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**THE EFFECT OF SIMULTANEOUS APPLICATION OF
CONTRAST TEMPERATURE THERAPY AND
INTERMITTENT COMPRESSION ON RECOVERY
FOLLOWING EXERCISE INDUCED MUSCLE DAMAGE**

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LIST OF ABBREVIATIONS

°C	degrees Celsius
beats.min ⁻¹	beats per minute
cm	centimetre(s)
HIMS	heart rate interval monitoring system
kg	kilogram(s)
km.h ⁻¹	kilometres per hour
m	metre(s)
min	minute(s)
ml	millilitre(s)
mm	millimetre(s)
mmHg	millimetres of mercury
mmHg.cm ⁻¹	millimetres of mercury per centimetre
N.m ⁻¹	Newtons per metre
nm	nanometre(s)
s	second(s)
SD	standard deviation
U.l ⁻¹	units per litre

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GLOSSARY OF TERMS

Contrast temperature therapy	The alternative application of cryotherapy and thermotherapy ^{93;102;133} .
Cryotherapy	Therapeutic treatments of cold aimed at lowering tissue temperature through the withdrawal of heat from the body ⁹⁴ .
Delayed onset muscle soreness	Pain or discomfort that typically occurs one to two days after unaccustomed loading of skeletal muscle and generally resolves within a week of the inciting activity ^{5;32} .
Exercise induced muscle damage	Muscle damage including cellular and subcellular disturbances, and particularly Z-line streaming, as a result of unaccustomed exercise, particularly if the exercise involves a large amount of eccentric (muscle lengthening) contractions ³⁸ .
Intermittent compression	Repetitive external mechanical squeezing of a limb usually by an inflatable cuff which is fitted around the limb ³³ .
Lengthening muscle actions	Elongation of a muscle while simultaneously producing tension. Also referred to as eccentric muscle actions ³² .
Recovery	The return of the muscle to its pre-exercise state following exercise ⁴⁶ .
Thermotherapy	Therapeutic treatments of heat (greater than 36 °C) that raises the core body temperature ^{13;176} .

ABSTRACT

Background: The high levels of training and competition associated with modern day sport induce physiological stresses that may be related to temporary impairments in athletic performance. The reduction in exercise performance may partially be attributed to delayed onset muscle soreness that may occur in association with exercise induced muscle damage. As a result, recovery has become an essential component of the training process. Although recovery is essentially a passive process, numerous recovery strategies have been developed in an attempt to accelerate the rate of recovery. Recently a new recovery modality has been developed which allows for the simultaneous application of contrast temperature therapy and intermittent compression. However, the effectiveness of this modality as a recovery strategy has yet to be determined.

Objective: To determine the effects of the simultaneous application of contrast temperature therapy with intermittent compression as a recovery strategy for the quadriceps muscle following exercise induced muscle damage in moderately active adult males.

Methods: Thirty healthy male volunteers between the ages of 20 and 40 years, who performed between three and five hours of physical activity per week were recruited for this study which had a randomised experimental design. Familiarisation of the testing procedures was conducted 7 to 10 days before the exposure to the exercise protocol which induced muscle damage. Medical and exercise related questionnaires were completed and body composition was measured. Participants were randomly assigned to either the experimental or control group. A repeated drop jump protocol was used to induce exercise induced muscle damage. Standard testing was performed 24 hours before, and 48 and 96 hours after this exercise protocol. The testing assessed self-reported muscle soreness, plasma creatine kinase activity, vastus lateralis muscle thickness measured by ultrasound imaging, heart rate recovery following a submaximal shuttle run test, quadriceps peak isometric torque and countermovement vertical jump height. The experimental group received simultaneous contrast temperature therapy and intermittent compression immediately after the exercise and at 24, 48, and 72 hours thereafter. This was applied through an inflatable cuff wrapped firmly around the thigh, through which cold (10 °C) or hot (40 °C) fluid was rhythmically pumped by a machine that controlled the temperature of the fluid. The control group did not receive any treatment after the exercise protocol.

Results: No significant differences were found between the experimental group (n=12) and control group (n=14) for any of the variables investigated. Significant increases in self-reported pain in both groups were recorded at 48 hours compared to baseline pain scores for perceived pain at rest, pain on activities of daily living, and pain on self-applied pressure to the quadriceps muscle ($p < 0.01$). Quadriceps muscle stretch showed significant increases in pain at 48 hours compared to baseline in only the control group ($p=0.003$). Plasma creatine kinase activity showed a significant difference over time ($F_{(2, 48)} = 1.14$; $p = 0.01$), and was significantly increased in both groups at 48 hours compared to baseline values ($p=0.009$). A significant difference over time was found for vastus lateralis muscle thickness ($F_{(2, 48)} = 1.18$; $p = 0.001$), which was significantly increased in both groups compared to baseline measurements at 48 hours ($p=0.0003$) and at 96 hours ($p=0.0003$). Countermovement vertical jump height ($F_{(2, 48)} = 1.46$; $p = 0.019$), and peak isometric quadriceps muscle torque for the dominant ($F_{(2, 48)} = 0.45$; $p = 0.003$) and non-dominant legs ($F_{(2, 48)} = 0.66$; $p = 0.001$) were found to have a significant difference over time. Countermovement vertical jump height was significantly decreased in both groups at 48 hours compared to baseline levels ($p=0.032$) while peak isometric force generated by the quadriceps muscle of the dominant leg was significantly increased at 96 hours compared to 48 hours ($p=0.003$) and increased at 96 hours compared to baseline ($p=0.038$) and 48 hours ($p=0.001$) for the non-dominant leg. No significant differences in heart rate recovery over time were found.

Conclusion: The simultaneous application of contrast temperature therapy and intermittent compression used in this study was ineffective in enhancing recovery following exercise induced muscle damage. No differences were noted in the indirect markers of exercise induced muscle damage compared to passive recovery. The need for further research into the modalities of contrast temperature therapy and intermittent compression, both individually and as a combination modality, in relation to exercise recovery was highlighted. In addition, it was noted that the application of intermittent compression to the whole leg rather than just the thigh, and the modification of the method of contrast temperature therapy application to enable an immediate transition between the hot and cold temperatures may improve the outcome of this combination therapy.

CHAPTER ONE

INTRODUCTION AND SCOPE OF THESIS

Athletes are regularly exposed to intense training and competition schedules which may result in exercise induced muscle damage^{10;80;156}. The most frequently reported symptoms of exercise induced muscle damage are soreness, swelling and stiffness⁷⁰. These symptoms are often collectively referred to as delayed onset muscle soreness⁷⁰. Exercise induced muscle damage is associated with high intensity exercise or unaccustomed exercise, particularly exercise associated with muscle lengthening under tension³⁸ and has a negative impact on athletic performance as it results in decreased force production, altered cardiorespiratory performance, metabolic impairment, and changes in neuromuscular control^{10;32;80}.

Recovery after exercise is important in minimising the negative effects of exercise induced muscle damage and enhancing the positive adaptations of the training process. Optimised recovery may allow for a higher level of subsequent performances of athletes over longer periods such as the duration of the sport's season⁸⁰. Inadequate or insufficient recovery has been associated with symptoms of fatigue, impairment of athletic performance, and an increased predisposition to injury^{10;156}. Recovery from symptoms of exercise induced muscle damage has been described as a passive process¹⁰. The use of a variety of active recovery modalities has become popular practice among athletes in an attempt to optimise the recovery process⁸⁰. Recovery strategies are commonly used in different sporting disciplines, despite a lack of evidence to support the efficacy of many of these interventions. Common recovery modalities include cryotherapy, thermotherapy, contrast temperature therapy, intermittent compression, hyperbaric oxygen therapy, non-steroidal anti-inflammatory drugs and analgesics, compression garments, stretching, massage, electrotherapy, homeopathy, vibration, and low intensity exercise. Recovery strategies may involve the combined or simultaneous application of different modalities^{10;32;80},

Several studies have reported beneficial findings for contrast temperature therapy on recovery through effects on indirect markers of exercise induced muscle damage^{46;94;114;132-134;170}, while others have shown no benefit of contrast temperature therapy on the same markers^{49;60;75;102;151;171}. It is theorised that the proposed beneficial effects of contrast temperature therapy may be related to vaso-pumping and hydrostatic pressure^{75;102}. However, the ability of contrast temperature therapy to achieve a significant vaso-pumping effect has been questioned as increased circulation has been reported to be only superficial, with the muscles being unaffected by temperature or circulatory changes⁷³. Similarly, although hydrostatic pressure has been shown to be important in the positive effects reported by some studies^{170;171;176}, it has been argued that temperature and not hydrostatic pressure alone is important in achieving positive results from contrast temperature therapy¹⁰². Intermittent compression has been shown to promote healing and improve circulation^{30;110;130}. Intermittent compression is thought to directly increase venous return and lymphatic drainage through intermittent squeezing of the limb by inflation and deflation cycles of a cuff fitted around the limb^{125;126}. The resultant increase in the arterio-venous pressure gradient, lowers peripheral resistance thereby increasing arterial blood flow⁶². The increase in arterial blood flow and increased blood and lymphatic drainage are thought to be the mechanism through which healing is promoted¹¹⁰.

The technology to simultaneously apply contrast temperature therapy and intermittent compression has only recently been developed. It is therefore not commonly used in clinical practice, and there is also a lack of research regarding this modality. It was theorised that the simultaneous application of these two recovery modalities would complement each other by enhancing the mechanisms through which they have been reported to achieve positive results on recovery from exercise. Therefore, the primary aim of this thesis was to determine the effects of the simultaneous application of contrast temperature therapy and intermittent compression on recovery following exercise induced muscle damage.

In preparation for the experimental study of the thesis, a comprehensive review of the literature pertaining to exercise induced muscle damage, exercise recovery, and recovery modalities, specifically contrast temperature therapy and intermittent compression will be presented (Chapter 2). This will be followed by a description of the study designed to answer the above question (Chapter 3). The summary and conclusion section will complete this thesis (Chapter 4).

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

Athletes are continually pushing physiological boundaries to improve exercise performance. However, sustained high training loads with insufficient recovery may be associated with symptoms of fatigue, a temporary impairment in athletic performance and an increased predisposition to injury^{10;156}. The reduction in exercise performance may partially be attributed to delayed onset muscle soreness that may occur in association with exercise induced muscle damage¹⁴¹. Recovery is therefore an important part of the training process^{129;169}, and is essentially a passive process during which the body naturally recuperates and returns to its pre-exercise state. Numerous active recovery modalities have been developed and implemented across a wide range of sports in an attempt to accelerate the natural recovery process^{25;32;51;80}.

This review will discuss the literature on delayed onset muscle soreness and exercise induced muscle damage, and will include the effects of exercise induced muscle damage on performance. The indirect markers of exercise induced muscle damage will be outlined. The physiological process of recovery from exercise induced muscle damage will be discussed and the various modalities that are used to enhance recovery will be introduced. The main focus of this review will be to examine the recovery modalities of contrast temperature therapy and intermittent compression. There is currently a lack of evidence for the combined use of contrast temperature therapy and intermittent compression, as the technology to support the simultaneous application of these two modalities is still relatively new. Contrast temperature therapy and intermittent compression will therefore be critically reviewed as separate recovery modalities.

The scientific and medical literature was searched using databases and online search engines including EBSCO, PubMed, CINAHL, and Google Scholar. The following keywords were used: "*contrast therapy*", "*contrast baths*", "*contrast temperature therapy*", "*contrast water immersion*", "*contrast temperature immersion*", "*hot and cold treatment*", "*hydrotherapy*", "*intermittent compression*", "*intermittent pneumatic compression*", "*cryotherapy*", "*cold water immersion*", "*hot water immersion*", "*thermotherapy*", "*exercise induced muscle damage*", "*DOMS*", "*exercise fatigue*", "*eccentric exercise*", "*exercise recovery*", "*muscle recovery*", "*recovery strategies*", "*recovery treatment*", "*athletic performance maintenance*", and "*DOMS management*".

2.2 EXERCISE INDUCED MUSCLE DAMAGE

Exercise induced muscle damage is a common occurrence following a bout of unaccustomed or intense exercise and in particular, exercise that involves predominantly lengthening muscle actions. During lengthening muscle actions, the muscle lengthens whilst simultaneously contracting against the lengthening force⁷⁰. Exercise induced muscle damage may be associated with delayed onset muscle soreness, morphological changes within the muscle fibres, increased muscle volume, increased limb circumference, decreased range of movement, decreased muscular strength, and leakage of myofibrillar proteins into the blood^{25;38;70}. The most frequently reported symptoms of exercise induced muscle damage are soreness, swelling and stiffness, and these symptoms are collectively referred to as delayed onset muscle soreness⁷⁰.

2.2.1 DELAYED ONSET MUSCLE SORENESS

Delayed onset muscle soreness describes the sensation of dull aching pain that develops after unaccustomed exercise, particularly exercise which involves high velocity muscle lengthening muscle actions or repetitive stretch shortening cycle actions^{10;32;54;156;170}. Delayed onset muscle soreness has also been described as muscle stiffness or tightness⁴⁴, and may be experienced during active or passive stretch, with muscle contraction, or on palpation of the affected muscles¹⁵⁷. Fast twitch muscle fibres may be more susceptible to delayed onset muscle soreness⁵¹, due to the selective activation of fast twitch muscle fibres during lengthening muscle actions¹²².

Delayed onset muscle soreness usually develops within six to 24 hours after exercise induced muscle damage, and peaks at between one and three days after the damaging bout of exercise. Typically, delayed onset muscle soreness resolves within seven to ten days after the onset^{19;32;51;54;140-142;170}. It is theorised that the pain associated with delayed onset muscle soreness may be related to an inflammatory response following muscle or connective tissue damage^{5;40;121}. However, although delayed onset muscle soreness is considered as an indirect marker of muscle damage, the symptoms may not accurately reflect the extent or time course of exercise induced muscle damage¹⁴¹.

2.2.2 MECHANISMS OF EXERCISE INDUCED MUSCLE DAMAGE AND DELAYED ONSET MUSCLE SORENESS

Immediately following intense exercise involving lengthening contractions, individuals usually experience an impairment of muscle function, including difficulty in controlling movements, reduced force output, increased tremor, and difficulty in moving the affected limb^{140;157}. Individuals usually experience feelings of fatigue and weakness rather than discomfort at this stage³². The symptoms of delayed onset muscle soreness develop over the next six to 12 hours and peak at 24 to 48 hours post exercise.

The exact mechanisms which are responsible for the symptoms of delayed onset muscle soreness are as yet unproven, and are likely to be multi-factorial^{38;172}. Previous studies have identified six theories that have been proposed to explain the cause of delayed onset muscle soreness. These theories include lactic acid accumulation, muscle spasm, connective tissue damage, muscle damage, enzyme efflux, and tissue inflammation^{42;85}. A brief overview of these theories will be discussed in the next section.

2.2.2.1 Lactic acid accumulation theory

The accumulation of toxic metabolic products, such as lactic acid, is thought to be a noxious stimulus which causes the perception of pain at a delayed stage^{5;74;85}. However, as shortening muscle actions are associated with a higher degree of metabolism than lengthening muscle actions, but do not result in the same high levels of delayed onset muscle soreness, this theory has been questioned⁸⁵. Furthermore, no relationship between lactic acid levels measured during or after exercise, and soreness ratings has been reported³². It has been suggested that lactic acid may contribute towards the sensation of acute pain immediately after intense exercise, but is possibly not the cause of pain that experienced 24 to 48 hours following exercise⁸⁵.

2.2.2.2 Muscle spasm theory

Following the observation of increased levels of resting muscle activity after exercise involving lengthening muscle actions^{12;43;85}, it was theorised that this increased resting muscle activity was due to tonic localised spasm of motor units. This leads to compression of local blood vessels causing ischaemia and accumulation of pain substances⁶¹. The stimulation of nociceptors was proposed to cause further reflex muscle spasm and prolonged ischaemic conditions¹². However investigations studying delayed onset muscle soreness using electromyography have been inconclusive. Some studies have shown an increase in electromyographic readings but no correlation to the perception of soreness¹², while other studies have failed to show any increase in electromyographic activity in muscles with delayed onset muscle soreness^{1;135}.

2.2.2.3 Connective tissue damage theory

The connective tissue damage theory is based on the premise that fast twitch fibres have an increased vulnerability to connective tissue damage during lengthening muscle actions³². Slow twitch fibres are bound by connective tissue that has a more robust structure than that of fast twitch fibres³². Furthermore there is a selective activation of fast twitch muscle fibres during lengthening muscle actions¹²². Attempts have been made to provide evidence for this theory, however there remains no real support for it. Although connective tissue disruption does occur, there is no definitive method of accurately and reliably measuring the extent of connective tissue damage or the mechanism that is responsible for it³².

2.2.2.4 Muscle damage theory

Muscle damage is caused by mechanical strain on the muscle. As muscle elongates while generating tension in an eccentric action, the ability to generate tension increases and a high load is distributed among the same number of fibres resulting in a higher force per fibre ratio when compared to a concentric (shortening) muscle contraction⁶⁸. High mechanical strain is placed on the muscle fibres as a result of this loading profile and is regarded as a major causative factor in muscle damage⁷⁰. Actin and myosin cross-bridges are detached mechanically in lengthening muscle actions which places higher strain and more stretch on the compliant portion of individual cross-bridges⁶⁸. These mechanical forces cause disruption of the contractile component of the muscle tissue resulting in Z-line streaming, Z-lines out of register, loss in thick myofilaments, loss of mitochondria in areas showing abnormalities, and derangement of filaments at the A-band^{5;68;70;78;106;135}. It has been suggested that distortion and streaming of the Z-band is due to force mismatch between adjacent sarcomeres rather than the total amount of force applied⁷⁸. This mechanical disruption is highest in fast twitch fibres which have the narrowest and weakest Z-lines³². The extent of muscle damage may be related more to fibre strain (elongation) rather than fibre stress (tension)⁷⁸.

The sensation of pain may be associated with the stimulation of nociceptors in the muscle connective tissue, around arterioles in capillaries, and the musculotendinous junction³². Intramuscular enzymes circulating in the blood such as creatine kinase have been used to quantify muscle damage in exercise induced muscle damage^{6;38;42;136;162}. However there is a dissociation between the peaking of blood creatine kinase activity and the time of peak muscle soreness^{36;37;72;135;136}. As such the muscle damage theory can only be accepted as a partial explanation of delayed onset muscle soreness³².

2.2.2.5 Enzyme efflux theory

Following damage to the sarcolemma, calcium from the sarcoplasmic reticulum accumulates in injured muscles⁵. It is proposed that this calcium accumulation leads to an inhibition of cellular respiration at the mitochondrial level and a subsequent reduction in adenosine triphosphate regeneration⁸⁵. The calcium accumulation is also thought to activate proteases and phospholipases which causes further damage to the sarcolemma and results in the production of leukotrienes and prostaglandins^{5,6}. As adenosine triphosphate is required for active transport of calcium back into the sarcoplasmic reticulum, the removal of calcium is delayed³². This results in increased muscle protein degeneration of the already weakened Z-lines and chemical stimulation of nociceptors³².

2.2.2.6 Tissue inflammation theory

Oedema formation and inflammatory cell infiltration have been found to be evident following repetitive lengthening muscle actions^{72;74;159}. Connolly et al⁵¹ proposed that the inflammatory response may lead to the synthesis of prostaglandin E₂ and leukotrienes. Prostaglandin release causes a sensation of pain through the sensitisation of type III and IV pain afferents due to the effects of chemical stimuli. Leukotrienes cause an increase in the vascular permeability which attracts neutrophils to the area⁵¹. Following injury, proteolytic enzymes from muscle fibres begin breaking down the lipid and protein structures of cells³².

Neutrophils and monocytes, which convert to macrophages, are attracted to the damaged area through the accumulation of bradykinin, histamine and prostaglandins, in addition to the rapid breakdown of damaged muscle fibres and connective tissue⁹². Neutrophils and macrophages are considered to be major contributors to the inflammatory response seen in exercise induced muscle damage and have been linked to the promotion of muscle damage⁵¹. The action of neutrophils generates free radicals which may exacerbate damage to the cell membrane⁵¹. The inflammatory response to muscle fibre damage also causes the movement of cells and fluid from the blood to interstitial spaces through the increased permeability of small blood vessels brought about following eccentric exercise^{41;159}. This may add to the sensation of pain through increasing osmotic pressure⁷⁷ and may be the cause of reduction in the strength approximately two days after the initial damage⁹⁷. Peak muscle soreness has been reported as coinciding with peak oedema levels⁸⁵.

2.2.2.7 Multifactorial Theories

It is widely accepted that no single theory adequately explains the mechanism of the onset of delayed onset muscle soreness³². This has led some researchers to propose a sequence of events that integrate aspects of the existing theories^{5;32;41;51;113;121;160}.

Cheung et al³² suggested an integration of the models that had previously been described^{5;6;159;160}: Lengthening muscle actions generating high tensile forces result in disruption of structural proteins within muscle fibres, particularly at the weakened Z-lines, together with excessive strain of connective tissue at the musculotendinous junction (connective tissue theory and muscle damage theory)^{5;6;42;76;78;135;136}. Calcium homeostasis is disturbed through damage to the sarcolemma^{5;85}. Accumulation of calcium slows adenosine triphosphate production and activates calcium-dependent proteolytic enzymes resulting in the further degradation of Z lines of sarcomeres, troponin and tropomyosin (enzyme efflux theory)^{5;6;85}. Circulating neutrophil levels are elevated within hours (tissue inflammation theory)⁹². From six to twelve hours post exercise induced muscle damage, intracellular components of damaged connective tissue and muscle tissue diffuse into the plasma and interstitium, attracting monocytes that then convert to macrophages^{5;85;92}.

