The Effects of Cold Water Immersion on Recovery Following the Demands Associated with Team-Sport Exercise

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ABSTRACT

The high-intensity, physically demanding nature of team-sport exercise invokes physiological perturbations often resulting in short- and long-term reductions in muscle function and increased symptoms of exercise-induced muscle damage (EIMD). When such exercise bouts are performed in hot environmental conditions and/or include exposure to intense physical collisions between opposing players, the physiological load of exercise can be exacerbated. Consequently, post-exercise recovery strategies including cold water immersion (CWI) are commonly implemented in order to minimise the deleterious symptoms associated with team-sport exercise, and optimise the quality of subsequent performance. Despite the increased popularity of CWI, minimal research has focused on the benefits following team-sport exercise and more specifically, there remains a paucity of research evaluating the subsequent effects of cold therapy on the recovery of neuromuscular function following the demands associated with team-sport exercise. Therefore, the aim of this thesis was to examine the effects of post-exercise cold therapy on the recovery of neuromuscular function following a variety of exercise conditions related to team-sport activity.

The initial investigation aimed to isolate skeletal muscle damage, commonly elicited during team-sport exercise, in a single-leg model to identify the specific effects of repeated eccentric muscle actions on neuromuscular function in the lower body (quadriceps) and the subsequent effects of post-exercise cold therapy (COLD). Ten resistance-trained males performed 6 x 25 maximal concentric/eccentric muscle contractions of the knee extensors (KE) followed by a 20-min recovery (COLD v control). Neuromuscular function (voluntary and evoked), together with perceived muscle soreness (MS) and pain, and blood markers for muscle damage were measured pre- and post-exercise, and immediately post-recovery, 2-h, 24-h and 48-h post-recovery. The results indicated that despite a post-exercise suppression in voluntary torque, a recovery of COLD did not significantly enhance maximal voluntary...
torque (MVC) and activation (VA) \((P>0.05)\). Further, post-exercise elevations in creatine kinase (CK), aspartate aminotransferase (AST) and c-reactive protein (CRP) were not significantly altered by COLD \((P>0.05)\); although, perceptions of pain were lower 48-h post-recovery following COLD compared to CONT. Therefore, when explicit eccentric muscle actions, isolated to a single-leg, elicited prolonged muscle damage, post-exercise COLD did not hasten the recovery of skeletal muscle function despite enhancing perceptions of pain.

Study two examined the effects of CWI following simulated team-sport exercise in the heat. Two sessions of a 2 x 30-min intermittent-sprint protocol (ISE) in 32°C and 52% humidity were performed, followed by a 20-min CWI or passive recovery (CONT). Neuromuscular function, perceived MS, CK, AST and CRP were measured pre- and post-exercise, and immediately post-recovery, 2-h and 24-h post-recovery; whilst core temperature \((T_{\text{core}})\), heart rate (HR), perceptions of exertion, thermal strain and thirst were recorded pre-during and post-exercise. MVC and VA were reduced post-exercise in both conditions and remained suppressed for the 24-h recovery period \((P<0.05)\), whilst CK, AST and CRP were elevated above pre-exercise values \((P<0.05)\). Implementation of post-exercise CWI produced a more rapid rate of reduction in \(T_{\text{core}},\) HR and MS, whilst increasing post-recovery MVC, VA and root mean square (RMS) of the electromyogram (EMG) signal \((P<0.05)\). In contrast, MVC and RMS measured at 24-h post-recovery were significantly higher in CONT compared to CWI \((P=0.05)\). Therefore, the results of study two indicated that following exercise in the heat, CWI accelerated the reduction in thermal and cardiovascular load, and improved acute recovery of MVC alongside increased central activation. However, the suppressed MVC 24-h post-recovery in CWI compared to CONT may suggest a detrimental prolonged effect of CWI after multi-joint exercise in the heat.

In the final investigation, the effects of CWI were examined following simulated collision-sport exercise. Three sessions consisting of a 2 x 30-min ISE with either tackling...
(T) or no tackling (CONT) were followed by a 20-min CWI intervention (TCWI) or passive recovery (TPASS or CONT). Every 6th min during the ISE, participants performed 5 x 10-m runs, receiving a shoulder-led tackle to the lower-body. Sprint time and distance covered during ISE were recorded, with MVC, VA, RMS, MS, CK, AST and CRP measured pre- and post-exercise, post-recovery, 2-h and 24-h post-recovery. Total distance covered during exercise was significantly greater in CONT (P=0.01), without differences between TPASS and TCWI (P>0.05). A recovery with TCWI resulted in immediate improvements in post-recovery MVC, VA and RMS (P<0.05); whilst M-wave amplitude and peak twitch were also significantly increased post-recovery and 2-h post-recovery, respectively in TCWI (P<0.05).

Although TCWI reduced perceptions of MS 2-h post-recovery, no significant effect on the elevation in CK, AST and CRP were observed (P>0.05). Accordingly, the results of the final study highlight that inclusion of body-collisions reduces exercise performance, while the use of CWI results in a faster recovery of MVC, VA and RMS and improves muscle contractile properties and perceptions of soreness following collision-based exercise.

In conclusion, the collective results of the three studies of the present thesis demonstrate that a single application of cold therapy is of no significant benefit to restore the reduction in skeletal muscle function following (single-joint) exercise inducing prolonged, localised musculoskeletal damage and trauma. However, when high-intensity, multi-joint, intermittent-sprint exercise includes exogenous load (heat and intense body collisions), post-exercise CWI is beneficial in attenuating the decline in acute voluntary force production. Specifically, it is likely that such improvements in MVC may be via ameliorated recovery of peripheral contractile apparatus and reduced internal thermal load, increasing central activation and skeletal muscle recruitment. Therefore, the benefits of cold therapy recovery may be dependent on the modality of exercise to recover from and with suppression in 24-h
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<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACSM</td>
<td>American College of Sports Medicine</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
</tr>
<tr>
<td>AST</td>
<td>Aspartate Aminotransferase</td>
</tr>
<tr>
<td>BF</td>
<td>Biceps Femoris</td>
</tr>
<tr>
<td>CD</td>
<td>Contraction Duration</td>
</tr>
<tr>
<td>CK</td>
<td>Creatine Kinase</td>
</tr>
<tr>
<td>CO₂</td>
<td>Carbon Dioxide</td>
</tr>
<tr>
<td>COLD</td>
<td>Cold Therapy</td>
</tr>
<tr>
<td>CON</td>
<td>Concentric</td>
</tr>
<tr>
<td>CONT</td>
<td>Control</td>
</tr>
<tr>
<td>CRP</td>
<td>C-reactive Protein</td>
</tr>
<tr>
<td>CV</td>
<td>Co-efficient of Variation</td>
</tr>
<tr>
<td>CWI</td>
<td>Cold Water Immersion</td>
</tr>
<tr>
<td>DOMS</td>
<td>Delayed Onset of Muscle Soreness</td>
</tr>
<tr>
<td>ECC</td>
<td>Eccentric</td>
</tr>
<tr>
<td>EIMD</td>
<td>Exercise Induced Muscle Damage</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyogram</td>
</tr>
<tr>
<td>GXT</td>
<td>Graded Exercise Test</td>
</tr>
<tr>
<td>HCO₃</td>
<td>Bicarbonate</td>
</tr>
<tr>
<td>HR</td>
<td>Heart Rate</td>
</tr>
<tr>
<td>ISE</td>
<td>Intermittent Sprint Exercise</td>
</tr>
<tr>
<td>ITT</td>
<td>Interpolated Twitch Technique</td>
</tr>
<tr>
<td>KE</td>
<td>Knee Extensor</td>
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<tr>
<td>La⁻</td>
<td>Lactate</td>
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<tr>
<td>Abbreviation</td>
<td>Description</td>
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<tr>
<td>--------------</td>
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</tr>
<tr>
<td>MS</td>
<td>Muscle Soreness</td>
</tr>
<tr>
<td>MVC</td>
<td>Maximal Voluntary Contraction</td>
</tr>
<tr>
<td>O$_2$</td>
<td>Oxygen</td>
</tr>
<tr>
<td>PAIN</td>
<td>Rating of Perception of Pain Threshold</td>
</tr>
<tr>
<td>Pt</td>
<td>Peak Twitch</td>
</tr>
<tr>
<td>RBE</td>
<td>Repeated Bout Effect</td>
</tr>
<tr>
<td>RMS</td>
<td>Root Mean Square</td>
</tr>
<tr>
<td>RPE</td>
<td>Rating of Perceived Exertion</td>
</tr>
<tr>
<td>RR</td>
<td>Rate of Relaxation</td>
</tr>
<tr>
<td>RTD</td>
<td>Rate of Torque Development</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for Social Sciences</td>
</tr>
<tr>
<td>T$_{core}$</td>
<td>Core Temperature</td>
</tr>
<tr>
<td>TCWI</td>
<td>Tackling and Cold Water Immersion</td>
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<tr>
<td>TCONT</td>
<td>Tackling and Passive Recovery</td>
</tr>
<tr>
<td>TPt</td>
<td>Time to Peak Torque</td>
</tr>
<tr>
<td>VA</td>
<td>Voluntary Activation</td>
</tr>
<tr>
<td>VL</td>
<td>Vastus Lateralis</td>
</tr>
<tr>
<td>VM</td>
<td>Vastus Medialis</td>
</tr>
<tr>
<td>VO$_2$</td>
<td>Volume of Oxygen</td>
</tr>
<tr>
<td>VO$<em>2$$</em>{max}$</td>
<td>Maximal Volume of Oxygen</td>
</tr>
<tr>
<td>$\frac{1}{2}$ RT</td>
<td>Half Relaxation Time</td>
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LIST OF SYMBOLS AND UNITS

± Plus or Minus
° Degrees
% Percentage
°C Degrees Celsius
cm Centimetre
Db Decibel
h Hour
Hz Hertz
kg Kilogram
m Metre
min Minute
mL Millilitre
mmol⁻¹ Millimol
ms Millisecond
mV Millivolt
N m⁻¹ Newton Metre
µL Micro Litre
s Second
W Watt
y Year
Certificate of Authorship

I, Monique Pointon, hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma at Charles Sturt University or any other educational institution, except where due acknowledgment is made in the thesis entitled **The Effects of Cold Water Immersion on Recovery Following the Demands Associated with Team-Sports Exercise**. Any contribution made to the research by colleagues with whom I have worked at Charles Sturt University or elsewhere during my candidature is fully acknowledged.

I agree that this thesis be accessible for the purpose of study and research in accordance with the normal conditions established by the Executive Director, Library Services or nominee, for the care, loan and reproduction of theses.”

* Signature: [Signature]  Date: [Date]

* Subject to confidentiality provisions as approved by the University.
CHAPTER 1

Introduction
Overview

Athletic training for competitive sport represents acute challenges intended to optimise prolonged improvements in performance and physiological function (Dawson et al. 2004). Regular bouts of intense training and competition often result in short- and long-term alterations in the function of the musculoskeletal, nervous, neuromuscular and metabolic systems (Reilly and Ekblom 2005; Westerblad et al. 1998). Short-term alterations may result from metabolic disturbances following higher-intensity exercise or exercise of high physiological demands (Westerblad et al. 2002). Alternatively, longer-lasting alterations may be related to exercise-induced muscle damage (EIMD) and trauma, resulting in delayed-onset muscle soreness (DOMS) (Cheung et al. 2003). In addition, many sports competitions follow tournament schedules requiring athletes to compete in matches over successive days (Ronglan et al. 2006; Spencer et al. 2005). When such bouts are performed in close succession, time available for full physiological and/or performance recovery is often inadequate (Montgomery et al. 2008b; Rowsell et al. 2009). In an aim to restore physical function, minimise short- and long-term performance impairment and maximise training adaptations following intense exercise bouts, implementation of post-exercise recovery procedures are now an accepted practice in most sports (Barnett 2006).

In particular, cold water immersion (CWI) has emerged as a popular post-exercise recovery intervention (Banfi et al. 2009; Kinugasa and Kilding 2009; Wilcock et al. 2006). Despite increased popularity, and evidence suggesting that cold therapy is beneficial in the treatment of acute musculoskeletal injuries (Ho et al. 1995; Merrick et al. 1999; Paddon-Jones and Quigley 1997); the effect of cold therapy on recovery of exercise performance and skeletal muscle function remains varied (Eston and Peters 1999; Ingram et al. 2009; Isabell et al. 1992; Peiffer et al. 2009a). The reason for a disparity of findings may in part be the result
of the range of different exercise modes used in research and the limited knowledge on specific mechanisms thought to result in improved recovery following CWI (Banfi et al. 2007; Peiffer et al. 2010b; Vaile et al. 2008c). As such, with a lack of understanding into the precise mechanisms responsible for observed benefits of post-exercise CWI, it is difficult to determine the exercise conditions under which post-exercise CWI may be beneficial and appropriate.

Current evidence highlights mixed findings of cold therapy on post-exercise recovery following a range of exercise modes, including exercise invoking EIMD (Vaile et al. 2008c), laboratory cycling protocols (Peiffer et al. 2010b; Vaile et al. 2010) and team-sports exercise (Rowsell et al. 2009; 2011), respectively. With such mode-based responses in mind, Vaile et al. (2008c) recently reported that following exercise-induced DOMS, isometric strength and squat jump performance was improved with CWI. Improvement in the recovery of voluntary force and a reduction in perceived muscle soreness was also demonstrated following prolonged intermittent shuttle running which induced symptoms of muscle damage (Bailey et al. 2007). Conversely, Jakeman et al. (2009) reported that CWI following a damaging bout of exercise had no beneficial effect on recovery of maximal voluntary force (MVC) or perceived soreness. Of further interest, Sellwood et al. (2007) reported that ice-water immersion did not minimize markers of DOMS, including perception of pain and tenderness, with ratings of pain increased 24-h post-recovery. Accordingly, the efficacy of CWI to assist post-exercise recovery, particularly following exercise-induced DOMS, remains the topic of some debate (Bailey et al. 2007; Rowsell et al. 2011; Vaile et al. 2010).

To date few studies demonstrating beneficial effects of cold therapy have been able to relate the post-CWI improvement in performance or perceptual recovery to any physiological, immunological, hematological or neuromuscular mechanism (Halson et al. 2008). The predominant research focus has thus far unsuccessfully focused on the effect of
CWI to alter peripherally-oriented mechanisms (Halson et al. 2008; Peiffer et al. 2010a, b; Vaile et al. 2008b). However, data on the efficacy of cold therapy on central mechanisms more aligned with neuromuscular recovery are limited, (Peiffer et al. 2009a). More specifically, traditionally investigations have unsuccessfully attempted to find a relationship with the beneficial effects of CWI to alterations in metabolic and blood markers of muscle damage as an explanation for improved performance (Halson et al. 2008; Peiffer et al. 2009a; Peiffer et al. 2010a, b; Vaile et al. 2008b). Although previous evidence has focused primarily on the peripheral effects of CWI, it is well known that fatigue-induced declines in exercise performance may result from a reduction in skeletal muscle force generation (Gandevia et al. 1995b), that may be of either central and/or peripheral origin (Enoka and Stuart 1992; Hakkinen and Komi 1983). To date, a paucity of research exists examining CWI recovery on alterations in skeletal muscle recruitment and activation (Peiffer et al. 2009a). Specifically, the possible relationship between CWI and the subsequent effects on centrally-mediated mechanisms, including skeletal muscle recruitment has not been investigated.

More recently, the effect of post-exercise CWI on restoring impaired parasympathetic function following supramaximal exercise has been investigated (Buchheit et al. 2009). Evidence highlighted that post-exercise CWI influenced vagal-modulation resulting in an increase in parasympathetic activity, possibly indicating a potential feedback mechanism from the periphery (Buchheit et al. 2009). While this feedback pathway following CWI may be evident for the control of cardiac muscle, minimal research has examined the role of CWI on skeletal muscle recruitment. Specifically, if CWI results in altered peripheral responses resulting in feedback to the central nervous system, indicating an improved physiological state, such feedback may provide a mechanism to help explain improvements in ensuing exercise performance. Whilst Peiffer (2010a) and Vaile (2008a) recently postulated that the beneficial effects of CWI on subsequent exercise performance in the heat were related to a
faster reduction in thermal and cardiovascular strain; the relationship between improved peripheral recovery influencing centrally-mediated mechanisms, and the possible associated improvements in exercise performance are not fully understood (Peiffer et al. 2009a); with minimal research examining team-sports exercise.

Team-sport athletes are often exposed to high volumes of intermittent-sprint exercise during training and competition (Coutts et al. 2009; Spencer et al. 2004). Such demands often result in increased EIMD and contractile trauma due to the high intensity and eccentric strain of the exercise bouts (Mohr et al. 2003; Reilly 1997). As such, post-exercise cooling strategies aiming to minimise the symptoms associated with EIMD and improve recovery of muscle function to ensure that performance in subsequent exercise sessions is optimal, have become popular (Barnett 2006; Ingram et al. 2009). However, current evidence highlights equivocal findings on post-exercise cold therapy improving the signs and symptoms associated with EIMD (Jakeman et al. 2009; Vaile et al. 2008c); specifically, it is unknown as to whether EIMD or the physiological demands resulting from team-sport exercise, reducing ensuing skeletal muscle function, can be improved with post-exercise cold therapy.

In addition to these acute musculoskeletal demands, team-sport athletes are frequently exposed to additional exogenous factors increasing the physical, physiological and perceptual demand of the exercise bout. For example, team-sport athletes may be required to perform training or competition in hot environments, particularly during pre-season training, generally occurring during summer months for most football codes. Additionally, given that many competitive athletic events occur in warm environmental conditions (e.g. World Championships, Olympics (~32°C Athens, Greece 2004) and Commonwealth Games, and pre-season training), physiological perturbations associated with exercise are augmented in hot environments (Marino 2004; Tucker et al. 2004), and may also require additional recovery time (Wendt et al. 2007). Although recent evidence documents decreases in body
temperature with concomitant improvements in exercise performance following CWI (Peiffer et al. 2010a; Vaile et al. 2010), an area which has had minimal focus has been the role of CWI on cerebral function and/or centrally-mediated mechanisms influencing subsequent exercise performance in the heat (Peiffer et al. 2009a). Accordingly, the use of CWI to speed recovery of these elevated demands following exercise in the heat may be of benefit for ensuing performance for athletes competing in warm conditions.

Further, many team-sport athletes are also exposed to repeated direct and indirect body collisions in excess of the regular physical demands of the specific exercise bout (McLellan et al. 2010; Takarada 2003). Team-sports such as rugby league, rugby union, American football and Australian football, consist of intense physical contacts between opposing players during training and match-play. Direct body collisions may increase the physiological demand of high-intensity, intermittent-sprint exercise, with an additional increased risk of musculoskeletal injuries (Dawson et al. 2004; McLellan et al. 2011). Although to date there is a dearth of controlled research studies examining the effects of physical collisions on team-sport activity (Singh et al. 2011). Regardless, implementation of post-exercise CWI following team-sport exercise has become popular aiming to minimise deleterious symptoms associated with intense exercise and/or collision-based loads (Banfi et al. 2007). Despite such popularity, there is a paucity of research examining the effect of CWI on recovery following team-sport exercise involving high-impact physical collisions (Banfi et al. 2007).

Accordingly, the aim of this thesis was to examine the efficacy of cold therapy implemented as a recovery strategy following various exercise-induced physical loads that may be present during normal team-sport activities of training or competition. More specifically, this thesis will investigate the effects of cold therapy on the recovery of central and peripheral skeletal muscle function following specific EIMD exercise and consequently
intermittent-sprint exercise involving exogenous loads of heat and body collisions, respectively.

**Statement of the Problem**

As team-sport exercise may involve EIMD, be performed in the heat and/or involve direct body collisions resulting in immediate and prolonged decrements in muscle function; recovery from such loads may be of importance in maintaining optimal performance during ensuing exercise, often within short time frames (24 h). Whilst the body of literature on the effects of post-exercise cold therapy for recovery is growing, evidence outlining potential mechanisms for observed improvements remain equivocal (Peiffer et al. 2009a; Vaile et al. 2010). Moreover, although implementation of cold therapy has emerged as a popular post-exercise recovery intervention in many sports (Barnett 2006; Wilcock et al. 2006), the effect of cold therapy on restoring physical and neuromuscular function, and improving subsequent exercise performance highlights varied benefits following various modes of exercise (Rowsell et al. 2009; Vaile et al. 2008b; Vaile et al. 2010). Furthermore, few studies demonstrating beneficial effects of cold therapy have been able to relate improvements in subsequent performance to any physiological, haematological or neuromuscular mechanisms (Halson et al. 2008). More specifically, although several studies have reported significant alterations in peripherally-mediated mechanisms, few have examined the effect of cold therapy recovery on alterations in skeletal muscle recruitment and central activation (Peiffer et al. 2009a), particularly following team-sport exercise. Therefore, with current evidence highlighting varied benefits of cold therapy recovery on subsequent exercise performance (Ingram et al. 2009; Montgomery et al. 2008b; Yamane et al. 2006) and a paucity of research examining cold therapy effects on the recovery of central- and peripheral neuromuscular
function (Peiffer et al. 2009a), the present thesis aimed to further elucidate the effect of post-exercise cold therapy on the recovery of neuromuscular function following exercise simulating team-sport demands.

Aims and Rationale

Study One

EIMD resulting from eccentric strain during high-intensity, intermittent-sprint exercise results in immediate and prolonged reductions in skeletal muscle function (Evans et al. 1990; Ronglan et al. 2006). As a result, implementation of post-exercise cold therapy has become increasingly popular (Barnett 2006). Although cold therapy is a well-documented treatment for acute musculoskeletal injury (Bailey et al. 2007; Yanagisawa et al. 2003a), evidence outlining performance and perceptual effects of cold therapy following EIMD is varied (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009), with minimal evidence demonstrating the effects of cold therapy on the recovery of neuromuscular function (Peiffer et al. 2009a). Therefore, the aim of the initial investigation was to determine the effects of cold therapy on the recovery of skeletal muscle function following high-velocity, single-joint eccentric exercise designed to result in EIMD, for which the signs and symptoms may be similar following team-sport training and match-play.

Study Two

It is well-established that exercise-induced increases in thermal strain result in alterations in central activation (Martin et al. 2004; Nybo and Nielsen 2001), muscle contractile function (Morrison et al. 2004) and exercise performance. Many sports involve repeated bouts of exercise over consecutive days, and when such events are performed in
warm environmental conditions performance decrements are more pronounced (Vaile et al. 2008a). In an effort to alleviate potential perturbations associated with exercise-induced increases in endogenous thermal load, implementation of CWI has become an increasingly popular post-exercise recovery strategy (Peiffer et al. 2010b; Vaile et al. 2008a). Recent investigations have reported improved recovery of thermal and cardiovascular strain, alongside concomitant improvements in subsequent exercise performance with CWI following exercise-induced increases in thermal load (Vaile et al. 2010; Wilcock et al. 2006; Yeargin et al. 2006). However, an area which has had minimal research focus and may explain the aforementioned results is the effect CWI has on the recovery of the peripheral and central mechanisms as related to contractile muscle function (Peiffer et al. 2009a). Evidence for the potential benefit for such an intervention is the observed faster return of maximal voluntary force and voluntary activation when external cooling returns core temperature ($T_{core}$) closer to pre-exercise values following passively-induced hyperthermia (Morrison et al. 2004). Therefore, the second study aimed to examine the effects of CWI following simulated team-sport exercise in the heat on the recovery of neuromuscular function; specifically central and peripherally-mediated mechanisms of skeletal muscle recruitment.

**Study Three**

Team-sports are characterized by intermittent bouts of high and low intensity activity (Meir et al. 1993), with many sports also involving regular physical collisions (n=20-40) throughout the course of training and/or match-play (Brewer and Davis 1995; Gissane et al. 2001). The combative nature of such sports, combining intermittent high-intensity activity and repeated blunt force trauma, may result in damage to skeletal muscle and post-exercise muscle soreness (Dawson et al. 2004; McLellan et al. 2011; Peake et al. 2005a), adversely affecting subsequent exercise performance (Ronglan et al. 2006). Despite the popularity of
post-exercise cold therapy, and although recent findings following team-sport exercise indicate potential benefits (Ingram et al. 2009; Rowsell et al. 2009), there remains a paucity of evidence examining the effects of direct body collisions during exercise on performance and the subsequent effect CWI has on recovery of physical function (Banfi et al. 2007). Accordingly, the primary aim of the final investigation was to examine the effects of CWI on recovery of skeletal muscle, physiological and perceptual functions following simulated, collision-based team-sport exercise. A secondary aim of this investigation was to quantify the effect of direct lower-body collisions (tackles) on ensuing intermittent-sprint performance.

**Research Questions and Hypotheses**

**Research Questions - Study One**

1. Does implementation of post-exercise cold therapy following lower-body single-joint, eccentric exercise improve the signs and symptoms of EIMD?

2. Does post-exercise cold therapy alter the recovery profile of neuromuscular function following peripheral contractile damage induced from high-velocity, single-joint eccentric exercise?

**Hypothesis - Study One**

As cold therapy is reported to reduce acute inflammatory responses (Bailey et al. 2007; Eston and Peters 1999), it was hypothesised that cold therapy implemented following a bout of single-joint, high-velocity eccentric exercise would attenuate the decline in skeletal muscle function and reduce deleterious signs and symptoms of EIMD.
Research Questions - Study Two

1. What are the effects of post-exercise CWI on neuromuscular, physiological and perceptual function following high-intensity intermittent-sprint exercise in the heat, and how does this affect acute and prolonged recovery of voluntary force production?

Hypothesis - Study Two

With the well documented restoration of maximal voluntary force production and activation when external cooling returns core temperature (T_{core}) closer to pre-exercise values following passively-induced hyperthermia (Morrison et al. 2004), it was hypothesised that CWI following a bout of intermittent-sprint exercise in the heat would enhance the rate of reduction in core temperature and thus ameliorate the decline in maximal voluntary force via improved recovery of voluntary activation and skeletal muscle recruitment resulting in improved performance.

Research Questions - Study Three

1. Does the inclusion of simulated direct physical collisions (tackles) affect prolonged, intermittent-sprint performance compared to a non-contact control?

2. What are the effects of post-exercise CWI on neuromuscular, physiological and perceptual function following the simulated demands of collision-based team-sport exercise, and how does this affect acute and prolonged recovery of voluntary force production?
Hypotheses - Study Three

It was hypothesised that implementation of direct physical collisions would result in a significant decrement in intermittent-sprint exercise performance and produce significant declines in neuromuscular function. Further, it was hypothesised that implementation of post-exercise CWI would attenuate the declines in skeletal muscle function while improving physiological and perceptual recovery.

Limitations

The limitations of this research include:

- Due to the subject population consisting of amateur male resistance-trained and team-sport athletes aged 18 – 23 y; the results can not necessarily be extrapolated to other populations such as the aged, youth, females or elite athletes.

- Participants will be required to complete food and activity diaries and replicate this for each testing session. Although the investigators will encourage research participants to keep this information accurate and up to date, this variable may not be completely controlled.

- Muscle mass, subcutaneous fat and skin thickness may impede the electrical stimulation data recorded throughout each investigation. Furthermore, the efficacy of cold therapy may also be impeded by these limitations.

- The third study will aim to replicate the intense nature and demands of team sports consisting of regular bouts of intense direct physical collisions; however, this aspect may possibly be limited by ethical constraints and an inability to enforce the intensity of collisions normally observed during on-field match-play. Throughout each exercise protocol, research participants will receive encouragement to perform at their
maximal, although complete control over the force produced during the tackling bouts may be difficult to impose.

- Transcranial magnetic stimulation noninvasively examines nerve propagation along the corticospinal tract, spinal roots, and peripheral nerves in humans allowing motor output to be mapped precisely (Hallet 2000). However, due to this equipment not being available at the time of data collection, the measurement of neuromuscular function in the current collection of studies is limited to peripheral nerve stimulation.

- Due to the recovery intervention consisting of cold water immersion or cold therapy the research studies were unable to implement a placebo control. As such, this may have some effect on the results of maximal voluntary activation and contraction, together with the perceptual ratings. The effect of this limitation will be discussed in the conclusion chapter.

**Delimitations**

The delimitations of the research include:

- The measurement of maximal isometric voluntary contractions (MVC) of the dominant leg knee extensors will be used as the main performance measure throughout each investigation. Although the measurement of MVC may not specifically relate to dynamic team sports performance, these measures will be implemented to provide reliable and valid data on neuromuscular function, both central and peripheral components. As such, this will limit the applicability to more dynamic activity, a common component of team-sports exercise. However, the determination of MVC will be implemented as it is the most reliable and valid measurement to examine the main aims of this thesis and determine the effects of exercise and cold therapy recovery on neuromuscular function. Measurements of voluntary activation, electromyography and twitch and motor evoked potential
properties are reliably determined via the measurement of MVC and as such this will be used as the main performance measure in all three investigations.

- Only male resistance-trained and team-sport athletes will be involved in each investigation. These research participants will be involved in 2-3 training session per week and compete in local rugby league/union and soccer teams.

- Participants will be required to avoid the consumption of food, alcohol and caffeine 3 h prior to each testing session.

- Participants will also be advised to avoid strenuous exercise 24 h prior to testing, and also during the designated recovery period (24 h or 48 h).

- Each investigation will be performed in an enclosed, controlled laboratory environment.

Note: To ensure consistency throughout the thesis, the formatting of information presented in Chapter’s 3, 4 and 5 have minor variations to that presented in the peer-reviewed publications due to different editorial requirements of specific journals.
CHAPTER 2

Literature Review
Overview

Athletes training for competitive sports, particularly at the elite level, are frequently exposed to demanding training and competition bouts (Dawson et al. 2002; Deutsch et al. 2007), potentially resulting in acute and prolonged reductions in athletic performance (Cheung et al. 2003; Ingram et al. 2009; Westerblad et al. 1998). In addition to repeated training bouts, many sports competitions follow a tournament schedule that can require athletes to compete in several matches over successive days (Ronglan et al. 2006; Spencer et al. 2005). When such exercise bouts are performed in close succession, time available for full physiological and performance recovery between sessions may be inadequate (Montgomery et al. 2008b; Rowsell et al. 2009) resulting in less than optimal ensuing performance (Gleeson et al. 1995; Häkkinen 1992). Specifically, a 2 - 7% decline in sprint and countermovement jump (CMJ) performance (Ronglan et al. 2006; Spencer et al. 2005), and increased markers of muscle damage and inflammatory cytokines (Montgomery et al. 2008a) have been observed during multi-day, team-sport competitive tournaments.

In an effort to restore physical function and enhance, or at least maintain, performance during subsequent exercise bouts (training and competition), post-exercise CWI has emerged as a popular recovery intervention (Banfi et al. 2009; Peiffer et al. 2010b; Vaile et al. 2010). Despite increased popularity, evidence demonstrating the efficacy of post-exercise cold therapy on attenuating declines in short- and long-term exercise performance, skeletal muscle function and altering cytokine responses are varied (Bailey et al. 2007; Halson et al. 2008; Ingram et al. 2009; Yamane et al. 2006). Further, the precise mechanisms responsible for observed improvements in recovery with cold therapy remain to be fully elucidated (Bailey et al. 2007; Vaile et al. 2008a), particularly following team-sport exercise (Ingram et al. 2009).
Of the studies demonstrating beneficial effects of post-exercise cold therapy, few have been able to relate the improvements to specific mechanisms (Bailey et al. 2007; Vaile et al. 2008a), with minimal focus on team-sports exercise (Ingram et al. 2009). In particular, although acute and prolonged declines in neuromuscular function are demonstrated following various exercise modalities (Kuitunen et al. 2004; McLellan et al. 2011; Ronglan et al. 2006), there is limited research examining the relationship between alterations in neuromuscular function with cold therapy and the subsequent effects on ensuing exercise performance (Peiffer et al. 2009a), particularly in a team-sport environment. Accordingly, a review of literature has identified the following areas for investigation:

i. The efficacy of cold therapy for recovery and performance enhancement following the demands associated with team-sports.

ii. The relationship between evidence of EIMD and post-exercise cold therapy.

iii. The association between exercise-induced elevations in thermal strain and post-exercise cold therapy altering the recovery of neuromuscular function.

iv. The relationship between muscle damage due to collisions in sport and subsequent performance and recovery with cold therapy.

v. The relationship between post-exercise cold therapy altering the recovery of neuromuscular function following the various demands associated with team-sport exercise, particularly with the inclusion of exogenous load (heat and body collisions).

Accordingly, a main aim of this thesis was to evaluate the effect of post-exercise cold therapy on the recovery of neuromuscular function following various demands associated with team-sport exercise. A fundamental component of each of the research studies investigated in the present thesis was the evaluation of neuromuscular function following various exercise modalities and the subsequent effects of post-exercise cold therapy on the
recovery profile of neuromuscular function. Therefore, to examine both central and peripheral skeletal muscle function, a range of neuromuscular measures were implemented and are discussed in more detail below. Subsequently, this review of literature will outline the physiological demands associated with team-sports exercise. Further, given that many athletic events are held in warm environments, and team-sport exercise often includes indirect and direct physical collisions, the associated effects on performance and physiological function under these conditions will be discussed. Finally, current evidence outlining the role of post-exercise cold therapy and the subsequent effects on the symptoms of EIMD, exercise performance, heat stress and collision-based sports will be discussed in detail.

**Assessment of Neuromuscular Function**

Muscle fatigue is defined as an exercise induced reduction in maximal voluntary force (Gandevia 2001). Although the precise mechanisms are not fully understood, it is accepted that central fatigue (including alterations in processes located above the neuromuscular junction) can be distinguished from peripheral fatigue (Gandevia et al. 1995b). A failure of force generating capacity may occur at various sites along the pathway from the central nervous system (CNS) to the intramuscular contractile apparatus (Kent-Braun 1999; Westerblad et al. 1998). Central fatigue refers to a progressive decline in the ability to activate muscle voluntarily and it is attributable to impairment at sites proximal to the neuromuscular junction (Gandevia et al. 1996). Peripheral fatigue is defined as a decrease in the capacity of the skeletal muscle to generate force because of action potential failure, excitation-contraction (E-C) coupling failure, or impairment of cross-bridge cycling, in the presence of unchanged or increasing neural drive (Bigland-Ritchie et al. 1986; Fuglevand et al. 1993; Hakkinen and Komi 1983; Taylor et al. 1997. The use of maximal voluntary contractions (MVC) and non-invasive transcutaneous electrical stimulation combined with
surface electromyography (EMG) allows examination of the sites of neuromuscular fatigue in humans (Gandevia, 2001). The assessment techniques are commonly implemented to examine alterations in maximal force-generating capacity during and following various exercise modalities and conditions (Bigland-Ritchie et al. 1986; Merton 1954; Søgaard et al. 2006), with the production of MVC reported to be highly reproducible with CV <7% and ICC of 0.90 (Place et al. 2007).

The contribution of central fatigue to a reduction in force generating capacity is commonly assessed by voluntary activation estimated by twitch interpolation of a single supramaximal electrical stimulus to the motor nerve during an isometric MVC (Gandevia 2001; Merton 1954). The interpolated stimulus evokes a superimposed twitch, the amplitude of which allows calculation of the percentage of voluntary activation and thus estimates the level of neural drive to the muscle (Allen et al. 1998; Gandevia et al. 1995a). A large interpolated twitch indicates low levels of VA and thus indicates the presence of central fatigue (Gandevia et al. 1995b). During isometric exercise, central fatigue may reflect inhibition at the motoneuronal level (Garland and McComas 1990) along with diminishing drive to the motor cortex (Gandevia et al. 1995b). Further, the determination of central fatigue is also assessed by the change in the electromyogram (EMG) signal of the active muscles during MVC (Millet and Lepers 2004) on the assumption that the changes reflect alterations in central neural drive (Bigland-Ritchie 1981). When the EMG value is used to evaluate the existence of central fatigue during an MVC, this value needs to be normalised by the maximal compound muscle action potential (M-wave) (Lepers et al. 2000). Therefore, to assess the contribution of central fatigue to alterations in the force-generating capacity of the muscle following exercise associated with the demands of team-sports in the present thesis and to evaluate the recovery profile of central fatigue, measures of MVC, VA and the EMG signal normalised to the M-wave will be assessed. Specifically, repeated, intermittent short-
duration MVC’s will be performed pre- and post-exercise and throughout the determined recovery period. Five repeated MVC’s, separated by a 5 s rest interval will be evaluated with superimposed twitch evoked at the point of peak torque during each MVC (represented schematically in Figure 2.1). A potentiated twitch will then be evoked during the 5 s rest interval between each contraction to allow calculation of the interpolated twitch technique (ITT) to provide an indication of voluntary activation (Gandevia 2001; Merton 1954; Thomas et al. 1989).
**Figure 2.1** Schematic representation of the protocol used for assessment of maximal voluntary contraction (MVC), voluntary activation and potentiated twitch properties throughout each of the studies. 5 x 5-s MVC’s were performed with superimposed electrical stimulation during the contraction followed by a potentiated twitch initiated during the 5-s rest interval between contractions. The arrow represents electrical stimulation of the femoral nerve.
Further, the presence of peripheral fatigue is commonly measured by comparing the force responses to electrical stimulation before and after a fatiguing exercise (Schillings et al. 2005). Accordingly, prior to the assessment of MVC rested twitch contractile and M-wave properties evoked by a single electrical stimulus will also be determined during the exercise and recovery time points and is considered as a reliable means to describe peripheral contractile fatigue (Millet et al. 2002). The M-wave is commonly used in human fatigue experiments as an index of the effectiveness of neuromuscular transmission and impulse propagation in muscle fibres (Bigland-Ritchie et al. 1982; Hicks and McComas 1989). The following characteristics of an evoked resting twitch are commonly determined to detect failure in E-C coupling or neuromuscular propagation: (1) peak potentiated twitch torque (Pt); (2) rate of torque development (RTD); (3) time to peak torque (TPt); (4) rate of relaxation (RR); (5) half relaxation time (1/2 RT); (6) contraction duration (CD) (Cannon et al. 2006; Place et al. 2007). Further, the duration, amplitude and latency of an M-wave provide an indication of neuromuscular transmission and excitability of the muscle fibre sarcolemma (Lepers et al. 2000). The decline in quadriceps twitch torque and prolongation of TPt and CD may indicate disruptions in E-C coupling (Behm and St-Pierre 1997; Edwards et al. 1977; Viitasalo and Komi 1981), while a reduced M-wave amplitude indicates a failure to drive the action potential to the muscle fibre. Therefore, evoked twitch contractile and M-wave properties will be determined throughout each study of the present thesis to assess the presence of peripheral fatigue following the demands associated with team-sports exercise and evaluate the effect of cold therapy on the recovery of peripheral reductions in force-generating capacity.

Of note, it is well documented that the application of cold sufficient to reduce muscle temperature alters contractile properties (Nastuk and Hodgkin 1950; Ranatunga et al. 1987), decreasing RTD and the speed of muscle relaxation (De Ruiter et al. 1999). The reasons for
these changes have been explained by a slowed adenosine triphosphate (ATP) hydrolysis
(Ferretti et al. 1992), slowed calcium (Ca\(^{2+}\)) release and uptake from the sarcoplasmic
reticulum (Kossler and Kuchler 1987) and a decreased Ca\(^{2+}\) sensitivity of the actomyosin
(Sweitzer and Moss 1990). As such, to account for potential differences in evoked responses
of the colder muscle temperatures, M-wave amplitude was normalised to the voluntary EMG
signal (Lepers et al. 2000). Further information on the effects of colder temperatures on
muscle function is discussed in more detail below.

*Physiological Responses to Team-Sport Exercise*

**Team-Sport, Game-Based Activity Patterns: Exercise Regulation and Fatigue**

Many team-sport athletes train and compete several times per week, often on
consecutive days, and engage in high-intensity, intermittent-sprint activity (Dawson et al.
2005; Dawson et al. 2002). During competitive matches, most athletes are required to
compete over prolonged periods (60-90 min) at moderate to high intensities (~65 - 85% VO\(_{2}\)\(_{\text{max}}\)), separated into equal halves by a 10-15 min half-time break (Reilly 1997). Due to the
intense nature and physiological demands of team-sports, exercise intensity during the second
half of competition is often altered (Coutts et al. 2010; Mohr et al. 2003; Reilly 1997), with
reductions in moderate- and high-intensity activity observed (Coutts et al. 2010; Duffield et
al. 2009a). In addition, with repeated bouts of high-intensity training sessions and/or
competitive matches performed over several consecutive days with minimal respite between
sessions, substantial fatigue and decrements in exercise performance may be observed
(Montgomery et al. 2008b; Ronglan et al. 2006; Spencer et al. 2005).

The activity patterns of many team-sports are intermittent in nature, consisting of
repeated bouts of brief (<6-s) maximal to near-maximal work interspersed with relatively
short (<60-s) moderate to low-intensity recovery periods (Glaister 2005; Spencer et al. 2005). In field-based team-sports (e.g. AFL, hockey, rugby and soccer), distances covered during games range from 5000 to 12000m (Bangsbo et al. 1991; Brewer and Davis 1995; Reilly and Borrie 1992; Wisbey et al. 2010). The match intensities and activity patterns of athletes during a competitive match are considerably influenced by player position (Bangsbo et al. 1991; Brewer and Davis 1995; Docherty et al. 1988), environmental factors, and the level of competition (Reilly 2000). The mean duration of high-intensity efforts during field-based sports is reported to be approximately 4-7-s (Bangsbo et al. 1991; Docherty et al. 1988; Mayhew and Wenger 1985) of which approximately 2-s is attributed to all-out sprinting (Bangsbo et al. 1991; Docherty et al. 1988). The mean exercise intensities of 60-75% $\text{VO}_2\text{max}$ (Bangsbo 1994; Boyle et al. 1994; Reilly 1997) and mean heart rates of 70-90% of maximum (Docherty 1982; Ekblom 1986) sustained over the prolonged duration of team-sports activity highlight both the aerobic and anaerobic nature of such exercise and that a substantial physiological load is present.

More specifically, during international men’s field hockey approximately 95% of game time is reported to be spent in moderate to low-intensity activities (standing, walking and jogging) (Spencer et al. 2004), similar to the 90-95% time spent in low-intensity motions for soccer (Bangsbo et al. 1991; Mayhew and Wenger 1985), Australian Rules football (Coutts et al. 2010; Wisbey et al. 2010) and rugby league (Meir et al. 1993). Spencer et al. (2004) reported that the remaining 5% of the player game time during elite men’s hockey was spent in the high-intensity motions of striding and sprinting. The mean sprint duration recorded was 1.8 ± 0.4s in accordance to that reported in soccer (2.0 ± 0.9 s) (Bangsbo et al. 1991), rugby (2.0 ± 0.4s) (Docherty et al. 1988) and Australian Rules football (2.4 ± 0.6s) (Dawson et al. 2002), but less than the 3.1 ± 0.3 s reported in women’s field-hockey (Lothian and Farrally 1994). Table 2.1 summarises the literature pertaining to time motion analysis of
various team sports. In addition to repeated bouts of sprinting followed by low-intensity activity throughout team-sports activity, athletes continually change direction approximately every 5 s during a competitive game (Spencer et al. 2004). The intermittent nature and continued change of direction, together with the eccentric muscle actions involved in repetitious accelerating and decelerating movements, suggests that considerable demands are placed on metabolic reserves, with increases in physiological load and exercise-induced muscle damage observed throughout team-sport competition (Spencer et al. 2004). As such, reductions in both sub-maximal and sprint performance are commonly observed during the second half of team-sport exercise (Balsom et al. 1992b; Mohr et al. 2003; Spencer et al. 2004).

During competitive match-play, recent time-motion analysis examining team-sports performance has indicated the presence of both ‘cumulative’ and ‘transient’ fatigue (Duffield and Coutts 2011). Indeed, match analysis of various team-sports has demonstrated reductions in total distance covered during the second half of matches in AFL (Coutts et al. 2010), rugby league (Sirotic et al. 2009), rugby union (Roberts et al. 2008) and soccer (Carling et al. 2008). Mohr et al. (2003) reported that the distance travelled in high-intensity running was significantly lower (14-45%) in the last 15 min of international soccer match-play compared to the first 15 min. Further evidence to suggest cumulative fatigue during match-play was observed recently by Rampinini et al. (2007) who demonstrated greater reductions in second half high-intensity running in athletes who completed more high-intensity running in the first half of Premier league soccer matches, accounting for approximately 25% reduction in total distance (~400 m). Similarly, Coutts et al. (2010) also reported that ~49% of the reduction in the total distance covered during second half of an AFL match compared to the first half was from high-intensity running. Therefore, the collective findings of the aforementioned studies demonstrate that cumulative fatigue exists during team-sport match-play and is often evident.
by the reduction in the total distance covered and high-intensity running throughout the second half of match-play.

