http://researchcommons.waikato.ac.nz/

Research Commons at the University of Waikato

Copyright Statement:

The digital copy of this thesis is protected by the Copyright Act 1994 (New Zealand).

The thesis may be consulted by you, provided you comply with the provisions of the Act and the following conditions of use:

- Any use you make of these documents or images must be for research or private study purposes only, and you may not make them available to any other person.
- Authors control the copyright of their thesis. You will recognise the author’s right to be identified as the author of the thesis, and due acknowledgement will be made to the author where appropriate.
- You will obtain the author’s permission before publishing any material from the thesis.
The use of intermittent sequential pneumatic compression for recovery following exercise

A thesis submitted in partial fulfillment of the requirements for the degree of

Master of Health, Sport and Human Performance

at

The University of Waikato

by

Adam Cranston

2019
Abstract

The modern athletic training schedule requires athletes to regularly complete intense training sessions that induce muscular damage. Recovery strategies are thought to reduce post-exercise fatigue and enhance subsequent performance, leading to the investigation and development of many different recovery strategies and protocols. Intermittent sequential pneumatic compression (ISPC) is a relatively new recovery strategy that has been proposed to enhance recovery post-exercise in the sport setting. However, the limited amount of current research on ISPC has returned contrasting findings, requiring further research to determine its efficacy for exercise recovery. Furthermore, technological advances in this area have seen the introduction of new devices, including the yet to be evaluated ISPC arm-sleeves. The first section of this thesis briefly examines the literature on commonly used recovery methods. It then provides an overview of the possible mechanisms for which ISPC is proposed to enhance recovery; compression, massage, temperature, and potentially via the placebo effect. Finally, it reviews the existing literature on ISPC in the sport and exercise-recovery setting. Part two of the thesis includes an original investigation whereby the efficacy of an ISPC device on the upper-body following a fatiguing exercise circuit was examined. Fifty resistance-trained athletes (37 male/13 female, mean ± SD; age = 27 ± 24 yrs) performed three physical performance tests (grip strength dynamometer, single arm medicine ball throw, preacher bench bicep curls to failure) pre and post exercise, and following a 30-minute recovery period. During the recovery period, participants were randomly assigned an experimental arm, which was placed in the ISPC device, and a control arm (no device). Participants completed a perceptual muscle soreness rating of four upper-body landmarks at the same time points and also 24 hours post-recovery. There was a statistically significant interaction between conditions for the
single arm medicine ball throw (p < 0.01) following the recovery period, however the effect size was deemed trivial. There was a small, non-significant effect (d = 0.22) for the bicep curls in favour of ISPC. The perceptual muscle soreness scales resulted in significant differences between conditions immediately post and 24 hours post across all muscle groups (p < 0.05), in favour of the ISPC condition. This study supports the use of an upper-body ISPC device to reduce muscle soreness for up to 24 hours following exercise, and may provide small benefits to strength-endurance when compared to a control trial. In conclusion, this thesis adds to the body of knowledge on ISPC as a recovery strategy in athletes. While the experimental study adds to the existing body of research already completed on ISPC, it is similar to previous literature outlined in the review, with conflicting results. Further research is required to investigate the efficacy of ISPC when compared to a sham condition.
# Table of Contents

Abstract...........................................................................................................................................ii  
Table of contents............................................................................................................................iv  
List of Tables........................................................................................................................................vi  
List of Figures........................................................................................................................................vii  
Abbreviations.......................................................................................................................................viii  
Acknowledgements............................................................................................................................ix  
Thesis Overview.................................................................................................................................x  
Chapter One – Literature review ......................................................................................................1  
  Introduction........................................................................................................................................2  
  Common recovery strategies.............................................................................................................8  
  Sleep..................................................................................................................................................8  
  Cold water immersion......................................................................................................................9  
  Hot water immersion......................................................................................................................12  
  Contrast water therapy....................................................................................................................13  
  Active recovery................................................................................................................................15  
  Whole body cryotherapy chamber..................................................................................................17  
  Compression Garments....................................................................................................................19  
  Nutrition and hydration....................................................................................................................20  
  Stretching........................................................................................................................................21  
Intermittent Sequential Pneumatic Compression................................................................................23  
  Proposed mechanisms for ISPC enhancing exercise recovery.........................................................23  
  ISPC compression............................................................................................................................23  
  Massage..........................................................................................................................................24  
  Temperature....................................................................................................................................26
List of Tables

Table 1 – The effect of ISPC on measures of performance
Table 2 – The effect of ISPC on perceptual measures
Table 3 – The effect of ISPC on physiological measures
Table 4 - Comparison of ISPC and CON conditions at all time points
Table 5 - Comparison of all measures (post-recovery and 24h post recovery) compared to post-exercise (pre-recovery) values
List of Figures

Figure 1 – Experimental testing protocol

Figure 2 – Anatomical landmarks for muscle palpation

Figure 3 – ISPC arm sleeve device and control arm setup for the recovery period
Abbreviations

ATP – Adenosine triphosphate
ADP – Adenosine diphosphate
CG – Compression garments
CK – Creatine kinase
CMJ – Counter-movement jump
CWI – Cold-water immersion
CWT – Contrast water therapy
DOMS – Delayed onset muscle soreness
GCS – Graduated compression stockings
HWI – Hot water immersion
ISPC – Intermittent sequential pneumatic compression
MD - Mean absolute difference
OCC – Vascular occlusion
SMD – Standardised mean difference
PCr – Phosphocreatine
Acknowledgements

First I would like to thank my parents for the continued support they have shown me over the years. Without this support the opportunity to further my education and complete this thesis would not have been possible.

Particular thanks must be given to my thesis supervisor Dr. Mathew Driller. The support and patience you have shown during this process has been unwavering. Without your guidance none of this would have been possible.

To my brothers, Tamanui, Pera, Jamison, and Stefan, you have each inspired and encouraged me in your own ways and without this I would not have embarked on this journey, thank you.

Next I would like to thank the management and staff of YMCA Gisborne and Anytime Fitness Gisborne. Thank you for letting me test participants in your facilities and have unrestricted access to all the equipment that was required. Also thank you to the staff in those facilities that personally went out of their way to find extra participants for the study contained in this thesis.

I would also like to thank Sport Gisborne for emailing out participant information sheets to various sport and fitness organizations in order to recruit participants.

Finally I would like to thank all those who took time out of their busy schedules to act as participants. Without you there would be no data and no study for this thesis to be based on.
Thesis Overview

The current thesis includes a chapter presented in the style of an individual journal article in its submitted format, therefore some information may be repeated. The thesis contains three chapters. Chapter One includes a review of the literature and introduces the reader to intermittent sequential pneumatic compression and its potential physiological mechanisms. Chapter Two contains an original investigation on the use of an intermittent pneumatic compression arm sleeve for recovery following exercise. Chapter Three summarizes the overall findings and provides both practical applications and recommendations for future research.
Chapter One:

The use of intermittent sequential pneumatic compression for recovery following exercise:

A literature review
Introduction

The high training loads required to excel in most modern athletic sports has meant that the role of accelerating recovery has become an important part of the training schedule (Argus, Driller, Ebert, Martin, & Halson, 2013; Coffey, Leveritt, & Gill, 2004; Halson, 2013; O'Donnell & Driller, 2015). Enhancing recovery is important for athletic performance due to its ability to speed physiological regeneration, improve the quality and load of subsequent training sessions, as well as decreasing the risk of fatigue and injury (Argus et al., 2013; Bompa, 1994). Fatigue is a complex and not thoroughly understood phenomenon within the human body (Powers & Howley, 2007). Muscle fatigue may vary based on the mode, duration, environmental conditions and intensity of exercise performed, which makes conclusive research on ISPC difficult to design and execute (Meeusen, Watson, Hasegawa, Roelands, & Piacentini, 2006; Powers & Howley, 2007).

Fatigue has been defined as the failure to maintain the required or expected force (Edwards, 1981). It can originate at different levels of the motor pathway and is generally divided into peripheral fatigue and central fatigue (Wan, Qin, Wang, Sun, & Liu, 2017). The central fatigue hypothesis suggests that the brain is the main limiting factor for performance and acts as a protective mechanism against excessive muscle damage (Bishop, Jones, & Woods, 2008).

Central fatigue originates in the central nervous system and limits performance by interfering with the number of functioning motor units or the frequency of motor unit stimulation (Powers & Howley, 2007; Wan et al., 2017). Motor units control the amount of muscle tissue that is activated during muscle contraction, so a reduction in
motor unit activity leads to reductions in force potential (Wan et al., 2017). The central fatigue hypothesis is based on the assumption that prolonged exercise influences the synthesis and metabolism of central monoamines such as serotonin, dopamine, and noradrenaline which has been proposed to augment lethargy and result in a reduction of motor unit recruitment (Meeusen et al., 2006). The role of serotonin in fatigue has received considerable attention due to its links to depression, sleepiness, and mood (Strüder & Weicker, 2001). Increases in serotonin during prolonged exercise has been shown to have an effect on the onset of fatigue (Davis, Alderson, & Welsh, 2000). Early work by Ikai and Steinhaus (1961) showed that a simple shout during exertion could increase ‘maximal’ strength and Ikai and Yabe (1969) showed that electrical stimulation of muscle fatigued by voluntary contractions caused increased tension development. These studies indicate there is a psychological component to the upper limits of voluntary strength and that optimal motivation or arousal is required to perform at the physiological limit (Ikai & Yabe, 1969). It is clear that the central nervous system is profoundly linked to exercise performance with roles that include ‘psyching up’ before exercise, the recruitment of motor units, and the continual feedback from receptors sensing tension, blood gasses, blood pressure, temperature, and other variables (Powers & Howley, 2007). Integration of these signals in the brain regulate power output in order to protect the organism (Powers & Howley, 2007).

Peripheral fatigue originates at or distal to the neuromuscular junction (Wan et al., 2017). Peripheral fatigue can occur through neural, mechanical, or energetic events that disrupt tension development (Fitts, 1994). Neural events that cause fatigue have been suggested to occur at the neuromuscular junction, the sarcolemma, the transverse tubules, or the sarcoplasmic reticulum (Powers & Howley, 2007). The neuromuscular
junction does not seem to be a viable site for fatigue however, as the action potential appears to reach the neuromuscular junction even when fatigued (Merton, 1954). The sarcolemma has been proposed as a potential site of fatigue due to its inability to maintain sodium and potassium concentrations during repeated stimulation (Powers & Howley, 2007). If this were the case, depolarization of the cell would occur causing a reduction of action potential amplitude (Sejersted & Sjøgaard, 2000). This depolarization of the sarcolemma may cause altered transverse tubule function such as a block of the transverse tubule action potential, which will in turn effect calcium release from the sarcoplasmic reticulum, and ultimately muscle contraction (Powers & Howley, 2007; Wan et al., 2017). The primary mechanical factor associated with fatigue is cross bridge cycling (Powers & Howley, 2007). Functional cross bridge cycling depends on the arrangement of actin and myosin, calcium (Ca) being available to bind with troponin to allow the cross bridge to bind to actin, and adenosine triphosphate (ATP) availability to activate the cross bridge to cause movement and dissociate the cross bridge from actin (Powers & Howley, 2007). A high hydrogen concentration may also cause fatigue by impacting on the cross bridge is several ways (Fitts, 1994; Sale, 1987; Wan et al., 2017). Hydrogen potentially reduces the force per cross bridge, reduces the force generated at a given Ca concentration, and inhibits sarcoplasmic reticulum Ca release (Powers & Howley, 2007). Energetics may play a role in fatigue through a mismatch in ATP generation and ATP utilization (Sale, 1987). As exercise begins ATP requirements increase and the body uses its phosphocreatine (PCr) stores to provide immediate resynthesis of ATP (Powers & Howley, 2007). As PCr stores become depleted, adenosine diphosphate (ADP) starts to build up and the myokinase reaction starts to generate ATP (Powers & Howley, 2007). As these products start to accumulate, glycolysis is activated to provide additional ATP, however this also causes hydrogen
accumulation (Banister & Cameron, 1990). If ATP demand continues to exceed supply, several reactions happen in the cell to limit work capacity and protect the cell from damage (Powers & Howley, 2007). The primary reaction is the accumulation of inorganic phosphate in the cell, which has been shown to inhibit maximal force by reducing the cross bridges ability to bind to actin (Fitts, 1994) and inhibiting Ca release from the sarcoplasmic reticulum (Allen & Westerblad, 2001). Phosphate may have a role in increasing efficiency by reducing the total ATP cost per unit of force (Nielsen & Nybo, 2003).