Mast cells and histamine production are activated and within hours there is a significant increase in neutrophils at the site of muscle damage⁹². Increased neutrophil activity generates free radicals which may exacerbate damage to the cell membrane (tissue inflammation theory)¹⁵⁹. Macrophages, after peaking in number at 48 hours after exercise induced muscle damage, produce prostaglandin E₂ which is responsible for sensitising type III and IV nerve endings to mechanical, chemical and thermal stimulation (tissue inflammation theory)⁹². Histamine, potassium and kinins accumulate from active phagocytosis and cellular necrosis⁵¹, together with elevated pressure from tissue oedema and increased local temperature caused activation of nociceptors within muscle fibres and muscle tendon junction (tissue inflammation theory)⁷⁷. In addition to the above mechanisms creating the sensation of delayed onset muscle soreness, movement may increase the sensation of pain as increasing intramuscular pressure could create a mechanical stimulus for nociceptors already sensitised by prostaglandins⁸⁵.

It is important to evaluate the degree of exercise induced muscle damage to better understand the mechanisms and consequences of muscle damage. This may be achieved through direct analysis of exercise induced muscle damage, or through indirect evaluation of the changes on structural, biochemical and functional levels^{32;38;54;60;141;156;174}.

2.2.3 INDICATORS OF EXERCISE INDUCED MUSCLE DAMAGE

Direct assessment of exercise induced muscle damage is difficult as analysis is only possible through muscle biopsy or magnetic resonance imaging, both of which have shortcomings³⁸. Muscle biopsy is an invasive technique, and muscle damage is not uniformly spread through the muscle. It is therefore possible to over- or under-estimate damage as the biopsy sample may not necessarily be an accurate representation of what is happening throughout the damaged muscles. In comparison, magnetic resonance imaging is not invasive and provides an understanding of what is occurring throughout the entire muscle. T2 relaxation time has frequently been used in assessing exercise induced muscle damage, as increased signal intensity may reflect increased water content, and therefore oedema within the damaged muscle³⁸. However, magnetic resonance imaging is expensive and often inaccessible, and the interpretation of other magnetic resonance imaging findings in relation to exercise induced muscle damage remains unclear¹⁶⁷.

Consequently, indirect indicators of muscle damage are frequently used in an attempt to identify and quantify exercise induced muscle damage. The indirect indicators of exercise induced muscle damage include increased levels of muscle enzymes and proteins in the blood^{140;156}, increased inflammatory markers in the affected muscle and in the blood³⁸, muscular soreness, decreased maximal muscle strength and power, decreased joint range of movement, muscle swelling or oedema^{32;54;140;141;156;157}, increased muscle cross-sectional area on ultrasound imaging¹⁴¹, and reduced heart rate recovery¹⁴.

Muscle enzymes that have been assessed as indirect markers of exercise induced muscle damage include lactate dehydrogenase, aspartate, aminotransferase, carbonic anhydrase isoenzyme II, and creatine kinase¹⁶². Muscle proteins that have been used as indicators of exercise induced muscle damage include myoglobin, heart fatty acid binding protein, troponin, and myosin heavy chain¹⁶². The most frequently used indirect markers of muscle damage are subjective muscle soreness, maximal voluntary contraction force, and plasma creatine kinase activity³⁸.

2.2.3.1 Muscle Soreness

Muscle soreness is commonly used as an indirect measure of muscle damage in studies investigating exercise induced muscle damage and delayed onset muscle soreness^{43;60;75;102;114;141;151;170;172}. Most frequently, this is achieved through the use of subjective rating of pain scales such as the visual analogue scale^{43;141;170;172}. The visual analogue scale is a commonly used tool to determine pain intensity and is simple for participants to complete¹²³. It has been found to be sensitive, has good test-retest reliability, and is used in a variety of applications to assess pain intensity^{84;123;177}. The visual analogue scale has been validated as accurate, reliable and appropriate in evaluating pain intensity following delayed onset muscle soreness when repeated measurement is involved¹⁴⁶. The use of a multidimensional visual analogue scale has been determined to provide a clear description of muscle pain associated with delayed onset muscle soreness⁴⁴. Using the visual analogue scale, various studies have been able to establish the time course of muscle pain associated with delayed onset muscle soreness, and assess various interventions on this muscle pain^{43;91;122;168}. However, the accuracy of muscle pain in reflecting the extent of exercise induced muscle damage has been questioned¹⁴¹.

2.2.3.2 Biochemical Measurement of Muscle Damage

Various blood variables have been assessed to quantify muscle damage and monitor subsequent recovery from that damage¹⁷². Plasma creatine kinase activity is the most commonly used blood marker to determine the extent of muscle damage^{75;80;102;151;170;172}. Creatine kinase has been shown to be a reliable indicator of muscle membrane permeability as this enzyme is found exclusively inside skeletal and cardiac muscle⁴². Following muscle damage, there is diffusion of creatine kinase from the damaged muscle into the interstitial fluid and plasma³². Plasma creatine kinase activity may then be measured as a reliable indicator of the magnitude of muscle damage³². Nosaka et al¹⁴¹ did however report a poor relationship between muscle soreness, plasma creatine kinase activity and the decrement of muscle function following the induction of delayed onset muscle soreness. Additionally, large variations in plasma creatine kinase activity have been reported for subjects who showed similar decreases in maximal voluntary contraction following exercise induced muscle damage¹⁴⁰. Although the level of plasma creatine kinase activity may be accurately determined¹⁶², several studies have reported large inter-individual variability in plasma creatine kinase activity following exercise induced muscle damage, which may potentially be related to the rate of creatine kinase clearance by the muscle and reticuloendothelial system^{40;100}.

2.2.3.3 Muscle Strength and Power

Maximal voluntary contraction is often the primary means of determining muscle function following exercise induced muscle damage^{24;154;172;174}. Isometric testing using an isokinetic dynamometer has been shown to be a valid, accurate and reliable method of measuring peak isometric torque and hence the force generated by the muscle^{98;109;163;174}. Test-retest accuracy and reliability has also been established for isokinetic testing across a wide range of populations and conditions^{4;29;45;119;161;164;165}. Isokinetic measurement of peak torque is therefore an accurate and reliable method of assessing muscle function change over time following exercise induced muscle damage^{47;48;131;154;174}. Warren et al¹⁷⁴ expressed that a significant advantage of using an isokinetic isometric test is that comparisons can be made both within and between individuals as torque measurements can be made at the exact same joint angle. As consistent reductions in strength have been observed over several days following exercise induced muscle damage^{24;43;71;154}, the ability to accurately measure and track these changes over this period of time is key to understanding the effect of recovery interventions on the time course of muscle function following exercise induced muscle damage¹⁷⁰.

While an isometric isokinetic test of knee extension is a good technique to measure the specific force generated by the quadriceps muscle, a functional test such as the countermovement vertical jump provides a functional method of recording the ability of the leg extensor muscles to generate power¹⁴⁵. Furthermore, isometric testing provides a measure of maximal static force generated by the muscle, whereas the countermovement vertical jump engages muscles in lengthening and shortening muscle contractions at relatively high angular velocities^{78;83}. The countermovement vertical jump also engages the stretch shortening cycle²⁵. The effect of fatigue and exercise induced muscle damage on the stretch shortening cycle has been extensively researched^{8;97;111;138}. The validity and reliability of the countermovement vertical jump in determining functional muscle performance has been well established^{89;90;145}. A high test-retest intraclass correlation coefficient ($r = 0.88$) was measured by Slinde et al¹⁵⁸ for the countermovement vertical jump. Byrne and Eston²³ have successfully used the countermovement vertical jump to investigate the time course of the effect of exercise induced muscle damage on vertical jump performance.

2.2.3.4 Muscle oedema

Intramuscular inflammation as a result of prostaglandin E₂ production and tissue oedema from increased vascular permeability caused by the production of leukotrienes are a well-documented events in exercise induced muscle damage^{32;51}. Peak muscle soreness has also been reported as coinciding with peak intramuscular oedema levels⁸⁵, peaking between days two and five and subsiding back to normal by ten days post exercise induced muscle damage^{43;71}. Measurement of swelling is usually conducted though limb girth measurements using an anthropometric tape^{75;170;172}. Although circumference measurements are reliable at measuring limb swelling¹⁷², it encompasses total limb volume and is not specific to intramuscular volume¹⁴¹.

Cross-sectional surface area has been reliably used to calculate the volume of individual muscles including the vastus lateralis with a mean intraclass correlation coefficient of 0.998¹⁴⁸ and a high test-retest reliability ($R^2 = 0.99$)⁶⁹. Although Dierking et al⁶⁶ found that cross-sectional surface area measured by ultrasound was not sensitive enough to detect any statistically significant changes as a result of exercise induced muscle damage, Chleboun et al³⁴ used ultrasound to measure cross-sectional surface area to track the time course of muscle volume increase related to exercise induced muscle damage. Ultrasound measurement has been used to quantify change in muscle thickness of various muscles^{20;56;141}. In a study investigating muscle wasting, both the thickness of the vastus lateralis muscle and the cross-sectional surface area were measured¹⁴⁹. The changes recorded on cross-sectional surface area were also reflected in the muscle thickness measurements indicating the usefulness of measuring muscle thickness to indicate a change in muscle volume.

2.2.3.5 Heart Rate Recovery

Heart rate recovery is a technique of indirectly determining autonomic nervous system functioning following exercise induced muscle damage¹⁴. Exercise induced muscle damage has been associated with a reduction in heart rate recovery due to parasympathetic reactivation and sympathetic withdrawal^{14;15}. The heart rate interval monitoring system (HIMS) is a submaximal multi-stage shuttle run test consisting of four 2-minute running stages, each of which are run at a progressively faster pace¹¹⁵. The running stages are each separated by a one minute rest period. Heart rate recovery is determined as the difference between the heart rate at the end of the fourth running stage and the heart rate 60 s later. The HIMS has been established as a valid and reliable method of measuring heart rate recovery and therefore autonomic nervous system regulation¹¹⁶. The day-to-day repeatability of the HIMS test for each of the running and recovery stages was shown to be high (95% confidence interval, R = 0.94 – 0.99). The variation in the heart rate was reported to be 5 – 8 beats.min⁻¹ for all the stages and 7 – 19 beats.min⁻¹ for the recovery periods. The standard error of measurement of submaximal heart rate was determined to be 1.1 – 1.4%¹¹⁶.

In summary it is therefore important to be able to quantify and monitor the amount of muscle damage caused through exercise induced muscle damage to understand the extent to which exercise performance may be affected by exercise induced muscle damage and delayed onset muscle soreness.

2.2.4 EXERCISE INDUCED MUSCLE DAMAGE AND PERFORMANCE

Delayed onset muscle soreness and other symptoms related to exercise induced muscle damage have a negative impact on athletic performance^{10;32;80}. The main factors resulting in decreased performance following exercise induced muscle damage are decreased force production, altered cardiorespiratory performance, metabolic impairment, and changes in neuromuscular control⁷⁰.

2.2.4.1 Force Production

A reduction in force production has the potential to negatively influence activities that require strength, power or speed⁵². The prolonged strength loss that occurs following a damaging bout of exercise is one of the most valid and reliable indirect indicators of exercise induced muscle damage¹⁷⁴. Isometric strength is reduced immediately after prolonged or repetitive muscle lengthening actions. Repetitive stretch shortening cycle exercise usually results in a force loss of 10% to 30%³⁸, whereas high force lengthening actions, such as maximal eccentric exercise of the elbow flexors may lead to force loss of 50% to 65%³⁹. The prolonged force loss may persist for up to two weeks after the onset of muscle damage, although recovery periods are variable^{38;70;169}. In addition, Miles et al¹²⁸ reported an immediate and prolonged increase in the movement time of the elbow flexor muscles to reach peak velocity following exercise induced muscle damage. This represents impairment in the ability of the involved muscle to generate rapid force. Immediate and long lasting reductions in vertical jump performance of up to 15% have also been observed for up to four days after exercise induced muscle damage, compared to baseline jump heights²⁴. The underlying mechanisms for the force loss associated with exercise induced muscle damage have not been clearly established. It is theorised that the decreased force production may be related to pain inhibition due to the symptoms of delayed onset muscle soreness^{32;143}. However, there is a poor relationship between the time course of muscle soreness and force loss after exercise induced muscle damage^{31;137;141}. Other proposed mechanisms for the reduction in force production after exercise induced muscle damage include decreased muscle pre-activation³⁵, and a reduction in the inherent ability of the muscle to generate force^{3;24;141}.

2.2.4.2 Cardiorespiratory Performance

Exercise induced muscle damage has been associated with a decrease in cardiorespiratory performance^{14;70;81}. Gleeson et al⁸¹ reported that 15 minutes of cycling at 80% of maximum oxygen consumption two days after performing an eccentric exercise protocol resulted in increased minute ventilation, breathing frequency, respiration exchange ratio, heart rate, and rating of perceived exertion, in comparison to values after performing a concentric exercise protocol. Similar findings were reported by Eston et al⁷⁰ when comparing the effects on perceived exertion and heart rate response to treadmill running at a given speed 48 hours after bouts of concentric or eccentric exercise. These findings indicate an elevated physiological response to endurance exercise as a result of exercise induced muscle damage²⁵. Exercise induced muscle damage may also result in a reduction in heart rate recovery following moderate to heavy exercise due to parasympathetic reactivation and sympathetic withdrawal¹⁴. An elevated heart rate during exercise and a reduced heart rate recovery after exercise are associated with an impaired athletic performance⁸¹.

2.2.4.3 Metabolic Impairment

Impaired muscle glycogen resynthesis following exercise induced muscle damage has been well documented^{7;25;104;175}. Asp et al⁷ observed a 23% reduction in work capacity of muscles with exercise induced muscle damage compared to a control group, two days following eccentric exercise. This was attributed to muscle glycogen depletion due to intense eccentric exercise, and a delayed return to pre-exercise levels due to impaired glycogen resynthesis after exercise induced muscle damage. Low muscle glycogen levels are associated with a decline in endurance performance and an increase in the perception of fatigue^{10;175}. As optimal pre-exercise muscle glycogen stores are required to achieve maximal exercise performance, reduced muscle glycogen resynthesis following exercise induced muscle damage may have a detrimental effect on subsequent athletic performance until muscle glycogen levels return to pre-exercise induced muscle damage levels^{104;175}.

2.2.4.4 Neuromuscular control

Several authors have indicated that exercise induced muscle damage may negatively influence muscle coordination, proprioception and muscle recruitment patterns^{18;32;65;153}. Deschenes et al⁶⁵ showed a reduction in neuromuscular efficiency following eccentric exercise. This was determined through the examination of average electrical muscle stimulation during maximal isokinetic performance following exercise induced muscle damage. This impairment was reported to outlast other indirect markers of exercise induced muscle damage such as delayed onset muscle soreness, strength loss and elevated plasma creatine kinase activity. It was proposed that increased central activation may be required to achieve pre-determined submaximal or maximal force production after muscle damage. Brockett et al¹⁸ reported that lengthening muscle actions may disturb proprioception, although it is unclear how exercise induced muscle damage influences muscle spindle and tendon organ function. Saxton et al¹⁵³ demonstrated that participants overestimated force production by 35% in force matching tasks following exercise induced muscle damage, compared to the control arm. The ability of the exercised arm to match angles produced by the control arm was also compromised. In addition, Cheung et al³² suggested that changes in recruitment patterns or the temporal sequencing of muscle activation patterns following exercise induced muscle damage may result in changes in muscle co-ordination. Although it is currently unclear how many of these neuromuscular impairments influence exercise performance, it is possible that these changes might contribute to the loss in force production after exercise induced muscle damage, or even to an increased risk of injury following muscle damage²⁵.

2.2.5 SUMMARY

Exercise induced muscle damage is a common occurrence, particularly after repetitive lengthening muscle actions or high intensity unaccustomed exercise^{43;172}. Muscle damage is associated with delayed onset muscle soreness, stiffness, swelling, decreased range of motion, increased metabolic rate, loss of muscle function, and a decrease in strength^{10;32;51}. Various methods exist to assess and monitor the symptoms associated with exercise induced muscle damage. These measurement instruments may also assess the effectiveness of various recovery interventions.

2.3 EXERCISE RECOVERY

Training for high performance sport requires the systematic application of a training load, followed by a period during which recovery and adaptation may occur¹¹⁵. An athlete may develop symptoms of overtraining or underperformance if there is any imbalance between training load and recovery. It is well-documented that training imposes physiological stress, which leads to disturbance of the intracellular homeostasis and transient physiological and metabolic changes. This is the primary signal for stimulating adaptation of muscle and other organs in the body that are associated with improved performance^{55;115}.

However, insufficient training does not induce adequate adaptations, and may be associated with reduced exercise performance^{79;87}. In contrast, overtraining may lead to a failure to adapt, resulting in symptoms of chronic fatigue and poor performance. Chronic fatigue may also be due to insufficient recovery after training or competition¹⁰. In addition, intense training and competition may also result in exercise induced muscle damage and delayed onset muscle soreness^{10;80;156}. Exercise induced muscle damage is associated with changes in muscle function and joint mechanics^{10;32}. These factors may lead to a reduction in exercise performance and may predispose the athlete to injury³². Exercise induced muscle damage may also be related to changes in muscle glycogen levels, altered metabolic and cardiorespiratory function, increased fatigue levels, and central nervous system adaptations^{7;14;81}. It is therefore important to balance training and competition loads with recovery to optimise performance^{10;41;80}.

Recovery has been defined as the point at which the athlete is able to train without constraints of sore muscles or an increased risk of injury. The main goal of recovery is to restore cellular function to pre-exercise levels²¹. Inadequate recovery may lead to poor performance, injury, illness, or overtraining^{25;112;172}. Optimal recovery may facilitate performance gains during training, and improved performance during competition^{10;60;70}. This may be particularly evident during events that take place over multiple days²⁶.

Although recovery may be interpreted as a passive process, many athletes make use of a variety of active recovery modalities in an attempt to enhance or accelerate the recovery process^{10;17;80}. The primary purpose of active recovery modalities is to shift the stress-recovery balance away from the induced stresses as a result of exercise, and towards recovery^{10;32}, to enable a more rapid return to pre-exercise performance levels. It is theorised that accelerated recovery may reduce the risk of injury, enable the athlete to tolerate higher training loads, or enhance the effect of a given training load, thereby increasing performance levels over time¹⁰.

2.3.1 PHYSIOLOGY OF RECOVERY

The goal of recovery is to restore cellular and muscle function to pre-exercise levels²¹. Exercise results in an increased metabolic rate and increased oxygen consumption. The cessation of exercise is associated with an initial rapid decline in oxygen consumption, followed by a more progressive decline in oxygen consumption over approximately one hour after exercise¹⁶. Lactate concentrations may increase up to 10 times pre-exercise levels during activity, and may take up to one hour to return to pre-exercise concentrations^{10;46}.

Historically, high concentrations of lactate and hydrogen ions in the muscle have been associated with muscle fatigue. However, this theory is not supported by current evidence, which has failed to identify a consistent relationship between recovery of post-exercise lactate concentrations and performance⁵⁰. It is proposed that decreased performance levels that occurred in the presence of increased lactate concentrations after exercise may be related to down-regulation of the central nervous system, rather than lactate acidosis having a detrimental effect on muscle contraction²⁷. As such, active recovery modalities aimed at lowering lactate concentrations may not be an effective method of accelerating exercise recovery.

Muscle glycogen concentrations decrease with exercise, and continue to decline after exercise has been terminated⁷. Exercise induced muscle damage has also been associated with glycogen depletion and delayed resynthesis¹¹⁷. As previously discussed (Section 2.2.4.3, page 19) optimal pre-exercise muscle glycogen stores are required for maximal exercise performance^{10;46;104;175}. Muscle glycogen resynthesis and restoring concentrations to pre-exercise levels are therefore important considerations for optimal recovery, particularly after a damaging bout of exercise¹⁰.

It has been proposed that minimising inflammation after muscle damage, or enhancing its rate of removal may have a positive effect on recovery¹⁰. However inflammatory processes are involved in both the damage and repair processes associated with exercise induced muscle damage¹⁷². Further, although the symptoms of delayed onset muscle soreness usually dissipate within five to seven days after exercise induced muscle damage, the muscle continues to regenerate, with signs of regeneration being present at two weeks after the damaging bout of exercise. After severe muscle injury, for example marathon running, signs of regeneration have been observed for up to 12 weeks after exercise induced muscle damage¹⁷³. Therefore the modification of the acute inflammatory process or the regeneration process may be inappropriate as this may compromise the restoration of cellular function and regeneration to pre-exercise levels¹¹⁷.

The adaptations associated with exercise induced muscle damage result in a decrease in athletic performance. Recovery after exercise induced muscle damage is important in both minimising the negative effects of exercise induced muscle damage as well as enhancing the positive adaptations of regular training. Many active recovery modalities have been investigated and are currently used by athletes and sports teams to accelerate recovery and improve exercise performance^{10;32;51;80}.

2.4 RECOVERY MODALITIES

Numerous studies have attempted to identify effective recovery interventions that may minimise the negative effects of exercise induced muscle damage and delayed onset muscle soreness, and optimise the recovery process. The recovery modalities that have been investigated include cryotherapy^{10;28;32;57;156;172;176}, thermotherapy^{172;176}, contrast temperature therapy^{10;80;172;176}, intermittent pneumatic compression^{28;110}, hyperbaric oxygen therapy¹⁰, non-steroidal anti-inflammatory drugs and analgesics^{10;32;51}, compression garments^{10;80}, stretching^{10;32;51}, massage^{10;32;51}, electrotherapy^{10;32;51}, homeopathy^{32;51}, vibration⁵¹ and low intensity exercise^{10;32;80}. Although there is a lack of substantial evidence regarding the effectiveness of the majority of these recovery interventions, the use of various recovery strategies and modalities remains a common practice throughout the sporting community^{10;80}. A detailed review of all of the different recovery modalities is beyond the scope of this literature review. This review will focus on the effects of contrast temperature therapy and intermittent compression with regards to recovery from exercise induced muscle damage and delayed onset muscle soreness.