Although evidence suggests that both ‘cumulative’ and ‘transient’ fatigue occurs in matches, it is apparent that athletes are also able to retain the capacity to achieve peak speeds when required throughout a match (Duffield and Coutts 2011). For example, Duffield et al. (2009a) recently demonstrated that the amount of very high-intensity running (i.e. >20 km·h\(^{-1}\)) and peak speeds achieved during each sprint were not altered in the last quarter of AFL match-play despite reductions in moderate-intensity running. Additionally, Bradley et al. (2009) reported no change in peak sprint velocities over the course of English Premier League soccer matches. Although many other studies have demonstrated that the total sprint distance is reduced towards the end of matches (Mohr et al. 2003, 2005), it is interesting to note that many athletes can preserve the ability to reach peak speeds, even toward the latter part of match-play (Carling et al. 2008; Coutts et al. 2010). Thus, although exercise intensity and performance may be reduced throughout the course of team-sport match-play, it is apparent that the self-paced nature of such exercise allows the regulation of exercise intensity and thus reductions in performance are observed primarily in non-critical intensities aiming to preserve the ability to maintain high-intensity running when necessary (Coutts et al. 2010).
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| Sirotic et al. (2009)         | Rugby League   | n = 39                           | • Video recordings over two rugby league seasons (2004-2005) from Elite and semi-elite competition | Mean Sprint duration  
- Elite = 2.1 ± 1.2 s  
- Semi-elite = 2.0 ± 0.9 s  
Time spent  
Running  
- Elite = 399.3 ± 106.6 s  
- Semi-elite = 329.1 ± 86.7 s                                                                 |
|                               |                | Elite n = 17                     |                                                                                               |                                                                           |
|                               |                | Semi-Elite n = 22                |                                                                                               |                                                                           |
|                               |                | Standing, walking, jogging, running, fast running, sprinting, LI running, HI running, VHI running |                                                                           |                                                                           |
| Cunniffe et al. (2009)        | Rugby Union    | n = 2                            | • Out of season competitive game  
• GPS  
• Accelerometer – Impact data  
• Standing and walking, jogging, cruising, striding, high-intensity running, sprinting | % of time spent in  
- Standing and walking = 72 ± 4%  
- Jogging = 18.6 ± 2.2%  
Severe (10°g) impacts  
- Backs = 4 ± 2  
- Forwards = 13 ± 6                                                                 |
|                               | (Elite)        | Forward n = 1                    |                                                                                               |                                                                           |
|                               |                | Back n = 1                       |                                                                                               |                                                                           |
| Roberts et al. (2008)         | Rugby Union    | n = 29                           | • English Premiership Rugby  
• Video recording  
• Standing, walking, jogging, medium-intensity running, HI-running, maximal speed running | Total Dist. Covered  
- Forwards = 5581 ± 692  
- Backs = 6127 ± 724  
Mean Sprint Duration  
- Forwards = 1.2 ± 0.3 s  
- Backs = 1.2 ± 0.3 s  
% of time spent  
- Walking = 81 ± 6%  
- Jogging = 33 ± 4%                                                                 |
|                               |                | Props & 2nd Row n = 8            |                                                                                               |                                                                           |
|                               |                | Back Row n = 6                   |                                                                                               |                                                                           |
|                               |                | Inside Backs n = 7               |                                                                                               |                                                                           |
|                               |                | Outside Backs n = 8              |                                                                                               |                                                                           |
| Deutsch et al. (2007)         | Rugby Union    | n = 29                           | • 8 Super 12 Rugby matches  
• Video recording  
• Locomotion (standing, walking, jogging, cruising, sprinting & utility)  
• Intense exertion (rucking/mauling, tackling, scrumming)  
• Discrete (kicking, jumping, passing) | % of time spent  
Walking  
- Forwards = 27.4 ± 1.2%  
- Backs = 50.2 ± 6.5%  
Jogging  
- Forwards = 21.0 ± 2.9%  
- Backs = 17.3 ± 3.7%  
Mean Sprint Duration  
- Forwards = 2.0 ± 0.8 s  
- Backs = 3.8 ± 0.4 s                                                                 |
|                               | (Super 12)     | Front Row Forward n = 9          |                                                                                               |                                                                           |
|                               |                | Back Row Forward n = 7           |                                                                                               |                                                                           |
|                               |                | Inside Back n = 7                |                                                                                               |                                                                           |
|                               |                | Outside Back n = 6               |                                                                                               |                                                                           |
| Duthie et al. (2006)          | Rugby Union    | n = 28                           | • 10 games of 2003 Super 12 Rugby competition  
• Video recordings  
• Standing, walking, jogging or striding | Total no. of sprints = 503  
- Forwards = 213 (13 ± 6 sprints p/game)  
- Backs = 288 (24 ± 7 sprints p/game)                                                                 |
<table>
<thead>
<tr>
<th>Study</th>
<th>Sport</th>
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<th>Notes</th>
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<tbody>
<tr>
<td>Burgess et al.</td>
<td>Soccer (A-League)</td>
<td>45</td>
<td>Mid-fielders 15, Defenders 15, Attackers 15</td>
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<td>(2006)</td>
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<td>Completion of at least one half of a game in 2002-2003</td>
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<td>Video recordings</td>
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<td>Walking, jogging, striding, sprinting, max. speed</td>
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<td>Mean sprint duration</td>
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<td>Forwards = 2.5 ± 1.6 s</td>
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<td>Backs = 3.1 ± 1.6 s</td>
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<tr>
<td>Duthie et al.</td>
<td>Super 12 Rugby</td>
<td>47</td>
<td>Forwards 16, Back Row 15, Inside Backs 9, Outside Backs 7</td>
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<td>Video recordings</td>
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<td>Rest (standing, walking &amp; jogging)</td>
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<td>Work (striding, sprinting, static exertion, jumping, lifting or tackling)</td>
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<td>Forwards = 2.2 ± 0.6 s</td>
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<td>Backs = 2.9 ± 0.6 s</td>
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<td>Spencer et al.</td>
<td>Elite field hockey</td>
<td>14</td>
<td>Australian men’s field hockey</td>
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<td>(2004)</td>
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<td>One international game</td>
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<td>Video recordings</td>
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<td>Standing, walking, jogging, striding, sprinting</td>
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<td>Mean Sprint Duration</td>
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<td>1.8 ± 0.4 s</td>
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<tr>
<td>Dawson et al.</td>
<td>AFL</td>
<td>11</td>
<td>Every AFL match played in Perth in 2000 season (22 games)</td>
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<td>(2004)</td>
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<td>Standing, walking, jogging, fast running, sprinting</td>
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<td>1.8 ± 0.4 s</td>
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<td>Forwards = 27 ± 7%</td>
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<td>Backs = 38 ± 10%</td>
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<td>Jogging</td>
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<td>Forwards = 20 ± 4%</td>
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<td>Standing, walking &amp; jogging = 95%</td>
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<td>Jogging = 37.8 ± 5.1%</td>
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<td>Work = 4:51 ± 1.16 min</td>
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<td>Rest = 83:21 ± 4.40 min</td>
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<td>Mean Sprint Duration</td>
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<td>Forwards = 2.2 ± 0.6 s</td>
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<td>Backs = 2.9 ± 0.6 s</td>
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<td>Tot. Dist. Covered</td>
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<td>13 600 – 17 000m</td>
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_AFL = Australian Football League; LI = Low Intensity; HI = High Intensity; VHI = Very High Intensity; GPS = Global Positioning System; % = Percentage;_
For team-sports in particular, most athletes do not fail to complete a match and as such the term fatigue specific to team-sports exercise may be used to describe the transient or progressive reduction in intensity during the event (Duffield and Coutts 2011). To date, most evidence examining the mechanisms causing fatigue for team-sport exercise have been extrapolated from laboratory simulations of intermittent-sprint exercise (Bangsbo et al. 2007; Reilly and Gilbourne 2003). Only recently have investigations examining the alterations in exercise intensity during team-sport exercise been applied to field settings (Coutts et al. 2010; Duffield et al. 2009a). Generally, fatigue can be defined as any exercise-induced reduction in the ability of a muscle or muscle group to generate force or power and has traditionally been divided into “central” and/or “peripheral” fatigue (Bigland-Ritchie and Woods 1984; Gandevia 2001). Peripheral fatigue occurs when there is interruption of the contractile element at or below the neuromuscular junction, while central fatigue is defined as a progressive reduction in neural drive of muscle during exercise, and thus a decline in ability of the nervous system to activate muscles maximally (Gandevia 2001; Millet and Lepers 2004).

Traditionally, the aetiology of fatigue during and following exercise has examined the mechanism during constant-paced, laboratory exercise. More recently, the focus of the manifestations of fatigue has shifted to more self-paced, internally regulated exercise, aiming to emulate the demands associated with exercise in an applied setting (Coutts et al. 2010; Duffield et al. 2009a). Regardless, the demands of high-intensity, intermittent-sprint team-sport exercise increases the physiological load which results in a reduction in intensity during exercise (Duffield et al. 2009a; Mohr et al. 2003) and can also impair performance during subsequent exercise bouts (Montgomery et al. 2008b; Rowsell et al. 2009). Accordingly, implementation of post-exercise recovery strategies, including CWI aim to minimise both transient and prolonged post-exercise fatigue associated with team-sport exercise (Ingram et
Consequently, this section of the review will focus on the physiological demands associated with team-sports exercise, the presence of ‘transient’ fatigue and the additional effects of exogenous load, specific to team-sports, has on exercise performance.

In addition to the presence of transient fatigue during match-play, many team-sports follow an intense schedule of consecutive day training bouts and tournament-based competition requiring teams to play several matches over successive days increasing the likelihood of residual fatigue (Odetoyinbo et al. 2007; Ronglan et al. 2006; Spencer et al. 2005). Involvement in competitive tournament-based matches over consecutive days results in increased perceptions of exertion and fatigue (Rowsell et al. 2009), and substantial decrements in sprint, agility and vertical jump performance (Montgomery et al. 2008b).

Ronglan et al. (2006) observed a 2-7% decline in sprint and CMJ performance in elite female handball players during a 3-day tournament. Furthermore, Spencer et al. (2005) demonstrated that repeated-bout sprint performance in matches decreased in elite male field hockey players over a 4-day tournament, with fewer efforts evident as matches progressed in the tournament. Rowsell et al. (2009) reported that decrements in physical test performance (CMJ and mean repeated sprint time) after team-sport match-play are consistent with decrements in exercise performance observed previously following competitive match-play (Dawson et al. 2005; Hoffman et al. 2003; Reilly and Rigby 2002). Furthermore, the 7% decline in CMJ performance over the course of the soccer tournament was similar to the decrement recently reported for handball players during a 3-day international tournament (Ronglan et al. 2006).

When such exercise is performed in a tournament situation, marked residual fatigue has been observed with decrements in striding and repeat effort running (Spencer et al. 2005). For example, Ronglan et al. (2006) observed decrements in 20-m sprint time and CMJ height over a 3-day international handball tournament, while the sprint and vertical jump performance of male basketball players was reduced during a 3-day tournament.
In addition, elite field hockey players performed fewer repeated-sprint bouts in consecutive matches during a 4-day tournament (Spencer et al. 2005). The progressive decrease in physical test performance observed by Rowsell et al. (2009) suggests that recovery between successive matches was insufficient for full restoration of physical performance. Recent studies have demonstrated that residual fatigue accumulated over successive matches can adversely affect team-sport performance (Ronglan et al. 2006; Spencer et al. 2005). As such, many team-sports athletes engage in strategies immediately post-exercise aiming to minimise fatigue and improve recovery in an effort to maximise performance during successive exercise bouts (2 - 48 h).

Further to the observed decrements in performance during tournament-based competition (Ronglan et al. 2006; Rowsell et al. 2009), markers of muscle damage have been shown to accumulate when matches are played on successive days (Hoffman et al. 2002; Rowsell et al. 2009; Viitasalo et al. 1995). The concentrations of creatine kinase (CK) and lactate dehydrogenase (LDH) increased over the course of the 4-day soccer tournament (Rowsell et al. 2009), remaining above baseline on day 5. These results are consistent with previous investigations (Hoffman et al. 2002; Viitasalo et al. 1995) and demonstrate the elevation in markers of muscle damage during competitive team-sport exercise played on successive days. Indeed, repeated moderate and rapid accelerations and decelerations performed during team-sports exercise place high physical demands on players with exercise-induced muscle damage from eccentric loading during decelerations and jumping commonly observed throughout the duration of a match (Lakomy and Haydon 2004). An increased rating of perceived exertion (RPE) has also been observed and suggested to be due to the increasing amount of exposure to EIMD over the duration of a soccer tournament, evidenced by increased concentrations of lactate dehydrogenase and CK (Rowsell et al. 2009). As a common problem for team-sport athletes is the time available for full physiological and
performance recovery between exercise sessions is often limited (Rowsell et al. 2009), interventions aiming to enhance or maximize recovery from previous competition/training are often implemented (Barnett 2006).

Physiological Responses Associated with Team-Sports Exercise: Transient Effects

First, repeated high-force stretch-shortening cycle muscle actions, consistent with many team-sport activities have been associated with disruption of the internal milieu of the body (Gaitanos et al. 1993) and reduced force-generating capacity (Bigland-Ritchie and Woods 1984; Miller et al. 1996) resulting in acute and long-term impairments in skeletal muscle force production (Nicol et al. 2006). Substrate depletion (e.g. muscle glycogen), increased muscle activity, metabolic by-product accumulation (e.g. bicarbonate and Lactate) and ion-shifts have traditionally been highlighted as explanations for alterations in skeletal muscle function following high-intensity, intermittent exercise (Balsom et al. 1992a; Gaitanos et al. 1993; King and Duffield 2009; Minett et al. 2010). Furthermore, the associations between disrupted peripheral factors of fatigue and the subsequent effect on central motor drive have also previously been described to contribute to acute reductions in post-exercise performance (Bigland-Ritchie et al. 1986; Garland and McComas 1990; Miller et al. 1996; Woods et al. 1987). During high-intensity intermittent-sprint exercise, a demonstrated decrease in muscle and blood pH and muscle glycogen levels (Gaitanos et al. 1993; Hargreaves et al. 1998; McCartney et al. 1986) have been associated with declines in electromyogram (EMG) and voluntary force; suggesting the presence of a feedback loop between intramuscular metabolism and central motor drive contributing to acute reductions in exercise intensity (Bigland-Ritchie et al. 1986; Garland and McComas 1990; Miller et al. 1996; Woods et al. 1987). Although controversy currently exists as to the precise mechanisms responsible for acute reductions in performance during prolonged, intermittent
exercise (Balsom et al. 1992b; Hargreaves et al. 1998; Millet et al. 2003; Sahlin et al. 1998), recent evidence highlights the role of anticipatory regulation in reducing exercise intensity as part of a protective mechanism either in response to (Gonzalez-Alonso et al. 1999) or anticipation of elevated physiological load i.e., increased endogenous heat production (Marino 2004; Tucker et al. 2004). Regardless, it is clear that repeated bouts of high-intensity, indicative of many team-sports, increases the physiological demand of exercise (Gaitanos et al. 1993; Hargreaves et al. 1998), altering the mechanical ability of skeletal muscle (Balsom et al. 1992b; Gaitanos et al. 1993) contributing to impairment in acute neuromuscular function (Nicol et al. 2006). Furthermore, although the high-intensity nature of team-sport exercise results in acute impairment in skeletal muscle function, intermittent-sprint running involving eccentric muscle actions can additionally result in prolonged reductions in skeletal muscle function (Lakomy and Haydon 2004).

Physiological Responses Associated with Team-Sports Exercise: Acute and Prolonged Neuromuscular Responses

The competitive demands of team-sports exercise impose strains on various physiological systems resulting in acute and prolonged reductions in neuromuscular function (McLellan et al. 2011; Montgomery et al. 2008b). The prolonged reduction in VJ, CMJ and MVC have been identified following one-off team-sport match play (Ascensão et al. 2011; Bailey et al. 2007; Ingram et al. 2009; McLellan et al. 2011) and also during repeated tournament-like competitions (up to 4 days) (King and Duffield 2009; Montgomery et al. 2008b; Rowsell et al. 2011). In a recent study by Ascensao et al. (2011), CMJ and squat jump was reduced at 24- and 48-h after a soccer match, whilst Kinugasa and Kilding (2009) reported an immediate reduction in VJ following 90-min soccer match-play. Similarly, following rugby league match-play, peak power, peak force and peak rate of force
development of a CMJ was also immediately reduced post-exercise and remained up to 24-h post-match. Further, when team-sport exercise is performance over consecutive days, reductions in neuromuscular function are more pronounced with declines in VJ observed following a 3-day basketball tournament (Montgomery et al. 2008b). Consequently, team-sport exercise results in both acute and prolonged impairment in neuromuscular function, which may remain evident up to 48-h post-exercise.

Furthermore, the ability to produce lower body maximal isometric force is also compromised for a prolonged period following team-sport exercise (Ascensão et al. 2011; Ingram et al. 2009). Specifically, following simulated team-sport exercise, Ingram et al. (2009) recently demonstrated prolonged reductions in isometric leg strength remaining evident up to 48-h post-exercise. Similarly, MVC was significantly reduced at 24 and 48-h following prolonged intermittent shuttle running and did not return to pre-exercise values until 168-h post-exercise (Bailey et al. 2007), with prolonged reductions in MVC (up to 24 h) supported more recently by Pournet et al. (2010) following exhaustive intermittent exercise. Accordingly, the aforementioned studies highlight that intermittent-sprint exercise places considerable demands on neuromuscular function and results in acute and prolonged reductions in skeletal muscle functions, which can remain evident up to 48-h post-exercise (Ascensão et al. 2011; Ingram et al. 2009). Although current evidence demonstrates significant reductions in VJ, CMJ and MVC following team-sport exercise, there remains a paucity of research outlining the effects of team-sport exercise on central activation (Girard et al. 2007). Therefore, with team-sport exercise resulting in acute and prolonged impairments in neuromuscular function, the focus on post-exercise recovery has become increasingly important (Ascensão et al. 2011; Ingram et al. 2009). However, to date minimal research has been able to fully elucidate the specific effects of post-exercise CWI on the recovery of
neuromuscular function following team-sport exercise, particularly examining central and peripheral mechanisms.

**Physiological Responses Associated with Team-Sport Exercise involving Exogenous Load: Heat and Contact-Based Sport**

In addition to the standard physiological response associated with team-sport exercise, inclusion of exogenous load such as performing in hot environmental conditions and involvement of indirect and direct body collisions exacerbates the physiological response to team-sport exercise (McLellan et al. 2011; Peiffer et al. 2009a). The heightened physiological responses during exercise in the heat and when match-play involves body collisions may result in acute alterations in the regulation of exercise intensity (Coutts et al. 2010) and may also delay the recovery back to optimal functioning (Wendt et al. 2007). Specifically, exercise in hot environmental conditions exacerbates the physiological stress of an exercise bout by increasing internal body temperature and sweat rates, with resultant reductions in blood volume and increased cardiovascular load compared to cooler conditions (Sawka 1992) with concomitant reductions in exercise performance (Duffield et al. 2009a; Tucker et al. 2004). Indeed, recent evidence highlights that the rise in core temperature ($T_{core}$) during team-sport exercise in warm conditions is associated with declines in moderate intensity activity, thus reducing the total distance covered during an Australian Football League (AFL) match (Duffield et al. 2009a). Furthermore, when the exogenous load involves direct physical collisions, heightened cytokine and endocrine responses are demonstrated to be a direct result of physical contacts (Zuliani et al. 1985), with prolonged reductions in neuromuscular function evident following collision-based sports such as rugby league (McLellan et al. 2011). Therefore, the presence of exogenous load (heat and body collisions) during team-sport exercise exacerbates the immediate and prolonged physiological stress associated with
high-intensity intermittent-sprint exercise. As a result, recovery interventions are often implemented in an effort to counter such physiological perturbations and aim to accelerate recovery back to optimal functioning in preparation for subsequent sessions (Dawson et al. 2005; Peiffer et al. 2009a). Accordingly, the following section will focus on the physiological responses associated with exercise performed in warm environmental conditions and team-sports involving direct physical collisions.

**Physiological Responses Associated with Exercise in the Heat**

Many elite team-sport and individual athletic events occur over prolonged periods (60-90 min) at relatively high-intensities (Reilly 1997). When exercise is performed in hot environmental conditions, elevated $T_{\text{core}}$ (>39°C) can increase perceptions of fatigue and thermoregulatory strain, limiting the duration of exercise and reducing time to exhaustion (Gonzalez-Alonso et al. 1999; Marino 2002; Nielsen et al. 1993; Nybo and Nielsen 2001). Indeed, it is well-established that both exercise- and passive-induced elevations in core (>39°C) (Martin et al. 2004; Thomas et al. 2006) and local tissue temperature (Cheung and Sleivert 2004; Thornley et al. 2003) result in a decrement in exercise performance (Gonzalez-Alonso et al. 1999; Morrison et al. 2004). These performance reductions may be via reductions in both the contractile function of the muscle (Hargreaves 2004) and central motor drive (Nybo and Nielsen 2001). The observed reduction in central nervous system (CNS) drive to active musculature, due to a down-regulation in muscle recruitment, is postulated to be part of an anticipatory regulation (Marino 2004; Tucker et al. 2004). This preventative mechanism is thought to reduce the production of metabolic heat (Martin et al. 2004; Thomas et al. 2006; Tucker et al. 2006) either in response to (Nybo and Nielsen 2001) or avoidance (Marino 2004; Tucker et al. 2004) of an increased thermal load. Although the increase in physiological demand of exercise in the heat and subsequent reduction in exercise
performance is well-documented following laboratory-based constant and self-paced exercise (Nybo and Nielsen 2001; Tucker et al. 2006), there is currently a paucity of research examining the specific physiological effects of team-sport exercise performed in hot environmental conditions (Duffield et al. 2009a; Edwards and Clark 2006).

In a laboratory setting, constant-load exercise performed in warm environments is well-established to induce high $T_{core}$, which are associated with reductions in exercise intensity and eventual cessation of exercise (Gonzalez-Alonso et al. 1999; Reilly et al. 2006). In contrast, self-paced exercise in the field only produces moderate increments in $T_{core}$ (Laursen et al. 2006), with exercise intensity adjusted based on perceptual and physiological responses aiming to ensure that work is completed without further excessive increases in internal thermal load (Marino 2004; Tucker et al. 2006). Further, previous team-sport, field-based data has indicated a plateau in $T_{core}$ (below 39.5°C) during soccer (Edwards and Clark 2006), American football (Godek et al. 2004) and AFL match-play (Duffield et al. 2009a). Duffield et al. (2009a) recently observed that the maintenance of $T_{core}$ was associated with reductions in moderate-intensity activity (7-14 km h$^{-1}$), although mean velocities of high-intensity and very high-intensity running were not reduced. Accordingly, these data suggest that unlike constant-paced exercise, athletes participating in field-based, team-sport exercise in warm environments may regulate exercise intensity to prevent excessive increases in $T_{core}$ predominantly via a reduction in moderate-intensity activity, to ensure the continuation of exercise, often until the end of a match.

Furthermore, the response in physiological demand and elevation in $T_{core}$ has previously been demonstrated to vary between recreational and professional team-sport match-play (Edwards and Clark 2006). Indeed, Edwards and Clark (2006) observed significant increases in $T_{core}$ from pre-exercise values during each half of a recreational soccer match; however, during a professional match, $T_{core}$ was only significantly increased above
pre-exercise values in the first half and unchanged in the second half of exercise. Unfortunately no performance measures were determined in this study and thus it is difficult to comment on the subsequent effects on exercise performance. However, it may be suggested that elite team-sport athletes are more efficient at regulating exercise intensity to avoid the rise in $T_{core}$ above critical values throughout the duration of a match performed in warm environments (Duffield et al. 2009a; Edwards and Clark 2006). Regardless, it is clear that team-sport exercise performed in hot environmental conditions results in increases in internal temperature which may be maintained at tolerable values via regulation of exercise intensity (Duffield et al. 2009a; Marino 2004; Tucker et al. 2004). However, with prolonged suppression of neuromuscular function, general fatigue and sleeplessness evident with the inability to tolerate imposed thermal load (Armstrong et al. 2007; Thomas et al. 2006), cooling strategies to counter such negative effects are commonly implemented (Marino 2002; Peiffer et al. 2009a).

Although the rise in $T_{core}$ during free-paced, field-based exercise are demonstrated to be below 39.5°C, some athletes often reach $T_{core}$ values approaching 40°C which is known to increase the risk of heat illness (Nybo and Nielsen 2001) and negatively affect exercise performance (Martin et al. 2004). High internal body temperatures resulting from exercise in the heat have been demonstrated to not only negatively impair acute exercise performance but also delay the recovery back to optimal functioning (Wendt et al. 2007). Indeed, exercise- and passively-induced heat stress, and the inability to tolerate imposed thermal loads has been demonstrated to result in prolonged suppression in voluntary force production and activation (Martin et al. 2004; Thomas et al. 2006). Interestingly, the return to optimal neuromuscular performance following passively-induced increases in thermal load has been observed only when cooling methods were introduced to increase the rate of reduction in $T_{core}$ (Thomas et al. 2006). Further, heat intolerance has also been linked to general fatigue, sleeplessness and
reductions in ensuing exercise performance (Armstrong et al. 2007); highlighting the importance of maintaining favourable internal body temperatures for optimal exercise performance, particularly when consecutive bouts of exercise are performed in warm environments. As it is common for many team-sports to train and/or compete, often over successive days, in hot environmental conditions and given the detrimental effects of high internal thermal loads on acute and long-term exercise performance, cooling strategies to counter the acute and prolonged effects of exercise-induced elevations in heat stress have become popular (Marino 2002; Quod et al. 2006). However, given the well documented association between laboratory exercise in hot conditions and attainment of higher physiological strain and reduced exercise performance (Gonzalez-Alonso et al. 1999), limited research has focused on the effects of post-exercise cooling on ameliorating physiological and performance recovery following self-paced exercise in the heat (Peiffer et al. 2009a; Peiffer et al. 2010b; Vaile et al. 2008a; Vaile et al. 2010), with no research to date examining the efficacy of post-exercise cooling following team-sport exercise in the heat.

**Physiological Responses Associated with Collision-Based Exercise**

Participation in physical collision sports such as rugby league, rugby union, Australian Football League (AFL), American Football and soccer involve high-intensity, intermittent-sprint exercise, resulting in damage due to direct impacts when opposing players collide with each other (Takarada 2003). The physically demanding nature of such sports significantly increases the physiological load associated with training and match-play (Gabbett et al. 2008) with players involved in 20-40 physical confrontations per game (Gissane et al. 2001). As such, subsequent reductions in exercise performance may be associated with stretch-shortening cycle (SSC) fatigue and exposure to repeated blunt force
trauma following competitive match-play (McLellan et al. 2011). Furthermore, large increases in muscle enzyme and endocrine responses, including CK and myoglobin, have been demonstrated to be a direct result of physical contact (Zuliani et al. 1985), with more elevations pronounced following competitive match play in American football (Hoffman et al. 2005; Hoffman et al. 2002) and rugby union (Takarada 2003). Due to the intense nature of repeated high-intensity activity, involving numerous physical impacts during training and match-play, many collision-sport athletes engage in post-exercise recovery strategies aiming to minimise potential reductions in exercise performance during subsequent bouts.

The physiological demands associated with physical contact sports are strongly correlated to the number of tackles performed (Gabbett et al. 2008; Takarada 2003) resulting in elevated cytokine and endocrine responses (McLellan et al. 2010) with marked increases in symptoms of EIMD (Takarada 2003). Indeed, during a competitive rugby union match, Takarada (2003) demonstrated a significant correlation between the number of tackles performed and peak CK measured 24-h post-match. Similarly, McLellan et al. (2010) reported an increase in plasma CK post-rugby league match-play with levels remaining elevated up to 120 h post-match; whilst American football also significantly increased plasma CK (Komi 2000). This peak plasma CK is similar to the responses reported in other sports involving high-impact collisions between players (Smart et al. 2008; Takarada 2003). Although the relationship between elevated CK response and athletic performance is unclear, McLellan et al. (2010) did observe a concomitant reduction in peak rate of force development (PRFD) 30 min post rugby league match. The authors postulated that the decrement in PRFD was causally related to the increase in plasma CK, in accordance with the findings of others (Andersson et al. 2008; Komi et al. 1996). Previously, Andersson et al. (2008) reported that CMJ height was reduced in the presence of a significant rise in plasma CK after elite female soccer match play; whilst Komi et al. (1996) reported an association between increase in
plasma CK and decreased drop jump performance. The results of the aforementioned studies demonstrate a significant reduction in subsequent exercise performance with an elevation in plasma CK as a result of high-intensity, body-collision activities and significant damage to skeletal muscle tissue as a result of repeated blunt force trauma (Andersson et al. 2008; McLellan et al. 2010).

Furthermore, the high-intensity physically demanding nature of collision sports results in significant neuromuscular fatigue, including impairment in excitation-contraction coupling (MacLaren et al. 1989) and reduced neural drive (McLellan et al. 2011; Strojnik and Komi 1998). McLellan et al. (2011) recently demonstrated a reduction in peak voluntary force 30-min post-rugby league match. In addition, a reduction in the rate of force development (RFD) of a peak twitch has also been demonstrated immediately post-rugby match and up to 48-h post-match and is postulated to reflect the influence of impaired excitation-contraction coupling resulting from high-impact physical collisions (MacLaren et al. 1989). Moreover, as a result of the large number of physical contacts during collision-based match-play (Brewer and Davis 1995; Gissane et al. 2001), musculoskeletal injuries including contusions, haematomas and strains are common, while joint injuries and muscular strains are more frequently sustained during training (Gabbett 2000; Gissane et al. 1997; Gissane et al. 1993; Meir et al. 1993; Stephenson et al. 1996). As a result, elevated symptoms of EIMD and reductions in neuromuscular function following collision-based exercise is documented to require a prolonged recovery phase of at least 5 days to achieve full recovery of skeletal muscle damage (McLellan et al. 2011). Therefore, in an effort to minimise injury risk, optimise recovery from skeletal muscle damage and improve subsequent performance, post-game (and training) recovery strategies are commonly implemented aiming to accelerate regeneration processes in preparation for subsequent sessions (Banfi et al. 2007; Dawson et al. 2005). Although CWI has emerged as a popular post-exercise recovery intervention, there
remains a paucity of research examining the specific effects of post-exercise CWI following collision-based, high-intensity intermittent-sprint activity.

**Exercise-Induced Muscle Damage (EIMD) Associated with Team-Sport Exercise**

As highlighted previously, team-sport exercise, particularly collision-based, induces prolonged symptoms of EIMD as a result of the eccentric component of intermittent-running, landing (Lakomy and Haydon 2004) and exposure to repeated physical collisions (McLean 1992; McLellan et al. 2010). Of the many symptoms that accompany EIMD, including muscle soreness, increased blood myocellular proteins (Armstrong et al. 1983), swelling and decreased range of motion (Clarkson et al. 1986); perhaps the most significant factor to athletic performance is the prolonged impairment of muscle function, most notably a reduction in force-generating capacity (Byrne et al. 2004; Clarkson and Sayers 1999).

The time course and severity of symptoms of EIMD can vary considerably depending on the duration, intensity, and type of exercise performed (Clarkson et al. 1986; Eston et al. 1994; Thompson et al. 1999). Although eccentric muscle actions are only one facet of team-sport exercise, severe EIMD resulting from high-velocity eccentric exercise produces immediate and prolonged reductions in muscle strength of more than 50% generally ascribed to excitation-contraction coupling impairment and myofibre damage (Allen 2001; Clarkson and Sayers 1999; Morgan and Allen 1999), which is generally restored after approximately 10 days (Clarkson et al. 1992; Newham et al. 1987). Therefore, in regard to subsequent exercise performance, the prolonged reduction in muscle function resulting from EIMD may be an important aspect to consider for optimal team-sports performance, particularly when ensuing bouts are within 24 – 48 h.
Initial manifestations of EIMD are disrupted sarcomeres and damage to components of the excitation-contraction (E-C) coupling system (Morgan and Allen 1999; Proske and Morgan 2001; Warren et al. 2001). Following these initial events, a process of muscle fibre degeneration and regeneration occurs (Armstrong 1984; Clarkson and Sayers 1999; Pyne 1994). Initial mechanical trauma is thought to mediate the inflammatory response and is characterised by infiltration of fluid and plasma proteins into the injured tissue for removal of damaged contractile proteins and cellular debris, before regeneration begins (Lowe et al. 1995; MacIntyre et al. 1996; Pyne). During these stages, the transient symptoms of delayed-onset muscle soreness (DOMS), muscle stiffness, and muscle swelling appear and subside. The progressive myofibril degeneration observed in some fibres following the initial insult (Jones et al. 1986) suggests secondary ischemic cellular damage (Swenson et al. 1996), with the release of intracellular proteins and infiltration of the tissue by neutrophils and macrophages between 6-12 h (Kuipers et al. 1983; Round et al. 1987). The peak in macrophages at 48 h aims to destroy necrotic tissue (MacIntyre et al. 2000) and sensitises type III and IV nerve endings to mechanical, chemical or thermal stimulation (Kuipers 1994). Increased sensations of DOMS and muscle pain with EIMD has therefore been suggested to result from elevated pressure from tissue oedema and increased local temperature activating nociceptors within the muscle fibres and the muscle tendon junction (Cheung et al. 2003).

One of the greatest concerns to the athlete following EIMD is the prolonged reduction in skeletal muscle function, resulting in decrements in exercise performance (Byrne and Eston 2002b). Indeed, isometric strength is reduced immediately post-eccentric exercise and recovery is gradual and prolonged over the ensuing 14 days (Clarkson et al. 1992; Newham et al. 1987). The magnitude and time course of strength loss appear dependent on the training history of the muscle group (Byrne et al. 2004) and the intensity and type of exercise performed (Eston et al. 1994; Thompson et al. 1999). Clarkson et al. (1992) reported that
following maximal eccentric exercise of the elbow flexors, an immediate 50-60% reduction in strength followed by a linear recovery to baseline by 2 weeks post-exercise occurred. Furthermore, Evans et al. (1990) observed significant decreases in isokinetic eccentric peak torque of the elbow flexors at 0 h (43.5%), 24 h (38.8%) and 48 h (-32%) following repetitive eccentric contractions. The suppression in peak torque required at least 14 days post-exercise to return to pre-exercise values. Other researchers have also reported a delayed return of eccentric peak torque of the elbow flexors following isokinetic eccentric exercise, with peak torque remaining 15% lower than initial peak torque until 7 d post-DOMS inducement (Yates and Armbruster 1990). Although the results of the aforementioned studies may not specifically relate to the precise manifestations of EIMD observed following team-sport exercise, the results do highlight the prolonged reductions in voluntary force production as a result of eccentric load, often present during team-sport exercise (Lakomy and Haydon 2004).

The majority of studies related to knee extensor (KE) eccentrically-induced fatigue have reported maximal voluntary contraction (MVC) impairment ranging from 11-30% (Brown et al. 1997; Eston et al. 2000; Hortobagyi et al. 1998; Newham et al. 1983). Byrne and Eston (2002a) reported a 20% decrease in MVC 1 h after completion of 100 barbell squats. Similarly, Friden et al. (1983) reported a 24.3% reduction in MVC 20 min after completion of 30 min eccentric cycling exercise; with a similar 19.6% reduction in MVC following KE eccentric exercise (Martin et al. 2005). Furthermore, Komi and Viitasalo (1977) demonstrated a 35% reduction in KE strength and a decrease in the rate of force development, which had not recovered 2 days after 40 maximal leg press eccentric actions. Similarly, a 30-40% reduction in KE strength with incomplete recovery (approximately 95%) 7 days after 100 repetitions of the eccentric phase of the barbell squat exercise performed with a load of 80% of concentric one repetition maximum was also observed (Byrne and Eston 2002b). Following multi-joint activity, Avela et al. (1999) reported a 30% reduction in
ankle extensor strength and rate of force development with full recovery by 2 and 4 days post-marathon running. Therefore, the consistent findings from research investigating the effects of eccentric exercise or prolonged stretch-shortening cycle (SSC) exercise on locomotor muscle groups are an immediate and prolonged reduction in strength and a decreased rate of force development, which may require up to 7-14 d to return to normal baseline levels (Byrne and Eston 2002b; Clarkson et al. 1992).

Although the precise mechanisms responsible for DOMS remains equivocal (Armstrong 1984; Clarkson and Sayers 1999; Friden and Lieber 1992), sites and mechanisms of failure in the neuromuscular system contributing to altered muscle function after eccentric exercise have been identified and demonstrated to be located peripherally (i.e. E-C coupling failure, redistribution of sarcomere lengths, damage to contractile machinery, impaired metabolism) rather than centrally (Newham et al. 1982; Rutherford et al. 1986; Saxton and Donnelly 1996). Evidence from studies employing the twitch interpolation technique have suggested that full voluntary activation (VA) can be achieved during isometric MVC following eccentric EIMD; thus indicating that the likely source of fatigue is peripheral in nature (Newham et al. 1987; Rutherford et al. 1986; Saxton and Donnelly 1996). However, evidence detailing the mechanisms responsible for immediate reductions in force production following eccentric exercise appears to support a contribution of both central and peripheral fatigue (Loscher and Nordlund 2002; Michaut et al. 2002). Indeed, Michaut et al. (2002) observed an immediate reduction in VA following eccentric exercise of the elbow flexors. Similarly, Löscher and Nordlund (2002) found some impairment of VA immediately after eccentric exercise, but this recovered within 5 min. Furthermore, Prasartwuth et al. (2005) observed an immediate reduction in MVC, together with reduced resting twitch torque following eccentric exercise of the elbow flexors. VA was reduced by 19 ± 6% immediately post-exercise but was not different from control values after 2 days. As such, the authors
suggested that reduced VA contributes to the early force loss following eccentric exercise, but inhibition of peripheral contractile function is likely responsible for prolonged reductions in force production (Prasartwuth et al. 2005).

Although immediate reductions in VA following eccentric exercise have been observed (Loscher and Nordlund 2002; Prasartwuth et al. 2005), the specific mechanisms for acute reductions in VA remain equivocal. It has previously been postulated that increased muscle soreness after eccentric exercise might impair VA, with pain contributing to inhibition of the motor cortex (Le Pera et al. 2001). However, Prasartwuth et al. (2005) and Lösch and Nordlund (2002) observed only immediate reductions in post-exercise VA, with levels returned to control values within 24-h post-exercise and muscle pain peaking 1-2 d following exercise. Hence, the different time course of muscle soreness and changes in VA make it unlikely that muscle pain directly causes the changes in voluntary drive following eccentric exercise. Alternatively, the firing of group III and IV muscle afferents has also been linked to reduced VA (Gandevia et al. 1996) with stimulation of these afferents possibly via metabolites such as bradykinin and histamines shown to increase in plasma after eccentric exercise (Blais Jr et al. 1999; O'Connor and Cook 1999). Regardless of the mechanisms, it appears that immediate reductions in skeletal muscle function following EIMD are due to a combination of failure of central motor drive and impaired peripheral contractile function (Prasartwuth et al. 2005; Saxton and Donnelly 1996). Furthermore, with prolonged reductions in force observed after EIMD with no change in VA, it appears that long lasting decrements in muscle function are a result of damage to peripheral contractile apparatus (Prasartwuth et al. 2005) which may result from the eccentric component of intermittent-sprint exercise (Bailey et al. 2007) or exposure to repeated physical collisions during team-sport exercise (McLean 1992; McLellan et al. 2011).
Further decrements in dynamic, multi-joint activity have also been reported in the presence of EIMD (Twist and Eston 2005). Observed reductions in the ability to generate peak power output (PPO) and maximally sprint over 10 m has been demonstrated following a plyometric exercise protocol (Twist and Eston 2005). Prolonged reductions in PPO were observed at 24, 48 and 72 h (18, 16 and 14%, respectively) with the findings relating to functional impairment in accordance to those of Byrne and Eston (2002b). The authors postulated that reductions in PPO and successive maximal intensity exercise following EIMD are partly due to subsequent damage of type II muscle fibres. With additional fibre recruitment during high-load eccentric exercise and likely subsequent damage (Eston et al. 1996; Friden et al. 1983; Jones et al. 1986; McHugh et al. 2000), it has been suggested that the inability to achieve PPO may also be attributed to the presence of muscle soreness and reduced central drive (Twist and Eston 2005). However, Proske (2005) has alternatively suggested that the presence of DOMS may in fact be part of a protective mechanism, relating to damaged contractile apparatus, to prevent further injury and damage.

Furthermore, impaired muscle glycogen resynthesis following EIMD has also been well documented (Asp et al. 1999; Asp et al. 1997; Costill et al. 1990; O'Reilly et al. 1987). This has primarily been attributed to an eccentrically damaged muscle having to work at a higher relative workload during concentric exercise, resulting in increased glycogen utilisation and thus decreased endurance capacity. The resting muscle glycogen content of type II fibres has been shown to be severely reduced, supporting the proposition that type II fibres are recruited and damaged during eccentric exercise (Asp et al. 1999). An impaired resynthesis of muscle glycogen following damaging exercise represents an obvious challenge to athletes involved in competition or athletic training where muscle glycogen content may be rate-limiting to increased exercise intensity. Reduced glycogen content in type II fibres may also suggest that there are similar implications for sports of a high intensity or intermittent
nature. Thus, with evidence demonstrating prolonged reductions in maximal strength and power (Byrne et al. 2004), impaired neuromuscular control (Miles et al. 1997), selective fibre type II damage, reflex inhibition and elevated muscle soreness, adversely affecting dynamic, multi-joint movements associated with athletic activity, the approach to optimising recovery following muscle-damaging exercise, aiming to allow an immediate return to training and further competition is of great importance.

The presence of EIMD and associated deleterious symptoms is a familiar experience for the elite and novice athlete. In particular, such experiences may be present during the pre-season when exercise is likely to be unaccustomed, or when activity during training and/or competition has a large eccentric component (Bailey et al. 2007; Thompson et al. 1999) or when exercise involves repeated exposure to intense physical contacts between opposing players as in collision-based sports (McLellan et al. 2011; Takarada 2003). With EIMD resulting in prolonged reductions in muscle strength and power (Byrne and Eston 2002a; Twist and Eston 2005), and increases in muscle soreness 24-72 h post-exercise (Armstrong 1984; Byrnes et al. 1985; Jones et al. 1986), many athletes engage in post-exercise recovery strategies aiming to alleviate deleterious symptoms of EIMD and attenuating declines in muscle function (Vaile et al. 2008c). More recently, attention has focused on the effect of cold therapy in aiding recovery from muscle-damaging exercise (Eston and Peters 1999; Howatson and Van Someren 2003; Yanagisawa et al. 2003a; Yanagisawa et al. 2003b). Although, the role of cold therapy as a treatment of sports-related injuries is well-documented (Bleakley et al. 2004), support for its specific application to EIMD, particularly following team-sport exercise, remains predominantly anecdotal.
Biochemical indirect markers of muscle damage and inflammation

EIMD is associated with structural damage of the sarcomeres (Friden et al. 1983; Newham et al. 1987), protein leakage from the injured muscle fibres (Sorichter et al. 1999), an acute inflammatory response (Fielding et al. 1993; MacIntyre et al. 1996) and DOMS (Faulkner et al. 1993; MacIntyre et al. 1996). Accordingly, the assessment of muscle protein responses in the blood, including creatine kinase (CK), asparate aminotransferase (AST) and C-reactive protein (CRP), to various exercise modalities are commonly implemented to provide indirect evidence for muscle damage and cell inflammation (Bailey et al. 2007; Halson et al. 2008; Warren et al. 1999). Increased cytokine levels have mainly been found after eccentric exercise with plasma levels of CK increased almost 40-fold 4 days following eccentric exercise (Bruunsgaard et al. 1997). CK is released by the muscle into the lymphatic system where it is transported to the thoracic duct and then enters the blood stream (Havas et al. 1997). Further, the local response to tissue injury and damage involves the production of cytokines that are released at the site of inflammation (Pedersen and Hoffman-Goetz 2000). These cytokines facilitate an influx of lymphocytes, neutrophils, monocytes and other cells that participate in the healing of the tissue. The local inflammatory response to tissue damage is accompanied by a systemic response (acute phase response) and includes the production of hepatocytes derived acute phase proteins such as CRP (Pedersen and Hoffman-Goetz 2000). However, it has recently been demonstrated that caution should be sort when using muscle proteins in the blood as indicators of muscle damage as blood concentration is a function of the interaction of production and clearance from the blood (Saxton and Donnelly 1996; Sorichter et al. 1995). Accordingly, Warren et al. (Warren et al. 1999) suggested that the measurement of MVC provides the best indication of muscle injury following EIMD. Therefore, measures of neuromuscular function were implemented as the primary means of evaluating muscle damage with measures of CK, AST and CRP used as additional evidence.
to indicate indirect levels of muscle damage and cell inflammation following the demands associated with exercise.

**Post-Exercise Cold Therapy**

**The Use of Cold Therapy as a Recovery Strategy**

The increased physical demands of competitive sports and the intense nature of consecutive day training and competition bouts has resulted in many amateur and professional athletes engaging in post-exercise CWI recovery (Barnett 2006). While anecdotal support for the benefits of CWI are generally positive, the effectiveness of post-exercise cold therapy on recovery of exercise performance, alterations in neuromuscular functions and perceptions of recovery remain varied (Bailey et al. 2007; Jakeman et al. 2009; Yamane et al. 2006). The rationale behind the implementation of post-exercise CWI is based on empirical evidence supporting the use of cold therapy following acute soft tissue injuries (Bleakley et al. 2004; Meussen and Lievens 1986), as well as benefits for reducing body temperature (Hadad et al. 2004). Although various forms of cold therapy have been suggested to be effective treatments to decrease metabolism, inflammation, blood flow, pain, and skin, muscle and intra-articular temperatures, as well as increase tissue stiffness (Merrick et al. 1999), the specific effects of CWI on the recovery profile and subsequent performance of athletes remain varied (Ingram et al. 2009; Yamane et al. 2006). This section of the review will therefore focus on the current effects of cold therapy on physiological and inflammatory responses, muscle function and also the effect of hydrostatic pressure on physiological alterations. Furthermore, current evidence on the specific effects of post-exercise cold therapy on recovery from various modes of exercise, primarily associated with team-sports
exercise, on the recovery of exercise performance, EIMD, exercise-induced thermal stress and contact sports will also be discussed.