One of the most prevalent markers for muscular fatigue in the literature is lactate. Despite ‘lactic acid’ often being blamed for causing fatigue during intense exercise, recent research has challenged this view (Barnett, 2006; Cairns, 2006; Robergs, Ghiasvand, & Parker, 2004; Westerblad, Allen, & Lannergren, 2002). It should be noted that “lactic acid” does not exist in the body and is instead a term that represents two ionic species; lactate ions and hydrogen ions (Cairns, 2006). To create cross bridge cycling between actin and myosin filaments, myosin adenosine triphosphatase uses ATP as an energy source for muscle contraction (Cairns, 2006). However as ATP stores are generally low, PCr stores in muscle are used to resynthesise and maintain ATP. PCr also quickly declines and other sources of fuel are required (Cairns, 2006). Muscle glycogen is then called upon with glycogenolysis being activated with the breakdown of carbohydrates leading to the formation of pyruvate and ATP (Cairns, 2006). With the presence of oxygen, pyruvate enters aerobic metabolism processes in the mitochondria (Cairns, 2006). However as exercise intensity increases, pyruvate production starts to exceed the amount of oxygen that is available to oxidise it, and when pyruvate is unable to be oxidised it converts to lactate (Cairns, 2006). Lactate and hydrogens relationship...
remains unclear with some thinking lactate is directly associated with the production of hydrogen (Sahlin, Harris, Nylind, & Hultman, 1976), and others believing hydrogen is formed during glycolytic reactions that involve ATP hydrolysis (Robergs et al., 2004). It is this relationship between lactate and hydrogen that has caused uncertainty in lactates role in fatigue. The lactic acid hypothesis states that the accumulation of lactate leads to acidosis in the working muscle which causes inhibition of contractile processes however much of the evidence for this theory are from correlation-type studies (Cairns, 2006). Many studies have used both lactate and performance metrics to quantify recovery and despite the widespread belief that the two are connected, these studies have continuously failed to find a connection between the two (Coffey et al., 2004; Hanson, Stetter, Li, & Thomas, 2013; Marcello, Fortini, & Greer, 2019; Vaile, Halson, Gill, & Dawson, 2008; Zelikovski, Kaye, Fink, Spitzer, & Shapiro, 1993). There is also some evidence that lactate may be advantageous to athletic performance as Brooks (2001) proposed with his ‘lactate shuttle hypothesis’. In this hypothesis lactate is released by the working muscle fibres and is utilized by other muscle fibres or cells as metabolic fuel. Also blood lactate has a half-life of between 15 and 90 minutes depending on exercise intensity and duration (Di Prampero, 1981; Karlsson & Saltin, 1971). This is a much shorter time frame than is typical between training sessions so lactates role in training recovery is likely to be minimal. Although these findings challenge the view that lactate is a cause of fatigue, we also know that it is related to decreased performance so would be unwise to completely disregard all studies that use lactate as a biomarker. With this knowledge we can better interpret findings that use lactate as a marker of fatigue.
The term “recovery” can be used to describe three different processes best distinguished by their time frames of action. The quickest form of recovery has been termed “immediate recovery” and is used to describe time finite proximal efforts (Bishop et al., 2008). An example of this would be a runner having one leg in immediate recovery between each stride. During this immediate recovery the non-active leg must regenerate ATP and remove the byproducts of bioenergetics (Bishop et al., 2008). The next form of recovery is “short term recovery” and would be used to describe the recovery between intervals or sets of high intensity work (Bishop et al., 2008). The third type of recovery, and the one that is most relevant to the current review, is termed “training recovery” and is used to describe the recovery between successive workouts or competitive events (Bishop et al., 2008). For most, this would be used to describe the ~24 hour period between training sessions but can also be used to describe the period between multiple training or competitive events on the same day.

Physical and psychological recovery is important in the training setting, in order to maximize training adaptations (Bompa, 1994). Inadequate recovery can lead to over-training (Barnett, 2006; Wan et al., 2017) illness and injury (Barnett, 2006; O'Donnell & Driller, 2015; Wan et al., 2017) and an inability to train at the required intensity or complete the required load in the next training session (Barnett, 2006; Northey, Rattray, Argus, Etxebarria, & Driller, 2016; O'Donnell & Driller, 2015). This makes strategies that could potentially mitigate these negative effects of inadequate recovery of particular concern to those where time to recover is limited. There are many recovery strategies that may be used by athletes and trainers including but not limited to: cold water immersion (CWI), active recovery, hot water immersion (HWI), massage, contrast water therapy (CWT), compression garments (CG), cryotherapy, sleep,
nutrition and hydration, and Intermittent Sequential Pneumatic Compression (ISPC) (Tavares, Healey, Smith, & Driller, 2017).

The purpose of this brief review is to give the reader an understanding of the literature related to recovery strategies used by athletes in the sport setting. It will review various established recovery methods before reviewing the literature on the use of ISPC.

**Common recovery strategies**

The following section will briefly outline the nine most common recovery strategies in the acute setting based on the research in team-sport athletes by Tavares et al. (2017); Venter (2014); Venter, Potgieter, and Barnard (2010).

*Sleep*

While there is a consensus on the importance of sleep to both cognitive and physiological function (Fullagar et al., 2015), athletes generally sleep less than non-athletes, and possibly less than what is required (O’Donnell, Beaven, & Driller, 2018; Walters, 2002). The bodies process of revitalization peaks during stage three and stage four sleep due to an increase in the secretion of anabolic hormones such as growth hormone and testosterone by the endocrine system (Venter, 2012; Walters, 2002). More than 95% of the daily production of growth hormone happens during non rapid eye movement sleep and is therefore considered the time where the body can repair and restore itself (Gunning, 2001). While sleep is seen by many as the main means of both physical and mental recovery (Venter, 2012), a full review on sleep for recovery is beyond the scope of this review as we are mostly concerned with acute recovery strategies.
Cold water immersion

CWI refers to immersion in water <15°C (Wilcock, Cronin, & Hing, 2006). The human body has several responses to water immersion such as changes to heart rate, peripheral resistance and blood flow, as well as alterations to skin, core, and muscle temperature (Wilcock et al., 2006). These changes may influence recovery through their effects on inflammation, immune function, muscle soreness, and perception of fatigue (Halson, 2013). CWI has become one of the most popular recovery interventions with a study by Tavares et al. (2017) that investigated the recovery habits of 58 (32 elite, 26 amateur) rugby players, finding 91% of elite and 54% of amateur rugby players commonly implemented it as a recovery strategy.

However, despite the popularity of CWI in the sport setting, there remains a lack of research to suggest an optimal treatment protocol (Bleakley & Davison, 2010). Athletes and sports practitioners are largely relying on anecdotal guidelines or results from individual studies. Some research suggests the use of intermittent cycles of one minute water immersions (Sellwood, Brukner, Williams, Nicol, & Hinman, 2007), whereas others suggest immersion for as little as 30 seconds is sufficient (Wilcock et al., 2006). There is also a lack of scientific rational for the optimal water temperature with athlete tolerance or preference often the determining factor (Bleakley & Davison, 2010). Vaile et al. (2008) looked at the effect of 15 minutes of intermittent immersion in 10° C, 15° C, 20° C, continuous immersion in 20° C water and active recovery on repeat cycling performance in the heat. While all CWI protocols produced favorable outcomes when compared to active recovery, there were no significant differences between the different water temperatures.
Machado et al. (2016) performed a systematic review and meta-analysis investigating the influence water temperature and immersion time had on muscle soreness. Data was collected on both immediate effects (<24 hours post exercise) and delayed effects (>24 hours post exercise). This review included nine studies published between 2007 and 2015 and included a total of 169 participants (male, n = 141; female, n = 28). It was found studies that used water temperatures between 11 and 15° C (moderate cold) presented favorable results for immediate effects when compared to water between 5 and 10° C (severe cold) (severe cold: mean absolute difference (MD)= 0.144, 95 % CI – 1.299, 1.526, p = 0.875; moderate cold: MD = 0.273, 95 % CI 0.107, 0.440, p = 0.001). There were similar findings for delayed effects (severe cold: MD = 0.057, 95 % CI – 1.483, 1.598, p = 0.942; moderate cold: MD = 0.317, 95% CI 0.102, 0.532 p = 0.004).

Immersion times were separated into short (<10min), medium (10-15min), and long (>15min). The medium immersion category was associated with the best results for immediate effects (short immersion: MD = 0.646, 95 % CI -0.360, 1.652, p =0.208; medium immersion: MD = 0.227, 95 % CI 0.139, 0.314, p <0.001; longer immersion: MD = -1.300, 95 % CI -2.927, 0.327, p = 0.117). The medium immersion category also had the best results on delayed effects (short immersion: MD = 0.728, 95 % CI -0.561, 2.017, p = 0.268; medium immersion: MD = 0.317, 95 % 0.102, 0.532, p = 0.004; longer immersion: MD = -2.200, 95 % CI -4.169, -0.231, p = 0.029). This study goes a long way to provide guidelines for optimal temperature and duration for reduction of muscle soreness following training or competition. However, further research is required investigating if these findings are similar for performance measures.

Bleakley et al. (2012) completed a systematic review on CWI that included a total of 17 studies with a total of 366 participants. Data on perceptual measures of muscle soreness,
and measures of maximal strength were collected. There were 12 studies that provided data on perceptual measures of muscle soreness after CWI and five studies that reported on maximal strength after CWI. The findings by Bleakley et al. (2012) on perceptual measures of muscle soreness revealed no significant differences between CWI and passive recovery groups at immediate follow-up (standardised mean difference (SMD) -0.07, 95% CI -0.43 to 0.28; 7 trials). At all four subsequent times, pooled results showed significantly lower levels of pain in the CWI group compared to passive recovery (24 hours: SMD -0.55, 95% CI -0.84 to -0.27, 10 trials); (48 hours: SMD -0.66, 95% CI -0.97 to -0.35; 8 trials); (72 hours: SMD -0.93, 95% CI -1.36 to -0.51; 4 trials); (96 hours: SMD -0.58; 95% CI -1.00 to -0.16; 5 trials). Measures of maximal strength taken from a range of time points after either CWI or passive recovery tended to favour the passive recovery group, however there were no significant differences between groups apart from at immediate follow up.

A meta-analysis by Leeder, Gissane, van Someren, Gregson, and Howatson (2012) investigated the effect of CWI on muscle soreness, creatine kinase (CK) levels, muscle power, and muscle strength. This meta-analysis included 14 studies with a total of 239 participants. CWI was found to have a moderate effect on alleviating muscle soreness post exercise (Hedges’ g = 0.525, p < 0.001). Sub-group analysis of different time points revealed CWI was effective at reducing muscle soreness at all time points up to 96 hours post exercise. Further sub-group analysis investigating CWI efficacy post eccentric and high intensity exercise found CWI to be effective at reducing muscle soreness. These results were consistent for both 24 and 48 hours post high intensity exercise and at 48 hours post-eccentric exercise. CWI was not however effective at reducing muscle soreness 24 hours post eccentric exercise. Investigation of CWI effect
on CK levels in the blood revealed a statistically significant reduction of CK post CWI (p = 0.022) although there was only a small effect size (Hedges’ g of 0.221). It was also found that CWI was effective at improving the rate of recovery of muscle power post exercise (Hedges’ g = 0.597, p = <0.001) and was found to be effective at all time points (24, 48, and 72 hours). CWI was not found to be effective for measures of muscular strength in this meta-analysis.

*Hot water immersion/thermotherapy*

HWI refers to immersion in water that raises the core body temperature (Wilcock et al., 2006) and occurs in water with a temperature >36°C (Greenleaf & Kaciuba-Uscilko, 1989). Like CWI, there is little research on the optimal treatment protocol for HWI. Brukner and Khan (2001) suggested a HWI duration of 10-20 minutes to aid in athletic recovery, however this time period does not appear to be based on a body of research and is unsubstantiated (Wilcock et al., 2006).

An increase in superficial tissue temperature may cause an increase in the cutaneous blood flow due to peripheral vasodilation and an increase in heart rate (Bonde-Petersen, Schultz-Pedersen, & Dragsted, 1992). This increase in cardiac output and lower peripheral resistance may then lead to an increase in blood flow (Bonde-Petersen et al., 1992) which in turn leads to an increase in the permeability of cellular, lymphatic, and capillary vessels (Baker, Robertson, & Duck, 2001), thus leading to an increase in metabolism, nutrient delivery, and waste removal from the cells (Côté, Prentice Jr, Hooker, & Shields, 1988).
There is also limited evidence that HWI may increase neural transmission (Cotts, Knight, Myrer, & Schulthies, 2004), increase proprioception and improve reaction time (Burke et al., 2001), and increase muscle elasticity (Brukner & Khan, 2001). While there are many anecdotal claims supporting the use of HWI, there remains little research-based evidence that supports them. While there are several hypothesized mechanisms of action for HWI, there is a lack of literature investigating its effect on athletic recovery and subsequent performance. Therefore further research is required before recommendations can be made.