Temperature therapies are popular recovery modalities, and include cryotherapy, thermotherapy, or contrast temperature therapy, which is a combination of hot and cold therapies^{80;102;172}. Temperature therapy is usually conducted in the form of water immersion, and is used widely across numerous sports codes^{46;94;171}. Anecdotally, many coaches and athletes support the use of different temperature therapies to improve recovery⁴⁶. However, there is little scientific evidence to support temperature therapy in improving recovery^{32;51;102;143;172;176}. Recent technological advances have made it possible to apply multiple modalities simultaneously. One such combination is the simultaneous application of contrast temperature therapy with intermittent compression.

2.5 CONTRAST TEMPERATURE THERAPY

Contrast temperature therapy consists of the alternate application of hot and cold treatment. Different contrast temperature therapy methods include alternating immersion in hot and cold baths, a hot shower and cold bath, or the application of hot and cold packs^{17;46;94}. The application of contrast temperature therapy varies between 30 and 300 seconds of treatment per hot or cold cycle, with the total duration of treatment being four to 30 minutes^{46;176}. Most commonly, it is applied in the hot to cold ratio of 3:1 or 4:1 for two or three cycles. The hot temperature ranges between 37 °C and 43 °C, and the cold temperature ranges between 8 °C and 15 °C^{17;46}. Summaries of recent studies investigating the effects of contrast temperature therapy as a recovery modality are presented in Tables 2.1 (page 26) and 2.2 (page 27).

The application of cryotherapy is associated with a reduction in skin and subcutaneous tissue temperature, which results in vasoconstriction of blood vessels and decreased swelling and inflammation⁴⁶. Thermotherapy results in increased tissue temperature and metabolite production, which leads to increased circulation to the treatment area and a reduction of muscle spasm^{46;94}. Contrast temperature therapy has been reported to have a number of effects including increased circulation, lactate clearance, and range of movement; and decreased plasma creatine kinase activity, swelling, pain, stiffness, and delayed onset muscle soreness^{46;94;114;132-134;170-172}. Contrast temperature therapy has also been associated with a perception of improved recovery⁴⁹. Although contrast temperature therapy is commonly used as a clinical treatment modality and as a recovery modality across a variety of sports, the underlying physiological mechanisms of contrast temperature therapy remain poorly understood⁹⁴.

Table 2.1: Outline of studies that have investigated contrast temperature therapy as a recovery modality

Authors	Study design	Total sample (contrast temperature therapy sample)	Age (years)	Inclusion criteria	Exclusion criteria	Results for contrast temperature therapy
Coffey et al ⁴⁹	Randomised multiple cross-over design	14 (14)	19-33	Highly active males	Females	Accelerated reduction in blood lactate concentration following high intensity running compared to passive recovery ($p < 0.05$). No difference in running performance
Dawson et al ⁶⁰	Repeated trials cross-over design	17 (17)	21-27	Semi-professional Australian Football League players	Not mentioned	No difference compared to passive recovery directly after match followed by active recovery the next day
French et al ⁷⁵	Match-paired, between-group design with repeated measures	26 (10)	18-30	Healthy men with > 1 year experience of resistance training	Females	No difference to passive recovery. Transient decrease in post exercise soreness at 1 hour ($p \leq 0.05$). No difference thereafter
Gill et al ⁸⁰	Randomised group selection	23 (groups not described)	22-28	Elite male rugby players	Females	Decreased creatine kinase activity at 36 and 48 hours post-match compared to passive recovery ($p < 0.05$)
Hamlin ⁸⁸	Randomised cross-over design	20 (20)	18-20	Junior representative rugby players	Not mentioned	Accelerated reduction in blood lactate concentration and lower heart rate in subsequent repeat sprint test, but little effect on subsequent performance compared to active recovery ($p < 0.05$).
Ingram et al ¹⁰²	Randomised cross-over design	11 (11)	21-33	Males athletes with team games experience	Females	Perceived pain less than passive recovery at 24hrs ($p < 0.05$)
Kuligowski et al ¹¹⁴	Randomised Sex-matched	56 (14)	18-24	University students	Upper extremity weight training in past 9 weeks Contraindications to hot/cold Poor health	Accelerated improvement of reduced passive range of movement ($p = 0.006$) and enhanced improvement of perceived muscle pain ($p = 0.001$) compared to passive recovery
Morton ¹³²	Randomised cross-over design	11 (11)	20-22	Moderate exercise 3-4 times per week	Regular high intensity exercise	Quicker reduction in blood lactate concentration compared to passive recovery ($p < 0.001$)
Robey et al ¹⁵¹	Semi-randomised cross-over design	20 (20)	18-22	Amateur rowers who had been competitive for at least a year	Not mentioned	No accelerated recovery compared to passive recovery
Vaile et al ¹⁷¹	Randomised cross-over design	12 (12)	27-36	Endurance trained male cyclists	Females	Better maintained 5 day performance for cycling sprint ($p < 0.01$) and time trial ($p < 0.05$), compared to passive recovery which dropped off. No changes in rating of perceived exertion or heart rate
Vaile et al ¹⁷²	Randomised cross-over design	38 (15)		Strength trained males	Females	Improved isometric force and peak power performance recovery at 24, 48 and 72hrs compared to passive recovery ($p < 0.05$). Decreased perceived pain and oedema at 24, 48 and 72hrs compared to passive recovery ($p < 0.01$)
Vaile et al ¹⁷⁰	Randomised cross-over design	13 (13)		Recreational athletes	Not mentioned	Less loss of muscle force with isometric squat in 24-48 hr period compared to passive recovery ($p < 0.05$). Smaller increase and quicker reduction in thigh volume compared to passive recovery ($p < 0.05$)

Table 2.2: Contrast temperature therapy protocols used by studies that have investigated contrast temperature therapy as a recovery modality

Authors	Method of exercise induced muscle damage induction	Method of contrast temperature therapy application	Hot/Cold temperature range (°C)	Duration	Frequency of application
Coffey et al ⁴⁹	High intensity treadmill running	Immersion to ASIS	10 / 42	1 min cold, 2 min hot, 5 times each	Once immediately following 1 st running block
Dawson et al ⁶⁰	Australian football league match	Hot shower and cold immersion to waist	12 / 45	1 min cold, 2 min hot, 5 x hot, 4 x cold	Once 15 – 20 minutes after exercise
French et al ⁷⁵	Concentric and eccentric resistance exercises	Seated in 50cm water depth with legs outstretched	8-10 / 37-40	1 min cold, 3 min hot, 4 x cold, 3 x hot	Once within 10 minutes after exercise
Gill et al ⁸⁰	Competitive rugby union match	Immersion to ASIS	8-10 / 40-42	1 min cold, 2 min hot, 3 times each	Once immediately following rugby match
Hamlin ⁸⁸	Repeated running sprint	Ice bath to hip and hot shower	8-10 / 38	1 min cold, 1 min hot, 3 times each	Once immediately after exercise
Ingram et al ¹⁰²	Simulated team sport exercise and shuttle run to exhaustion	Immersion to umbilicus	10 / 40	2 min cold, 2 min hot x 3	Once immediately after exercise
Kuligowski et al ¹¹⁴	Eccentric elbow flexor resistance exercise	Immersion of arms to mid deltoid	12.8 / 38.9	1 min cold, 3 min hot, x 6	Once immediately after exercise
Morton ¹³²	Intense cycling	Immersion to gluteal fold	12 / 36	9 min hot, 1 min cold then 4 min hot, 1 min cold x 4	Once immediately after exercise
Robey et al ¹⁵¹	Strenuous stair-climb running	Hot shower and cold immersion to waist	12 / 40	2 min hot, 1 min cold x 5	Post exercise and at 24 and 48 hours
Vaile et al ¹⁷¹	Fatigue inducing cycling protocol	Immersion to neck	15 / 38	1 min cold, 1 min hot x 7	Once Immediately after exercises for each of 5 days of trial
Vaile et al ¹⁷²	Eccentric leg press resistance exercise	Immersion to neck	15 / 38	1 min cold, 1 min hot x 7	Immediately and once a day for 72hrs
Vaile et al ¹⁷⁰	Eccentric leg press resistance exercise	Immersion to ASIS	8-10 / 40-42	1 min cold, 2 min hot x 5	Once immediately after exercise

2.5.1 PHYSIOLOGICAL MECHANISMS OF CONTRAST TEMPERATURE THERAPY

It is theorised that the underlying physiological mechanisms of contrast temperature therapy may be related to repetitive vasodilation and vasoconstriction from the alternate cryotherapy and thermotherapy application, which results in a “pumping action”. This may assist recovery through the removal of metabolites such as lactate and hydrogen ions, the reduction of intracellular fluid, and the removal of cellular debris^{46;134;171;176}. However, contrast temperature therapy may only affect subcutaneous tissue temperature^{10;133;134}. Higgins and Kaminski⁹³ reported that intramuscular temperature at a depth of four centimetres below the surface of the skin did not fluctuate following repeated contrast temperature therapy with one-minute immersions into hot and cold water. The ability to achieve a vaso-pumping effect without a change in intramuscular temperature has subsequently been questioned¹⁷⁶.

Fiscus et al⁷³ demonstrated significantly increased arterial blood flow in the lower leg with a 20-minute contrast temperature therapy application consisting of a 4:1 hot to cold ratio of water immersion, compared to 20 minutes of no immersion. There was a fluctuation in circulation, with increased blood flow during the change from cold to hot water immersion, and decreased blood flow during cold water immersion. The decreased blood flow during the cold water immersion cycle represented a return to baseline levels of circulation, rather than true vasoconstriction. It was proposed that the fluctuations in blood flow were due to changes in cutaneous circulation rather than intramuscular circulation, and were therefore unlikely to result in a vaso-pumping action. In addition, it has been reported that immersion into ice water with high body temperatures such as following intense exercise or after immersion in hot water may cause vasodilation rather than vasoconstriction, as a “shock response”. However, the underlying mechanisms for this “shock response” are not clearly understood^{13;108}.

Furthermore, it has also been suggested that the reduction in swelling associated with contrast temperature therapy may not be related to circulatory changes, but that other mechanisms may be involved^{53;133;134}. Cotts et al⁵³ reported that a reduction in swelling may be achieved through cellular debris and fluid removal via the lymphatic system. However, the lymphatic system acts independently of circulatory changes and requires either gravity or muscle contraction to move lymphatic fluid. It is therefore unclear how contrast temperature therapy would enhance lymphatic drainage through temperature change alone.

As contrast temperature therapy is most commonly implemented as contrast water immersions, several authors have suggested that any benefits of contrast temperature therapy may be related to the effects of hydrostatic pressure rather than the alternating temperature application^{93;133;134}. Vaile et al¹⁷⁰ observed significant reductions in thigh volume for up to 72 hours after exercise induced muscle damage following 15 minutes of contrast temperature therapy using water immersion to the level of the anterior superior iliac spine, compared to a passive recovery group. The mean thigh girth measurements increased by $0.6 \pm 0.6\%$ in the contrast temperature therapy group, compared to $2.3 \pm 0.8\%$ in the passive recovery group. The significantly lower thigh volume in the contrast temperature therapy group was interpreted as a reduction in oedema within the thigh muscles. In contrast, French et al⁷⁵ found no difference in mean thigh girth between a contrast temperature therapy group immersed to 0.5 m and a control group after a damaging bout of exercise. Furthermore, Cote et al⁵² reported a 27% increase in oedema over three consecutive days of contrast temperature therapy treatment using localised water immersion on acute ankle sprain injuries. Wilcock et al¹⁷⁶ proposed that as hydrostatic pressure increases by $0.74 \text{ mmHg.cm}^{-1}$. Hydrostatic pressure causes an inward and upward displacement of fluid within the body. Therefore, differences in results between studies investigating contrast temperature therapy may be due to higher hydrostatic pressures generated in the studies that immerse in a greater depth of water. Water immersion contrast temperature therapy may reduce oedema through increased extracellular fluid transfer into the vascular system, and increased cardiac output.

Hydrostatic pressure is not the only mechanism that should be considered with regards to the physiological changes seen with contrast temperature therapy. Fiscus et al⁷³ investigated arterial blood flow in cold water immersion, warm water immersion and contrast temperature therapy immersion. The immersion depth was the same for each intervention but the arterial blood flow measured during immersion was different for each condition. Contrast temperature therapy produced fluctuations in blood flow throughout the 20 minute immersion period; warm water immersion resulted in a significant increase in blood flow compared to the control condition (no immersion); and cold water immersion was not significantly different to the control condition. The differences in arterial blood flow were attributed to the temperature differences.

Bonde-Peterson et al¹³ observed circulatory differences following warm water immersion, cold water immersion, thermoneutral water immersion and a control (no immersion). They reported a trend to increased stroke volume with immersion compared to no immersion, with the thermoneutral and cold water immersion having the highest effect. Heart rate was decreased by 15% for thermoneutral and cold water immersion, whereas heart rate was increased by 32% for hot water immersion. Significant increases in cardiac output were also reported for thermoneutral water immersion (18%) and hot water immersion (44%). Cold water immersion had no effect on cardiac output. Arterial pressure and total peripheral resistance were significantly increased with cold water immersion while a significant decrease in total peripheral resistance was found for hot water immersion. Thermoneutral water immersion had insignificant effects on arterial pressure and total peripheral resistance. The immersion depth was the same for each condition, which led the authors to conclude that temperature had an overriding effect on hydrostatic pressure and temperature therefore was responsible for the differences between the groups¹³. It is however unclear whether circulatory changes have any effect on recovery from exercise induced muscle damage.

Several studies have investigated modalities of cold water immersion, contrast temperature therapy immersion, hot water immersion and thermoneutral water immersion as recovery modalities^{156;171;172}. Although the depth of water immersion, and therefore hydrostatic pressure, was the same for the different temperature modalities, differences in results were recorded between groups. Vaile et al¹⁷¹ reported that cold water immersion and contrast temperature water immersion improved recovery from high intensity cycling, enabling subjects to better maintain performance over five days of cycling, compared to hot water immersion and passive recovery. Vaile et al¹⁷² found that cold water immersion and contrast temperature water immersion were effective at improving recovery of isometric force and dynamic power, and reducing local oedema following delayed onset muscle soreness compared to passive recovery, while hot water immersion was only effective in recovery of isometric force. Sellwood et al¹⁵⁶ investigated cold water immersion compared to tepid water immersion (24 °C). The authors reported no difference between the two modalities to recovery from delayed onset muscle soreness, with the exception that the cold water immersion group experienced a greater amount of pain associated with delayed onset muscle soreness than tepid water immersion. Unfortunately none of these studies specifically compared contrast temperature therapy immersion to thermoneutral water immersion with regards to recovery from exercise induced muscle damage. These studies do however indicate that temperature, in addition to hydrostatic pressure, may be an important component of contrast temperature therapy.

In summary, the proposed physiological mechanisms of contrast temperature therapy are not well-understood. Contrast temperature therapy has little effect on deep muscle temperature^{93;94}. Contrasting temperature has been shown to only cause an increase in superficial circulation^{10;133;134} placing doubt on the theory of vaso-pumping^{93;176}. Hydrostatic pressure has been proposed as an important component in the positive effects measured in studies investigating the effects of contrast temperature therapy on recovery^{93;133;134}. However, hydrostatic pressure has also been questioned as the sole mechanism through which contrast temperature therapy achieves results¹⁰². Various studies investigating the effect of water immersion modalities on exercise recovery have achieved different results for different temperatures of water immersion while using the same depth of water, thereby generating the same amount of hydrostatic pressure^{102;156;171;172;176}. This indicates that the temperature of contrast temperature therapy is also an important factor in achieving positive effects on recovery. The next section of the review will focus on the effects of contrast temperature therapy on recovery and exercise performance.

2.5.2 EFFECTS OF CONTRAST TEMPERATURE THERAPY ON RECOVERY AND EXERCISE PERFORMANCE

A recent systematic review of contrast temperature therapy concluded that there was insufficient scientific evidence to support the use of contrast temperature therapy to aid recovery⁹⁴. This was mainly due to the poor methodological quality of the studies investigating contrast temperature therapy. Numerous studies have investigated the effects of contrast temperature therapy on exercise recovery. Table 2.3 (page 33) provides a summary of the effects of contrast temperature therapy on different outcome measures associated with recovery and exercise performance.

2.5.2.1 Muscle soreness

Numerous studies have determined the effects of contrast temperature therapy on perceived pain following exercise induced muscle damage or intense exercise^{60;75;102;114;151;170;172}. All studies used self-reported pain scales to quantify pain levels. Dawson et al⁶⁰ and Robey et al¹⁵¹ used a seven point Likert scale to measure pain, while Ingram et al¹⁰² used a 10 point Likert scale. Ten centimetre visual analogue scales were also utilised^{75;170;172}, as was a 12 cm graphic pain rating scale¹¹⁴. Pain levels were recorded for 48 hours^{60;75;102}, 72 hours^{151;170;172}, or 96 hours¹¹⁴ after the application of the recovery modalities. Four of the studies failed to show any benefit of contrast temperature therapy over passive recovery for recovery of muscle soreness following exercise induced muscle damage^{60;75;151;170}. In contrast, Ingram et al¹⁰² demonstrated a significant decrease in pain following contrast temperature therapy at 24 hours after exercise induced muscle damage, compared to passive recovery. Vaile et al¹⁷² also reported a reduction in pain at 24, 48 and 72 hours post-exercise after contrast temperature therapy, compared to passive recovery. Furthermore, Kuligowski et al¹¹⁴ observed a significantly improved rate of reduction in pain after exercise induced muscle damage following contrast temperature therapy, compared to passive recovery.

Table 2.3: Effects of contrast temperature therapy on outcome measures

Authors	Muscle soreness	Plasma creatine kinase activity	Other blood markers of muscle damage	Swelling (limb volume)	Blood lactate	Blood pH	Heart Rate	Core Temp	Perceived exertion	Passive range of movement	Active range of movement	Endurance performance	Sprint performance	Peak Force	Explosive power
Coffey et al ⁴⁹					+	0	0		0			0			
Dawson et al ⁶⁰	0										0		0		0
French et al ⁷⁵	0	0	0	0						0			0	0	0
Gill et al ⁸⁰		+													
Hamlin ⁸⁸					+		+						0		
Ingram et al ¹⁰²	+	0	0										0	0	
Kuligowski et al ¹¹⁴	+									+	0			0	
Morton ¹³²					+										
Robey et al ¹⁵¹	0	0										0		0	
Vaile et al ¹⁷¹							0	0	0			+	+		
Vaile et al ¹⁷²	+	0	0	+										+	+
Vaile et al ¹⁷⁰	0	0		+										+	+

Key: 0 : no beneficial effect of contrast temperature therapy on recovery
 + : positive effect of contrast temperature therapy on recovery
 - : negative effect of contrast temperature therapy on recovery

2.5.2.2 Biochemical markers of muscle damage

Six of the studies investigating the effects of contrast temperature therapy on recovery evaluated biochemical markers of muscle damage^{75;80;102;151;170-172}. All studies investigated either plasma creatine kinase activity or interstitial creatine kinase activity as indirect markers of muscle damage. Plasma myoglobin^{75;102;172}, C-reactive protein¹⁰², interleukin-6¹⁷² and lactate dehydrogenase¹⁷² concentrations were also assessed. Gill et al⁸⁰ was the only study that determined positive effects of contrast temperature therapy on biochemical markers of muscle damage, where an increased rate of reduction in interstitial creatine kinase activity was observed following contrast temperature therapy, compared to no treatment. None of the other studies showed a positive or negative effect of contrast temperature therapy on any of the biochemical markers of muscle damage^{75;102;151;170;172}.

Gill et al⁸⁰ compared the effect of various recovery modalities on recovery after a rugby match. These consisted of passive recovery, active recovery of seven minutes of low intensity stationary cycling, three cycles of contrast temperature therapy immersion to the level of the anterior superior iliac spines for one minute cold (8 – 10 °C) and three minutes of hot (40 – 42 °C), and compression garments. Interstitial creatine kinase activity was reduced following contrast temperature therapy, active recovery and compression garments, compared to passive recovery at 36 and 48 hours post-match. There were no significant differences in interstitial creatine kinase activity between the three recovery modalities. Rugby is a physical game that involves a large number of collisions between players. It is therefore a possibility that muscle damage as a result of physical impacts in addition to that caused from the exercise itself, had an effect on interstitial creatine kinase concentration⁸⁰. In addition, all of the other studies have measured plasma creatine kinase activity rather than interstitial creatine kinase activity⁸⁰, making these data difficult to compare.

2.5.2.3 Swelling

Three studies used limb girth measurements of the thigh^{75;170;172} and lower leg⁷⁵ as an indirect quantification of swelling following exercise induced muscle damage. Measurements were taken at the midpoint of the thigh and around the calf muscle where the circumference was the greatest⁷⁵; or at three sites on the thigh, including above the knee, mid-thigh and sub-gluteal^{170;172}. French et al⁷⁵ reported no benefit of contrast temperature therapy over passive recovery, whereas the other two studies^{170;172} found that the mid-thigh girth was decreased following contrast temperature therapy when compared to passive recovery. The authors suggested that the decreased volume may be due to an increase in the reabsorption of interstitial fluid due to the hydrostatic pressure of the water^{170;172}. They also suggested that a reduction in post exercise swelling is considered to improve the contractile function of muscle and may also decrease secondary muscle damage. On examination of the immersion methods used in the three studies, it is noted that French et al⁷⁵ made use of an immersion protocol sitting in 50 cm of water whereas both of the Vaile studies^{170;172} used standing in deeper water, to the neck and to the level of the anterior superior iliac spine respectively. Compression is known to have a significant effect on swelling¹¹ and since hydrostatic compression would be much greater in deeper water, it would therefore have a greater effect on swelling. This is a possible explanation for the difference in results found by the studies.

