no change in the muscle activation capability of the subjects. Komi and Rusko (1974) however concluded that a decrease in EMG occurred when eccentric muscle actions are maximal and muscle fatigue post-exercise results in inhibition of some motor neurons, or a change in the form of the motor unit potential of the active muscle. As yet, the relationship between changes in EMG following eccentric exercise, voluntary muscle activation patterns and muscle compound action potential (M-wave) have not been investigated.

Disruption to the structure and function of the extrafusal fibres within a muscle which has undergone eccentric exercise is considered to be a key factor contributing to sustained strength loss and weakness observed during EIMD (Warren et al., 2001). However, little consideration has been given to the effects of eccentric contractions on the intrafusal fibres. Although they have not directly tested spindle activity, previous authors have demonstrated proprioceptive based tasks to be altered in association with EIMD, and cite damage to or dysfunction of, the muscle spindle and/or Golgi tendon organs as factors contributing to altered function (Brockett et al., 1997; Pearce et al., 1998). Understanding and limiting activity that has a potential to alter proprioception and the pathways that are proprioception controlled, may prove important in the prevention of injury both in the sporting and non-sporting sectors. Altered proprioceptive and reflex functioning is already an area of focus as a potential causal

factor in falls (Richardson & Hurvitz, 1995; Schieppati, Tacchini, Nardone, Tarantola & Corna. 1999).

The current investigation therefore aimed to examine:

1. Whether changes to the function of intrafusal muscle fibres occur in response to downhill backward walking.
2. Whether a change in muscle activation occurs following downhill backward walking.
3. The effects of a bout of downhill backward walking on performance of various proprioceptive tasks of the foot plantarflexors.
4. The relationships that may exist between reflex responses, activation changes and proprioceptive errors following a bout of downhill backward walking.

6.2 Methods

6.2.1 *Subjects*

A total of 12 subjects were recruited for the study with a mean (± SEM) age, height and weight of 28.2 ± 2.4 years, 170.0 ± 2.3 cm and 70.2 ± 3.1 kg respectively. As with previous investigations, an even gender balance was maintained (6 males and 6 females) although no gender distinctions were made throughout the data analysis due to the small sample size. Testing was conducted for each variable on both the exercised and non-exercised limb.

6.2.2 *Testing Schedule and Exercise Protocol*

Testing was conducted for each subject on seven occasions over a 10-day period (Table 8). The exercise protocol consisted of 60 minute downhill backward walking (modified Trackmaster treadmill, JAS manufacturing, TX : USA) as described in Section 4.2.3. Each subject performed the exercise protocol on one occasion, with the stepping (eccentrically biased) limb being the left limb.

Table 8.
Testing Schedule for Criterion Protocol Measures

	Baseline		Exercise	Post-exercise				
	Time (hours)							
	72	48	0	0.5	24	48	72	96
Criterion measures	√	√		√	√	√	√	√

6.2.3 *H-reflex*

H-wave responses (also see Chapter 3) for both limbs were elicited by stimulation of the tibial nerve in the popliteal fossa using a DS7 Digitimer stimulator (Digitimer, Hertfordshire : UK). Single electrical pulses (100μs duration) were delivered via a Medelec bipolar stimulator (AMA medical products, Western Australia : AUS) to the tibial nerve at 10-second intervals. The stimulator position in the popliteal fossa was adjusted to elicit the peak H-wave response for a given stimulus intensity before being held firmly in position with velcro strapping around the thigh. Stimulator placement was conducted first in the exercised leg, then in the non-exercised leg, with reflex responses then being recorded simultaneously in both limbs.

Following a similar method to that described by Sacco, Newberry, McFadden, Brown and McComas (1997), two stimuli were delivered at increasing intensities of 0.1 mA, with 10-second intervals between levels. This procedure was performed from an intensity low enough to elicit a useful H-wave until the H-wave began to be obscured by the M-wave. Results were analysed offline with the H-wave peak – to – peak amplitude for each stimulus recorded and the average of the results for each stimulation intensity calculated. For statistical analysis across time, the peak H-wave amplitude was determined, with baseline data representing the mean of the two results from baseline testing.

6.2.4 *Muscle Compound Action Potential and Contractile Properties*

M-wave responses for both limbs were elicited by stimulation of the tibial nerve in the popliteal fossa following the same method as that described in the previous section for the H-wave response. Once the peak H-wave had been detected the stimulus intensity was then increased at 0.5mA increments until no further increases in either plantarflexor strength or M-wave amplitude was seen. Results were analysed off line with the M-wave peak – to – peak amplitude for each stimulation recorded and the average of the results for each stimulation intensity calculated. For statistical analysis across time, peak M-wave amplitude was determined, with baseline data representing the mean of the two results from baseline testing.

Ankle plantarflexor torque recordings corresponding to the twitches associated with the final M-waves were analysed off line for the contractile properties of twitch peak torque

(P_T), contraction time (time to peak torque; TTP) and half relaxation time (T_H). P_T represented the maximal torque produced during stimulation. TTP was measured as the time elapsed between the initial rise in torque until the point of peak torque. T_H was calculated as the time elapsed between the points where twitch torque was equal to 90% and 45% of P_T. Baseline values used during statistical analysis represent the mean results for each variable from the two baseline testing occasions.

6.2.5 *Tendon Tap Reflex*

An Achilles tendon tap (Martini & Welch, 1998, p. 78) was elicited in both the exercised and non-exercised limbs with the subsequent EMG responses (peak – to – peak amplitude for soleus and gastrocnemius muscles) and peak torque outputs recorded. Eight consecutive tendon taps were performed using a patella tendon hammer (AMA medical products, Western Australia : AUS), with 10-seconds allowed between each tendon tap. The striking distance from which the tap was elicited was controlled for throughout the testing procedures however the force of the tap was not mechanically controlled. The same individual however was responsible for the testing on each occasion with the testing variability of this procedure reported in Chapter 8. Throughout testing, the tendon tap responses were elicited in the exercised limb first for all subjects. Voltage changes recorded during the procedure were analysed offline post – test with the maximum and minimum results from the eight trials discarded, and the remaining six results averaged for statistical analysis. Baseline results used for comparison across time represent the mean results of the two baseline testing occasions.

6.2.6 *Maximal Voluntary Isometric Plantarflexor Strength*

Maximal voluntary isometric strength of the plantarflexors for both the exercised and non-exercised limbs was determined with using the DAD (also see Chapter 3). Subjects performed three maximal efforts lasting approximately 5-seconds, starting with the exercised limb on each occasion and alternating between limbs, with the mean result from the three trials used during statistical comparisons. Each trial performed was stored as a voltage output via AmLab with results analysed offline post – test. From each 5-second trial, a 0.5-second window representing the peak torque produced during the trial was exported to a spreadsheet (Microsoft Excel) where the values for torque and avEMG were averaged (also see Chapter 3). The baseline results used for statistical analysis represent the peak result produced during the two baseline testing occasions.

To verify that subjects were maximally activating the plantarflexors during the voluntary strength task, a single supramaximal twitch was delivered via the stimulating electrode positioned during the H-reflex protocol within the third trial effort for each limb. Using previously described methods the central activation ratio (CAR) was then calculated (Kent-Braun & Le Blanc, 1996). The additional measure of resting muscle tension was also determined in conjunction with the voluntary strength protocol. The resting tension was taken as the torque produced at rest by the plantarflexors with the limb secured at 10° dorsiflexion prior to performing a maximal voluntary contraction.

6.2.7 *Vertical Jump*

Single limb vertical jump height was determined in both limbs using the squat jump technique (Sale, 1991, p. 64), with a countermovement performed. Jump height being determined as the difference between standing height and jump height using a jump – and – reach board (Acromat, South Australia : AUS). The limb tested first on each testing occasion was randomised with three consecutive trials performed on each limb. An average of the three trials was used for statistical analysis, with baseline representing the mean of the results obtained during baseline testing.

6.2.8 *Torque Perception*

Ankle torque perception was determined with both ankles positioned in 10° dorsiflexion. Subjects were asked to match a target force calculated as 30% of their pre-exercise MVC with both the exercised limb and non-exercised limbs acting as a reference limb. Subjects were given a visual target for the reference limb before being instructed to 'match' the force in the contralateral (matching) limb for which no visual feedback was given. Three trials were performed for each condition, with matching to the exercised limb always the first condition presented. As with the isometric maximal voluntary strength task, avEMG recordings for both limbs were made simultaneously with the torque recordings for each force perception trial.

During the task, subjects were given 8-seconds in which to complete each match with all results recorded as a voltage output using AmLab. The outcome variables for each force perception trial were torque output, soleus avEMG and gastrocnemius avEMG for both the target and the matching limb. Results were then analysed offline post – test and a 0.5-second period representing the closest match for each trial exported to Excel. The closest match referred to that time where the torque difference between the target

and the matching limb was the smallest, given that the target limb torque was within 5% of the 30% MVC torque required. Each 0.5-second trial sample was averaged, with the mean of the three trials used for statistical analysis. The baseline results used during statistical analysis representing the closest matching result from the two baseline testing occasions.

6.2.9 *Joint Position Perception*

Each subject performed a joint position perception task with both the exercised and non-exercised limbs acting as the reference on separate occasions for the two joint angles of 10° dorsiflexion and 10° plantarflexion. Subject positioning was the same as that previously described (see Chapter 3) with the exception that the foot was secured using only the padded elastic strap positioned over the mid region of the foot. This allowed the footplate to move freely with the foot during ankle movements without restricting range of motion.

Subjects had the exercised limb moved to and held constant at one of the two angles and, after a period of 5-seconds, were asked to 'match' the angle with the contralateral (non-exercised) limb. Three attempts were allowed at each angle with 5-seconds between each attempt during which time both limbs were relaxed. The same procedure was then performed using the non-exercised limb as the reference limb, matching with the exercised limb. On each occasion the exercised limb was used as the reference (or target) limb first with 10° dorsiflexion always the first reference angle presented. Presentation of the reference angles then alternated between plantarflexion and dorsiflexion throughout the procedure. An average of the three trials for each angle was used for statistical analysis across time, with baseline values representing the best result from the two baseline testing occasions.

6.2.10 *Range of Motion and Relaxed Ankle Angle*

Both range of motion (ROM) and relaxed angle of the ankle were determined using the DAD immediately following the joint position perception. Following the last angle match, subjects were instructed to 'go to full plantarflexion' for 5 seconds, followed immediately by 'full dorsiflexion' for 5 seconds before being instructed to 'relax', with both limbs moving simultaneously. Instructions for the ROM task always being delivered in the same order to prevent a possible order effect on the relaxed ankle angle.

Baseline results used for statistical analysis represent an average of results from the two baseline testing occasions for both ROM and relaxed ankle angle.

6.2.11 *Statistical Analysis*

Each criterion variable was analysed using a repeated measures (time) ANOVA. Where a significant p value ($p<0.05$) was obtained, post hoc analysis consisted of a simple contrast (to baseline) to determine those time intervals that were significantly different from baseline. To assess the existence of relationships between the testing variables a Pearson Product moment correlation coefficient was calculated. As previously mentioned, the reliability of each measure was calculated from data collected over two testing occasions, the results for which are reported in Chapter 8.

6.3 Results

6.3.1 *Presentation of Results*

The results for each variable are presented in one of two graphical formats. Where no significant changes from baseline for either limb, or a significant change from baseline for both limbs occurred, the results are shown as mean ± SEM across time at each testing time intervals. Where a significant change from baseline occurred for one limb only the results for that limb are also presented as mean ± SEM across time at each testing time intervals. A shaded box area then represents the results for the remaining limb. The shaded boxes representing the baseline mean ± SEM, and thus indicates that value range from which no significant changes across time occurred for that limb.

6.3.2 *H-reflex and Tendon Tap EMG Responses*

No significant changes were recorded for the H-wave of the gastrocnemius (Figure 41) or soleus (Figure 42) muscle for either the exercised or the non-exercised limb across time. There was however a trend for the soleus H-wave to increase 0.5 hours post-walk followed by a reduction at 48 hours post-walk, before returning to baseline values by the 96-hour time interval. No clear trends were noted for the gastrocnemius H-wave for either limb, or the soleus H-wave of the non-exercised limb.

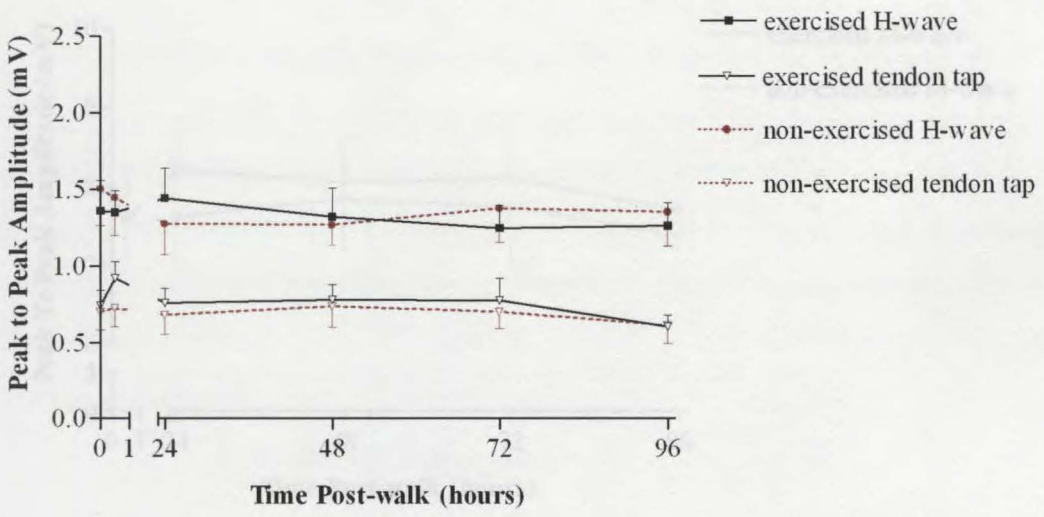


Figure 41. Hoffmann (n = 12) and Achilles tendon tap (n = 8) reflex responses (mean ± SEM) of gastrocnemius for the exercised and non-exercised limbs across time.

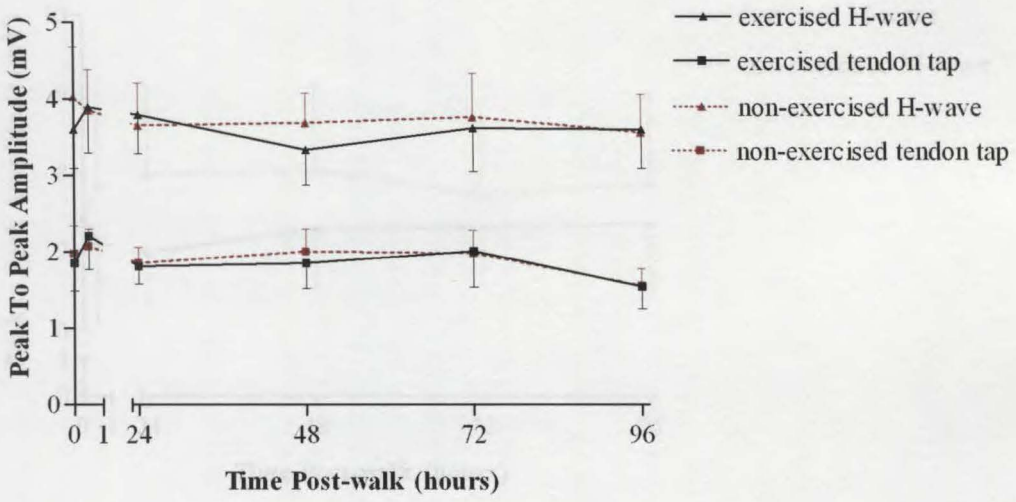


Figure 42. Hoffmann (n = 12) and Achilles tendon tap (n = 8) reflex responses (mean ± SEM) of soleus for the exercised and non-exercised limbs across time.

6.3.3 *Muscle Compound Action Potential*

As with the H-reflex, no significant changes from baseline were recorded in relation to the soleus or gastrocnemius maximal M-wave for either the exercised or non-exercised limb post-walk (Figure 43 and 44). While a general decline in the M-waves of the exercised limb was observed immediately post-walk, the maximum deviation (positive or negative) from baseline recorded at any time interval was approximately 0.27 mV or 6% (exercised limb; soleus). An example of the trace recordings for both the H-reflex and a maximal M-wave are given in Figure 45.

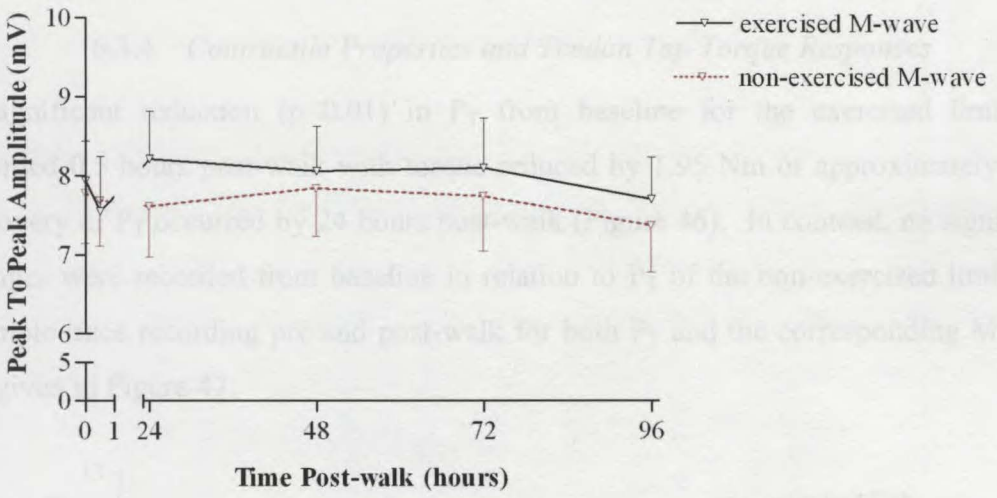


Figure 43. Maximal M-wave (n = 12) responses (mean ± SEM) of soleus for the exercised and non-exercised limbs across time.

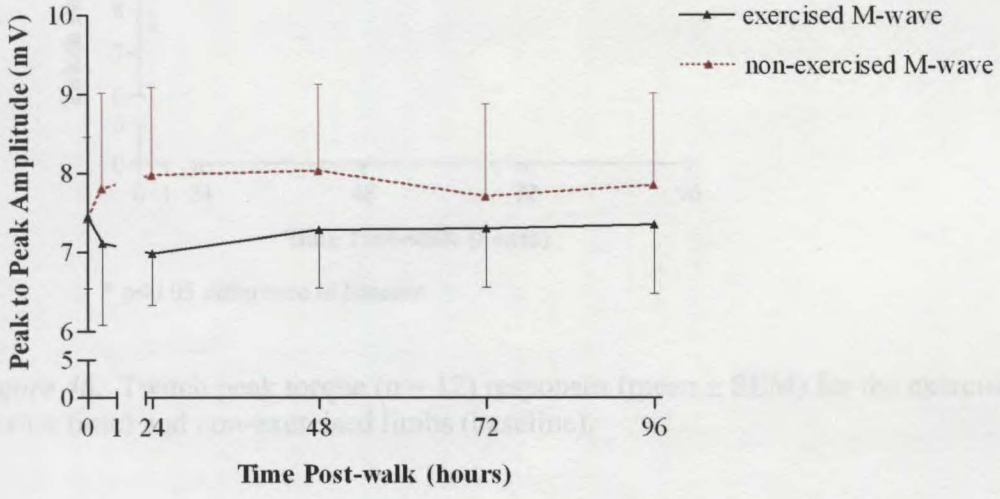
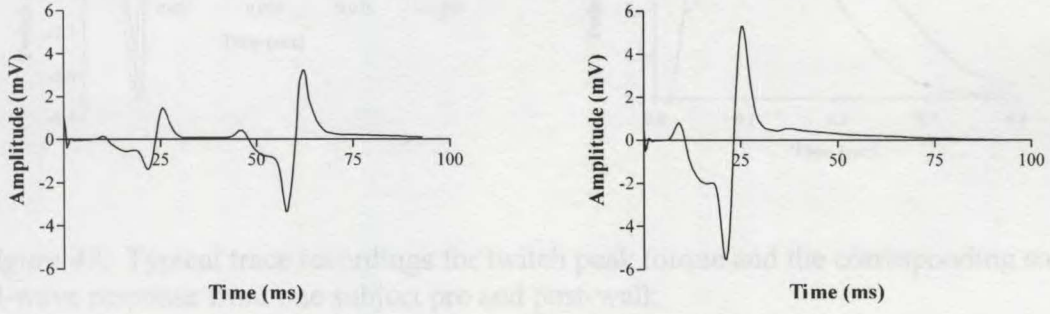


Figure 44. Maximal M-wave (n = 12) responses (mean ± SEM) of gastrocnemius for the exercised and non-exercised limbs across time.

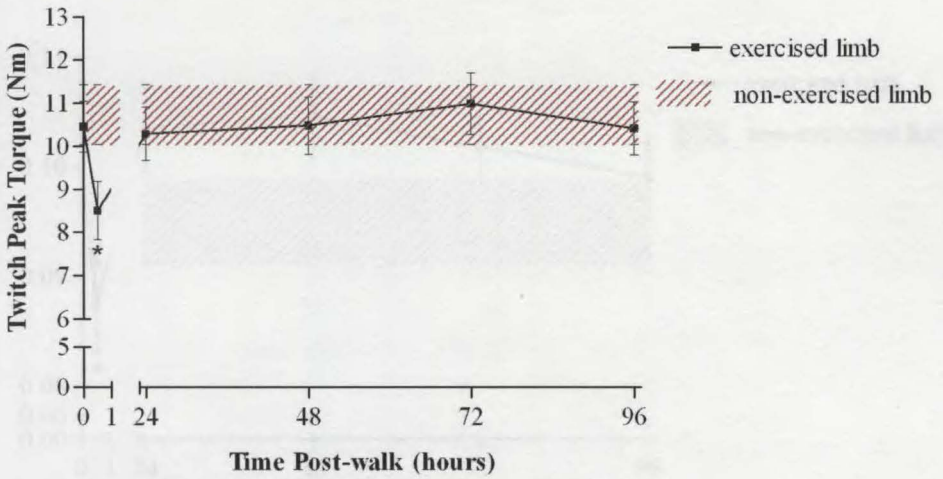


(a) (b)

Figure 45. Sample trace recordings of the H-wave and M-wave at low intensity stimulation (a) and the maximal M-wave (b).

6.3.4 *Contractile Properties and Tendon Tap Torque Responses*

A significant reduction ($p<0.01$) in P_T from baseline for the exercised limb was recorded 0.5 hours post-walk with torque reduced by 1.95 Nm or approximately 17%. Recovery of P_T occurred by 24 hours post-walk (Figure 46). In contrast, no significant changes were recorded from baseline in relation to P_T of the non-exercised limb. An example trace recording pre and post-walk for both P_T and the corresponding M-wave are given in Figure 47.



* $p<0.05$ difference to baseline

Figure 46. Twitch peak torque (n = 12) responses (mean ± SEM) for the exercised (across time) and non-exercised limbs (baseline).