**Physiological Responses to Cold Therapy: Cooler Temperatures and Hydrostatic Pressure**

The implementation of cold therapy (ice packs, CWI etc) results in a number of physiological responses to numerous body systems (Bleakley and Davison 2009). The most common physiological response with cold therapy is a reduction in skin temperature (Weston et al. 1994). Dependent upon the mode and duration of cold therapy application, intramuscular temperature is also reduced resulting in a peripheral vasoconstrictive response (Ho et al. 1994; Karunakara et al. 1999; Marsh and Sleivert 1999). The associated redistribution of blood flow to the core (Ho et al. 1994; Karunakara et al. 1999; Marsh and Sleivert 1999) stimulates venous return, thus increasing central blood volume (Bonde-Petersen et al. 1992). Cold therapy administered at temperatures lower than 15°C have also been shown to induce substantial alterations on numerous body systems including ‘cold shock response’ characterised by hyperventilation, extreme activation of the sympathetic nervous system, increasing the release of noradrenaline in the blood (Leppäluoto et al. 2005) and resultant tachycardia (Tipton 1989). This response normally reaches a peak within the first 30 s of <15°C CWI, with many individuals adapting after approximately 3 min (Datta and Tipton 2006). Other physiological responses associated with superficial application of cold include a decrease in local metabolic function, reduced oedema formation (Dolan et al. 1997) as well as reduced nerve conduction velocity, muscle spasm and increased local anaesthetic effects (Weston et al. 1994). As such, cold therapy is thought to play a significant role in minimising the inflammatory response (Burke et al. 2000; Meeusen and Lievens 1986). Furthermore, recently observed benefits of post-exercise cold therapy have been
postulated to relate to the resultant effect of cold temperatures on blood flow, and if CWI is the modality implemented, the additional effects of hydrostatic pressure applied to the body during immersion in water is also thought to contribute to altered physiological function (Vaile et al. 2008c; Vaile et al. 2010; Wilcock et al. 2006). Therefore, the reduction in metabolic function, oedema formation and subsequent reductions in the inflammatory response are the proposed rationale for the implementation of post-exercise cold therapy following athletic events (Wilcock et al. 2006).

In addition to the demonstrated physiological responses to colder temperatures, hydrostatic pressure exerted on the body due to water immersion produces significant physiological alterations which have been postulated to be beneficial to recovery (Wilcock et al. 2006). Cardiovascular responses to body cooling and water immersion include increases in peripheral vascular resistance, blood pressure and stroke volume, and reductions in heart rate, cardiac output and peripheral blood flow (Bonde-Petersen et al. 1992; Sramek et al. 2000; Wilcock et al. 2006). The redistribution of blood from the periphery to the core has been suggested to result from both peripheral vasoconstriction (Marsh and Sleivert 1999) and the hydrostatic pressure exerted on the body during water immersion (Wilcock et al. 2006). The increased blood flow throughout the body results in a concomitant increase in central blood volume and venous return and is thereby suggested to improve cardiac efficiency and blood delivery to the working muscles (Marsh and Sleivert 1999). Wilcock et al. (2006) suggested that the hydrostatic pressure exerted on the body while immersed in water causes a displacement of fluid from the peripheries into the central cavity, thus enhancing the return of fluid from the muscles into the blood and increasing central blood volume, stroke volume and cardiac output. The increase in central circulatory volume is thought to be beneficial in reducing cardiac stress due to the increased venous return and stroke volume (Maw et al. 2000) and improved muscle blood flow (Bonde-Petersen et al. 1992).
Evidence of reduced cardiac stress following CWI has been observed with resultant increases in vagal-related heart rate variability (HRV) indices (Buchheit et al. 2009; Lemaitre et al. 2008). These higher blood pressures activate arterial high pressure and cardiopulmonary low pressure baroreflexes, which is thought to enhance vagal nerve activity and inhibit sympathetic nerve activity, leading to a bradycardia (Liu et al. 2008; Pump et al. 2001) and in increase in vagal-related HRV indexes (Buchheit et al. 2009; Spinelli et al. 1999). Indeed, Schniepp et al. (2002) observed a reduction in maximum and average heart rates after CWI, consistent with other investigations (Bergh and Ekblom 1979b; Blomstrand et al. 1984; Suzuki et al. 1980). It has been postulated that improved cardiac efficiency is likely due to a combination of cold-induced peripheral vasoconstriction, hydrostatic pressure and the passive nature of CWI (Buchheit et al. 2009; Schniepp et al. 2002). Accordingly, one rationale for implementing post-exercise CWI to improve recovery has been based on the proposed beneficial effects of combined hydrostatic pressure and cold temperatures reducing cardiac strain and improving blood flow throughout the body (Wilcock et al. 2006).

**The Effects of Cold Therapy following Acute Injury and the Associated Inflammatory Response**

Further to the highlighted immediate beneficial physiological responses there is currently substantial evidence supporting the use of cold therapy as a means of reducing the inflammatory response (Eston and Peters 1999; Hiruma et al. 1999; Yanagisawa et al. 2003a). Indeed, cold therapy is well documented to be beneficial for the treatment of acute soft tissue injury by providing pain relief, muscle spasm reduction and a modification of the inflammatory response (Knight 1989; Knight et al. 2000; Prentice 1990). Accordingly, cold application immediately post-injury is primarily used to reduce cell metabolism (Kowal 1983; Prentice 1990), necrosis, oedema formation and neutrophil migration; thereby
minimising secondary hypoxic injury and the degree of tissue damage (Eston and Peters 1999; Hiruma et al. 1999; Knight 1989; Knight et al. 2000; Yanagisawa et al. 2003b). The reduction in metabolic activity in the cells that survive the original trauma renders them less susceptible to hypoxia (secondary trauma) and hence the total number of cells traumatised is potentially reduced (Knight 1976). Further, it is thought that cold reduces blood flow by increasing viscosity and causing localised vasoconstriction, which further reduces blood flow and less fluid volume at the injury site, thus resulting in a reduction in oedema formation (Knight 1976). An additional beneficial effect of cold therapy on acute musculoskeletal injury is the reduction in muscle spasm by decreasing motor and sensory nerve conduction velocity, and thus reducing pain through cold-induced anaesthesia (Prentice 1982; Prentice 1990). Hence, traditionally cold therapy has been implemented to provide beneficial effects for the acute recovery of soft tissue injury and according to this rationale, cold therapy has more recently been implemented as a post-exercise recovery strategy in an aim to minimise similar deleterious symptoms associated with training and competition.

Furthermore, implementation of cold therapy following acute musculoskeletal injury has also demonstrated beneficial effects in reducing immediate and prolonged perceptions of pain and tenderness (Meeusen and Lievens 1986; Prentice 1990). The application of cold sufficient to lower muscle temperatures by 10-15°C has been shown to reduce motor nerve conduction velocity continuing with decreasing temperature until nerve fibre conduction ceases completely (Swenson et al. 1996). The additional reduction in muscle spindle activity, stretch-reflex response, and spasticity are also thought to contribute to the inhibition of the pain-spasm cycle (Meussen and Lievens 1986). Some authors attribute the reduced pain perception to the localised analgesic effects of cooling rather than inhibition of muscle damage (Denegar and Perrin 1992; Gulick et al. 1996; Meeusen and Lievens 1986). Although there is substantial support for an analgesic effect of cold (Denegar and Perrin 1992; Gulick
et al. 1996; Meeusen and Lievens 1986; Weston et al. 1994), the duration of this analgesia is limited to 1-3 h (Meeussen and Lievens 1986) and accordingly this mechanism may only account for the acute reductions in perceptions of muscle soreness and pain. From an athletic point-of-view, the reduction in pain and muscle soreness (MS) with cold therapy, regardless of the mechanism, together with reduced inflammation and secondary muscle damage has provided a strong rationale to implement post-exercise cold therapy into recovery regimes. However, to date there is a paucity of evidence outlining the beneficial effects of post-exercise cold therapy on reducing MS and inflammation following activity specific to team-sports (Ingram et al. 2009).

Although there is agreement that cold therapy serves an important role in the initial treatment of the acute inflammatory phase after a soft tissue trauma (Knight 1989; Knight et al. 2000; Prentice 1990), the repression of the natural occurring inflammatory mechanism has recently been suggested to be detrimental to muscle repair and adaptation (Lapointe et al. 2002; Yamane et al. 2006). Tidball (2005) recently suggested that muscle inflammation is a functionally beneficial response in the repair and adaptive process. Cellular and connective tissue hypotheses state that adaptation proceeds from successful muscle repair and/or reorganization of several contractile and structural components such as the sarcomeres (Lynn et al. 1998), the extracellular matrix (Stauber et al. 1990), and the cytoskeleton (MacIntyre et al. 1995), making the muscle less vulnerable to further EIMD. The repair process is presumably partially dependent on the inflammatory response triggered by the initial mechanical damage (MacIntyre et al. 1995; Smith 1991). Generally, inflammation serves to remove damaged muscle tissue by recruiting neutrophils and macrophages, but it is also likely important for the process governing muscle repair and/or reorganization after trauma (Armstrong 1984; MacIntyre et al. 1995; Tidball 2005). While neutrophils have been linked to the promotion of muscle damage, their role in processing and removal of damaged tissue
may also be important in muscle regeneration (Kuipers et al. 1983; Round et al. 1987).

Similarly, whilst macrophages can injure muscle cells, there is increasing evidence to support
the existence of macrophage-derived factors influencing muscle growth and regeneration
(Tidball 2005). Accordingly, although traditional evidence outlines that cold therapy is
beneficial in the acute recovery of soft tissue injury (Myrer et al. 1997; Yanagisawa et al.
2003b), the prolonged effects of cold therapy on muscle repair and adaptations, particularly
in an athletic setting are unknown. Barnet et al. (2006) recently eluded that application of a
recovery modality, such as cold, designed to reduce inflammation has recently been
suggested not be in the best interests of the athlete. As such, although CWI is widely utilised
as a treatment modality following acute soft tissue injury (Myrer et al. 1997; Yanagisawa et
al. 2003a), there remains some controversy over the effectiveness of cold therapy in
improving the prolonged symptoms associated with skeletal muscle damage and trauma
(Farry et al. 1980).

Evidence to support the negative effects of repressing the inflammation response via
cooling modalities (Farry et al. 1980; Fu et al. 1997) and non-steroid anti-inflammatory drugs
(NSAID’s) (Lapointe et al. 2002) following musculoskeletal trauma on muscle repair and
adaptation have been observed in animal studies. Certainly, cooling after exercise in an
endurance-training program imposing exhaustive running upon rats for 5 days per week was
found to enhance rather than reduce ultra-structural damage after 7 weeks (Fu et al. 1997),
suggesting a detrimental effect of post-exercise cooling on muscle repair and adaptation.
Further, Farry et al. (1980) found that no regimen of cooling in rats lessened oedema and that
a detrimental effect with temperatures below 15°C existed. These authors also reported that
the volumes of non-traumatised and traumatised limbs were greater 48 h after two 20-min
applications of crushed ice than in the control condition. In addition, although Lapointe et al.
(2002) examined the effects of NSAID’s on muscle repair and adaptation, the repression of
the inflammatory response with NSAID’s significantly impaired the repeated bout effect. Treatment with NSAID affected in parallel the concentration of macrophage subpopulations and the adaptive response which likely reflected an impairment of skeletal muscle repair and adaptation (Lapointe et al. 2002). Accordingly, Lapointe et al. (2002) postulated that the decreased inflammatory response altered repair likely resulted in an incomplete or inadequate muscle adaptation.

Although the aforementioned studies relate to investigations in humans, Isabell et al. (1992) demonstrated that post-exercise ice massage following 300 concentric/eccentric contractions of the elbow flexors resulted in the highest peak soreness and CK levels and the lowest ROM compared to an exercise group. Isabell et al. (1992) observed no effect of cold therapy on indices of muscle damage and suggested repeated cold therapy may in fact be detrimental to muscle repair and adaptation over a prolonged period with levels of CK significantly higher 120 h post-exercise following ice massage compared to a control and exercise group. In agreement, Yamane et al. (2006) recently examined the effect of cold therapy on endurance or forearm flexor resistance training on untrained men over a period of 4-6 weeks. Cold therapy comprised of one or two 20 min CWI for the endurance training and one 20 min CWI for the resistance training following each training session. The results indicated that post-exercise cooling lessened the effects of training reductions in VO$_{2\text{max}}$, ventilatory threshold and arterial diameter observed post-training. Accordingly, Yamane et al. (2006) suggested that CWI may actually have negative effects on muscle adaptation and repair by retarding any post-training adaptive processes associated with improvement in performance (Yamane et al. 2006).

Although evidence supports the effect of cold therapy in the treatment of acute soft tissue injury (Myrer et al. 1997; Yanagisawa et al. 2003b), myofibre micro-damage and cellular and humoral events induced by endurance and strength training within skeletal
Chapter 2

muscles are considered as physiological preconditions not only for repair processes, such as myofibre regeneration, but also for the adaptive processes leading to improved muscular performance. Thus, lowering muscle temperature by cold therapy may indeed interfere with these regenerative processes and may retard rather than support the desired improvement of muscular performance (Yamane et al. 2006). While the exact mechanisms remain to be elucidated, these results challenge the position that the acute inflammatory process must be repressed to favour recovery from muscle damage (Lapointe et al. 2002). Accordingly, further investigations evaluating the effect of post-exercise cold therapy on muscle repair and adaptation processes, particularly in an athletic setting, is required.

Colder Muscle Temperatures: The Effects on Peripheral Muscle Function

The effects of cooler muscle temperature on the production of skeletal muscle force and neural conduction are often contradictory (Edwards et al. 1992; Oska et al. 1997). Generally, in cooler muscle fibres there is an extended time of relaxation that reflects prolongation of cross-bridge attachment resulting in a reduction in cross-bridge cycling (Ferretti et al. 1992). An impairment in the activation of motor units during a short time interval has also been observed, suggested to be due to the lower nerve impulse frequency (Vanggaard 1975). Further, the maximum tetanic tension produced during muscle cooling has been demonstrated to be depressed with the reduction more pronounced on cooling below 20˚C (Davies et al. 1982; Ranatunga and Wylie 1983). Additionally, the external cooling of muscle temperature to 24˚C and lower has been shown to significantly reduce the force and power generating abilities of both human and animal skeletal muscle (Davies and Young 1983; Holewijn and Heus 1992; Ranatunga et al. 1987). Indeed, the rate of force development (RFD) of an evoked twitch was significantly slowed by 22% when isolated rat muscle was
cooled to 25°C (Faulkner et al. 1990). Drinkwater and Behm (2007) also showed that the rate of isometric twitch and tetanic force development declined by a similar 50 and 46%, respectively when the plantar flexors were cooled to 22°C in humans. Furthermore, Ranatunga et al. (1987) found that time to peak twitch almost doubled in cooling the skin to 12.5°C. As such, any cooling modality which reduces the temperature of the muscle fibre results in a delayed conduction velocity and RTD resulting in significant reductions in immediate production of force (Davies and Young 1983; Ranatunga et al. 1987). Consequently, it may be viewed as deleterious to peak power output if the rate and force production of skeletal muscle fibres are reduced.

In addition to the prolongation of RFD, another characteristic of muscle cooling on twitch contractile properties is the prolongation of the half relaxation time (½ RT). Drinkwater and Behm (2007) illustrated a 132% increase in the ½ RT of single evoked twitch while ½ RT of tension developed by tetanic stimulation was 119% longer when human plantar flexors were cooled to 22°C. Similarly, at a skin temperature of 12.5°C, Ranatanga et al. (1987) illustrated a 200% increase in ½ RT. Faulkner et al. (1990) postulated that the prolongation of relaxation may be caused by slowed adenosine triphosphate (ATP) binding to the ATP-binding site on the myosin head thus causing slower detachment of the myosin cross-bridges. In addition, slowing of the sarcoplasmic reticulum ATPase is also thought to slow the active process of re-uptake of calcium out of the sarcoplasm, thereby prolonging contraction (Drinkwater and Behm 2007; Kossler and Kuchler 1987). Accordingly, in regard to an athletic setting, cooling a muscle to below 20°C may have negative consequences on the ability to produce immediate force and power (Davies and Young 1983; Ranatunga et al. 1987).

More recently, Davies et al. (1982) reported some observations on the effects of temperature on the contractile properties of the triceps surae muscle. The authors observed
that under the coldest conditions (muscle temperature of 24°C) the tension and evoked twitch tension was significantly reduced. A slower rate of tension development and decreased velocity of muscle shortening under cold conditions also has been reported by several authors (Bigland-Ritchie et al. 1992; Cornwall 1994; Sargeant 1987). Clarke and Royce (1962) reported that 10 min cold application increased the time necessary to reach 95% of maximum force in forearm flexor muscle, which has since been supported by Davies and Young (1983). Further, Bigland-Ritchie et al. (1992) reported a lengthened M-wave duration in locally cooled finger muscles when the muscle temperature fell below 28°C. It has been postulated that changes in the rate processes with decreasing temperature may be a consequence of the effect of temperature on metabolic rate (Bergh and Ekblom 1979b; Faulkner et al. 1990) and the maximal rate of ATP hydrolysis (Edwards et al. 1972). Slowed conduction velocity resulting from cooler muscle temperatures is also thought to be due to impairment of Ca²⁺ release from the sarcoplasmic reticulum (Kossler and Kuchler 1987), reduced Ca²⁺ sensitivity (Sweitzer and Moss 1990), and/or impaired kinetics of the muscle fibre action potentials (Edwards et al. 1972; Segal and Faulkner 1985; Ward and Thesleff 1974). Therefore, reduced force and power provides clear evidence that electrical and mechanical muscle response is affected by cold.

**Colder Muscle Temperatures: The Effects on Voluntary Force Production**

Cooling also has been reported to modify the neuromuscular adjustments during voluntary contractions (Clarke et al. 1959; Davies et al. 1982). Previous studies examining human voluntary contractile performance at altered muscle temperatures have observed that the endurance time of sustained, submaximal, voluntary contractions are temperature dependent (Clarke et al. 1959; Edwards et al. 1972). Specifically, optimal force development
seems to occur at peripheral temperatures of 18-26°C, although such evidence remains equivocal (Binkhorst et al. 1977; Davies et al. 1982). Maximal voluntary force was found to be depressed when exposed to muscle temperatures below ~20°C (Clarke et al. 1959). Binkhorst, Hooft and Vissers (1977) found that within a muscle temperature range of 22-38°C the maximum force was not significantly altered but the maximum shortening velocity was increased with temperature. Although a peripheral temperature range of 18-26°C has been described for optimal voluntary force production (Edwards et al. 1972), Davies et al. (1982) reported that local cooling to 24°C of the triceps surae reduced MVC. At cooler temperatures, Holewijn and Heus (1992) observed a fall in the maximal grip force after 30 min local cooling (15°C), and Oska et al. (1997) found reduced maximal muscle performance during dynamic exercise performed at a lowered room air temperature (5-10°C). In contrast, Meigal et al. (2003; 1998) reported that exposure to cold (10°C) room air for 30 min did not impair MVC during isometric elbow flexion or handgrip. Edwards et al. (1992) also showed that local muscle cooling did not modify MVC and even prolonged an isometric voluntary contraction sustained until fatigue. Coulange et al. (2006) concluded that total body immersion in cold water did not affect MVC and endurance to fatigue but altered the neuromuscular propagation in the coolest muscle and enhanced fatigue–induced EMG changes in the leg muscles. To further demonstrate equivocal findings, McGown (1967) demonstrated that a cold treatment significantly increased the force production of the quadriceps musculature when applied immediately before isometric leg extension. Therefore, although the literature suggests colder muscle temperatures slow peripheral nerve conduction velocity (Drinkwater and Behm 2007), equivocal findings are present regarding the ability to produce maximal voluntary force following cold therapy (Holewijn and Heus 1992; McGown 1967)
It is also possible that the effects of cold therapy on voluntary force production are dependent on the temperature of the muscle, with pronounced decrements only observed once muscle temperatures get very cold (<24°C) (Davies et al. 1982; Ranatunga 1984; Ranatunga et al. 1987). Indeed, Ranatanga et al. (1984) did not find any decrement in voluntary force at a muscle temperature of 25°C but found it had decreased by 30% in the 12-15°C range. In a latter study by the same authors, Ranatanga et al. (1987) demonstrated an 8% increase in voluntary force with cooling the first dorsal interrossues muscle to 25°C; however, once temperatures decreased below 20°C, there were clear decrements in voluntary force. Davies et al. (1982) cooled the triceps surae to 24°C and found an 18% decrement in voluntary force. Similarly, in cooling the finger flexors to below 27°C, Cornwall (1994) demonstrated a decrease of isometric grip strength by 14.8% in males and 30.5% decrease in females.

Holewijn (1992) found that immersion in 15°C water for 30 min resulted in a significant 21.8% decline in maximal force of grip strength. Clarke et al. (1959) found that MVC of the finger flexors did not change with no less than 30 min immersion in a water temperature of 18°C, but fell by 40% only once the water temperature was 2°C. Oska et al. (2002) demonstrated that the decrement in MVC after a fatigue protocol was more pronounced in mildly cooled muscle (29°C, 17% decrement) than in normo-thermic muscle (34°C, 15% decrement). Whilst the temperature at which cooling begins to impair maximal voluntary force is not exact, the general consensus is that muscle temperature above 27°C will not inhibit maximal force (Davies et al. 1982) with temperatures below 24°C generally impairing voluntary force. Thus, this suggests that the reduction in voluntary force production in colder muscle temperatures may be due to slowed RTD (Drinkwater and Behm 2007) and a reduction in tetanic tension (Davies et al. 1982). The slowed twitch contractile responses and reductions in voluntary force production in muscle temperatures <24°C may have significant
implications for athletic performance, particularly when maximal force is required soon after post-exercise cold therapy.

A decrease in muscle temperature with cold therapy has also resulted in a reduction in short-term power output (Bergh and Ekblom 1979b; Schniepp et al. 2002). Schniepp et al. (2002) found that CWI resulted in a significant decrement in short-term power output in elite cyclists and attributed the decline in power output to a variety of physiological effects that decreased temperature has on muscle function. Similarly, Bergh and Ekblom (1979b) observed a 4-6% decline in power output during maximum cycling per 1°C reduction when muscle temperature decreased from 38 to 30°C; which was attributed to a slower rate of tension development. The reduction in power output has also been attributed to increased muscle stiffness and greater mechanical resistance in colder muscles (Faulkner et al. 1990).

As such, the collective results of the aforementioned studies demonstrate that colder muscle temperatures <24°C result in significantly slowed RTD, conduction velocity and duration of an evoked M-wave and twitch (Bigland-Ritchie et al. 1992; Drinkwater and Behm 2007), together with decreases in voluntary force production (Cornwall 1994) and power output (Bergh and Ekblom 1979a; Schniepp et al. 2002). Therefore, when maximal force or power is required in close succession, implementation of post-exercise cold therapy may be questioned due to observed decrements in skeletal muscle function. Despite this, the effect of post-exercise cold therapy on the recovery of skeletal muscle function following athletic performance such as team-sport exercise, particularly when maximal force and power is not required until next-day training or competition, remains equivocal.
Post-Exercise Cold Therapy: The Effects on Subsequent Exercise Performance

As athletes are often exposed to tournament schedules, or training and matches in close succession, implementation of post-exercise cold therapy is now commonly employed in many sports (Bailey et al. 2007; Kinugasa and Kilding 2009; Rowsell et al. 2009; Vaile et al. 2008a). Despite increased popularity of cold therapy, the effects on improving subsequent exercise performance remain equivocal (Gill et al. 2006; Ingram et al. 2009; Montgomery et al. 2008b; Yamane et al. 2006). Recently, the ameliorated recovery of maximal force production and exercise performance have been observed following post-exercise cold therapy (Bailey et al. 2007; Ingram et al. 2009; Lane and Wegner 2004; Vaile et al. 2008a, b, c). For example, following 90 min of intermittent shuttle running, post-exercise CWI reduced decrements in isometric MVC of the KE (Bailey et al. 2007). Similarly, Ingram et al. (2009) reported a facilitated return to baseline isometric leg extension and flexion force values with CWI when compared to contrast water therapy or a control condition. In addition, the ability to maintain power output and performance during subsequent high-intensity, intermittent cycling sessions was demonstrated following CWI when sessions were separated by 24 h (Lane and Wegner 2004) or performed in the heat (Vaile et al. 2008a). Thus, when cold therapy follows multi-joint, high-intensity intermittent-sprint exercise, positive effects on subsequent exercise performance have been observed (Bailey et al. 2007; Ingram et al. 2009; Vaile et al. 2008a). Table 2.2 provides a summary of the current literature reporting the effects of post-exercise cold therapy on the recovery of subsequent exercise performance.

Positive effects of cold therapy implemented prior to a bout of strength training have also been observed (Burke et al. 2000; McGown 1967). Burke et al. (2000) demonstrated that cold therapy employed prior to isometric strength training resulted in greater improvements in force production. Earlier studies have reported similar findings when strength training was combined with pre-exercise cold therapy (Grose 1958; McGown 1967) with some authors
postulating that increased force production present following cold therapy was due to impaired sensory perception (Bennett 1984; Holewijn and Heus 1992; Ruiz et al. 1993) associated with decreased nerve conduction velocity and cold-induced anaesthesia (Ingersoll et al. 1992; LaRiviere and Osternig 1990). Whilst the precise mechanism for the observed improvement is unknown, and in contrast to the previous section on temperature and muscle force production, the use of cold therapy prior to strength training has demonstrated superior gains in force production when compared with thermal therapy and passive recovery (Burke et al. 2000; Ruiz et al. 1993).

Previous studies have demonstrated positive effects of cold therapy on subsequent performance following a single bout of exercise, along with reduced perceived general fatigue and leg soreness (Rowsell et al. 2009; Rowsell et al. 2011). However, when consecutive bouts of exercise are performed over successive days in a tournament schedule, the effects on subsequent exercise performance remains equivocal (Montgomery et al. 2008b; Rowsell et al. 2009). Montgomery et al. (2008b) demonstrated attenuated decreases in 20-m sprint test and line-drill performance during a 3-day basketball competition with CWI recovery. Further, improvements in the recovery of high-intensity cycling performed over consecutive days have also been observed previously (Vaile et al. 2008b). However, whilst Rowsell et al. (2009) demonstrated lower ratings of fatigue and ameliorated perception of leg soreness following repeated match-play during a 4-day soccer tournament, CWI did not significantly improve subsequent physical performance (CMJ and 12 x 20-m repeated sprint test). This finding is in accordance with previous research reporting that CWI (10 min at 10°C) after 90 min of intermittent shuttle running did not enhance vertical jump performance in young active males at 24 and 48 h of recovery compared with a non-immersion control group (Bailey et al. 2007). In a similar finding, although lower ratings of muscle soreness were observed, CWI (10 min at 9.3 ± 1.6°C) demonstrated no significant benefit to exercise
performance when two bouts of high-intensity, simulated team-sports exercise were separated by 24 h (King and Duffield 2009). Moreover, Coffey et al. (2004) also demonstrated no significant improvement in run time-to-exhaustion after CWI (10°C for 5 min) with Parouty et al. (2010) (14°C for 5 min) further demonstrating a slower swim time during a consecutive sprint compared to passive recovery. Collectively, although CWI recovery appears to be beneficial in enhancing the perceptions of recovery and muscle soreness (Rowsell et al. 2009; Rowsell et al. 2011), the positive effects on performance over consecutive days of exercise remain equivocal (King and Duffield 2009; Rowsell et al. 2009; Rowsell et al. 2011).
Table 2.2 Summary of the Literature Examining Post-Exercise Cold Water Immersion following Various Exercise Modalities

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Exercise Protocol</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pournet et al. (2011)</td>
<td>N = 41</td>
<td>- 20 min exhaustive, intermittent exercise followed by 15 min recovery</td>
<td>• CWI = 15 min at 10°C</td>
<td>CWI and CTWT improved MVC 1 h post-exercise compared to pre-exercise values</td>
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<tr>
<td></td>
<td>Elite athletes (Football, Rugby, Volleyball)</td>
<td></td>
<td>• TWI = 15 min at 36°C</td>
<td>CWI blunted the rise in leukocytes 1 h post-exercise and plasma CK 24 h post.</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• CTWT = alternating between 1 min 30 s in cold (10°C), 1 min 30 s in hot (42°C) for total 15 min</td>
<td></td>
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<tr>
<td>Parouty et al. (2010)</td>
<td>N = 10</td>
<td>- 2 x 100 m swimming sprints interspersed by 30 min recovery</td>
<td>• CWI = 5 min at 14°C</td>
<td>CWI improved perceptions of recovery</td>
</tr>
<tr>
<td></td>
<td>5 F 5 M</td>
<td></td>
<td>• Cont = 5 min out of water seated rest in 28°C</td>
<td>CWI resulted in slower swimming performance but ‘likely’ lower peak HR</td>
</tr>
<tr>
<td>Heyman et al. (2009)</td>
<td>N = 13</td>
<td>- 2 x climbing tests to volitional exhaustion separated by 20 min recovery</td>
<td>• Passive rest</td>
<td>ACT &amp; CWI maintained performance in second bout of exercise</td>
</tr>
<tr>
<td></td>
<td>Well trained climbers (F)</td>
<td></td>
<td>• ACT = cycling at 30-40 W</td>
<td>CWI reduced Tsk</td>
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<td></td>
<td></td>
<td></td>
<td>• EMS of forearm muscles</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>• CWI of forearms &amp; arms (3 x 5 min at 15 ± 1°C)</td>
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<tr>
<td>King and Duffield (2009)</td>
<td>N = 10</td>
<td>- Simulated team-sport exercise circuit (4 x 15 min) performed on consecutive days separated by 24 h recovery period</td>
<td>• 15 min passive seated rest</td>
<td>Trends for attenuated decline in 5 x 20 m sprint &amp; VJ with CTWT &amp; CWI</td>
</tr>
<tr>
<td></td>
<td>Club netball players (F)</td>
<td></td>
<td>• ACT = 15 min shuttle run at 40% VO2max</td>
<td>ACT significantly elevated HR, RPE &amp; MS in session 2 compared to other conditions</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• CWI = 2 x 5 min immersion (9.3 ± 1.6°C, 2.5 min out)</td>
<td>No change in performance in session 2 (24 h) in any condition</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• CTWT = 4 x 1 min immersion (9.7 ± 1.4°C), 2 min warm shower (39.1 ± 2.0°C)</td>
<td></td>
</tr>
<tr>
<td>Ingram et al. (2009)</td>
<td>N = 11</td>
<td>- 80 min simulated team-sport exercise followed by 20 min shuttle run to exhaustion</td>
<td>Upon completion of exercise and 24 h post-exercise</td>
<td>CWI lowered MS and reduced the decrement in isometric leg extension</td>
</tr>
<tr>
<td></td>
<td>Team-sport athletes (M)</td>
<td></td>
<td>• CWI = 2 x 5 min immersion (10°C) separated by 2.5 min upright seating at room temp (22°C)</td>
<td>and flexion strength in 48 h recovery period</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>CWI resulted in a more rapid return to</td>
</tr>
<tr>
<td>Study</td>
<td>Participants (N)</td>
<td>Protocol/Condition</td>
<td>Recovery</td>
<td>Performance Changes</td>
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<tr>
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</table>
| Vaile et al. (2008b)         | N = 12           | Endurance trained cyclists (M) | 4 RM sessions | - 5 consecutive exercise days of 105 min duration consisting of 66 maximal effort sprints plus 9 min cycling TT | Recovery performed on each of the 5 exercise days:  
  - CWI = 14 min full body at 15°C  
  - HWI = 14 min full body at 38°C  
  - CTWT = full body cold (15°C) for 1 min, hot (38°C) 1 min repeated 7 times | Sprint (0.1–2.2%) and TT (0.0–1.7%) performance enhanced across 5 day trial following CWI & CTWT  
  T<sub>r</sub> reduced with CWI  
  No diff in HR |
| Montgomery et al. (2008a)    | N = 29           | Basketball players (M) | 3 day basketball tournament | One game each day | Recovery performed on each of the 5 exercise days:  
  - CHO & stretching (n=9)  
  - CWI at 11°C for 5 x 1 min (n=10)  
  - Full leg compression at 18 mmHg for ~18 h (n=10) | CWI & compression had little benefit on reducing biomarkers (CK, MYO, IL-6, FABP) 6 h after game, but had moderate effect on reducing biomarkers at the end of the competition (3 days)  
  CWI produced acute analgesic effect |
| Montgomery et al. (2008b)    | N = 29           | Basketball players (M) | 3 day basketball tournament | One game each day | Recovery performed on each of the 5 exercise days:  
  - CHO & stretching (n=9)  
  - CWI at 11°C for 5 x 1 min (n=10)  
  - Full leg compression at 18 mmHg for ~18 h (n=10) | CWI better maintained 20 m acceleration (0.5 ± 1.4%) after 3 days compared with compression (3.2 ± 1.6%)  
  CWI similar benefits to maintaining line drill performance as compression  
  CWI resulted in smallest decrement in flexibility |
| Bailey et al. (2007)         | N = 20           | Healthy (M)        | 90 min intermittent shuttle run |  
  - 10 min intermittent shuttle run immediately post-exercise | Recovery performed on each of the 5 exercise days:  
  - CWI = 10 min at 10 ± 0.5°C  
  - Cont = non-immersion passive rest | CWI reduced MS 1, 24 & 48 h post-exercise and resulted in smaller decrements in MVC of KF 24 & 48 h compared with cont  
  CWI reduced MYO 1 h after exercise but did not alter CK |
| Lane and (2007)              | N = 10           | Repeated bouts of HI cycling (18 min) | 15 min immediately post-exercise |  
  - Repeated bouts of HI cycling (18 min)  
  - 15 min immediately post-exercise | Recovery performed on each of the 5 exercise days:  
  - CWI = 10 min at 10 ± 0.5°C  
  - Cont = non-immersion passive rest | Significant decline in total work |
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants</th>
<th>Interventions</th>
<th>Results</th>
</tr>
</thead>
</table>
| Wegner (2004)              | Physically active     | of varying work at resistance of 80g.kg of BW separated by 24 h               | • ACT = cycling at 30% VO$_2$max  
• CWI = immersion of legs in 15°C  
• Cont = seated rest  
completed between exercise bout 1 and 2 only in cont |
| Schniepp et al. (2002)     | N = 10 Well trained   | - 2 RM sessions  
- 2 maximum effort sprints (~30 s) separated by 15 min recovery           | • CWI = 15 min at 12°C up to iliac crest  
• Cont = 15 min seated rest  
Time to PP not difference between conditions  
Max. and average power declined by 13.7 & 9.5% in CWI compared to 4.7 & 2.3% in cont, respectively  
CWI produced greater decline in MHR compared to cont (8.1 V 2.4%) |
| Burke et al. (2000)        | N = 45 (N = 21 F  
N = 24 M)              | - 1 set of 4 repetitions (60% MVC, 70% MVC, 80% MVC, 100% MVC)  
- 2 s build up to MVC and 6 s hold  
- 5 consecutive days of lower limb static contractions  
All prior to isometric strength training | • Cont = 10 min seated rest  
• CWI = 10 min standing in cold batch to gluteal fold (8 ± 1°C)  
• Hot = 10 min standing in hot bath to gluteal fold (43 ± 1°C)  
CWI resulted in greater increase in MVC compared to cont & hot |

M = Male; F = Female; MVC = maximal voluntary contraction; CWI = cold water immersion; TWI = temperate water immersion; HWI = hot water immersion; CTWT = contrast temperature water therapy; EMS = electromyostimulation; ACT = active; CHO = carbohydrate; HR = heart rate; MHR = maximum heart rate; PP = peak power; RM = repeated measures; HI = high-intensity; BW = body weight; VO$_2$max = maximal oxygen consumption; CK = creatine kinase; MYO = myoglobin; IL-6 = interleukin 6; FABP = free-fatty acid building protein; KF = knee flexor; MS = muscle soreness; TT = time trial; VJ = vertical jump; RPE = rate of perceived exertion; $T_{sk}$ = skin temperature.
To further highlight equivocal findings, several studies have also demonstrated negative effects of CWI on the recovery of exercise performance (Crowe et al. 2007; Peiffer et al. 2009a; Schniepp et al. 2002). Indeed, Peiffer et al. (2009a) recently observed decrements in maximal and average sprint power (-9.0% and -7.2%, respectively) following post-exercise CWI (12˚C for 15 min), together with decrements in neuromuscular function. Similarly, post-exercise whole-body CWI (12-14˚C for 15-min) resulted in a reduction in sprint cycling performance, illustrated by a decreased peak and average power of repeated 30-s sprints performed immediately after immersion (Crowe et al. 2007; Schniepp et al. 2002). The authors postulated that the most likely cause of the reduction in cycle sprint power output following CWI was a decrease in the contractile speed of the cooled muscle during maximal contraction (Bigland-Ritchie et al. 1992). Indeed, as previously outlined, cold exposure can increase action potential propagation time in the muscle (Bergh and Ekblom 1979a; Bigland-Ritchie et al. 1992) and decrease dynamic contractile force by 4-6% for each 1˚C decrease in muscle temperature (Bergh and Ekblom 1979a). This reduction in acute anaerobic performance complements earlier data indicating that cooling may have negative effects on anaerobic sprinting-events requiring optimal muscle temperature for high power output (Holewijn and Heus 1992; Sargeant 1987), particularly when exercise bouts are performed in close succession.

Although the effects of CWI recovery on subsequent exercise performance remain equivocal (Bailey et al. 2007; Ingram et al. 2009; King and Duffield 2009), the present literature outlines that the benefits of post-exercise CWI on ensuing performance may be dependent upon the modality of exercise. When longer duration, multi-joint exercise is performed, CWI appears to be beneficial (Bailey et al. 2007; Ingram et al. 2009); however, when shorter bouts of high-intensity exercise are performed (e.g. 30-s sprints), cooler muscle
temperatures resulting from CWI appear to be detrimental to the production of high anaerobic power output (Crowe et al. 2007; Peiffer et al. 2009a; Schniepp et al. 2002).

**Post-Exercise Cold Therapy: The Effects on Signs and Symptoms of EIMD**

EIMD resulting from the eccentric component of intermittent-running and the landing phase during team-sport exercise, or when direct physical collisions occur during exercise results in immediate and prolonged reductions in muscle function, most notably a reduction in force-generating capacity (Byrne et al. 2004; McLellan et al. 2011). Further symptoms of EIMD include increased perceptions of pain and tenderness, muscle stiffness and DOMS (Clarkson et al. 1992; Eston and Peters 1999), which are the most commonly reported components of sport-related injuries (Byrne et al. 2004). Implementation of post-exercise cold therapy has recently indicated beneficial effects on reducing post-exercise muscle soreness after strenuous eccentric muscle activity (Bailey et al. 2007; Eston and Peters 1999; Halson et al. 2008; Vaile et al. 2008c) and team-sport exercise (Yanagisawa et al. 2003a); however, findings remain equivocal on the recovery of skeletal muscle function (Isabell et al. 1992; Paddon-Jones and Quigley 1997; Sellwood et al. 2007).

Previous research has suggested that CWI may be an effective treatment for muscle damage (Eston and Peters 1999; Vaile et al. 2008b, c) by reducing the inflammatory response and consequent secondary hypoxic injury (Takarada 2003; Thompson et al. 2003). Indeed, Eston and Peters (1999) demonstrated beneficial effects of CWI recovery (15 min at 15˚C every 12 h) on reducing plasma CK and muscle stiffness following damage-inducing eccentric exercise of the elbow flexors. Using a similar exercise protocol, Yanagisawa et al. (2003a; 2003b) also reported a reduction in exercise-induced muscle oedema as well as a tendency for reduced muscle soreness and CK activity with post-exercise CWI (15 min at
5˚C). Furthermore, when CWI followed multi-joint prolonged intermittent shuttle running exercise, Bailey et al. (2007) reported a reduction in serum myoglobin response 1-h post-exercise and diminished perceptions of muscle soreness up to 48-h post-exercise. Based on an attenuated decline in peak knee flexion torque, Bailey et al. (2007) postulated that CWI mediated a reduced inflammatory response; thus reducing subsequent secondary muscle damage evidenced by an attenuated efflux of myoglobin. Accordingly, these collective results suggest that when single or multi-joint exercise involves eccentric muscle actions (explicitly or via intermittent shuttle running), post-exercise CWI is beneficial in reducing markers of CK and myoglobin, and reducing perceptions of muscle soreness (Bailey et al. 2007; Eston and Peters 1999).

In contrast, several studies documenting elevations in post-exercise markers of muscle damage following concentric (Halson et al. 2008) and eccentric based exercise (Paddon-Jones and Quigley 1997; Sellwood et al. 2007) have reported no benefits of CWI on reducing symptoms of EIMD. Following a high-intensity, combined fixed- and self-paced cycling protocol, Halson et al. (2008) reported significant increases in CK, CRP, IL-6 and TNF-α; although, the elevation of these variables were not affected by CWI recovery. Furthermore, Ingram et al. (2009) reported no significant difference in the elevation of CK and CRP; despite lower muscle soreness ratings, reduced decrements in isometric leg strength, and faster 10 x 20-m sprint times. In addition, when single-joint bouts of eccentric leg extension (Sellwood et al. 2007) and eccentric elbow flexion (Paddon-Jones and Quigley 1997) were performed, CWI of the exercised limbs offered no benefit in the recovery of muscle performance or subjective muscular pain compared with a control. Furthermore, Paddon-Jones and Quigley (1997) observed DOMS in eight resistance-trained males after performing 64 eccentric actions of the elbow flexors. The results indicated that following 5 x 20 min ice water immersions, with 60 min recovery between each immersion, no significant differences
in muscle soreness, isometric and isokinetic torque, or limb volume were evident between the experimental and control arms over a 96 h recovery period. Similarly, no differences in the perception of muscle soreness were reported following studies using single ice massage applications of 15-20 min duration either immediately, 24 h or 48 when exercise induced DOMS (Gulick et al. 1996; Yackzan et al. 1984). Of note, Isabell et al. (1992) observed no effect of ice massage on improving indices of skeletal muscle damage and suggested that repeated cryotherapy may actually be contra-indicatory to the prolonged repair and adaptation of skeletal muscle function. Therefore, it appears that the effects of various modalities of post-exercise cold application following single- and multi-joint exercise inducing prolonged muscle damage demonstrate equivocal findings on reducing perceptions of pain and MS, reducing cytokine and endocrine responses. Interestingly, of the studies reporting no significant benefit on reducing the symptoms of EIMD, either ice water immersion (Padden-Jones and Quigley 1997; Sellwood et al. 2007) or ice massage (Gulick et al. 1996; Yackzan et al. 1984) have been implemented as the post-exercise intervention; resulting in potential detrimental effects on prolonged muscle repair and adaptation observed (Isabell et al. 1992). In contrast, when post-exercise recovery consists of cold water immersion following either single or multi-joint exercise eliciting significant muscle damage, perceptions of MS, concentrations of CK and myoglobin, and recovery of voluntary torque were all enhanced (Bailey et al. 2007; Eston and Peters 1999; Yanagisawa et al. 2003b). Thus, it may be suggested that the colder temperatures of ice therapy were of no benefit, and possibly detrimental to the recovery of symptoms of EIMD compared to post-exercise cold therapy. Furthermore, of the studies demonstrating no beneficial effect on attenuating the elevation of endocrine and cytokine responses (Halson et al. 2008; Ingram et al. 2009), the exercise modality to recovery from primarily consisted of concentric-based exercise and thus the level of reductions in exercise performance may not
have been sufficient to observe any significant benefits of post-exercise CWI. However, as Ingram et al. (2009) observed reduced decrements in isometric voluntary torque and faster 10 x 20-m sprint time following CWI, further research examining the effects of post-exercise cold therapy after exercise indicative of team-sports is required.