**Contrast water therapy**

CWT involves alternating between a hot >36°C (Greenleaf & Kaciuba-Uscilko, 1989) and cold <15°C (Wilcock et al., 2006) water. Protocols vary between 30 seconds and 6 minutes of immersion in one temperature extreme, immediately followed by 30s-6mins in the other temperature extreme (Wilcock et al., 2006). This is repeated and may last between 4-30 minutes in total duration (Wilcock et al., 2006). CWT is hypothesized to cause alternating vasoconstriction and vasodilation which is thought to have a similar effect to muscle pumping, increasing blood flow and metabolite removal, thus enhancing recovery (Wilcock et al., 2006).

Bieuzen, Bleakley, and Costello (2013) performed a systematic review of 13 studies investigating CWT and found that CWT resulted in significantly greater improvements in muscle soreness when compared to passive recovery. The authors also found CWT to be more effective at reducing loss of muscle strength post exercise when compared to passive recovery.
A study by Gill, Beaven, and Cook (2006) analyzed post match CK levels in rugby players, and found a positive response to CWT, active recovery, and CG, in the reduction of CK when compared to a control condition (active recovery = 88.2% reduction, CWT = 85% reduction, CG = 84.4% reduction, passive recovery = 39% reduction). Transdermal exudate samples were obtained 3.5 hours before, immediately after, 36 and 84 hours post competitive rugby matches for CK analysis. This process was repeated over a four game period. The authors suggest that caution should be applied towards generalizing these findings to recovery from high intensity exercise as rugby has a high level of direct impact, which likely causes a greater amount of CK post match.

A study by Coffey et al. (2004) found a 15-minute CWT protocol to have no effect on performance compared to active recovery and passive recovery. Participants completed two pairs of treadmill runs to exhaustion with the first run at 120% and the second at 90% of peak running speed. The two runs were separated by 15 minutes of standardised rest. After the second run participants completed a 15-minute recovery protocol of active recovery, passive recovery, or CWT. Four hours after the start of the test protocol, participants completed an additional two run to exhaustion tests. No performance benefits for CWT when compared to active and passive recovery were found.

Current literature suggests that CWT may be effective at reducing muscle soreness, markers of muscle damage such as CK, and strength loss. However it may not have an effect on subsequent running performance. Careful consideration towards type, and intensity of exercise should be given before generalizing these findings as these
variables have different physiological outcomes which are likely impacted differently from recovery strategies.

*Active recovery*

Active recovery involves exercising at a moderate intensity during recovery from high intensity exercise (Fairchild et al., 2003). Active recovery is widely considered efficacious, however, much of the literature supporting this claim are based on its effect on the rate of post exercise lactate removal (Barnett, 2006). As discussed earlier in this literature review, lactate has been challenged as a valid marker of fatigue. This makes findings that use lactate as the sole marker of fatigue recovery questionable (Barnett, 2006; Bond, Adams, Tearney, Gresham, & Ruff, 1991; Cairns, 2006; Robergs et al., 2004; Westerblad et al., 2002).

Ortiz Jr, Elder, Elder, and Dawes (2019) performed a systematic review of active recovery that included 26 articles with a total of 471 participants. The authors found an active recovery protocol of 6 - 10 minutes to be the most effective duration with positive outcomes for sport specific performance metrics. This was in comparison to active recovery strategies of short (30 seconds – 2 minutes) and moderate (10 minutes – 72 hours) durations, which had negligible or negative effects on athletic performance. There has been a lack of research on the 3 – 5 minute window for active recovery so further research is required investigating this time frame before solid conclusions can be made on an optimal duration for active recovery. A lack of evidence remains for an optimal intensity at which active recovery should be performed with percentage of heart rate max, VO$_{2\text{max}}$, and maximal aerobic speed the general markers of prescribing intensity. The authors found a large body of evidence that suggests that markers of
lactate is an unreliable indicator of enhanced subsequent performance with several studies reporting active recovery to cause significantly lower lactate levels while having no effect on performance (Franchini, de Moraes Bertuzzi, Takito, & Kiss, 2009; Ouergui, Hammouda, Chtourou, Gmada, & Franchini, 2014; Toubekis, Peyrebrune, Lakomy, & Nevill, 2008; Toubekis, Smilos, Bogdanis, Mavridis, & Tokmakidis, 2006; Watts, Daggett, Gallagher, & Wilkins, 2000). There was some evidence of improved performance measures with several studies reporting improvements in counter-movement jump (CMJ) and timed swim performances (Greenwood, Moses, Bernardino, Gaesser, & Weltman, 2008; Jemni, Sands, Friemel, & Delamarche, 2003; Rasooli, Jahromi, Asadmanesh, & Salesi, 2012; Rey, Lago-Peñas, Casáis, & Lago-Ballesteros, 2012; Toubekis et al., 2008; West et al., 2013). However, there is evidence that active recovery interventions may have a positive psychological effect by increasing an athlete’s perception of recovery, which may positively influence subsequent performance. It is clear from this review that further investigation of active recovery is required. Future research should focus on; the optimal intensity of active recovery, active recovery performed in the 3 – 5 minute time frame, and physiological markers of recovery other than lactate.

A study by Lum, Landers, and Peeling (2010) looked at the effects of an active recovery swim session on subsequent running performance of nine well-trained triathletes. The triathletes completed a high intensity running session followed 10 hours later by either a swim session that consisted of 20 x 100m at 90% of 1km time trial speed or passive recovery. At 24 hours post the initial session, participants completed a time to fatigue run test. The swim trial participants ran for 830 ± 98s, in comparison passive trial participants ran for 728 ± 183s. It is difficult however to isolate this positive effect
solely to active recovery as the authors suggest improvements may have been from hydrostatic benefits of water such as an increase in venous return and blood flow.

An additional consideration is whether active recovery affects the rate of glycogen resynthesis between recovery sessions (Barnett, 2006). Bonen, Ness, Belcastro, and Kirby (1985) found a higher rate of glycogen resynthesis during passive recovery when compared to active recovery, which suggests that active recovery may limit glycogen resynthesis. Active recovery has been shown to increase lactate oxidation in the working muscle (Bangsbo, Graham, Johansen, & Saltin, 1994) which may be detrimental to glycogen resynthesis as lactate has the potential to be a major carbon source for the replenishment of glycogen when food is not available (Fairchild et al., 2003).

While there are many studies that support the use of active recovery as a method for recovery, the fact that a large amount of these studies use lactate as their main marker of recovery is problematic. If lactate is in fact not a cause of fatigue, then active recovery may simply be taking away valuable time and energy from the athlete that could be spent on other acute recovery methods.

*Whole-body cryotherapy chamber*

Cryotherapy is defined as body cooling for therapeutic purposes (Bleakley, Bieuzen, Davison, & Costello, 2014). Cryotherapy in the sport and exercise setting has traditionally been applied through ice packs, and the previously discussed CWI. Recently a new form of cryotherapy has emerged, termed whole-body cryotherapy. Whole-body cryotherapy involves exposing the body to extremely cold dry air
(typically between -100°C and -140°C) by entering an environmentally controlled room or chamber for a short amount of time (typically between two and five minutes). To minimize the risk of cold related injury, individuals wear minimal clothing, gloves, a woolen headband to cover the ears, a mask covering the nose and mouth, and dry shoes and socks, and all sweat is removed before they enter the chamber (Banfi, Lombardi, Colombini, & Melegati, 2010; Bleakley et al., 2014). The purported effects of whole body cryotherapy include decreased tissue temperature, reduction in inflammation, analgesia, and enhanced recovery following exercise.

Bleakley et al. (2014) completed a review evaluating the current literature on whole-body cryotherapy. They found evidence that whole-body cryotherapy had a similar effect on skin and intramuscular (at 2cm depth) temperature when compared to CWI, and had a smaller effect than application of ice packs. They found evidence from three studies that whole-body cryotherapy does not affect markers of muscle damage (CK, lactate dehydrogenase, and aspartate aminotransferase) after exercise, however controlled studies suggest that whole body cryotherapy may have a positive influence on inflammatory mediators, antioxidant capacity and autonomic function.

While there is a small amount of evidence that supports whole body cryotherapy, it currently appears that the benefits are not significantly greater than other forms of cooling such as CWI or ice packs. Given the high cost of whole body cryotherapy, the current recommendation would be to utilize CWI or ice packs before whole body cryotherapy.
Compression garments

Compression has been used in the clinical setting for many years with applications that include treatment of oedema, pulmonary embolism, stasis and deep vein thrombosis, and in management of wound scars and venous leg ulcers (MacRae, Cotter, & Laing, 2011). CG’s have begun to be used as a recovery method in the sports and exercise environment as they are thought to influence recovery time by improving venous return, which in turn can assist in the removal of metabolic waste (Davies, Thompson, & Cooper, 2009). There are many commercially available CG’s that are marketed as a means to improve performance and recovery from exercise despite these claims often lacking satisfactory supporting evidence with equivocal findings in the literature (MacRae et al., 2011).

Hill, Howatson, Van Someren, Leeder, and Pedlar (2014) completed a meta-analysis of 12 compression recovery studies that included a total of 205 participants. The authors found the use of CG’s to have a moderate benefit in reducing delayed onset of muscle soreness (DOMS), recovery of muscle strength post exercise, recovery of muscle power following exercise and reducing concentrations of CK post exercise. MacRae et al. (2011) assessed 19 studies with a total of 273 participants to investigate the effect of wearing CG’s on indicators of recovery from exercise. From these studies it was concluded that wearing CG’s provides limited physiological or performance effects although reports of detrimental effects were rare. Some evidence was found that CG’s may alter local blood flow, and protein or metabolite clearance, mitigate swelling and reduce perceived muscle soreness but findings were often isolated and inconclusive. The level of compression ranged from ~7mmHg to 33mmHg with seven studies not reporting the level of compression.
Bochmann et al. (2005) investigated the effect of a compression sleeve (13-23 mmHg) on arterial inflow to the forearm. They found a 200% increase in arterial perfusion when compared to a control (contralateral forearm with no compression sleeve). Increases were maintained from 10 minutes to 3 hours and were also present during 70 minutes of light rhythmic hand exercises with both hands. The authors found that an applied pressure of 20mmHg had the biggest influence on arterial perfusion. Unfortunately this study did not contain any performance measures to assess the effect this increase in arterial perfusion had on subsequent performance.

The simplicity and lack of effort of wearing CG’s should be taken into consideration when concluding whether they are worth using as a recovery method. In the study by Gill et al. (2006) it was shown that CG’s had only a slightly inferior effect on recovery (judged by clearance of CK) compared to active recovery and CWI. However athlete adherence to being prescribed CG use is likely to be higher than alternative recovery strategies as CG’s requires no additional energy expenditure or discomfort.

Nutrition and hydration

Literature clearly shows that not consuming an adequate amount of calories and/or enough of the right types of macronutrients may impede adaptation to training while a proper balanced diet augments physiological adaptations to training (Kreider et al., 2010). Athletes need to ensure they are consuming enough calories to meet the energy demands of their training and competition to offset energy expenditure (Leutholtz & Kreider, 2001). The average daily caloric intake for an untrained individual ranges between 1900-3000kcal/day whereas up to 12,000kcal/day can be required for
endurance athletes such as competitors in the Tour de France (Leutholtz & Kreider, 2001). Maintaining an energy deficient diet can lead to issues such as loss of lean body mass, illness, overtraining syndrome, and reductions in performance (Kreider, Fry, & O'Toole, 1998). Athletes also need to be consuming adequate proportions of carbohydrates, protein, and fat to fuel and recover from exercise. Protein is important for recovery as if an athlete has a protein insufficient diet, they will maintain a negative nitrogen balance, which may then result in an increase in protein catabolism and slow recovery (Kreider et al., 2010). If protein inefficiencies are not addressed it can lead to muscle wasting and training intolerance (Leutholtz & Kreider, 2001). Fat is an important macronutrient in an athletes diet to replenish intramuscular triacylglycerol stores and to provide essential fatty acids (Venkatraman, Leddy, & Pendergast, 2000).

Restoration of body fluids are also an integral part of the training recovery process (Bishop et al., 2008) as prior dehydration can be detrimental to performance (Barnett, 2006). Rehydration enhances restoration of electrolytes so inclusion of sodium and potassium in post training beverages is also recommended (Bishop et al., 2008). Following dehydration by 2% body mass, an appropriate rehydration strategy that has sufficient volume and sodium content can restore net fluid balance and plasma volume within four hours (Barnett, 2006).

**Stretching**

Despite stretching being one of the most used acute recovery strategies, with one study (Tavares et al., 2017) finding that 100% of surveyed elite rugby players and 77% of surveyed amateur rugby players use it for recovery, literature examining its effects as a recovery method is sparse.
Dawson, Gow, Modra, Bishop, and Stewart (2005) investigated stretching immediately after an Australian football match and found that stretching significantly improved power output during a 6 second cycle sprint 15 hours after the match when compared to a control. Stretching has also been shown to improve range of motion and reduce muscle soreness following a muscle damaging protocol when compared to a control (Kokkinidis, Tsamourtas, Buckenmeyer, & Machairidou, 1998).