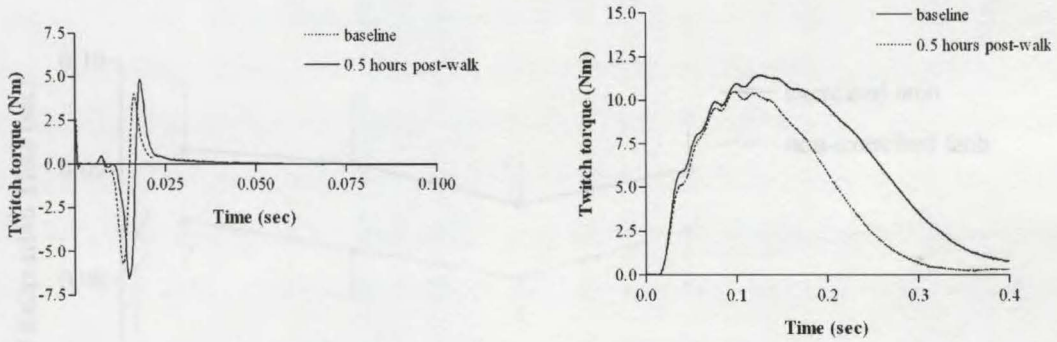
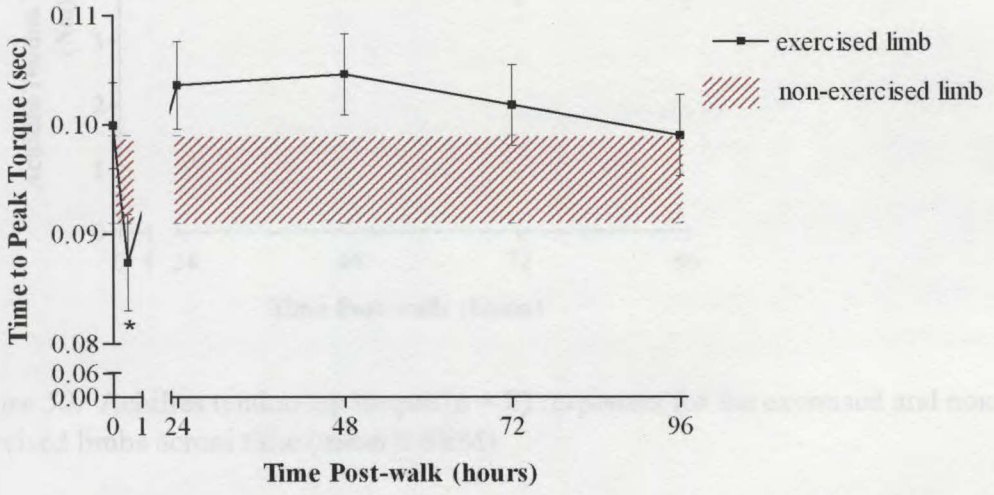


Figure 47. Typical trace recordings for twitch peak torque and the corresponding soleus M-wave response from one subject pre and post-walk

For the exercised limb, a significant reduction ($p<0.01$) in TTP from baseline was recorded 0.5 hours post-walk, with a shortening of 0.013 seconds or approximately 12% recorded (Figure 48). At all other time intervals post-walk for the exercised limb, TTP

increased by approximately 3 – 6% compared to baseline. No significant changes from baseline were recorded for the non-exercised limb across time.

No significant changes were recorded in relation to baseline values for either limb post-walk for T_H (Figure 49). For the non-exercised limb, variations from baseline across the time intervals ranged from 0.6% - 5.3%, while in the exercised limb a range of 3.4% - 10.4% was recorded. The greatest variations of 10.4% and 7.9% for the exercised limb were recorded 24 and 48 hours post-walk. It is notable that all results post-walk of the exercised limb increased from baseline with the exception of 0.5 hours.



* p<0.05 difference to baseline

Figure 48. Time to peak (n = 12) responses (mean ± SEM) for the exercised limb (across time) and the non-exercised limb (baseline).

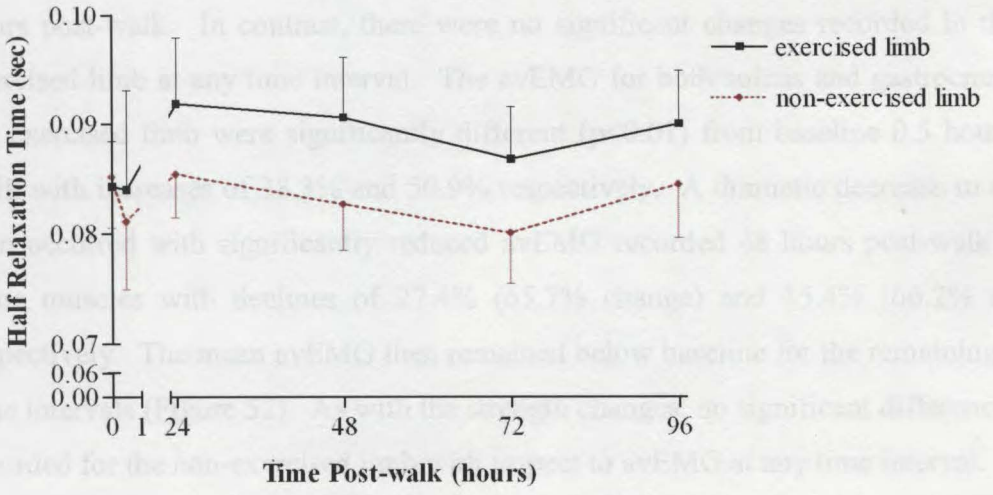


Figure 49. Half-relaxation time (n = 12) responses across time (mean ± SEM) for the exercised and non-exercised limbs across time.

No significant changes from baseline were recorded for either the exercised or non-exercised limb with regards to tendon tap torque output (Figure 50). The greatest variation from baseline of 8% was seen in the exercised limb 24 hours post-walk. The trend for the exercised limb however, followed those for twitch peak torque of the exercised limb.

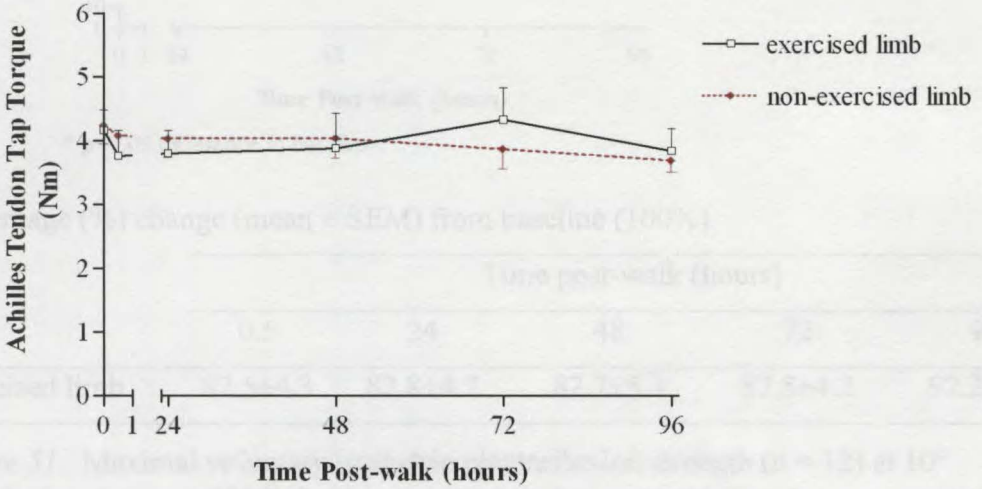
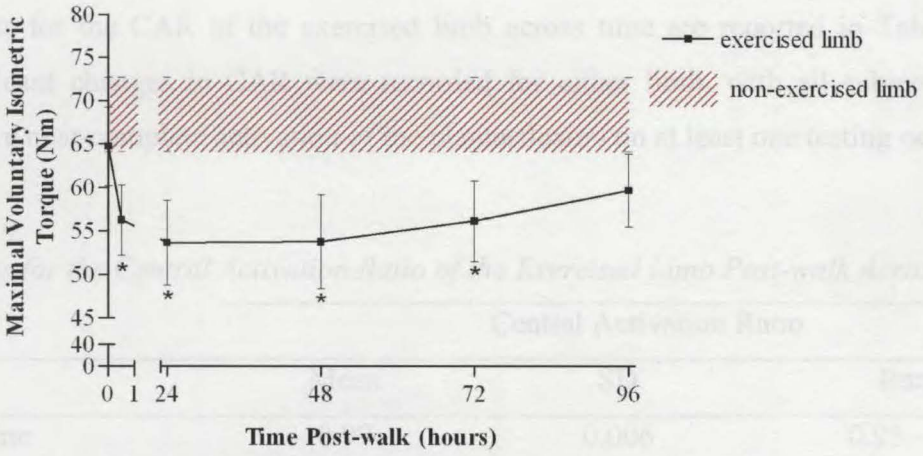


Figure 50. Achilles tendon tap torque (n = 8) responses for the exercised and non-exercised limbs across time (mean ± SEM)

6.3.5 *Maximal Voluntary Isometric Plantarflexion Strength*

Maximal voluntary isometric plantarflexion strength for the exercised limb significantly decreased from baseline 0.5, 24, 48 and 72 hours post-walk with declines of 12 - 17% (Figure 51). The mean strength of the exercised limb was still reduced some 6% at 96 hours post-walk. In contrast, there were no significant changes recorded in the non-exercised limb at any time interval. The avEMG for both soleus and gastrocnemius in the exercised limb were significantly different ($p<0.01$) from baseline 0.5 hours post-walk with increases of 38.3% and 50.9% respectively. A dramatic decrease in avEMG then occurred with significantly reduced avEMG recorded 48 hours post-walk for the same muscles with declines of 27.4% (65.7% change) and 15.4% (66.2% change) respectively. The mean avEMG then remained below baseline for the remaining testing time intervals (Figure 52). As with the strength changes, no significant differences were recorded for the non-exercised limb with respect to avEMG at any time interval.

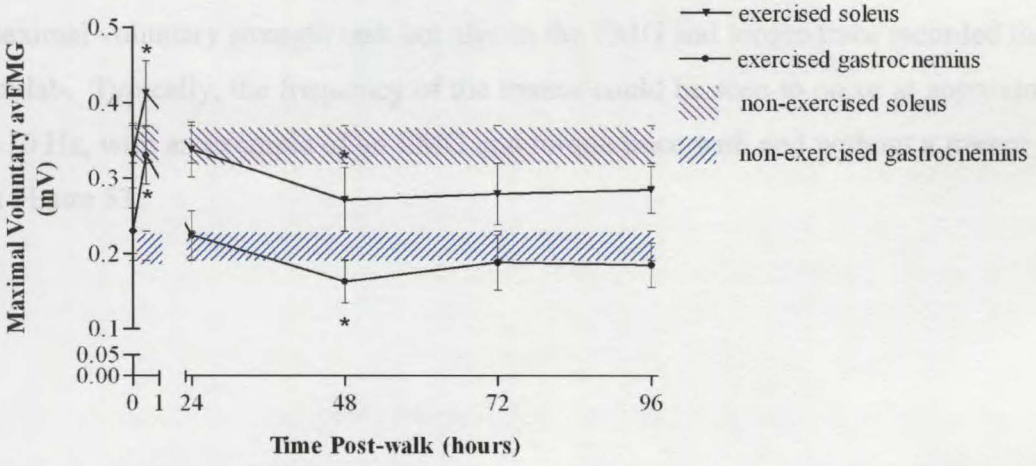


* $p<0.05$ difference to baseline

Percentage (%) change (mean ± SEM) from baseline (100%)

	Time post-walk (hours)				
	0.5	24	48	72	96
Exercised limb	87.5±4.3	82.8±4.7	82.7±5.3	87.5±4.2	92.2±4.7

Figure 51. Maximal voluntary isometric plantarflexion strength (n = 12) at 10° dorsiflexion (mean ± SEM) for the exercised limb (across time) and non-exercised limb (baseline).



* $p<0.05$ difference to baseline

Percentage (%) change (mean ± SEM) from baseline (100%)

	Time post-walk (hours)				
	0.5	24	48	72	96
Soleus	125.1±8.9	102.4±4.2	82.1±6.3	84.5±4.3	86.3±3.7
Gastrocnemius	143.5±7.3	97.4±2.2	70.8±5.9	81.7±3.5	80.4±3.6

Figure 52. Maximal voluntary avEMG (n = 12) of soleus and gastrocnemius (mean ± SEM) for the exercised limb (across time) and non-exercised limb (baseline).

Results for the CAR of the exercised limb across time are reported in Table 9. No significant changes in CAR were recorded for either limb, with all subjects able to achieve near complete activation of the plantarflexors on at least one testing occasion.

Table 9.
Results for the Central Activation Ratio of the Exercised Limb Post-walk Across Time

	Central Activation Ratio		
Time	Mean	SD	Range
Baseline	0.99	0.006	0.95 – 1.00
0.5 hours post-walk	1.00	0.004	0.97 – 1.00
24 hours post-walk	1.00	0.003	0.98 – 1.00
48 hours post-walk	0.99	0.012	0.96 – 1.00
72 hours post-walk	1.00	0.006	0.97 – 1.00
96 hours post-walk	0.98	0.009	0.96 – 1.00

Although the exact frequency was not analysed, a muscle tremor was observed in all subjects following the exercise protocol and in some subjects was present up to 48 hours post-walk. The tremor was observed not only by limb movement during the maximal voluntary strength task but also in the EMG and torque trace recorded through Amlab. Typically, the frequency of the tremor could be seen to occur at approximately 8-10 Hz, with an example of an EMG and torque trace with and without a tremor given in Figure 53.

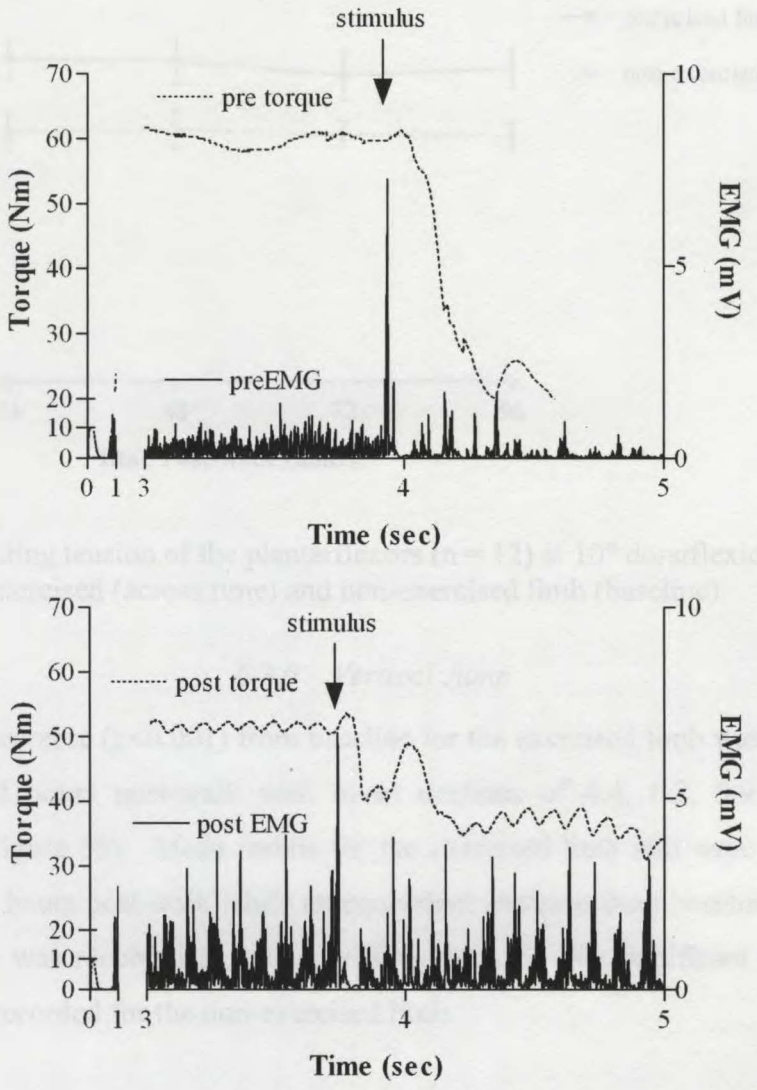


Figure 53. Sample of a typical EMG and torque trace recording pre-walk and 0.5 hours post-walk demonstrating the differeneces in the trace with and without a muscle tremor.

While no significant changes were recorded for the resting tension of either limb post-exercise, there was a clear trend for an increase in tension of the exercised limb, particularly at the 0.5 – 48 hour post-walk time intervals, as illustrated by Figure 54. In contrast there was no obvious change in the resting tension of the non-exercised limb post-walk.

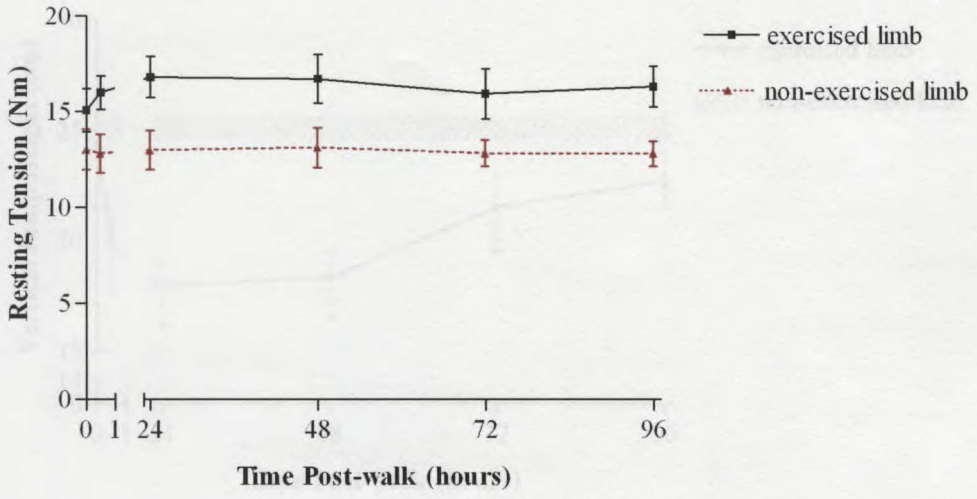
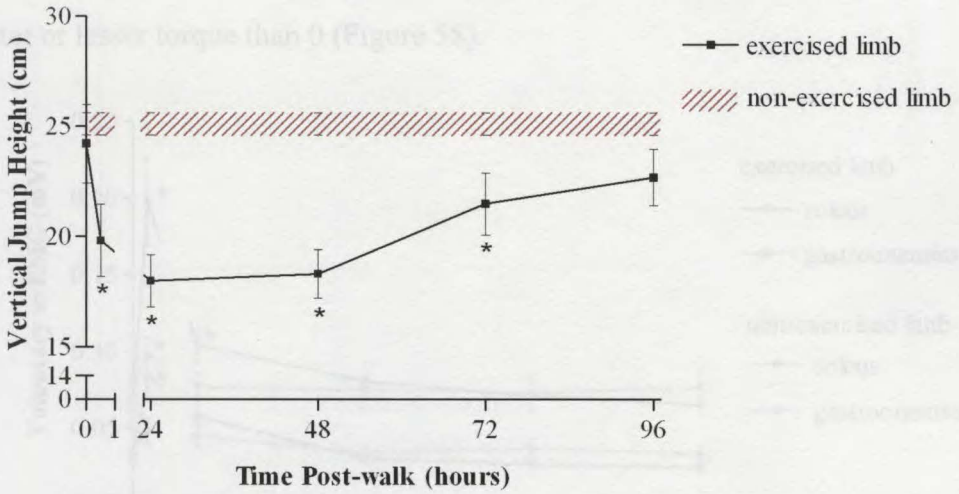


Figure 54. Resting tension of the plantarflexors (n = 12) at 10° dorsiflexion (mean ± SEM) for the exercised (across time) and non-exercised limb (baseline).

6.3.6 *Vertical Jump*

A significant decrease ($p<0.001$) from baseline for the exercised limb was recorded 0.5, 24, 48 and 72 hours post-walk with mean declines of 4.4, 6.2, 5.9 and, 2.7 cm respectively (Figure 55). Mean results for the exercised limb still were reduced from baseline by 96 hours post-walk while an equivalent increase from baseline for the non-exercised limb was recorded at the same time interval. No significant changes from baseline were recorded for the non-exercised limb.



* $p<0.05$ difference to baseline

Percentage (%) change (mean ± SEM) from baseline (100%)

	Time post-walk (hours)				
	0.5	24	48	72	96
Exercised limb	79.2±1.8	75.0±2.5	75.9±6.0	87.5±3.8	91.7±5.5

Figure 55. Vertical jump (n = 8) results (mean ± SEM) for the exercised (across time) and non-exercised (baseline) limbs.

6.3.7 *Torque Perception*

Significant increases ($p<0.05$ non-exercised; $p<0.001$ exercised) from baseline for soleus avEMG were recorded 0.5 hours post-walk for both limbs, with the exercised limb avEMG still increased 24 hours post-walk. Similarly, the gastrocnemius avEMG of the exercised limb was significantly increased ($p<0.001$) 0.5 hours post-walk, returning to baseline values 24 hours post-walk. No significant differences from baseline were recorded for the gastrocnemius avEMG of the non-exercised limb. The same trends occurred regardless of whether the exercised limb was the target, (Figure 56) or matching limb (Figure 57). When matching to the exercised limb a significant increase from baseline ($p<0.05$) in the non-exercised limb (matching) torque was recorded 0.5 and 24 hours post-walk, with the mean torque produced increasing by approximately 20% from baseline matching torques at both time intervals (Figure 58). Conversely, torques produced in the exercised limb while matching to the non-exercised limb were underestimated or less (approximately 6%) than those for baseline at 48 to 96 hours post-walk, although these differences were not significant. As no differences across time were recorded for the target limbs the results are presented as a torque error

from the target limb, thus the target limb is represented as 0, and the matching limb as a greater or lesser torque than 0 (Figure 58).

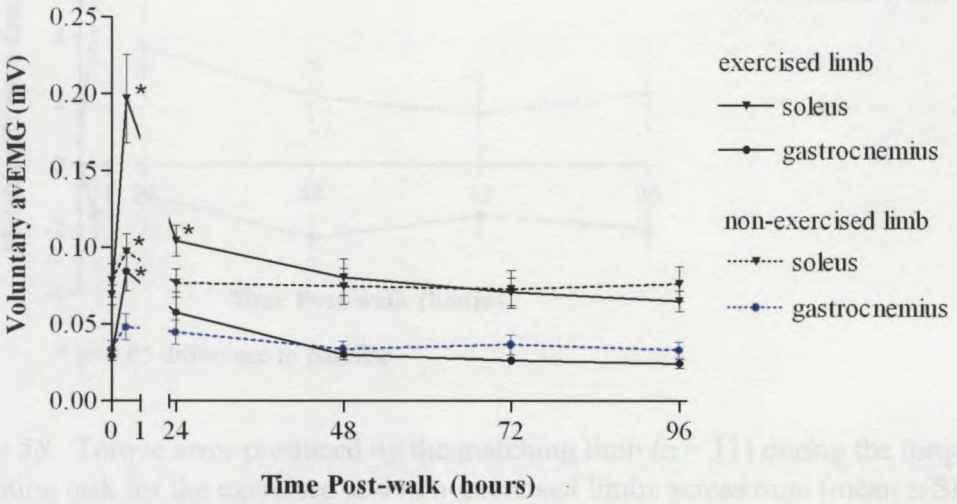


Figure 56. Voluntary avEMG (n = 10) responses of soleus and gastrocnemius during the 30% MVC force perception task for the exercised (target) and non-exercised (matching) limbs across time (mean ± SEM).