**Post-Exercise Cold Therapy: Effects following Exercise in the Heat**

Given that many athletic events may occur in warm climates and the increased physiological perturbations associated with exercise in the heat; cooling strategies to counter the acute and prolonged effects of exercise-induced elevations in heat stress have become popular (Marino 2002; Quod et al. 2006). Despite such association, research detailing the efficacy of CWI on the recovery from exercise-induced heat stress in particular remains relatively novel (Peiffer et al. 2009a; Peiffer et al. 2010a, b; Vaile et al. 2008a; Vaile et al. 2010). Previous research has demonstrated that cooling hyperthermic athletes in various water temperatures (2-20˚C) results in safe and efficient cooling to reduce thermoregulatory load (Proulx et al. 2006). CWI applied after a bout of exercise in the heat can decrease $T_{core}$ at a rate that is faster than heat loss occurring under normal convective conditions (Clements et al. 2002; Mitchell et al. 2001; Peiffer et al. 2009a; Yeargin et al. 2006). Recently, Vaile et al. (2008b) and Yeargin et al. (2006) have shown that CWI used immediately after exercise can result in a greater decrease in rectal temperature ($T_{re}$). Yeargin et al. (2006) used a 12 min CWI intervention (14˚C) 4 min after the termination of exercise in the heat and observed a mean core cooling rate of 0.08˚C min$^{-1}$. In addition, immediate post-exercise cooling resulted in a mean core cooling rate of 0.05˚C min$^{-1}$ after 15 min of CWI at 14˚C (Vaile et al. 2008b). The greater cooling rates observed by Vaile et al. (2008b) and Yeargin et al. (2006) were postulated to be due to peripheral blood pooling that might have occurred immediately.
following exercise, potentially contributing to the greater core cooling. As such, implementation of post-exercise cooling following exercise in the heat is a safe and efficient mode of reducing the rise in $T_{\text{core}}$. Provided in Table 2.3 is a summary of the relevant literature pertaining to the current literature examining the effects of post-exercise cold therapy on recovery following exercise-induced elevations in thermal strain.

CWI has been implicated to have a positive effect on reducing exercise- and passively-induced elevations in $T_{\text{core}}$ with concomitant increases in exercise performance upon the return of body temperature to resting values (Morrison et al. 2004; Peiffer et al. 2010b; Thomas et al. 2006; Vaile et al. 2008a). Indeed, the return to optimal performance of voluntary force and activation were facilitated when cooling methods were introduced to speed the rate of reduction in $T_{\text{core}}$ (Morrison et al. 2004; Thomas et al. 2006). Thomas et al. (2006) demonstrated that passively-induced elevations in $T_{\text{core}}$ resulted in a reduction in voluntary force production. However, torque and VA were restored to baseline values only when $T_{\text{core}}$ was lowered back to normal values via a cooling garment. In agreement, Morrison et al. (2004) observed an 13% and 11% reduction in MVC and VA, respectively when subjects were passively heated to 39.4˚C. Only once cooling lowered $T_{\text{core}}$ back to resting values did VA and MVC return to baseline values. Morrison et al. (2004) postulated that the reduction in MVC and VA was directly influenced by an increase in $T_{\text{core}}$ and thus the faster rate of reduction in $T_{\text{core}}$ with cooling resulted in a greater recovery of force production and activation.
### Table 2.3 Summary of Literature Examining Post-Exercise Cold Water Immersion Following Exercise in the Heat

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Exercise Protocol</th>
<th>Environmental Temperature</th>
<th>Intervention</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peiffer et al. (2010a)</td>
<td>N = 10 Male cyclists</td>
<td>- 2 RM sessions</td>
<td>35°C 40% RH</td>
<td>• 15 min seated recovery (Cont) • 5 min CWI</td>
<td>- $T_{re}$ lower in CWI (0.5 ± 0.4°C) • PO greater in second TT with CWI (327.9 ± 55.7 W) compared to Cont (288.0 ± 58.8 W)</td>
</tr>
<tr>
<td>Peiffer et al. (2010b)</td>
<td>N = 10 Male cyclists</td>
<td>- 2 RM sessions</td>
<td>35.0 ± 0.3°C 40.0 ± 3.0% RH</td>
<td>• 5 min CWI (14°C) • 20 min seated in room air (Cont; 35°C)</td>
<td>- No change in $T_{re}$ • After second TT $T_{re}$ lower in CWI V Cont • No diff. in maximal isokinetic concentric torque</td>
</tr>
<tr>
<td>Vaile et al. (2010)</td>
<td>N = 10 Endurance trained male cyclists</td>
<td>- 2 RM sessions</td>
<td>32.8 ± 1.1°C 43.6 ± 1.8% RH</td>
<td>• 15 min ACT (cycling at 40% of PPO) • 15 min full-body CWI (15°C)</td>
<td>- Significant decline in performance with ACT but not with CWI • $T_{re}$ reduced immediately after CWI and at the end of 40 min passive recovery until the end of exercise bout 2 • Reduced limb blood flow (leg &amp; arm) and HR with CWI</td>
</tr>
<tr>
<td>Peiffer et al. (2009b)</td>
<td>N = 12 Male cyclists</td>
<td>- 4 RM sessions</td>
<td>40°C 40% RH</td>
<td>• 5, 10 or 20 min CWI (14°C) • 20 min seated at room temperature (24°C)</td>
<td>- Greater rate of reduction in $T_{re}$ with CWI • $T_{mus}$ reduced with 45 min after CWI • Time to exhaustion lower for all water immersion durations • $T_{mus}$ lower following 10 &amp; 20 min immersion compared to 5 min • Isometric and isokinetic torque not altered by any duration of CWI</td>
</tr>
<tr>
<td>Peiffer et al. (2009a)</td>
<td>N = 10 Well trained male cyclists</td>
<td>- 2 RM sessions</td>
<td>32.2 ± 0.7°C 55.0 ± 2.4% RH</td>
<td>• 20 min CWI (14°C) • 20 min passive rest (Cont; 24°C)</td>
<td>- $T_{re}$ reduced in CWI 50 min post-TT • $T_{re}$ reduced with CWI • Greater reduction in post-TT MVC and SMVC with CWI compared to Cont • 9% smaller femoral vein diameter with CWI</td>
</tr>
<tr>
<td>Buchheit et al. (2009)</td>
<td>N = 10 Male cyclists</td>
<td>- 2 x supramaximal cycling interspersed by 20 min passive recovery in which time 5 min of CWI or passive seating was performed</td>
<td>35.0 ± 0.3°C 40.0 ± 3.0% RH</td>
<td>• 5 min CWI at 14°C • 5 min seated rest in 35°C, 40% RH (cont)</td>
<td>- No effect of CWI on $T_{re}$ cycling performance of HR recovery • Vagal-related HR variability decreased after exercise 1 and further after exercise 2 under cont but not CWI</td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
<td>Protocol Details</td>
<td>Results</td>
<td></td>
<td></td>
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</tbody>
</table>
| Vaile et al. (2008a)         | N = 10       | Well trained male cyclists, 5 RM sessions, 30 min cycling task, 15 min CP at 75% PPO, 15 min TT, 15 min recovery, 40 min passive rest, repeat 30 min cycling task | • Intermittent (5 x 1 min in, 2 min out) CWI  
  o 10°C  
  o 15°C  
  o 20°C  
• Continuous CWI at 20°C  
• ACT cycling (40% VO2peak; 31.1 ± 2.6°C, 48.0 ± 4.2% RH)  
- ACT resulted in 4.1 ± 1.8% less total work completed in second exercise task. No change in work with CWI  
- CWI reduced thermal strain, mean Tb and RPE |
| Halson et al. (2008)         | N = 11       | Endurance trained male cyclists, 2 RM sessions, ~40 min simulated TT, 3 x CWI at 11.5°C for 60 s, Passive rest in 24.2 ± 1.8°C.  
(Cont) | • 3 x CWI at 11.5°C for 60 s  
  • Passive rest in 24.2 ± 1.8°C  
  • CWI reduced HR, Tb, Tsk  
  • No diff. between CWI and Cont for La, glucose, pH, catecholamines, cortisol, testosterone, CK, CRP, IL-6, IGL-1  
- CWI resulted in a faster 2 mile performance time compared to Mock  
- CWI and IWI reduced Tsh and HR |
| Yeargin et al. (2006)        | N = 15       | High trained, heat acclimated distance runners, 3 RM sessions, ~90 min hilly trail run, 12 min recovery, 30 min passive rest, 2 mile race, 13.98°C  
(Cont) | • CWI = 13.98°C  
  • IWI = 5.23°C  
  • Mock = seated in tub with no water at 29.5°C room temp.  
- CWI resulted in a faster 2 mile performance time compared to Mock  
- CWI and IWI reduced Tsh and HR |
| Clements et al. (2002)       | N = 17       | Highly trained, heat acclimated distance runners, 3 RM sessions, ~19 km hilly trail run (~86 min), 12 min recovery, 27 ± 1°C  
(Cont) | • CWI = 14.03 ± 0.28°C  
  • IWI = 5.15 ± 0.20°C  
  • Mock = no water, 28.88 ± 0.26°C  
- No diff. in cooling rates found at start of immersions until 8 min  
- CWI & IWI provided similar cooling rates which were greater than mock at 8-12 min  
- Tsh similar between CWI & IWI at end of immersion  
- IWI reduced Tsh at 6-10 min post-immersion compared to CWI and mock |

Tb = rectal temperature; Tmus = muscle temperature; Tb = body temperature; Tsk = skin temperature; CWI = cold water immersion; IWI = ice water immersion; ACT = active; PO = power output; PPO = peak power output; TT = time trial; RM = repeated measures; CP = constant paced; RH = relative humidity; HR = heart rate; MVC = maximal voluntary contraction; SMVC = superimposed maximal voluntary contraction; VO2peak = peak oxygen consumption; RPE = rate of perceived exertion; La = lactate; CK = creatine kinase; CRP = c-reactive protein; IL-6 = interleukin 6; IGL-1 = insulin-like growth factor 1; W = women; M = men
Although the aforementioned studies are related to passively-induced elevations in $T_{\text{core}}$, similar positive effects of cooling following exercise-induced increases in $T_{\text{core}}$ have been observed (Peiffer et al. 2010b; Vaile et al. 2008a; Vaile et al. 2010; Yeargin et al. 2006). Yeargin et al. (2006) demonstrated that 12 min of whole-body CWI (14°C) after 90 min of running in the heat significantly reduced the time to complete a 2-mile running time trial compared with a control condition. An improvement of 6% in performance times observed by Yeargin et al. (2006) is similar to the results previously mentioned by Marsh and Sleivert (1999), and Olschewski and Bruck (1988), and Lee and Haymes (1995) during constant-paced exercise. The improved running performance occurred with a mean rectal temperature ($T_{\text{re}}$) that was 0.5°C lower than in the control condition. Peiffer et al. (2010a) demonstrated a comparable 0.6°C reduction in $T_{\text{re}}$ with CWI prior to a second cycling time trial and observed a significantly greater power output resulting in a faster completion of the subsequent 4-km cycling time-trial. In addition, RPE during the second constant-pace session was significantly lower after CWI and paralleled the reduction in $T_{\text{re}}$ (Peiffer et al. 2010a). These studies highlight that effective reduction in $T_{\text{core}}$ with cooling following exercise in the heat result in a concomitant enhancement of subsequent exercise performance, particularly in warm thermal environments.

Similarly, Vaile et al. (2008a) found that CWI performed between two high-intensity cycling bouts maintained repeated performance in hot environmental conditions compared with active recovery. More recently, Vaile et al. (2010) demonstrated similar results with post-exercise CWI maintaining performance during high-intensity cycling in hot conditions compared to an active recovery. Vaile et al. (2008a; 2010) postulated that a reduction in mean body temperature and heart rate following all CWI protocols may have resulted in a decrease in peripheral blood flow and therefore produced a greater volume of blood available centrally or to the working muscle (Lee and Haymes 1995; Marsh and Sleivert 1999) resulting in
improved exercise performance. The faster performance times may also support the suggestions by Marino (2002) that CWI may attenuate the reduction in motor recruitment allowing the maintenance of a higher exercise intensity during subsequent exercise. Growing evidence indicates a complex role of central regulation of voluntary muscle activation, and therefore performance, in response or anticipation of the internal thermal load (Marino 2004; Tucker et al. 2004). Accordingly, the act of cooling may alleviate this thermal load via reductions in body temperature, and allow an increase in the activation and recruitment of muscle force (Marino 2004; Tucker et al. 2004), thereby improving recovery and subsequent exercise performance in hot conditions. Cooling following passively-induced increases in thermal stress have highlighted increased voluntary force production and activation when \( T_{\text{core}} \) values were returned to resting (Morrison et al. 2004; Thomas et al. 2006). However, there is currently a paucity of research examining the relationship between cooling reducing \( T_{\text{core}} \) and concomitant enhancement of voluntary force and activation contributing to improvements in subsequent exercise performance after exercise-induced elevations in thermal stress (Peiffer et al. 2009a), particularly relating to team-sports performance.

In addition, observed improvements in exercise performance following CWI after exercise in hot environmental conditions may also be a result of improved perceptions of thermal sensation (Vaile et al. 2008a; White et al. 2002), reductions in post-exercise heart rate and cardiovascular strain (Arngrimsson et al. 2004; Yeargin et al. 2006) potentially contributing to increased central motor drive. Vaile et al. (2008a) observed a significantly reduced heart rate during 40 min of passive rest in the heat following all CWI protocols compared with active recovery. Similarly, Halson et al. (2008) demonstrated an immediate 3-15% decrease in heart rate following CWI and suggested that the decline resulted from an increase in central blood volume expansion and an increase in stroke volume. In support, Buchheit et al. (2009) demonstrated that CWI implemented before and after supramaximal
exercise in the heat (35.0 ± 0.3°C; 40.0 ± 3.0% RH) accelerated the restoration in parasympathetic function, likely in response to vasoconstriction and hydrostatic pressure of CWI activating cardiopulmonary baroreceptors thus enhancing vagal nerve activity and inhibiting sympathetic activation. In accordance, Vaile et al. (2010) more recently demonstrated significant reductions in $T_{re}$, leg blood flow, arm blood flow and heart rate following CWI compared with active recovery. Furthermore subsequent cycling performance was reduced following active (-1.8%) but maintained following CWI. The authors postulated that the resultant reduction in thermal and cardiovascular strain with CWI contributed to the improved subsequent exercise performance (Halson et al. 2008; Vaile et al. 2008a; Vaile et al. 2010). Whilst the attenuation in thermal strain with cooling is well documented to improve subsequent exercise performance following passively-induced elevations in $T_{core}$ (Morrison et al. 2004; Thomas et al. 2006), minimal research has focused on the relationship between cooling reducing exercise-induced elevations in thermal stress and the subsequent effect on exercise performance (Duffield et al. 2009b). Further, the relationship between cooling reducing cardiovascular strain following exercise in the heat contributing to increased central motor drive and thus improving subsequent exercise performance remains unknown.

Collectively, the aforementioned studies have reported beneficial effects of CWI recovery following exercise in the heat; however, contrasting findings demonstrating negative effects of cooling on exercise performance have also been observed (Crowley et al. 1991; Peiffer et al. 2009a; Schniepp et al. 2002). For example, Peiffer et al. (2009a) recently demonstrated that despite significant reductions in skin and $T_{re}$ following CWI recovery after 90 min cycling in the heat, greater decreases in MVC and VA were evident compared with a control condition. A 13% decrease in maximal isometric force was observed immediately post-time trial and suggests that post-exercise CWI impaired muscle function when assessed immediately post-CWI. A decrease in vessel diameter immediately post-exercise, with a
further decrease following CWI lasting for 90 min after the time-trial (TT) was observed in Peiffer et al. (2009a), which the authors suggested was likely a vasonconstrictive response to the cold exposure (Park et al. 1999). Peiffer et al. (2009a) speculated that the magnitude of the decrease in vessel diameter after CWI (~12%) would have lead to a decrease in femoral vessel blood flow (~24%) away from the recovering muscle (Walther et al. 2006), suggesting a possible negative effect CWI in this case. An explanation for observed reductions in post-CWI MVC may relate to the direct association between lowered $T_{\text{mus}}$ reducing muscular force production (Bergh and Ekblom 1979a; Oska et al. 1997). Therefore, Peiffer et al. (2009a) speculated that the decrease in muscle force output observed in their study was due to lower $T_{\text{mus}}$ after the 20 min of CWI.

Similarly, Cheung and Sleivert (2004) examined the effect of post-exercise whole-body cooling (cooling garment) on isokinetic knee extension torque. Isokinetic torque was decreased by 10% irrespective of changes in $T_{\text{core}}$. The authors postulated that the reduction in torque was most likely due to decreases in $T_{\text{mus}}$ (Bergh and Ekblom 1979a). However, this conclusion is difficult to verify as the participants in this study had thermoneutral (<38˚C) core temperatures. Although, it is well-documented that exposure to CWI can rapidly decrease muscle temperature with concomitant reduction in muscular force output (Bergh and Ekblom 1979a; Howard Jr et al. 1994). Under these cooler muscle temperatures, additional motor units must be recruited to produce similar levels of muscular force output (Rome 1990). As such, without an adequate warm-up, force production and power output immediately following CWI may be impaired (Bergh and Ekblom 1979a). Similar findings have also been reported in non-hyperthermic conditions, with reductions in peak and average power output during the second of two cycle sprints performed in <27˚C after 15 min of CWI (Crowe et al. 2007; Schniepp et al. 2002).
In conclusion, the results of the outlined studies highlight that the benefits of post-exercise CWI following exercise in the heat may be time dependent. As such, when maximal force and/or power is required immediately following CWI after exercise in the heat, muscle force output is reduced (Peiffer et al. 2009a), likely due to colder muscle temperatures inhibiting contractile function (Bergh and Ekblom 1979a; Oska et al. 1997). However, when subsequent exercise performance is completed with a prior adequate warm-up or the duration between exercise bouts is extended after CWI, the enhanced reduction in T_{core} and thus cardiovascular and thermal strain results in improved exercise performance (Vaile et al. 2008a; Vaile et al. 2010). Although this is a common observation in the current literature, there remains a paucity of research examining the potential relationship between reductions in post-CWI T_{core} influencing central motor drive to explain observed improvements in subsequent exercise performance. More specifically, this potential neuromuscular mechanism has not been evaluated following team-sport exercise in the heat with post-exercise CWI.

Post-Exercise Cold Therapy: The Effects following Collision-Based Exercise (direct and non-direct)

In addition to team-sport athletes being required to perform in warm environmental conditions, sports including rugby league, rugby union, Australian and American Football and soccer also involve exogenous load consisting of regular indirect and direct physical collisions between opposing players throughout the course of training and/or match-play (Brewer and Davis 1995; Gissane et al. 2001). The combative nature of such sports, combining intermittent high-intensity activity and repeated blunt force trauma, may result in micro-damage to skeletal muscle and post-exercise muscle soreness, in excess of the normal rigours of team-sport demands (Dawson et al. 2004; McLellan et al. 2010). Accordingly, post-exercise CWI has become increasingly popular following collision-based exercise
despite a paucity of research fully elucidating the benefits following high-intensity physical collision sports (Banfi et al. 2007; Rowsell et al. 2009; Rowsell et al. 2011). Although Gill et al. (2006) previously examined the specific effects of post-exercise recovery strategies (contrast water therapy, compression garments, active and passive recovery) in rugby players, Banfi et al. (2007) is the only investigation to examine the effects of CWI recovery following rugby (collision-based) exercise. Whilst no performance results were reported, CWI stabilised CK values, when combined with an initial active recovery. The combined effect of post-exercise CWI and active recovery has also been examined following 90-min of soccer match-play (Kinugasa and Kilding 2009). Although CWI combined with active recovery (cycling) resulted in enhanced perceptions of recovery, no significant effect was observed for VJ height (Kinugasa and Kilding 2009). These results are in accordance with Rowsell et al. (2009) who reported reduced perceptions of leg soreness and general fatigue with CWI recovery during a 4-day soccer tournament, without any improvement in physical performance (CMJ performance and repeated sprint time). Similarly, Ascensao et al. (2011) reported that CWI following a one-off soccer match, reduced 24-h soreness and attenuated increases in CK, myoglobin and CRP concentrations. As such, the results of the aforementioned studies demonstrate beneficial effects of CWI when combined with active recovery on enhancing perceptions of recovery following team-sport exercise. Although recent evidence following simulated team-sport exercise indicates potential benefits of CWI recovery (Ingram et al. 2009), there remains a paucity of evidence examining the specific effects of CWI alone on physiological, performance and perceptual recovery following direct collision-based team-sport exercise. As a summary, Table 2.4 outlines the current literature examining the effects of various recovery strategies following bouts involving collision-based (team-sport) exercise.
### Table 2.4 Summary of Literature Examining Post-Exercise Recovery Strategies Following Collision-Based Exercise

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Methods</th>
<th>Interventions</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rowsell et al. (2011)</td>
<td>N = 20 Junior elite soccer players (M)</td>
<td>- 4-day soccer tournament</td>
<td>• CWI (n=10) = 5 x 1 min at 10°C (1 min seated at 24°C)</td>
<td>- CWI reduced perceptions of leg soreness and general fatigue, and attenuated the decline in total distance run compared to TWI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- One full match each day</td>
<td>• TWI (n=10) = 5 x 1 min at 34°C, 1 min seated at 24°C</td>
<td></td>
</tr>
<tr>
<td>Ascensao et al. (2011)</td>
<td>N = 20 Junior Soccer (M)</td>
<td>- One-off friendly soccer match</td>
<td>• CWI = 10 min at 10°C (n=10)</td>
<td>- CWI reduced CK, MYO, CRP and adductor MS 30 min post match</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• TWI = 10 min at 35°C (n=10)</td>
<td>- CK, CRP, quadriceps strength &amp; perceptions of MS were reduced 24 h post-exercise with CK &amp; CRP remaining lower at 48 h post-exercise with CWI compared to TWI</td>
</tr>
<tr>
<td>Rowsell et al. (2009)</td>
<td>N = 20 Junior elite soccer players</td>
<td>- 4- day simulated soccer tournament</td>
<td>• CWI = 20 min at 10 ± 0.5°C</td>
<td>- CWI reduced perceptions of leg soreness and general fatigue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- One match per day</td>
<td>• TWI = 20 min at 34 ± 0.5°C</td>
<td>- No change in CK &amp; LDH</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>- No change in physical test performance (CMJ, 12 x 20m RSA)</td>
</tr>
<tr>
<td>Kinugasa and Kilding (2009)</td>
<td>N = 28 Junior Soccer (M)</td>
<td>- 3 soccer matches played on separate days with a recovery after each match</td>
<td>• Passive = static stretching (7 min) &amp; sustained leg raise (2 min)</td>
<td>- Immediate perceived quality of recovery higher after Comb compared to CTWT &amp; passive</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• CTWT = 1 min immersion to mesosternale in cold (12°C), followed by hot shower (38°C) for 2 min repeated 3 times</td>
<td>- Perceived lighter legs with Comb compared to passive at 24 h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Comb = CWI for 1 min, followed by ACT cycle (60-80 rpm, 90-110 W) for 2 min repeated 3 times</td>
<td>- No effect on VJ</td>
</tr>
<tr>
<td>Banfi et al. (2007)</td>
<td>N = 30 National rugby</td>
<td>- Elite rugby union training sessions</td>
<td>• Passive rest</td>
<td>- ACT followed by CWI stabilised CK activity</td>
</tr>
<tr>
<td>Gill et al. (2006)</td>
<td>N = 23 Elite rugby players (M)</td>
<td>- Four competition weeks of the New Zealand National Provincial Championship were monitored. - Randomly assigned recovery performed immediate post-match</td>
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<tr>
<td></td>
<td></td>
<td>• ACT = 10 min cycling at 180 W followed by CWI of legs for 10 min</td>
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<tr>
<td></td>
<td></td>
<td>• CWI = 10 min immersion of the legs followed by 10 min ACT</td>
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<td></td>
<td></td>
<td>• Passive = 9 min seated rest</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>• ACT = 7 min low-intensity cycle exercise (80-100 rpm, ~150 W)</td>
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<td></td>
<td></td>
<td>• CTWT = Alternating between immersion in cold water (8-10°C) for 1 min and hot water (40-42°C) for 2 min for total ~9 min</td>
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<td></td>
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<tr>
<td></td>
<td></td>
<td>• Compression garment = Lower body compression garment (Skins®) for ~12 h</td>
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<tr>
<td></td>
<td></td>
<td>- Passive recovery resulted in significantly less CK recovery compared to ACT, CTWT and Compression. - No diff observed in CK activity between ACT, CTWT and compression</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Dawson et al. (2005)</th>
<th>N = 12 AFL</th>
<th>- 12 semi-professional AFL matches - Next day training performance after game</th>
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<tr>
<td></td>
<td></td>
<td>• Control = passive rest</td>
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<tr>
<td></td>
<td></td>
<td>• Stretch = 15 min gentle static stretch of legs &amp; back</td>
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<tr>
<td></td>
<td></td>
<td>• Pool walking = 15 min easy walking in 28°C pool</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Hot/cold = standing in hot (~45°C) shower for 2 min followed by standing in waist deep cold water (~12°C) for 1 min repeated 5 times</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- No diff in MS ratings between any conditions - VJ, 6-s work and power significantly reduced 15 h from baseline in control only</td>
</tr>
</tbody>
</table>

RSA = repeated sprint ability; CMJ = countermovement jump; VJ = vertical jump; AFL = Australian Football League; LDH = lactate dehydrogenase; CK = creatine kinase; MYO = myoglobin; CRP = c-reactive protein; MS = muscle soreness; M = male; CWI = cold water immersion; TWI = temperate water immersion, CTWT = contrast temperature water therapy; ACT = active
Furthermore, although the specific effects of CWI was not investigated, Gill et al. (2006) and Dawson et al. (2005) examined the effect of various recovery modalities following physical collision sports of rugby and AFL match-play, respectively. Gill et al. (2006) examined four recovery interventions (passive, contrast water therapy, compression garments and active recovery) on the rate and magnitude of muscle damage recovery following competitive rugby matches. CK activity was significantly increased post-match with the magnitude of recovery significantly worse in passive recovery compared to the other modalities. Similarly, Dawson et al. (2005) reported that measures of vertical jump, 6-s cycling work and power following AFL match-play were only significantly lower than baseline values in the control condition (passive recovery) compared to stretching, pool walking and contrast water therapy. As such, these findings collectively demonstrate that recovery with active, contrast water therapy, compression garments or stretching following collision-based exercise is superior in ameliorating perceptual and performance variables compared to a passive recovery. Although these studies did not specifically evaluate the effects of CWI following collision-based exercise, the results do highlight that recovery modalities are superior to maintaining performance and improving perceptions of recovery compared to passive rest. As such, with CWI commonly implemented in an athletic setting following collision-based exercise (rugby league, union, AFL etc), due to the paucity of research examining the specific effects of post-exercise CWI in such a setting, further research is warranted.

Post-Exercise Cold Therapy: The Effects on Perceptions of Recovery

A common observation in the literature implementing CWI recovery following multi-joint exercise is the improvement in subjective reports of heightened recovery (Halson et al.
Perceptions of general fatigue, muscle soreness and DOMS are commonly reported to be reduced following CWI (Halson et al. 2008; Rowsell et al. 2009; Vaile et al. 2008b). Indeed, Halson et al. (2008) reported lower ratings of general fatigue and leg soreness, together with an increased perception of physical and mental recovery after CWI was implemented following a simulated 20-min cycling time-trial. Similarly, Rowsell et al. (2009) reported that following CWI, athletes reported less general fatigue and leg soreness over the duration of 4-day soccer tournament compared with thermoneutral water immersion.

Potential mechanisms to explain reduced perceptions of MS and DOMS following CWI have been related to the analgesic effect of cold therapy (Meeusen and Lievens 1986). Whilst this mechanism is plausible, the duration of such analgesia is limited to 1-3 h post-CWI (Meeusen and Lievens 1986). Currently, the precise mechanisms to explain prolonged reductions in MS with CWI (24 - 48 h) have been speculative and relate to a reduction in the extent of skeletal muscle damage as a result of CWI (Eston and Peters 1999). However, to date no research has fully elucidated this potential mechanism. An alternative mechanism to explain prolonged reductions in MS following CWI may also relate to the potential placebo effect of the intervention (Beedie 2007; Wilcock et al. 2006). Indeed, of the studies demonstrating enhanced perceptions of MS with CWI, no study incorporated a placebo control into their investigation and thus a treatment effect cannot be dismissed (Bailey et al. 2007; Halson et al. 2008; Rowsell et al. 2009). Whilst the potential placebo effect cannot be discounted, it is difficult to incorporate a placebo control to minimise the potential treatment effect of such an intervention. Regardless, for athletic performance, it has previously been demonstrated that athletes perform better when they believe they received beneficial treatment (Beedie 2007; Clark et al. 2000). Although a placebo effect of CWI may results in reductions perceptions of prolonged MS and fatigue, more importantly, the relationship between enhanced perceptions of recovery with CWI influencing subsequent athletic performance remains to be elucidated.
CONCLUSION

Team-sport exercise commonly induces prolonged reductions in muscle function and increased perceptions of MS and fatigue. When such exercise is performed with additional exogenous load, including competing in the heat or exposure to repeated intense physical collisions, the extent of the reductions in muscle function, EIMD and increases in physiological load can be more pronounced. As such, acute and prolonged exercise performance is often reduced until appropriate recovery back to optimal physiological functioning occurs. Therefore, many team-sports implement post-exercise CWI in an effort to minimise deleterious symptoms associated with training and competition, aiming to ensure that performance in subsequent bouts of exercise is optimal. Although recent evidence highlights beneficial effects of CWI on reducing physiological load and enhancing subsequent exercise performance, the precise mechanisms influencing such improvements have not been fully elucidated. In particular, the effect and subsequent influence post-exercise CWI has on altering central and peripheral neuromuscular function following exercise indicative of team-sports remains unknown. Therefore, this thesis aims to evaluate the effect of post-exercise CWI on the recovery of neuromuscular function following exercise modalities common to team-sports. Specifically, the effects of post-exercise cold therapy following EIMD will firstly be examined. Following this, implementation of CWI following intermittent-sprint exercise in the heat and involving direct physical collisions, respectively will be evaluated.
CHAPTER 3

Cold Application for Neuromuscular Recovery Following Intense Lower-Body Exercise

As accepted for publication into the European Journal of Applied Physiology.

ABSTRACT

This study examined the effects of cold therapy (COLD) on recovery of voluntary and evoked contractile properties following high-intensity, muscle-damaging and fatiguing exercise. Ten resistance-trained males performed 6 x 25 maximal concentric/eccentric muscle contractions of the dominant knee extensors (KE) followed by a 20-min recovery (COLD v control) in a randomized cross-over design. Voluntary and evoked neuromuscular properties of the right KE, ratings of perceived muscle soreness (MS) and pain, and blood markers for muscle damage were measured pre- and post-exercise, and immediately post-recovery, 2-h, 24-h and 48-h post-recovery. Exercise resulted in decrements in voluntary and evoked torque, increased MS and elevated muscle damage markers ($P<0.05$). Measures of maximal voluntary contraction (MVC) or voluntary activation (VA) were not significantly enhanced by COLD ($P>0.05$). Activation of right KE decreased post-exercise with increased activation of biceps femoris (BF) ($P<0.05$). However, no significant differences were evident between conditions of activation of KE and hamstrings at any time point ($P>0.05$). No significant differences were observed between conditions for creatine kinase or asparate aminotransferase ($P>0.05$). However, perceptual ratings of pain were significantly ($P<0.05$) lower following COLD compared to control. In conclusion, following damage to the contractile apparatus, COLD did not significantly hasten the recovery of peripheral contractile trauma. Despite no beneficial effect of COLD on recovery of MVC, perceptions of pain were reduced following COLD.
INTRODUCTION
Cold therapy is an accepted form of treatment for acute soft tissue injury to help alleviate pain, muscle soreness (Bailey et al. 2007) and inflammation (Yanagisawa et al. 2003b). More recently, cold therapy has been utilised in many sports as a post-exercise recovery strategy to reduce potential detrimental effects of soreness and damage as a result of training and competition (Wilcock et al. 2006). With athletes often involved in repeated bouts of high-intensity training and/or competition performed within 48 h or over consecutive days, the potential for inducing sustained muscle damage can be augmented. Therefore, to ensure that subsequent training sessions are performed with maximal effort, it is common for athletes to engage in post-exercise recovery strategies. In particular, cold therapy is commonly used as a recovery strategy aiming to alleviate and/or minimise possible perturbations in performance associated with training and competition bouts (Wilcock et al. 2006).

Evidence outlining the performance and perceptual effects of cold therapy following exercise-induced muscle damage (EIMD) are varied (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009; Vaile et al. 2008c). Vaile et al. (2008c) recently reported that following exercise-induced delayed onset muscle soreness (DOMS), isometric strength and squat jump performance was improved with cold water immersion (CWI) recovery. Improvement in the recovery of voluntary force and a reduction in perceived muscle soreness was also demonstrated following prolonged intermittent shuttle running which induced symptoms of muscle damage (Bailey et al. 2007). Despite the aforementioned studies demonstrating beneficial effects of CWI, Jakeman et al. (2009) recently reported that CWI following a damaging bout of exercise had no beneficial effect on recovery of maximal voluntary force (MVC) or perceived soreness. Interestingly, Sellwood et al. (2007) reported that ice-water immersion did not minimise markers of DOMS, including perception of pain and tenderness, with ratings of pain increased 24-h post-recovery. As such, evidence that cold
therapy maintains or improves recovery of performance and perceptions of soreness remains equivocal (Bailey et al. 2007; Eston and Peters 1999; Jakeman et al. 2009; Vaile et al. 2008c).

To date few studies demonstrating beneficial effects of cold therapy have been able to relate the improvement in performance or perceptual recovery to physiological, immunological, hematological or neuromuscular mechanisms (Halson et al. 2008). More specifically, data on the efficacy of cold therapy on neuromuscular recovery is limited (Peiffer et al. 2009a). Following DOMS-inducing exercise, significant reductions in maximal voluntary force have been reported (Brown et al. 1997; Paddon-Jones and Quigley 1997), which have primarily been attributed to peripheral muscle fatigue (Sayers et al. 2003). However, data describing the role of centrally-mediated fatigue mechanisms following eccentric exercise is limited (Behm et al. 2001; Pasquet et al. 2000). Therefore, the aim of this investigation was to determine the effects of cold therapy on the recovery of voluntary and evoked contractile properties following high-intensity eccentric exercise designed to result in contractile damage and perceived soreness. As cold therapy is well-known to reduce acute inflammatory responses (Bailey et al. 2007; Eston and Peters 1999) it was hypothesised that potential improvements in performance and perceptual recovery following a bout of EIMD would be present due to COLD recovery.

**METHODS**

**Participants**

Ten resistance trained male athletes (mean ± SD) aged 21 ± 1.6 yr, height 182.2 ± 3.6 cm and body mass 87.3 ± 9.3 kg were recruited as participants. At the time of testing, participants regularly completed upper and lower body strength training 3-4 times per week for the past 4.4 ± 1.1 y and competed in team sport competition and training (rugby league/union) at least twice per week. Testing was completed during the mid-season of
competition so the potential for the exercise to be unaccustomed was minimized. All participants were informed of the requirements of the study and verbal and written consent was gained prior to the commencement of testing. Human ethics clearance was granted by the Institutional Ethics Committee prior to the completion of any testing procedures.

**Overview**

Participants were required to undertake a familiarization session followed by two experimental sessions in a randomized, crossover design. The two sessions were identical apart from the implementation of the recovery intervention. Each session consisted of an exercise protocol of single-leg maximal concentric/eccentric contractions followed by a cold therapy (ice cuffs) or a control condition (no recovery) intervention. Neuromuscular performance was measured pre-exercise, post-exercise, post-recovery and again 2-, 24- and 48-h post-recovery. Venous blood samples were collected for markers of muscle damage and inflammation; whilst perceptual ratings of soreness and pain were also recorded at each of the aforementioned time points. Each testing session was conducted in an enclosed laboratory (dry bulb temperature 20.2 ± 1.4°C) with sessions performed at the same time of day to minimize diurnal variation. The repeated bout effect (RBE) has previously been demonstrated to last up to 6-12 months following unaccustomed exercise (Nosaka et al. 2001). In order to account for any potential RBE, participants recruited were resistance-trained so the exercise was unlikely to be unaccustomed and a period of 6 weeks was allowed between the completion of respective conditions (Ebbeling and Clarkson 1989; McHugh et al. 1999). During the 6 week period between experimental conditions, participants were required to maintain normal training status and avoid unaccustomed exercise in the week prior to each testing session. Participants were required to present in a rested state and avoid any consumption of food or drink (including caffeine) 3-h prior to testing and refrain from
alcohol consumption 24-h prior to testing. All food, drink and physical activity in the 24-h prior to the first testing session and during the 48-h recovery period was recorded and replicated for all testing sessions. Participants refrained from any strenuous activity during the 48-h recovery period and activity diaries were monitored throughout.

**Exercise Protocol**

Upon completion of pre-exercise neuromuscular tests, an intense single-leg exercise protocol consisting of a series of maximal concentric (CON) and eccentric (ECC) contractions of the knee extensor (KE) muscles of the dominant leg were performed. The exercise protocol consisted of 6 x 25 maximal CON/ECC KE contractions (total contractions: CON =150, ECC = 150) at an angular velocity of 60°/s and 120°/s respectively with each set separated by 1-min rest (represented schematically in Figure 3.1). Pilot testing indicated that a total of 150 CON and 150 ECC contractions induced acute muscle fatigue and DOMS in trained participants as evidenced by a reduction in MVC and prolonged muscle soreness (up to 48-h post-exercise). The maximal CON/ECC contractions were performed within a range of 15° to 80° knee flexion (0° being full extension). During the exercise protocol, participants were instructed to produce maximal effort as fast as possible in both the CON and ECC phases of the contraction and continue exerting maximal effort throughout the full range of motion. Strong verbal encouragement was consistently provided during all voluntary efforts with extra encouragement provided during the final contractions to promote maintenance of maximal effort. Participants also received continual visual feedback of performance from the computer monitor. At the completion of the exercise protocol, post-exercise neuromuscular, physiological and perceptual measures were performed prior to participants engaging in the respective recovery intervention.
**Figure 3.1** Schematic diagram of the exercise protocol and time-points for measures.

### Recovery Interventions

Within 10 min of completing the exercise protocol, the recovery intervention was administered in a designated area of the laboratory. To isolate the cooling to the limb exercised, cold therapy (COLD) consisted of the application of ice cuffs (AirCalf, Coleman Co Inc, Wichita, Kansas USA) covering the entire surface of the exercised leg (quadriceps, knee and calf). Once the three cuffs were applied to the exercised leg, participants rested in a supine position for a 20-min duration (Figure 3.2). Temperature of the ice water remained consistent and was monitored using a thermometer (water temperature = 0.5°C). For the control (CONT) condition, participants rested in a supine position for 20 min with no ice cuff application. Skin temperature was measured in both conditions at 5-min intervals by a skin thermistor (Monotherm 4070, Mallinkrodt, St. Louis, MO, USA) placed on the anterior surface of the thigh.

<table>
<thead>
<tr>
<th>Warm-Up</th>
<th>25 CON/ECC</th>
<th>25 CON/ECC</th>
<th>25 CON/ECC</th>
<th>25 CON/ECC</th>
<th>COLD vs CONT</th>
<th>1 min rest</th>
<th>1 min rest</th>
<th>1 min rest</th>
<th>1 min rest</th>
<th>Post Ex</th>
<th>Post Rec</th>
<th>2h Post Rec</th>
<th>24h Post Rec</th>
<th>48h Post Rec</th>
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</table>

*Note: For the COLD condition, the ice cuffs were applied to the exercised leg, and participants rested in a supine position for 20 min. For the control (CONT) condition, participants rested in a supine position for 20 min with no ice cuff application.*
Figure 3.2 Application of ice cuffs following CON/ECC exercise protocol for 20 min duration
**Instrumentation**

For the exercise protocol and neuromuscular tests, participants were seated on an isokinetic dynamometer (Humac Norm isokinetic dynamometer, Ausmedic, CSMi Medical Solutions, Stoughton, MA) linked to a BNC2100 terminal block connected to a signal acquisition system (PXI1024; National Instruments, Austin, Texas). A/D conversion for torque and electromyographic data was performed at 16-bit resolution and synchronously sampled all data at a rate of 1 kHz. Participants were seated upright with a 90° hip angle on the dynamometer chair and securely fastened by adjustable straps tightly across the chest and pelvis with the distal right leg fixed to the dynamometer lever arm. The axis of rotation of the dynamometer was aligned to the lateral epicondyle of the femur, indicating the anatomical joint axis of the knee. Torque was measured and recorded instantaneously. Lever arm length, chair length and dynamometer height were recorded during familiarization for accurate re-positioning during subsequent testing sessions.

**Neuromuscular Tests**

**Muscle Activation**

Muscle activation was achieved by stimulating the femoral nerve using a felt pad bar cathodal electrode with a tip spacing of 30 mm (Nicolet Biomedical, Madison, WI, USA) positioned at the medio-anterior aspect of the upper thigh, directly below the inguinal fold. The anode was a 90 x 50 mm reusable self-adhesive gel pad electrode (Verity Medical, Ltd., Stockbridge, Hampshire, UK) and positioned opposite the cathode on the medio-posterior aspect of the upper thigh, directly below the gluteal fold. The current applied to the femoral nerve was delivered by a Digitimer DS7AH stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK) using a single square-wave pulse with a width of 200 μs (400 V with a
current of 100-450mA) that was driven by a custom designed instrument using LabView software (version 8.0, LabView; National Instruments). Initially, the current was manually applied in incremental steps until a twitch of moderate amplitude was observed. Following this, the position of the stimulating electrode was adjusted until the site most responsive to the stimulation was located. The electrode was then securely fastened in position using a Velcro strap with a constant force of 1.5 kg/f applied via an algometer (Pain Test™ FPI Algometer, Wagner Instruments, Greenwich, CT). The stimulus intensity was gradually increased until a plateau in twitch amplitude and M-wave response was achieved with the stimulus intensity increased by a further 25% to ensure that supramaximal stimulation was applied to the nerve.

**Maximal Voluntary Isometric Contractions (MVC)**

Prior to muscle activation, participants completed a warm-up consisting of sub-maximal voluntary contractions (50, 60, 80 and 90%, respectively). Participants then performed 5 x 5-s maximal voluntary contractions (MVC) with 5-s rest between each contraction with the knee flexed at 65° (0° being full extension). Participants were instructed to produce a maximal effort for the entire 5 s after which time they were told to relax until the next MVC. A superimposed electrical twitch was delivered during each MVC when a reduction in peak force was observed. During each contraction, the trigger for stimulation was manually primed within 1-2 s after initiation of each contraction. Once primed, the stimulus was automatically triggered when customized LabView software detected a decline in peak force. Manual priming of the trigger was necessary to prevent premature stimulation prior to the attainment of peak force. When primed, the decline in peak force necessary to automatically trigger the stimulus was <1%. Within 3 s following each superimposed contraction, a second stimulus was delivered with the muscle at complete rest to obtain a
potentiated twitch and M-wave response. Mean voluntary isometric torque was determined as the average peak isometric torque produced over the 5 contractions (mean MVC). Mean torque values were determined during the 25 ms preceding the delivery of the electrical stimulus.

**Voluntary Activation (VA)**

VA was calculated using the twitch interpolation technique (Allen et al. 1995). Peak superimposed torque following the delivery of the stimulus during the MVC’s was determined as the peak torque value produced during the 50-150 ms period subsequent to the delivery of the stimulus. Interpolated twitch torque was subsequently determined as peak superimposed torque minus voluntary peak torque. VA was determined by expressing the interpolated twitch torque (ITT) as a percentage of the peak potentiated evoked twitch torque (Pt) obtained at rest between contractions using the following equation: $\text{VA (\%)} = [1 – (\text{ITT/Pt})] \times 100$. Mean VA was determined from the average of all 5 superimposed contractions.

**Potentiated Evoked Twitch Contractile and M-wave Properties**

Torque-time curves from the potentiated evoked twitch contractions were averaged across all trials with mean data used to determine the following characteristics: (1) peak potentiated twitch torque (Pt); (2) the rate of torque development (RTD); (3) time to peak torque (TPt); (4) the rate of relaxation (RR); (5) half-relaxation time (1/2 RT); and (6) contraction duration (CD) (Wilder and Cannon 2009). Torque onset was determined as the point at which torque increased beyond 2 standard deviations above the mean torque value calculated over a 50-ms period immediately prior to stimulation (Wilder and Cannon 2009).
Potentiated M-wave data were averaged across the 5 trials with the mean used to determine (1) peak to peak amplitude; (2) duration; and (3) latency.

**Surface Electromyography (EMG)**

Surface EMG data were obtained from the vastus lateralis (VL), vastus medialis (VM) and the antagonist biceps femoris (BF) (Figure 3.3). Voluntary EMG data were obtained during the exercise protocol and during assessment of MVC pre-exercise, post-exercise and post-recovery period. EMG signals were sampled using differential surface electrodes (Bagnoli-16, Delsys Inc, Boston, MA) positioned on VL, VM and BF according to Cram and Kasman (1998). The electrode placement sites were marked with permanent pen and participants were asked to maintain these placement sites between testing sessions. A reusable self-adhesive electrode was attached to the patella of the opposing limb to ground the signals. The EMG cables were taped during the exercise protocol to prevent movement artifacts in the EMG signal. Voluntary EMG signals were pre-amplified and bandpass filtered, with a bandwidth frequency ranging from 20 to 450 Hz (common mode rejection ratio > 90dB; impedance input = 100MΩ; gain = 1000).

Voluntary EMG signals were quantified using the root mean square (RMS). RMS amplitude was calculated as the average of the 25 ms preceding the superimposed twitch during MVC. The EMG signal was then averaged between vasti muscles to provide a global indication of total KE motor unit activity. For processing, voluntary EMG data were normalized against the peak to peak M-wave amplitude with average RMS data expressed as a percentage of the average VM/VL M-wave amplitude.