2.5.2.4 Blood lactate

The three studies that investigated the effect of contrast temperature therapy on blood lactate concentration following exercise induced muscle damage all found contrast temperature therapy to be beneficial in decreasing the elevated post exercise induced muscle damage blood lactate levels^{49;88;132}. All three studies sampled blood from the finger tip for blood lactate analysis^{49;88;132}. Coffey et al⁴⁹ investigated recovery following a treadmill run to exhaustion over a four hour period. Contrast temperature therapy (n = 14) consisted of immersion to the level of the anterior superior iliac spines, with one minute of cold water immersion (10 °C) and two minutes of hot water immersion (42 °C) for five repetitions.

Contrast temperature therapy showed a quicker return to normal blood lactate concentrations after exercise than passive recovery. Hamlin⁸⁸ compared the effectiveness of six minutes of either active recovery or contrast temperature therapy on recovery following a repeated sprint test. Twenty junior representative rugby players (17 male, three female) were involved in this randomised crossover study. Contrast temperature therapy consisted of an ice bath (8 - 10° C) with immersion to the hip for one minute alternated with a hot shower (38° C) for one minute, repeated three times. Active recovery took the form of six minutes of slow jogging at 6.8 km.h⁻¹. Contrast temperature therapy significantly lowered blood lactate concentrations compared to active recovery. Morton¹³² investigated the effects of contrast temperature therapy, cold water immersion and passive recovery on recovery from intense anaerobic exercise with moderately exercised individuals (n = 11). Contrast temperature therapy consisted of immersion to the level of the gluteal folds, initially for nine minutes of hot (36° C) and one minute cold (12° C), followed by four cycles of four minutes hot and one minute cold. The contrast temperature therapy group showed a quicker reduction in blood lactate concentrations, compared to cold water immersion and passive recovery.

It is noted that all three of these studies investigated short term recovery from relatively low impact exercise, when compared to the exercise induced muscle damage protocols used by other studies investigating the effects of contrast temperature therapy on recovery. The three studies made use of treadmill running⁴⁹, repeated sprint running⁸⁸ and intense cycling¹³² respectively to induce muscle fatigue and damage. These methods produce relatively less muscle damage than exercise protocols utilising high intensity lengthening muscle actions and stretch shortening cycles^{19;141}. Unfortunately, none of the studies that utilised these more damaging protocols measured blood lactate as an outcome measure in their studies to provide a comparison^{75;114;170;172}. It is also noted that no performance benefits associated with the decreased blood lactate concentrations following recovery with contrast temperature therapy^{49;88}.

2.5.2.5 Blood pH

High intensity exercise produces lactate and hydrogen ions which may be associated with a reduction in blood pH¹²⁴. Increased hydrogen ion concentrations and decreased blood pH have been implicated in the reduction of the force and velocity of muscle contractions, and as potential contributing factors to muscle fatigue⁴⁹. Coffey et al⁴⁹ extracted blood from a fingertip site to assess blood pH as a variable for recovery from exercise induced muscle damage. No differences were noted in blood pH between the contrast temperature therapy and passive recovery group following recovery from repeated four one-hour treadmill runs.

2.5.2.6 Heart rate

Three studies investigated different aspects of heart rate using contrast temperature therapy as a recovery modality^{49;88;171}. Coffey et al⁴⁹ investigated heart rate recovery following two sets of treadmill running to exhaustion, separated by active recovery, contrast temperature therapy or passive recovery. There were no differences in heart rate between the contrast temperature therapy and passive recovery groups during the first set of running, the recovery phase, or the second set of running. Vaile et al¹⁷¹ used heart rate as a measure of next day performance following strenuous cycling over a five day period. Each day the cycling was followed by a cold water immersion (15 °C for 14 minutes), hot water immersion (38 °C for 14 minutes), contrast temperature therapy (15 °C and 38 °C for one minute each repeated seven times) or passive recovery. Immersion was standing with the water up to the level of the neck. No significant differences in heart rate were found between contrast temperature therapy and passive recovery. Hamlin⁸⁸ studied the effects of contrast temperature therapy compared to active recovery as recovery modalities applied between two bouts of repeated sprints. Contrast temperature therapy was reported to have a beneficial effect on average heart rate measured during both the recovery between bouts and on the average heart rate during the second bout of repeated sprints, compared to active recovery. However, there was no performance difference noted. Unfortunately the study did not include a passive recovery group for comparison.

2.5.2.7 Core temperature

Vaile et al¹⁷¹ used core temperature as an outcome measure as part of a study investigating the effect of water immersion on next day cycling performance over five days of strenuous cycling. The water immersion consisted of 14 minutes of cold water immersion, hot water immersion, contrast temperature therapy (immersion to the neck for one minute at 15° C and one minute at 38° C for seven cycles) or passive recovery. Core temperature was measured before and after cycling, before and after the recovery modality, and 15 minutes after recovery. There were no significant differences in core temperature between contrast temperature therapy and passive recovery. It was theorised that the short-duration applications of heating and cooling required for contrast temperature therapy may not influence core temperature. However, further studies are required to confirm this hypothesis.

2.5.2.8 Perceived exertion

Two studies investigated the effects of contrast temperature therapy on perceived exertion during recovery, using different measurement tools. Vaile et al¹⁷¹ asked participants to rate their exertion on a scale of zero to ten on several occasions throughout the study, before and after the recovery interventions. The participants performed a fatigue inducing cycling protocol over a five day period. Coffey et al⁴⁹ utilised a perceived exertion rating method where participants were instructed to rate stress, discomfort and fatigue that remained in their legs at that specific moment¹⁵⁰. This was also completed on various occasions throughout the exercise protocol consisting of high intensity treadmill running. In both studies there were no significant differences in perceived exertion during recovery following the use of contrast temperature therapy, compared to passive recovery.

2.5.2.9 Range of movement

Both active and passive ranges of movement have been used in studies investigating the effect of contrast temperature therapy on recovery. French et al⁷⁵ evaluated the passive range of movement into the movements of hip flexion, extension and abduction, knee flexion and extension, and ankle dorsiflexion. Images were digitised and analysed using computer software. No significant differences were found between contrast temperature therapy (one minute at 8 - 10 °C and three minutes at 37 - 40 °C for four cycles of cold and three cycles of hot water immersion seated at a depth of 50 cm) and passive recovery groups.

Active range of movement was assessed using the sit and reach test by Dawson et al⁶⁰. No significant differences were found between contrast temperature therapy (three minutes shower at 45 °C and one minute cold water immersion at 12 °C to the level of the waist, for five cycles of hot and four cycles of cold) and passive recovery groups. Kuligowski et al¹¹⁴ assessed both active and passive range of movement using a goniometer. Resting elbow flexion and active elbow flexion and extension were measured following exercise induced muscle damage. The contrast temperature therapy recovery group showed a quicker return to baseline of resting elbow flexion than passive recovery. Contrast temperature therapy consisted of immersion to the level of the mid deltoid muscle for six cycles of cold for one minute (12.8 °C) and three minutes of hot (38.9 °C). No differences were found for active elbow flexion and extension range of movement.

2.5.2.10 Endurance and sprint performance

Previous studies have investigated the effects of contrast temperature therapy on endurance performance^{49;151;171} and sprint performance^{60;75;88;102;171}. There were no significant differences in exercise performance following contrast temperature therapy or passive recovery during two sets of treadmill running to exhaustion⁴⁹; a 2 km ergometer time trial following strenuous stair-climb running¹⁵¹; a six second cycling sprint following an Australian football match⁶⁰; 10 m and 30 m running sprints following a bout of exercise that induced muscle damage⁷⁵; two sets of repeated 40m running sprints (compared to active recovery)⁸⁸; and repeated 20 m running sprints following simulated team sports exercise¹⁰². Only Vaile et al¹⁷¹ reported a benefit of contrast temperature therapy over passive recovery for average cycling power on a repeated sprint and a nine minute time trial over five consecutive days of testing.

Ingram et al¹⁰² investigated recovery from simulated team sport exercise followed by 20 m shuttle runs to exhaustion. Contrast temperature therapy consisted of immersion to the waist for three cycles of two minutes at 10 °C and two minutes at 40 °C. There was no difference in the ability to perform the protocol for a second time following either contrast temperature therapy or passive recovery. Robey et al¹⁵¹ also showed no benefit in performance recovery of contrast temperature therapy over passive recovery following strenuous stair climbing. A contrast therapy protocol of a hot shower at 40 °C for two minutes and 12 °C cold water immersion to the waist for 1 minute, repeated five times was used.

2.5.2.11 Peak force and explosive power

There were no significant differences in recovery of peak force as measured by a five repetition maximum back squat⁷⁵; maximal voluntary contraction of the quadriceps, hamstrings and hip flexors¹⁰²; maximal voluntary isometric elbow flexion¹¹⁴; and concentric isometric leg extension peak torque¹⁵¹ following contrast temperature therapy, compared to passive recovery. However, there were significant improvements in maximal isometric squat with the implementation of contrast temperature therapy, compared to passive recovery following exercise induced muscle damage^{170;172}. Two studies found no significant differences in muscle power following contrast temperature therapy, compared to passive recovery^{60;75}. These studies used the vertical jump⁶⁰ and countermovement jump tests⁷⁵ to assess recovery after exercise induced muscle damage. A further two studies demonstrated a positive effect of contrast temperature therapy on recovery compared to passive recovery, by utilising a weighted squat jump test to measure power. Interestingly, these are the same two studies that reported positive findings for contrast temperature therapy over passive recovery for peak force^{170;172}.

Vaile et al¹⁷⁰ studied 13 recreational athletes to compare the effects of contrast temperature therapy and passive recovery on various markers of exercise induced muscle damage. Contrast temperature therapy consisted of five cycles of cold water for one minute at 8 – 10 °C and hot water for two minutes at 40 – 42 °C. Peak force and peak power were significantly higher at 24 and 48 hours post-exercise following contrast temperature therapy, compared to passive recovery. It was proposed that contrast temperature therapy was associated with a more rapid recovery of power production after muscle damage, compared to passive recovery. Vaile et al¹⁷² also observed the effects of contrast temperature therapy on recovery following eccentric leg press exercises in 15 strength trained males. Contrast temperature therapy was administered through immersion to neck with alternating durations of one minute in cold water (15 °C) and hot water (38 °C) for seven cycles. There were significantly improved recovery rates for muscle power and isometric force production after contrast temperature therapy, compared to passive recovery. This is further supported in that both of the Vaile studies^{170;172} that investigated the effect of contrast temperature therapy on recovery of limb volume following delayed onset muscle soreness inducing exercise, found a significant improvement over passive recovery with the use of contrast temperature therapy by means of immersion.

In contrast, Dawson et al⁶⁰ used a repeated crossover design to compare the effects of passive recovery, stretching, active recovery consisting of pool walking for 15 minutes, and contrast temperature therapy on recovery in 17 semi-professional Australian football league players. Contrast temperature therapy comprised of a hot shower (45 °C) for two minutes and an ice bath with immersion to waist (12 °C) for one minute, for five cycles of hot and four of cold. There were no significant differences in explosive power between the contrast temperature therapy and passive recovery groups. Further, French et al⁷⁵ compared the effects of contrast temperature therapy, compression garments and passive recovery on recovery in healthy males following eccentric resistance exercises to induce muscle damage. Contrast temperature therapy was implemented through water immersion in 50 cm of water with the legs outstretched. Four cycles of cold water immersion for one minute (8 – 10 °C) and three cycles of hot water for three minutes (37 – 40 °C) were performed. There were no significant differences in countermovement jump performance between the contrast temperature therapy and passive recovery groups.

2.5.2.12 Summary of literature: Effects of contrast temperature therapy on recovery and exercise performance

In summary, most studies demonstrated some positive effects of contrast temperature therapy on the markers of recovery^{49;80;88;102;114;132;170-172}. There were three studies that failed to identify any benefit for contrast temperature therapy on recovery^{60;75;151}. In addition, no negative effects of contrast temperature therapy on recovery were reported by any of the studies. Few studies showed any effect of contrast temperature therapy on recovery for sprint or endurance performance, peak force production, or explosive power production. Only three studies reported positive effects of contrast temperature therapy on recovery for performance measures¹⁷⁰⁻¹⁷².

When reviewing studies that have investigated the effects of contrast temperature therapy on recovery, it is evident that there are many differences in protocols or exercise conditions used to induce muscle damage and delayed onset muscle soreness. These include high intensity exercise^{49;88;132;151;171}, competitive sport or simulated sports exercise^{60;80;102}, and eccentric exercise protocols^{75;114;170;172}. These exercise protocols and conditions may produce exercise induced muscle damage with different etiologies^{41;42;141}. There is also no evidence as to whether a larger amount of exercise induced muscle damage would make it easier or more difficult to demonstrate a positive effect of an intervention on recovery.

There are also large variations in the method of application of contrast temperature therapy. The temperature of the hot and cold applications are different between studies, as is the length of time for which the hot and cold are applied as well as the number of cycles implemented. Furthermore, the way in which contrast temperature therapy is applied is far from uniform. The depth of immersion varies considerably while sometimes a hot shower is used to apply the hot component of contrast temperature therapy. As the depth of immersion directly affects the amount of hydrostatic pressure applied on the body, and the volume of hydrostatic pressure generated varies greatly between studies, the varied results reported in the studies reviewed may be partially attributed to the differences in contrast temperature therapy application (Table 2.2, page 27).

On comparing the differences in the contrast temperature therapy protocols of the studies that reported benefits on recovery for performance variables¹⁷⁰⁻¹⁷² and those that found no benefits of contrast temperature therapy on recovery^{60;75;151}, it was noted that the depth of immersion for the application of the contrast temperature therapy was greater in the studies that reported a benefit. As a greater depth of immersion results in a higher degree of hydrostatic compression, it would indicate that compression is an important component in enhancing recovery through the application of contrast temperature therapy.

There appears to be some need for standardisation of studies investigating the effects of contrast temperature therapy on recovery from exercise induced muscle damage. This includes the protocols used to induce exercise induced muscle damage, the application of contrast temperature therapy, and the outcome measures used to assess the effect of contrast temperature therapy on recovery. This will allow for direct comparison to be made between studies investigating contrast temperature therapy as well as those investigating the effect of other exercise recovery modalities. There is also a need to understand how changes in the various indirect markers of exercise induced muscle damage may help with recovery.

2.6 INTERMITTENT COMPRESSION

Intermittent compression is a modality that is used to promote tissue healing, reduce oedema and increase blood flow in various medical conditions^{30;58;110;144}. Intermittent compression is most commonly applied through a pneumatically inflatable cuff, which may be inflated to apply an external pressure to the limb. The pressure is intermittently released and then re-inflated to provide a pumping mechanism¹³⁰.

The concept of exerting external pressure on the legs in an attempt to improve circulation has been experimented with since the early 19th century¹¹⁰. Intermittent pneumatic compression is most frequently used in the prevention of deep vein thrombosis, and in the treatment of lymphoedema and venous ulcers^{11;30;59;105;110;130;139}. Relatively few studies have investigated the role of intermittent compression in healing of fractures and soft tissue injuries^{2;28;30;58;144}. In addition, there is currently a lack of evidence regarding the use of intermittent compression as a recovery modality.

However, there is currently no consensus regarding the optimal dosage of intermittent compression¹³⁰. Compression pressure ranges of 30 mmHg to 120 mmHg have been used clinically to treat venous ulcers, deep vein thrombosis, chronic venous insufficiency, and arterial disease¹³⁰ as well as fracture³⁰, soft tissue healing² and exercise induced muscle damage³³. Lymphoedema has been treated with pressures ranging between 35 mmHg and 180 mmHg¹³⁰. In the laboratory pressures ranging from 20 mmHg and 320 mmHg have been tested with positive results on blood flow, lymphatic flow and tissue healing^{30;58;125;126;144}. Treatment durations of between 45 minutes¹⁰⁵ and two hours¹⁰⁵ have been reported.

There is a wide variation in the inflation and deflation cycles that have been used both clinically and in laboratory testing. Morris¹³⁰ reported that the deflation time is generally greater than inflation time and that the deflation time is mostly greater than 40 s. It was also reported that compression for less than 20 seconds is sufficient to empty blood from the compressed veins. However, there is little research comparing the effects of compression intensity, duration and timing on blood flow.

McGeown et al¹²⁶ investigated the effect of intermittent compression on lymphatic flow and reported that there was no difference in flow using inflation times of between one second and 18 seconds and that lymph flow increased as the deflation time increased from 0.5 seconds to eight seconds. Nikolovska et al¹³⁹ compared a “fast” and “slow” intermittent compression protocol for ulcer healing. The “fast” protocol of inflating over 0.5 seconds, holding inflation for six seconds and then deflating for 12 seconds yielded an 86% positive result compared to a 61% positive result achieved by the “slow” protocol of inflating over 60 seconds, holding inflation for 30 seconds and then deflating for 90 seconds.

2.6.1 PHYSIOLOGICAL MECHANISMS OF INTERMITTENT COMPRESSION

The underlying mechanisms of intermittent compression that may facilitate healing remain unclear¹¹⁰. It is hypothesised that compression of a limb may lead to improved venous return and increased venous peak flow velocity⁵⁸. These factors may result in increased shearing stress on endothelium⁵⁹, causing the production of nitric oxide¹¹⁸. Nitric oxide results in vasodilation by inhibiting smooth muscle contractions, which further enhances venous return¹¹⁰. Improved venous return increases the arterio-venous pressure gradient and lowers peripheral resistance⁶², thereby increasing arterial blood flow¹¹⁰. Intermittent compression also results in improved lymphatic drainage and accelerated healing through the removal of oedema from injured soft tissues^{105;125;126;166}.

Challis et al³⁰ demonstrated significant improvements in post-radial fracture grip strength in participants who received four weeks of intermittent pneumatic compression therapy, compared to a control group. Park and Silva¹⁴⁴ determined that increased healing of fractures in a rat model may be related to increased blood flow associated with intermittent pneumatic compression. In addition, Dahl et al⁵⁸ observed that intermittent compression improved tendon healing, with increased fibroblast density and a higher degree of organised parallel collagen fibres, in rats that received intermittent pneumatic compression, compared to a control group. The improved tendon healing was attributed to a 100% increase in the level of sensory neuropeptides in the severed tendons, which may be associated with strong growth promotion. Further, it was theorised that intermittent pneumatic compression may improve neurovascular ingrowth and fibroblast proliferation through the endogenous production of the sensory neuropeptide substance P, thereby accelerating the healing process.

Airaksinen et al² compared the rehabilitation of 44 acute ankle sprains treated either with elastic bandage compression or elastic bandage compression combined with intermittent pneumatic compression. Lower limb dysfunction was assessed through measurements of oedema, ankle range of movement, and limb dysfunction. Measurements were taken on admission to the study, after one week of treatment, and after a four-week follow-up. Elastic bandage compression combined with intermittent pneumatic compression produced significantly faster rehabilitation of oedema, ankle range of movement, and limb dysfunction during the four-week follow-up, compared to elastic bandage compression alone. It was also concluded that intermittent pneumatic compression treatment is effective in acute post-traumatic therapy.

2.6.2 EFFECTS OF INTERMITTENT COMPRESSION ON RECOVERY AND PERFORMANCE

Although there is some evidence that intermittent compression may be beneficial in promoting healing^{2;30;58;110}, there is a lack of evidence regarding the use or effects of intermittent compression as a recovery modality. Chleboun et al³³ investigated the effects of intermittent pneumatic compression on swelling, stiffness and strength loss of the elbow flexors following high velocity muscle lengthening actions of the elbow flexor muscle of female college students. The sample was randomly divided in to an exercise group that performed the exercise protocol, and a non-exercise group that underwent the intervention only without performing the exercise protocol. Muscle circumference, stiffness and strength were measured before the exercise protocol, immediately following the exercise protocol, and then daily for five days both before and after the application of intermittent compression, which also commenced directly after the exercise protocol. Intermittent compression was applied using a single chamber pneumatic sleeve fitted over the entire length of the arm. Pneumatic compression was applied at 60 mmHg for 20 minutes, with 40 second inflation and 20 second deflation cycles.

Muscle circumference and stiffness were increased and strength was decreased for the duration of the five day follow-up period. Decreased muscle circumference and stiffness were recorded immediately following intermittent compression compared to the pre-intervention values for each day, most notably on days two and three post exercise. Strength was not affected following the application of intermittent compression. However over the duration of the study, there were no significant differences in muscle circumference and stiffness between the intermittent compression and control groups. It was concluded that intermittent pneumatic compression may be effective in temporarily reducing muscle swelling and stiffness associated with exercise induced muscle damage. The use of intermittent compression as a recovery modality therefore requires further investigation to understand the effects of the modality on recovery from exercise induced muscle damage, as well as the underlying mechanisms of action of intermittent compression¹³⁰.