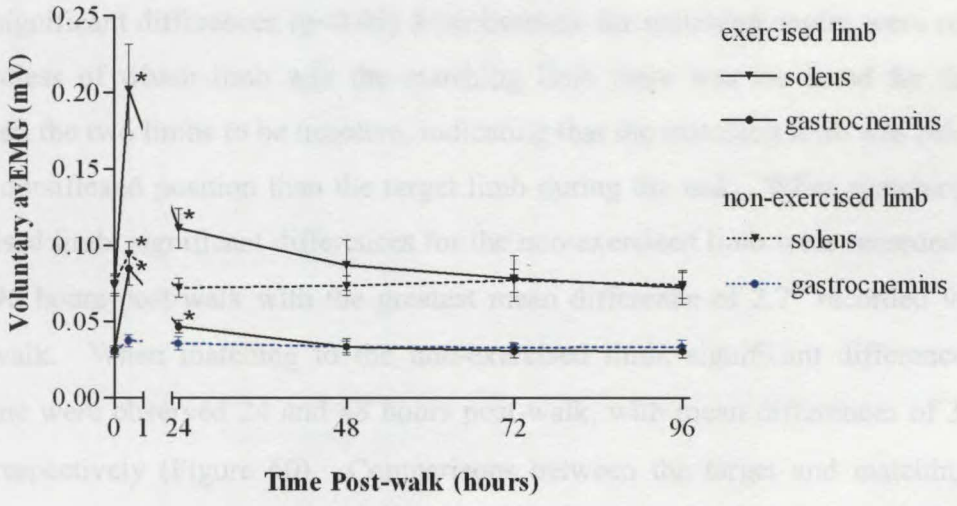


Figure 57. Voluntary avEMG (n = 11) responses of soleus and gastrocnemius during the 30% MVC force perception task for the exercised (matching) and non-exercised (target) limbs across time (mean ± SEM).

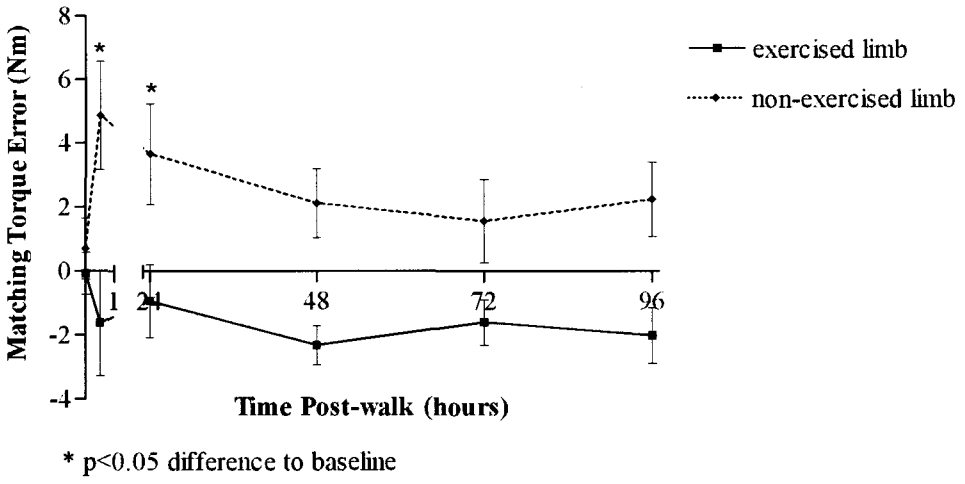
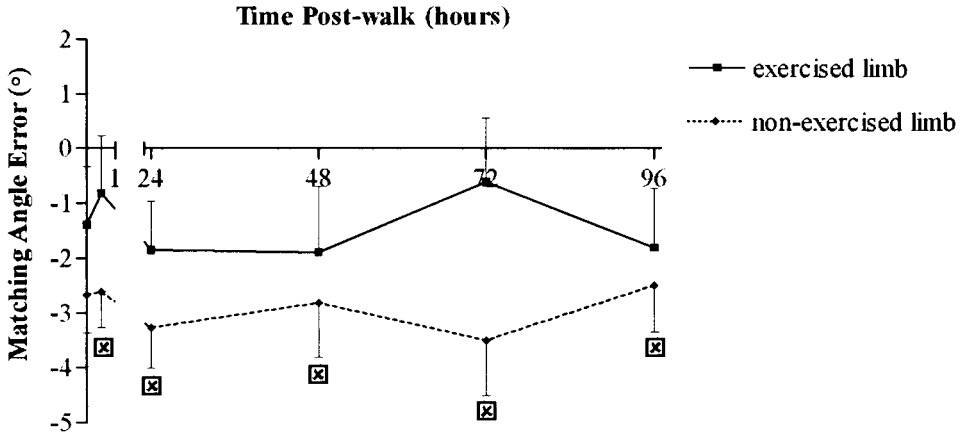


Figure 58. Torque error produced by the matching limb (n = 11) during the torque perception task for the exercised and non-exercised limbs across time (mean ± SEM).

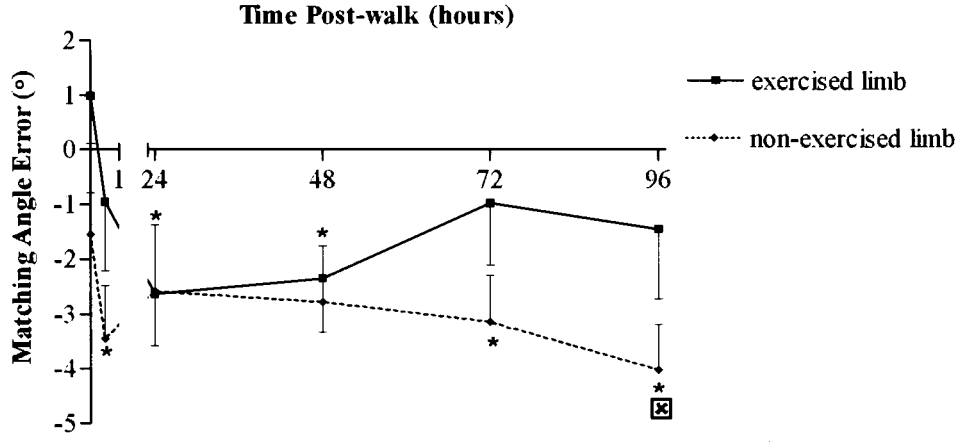
6.3.8 *Joint Position Perception*

When matching for dorsiflexion angle, no significant differences from baseline were recorded for either limb irrespective of whether the limb was the target or matching limb (Figure 59). However, for the matching limb during the plantarflexion matching task, significant differences ($p<0.05$) from baseline for matching angles were recorded. Regardless of which limb was the matching limb there was the trend for the error between the two limbs to be negative, indicating that the matching limb was placed in a more dorsiflexed position than the target limb during the task. When matching to the exercised limb, significant differences for the non-exercised limb were recorded 0.5, 72 and, 96 hours post-walk with the greatest mean difference of 2.7° recorded 96 hours post-walk. When matching to the non-exercised limb, significant differences from baseline were observed 24 and 48 hours post-walk, with mean differences of 3.9° and 3.4° respectively (Figure 60). Comparisons between the target and matching limbs revealed significant differences 96 hours post-walk when matching a plantarflexed angle to the exercised limb, and all time intervals post-walk when matching a dorsiflexed angle to the exercised limb. No significant differences between limbs were recorded when the non-exercised limb was the target limb.



⊠ p<0.05 difference between target and matching limb

Figure 59. Dorsiflexion angle matching error (n = 12) of the exercised and non-exercised limbs across time (mean ± SEM).



* p<0.05 difference to baseline

⊠ difference between target and matching limb

Figure 60. Plantarflexion angle matching error (n = 12) of the exercised and non-exercised limbs across time (mean ± SEM).

6.3.9 *Relaxed Ankle Angle*

No significant changes from baseline were recorded in either limb across time in relation to relaxed ankle angle, with values varying by approximately 1.3° in the non-exercised limb and 0.7° in the exercised limbs. The relaxed ankle angles were also similar between the two limbs at each interval across time (Figure 61).

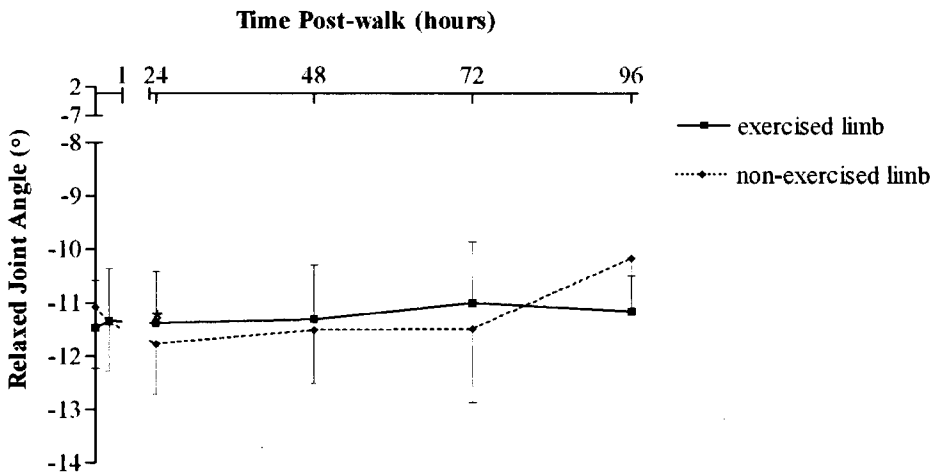


Figure 61. Relaxed ankle joint angle (n = 12) across time (mean ± SEM) for the exercised and non-exercised limbs.

6.3.10 *Range of Motion (ROM)*

While there was a general trend for ankle ROM to be reduced 0.5 hours post-walk in both the exercised and non-exercised limbs (2.3 and 1.5° respectively), these variations were not significantly different from baseline (Figure 62). An overall variation of approximately 3° was observed in the non-exercised limb and 4° in the exercised limb across the testing time intervals. As with relaxed ankle angle, ROM between the two limbs was similar across each testing interval with the greatest variation between limbs of approximately 2° seen 24 hours post-walk.

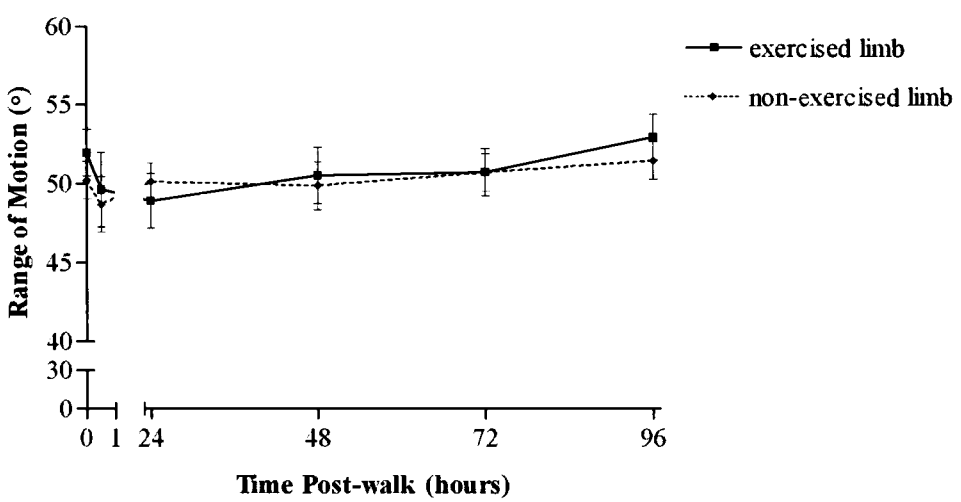
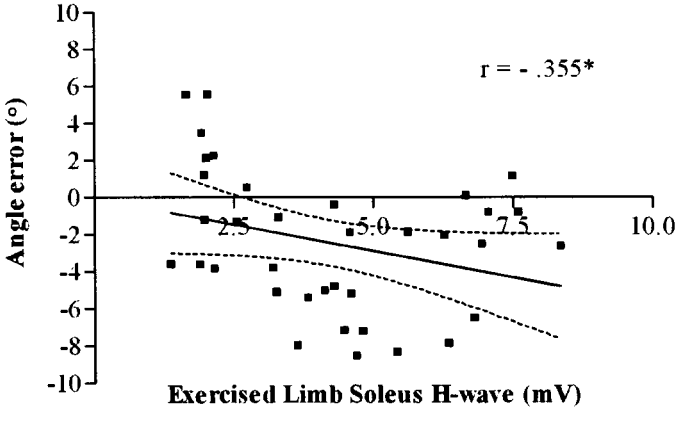


Figure 62. Ankle range of motion (n = 11) across time (mean ± SEM for the exercised and non-exercised limbs.

6.3.11 *Relationships between Variables*

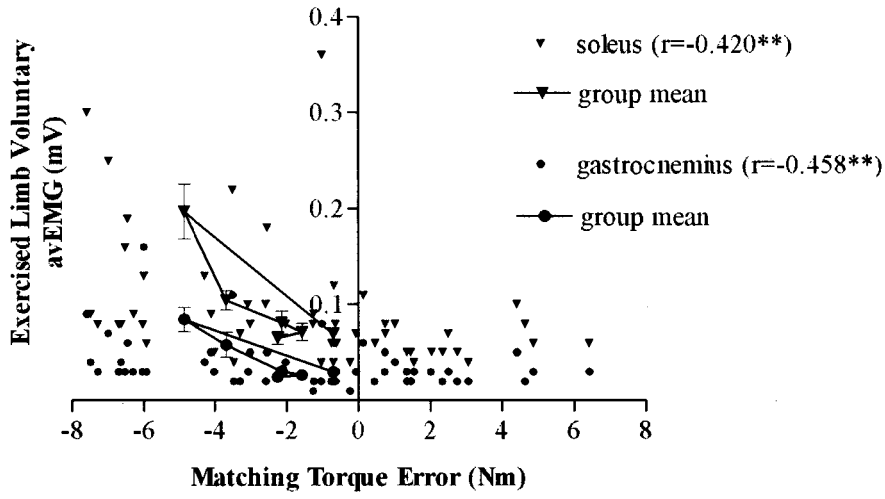
While no significant changes in H-wave amplitude occurred for the exercised limb following the walking protocol, a significant relationship ($p<0.05$) was observed between the variation in the soleus H-wave of the exercised limb, and the error produced during the position perception task when matching to the non-exercised ($r = -.355$) limbs (Figure 63). The negative relationship indicating that, as H-wave amplitude increased, the error produced during the matching task became more decreased. A significant relationship ($p<0.05$) was also apparent between the gastrocnemius H-wave amplitude of the exercised limb and the error produced during the position perception task when the exercised limb acted as the target limb ($r = -.343$).

Highly significant ($p<0.01$) relationships were demonstrated between the torque error produced during the torque perception task, and the changes in voluntary avEMG of the exercised limb (during the torque perception task). The trend indicating that as avEMG increased from baseline, the torque produced by the matching limb was reduced, causing a negative increase in the error produced. This trend also occurred for both the soleus and the gastrocnemius muscles of the exercised limb (Figure 64). A similar trend occurred in relation to soleus avEMG of the exercised limb when matching to the non-exercised limb with a significant ($p<0.05$) correlation occurring between the two variables ($r=.388$). In opposition to the results during the torque perception task when matching to the exercised limb, matching to the non-exercised limb demonstrated a positive relationship. This resulted from an increase in avEMG corresponding to an increase in the matching torque produced by the exercised limb.



* correlation significant at $p<0.05$ level

Figure 63. Relationship between soleus H-wave amplitude of the exercised limb and matching angle error for all testing time interval post-walk combined (n=36).



** correlation significant at p<0.01 level

Figure 64. Relationship between matching torque error and the voluntary avEMG of the exercised limb for all testing time intervals post-walk combined (n=60).

6.4 Discussion

While much of the earlier work relating to eccentric exercise focused on the time course changes in soreness, strength and CK efflux (Abraham, 1977; Newham, Jone et al., 1983), more recent works have begun investigating the effects of eccentric exercise on functional and proprioceptive tasks (Sargeant & Dolan, 1987; Saxton et al., 1995; Weerakkody, et al., 2003). As demonstrated during previous investigations, following a 60-minute downhill backward walking protocol subjects typically experienced DOMS in the 24-96 hour period following exercise (Chapter 4; Jones et al., 1997). While not specifically measured in the present investigation, subjects often reported soreness that was most intense when rising in the morning or after a period of non-weight bearing (for example: after sitting). Despite experiencing pain post-walk, subjects were able to complete the testing procedures without undue difficulty. The aim of the present investigations therefore was to examine the influence of downhill backward walking on both functional and proprioceptive responses.

6.4.1 *EMG responses*

6.4.1.1 *Reflex EMG responses*

Similar to previous work by Avela, Kyröläinen and Komi (1999), the present findings demonstrated no significant changes in the H-reflex following a bout of eccentric

exercise. While the fore-mentioned authors found H-wave peak – to – peak amplitude to be significantly reduced during and immediately following passive stretching of triceps surae, recovery to baseline occurred within 4 minutes of completion of the stretching protocol. Based on this time line it would be expected that any change in H-wave amplitude induced by the current exercise protocol would have resolved by the first post-walk testing interval conducted 0.5 hours (30 minutes) following exercise. However, it may be possible that disruption to the muscle spindles within the exercised muscle did occur; yet no changes in the H-wave were recorded due to the nature of the test. Stimulating the tibial nerve evokes responses from both the Ia afferents (H-wave) and the α-motoneurons (M-wave) associated with the innervated muscles. While the M-wave represents the activation of motor units, the H-wave represents the reflex response to the orthodromic volley travelling in the Ia fibres originating in the muscle spindles. As the stimulation intensity increases, the amplitude of the H-wave is reduced until it is eventually absent due to the antidromic impulses from the motor units. Thus, while the peak M-wave represents the maximal recruitment of the motor units, the peak H-wave recorded represents the maximal response of the Ia afferents before the antidromic effect occurs. Thus the H-wave represents a measure of the potency of the Ia afferent reflex pathway, rather than the spindle apparatus per se and therefore a change in the H-wave would only be expected if there was a post activation depression of the α-motoneurons (Hultborn et al., 1996; Wood, Gregory & Proske, 1996).

During voluntary muscle activation, motor unit recruitment occurs in the order of ascending motoneuron size (Olson et al., 1968), therefore during a low intensity submaximal contraction force generation occurs largely due to the recruitment of slow twitch type I motor units. As previously explained (see section 4.4) the exercise protocol employed in the current investigation was considered to be of relatively low intensity, or submaximal, in nature and would therefore have been expected to recruit lower threshold motor units preferentially. However, it has been suggesied that during force generation of stimulated muscle, recruitment order is reversed, with Type II units having a lower threshold for activation via electrical stimulation (Davies, Weigner & Young, 1993) than Type I units. Therefore, the possibility exists that if the Ia afferents disrupted following the exercise protocol were not contributing to the recorded H-wave either pre or post-exercise during the stimulation process before the H-wave was reduced by the increased motor unit recruitment associated with electrical stimulation, no difference in the peak H-wave would result. Investigation of H-wave amplitude

following an exercise protocol that was maximal or fatiguing, and would recruit a greater proportion of high threshold motor units, would be more likely to have results in H-wave differences post-exercise. Such a result being reported previously following a fatiguing exercise protocol of triceps surae (Pougnault, 2002).

6.4.1.2 *Voluntary EMG responses*

The increases in voluntary EMG post-exercise are unlikely related to the factors contributing to the soreness experienced by the subjects, given that the increases in voluntary EMG were recorded 0.5 hours post-walk, well before the typical onset of soreness (see also Chapter 4). Furthermore, in the present investigation the degree of increase in voluntary EMG seen 0.5 hours post-walk was not an indicator for the severity of soreness reported 24 - 48 hours post-walk. The increase in avEMG recorded during both the maximal voluntary strength and torque perception protocols can be explained by a change in the recruitment strategy of the exercised muscles, rather than alteration in the activation potential of the muscle, or changes associated with muscle soreness.

This conclusion is based on the lack of alteration in either the CAR or M-wave amplitudes, while significant variations in voluntary EMG were recorded during both the maximal and submaximal tasks. Similar to the findings of Saxton et al (1995), subjects in the present study demonstrated a pronounced muscle tremor (approximately 10 - 12 Hz) in the exercised limb following the walking protocol at 0.5 hours, which was reduced or absent by 24 hours post-walk. Considered to occur as a consequence of motor unit synchronisation, muscle tremors are known to result in an increase in the peak – to – peak EMG (Furness, Jessop & Lippold, 1977). Similar EMG responses have been observed in tremors associated with fatiguing exercise (Rodacki, Fowler & Bennett, 2002; Viitasalo, Hamalainen, Mononen, Salo & Lahtinen, 1993). Tremors with a frequency between 7 and 12 Hz are generally referred to as a physiological tremor, with the enhancement of EMG amplitude occurring through the operation of the segmental stretch reflex producing rhythmic contractions (Aminoff, 1998, p. 534). This is further supported by the observations that the strongest tremors and the greatest increases in voluntary EMG in the present investigation occurred at the same time interval as the trend for an increase in the soleus H-wave amplitude, and tendon tap reflex EMG responses for both soleus and gastrocnemius.

While it appears unlikely that the increases in voluntary EMG were the result of an alteration in the central activation of the exercised muscle, it cannot be ruled out as a contributing factor. Previous investigations by Kent-Braun and Le Blanc (1996) have demonstrated that superimposed-high frequency trains of stimuli are a more sensitive indicator of maximal activation potential than either superimposed single (as employed in the current study) or paired stimulus techniques. Given this, the possibility exists that as least part of the voluntary EMG increase recorded post-walk may have occurred as a result of an increase in motor unit recruitment. The CAR assessed using the three techniques (single, paired and train stimuli) however accounted for only a 4% difference in subjects' activation potential, much less than the 30% plus increase in voluntary EMG recorded during the present study.

It is also possible that the reduction in voluntary EMG recordings 48 – 96 hours post-walk may have resulted from inhibition of voluntary activation, not detected using single stimulus twitch interpolation for the same reason as those outlined for the increases in voluntary EMG previously. The absence of a change in peak M-wave amplitude or the CAR at these same time intervals however indicates that inhibition was not an major factor. This supports the previous work of Behm et al. (2001) who found no direct relationship between reductions in force, central activation and voluntary EMG following eccentric exercise. It should also be noted that while single twitch interpolation is not as sensitive as train stimuli in detecting central activation changes, it is sensitive to detecting a change in the ratio following an exercise intervention (Kent-Braun & Le Blanc, 1996). Thus had either an increase or decrease in central activation strategies of the exercised muscle occurred post-walk, a corresponding change in the CAR should have been recorded. What remains unclear is if damaged muscle fibres with a reduced force generating capacity are still able to transmit action potentials and therefore contribute to surface EMG investigations. While it has been suggested this is the case (Brown et al., 1996; Warren et a., 1993), a decrease in voluntary EMG due to muscle fibre damage would be expected even where the central activation ratio remains unchanged, such as that reported in the present study, if action potentials are not transmitted along fibres damaged following eccentric exercise. This being the case it would also be expected that the fibre type damaged may contribute to the changes in post-exercise EMG given that EMG amplitude has been shown to correlate with the number and type of motor units activated within the recording area (Suzuki, Conwit, Stashuk, Santarsiero & Metter, 2002).

At present debate exists as to whether preferential damage occurs to slow or fast twitch muscle fibres in responses to eccentric exercise (Appell, Soares & Duarte, 1992; Armstrong et al., 1983; Leiber & Friden, 1999; Mair et al., 1992). While the findings of the present study do not contribute to the body of knowledge on this question, it does add to the known literature in relation to EMG changes following eccentric exercise. Investigators employing an exercise protocol of a maximal nature have generally reported no change in post-exercise EMG responses (Day et al., 1998; Deschenes et al., 2000; Pearce et al., 1998). In contrast the present study and previous work employing exercise protocols consisting of submaximal eccentric contractions have demonstrated increases in EMG activity post-exercise (Kroon & Naeji, 1991; Newham et al., 1983). It is therefore suggested that the differences in EMG data obtained following eccentric exercise may be related to the recruitment pattern employed during the exercise protocol, resulting in preferential damage to the slow, Type I motor unit population.