All data were processed offline with the determination of peak and mean MVC, VA, potentiated twitch and M-wave properties, and RMS achieved using Matlab software (version R2010a; The MathWorks Inc. Natick, MA). For the isometric contractions, correction for the
effect of gravity on the lower leg during the superimposed and potentiated evoked
contractions was performed by calculating the mean load applied to the force transducer
during the 50-ms period immediately prior to force onset. The mean load applied to the
transducer during this period was used to offset force data. Once corrected for the effect of
gravity, force data were then multiplied by lever arm length and expressed in units of torque
(N m$^{-1}$).
Figure 3.3 Neuromuscular assessment of MVC, VA, EMG, Evoked Twitch and M-wave on the Humac Isokinetic Dynamometer.
Physiological Measures

Venous Blood Measures

On arrival, a 5 mL sample of venous blood, taken from the antecubital vein, was obtained pre-exercise and post-recovery intervals (immediately, 2, 24 and 48 h) for the measurement of creatine kinase (CK), C-reactive protein (CRP) and aspartate aminotransferase (AST). Using an evacuated venipuncture system and serum separator tubes (Monovette, Sarstedt, Numbrecht, Germany) samples were allowed to clot at room temperature prior to centrifugation for 10 min at 4000 rpm. Plasma was then extracted and stored at -20°C until analysis. Before analysis the plasma was allowed to reach room temperature and mixed gently via inversion. CK, CRP and AST were analyzed according to manufacturer’s instructions provided in the respective assay kits (Dimension Xpand spectrophotometer, Dade Bearing, USA). Intra-assay Coefficients of Variance were < 5% for all venous blood analyses.

Perceptual Measures

Following collection of resting blood variables, perceptual rating of perceived exertion (RPE) and muscle soreness (MS) were determined using modified 10-point Likert scales (RPE: 0=very, very light and 10 = very, very hard; MS: 0 = no soreness and 10 = very very sore). RPE was determined pre- and post-exercise, whilst MS was determined pre- and post-exercise and throughout the recovery period (post-recovery, 2-h, 24-h and 48-h post-recovery). Assessment of perceived rating of pain threshold was determined via an algometer (Pain Test™ FPI Algometer, Wagner Instruments, Greenwich, CT) positioned on the belly of the VM of the dominant leg. The tip of the algometer (1 cm diameter) was held by the tester and positioned perpendicular to the skin surface on the VM. The force applied
was gradually ramped and subjects were required to notify the tester as soon as “pain” onset occurred. The value was recorded in kg/f and the testing site was marked with permanent pen to ensure re-test reliability. Only one tester performed this measure to account for possible variability in the speed and consistency of force delivered.

**Statistical Analysis**

Data recorded from neuromuscular, physiological and perceptual measures are reported as means ± SD; whilst data for plasma CK, AST and CRP, mean and peak MVC and VA are reported as a percentage change from pre-exercise values. A repeated measures analysis of variance (ANOVA) (condition x time) was used to determine significant difference between conditions and over time for each recovery intervention. Mauchly’s Test of Sphericity was performed indicating normal distribution of data. Pairwise comparisons were used to determine the time points for significance which was set at p < 0.05. All data collected were analyzed using SPSS™ version 16.0 (Statistical Package for the Social Sciences, Chicago, Il, USA).

**RESULTS**

**Maximal Voluntary Contraction and Voluntary Activation**

Mean MVC was significantly reduced following the exercise protocol in both conditions and remained significantly lower than pre-exercise values for the duration of the 48-h recovery period (P<0.01; Figure 3.4). Despite the post-exercise suppression of MVC, there were no significant differences (P>0.05) in mean MVC between the respective recovery conditions at any time point over the ensuing 48-h recovery period. Further, no significant difference was present for the increase in percentage of pre-exercise mean MVC immediately
following COLD compared with CONT (83 ± 10 v 77 ± 12% respectively; \(P=0.23\)). Mean VA was significantly reduced from pre-exercise values at post-recovery (\(P<0.05\)) and 2-h post-recovery time-points (\(P<0.05\)) in CONT only (Figure 3.4). Despite this reduction, no significant difference was observed between recovery conditions at any time point (\(P>0.05\)).

**Potentiated Twitch Contractile Properties**

All potentiated twitch properties were significantly reduced post-exercise and during the recovery period (\(P<0.01\)) compared to pre-exercise values in both conditions. Further, Pt, TPt and RTD remained significantly reduced 2-h and 24-h post-recovery (\(P<0.001\)), while TPt remained significantly slower 48-h post-recovery (\(P<0.01\)). Following COLD recovery, CD was significantly longer (\(P<0.04\)) compared with CONT (Figure 3.5). Although there was a trend for a slower \(\frac{1}{2}\) RT immediately post-recovery following COLD compared to CONT (\(P=0.10\)), which was also present at 2-h and 24-h post-recovery (\(P=0.18\)), these differences were not significantly different (Figure 3.5).
Figure 3.4 (a) Mean voluntary torque (MVC), (b) percentage of pre-exercise values for MVC (% Pre MVC) and, (c) percentage mean voluntary activation (% Mean VA) for cold therapy (COLD) and control (CONT), respectively

No Significant differences between conditions ($P>0.05$)

^ Significant time effect from pre-exercise values ($P<0.05$)
Figure 3.5 Evoked twitch properties of the right knee extensors. 
(a) peak twitch, (b) time to peak twitch, (c) rate of torque development, (d) rate of relaxation, 
(e) half relaxation time and, (f) contraction duration for cold therapy (COLD) and control 
(CONT), respectively

* Significant difference between conditions ($P<0.05$)

^ Significant time effect from pre-exercise values ($P<0.05$)
**Voluntary EMG**

Voluntary EMG (RMS) expressed as a percentage of the M-wave amplitude was not significantly different between conditions at any time point ($P>0.05$). Mean RMS of VM and VL was significantly reduced post-exercise, 2-h and 24-h post-recovery compared to pre-exercise values in both conditions ($P<0.04$). A significant increase in RMS of BF was evident post-exercise, post-recovery and 2-h post-recovery in both groups compared to pre-exercise values ($P<0.01$; Figure 3.6). No significant differences were evident between recovery conditions at any time point for mean RMS of VM, VL or BF ($P>0.05$).

**Potentiated M-wave Properties**

The duration of the M-wave in VM was significantly increased following COLD post-recovery ($P<0.01$) (2.85 ± 7.65 v 5.7 ± 8.99 ms) and 48-h post-recovery ($P<0.05$) (2.9 ± 5.42 v 5.0 ± 6.5 ms). No significant differences were evident between recovery conditions at any time point for M-wave amplitude and latency in VM and VL ($P>0.05$).
Figure 3.6 (a) Mean value for root mean square (RMS) of vastus medialis (VM) and vastus lateralis (VL), (b) RMS of biceps femoris (BF) for cold therapy (COLD) and control (CONT), respectively

No significant differences between conditions ($P>0.05$)

^ Significant time effect from pre-exercise values ($P<0.05$)
Physiological Measures

Skin temperature was reduced during COLD with 17.6 ± 2.0°C at 0 min and 11.4 ± 0.7°C at 20 min (P<0.05). Skin temperature during CONT was unchanged (21.2 ± 1.2°C at 0 min and 22.7 ± 0.8°C at 20 min) (P>0.05). Plasma CK values were significantly increased from pre-exercise values at 2-h (P<0.05) and 24-h post-recovery (P<0.05) in CONT only. A significant increase in AST from pre-exercise values at 24-h post-recovery was also evident in CONT only (P<0.05). There was no significant change in CRP as a result of the exercise protocol and no significant difference was observed between conditions (P>0.05). At 48-h post-recovery, plasma CK as a percentage of pre-exercise values were 130 ± 105% following COLD and 165 ± 131% following CONT; although these values were not significantly different (P=0.34; Figure 3.7). Percentage of pre-exercise values in AST were significantly lower 24-h (P<0.05) and 48-h post-recovery (P<0.05) following a recovery of COLD compared with CONT (Figure 3.7).

Perceptual Measures

Rating of pain and perceptions of MS were significantly increased as a result of the exercise protocol and did not return to pre-exercise values in the 48-h recovery period in both conditions (P<0.01). Following a recovery of COLD, perceptual ratings of algometer-induced PAIN occurred at a significantly higher rating of kg/f 48-h post-recovery compared to CONT (P<0.05; Figure 3.8). Despite this, no significant differences were evident between recovery conditions for perceptions of MS at any time point (P>0.05; Figure 3.8).
Figure 3.7 Percentage of pre-exercise values for plasma venous blood responses for (a) creatine kinase (CK), (b) aspartate aminotransferase (AST) and (c) C-reactive protein (CRP) for cold therapy (COLD) and control (CONT), respectively.

* Significant difference between conditions ($P<0.05$)
Figure 3.8 Perceptual ratings of (a) pain threshold and, (b) muscle soreness on a 10-point Likert scale for cold therapy (COLD) and control (CONT), respectively

* Significant difference between conditions ($P<0.05$)

^ Significant time effect from pre-exercise values ($P<0.05$)
DISCUSSION

The intense nature of the exercise protocol resulted in significant post-exercise declines in muscle function and increased markers of muscle damage. Post-exercise MVC remained below pre-exercise values up to 48-h following exercise. Despite the observed reduction in MVC, minimal changes in voluntary activation were evident. Further, the exercise protocol resulted in significant reductions in twitch contractile properties and elevations in CK and AST, indicating that the likely origin of muscle fatigue was primarily peripheral in nature (Allen et al. 1995; Bigland-Ritchie et al. 1986). Despite the interruption and suppression of muscle contractile function, COLD did not significantly hasten the recovery of peripheral contractile damage. Apart from a significant reduction in perception of pain at 48-h post-recovery, COLD recovery did not significantly alter indices of exercise-induced muscle damage. The results of the present study therefore suggest that a single application of COLD is of no significant benefit to restore the reduction in muscle function and contractile damage following high-intensity eccentric exercise.

To date, evidence outlining performance benefits of cold therapy as a recovery strategy remain varied (Eston and Peters 1999; Vaile et al. 2008c). In agreement with the results of the present study, Jakeman et al. (2009) recently reported that a single bout of CWI following strenuous plyometric exercise resulted in no significant effect on restoration of concentric muscle strength. It may be possible that implementation of a single application of cold therapy was not sufficient to induce beneficial changes following damage to muscle contractile properties as a result of eccentric loading. Although, when repeated applications of cryotherapy were administered post-damaging exercise, results have indicated similar findings (Eston and Peters 1999; Howatson et al. 2005). Howatson et al. (2005) demonstrated that repeated application of ice massage was ineffective in reducing markers of muscle damage and enhancing the recovery of isometric and isokinetic strength of the elbow flexors. Similarly, despite a reduction in muscle stiffness, Eston and Peters (1999) reported no benefit
of repeated CWI on the recovery of strength loss following EIMD. In accordance with results of the present study it seems likely that following exercise resulting in damage and trauma to skeletal muscle fibres, a single bout of COLD does not significantly hasten the recovery of muscle contractile damage (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009).

Findings from the aforementioned research are in opposition to recent studies demonstrating beneficial effects of CWI on recovery of voluntary force (Bailey et al. 2007; Ingram et al. 2009; Vaile et al. 2008c). These investigations report hastened recovery of muscle function following CWI recovery after multi-joint, high-intensity exercise, often involving increases in endogenous thermal load (Peiffer et al. 2009a; Vaile et al. 2008a; Vaile et al. 2010) and exercise simulating the demands of team-sports (Ingram et al. 2009). Studies which have reported no effect of COLD on recovery of muscle function often elicit EIMD via single-joint modalities, frequently causing trauma to muscle contractile properties (Howatson et al. 2005; Isabell et al. 1992; Jakeman et al. 2009). As such, due to eccentric exercise eliciting damage of muscle contractile properties it may be that COLD does not affect immediate repair of contractile trauma. Rather, other mechanisms may be responsible for the observed improvement in the recovery of voluntary force observed in previous studies, such as reductions in thermal and cardiovascular strain (Peiffer et al. 2009a; Vaile et al. 2008a).

Accordingly, given the different responses of COLD following different modes of exercise, further research to fully elucidate the effects of COLD recovery following exercise-induced damage to muscle contractile properties is warranted.

Despite a reduction in post-exercise MVC, VA was not significantly altered by the exercise protocol. Given the reductions in twitch contractile properties and elevated blood markers indicative of muscle damage, the likely cause of reduced MVC is due to a reduction in peripheral contractile ability (Allen et al. 1995; Bigland-Ritchie et al. 1986). The effect of
eccentric contractions on central activation and the time-course for the recovery of VA is not fully understood (Endoh et al. 2005; Prasartwuth et al. 2006; Prasartwuth et al. 2005). That said, a reduction in post-recovery VA compared to pre-exercise values was only observed in CONT in the present study. To our knowledge, recovery of VA following COLD has not been examined following EIMD. Previous evidence has demonstrated an increase antagonistic effect following fatiguing exercise to assist protection against further damage to the agonist, reducing agonist MVC (Psek and Cafarelli 1993; Rothmuller and Cafarelli 1995). It is interesting to note in the present study that COLD resulted in a trend for a reduction in hamstring RMS compared to CONT. However, as these results were not significant, it is difficult to speculate on the effects of a reduced reliance upon co-activation of the antagonist muscle in the recovery of muscle function and central activation. As such, the effects of COLD on the alteration in neural activation pattern following EIMD remains unknown and further research is warranted.

In accordance with previous research, the intense nature of the eccentric contractions resulted in significant elevation in indirect markers of muscle damage and cell inflammation (Eston and Peters 1999; Goodall and Howatson 2008; Howatson et al. 2005; Vaile et al. 2008c). A recovery of COLD did not significantly affect the presence of CK in the blood at any time point. These results are in accordance with previous research demonstrating no significant effect of COLD on CK appearance following EIMD (Bailey et al. 2007; Goodall and Howatson 2008; Howatson et al. 2005; Jakeman et al. 2009). Despite only a single application of post-exercise COLD implemented in the present study, repeated application of CWI (Goodall and Howatson 2008) and ice massage (Howatson et al. 2005) have also demonstrated no effect on CK appearance. Despite the lack of significance, it is interesting to note that following COLD recovery, the percentage change of AST and CK were respectively smaller compared to CONT. Consequently, while CK presence in the blood is only an
indirect marker of muscle damage, it may be that the application of COLD had some success in blunting post-exercise CK and AST release rate (Eston and Peters 1999). Eston and Peters (1999) have previously demonstrated a positive effect of CWI on the appearance of CK and postulated that the observed reduction in CK efflux was possibly due to a decreased permeability of blood and lymph vessels; thus resulting in an attenuated inflammatory response and reduced rate of post-exercise damage to the muscle tissue. As such, with smaller elevations in AST and CK appearance observed following COLD in the present study, further research examining the effect of COLD on the efflux of indirect muscle damage markers is warranted.

The exercise protocol reduced algometer-induced pain threshold and increased perceptions of MS, which did not return to pre-exercise values in the 48-h recovery period. However, immediately following a recovery of COLD, subjects could withstand a greater amount of force on the belly of the VM until a subjective pain threshold was reached. It has previously been shown that the application of cold through various interventions (ice massage, ice packs) stimulates an analgesic effect resulting in a decreased perception of pain (Cheung et al. 2003; Meeusen and Lievens 1986). A decrease in tissue temperature also results in a reduced nerve conduction velocity and activity of the muscle spindle (Meeusen and Lievens 1986), as evidenced in the present study by a delayed CD, TPt and twitch latency after COLD. The acute relief of pain via the application of COLD is postulated to be due to an analgesic effect; however, the duration of this analgesia is limited to 1-3 h (Meeusen and Lievens 1986). Alternatively, given the lack of condition-induced change in twitch contractile properties by 24 h post-exercise, with lingering reductions of MS and PAIN in COLD at the same time point, a perceptual or placebo effect may have been present.

In conclusion, the aim of the current investigation was to determine the effects of cold therapy on the recovery of voluntary and evoked contractile properties following high-
intensity, eccentric exercise. The exercise protocol resulted in a prolonged reduction in muscle function and induced symptoms of EIMD. Whilst it was hypothesized that COLD recovery may ameliorate potential indices of EIMD following intense eccentric exercise, COLD did not significantly hasten the recovery of twitch contractile damage, presence of blood markers of muscle damage or voluntary force in the present study. Although cold therapy is commonly implemented in an athletic environment in an effort to reduce the effects of soreness and damage, the present study demonstrated that despite improved perceptions of pain, COLD did not significantly improve deleterious effects of EIMD.
CHAPTER 4

Cold Water Immersion Recovery Following Intermittent-Sprint Exercise in the Heat

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ABSTRACT

This study examined the effects of cold water immersion (CWI) on recovery of neuromuscular function following simulated team-sport exercise in the heat. Ten male team-sport athletes performed two sessions of a 2 x 30-min intermittent-sprint protocol (ISE) in 32°C and 52% humidity, followed by a 20-min CWI intervention or passive recovery (CONT) in a randomized, crossover design. The ISE involved a 15-m sprint every minute separated by bouts of hard-running, jogging and walking. Voluntary and evoked neuromuscular function, ratings of perceived muscle soreness (MS) and blood markers for muscle damage were measured pre- and post-exercise, and immediately post-recovery, 2-h and 24-h post-recovery. Measures of core temperature ($T_{core}$), heart rate (HR), capillary blood and perceptions of exertion, thermal strain and thirst were also recorded at the aforementioned time points. Post-exercise maximal voluntary contraction (MVC) and activation (VA) were reduced in both conditions and remained below pre-exercise values for the 24-h recovery ($P<0.05$). Increased blood markers of muscle damage were observed post-exercise in both conditions and remained elevated for the 24-h recovery period ($P<0.05$). Compared with CONT, the rate of reduction in $T_{core}$, HR and MS was enhanced with CWI whilst post-recovery MVC and VA were increased ($P<0.05$). In contrast, 24-h post-recovery MVC and VA were significantly higher in CONT compared with CWI ($P=0.05$). Following exercise in the heat, CWI attenuated thermal and cardiovascular strain, and improved acute recovery of MVC along with increased VA. However, despite improved acute recovery of MVC, CWI resulted in an attenuated voluntary force 24 h post-recovery.
INTRODUCTION

It is well established that exercise-induced increases in thermal strain result in alterations in central activation (Martin et al. 2004; Nybo and Nielsen 2001), muscle contractile function (Morrison et al. 2004) and exercise performance (Tucker et al. 2004). Many sports require repeated bouts of exercise over consecutive days, often during training and competition or tournament schedules, and when such events are performed in warm environmental conditions performance decrements are more pronounced (Vaile et al. 2008a). In an effort to alleviate potential perturbations associated with exercise-induced increases in endogenous thermal load during intermittent team-sport exercise, post-exercise recovery strategies are often employed. In particular, implementation of cold water immersion (CWI) has become an increasingly popular post-exercise recovery strategy (Peiffer et al. 2010b; Vaile et al. 2008a). Despite this, current evidence highlights equivocal benefits of CWI on the recovery of exercise performance following exercise-induced muscle damage (Vaile et al. 2008c), laboratory cycling protocols (Peiffer et al. 2010b; Vaile et al. 2010) and team-sports exercise (Rowsell et al. 2009; 2011). Performance decrements have been associated with exercise-induced elevations in thermal strain altering central and peripheral neuromuscular function (Martin et al. 2004; Tucker et al. 2004); although, the effects of CWI on the association between endogenous load and recovery of skeletal muscle contractile function contributing to performance benefits remains equivocal.

Evidence to date suggests CWI decreases body temperature (Peiffer et al. 2010a; Vaile et al. 2010), reduces the inflammatory response (Knight 1989) and minimizes secondary muscle damage responses (Knight 1989; Knight et al. 2000). Despite evidence to suggest that CWI is beneficial in the treatment of acute soft tissue injury (Bleakley et al. 2004), research outlining the efficacy of CWI facilitating recovery of muscle function and performance following exercise-induced muscle damage (EIMD) and high-intensity, constant

Investigations examining multi-joint, high-intensity exercise, often involving increases in endogenous thermal load (Peiffer et al. 2010a; Vaile et al. 2008a), team-sport exercise (Ingram et al. 2009; Rowsell et al. 2009; 2011), and self-paced cycling (Peiffer et al. 2010b; Vaile et al. 2008a, b) have also reported equivocal benefits on the recovery of muscle function following CWI. Recently, it has been reported that CWI following exercise in the heat maintained subsequent cycling performance (Vaile et al. 2008a) and resulted in a faster 4-km time trial (Peiffer et al. 2010a). However, despite recovery with CWI reducing muscle and rectal temperature, the rate of recovery in isokinetic strength following a 1-km cycling time trial was not altered with CWI (Peiffer et al. 2010b). Moreover, CWI has been reported to have negative effects on recovery of neuromuscular function resulting in a 13% decrease in maximal voluntary isometric force when compared with passive recovery (Peiffer et al. 2009a). The mechanisms associated with improvements in exercise performance associated with CWI recovery following exercise-induced increase in thermal load have primarily been linked to facilitated recovery of physiological (Vaile et al. 2010; Yeargin et al. 2006) and cardiovascular function (Wilcock et al. 2006). Despite such findings, an area which has had minimal focus has been the effect of CWI on recovery of neuromuscular function (Peiffer et al. 2009a), specifically central and peripheral components following exercise in the heat.

Although reduced performance following exercise in the heat is associated with alterations in central and peripheral neuromuscular function (Martin et al. 2004; Tucker et al. 2004), the contribution of central- and peripherally-mediated mechanisms influencing improvements in subsequent recovery of contractile function in the heat following CWI has
not been investigated. Therefore, the present study aimed to determine the effects of CWI following simulated team-sport exercise in the heat on the recovery of neuromuscular function, and associated effects on other physiological, performance and perceptual variables. With the return of maximal voluntary force and activation observed only when cooling reversed core temperature ($T_{\text{core}}$) back to normal values (~37.4°C) following passively-induced hyperthermia (Morrison et al. 2004), we hypothesized that CWI following a bout of intermittent-sprint exercise in the heat would ameliorate the subsequent decline in maximal voluntary force due to an enhanced reduction in $T_{\text{core}}$ and improved recovery of central activation.

**METHODS**

**Participants**

Ten male team sport athletes (rugby league/union) aged (mean ± SD) 19.9 ± 1.1 y, height 179.6 ± 3.8 cm and body mass 78.9 ± 6.3 kg were recruited as participants for this study. At the time of testing, participants completed 3-4 training sessions per week and competed in team sport competition at least once per week. All participants were informed of the requirements of the study and verbal and written consent was gained prior to the commencement of testing. Human ethics clearance was granted by the Institutional Ethics Committee prior to the completion of any testing procedures.

**Overview**

Participants completed an initial session to ensure familiarity with all measures and procedures. The two testing sessions were identical apart from the recovery intervention implemented and were completed in a randomized, crossover order separated by at least 7 days. Each experimental session consisted of a prolonged high-intensity, intermittent-sprint
exercise (ISE) protocol (2 x 30 min halves) performed in an enclosed laboratory on a 20-m synthetic running track. The ISE protocol and ensuing recovery conditions were performed in 32.4 ± 1.5°C, 51.1 ± 6.2% relative humidity for CONT and 32.4 ± 1.3°C, 53.8 ± 8.5% relative humidity for CWI. The ISE was followed by cold water immersion (CWI) or a passive recovery (CONT), with each testing session performed at the same time of day to minimize diurnal variation. Neuromuscular performance and repeated sprint ability (RSA) were measured pre- and within 5-min post-exercise, post-recovery and again 2-h and 24-h post-recovery. Participants were required to present in a rested state and avoid consumption of food or drink (including caffeine) 3-h prior to testing and refrain from alcohol consumption 24-h prior to testing. All food, drink and physical activity in the 24-h prior to the first testing session and during the 24-h recovery period following the exercise protocol were recorded. Participants refrained from any strenuous activity during the 24-h recovery period and activity diaries were monitored throughout. Food, drink and activity prior to and during the first testing session were replicated for all testing sessions. During the ISE protocol 500 mL of water was provided to be consumed ad libitum with complete consumption ensured during each testing session.

Exercise Protocol

Upon completion of pre-exercise neuromuscular tests (detailed subsequently), participants completed a warm-up involving running at increasing speeds over a 15-m running track for a period of 3 min, followed by 3 maximal 15-m sprints. The exercise protocol consisted of 2 x 30-min halves, interspersed by 10-min passive recovery. The exercise protocol involved a 15-m maximal sprint, (with a subsequent 5-m deceleration zone before impacting with a large mat) performed every min, followed by sub-maximal exercise of varying intensities in a self-paced, shuttle-run format (hard running, jogging, walking) for
the remainder of the minute (Duffield and Marino 2007). Only one exercise mode of hard running, jogging or walking (rotated each minute) was completed each minute before returning to the starting position to complete the ensuing sprint. Every 6th rotation, following the maximal sprint, participants completed 8 consecutive double-leg bounds, covering as much distance as possible (distance determined for each exercise mode using 1-m markings alongside the 15-m synthetic track) (See Appendix B). Maximal 15-m sprint performance during the exercise protocol was assessed with infra-red timing gates (Speed-Light, Swift, Australia) with mean sprint time calculated. As the exercise intensity was set by the participants, distances covered during the exercise protocol were monitored by the investigators and appropriate encouragement was provided to ensure similar workloads were performed during each testing session. Intra-class correlations (r) and coefficients of variation (CV) for total distance covered, sprint times and hard running were r=0.82-0.96 and CV=1.5-3.2%, respectively.

Recovery Interventions

Within 10 min of completing the exercise protocol, the recovery intervention was administered in a designated area of the laboratory. Cold water immersion (CWI) consisted of immersion in an ice bath (plunge pool) (8.9 ± 0.9°C) to a level of the umbilicus for 9 min followed by 1 min seated at room air temperature (Figure 4.1). This procedure was repeated twice for a total duration of 20 min (Peiffer et al. 2009a; Peiffer et al. 2010b). For the passive recovery (CONT), participants remained seated in the laboratory for 20 min (Figure 4.1).
Figure 4.1 (a) Seated passive recovery and (b) cold water immersion to the level of iliac crest for 20-min duration
Chapter 5

Procedures

Neuromuscular Tests

For the neuromuscular tests, participants were seated on an isokinetic dynamometer (Humac Norm isokinetic dynamometer, Ausmedic, CSMi Medical Solutions, Stoughton, MA) linked to a BNC2100 terminal block connected to a signal acquisition system (PXI1024; National Instruments, Austin, Texas). A/D conversion for torque and electromyographic data was performed at 16-bit resolution and synchronously sampled all data at a rate of 1kHz. Participants were seated upright with a 90° hip angle on the dynamometer chair and securely fastened by adjustable straps tightly across the chest and pelvis with the distal right leg fixed to the dynamometer lever arm. The axis of rotation of the dynamometer was aligned to the lateral epicondyle of the femur indicating the anatomical joint axis of the knee. Torque was measured and recorded instantaneously. Lever arm length, chair length and dynamometer height were recorded during familiarization for accurate repositioning during subsequent testing sessions.

Muscle Activation

Muscle activation was achieved by stimulating the femoral nerve using a felt pad bar cathodal electrode with a tip spacing of 30 mm (Nicolet Biomedical, Madison, WI, USA) positioned at the medio-anterior aspect of the upper thigh, directly below the inguinal fold. The anode was a 90 x 50 mm reusable self-adhesive gel pad electrode (Verity Medical, Ltd., Stockbridge, Hampshire, UK) and positioned on the medio-posterior aspect of the upper thigh, directly below the gluteal fold, opposite the cathode. The current applied to the femoral nerve was delivered by a Digitimer DS7AH stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK) using a single square-wave pulse with a width of 200 µs (400 V with a current of 100-450mA) that was driven by a custom designed instrument using LabView.
software (version 8.0, LabView; National Instruments). Initially, the current was manually applied in incremental steps until a twitch of moderate amplitude was observed. Following this, the position of the stimulating electrode was adjusted until the site most responsive to the stimulation was located. This location was marked with a permanent pen to ensure identical placement for subsequent testing. The electrode was then securely fastened in position using a Velcro strap with a constant force of 1.5 kg/f applied via an algometer (Pain Test™ FPI Algometer, Wagner Instruments, Greenwich, CT). The stimulus intensity was gradually increased until a plateau in twitch and M-wave amplitude was achieved. The stimulus intensity was then increased by a further 25% to ensure that supramaximal stimulation was applied to the nerve.

**Maximal Voluntary Isometric Contractions (MVC)**

A 5-min low-intensity warm-up at 60 W on a cycle ergometer (Monark 818E, Varberg, Sweden) was initially performed prior to the measurement of MVC pre-exercise, 2-h and 24-h post-recovery. During neuromuscular testing, participants performed 5 x 5-s MVC with 5-s rest between each contraction with the knee flexed at 65° (0° being full extension). Participants were instructed to produce a maximal effort for the entire 5 s at which time they were told to relax until the next MVC. A superimposed electrical twitch was delivered during each MVC when a reduction in peak force was observed. During each contraction, the trigger for stimulation was manually primed within 1-2 s after initiation of each contraction. Once primed, the stimulus was automatically triggered when customized LabView software (version 8.0, LabView; National Instruments) detected a decline in peak force. Manual priming of the trigger was necessary to prevent premature stimulation prior to the attainment of peak force. When primed, the decline in peak force necessary to automatically trigger the stimulus was <1%. Further, within 3 s following each superimposed contraction, a second
stimulus was delivered with the muscle at complete rest to determine potentiated twitch properties. Mean voluntary isometric torque was determined by averaging the peak isometric torque produced over the 5 contractions (mean MVC). Mean torque values were determined during the 25 ms preceding the delivery of the electrical stimulus.

**Voluntary Activation (VA)**

VA was calculated using the twitch interpolation technique (Allen et al. 1995). Peak superimposed torque following the delivery of the stimulus during the MVC’s was determined as the peak torque value produced during the 50-150 ms period subsequent to the delivery of the stimulus. Interpolated twitch torque was subsequently determined as peak superimposed torque minus voluntary peak torque and calculated to four decimal places. VA was determined by expressing the interpolated twitch torque (ITT) as a percentage of the peak potentiated evoked twitch torque (Pt) obtained at rest between contractions using the following equation: $\text{VA (\%) = } [1 – (\text{ITT/Pt})] \times 100$. Peak VA was determined from the peak isometric contraction and mean VA was determined from the average of all 5 superimposed contractions. Both the mean and peak VA of the 5 contractions were used for subsequent analyses.

**Potentiated Evoked Twitch Contractile and M-wave Properties**

Potentiated twitch and M-wave properties were determined from an electrical stimulus initiated ~3-s following the superimposed contraction on the resting muscle. Torque-time curves from the potentiated evoked twitch contractions were averaged across all trials with mean data used to determine the following characteristics: (1) peak potentiated twitch torque (Pt); (2) the rate of torque development (RTD); (3) time to peak torque (TPT); (4) the
rate of relaxation (RR); (5) half-relaxation time (1/2 RT); and (6) contraction duration (CD) (Cannon et al. 2006). Torque onset was determined as the point at which torque increased beyond 2 standard deviations above the mean torque value calculated over a 50-ms period immediately prior to stimulation (Wilder and Cannon 2009). Potentiated M-wave data was averaged across the 5 trials with the mean used to determine: (1) peak to peak amplitude; (2) duration; and (3) latency (Saboisky et al. 2003).

**Surface Electromyography (EMG)**

Surface EMG data were obtained from the vastus lateralis (VL), vastus medialis (VM) and the antagonist biceps femoris (BF). Voluntary EMG data were obtained during the assessment of MVC pre-, post-exercise and all post-recovery assessments. EMG signals were sampled using differential surface electrodes (Bagnoli-16, Delsys Inc, Boston, MA) and positioned on VL, VM, BF according to Cram and Kasman (1998). Low impedance was obtained by shaving, abrading and cleaning the skin prior to positioning of the electrodes at each testing time point. The electrode placement sites were marked with permanent pen to ensure identical placement for subsequent testing sessions. In addition, a reusable self-adhesive electrode was attached to the patella of the opposing limb and acromion process for the arm to ground the signals. EMG signals were pre-amplified and bandpass filtered, with a bandwidth frequency ranging from 20 to 450 Hz (common mode rejection ratio > 90dB; impedance input = 100MΩ; gain = 1000).

Voluntary EMG signals were quantified using the root mean square (RMS) amplitude calculated as the average of the 25-ms preceding the superimposed twitch during MVC. The EMG signal was then averaged between vasti muscles to provide a global indication of total KE motor unit activity. For processing, voluntary EMG data were normalized against the peak to peak M-wave amplitude with average RMS data expressed as a percentage of the
average VM/VL M-wave amplitude. All data were processed offline with the determination of mean MVC, VA, potentiated twitch and M-wave properties, and RMS achieved using Matlab software (version R2010a; The MathWorks Inc. Natick, MA). For the isometric contractions, correction for the effect of gravity on the lower leg during the superimposed and potentiated evoked contractions was performed by calculating the mean load applied to the force transducer during the 50-ms period immediately prior to force onset. The mean load applied to the transducer during this period was used to offset force data. Once corrected for the effect of gravity, force data were then multiplied by lever arm length and expressed in units of torque (N m⁻¹).

Repeated Sprint Ability

Participants performed a repeated sprint exercise protocol prior to, post-exercise, post-recovery and 2 h and 24 h post-exercise. The RSA protocol consisted of 5 x 15-m maximal sprints performed every 20-s. Maximal 15-m sprint times were assessed with infra-red timing gates (Speed-Light, Swift, Australia) and the percentage decline within each 5 x 15-m bout was calculated ((total time/(fastest time · sprint n) · 100)).

Capillary and Venous Blood Measures

On arrival, a 100 μL sample of capillary blood was obtained for the measurement of Lactate (La⁻), pH and bicarbonate (HCO₃⁻) with further samples obtained immediately post-exercise and post-recovery intervention. A 5 mL sample of venous blood was obtained from the antecubital vein pre-exercise and at post-recovery intervals (immediately, 2 and 24 h) for the measurement of creatine kinase (CK), C-reactive protein (CRP), aspartate aminotransferase (AST) as markers of muscle damage and cell inflammation. Using an
evacuated venipuncture system and serum separator tubes (Monovette, Sarstedt, Numbrecht, Germany), samples were allowed to clot at room temperature prior to centrifugation for 10 min at 4000 rpm. Serum was then extracted and stored at -20°C until analysis. Before analysis the serum was allowed to reach room temperature and mixed gently via inversion. CK, CRP and AST were analyzed according to manufacturer’s instructions provided in the respective assay kits (Dimension Xpand spectrophotometer, Dade Bearing, USA). Intra-assay Coefficients of Variance were < 5% for all venous blood analyses

**Nude Mass, Heart Rate and Core Temperature**

Nude mass was recorded on arrival and immediately after the exercise protocol on a set of calibrated scales (HW 150 K, A & D, Tokyo, Japan accurate to 10 g). Heart rate (HR) was determined with a heart rate monitor and wrist watch receiver (F1, Polar Electro-Oy, Finland). Core temperature ($T_{core}$) was measured with a telemetric capsule (Vital Sense, MiniMitter, Oregon, USA) ingested 4-h prior to each testing session to ensure it had passed into the gastrointestinal tract. $T_{core}$ was assessed with a hand-held monitor that telemetrically received measures from the ingested capsule (VitalSense, Mini Mitter, Oregon, USA). HR and $T_{core}$ were measured prior to exercise, every 5 min during the exercise protocol, immediately post-recovery and 2-h post-recovery.

**Perceptual measures**

Perceptual measures of Rating of Perceived Exertion (RPE) was determined using the Borg 6-20 Scale (Morrison et al. 2004). Muscle soreness (MS), thirst and thermal strain were all determined using 10-point Likert scales (MS: 0 = no pain and 10 = very very sore; thirst: 0 = not thirsty and 10= extremely thirsty; thermal strain: 0 = unbearably cold and 10 =
unbearably hot). RPE, thirst and thermal strain were determined pre- and post-exercise and every 5-min during the exercise protocol, whilst MS was determined pre- and post-exercise and throughout the recovery period (post-recovery, 2 h and 24 h).

**Statistical Analysis**

Data recorded from neuromuscular, physiological and perceptual measures are reported as means ± SD. A repeated measures analysis of variance (ANOVA) (condition x time) was used to determine significant difference between conditions and over time for each recovery intervention. Significant difference (P<0.05) between time points were determined using planned within-subject contrasts. Mauchly’s Test of Sphericity was performed to test for the homogeneity of variance (Portney and Watkins 2009). All data collected were analyzed using SPSS™ version 16.0 (Statistical Package for the Social Sciences, Chicago, Il, USA).

**RESULTS**

**Distance Covered and Sprint Time**

There were no significant differences between conditions for the total distance covered during the exercise protocol (P=0.80; 4207 ± 550 m CONT v 4170 ± 342 m CWI). Total distance covered for hard running was 1843 ± 224 m CONT and 1842 ± 180 m CWI (P=0.10). Distance covered for jogging was 1449 ± 223 m CONT and 1436 ± 133 m CWI (P=0.78); whilst 915 ± 122 m CONT and 892 ± 88 m CWI were covered during walking (P=0.54). There were no significant differences between conditions for double leg bound distance at any time point (134.8 ± 10.1 m CONT v 134.5 ± 9.2 m CWI; P=0.30-0.80). Further, total time for maximal 15 m sprints during the exercise protocol was 176.1 ± 8.2 s CONT and 175.2 ± 3.2 s CWI (P=0.10-0.90).
**Repeated Sprint Ability**

Mean sprint time and percentage decline in 5 x 15-m maximal sprints were significantly increased post-exercise and remained above pre-exercise values up to 2 h post-recovery in both conditions ($P<0.05$; Table 4.1). No significant differences were evident between recovery conditions for repeated 5 x 15-m sprints during any time point ($P>0.05$).

**Maximal Voluntary Contractions and Activation**

Post-exercise mean MVC and VA were significantly reduced compared to pre-exercise values in both conditions ($P<0.01$; Figure 4.2). Mean VA did not return to baseline values until 2-h post-recovery, with MVC remaining below pre-exercise values for the 24-h recovery period ($P<0.01$; Figure 4.2) in both conditions. Compared to CONT, mean MVC and VA were significantly higher post-recovery following CWI ($P<0.01$). However, 24-h post-recovery mean MVC was greater in the CONT condition compared to CWI ($P<0.05$).

**Potentiated Twitch and M-wave Properties**

The exercise protocol resulted in significantly reduced Pt, RR and TPt ($P<0.05$; Table 4.2). Pt and RR remained below pre-exercise values post-recovery and 2-h post-recovery in both conditions ($P<0.01$). Despite the post-exercise reduction in Pt and TPt, no significant differences were evident between the recovery conditions at any time point ($P>0.05$; Table 4.2). Compared to CONT, CWI resulted in a significantly reduced RR and CD post-recovery ($P<0.01$), which remained evident 24-h post-recovery for RR only ($P<0.01$). Exercise did not significantly alter the duration and latency of the M-wave ($P>0.05$; Table 4.3). Compared to CONT, post-recovery M-wave duration was significantly increased following CWI ($P<0.05$; Table 4.3). No significant differences were evident.
between respective recovery conditions at any time point for M-wave latency ($P>0.05$; Table 4.2).

**Voluntary EMG**

Voluntary EMG (RMS) expressed as a percentage of the M-wave amplitude was not significantly different between conditions at any time point ($P>0.05$). Post-exercise mean RMS of VM/VL was significantly reduced compared to pre-exercise values in both conditions ($P<0.05$; Figure 4.3). Compared to CONT, mean RMS of VM/VL were significantly increased post-recovery in CWI ($P<0.05$; Figure 4.3). However, 24-h post-recovery, mean RMS values of VM/VL were significantly increased in CONT compared to CWI ($P<0.05$; Figure 4.3). Absolute RMS of BF was not altered by the exercise protocol and no significant differences were evident between conditions at any time point ($P>0.05$; Figure 4.3).
Figure 4.2 Mean ± SD (a) mean voluntary torque (MVC) and, (b) activation (VA) for cold water immersion (CWI) and passive recovery (CONT)

* Significant difference between conditions ($P<0.05$)

^ Significant time effect from pre-exercise values ($P<0.05$)
Table 4.2 Mean ± SD Potentiated twitch properties for cold water immersion (CWI) and passive recovery (CONT). Pt; peak twitch, TPt; time to peak twitch, $\frac{1}{2}$ RT; half relaxation time, RTD; rate of torque development, RR; rate of relaxation, CD; contraction duration and Lat; latency.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
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<th>Post Rec</th>
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<tbody>
<tr>
<td><strong>Pt (N·m)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>43.1 ± 7.1</td>
<td>41.9 ± 10.3</td>
<td>33.9 ± 11.8 $^\wedge$</td>
<td>30.7 ± 9.2 $^\wedge$</td>
<td>34.9 ± 10.1 $^\wedge$</td>
</tr>
<tr>
<td><strong>TPt (ms)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>95.3 ± 9.6</td>
<td>99.8 ± 14.8</td>
<td>87.8 ± 14.7 $^\wedge$</td>
<td>84.2 ± 11.2</td>
<td>86.1 ± 6.0</td>
</tr>
<tr>
<td><strong>$\frac{1}{2}$ RT (ms)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>81.6 ± 11.2</td>
<td>81.6 ± 12.9</td>
<td>73.5 ± 9.7</td>
<td>72.6 ± 13.1</td>
<td>86.2 ± 8.9</td>
</tr>
<tr>
<td><strong>RTD (N·m s$^{-1}$)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>454.4 ± 64.6</td>
<td>437.7 ± 65.5</td>
<td>402.8 ± 156.4</td>
<td>438.5 ± 80.1</td>
<td>362.3 ± 142.9</td>
</tr>
<tr>
<td><strong>RR (N·m s$^{-1}$)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>-273.6 ± 71.7</td>
<td>-270.3 ± 91.1</td>
<td>-249.6 ± 61.9 $^\wedge$</td>
<td>-271.2 ± 77.8 $^\wedge$</td>
<td>-176.5 ± 54.6 $^\wedge$</td>
</tr>
<tr>
<td><strong>CD (ms)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>176.8 ± 9.9</td>
<td>181.4 ± 16.1</td>
<td>162.5 ± 13.4</td>
<td>163.4 ± 13.0</td>
<td>168.9 ± 10.5 $^*$</td>
</tr>
<tr>
<td><strong>Latency (ms)</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
<td><strong>CWI</strong></td>
<td><strong>CONT</strong></td>
</tr>
<tr>
<td></td>
<td>22.4 ± 6.5</td>
<td>21.2 ± 6.0</td>
<td>20.5 ± 7.9</td>
<td>21.4 ± 8.1</td>
<td>24.9 ± 6.2</td>
</tr>
</tbody>
</table>

* Significant difference between conditions ($P<0.05$)

$^\wedge$ Significant time effect from pre-exercise values ($P<0.05$)
Table 4.3 Mean ± SD Potentiated M-wave properties for cold water immersion (CWI) and passive recovery (CONT).

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
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<th>Post Rec</th>
<th>2h Post</th>
<th>24h Post</th>
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</thead>
<tbody>
<tr>
<td><strong>Latency (ms)</strong></td>
<td></td>
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</tr>
<tr>
<td>CONT</td>
<td>10.9 ± 1.9</td>
<td>10.2 ± 1.8</td>
<td>11.3 ± 1.7</td>
<td>9.6 ± 1.9</td>
<td>11.1 ± 1.7</td>
</tr>
<tr>
<td>CWI</td>
<td>11.1 ± 4.2</td>
<td>11.2 ± 1.8</td>
<td>12.2 ± 3.0</td>
<td>11.7 ± 2.5</td>
<td>11.0 ± 2.6</td>
</tr>
<tr>
<td><strong>Duration (ms)</strong></td>
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<td></td>
</tr>
<tr>
<td>CONT</td>
<td>4.9 ± 1.9</td>
<td>4.2 ± 0.9</td>
<td>3.6 ± 1.1</td>
<td>* 4.6 ± 1.4</td>
<td>4.4 ± 1.5</td>
</tr>
<tr>
<td>CWI</td>
<td>5.2 ± 2.0</td>
<td>5.4 ± 2.4</td>
<td>4.9 ± 1.2</td>
<td>4.8 ± 2.1</td>
<td>4.5 ± 1.2</td>
</tr>
<tr>
<td><strong>Amplitude (mV)</strong></td>
<td></td>
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</tr>
<tr>
<td>CONT</td>
<td>1.2 ± 0.2</td>
<td>1.3 ± 1.1</td>
<td>0.9 ± 0.2</td>
<td>1.0 ± 0.4</td>
<td>1.0 ± 0.3</td>
</tr>
<tr>
<td>CWI</td>
<td>1.0 ± 0.3</td>
<td>1.0 ± 0.4</td>
<td>1.2 ± 0.4</td>
<td>1.0 ± 0.3</td>
<td>1.0 ± 0.3</td>
</tr>
</tbody>
</table>

* Significant difference between conditions (P<0.05)
Figure 4.3 Mean ± SD of Root Mean Square (RMS) for (a) average vastus medialis and vastus lateralis and, (b) biceps femoris for cold water immersion (CWI) and passive recovery (CONT).