2.7 COMBINED APPLICATION OF CONTRAST TEMPERATURE THERAPY AND INTERMITTENT COMPRESSION THERAPY

There is a lack of evidence regarding the use of combined contrast temperature therapy and intermittent compression as a recovery modality. However, there is some evidence for the simultaneous use of cryotherapy with compression as a therapeutic modality. The positive effects of cryotherapy appear to be enhanced when combined with either static compression or intermittent compression^{9;28;57;64;95;96;103;107}. Janwantanakul¹⁰³ investigated the effects of ice packs that had been strapped to the mid-thigh using varying amounts of compression, ranging from 0 mmHg to 44 mmHg, on tissue cooling. There were significant increases in both the rate and magnitude of tissue cooling with cryotherapy and compression, compared to cryotherapy alone. In addition, a higher compressive force during simultaneous compression and cryotherapy was associated with a more rapid reduction in tissue temperature. These results supported the findings of a previous *in vivo* animal study that investigated the cooling properties of ice packs attached to the hind leg of anaesthetised dogs both with and without compression⁹. In this study, tissue hypothermia peaked after 20 minutes of ice pack application and persisted for at least an hour after cryotherapy. Hyperthermia was significantly increased when compression was applied with cryotherapy, compared to cryotherapy alone.

2.8 SUMMARY OF THE LITERATURE

Exercise induced muscle damage is a common occurrence following intense or unaccustomed exercise^{10;32;51}. Exercise induced muscle damage may result in different physiological effects which may negatively influence sporting performance¹⁰. Recovery after exercise is an essential process that minimises the negative effects of exercise induced muscle damage, and enhances the positive effects of training and competition^{129;169}. Many recovery strategies have been developed in an attempt to both accelerate and optimise recovery. There is conflicting evidence regarding the efficacy of these modalities and as yet, no modality has been established to be more effective than another¹⁰.

There is evidence to support the therapeutic effects of both contrast temperature therapy and intermittent compression, although the underlying physiological mechanisms of action for both modalities remain unclear^{2;30;49;58;88;102;110;114;132;170-172}. The use of contrast temperature therapy is also prevalent among the sporting population¹⁰. However, current literature provides equivocal evidence for the use of both contrast temperature therapy and intermittent compression as recovery modalities. The conflicting evidence for contrast temperature therapy and intermittent compression as recovery interventions may be due to methodological differences in the application of interventions, as well as the numerous potential methods of application of contrast temperature therapy and intermittent compression. It is therefore evident that further research is required to understand the effects of contrast temperature therapy and intermittent compression on recovery from exercise induced muscle damage.

The study, to be discussed in Chapter 3, was designed to investigate the effects of the combined application of contrast temperature therapy and intermittent compression on recovery following exercise induced muscle damage. The information gained from this study will assist in establishing the efficacy of the combined application of contrast temperature therapy and intermittent compression as a recovery modality. This has clinical and practical implications for recovery strategies for sports teams, and for both recreational and elite athletes.

CHAPTER THREE

THE EFFECT OF SIMULTANEOUS APPLICATION OF CONTRAST TEMPERATURE THERAPY AND INTERMITTENT COMPRESSION ON RECOVERY FOLLOWING EXERCISE INDUCED MUSCLE DAMAGE

3.1 INTRODUCTION

Intense or unaccustomed exercise leads to exercise induced muscle damage and delayed onset muscle soreness, which may negatively influence exercise performance^{10;32;169;172}. Recovery after exercise induced muscle damage is essentially a passive process. Various recovery modalities have been developed in an attempt to accelerate recovery, and to facilitate a more rapid return to training and competition^{10;32;51;80}. There is equivocal evidence for the effects of contrast temperature therapy on recovery. Some studies have identified positive effects of contrast temperature therapy on muscle soreness, range of motion, muscle force production, and exercise performance^{49;80;88;102;114;132;170-172}. In contrast, other studies have found no change in indirect markers of recovery from exercise induced muscle damage after contrast temperature therapy^{60;75;151}. However, intermittent compression may be associated with temporary reductions in muscle stiffness and swelling following exercise induced muscle damage^{114;171;172}. There is also limited evidence for the effects of combined application of contrast temperature therapy and compression on recovery. Janwantanakul¹⁰³ demonstrated improvements in the rate and magnitude of tissue cooling with cryotherapy and compression, compared to cryotherapy alone. It may therefore be theorised that the combined application of contrast temperature therapy and intermittent compression may enhance physiological adaptations and provide therapeutic effects, which may accelerate recovery after muscle damage. Accordingly, the purpose of this study was to determine the effects of the simultaneous application of contrast temperature therapy and intermittent compression on recovery following exercise induced muscle damage.

3.2 AIM AND OBJECTIVES

3.2.1 AIM

The aim of this study was to investigate the effects of the simultaneous application of contrast temperature therapy and intermittent compression on recovery following exercise induced muscle damage.

3.2.2 SPECIFIC OBJECTIVES

- To determine whether a repeated drop-jump protocol induced muscle damage in an experimental group that received simultaneous contrast temperature therapy and intermittent compression, and a control group that did not receive any treatment after the exercise protocol.
- To determine differences in outcome measures, including muscle soreness, plasma creatine kinase activity, heart rate recovery, quadriceps peak isometric torque, countermovement vertical jump performance, and muscle thickness, between the experimental and control groups during the recovery period after exercise induced muscle damage.
- To determine differences in outcome measures between groups over time during the recovery period after exercise induced muscle damage.

3.3 METHODS

3.3.1 PARTICIPANTS AND STUDY DESIGN

The study had a true experimental design with single blinding. Thirty healthy, active male volunteers were recruited for this study through advertisements placed at local gyms, sports clubs and university campuses, and by word of mouth. Participants were randomly assigned to either an experimental group that received simultaneous contrast temperature therapy and intermittent compression, or a control group that received no treatment.

3.3.1.1 Sample size determination

Data from previous studies measuring heart rate recovery were used to ensure that the sample size would provide sufficient statistical power¹¹⁶. Heart rate recovery was selected to determine the required sample size, as this parameter has the greatest degree of standard deviation of all the parameters measured in this study. Required sample size for heart rate recovery was calculated using a smallest meaningful difference of 9 beats.min⁻¹, and a standard deviation of 6 beats.min⁻¹. With statistical significance accepted as $p < 0.05$, groups of 9, 11, and 13 participants would provide 80%, 90% and 95% statistical power for heart rate recovery respectively. Therefore, 15 participants were randomly allocated to the experimental and control groups respectively to ensure sufficient statistical power should some participants fail to complete the testing procedure.

3.3.1.2 Inclusion criteria

Participants included healthy, active males between the age of 20 and 40 years, who performed between three to five hours of physical activity per week.

3.3.1.3 Exclusion criteria

Participants that reported any relevant medical or surgical history, including a history of lower limb or lumbar spine injury or pathology, or the use of any non-steroidal anti-inflammatory drugs or analgesics were excluded from the study.

Participants who had performed any lower limb weight training in the 12 weeks prior to the study were excluded, due to the protective effects of previous lengthening muscle actions on delayed onset muscle soreness^{19;122;142;156}. Participants were also excluded from the study if they performed any exercise or used any other recovery modalities during the testing period that could have confounded the recovery following exercise induced muscle damage.

Participants who presented with contraindications to contrast temperature therapy or intermittent compression of the lower limb, including open wounds, infections, malignant tumours, vascular diseases, deep vein thrombosis, thrombophlebitis, pulmonary oedema, congestive heart failure, impaired sensation, arteriosclerosis, hypertension, cryoglobinaemia, and Raynaud's disease were also excluded from the study^{63;120;156}.

Females were excluded from this study, as the menstrual cycle has been shown to alter isometric endurance due to a cyclic variation in muscle temperature. The menstrual cycle may also have direct effects on muscle and circulation, which may influence performance measurements¹⁴⁷.

3.3.1.4 Randomisation

The participants were assigned to either the experimental group (n = 15) or the control group (n = 15) stratified according to their body mass. This was done to ensure a higher probability of the experimental and control groups containing participants of similar anthropometric characteristics. Prior to randomisation participants were ranked from heaviest to lightest body mass. The heaviest participant was randomly allocated to the control group by the flipping of a coin. Thereafter, participants were alternately allocated to the experimental group and the control group respectively.

3.3.2 TESTING PROCEDURE

The study was granted ethical clearance by the University of Cape Town Faculty of Health Sciences Human Research Ethics Committee (HREC REF: 198/2009) (Appendix I, page 110). All participants completed an informed consent form before the study (Appendix II, page 112). Participants visited the laboratory for a familiarisation session seven to 10 days before the start of the intervention. Participants completed questionnaires to determine their medical history, current physical activity levels and contraindications to contrast therapy or intermittent compression to the lower limbs (Appendix III, page 118). Body composition measurements were performed and participants were familiarised with all the tests. The familiarisation process was conducted to reduce error associated with participants performing unaccustomed exercise. Standard testing, which included the assessment of muscle soreness, plasma creatine kinase activity, heart rate recovery, quadriceps peak isometric torque, countermovement vertical jump performance, and muscle thickness was performed 24 hours before, and 48 and 96 hours after the exercise protocol designed to induce exercise induced muscle damage. The experimental group had simultaneous contrast temperature therapy and intermittent compression immediately after the exercise and at 24, 48, and 72 hours thereafter. The control group did not receive any treatment after the exercise protocol.

Participants were requested to avoid any medication and strenuous training for the duration of the study. Compliance with study instructions was assessed before each testing or intervention session. Participants were questioned about their compliance with instructions regarding medication use, exercise, and the use of alternative recovery modalities at the start of each session. Test results were recorded on a standardised recording sheet (Appendix IV, page 122). Testing occurred at a similar time (to within one hour) for each participant for the duration of the study. To ensure single blinding, one investigator applied the simultaneous contrast temperature therapy and intermittent compression intervention. A second investigator tested the participants before and after the intervention, and was blinded to the participants' groups. The study design is summarised in Figure 3.1.

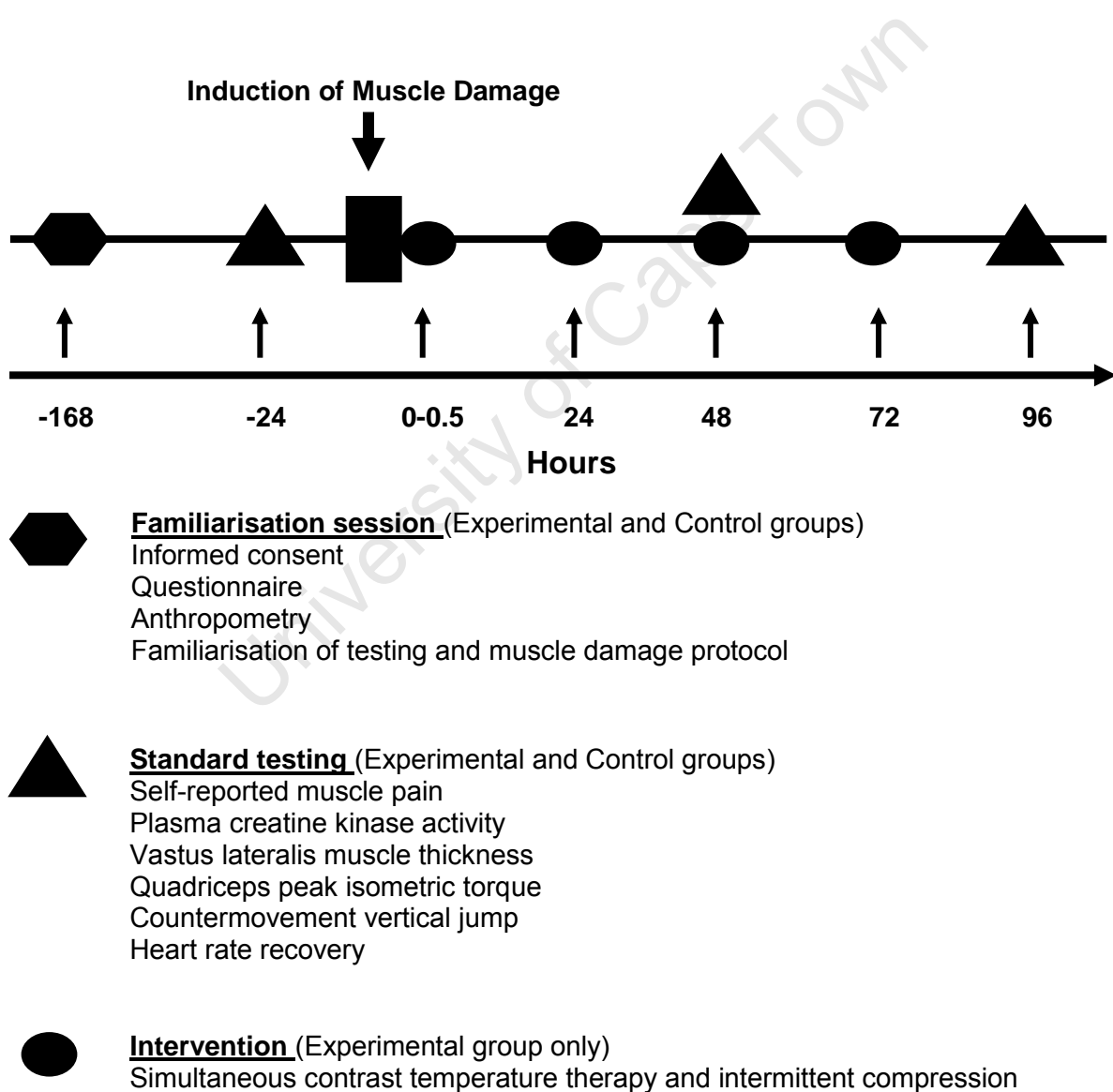


Figure 3.1: Study design.

3.3.2.1 Informed consent and questionnaires

Participants were informed about the purpose of the study, the testing procedures, the possible risks associated with participation in the study, and their right to withdraw from the study at any stage. All participants were required to complete an informed consent form prior to participation in this study (Appendix II, page 112). Participants were also required to complete questionnaires to determine their medical history, current physical activity levels and any contraindications to contrast temperature therapy or intermittent compression to the lower limbs^{63;120;156} (Appendix III, page 118).

3.3.2.2 Anthropometry

Body mass (kg) was recorded using a calibrated scale (Beurer PS07, Beurer GmbH, Ulm, Germany) and stature (cm) was recorded using a stadiometer (Seca 217, Seca Medizinische Waagen und Messsysteme, Hamburg, Germany). Body fat was expressed as the sum of seven skinfolds (mm) (biceps, triceps, subscapular, suprailiac, calf, thigh and abdomen), measured using a skinfold calliper (Harpenden Skinfold Calliper, Quality Measurement Ltd, Burgess Hill, United Kingdom)¹⁵². Body fat was also expressed as a percentage of body mass⁶⁷.

3.3.2.3 Muscle pain

Muscle pain was assessed subjectively using the multidimensional visual analogue scale. Muscle pain was measured 24 hours before, and at 48 and 96 hours after the exercise protocol. Participants rated muscle pain in the quadriceps muscle group according to “*general pain at rest*”, “*pain during activities of daily living*”, “*pain during a passive stretch*”, and “*pain when pressure is applied to the muscle*”. For “*general pain at rest*”, participants were asked to reflect on the pain in their thigh muscles while sitting relaxed on a chair. For “*pain during activities of daily living*”, participants were asked to reflect on the pain in their thigh muscles that they had experienced with normal daily activities. For “*pain during a passive stretch*”, participants were asked to reflect on the pain in their thigh muscles while using their hand to pull their heel towards their buttock in a standing position. For “*pain when pressure is applied to the muscle*”, participants were asked to reflect on the pain in their thigh muscles while they palpated the thigh muscles themselves.

Participants were required to rate their pain in each of the aforementioned categories by making a mark on a 10 cm line, where 0 cm represented “*no pain*” and 10 cm represented “*maximal pain*”. The pain score for each activity was recorded as distance (measured in cm) from the “*no pain*” mark of the pain rating scale to the participants mark. It has been established that the multidimensional visual analogue scale provides a clear description of muscle pain associated with exercise induced muscle damage⁴⁴.

3.3.2.4 Plasma creatine kinase activity

A phlebotomist withdrew a 5 ml blood sample from an antecubital vein 24 hours before, and at 48 and 96 hours after the exercise protocol. Samples were collected in plain standard gel separating vacutainer tubes that did not contain any preservative. Immediately following collection the samples were centrifuged at 3500 rpm at room temperature. The samples were analysed on the automated Beckman Coulter Synchron CX5 Pro chemistry analyser (Beckman Coulter Inc, Brea, CA, USA). The acceptability of the reagent was determined by ensuring that the appropriate quality control material was run facilitating acceptance of the results. Creatine kinase reagent was used to measure creatine kinase activity in the serum samples by an enzymatic rate method. In the reaction creatine kinase catalysed the transfer of a phosphate group from the creatine phosphate substrate to adenosine diphosphate. The subsequent formation of adenosine triphosphate was measured through the use of two coupled reactions catalysed by hexokinase and glucose 6-phosphate dehydrogenase, which resulted in the production of reduced β -nicotinamide adenine dinucleotide from β -nicotinamide adenine dinucleotide. The creatine kinase assay contained the activator monothioglycerol.

The Beckman Coulter Synchron CX5 Pro chemistry analyzer automatically proportioned the appropriate sample and reagent volumes into the cuvette. The ratio used was one part sample to 20 parts reagent. The system monitored the change in absorbance at 340 nm. This change in absorbance is directly proportional to the activity of creatine kinase in the sample, and was used to calculate plasma creatine kinase activity.

3.3.2.5 Vastus Lateralis Muscle Thickness

B-mode ultrasound images of the mid-belly of the vastus lateralis muscle of the dominant leg were obtained using a soft tissue ultrasound machine (SonoSite Micromaxx, SonoSite Inc, Bothell, WA, USA) 24 hours before, and at 48 and 96 hours after the exercise protocol. Participants were positioned in supine with the knees straight and the thigh exposed. The mid-belly of the vastus lateralis was determined as the mid distance between the anterior superior iliac spine and the lateral femoral condyle. The midpoint was marked with a permanent marker to ensure consistency of measurements between tests. Hypoallergenic transmission gel was placed on the transducer head, which had a 5 cm linear array, and an ultrasound image was recorded with the vastus lateralis in a relaxed state. The thickness of the vastus lateralis was assessed on the ultrasound images by measuring the distance between the subcutaneous fat layer and the femur¹⁴¹. The lightest amount of pressure possible was used to apply the transducer head to the skin covered in hypoallergenic transmission gel so as to not compress the muscle tissue⁵⁶. The maximum measurement obtained from three images was recorded. Soft tissue ultrasound has been found to be accurate and reliable method of measuring vastus lateralis muscle thickness^{20;56;141}, and has been effectively used in studies investigating exercise induced muscle damage to calculate muscle volume^{69;148;149}.

3.3.2.6 Heart rate recovery

Heart rate recovery was assessed using the submaximal HIMS test 24 hours before, and 48 and 96 hours after the exercise protocol. In brief, the HIMS test consists of four 2-minute running stages of increasing intensity (8.4, 9.6, 10.8, and 12 km.h⁻¹) interspersed with recovery periods (one minute after each stage). During the rest periods, participants were required to stand upright and motionless with their arms resting by their sides. The pace of each of the four running stages was set by a pre-recorded auditory signal. The test was performed on a rubberised indoor floor between two lines 20 m apart¹¹⁶. Heart rate was recorded throughout the test using a Polar 601 heart rate monitor (Polar Electro, Oy, Finland). Heart rate recovery was defined as the difference between the heart rate at the end of the fourth running stage and 60 s later. Lamberts et al¹¹⁶ established the validity and reliability of the HIMS test in measuring heart rate recovery.

3.3.2.7 Peak isometric quadriceps torque

A Cybex isokinetic dynamometer (CSMI Solutions, Stoughton, USA) was used to evaluate peak isometric quadriceps torque 24 hours before, and 48 and 96 hours after the exercise protocol. The participants were positioned on the Cybex chair with the back of their knees against the front of the seat of the chair. The back rest was set at 95° and adjusted so that it fitted comfortably against the participants' lower back. The five point shoulder harness was fitted firmly to secure the participant in the chair. The thigh of the limb being tested was secured to the chair using a strap passing over the thigh and attaching to the chair on either side of the thigh. The testing lever was attached to the front of the lower leg by a strap passing around the back of the leg. The length of the testing lever was set so that the lower edge of the pad on the front of the leg rested against the top of the foot when the foot was held at 90°. Isometric quadriceps muscle strength testing was performed at 60° of knee flexion. All settings on the seat and testing lever were recorded for each participant so that the exact testing position could be reproduced for each test. Both legs were tested and the dominant leg was tested first.

The participants performed a warm up set of five repetitions of isometric knee extension. Each isometric contraction was sustained for a five-second period. Participants then performed the maximal isometric test, which consisted of five repetitions of maximal isometric knee extension. Each contraction was maintained for a five-second period, and the maximum peak isometric torque ($\text{N}\cdot\text{m}^{-1}$) for the best of the five repetitions was recorded¹⁵⁶. Standard verbal encouragement was provided during the test. The validity and reliability of the measurement of quadriceps peak isometric torque using a Cybex isokinetic dynamometer has previously been established^{98;109;163}.

3.3.2.8 Countermovement vertical jump

Functional muscle power was assessed through a countermovement vertical jump test⁸⁹ 24 hours before, and 48 and 96 hours after the exercise protocol. To record the maximal standing reach, participants were instructed to stand with their dominant side against a wall, and stretch up as far as possible with their dominant hand while keeping the feet flat on the floor. The tips of the fingers were coated in chalk. The chalk marks left by the fingers were used as indicators of the height reached on the maximal standing reach and vertical jump height. The countermovement vertical jump was performed in the same position as for the standing reach. Participants were instructed to bend their knees to 90° and swing their arms backwards. Without pausing at the bottom of the bend, they were instructed to swing their arms forwards and jump as high as possible while reaching up with their dominant hand and making a mark with their chalked fingers as high up the wall as possible. An investigator monitored the jumps to ensure participants performed each jump correctly. Participants were given standard verbal encouragement during the test. Participants were required to perform the countermovement vertical jump three times and the maximum jump height from the best of the three jumps was recorded. The jump height was calculated as the difference between the maximal standing reach height and the maximal countermovement jump height. The jump height represented the functional capacity of the quadriceps and other leg extensor muscles to generate power¹⁴⁵. The validity and reliability of the countermovement vertical jump in determining functional muscle performance has previously been established^{89;90;145}.