6.4.2 *Voluntary Strength Following Eccentric Exercise*

Similar to previous work by Warren, Ingalls et al. (1999), the current study demonstrated reductions in both voluntary and stimulated torque in the absence of changes in M-wave amplitude. For this to occur it requires that the force generating capacity of the muscle be reduced without a reduction in activation potential (Kent-Braun, 1997). Thus, the plasmalemma and t-tubles of the exercised muscle fibres are still able to transmit action potentials in the presence of large reductions in torque output (Ingalls et al., 1998). Given that damage and disruption to the contractile mechanisms (Crenshaw et al., 1994; McNeil & Khakee, 1992) of eccentrically exercised muscle has been well demonstrated, it seems reasonable for this observation to occur. Additionally, while the time course for recovery was different, the current study demonstrated similar levels (approximately 20%) of disruption to both voluntary and stimulated torque. From this, the reduction in the force generating capacity of the exercised muscle occurs at a point peripheral to the point of stimulation (Baker, Kostov, Miller & Weiner, 1993; Bigland-Ritchie, Jones, Hosking & Edwards, 1978).

In recent years, it has been suggested that the major component of the initial strength loss following eccentric exercise results from failure of the excitation-contraction coupling process (for a review see Morgan & Allen, 1999). The loss of contractile

proteins and disruption to the contractile mechanics of the muscle prolonging the recovery of strength while regeneration of the damaged muscle begins to occur (Balnave & Allen, 1995; Warren et al., 2001). While the exact mechanisms for the strength losses reported cannot be elucidated from the present results, they do lend support to the idea of multiple factors influencing strength loss following eccentric exercise. Had the time course of recovery been the same for both stimulated and voluntary torque it could have been concluded that the torque declines occurred solely due to failure of the excitation-contraction coupling process. However, since reductions in voluntary torque outlasted the reductions in stimulated torque, the declines must be attributed to additional mechanisms.

6.4.3 *Vertical Jump Performance following Eccentric Exercise*

Similar to the findings of Byrne and Eston (2002), a reduction in vertical jump height was observed following a bout of eccentric exercise. Byrne et al. (2002) demonstrated a mean reduction in vertical jump height of approximately 5% of pre-exercise levels following 100 barbell squats at 70% body mass load. This value being considerably less than the 25% reported in the present study, and the 16% reported previously by Farr et al. (2002) following a downhill walking protocol. All three studies however, demonstrated a 3-4 day time course for recovery of vertical jump performance following the exercise protocol.

The study of Byrne and Eston (2002) gave support to previous work indicating that elastic energy may be responsible for the maintenance of vertical jump performance when the force-generating capacity of a muscle is otherwise reduced by the effects of EIMD (Hortobagyi, Lambert & Kroll, 1991). Their results showed a loss in strength of approximately 20% immediately following eccentric exercise compared with a 5% decline in jump performance. This idea of elastic energy contributions was also concluded based on the additional observations of a greater deficit in squat jump performance compared with countermovement and drop jump performance following exercise. Both the countermovement and drop jump involving an eccentric-concentric muscle action versus the concentric only action of the squat jump, contributing to greater power production in the pre-stretched muscle via elastic energy storage within the muscle and tendon (Bobbert et al., 1996). This elastic energy *or* strain energy principle, is based on the theory that when a muscle is stretched forcibly prior to shortening (contracting) as commonly seen in the stretch-shortening cycle, the parallel

and series elastic components of the muscle store energy. When released in combination with the tension produced by the muscles contractile elements, it produces elastic recoil that results a stronger and more powerful shortening contraction than that of concentric tension alone (Kreighnaum & Barthels, 1990, p. 77; Kuitunen, Komi & Kyröläinen, 2002).

As only the countermovement vertical jump was performed as a criterion measure in the present study the results obtained cannot be directly applied to support or refute the idea of a maintenance in elastic energy storage and reutilisation. It should be noted however, that unlike Byrne and Eston (2002), the declines in vertical jump performance were greater than those seen for maximal voluntary torque. This is similar to the observations of Farr et al (2002) who noted a peak decline in countermovement vertical jump height of 16% (from baseline) compared to a reduction in maximal voluntary isometric strength of 9%.

Based on the literature and the current results it appears that three main possibilities exist as to why vertical jump performance is decreased following eccentric exercise. The most likely reasoning relates to the loss of strength associated with EIMD. As vertical jump performance is closely correlated with both force and power (Ashley & Weiss, 1994), it seems reasonable that a reduction in maximal voluntary strength would alter vertical jump performance. A similar pattern in the time course of recovery for both strength and jump height were seen in the present study.

The significant changes in both proprioceptive function and voluntary EMG observed in the present study may have further contributed to a decline in jump performance. It has been shown that if the control or co-ordination of the movements performed during the jump task is compromised, then the maximal height achievable by that movement will be compromised (Bobbert et al., 1996). The tremor experienced by subjects post-walk coupled with the alterations in both strength, joint position, and range of movement, may have resulted in alterations in neural input and therefore the control of the vertical jump pattern performed (Rodacki et al., 2002). While it has been shown that a generalised motor program exists for the performance of a vertical jump, the output of that motor program (that is jump height) is dependent on the settings of that program, in particular the amplitude and timing of the control signals (van Zandwijk, Bobbert, Munneke & Pas, 2000).

Finally, if the countermovement of the jump pattern itself is altered then the maximal height achievable will also be influenced (Bobbert et al., 1996). In the present study, the actual depth of the countermovement was not strictly controlled and therefore the possibility exists that if the depth of the countermovement was altered in the post-exercise period, then jump height would also be affected. The increases in muscle soreness, resting tension, and muscle compliance of the plantarflexors are factors that may have influenced the depth of the countermovement performed post-walk, thus decreasing the height of the subsequent vertical jump (Hunter & Marshall, 2002; Proske & Morgan, 2001). The degree to which each of the above factors influenced the eventual drop in vertical jump performance however can only be elucidated with further testing of biomechanical and physiological parameters.

6.4.4 *Functional Proprioceptive Changes*

6.4.4.1 *Torque Perception*

Similar to the changes observed in 'sense of effort' following fatigue (Jones & Hunter, 1983), the present study showed evidence of a significant increase in 'sense of effort' during a force perception task following eccentric exercise. This was indicated by an increase or overestimation in the matching force produced in the non-exercised limb to a target force in the exercised limb, and the reverse when matching to the non-exercised limb. The findings are consistent with those of Carson, Reik and Shahbazpour (2002) and Proske et al. (2003) following eccentric exercise of triceps and biceps brachii. Force perception during effort is controlled by both peripheral and central components. Peripherally, the Golgi tendon organs provide afferent feedback on muscle tension, while the centrally mediated 'sense of effort' originates from corollary discharge of the motor command and descending neuronal output controlling active muscles (McCloskey, Gandevia, Potter & Colebatch, 1983).

It has previously been suggested that force perception errors following eccentric exercise may result from changes in the functioning of the Golgi tendon organ (Saxton et al., 1995). However, results from the present study demonstrated significant changes in the force perception while the responses of the tendon organ appeared to remain unaltered. Following the eccentric exercise protocol, tendon and H-reflex responses of both the EMG from soleus and gastrocnemius, and the torque output of the plantarflexors remained stable during an Achilles tendon tap and H-reflex protocol.

These results combined indicate that the responses of both the muscle spindles and the Golgi tendon organs remained unaltered. As the Golgi tendon organ continuously signal information about muscle tension, any changes in signalling post-walk would be expected to simulate an increase or decrease in active muscle tension. An increase in signalling would be expected to result in inhibition of the α-motoneruons, similar to that seen with autogenic inhibition, therefore decreasing the response of the tendon tap reflex. A decrease in the signalling, decreasing inhibition and increasing the tendon tap reflex response (Kingsley, 2000, p. 216; Martini, 1998,p. 438; Pritchard & Alloway, 1999, pp. 11-112). What cannot be estimated however, is the degree to which synaptic inhibition or excitation may have had on the tendon reflex pathways, given that both the muscle spindles and the Golgi tendon organs act on the α-motor neurons (Enriquez-Denton et al., 2002; Nichols, 1999). Gregory, Brockett, Morgan, Whitehead and Proske (2002) reported that signalling from tendon organs remains relatively stable during either active or passive movements in the presence of disruption to the force generating capacity from both eccentric and fatiguing exercise. It has been estimated that the peripheral input from the tendon organ contributes an estimated 30% to the total sense of effort during force perception (Cafarelli & Bigland-Ritchie, 1979; Cafarelli & Kostka, 1981). In the context of the current investigation, this means that large alterations in the functioning of the tendon organs would be required to have a significant influence on force perception following eccentric exercise.

Based on results from force perception tasks following fatiguing exercise, the increase in matching forces are generally associated with an increased 'sense of effort' corresponding to a decrease in the maximal force generating capacity of the exercised muscle. That is, the greatest errors in matching are recorded at the same time intervals as the greatest reductions in the maximal voluntary strength of the exercised limb (Cafarelli & Layton-Wood, 1986). While a significant relationship between torque error and maximal voluntary torque of the target was recorded in the current study (to exercised limb as target: $r=.530$ and non-exercised limb as target $r=.464$: $p<0.001$), the greatest matching error was recorded 0.5 hours post-walk, while the greatest reductions in strength were recorded 48 hours post-walk. The timing of the errors in force perception however were parallel and strongly correlated with the changes in avEMG recorded for the exercised limb post-walk. As surface voluntary EMG is considered to be an indication of descending motor neuron command (Jones, 1986), the increase in avEMG observed during the torque perception task could be considered to represent an

increase in the central drive of the muscle. The avEMG of both soleus and gastrocnemius of the exercised limb being significantly increased 0.5 hours following the eccentric exercise protocol, with soleus avEMG still increased 24 hours post-walk, observable during both the matching and the voluntary strength task. During a matching task, the increased central drive for the exercised limb is transferred as an increased 'sense of effort', resulting in either an overestimation (when the target limb) or underestimation (when the matching limb) of the matching force. As the central drive is returned to normal or baseline values, so too does the error produced during the force perception task (Carson et al., 2002).

As previously mentioned, motor unit synchronisation manifested as a muscle tremor was observed for all subjects post-walk in the exercised limb. This tremor may have produced a similar effect to that observed when vibration is applied to the active muscle of a target limb. During a force perception task, vibration results in an increase in the matching torque error (Cafarelli & Kostka, 1981). Vibration also resulted in increase in EMG amplitude as a result of motor unit synchronisation (Jones & Hunter, 1985), similar to the observations in the present investigation. The results of vibration based experimentation with fatigue also supports a strong centrally mediated control of force sensation (Cafarelli & Layton-Wood, 1986) as suggested by the current data. What cannot be clearly distinguished from the current results is the contribution that an increased 'sense of effort' versus motor unit synchronisation would have on the error produced during the matching task. The overall error produced in the period following eccentric exercise is likely a combination of both phenomenon.

6.4.4.2 *Position Perception*

Similar to previous findings (Saxton et al., 1995), the current study demonstrated a significant alteration in angle position perception when matching a plantarflexed ankle angle following a bout of eccentric exercise. The lack of any change in the control dorsiflexed position (in the current study) supporting that the change in the plantarflexed position was the result of the exercise and not daily variations. While it has been suggested that such errors may result from disruption of muscle spindle function (Brockett et al., 1997), the present results suggest that this is not the case with no change in either the H-reflex or the tendon tap reflex being recorded. It has also been suggested that a change in position sense may be related to a change in the resting joint angle and range of motion. This was also not demonstrated in the present findings

with both parameters unaltered following the exercise protocol. However, the significant correlations between the angle error, H-reflex and tendon reflex responses recorded indicated that while neither reflex appears to be significantly altered, minor variations (but not significant changes) may be sufficient to influence proprioceptive responses.

Work relating to the strength loss observed following eccentric exercise has demonstrated that an increase in passive muscle torque occurs immediately following exercise, remaining elevated up to 96 hours post-exercise (Whitehead et al., 2001). This is accompanied by a shift in the muscle's optimum length for active tension due largely to sarcomere disruption. Additionally, increases in muscle stiffness (Howell et al., 1993) and swelling (Clarkson et al., 1992) have also been shown to occur in response to eccentric exercise. While no definitive conclusions can be made, the results suggest that a change in passive tension and compliance of the exercised muscle following the walk protocol may partly contribute to the disruption in position sense post-exercise.

As with torque perception, it is also possible that the muscle tremor observed following the exercise bout may have influenced the ankle position perception. In subjects where spindle function is impaired by neuropathy the error produced during an angle-matching task with vibration is less than that in unaffected subjects (van Deursen, Sanchez et al., 1998). Vibration of the muscle tendon is also shown to produce an illusion of movement, similar to that expected when a stretch is applied to the muscle (Goodwin, McCloskey & Matthews, 1972). The muscle tremor experienced by subjects in the current study replicating a vibration intervention and therefore influencing position perception in a similar manner as if vibration had been applied to the muscle during the matching task.

6.4.4.3 *Testing Limitations*

It must be highlighted that testing in the present investigation was focused on triceps surae due to the ease with which surface EMG could be employed. It is therefore unknown to what extent changes may have occurred in the other muscles that act to plantarflex the foot. Peroneus longus, peroneus brevis, plantaris, tibialis posterior, flexor hallusic longus and flexor degitorum longus all act along with gastrocnemius and soleus to plantarflex the foot (Martini, 1998, p. 358-359; Spence & Mason, 1992, p. 326). It is assumed that these muscles were active during the exercise protocol and

contributed feedback during the proprioceptive tasks. What cannot be stated is the degree to which these muscles were influenced by the exercise protocol and the extent to which proprioceptive and activation responses of these muscle were different from those recorded in gastrocnemius and soleus. Further investigation of these muscles using this model of EIMD may contribute further to the understanding of the functional proprioceptive changes observed in the present study.

6.5 Conclusions

It appears unlikely that changes in proprioception following a bout of submaximal eccentric exercise occur solely as a result of disruption to the functioning of the key muscle receptors: the muscle spindles and Golgi tendon organs. The reflex response from both receptors, remaining stable following downhill backward walking. The errors are more likely the result of alterations in the passive tension and compliance of the exercised muscle, and changes in 'sense of effort' in response to the exercise. Voluntary EMG activity and strength production of triceps surae were also altered in addition to reductions in twitch peak torque. As with the changes in proprioception, it is suggested that the voluntary EMG results occurred due to an alteration in the activation pattern of the exercised muscle. In contrast, reductions in the force generating capacity of the exercised muscles appear to represent peripheral alterations within the muscle. The changes in proprioception and muscle activation, along with the reductions in maximal voluntary torque, are likely to contribute to observed reductions in performance of a countermovement vertical jump.

CHAPTER 7 PROPRIOCEPTIVE RESPONSES AND MUSCLE ACTIVATION OF TRICEPS SURAE FOLLOWING A REPEATED BOUT OF DOWNHILL BACKWARD WALKING

7.1 Introduction

In the previous chapter alterations in proprioceptive function of triceps surae were demonstrated following a bout of downhill backward walking. The loss in accuracy of proprioception was correlated with changes in the pattern of voluntary activation of the triceps surae.

To date much of the work relating to the 'repeated bout effect' has been conducted on the biceps and quadriceps, and has focused on the protection of soreness, plasma CK and maximal voluntary strength following a second bout of similar exercise. A literature review revealed no published studies of the protective mechanism in relation to proprioceptive changes associated with EIMD. While recent work has demonstrated a protective response in regard to the altered pattern of voluntary muscle activation of eccentrically exercised muscles (McHugh et al., 2001; Warren, Hermann, Ingalls, Masselli & Armstrong, 2000), whether such an effect occurs for alterations in proprioceptive function is not known.

Understanding the extent to which adaptation occurs in response to repeated eccentric exercise has the potential to provide further understanding of the mechanisms responsible for the phenomenon of the repeated bout effect. It would also seem crucial to examine the effects of repeated eccentric exercise on proprioception as a possible contributor to the likelihood for injury for athletes during both training and competition.

The aims of this investigation therefore were to examine:

1. Whether alterations in muscle activation (surface EMG) occur following a repeated bout of downhill backward walking.
2. Whether differences in the errors produced during a proprioceptive task occur following a repeated bout of downhill backward walking.
3. Whether alterations in muscle activation (surface EMG) and proprioceptive task performance between two downhill backward walking bouts follow the 'repeated bout effect'.

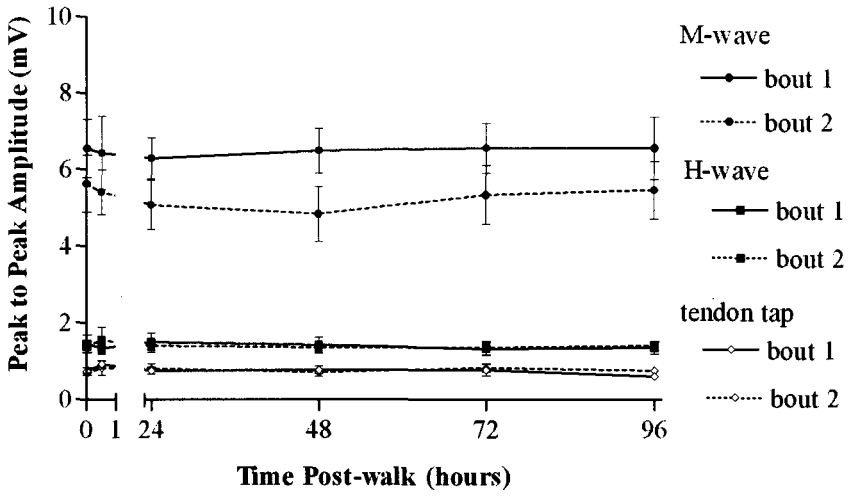


Figure 65. M-wave (n=10), Hoffmann (n = 10) and Achilles tendon (n = 8) reflex EMG responses (mean ± SEM) of gastrocnemius across time following two separate exercise bouts.

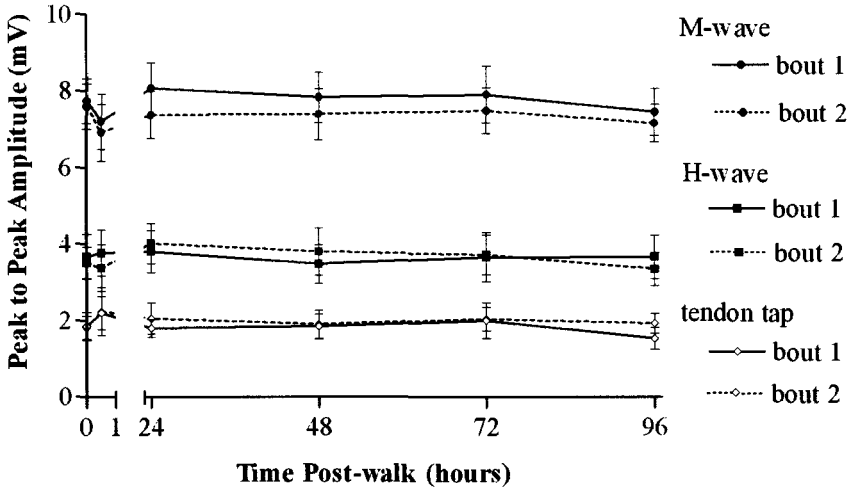


Figure 66. M-wave (n=10), Hoffmann (n = 10) and Achilles tendon (n = 8) reflex EMG responses (mean ± SEM) of soleus across time following two separate exercise bouts.

7.3.2 *Reflex Torque Responses and Contractile Properties*

Following the initial exercise bout a significant ($p<0.001$) reduction in T_0 of approximately 2.0 Nm or 20% was recorded 0.5 hours post-walk with a similar reduction ($p<0.01$) of 2.5 Nm or 24% recorded at the same time interval following the repeated exercise bout. Torque had recovered by 24-hours following both exercise bouts, with no significant differences between the two bouts at any testing time interval (Figure 67). A similar trend for a reduction in Achilles tendon tap torque was observed at the first testing time interval following both exercise bouts, although no significant

changes either from baseline or between bouts were recorded in relation to this variable (Figure 67).

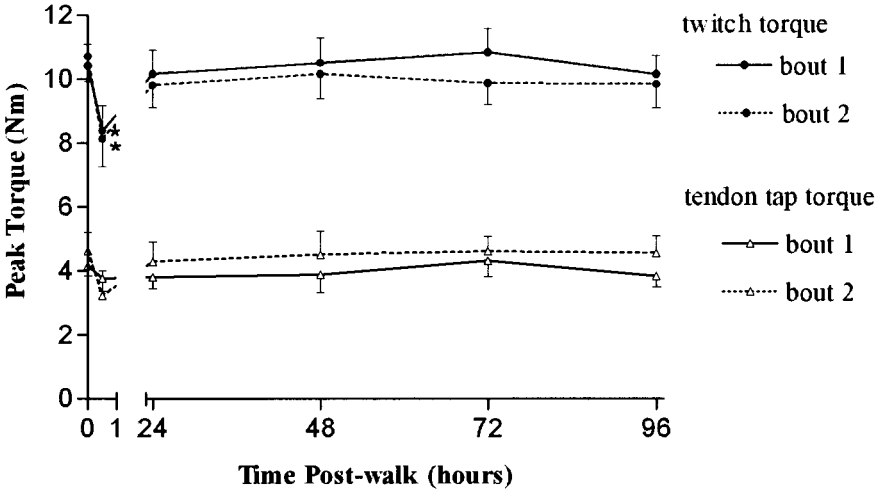


Figure 67. Stimulated twitch (n = 10) and Achilles tendon (n = 8) reflex responses of torque output (mean ± SEM) across time following two separate exercise bouts.

Following the initial exercise bout a significant reduction ($p<0.001$) from baseline 0.5 hours post-walk for TTP of approximately 0.011 ms or 11% was recorded, with an significant increase from baseline of 0.005ms or 5% recorded 24 hours post-walk. A significant increase from baseline recorded at this time interval indicating a slowing in the contractile rate of the exercised muscles (Figure 68). All other time intervals showed no significant variation from baseline. While a greater reduction for TTP of 0.015 ms or 15% was recorded 0.5 hours following the repeated exercise bout, this result did not prove significantly different ($p=0.09$) from baseline (Figure 68). Nor were differences between the two exercise bouts recorded at any time interval post-walk.

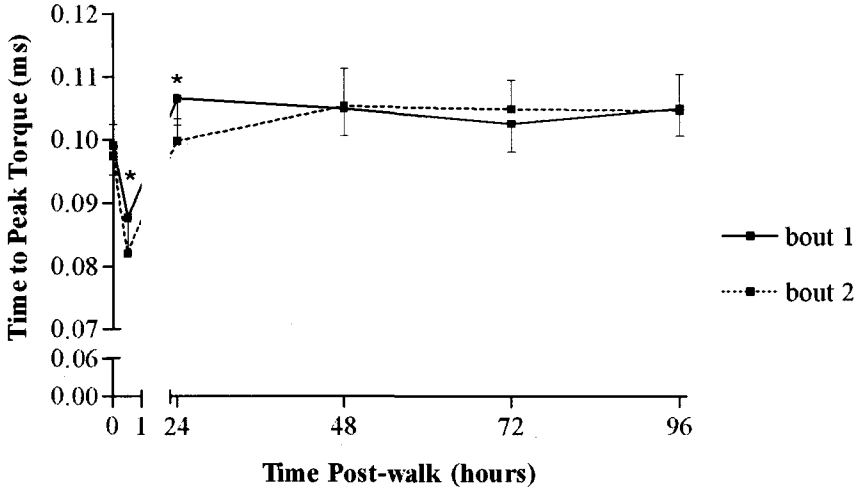


Figure 68. Time to peak (n = 10) responses across time (mean ± SEM) following two separate exercise bouts.