* Significant difference between conditions ($P<0.05$)

^ Significant time effect from pre-exercise values ($P<0.05$)
Chapter 5

_Nude Body Mass, Heart Rate and Core Temperature_

Nude body mass was significantly reduced post-exercise ($P<0.01$) in CONT ($1.60 \pm 0.50$ kg) and CWI ($1.48 \pm 0.41$ kg), with no significant differences evident between the conditions ($P=0.50$). $T_{\text{core}}$ and HR were significantly elevated post-exercise compared to pre-exercise values in both conditions ($P<0.01$; Figure 4.4 and 4.5, respectively). Following CWI, absolute $T_{\text{core}}$ values were not significantly different compared to CONT ($P=0.50$); however, the relative rate of change in $T_{\text{core}}$ was significantly faster post-recovery and 2 h post-recovery compared to CONT ($P<0.05$; Figure 4.4). In addition, CWI resulted in a significantly lower absolute HR and percentage of maximal HR post-recovery compared to a CONT ($P<0.05$; Figure 4.5).
Figure 4.4 Mean ± SD (a) Core temperature and (b) rate of change in core temperature from post-exercise values for cold water immersion (CWI) and passive recovery (CONT).

* Significant difference between conditions ($P<0.05$)
Figure 4.5 Mean ± SD (a) heart rate and (b) percentage of age-predicted maximal heart rate (MHR) for cold water immersion (CWI) and passive recovery (CONT).

* Significant difference between conditions ($P<0.05$)
**Venous and Capillary Blood Variables**

Significant post-exercise increases in CK, AST and La were evident, with CK values remaining elevated during the 24-h recovery period \((P<0.03)\) as presented in Table 4.3. Despite exercise-induced elevations in CK, AST and La, no differences were evident between recovery conditions at any time point \((P>0.05; \text{ Table 4.4})\). CRP was not significantly altered by the exercise protocol and no significant differences were observed between recovery conditions at any time point \((P>0.05; \text{ Table 4.4})\). Further, no significant differences were evident between the conditions for the \% change in CK, AST and CRP at any time point \((P>0.05; \text{ Table 4.4})\). Post-exercise pH and \(\text{HCO}_3^-\) decreased significantly from pre-exercise values in both conditions \((P<0.05; \text{ Table 4.4})\); however, no significant differences were evident between conditions at any time point \((P>0.05)\).

**Perceptual Measures**

Ratings of MS were significantly increased post-exercise and remained elevated above pre-exercise values during the 24-h recovery period \((P<0.01)\). Compared to CONT, perceptions of MS were significantly reduced immediately post-recovery in CWI \((P<0.02; 4.5 \pm 0.9 \text{ CWI v 5.7 } \pm 0.9 \text{ CONT})\). Ratings of thermal strain, perceived exertion and thirst were significantly increased during the exercise protocol \((P<0.01)\). Compared to CONT, thermal strain was significantly lower immediately following CWI \((P<0.01; 3.5 \pm 1.2 \text{ CWI v 6.2 } \pm 0.9 \text{ CONT})\); whilst, no significant differences were evident between recovery conditions at any time point for thirst and RPE \((P>0.05)\).
### Table 4.4 Mean ± SD Absolute and percentage (%) of pre-exercise values for capillary and venous blood variables for cold water immersion (CWI) and passive recovery (CONT).

Lactate (La\(^{-}\)), pH, bicarbonate (HCO\(_{3}\)), creatine kinase (CK), aspartate aminotransferase (AST) and c-reactive protein (CRP).

<table>
<thead>
<tr>
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<th>Pre</th>
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<th>Post-Rec</th>
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<tr>
<td><strong>La(^{-}) (mmol.(^{-})L)</strong></td>
<td></td>
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<tr>
<td>CONT</td>
<td>1.18 ± 0.15</td>
<td>4.62 ± 1.17</td>
<td>1.43 ± 0.47</td>
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</tr>
<tr>
<td>CWI</td>
<td>1.14 ± 0.14</td>
<td>4.73 ± 1.50</td>
<td>1.80 ± 0.60</td>
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<tr>
<td><strong>pH (mmol.(^{-})L)</strong></td>
<td></td>
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<tr>
<td>CONT</td>
<td>7.36 ± 0.03</td>
<td>7.37 ± 0.02</td>
<td>7.39 ± 0.02</td>
<td></td>
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</tr>
<tr>
<td>CWI</td>
<td>7.37 ± 0.02</td>
<td>7.37 ± 0.05</td>
<td>7.40 ± 0.03</td>
<td></td>
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</tr>
<tr>
<td><strong>HCO(_{3}) (mmol.(^{-})L)</strong></td>
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</tr>
<tr>
<td>CONT</td>
<td>22.29 ± 0.69</td>
<td>19.70 ± 1.44</td>
<td>22.42 ± 0.79</td>
<td></td>
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</tr>
<tr>
<td>CWI</td>
<td>22.19 ± 0.65</td>
<td>19.59 ± 1.82</td>
<td>21.87 ± 0.54</td>
<td></td>
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</tr>
<tr>
<td><strong>CK (IU/L)</strong></td>
<td></td>
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</tr>
<tr>
<td>CONT</td>
<td>308.1 ± 189.7</td>
<td>418.8 ± 205.5</td>
<td>394.5 ± 209.1</td>
<td>419.3 ± 228.3</td>
<td>551.7 ± 359.5</td>
</tr>
<tr>
<td>CWI</td>
<td>359.1 ± 157.9</td>
<td>477.1 ± 199.9</td>
<td>489.0 ± 208.2</td>
<td>541.5 ± 252.3</td>
<td>708.9 ± 362.7</td>
</tr>
<tr>
<td><strong>CK (%) change</strong></td>
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<tr>
<td>CONT</td>
<td>146.7 ± 30.8</td>
<td>135.2 ± 20.0</td>
<td>143.8 ± 26.0</td>
<td>194.7 ± 106.0</td>
<td></td>
</tr>
<tr>
<td>CWI</td>
<td>135.6 ± 13.5</td>
<td>141.5 ± 28.1</td>
<td>160.5 ± 50.5</td>
<td>219.7 ± 118.5</td>
<td></td>
</tr>
<tr>
<td><strong>AST (IU/L)</strong></td>
<td></td>
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</tr>
<tr>
<td>CONT</td>
<td>25.30 ± 3.86</td>
<td>32.75 ± 3.56</td>
<td>33.25 ± 3.51</td>
<td>31.88 ± 3.79</td>
<td>32.50 ± 4.13</td>
</tr>
<tr>
<td>CWI</td>
<td>33.13 ± 5.43</td>
<td>39.00 ± 7.67</td>
<td>40.00 ± 5.42</td>
<td>37.13 ± 6.16</td>
<td>39.13 ± 5.42</td>
</tr>
<tr>
<td><strong>AST (%) change</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>CONT</td>
<td>110.7 ± 25.5</td>
<td>111.5 ± 27.5</td>
<td>106.6 ± 23.5</td>
<td>106.9 ± 33.6</td>
<td></td>
</tr>
<tr>
<td>CWI</td>
<td>117.2 ± 18.5</td>
<td>135.8 ± 32.5</td>
<td>126.5 ± 41.1</td>
<td>128.5 ± 32.2</td>
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</tr>
<tr>
<td><strong>CRP (IU/L)</strong></td>
<td></td>
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</tr>
<tr>
<td>CONT</td>
<td>1.6 ± 0.9</td>
<td>1.8 ± 1.3</td>
<td>1.7 ± 1.2</td>
<td>1.7 ± 1.1</td>
<td>2.1 ± 1.3</td>
</tr>
<tr>
<td>CWI</td>
<td>1.9 ± 1.2</td>
<td>2.0 ± 1.2</td>
<td>1.9 ± 1.3</td>
<td>2.0 ± 1.0</td>
<td>2.5 ± 1.3</td>
</tr>
<tr>
<td><strong>CRP (%) change</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>100.6 ± 24.0</td>
<td>98.7 ± 24.9</td>
<td>104.0 ± 19.7</td>
<td>133.6 ± 45.2</td>
<td></td>
</tr>
<tr>
<td>CWI</td>
<td>106.7 ± 9.1</td>
<td>100.7 ± 28.6</td>
<td>116.8 ± 31.4</td>
<td>161.0 ± 83.1</td>
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* Significant difference between conditions (P<0.05)

\(^{\wedge}\) Significant time effect from pre-exercise values (P<0.05)
DISCUSSION

This investigation aimed to examine the efficacy of CWI on the recovery of performance and physiological function following simulated team-sport exercise in the heat. Following exercise-induced reductions in muscle contractile force generation, CWI significantly improved acute recovery of MVC and VA. Furthermore, a faster reduction in post-exercise $T_{core}$ and HR, together with reduced perceptions of muscle soreness and thermal strain were evident following CWI. The reduction in post-exercise thermal and cardiovascular strain likely contributed to the enhanced acute recovery of MVC (Yeargin et al. 2006), and the novel finding of increased central activation (VA) and motor unit recruitment (RMS) observed in the present study. Despite initial improvements in MVC and VA following CWI, we show for the first time the suppression of voluntary force and RMS 24-h post-recovery following CWI compared to passive recovery. That is, MVC was 85±18% of pre-exercise values in CONT compared to 74±13% in CWI; thus resulting in an 11% decline in voluntary force 24-h post-recovery. However, despite altered muscle function, no changes in RSA were evident following cooling; highlighting the divergence in isolated joint versus whole-body exercise performance. Accordingly, based on the results of the present study, implementation of CWI on the recovery of neuromuscular and contractile function following team-sport exercise in the heat may be time and mode dependent providing immediate beneficial effects to recovery of isometric MVC; however, counterproductive to long-term recovery of single-joint isometric voluntary force production.

Simulated team-sport exercise in the heat resulted in prolonged reduction in MVC and VA which is commonly observed following exercise-induced elevations in thermal strain (Martin et al. 2004; Nybo and Nielsen 2001; Thomas et al. 2006). Impaired performance with increased thermal load has previously been associated with a reduction in centrally-mediated recruitment and activation (Martin et al. 2004; Nybo and Nielsen 2001; Thomas et al. 2006).
ISE in the current study also resulted in reduced peak twitch contractile force, evident up to 2 h post-recovery. Therefore, reductions in post-exercise MVC likely resulted from a combination of reduced neural activation of skeletal musculature (Nybo and Nielsen 2001) and suppression of peripheral contractile ability (Hargreaves 2004).

In accordance with previous research (Peiffer et al. 2010a; Vaile et al. 2008a; Vaile et al. 2010), implementation of CWI recovery following exercise-induced elevation in thermal strain, resulted in a faster rate of reduction in $T_{\text{core}}$ and HR, with subsequent improvements in exercise performance. Indeed, Vaile et al. (2010) and Peiffer et al. (2010a) recently demonstrated that repeated cycling performance in the heat was improved or at least maintained following recovery with CWI. Peiffer et al. (2010a) demonstrated smaller reductions in power output and thus a faster completion of a subsequent 4-km cycling time trial with CWI compared to a seated recovery ($1.5 \pm 0.2\%$ CWI v $14 \pm 1.0\%$ control). Similar to the results of the present study, Peiffer et al. (2010a) and Vaile et al. (2010) observed a faster reduction in $T_{\text{core}}$ and HR and postulated that the mechanisms for improved exercise performance were related to CWI reducing post-exercise thermal and cardiovascular strain. Whilst the faster reduction in $T_{\text{core}}$ is a possible explanation for the observed performance enhancement, a further explanation may include the role of enhanced centrally-mediated skeletal muscle recruitment. Accordingly, a novel finding of this investigation was the ameliorated recovery of central activation; together with increased RMS following CWI. Thus, acute improvements in post-recovery MVC are likely a result of centrally-mediated mechanisms increasing skeletal muscle activation and recruitment based on CWI producing a faster reduction in internal thermal load. Therefore, CWI interventions to hasten recovery of increased thermal load may also act to negate the reported centrally-mediated suppression of VA (Morrison et al. 2004); hence allowing greater skeletal muscle recruitment and improved acute MVC performance following post-exercise CWI.
Further to improvements in central activation, CWI recovery resulted in altered peripheral contractile properties. Slower CD, RR and TPt of the potentiated twitch and duration of the M-wave were evident following CWI compared to CONT. In order to control for the effects of temperature on the evoked signal, M-wave amplitude was normalized to the voluntary EMG signal. Upon normalization, no differences between conditions were evident. Unfortunately, a limitation of the present study was that temperature of the muscle during evoked twitches was not measured. Despite this, it is well-known that reduced muscle temperature with the application of cold significantly alters muscle contractile function and slows nerve conduction velocity (Eston and Peters 1999). Implementation of a re-warm up in the present study performed prior to 2-h and 24-h post-recovery measurements ensured a similar level of potentiation in the evoked signal to pre-exercise measurements. With no differences evident between the conditions for twitch and M-wave duration at 2-h and 24-h post-recovery, the re-warm up was sufficient to counter potential negative effects of cold temperatures on the evoked signal. Regardless of the potential for such changes in evoked twitch contractile properties and whilst the effect of temperature remained evident (post-recovery), the immediate recovery of voluntary force (MVC) following CWI was enhanced compared to a passive recovery. Although CWI slowed the response to the evoked signal, increased VA and RMS highlights the influence of central control in enhancing voluntary force production. Thus, results of the present study indicate that ameliorated recovery of acute MVC is likely due to other factors including reductions in whole-body endogenous thermal and cardiovascular strain (Yeargin et al. 2006), and improved perceptions of thermal recovery and MS.

The ISE protocol in the current study resulted in significant reductions in muscle contractile properties (Pt and M-wave amplitude) and prolonged elevations in CK and AST. Recent studies examining the influence of CWI on markers of muscle damage after a
simulated cycling time-trial in the heat (Halson et al. 2008) and a 90-min rugby training session (Banfi et al. 2007) have reported no effect of CWI on the appearance of CK and CRP. However, these studies measured the appearance of CK and CRP immediately post-recovery (within 40 min of exercise cessation) and therefore the peak expression of such markers is unlikely to have been evident, which may explain the lack of difference between conditions. Regardless, in accordance with previous research, the results of the present investigation also demonstrated that CWI was ineffective in reducing the immediate and prolonged (24 h) appearance of blood markers of muscle damage (Bailey et al. 2007; Halson et al. 2008; Rowsell et al. 2009). Further, a common finding in the literature is the improved perception of MS for up to 48 h following the post-exercise use of CWI recovery interventions (Ascensão et al. 2011; Parouty et al. 2010). For example, Parouty et al. (2010) recently reported an immediate increase in the perception of recovery when CWI was performed between 2 x 100-m swimming sprints. Further, both acute (30 min) and prolonged (24 h) reductions in leg soreness were observed when CWI immediately followed a one-off soccer match (Ascensão et al. 2011). Similarly, the results of the present study demonstrated an immediate reduction in MS following CWI. Whether the improved perception of recovery from CWI contributes to the observed acute improvements in MVC and VA is unknown, and further research may be required to fully elucidate the role of perceptual recovery.

Despite acute improvements in MVC and VA, suppression of force production and global KE RMS was evident 24-h post-recovery following CWI compared to CONT. The decrement in exercise performance 24-h post-recovery contrasts previous investigations examining CWI following intermittent-sprint exercise (Bailey et al. 2007; Ingram et al. 2009). Possible explanations for differences between the present study and previous investigations may be due to the incorporation of double leg bounds, resulting in greater exercise-induced muscle damage (EIMD) compared to intermittent running and cycling
exercise in previous studies (Bailey et al. 2007; Ingram et al. 2009). Indeed, previous investigations reporting no benefit of CWI on recovery of muscle function and strength loss often elicit EIMD via single-joint modalities, frequently causing trauma to muscle contractile properties (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009). Thus, the implementation of cold therapy post-damaging exercise may not be beneficial to the repair of contractile trauma (Yamane et al. 2006). Although the application of cold has been shown to reduce acute inflammation following musculoskeletal injury (Knight 1989), it has recently been suggested that repressing acute inflammation negatively affects repair and regenerative processes of skeletal muscle and may be detrimental to prolonged muscle performance (Barnett 2006; Yamane et al. 2006). As such, although CWI was effective in reducing Tcore, HR and thermal strain, resulting in enhanced short-term recovery of voluntary force, CWI was ineffective in maintaining long-term recovery of isometric muscle function compared to CONT. Despite CWI-induced decrements in 24-h post-recovery single-joint MVC, RSA did not differ. Accordingly, from an applied perspective, while CWI may result in the reduction of single-joint isometric MVC, the ability to produce repeated maximal effort sprints was not compromised.

In conclusion, CWI recovery following simulated team-sport exercise in the heat enhanced the rate of reduction in Tcore, HR and thermal strain, resulting in improved acute recovery of MVC. With an increase in VA and RMS observed post-CWI, it is likely that reductions in thermal and cardiovascular strain improved centrally-mediated mechanisms increasing skeletal muscle recruitment contributing to ameliorated short-term recovery of MVC. Despite acute improvements to the recovery of MVC, CWI resulted in a decrement in voluntary force production and global KE RMS 24 h post-recovery. The precise mechanisms responsible for observed decrements in force production 24-h post-recovery with CWI are unknown and therefore further research is required to fully elucidate the long-term effects of
CWI on muscle repair and adaptation processes necessary for improved isometric muscle function. However, regardless of the recovery of isolated skeletal muscle contractile function, no differences in repeated sprint ability were evident. Accordingly, practitioners should be aware of the mode and duration specific responses to CWI as a recovery intervention following team-sport exercise in the heat.
CHAPTER 5

Cold Water Immersion Recovery Following Simulated Collision Sport Exercise

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ABSTRACT

This investigation examined the effects of cold water immersion (CWI) recovery following simulated collision-sport exercise. Ten male rugby athletes performed three sessions consisting of a 2 x 30-min intermittent-sprint protocol (ISE) with either tackling (T) or no tackling (CONT), followed by a 20-min CWI intervention (TCWI) or passive recovery (TPASS and CONT) in a randomized order. The ISE consisted of a 15-m sprint every minute separated by self-paced bouts of hard-running, jogging and walking for the remainder of the minute. Every 6th rotation, participants performed 5 x 10-m runs, receiving a shoulder-led tackle to the lower-body on each effort. Sprint time and distance covered during ISE were recorded, with voluntary (MVC) and evoked neuromuscular function (VA), electromyogram (RMS), ratings of perceived muscle soreness (MS), capillary and venous blood markers for metabolites and muscle damage measured pre- and post-exercise, and immediately post-recovery, 2-h and 24-h post-recovery. Total distance covered during exercise was significantly greater in CONT (P=0.01), with no differences between TPASS and TCWI (P>0.05). TCWI resulted in increased MVC, VA and RMS immediately post-recovery (P<0.05). M-wave amplitude and peak twitch was significantly increased post-recovery and 2-h post-recovery, respectively in TCWI (P<0.05). Whilst TCWI had no effect on the elevation in blood markers for muscle damage (P>0.05), lactate was significantly reduced post-recovery compared to TPASS (P=0.04). CWI also resulted in reduced MS 2-h post-recovery compared to TPASS (P<0.05). The introduction of body-contact reduces exercise performance, while the use of CWI results in a faster recovery of MVC, VA and RMS and improves muscle contractile properties and perceptions of soreness following collision-based exercise.
INTRODUCTION

Team-sports are characterised by intermittent bouts of high intensity activity, separated by short bouts of low intensity activity (Meir et al. 1993). Many team-sports such as rugby league, rugby union, Australian and American Football and soccer also involve regular collisions between opposing players throughout the course of training and/or match-play. For example, participation in rugby league and rugby union requires players to be exposed to numerous (n=20-40) direct physical collisions and tackles (Brewer and Davis 1995; Gissane et al. 2001) throughout training and/or competition. The combative nature of such sports, combining intermittent high-intensity activity and repeated blunt force trauma, may result in micro-damage to skeletal muscle and post-exercise muscle soreness (Dawson et al. 2004; McLellan et al. 2010). However, to date few studies have attempted to quantify the effect of body-contact on ensuing exercise performance and those that have, report minimal effect of tackling due to insufficient tackling load (Singh et al. 2010). Regardless, with many team-sports training and competing over successive days, often with collision based exercise, time available for physiological recovery can be limited (Rowsell et al. 2009). As repeated collisions may result in potential residual muscle soreness and damage (Peake et al. 2005b), such outcomes can adversely affect subsequent exercise performance (Ronglan et al. 2006). Accordingly, interventions such as cold water immersion (CWI) that are aimed at improving post-exercise recovery have become increasingly popular in many team-sports.

Although the body of literature on the effects of CWI recovery is growing, findings on potential benefits remain equivocal (Eston and Peters 1999; Ingram et al. 2009; Rowsell et al. 2009). In particular, and despite increased popularity, there is a paucity of research examining the effect of CWI following high-intensity, physical collision sport (Banfi et al. 2007). To date, Banfi et al. (2007) is the only investigation to examine the effects of CWI recovery following rugby (collision-based) exercise. While no performance results were
reported, CWI stabilised venous blood creatine kinase (CK) values, although CWI was combined with an initial active recovery. As such, the effect of CWI alone on performance and physiological recovery from rugby, or more specifically, collision-based exercise, remains unknown. Recently, Rowsell et al. (2009) have demonstrated a reduced perception of leg soreness and general fatigue with CWI recovery during a 4-day soccer tournament without any improvement in physical performance. Conversely, Ingram et al. (2009) recently demonstrated ameliorated recovery of sprint performance and maximal voluntary force (MVC) 48-h post-recovery with CWI following 80-min simulated team-sport exercise (without collisions) compared to contrast water immersion and passive recovery. Although findings regarding CWI recovery following team-sport exercise indicate potential benefits (Ingram et al. 2009; Rowsell et al. 2009), there remains a paucity of evidence examining the effects of exercise-induced direct body collisions on performance and the subsequent result CWI has on physiological, performance and perceptual recovery.

Despite reports of increased damage and physiological load with collision-based exercise (Duthie et al. 2003; Gabbett et al. 2008), few studies quantify the subsequent effects on performance (Singh et al. 2010). Moreover, with many professional team-sports implementing CWI recovery into training regimes, minimal evidence to support such practice following direct body contact sport exists. Accordingly, the aim of this investigation was two-fold. The initial aim was to examine the effects of CWI on recovery of neuromuscular, physiological and perceptual function following simulated collision-based team-sport exercise; and second, to quantify the effect of direct lower-body collisions (tackles) on intermittent-sprint exercise performance. It was hypothesised that implementation of direct body collisions would result in a significant decrement in exercise performance. Further, we hypothesized that CWI would result in an enhanced recovery of physiological, perceptual and performance variables.
METHODS

Participants

Ten male, club-level, team sport athletes (rugby league/union) aged (mean ± SD) 21.0 ± 1.7 y, height 182.9 ± 6.1 cm and body mass 87.2 ± 7.7 kg were recruited as participants for this study. At the time of testing, participants completed 3-4 training sessions per week and competed in rugby league/union competition at least once per week. All participants were informed of the requirements of the study and verbal and written consent was gained prior to the commencement of testing. Human ethics clearance was granted by the Institutional Ethics Committee prior to the completion of any testing procedures.

Overview

Participants completed an initial session to ensure familiarity with all physiological, neuromuscular and perceptual procedures, together with an explanation and demonstration of the tackling procedure to be received. This was followed by three testing sessions in a randomized order separated by at least 7 days. The testing sessions were identical apart from the implementation of tackling within the exercise protocol and the use of a post-exercise recovery intervention. Each experimental testing session consisted of a high-intensity intermittent-sprint exercise (ISE) protocol (2 x 30 min halves) performed in an enclosed laboratory on a 20-m synthetic running track at 20.3 ± 1.1°C and 37.0 ± 1.1% relative humidity. Three testing conditions were implemented and consisted of ISE with tackling and CWI recovery (TCWI), tackling with passive recovery (TPASS) and no tackling with passive recovery (CONT). The aim of implementing three conditions was twofold. First, the investigation aimed to examine the effects of CWI recovery following ISE involving intense body collisions (TPASS v TCWI) and, second to examine the effect of the additional load of body collisions on exercise performance (TPASS V CONT) to confirm an effect of the
tackling protocol used. For the tackling conditions (TPASS and TCWI), the ISE protocol included five bouts (repeated every 10 s) of intense body collisions (tackles) performed every 6th min (total of 40 tackles). During the control condition (CONT), no tackles were implemented; rather participants completed 5 x 10-m jogs along the synthetic track. The ISE was followed by 20-min of cold water immersion (TCWI) or passive recovery (TPASS and CONT) intervention with each testing session performed at the same time of day to minimize diurnal variation.

Neuromuscular function was measured pre- and post-exercise, and again immediately post-recovery, 2-h and 24-h post-recovery. Participants were required to present in a rested state and avoid consumption of food or drink (including caffeine) 3-h prior to testing and refrain from alcohol consumption 24-h prior to testing. All food, drink and physical activity in the 24-h prior to the first testing session and during the 24-h recovery period following the exercise protocol were recorded. Participants refrained from any strenuous activity during the 24-h recovery period and food and activity diaries were monitored throughout. Food, drink and activity prior to and during the first testing session were replicated for all testing sessions. During the ISE protocol 500 mL of water was provided to participants to consume *ad libitum* with full consumption monitored during each testing session.

**Exercise Protocol**

Upon completion of pre-exercise neuromuscular tests (detailed subsequently), participants completed a warm-up involving running at increasing speeds over a 15-m running track for a period of 3 min, followed by 3 maximal 15-m sprints. The exercise protocol consisted of 2 x 30-min halves, interspersed by 10-min passive recovery. The exercise protocol involved a 15-m maximal sprint, (with a subsequent 5-m deceleration zone before impacting with a large mat) performed every min, followed by sub-maximal exercise
of varying intensities in a self-paced, shuttle-run format (hard running, jogging, walking) for
the remainder of the minute (Duffield and Marino 2007). Only one exercise mode of hard
running, jogging or walking (rotated each minute) was completed each minute before
returning to the starting position to complete the ensuing sprint. Every 6\textsuperscript{th} rotation following
the maximal sprint, participants received 5 intense lower-body tackles performed by a trained
research assistant. The research assistant was 10-m from the start line and to replicate training
drills was in a kneeling position. Participants were required to run hard for the 10-m before
making contact with the tackling research assistant (jog back to start line in TPASS
condition). The research assistant leaned back as the participants neared and lunged into the
tackles upon contact aiming to provide direct body contact with their shoulder into the
participant’s mid-section of the lower leg, forcing them to the ground (Figure 5.1).
Participants were then required to jog back to the start line and immediately commence the
ensuing tackling bout. Maximal 15-m sprint performance during the exercise protocol was
assessed with infra-red timing gates (Speed-Light, Swift, Australia) and mean sprint time was
calculated. As the exercise intensity was set by the participants, distances covered during the
exercise protocol were monitored by the investigators and appropriate encouragement was
provided to ensure similar workloads were performed during each testing session. Distance
covered for each respective sub-maximal exercise bout was determined using 1-m markings
alongside the 15-m synthetic track. From pilot data collection (n=10), intra-class correlations
(r) and coefficients of variation (CV) for distance covered were r=0.82-0.96 and CV=1.5-
3.2%, respectively.
Figure 5.1 Outline of the tackling procedure performed every 6th rotation during the exercise protocol.
**Recovery Interventions**

Within 10 min of completing the exercise protocol, the recovery intervention was administered in a designated area of the laboratory (20.3 ± 1.1°C, 37.0 ± 1.1% relative humidity). Cold water immersion (TCWI) consisted of seated immersion in an ice bath (plunge pool) (9.2 ± 0.2°C) to a level of the iliac crest for 9 min followed by 1-min seated at room air temperature (Vaile et al. 2008a). This procedure was repeated twice for a total duration of 20 min. For the passive recovery (TPASS and CONT), participants remained seated in the laboratory for 20 min.

**Instrumentation**

**Assessment of Neuromuscular Function**

A 5-min low-intensity warm-up at 60 W on a cycle ergometer (Monark 818E, Varberg, Sweden) was performed prior to the measurement of neuromuscular function at pre-exercise, 2-h and 24-h post-recovery time-points to ensure similar levels of potentiation as during the post-exercise measurement. For the measurement of neuromuscular function, participants were seated on an isokinetic dynamometer (Humac Norm isokinetic dynamometer, Ausmedic, CSMi Medical Solutions, Stoughton, MA) linked to a BNC2100 terminal block connected to a signal acquisition system (PXI1024; National Instruments, Austin, Texas). A/D conversion for torque and electromyographic data was performed at 16-bit resolution and synchronously sampled all data at a rate of 1kHz. Participants were seated upright with a 90° hip angle and securely fastened by adjustable straps tightly across the chest and pelvis with the distal right leg fixed to the dynamometer lever arm. The axis of rotation of the dynamometer was aligned to the lateral epicondyle of the femur, indicating the anatomical joint axis of the knee. Lever arm length, chair length and dynamometer height
were recorded during familiarization for accurate re-positioning during subsequent testing sessions.

Muscle activation was achieved by stimulating the femoral nerve using a felt pad bar cathodal electrode with a tip spacing of 30 mm (Nicolet Biomedical, Madison, WI, USA) positioned at the medio-anterior aspect of the upper thigh, directly below the inguinal fold. The anode was a 90 x 50 mm reusable self-adhesive gel pad electrode (Verity Medical, Ltd., Stockbridge, Hampshire, UK) and positioned on the medio-posterior aspect of the upper thigh, directly below the gluteal fold, opposite the cathode. The current applied to the femoral nerve was delivered by a Digitimer DS7AH stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK) using a single square-wave pulse with a width of 200 µs (400 V with a current of 100-450mA) that was driven by a custom designed instrument using LabView software (version 8.0, LabView; National Instruments). Initially, the current was manually applied and gradually increased until a plateau in twitch and M-wave amplitude was achieved in which time the stimulus intensity was increased by a further 25% to ensure supramaximal stimulation. The site most responsive was marked with a permanent pen to ensure identical placement for subsequent testing. The electrode was then securely fastened in position using a Velcro strap with a constant force of 1.5 kg/f applied via an algometer (Pain Test™ FPI Algometer, Wagner Instruments, Greenwich, CT).

For the measurement of voluntary torque, participants performed 5 x 5-s maximal voluntary isometric contractions (MVC) separated by 5-s rest between each contraction with the knee flexed at 65° (0° being full extension). The production of maximal effort was encouraged for the entire 5 s during which time a superimposed electrical twitch was delivered when a reduction in peak force was observed. The trigger for stimulation was manually primed within 1-2 s after initiation of each contraction and once primed, the stimulus was automatically triggered when customized LabView software detected a decline
in peak force (<1%). Within 3 s following each superimposed contraction, a second stimulus was delivered to the rested muscle to determine potentiated twitch properties. Mean MVC was determined as the peak isometric torque produced during the 25 ms preceding the delivery of the electrical stimulus averaged over the 5 contractions.

Voluntary activation (VA) was calculated using the twitch interpolation technique (Allen et al. 1995). Peak superimposed torque was determined during the 50-150-ms period subsequent to the delivery of the stimulus during each MVC. Interpolated twitch torque was subsequently determined as peak superimposed torque minus voluntary peak torque. VA was determined by expressing the interpolated twitch torque (ITT) as a percentage of the peak potentiated evoked twitch torque (Pt) obtained at rest between contractions using the following equation: VA (%) = [1 – (ITT/Pt)] x 100. Mean VA was determined from the average of all 5 superimposed contractions.

Potentiated twitch and M-wave properties were determined from the electrical stimulus initiated ~3 s following the superimposed contraction on the rested muscle. Torque-time curves from the potentiated evoked twitch contractions were averaged across all trials with mean data used to determine the following characteristics: (1) peak potentiated twitch torque (Pt); (2) the rate of torque development (RTD); (3) time to peak torque (TPt); (4) the rate of relaxation (RR); (5) half-relaxation time (1/2 RT); and (6) contraction duration (CD) (Cannon et al. 2006). Torque onset was determined as the point at which torque increased beyond 2 standard deviations above the mean torque value calculated over a 50-ms period immediately prior to stimulation (Wilder and Cannon 2009). Potentiated M-wave data was averaged across the 5 trials with the mean used to determine: (1) peak to peak amplitude; (2) duration; and (3) latency.

Surface electromyography (EMG) data was obtained from the vastus lateralis (VL), vastus medialis (VM) and the antagonist biceps femoris (BF) for the dominant leg. EMG
signals were sampled using differential surface electrodes (Bagnoli-16, Delsys Inc, Boston, MA) positioned on VL, VM, and BF according to Cram and Kasman (1998), with a reusable self-adhesive electrode attached to the patella of the opposing limb to ground the signals. Low impedance was obtained by shaving, abrading and cleaning the skin prior to positioning of the electrodes at each testing time point. Electrode placement sites were marked with permanent pen to ensure identical placement for subsequent testing sessions. Voluntary EMG data was obtained during the assessment of MVC with the EMG cables taped to prevent movement artifacts in the EMG signal. EMG signals were pre-amplified and bandpass filtered, with a bandwidth frequency ranging from 20 to 450 Hz (common mode rejection ratio > 90dB; impedance input = 100MΩ; gain = 1000).

Voluntary EMG signals were quantified using the root mean square (RMS). RMS amplitude was calculated as the average of the 25-ms preceding the superimposed twitch during MVC. The EMG signal was then averaged between vasti muscles to provide a global indication of total knee extensor (KE) motor unit activity. All data were processed offline with the determination of MVC, VA, potentiated twitch and M-wave properties, and RMS achieved using Matlab software (version R2010a; The MathWorks Inc, Natick, MA). For the MVC, correction for the effect of gravity on the lower leg during the superimposed and potentiated evoked contractions was calculated as the average load applied to the force transducer during the 50-ms period immediately prior to force onset and subsequently used to offset force data. Once corrected for gravity, force data were then multiplied by lever arm length and expressed in units of torque (N·m⁻¹).

**Capillary and Venous Blood Measures**

On arrival, a 100 µL sample of capillary blood was obtained for the measurement of Lactate (La⁻), pH and bicarbonate (HCO₃⁻) with further samples obtained at half-time, post-
exercise and post-recovery intervention. A 5 mL sample of venous blood from the antecubital vein was obtained pre-exercise and post-recovery intervals (immediately, 2 and 24 h) for the measurement of creatine kinase (CK), C-reactive protein (CRP), aspartate aminotransferase (AST) as markers of muscle damage and cell inflammation. Using an evacuated venipuncture system and serum separator tubes (Monovette, Sarstedt, Numbrecht, Germany), samples were allowed to clot at room temperature prior to centrifugation for 10 min at 4000 rpm. Serum was then extracted and stored at -20°C until analysis. Before analysis the serum was allowed to reach room temperature and mixed gently via inversion. CK, CRP and AST were analyzed according to manufacturer’s instructions provided in the respective assay kits (Dimension Xpand spectrophotometer, Dade Bearing, USA). Intra-assay Coefficients of Variance were < 5% for all venous blood analyses.

**Physiological and Perceptual Measures**

Heart rate (HR) was recorded with a heart rate monitor and wrist watch receiver (F1, Polar Electro-Oy, Finland). HR was measured prior to exercise, every 5 min during the exercise protocol and immediately post-recovery. Perceptual measures of rate of perceived exertion (RPE) was determined using the Borg 6-20 Scale (Morrison et al. 2004) with perceived muscle soreness (MS) determined using a 10-point Likert scale (MS: 0 = no pain and 10 = very very sore). RPE was determined pre- and post-exercise and every 5-min during the exercise protocol, whilst MS was determined pre- and post-exercise and throughout the recovery period (post-recovery, 2 h and 24 h).
Statistical Analysis

Data recorded from neuromuscular, physiological and perceptual measures are reported as means ± SD. A repeated measures analysis of variance (ANOVA) (condition x time) was used to determine significant difference between conditions and over time for each recovery intervention. Pairwise comparisons were used to determine the time points for significance which was set at P<0.05. To examine the effect of the recovery intervention, comparisons were made between TPASS and TCWI; whilst the inclusion of tackling on exercise performance was determined with comparisons between TPASS and CONT. In addition, the magnitude of change from pre-exercise values within each condition was determined for neuromuscular, physiological and perceptual variables. Mauchly’s Test of Sphericity was performed to test for the homogeneity of variance (Portney and Watkins 2009). All data collected were analyzed using SPSS™ version 16.0 (Statistical Package for the Social Sciences, Chicago, II, USA).

RESULTS

Distance Covered and Sprint Time

Total distance covered during the exercise protocol was 4178 ± 195 m, 4212 ± 201 m and 4584 ± 385 m for TPASS, TCWI and CONT, respectively. A significantly greater distance was covered during CONT compared to TPASS and TCWI (P<0.01) with no differences evident between TPASS and TCWI (P>0.05). Mean sprint time was significantly increased throughout the duration of the exercise protocol in all conditions (P<0.01; Figure 5.1). TPASS and TCWI resulted in a significantly slower mean sprint time compared to CONT (P<0.05, respectively). Sprint time after the tackle bout was significantly increased throughout the duration of the exercise protocol with TPASS and TCWI resulting in a significantly slower time compared to CONT (P<0.01; Figure 5.2).
Figure 5.2 Mean ± SD (a) Mean sprint time during the exercise protocol and (b) mean sprint time after each tackle bout for CONT: no tackling, passive recovery, TPASS: tackling and passive recovery and TCWI: tackling and cold water immersion.

^ Significant time effect from pre-exercise values in all conditions (P<0.05)
# Significant difference between TPASS and TCWI v CONT (P<0.05)
‡ Tackling bout
Neuromuscular Function

The exercise protocol resulted in a significant reduction in MVC and VA in all conditions \((P<0.01;\) Figure 5.3a and Figure 5.3b, respectively) and remained below pre-exercise values for the 24-h recovery period. The additional load of tackling resulted in a prolonged reduction in VA with a significant reduction 2-h post-recovery in TPASS and TCWI \((P<0.05)\) compared to CONT. A recovery of CWI resulted in a significantly increased MVC and VA post-recovery compared to TPASS \((P<0.05)\). No significant differences were evident between the conditions at any other time point \((P>0.05)\).

Mean RMS of VM/VL was significantly reduced post-exercise and post-recovery in all conditions \((P<0.05;\) Figure 5.3c), with a reduction in RMS also observed 2-h post-recovery in TPASS and CONT only \((P<0.01)\). The additional load of tackling resulted in a significantly increased post-exercise RMS of the BF in TPASS and TCWI compared to CONT \((P<0.05;\) Figure 5.3d). A recovery of CWI resulted in a significant increase in RMS of VM/VL compared to TPASS and CONT post-recovery and 2-h post-recovery \((P<0.05)\).

Significant reductions in post-exercise and post-recovery Pt and RTD were observed in all conditions compared to pre-exercise values \((P<0.01;\) Table 5.1). At 2-h post-recovery, Pt remained decreased compared to pre-exercise values in TPASS and CONT only \((P<0.05)\). Apart from a reduction in RTD \((P<0.05)\), the exercise protocol did not significantly alter T Pt, latency, \(\frac{1}{2}\) RT, RR and CD. However, a recovery of CWI resulted in a significantly slower \(\frac{1}{2}\) RTD, RR and CD post-recovery compared to TPASS \((P<0.05;\) Table 5.1).
Figure 5.3 Mean ± SD (a) Maximal voluntary contraction, (b) voluntary activation, (c) root mean square (RMS) EMG of mean vastus lateralis and vastus medialis, and (d) RMS EMG of biceps femoris for CONT: no tackling, passive recovery, TPASS: tackling, passive recovery and, TCWI: tackling, cold water immersion recovery.

^ Significant time effect from pre-exercise values in all conditions (P<0.05)
^+ Significant time effect from pre-exercise values in TPASS and CONT only (P<0.05)

* Significant difference between TPASS and TCWI (P<0.05)
# Significant difference between TPASS and TCWI v CONT (P<0.05)
§ Significant difference between TPASS and CONT v TCWI (P<0.05)
### Table 5.1 Mean ± SD Potentiated twitch properties.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>Post Rec</th>
<th>2h Post</th>
<th>24h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt (N.m)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>29.7±4.9</td>
<td>23.5±5.1</td>
<td>26.8±4.8</td>
<td>27.6±4.6</td>
<td>28.6±5.0</td>
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<td>TPASS</td>
<td>29.5±4.8</td>
<td>24.3±4.4</td>
<td>26.1±4.4</td>
<td>27.9±4.4</td>
<td>28.1±4.5</td>
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<tr>
<td>TCWI</td>
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<td>24.9±4.7</td>
<td>24.9±4.9</td>
<td>28.3±5.8</td>
<td>29.8±5.7</td>
</tr>
<tr>
<td>TPT (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>CONT</td>
<td>86.1±16.8</td>
<td>83.4±15.4</td>
<td>86.8±12.2</td>
<td>93.4±16.6</td>
<td>89.6±9.9</td>
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<tr>
<td>TPASS</td>
<td>91.8±18.2</td>
<td>94.0±13.9</td>
<td>97.4±27.7</td>
<td>87.8±17.0</td>
<td>92.3±16.8</td>
</tr>
<tr>
<td>TCWI</td>
<td>91.4±15.8</td>
<td>93.7±18.3</td>
<td>90.4±13.5</td>
<td>86.3±10.2</td>
<td>94.0±11.9</td>
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<td>½ RT (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>49.3±9.4</td>
<td>43.4±5.5</td>
<td>44.7±4.9</td>
<td>49.1±8.6</td>
<td>53.0±13.4</td>
</tr>
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<td>TPASS</td>
<td>53.0±8.0</td>
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<td>51.5±10.4</td>
<td>52.1±9.9</td>
<td>57.0±11.7</td>
</tr>
<tr>
<td>TCWI</td>
<td>49.7±7.9</td>
<td>43.0±3.2</td>
<td>58.6±6.8</td>
<td>*</td>
<td>51.6±8.2</td>
</tr>
<tr>
<td>RTD (N.m s⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>352.6±60.8</td>
<td>284.8±54.2</td>
<td>312.8±55.3</td>
<td>306.1±80.2</td>
<td>326.0±80.9</td>
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<td>TPASS</td>
<td>343.6±70.2</td>
<td>260.5±39.4</td>
<td>283.0±70.8</td>
<td>329.4±70.1</td>
<td>309.5±67.5</td>
</tr>
<tr>
<td>TCWI</td>
<td>338.5±92.2</td>
<td>276.8±78.5</td>
<td>284.8±83.3</td>
<td>333.4±80.8</td>
<td>323.2±75.6</td>
</tr>
<tr>
<td>RR (N.m s⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>315.5±72.3</td>
<td>277.3±59.5</td>
<td>307.6±62.1</td>
<td>296.0±80.7</td>
<td>287.9±77.7</td>
</tr>
<tr>
<td>TPASS</td>
<td>295.5±64.4</td>
<td>261.8±55.6</td>
<td>266.2±68.3</td>
<td>280.9±62.9</td>
<td>259.9±70.7</td>
</tr>
<tr>
<td>TCWI</td>
<td>295.6±89.5</td>
<td>297.5±59.1</td>
<td>225.8±87.8</td>
<td>*</td>
<td>286.2±83.9</td>
</tr>
<tr>
<td>CD (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>135.3±22.2</td>
<td>126.8±17.5</td>
<td>131.5±15.9</td>
<td>142.5±20.9</td>
<td>142.6±16.5</td>
</tr>
<tr>
<td>TPASS</td>
<td>144.8±22.6</td>
<td>142.5±20.2</td>
<td>148.9±30.0</td>
<td>139.7±21.5</td>
<td>148.7±24.9</td>
</tr>
<tr>
<td>TCWI</td>
<td>146.0±27.6</td>
<td>136.7±18.8</td>
<td>156.9±25.3</td>
<td>*</td>
<td>137.9±15.0</td>
</tr>
<tr>
<td>Lat (ms)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>36.9±14.7</td>
<td>36.9±14.7</td>
<td>37.0±11.4</td>
<td>34.7±9.7</td>
<td>31.4±12.8</td>
</tr>
<tr>
<td>TPASS</td>
<td>32.8±15.6</td>
<td>22.8±14.2</td>
<td>30.9±12.9</td>
<td>35.7±12.2</td>
<td>31.8±16.5</td>
</tr>
<tr>
<td>TCWI</td>
<td>32.8±16.5</td>
<td>28.6±15.4</td>
<td>30.9±14.0</td>
<td>33.3±10.1</td>
<td>25.5±10.3</td>
</tr>
</tbody>
</table>

^ Significant time effect from pre-exercise values in all conditions (P<0.05)

^+ Significant time effect from pre-exercise values in TPASS and CONT only (P<0.05)

* Significant difference between TPASS and TCWI (P<0.05)
Amplitude, latency and duration of the M-wave were not significantly altered by the exercise protocol in any condition \((P>0.05\); Table 5.2). TCWI did however, result in a significantly higher amplitude in VM compared to CONT post-recovery, 2-h and 24-h post-recovery \((P<0.01\)). The duration and latency of the M-wave in VM was significantly slower following TCWI post-recovery compared to TPASS \((P<0.05\); Table 5.2). No significant differences were evident between the conditions for duration and amplitude in VL.