Kinugasa and Kilding (2009) performed a study comparing three post match recovery strategies on youth soccer players. 28 participants played three 90-minute soccer games on different days followed by either CWT, a combined recovery protocol (CWI and active recovery) or a protocol whereby they completed seven minutes of static stretching and two minutes with their legs raised above heart level. They found that static stretching was not as effective as CWT or the combination protocol at improving the subjects perceived recovery. However it is difficult to isolate these finding’s on static stretching as static stretching was combined with an elevation protocol. Montgomery et al. (2008) found similar results where a recovery strategy, which involved static stretching and carbohydrate intake, was not as effective as CWI at improving performance measures (20m sprint, basketball specific running drill, sit and reach test) following basketball games. In a review article by Barnett (2006) it was concluded that there was no apparent long or short-term benefit to static stretching as a recovery modality.
Intermittent, Sequential Pneumatic Compression (ISPC)

A relatively new recovery strategy in the athletic setting is the use of ISPC. ISPC involves inflatable sleeves being placed on the arms or legs. These sleeves have several separate chambers, which inflate sequentially from distal to proximal aspects of the limb, mimicking the anatomical muscle venous pump system (O'Donnell & Driller, 2015). The use of ISPC, has been explored as a potential tool for recovery due to its ability to exert up to four times as much pressure (~80mmHg) (Hanson et al., 2013) as CG’s. The use of ISPC originated from the medical field where it is used as a means to treat various ailments including deep venous thrombosis (Chen, Frangos, Kilaru, & Sumpio, 2001; Marcello et al., 2019; Morris & Woodcock, 2010), lymphedema (Airaksinen, 1989; Chen et al., 2001; O'Donnell & Driller, 2015; Overmayer & Driller, 2017; Winke & Williamson, 2018; Zelikovski et al., 1993), and posttraumatic edema (Airaksinen, 1989; Airaksinen, Kolari, & Miettinen, 1990; Overmayer & Driller, 2017). Research on the efficacy of ISPC devices in the sport setting remains inconclusive, with mixed findings (Airaksinen, 1989; O'Donnell & Driller, 2015; Overmayer & Driller, 2017; Wiener, Mizrahi, & Verbitsky, 2001; Zelikovski et al., 1993).

Proposed mechanisms for ISPC enhancing exercise recovery

ISPC compression

The primary mechanism that is often attributed to ISPC’s efficacy is compression (Airaksinen, 1989; Chen et al., 2001; O'Donnell & Driller, 2015; Overmayer & Driller, 2017; Winke & Williamson, 2018). As discussed earlier in this review, compression is a strategy that has been used in therapeutic medicine for many years (MacRae et al.,
A potential mechanism for compression is increased blood flow. Increased blood flow has been attributed to the myogenic response which is as follows: garment or equipment applies compression of underlying tissues which reduces transmural pressure in local arterioles, causing vessels to relax (i.e dilate) thereby increasing flow (MacRae et al., 2011). However Styf (1990) reported reductions in muscle blood flow with external compression of the tibialis anterior during dorsiflexion. Muscle blood flow decreased from 35 ± 10 mL/100g⁻¹/min⁻¹ without compression to 11 ± 5 mL/100g⁻¹/min⁻¹ with compression. Applied pressures were not reported in this study. These data indicate that an “ideal” pressure range may exist for an increase in blood flow from compression. Pressures too low likely result in little to no change whereas pressures too high result in adverse effects (Weerapong, Hume, & Kolt, 2005). Another reported mechanism for compression is metabolite clearance. The two most common measured metabolites are creatine kinase (CK) and lactate. CK levels are used as a marker of fatigue as strenuous exercise that damages skeletal muscle cell structure results in an increase of CK in the blood (Gill et al., 2006). Several studies have investigated compressions effect on removal of these metabolites with positive findings (Duffield & Portus, 2007; Gill et al., 2006; Hill et al., 2014; Kraemer, Bush, Wickham, Denegar, Gomez, et al., 2001; Kraemer, Bush, Wickham, Denegar, Gómez, et al., 2001; Kraemer et al., 2010).

**Massage**

The sequential compression of an ISPC device is similar to techniques applied through massage therapy to treat muscle fatigue (Marcello et al., 2019; Zelikovski et al., 1993). Massage has been defined as “a mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well-being”
Massage is commonly used in sports for both performance enhancement and recovery as it is believed to increase blood flow, reduce muscle tension, reduce neurological excitability and increase the sense of well-being (Weerapong et al., 2005). Despite its popularity, there is limited scientific evidence that it enhances performance, enhances recovery or prevents injury (Weerapong et al., 2005). Much like many other recovery interventions, the effects of massage are likely due to several different mechanisms, which makes pinpointing specific adaptations difficult. There have been biomechanical, physiological, neurological, and psychological mechanisms proposed as contributing factors for the positive effect of massage on recovery with no one factor likely to be working in isolation.

Massage is thought to decrease tissue adhesion through application of mechanical pressure on muscle tissue (Weerapong et al., 2005). It is thought that through mobilizing and elongating shortened or adhered connective tissues there is an increase in muscle-tendon compliance. Improved muscle compliance results in a less stiff muscle tendon-unit (Weerapong et al., 2005). A reported benefit from massage in regards to enhancement of athletic performance is an increase in blood circulation (Weerapong et al., 2005). However, scientific evidence does not support this hypothesis with studies either having design flaws or reporting no findings of significance. Another possible physiological mechanism for massages effect is hormonal changes. Positive changes in serotonin and cortisol levels after massage have been reported (Field, Grizzle, Scafidi, & Schanberg, 1996; Hernandez-Reif, Field, Krasnegor, & Theakston, 2001; Ironson et al., 1996), however the exact mechanism as to why this occurs is unclear. There is some evidence that massage can increase parasympathetic activity by reducing heart rate and blood pressure (Groer et al., 1994; Labyak & Metzger, 1997; Longworth, 1982),
increase relaxation substances such as endorphins (Kaada & Torsteinbø, 1989), and increase heart rate variability (Delaney, Leong, Watkins, & Brodie, 2002).

Massage is often used as a method to relieve pain (Hernandez-Reif et al., 2001; Weerapong et al., 2005). The possible mechanisms for this are neurological, physiological, and mechanical. Neurological mechanisms may include activation of neural gating mechanisms in the spinal cord (Weerapong et al., 2005). It is hypothesized that tactile information from massage stimulates large fast nerve fibers and blocks smaller slower nerve fibers that detect pain, presumably from local lateral inhibition in the spinal cord, however there is minimal scientific evidence supporting this claim (Weerapong et al., 2005).

**Temperature**

Despite not being considered as a primary physiological mechanism of ISPC, increasing muscle temperature of the treated limb may play a role in performance enhancement when said performance takes place shortly after treatment. ISPC is likely to increase muscle temperature of the treated limb through both superficial skin friction causing hyperaemia within the massaged area (Longworth, 1982; Weerapong et al., 2005) and the fact that the structure of ISPC devices likely act as heat trap for escaping body heat.

Increased muscle temperature and core temperature have been shown to potentially improve muscle force and power production (Faulkner et al., 2012), improved muscle blood flow (Chiesa et al., 2015; Clarke, Hellon, & Lind, 1958), augmented muscle glycogen and carbohydrate utilization (Starkie, Hargreaves, Lambert, Proietto, & Febbraio, 1999), accelerated oxyhaemoglobin dissociation (Barcroft & King, 1909),
increased metabolic rate and enzymatic reactions (Gray, De Vito, Nimmo, Farina, & Ferguson, 2006), and changes in both mechanical efficiency and muscle fiber conduction velocities (Ferguson, Ball, & Sargeant, 2002; Gray et al., 2006).

There is evidence that elevated skin and intramuscular temperature may provide performance benefits to measures of power (Mohr, Krstrup, Nybo, Nielsen, & Bangsbo, 2004). An increase in muscle temperatures following a hot water (42.8 degrees) immersion protocol resulted in an increase of ATP turnover and muscle fiber contraction velocity (Racinais, Cocking, & Périard, 2017). The increase in ATP turnover rate was likely due to an upregulation of cross-bridge cycling, as myofibrillar ATP synthase activity is dependent on temperature (He, Bottinelli, Pellegrino, Ferenczi, & Reggiani, 2000; Stienen, Kiers, Bottinelli, & Reggiani, 1996). This mechanism has been observed during both intense dynamic exercise (Febbraio, Carey, Snow, Stathis, & Hargreaves, 1996) and isometric contractions (Edwards et al., 1972).

*Placebo or “belief” effect*

An important consideration when looking at the reported benefits of a new and novel recovery device is the placebo effect. The term ”placebo effect” was first recognized in the medical field where it was first used to define any medicine or treatment that was adapted more to please than to be of benefit to the patient (De Craen, Kaptchuk, Tijssen, & Kleijnen, 1999). Historically the placebo effect was thought of as a result of biases in subjective symptom reporting however there is increasing evidence that there are specific neurobiological mechanisms that mediate these effects (Halson & Martin, 2013; Scott et al., 2008). The placebo effect may be-related to psychological or social changes associated with expectation, reward, hope, and a reduction of anxiety and stress.
(Benedetti, 2013). Neurobiological changes such as increases and decreases in the metabolic activity of the rostral anterior cingulate, a cognitive-emotional integrative region of the brain, have also been proposed as contributors to the placebo effect (Scott et al., 2008). Additionally the placebo effect has been shown to increase the correlations between the anterior cingulate and periaqueductal grey matter activity which is centrally involved in opioid-mediated antinociception (Scott et al., 2008). The neurobiology of the placebo effect was initially examined by Levine, Gordon, and Fields (1978) in a study where placebo analgesia was shown to be blocked by the opioid-receptor antagonist naloxone. Following oral surgery patients pain ratings were reduced by 39% when given a placebo however the placebo effect was non-existent following administration of naloxone. In the sport science setting there are a number of studies that have documented the placebo effects associated with commonly used and widely accepted ergogenic aids such as carbohydrate drinks (Clark, Hopkins, Hawley, & Burke, 2000), caffeine (Beedie, Stuart, Coleman, & Foad, 2006), bicarbonate (McClung & Collins, 2007) and altitude training (Garvican et al., 2011). For research on sport and exercise recovery a strong likelihood exists that research participants’ beliefs and expectations contribute to performance outcomes (Halson & Martin, 2013). Given the nature of ISPC, designing a study that has a true placebo condition is very difficult, hence why many studies have not incorporated one into their study design. Therefore, it is very difficult to ascertain whether positive effects from ISPC studies are due to the proposed mechanisms or from the placebo effect.
ISPC literature

Clinical measures

The initial scientific studies looking at ISPC were largely in the clinical setting. Hartman et al. (1982) investigated ISPC as a potential treatment for deep vein thrombosis in patients undergoing surgery for either hip replacement or hip fracture. 104 participants were separated into two even groups, one where participants had ISPC treatment accompanied by elevation, and a control group where participants completed elevation only. Thrombi was identified and located through the use of Doppler ultrasound, phleborheography, and radioiodinated fibrinogen scanning. Deep vein thrombosis developed in 10 of the control group (19%) with only a single case developing in the ISPC group (2%). This difference was statistically significant (p < 0.05).

ISPC has also been investigated as a possible treatment for lymphatic disorders. Dini et al. (1998) performed a randomized study comparing a pneumatic compression protocol to a control protocol in patients with post mastectomy lymphedema. Circumference was taken from seven different sites on the affected arm and compared to the same sites on the unaffected arm as a control. The differences between arms were added up and the result was designated ‘delta’ and was the metric recorded. The pneumatic compression was applied in two cycles of two weeks each with five two-hour sessions per week, separated by a five-week interval. The control group received no treatment. Although the pneumatic compression group had a 25% reduction in mean delta values, a result that reached significance (p = 0.009), and the control group only had a 20% reduction in
mean delta values, a result that failed to reach significance ($p = 0.33$), there was a lack of significance when comparing the two groups ($p = 0.084$).

Airaksinen (1989) evaluated changes in posttraumatic ankle joint mobility, pain and edema after ISPC treatment. Participants were outpatients who had suffered lower leg fractures and had their lower limb in an immobilizing cast for six to 12 weeks. All participants still had marked edema and reduced mobility two weeks after removal of the cast. The trial group consisted of 22 patients who were given a 75-minute ISPC treatment on five consecutive days. The control group was made up of 12 patients who were given no treatment. The trial group increased ankle mobility by 11.9° with the control group only increasing ankle mobility by 1.0°, a difference that was highly significant ($p <0.001$). The trial group had a greater reduction in edema with a 170ml reduction compared to 15ml in the control group ($p <0.001$). Additionally the trial group experienced markedly greater pain relief as judged by a visual analogue scale with a drop from 3.6 to 2 compared to the control group where pain scales dropped from 4.9 to 4.6, the difference between the two groups was highly significant ($p <0.001$).