No significant differences from baseline or between bouts was recorded in relation to half relaxation time, with no clear trends being observed following either the initial or repeated exercise bout. The greatest deviation from baseline of approximately 0.01 ms or 10% recorded 24 hours following the initial and 72 hours following the repeated exercise bout (Figure 69).

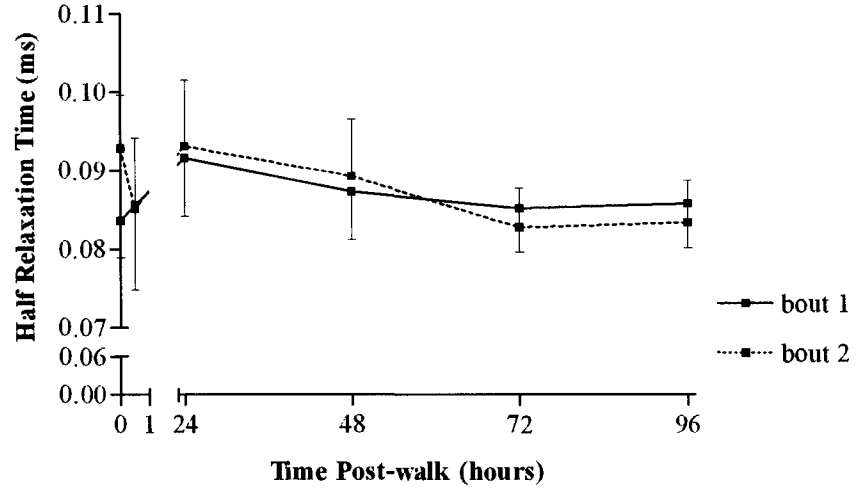


Figure 69. Half relaxation time (n = 10) responses across time (mean ± SEM) following two separate exercise bouts.

7.3.3 *Maximal Voluntary Isometric Strength*

Maximal voluntary isometric strength of the plantarflexors measured using the DAD was significantly reduced ($p<0.001$) from baseline at all time intervals post-walk

following both exercise bouts. Peak strength reductions of approximately 15 Nm (22%) following the initial exercise bout at 48 hours post-walk, and approximately 14 Nm (21%) following the repeated exercise bout at 0.5 hours post-walk (Figure 70) were recorded. While a recovery in strength was observed 0.5 - 24 hours post-walk following the repeated bout, no significant differences in responses were recorded between the two bouts with strength still reduced by approximately 10% 96 hours post-walk for both bouts. Although there was a tendency for the second bout losses to recover more rapidly than the first.

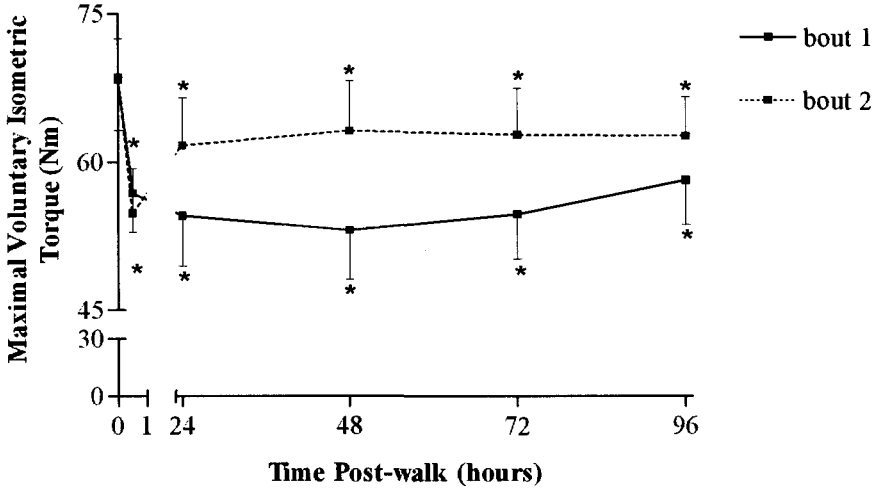


Figure 70. Maximal voluntary isometric strength (n = 12) responses across time (mean ± SEM) following two separate exercise bouts.

Following the initial exercise bout, voluntary avEMG increased significantly ($p<0.001$) in both soleus and gastrocnemius at 0.5 hours post-walk (30% and 43% above baseline respectively). At 48 hours post-walk for gastrocnemius, and 48 and 72 hours post-walk for soleus, significant decreases in avEMG from baseline were recorded representing a reduction in avEMG of 73% and 48% respectively. In contrast, no significant differences from baseline were recorded following the repeated exercise bout with the greatest variation from baseline of approximately 12% occurring 0.5 hours post-walk for both muscles (Figure 71). As reported in the previous chapter, a muscle tremor was observed in all subjects post walk for both the initial and the repeated exercise bout. The typical duration of the tremor however was less following the repeated exercise bout compared to the initial bout with most subjects experiencing the tremor at only the 0.5 hour post-walk time interval.

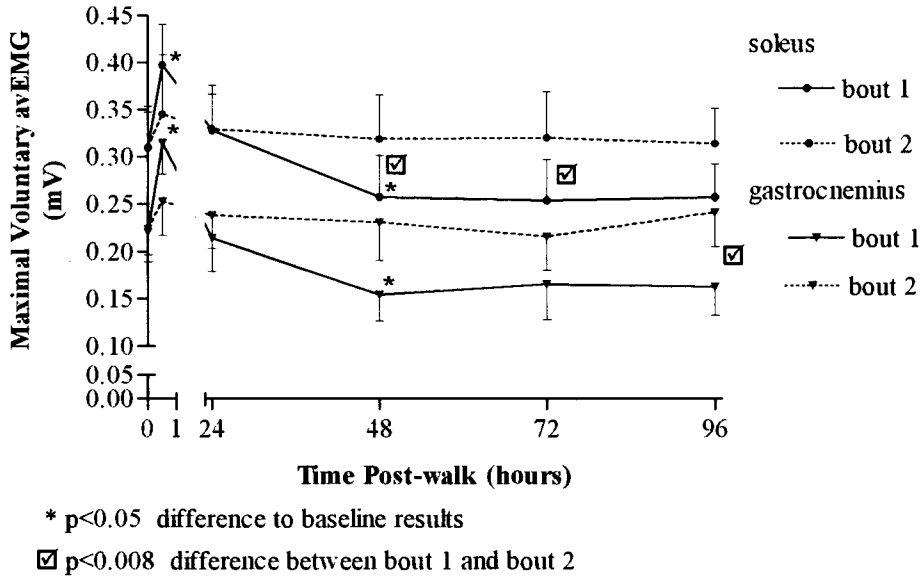


Figure 71. Maximal voluntary avEMG responses of soleus and gastrocnemius (n = 12) across time (mean ± SEM) following two separate exercise bouts.

A significant difference ($p<0.0083$) in avEMG between the two bouts occurred 48 and 72 hours post-walk for soleus, and 96 hours post-walk for gastrocnemius. The sustained reduction in avEMG observed following the initial exercise bout was not evident following the repeated exercise bout, with only one post-walk time interval falling below baseline values (gastrocnemius at 72 hours post-walk by 4%).

7.3.4 *Vertical Jump*

A significant decrease ($p<0.01$) from baseline was recorded for vertical jump height following the initial exercise bout at all time intervals between 0.5 and 72 hours post-walk inclusive, with a peak reduction of 6.2 cm (25%) recorded 24 hours post-walk. Following the repeated exercise bout however, a significant decrease ($p<0.05$) from baseline was observed only 0.5 hour post-walk with a reduction of 2.2 cm or approximately 10% recorded (Figure 72). No differences however, were recorded between the two bouts for either limb at any time interval post-walk. For both the exercised and non-exercised limb, a significant increase from baseline ($p<0.05$) was recorded 96 hours, and 72 and 96 hours post-walk respectively, with both limbs demonstrating an increase of approximately 2 cm or 7% by the final testing interval (post bout 2).

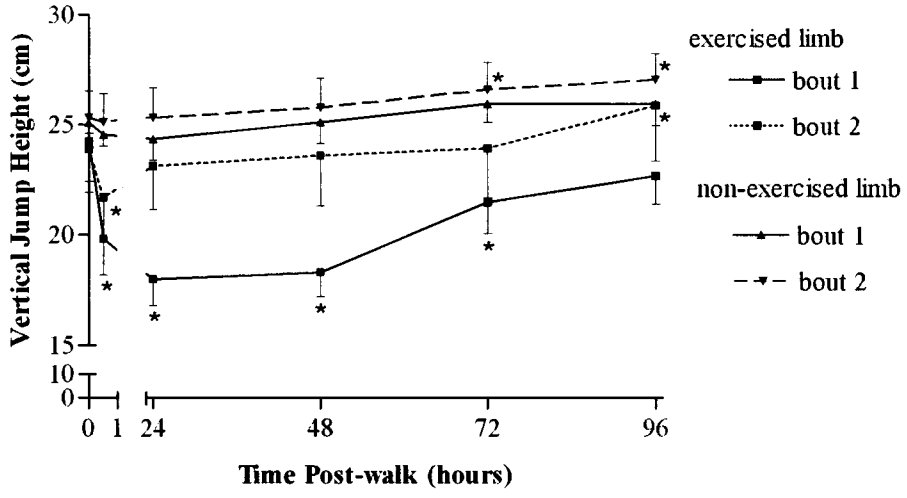


Figure 72. Vertical jump height (n = 8) results across time (mean ± SEM) for the exercised and non-exercised limbs following two separate exercise bouts.

7.3.5 *Torque Perception*

Following both the initial and the repeated exercise bouts, a significant change in the torque produced by the matching limb compared with baseline occurred (Figure 73). Where the exercised limb was the matching limb, a peak error of approximately -20% was produced 0.5 hours post-walk ($p<0.05$) compared with a 2 – 4% error during baseline testing. A similar error change of 5% (baseline) to +15 - 17 % (0.5 hours post-walk) occurring when the non-exercised limb was the matching limb (Figure 74).

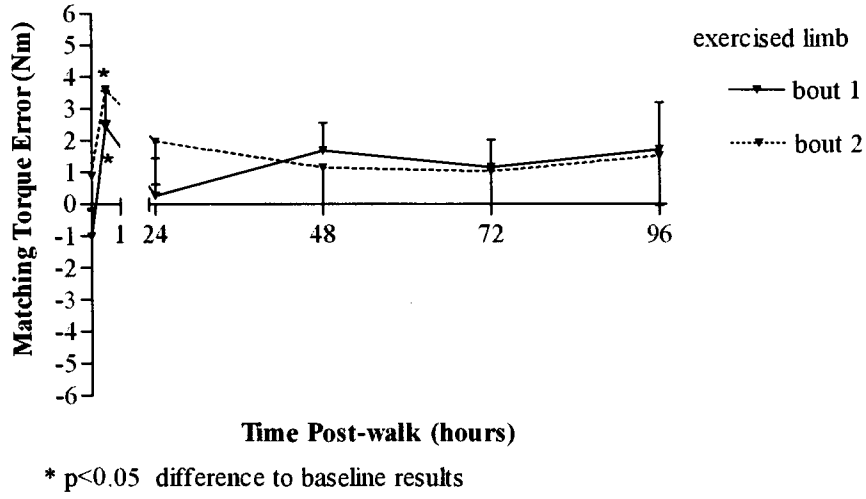


Figure 73. Torque error produced by the matching limb during the torque perception task for the exercised limb (n = 10) across time (mean ± SEM) following two separate exercise bouts

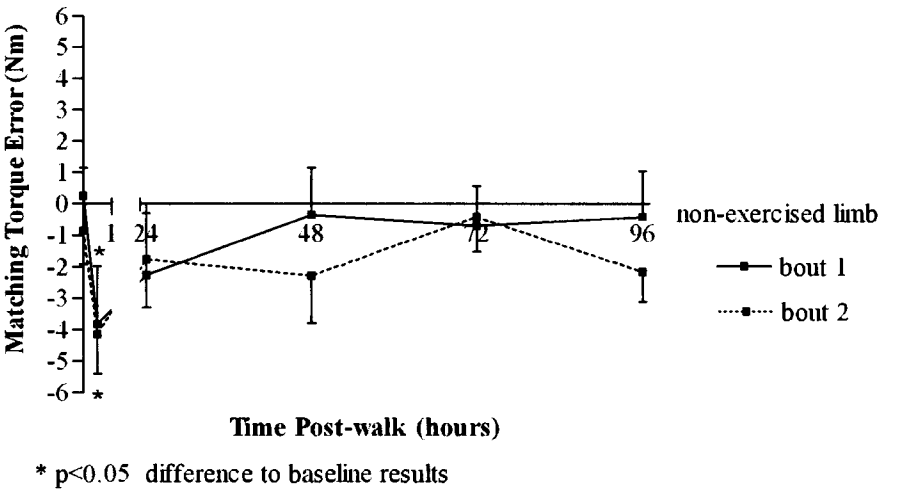
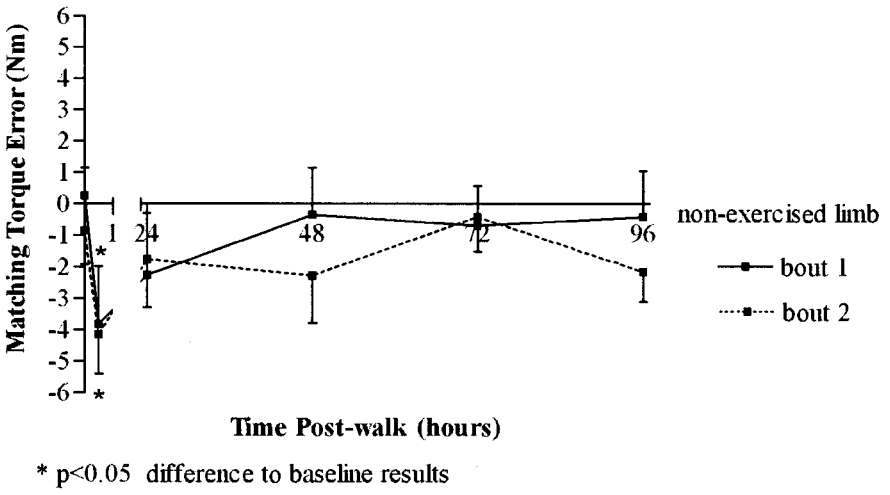


Figure 74. Torque error produced by the matching limb during the torque perception task for the non-exercised limb (n=10) across time (mean SEM) following two separate exercise bouts

During the torque perception task a significant increase ($p<0.05$) in the avEMG of both soleus and gastrocnemius was recorded 0.5 hours post-walk following both exercise bouts regardless of whether the exercised limb was the target or matching limb during the task (Figure 75 and 76). These increases were still evident 24 hours post-walk in soleus following the initial exercise bout during both the target and matching tasks (Figure 76), and gastrocnemius when the exercise limb was the matching limb (Figure 75). Following the initial exercise bout increases in avEMG of 140 - 160% occurred for the exercise limb. While these were reduced by approximately 20 – 30% following the repeated exercise bout, no significant differences between the peak avEMG responses for either soleus or gastrocnemius were recorded between bouts.



* p<0.05 difference to baseline results

Figure 74. Torque error produced by the matching limb during the torque perception task for the non-exercised limb (n=10) across time (mean SEM) following two separate exercise bouts

During the torque perception task a significant increase ($p<0.05$) in the avEMG of both soleus and gastrocnemius was recorded 0.5 hours post-walk following both exercise bouts regardless of whether the exercised limb was the target or matching limb during the task (Figure 75 and 76). These increases were still evident 24 hours post-walk in soleus following the initial exercise bout during both the target and matching tasks (Figure 76), and gastrocnemius when the exercise limb was the matching limb (Figure 75). Following the initial exercise bout increases in avEMG of 140 - 160% occurred for the exercise limb. While these were reduced by approximately 20 – 30% following the repeated exercise bout, no significant differences between the peak avEMG responses for either soleus or gastrocnemius were recorded between bouts.

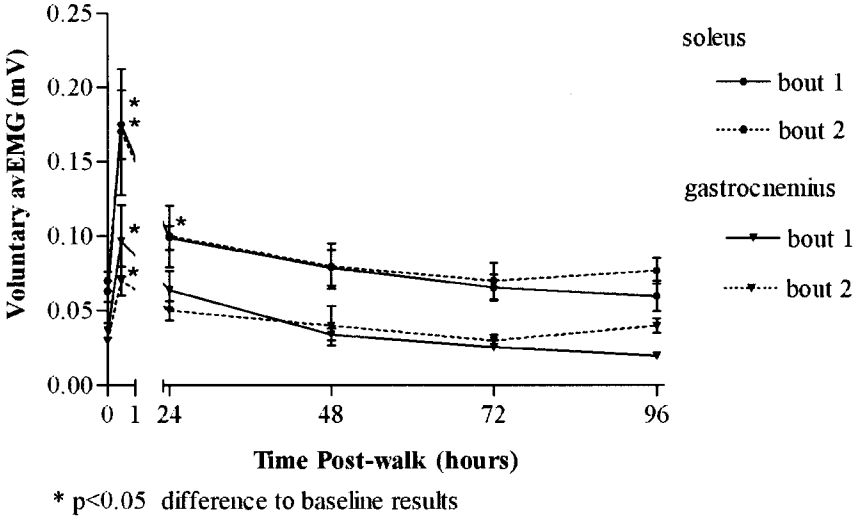


Figure 75. Voluntary avEMG responses across time (mean ± SEM) of soleus and gastrocnemius for the exercised limb (n = 10) during the force perception task where the exercised limb is the target limb following two separate exercise bouts.

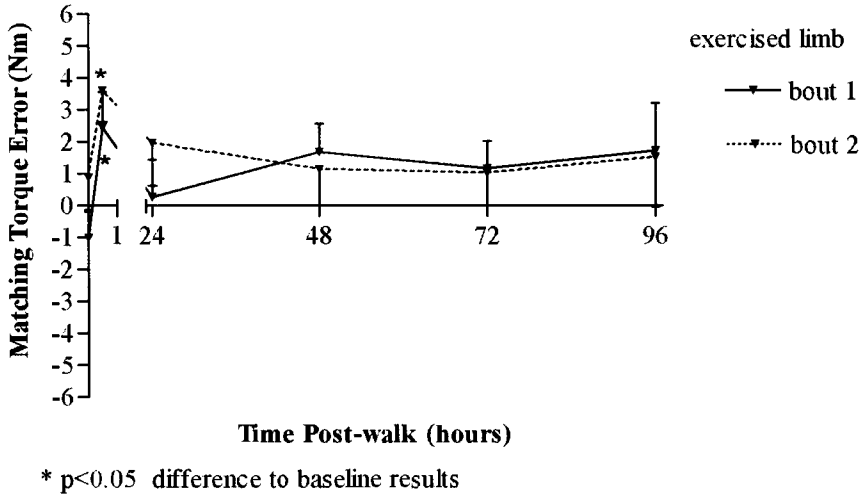


Figure 76. Voluntary avEMG responses across time (mean ± SEM) of soleus and gastrocnemius for the exercised limb (n = 10) during the force perception where the exercised limb is the matching limb following two separate exercise bouts.

7.3.6 *Joint Position Perception*

When matching for joint position with the ankle at a 10° dorsiflexed angle, no significant differences from baseline were recorded for the target or the matching limb at any time interval following either exercise bout. Greater errors were produced however when matching was conducted to the exercised limb, the average error across the two bouts being 2.8° as opposed to 1.2° when the exercised limb acted as the matching limb. When the exercised limb was the reference limb, comparisons between the target and matching angle at each time interval revealed significant differences 0.5 –

96 hours post-walk for the initial exercise bout, and baseline – 24 hours and 72 hours post-walk for the repeated exercise bout (Figure 77). No differences were observed between the two limbs when matching for dorsiflexion angle where the non-exercised limb acted as the reference limb (Figure 78).

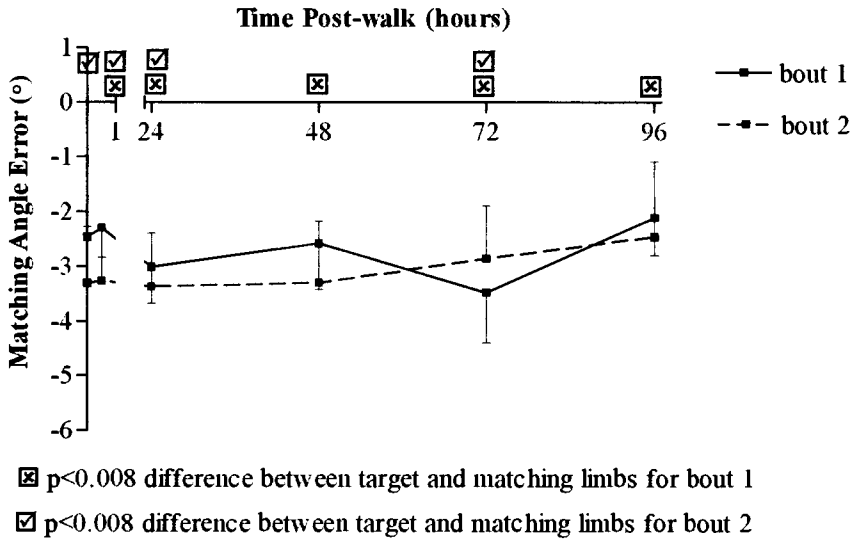


Figure 77. Dorsiflexion angle matching error (n = 12) of the non-exercised limb across time (mean ± SEM) following two separate exercise bouts.

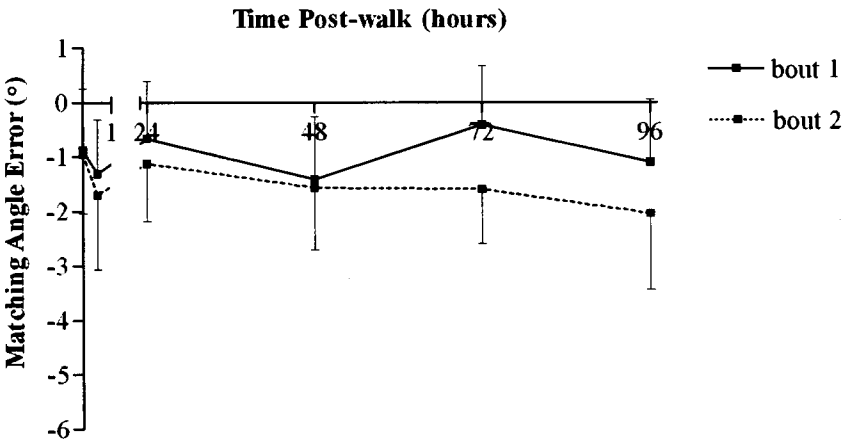
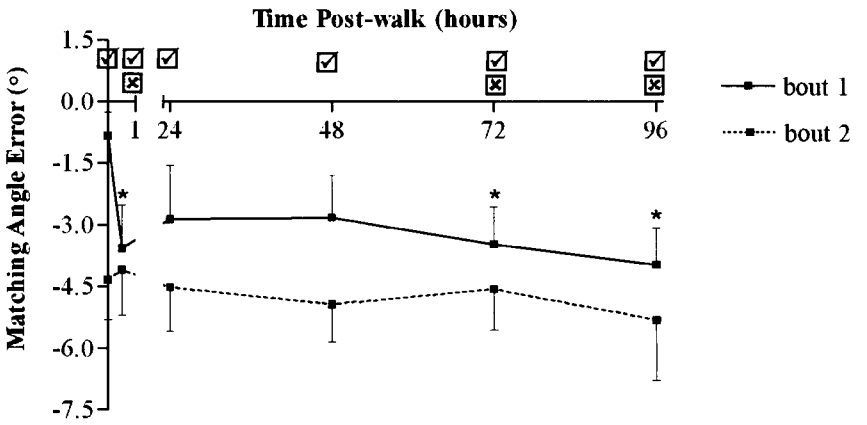


Figure 78. Dorsiflexion angle matching error (n = 12) of the exercised limb across time (mean ± SEM) following two separate exercise bouts.