**Capillary Blood, Venous Blood and Heart Rate**

ISE resulted in a significant reduction in pH and \(\text{HCO}_3^-\) compared to pre-exercise values with greater reductions evident in TPASS and TCWI compared to CONT \((P<0.05\); Table 5.3). \(\text{La}^-\) was significantly elevated post-exercise in all conditions \((P<0.01\) with significantly greater elevations in TCWI and TPASS post-recovery compared to pre-exercise values \((P<0.05\); Table 5.3). TPASS resulted in a significantly lower \(\text{La}^-\) post-recovery compared to TCWI \((P<0.04\). There were no significant differences between recovery conditions at any time point for pH and \(\text{HCO}_3^-\) \((P>0.05\). CK, AST and CRP were all significantly elevated as a result of the exercise protocol in all conditions \((P<0.05\); Table 5.3). However, no significant differences were evident between the recovery conditions at any time point \((P>0.05\). Post-exercise heart rate was significantly increased compared to pre-exercise values in all conditions \((P<0.01\); Figure 5.4a; pg187). No significant differences were evident between the conditions at any time point \((P>0.05\).
Table 5.2 Mean ± SD Potentiated M-wave properties in vastus medialis for CONT: no tackling, passive recovery, TPASS: tackling, passive recovery and TCWI: tackling, cold water immersion recovery.

<table>
<thead>
<tr>
<th></th>
<th>Pre</th>
<th>Post</th>
<th>Post Rec</th>
<th>2h Post</th>
<th>24h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Latency (ms)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>10.1±2.0</td>
<td>9.9±1.8</td>
<td>10.2±1.9</td>
<td>10.3±1.8</td>
<td>11.0±3.2</td>
</tr>
<tr>
<td>TPASS</td>
<td>10.8±1.7</td>
<td>10.5±2.3</td>
<td>11.5±2.3</td>
<td>11.8±1.8</td>
<td>11.2±1.7</td>
</tr>
<tr>
<td>TCWI</td>
<td>11.0±1.8</td>
<td>10.5±2.5</td>
<td>11.7±2.6 *</td>
<td>10.3±2.4</td>
<td>10.4±2.4</td>
</tr>
<tr>
<td><strong>Amplitude (ms)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>4.1±2.1</td>
<td>3.5±2.3</td>
<td>3.9±2.5</td>
<td>3.9±2.6</td>
<td>4.0±2.5</td>
</tr>
<tr>
<td>TPASS</td>
<td>4.9±3.1</td>
<td>5.0±2.7</td>
<td>5.0±2.4</td>
<td>5.0±2.7</td>
<td>5.3±2.7</td>
</tr>
<tr>
<td>TCWI</td>
<td>5.4±1.9</td>
<td>5.3±1.8</td>
<td>6.4±2.0 §</td>
<td>5.9±1.7 §</td>
<td>5.9±2.0 §</td>
</tr>
<tr>
<td><strong>Duration (mV)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>3.8±0.8</td>
<td>3.9±1.1</td>
<td>4.0±0.9</td>
<td>3.9±1.4</td>
<td>4.0±1.1</td>
</tr>
<tr>
<td>TPASS</td>
<td>4.6±2.0</td>
<td>3.7±1.2</td>
<td>3.6±0.7</td>
<td>4.2±1.1</td>
<td>3.8±0.8</td>
</tr>
<tr>
<td>TCWI</td>
<td>3.7±0.8</td>
<td>3.5±0.6</td>
<td>5.0±1.5 *</td>
<td>3.9±0.7</td>
<td>3.9±0.8</td>
</tr>
</tbody>
</table>

§ Significant difference between TPASS and CONT v TCWI (P<0.05)
* Significant difference between TPASS and TCWI (P<0.05)
Table 5.3 Mean ± SD Capillary and Venous Blood variables for Lactate (La$^-$), pH, bicarbonate (HCO$_3^-$), creatine kinase (CK), aspartate aminotransferase (AST) and c-reactive protein (CRP) for CONT: no tackling, passive recovery, TCONT: tackling, passive recovery, TCWI: tackling, cold water immersion recovery.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre</th>
<th>Post-Ex</th>
<th>Post-Rec</th>
<th>2h Post</th>
<th>24h Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>La$^-$ (mmol.L$^{-1}$)</td>
<td>CONT 1.62±0.48</td>
<td>4.46±2.14 ^</td>
<td>1.78±0.64</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPASS 1.38±0.28</td>
<td>5.75±2.62 ^</td>
<td>2.17±0.94 ^*</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCWI 1.56±0.17</td>
<td>5.39±2.91 ^</td>
<td>2.66±1.22 ^#</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH (mmol.L$^{-1}$)</td>
<td>CONT 7.34±0.03</td>
<td>7.33±0.03 ^#</td>
<td>7.36±0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPASS 7.34±0.02</td>
<td>7.32±0.04 ^#</td>
<td>7.35±0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCWI 7.34±0.02</td>
<td>7.32±0.03 ^#</td>
<td>7.34±0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCO$_3^-$ (mmol.L$^{-1}$)</td>
<td>CONT 21.6±1.4</td>
<td>19.2±1.2 ^ #</td>
<td>21.3±0.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPASS 21.3±1.4</td>
<td>18.8±2.0 ^ #</td>
<td>20.9±0.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCWI 21.6±1.3</td>
<td>18.6±1.4 ^ #</td>
<td>20.6±0.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK (IU/L)</td>
<td>CONT 279±282</td>
<td>389±337 ^</td>
<td>472±398 ^</td>
<td>475±374 ^</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPASS 281±193</td>
<td>418±256 ^</td>
<td>457±247 ^</td>
<td>503±357 ^</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCWI 247±143</td>
<td>386±176 ^</td>
<td>467±209 ^</td>
<td>567±346 ^</td>
<td></td>
</tr>
<tr>
<td>AST (IU/L)</td>
<td>CONT 32±19</td>
<td>39±18 ^</td>
<td>39±18 ^</td>
<td>38±18 ^</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPASS 31±13</td>
<td>38±15 ^</td>
<td>40±14 ^</td>
<td>39±14 ^</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCWI 31±12</td>
<td>38±12 ^</td>
<td>40±11 ^</td>
<td>40±12 ^</td>
<td></td>
</tr>
<tr>
<td>CRP (IU/L)</td>
<td>CONT 1.6±0.7</td>
<td>1.8±0.7 ^</td>
<td>1.6±0.6</td>
<td>1.9±0.8 ^</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TPASS 1.9±1.3</td>
<td>2.1±1.5 ^</td>
<td>2.4±2.0 ^</td>
<td>2.5±2.4 ^</td>
<td></td>
</tr>
<tr>
<td></td>
<td>TCWI 1.9±1.5</td>
<td>2.0±1.5 ^</td>
<td>1.9±1.6</td>
<td>3.2±3.0 ^</td>
<td></td>
</tr>
</tbody>
</table>

^ Significant time effect from pre-exercise values in all conditions (P<0.05)
# Significant difference between TPASS and TCWI v CONT (P<0.05)
* Significant difference between TPASS and TCWI (P<0.05)
Perceptual Measures

Rating of MS was significantly increased at all time points in all conditions compared to pre-exercise values \( P<0.01 \); Figure 5.4c). A recovery of CWI resulted in a significantly lower MS 2-h post-recovery compared to CONT \( P<0.05 \) and TPASS \( P<0.05 \). No significant difference was evident between the conditions at any other time point \( P>0.05 \). The additional load of tackling (TPASS and TCWI) resulted in a significantly increased RPE during the first and second half of exercise compared to CONT \( P<0.05 \); Figure 5.4b). In addition, RPE measured after the tackles significantly increased throughout the duration of exercise, with TPASS and TCWI significantly higher than CONT \( P<0.05 \).
Figure 5.4 Mean ± SD (a) heart rate, (b) rate of perceived exertion (RPE) and (c) rate of perceived muscle soreness for CONT: no tackling, passive recovery, TCONT: tackling, passive recovery and, TCWI: tackling, cold water immersion recovery.

^ Significant time effect from pre-exercise values in all conditions ($P<0.05$)

# Significant difference between TPASS and TCWI v CONT ($P<0.05$)

§ Significant difference between TPASS and CONT v TCWI ($P<0.05$)
DISCUSSION

This investigation examined the effects of CWI on recovery of muscle function following simulated, collision-based team-sport exercise. The effect of the additional load of tackling on exercise performance was also examined. The exercise protocol sufficiently induced a significant loss in muscle function, as evidenced by reductions in MVC and VA; which did not return to pre-exercise values during the 24 h recovery period. Further evidence for exercise-induced muscle damage was also observed by significantly elevated blood markers of muscle damage (CK, AST and CRP), increased perceptions of soreness and reductions in potentiated M-wave amplitude and twitch contractile properties. Despite the significant loss in muscle function, recovery by CWI enhanced the immediate restoration of MVC and VA, alongside an ameliorated recovery of RMS, Pt and MS. Accordingly, a novel finding of this study was that the improvement in acute post-exercise MVC following high-intensity, collision-based exercise is likely attributed to an interaction of enhanced central activation and improved recovery of peripheral contractile function. A further novel finding was the observation of a reduction in intermittent-sprint exercise performance as a direct result of the presence of repeated collision-based body contacts.

To date, Singh et al. (2010) is the only study to examine the effects of body-contact on ensuing exercise performance. The implementation of simulated body-collisions into an intermittent-sprint exercise protocol resulted in no greater decrement in performance compared to a non-contact control. However, the authors acknowledged that the lack of effect on performance may have been due to the small volume of contacts and simulated use of body-collisions (tackle bags and bump pads) resulting in limited physically damaging body-on-body contact. In the present study, the incorporation of higher intensity, direct lower-body contact (tackling) resulted in the reduction of performance compared to intermittent-sprint exercise alone. Total distance covered was significantly lower, concomitantly with
reduced mean sprint times in TCONT and TCWI compared to CONT. Consequently, direct body contact results in a reduction in ensuing sprint performance and also in sub-maximal distances covered. The additional tackling load elicited increased physiological responses, as evidenced by a greater reduction in pH and HCO$_3^-$ together with elevated La$^-\text{ values.}$ Moreover, the incorporation of tackling resulted in greater central fatigue compared to the control condition, as observed by a greater reduction in VA. Accordingly, it appears that the presence of repeated direct and forceful body-contact results in a reduction of intermittent-sprint performance for team-sport athletes.

Despite limited research in this area, it is perhaps not unexpected that the presence of simulated tackling results in greater physiological load and reduced performance for team-sport exercise. Interestingly, the tackling condition also resulted in an increased co-activation of the antagonist muscle during post-exercise MVC, with an increase in hamstring RMS evident compared to CONT. To date, no studies have reported the effect of collision-based exercise on neuromuscular responses. However, previous research has reported a reduction in knee extensor force production due to an increased antagonistic effect during fatiguing isometric contractions (Psek and Cafarelli 1993). The reported increase in central drive to the antagonist muscle was postulated as a protective mechanism against damage to the agonist (Psek and Cafarelli 1993). This purported protective effect may explain the present findings, with post-exercise reductions in MVC, VA and concomitant increases in antagonist RMS observed following intense lower-body collisions. Previous research reports that increased volume of collisions during exercise increases the physiological demands of the exercise bout (Duthie et al. 2003; Gabbett et al. 2008). Accordingly, the present study adds to these findings in that incorporating body-contact into any training (or competitive) environment may result in exacerbated performance reductions, possibly via reductions in central
recruitment of skeletal musculature. In addition, the present data also highlights the effective
use of a body-contact intervention in the current study.

Regardless of the effects of body-contact on performance, of primary interest, the
present study highlights the short-term benefits of CWI following collision-based exercise.
These results are in agreement with previous research demonstrating improved recovery of
MVC with CWI following intermittent-sprint (Bailey et al. 2007; Ingram et al. 2009) and
DOMS-inducing exercise (Vaile et al. 2008c). Recently, Ingram et al. (2009) demonstrated
that following 80-min of simulated team-sport exercise, CWI recovery resulted in a smaller
decrement in MVC compared to contrast water therapy and a control condition. Vaile et al.
(2008c) reported an improvement in isometric squat strength 48 h and 72 h following CWI
compared to a passive recovery after DOMS-inducing exercise. Similarly, Bailey et al.
(2007) reported a lower decrement in MVC of the knee flexors at 24 h and 48 h post-recovery
with CWI. The authors in the aforementioned studies postulated that the mechanisms to
explain ameliorated recovery of MVC were due to enhanced recovery of deleterious
symptoms associated with exercise-induced muscle damage and fatigue. Although possible, a
further explanation is the possibility of centrally-mediated mechanisms aiding recovery of
exercise performance (Gandevia 2001). To support this notion, in the present study recovery
of VA and RMS were improved alongside MVC with CWI. Accordingly, the recovery of
MVC following high-intensity, simulated rugby exercise in the current study is possibly
explained by the role of increased centrally-mediated skeletal muscle recruitment in
improving voluntary force (Gandevia 2001).

Despite the significant reduction in exercise performance and muscle function as a
result of the tackling load, CWI recovery ameliorated the decline in MVC and VA, alongside
increased RMS of the agonist muscle group and improved perceptions of MS. Although
Eston and Peters (1999) previously reported a faster return to baseline strength values
following CWI and postulated a reduction in eccentric exercise muscle damage; to date, the improvement in central activation following CWI has not been investigated, particularly following collision-based, team-sport exercise. As such, the results of the present study highlight an increase MVC and a novel finding of an increase in VA and RMS, together with improvements in Pt and M-wave amplitude following CWI. Such findings may suggest that improvements in MVC are a result of enhanced recovery of peripheral contractile ability as well as increased central activation. Although the causal relationship between ameliorated recovery of muscle contractile function and physiological parameters influencing central motor drive is unknown; the enhanced recovery of central and peripheral muscle function following CWI in the present study highlights the need for further investigation examining the potential relationship between these variables.

Whilst CWI recovery resulted in an increase in Pt and M-wave amplitude, ½ RT, RR, CD, M-wave duration and latency were slower compared to TCONT and CONT. Unfortunately, a limitation of the present study was that the temperature of the muscle during evoked twitches was not measured. Despite this, it is well-known that the application of cold significantly slows motor and sensory nerve conduction velocity (Algafly and George 2007; Eston and Peters 1999; Swenson et al. 1996), reduces neuromuscular transmission (lengthened duration of the compound-evoked muscle potential, M wave) and lengthens contraction and half-relaxation time of evoked twitches (Bigland-Ritchie et al. 1992). Regardless of such changes in evoked twitch contractile properties, the recovery of MVC following CWI was enhanced immediately post-recovery compared to a passive recovery (TCONT and CONT). Although CWI resulted in a slower response to the evoked signal, an increased VA and RMS following CWI may highlight the influence of central modulation in enhancing post-recovery MVC, possibly due to other factors including alterations in muscle contractile properties and enhanced perceptions of muscle soreness.
Significant elevations in CK, AST and CRP suggests that considerable muscle damage and cell inflammation was present (Peake et al. 2005b). However, with no significant differences evident between the tackling conditions and CONT, it is likely that the cause of increase in CK, AST and CRP was due to high-intensity nature of the exercise. Blood markers of muscle damage remained elevated with values significantly higher 24 h post-recovery compared to pre-exercise values in all conditions, which has previously been shown following rugby league (McLellan et al. 2011) and union match-play (Takarada 2003). The lack of observed difference in the appearance of markers of muscle damage in the present study compared to those evident in McLellan et al. (2010) and Takarada (2003) may be due to the aforementioned studies examining actual match-play, and as such considerably greater impact force compared to the simulated tackle may have been evident. CWI recovery had no significant effect on the appearance of CK compared to a passive recovery, which corroborates previous research using exercise-induced muscle damage (Bailey et al. 2007; Howatson et al. 2005; Jakeman et al. 2009) and simulated team-sport exercise (Rowsell et al. 2009). Rowsell et al. (2009) recently reported that CWI recovery following each match during a 4-day simulated soccer tournament did not significantly alter the elevation in CK or lactate dehydrogenase. Similarly, despite a post-exercise reduction in myoglobin, Bailey et al. (2007) reported that CWI had no significant effect on CK response following a 90-min intermittent shuttle running. Furthermore, a recovery of CWI did not significantly alter the elevation of AST and CRP in the present study. The lack of difference in the CRP response is also supported by Halson et al. (2008) who demonstrated that following a 40-min simulated cycling time-trial, CWI recovery did not significantly alter the presence of CRP and additional blood markers of muscle damage and inflammation. The results of the present study support the aforementioned studies and suggest that CWI is not effective in reducing
the appearance of indirect blood markers of muscle damage and inflammation in the 24 h recovery period after intense, high-impact collision-based exercise.

Finally, in agreement with previous studies (Bailey et al. 2007; Halson et al. 2008; Rowsell et al. 2009), CWI recovery resulted in a significant reduction in perception of muscle soreness evident 2-h post-recovery. Rowsell et al. (2009) recently demonstrated reductions in perceptions of general fatigue and leg soreness following CWI recovery during a 4-day simulated soccer tournament. Similarly, lower ratings of general fatigue and leg soreness were reported when CWI was implemented following a simulated 20-min cycling time-trial (Halson et al. 2008). Previous investigators suggest that athletes perform better when they believe they have received beneficial treatment (Beedie 2007; Clark et al. 2000). Therefore, it is possible that the enhanced perception of muscle soreness observed in the current study might have arisen due to a potential placebo effect of CWI. Although the placebo effect is a possible explanation for the observed reduction in perceptions of muscle soreness, the acute recovery of MVC, VA, RMS and Pt were improved following CWI. As such, despite the possibility of a placebo effect enhancing perceptions of muscle soreness, CWI implemented in the current study did elicit improvements in acute recovery of peripheral contractile function following intense, collision-based exercise.

CONCLUSION

High-intensity, intermittent-sprint exercise with the additional load of intense body-contact, simulating collision sports (rugby union/rugby league), resulted in a significant reduction in exercise performance, muscle function and increased soreness. In comparison to intermittent-sprint exercise alone, the incorporation of lower-body tackles (collisions) resulted in an increased physiological load and greater decrement in exercise performance. Despite the reduction in muscle function and exercise performance, CWI improved the acute
recovery of MVC compared to a passive recovery. Moreover, CWI resulted in improvements in VA, RMS, perceptions of MS and potentiated M-wave and twitch responses. Such improvements following CWI are likely due to an interaction between alterations in the state of peripheral contractile ability and enhanced skeletal muscle recruitment via increased central activation. Accordingly, the CWI-induced improvement in recovery of voluntary force and activation together with enhanced perception of MS would suggest an effective implementation of this recovery strategy in contact-based sports.
CHAPTER 6

Discussion
OVERVIEW:

Team-sport exercise consists of frequent bouts of high-intensity exercise, separated by low intensity exercise (Coutts and Duffield 2010; Duthie et al. 2006; McLean 1992), often with large eccentric components (jumping, sprinting etc) and exposure to intense physical collisions between opposing players (Dawson et al. 2002; Duthie et al. 2005). The high-intensity, physically demanding nature of team-sports exercise may result in prolonged reductions in muscle function, increased symptoms of EIMD and elevated blood markers of muscle damage (Gill et al. 2006; McLellan et al. 2010). Accordingly, strategies including cold therapy are often implemented to counter deleterious residual effects of training or competition bouts aiming to maximise subsequent exercise performance. To date, evidence outlining the short- and long-term effect of cold therapy on recovery of exercise performance, skeletal muscle function, perceptions of recovery and endocrine responses remains equivocal; potentially due to mode-specific responses to the exercise performed or conditions encountered (Eston and Peters 1999; Isabell et al. 1992; Peiffer et al. 2009a). Accordingly, the aim of this thesis was to examine the effect of post-exercise cold therapy on recovery of skeletal muscle function following exercise conditions indicative of team-sports.

EIMD resulting from eccentric muscle actions, high-intensity sprinting and physical collisions during team-sport exercise results in immediate and prolonged reductions in skeletal muscle function (Evans et al. 1990; Ronglan et al. 2006). As a result, implementation of post-exercise cold therapy following EIMD resulting from team-sport exercise has become increasingly popular (Barnett 2006). Although cold therapy is a well-documented treatment for acute musculoskeletal injury (Bailey et al. 2007; Yanagisawa et al. 2003a), there is a paucity of evidence outlining the effects of cold therapy on recovery of peripheral muscle function following EIMD (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009). As such, in order to specifically examine the effects of cold therapy recovery on
skeletal muscle function, a single-joint exercise protocol inducing EIMD via eccentric contractions was completed in the initial investigation.

In addition to the presence of EIMD, it is common for team-sport athletes to train and compete in warm environmental conditions; particularly during major competitions and pre-season training. Although exercise-induced elevations in thermal load are well established to negatively affect neuromuscular function (Armstrong et al. 2007; Wendt et al. 2007), few studies to date have quantified the effect of post-exercise cold therapy on the recovery of neuromuscular function following high-intensity exercise in the heat (Peiffer et al. 2009a), particularly for team-sport exercise. Accordingly, the second investigation aimed to elucidate the effect of post-exercise CWI on the recovery of neuromuscular function following high-intensity, team-sport exercise in the heat.

Finally, team-sport exercise, particularly rugby league, rugby union, Australian Football League and American Football, consist of high-intensity ISE interspersed by high impact, physical collisions (Dawson et al. 2002; Duthie et al. 2005). As a result, exposure to direct physical collisions has been demonstrated to increase prolonged muscle soreness and reduce skeletal muscle function (Duthie et al. 2003; Gabbett et al. 2008). Despite reports of the presence of muscle damage and increased physiological load with collision-based exercise (Duthie et al. 2003; Gabbett et al. 2008), few studies have quantified the subsequent effects of increased load on exercise performance (Singh et al. 2010, 2011). Further, many professional team-sports implement CWI in recovery regimes, although there is minimal evidence to support such practice following collision-based exercise (Banfi et al. 2007).

Accordingly, the primary aim of the final investigation was to examine the effects of CWI on the recovery of skeletal muscle function and symptoms of damage and soreness following simulated collision-based team-sport exercise. A secondary aim of this investigation was to
quantify the effect of direct lower-body collisions (tackles) on ensuing intermittent-sprint performance.

**Summary of the main findings**

The initial investigation consisted of a high-velocity, CON/ECC exercise protocol of the dominant leg KE, with measures of MVC, VA, voluntary EMG, evoked twitch contractile and M-wave properties measured pre- and post-exercise, and throughout a 48-h recovery period (immediately, 2 h, 24 h and 48 h post-recovery). Indirect blood markers of muscle damage and cell inflammation, perceptual ratings of pain and MS were also determined during the aforementioned time points. Although exercise resulted in prolonged reductions in skeletal muscle function and elevated blood markers of muscle damage, implementation of cold therapy did not significantly hasten the recovery of suppressed muscle contractile function. Apart from a significant reduction in the perception of pain observed at 48-h post-recovery, COLD did not significantly alter indices of EIMD. However, it was interesting to note that despite not reaching significance, a large trend for a higher percentage of pre-exercise MVC was observed post-recovery in COLD compared to CONT ($P=0.08$).

Regardless, the findings of study one suggest that a single application of COLD is of no significant benefit to restore the reduction in skeletal muscle function and contractile damage following eccentric exercise invoking EIMD. Specifically, it is apparent that when significant damage and trauma is present in the peripheral contractile apparatus, post-exercise COLD application does little to restore the reduction in skeletal muscle function.

The second investigation examined the efficacy of CWI on the recovery of neuromuscular function following simulated team-sport exercise in the heat. Measures of MVC, VA, voluntary EMG, potentiated twitch contractile and M-wave properties, blood markers of muscle damage, perceptual ratings of pain and muscle soreness were determined
pre- and post-exercise, and throughout a 24-h recovery period (immediately, 2-h and 24-h post-recovery), whilst HR, Tcore and perceptual ratings of thermal strain, thirst and exertion were recorded throughout the exercise protocol. The results of the second study demonstrated that following prolonged ISE in the heat, CWI provided a significant improvement in the acute recovery of MVC and VA. Additionally, CWI produced a faster reduction in Tcore and HR, together with reduced perceptions of MS. A novel finding of this study was that immediately following CWI, VA and RMS were increased compared to CONT and as such, the resultant increase in neural drive and skeletal muscle motor unit recruitment likely contributed to the observed improvements in muscle function. Despite initial improvements in MVC and VA following CWI, an additional novel finding of this investigation was the suppression of voluntary force and RMS 24-h post-recovery compared to passive recovery. Accordingly, implementation of post-exercise CWI following team-sport exercise in the heat provided immediate beneficial effects to voluntary force production and activation; although was counterproductive to prolonged recovery of muscle function. Therefore, when the post-exercise reduction in muscle function may be predominantly of ‘central’ origin following exercise in the heat, CWI may produce immediate benefits to restore the suppression of voluntary force via reductions in thermal and cardiovascular strain; however, the mechanisms for prolonged decrements in force production remain unknown.

The final investigation examined the efficacy of CWI on the recovery of neuromuscular function following simulated team-sport exercise involving direct physical collisions. A high-intensity ISE protocol interspersed with lower-body, shoulder-driven tackles was completed, followed by post-exercise CWI or passive recovery. Measures of MVC, VA, voluntary EMG, HR, RPE, potentiated twitch contractile and M-wave properties were determined pre- and post-exercise, and throughout the 24-h recovery period (immediately, 2-h and 24-h post-recovery). The results of the final study demonstrated a
significant reduction in post-exercise muscle contractile function and elevated symptoms of EIMD, which did not return to pre-exercise values during the 24-h recovery period. However, implementation of CWI attenuated the decline in MVC, VA, RMS, Pt and MS. Accordingly, although exposure to intense lower-body collisions during exercise results in reductions in skeletal muscle function and elevated markers of muscle damage, the implementation of post-exercise CWI ameliorates the recovery of VA and RMS, and improves voluntary force production and peripheral muscle contractile function. Therefore, a novel finding of this study was that the improvement in acute post-exercise MVC following high-intensity, collision-based exercise is likely attributed to an interaction of enhanced central activation and improved recovery of peripheral contractile function. A further novel finding of this study was the observation of a reduction in ISE performance as a direct result of the presence of repeated lower-body physical collisions.

Effect of CWI on recovery of skeletal muscle function

Following single- and multi-joint exercise in the present collection of studies, measures of skeletal muscle function were significantly reduced and remained below pre-exercise values for the duration of the recovery period in each investigation (24 h and 48 h). It is likely that the cause of such reductions in each study were related to a combination of impaired central and peripheral function. More specifically, single-joint eccentric exercise in Study One elicited predominant reductions in peripheral contractile function with minimal alterations in voluntary activation. Additionally, prolonged elevations in CK and AST suggest that the likely cause of post-exercise reductions in MVC in Study One were predominantly a result of impaired peripheral function (Bigland-Ritchie et al. 1986). In Study Two and Three (whole-body model) respectively, reduced voluntary force production is suggested to be the result of a combination of altered central and peripheral neuromuscular
function; with observed reductions in VA, RMS, Pt, M-wave amplitude and elevations in CK and AST. The subsequent effects of post-exercise cold therapy seemed to be dependent on the mode and type of exercise-induced fatigue, and hence resulted in varying effects on the recovery of musculoskeletal function. Specifically, following repeated single-joint, high-velocity eccentric contractions in the initial study, implementation of COLD did not significantly hasten the recovery of suppressed muscle contractile function or alter indices of EIMD. In contrast, following high-intensity, whole-body, multi-joint intermittent-sprint activity involving exogenous load (heat and body collisions), implementation of CWI provided a significant improvement in the acute recovery of MVC and VA. As such, the effect of CWI on the recovery of skeletal muscle function seems dependent upon the type of exercise-induced responses to recover from; and in a generic sense, whether the fatigue causing reductions in voluntary force are essentially peripheral or central in nature. With such findings in mind, the rationale of this thesis was to explore neuromuscular responses to cold therapy on post-exercise recovery and quantify central and peripheral influences to explain ergogenic qualities of post-exercise cooling.

In accordance with previous research, cold therapy implemented following localized exercise inducing muscle damage did not significantly attenuate reductions in skeletal muscle function (Howatson et al. 2005; Jakeman et al. 2009). Indeed, Jakeman et al. (2009) recently reported that a single bout of CWI following 10 x 10 CMJ resulted in no significant effect on the restoration of concentric muscle strength. In another study, when repeated applications of cold therapy were administered following damaging exercise, Howatson et al. (2005) demonstrated that ice massage was ineffective in reducing markers of muscle damage and enhancing the recovery of isometric and isokinetic strength of the elbow flexors. Similarly, despite a reduction in muscle stiffness, Eston and Peters (1999) reported no benefit of repeated CWI on the recovery of strength loss following EIMD. Thus, in accordance with the
results of the initial study, a single bout of cold therapy following exercise invoking significant damage and trauma to a localised skeletal muscle group, does not significantly hasten the recovery of skeletal muscle contractile damage (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009).

Although post-exercise cold therapy did not significantly hasten the recovery of muscle function following single-joint EIMD, the results of study two and three demonstrated ameliorated recovery of acute MVC following prolonged, whole-body, high-intensity, intermittent-sprint activity. In agreement, recent studies have also observed beneficial effects on the recovery of post-exercise voluntary force following CWI after whole-body exercise (Bailey et al. 2007; Ingram et al. 2009; Vaile et al. 2008c). The results of study two demonstrated that post-exercise CWI resulted in a faster reduction in $T_{core}$ and HR, concurring with Vaile et al. (2010) and Peiffer et al. (2010a), who respectively, observed subsequent improvements in cycling performance. Specifically, Peiffer et al. (2010a) observed smaller reductions in power output and thus a faster completion of a subsequent 4-km cycling time trial with CWI compared to a seated recovery ($1.5 \pm 0.2\%$ CWI v $14 \pm 1.0\%$ control); whilst Vaile et al. (2010) observed a maintained cycling performance between the first and second bout of exercise with CWI compared with an active recovery. Whilst previous investigations have postulated that enhanced subsequent exercise performance is likely due to the faster reduction in thermal strain (Peiffer et al. 2010b; Vaile et al. 2008a; Yeargin et al. 2006) or a restoration of parasympathetic reactivation (Buchheit et al. 2009); the novel finding of study two further elucidated the role of enhanced centrally-mediated skeletal muscle recruitment in increasing acute recovery of MVC in response to reduced thermal load. Indeed, post-exercise CWI in the heat results in a faster reduction in $T_{core}$ and HR with concomitant increases in central activation, RMS and MVC. Thus, acute improvements in post-recovery MVC in study two are likely a result of centrally-mediated
mechanisms increasing skeletal muscle activation and recruitment based on CWI producing a faster reduction in internal thermal and/or cardiovascular load (Nybo and Nielsen 2001). In agreement, Buchheit et al. (2009) recently demonstrated that CWI following supramaximal exercise in the heat resulted in an improved restoration of parasympathetic function. Buchheit et al. (2009) proposed that the observed changes in vagal-related heart rate variability indexes were the combined result of water immersion and cooling. It was proposed the increased hydrostatic pressure and reduced temperature result in an increased intra-thoracic pressure and increased central blood volume, resulting in increased stimulation of blood vessel baroreceptors. The increased central blood volume is proposed to result in increased vagal tone, consequently reducing sympathetic stimulation and heart rate variability. Whilst we can only speculate in the present study, the results of study two and three, together with those of Buchheit et al. (2009), may suggest a possible feedback mechanism from the periphery following CWI to influence central function. As such, the observed reduction in $T_{core}$ and HR along with increased parasympathetic activity following CWI may highlight the central regulation of motor recruitment based on CWI alterations in the periphery. However, further research is required to fully elucidate this area. Whilst the measurement of MVC does not specifically indicate whole-body performance, it does represent the extent to which voluntary skeletal muscle function operates, which may then relate to whole-body performance improvements observed in previous research (Peiffer et al. 2010a; Vaile et al. 2010). Therefore, although the present study demonstrated improvements in acute MVC via ameliorated recovery of VA and RMS, further research examining the relationship between improvements in MVC of isolated muscle groups relating to enhance whole-body performance is warranted.

Similarly, CWI implemented following ISE involving intense lower-body collisions in study three resulted in ameliorated recovery of VA and RMS, with concomitant increases
in acute MVC. Further, the recovery of Pt and M-wave amplitude were improved with CWI, with additional enhanced perceptions of MS. Although the precise relationship has not been investigated previously, improvements in the recovery of central and peripheral function with CWI following simulated collision-based exercise may indicate that acute improvement in MVC is likely due to an interaction of increased peripheral contractile ability, influencing improvements in skeletal muscle recruitment and activation. Indeed, the influence of feedback from the muscle to the CNS resulting in alterations in central motor drive have previously been considered following sustained MVC (Kent-Braun 1999). Input from Golgi tendon organs and small-diameter muscle afferents are known to be an important source of afferent information signalling an altered state of the contractile apparatus (Gandevia 2001). Previously, nociceptor stimuli transmitted via small-diameter afferents were shown to reduce voluntary drive during sustained contractions (Gandevia et al. 1996). Despite the precise relationship between the state of peripheral contractile function, muscle pain and subsequent effects on voluntary drive remaining unknown, the findings of study three may suggest that with increases in Pt and M-wave amplitude, enhanced perceptions of MS and smaller elevations in CK and AST, less noxious stimuli triggering group III and IV afferents may of been evident (Kniffki et al. 1978). As a result, afferent feedback to higher centres indicating an improved state of peripheral contractile ability may be present, thus positively contributing to increased central motor drive excitability and voluntary force production (Kent-Braun 1999). Accordingly, CWI following collision-based exercise may result in an improved contractile state allowing for increased activation via centrally mediated regulation.

The effects of post-exercise cold therapy on the recovery of MVC following EIMD in study one and three in the present collection of studies may be viewed as a contrasting finding. Indeed, COLD implemented following a bout of single-joint eccentric exercise resulted in no significant benefit in the recovery of peripheral muscle function. However,
when whole-body, multi-joint ISE involving direct physical collisions (tackles) elicited EIMD, post-exercise CWI improved the recovery of acute voluntary force via increases in VA and RMS, together with beneficial alterations in peripheral contractile ability. Possible explanations for the observed differences in the present findings may relate to the type of exercise-induced fatigue and damage invoked in the respective protocols. Indeed, following eccentric exercise in study one, reductions in voluntary force production were the result of significant peripheral contractile fatigue and trauma with minimal alterations in central activation, which is in accordance with previous investigations (Sayers et al. 2003). Conversely, whilst ISE involving direct physical collisions in study three also resulted in declines in peripheral contractile function, post-exercise reductions in central activation and recruitment were also observed that contributed to reductions in MVC. Although both exercise protocols resulted in elevated responses in CK and AST, it may be suggested that collision-based exercise resulted in less damage to the contractile apparatus compared to explicit forcibly lengthened eccentric exercise. Such forced activity is demonstrated to result in disrupted sarcomeres in myofibrils and damage to components of the excitation-contraction (E-C) coupling system, reducing the force-generating capacity of the muscle (Morgan and Allen 1999; Proske and Morgan 2001; Warren et al. 2001). As the physical collisions performed in study three were indicative of training forces rather than match-play intensities, it may be that less damage and trauma was induced to the peripheral contractile apparatus compared to explicit eccentric contractions in study one. Thus, it is postulated that the lack of observed effect of post-exercise COLD in study one may be due to increased EIMD invoked via eccentric contractions compared to physical collisions. Further, the observed improvement in post-recovery MVC in study 3, which was absent in study 1, may therefore relate to the presence of significant reduction in VA rather than presence of EIMD.
Despite acute improvements in MVC and VA, suppression of voluntary force production and global KE RMS were evident 24-h post-recovery following CWI compared to CONT in study two. The decrement in performance 24-h post-recovery contrasts with previous investigations examining CWI following ISE (Bailey et al. 2007; Ingram et al. 2009). Possible explanations for differences between the present study and previous investigations may be due to the incorporation of double leg bounds resulting in greater EIMD due to eccentric muscle actions and the landing phase of the bounds (Lakomy and Haydon 2004) compared to intermittent running and cycling exercise in previous studies (Bailey et al. 2007; Ingram et al. 2009). Indeed, previous investigations, including the initial study in the present thesis, reporting no benefit of cold therapy on recovery of skeletal muscle function and strength loss often elicit EIMD via eccentric exercise, frequently causing negative alterations and trauma to muscle contractile properties (Eston and Peters 1999; Howatson et al. 2005; Jakeman et al. 2009). Although the application of cold has been shown to reduce acute inflammation following musculoskeletal injury (Knight 1989), the repression of the inflammatory response has recently been suggested to negatively affect repair and regenerative processes of skeletal muscle and may be detrimental to prolonged muscle performance (Barnett 2006; Yamane et al. 2006). Thus, the implementation of cold therapy post-damaging exercise may not be beneficial to the prolonged repair of contractile trauma elicited via eccentric contractions (Yamane et al. 2006). As such, results of study two demonstrate that although CWI was effective in reducing $T_{core}$, HR and perceptions of thermal strain resulting in enhanced short-term recovery of voluntary force, the long-term recovery of muscle function with CWI compared to CONT was reduced, suggesting that beneficial effects of CWI following ISE in the heat may be time dependent and possibly counterproductive to repair and adaptation processes of muscle function (Yamane et al. 2006).
Indeed, potential detrimental effects of cold therapy on the repair and adaptation processes of skeletal muscle have been observed (Isabell et al. 1992; Yamane et al. 2006). For example, Isabell et al. (1992) observed no effect of cold therapy on indices of muscle damage and suggested repeated cold therapy may in fact be detrimental to muscle repair and adaptation over a prolonged period. In agreement, Yamane et al. (2006) recently examined the effect of cold therapy on endurance or forearm flexor resistance training on untrained men over a period of 4-6 weeks. The results indicated that post-exercise cooling lessened the effects of training by interfering with myofibre micro-damage, and cellular and humoral events induced by endurance and strength training which are considered as physiological preconditions for myofibre repair and regeneration processes leading to improved muscular performance (Yamane et al. 2006). Although evidence supports cold therapy in the treatment of acute soft tissue injury (Myrer et al. 1997; Yanagisawa et al. 2003b), Yamane et al. (2006) suggested that lowering muscle temperature and minimising inflammatory processes by cold therapy may indeed interfere with these regenerative processes and may retard rather than support the desired improvement of muscular performance. While the exact mechanisms remain to be elucidated, these results provide some challenge to the position that the acute inflammatory process must be repressed to favour recovery from muscle damage (Lapointe et al. 2002). Accordingly, with the reduction of MVC, VA and RMS observed in study two, further investigations evaluating the prolonged effect of post-exercise cold therapy on muscle repair and adaptation processes following EIMD are warranted.

Therefore, the overall results of the present collection of investigations suggest that application of cold therapy following single-joint exercise or exercise inducing significant (peripheral) skeletal muscle contractile damage and trauma is of no significant benefit in the restoration of the reduction in muscle function to improve deleterious symptoms associated with EIMD. However, when exercise increases physiological load due to exogenous factors
(heat and direct body collisions), implementation of CWI improves the immediate recovery of voluntary force production. With concomitant increases in post-recovery VA and RMS following CWI, the novel findings of study two and three, respectively highlight the influence of central activation and motor unit recruitment in enhancing subsequent production of voluntary force. As CWI produced a faster rate of reduction in $T_{core}$ and HR in study two, and attenuated the decline in Pt and M-wave amplitude in study three, suggests that the reduction in internal thermal/cardiovascular load and improved recovery of peripheral contractile ability influenced the increase in acute voluntary force production via afferent feedback to higher centres (Kent-Braun 1999). Although improvement in acute MVC was observed following CWI, decrements in long-term recovery of MVC and RMS were evident in study two. Although this was only observed in study two, it is suggested that the beneficial effects of CWI may be dependent on the time period required for recovery; however, further research is required to further elucidate the prolonged effects of post-exercise cold therapy.

**Effect of CWI recovery on physiological markers of exercise induced stress**

The single- and multi-joint exercise modalities completed in the present collection of studies elevated blood markers of muscle damage, cell inflammation and metabolic acidosis. In addition, the high-intensity ISE protocols resulted in significant increases in HR, RPE and perceptions of MS, with pronounced elevations in $T_{core}$ resulting from exercise in the heat. Implementation of post-exercise CWI reduced thermal and cardiovascular strain and enhanced perceptions of MS in accordance with previous investigations (Peiffer et al. 2010b; Vaile et al. 2008a; Yeargin et al. 2006). However, the appearance of CK, AST and CRP, and blood markers of metabolic acidosis were not significantly altered by cold therapy, which concurs with previous studies already reported (Bailey et al. 2007; Halson et al. 2008).
In accordance with previous research, cold therapy implemented following a bout of EIMD and high-intensity ISE did not significantly alter the appearance of CK, AST or CRP (Bailey et al. 2007; Banfi et al. 2007; Halson et al. 2008). Although a single bout of cold therapy was implemented in the present collection of studies, repeated applications of CWI (Goodall and Howatson 2008) and ice massage (Howatson et al. 2005) have also demonstrated no effect on post-exercise CK appearance. Recent studies examining the influence of CWI on markers of muscle damage following multi-joint activity, including simulated cycling time-trial in the heat (Halson et al. 2008) and a 90-min rugby training session (Banfi et al. 2007) have also reported no effect of CWI on CK and CRP appearance. Accordingly, the collective results of the present investigations complement previous findings, suggesting that cold therapy recovery is ineffective in significantly altering the appearance of indirect blood markers of muscle damage and cell inflammation following EIMD (Bailey et al. 2007; Howatson et al. 2005; Jakeman et al. 2009) and simulated team-sport exercise (Rowsell et al. 2009).

Despite the lack of significance between recovery conditions, it is interesting to note that AST was significantly elevated above pre-exercise values 24-h post-recovery in CONT in both study one and two, with similar elevations in CK in study one. Furthermore, the percentage of pre-exercise AST was significantly reduced 24-h post-recovery in COLD compared to CONT. Although not significant ($P=0.34$), the percentage of pre-exercise CK values were 130% (±105%) in COLD v 165% (±131%) in CONT at 48-h post-recovery in the initial study. Consequently, whilst CK and AST presence in the blood are only indirect markers of muscle damage and cell inflammation (Friden et al. 1983), it may be that the application of cold therapy following EIMD and ISE in the heat in study one and two respectively, had some success in blunting the relative post-exercise CK and AST release rate. Eston and Peters (1999) have previously demonstrated a positive effect of CWI on the
appearance of CK and postulated that the observed reduction in CK efflux was possibly due to a decreased permeability of blood and lymph vessels; thus resulting in an attenuated inflammatory response and reduced rate of post-exercise damage to the muscle tissue. An explanation for the lack of observed significance in the present investigations may be due to the large inter-individual variability in these measures (Clarkson and Ebbeling 1988). However, with smaller non-significant elevations in AST and CK appearance observed following cold therapy in study one and two, further research examining the effect of cold therapy on the efflux of indirect muscle damage markers following both EIMD and ISE in the heat may be warranted, particularly examining the relationship of attenuated inflammatory responses on prolonged recovery of skeletal muscle function and adaptation.

Post-exercise CWI following exercise in the heat also produced a faster rate of reduction in $T_{core}$ and HR, in accordance with previous research (Peiffer et al. 2009a; Peiffer et al. 2010b; Vaile et al. 2008a). The faster reduction in thermal and cardiovascular strain are postulated to be due to a combination of peripheral vasoconstriction redistributing cooler blood to the core, increasing central blood volume (Buchheit et al. 2009; Marsh and Sleivert 1999) and thus reducing $T_{core}$ and HR, respectively. In addition, the hydrostatic pressure associated with immersion in cold water is also thought to contribute to the redistribution of blood towards the core, resulting in increased venous return and available blood volume (Wilcock et al. 2006). The resultant increase in central blood volume is thought to stimulate central baroreceptors (Park et al. 1999; Pump et al. 2001) which may enhance vagal nerve activity and inhibit sympathetic activity, leading to bradycardia (Pump et al. 2001) and improve vagal-related heart-rate variability control (Buchheit et al. 2009; Spinelli et al. 1999).

Following CWI in study two, the faster reduction in $T_{core}$ and HR occurred at the same time as increased post-recovery MVC, VA and RMS. As the temperature centre in the
hypothalamus is known to integrate both efferent and afferent signals controlling behavioural and autonomic thermoregulatory response (Nybo and Nielsen 2001), it is possible that the hypothalamus influenced both sensory and motor areas responsible for perception of effort and muscle activation (Bligh 1998). Although there is minimal evidence on the relationship between post-exercise cooling following exercise-induced elevations in T\textsubscript{core} and the central regulation of voluntary force and activation; cooling following passively-induced elevations in T\textsubscript{core} have previously been shown to return MVC and VA to baseline values (Morrison et al. 2004; Thomas et al. 2006). Morrison et al. (2004) and Thomas et al. (2006) postulated that the faster reduction in T\textsubscript{core} directly influenced increases in MVC and VA. Therefore, with CWI resulting in a greater rate of reduction in T\textsubscript{core} and HR, it is possible that afferent feedback to the CNS indicating reduced thermal and cardiovascular strain contributed to an increased motor unit recruitment and muscle activation, thus increasing MVC (Morrison et al. 2004; Nybo and Nielsen 2001; Thomas et al. 2006) in study two.

In summary, the findings of the present collection of studies demonstrate that post-exercise cooling following EIMD and ISE involving exogenous load (heat and body collisions) does not significantly alter blood markers of muscle damage and metabolic acidosis. Despite a lack of significance, large trends for smaller elevations in CK and AST evident in study one and two may suggest a possible blunting of CK and AST release rate with post-exercise cooling. However, further research to fully elucidate the effect of post-exercise cooling on minimising the inflammatory response following team-sport exercise may be warranted, particularly as recent evidence has indicated detrimental effects of cooling on muscle repair and adaptation (Isabell et al. 1992; Yamane et al. 2006). In accordance with previous findings (Peiffer et al. 2009a; Peiffer et al. 2010b; Vaile et al. 2008a), CWI following exercise in the heat produced a faster rate of reduction in T\textsubscript{core} and HR, with concomitant increases in post-recovery MVC, VA and RMS. As such, the enhanced recovery
of acute MVC is suggested to be a result of afferent feedback to the CNS signalling a reduction in thermal and cardiovascular strain; thus contributing to an increase in skeletal muscle recruitment and activation noted in the earlier section.