In another study by Airaksinen et al. (1990) the differences between treating acute ankle sprains with elastic bandages alone, or a combination of elastic bandages and ISPC was investigated in 44 outpatients with acute ankle sprains. Edema, degree of ankle motion, pain, and limb dysfunction was measured at the initiation of the patient into the study, after treatment for one week, and after a four-week follow up. Edema was measured by the difference between the affected leg and the contralateral leg. Edema decreased faster in the study group than the control group with volume of edema
only 33mL (SE ± 6) after five treatments compared to the control group where edema was 80mL (SE ± 7), a difference that was highly significant (p <0.001). Ankle range of motion was improved markedly more in the ISPC group than in the control group at both one week of treatment (p <0.001) and at the four week follow up (p <0.001). There were similar findings favoring the ISPC group for pain after both one-week of treatment (p <0.001) and at the four week follow up (p <0.001) and for limb function at both the one week and four week time points (p <0.001).

Morris and Woodcock (2002) analyzed the effect ISPC had on arterial blood flow. They collected data from 19 healthy subjects and 17 subjects with peripheral arterial disease. A Doppler ultrasound was used to measure common femoral artery blood flow velocities during 10 minutes of ISPC of the calf and thigh while the subject was in a supine position. They found that during the compression phase of ISPC blood flow velocity decreased slightly (15% in healthy subjects and 6% in patients) and increased on release (21% in healthy subjects and 29% in patients) with an overall increase in blood flow of 1% and 2% respectively.

Performance measures

Researchers have begun to investigate ISPC in the sport performance setting as it has been hypothesized that its mechanisms for preventing deep vein thrombosis and lymphedema may also be efficacious in decreasing muscular fatigue and improving subsequent performance. Studies have primarily focused on ISPC’s efficacy on
endurance (see table 1), however measures of muscular strength, muscular power and strength endurance have also been used (see table 1).

Wiener et al. (2001) studied tibialis anterior recovery through the use of an ISPC device on the legs. Eight male participants performed a 12-minute exercise protocol aimed at fatiguing the tibialis anterior. The recovery protocol involved participants sitting with one leg in an ISPC device using 80mmHg of pressure and the other acting as a control (no ISPC). Following the recovery protocol the ISPC leg resulted in a higher power output (judged by an electro-myography) of the tibialis anterior muscle than the control leg while performing a weight-lifting exercise. The authors concluded that ISPC treatment of a fatigued muscle following a sustained effort improves its contractile capacity compared to passive recovery.

Zelikovski et al. (1993) also investigated ISPC’s effect on a performance measure. Eleven healthy male participants performed two maximal incremental exercise tests to exhaustion on electronically braked cycle ergometers. Test one involved participants cycling at a constant workload (80% of VO2 max) until exhaustion. Participants then completed either a 20-minute ISPC (leg sleeves) recovery protocol or a control condition where they sat passively. The authors reported a 45% prolongation of exercise duration for the ISPC condition when compared to the control condition (ISPC = 10.9 min bout 1, 8.7 min bout 2, CON = 11.6 min bout 1, 6.4 min bout 2), a difference that was significant (p < 0.004).
O'Donnell and Driller (2015) performed a study investigating the effect of ISPC on recovery between exercise bouts. The participants were 10 well-trained male triathletes. Participants completed 40-minutes of a high intensity interval session on a cycle ergometer followed by 30 minutes of either passively sitting (control) or sitting with ISPC sleeves applied to the legs. Following the recovery period, participants completed a 5km run time trial test on a treadmill. This study resulted in no statistically significant findings ($p > 0.05$), between the use of an ISPC device and a control condition for performance recovery.

Overmayer and Driller (2017) examined the efficacy of ISPC on recovery of performance measures in 21 trained cyclists. The authors used a 20 minute cycling bout to simulate the conditions experienced in a scratch race, then a 4-minute cycling time trial to simulate the conditions of an individual pursuit as experienced in an Omnium track cycling competition. Either 30 minutes of recovery in ISPC boots or passive recovery separated the exercise bouts. Total power output (watts) and average power output (watts) during the 4-minute time trial was used as the performance measures. No statistically significant differences were found between ISPC and control conditions for total power output (both CON and ISPC: 221 [SD 50] W) between ISPC and control conditions ($p >0.05$). Likewise there were no significant differences between conditions for average power output ($p >0.05$).

Northey et al. (2016) compared vascular occlusion (OCC), ISPC and a passive control on recovery following a resistance-training bout (10 sets of 10 repetitions of back squat). Twelve strength trained male participants participated in this randomized crossover
study. Performance measures included isokinetic torque of the quadriceps measured by an isokinetic dynamometer; squat jump height and CMJ height. These performance measures were taken before exercise, immediately after exercise, one hour and 24 hours post exercise. There were no statistically significant differences between conditions for peak isokinetic torque of the quadriceps (p = 0.561), squat jump (p = 0.843), or CMJ (p = 0.879).

Chleboun et al. (1995) investigated maximum voluntary isometric strength of the elbow flexors following an exhaustive eccentric weight training protocol in 22 female college students. Participants were untrained and had not been lifting weights or completing regular arm exercises for six months before the study. Muscle damage was induced by participants completing three sets of eccentric exercise that were equal to 90%, 80%, and 70% of the isometric maximal voluntary contraction with two minutes between each set. As many repetitions as possible were completed where the participants could maintain an eccentric contraction of five seconds. Strength was assessed using a cable and pulley system with a strain gauge attached to the cable. Subject’s isometric maximum voluntary contractions were measured pre exercise, immediately post exercise and daily for five days post exercise. The authors found no significant changes in strength between ISPC and control at any time point.
Table 1. The effect of ISPC on measures of performance

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Sample size</th>
<th>Participant description</th>
<th>Groups/interventions</th>
<th>Measures</th>
<th>Effect of ISPC on performance measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wiener et al. (2001)</td>
<td>Pre-post single group design.</td>
<td>8; M</td>
<td>Healthy non-athletes</td>
<td>ISPC – 3 min at 80mmHg CON – passive recovery</td>
<td>BMG of tibialis anterior during sustained weightlifting protocol</td>
<td>↑</td>
</tr>
<tr>
<td>Zelikovski et al. (1993)</td>
<td>Pre-post crossover design</td>
<td>11; M</td>
<td>Healthy men</td>
<td>ISPC – 20 min at 50mmHg CON – 20 min passive rest</td>
<td>Time to exhaustion</td>
<td>↑</td>
</tr>
<tr>
<td>O’Donnell and Driller (2015)</td>
<td>Randomized crossover design</td>
<td>10; M</td>
<td>Well trained triathletes</td>
<td>ISPC – 30 min at 80mmHg CON – 30 min seated</td>
<td>5kmTT (treadmill)</td>
<td>↔ → Trivial</td>
</tr>
<tr>
<td>Overmayer and Driller (2017)</td>
<td>Counterbalanced crossover design</td>
<td>13; M, 8; F</td>
<td>Trained cyclists</td>
<td>ISPC – 30 min at 80mmHg CON – 30 min seated</td>
<td>Cycle 4minTT (average power output)</td>
<td>↔ → Trivial</td>
</tr>
<tr>
<td>Northey et al. (2016)</td>
<td>Randomized cross-over design</td>
<td>12; M</td>
<td>Healthy males</td>
<td>ISPC – 45 min at 80mmHg OCC – 12 min alternating between legs every 3 min at 220mmHg CON – 45 min lying down</td>
<td>Muscle dynamometry, vertical jump SJ and CMJ</td>
<td>↔</td>
</tr>
<tr>
<td>Chleboun et al. (1995)</td>
<td>Repeated measures design</td>
<td>22; F</td>
<td>College students</td>
<td>Exercise group Non exercise group</td>
<td>Isometric MVC of elbow flexors</td>
<td>↔</td>
</tr>
</tbody>
</table>

M = Male; F = Female; ISPC = Intermittent Sequential Pneumatic Compression; CON = Control; 5kmTT = 5 kilometer time trial; 4minTT = 4 minute time trial; SJ = Squat jump; CMJ = Counter movement jump; MVC = Maximum voluntary contractions; ↑ indicates a significant improvement in performance from intermittent sequential pneumatic compression use; ↔ indicates a non significant change in performance from intermittent sequential pneumatic compression; - indicates effect size not reported

Perceptual measures

Researchers have also investigated ISPC’s effect on perceptual measures of recovery and pain. Perceptual methods that have been included in the literature include total quality recovery scales, perceived recovery scales, Likert scales, and 100mm visual analog scales (see table 2).
O'Donnell and Driller (2015) also examined perceptual measures in their study on ISPC’s effect on recovery in well-trained triathletes. A total quality recovery scale, which ranged from 6 (very, very poor recovery) to 20 (very, very good recovery), was used to rate the participants perceived level of recovery following 40 minutes of intervals on a cycle ergometer. Participants completed the scale at 10, 20, and 30 minutes into a 30-minute recovery period where an ISPC recovery protocol or passive recovery protocol was implemented. The authors found no statistically significant differences (p > 0.05) between the use of an ISPC device and the control trial with an unclear effect between trials for perceived recovery.

Perceptual measures of recovery were also examined in the previously discussed Overmayer and Driller (2017) study. Like O'Donnell and Driller (2015), a total recovery scale was used to assess participants perceived level of recovery at 10, 20, and 30 minutes into their respective recovery protocols (ISPC or passive control). The authors found a small effect size (d = 0.27) but no significant two-way interaction between ISPC and control recovery conditions for pre recovery and post recovery total quality recovery scores (p = 0.7).

The aforementioned study by Northey et al. (2016) also included analysis of perceptual measures. Participants completed a perceived recovery status scale and a perceived soreness scale before exercise, immediately after exercise, one hour and 24 hours post exercise. Five minutes post recovery; participants completed a recovery protocol rating on a five point Likert scale. The authors reported no significant differences in perceived recovery status between conditions (ISPC, OCC, control) at any of the four time points.
Likewise there were no significant differences in perceived soreness between conditions at any time point (pre [p = 0.428], post [p = 0.735], 1 hour [p = 0.145], and 24 hours [p = 0.275]. Despite the lack of findings for both perceptual and performance measures in this study, participants ranked ISPC recovery significantly better than the control condition (ISPC mean rank = 1.58, p = 0.030; control mean rank = 2.50).

Winke and Williamson (2018) investigated the differences between an upper limb ISP device and a continuously worn upper limb compression sleeve. Participants included eight recreationally fit college students to measure perceived muscle soreness over a five-day recovery period following a muscle damaging protocol. Measurements were taken pre test, 24 hours post test and for the following four days. Participants completed each recovery protocol with at least one week separating trials. Subjective ratings of muscle soreness were higher in the compression garment group during both elbow flexion (p = 0.015) and elbow extension (p = 0.005). A significant limitation of this study was that they had no control condition for comparison.
Table 2. The effect of ISPC on perceptual measures

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Sample size</th>
<th>Participant description</th>
<th>Groups/interventions</th>
<th>Measure</th>
<th>Level of significance and effect size of findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>O’Donnell and Driller (2015)</td>
<td>Randomized crossover design</td>
<td>10; M</td>
<td>Well trained triathletes</td>
<td>ISPC – 30 min at 80mmHg</td>
<td>TQR</td>
<td>↔ Unclear</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CON – 30 min seated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overmayer and Driller (2017)</td>
<td>Counterbalanced crossover design</td>
<td>13; M, 8; F</td>
<td>Trained cyclists</td>
<td>ISPC – 30 min at 80mmHg</td>
<td>TQR</td>
<td>↔ Small</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CON – 30 min seated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Northey et al. (2016)</td>
<td>Randomized crossover design</td>
<td>12; M</td>
<td>Strength trained</td>
<td>ISPC - 45min at 80mmHg</td>
<td>PRS, PSS</td>
<td>↔</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>OCC – 12min alternating between legs every 3min at 220mmHg</td>
<td>PSS Likert scale</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CON – 45min supine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Winke and Williamson (2018)</td>
<td>Randomized crossover design</td>
<td>4; M, 4; F</td>
<td>College students</td>
<td>ISPC - 20 min at 100mmHg 24 hours post exercise + following 4 days CS – worn continuously</td>
<td>100mm visual analog scale</td>
<td>↑ Small</td>
</tr>
</tbody>
</table>

M = Male; F = Female; ISPC = Intermittent Sequential Pneumatic Compression; CON = Control; OCC = Vascular Occlusion; TQR = Total Quality recovery scale; PRS = Perceived Recovery Scale; PSS = Perceived soreness scale; ↑ indicates a significant improvement in performance from intermittent sequential pneumatic compression use; ↔ indicates a non significant change in perceptual measures from intermittent sequential pneumatic compression; - indicates effect size not reported

**Mechanistic measures**

Studies have also investigated the mechanisms of ISPC. These are important to consider as they provide context and a deeper understanding of the performance and perceptual findings. The primary mechanistic measure used in existing literature was blood lactate levels, however there were also measures of pyruvate, ammonia, hydrogen, bicarbonate, and heart rate (see table 3).