In contrast, when matching to either the exercised limb or non-exercised limb with the target limb at a 10° plantarflexed angle, a significant difference from baseline for the matching limb was recorded following the initial exercise bout but not the repeated exercise bout. When matching to the exercised limb, a significant difference from baseline for the non-exercised limb was recorded 0.5, 72 and 96 hours post-walk with the greatest error of 4° observed 96 hours post-walk (Figure 79). Comparisons between

the limbs when matching to the exercised limb at a plantarflexed angle revealed significant differences 0.5, 72 and 96 hours post-walk for bout one, and at all time intervals including baseline following the repeated exercise bout.



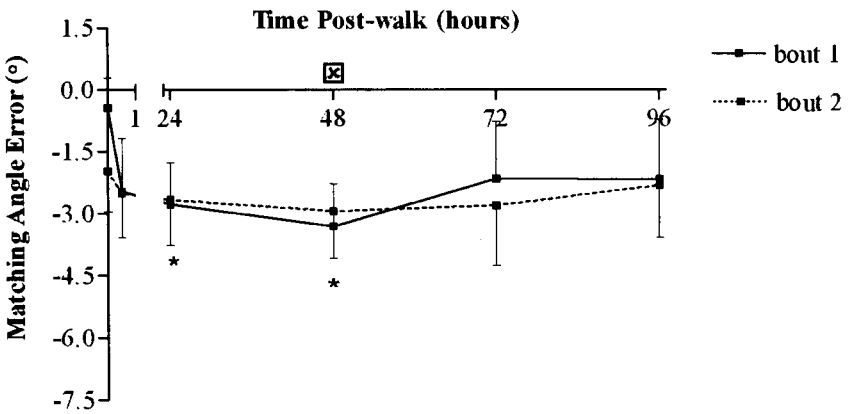
* p<0.05 difference to baseline results

☒ p<0.008 difference between target and matching limbs for bout 1

☑ p<0.008 difference between target and matching limbs for bout 2

Figure 79. Plantarflexion angle matching error (n = 12) of the non-exercised limb across time (mean ± SEM) following two separate exercise bouts.

Significant differences from baseline for the initial bout only were recorded at 24 and 48 hours post-walk for the exercised limb, when matching was conducted to the non-exercised limb, with the greatest error of 3° recorded 48 hours post-walk (Figure 80). Differences between the matching and target limb also only being recorded following the initial exercise bout at 48 hours post-walk



* p<0.05 difference to baseline results

☒ p<0.008 difference between target and matching limbs for bout 1

Figure 80. Plantarflexion angle matching error (n = 12) of the exercised limb across time (mean ± SEM) following two separate exercise bouts.

It was also noted that regardless of whether matching was conducted to the exercised or non-exercised limbs, the error produced during the matching was negative. Thus, the matching limb tended to be placed in a more dorsiflexed position compared to that of the target limb during the matching task. This trend being consistent across bouts and matching angles.

7.3.7 *Relaxed Ankle Angle*

A variation in joint angle of 0.7° and 1.7° was recorded for relaxed ankle angle following the initial and repeated exercise bouts respectively, with a general trend for joint angle to be increased at the 24 – 72 hour time intervals. While a greater variation in joint angle was observed following the repeated exercise bout no significant differences from baseline were recorded following either exercise bout, or between the two bouts at any time interval (Figure 81).

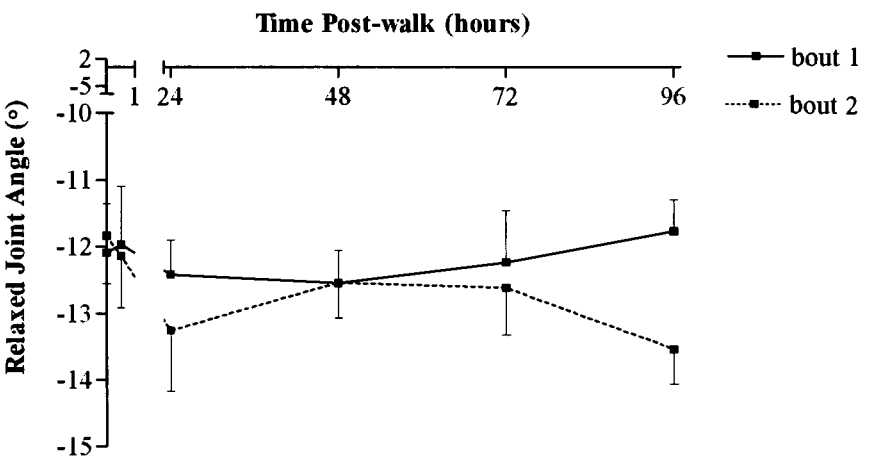


Figure 81. Results for relaxed ankle angle (n = 10) across time (mean ± SEM) following two separate exercise bouts.

7.3.8 *Range of Motion*

While there was a general trend for a greater range of motion at all time intervals following the repeated exercise bout, no significant differences occurred between the two bouts. Nor were any significant differences seen at any time interval in relation to baseline following either exercise bout (Figure 82). The smallest range of motion was recorded 24 hours post-walk, and the largest at 96 hours post-walk for both exercise bouts with recordings of 49.6 ± 1.9°, 51.3 ± 1.5°, 52.5 ± 1.7° and 52.8 ± 1.0° respectively.

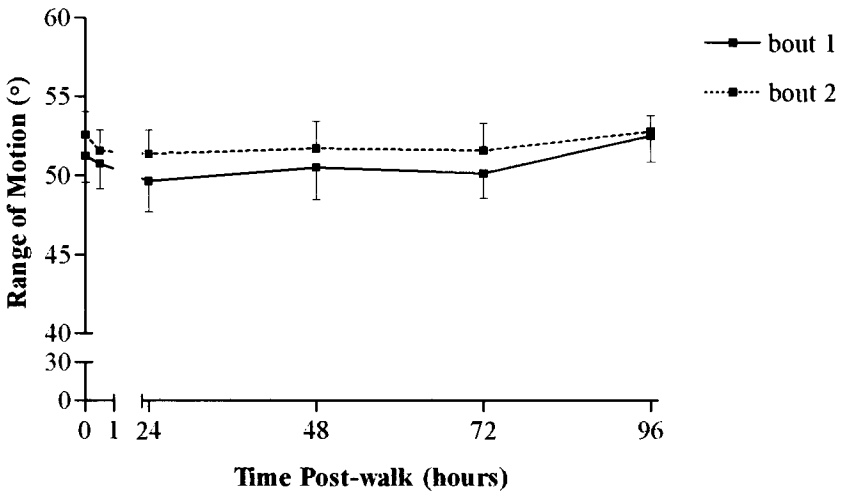


Figure 82. Results for ankle range of motion (n = 9) across time (mean ± SEM) following two separate exercise bouts.

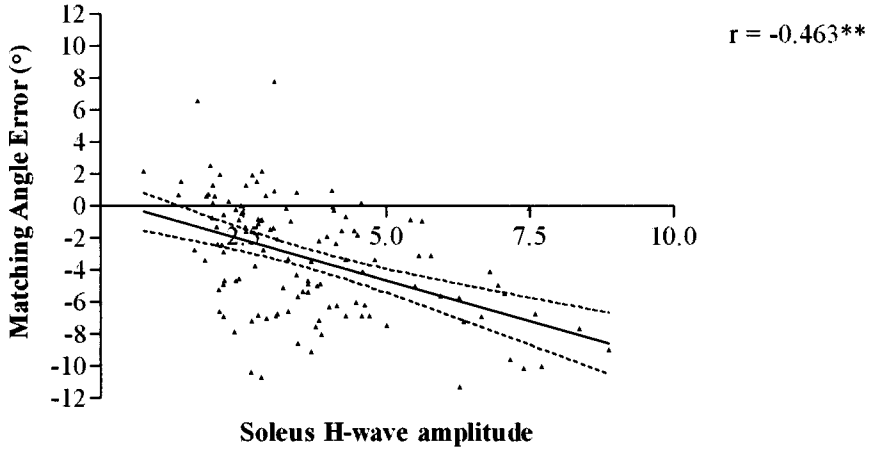
7.3.9 *Relationships between variables*

A significant negative correlation ($p<0.01$) was observed between the error produced in the angle perception task (exercised limb as target) and the H-wave amplitude of the soleus for the exercised limb, when results were considered as two separate bouts ($r = -.473$, bout 1; $r = -.499$, bout 2) or combined ($r = -.463$; Figure 83). This relationship occurring following both exercise bouts despite a significant change from baseline for angle matching error occurring following the initial exercise bout only, and no significant changes from baseline recorded at any time interval post-walk for H-wave amplitude.

The error produced during the torque perception task (non-exercise limb as target) with all results combined (Figure 84) was significantly correlated ($p<0.05$) with the avEMG of the exercised limb for both soleus ($r = -.258$) and gastrocnemius ($r = -.278$). The negative correlation showing that as the amplitude of EMG was increased, the error produced during the task was increased so as to produce an underestimation of the target torque.

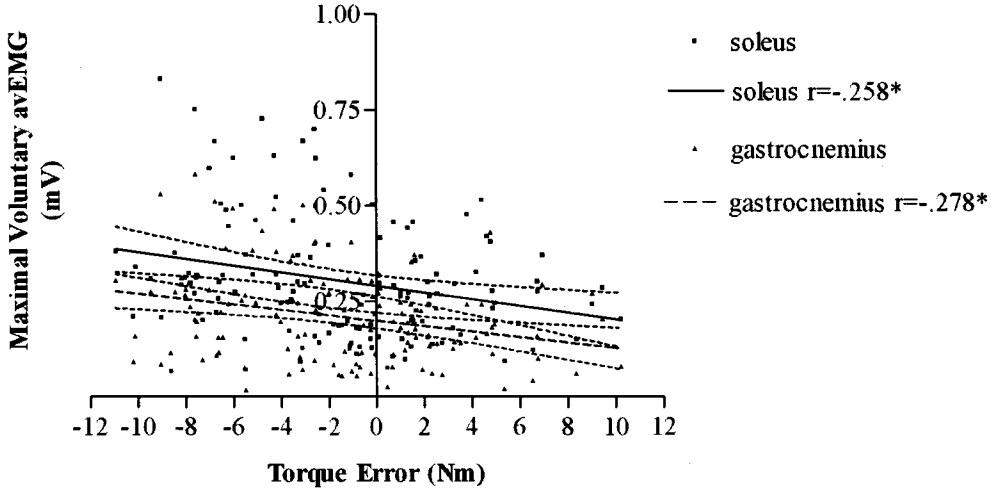
When considered as separate bouts, significant correlations ($p<0.05$) between the avEMG of soleus and gastrocnemius, and the error produced during the matching tasks of $r = -.297$ and $r = -.279$ respectively, were recorded following the initial exercise bout. Following the repeated bout however, a significant correlation between torque error and

the avEMG of gastrocnemius only was recorded (r= -.267), the correlation between torque error and soleus avEMG reduced to r = -.211 (p>0.05).



** correlation significant at p<0.01 level

Figure 83. Relationship between soleus H-wave amplitude of the exercised limb and matching angle error for all testing time intervals post-walk following two eccentric exercise bouts (n=120).



* correlation significant at p<0.05 level

Figure 84. Relationship between matching torque error and the voluntary avEMG of the exercised limb for all testing time intervals post-walk following two eccentric exercise bouts (n=132).

7.4 Discussion

The mechanisms responsible for the phenomenon of the repeated bout effect are not well understood. Many investigations relating to the protection observed with repeated eccentric contractions have concentrated on the area of adaptation to voluntary strength

loss, soreness and plasma CK. The present study of the influence of repeated eccentric exercise on proprioception is in this sense unique.

7.4.1 *Strength Changes and the Repeated Bout Effect*

As previously demonstrated by both earlier studies (Chapter 5) and other investigators (Brown et al., 1997; Nosaka et al., 2001), a protective effect was observed in relation to strength declines with repeated eccentric exercise. The repeated exercise strength declines were less than those following the initial bout, with a more rapid recovery also observed. The voluntary EMG findings of the current study lend support to the idea that the force recovery with repeated eccentric exercise may be mediated in part by neural factors (Hortobagyi et al., 1996; Komi & Buskirk, 1972). Following the repeated exercise bout, no significant changes from baseline were recorded in voluntary EMG for either soleus or gastrocnemius. This is in contrast to the significant changes recorded following the initial exercise bout attributed primarily to changes in the activation strategy of the exercised muscle. Therefore, voluntary EMG in addition to voluntary strength demonstrated a repeated bout effect when compared to the response seen following the initial exercise bout. This result is similar to that observed by Hortobagyi et al. (1998) for EMG activity of the quadriceps following repeated eccentric exercise. These investigators reporting a decrease in voluntary EMG following the initial exercise bout, and no changes following the repeated exercise bout.

It has been suggested that following an initial bout of eccentric exercise, the control strategy of the exercising muscle is altered for subsequent eccentric exercise, reducing the larger than normal mechanical stresses on active fibres. This in turn reduces the amount of fibre damage sustained during the exercise and results in a lesser degree of force loss following the repeated bout (Warren et al., 2000). While EMG activity during the walking protocol was not examined in the present investigation, the differences in post-exercise EMG results suggests that the impact of the second exercise bout was not as great as the first despite the stimulus being the identical, and thus suggests the muscle was better able to cope with the repeated exercise bout.

It has been suggested that the pattern of neural control during eccentric contractions is different to that of either concentric or isometric contractions (Enoka, 1996; Kay, St Clair Gibson, Mitchell, Lambert & Noakes, 2000). Following training programs with an eccentric component, both an increase (Hortobagyi et al., 1996; Komi & Buskirk,

1972; McHugh et al., 2001) and decrease (Bishop, Trimble, Bauer & Kaminski, 2000) in activation of the active muscle has been reported although an increase in EMG is the more common observation. Additionally, Hortobagyi, Lambert and Hill (1997) reported a greater cross education in the vastus lateralis and biceps femoris following eccentric versus concentric training of the quadriceps. Ipsilateral exercise training with eccentric actions resulted in a greater increase in strength and muscle activation of the contralateral homologous muscle group than ipsilateral training with concentric contractions. In the present study, it could be seen that while there were no significant differences between the two bouts at baseline for avEMG, differences were seen in both the soleus and gastrocnemius for at least one time interval following the repeated exercise bout. These differences occurred despite similar strength readings at the same time interval.

Whether a change in muscle activation strategy is responsible for protection of strength with repeated exercise is still open to question since, the repeated bout effect occurs when electrical stimulation of eccentric contraction eliminates the neural input of damaging exercise (Sacco & Jones, 1992). Work by Nosaka et al. (2002) has demonstrated that peripheral (i.e. muscular) adaptations were more likely to explain the protection of maximal voluntary strength with repeated exercise. To fully understand the mechanism for strength protection with respect to EMG changes occurring during the present model it would be necessary to examine changes in both strength and the pattern of muscle activation before, during and after the exercise protocol, rather than just pre – test and post – test.

Of interest is the observation that the present investigation demonstrated no repeated bout effect in relation to stimulated torque. This would suggest that similar mechanisms are responsible for strength losses following both the initial and the repeated exercise bout. While a steeper recovery of voluntary strength was observed following the repeated exercise bout, a similar level of strength loss was observed immediately post-walk. In contrast, the level of stimulated strength loss both immediately post-walk and during the 4-day recovery period remained the same between the two bouts. As it has been suggested that the initial strength loss occurs due to failure of the E-C coupling process (Warren et al, 2001), the consistent result between the bouts could indicate that the repeated bout effect does not occur in relation to this E-C coupling failure. The repeated bout effect however can be applied to the prolonged strength loss, which is

thought to occur as a result of damage and disruption to the contractile mechanisms of the muscle (Crenshaw et al., 1994; McNeil & Khakee, 1992). This would explain the finding of no differences in immediate strength loss with a repeated eccentric exercise bout compared with the observed protection of prolonged strength loss.

7.4.2 *Protection of Vertical Jump Performance*

The protective effect observed in relation to vertical jump performance was similar to that seen with respect to maximal voluntary strength following a repeated bout of eccentric exercise. That is, while reductions were observed following both exercise bouts, the time course for recovery following the second bout was shorter.

As previously discussed (section 6.4.3) a possible factor leading to vertical jump performance declines following eccentric exercise is disruption of the control or co-ordination of the movements required to execute the jump task without compromising the maximal height achievable (Rodacki et al., 2002; van Zandwijk et al., 2000). The change in voluntary EMG contributing to the initial disruption of jump performance and the delayed appearance of soreness altering jumping technique across time. This being the case, the protective effect recorded for vertical jump would be expected due to the observed protection for both voluntary EMG and soreness following the repeated exercise bout. Although protection of maximal voluntary EMG occurred during the strength task, voluntary EMG during the submaximal torque perception task was significantly altered immediately post-walk for both exercise protocols and may have influenced jump performance at the first testing time interval following both exercise bouts.

In addition to the maintenance of jumping movement, the more rapid recovery of voluntary strength would also be expected to contribute to the lesser changes in vertical jump performance. The protective effect observed for isometric maximal voluntary strength (section 6.3.4) and that previously shown for isokinetic voluntary strength (section 5.3.3.2) influencing the relationship typically seen between voluntary strength and vertical jump height (Bobbert & Van Soest, 1994).

Following a bout of eccentric exercise it was also suggested (section 6.4.3) that changes in both the resting joint angle of the ankle and the range of movement around the ankle joint may have influenced the subject's ability to perform the movement required for the

vertical jump protocol. This restriction in movement would further contribute to the decline in vertical jump height. Following the repeated bout of exercise, the variation seen in both joint angle and range of movement were less than that following the initial bout, which may have also contributed to the apparent protective mechanism seen for vertical jump performance following a second bout of eccentric exercise.

7.4.3 *Effect of Repeated Eccentric Exercise on Proprioception*

According to the repeated bout effect, a variable should show a smaller change following repeated exercise, or a more rapid recovery, compared to the initial bout. The results of the present study show that proprioceptive function does not exhibit the repeated bout effect, at least in relation to joint position perception or torque perception of the foot plantarflexors, with similar effects seen following both the initial and repeated exercise bouts.

7.4.3.1 *Torque Perception*

In the previous chapter (section 6.4.4.1) it was suggested that a possible source of the alteration in force perception following a single bout of eccentric exercise was an increase in 'sense of effort' as evident by an increase in avEMG coupled with a decrease in maximal voluntary strength. An additional hypothesised factor is the presence of muscle tremor post-exercise which could potentially influence force perception.

Following the repeated eccentric exercise bout, peak torque error occurred at the same time as the peak increase in avEMG during the force perception task and the nadir in maximal voluntary strength. Additionally, a significant increase in avEMG during the force perception task was recorded following both exercise bouts. While a significant increase in avEMG during the voluntary strength task was not observed following the repeated exercise bout, a muscle tremor was still present in 8 of the 10 subjects following the repeated exercise bout with all reporting that the severity of the tremor was less than that following the initial exercise bout. For a second time, this occurred despite the absence of any significant change in either the reflex EMG or torque produced during an Achilles tendon tap reinforcing the suggestion that the force perception errors were not the result of changes in the functioning of the Golgi tendon organs. As previously discussed (see section 6.4.4.1), the relative contribution of the tendon organ to force perception is relatively low (~30%), with signalling stable following eccentric exercise.

The extent to which this altered force perception would continue with further eccentric exercise bouts is not known, but it would seem reasonable that if further neuromuscular adaptation occurs, such as that seen with the maximal strength protocol, a follow on effect may be seen during submaximal contractions and therefore the force perception task. As much of the work relating to the repeated bout effect examines only one repeat of the initial exercise, it is not known if or when a complete adaptation to force perception, and those variables influencing it would be expected. From a training and sporting view it may be important to examine the repeated bout effect further beyond the traditionally tested two exercise bouts.

7.4.3.2 *Position Perception*

Across the variables examined in the present study, the time elapsed between the initial and repeat exercise bout was typically sufficient to allow for complete recovery of a variable where that variable had been significantly changed from baseline post-walk. This being evident by the lack of any difference between the two baselines, or any difference between the matching and reference limbs during the force perception task at baseline. For joint position perception however, a significant difference at baseline for the matching limb was observed in relation to position perception when the exercised limb acted as the target limb in a plantarflexed position following the second bout. Baseline results for bout two were more similar to the results at 96 hours post-walk for bout 1 than the initial baseline results (pre-walk bout 1). As post-walk results for both exercise bouts followed a similar pattern, this meant that significant differences from baseline were only recorded following the initial exercise bout.

Earlier it was suggested that changes in the position perception task following exercise may reflect alteration in multiple physiological measures (see section 6.4.4.2). These include those associated with the H-reflex, the Achilles tendon reflex, passive tension, muscle compliance and motor unit synchronisation. As each of these variables showed no significant differences between baseline values for the two exercise bouts, it is unclear why a difference was recorded between baselines for the position perception task. Again this may be due to a combination of numerous factors, but this being the case it is doubtful if more specific explanations for the proprioception changes could be made without more sophisticated and precise measurements. For example, an actual visual examination of muscle spindles using biopsy and histological techniques, rather

than a test of spindle function, or the more invasive use of needle EMG may be required. Given that greater changes in many of these variables are typically reported following maximal eccentric exercise (particularly reflex responses), examining the repeated bout effect on proprioception following maximal exercise may provide a better insight into the mechanisms at play.

It must again be mentioned that testing in the present investigation was limited to gastrocnemius and soleus, however plantarflexion of the foot is controlled by a total of 8 muscles. It is therefore possible that a muscle not specifically tested controlled the mechanisms responsible for the proprioceptive alterations. Any further investigation of the apparent lack of protection in functional proprioceptive tasks would therefore need to incorporate these muscles

7.5 Conclusions

While the repeated bout effect is widely studied, little (or no) consideration in the past has been given to the functional proprioceptive changes that occur with repeated eccentric exercise. The current results suggesting that the repeated bout effect may not occur to the same extent in these tasks as they do with the more typically examined indicators of voluntary strength, DOMS and plasma CK. The repeated bout effect may also be limited in its application to muscle activation during submaximal contractions even though applying to maximal contractions, following a submaximal exercise protocol. A relationship between the 'unprotected' activation changes and the 'unprotected' proprioceptive changes was apparent. From a practical point of view, it would seem necessary to further investigate the repeated bout effect in relation to proprioception as a possible mechanism for the prevention of injury in both competition and training. Extending research past the typical time of two exercise bouts may also prove imperative in the understanding of proprioceptive changes following eccentric exercise, as protection in the variables influencing proprioception may occur at a slower rate than the more conventionally measured variables.