3.3.3 EXERCISE PROTOCOL INDUCING MUSCLE DAMAGE

A repeated drop-jump protocol was used to induce muscle damage in the quadriceps muscle group. Prior to the exercise protocol, participants were required to stand facing a wall with both arms extended above the head. A mark was made 20 cm above each participant's fingertips. A bench, measuring 50 cm in height, was placed 1.5 m away from the wall. Participants then performed a weighted warm-up, according to the protocol described by Burkett et al²². To perform the drop-jump, participants were instructed to jump off the bench and land with the legs slightly apart. On landing, participants then bent both knees, touched the ground with the fingertips, and immediately jumped vertically to touch the mark on the wall with both hands.

The drop-jump protocol was monitored to ensure participants completed each jump correctly. The participants were required to perform seven sets of 10 repetitions of this exercise. Each set was be separated by a one-minute rest period. Participants were given standard verbal encouragement during the test. The validity and reliability of this stretch shortening cycle exercise protocol in the development of delayed onset muscle soreness has previously been established¹⁵⁷.

3.3.4 CONTRAST TEMPERATURE THERAPY WITH INTERMITTENT COMPRESSION INTERVENTION

Simultaneous contrast temperature therapy and intermittent compression (Zamar Healthcare Shock device, ZAMAR S.r.l., Suzzara, Italy) was applied to the quadriceps muscle of the experimental group at 0, 24, 48 and 72 hours after the drop-jump protocol. A compression sleeve was firmly applied to the upper leg over the quadriceps muscles of both lower limbs. The Zamar device was set with the lower temperature at 10 °C and the upper temperature at 40 °C. As the Zamar device uses a closed system to circulate the fluid through the heating and cooling mechanisms, it was not possible to confirm the accuracy of the pre-set temperatures. The manufacturer's specifications state that the digital thermometer that is installed in the device has been calibrated at the time of manufacture and has a tolerance of 0.5 °C. The contrast temperature therapy protocol consisted of three cycles of cold therapy followed by hot therapy, with a one-minute pause at the hot and cold extremes. The Zamar device automatically adjusted the temperature of the liquid flowing through the sleeves. It took approximately five minutes to adjust the temperature from 10 °C to 40 °C, and approximately 10 minutes to adjust the temperature from 40 °C to 10 °C. The delayed temperature change was a limitation of the Zamar device. Furthermore, the Zamar uses a heat exchange mechanism to heat or cool the fluid. This means that variables such as the temperature of the limb and ambient temperature affect the time taken for the Zamar to reach its target temperature. Therefore, the total duration of the intervention was approximately 50 minutes. Previous studies have demonstrated positive effects of contrast temperature therapy using similar applications of hot and cold temperatures⁹⁴.

During the application of contrast temperature therapy, the Zamar device cyclically increased the compressive pressure of the sleeve by 10 mmHg. The pressure was increased for a period of seven seconds, and was then returned to the baseline level for three seconds. This intermittent compression was continued for the full duration of the intervention. In addition, firm application of the compression sleeves gave a baseline compressive force of between 40 mmHg and 50 mmHg. The additional cyclical pressure increase within the compression sleeves therefore resulted in a maximum compressive force of 50-60 mmHg. These pressures have been previously determined to be safe and effective for the application of intermittent compression⁶³. The compressive forces generated by the sleeve were measured by placing a sphygmomanometer cuff, inflated to 10 mmHg, under the sleeve before it was firmly applied and operated. The rise in pressure measured by the sphygmomanometer cuff represented the forces generated by the application of the sleeve and the intermittent compression generated by the Zamar device.

3.3.5 STATISTICAL ANALYSES

Statistical analyses were performed using Statistica software (StatSoft, Inc. (2007). STATISTICA (data analysis software system), version 8.0. www.statsoft.com). The Levene's test was used to determine whether the sample variances were similar. Differences in descriptive variables between the experimental and control groups were assessed using an independent t-test. Statistical significance for the two main effects of group and time, and the interaction (group x time) of all other variables were assessed using a two-way analysis of variance (ANOVA) with repeated measures. Unequal N HSD *post hoc* comparisons were performed where necessary. A Mann-Whitney U test was used to assess differences in the pain scores between groups. A Friedman's ANOVA and Dunn's Multiple Comparison test were used to assess differences in the pain scores within groups over time. All data are presented as the mean \pm standard deviation (SD). Statistical significance was accepted as $p < 0.05$.

3.3.6 ETHICAL CONSIDERATIONS

The study was performed in accordance with the principles of the Declaration of Helsinki (Seoul version, 2008). The study was submitted and approved by the Faculty of Health Sciences Human Research Ethics Committee, University of Cape Town (HREC REF: 198/2009) (Appendix I, page 110). All participants were required to complete an informed consent form prior to completing the questionnaire (Appendix II, page 112). The informed consent form explained the purpose and procedure of the study, how confidentiality would be ensured, and the right to withdraw from the study without reason or prejudice. All data were kept confidential and anonymous.

3.3.6.1 Risks to participants

3.3.6.1.1 Anthropometry and muscle pain

There were no potential risks to the participants that may be associated with mass, stature, skinfold and muscle pain measurements.

3.3.6.1.2 HIMS test, isometric quadriceps strength test, countermovement vertical jump test and drop jump exercise protocol

The isokinetic isometric strength test and countermovement vertical jump test involve maximal contractions of primarily the quadriceps muscle group. The risk associated with these tests is equivalent to the risk associated with normal gym training, primarily muscle injury. The drop jump exercise protocol designed to create exercise induced muscle damage involves strong eccentric contractions of primarily the quadriceps muscle group, while the HIMS test is a submaximal shuttle running test. The risks related to these tests are also primarily muscle injury and the risk associated with unaccustomed exercise, such as painful and stiff quadriceps muscles. The risks to the participants in this study were minimised through thorough familiarisation with all the equipment and testing procedures. Participants were also required to perform a standardised warm up before the tests to reduce the risk of any musculoskeletal injury that may be associated with maximal or submaximal contractions of muscles. In addition, all the tests were carefully controlled, thereby reducing improper use of the equipment and minimising the risk of injury to the participants. Furthermore, participants with previous or current musculoskeletal injuries were excluded from this study, therefore reducing the risk of injury associated with the testing procedures.

3.3.6.1.3 Blood samples

Blood samples were drawn for the analysis of plasma creatine kinase activity. When working with blood, potential risks include infection with blood borne diseases including hepatitis and human immunodeficiency virus. To minimise these potential risks, a trained phlebotomist performed the procedures. Furthermore, sterile equipment was used for these procedures, and good clinical practice was strictly adhered to throughout testing.

3.3.6.1.4 Contrast temperature therapy with intermittent compression

Contrast temperature therapy involves both the heating and cooling of neuromuscular structures. Due to the heating and cooling effects and the change in pressure applied, the contraindications include open wounds, infections, malignant tumours, vascular diseases, deep vein thrombosis, thrombophlebitis, pulmonary oedema, congestive heart failure, impaired sensation, arteriosclerosis, hypertension, Raynauds disease and cryoglobinaemia^{63;120}. To reduce these risks, these conditions were part of the exclusion criteria. Participants were screened for these conditions during the completion of the informed consent forms and questionnaires. In addition, the temperatures used for the contrast temperature therapy were from 10 °C to 40 °C, and there was little risk of ice burn or heat burn to the skin.

Compression at pressures higher than diastolic blood pressure for prolonged periods of time can become uncomfortable. Pressures beyond this point may also occlude circulation. Pressures generated by the Zamar Healthcare Shock device did not exceed 80 mmHg. The pressure was intermittently increased and decreased and did not remain above 60 mmHg for more than ten seconds before being lowered. This is consistent with the recommendations for safe intermittent compression application⁶³. Furthermore, participants undergoing the intervention were continuously monitored for distal lower limb signs of significantly decreased circulation and unexpected discomfort. This further lowered any risks still presenting. Prior to the intervention, the Zamar Healthcare Shock unit was tested for accurate calibration of both the sleeve pressures and temperatures.

3.3.6.2 Benefits to participants

Participants received a full summary of their individual results, as well as the overall findings from this study. The individual results included information regarding body composition measurements, countermovement vertical jump heights, quadriceps peak concentric force production, and heart rate recovery data (Appendix V, page 126).

3.4 RESULTS

3.4.1 PARTICIPANTS

The descriptive characteristics of participants are shown in Table 3.1. At the start of the study 30 participants were recruited and matched pairs were randomly assigned to the experimental and control groups. One participant from the control group was excluded as he could not complete the baseline HIMS test due to a sudden onset of lower back pain, reducing the control group to 14 participants. In the experimental group, three participants were excluded. One experienced anterior knee pain and could not complete the isokinetic knee test, the second did not attend all the intervention sessions, and the third did not adhere to the exercise guidelines for the duration of the study period. The sample size chosen was calculated to have a statistical power of 95% for heart rate recovery, which was the variable with the greatest expected degree of standard deviation. The reduced sample size reduced the statistical power to 90% for heart rate recovery. There was a significant difference between groups in age, with participants in the control group being older than participants in the experimental group ($p = 0.02$). There were no significant differences between groups for any other descriptive variables.

Table 3.1: Descriptive characteristics of participants in the experimental ($n = 12$) and control groups ($n = 14$). Data are expressed as mean \pm standard deviation

VARIABLE	EXPERIMENTAL	CONTROL
Age (years)	23.2 \pm 3.5	28.4 \pm 6.3*
Body mass (kg)	86.5 \pm 17.4	87.8 \pm 18.2
Stature (cm)	182.3 \pm 6.0	180.5 \pm 8.7
Body mass index	26.0 \pm 5.2	26.9 \pm 5.2
Sum of skinfolds (mm)	121.0 \pm 55.3	108.4 \pm 43.8
Body fat (%)	20.4 \pm 6.1	19.2 \pm 5.0
Lean body mass (kg)	68.0 \pm 9.5	70.3 \pm 11.1

* $p = 0.02$

3.4.2 MUSCLE PAIN

Participants in both the experimental and control groups experienced muscle pain 48 hours after the exercise protocol (Figure 3.2). There were no significant differences in pain scores between groups specifically, at 48 hours there were significant increases in “perceived pain at rest” [experimental group ($p = 0.005$); control group ($p = 0.001$)], “pain during activities of daily living” [experimental group and control group ($p = 0.001$)], “pain on passive stretch” [control group ($p = 0.003$)], and “pain on palpation” [experimental group ($p = 0.005$); control group ($p = 0.001$)], compared to baseline values. At 96 hours pain levels in both the experimental and control groups had decreased and were not significantly different from baseline levels.

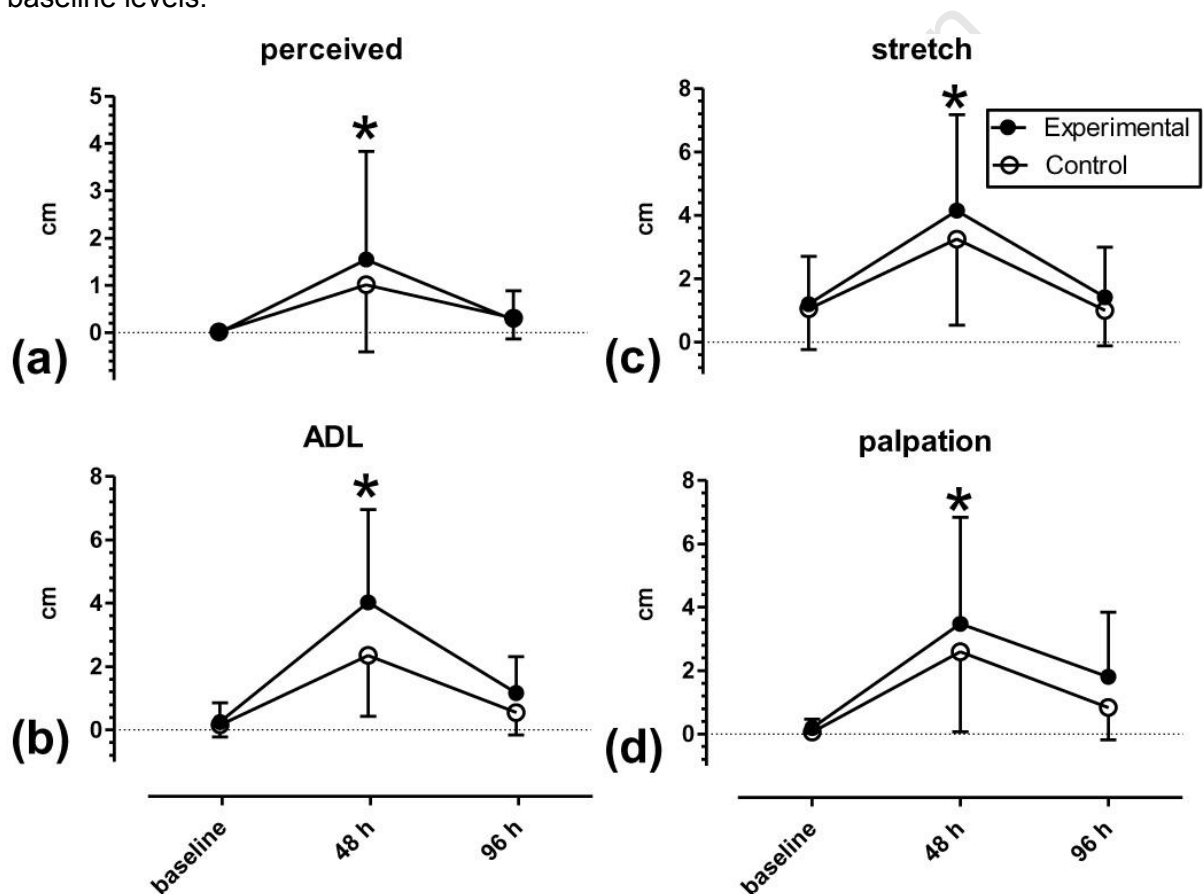


Figure 3.2: Subjective (a) pain at rest, (b) pain during activities of daily living, (c) pain on quadriceps muscle stretch, and (d) pain on pressure to the quadriceps muscle (cm) of participants in the experimental (-●-) ($n = 12$) and control (-○-) ($n = 14$) groups. Tests were conducted 24 hours before (baseline), and at 48 and 96 hours after exercise induced muscle damage. Data are expressed as mean \pm SD.

Significant Differences:

- a) * 48 hours vs. baseline ($p = 0.005$) in experimental group; ($P = 0.001$) in control group
- b) * 48 hours vs. baseline ($p = 0.001$) in experimental group; ($p = 0.001$) in control group
- c) * 48 hours vs. baseline ($p = 0.003$) in control group
- d) * 48 hours vs. baseline ($p = 0.005$) in experimental group; ($p = 0.001$) in control group

3.4.3 PLASMA CREATINE KINASE ACTIVITY

There was a significant difference in plasma creatine kinase activity over time in both groups ($F_{(2, 48)} = 1.14$; $p = 0.01$) (Figure 3.3). Plasma creatine kinase activity increased in both groups at 48 hours, compared to baseline values ($p = 0.009$). There were no significant differences between groups.

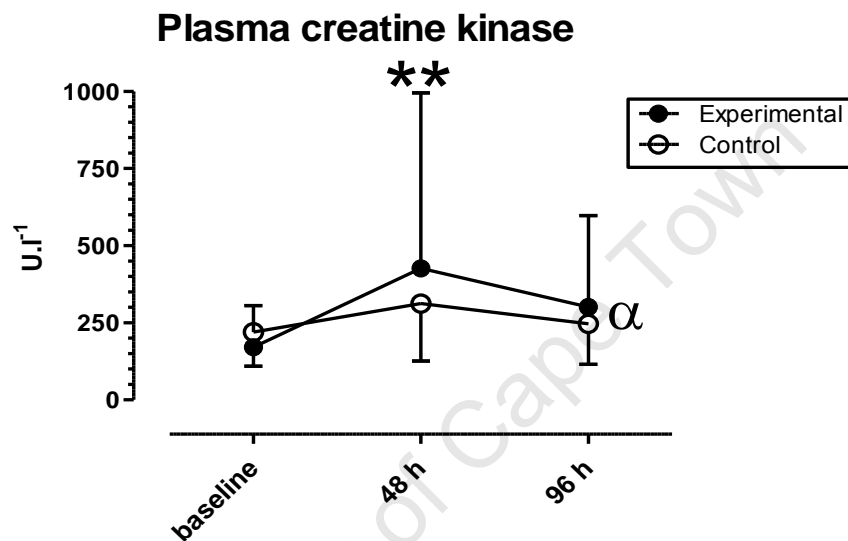


Figure 3.3: Plasma creatine kinase activity (U.l⁻¹) of participants in the experimental (-●-) ($n = 12$) and control (-○-) ($n = 14$) groups. Tests were conducted 24 hours before (baseline), and at 48 and 96 hours after exercise induced muscle damage. Data are expressed as mean \pm SD.

Significant Differences:

- α main effect of time ($p = 0.01$)
- ** 48 hours vs. baseline ($p = 0.009$)

3.4.4 VASTUS LATERALIS MUSCLE THICKNESS

The differences in vastus lateralis muscle thickness between the experimental and control groups are shown in Figure 3.4. There were no significant differences between groups, however there was a significant difference in the measurement over time ($F_{(2, 48)} = 1.18$; $p = 0.001$). Vastus lateralis muscle thickness was significantly increased in both groups at 48 hours compared to baseline values ($p = 0.003$), and at 96 hours compared to baseline levels ($p = 0.003$).



Figure 3.4: Muscle thickness (cm) of participants in the experimental (-●-) ($n = 12$) and control (-○-) ($n = 14$) groups. Tests were conducted 24 hours before (baseline), and at 48 and 96 hours after exercise induced muscle damage. Data are expressed as mean \pm SD.

Significant Differences:

- α main effect of time ($p = 0.001$)
- ** 48 hours vs. baseline ($p = 0.003$)
- ** 96 hours vs. baseline ($p = 0.003$)

3.4.5 HEART RATE RECOVERY

There were no significant differences in heart rate recovery either between groups or over time (Figure 3.5).

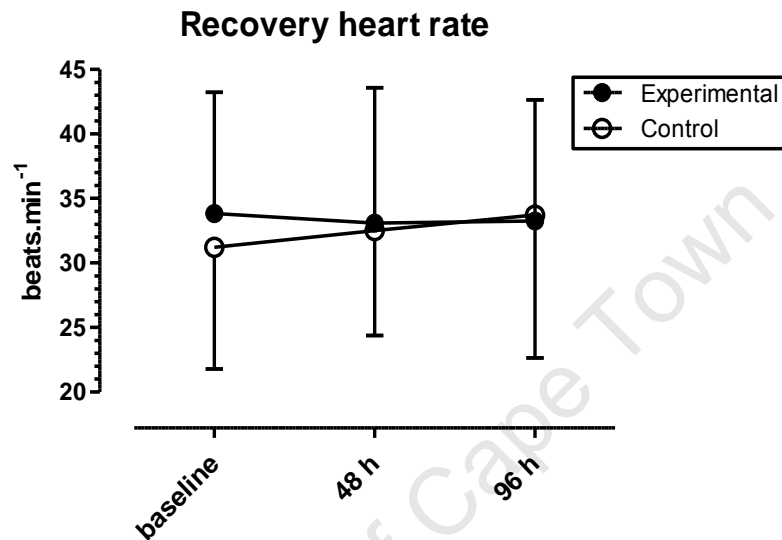


Figure 3.5: Recovery heart rate (beats.min⁻¹) of participants in the experimental (-●-) (n = 12) and control (-○-) (n = 14) groups. Tests were conducted 24 hours before (baseline), and at 48 and 96 hours after exercise induced muscle damage. Data are expressed as mean ± SD.

3.4.6 COUNTERMOVEMENT VERTICAL JUMP HEIGHT

There were no significant differences in vertical jump performance between groups, however there was a significant difference in measurement over time ($F_{(2, 48)} = 1.46$; $p = 0.019$). Vertical jump height was significantly decreased in both groups at 48 hours, compared to baseline values ($p = 0.032$) (Figure 3.6).

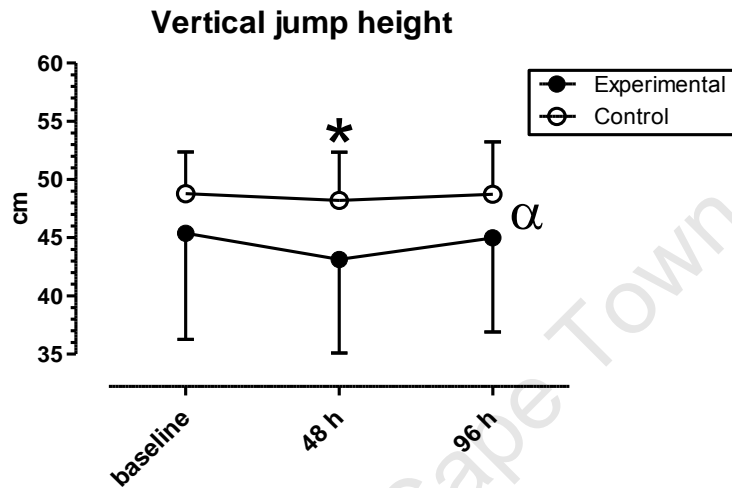


Figure 3.6: Vertical jump height (cm) of participants in the experimental (-●-) ($n = 12$) and control (-○-) ($n = 14$) groups. Tests were conducted 24 hours before (baseline), and at 48 and 96 hours after exercise induced muscle damage. Data are expressed as mean \pm SD.