O’Donnell and Driller (2015) took lactate samples from participants following the aforementioned exercise protocol of 40-minutes of intervals on a cycle ergometer, and at the end of a 30 minute recovery period. They found a small ($d = 0.43$), but not
statistically significant ($p = 0.13$) change in blood lactate concentrations between trials in favor of ISPC.

Marcello et al. (2019) investigated heart rate and lactate levels during two 60-minute steady state cycle rides with a 30-minute ISPC or CON recovery intervention between rides. Participants performed the cycle rides at 60% of their functional threshold power with heart rates being collected at every 15-minute time point and lactate being measured at the 30-minute time point. They found no significant differences between conditions for heart rates but interestingly found a significant ($p = 0.041$) elevation in lactate for the ISPC treatment.

Hanson et al. (2013) investigated blood lactate concentrations prior to a one-minute sprint on a stationary bike, immediately after the sprint, and then 20 minutes after their recovery intervention (passive sitting, active recovery or ISPC). They found that ISPC significantly decreased blood lactate when compared to the passive recovery group ($p = 0.04$) however there was no significant differences between the ISPC group and the active recovery group ($p = 0.85$).

As previously discussed, Zelikovski et al. (1993) compared ISPC to a control protocol’s effect on recovery following an exhaustive exercise bout on a cycle ergometer. A chemical analysis of blood lactate, pyruvate, ammonia, pH, and bicarbonate was performed to analyse ISPC’s effect on removal of metabolites. Despite the authors reporting a significant increase in performance (maximal incremental cycle ergometer test to exhaustion), blood lactate, pH, bicarbonate, and ammonia were not significantly
different between the two recovery protocols. There were however higher levels of blood pyruvate during the control protocol than during the ISPC protocol (p < 0.05).

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Sample size</th>
<th>Participant description</th>
<th>Groups/interventions</th>
<th>Mechanism</th>
<th>Effect of ISPC on performance measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>O’Donnell and Driller (2015)</td>
<td>Randomized crossover</td>
<td>10; M</td>
<td>Well trained triathletes</td>
<td>ISPC – 30 min at</td>
<td>La</td>
<td>Small</td>
</tr>
<tr>
<td></td>
<td>design</td>
<td></td>
<td></td>
<td>80 mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CON – 30 min seated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marcella et al. (2019)</td>
<td>Randomized crossover</td>
<td>11; M, 1; F</td>
<td>Trained</td>
<td>ISPC – 30 min at</td>
<td>La</td>
<td>HR</td>
</tr>
<tr>
<td></td>
<td>design</td>
<td></td>
<td></td>
<td>80 mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CON – 30 min seated</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hanson et al. (2013)</td>
<td>Randomized controlled</td>
<td>21; F</td>
<td>Student athletes</td>
<td>ISPC - 20 min (pressure</td>
<td>La</td>
<td></td>
</tr>
<tr>
<td></td>
<td>clinical trial</td>
<td></td>
<td></td>
<td>unreported)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Active - 20 min on</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>cycle at 40% HRR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zelikovski et al. (1993)</td>
<td>Pre-post crossover</td>
<td>11; M</td>
<td>Healthy men</td>
<td>ISPC – 20 min at</td>
<td>La, Pyr, NH3, pH, HCO3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>design</td>
<td></td>
<td></td>
<td>50 mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CON – 20 min passive</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>rest</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

M = Male; F = Female; ISPC = Intermittent Sequential Pneumatic Compression; CON = Control; La = Lactate; HR = Heart Rate; Pyr = Pyruvate; NH3 = ammonia; pH = Potential hydrogen; HCO3 = bicarbonate † indicates a significant improvement in physiological measures from intermittent sequential pneumatic compression use; ↔ indicates a non significant change in performance from intermittent sequential pneumatic compression; - indicates effect size not reported

**Literature review conclusion**

Given the physical demands and competitiveness of most modern sports, recovery has become an integral component in the overall periodization and training schedule. This literature review briefly analysed some of the most commonly used acute recovery methods. Contradicting evidence was found for most techniques, including the use of CWI, CWT, and CG for recovery. In addition, there was limited support for hot water immersion or active recovery and further research is required before these can be
recommended as effective recovery modalities. Whole body cryotherapy chambers had some support but their level of efficacy currently does not appear to be any greater than less-expensive and more practical cryotherapy options.

When investigating ISPC’s effect on performance measures, only limited support was found with only two out of the six studies reviewed reporting positive findings (see table 1). Additionally limited support for ISPC use for recovery was found when investigating perceptual measures with only one from four studies finding a positive result from the use of ISPC (see table 2). Physiological markers of recovery from ISPC use were also inconclusive (see table 3). Most existing literature on ISPC’s effect on performance measures have focused on endurance. While these findings are applicable to sports where endurance is the main marker of performance, many sports require effective recovery of other physical measures in order to be successful. The current investigation will investigate ISPC’s effect on strength endurance, power, and maximal strength. These are important indicators of physical readiness in many sports and have been under-investigated at this time.

Existing literature has also mostly focused on ISPC’s effect on lower limbs. Only one paper (Winke & Williamson, 2018) was found that investigated ISPC use on the upper limb. Research on this topic is of great importance given the amount of sports where effective recovery strategies for the upper limbs is of concern (paddling, throwing, grappling, and climbing events), and the fact that several upper limb ISPC devices are already available on the market.
Chapter Two:

Investigating the use of an intermittent sequential pneumatic compression arm sleeve for recovery following exercise.

(Under review in the Journal of Strength and Conditioning Research)
Abstract

Objective: The current study aimed to investigate the efficacy of an intermittent sequential pneumatic compression (ISPC) device placed on the arm following a fatiguing upper-body exercise circuit. Methods: Fifty resistance-trained athletes (37 male/13 female, mean ± SD; age = 27 ± 24 yrs) performed three physical performance measures (grip strength dynamometer, single arm medicine ball throw, preacher bench bicep curls to failure) pre and post exercise, and following a 30-minute recovery period. During the recovery period, participants were randomly assigned an experimental arm, which was placed in the ISPC device, and a control arm (no device). Participants completed a perceptual muscle soreness rating of four upper-body landmarks at the same time points and also 24 hours post-recovery. Results: There was a statistically significant interaction between conditions for the single arm medicine ball throw (p < 0.01) following the recovery period, however the effect size was deemed trivial. There was a small, non-significant effect (d = 0.22) for the bicep curls in favour of ISPC. The perceptual muscle soreness scales resulted in significant differences between conditions immediately post and 24 hours post across all muscle groups (p < 0.05), all in favour of the ISPC condition. Conclusion: This study supports the use of an upper-body ISPC device to reduce muscle soreness for up to 24 hours following exercise, and may provide small benefits to strength-endurance when compared to a control trial.

Key words: fatigue, muscular performance, dynamic compression, delayed onset muscle soreness.
Introduction

The high training loads required to excel in most modern athletic sports has meant that the role of accelerating recovery has become an integral part of the training schedule (Argus et al., 2013; Coffey et al., 2004; O'Donnell & Driller, 2015). Enhancing recovery is important for athletic performance due to its ability to speed physiological regeneration, improve the quality and load of subsequent training sessions, as well as decreasing the risk of fatigue and injury (Argus et al., 2013; Bompa, 1994). Recovery is of utmost importance to competitive sports that require athletes to compete multiple times in a single day, as it has been shown that blood lactate (BLa) levels as low as 5 mmol.L⁻¹ may result in impaired subsequent performance in trained athletes (Bishop, Bonetti, & Dawson, 2001). This makes strategies that could potentially mitigate these negative effects of metabolic acidosis of particular concern to those where time to recover is limited.

Compression garments are thought to be one strategy that may influence recovery time by improving venous return, which in turn can assist in the removal of metabolic waste (Davies et al., 2009). However, the efficacy of compression garments remains equivocal. Hill et al. (2014) completed a meta-analysis of 12 compression recovery studies that included a total of 205 participants. The authors found the use of compression garments to have a moderate benefit in reducing delayed onset of muscle soreness (DOMS), recovery of muscle strength post exercise, recovery of muscle power following exercise and reducing concentrations of creatine kinase post exercise. MacRae et al. (2011) assessed 19 studies with a total of 273 participants to investigate the effect of wearing compression garments on indicators of recovery from exercise. From these studies it was concluded that wearing compression garments provides limited physiological or performance effects although reports of detrimental effects were rare. Some evidence
was found that compression garments may alter local blood flow, and protein or metabolite clearance, mitigate swelling and reduce perceived muscle soreness but findings were often isolated and inconclusive. The level of compression ranged from ~7mmHg to 33mmHg with seven studies not reporting the level of compression.

The use of intermittent sequential pneumatic compression (ISPC), have recently been explored as a potential tool for recovery, ISPC devices originated from the medical field where similar devices have been used in the treatment of lymphedema (Zelikovski et al., 1993) and other venous insufficiencies. A potential mechanism for ISPC devices efficacy is their mimicking of the anatomical muscle venous pump system through inflation of separate chambers in a distal to proximal sequential pattern (O'Donnell & Driller, 2015), perhaps further increasing blood-flow during recovery from exercise.

Research on the efficacy of ISPC devices in the sport setting remain inconclusive, with mixed findings (Airaksinen, 1989; O'Donnell & Driller, 2015; Overmayer & Driller, 2017; Wiener et al., 2001; Zelikovski et al., 1993). O'Donnell and Driller (2015) performed a study where 10 well-trained male triathletes completed 40-minutes of intervals on a cycle ergometer followed by 30 minutes of either passively sitting (control) or sitting with ISPC sleeves applied to the legs. Following the recovery period, participants completed a 5km run test on a treadmill. The authors found no statistically significant differences (p > 0.05) between the use of an ISPC device and the control trial for any of the measured variables, with a trivial effect (d = 0.07) on performance, an unclear effect between trials for perceived recovery and a small (d = 0.43), but not statistically significant (p = 0.13) change in blood lactate concentration between trials. Wiener et al. (2001) studied tibialis anterior recovery through the use of an ISPC device on the legs. Eight male participants performed a 12-minute exercise protocol aimed at fatiguing the tibialis anterior. The recovery protocol involved participants sitting with
one leg in an ISPC device using 80mmHg of pressure and the other acting as a control (no ISPC). Following the recovery protocol the ISPC leg resulted in a higher power output (judged by an electro-myography) of the tibialis anterior muscle than the control leg while performing a weight-lifting exercise. Overmayer and Driller (2017) assessed the efficacy of ISPC on 30-minutes of recovery in trained cyclists between a 20-minute and a four-minute performance test on a cycle ergometer. No statistically significant differences were found between ISPC and control conditions for any of the measured performance or perceived recovery variables (p < 0.05). Zelikovski et al. (1993) also investigated ISPC’s effect on exercise performance. Eleven male participants performed two maximal incremental exercise tests to exhaustion, separated by 20-minutes of ISPC use (leg sleeves) or a passive control. The authors reported a 45% prolongation of exercise duration from bout one to bout two for the ISPC condition when compared to the control. Northey et al. (2016) compared vascular occlusion, ISPC and a passive control on recovery following a resistance-training bout (10 sets of 10 repetitions of back squats). Participants completed perceptual (perceived recovery and muscle soreness) and performance (isokinetic dynamometer, squat jump height and countermovement jump height) measures before exercise, immediately after exercise, one hour and 24 hours post exercise. The authors found no evidence of the ISPC intervention improving any of the performance or perceptual measures.

While there is a growing body of contradictory literature on the use of ISPC devices for recovery following exercise in the lower body, there remains a paucity of research on the efficacy of ISPC devices for the upper-body. There are many sporting events that require expedited recovery of the upper-body for repeated performance within a short time frame. Therefore, the aim of the current study is to investigate the efficacy of using
ISPC arm sleeves for recovery following upper-body exercise on perceived muscle soreness and physical performance variables.

**Methods**

*Participants*

Fifty resistance-trained athletes volunteered to participate in the study (37 male, 13 female, mean ± SD = 27 ± 24 yrs, 171 ± 35 cm, 86 ± 83 kg). All participants were taking part across a range of sports at the time of the study and were required to be performing at least three resistance-training sessions per week for at least six months in order to participate in this study. All participants provided informed written consent before taking part in the study and ethical approval for this study was obtained from the institutions Human Research Ethics Committee.