CHAPTER 8 RELIABILITY OF CRITERION PROTOCOL AND VALIDATION OF THE DUAL ANKLE DYNAMOMETER

8.1 Introduction

Reliability, or reproducibility, refers to the amount of variation (being either biological variation or experimental error) that occurs between testing trials in one session, or between results from two or more testing sessions. It therefore indicates the stability, consistency, and equivalence with which a given result can be reproduced across time (Porter & Hamm, 1986, p. 449). While standard deviation is often used to report the degree of variation between two or more trials, it can only be effectively applied to several trials from the same subject. Most often, a test – retest technique is employed whereby the results from the same test procedure conducted on two separate occasions are compared. Based on the results, calculation of the test – retest correlation is sensitive to the range of values over the trials and also has limited use for a small number of trials (ie two) by many subjects (Malim & Birch, 1997, p. 47). The calculation of the co-efficient of variation however, allows for comparison where the deviation between the subjects within the trials is large (Sale, 1991, p. 75).

While reliability refers to the consistency of a measure, validity is concerned with the extent of the relationship between a concept and an indicator (Carmines & Zeller, 1979, p. 12). That is, the extent to which a test or instrument actually measures what it is claimed to measure (Malim & Birch, 1997, p. 47). As many of the protocols used in the current studies employed a purpose built, one-of-a-kind apparatus it was necessary to establish the normal variation and reliability of the testing procedures, and to establish the validity of the measurements obtained from the equipment. This chapter therefore examines the reliability of each variable compared with the reliability of similar measures throughout the literature, and the validity of the DAD used throughout the testing protocols by comparison of the results with a Cybex isokinetic dynamometer.

8.2 Methods

The reliability of each testing variable described in the previous chapters was determined by calculation of the coefficient of variation of method error (Sale, 1991, p. 75). Test – retest values for each variable were taken from the results obtained from the two days of testing conducted with a 48-hour interval between tests. As an additional

measure for comparison with reliability studies throughout the literature, the intra-class correlation coefficient (ICC) for each testing protocol was also determined. Finally, paired sample t-tests were conducted to determine if any significant differences between the two testing occasions occurred for any of the criterion testing variables. The procedures for all variables have been described in detail in the previous chapters (Chapters 4 – 7)

8.3 Results

8.3.1 *Reliability*

The coefficient of variation of method error (CV : %) for single repeated measures of all criterion testing variables was calculated from two testing occassions (Sale, 1991, p. 76). The mean CV (%) of all testing variables for the exercised limb (13.68%) was slightly higher than that of the non-exercised limb (12.34%) with the greatest variation in results seen during the H-reflex protocol (34.35%). The test – retest ICC results ranged from a poor/moderate +0.57 for the avEMG variables to a high +0.98 for the maximal voluntary strength protocols. Results for the test – retest intraclass correlation and calculated CV (%) for both the exercised and non-exercise limbs are given in Table 5. Paired sample t-tests conducted between the baseline values for both limbs revealed no statistical difference for any of the criterion testing variables. Reliability of the criterion measure CK was calculated as being 23.5% with a range of 24 – 415 U/l recorded across testing [section 4.2.4; n = 20].

Table 12.

ICC and Coefficient of Variation of Method Error (%) for All Testing Variables

	Exercised limb		Non-exercised limb	
Variable	ICC	CV (%)	ICC	CV (%)
Contractile Properties [section 6.2.4; n = 12]				
Twitch peak force	+0.88	6.18	+0.89	7.76
Time to peak	+0.85	8.26	+0.93	5.95
Half relaxation time	+0.83	7.64	+0.95	4.93
Range of Motion [section 6.2.10; n = 11]	+0.95	2.67	+0.94	2.33
Relaxed Joint Angle [section 6.2.10; n = 11]	+0.90	12.97	+0.96	10.84

Table continues

Table 5 (continued)

	Left limb		Right limb	
Criterion Variable	ICC	CV (%)	ICC	CV (%)
Angle Position Perception – exercised limb as target [section 6.2.9; n = 12]				
Plantarflexion	+0.77	7.40	+0.86	22.81
Dorsiflexion	+0.97	6.37	+0.75	21.86
Angle Position Perception – non-exercised limb as target [section 6.2.9; n=12]				
Plantarflexion	+0.80	28.13	+0.94	6.37
Dorsiflexion	+0.86	30.63	+0.97	6.94
H-reflex [section 6.2.3; n = 12]				
Gastrocnemius H wave	+0.76	34.35	+0.91	23.45
Soleus H wave	+0.91	21.54	+0.96	15.21
Gastrocnemius M wave	+0.96	11.13	+0.97	10.91
Soleus M wave	+0.94	6.93	+0.98	6.45
Maximal Voluntary Strength – Cybex [section; 4.2.6; n = 15]				
Plantarflexion	+0.98	6.20	–	–
Dorsiflexion	+0.97	5.96	–	–
Maximal Voluntary Strength – DAD [sections 3.3.2, 3.3.5, and 6.2.6; n = 12]				
Torque	+0.95	7.95	+0.96	6.79
Gastrocnemius avEMG	+0.70	25.05	+0.57	27.39
Soleus avEMG	+0.93	12.36	+0.94	9.91
Tendon Tap [section 6.2.5; n = 8]				
Strength	+0.96	9.41	+0.94	7.82
Gastrocnemius EMG amplitude	+0.93	15.48	+0.97	11.31
Soleus EMG amplitude	+0.96	18.29	+0.98	11.27
Torque Perception – exercised limb as target [section 6.2.8; n = 11]				
Torque	+0.98	3.07	+0.84	16.84
Gastrocnemius avEMG	+0.75	25.01	+0.94	18.01
Soleus avEMG	+0.85	14.83	+0.95	14.06
Torque Perception – non-exercised limb as target [section 6.2.8; n = 11]				
Torque	+0.87	13.70	+0.98	4.67
Gastrocnemius avEMG	+0.76	21.92	+0.79	24.98
Soleus avEMG	+0.84	16.54	+0.80	19.19
Vertical Jump [section 6.2.7; n = 8]	+0.94	7.03	+0.88	2.66

8.3.2 *Validity*

A strong correlation was found between the Cybex 6000 isokinetic dynamometer and the DAD (r = +0.93; p<0.001; Figure 85). Both apparatus being used throughout testing for the measurement of maximal voluntary isometric strength of the plantarflexors.

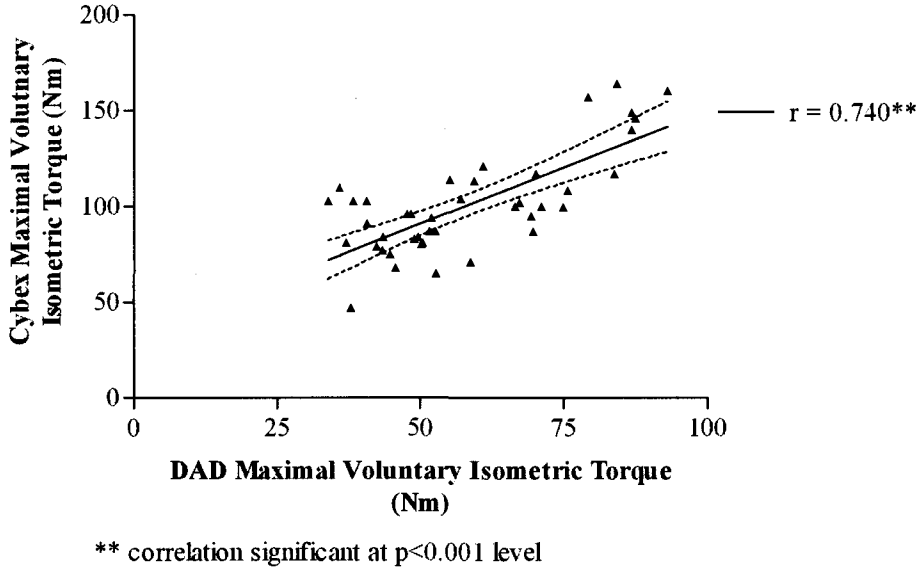


Figure 85. Relationship between strength measured on the Cybex strength and DAD (n = 24) from two testing time intervals.

8.4 Discussion

8.4.1 *Reliability Comparisons*

According to Malim and Birch (1997, p. 47) a good test – retest correlation will yield a result of +0.80 or +0.90. The current results producing 46/54 (85%) results with a correlation above +0.80, 33/46 (72%) of these being above +0.90. The correlation results for plantarflexion strength using the Cybex 6000 isokinetic dynamometer for the current testing population was considerably higher (+0.98 versus +0.74) than those reported by Hsu, Tang and Jan (2002) using a stroke patient population. The correlation results for maximal strength, both voluntary and stimulated (using the DAD) were similar to those previously reported by Allen, Gandevia and McKenzie (1995) for the biceps, and comparable to the results obtained for plantarflexion using the Cybex. The coefficient of variation results for stimulated voluntary strength and contractile properties (approximately 5 – 10%) also comparable to those reported by Gerrits, Hopman, Sargeant and de Haan (2001) in human paralysed and non-paralysed quadriceps muscle.

In the present study, ICC results for the soleus EMG demonstrated greater reliability than gastrocnemius yielding strong correlations above +0.90 between the two test occasions. In contrast only weak (+0.50) to moderate (+0.70) correlations were recorded for gastrocnemius. The results for soleus being similar to previous work reporting ICC results ranging from +0.80 to +0.96 for the quadriceps muscles during knee extension (Larsson, Karlsson, Eriksson & Gerdle, 2003; Pincivero, Green, Mark & Campy, 2000). Larsson et al (2003) also reporting ICC results of +0.93 for peak torque reliability of the knee extensors. The greater reliability of soleus EMG results over gastrocnemius EMG results consistent across all testing protocols for both voluntary and stimulated EMG possibly reflects differences in both muscle fibre architecture and innervation (Loeb & Gans, 1986, p. 58-59; Vandervoot & McComas, 1983).

High ICC results were demonstrated for both the soleus and gastrocnemius muscles with respect to peak H-wave and M-wave amplitude, with values ranging from +0.76 (gastrocnemius H-wave) to +0.98 (soleus M-wave). ICC values generally tending to be higher for M-wave recordings than for the H-wave of either muscle. These results correspond to those reported by Hoffman, Palmieri, Ingersoll, Hopkins and Wagie (2003) who demonstrated ICC results for soleus ranging from +0.96 when testing sessions were separated by 1 hour, through to +0.79 for testing sessions conducted on subsequent days. Other ICC values for peak H-wave and M-wave amplitudes in the literature ranging from +0.56 to +0.99 (Ali & Sabbahi, 2001; Palmieri, Hoffman & Ingersoll, 2002; Williams, Sullivan, Seaborne & Morelli, 1992)

Jump height ICC results for double-leg vertical jumps as high as +0.99 (Pauole, Madole, Garhammer, Lacourse & Rozenek, 2000) have been reported in the literature. Harman, Rosenstein, Frykman & Rosenstein (1990) also reporting excellent test – retest reliability (Cronbach α reliability) for various biomechanical descriptive variables using a countermovement double-leg jump technique. The current results for CV% showed vertical jump height performed on the non-exercised limb (right limb: 2.66%) to be more reliable than the exercised limb (left limb: 7.03 %). These CV results while lower than those reported for a double-limb jump technique, still demonstrated good test – retest reliability. The current results are more comparable to those reported by Farr, Nottle, Nosaka & Sacco (2002) also for a single-limb technique, where jumping limb dominance was randomised, reporting coefficient of variations of 7.5 to 8.1%.

The 13 – 16% CV for torque perception in the present study were higher than those reported previously by Jones and Hunter (1983), who demonstrated a CV% of 5% between three experimental sessions. Despite this variation, the torques produced during the matching were on average within 3% of the required 30% MVC torque, with both over and under – estimations of torques occurring during the baseline testing for various subjects. This 3% MVC torque variation is comparable to the 3% (Jones & Hunter, 1985) and 7.3% (Jones, 1989) reported previously under normal / controlled conditions. The variation seen in the voluntary avEMG during the torque perception tasks were similar to those recorded for avEMG during the voluntary torque protocol. As a strong linear force-EMG relationship has previously been demonstrated (Cafarelli & Bigland-Ritchie, 1979), particularly at low level (<50% MVC) contractions (Solomonow et al., 1990), any variation in both maximal voluntary strength and matching torque would be expected to influence the avEMG recorded during the matching tasks. Overall, the current study demonstrated moderate (+0.75) to strong (+0.90) test – retest ICC values across the testing for avEMG of both soleus and gastrocnemius muscles.

The ICC results for position perception in the present study were on average greater than those reported previously by Lonn, Crenshaw, Djupsjobacka & Johansson (2000) for limb position of the arm. These authors reporting modest correlations of +0.40 to +0.61, with the current study showing a range of +0.75 to +0.86 for the matching limb. A study by Berenberg, Shefner and Sabol (1987) demonstrated an average matching error in 22 subjects of 2.46 ± 0.87° using a comparable methodology to the present investigation that demonstrated an average error of 1.26 ± 0.81° during baseline testing. This being similar to the 0.6 – 1.1° precision that has been demonstrated in the shoulder and elbow during position sense of the hands (van Beers, Sittig and Denier van der Gon, 1998).

8.4.2 *Equipment Validation – The Dual Ankle Dynamometer*

Compared to previous findings, the torque outputs recorded during the current study were both lower at their initial baseline levels, and the recovery of post-walk torque reductions more delayed. With the ankle positioned at maximal dorsiflexion, and the knee at full extension, Seliger, Dolejš and Karas (1980) reported peak torque ranging from 81 – 188 Nm. These values are similar to those of Trappe, Trappe, Lee and Costill (2001) who reported values of 87 – 211 Nm with the ankle at 10° dorsiflexion and the

knee at 30° flexion. The latter authors stating that peak plantarflexion isometric strength occurs between the ankle angle ranges of 4° dorsiflexion to 2° plantarflexion, with the strength highly dependant on both knee and ankle angle. Peak torque results from the present study demonstrated ranges from 39 – 83 Nm using the DAD with the ankle at 10° dorsiflexion and the knee at 90° flexion. For the same individuals peak torque ranges of 47 – 164 Nm were recorded with subjects knee and ankle positioning the same, but with the subjects in a supine position rather than seated. This difference can be explained by the different methods used to secure the subjects foot during each protocol. During testing on the Cybex, the subjects ankle was strapped such that heel lift was fully restricted allowing for greater exertion against the footplate, and subjects were allowed to hold onto the testing apparatus during exertion. During testing on the DAD, subjects were restricted from holding the apparatus during exertion, and the foot strapping allowed for some heel lift during a voluntary contraction. Despite these differences, the strong correlation between the two apparatus and the consistency of the testing protocols showed the DAD to be a valid measure of plantarflexion torque.

8.5 Conclusions

While a large number of the protocols used throughout testing involved the use of a custom built dual ankle dynamometer, the results obtained were well correlated with those obtained on the more conventional Cybex 6000 isokinetic dynamometer. Additionally, the favourable ICC results and relatively low coefficients of variation demonstrated that most testing variables were reproducible. The greatest variations recorded for proprioceptive and EMG variables, however, these were still comparable to those reported previously throughout the literature and considered acceptable for the purposes of the studies presented in this thesis.

CHAPTER 9 SUMMARY AND RECOMMENDATIONS

EIMD is a widely studied yet poorly understood consequence of eccentric exercise, with a range of possible mechanisms proposed to explain the phenomenon. As much of the work relating to EIMD had focused on responses to maximal eccentric contractions, the need to examine responses to submaximal contractions was highlighted, given that many activities both sporting and everyday have a substantial submaximal eccentric component. From this, a downhill backward walking model devised to isolate the foot plantarflexors, was shown to be an effective submaximal eccentric exercise protocol for the triceps surae. Responses closely mirrored those observed following both submaximal and maximal eccentric exercise protocols in other muscle groups.

With the scarcity of information often contradictory, a main focus of the current studies was the muscle activation and control changes that occur in association with EIMD. Using the downhill backward walking model, it was demonstrated that proprioceptive responses as measured by both position and force perception tasks were significantly altered post-exercise. The changes in proprioception were primarily attributed to alterations in muscle activation and control, rather than a consequence of disrupted functioning of the reflex pathways relating to position and force perception. The changes observed in proprioception were likely a combination of changes in a number of variables, rather than a change in one single variable alone.

The present investigation also exposed the need for more practical testing following eccentric exercise in addition to the commonly measured indicators of voluntary strength, plasma CK and soreness. In order to understand the mechanisms responsible for these common indicators it appears necessary to examine a wider range of associated variables to fully understand the extent to which everyday functioning and movement may be affected by EIMD. Additionally, closer examination and measurement of variable changes occurring during an eccentric exercise protocol rather than typical pre and post measures may provide a greater understanding of the causational mechanisms of EIMD.

A major finding of the investigation was that the typically seen protection associated with EIMD and the repeated bout effect did not apply to changes seen post-exercise in functional proprioceptive tasks due largely to the lack of protection in activation changes following the repeated exercise bout. As a result it of this, it was noted that it might be necessary to examine changes in proprioceptive and reflex responses following more damaging maximal eccentric contraction protocols. Maximal eccentric exercises have previously been shown to elicit a greater impact on the mechanisms that control proprioceptive functioning. Examining the repeated bout effect past the typically recorded two exercise bouts, also has the potential to reveal more relating to the mechanisms responsible for this observed protective response for the common indicators of EIMD, and the lack of protection of other variables.

It is recognised that a limitation of the investigations conducted in the present work relate to the design of the DAD in that it only allowed for strength testing with the knee in a flexed position. As the damage model used an extended knee position it would be expected that this would have preferentially loaded the gastrocnemius muscle. The flexed knee testing position however would greatly limit the contribution of the gastrocnumius to the strength testing. Further investigations employing strength testing in both positions would therefore be advised to ensure the specificity of the exercise and testing protocols. Having said this however, given that much of the testing required a relaxed muscle, or the production of submaximal contractions, it is suggested that the difference in the damage and the testing positions would have had little influence on a majority of the testing protocol. It was the intent to examine the influence of a submaximal eccentric exercise protocol on proprioception and activation. Testing was also conducted on the soleus muscle (in addition to the gastrocnemius muscle) which would have been active and under strain during the walking protocol and therefore subjected to repetitive, submaximal eccentric contractions. The similarities in the results between the recordings from soleus and gastrocnemius suggest that the testing position was likely to have little effect on many of the testing procedures.

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APPENDIX A CONSENT FORM

Proprioceptive Changes Associated with Exercise Induced Muscle Damage (EIMD)

Information Sheet and Consent Form

Thank you for your interest in my research into the area of exercise induced muscle damage. The goal of the following information is to fully inform you of the aim and the nature of the study. Should you have any questions relating to any of the information please don't hesitate to contact me for a further explanation.

The main aim of the study is to determine what effect exercise induced muscle damage has on an individual's perception of movement and body control. Exercise induced muscle damage is a commonly experienced phenomena amongst both the social and the elite athlete. The most recognisable sign of exercise induced muscle damage is delayed onset muscle soreness. This is the soreness and stiffness you have probably felt the day after the first game of the new sports season or the day after a hard day gardening in the back yard. What I am specifically interested in is determining whether during the time an individual is experiencing this soreness and stiffness, their movement and muscle control is affected. The results of this research hopefully will lead to a greater understanding of the mechanisms of exercise induced muscle damage, and knowledge relating to any increased risk of injury associated with exercise induced muscle damage.

If you agree to participate in the study there are five tasks which you will asked to complete over 7 (or 13) days of testing.

- A strength testing task of the calf muscles - to determine your maximal calf strength
- A matching task for ankle strength and position - to examine your muscle control without any visual stimulus
- A tracking task for ankle movement - to examine your muscle control with a visual stimulus
- A vertical jump test – to examine the degree of functional strength loss
- Testing for reflex responses in the calf muscles - to examine the responses of the mechanisms that control balance and movement (some discomfort may be experienced during this procedure as it does involve low levels of electrical stimulation however each stimulation is of short duration)

A demonstration and familiarisation period of all testing procedures will occur before you begin testing so that you are fully aware of what is involved with each procedure. You will also be free to withdraw at any stage and for any reason without prejudice.

You will also be asked to complete an exercise task which will involve 60 minutes of backwards downhill walking on a treadmill at pace of 30-35 steps per minute.

Blood samples from a finger prick will be taken on all occasions for measurement of the muscle enzyme creatine kinase. This is a commonly used indicator of the existence of exercise induced muscle damage. As the study is aimed at examining exercise induced muscle damage you may experience some degree of muscle soreness and stiffness in the days following the treadmill walk. This should last no longer than 5-7 days, and as mentioned above this may be an experience that you are already very familiar with. Therefore, monitoring of delayed onset muscle soreness, muscle tenderness and relaxed ankle angle will also occur regularly throughout the testing period.

No direct comparisons between different individuals participating in the study will be made at any stage of the testing. The only comparisons will occur between your results from one day to another, and the differences in results between your left and right legs. You are therefore not in competition with any other individuals in the study and will in no way be made to feel that your results are inadequate or wrong.

As the study is aimed at assessing any changes that may occur across a period of time it is asked that during the study you do not make any major changes to your diet or exercise activities this may influence your results. Additionally, as the study does involve an exercise protocol, it is required that all subjects be healthy at the time of testing. For this reason you will be asked to complete a medical questionnaire prior to the commencement of testing. In some instances it may be asked that you undergo a medical examination (any cost will be incurred by the university and not yourself) to reduce the risk of harm to yourself resulting from participation in the study.

All personal information and test results recorded will remain confidential and will not be used for any purpose other than the current study. All information will be kept under lock and key, with each individual being assigned a number known only to the researchers. Additionally, no data analysis will include any name or information that may identify you specifically as a subject.

Again, if there are any questions relating to the above information please don't hesitate to contact me for further clarification.

Sincerely

Carmel Nottle
PhD candidate
Phone: [redacted]
Email: [redacted]
Post: Carmel Nottle
SOBSS
Edith Cowan University
100 Joondalup Drive
Joondalup 6027

Declaration

I _____ have read the informed consent, have completed a medical questionnaire, and have had all questions relating to the study answered to my satisfaction.

I agree to participate in this study realising that I am free to withdraw at any time, for any reason with out prejudice.

I agree that the research data obtained from this study may be published, provided I am no identifiable in any way

Participant _____ Date _____

Investigator _____ Date _____

APPENDIX B MEDICAL QUESTIONNAIRE

Medical Questionnaire

The following questionnaire is designed to establish a background of your medical history, and identify any injury or illness that may influence your testing or performance.
Please answer all questions as accurately as possible and if you are unsure about any thing please ask.
All information provided is strictly confidential.

Personal Details

Name: _____ ID: _____

DOB: _____ Gender _____

Medical History

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

			If you answered YES please give details
High or abnormal blood pressure	Y	N	
High cholesterol	Y	N	
Rheumatic fever	Y	N	
Heart abnormalities	Y	N	
Asthma	Y	N	
Diabetes	Y	N	
Epilepsy	Y	N	
Recurring back pain	Y	N	
Recurring neck pain	Y	N	
Severe allergies	Y	N	
Any infectious diseases	Y	N	
Any neurological disorders	Y	N	
Any neuromuscular disorders	Y	N	

Current Health

			If you answered YES please give details
Are you currently on any medications?	Y	N	
Have you had the flu in the last two weeks?	Y	N	
Have you recently had any injuries?	Y	N	
Do you have any recurring muscle or joint injuries?	Y	N	
Have you had any ankle problems in the last 6 months?	Y	N	

Have you participated in a resistance training program in the last 6 months? Y N

Is there any other condition not previously mentioned which may affect your exercise performance? Y N

Family History

Are any of the following known to exist in your family?

			If you answered YES please give details
Cardiac disease	Y	N	
Pulmonary disease	Y	N	
Stroke	Y	N	

Lifestyle Habits

Do you exercise regularly? Y N
If YES how many hours per week? _____

Do you smoke tobacco or any other nicotine products Y N
If YES please how much per day? _____

Do you consume alcohol? Y N
If YES how many standard drinks per week? _____

Do you consume tea and or coffee? Y N
If YES how many cups per day? _____

Do you take any recreational drugs? Y N
If YES how much or how often per 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.