Significant Differences:

- α main effect over time ($p = 0.019$)
- * 48 hours vs. baseline ($p = 0.032$)

3.4.7 QUADRICEPS PEAK ISOMETRIC TORQUE

3.4.7.1 Dominant leg

There were no significant differences in quadriceps peak isometric torque for the dominant leg between groups, however there was a significant difference in measurement over time ($F_{(2, 48)} = 0.45$; $p = 0.003$) (Figure 3.7a, page 75). Peak isometric torque was significantly increased at 96 hours, compared to values at 48 hours ($p = 0.003$).

3.4.7.2 Non-dominant leg

There were also no significant differences in quadriceps peak isometric torque for the non-dominant leg between groups, however there was again a significant difference in measurement over time ($F_{(2, 48)} = 0.66$; $p = 0.001$) (Figure 3.7b, page 75). Peak isometric torque was significantly increased at 96 hours, compared to values at baseline ($p = 0.038$), and 48 hours ($p = 0.001$).

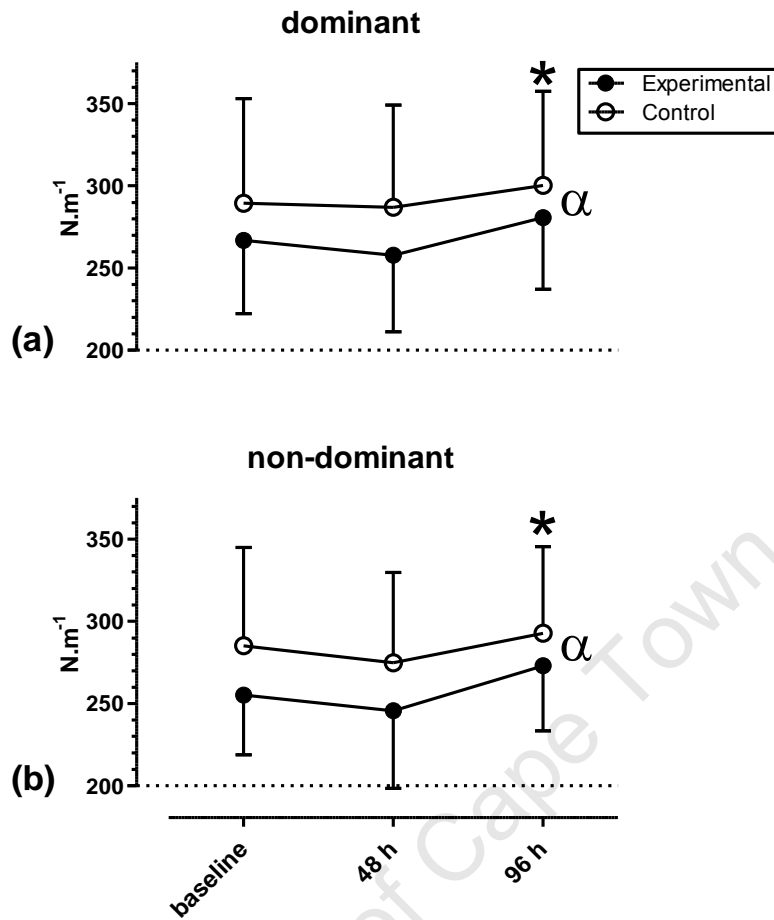


Figure 3.7: Quadriceps peak isometric torque ($N.m^{-1}$) of participants (a) dominant leg and (b) non-dominant leg in the experimental (\bullet) ($n = 12$) and control (\circ) ($n = 14$) groups. Tests were conducted 24 hours before (baseline), and at 48 and 96 hours after exercise induced muscle damage. Data are expressed as mean \pm SD.

Significant Differences:

Dominant leg (a)

α main effect over time ($p = 0.003$)

* 96 hours vs. 48 hours ($p = 0.003$)

Non-dominant leg (b)

α main effect over time ($p = 0.001$)

* 96 hours vs. baseline ($p = 0.038$) and 96 hours vs 48 hours ($p = 0.001$)

3.5 DISCUSSION

This was the first study to examine the effect of the simultaneous application of contrast temperature therapy and intermittent compression on exercise induced muscle damage. The development of a research intervention is challenging, as the precise pathology of exercise induced muscle damage is unclear. The use of indirect markers of muscle damage including muscle soreness and plasma creatine kinase activity are used to quantify the amount and extent of muscle damage.⁸⁶ The drop jump protocol used in this study was effective in inducing muscle damage, as it resulted in both muscle soreness (Figure 3.2, page 69) and increased plasma creatine kinase activity (Figure 3.3, page 70). This is consistent with previous research that has used the similar protocols for the induction of exercise induced muscle damage^{19;157;170;172}.

3.5.1 PARTICIPANTS

There was a significant difference in age between participants in the experimental group and the control group (Table 3.1, page 68). Muscle compliance, or passive muscle stiffness, has been identified as an important moderating factor in muscle damage¹²⁷. It was observed that a more compliant muscle experiences less exercise induced muscle damage than stiffer muscles. Flexibility, which is highly correlated with muscle compliance⁸², has been shown to decrease with increasing age⁷⁰. This decrease in flexibility has not been identified in males between the ages of 20 and 40 years of age. It may be proposed that that the significant difference in age between groups in this study would be unlikely to influence the extent of muscle damage and delayed onset muscle soreness, as the age range of participants in both groups was relatively small. However, this study did not measure muscle flexibility, and further studies are required to confirm this hypothesis.

3.5.2 MUSCLE PAIN

This study showed significant increases in all categories of pain scores in both the experimental and control groups at 48 hours after exercise induced muscle damage, compared to baseline values, with the exception of pain on quadriceps stretch. However, there were no significant differences in pain scores between groups (Figure 3.2, page 69). A significant difference over time for pain on stretch was found at 48 hours in the control group, but not in the experimental group. As the difference was only over time and not between groups, and this only occurred in one of the four measures of the multi-dimensional scale used to assess perceived pain, this finding is unlikely to be of any significant clinical relevance. For all measures, the muscle pain peaked at the 48 hours testing and had returned to baseline level by the 96 hours measurement. The time course for muscle pain measured in this study is consistent with studies investigating delayed onset muscle soreness following muscle damaging protocols^{19;32;40;137;141}.

There is equivocal evidence for the effects of contrast temperature therapy on muscle pain following exercise induced muscle damage with pain improving^{102;114;172} or remaining unchanged with contrast temperature therapy^{60;75;151;170}. However, the studies that reported no significant differences in pain scores after contrast temperature therapy all produced lower levels of hydrostatic compression, compared to the studies that demonstrated significant reductions in pain^{102;172}. Dawson et al⁶⁰ and Robey et al¹⁵¹ used cold water immersion to the waist level, but applied heat through the use of a hot shower. The use of a hot shower rather than hot water immersion would result in no hydrostatic force being applied for the hot component of contrast temperature therapy. In addition, French et al⁷⁵ used contrast water immersion in 50 cm of water with legs outstretched. In contrast, Ingram et al¹⁰² used immersion to the umbilicus for the hot and cold component, while Vaile et al¹⁷² used contrast water immersion to neck level. As a greater depth of water immersion results in a higher level of hydrostatic compression, the application of contrast water immersion to these levels would result in much higher levels of hydrostatic compression, compared to lower levels of immersion¹⁷⁶.

Further, Vaile et al¹⁷⁰ used a contrast water immersion protocol with immersion to the level of the anterior superior iliac spine in recreational athletes, but were unable to demonstrate a reduction in pain compared to the control group, whereas Ingram et al¹⁰², evaluated athletes with team games experience and found a positive effect of contrast temperature therapy on pain using immersion to the level of the umbilicus. It is unclear whether athletes with different exercise profiles may have different responses to recovery interventions following exercise induced muscle damage, and this theory requires further investigation³⁸.

The third study to report a positive effect of contrast temperature therapy on recovery used an immersion protocol for the arm to the level of the mid deltoid muscle, following lengthening contractions of the elbow flexor muscles. Although this level of immersion produced less hydrostatic compression than some of the other studies that did not find a positive effect of contrast temperature therapy on muscle pain, this was the only study to make use of a whirlpool in which to apply the contrast temperature therapy. It is unclear as to whether the agitation of the water in the whirlpool may have contributed to the positive finding in this study. Moreover, although delayed onset muscle soreness is the most frequently reported symptom of exercise induced muscle damage⁷⁰, it may not necessarily reflect the magnitude of muscle damage²⁵, or long-term changes in neuromuscular function which occur after exercise induced muscle damage. For example, neuromuscular function is disturbed for approximately 11 days after an ultramarathon³¹. In addition, signs of regeneration have been observed in the muscle of runners 12 weeks after a marathon, despite the absence of pain¹⁷³. However, this study found that all physical, biochemical, and physiological outcome measures had returned to baseline values by 96 hours after the damaging bout of exercise.

3.5.3 PLASMA CREATINE KINASE ACTIVITY

In this study, plasma creatine kinase activity was significantly elevated in both groups at 48 hours after muscle damage, with no significant difference in plasma creatine kinase activity between groups (Figure 3.3, page 70). The increase and time course of changes in plasma creatine kinase activity is similar to that observed in other studies, which have used similar protocols to induce exercise induced muscle damage^{38;169}. Exercise protocols that induce muscle damage through repetitive stretch shortening cycles have reported plasma creatine kinase activity increases of between 100 and 600 U.l⁻¹ peaking at 12 to 24 hours post exercise^{26;155}. However, there appears to be large inter-individual variability in plasma creatine kinase activity in response to muscle damaging exercise^{40;100}. The findings of this study with regards to the response of plasma creatine kinase activity are also consistent with previous studies, where plasma creatine kinase activity was not altered compared to control conditions following contrast temperature therapy interventions^{75;102;151;170;172}. These studies used methods of inducing exercise induced muscle damage consisting of either repetitive stretch shortening cycles or high force lengthening muscle contractions. Although these two methods of exercise induced muscle damage induction result in differing plasma creatine kinase responses, neither method was able to show an effect on plasma creatine kinase activity following contrast temperature therapy.

In contrast, Gill et al⁸⁰ reported decreased transdermal exudate creatine kinase activity at 36 and 48 hours following a rugby match when using contrast temperature therapy, compared to no treatment. Rugby is a sport where significant muscle damage is experienced both as a result of physical contact between players, and due to exercise induced muscle damage after intense exercise. These combined mechanisms may lead to higher levels of plasma creatine kinase activity. Indeed, both the baseline and post-exercise creatine kinase activity levels were three to five times higher after rugby, compared to standard and non-contact exercise protocols that induce exercise induced muscle damage. Further, the rate of appearance and clearance of creatine kinase activity in the transdermal exudate may be different to that of blood plasma and therefore incomparable to that of plasma creatine kinase activity. This finding may indicate that contrast temperature therapy might be a useful intervention following activities which involve both physical contact and exercise induced muscle damage inducing exercise. Further research is needed to confirm this observation.

In this study, there were also wide intra-individual variations in plasma creatine kinase activity. Variations in plasma creatine kinase activity during the study may also be related to differences in the rate of creatine kinase clearance by muscle and the reticuloendothelial system. This has been shown to be independent of physical activity, gender or mass³⁸. Unfortunately the design of this study cannot provide an explanation for the large degree of intra-individual variation in creatine kinase activity. Further studies are needed to investigate this finding.

3.5.4 VASTUS LATERALIS MUSCLE THICKNESS

There was a significant increase in vastus lateralis mid-belly thickness in both groups at 48 and 96 hours after exercise induced muscle damage, compared to baseline values. There were no differences in muscle thickness between groups (Figure 3.4, page 71). The change in muscle thickness reflects increased muscle volume, which may be associated with intramuscular oedema¹⁷⁰. As previously discussed (Section 2.2.2, page 7), mechanical strain of the muscle may be associated with the disruption of contractile components of the muscle and the muscle membrane. The subsequent inflammatory response and a movement of leukotrienes, neutrophils and fluid from the blood into interstitial spaces results in intramuscular oedema^{41;51}.

Only three studies have investigated the effects of contrast temperature therapy on swelling or limb volume after exercise induced muscle damage. Vaile et al¹⁷² and Vaile et al¹⁷⁰ showed significant reduction of thigh volume following contrast temperature therapy while French et al⁷⁵ failed to show any effect compared to no treatment. The mechanism through which contrast temperature therapy is most likely to have an effect on oedema is due to hydrostatic compression by increasing venous and lymphatic drainage¹⁷². The reduced amount of hydrostatic compression used by French et al⁷⁵ in comparison to Vaile et al¹⁷² and Vaile et al¹⁷⁰ has been discussed (Section 2.5.1, page 28). A lower hydrostatic pressure may account for the differing results between these studies with regards to thigh muscle girth and volume. Unfortunately, this study did not assess changes in muscle thickness after 96 hours. The time course of recovery of muscle thickness to baseline levels was not clearly established. This finding also provides evidence to support tissue changes in response to exercise induced muscle damage in the absence of delayed onset muscle soreness^{32;51;54;170}.

Further, Chelboun et al³³ used a compressive pressure of 60 mmHg to apply intermittent compression after lengthening actions of the elbow flexor muscles to induce exercise induced muscle damage. Limb circumference was measured in an experimental group that received intermittent compression and a control group before and after intermittent compression. Chelboun et al³³ reported a significant reduction in limb circumference after each application of intermittent compression compared to the measurement before the intervention. The control group that did not undergo the exercise protocol to induce exercise induced muscle damage showed no change in upper limb circumference before and after intermittent compression. This shows that even though intermittent compression was able to reduce arm muscle volume in the intervention group after the intermittent compression compared to before, there was still a gradual increase in arm circumference until the fourth day following induction of exercise induced muscle damage. This is consistent with the findings of this study for both the intervention and control group. The relevance of the ability of intermittent compression to reduce oedema related to exercise induced muscle damage in the short term needs to be investigated further with regards to recovery.

3.5.5 HEART RATE RECOVERY

In this study, there were no differences in heart rate recovery between the groups or over time (Figure 3.5, page 72). Previous studies have reported a decrease in heart rate recovery associated with fatigue^{14;15}. Heart rate recovery is a measure of the autonomic nervous system's ability to cope with stress. With exercise, the sympathetic nervous system is activated, causing an increase in heart rate. At the conclusion of the exercise, the parasympathetic nervous system becomes more active to allow for recovery, while the sympathetic nervous system withdraws¹⁵. When the body is under stress, there is a delayed re-activation of the parasympathetic nervous system. This can be measured as a reduced heart rate recovery.

Coffey et al⁴⁹ and Vaile et al¹⁷¹ were unable to establish any positive effects of contrast temperature therapy on heart rate recovery. However, Coffey et al⁴⁹ investigated contrast temperature therapy as a recovery modality for repeated running performance. Vaile et al¹⁷¹ reported on next day performance following strenuous training using a fatigue inducing protocol. The amount of exercise induced muscle damage induced using these protocols may be less than that induced using lengthening muscle contractions and as such, the results may not be directly comparable with the findings in this study.

3.5.6 COUNTERMOVEMENT VERTICAL JUMP HEIGHT

In this study, there were no significant differences in vertical jump performance between groups. However, there was a significant reduction in vertical jump height at 48 hours after exercise induced muscle damage, compared to baseline values (Figure 3.6, page 73). This is consistent with research indicating that exercise induced muscle damage leads to a decrease in performance requiring explosive muscle power^{24;157}. It has been suggested that this decrease in power is due to pain inhibition as a result of delayed onset muscle soreness or decreased muscle pre-activation as a result of exercise induced muscle damage^{32;35}.

Dawson et al⁶⁰, French et al⁷⁵ and Vaile et al¹⁷⁰ were unable to observe any benefit of contrast temperature therapy for explosive muscle power activities, while Vaile et al¹⁷² showed a positive effect for contrast temperature therapy over the control. Once again, studies that used contrast temperature therapy protocols that produced lower levels of hydrostatic compression did not find positive effects^{60;75}. In addition, Vaile et al¹⁷² used strength trained athletes while Vaile et al¹⁷⁰ used recreational athletes. This may have resulted in a lower level of exercise induced muscle damage in the strength trained athletes compared to the recreational athletes as the prior strength training may have protection against the negative effects of exercise induced muscle damage through the repeated bout effect, thereby reducing the negative effect on explosive power production.

3.5.7 QUADRICEPS PEAK ISOMETRIC TORQUE

There were also no significant differences in peak isometric torque between the experimental and control groups. However, peak isometric torque was significantly increased at 96 hours after exercise induced muscle damage, compared to 48 hours and baseline values (Figure 3.7, page 75). Previous studies have recommended maximal voluntary torque as one of the preferable methods of measuring the effects of exercise induced muscle damage^{131;174}. In addition, most studies have reported a decrease in peak force production by between 10 and 30% as a result of exercise induced muscle damage³⁸. Although care was taken to familiarise the volunteers to the equipment as well as methods and techniques to be used in the standardised testing, the significant increase in peak isometric torque recorded at 96 hours as well as the lack of a significant decrease at 48 hours noted in both the experimental group and control group indicates the possibility of a learning or training effect associated with isometric isokinetic testing^{131;163}.

In this study, participants attended a single familiarisation session for orientation with the equipment and testing procedure utilities for the maximal isometric contraction test. Participants were also able to practice the testing procedure to become accustomed to the concept of achieving and maintaining voluntary isometric force. However, this may not have been sufficient for the participants to achieve maximal isometric force with full quadriceps activation. Morton et al¹³¹ employed multiple familiarisation sessions to obtain a maximal isometric contraction. During these familiarisation sessions, percentage of voluntary muscle activation was determined using a twitch interpolation technique. Participants returned for further familiarisation sessions until a voluntary muscle activation of more than 95% was achieved. It was established that participants required between two and six sessions to achieve 95% of voluntary muscle activation¹³¹. It was reported that any initial “learning” or “training” effect was alleviated during repeated familiarisation sessions. It is therefore evident that the single familiarisation session employed in this study may not have been adequate to eliminate a learning effect. Future studies may benefit from the use of an objective measure to ensure adequate familiarisation with maximal isometric testing procedures.

Previous studies investigating contrast temperature therapy as a recovery modality for exercise induced muscle damage have reported no effect^{75;102;114;151} or positive effects^{170;172} on reduction of the magnitude of loss of peak force production or a quicker return to pre-exercise induced muscle damage peak force, compared to no recovery. The protocols of three of the studies that did not show a quicker recovery of reduced peak force production following contrast temperature therapy have been previously discussed (Section 2.5.2.12, page 42) and the lower hydrostatic compression forces generated in these studies have been highlighted^{75;102;151}. This may be a factor in not achieving a quicker return to pre-exercise induced muscle damage peak force in those studies compared to the studies that showed an enhanced recovery effect^{170;172}.

Kuligowski et al¹¹⁴ did not report a reduced loss of peak force production or a quicker return to pre-exercise induced muscle damage peak force even though the hydrostatic compression force generated was similar to those who did report positive effects on recovery. However, the study of Kuligowski et al¹¹⁴ was the only study to utilise a high force eccentric exercise protocol to induce muscle damage. The studies that reported enhanced effects on peak force recovery utilised repeated stretch shortening cycle exercise to induce exercise induced muscle damage. High force eccentric exercise protocols have been associated with higher degrees of peak force loss and prolonged recovery times, as well as higher levels of plasma creatine kinase activity³⁸.

3.5.8 LIMITATIONS OF THE STUDY

The main limitation of this study was the method of application of simultaneous contrast temperature therapy and intermittent compression. The treatment was applied through a sleeve that was fitted to the participant's thigh, and the thighs were treated in isolation. This may limit the effectiveness of both the contrast temperature therapy and intermittent compression, as the lower legs and feet were not treated with the thighs. The majority of contrast temperature therapy studies have used contrast baths, where participants are submerged at least to the waist level⁹⁴. The intermittent compression sleeves also usually cover the whole limb, including the foot¹³⁰. In addition, in this study there was a relatively long transition period between the hot and cold cycles. It took approximately five minutes to change from cold to hot temperature therapy. Fiscus et al⁷³ found significant changes in arterial blood flow with contrast therapy where there is an instantaneous change from cold to hot temperatures. It is unclear whether a potential change in circulation would be as marked in this study, due to the time taken for the change of temperature to occur. However, it is only recently that technological advances have allowed for the simultaneous application of contrast temperature therapy and intermittent compression. Improvements to the technology such as the use of a full leg length compression sleeve and the ability to achieve immediate transitions between temperatures would justify further research into this modality as a recovery strategy.

A potential limitation of this study was that the effectiveness of the recovery intervention was only assessed during the acute recovery period, where participants still experience pain and elevated plasma creatine kinase activity. In addition, current laboratory studies generally induce moderate to severe muscle damage in specific muscle groups to monitor the recovery process in isolation and to determine the efficacy of recovery interventions. However, in practice recovery is an ongoing process that occurs simultaneously during training and competition. This may influence both the recovery process and the effect of recovery modalities. Fatigue may also alter the response to recovery modalities. Therefore field studies that examine the effects of repeated bouts of exercise and competition on recovery should be conducted to determine the practical efficacy of different recovery interventions.