*Design*

The current study implemented a pre-post single group design that utilized an experimental arm and a control arm. Participants were allocated experimental and control arms in a randomized, counter-balanced order. Participants completed performance and perceptual measures pre and post a fatiguing bout of resistance exercises and then again immediately following the recovery intervention (figure 1). Participants completed all testing within a 1.5 hour period. Participants were asked to refrain from completing upper-body exercise for 24 hours pre and 24 hours post the study to limit any influence on the performance or muscle soreness measure.
Warm up + familiarization
5 minutes of self-paced warm up and muscular activation plus familiarisation of testing procedures

Pre-test
1. Grip strength dynamometer
2. Single arm medicine ball throw
3. Preacher bench bicep curls
4. Bar hangs
5. Hand-grip crushers

Fatiguing exercise circuit
1. Reverse grip battle rope waves
2. Farmers carry medicine ball throw
3. Chin-ups
4. Bar hangs

Post-exercise test
1. Grip strength dynamometer
2. Single arm medicine ball throw
3. Preacher bench bicep curls

Recovery period (30 min)
ISPC device on one arm (ISPC), no device on other arm (CON).

Post-exercise test
1. Grip strength dynamometer
2. Single arm medicine ball throw
3. Preacher bench bicep curls

**Figure 1.** Experimental testing protocol. ISPC = Intermittent sequential pneumatic compression

**Procedures**

*Perceived muscle soreness*

Participants palpated their own muscle groups after being shown the correct palpation technique by the researcher. Four muscle groups were used to determine local muscle soreness in both right and left arms; triceps brachii longhead, biceps brachii, extensor digitorum and flexor carpi radialis (figure 2). All measures were taken while the arm was in a relaxed but extended position. Participants were instructed to provide a pain score ranging from 0-10 (0 = no soreness, 10 = severe/maximal soreness). This procedure was completed at the very start of the trial to provide a baseline pain measure, after the fatiguing exercise, following the recovery period and 24-hours post.
Figure 2. Anatomical landmarks for muscle palpation. a) triceps brachii longhead, b) biceps brachii, c) extensor digitorum, d) flexor carpi radialis

Performance measures

As a warm-up protocol, participants completed two minutes on an arm-grinder ergometer at 50% of perceived maximum intensity (n = 36) or 500 meters on a rowing ergometer at 50% of perceived maximum intensity (n = 14). This was selected by the participant based on personal preference and familiarity with the equipment. Participants then performed ten internal and external shoulder rotations per arm with a theraband, 10 bicep curls with the same theraband and ten-forearm supination/pronation’s per arm with a 3kg dumbbell in their hand. Participants were then familiarised with the grip strength dynamometer and were given an opportunity to practice using the device and making sure the trigger was in a suitable position for each individual’s hand size.

The current study included three performance tests. These included the grip strength dynamometer, single arm medicine ball throw, and maximum repetition single arm preacher bench bicep curls. The grip strength dynamometer was selected as a measure of strength, the single arm medicine ball throw was selected as a measure of upper-body power and the preacher bench bicep curls were selected to measure muscular strength-
endurance. Participants performed the performance tests at three different stages of the trial (pre, post-exercise and post-recovery – figure 1).

**Grip strength dynamometer**

As described in previous studies (Chen, Cui, Wu, & Xie, 2013; Gunawardhana & Silva, 2018; Sneade & Furnham, 2016) grip strength was tested using a grip strength dynamometer (CAMRY, electronic hand dynamometer, model EH101, China). Participants stood in an upright position with the arm bent at a 90-degree angle with the dynamometer screen facing away to blind participants from visual feedback of their scores, as it has previously been shown that visual feedback can produce significant increases in grip strength (Weinstock-Zlotnick, Bear-Lehman, & Yu, 2011). The participant squeezed the dynamometer with maximal effort over a period of 5 seconds. Participants alternated arms between trials and completed this process twice per arm, with 30-seconds rest between attempts. The hand-grip strength was measured in kilograms (kg).

**Single-arm medicine ball throw**

Participants sat with their back against a wall and their legs fully extended. The participant threw the medicine ball as far as they could with the distance being recorded using a measuring tape. Similar methods have been used previously to assess upper extremity power (Mayhew et al., 1993). This process was repeated twice per arm with the best throw of the two recorded. Males used an 8 kg medicine ball and females used a 4 kg medicine ball. Measurements were recorded in meters and rounded to the nearest centimeter.
**Preacher bench bicep curl**

Participants sat on the preacher curl bench with their armpit set at the apex of the bench. Participants started with their arm in a 50-degree angle of flexion. The researcher asked if the participant was ready and upon confirmation, started a metronome that was set to 30 bpm as has been used in previous studies (Hurley, Hatfield, & Riebe, 2013). On the first beep the participant would extend the arm until it reached a 160-degree angle of extension. Participants would hold this position until the next beep where the participant would contract the biceps brachii muscle until the arm was back in the fully flexed (50-degree) position. This process was repeated until muscular fatigue prevented completion of any more repetitions in time with the metronome. Participants would then rest for 30 seconds before switching arms and repeating the process on the other side. The researcher did not disclose how many repetitions the participant had completed. Males used a 12 kg dumbbell and females used a 6 kg dumbbell.

**Fatiguing Exercise Circuit**

The fatiguing exercise circuit consisted of five different exercises; reverse grip battle rope waves, a farmers carry, chin ups, bar hangs and hand-grip crushers. The battle rope waves were performed for one minute with as many repetitions as possible being performed in that one minute. The farmers carry was completed by walking twenty meters holding a dumbbell in each hand (20 kg females, 30 kg males). The supinated grip chin ups involved participants completing as many repetitions as they could before muscular fatigue prevented completion of another repetition. For participants who could not complete a single repetition, only the eccentric portion of the movement was completed. This was achieved by using a box to take away the concentric portion of the
movement. For the chin-up bar hangs, participants hung onto a chin up bar for as long as possible with their hands in a pronated grip. Once grip strength failed the participant would let go of the bar. For the hand-grip crusher exercise, participants squeezed the hand grip crushers (York Hand Grip – Strong, England) as many times as possible with one rep being performed per second until the participant felt no more repetitions could be completed.

Recovery protocol

Participants remained in a passive seated position with the ISPC arm sleeve device (RecoveryPump, LLC, USA) on their trial arm with it set at an approximate 70-degree angle (see figure 3). Their control arm was resting at a similar angle. The ISPC device has four chambers that inflate distally to proximally with an inflation time of 26 seconds and a deflation time of 15 seconds. It was set to a pressure of 80 mm Hg. The ISPC device covers the entire structure of the arm from the fingers to the pectoralis major (see figure 3). The ISPC device has an opening at the distal end that allows for the fingers to exit if the arm is longer than the ISPC device (84cm). The duration of the recovery protocol was 30- minutes.
**Statistical analysis**

Statistical analyses were performed using the Statistical Package for Social Science (SPSS 25.0 IBM Corp, Armonk, NY, USA). A two-way repeated measures ANOVA was used for the investigation of the effect of the two treatments (ISPC or CON) at different time points (pre exercise, post exercise, post recovery, 24 hours post). A Bonferroni adjustment was made if significant main effects were present. This was completed for all variables (four site muscle soreness scales, grip strength dynamometer single arm medicine ball throw, and preacher bench biceps curl). Analysis of the studentised residuals was verified visually with histograms and also by the Shapiro-Wilk test of normality. Statistical significance was set at $p < 0.05$ and all data are presented as means and standard deviation (±SD) unless stated otherwise. Additionally, effect size statistics were performed to determine differences between ISPC and CON groups across time points. For these measures the standardized change in mean between time points was calculated and expressed as standardised (Cohen’s $d$) effects. The magnitude of each effect size was interpreted using thresholds of 0.2, 0.6, 1.2 and 2.0 for small, moderate, large, and very large (Cohen, 1988). An effect size of $<0.2$ was considered trivial. Where the 90% confidence limits overlapped the thresholds for small positive and small negative values the effect was considered unclear (Batterham & Hopkins, 2006).
Results

Mean pressure (± SD) applied at each landmark by the ISPC device in a cohort of the study population (n=23), as identified using the Kikuhime pressure monitor was triceps brachii longhead = 76 ± 26 mmHg, biceps brachii = 80 ± 30 mmHg, extensor digitorum = 79 ± 62 mmHg, flexor carpi radialis = 78 ± 74 mmHg, with a total mean pressure of 78 ± 81 mmHg.

There were no significant differences at baseline between conditions for any of the measured variables (p > 0.05).

There was a statistically significant two-way interaction between treatment and time for single arm medicine ball throw (p < 0.01), with a significant difference between trials at the post recovery time point in favour of ISPC (3.30m ± 0.83m and 3.15m ± 0.73m, for ISPC and CON, respectively). There were no significant differences between conditions for any other performance measure despite preacher bench biceps curl displaying a small change in effect size. The other two performance variables displayed a trivial change in effect size (table 4).
Table 4: Comparison of ISPC and CON conditions at all time points. Data is presented as means ± SD.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post exercise</th>
<th>Post recovery</th>
<th>24 hours post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ISPC</td>
<td>CON</td>
<td>ISPC</td>
<td>CON</td>
</tr>
<tr>
<td>Grip strength dynamometer (kg)</td>
<td>49.7 ± 13.0</td>
<td>49.8 ± 12.4</td>
<td>40.1 ± 11.0</td>
<td>38.9 ± 10.1</td>
</tr>
<tr>
<td>Single arm medicine ball throw (m)</td>
<td>3.2 ± 0.8</td>
<td>3.2 ± 0.7</td>
<td>3.1 ± 0.8</td>
<td>3.1 ± 0.8</td>
</tr>
<tr>
<td>Preacher bench bicep curls (reps)</td>
<td>19.1 ± 8.5</td>
<td>21 ± 9.8</td>
<td>11.1 ± 4.3</td>
<td>11.8 ± 5.6</td>
</tr>
<tr>
<td>Triceps brachii longhead soreness (0-10)</td>
<td>1.9 ± 2.0</td>
<td>1.9 ± 1.9</td>
<td>3.0 ± 2.0</td>
<td>2.9 ± 2.2</td>
</tr>
<tr>
<td>Biceps brachii soreness (0-10)</td>
<td>1.9 ± 1.9</td>
<td>1.7 ± 1.7</td>
<td>3.3 ± 2.1</td>
<td>3.1 ± 2.2</td>
</tr>
<tr>
<td>Extensor digitorum soreness (0-10)</td>
<td>2.8 ± 2.0</td>
<td>2.6 ± 1.8</td>
<td>3.9 ± 2.2</td>
<td>3.7 ± 2.2</td>
</tr>
<tr>
<td>Flexor carpi radialis soreness (0-10)</td>
<td>1.4 ± 1.5</td>
<td>1.3 ± 1.4</td>
<td>2.7 ± 2.2</td>
<td>2.9 ± 2.3</td>
</tr>
</tbody>
</table>

Abbreviations: ISPC, Intermittent sequential pneumatic compression; CON, Control.

A significant two-way interaction between treatment and time was found for biceps brachii soreness, triceps brachii longhead soreness, extensor digitorum soreness, and flexor carpi radialis soreness (p < 0.05). A statistically significant interaction between ISPC and CON at the post recovery time point was found for biceps brachii soreness, triceps brachii longhead soreness, extensor digitorum soreness, and flexor carpi radialis soreness (p < 0.05). A statistically significant interaction between ISPC and CON was also found at the 24 hour post trial time point for biceps brachii soreness, triceps brachii longhead soreness, extensor digitorum soreness, and flexor carpi radialis soreness (p < 0.05).

Effect size analysis revealed small benefits to the ISPC intervention when comparing post exercise to post recovery for biceps brachii soreness (d = 0.22), triceps soreness (d
Comparison of 24 hours post to post exercise revealed small benefits for the ISPC intervention for the biceps brachii soreness ($d = -0.48$), triceps soreness ($d = -0.27$), and extensor digitorum soreness ($d = -0.32$). All other measures resulted in trivial effect sizes (table 5).