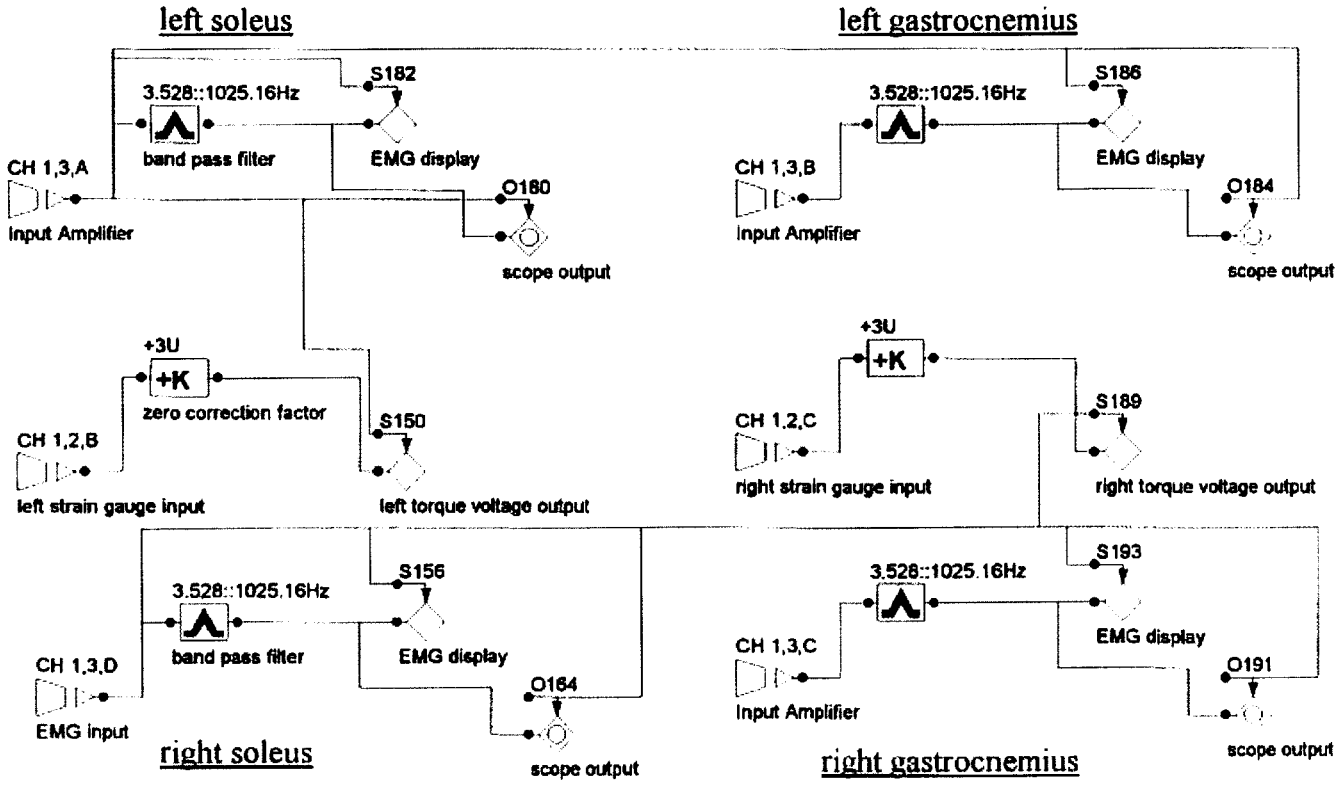
Participant
Name: _____ Date: _____

Signature: _____

Practitioner
Name: _____ Date: _____

Signature: _____

APPENDIX C AMLAB SCHEMATICS



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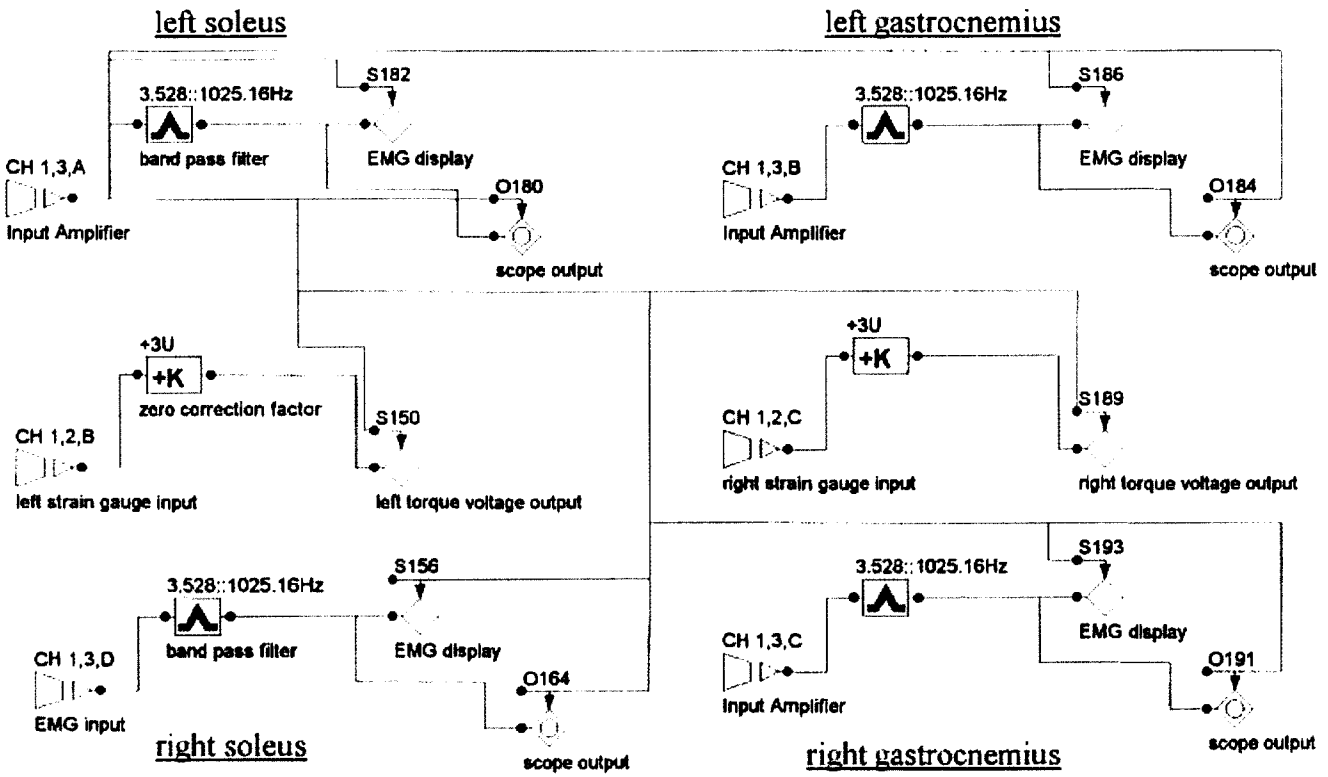

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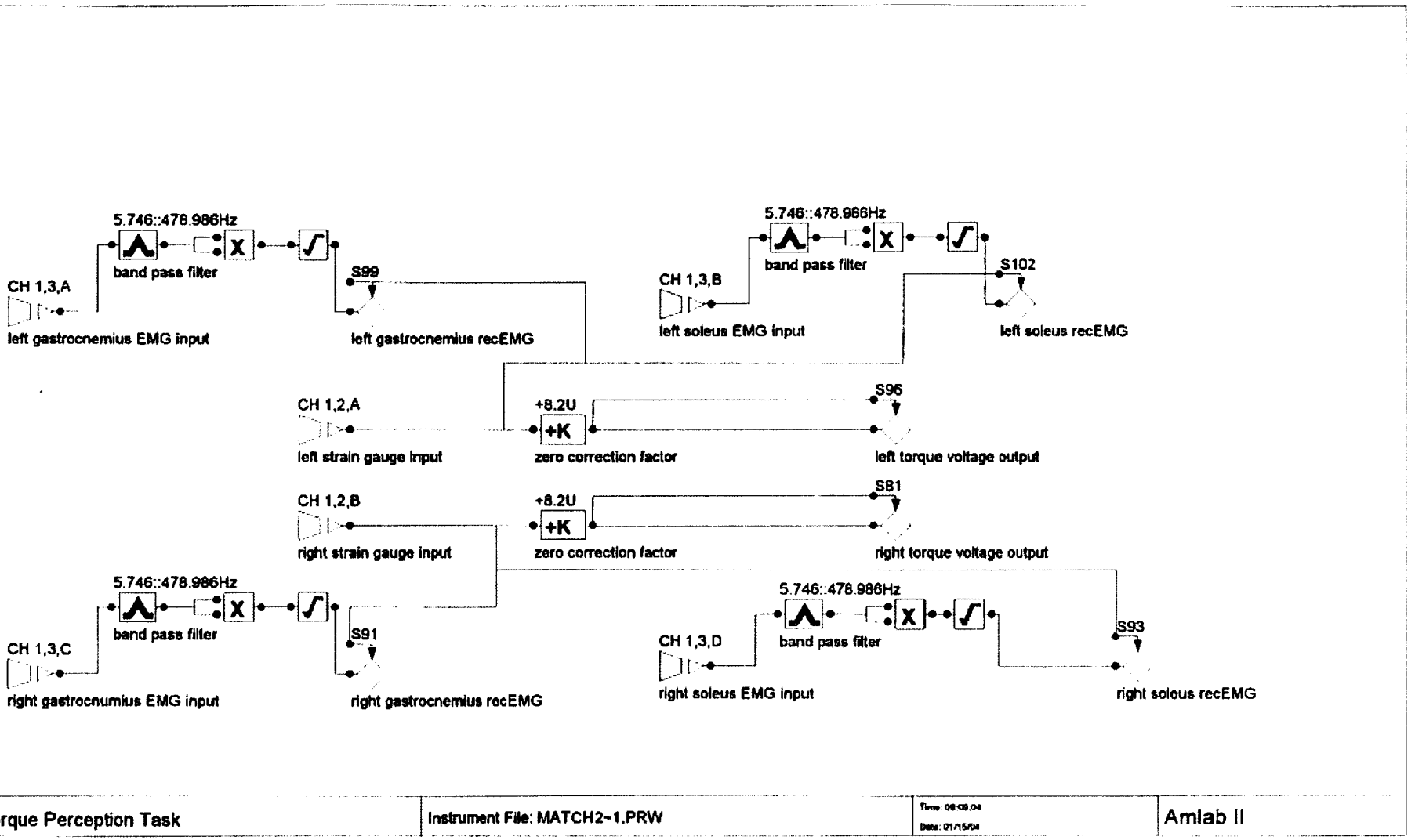
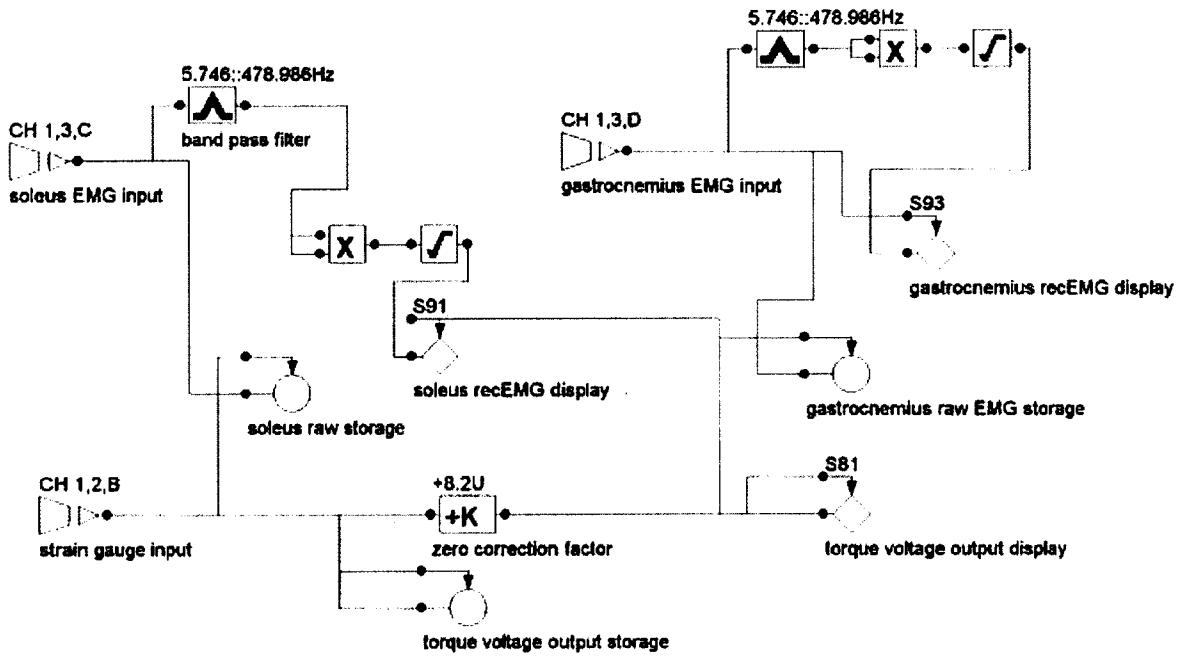

5.746::478.986Hz
band pass filter
CH 1,3,A
left gastrocnemius EMG input
S99
left gastrocnemius recEMG
5.746::478.986Hz
band pass filter
CH 1,3,B
left soleus EMG input
S102
left soleus recEMG
CH 1,2,A
left strain gauge input
+8.2U
+K
zero correction factor
S96
left torque voltage output
CH 1,2,B
right strain gauge input
+8.2U
+K
zero correction factor
SB1
right torque voltage output
5.746::478.986Hz
band pass filter
CH 1,3,C
right gastrocnumius EMG input
S91
right gastrocnemius recEMG
5.746::478.986Hz
band pass filter
CH 1,3,D
right soleus EMG input
S93
right soleus recEMG
Title: Torque Perception Task
Instrument File: MATCH2~1.PRW
Amlab II



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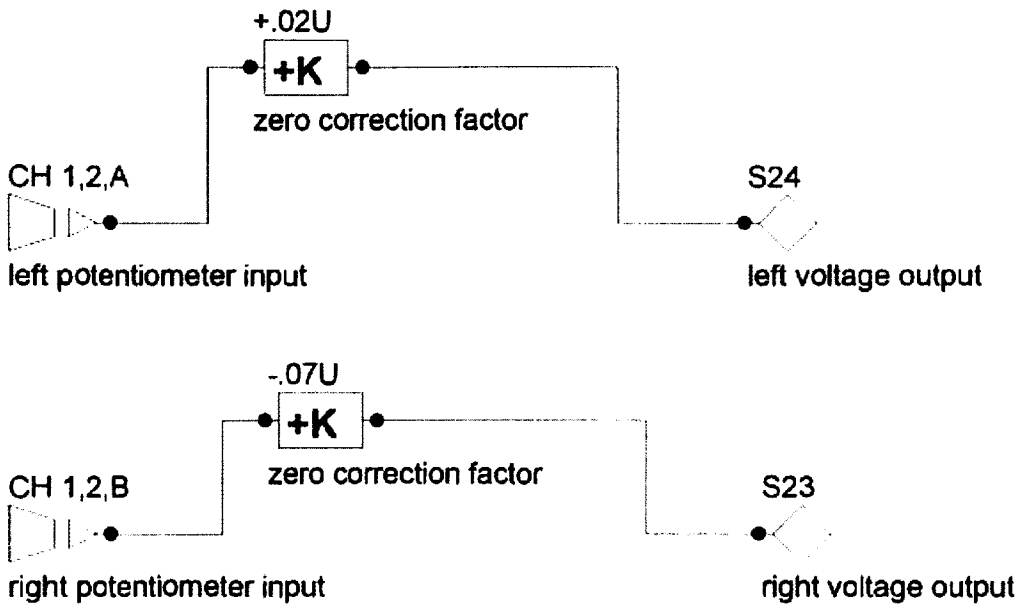

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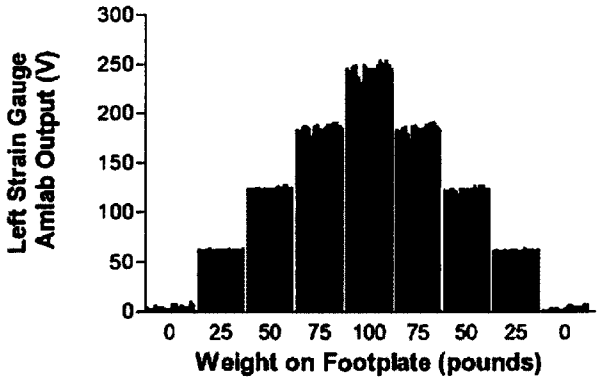
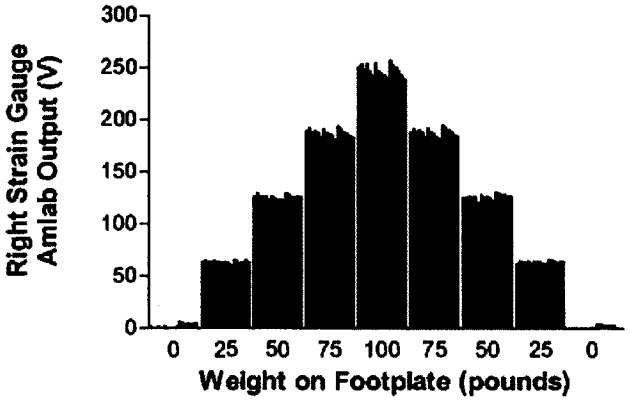
APPENDIX D STRAIN GAUGE CALIBRATION RECORDS





		AMLAB Output (V)																
		Calibration Date																
	Weight	12/2/01	13/2/01	21/2/01	2/3/01	2/4/01	19/4/01	21/8/01	1/9/01	13/11/01	1/8/02	1/24/02	2/20/02	8/03/02	21/03/02	5/04/02	5/05/02	16/05/02
Left Strain Gauge	0	3.84	2.37	3.01	6.06	4.44	5.32	3.10	2.37	3.11	7.54	8.28	5.32	6.06	5.32	5.32	9.76	7.54
	25	62.86	62.15	62.13	62.86	63.60	62.12	62.13	62.86	62.86	62.13	62.86	63.60	62.12	62.86	62.86	62.13	63.60
	50	123.51	123.51	124.25	124.99	124.99	122.03	124.25	123.51	124.25	122.77	124.25	126.47	124.99	127.21	127.47	124.99	124.25
	75	183.42	181.94	186.38	187.15	184.90	176.28	186.38	187.89	184.16	183.42	185.63	187.86	188.59	190.07	190.07	187.12	184.89
	100	244.80	241.80	247.02	247.76	235.92	230.01	248.50	245.54	244.06	244.06	244.80	248.50	252.94	249.98	253.67	248.50	244.06
	75	182.67	181.94	184.89	186.37	179.72	173.06	187.12	187.89	184.90	184.16	185.63	188.59	189.33	190.81	191.55	187.12	184.89
	50	122.77	122.03	123.51	124.25	122.03	118.33	124.99	123.51	124.99	123.51	124.25	127.21	124.99	127.95	127.95	124.99	124.99
	25	61.38	62.86	61.38	61.38	62.86	61.38	62.86	62.86	63.60	62.13	62.13	64.34	62.86	63.60	63.60	62.13	63.60
	0	2.36	0.90	2.27	3.84	2.96	3.84	2.36	1.58	3.11	6.06	6.80	4.58	5.32	4.58	4.58	7.54	6.80
Right Strain Gauge	0	2.37	0.00	0.89	1.63	0.74	1.62	0.89	0.89	0.89	1.63	6.06	5.32	3.84	4.58	3.84	4.58	4.58
	25	64.34	65.08	64.34	64.34	65.08	64.34	63.60	63.60	63.60	62.86	60.64	85.82	65.08	62.86	63.60	65.82	63.60
	50	127.21	130.17	127.21	127.21	127.95	124.99	127.21	125.73	124.73	124.25	123.51	130.17	129.43	126.47	126.47	127.21	126.46
	75	190.07	193.03	188.59	190.82	188.59	184.16	191.55	188.59	187.12	185.64	181.94	194.51	192.29	189.33	187.11	185.64	184.15
	100	251.45	254.42	249.24	254.52	248.00	243.32	255.16	247.76	245.54	243.32	240.36	258.15	252.94	250.72	247.02	243.32	239.62
	75	188.59	191.55	189.33	190.82	188.59	183.42	192.29	188.59	187.12	184.16	182.67	195.25	193.03	190.81	189.33	186.38	184.89
	50	125.73	126.69	127.21	126.47	127.21	121.29	128.69	126.47	127.21	125.73	122.71	130.91	130.17	128.67	128.69	127.21	127.21
	25	62.13	65.08	64.34	62.86	65.08	62.86	64.34	64.34	64.35	62.60	62.12	65.82	65.83	65.08	62.82	65.08	64.63
	0	-0.58	0.00	0.74	-0.59	0.00	0.14	-0.59	-0.59	0.89	-0.59	4.59	4.58	3.10	3.10	3.10	2.36	3.10

APPENDIX E POTENTIOMETER CALIBRATION RECORDS

		Amlab Output (V)								
		Calibration Date								
	Angle	12-Feb-01	2-Mar-01	2-Apr-01	21-Aug-01	13-Nov-01	8-Jan-02	20-Feb-02	21-Mar-02	5-Apr-02
Left Potentiometer	-20	-1.044	-1.043	-1.044	-1.040	-1.041	-1.042	-1.043	-1.042	-1.043
	-15	-0.781	-0.781	-0.781	-0.779	-0.779	-0.780	-0.782	-0.782	-0.781
	-5	-0.261	-0.260	-0.262	-0.262	-0.261	-0.261	-0.262	-0.261	-0.262
	0	0.001	0.002	0.001	0.001	0.002	0.001	0.002	0.001	0.002
	5	0.262	0.260	0.260	0.262	0.259	0.261	0.262	0.263	0.261
	10	0.523	0.523	0.521	0.523	0.518	0.521	0.523	0.523	0.521
	15	0.779	0.782	0.780	0.783	0.782	0.780	0.781	0.781	0.781
	20	1.047	1.042	1.043	1.042	1.042	1.042	1.041	1.042	1.041
	25	1.310	1.300	1.310	1.320	1.320	1.310	1.310	1.320	1.300
	30	1.559	1.560	1.557	1.557	1.557	1.559	1.561	1.562	1.560
	-20	-1.046	-1.044	-1.045	-1.038	-1.043	-1.044	-1.044	-1.043	-1.046
	-15	-0.783	-0.782	-0.782	-0.777	-0.781	-0.782	-0.783	-0.783	-0.784
	-5	-0.263	-0.261	-0.263	-0.260	-0.263	-0.263	-0.263	-0.262	-0.265
Right Potentiometer	0	-0.001	0.001	0.000	0.003	0.000	-0.001	0.001	0.000	-0.001
	5	0.260	0.259	0.259	0.264	0.257	0.259	0.261	0.262	0.258
	10	0.521	0.522	0.520	0.525	0.516	0.519	0.522	0.522	0.518
	15	0.777	0.781	0.779	0.785	0.780	0.778	0.780	0.780	0.778
	20	1.045	1.041	1.042	1.044	1.040	1.040	1.040	1.041	1.038
	25	1.308	1.299	1.309	1.322	1.318	1.308	1.309	1.319	1.297
	30	1.557	1.559	1.556	1.559	1.555	1.557	1.560	1.561	1.557