A further limitation of this study is that the control group was untreated which means that the placebo effect was not controlled for. The placebo effect in randomised trials has been defined as the difference between placebo and no treatment¹⁰⁰. Hróbjartsson and Gøtzsche¹⁰¹ reported that placebo interventions did not have important clinical effects in general, although meta-regression analyses did show larger placebo effects associated with some study designs including those with patient-reported outcomes and observer-reported outcomes involving patient cooperation. Some aspects of this study did incorporate these components. This means that the findings of the experimental group may have been influenced by a placebo effect. As there were no significant differences between groups in this study, the placebo effect, if present, may not have had a significant influence on the experimental group findings. However, this is purely speculative and further studies need to be conducted with a placebo-treated control group to confirm this theory.

This study was single blinded. As such, the control group was aware that they were part of the control group and were not receiving the intervention. This may have served to demoralise the control group as they were not receiving the intervention, or conversely, may have provided additional motivation for the control group to make up for not receiving the intervention. These phenomena may have affected the results for perceived pain, isokinetic strength, and countermovement vertical jump height, but would have had no effect on vastus lateralis muscle thickness, heart rate recovery, and creatine kinase activity. The effect of this motivation or demotivation is therefore a potential limitation of this study.

Finally, the intra-rater reliability of the investigator was not established for ultrasound measurement of the vastus lateralis muscle. The investigator did however receive extensive training from an experienced ultrasonographer. To further reduce the possibility of error, the largest of the three measurements obtained on each occasion was used. This is because the possibility exists that the pressure of the transducer head on the limb may compress the muscle and result in the measurement being smaller than it really is. As the results for vastus lateralis muscle thickness followed the same pattern as all the other variables for both the experimental and control groups, it is unlikely that the ultrasound measurements obtained were unreliable.

3.6 CONCLUSION

In conclusion, the simultaneous application of contrast temperature therapy and intermittent compression used in this study was ineffective in enhancing recovery following exercise induced muscle damage. This is based on the finding that there were no differences in muscle pain, plasma creatine kinase activity, vastus lateralis muscle thickness, heart rate recovery, countermovement vertical jump height, and quadriceps peak isometric torque between the group which received treatment of simultaneous contrast temperature therapy and intermittent compression, and the control group.

It is recommended that future studies should investigate the long-term effects of recovery interventions to accurately demonstrate potential positive or negative effects on repeated measures of exercise performance. Further, it is recognised that although current evidence-based parameters for the contrast temperature therapy and intermittent compression applications were used in this study, much research is still required to determine the optimal application of both contrast temperature therapy and intermittent compression. It may also be proposed that future studies should recruit participants from a specific sporting code to limit potential variability in exercise performance between participants⁹⁹.

CHAPTER FOUR

SUMMARY AND CONCLUSION

Exercise induced muscle damage occurs following unaccustomed or intense exercise and in particular, exercise that involves predominantly lengthening muscle actions⁷⁰. The negative effects of exercise induced muscle damage on athletic performance have been well documented^{10;32;80;169;172}. As a result, active recovery modalities are becoming increasingly popular within sport as athletes attempt to find a competitive edge over their opponents. The ability to enhance the passive recovery process may not only result in a quicker return to baseline levels of function following competition, but may also enable the athlete to cope with a higher training load thereby generating a higher training effect and increasing performance over time¹⁰.

Research into the effects of various commonly utilised recovery modalities is ongoing and results are unclear^{10;80}. In addition, technology continues to develop new ideas and methods of recovery such as the technology to simultaneously apply contrast temperature therapy and intermittent compression. This recovery strategy seeks to provide the claimed benefits of contrasting temperature with the mechanical pumping effect of intermittent compression. Literature investigating the effects of contrast temperature therapy on recovery are largely equivocal⁹⁴, while there is a lack of scientific research into the effects of intermittent compression on recovery³³.

Therefore, the overall aim of this study was to investigate the effects of the simultaneous application of contrast temperature therapy with intermittent compression as a recovery strategy following exercise induced muscle damage in moderately active adult males. Based on the evidence provided in this thesis, the study objectives, as described in Section 3.2.2 (page 51), may be answered as follows:

To determine whether a repeated drop-jump exercise protocol induced muscle damage in an experimental group, who received treatment consisting of contrast temperature therapy with simultaneous intermittent compression, and a control group, who did not receive any treatment after the exercise protocol.

In this study, both the experimental group and control group showed significant changes in the majority of the indirect markers of exercise induced muscle damage at 48 hours, and in some cases at 96 hours following the repeated drop jump protocol. Self-reported muscle pain, plasma creatine kinase activity, vastus lateralis muscle thickness, and countermovement vertical jump height all revealed changes that are consistent with exercise induced muscle damage thereby supporting the presence of muscle damage. The resolution of these changes over the duration of the study period is also consistent with the time frames expected following exercise induced muscle damage.

To determine differences in outcome measures, including muscle soreness, heart rate recovery, muscle power, plasma creatine kinase activity, and muscle thickness, between the experimental group and control group.

None of the outcome measures revealed differences between the experimental group and control group throughout the study period. This indicates that the application of contrast temperature therapy with simultaneous intermittent compression, or the method by which the intervention was applied, provided no benefits for recovery over passive recovery.

To determine differences in outcome measures between groups over time during the acute recovery period after exercise induced muscle damage.

With the exception of heart rate recovery, an effect over time was found within both the experimental group and control group for all other indirect markers of exercise induced muscle damage, but not between groups. An implication of this finding is that the method of simultaneous application of contrast temperature therapy and intermittent compression applied in this study neither reduces the level of exercise induced muscle damage nor aids in recovery following exercise induced muscle damage compared to passive recovery.

Based on the findings of this study, modifications to the method of application of simultaneous contrast temperature therapy and intermittent compression have been recommended to further evaluate the effects of this combined modality as a recovery intervention. Recommendations include the ability to achieve an immediate transition from hot to cold and *vice versa* to optimise the effects of the contrast temperature therapy component, and the use of a full leg length compressive sleeve rather than just a thigh sleeve in order to enhance the application of the intermittent compression component. In addition, further research is necessary to identify the optimal parameters regarding temperature and compression pressures for the combined application of these modalities. The findings of the clinical application of the simultaneous contrast temperature therapy with intermittent compression as conducted in this study do not support the practical use of this combination therapy as a recovery modality.

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APPENDIX I

UNIVERSITY OF CAPE TOWN



**Health Sciences Faculty
Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6626 • Facsimile [021] 406 6411
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07 October 2009

REC REF: 198/2009

Mr W Holroyd
Health & Rehab

Dear Mr Holroyd

PROJECT TITLE: THE EFFECT OF SIMULTANEOUS CONTRAST TEMPERATURE THERAPY AND VARIABLE INTERMITTENT COMPRESSION ON RECOVERY FOLLOWING EXERCISE-INDUCED MUSCLE DAMAGE.

Thank you for addressing the issues raised by the Research Ethics Committee.

It is a pleasure to inform you that the Ethics Committee has **formally approved** the above-mentioned study.

Approval is granted for one year till the 15th October 2010.

Please add the UCT REC Contact details, Prof M Blockman 021 4066338 to the Informed Consent Form.

Please submit an annual progress report if the research continues beyond the expiry date. Please submit a brief summary of findings if you complete the study within the approval period so that we can close our file.

Please note that the ongoing ethical conduct of the study remains the responsibility of the principal investigator.

Please quote the REC. REF in all your correspondence.

Yours sincerely

signature removed

PROFESSOR M BLOCKMAN
CHAIRPERSON, HSF HUMAN ETHICS

Federal Wide Assurance Number: FWA00001637.

S Thomas

Institutional Review Board (IRB) number: IRB00001938

This serves to confirm that the University of Cape Town Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical Research Council (MRC-SA), Food and Drug Administration (FDA-USA), International Convention on Harmonisation Good Clinical Practice (ICH GCP) and Declaration of Helsinki guidelines.

The Research Ethics Committee granting this approval is in compliance with the ICH Harmonised Tripartite Guidelines E6: Note for Guidance on Good Clinical Practice (CPMP/ICH/135/95) and FDA Code Federal Regulation Part 50, 56 and 312.

APPENDIX II

INFORMED CONSENT FORM

Dear Participant

The MRC/UCT Research Unit for Exercise Science and Sports Medicine will be conducting a study to investigate the effect of the simultaneous application of contrast temperature therapy and intermittent compression on the recovery from exercise induced muscle damage. Both contrast therapy (the alternating application of hot and cold treatments) and intermittent compression (compressive pressure that rhythmically builds up on the limb and then releases) are widely used by athletes and sports teams to assist in recovering from exercise. This study aims to validate the use of these two techniques simultaneously. The research project adheres to the Helsinki Declaration of 2008 and has been approved by the UCT Research Ethics Committee (REC REF: 198/2009).

The research project will involve you attending a maximum of seven appointments of about an hour long, over a two week period. The appointments will be at the Centre for Sports Medicine Umhlanga. The following procedures and tests will be conducted at each appointment:

First appointment

- Complete a questionnaire regarding medical history and physical activity levels.
- Body mass and stature measurements.
- Measurement of body composition involving skinfold thicknesses using skinfold callipers to determine fat content.
- Familiarisation with the testing that will be conducted during the study.

Second Appointment – 6 days following first appointment

- The following standardised testing will be conducted to obtain a baseline measurement
 - Blood samples will be taken from a vein on the front of the elbow on three occasions during the study to measure plasma creatine kinase activity (an indicator of muscle damage). Good clinical practice and sterile procedures will be strictly adhered to.
 - Soft tissue ultrasound imaging will be used to measure the thickness of the vastus lateralis (outer thigh muscle).
 - The rating of thigh muscle pain while doing 4 simple activities.
 - The strength of the muscles on the front of the thigh will be determined using an isokinetic machine. This machine measures the amount of force the thigh muscle can produce to straighten the knee.
 - A countermovement vertical jump will be used as a functional measure of thigh muscle power. This involves measuring the height of the jump.
 - 1 minute heart rate recovery (using a heart rate monitor) will be measured following a 13 minute submaximal shuttle run test (HIMS).

Third Appointment – the day after the second appointment

- You will be required to perform an exercise that involves jumping down from a 50cm high box to the ground and then jumping up again to touch a pre-marked area on the wall. This exercise is designed to create soreness in the muscles as a result of the intensity and strength required to perform the exercise. This will result in a feeling of achiness and stiffness of the thigh muscles.
- You will then be randomly allocated to two groups. The one group will then receive the intervention while the second will not.
 - The intervention consists of 50 minutes of contrast temperature therapy with intermittent compression. This involves having a sleeve fitted over both the thigh muscles. The sleeve will be intermittently cooled to 10°C and heated to 40°C. The sleeve will provide rhythmic compression to the thigh.
 - Only the intervention group will be required to attend the sessions were the intervention is applied.

Fourth Appointment – the day after the third appointment

- Only the intervention group will be required to attend this appointment.
- The intervention will be applied as described above.

Fifth Appointment – the day after the fourth appointment

- Both groups are required to attend this appointment
- Standardised testing as described on the second appointment
- The intervention group will undergo the intervention as described for the third appointment

Sixth Appointment – the day after the fifth appointment

- Only the intervention group will be required to attend this appointment.
- The intervention will be applied as described for the third appointment

Seventh Appointment – the day after the sixth appointment

- Standardised testing as described on the second appointment

TIME COMMITMENTS

The following table summarises the time commitments required to participate in this study. The total time commitment necessary for this study over the two week period is approximately 6 hours.

Table: Time commitments

Appointment	Testing procedure	Time needed
1	Fill in questionnaire Body composition assessment Familiarisation of testing techniques	45 min
2	Standard exercise testing	45 min
3	Jumping exercise protocol Hot / Cold treatment for intervention group only	20 min 50 min
4	Hot / Cold treatment for intervention group only	50 min
5	Standard exercise testing Hot / Cold treatment for intervention group only	45 min 50 min
6	Hot / Cold treatment for intervention group only	50 min
7	Standard exercise testing	45 min

Both the intervention group and the control group will be required to undergo the standard testing and the exercise routine to create muscle soreness. In addition, the intervention group will be required to attend all the intervention sessions where the hot/cold compression treatment is applied. The control group is not required to attend the intervention sessions.

POSSIBLE RISKS

There are no potential risks that may be associated with mass, stature, skinfold measurement, and muscle pain measurements. The vertical jump and isokinetic strength tests involve maximal contractions of muscle groups. A jumping exercise routine is also utilised to induce muscle damage, resulting in painful and stiff thigh muscles. The risk related to with all these tests is equivalent to the risk associated with normal gym training, primarily muscle injury. The risk will be minimised through thorough familiarisation with all equipment, and the implementation of a controlled warm up before these exercises and testing procedures. Any injury that occurs during testing will receive insurance cover in the form of the UCT public liability policy.

Blood samples will be drawn for the analysis of plasma creatine kinase activity and blood glucose oxidation. As always when working with blood, potential risks include infection with blood borne diseases including hepatitis and HIV. In order to minimize these potential risks, a trained phlebotomist will perform the procedures. Furthermore, sterile equipment will always be used for these procedures, and good clinical practice will be strictly adhered to.

Contrast temperature therapy involves the heating and cooling of the body's tissues. Due to the heating and cooling effects and the change in pressure applied, there may be some complications in people suffering from infections, open wounds, circulatory disorders, cardio-respiratory conditions or impaired sensation. To minimise these risks, if you suffer from any of these types of conditions, you will be excluded from this study.

ANTICIPATED BENEFITS

You will receive a full summary of your individual results, as well as the overall findings from this study. The individual results will include information regarding body composition measurements, peak quadriceps strength during the isometric isokinetic test, height obtained in the countermovement vertical jump test, and heart rate recovery data following the HIMS test.

PRIVACY AND CONFIDENTIALITY

All records and results generated within this study will be stored in a computer database in a secure facility, and in a manner that maintains confidentiality. All participants will remain anonymous in any ensuing publication.

CONTACT INFORMATION

Investigator Name	Telephone	Email
Wayne Holroyd	(031) 560 5557	wayne@centreforsportsmedicine.co.za
Theresa Burgess	(021) 406 6171	theresa.burgess@uct.ac.za

I confirm that the exact procedures and possible complications of the above tests have been explained to me. I understand that I may ask questions at any time during the testing procedures. I realise that I am free to withdraw from the study without prejudice at any time, should I choose to do so. I have been informed that the personal information required by the researchers will be held in strict confidentiality. In addition, I know that the information derived from the testing procedures will remain confidential and will be revealed only as a number in statistical analyses.

I have carefully read this form. I understand the nature, purpose and procedure of this study. I agree to participate in this research project of the MRC/UCT Research Unit for Exercise Science and Sports Medicine.

Full name of participant: _____

Signature of participant: _____

Full name of witness: _____

Signature of witness: _____

Date: _____

APPENDIX III

QUESTIONNAIRE

Name: _____

Date of Birth: _____

Age: _____

Cell no.: _____

Email: _____

Medical and Surgical History (last 2 years): _____

Present/previous injuries to lower limbs, pelvis or spine: _____

Current medication: _____

Are you currently receiving any massage, soft tissue or physiotherapy treatment?

Yes No

Please give details of treatment: _____

Do you have a history of lower limb muscle pathology or injury, or neurological disease involving the lower limbs?

Yes No

Have you ever been diagnosed with any of these disorders?

- | | | | |
|------------------------|--------------------------|--------------------------|--------------------------|
| Coronary Heart Disease | <input type="checkbox"/> | Asthma | <input type="checkbox"/> |
| Diabetes | <input type="checkbox"/> | Rheumatoid Arthritis | <input type="checkbox"/> |
| Thyroid Disease | <input type="checkbox"/> | Renal Disease | <input type="checkbox"/> |
| Thrombophlebitis | <input type="checkbox"/> | High Blood Pressure | <input type="checkbox"/> |
| Tuberculosis | <input type="checkbox"/> | Osteoporosis | <input type="checkbox"/> |
| Osteoarthritis | <input type="checkbox"/> | Cancer | <input type="checkbox"/> |
| High Cholesterol | <input type="checkbox"/> | Stroke | <input type="checkbox"/> |
| Vascular diseases | <input type="checkbox"/> | Impaired sensation | <input type="checkbox"/> |
| Arteriosclerosis | <input type="checkbox"/> | Raynaud's disease | <input type="checkbox"/> |
| Cryoglobinaemia | <input type="checkbox"/> | Deep vein thrombosis | <input type="checkbox"/> |
| Pulmonary oedema | <input type="checkbox"/> | Congestive heart failure | <input type="checkbox"/> |
| Hypertension | <input type="checkbox"/> | | |

Have you had any of the following?

Cardiac Surgery Yes No

Spinal Surgery: Yes No

Fractures or broken bones: Yes No

Please specify where: _____

Have you been ill in the past 3 weeks?

Yes No

Please specify details of you're the illness: _____

Have you taken any medication in the past 3 months?

Yes No

Please specify details of the medication: _____

Is there any medication that you regularly take to manage pain/injuries e.g.: paracetamol, anti-inflammatories?

Yes No

Please specify details: _____

Do you exercise between 3 and 5 hours a week?

Yes No

Have you done any weight training for your legs over the past 3 months?

Yes No

Are you able to attend the testing sessions at the Centre for Sports Medicine Umhlanga as described to you in the document of informed consent?

Yes No

Signature: _____

Date: _____

Thank you for your co-operation in completing this questionnaire^{63;120;156}.

APPENDIX IV

DATA COLLECTION FORM

Name: _____

Date: _____

ANTHROPOMETRY

Body mass _____

Stature _____

BMI _____

Dominant leg _____

SKINFOLD MEASUREMENT (mm)

Triceps _____

Biceps _____

Sub-scapular _____

Supra-iliac _____

Thigh _____

Calf _____

Abdominal _____

FAMILIARISATION CHECK SHEET

Name: _____

Date: _____

Warm-up procedure

Delayed onset muscle soreness induction protocol

Blood test

Ultrasound measurement

Countermovement vertical jump test

Isometric knee extension test

HIMS test

Muscle pain scoring

Instruction on permitted exercise during study

Instruction on medication that is not permitted as part of the study

Instruction on recovery modalities that are not permitted during study

STANDARDISED TESTING RECORDING FORM

Name: _____

Date: _____

Adherence to exercise restriction

Adherence to medication restriction

Adherence to recovery modality restriction

Perceived Muscle Pain (completed by participant)

- General pain at rest

- Pain during activities of daily living

- Pain during a passive stretch

- Pain when pressure is applied to the muscle

Ultrasound Vastus Lateralis Measurement

1. _____

2. _____

3. _____

Countermovement Vertical Jump

Standing reach height: _____

Countermovement vertical jump height

1. _____ 2. _____ 3. _____

Isometric quadriceps peak torque test

Peak isometric torque dominant leg

Peak isometric torque non-dominant leg

HIMS Test completed

HIMS Test heart rate file saved to computer

Creatine kinase activity blood test completed

University of Cape Town

APPENDIX V

PARTICIPANT FEEDBACK FORM

Dear _____

Thank you for participating in this study. Your assistance is greatly appreciated. Following is a report on your results from the testing that was performed, together with a summary of the findings of the study.

ANTHROPOMETRY (BODY MEASUREMENTS)

VARIABLE	YOUR MEASUREMENT	STUDY AVERAGE
Age (years)		
Body mass (kg)		
Stature (cm)		
Body mass index (BMI)		
Sum of skinfolds (mm)		
Body fat (%)		
Lean body mass (kg)		

You were randomly allocated to the **EXPERIMENTAL / CONTROL** group

TESTING RESULTS

General pain at rest

	BASELINE	48 HOURS	96 HOURS
Your score			
Experimental group average			
Control group average			

Pain during activities of daily living

	BASELINE	48 HOURS	96 HOURS
Your score			
Experimental group average			
Control group average			

Pain during a passive stretch

	BASELINE	48 HOURS	96 HOURS
Your score			
Experimental group average			
Control group average			

Pain when pressure is applied to the muscle

	BASELINE	48 HOURS	96 HOURS
Your score			
Experimental group average			
Control group average			

Plasma creatine kinase activity ($\text{U}\cdot\ell^{-1}$) (blood marker of muscle damage)

	BASELINE	48 HOURS	96 HOURS
Your measurement			
Experimental group average			
Control group average			

Vastus Lateralis muscle thickness (cm) (outer thigh)

	BASELINE	48 HOURS	96 HOURS
Your measurement			
Experimental group average			
Control group average			

Countermovement vertical jump height (cm)

	BASELINE	48 HOURS	96 HOURS
Your measurement			
Experimental group average			
Control group average			

Isometric quadriceps peak torque – Dominant leg ($\text{N}\cdot\text{m}^{-1}$) (thigh muscle strength)

	BASELINE	48 HOURS	96 HOURS
Your measurement			
Experimental group average			
Control group average			

Isometric quadriceps peak torque – Non-dominant leg (N.m⁻¹)

	BASELINE	48 HOURS	96 HOURS
Your measurement			
Experimental group average			
Control group average			

Heart rate recovery (beats.min⁻¹)

	BASELINE	48 HOURS	96 HOURS
Your measurement			
Experimental group average			
Control group average			

RESULTS

The exercise protocol used in the study resulted in all subjects in both groups developing symptoms of exercise induced muscle damage. No differences were found for any of the variables between the group which received treatment of simultaneous contrast temperature therapy and intermittent compression, and the control group. This means that the treatment was ineffective at reducing the amount of exercise induced muscle damage or enhancing recovery from exercise induced muscle damage compared to passive recovery.

PRACTICAL RECOMMENDATIONS

The use of simultaneous contrast temperature therapy and intermittent compression is not clinically indicated to assist in recovery from exercise induced muscle damage. Although there is some scientific evidence to support the use of these modalities individually to assist in promoting recovery from exercise, the results of this study show no benefit over passive recovery for their simultaneous use.