Table 5: Comparison of all measures (post-recovery and 24h post recovery) compared to post-exercise (pre-recovery) values. Data presented as raw difference in values (mean ±90% confidence intervals) with Cohen’s $d$ effect sizes (and 90% confidence intervals) for comparison between ISPC and CON conditions. * significant difference between trials (p < 0.05)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Post-exercise – Post-recovery</th>
<th>Post exercise – 24 hours post-recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\Delta$ ISPC – $\Delta$ CON</td>
<td>$\Delta$ ISPC – $\Delta$ CON</td>
</tr>
<tr>
<td>Grip strength dynamometer (kg)</td>
<td>$-1.0 \pm 0.9$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$-0.90 \pm 0.09$, Trivial</td>
<td></td>
</tr>
<tr>
<td>Single arm medicine ball throw (m)</td>
<td>$0.2 \pm 0.1^*$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$0.19 \pm 0.08$, Trivial</td>
<td></td>
</tr>
<tr>
<td>Preacher bench bicep curls (reps)</td>
<td>$1.1 \pm 1.0$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$0.22 \pm 0.21$, Small</td>
<td></td>
</tr>
<tr>
<td>Triceps brachii longhead soreness (0-10)</td>
<td>$-0.6 \pm 0.3^*$</td>
<td>$-0.6 \pm 0.4^*$</td>
</tr>
<tr>
<td></td>
<td>$-0.28 \pm 0.14$, Small</td>
<td>$-0.27 \pm 0.17$, Small</td>
</tr>
<tr>
<td>Biceps brachii soreness (0-10)</td>
<td>$-0.8 \pm 0.3^*$</td>
<td>$-1.0 \pm 0.3^*$</td>
</tr>
<tr>
<td></td>
<td>$-0.37 \pm 0.13$, Small</td>
<td>$-0.48 \pm 0.15$, Small</td>
</tr>
<tr>
<td>Extensor digitorum soreness (0-10)</td>
<td>$-0.6 \pm 0.4^*$</td>
<td>$-0.7 \pm 0.4^*$</td>
</tr>
<tr>
<td></td>
<td>$-0.24 \pm 0.17$, Small</td>
<td>$-0.32 \pm 0.20$, Small</td>
</tr>
<tr>
<td>Flexor carpi radialis soreness (0-10)</td>
<td>$-0.2 \pm 0.3^*$</td>
<td>$-0.2 \pm 0.3^*$</td>
</tr>
<tr>
<td></td>
<td>$-0.11 \pm 0.12$, Trivial</td>
<td>$-0.08 \pm 0.15$, Trivial</td>
</tr>
</tbody>
</table>

Abbreviations: ISPC, Intermittent sequential pneumatic compression; CON, Control.
Discussion

The current study showed trends towards performance benefits from use of an upper-body ISPC device. However, the performance measures that showed positive trends either had a significant but trivial effect size (single-arm medicine ball throw), or had a small but non-significant effect (preacher bench bicep curl). These findings show the need for further research before unequivocal conclusions can be made for upper-body ISPC efficacy for recovery of power and strength-endurance. Also supported by the current study is the use of an upper-body ISPC device to reduce perceived muscle soreness for up to 24 hours following exercise with localised upper-body soreness sites resulting in small, yet significant benefits.

In comparable research on an ISPC devices and muscular endurance, O'Donnell and Driller (2015) found no significant differences for any of the measured performance or physiological variables (5km performance on a treadmill, blood lactate concentration) following ISPC device application to the legs for 30 minutes. Overmayer and Driller (2017) also found no significant differences when comparing performance variables during a four-minute maximum cycling test on a cycle ergometer after using an ISPC device on the legs for 30 minutes. Alternatively, Zelikovski et al. (1993) found enhancements of measures of muscular endurance through a maximal incremental exercise test and Wiener et al. (2001) found enhancements of muscular endurance via electro-myography on the tibialis anterior muscle during a sustained weight lifting protocol. Although it is difficult to make conclusions based on the fact that the current study was primarily assessing strength-endurance whereas O'Donnell and Driller (2015), and Overmayer and Driller (2017) focused more on aerobic endurance, comparisons can still be made as metabolic acidosis in the muscle may be a limiting factor for
performance in the tests used (Overmayer & Driller, 2017; Wiener et al., 2001). Of note is the training status of the participants in these studies. The two studies that found no significant differences used trained to well-trained athletes whereas the two studies that found differences used “healthy” men. It has been shown that trained athletes have quicker recovery rates between exercise bouts than those who are lesser trained (O'Donnell & Driller, 2015). Therefore, it is possible that when the participants are well-trained, there is less to recover from, leaving less potential benefit when using any recovery intervention. It is possible that this factored into the results of the current study, as participants were trained but not necessarily well-trained which may be why only small trends to improved endurance performance were found.

The current studies findings on strength are in agreement with those of Northey et al. (2016). Both studies used muscle dynamometry devices to measure isometric force produced by the forearms and quadriceps respectively. Both studies returned non significant (p > 0.05) findings for use of an ISPC device as a means to enhance recovery of strength. However Northey et al. (2016) returned conflicting findings to the current studies findings on power. Northey et al. (2016) used squat jump and countermovement jump heights to assess power and found no significant changes between conditions, whereas this study found a significant (p < 0.01) interaction for ISPC use to improve upper-body power. There are several possible mechanisms that could contribute to these findings such as an increased range of motion in the joint, increased skin and intramuscular temperature, and increased blood flow (Weerapong et al., 2005), but all remain speculative without further investigation. An increased range of motion of the shoulder joint may have contributed to improvements in the single arm medicine ball throw. There is evidence that elevated skin and intramuscular temperature may provide performance benefits to measures of power (Mohr et al., 2004). While temperature
differences between arms was not recorded in the current study, the combination of the massaging effect of the ISPC device and the encasing of the arm in the sleeve likely elevated the temperature of the ISPC arm in comparison to the CON arm which remained passive for 30 minutes.

Whilst the current study did not analyze the specific mechanisms involved in ISPC recovery, we can speculate based off findings from previous research. ISPC has been suggested to increase venous blood flow and venous return and therefore accelerate the removal of metabolites from the muscles (Zelikovski et al., 1993). A common way to assess this is to evaluate changes in blood lactate concentration at different time points. O'Donnell and Driller (2015) found a small ($d = 0.43$), but not statistically significant ($p = 0.13$) change in blood lactate concentrations between experimental and control trials.

The build-up of metabolic waste may be a limiting factor for muscular endurance (Hogan & Welch, 1984), therefore these findings suggest a link between ISPC removing metabolites from the muscle and performance improvements in activities that require muscular endurance. However Zelikovski et al. (1993) found limited mechanistic justification (through measurement of BLa, bicarbonate, pH, ammonia, and pyruvate) for their findings despite finding a 45% performance increase between ISPC and CON, indicating future research is required.

The current study reported significant findings for perceptual levels of muscle soreness for up to 24 hours post recovery. This may be due to the proposed ability of ISPC to increase local blood and lymph flow via the sequential compression of the targeted muscles. An increase in local blood flow may accelerate the removal of metabolic waste that could otherwise accumulate in the muscle (Overmayer & Driller, 2017). Other possible mechanisms for the muscle soreness findings is a decrease in oedema or
swelling through compression of the muscle (Weerapong et al., 2005). The current studies findings are in contrast to the previous research by Northey et al. (2016) where perceived recovery was not improved through the use of an ISPC device. Northey et al. (2016) used the same perceptual muscle soreness scale as the current study at one-hour post recovery intervention and 24 hours post study but no statistically significant changes were found at either time point. A potential reason for the differences in the Northey study and the current study could be due to the other interventions used. In the current study, there were only two interventions (ISPC and CON), whereas, in the Northey et al. study, there were three interventions (ISPC, vascular occlusion, CON). It is possible that when multiple interventions are tested, the placebo effect is reduced. The current study did not control for the possible placebo effect, therefore, this cannot be discounted when considering perceptual measures such as muscle soreness. Future studies should look at performing a study design that includes ISPC, CON and sham conditions to reduce the placebo effect and perform the conditions on both arms instead of just one. An additional consideration for future studies is to incorporate a more endurance-based measure that involves a greater portion of the upper-body rather than a single muscle group, for example a rowing ergometer test.
Chapter Three:

Practical applications, future directions, limitations, and conclusions
Practical applications

Based on the original study in Chapter two of this thesis, upper-body ISPC may be beneficial for use by those who are looking for quick recovery during upper-body power events. Sports such as track and field throwing events or sport climbing events may benefit from ISPC use between attempts to enhance recovery of upper-body power. Despite failing to reach statistical significance (p > 0.05), there is also evidence that upper-body ISPC had a small effect on muscular strength-endurance. These findings are applicable for sports that include muscular endurance elements such as climbing, grappling, or paddling. This study also found evidence that upper-body ISPC use reduced perceived muscle soreness for up to 24 hours post use, which would be applicable to any multi-day sporting event where upper-body muscle soreness may impair performance. Furthermore, the reduced muscle soreness associated with ISPC may influence athletes to participate more regularly in physical activity or training, as they do not feel as sore or as tired.

Future directions

This research may act as a reference for future investigation into ISPC. Future research might consider the following recommendations:

- Incorporating the use of a sham condition to control for the possible placebo effect.
- Apply the ISPC sleeves to both arms instead of just one arm.
- Perform a randomized crossover design study using ISPC, sham, and control conditions.
• Compare the use of ISPC with other common recovery methodologies such as compression garments and cold-water immersion.

• Incorporate some mechanistic measures such as creatine kinase to indicate muscle damage, or blood flow measures using ultrasound/strain gauge plethysmography to identify any differences in venous return.

• Comparison of different levels of pressure exerted by the ISPC device (e.g. 20, 40, 60, 80, 100 mmHg) for a range of physical and physiological measures would be an important area for future studies.

• Incorporate a more endurance-based measure that involves a greater portion of the upper-body rather than a single muscle group, for example a rowing ergometer or arm-grinder test.

Limitations

The study contained in this thesis (chapter 2) has several design limitations. Firstly, it failed to provide a sham/placebo condition. However, a valid placebo condition is very difficult to incorporate into a study of an external device where participants are very kinesthetically aware of what condition they are undertaking. Even with a device that produces lower levels of pressure would make it easy to differentiate for participants, and indeed, may still exert a physiological effect. Perhaps a placebo condition not related to the intervention (e.g. a “recovery drink”) would be more appropriate. There was a lack of valid and objective measures of mechanisms of action in our original study. The current study design used one arm as the study condition and the other as the control condition. A valid measure would be required to indicate mechanisms of action local to the test arm as opposed to a systemic mechanism. Two common tests used to assess muscle damage are creatine kinase and lactate levels in the blood, however these
are both indicators of systemic damage. In order to get valid and objective measures of muscle damage, an alternative study design would have had to be implemented where both arms were used in a crossover study design. However a crossover design would likely limit the amount of participants, as multiple sessions would be required. A mechanism that could have been investigated in the current study design is blood flow. Unlike creatine kinase or lactate samples that indicate systemic damage, blood flow can be assessed regionally and could therefore be used to investigate the difference ISPC use had on regional blood flow between the two arms.

Conclusion

Chapter 1 of this thesis aimed to provide a brief overview of existing literature that has investigated the mechanisms of fatigue and the acute strategies that can be used to adequately treat the symptoms of fatigue. Findings from this literature review returned contradicting evidence for most acute recovery strategies. Findings on CWI, CWT, and CG largely indicated these strategies may be beneficial recovery strategies whereas supporting literature for the use of hot water immersion or active recovery was scarce. Literature on whole body cryotherapy chambers were supportive of the strategy when compared to control trials, however, the current level of benefit does not seem to be any greater than less expensive methods of cryotherapy (e.g. water immersion). The study contained in this thesis (chapter 2) supports the use of an upper-body ISPC device for 30 minutes post-exercise to reduce muscle soreness for up to 24 hours. It also shows that an upper-body ISPC device provides significant benefits to upper-body power and small benefits to strength-endurance measures when compared to a control trial. While
future research is warranted, we would suggest that ISPC has the potential to benefit aspects of performance recovery in trained athletes.
References


Dawson, B., Gow, S., Modra, S., Bishop, D., & Stewart, G. (2005). Effects of immediate post-game recovery procedures on muscle soreness, power and flexibility levels over the next 48 hours. *Journal of Science and Medicine in Sport, 8*(2), 210-221.


temperature and sprint performance during soccer matches – beneficial effect of

Montgomery, P. G., Pyne, D. B., Hopkins, W. G., Dorman, J. C., Cook, K., & Minahan,
C. L. (2008). The effect of recovery strategies on physical performance and
cumulative fatigue in competitive basketball. *Journal of Sports Sciences, 26*(11),
1135-1145.

Morris, R. J., & Woodcock, J. P. (2002). Effects of supine intermittent compression on
arterial inflow to the lower limb. *Archives of Surgery, 137*(11), 1269-1273.

Morris, R. J., & Woodcock, J. P. (2010). Intermittent pneumatic compression or
graduated compression stockings for deep vein thrombosis prophylaxis?: A
systematic review of direct clinical comparisons. *Annals of surgery, 251*(3),
393-396.

Medicine, 33*(1), 1-11.

Vascular occlusion and sequential compression for recovery after resistance

compression on recovery between exercise bouts in well-trained triathletes.
*Journal of Science and Cycling, 4*(3), 19.

review on understanding sleep for elite athletes. *Nature and Science of Sleep, 10*,
243.


Appendices
Appendix 1 – ethics approval

15-8-2018

Adam Cranston
By email: adamcranston@ymail.com

Dear Adam

UoW HREC(Health) 2018#57: Investigating the use of an intermittent sequential pneumatic compression arm sleeve for recovery following exercise

Thank you for submitting your amended application HREC(Health) 2018#57 for ethical approval.

We are now pleased to provide formal approval for your project within the parameters outlined within your application.

Please contact the committee by email (humanethics@waikato.ac.nz) if you wish to make changes to your project as it unfolds, quoting your application number with your future correspondence. Any minor changes or additions to the approved research activities can be handled outside the monthly application cycle.

We wish you all the best with your research.

Regards,

[Signature]

Karsten Zegwaard PhD
Acting Chairperson
University of Waikato Human Research Ethics Committee