**Effect of CWI recovery on perceptual markers of exercise induced stress**

The high-intensity, muscle-damaging and ISE protocols implemented in the present collection of studies resulted in significant increases in perceptions of MS and pain which remained above pre-exercise values for the duration of the recovery period (24 h and 48 h). Furthermore, RPE was significantly increased post-exercise in all three studies, whilst increased ratings of thermal strain and thirst were evident post-exercise in study two. Following implementation of CWI, perceptions of MS were significantly reduced post-recovery and 2-h post-recovery in study two and three, respectively, with perceptions of algometer-induced pain threshold significantly reduced 48-h post-recovery. Furthermore, CWI implemented following exercise in the heat significantly reduced perceptions of thermal strain compared to a passive recovery. Therefore, the collective results of the aforementioned studies demonstrate that implementation of post-exercise cold therapy following EIMD and ISE involving exogenous load (heat and body collisions) significantly reduces acute and prolonged perceptions of MS and pain.

In agreement with previous findings, application of cold therapy via various interventions (ice massage, ice packs, cold water immersion) stimulates an analgesic effect resulting in a decreased perception of pain or soreness (Cheung et al. 2003; Meeusen and Lievens 1986). The proposed analgesic effect may explain the acute reductions in perceptions of MS following cold therapy in study two and three; however, the duration of this analgesia is reported to be limited to 1-3 h (Meeusen and Lievens 1986). Alternative explanations for the observed reductions in MS may also relate to the change in hydrostatic pressure.
associated with water immersion resulting in decreases in tissue oedema (Wilcock et al. 2006). However, the recent work of Ascensao et al. (2011) provides support for an acute analgesic effect, rather than hydrostatic pressure, as reductions in MS were observed following CWI but not following thermoneutral immersion. Whilst the analgesic effect of cold may explain acute reduction in MS, the prolonged reduction in MS and pain following cold therapy may relate to the decrease in post-exercise oedema due to cold temperatures and hydrostatic pressure, potentially reducing the likelihood of secondary muscle damage to tissues (Eston and Peters 1999; Vaile et al. 2008c; Wilcock et al. 2006). Despite plausibility, the current studies did not incorporate a placebo control for cooling and thus a treatment effect contributing to enhanced perceptions of MS cannot be dismissed, particularly given the lack of condition-induced changes in twitch contractile properties and blood markers of muscle damage at 24- and 48-h post-recovery.

Given that cold therapy only resulted in trends for reduced perceptions of MS following EIMD in the initial study, the beneficial effects of cold therapy on perceptual recovery may also be dependent on the mode of exercise to recover from and the method of cold therapy used. For instance, in accordance with previous findings, localised EIMD following high-velocity eccentric exercise resulted in prolonged reductions in skeletal muscle function and elevated blood markers of muscle damage, with implementation of cold therapy not improving symptoms of EIMD (Howatson et al. 2005; Jakeman et al. 2009). In contrast, post-exercise CWI in study two and three significantly improved the acute recovery of MVC, VA and RMS, together with increases in Pt and M-wave amplitude. Thus, a possible explanation for the lack of condition-induced changes in study one may be due to the method of cold therapy applied, administered via the application of ice cuffs to the exercised limb compared to CWI implemented in study two and three. As the exercised limb in study one involved a single-joint, isolated muscle group, immersion on one leg was not possible.
Although the application of ice cuffs provided inflated pressure, this may not have been to the extent of pressure exerted during water immersion. As such, application of ice cuffs may have not produced sufficient reductions in muscle temperature to elicit alterations in peripheral contractile function. Indeed, although CD was significantly slowed post-recovery with COLD, no treatment effects were evident in other twitch contractile properties in study one. Further, the additional effect of greater whole-body hydrostatic pressure due to water immersion in study two and three may of elicited greater beneficial physiological changes in accordance with previous findings (Vaile et al. 2008c; Wilcock et al. 2006).

Furthermore, an alternative explanation for the lack of significant condition-induced reductions in perceptions of soreness in study one may also be a result of the significant interruption and damage of peripheral contractile apparatus resulting from eccentric contractions, causing disruption of myofibrils and components of E-C coupling system (Morgan and Allen 1999; Proske and Morgan 2001) making it difficult for any intervention to improve perceptions of MS and pain. With significant localised exercise-induced skeletal muscle damage and trauma evident in study one, implementation of ice cuff therapy was not sufficient to hasten the recovery of peripheral contractile damage and enhance perceptions of MS and pain. Despite reductions in skeletal muscle function following eccentric exercise in study one were primarily the result of interruptions to peripheral contractile ability, multi-joint ISE exercise in study two and three, elicited additional reductions in central activation. Although the precise mechanism responsible for observed improvements in post-recovery peripheral contractile ability (Pt and M-wave amplitude) with CWI in study two and three are unknown, the effect of hydrostatic pressure reducing local tissue oedema and secondary muscle damage may be a possible explanation (Wilcock et al. 2006), thus resulting in lower perceptions of MS and pain. Therefore, with beneficial alterations in peripheral contractile ability and enhanced perceptions of MS with post-exercise CWI in study two and three, the
increased central activation and motor unit recruitment, allowing increased voluntary force production was likely a result of afferent feedback to the CNS indicating improved perceptions of soreness and peripheral function (Kent-Braun 1999). As such, the collective results of the present studies highlight that beneficial effects of post-exercise cold therapy on reducing perceptions of MS, pain and thermal strain are likely to be dependent upon the exercise modality inducing fatigue and damage, together with the method of cold application.

CONCLUSION

The collective results of the present studies demonstrate that cold therapy is of no significant benefit in restoring the reduction in skeletal muscle function following (single-joint) exercise inducing prolonged, localized musculoskeletal damage and trauma. However, for high-intensity, multi-joint ISE including exogenous load (heat and intense body collisions), recovery with CWI is beneficial in improving the restoration of acute voluntary force production. Such improvements in voluntary force are potentially via afferent feedback to higher centres indicating ameliorated recovery of peripheral contractile apparatus and reductions in internal thermal load, thus increasing central activation and skeletal muscle recruitment. Although short-term benefits of CWI were observed with increased voluntary force production, the resultant reduction in long-term (24 h) MVC and RMS may suggest that CWI could be counterproductive to prolonged recovery of skeletal muscle function (Isabell et al. 1992; Yamane et al. 2006). Therefore, the overall results of the present collection of studies demonstrate that implementation of CWI improves acute recovery of skeletal muscle function following whole-body high-intensity, ISE involving exogenous load; however is not beneficial when single-joint exercise eccentric exercise elicits prolonged, localised musculoskeletal trauma. Therefore, the benefits of cold therapy recovery may be dependent on the modality of exercise to recover from. Furthermore, although CWI improved immediate
recovery of MVC via enhanced central activation and skeletal muscle recruitment, the prolonged recovery of voluntary force production was reduced with CWI. As such, the benefits of CWI may also be time dependent.
CHAPTER 7

Conclusions and Summary
RESEARCH QUESTIONS ADDRESSED

Overview

The principal purpose of the thesis was to examine the effects of post-exercise cold therapy on recovery following the various demands associated with team-sports exercise. In particular, the evaluation of neuromuscular function following cold therapy was of primary interest. Therefore, based on the evidence provided in this thesis, the research questions posed in Chapter One are addressed as follows:

Research Questions – Study One

1. Does implementation of post-exercise cold therapy following lower-body single-joint, eccentric exercise improve the signs and symptoms of EIMD?

Despite a significant reduction in the perception of pain evident 48-h with cold therapy, no other benefits in the restoration of skeletal muscle function were observed in the present study following prolonged EIMD. Therefore, cold therapy implemented following lower-body, single-joint exercise inducing prolonged, localised musculoskeletal damage and trauma is of no significant benefit to improve the signs and symptoms of EIMD.

2. Does post-exercise cold therapy alter the recovery profile of neuromuscular function following peripheral contractile damage induced from high-velocity, single-joint eccentric exercise?

The intense nature of the exercise protocol in study one resulted in prolonged reductions in muscle contractile function. Despite this, implementation of post-
exercise cold therapy did not significantly hasten the recovery of peripheral or central mechanisms of muscle function following peripheral contractile damage.

Research Questions – Study Two

1. What are the effects of post-exercise cold water immersion on neuromuscular, physiological and perceptual function following high-intensity intermittent-sprint exercise in the heat, and how does this affect acute and prolonged recovery of voluntary force production?

High-intensity, intermittent-sprint exercise in the heat resulted in acute and prolonged reductions in skeletal muscle function, with concomitant increases in physiological (HR and $T_{\text{core}}$) and perceptual strain (MS). However, implementation of post-exercise CWI provided an immediate improvement in the recovery of MVC and VA. Further, faster rates of reduction in $T_{\text{core}}$ and HR, together with reduced perceptions of MS were also evident following CWI. These results are in line with the recent findings of Buchheit et al. (2009) who observed an increase in parasympathetic activity with CWI. As such it may be suggested that CWI influences vagal modulation, resulting in a positive effect on central function and increasing central motor drive, as was observed in the current study through increased voluntary activation and contraction. The implementation of CWI following prolonged intermittent-sprint exercise in the heat was beneficial in attenuating the acute declines in voluntary force production, likely resulting from reductions in thermal strain and heart rate allowing an increase in voluntary activation and motor unit recruitment. However, a suppression of MVC and RMS was also observed 24-h post-recovery with CWI compared to passive recovery. Although further research is required to examine the long-term effects of
CWI, it may be suggested that whilst post-exercise CWI is beneficial for acute recovery it may have a negative effect on prolonged recovery due to potential detrimental effects on muscle repair and adaptation (Yamane et al. 2006).

**Research Questions – Study Three**

1. **How does the inclusion of simulated direct physical collisions (tackles) impact on prolonged, intermittent-sprint performance compared to a non-contact control?**

   High-intensity, intermittent-sprint exercise in study two was sufficient to induce a prolonged reduction in skeletal muscle function, with an elevation in CK, AST, CRP, perceptions of MS and reductions in potentiated M-wave amplitude and twitch contractile properties also evident. Further, the additional tackling load elicited increased physiological responses (greater reductions in pH and HCO₃⁻ together with elevated La⁻ values) and heightened central fatigue compared to the control condition. Moreover, the incorporation of intense lower-body physical collisions resulted in the reduction in total distance covered and mean sprint times compared to intermittent-sprint exercise alone. Consequently, inclusion of repeated direct and forceful body-contact during intermittent-sprint exercise results in a reduction in ensuing sprint performance and elicits increased physiological and neuromuscular stress.

2. **What are the effects of post-exercise cold water immersion on neuromuscular, physiological and perceptual function following the simulated demands of collision-based team-sport exercise, and how does this affect acute and prolonged recovery of voluntary force production?**
Although the inclusion of repeated, intense physical collisions during intermittent-sprint exercise exacerbates the physiological load of exercise and results in significant reductions in repeated sprint performance and voluntary activation, recovery by CWI enhances the immediate restoration of MVC and VA, together with ameliorated recovery of RMS, Pt and MS. Therefore, post-exercise CWI following high-intensity, collision-based exercise attenuates the acute decline in neuromuscular function, whilst improving the recovery of physiological and perceptual function.

CONCLUSIONS AND SUMMARY

The collective findings of the present studies demonstrate that post-exercise cold therapy is of no significant benefit for the restoration in the reduction in skeletal muscle function following (single-joint) exercise inducing prolonged, localized musculoskeletal damage and trauma. However, when high-intensity, multi-joint ISE includes exogenous load (heat and intense body collisions), recovery with CWI is beneficial in attenuating the decline in acute voluntary force production. The results of the present collection of studies is summarised in table 7.1 and highlights the acute and prolonged effects of CWI following single- and multi-joint exercise. Such improvements in voluntary force are potentially via afferent feedback to higher centres indicating ameliorated recovery of peripheral contractile apparatus and reductions in internal thermal and/or cardiovascular load, thus increasing central activation and skeletal muscle recruitment. Although short-term benefits of CWI were observed with increased voluntary force production, the resultant reduction in long-term (24 h) MVC and RMS may suggest that CWI could be counterproductive to prolonged recovery of skeletal muscle function (Isabell et al. 1992; Yamane et al. 2006). Therefore, the overall results demonstrate that implementation of post-exercise CWI improves acute recovery of skeletal muscle function following whole-body high-intensity ISE involving exogenous load;
however, is not beneficial when single-joint eccentric exercise elicits prolonged, localised musculoskeletal trauma. Furthermore, the benefits of cold therapy recovery are likely dependent on the modality of exercise to recover from and may also be time dependent.
Table 7.1 Summary of the changes to the following variables after cold water immersion. Maximal voluntary contraction (MVC), voluntary activation (VA), root mean square of the M-wave (RMS), amplitude (Amp), latency (Lat), duration (Dur), peak twitch (Pt), time to peak torque (TPt), rate of relaxation (RR), contraction duration (CD), half relaxation time (1/2 RT), creatine kinase (CK), aspartate aminotransferase (AST), c-reactive protein (CRP), muscle soreness (MS), core temperature (T<sub>core</sub>), heart rate (HR).

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<thead>
<tr>
<th>Study One</th>
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<td><strong>Immediate</strong></td>
<td><strong>Acute (2 h)</strong></td>
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<td><strong>Study Two</strong></td>
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- No change
- Improved
- Detrimental
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<tr>
<th>Study Three</th>
<th>MVC</th>
<th>VA</th>
<th>RMS</th>
<th>MVC</th>
<th>VA</th>
<th>RMS</th>
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<td><strong>M-wave properties</strong></td>
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Practical Applications

The overall findings demonstrate that the efficacy of post-exercise cold therapy on the recovery of skeletal muscle function is dependent upon the type of exercise-induced fatigue. For instance, during athletic training when explicit single-joint eccentric contractions induce significant musculoskeletal damage and trauma or when specific EIMD is present, post-exercise cold therapy may be of little benefit to restore immediate and prolonged reductions in skeletal muscle function and improve deleterious symptoms of EIMD. However, when multi-joint ISE involving exogenous load (heat and body collisions) is performed post-exercise CWI may improve the recovery of voluntary force by increasing skeletal muscle recruitment. Thus, when competition or training events are performed in close succession in environments which increase the physiological load of exercise, particularly in the heat or exercise involving direct physical collisions, CWI may be of use to reduce the physiological load imposed and improve the ability to produce maximal voluntary force. However, the present collection of studies also observed a decrement in MVC and RMS 24-h post-CWI after exercise in the heat. With previous research indicating potential negative effects of post-exercise CWI on muscle repair and adaptation (Isabell et al. 1992; Yamane et al. 2006), these collective results challenge the position that reductions in acute inflammation are necessary to favour the long-term recovery of EIMD (Lapointe et al. 2002) and thus caution is required when implementing post-exercise CWI when the aim of exercise is to elicit adaptations for improved muscular performance.

Although the measurement of MVC, VA and RMS implemented in the current studies provided an indication of alterations in skeletal muscle function on a single muscle group, acute improvements in voluntary force production following cold therapy observed in study two and three may also relate to more whole-body performance; particularly as increases in central activation and motor unit recruitment contributed to increases in voluntary force.
production. Thus, the ability to centrally regulate skeletal muscle recruitment based on CWI reducing physiological load may relate to improvements in more whole-body dynamic exercise requiring an increase in production of voluntary force from a number of joints/limbs e.g. maximal sprinting and jumping. However, further research is required to fully elucidate the effects of post-exercise cold therapy on improvements in skeletal muscle function and the direct implications on whole-body exercise performance.

A common finding in the current collection of studies and previous literature favours post-exercise CWI in enhancing the perception of recovery and muscle soreness. Although the results of study one indicated no significant benefit of post-exercise cold therapy on the recovery of neuromuscular function, the prolonged perception of pain was enhanced. Further, in both study two and three involving whole-body exercise, CWI not only improved the recovery of central and peripheral mechanisms of muscle function, but also enhanced the acute and prolonged perception of muscle soreness. Whilst the relationship between improvements in the perception of recovery and enhanced exercise performance is unknown, from a practical point of view, a coach and athlete may favour the implementation of post-exercise CWI based on the improved perception of muscle soreness and pain alone.

**Recommendations for future research**

- Notably, despite not reaching significance in the initial study, a large trend for a higher percentage of pre-exercise MVC was observed post-recovery in COLD compared to CONT \((P=0.08)\). As such, although the overall results in study one indicate that post-exercise COLD does not significantly hasten the recovery of skeletal muscle function following high-velocity, single-joint eccentric exercise; further research examining the potential trends for a faster return to pre-exercise voluntary force values may be required following this type of exercise. As explicit
single-joint eccentric contractions may only be utilised in athletic training, the ability to recover at a faster rate to produce optimal voluntary force may aid subsequent training quality, and thus further research examining this facet of athletic performance is warranted.

- As noted previously, a limitation of the current collection of studies was the inability to measure cerebral function with TMS. Therefore, future research directions would benefit by implementing this measure to allow motor output to be mapped precisely and provide further evidence of the effect of CWI on cerebral function.

- Although the collective findings of the present studies indicate that post-exercise CWI following multi-joint high-intensity, intermittent sprint exercise involving exogenous load (heat and physical collisions) improve the immediate recovery of voluntary force production, the observed reduction in MVC 24-h post-recovery with CWI after exercise challenges the beneficial effects of CWI, particularly the prolonged effects on muscle repair and adaptation. Thus, with post-exercise CWI commonly implemented following training and competition in many amateur and professional team-sports, the prolonged effects following specific training sessions aiming to provide an acute stimulus for chronic adaptation is warranted.

- Furthermore, repeated applications are often implemented throughout the course of a competitive season. As such, the effects of chronic use of cold therapy following various modalities specific to professional team-sports may be of benefit, particularly as the results of the thesis indicate a detriment in prolonged production of voluntary force.
CHAPTER 8

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APPENDIX A

Information and Informed Consent Documents
Investigator Responsibilities - Participants Rights

1) As a subject you are free to withdraw your consent to participate at any time.

2) The researchers will answer any questions you may have in regard to the study at any time.
Questions concerning the study can be directed to:

Ms Monique King
PhD Student
Ph: (02) 6338 6101
Ah: 0438 421 849
School of Human Movement Studies
Charles Sturt University
Email: moking@csu.edu.au

Dr Rob Duffield
Ph: (02) 6338 4939
School of Human Movement Studies
Charles Sturt University
Email: rduffield@csu.edu.au

I, (print your name)_________________________________ have read the information contained within this consent form and any questions I have asked have been answered to my satisfaction.

I agree to participate in this project, realising I can withdraw at any time without being subject to any penalty or discriminatory treatment.

I agree that the purpose of this research and potential risks or discomforts involved with the testing procedures have been sufficiently explained to me.

I also agree that research data and any photos gathered for this study may be published or taken providing my name and confidential details are not used.

I have read the aforementioned criteria, been provided with written explanations of the procedures and understand my rights as a participant.

Testing sessions will involve:

- Capillary and Venous blood samples
- Pre-exercise muscle function assessment (electrical stimulation)
- Exercise Protocol
- Post-exercise muscle function assessment (electrical stimulation)
- Recovery Intervention
- 1, 24 and 48 h post-exercise assessment
  - capillary and venous blood samples
  - muscle function measures (electrical stimulation)

__________________________________________________________
Signature of participant (and parent/guardian if under 18 years of age)

__________________________
Date

__________________________________________________________
Signature of investigator

__________________________
Date
INFORMATION SHEET

Cold Application for Neuromuscular Recovery Following Intense Lower-Body Exercise

Purpose of the Study:
The purpose of this investigation is to examine the effect of cryotherapy (ice cuffs) as a recovery strategy on the acute and prolonged recovery of evoked and voluntary muscle activation following intense, lower-body, exercise.

Procedures:
Participants will be required to attend three sessions at the same time of day, separated by at least 35 days. All testing sessions will be conducted in the Human Performance Laboratory, School of Human Movement, Charles Sturt University. Participants will be required to present in a rested state and avoid the consumption of food, alcohol and caffeine at least 3 h prior to testing (not including water). Participants will also be required to record all food, drink and activity in the 24 h prior to each testing session and replicate this for all testing sessions. If participants are taking any form of supplements for training (e.g. protein, creatine etc), they are required to cease consumption approximately four days prior to testing and not recommence supplementation until after the 48 h post-measures have been taken. This procedure will be required for each testing session.

The first testing session will allow participants to become familiar with the exercise protocol and all experimental procedures. The ensuing two sessions will consist of an intense, single-limb, exercise protocol with a recovery of either cryotherapy or a control condition (no recovery). Each session will involve a single-leg dynamic exercise protocol (isokinetic dynamometer) followed by a 20-min recovery condition. Measures of muscle strength, muscle activation (electrical stimulation), blood parameters and psychological measures will be recorded pre- and post-exercise, and again at 1, 24 and 48 h post-exercise (recovery).

Exercise Protocol
Each testing session will involve a warm-up on the isokinetic dynamometer involving submaximal isometric contractions of the right quadriceps. This will be followed by the completion of pre-exercise muscle function tests prior to the completion of an intense, single-limb (dominant leg) dynamic exercise protocol. The dynamic exercise protocol will consist of repeated maximal concentric (CON) and eccentric (ECC) knee extensor muscle contractions performed on the dominant leg. The exercise protocol will involve:
6 sets of 25 maximal CON (60°/s)/ECC (120°/s) contractions

At the completion of the exercise protocol, participants will complete post-exercise neuromuscular tests and engage in the recovery condition (cryotherapy or control) for a duration of 20-min.

Experimental procedures
Participants will perform two testing sessions for the respective conditions (cryotherapy and control) separated by at least 35 days. Each testing session will be identical apart from the recovery intervention implemented. Following the warm-up, participants will perform the pre-exercise muscle strength and activation measures consisting of resting electromyogram (EMG) (single stimulus x 6) and 5 x 5-s sustained maximal isometric voluntary contractions (MVC) with superimposed twitch (2 pulses at 50Hz separated by 10ms interval) followed by a potentiated twitch (2 pulses at 50Hz separated by 10ms interval). These tests will be performed on both the right and left quadriceps (knee extensors) and the right arm (biceps brachii) (isokinetic dynamometer). These measures will again be recorded immediately after exercise, post-recovery, 1, 24 and 48 h post-recovery.

On arrival to the laboratory, a 100µL sample of capillary blood will be collected from the earlobe for the measurement of lactate, pH and bicarbonate as indicators of metabolic acidosis. This procedure is harmless; however it does involve a small incision to the earlobe with a sterilised lancet which may cause a brief, marginal sensation of pain or discomfort. Further, a venous blood sample will be obtained from the antecubital vein for the measurement of creatine kinase, C-reactive protein, aspartate transminase, myoglobin and troponin as markers of muscle damage and cell inflammation. This procedure will be conducted by a trained pathologist with the participant in a supine position for the duration of the procedure. This procedure may cause some discomfort, however is transient and is a commonly performed procedure in the laboratory. Venous blood will be obtained at rest, immediately post-intervention, 1, 24 and 48 h post-recovery. Perceptual measures of ratings of perceived exertion, rating of muscle soreness and rating of pain threshold (algometer) will also be obtained throughout.

Pre- and post-exercise muscle strength and activation measures on the isokinetic dynamometer will involve electrical stimulation of the muscle as a method to determine both muscle and neural fatigue. This electrical stimulation is performed by way of two adhesive pad electrodes placed over the femoral nerve for the quadriceps, and over the origin and insertion of the biceps brachii. This procedure requires participants to remain in a rested state whilst the electrical stimulus (2 pulses at 50Hz) is delivered to the muscle (resting twitch). Further, participants will also be required to maximally contract either the leg or arm against a fixed resistance, to which a short (<0.3-s) electrical stimulus (2 pulses at 50Hz) will be applied at maximal force during the contraction.

Prior consent will be gained for any visual recording of any testing session (photographs/videos etc.) from the subjects involved and these recordings will remain under
confidential storage and only published with the express permission of the subject involved. If the data is published in any form, participant names and distinguishing features will remain confidential.

**Experimental Conditions**
Following the exercise protocol participants will be required to complete a recovery of either cryotherapy or a control condition (no recovery). Each condition will be 20-min in duration and will involve:

**Cold therapy:** During this recovery condition, leg ice cuffs will be applied and wrapped around the surface of the right quadriceps, knee and calf. The temperature if the ice cuffs will be maintained at ~5°C for the duration of recovery. Participants will be required to remain in a seated position for the duration of recovery (20 min).

**Control:** Subjects will be required to be seated in the same position as during the cryotherapy at room air temperature in a designated area for 20-min.

**Participant Benefit:**
Subjects will receive information pertaining to their physical ability and performance on the exercise protocol.

**Risks and Burdens to the Participant:**
- Participants will find the exercise protocol taxing and difficult, however as all participants will be trained and previously familiarised with the protocol, the exercise will be no more than normal training sessions involving strengthening exercise.
- The electrical stimulation given during the measurement of muscle activation may cause a transient feeling of pain or discomfort; however this does not result in any long term (more than a few seconds) pain. Further, all participants will be familiarised and aware of this procedure prior to testing, and this procedure if often used in our laboratories with Ethics clearance gained on many prior occasions.
- Finally, capillary blood samples will be collected from the earlobe which requires a small incision with a sterilised lancet. This procedure may provide a momentary feeling of pain or discomfort, however this is only brief and no side effects are present. Venous blood samples will be collected from the antecubital vein. This procedure will be conducted by a trained pathologist with the participant in a supine position for the duration of the procedure. This procedure may cause some discomfort, however is transient and is a commonly performed procedure in the laboratory.
- This data will be published in international peer-reviewed journals; however, no individual participant information or results will be included.

**Time Commitments:**
Each testing session will take approximately no more than two hours, with post-recovery measures at 1, 24 and 48 h only taking approximately ½ h each. These sessions will be organised for a time that is convenient for both subject and investigator. Experimental conditions will be separated by at least 35 days. However, if for some reason, the participant can not finish all testing requirements, they are free to withdraw from the project at any time without penalty or discriminatory treatment.
Contact Details:
If participants have any queries throughout the testing procedures, they can contact the researcher on:

Ms Monique King  
School of Human Movement  
Charles Sturt University  
Ph: 6338 6101 (wk)  
0438 421 849 (ah)  
Email: moking@csu.edu.au

Note: Charles Sturt University’s Ethics in Human Research Committee has approved this project. If you have any complaints or reservations about the ethical conduct of this project, you may contact the Committee treatment through the executive Officer:

The Executive Officer  
Ethics in Human Research Committee  
Academic Secretariat  
Charles Sturt University  
Private Mail Bag 29  
Bathurst, NSW, 2795.

Tel: (02) 6338 4628  
Fax: (02) 6338 4194

Any issues you raise will be treated in confidence and investigated fully and you will be informed of the outcome.
COLD WATER IMMERSION RECOVERY FOLLOWING INTERMITTENT-SPRINT EXERCISE IN THE HEAT

Contact Details:
If participants have any queries throughout the testing procedures, they can contact the researcher on:

Ms Monique King
PhD Student
School of Human Movement
Charles Sturt University
Ph: (02) 6338 6101 (wk)
0438 421 849 (ah)
Email: moking@csu.edu.au

Dr Rob Duffield
School of Human Movement
Charles Sturt University
Email: rduffield@csu.edu.au

Participants
Participants involved in the study must be actively involved in a team sport (e.g. rugby league, rugby union, AFL or soccer) and regularly participate in training sessions (~2-3 sessions per week).
Participants will be aged between 18-25 yr, be non-smokers and be presented in a healthy state.

Purpose of the Study:
The purpose of this investigation is to examine the effect of cold water immersion used as a recovery strategy on the recovery of exercise performance following high-intensity, intermittent-sprint exercise, simulating the demands of team sports, performed in warm conditions.

Procedures:
Participants will be required to attend four sessions at the same time of day, separated by at least 14 days. All testing sessions will be conducted in the Human Performance Laboratory, School of Human Movement, Charles Sturt University. Participants will be required to present in a rested state and avoid the consumption of food, alcohol and caffeine at least 3 h prior to testing (not including water). Participants will also be required to record all food, drink and activity in the 24 h prior to each testing session and replicate this for all testing sessions. Within 24 – 48 h prior to each testing session, participants will be given a telemetric core temperature pill to ingest. Participants will be provided with a complete explanation of the procedures and the purpose of the pill and safety procedures. Participants will then need to arrange to consume the core temperature pill 4-h prior to each ensuing session. After ingestion, the telemetric core temperature pill will pass within 24 h with normal bowel movement. Participants will be advised they cannot have an MRI while the pill is in the body until it has passed and will also be given a bracelet warning them of this.
The first testing session will allow participants to become familiar with the exercise protocol and all experimental procedures. The ensuing three sessions will consist of a high-intensity, intermittent-sprint exercise protocol of 2 x 30 min halves. Participants will be required to complete the three conditions in a randomised order which will consist of the exercise protocol performed in warm conditions (~33°C) with a passive recovery (W-CONT), the exercise performed in warm conditions with cold water immersion as a recovery (W-CWI) and the exercise performed in moderate conditions (~22°C) with passive recovery (CONT). Each session will involve a 2 x 30-min high-intensity, intermittent-sprint exercise protocol followed by a 20-min recovery of either cold water immersion (CWI) or passive (CONT). The exercise protocol will consist of repeated bouts of sprinting, hard running, jogging, walking and bounding to simulate the game demands of team sports in either warm or moderate environmental conditions. Measures of exercise performance (sprint time and distance covered), muscle strength, muscle activation (electrical stimulation), blood parameters and psychological measures will be recorded pre- and post-exercise, and again at 1, 2 and 24 h post-exercise (recovery).

**Exercise Protocol**
Each testing session will involve a low-intensity warm-up of the right leg on a leg strength testing machine prior to the assessment of resting muscle function tests. Following the completion of pre-exercise muscle function tests, participants will perform the 2 x 30 min exercise protocol. Prior to beginning the exercise, participants will complete a warm-up involving running at increasing speeds back-and-forth over a 20-m running track for a period of 3-min. This will be followed by 3 maximal 20-m sprints. Participants will then complete the 2 x 30 exercise protocol which will consist of a 20-m maximal sprint (~4-s) every minute. For the remainder of the minute, participants will complete a sequence of shuttle runs incorporating “hard running”, “jogging”, “walking” and “bounds”. During the hard running phase, participants will be required to cover as much distance as possible. Approximately 10-s prior to the next sprint, participants will be asked to jog back to the start line. During the “bounds” phase, participants will be required to complete 10 consecutive, double-leg bounds (frog jumps) with an aim to cover as much distance as possible. Below is a diagram outlining the sequence of the shuttles during the exercise protocol.

```
SPRINT ➔ HARD RUN ➔ SPRING ➔ JOG ➔ SPRING ➔ WALK ➔ SPRING ➔ BOUNDS
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There will be a 10-min rest interval between the 30-min halves in which participants will be required to rest in a seated position. During this time physiological measures will be recorded (capillary blood, heart rate, core temperature, skin temperature, rating of perceived exertion, rating of thirst and rating of thermal strain). Once participants have rested, the second half of the exercise protocol will be completed.

**Experimental procedures**
Participants will perform three testing sessions for the respective conditions (W-CONT, W-CWI and CONT) separated by at least 14 days. Each testing session will be identical apart from the recovery intervention implemented and the specific environmental temperature. Following the warm-up, participants will perform pre-exercise muscle strength and activation measures consisting of a resting electrical stimulus (x 6) and 4 x maximal voluntary contractions (MVC) with electrical stimulus on the right leg. These measures will again be recorded immediately after, 1, 2 and 24 h post-exercise.

On arrival to the laboratory, a 100µL sample of capillary blood will be collected from the earlobe for the measurement of lactate, pH and bicarbonate as indicators of metabolic acidosis. This procedure is
harmless; however it does involve a small incision to the earlobe with a sterilised lancet which may cause a brief, marginal sensation of pain or discomfort. Further, a 5mL sample of venous blood will be obtained from the antecubital vein for the measurement of markers of muscle damage and cell inflammation. This procedure will be conducted by a trained phlebotomist with the participant in a supine position for the duration of the procedure. This procedure may cause some discomfort, however is transient and is a commonly performed procedure in the laboratory. Venous blood will be collected at rest, 1, 2 and 24 h post-exercise. Physiological and psychological measures of capillary blood, heart rate, core temperature, skin temperature, rating of perceived exertion, rating of thirst and rating of thermal strain will also be obtained throughout. Heart rates will be measured with a Polar heart rate chest monitor and wrist watch receiver. Core (internal body) temperature will be measured with Vital-Sense temperature monitor that will detect body temperature from pill ingested by the participant prior to the testing session.

Pre- and post-exercise muscle strength and activation measures will involve electrical stimulation of the muscle. This electrical stimulation is performed by way of two adhesive pad electrodes placed at the top and bottom of the rectus femoris muscle for the quadriceps (leg). This procedure requires participants to maximally contract their quadriceps muscle against a fixed resistance, to which a short (<0.3-s) electrical stimulus will be applied. This will also occur during the maximal voluntary contractions, in which the electrical stimulus will be applied at peak force during the contraction. This may cause discomfort, however this is transient and the procedure has been performed quite often in our laboratory.

Prior consent will be gained for any visual recording of any testing session (photographs/videos etc.) from the subjects involved and these recordings will remain under confidential storage and only published with the express permission of the subject involved. If the data is published in any form, participant names and distinguishing features will remain confidential

Experimental Conditions

Following the exercise protocol participants will be required to complete a recovery of either cold water immersion or a control condition (no recovery). Each condition will be 20-min in duration and will involve:

W-CWI: The subject will perform the exercise protocol in warm environmental conditions (~33°C) followed by a recovery of cold water immersion (CWI). For the CWI, the subject will be immersed in an ice bath (plunge pool) at ~10°C up to a level of the iliac crest for 9-min followed by 1-min seated at room air temperature. This procedure will be repeated twice for a total duration of 20-min.

W-CONT: The subject will perform the exercise protocol in warm environmental conditions (~33°C) followed by a passive recovery (CONT). For the CONT, subjects will be required to be seated in the same position as during the CWI in warm environmental conditions (~33°C) in a designated area for 20-min.

CONT: The subject will perform the exercise protocol in moderate environmental conditions (~22°C) followed by a passive recovery. Subjects will be required to be seated in the same position as during the CWI in moderate environmental conditions (~22°C) in a designated area for 20-min.

Participant Benefit:
Subjects will receive information pertaining to their physical ability and performance on the exercise protocol.

Risks and Burdens to the Participant:
Appendices

- Participants will find the exercise protocol taxing and difficult as it involve high-intensity exercise, designed to induce fatigue and a decrement performance; however as all participants will be trained and previously familiarised with the protocol, the exercise will be no more than normal training sessions involving high-intensity, intermittent-sprint exercise.
- The electrical stimulation given during the measurement of muscle activation may cause a transient feeling of pain or discomfort; however this does not result in any long term (more than a few seconds) pain. Further, all participants will be familiarised and aware of this procedure prior to testing, and this procedure if often used in our laboratories with Ethics clearance gained on many prior occasions.
- Capillary blood samples will be collected from the earlobe which requires a small incision with a sterilised lancet. This procedure may provide a momentary feeling of pain or discomfort, however this is only brief and no side effects are present.
- Finally, venous blood will be collected from the antecubital vein which requires a small incision with a sterilised needle. This procedure may provide a momentary feeling of pain or discomfort, however this is only brief and no side effects are present.
- While the procedure of ingesting a telemetric core temperature pill is harmless, participants may be apprehensive about the ingestion of a foreign object. This procedure is however often used in our laboratories with Ethics clearance gained on many prior occasions.
- This data will be published in international peer-reviewed journals; however no individual participant information or results will be included.

Time Commitments:
Each testing session will take approximately no more than two hours, with post-exercise measures at 1, 2 and 24 h only taking approximately half an hour each. These sessions will be organised for a time that is convenient for both subject and investigator. Participants will also be required to complete a food and activity diary prior to testing and replicate this during each testing session. The completion of this diary will require a small amount of time to fill in and may be a possible burden to the participant. Experimental conditions will be separated by at least 14 days. However, if for some reason, the participant can not finish all testing requirements, they are free to withdraw from the project at any time without penalty or discriminatory treatment.

Note: Charles Sturt University’s Ethics in Human Research Committee has approved this project. If you have any complaints or reservations about the ethical conduct of this project, you may contact the Committee treatment through the executive Officer:

The Executive Officer
Ethics in Human Research Committee
Academic Secretariat
Charles Sturt University
Private Mail Bag 29
Bathurst, NSW, 2795.

Tel: (02) 6338 4628
Fax: (02) 6338 4194

Any issues you raise will be treated in confidence and investigated fully and you will be informed of the outcome.
INFORMATION SHEET

Cold Water Immersion Recovery Following Simulated Collision Sport Exercise

Contact Details:
If participants have any queries throughout the testing procedures, they can contact the researcher on:

Ms Monique King
PhD Student
School of Human Movement
Charles Sturt University
Ph: (02) 6338 6101 (wk)
Email: moking@csu.edu.au

Dr Rob Duffield
School of Human Movement
Charles Sturt University
Ph: (02) 6338 4939
Email: rduffield@csu.edu.au

0438 421 849 (ah)

Participants
Participants involved in the study must be actively involved in a contact team sport (e.g. rugby league, rugby union, AFL) and regularly participate in training and competitive sessions (~3-4 sessions per week). Participants must be aged between 18-25 yr, be non-smokers and present in a healthy and rested state (i.e. no physical exercise 24h prior to testing).

Purpose of the Study:
The purpose of this investigation is to examine the effect of cold water immersion used as a strategy for the recovery of exercise performance following high-intensity, intermittent-sprint exercise, simulating the demands of team sports that incorporate body-on-body contact.

Procedures:
Participants will be required to attend three sessions at the same time of day, separated by at least 14 days between each session. All testing sessions will be conducted in the Human Performance Laboratory, School of Human Movement, Charles Sturt University. Participants will be required to present in a rested state and avoid the consumption of food, alcohol and caffeine at least 3 h prior to testing (not including water). Participants will also be required to record all food, drink and activity in the 24 h prior to each testing session and replicate this for all testing sessions.

The first testing session will allow participants to become familiar with the exercise protocol and all experimental procedures. The ensuing two sessions will consist of a high-intensity, intermittent-sprint exercise protocol of 2 x 30 min halves. Participants will be required to complete the two conditions in a counter-balanced order which will consist of the exercise protocol performed in moderate conditions (~23°C) with a passive recovery (CONT) or with a recovery of cold water immersion (CWI). Each
session will involve a 2 x 30-min high-intensity, intermittent-sprint exercise protocol simulating the demands of contact sports followed by a 20-min recovery of either CWI or CONT. The exercise protocol will consist of repeated bouts of sprinting, hard running, jogging, walking and tackling to simulate the game demands of team sports, particularly sports consisting of intense body-on-body contact. Measures of exercise performance (sprint time and distance covered), muscle strength, muscle activation (electrical stimulation), blood parameters and perceptual measures will be recorded pre- and post-exercise, post-recovery and 2 and 24 h post-recovery.

**Exercise Protocol**

Each testing session will involve a low-intensity warm-up on a leg strength testing machine prior to the assessment of resting muscle function tests. Following the completion of pre-exercise muscle function tests, participants will perform the 2 x 30 min exercise protocol. Prior to beginning the exercise, participants will complete a warm-up involving running at increasing speeds back-and-forth over a 15-m running track for a period of 3-min. This will be followed by 5 maximal 15-m sprints. Participants will then complete the 2 x 30 min exercise protocol which will consist of a 15-m maximal sprint (~4-s) every minute. For the remainder of the minute, participants will complete a sequence of shuttle runs incorporating “hard running”, “jogging”, “walking” and “tackling”. During the hard running phase, participants will be required to cover as much distance as possible. Approximately 10-s prior to the next sprint, participants will be asked to jog back to the start line. During the “tackling” phase, participants will be required to complete the 15-m maximal sprint, jog backwards to the 10-m mark, run forwards toward the tackler and receive a tackle to the lower body. This will be performed 5 times. During the tackling phase a trained research assistant (rugby player) will provide the tackle in a safe manner. The research assistant will be wearing head, shoulder and chest protective gear and will tackle the participant to the ground which will be covered with padded gymnasium mats. Below is a diagram outlining the sequence of the shuttles during the exercise protocol.

![Diagram](image)

- The tackling drill has been adapted from common drills performed during training sessions in both amateur and professional teams. Both the research assistant and participant are required to be experienced in training and competing in sports involving tackling, whilst a thorough familiarisation session will also be conducted prior to testing to ensure participants and research assistants are familiar with and comfortable with all testing procedures. Correct and safe tackling technique will be outlined and practised during the familiarisation session ensuring that participants are aware of the risks associated and how to avoid such hazards.

There will be a 10-min rest interval between the 30-min halves in which participants will be required to rest in a seated position. During this time measures of blood, heart rate and rating of perceived exertion will be recorded. Once participants have rested, the second half of the exercise protocol will be completed.

**Experimental procedures**

Participants will perform two testing sessions for the respective conditions (CONT and CWI) separated by at least 14 days. Each testing session will be identical apart from the recovery intervention implemented. Following the warm-up, participants will perform pre-exercise muscle strength and activation measures consisting of a resting electrical stimulus (x 6) and 5 x maximal voluntary contractions (MVC) with electrical stimulus on the right and left leg, and dominant arm.
These measures will again be recorded immediately after exercise, post-recovery, 2 and 24 h post-recovery.

On arrival to the laboratory, a 100μL sample of capillary blood will be collected from the earlobe for the measurement of lactate, pH and bicarbonate. This procedure is harmless; however it does involve a small incision to the earlobe with a sterilised lancet which may cause a brief, marginal sensation of pain or discomfort. Further, a 5mL sample of venous blood will be obtained from the antecubital vein (forearm) for the measurement of markers of muscle damage and cell inflammation. This procedure will be conducted by a trained pathologist with the participant lying down for the duration of the procedure. This procedure may cause some discomfort, however the pain does not have any lasting affect and is a commonly performed procedure in the laboratory. Venous blood will be collected at rest, post-recovery, 2 and 24 h post-recovery. Physiological and perceptual measures of capillary blood, heart rate, skin temperature, rating of perceived exertion and rating of pain threshold will also be obtained throughout. Heart rates will be measured with a Polar heart rate chest monitor and wrist watch receiver. Skin temperature will be measured via skin thermistors placed on the quadriceps of the right and left leg. Pain threshold will be measured via an Algometer placed on the belly of the quadriceps. A consistent pressure will be applied to the muscle with the participant rating the change in sensation from initial feeling of the instrument to pain. The procedure is harmless; however, it may cause a brief sensation of pain or discomfort.

Pre- and post-exercise muscle strength and activation measures will involve electrical stimulation of the muscle. This electrical stimulation is performed placing electrodes just below the inguinal fold (groin) at the top of the quadriceps muscle to target the femoral nerve. This procedure requires participants to maximally contract their quadriceps muscle against a fixed resistance, to which a short (<0.3-s) electrical stimulus will be applied. This will also occur during the maximal voluntary contractions, in which the electrical stimulus will be applied when the subject reaches peak force during the contraction. For the dominant arm, electrical stimulation will be applied by way of two adhesive electrodes positioned at the top and the bottom of the bicep. The participant will then perform the same procedure as for the quadriceps. These procedures may cause some discomfort, however this is transient and the procedure has been performed quite often in our laboratory.

Prior consent will be gained for any visual recording of any testing session (photographs/videos etc.) from the subjects involved and these recordings will remain under confidential storage and only published with the express permission of the subject involved. If the data is published in any form, participant names and distinguishing features will remain confidential.

Experimental Conditions
Following the exercise protocol participants will be required to complete a recovery of either cold water immersion or a control condition (passive recovery). Each condition will be 20-min in duration and will involve:

CWI: The subject will perform the exercise protocol in moderate environmental conditions (~20°C) followed by a recovery of cold water immersion (CWI). For the CWI, the subject will be immersed in an ice bath (plunge pool) at ~10°C up to a level of the iliac crest for 9 min followed by 1 min seated at room air temperature. This procedure will be repeated twice for a total duration of 20-min.

CONT: The subject will perform the exercise protocol in moderate environmental conditions (~20°C) followed by a passive recovery. Subjects will be required to be seated in the same position as during the CWI in a designated area for 20-min.
Participant Benefit:
Subjects will receive information pertaining to their physical ability and performance on the exercise protocol.

Risks and Burdens to the Participant:
- Participants will find the exercise protocol taxing and difficult as it involves high-intensity exercise with several body-on-body contact (tackles), designed to induce a decrement performance, fatigue and muscle damage typical from a normal game of rugby; however, as all participants will be trained in contact sports and previously familiarised with the protocol, the exercise will be no more than a difficult training session (from which the tackling procedure has been adapted) involving high-intensity, intermittent-sprint exercise consisting of tackling movements.
- The electrical stimulation given during the measurement of muscle activation may cause a transient feeling of pain or discomfort; however this does not result in any long term (more than a few seconds) pain. Further, all participants will be familiarised and aware of this procedure prior to testing, and this procedure if often used in our laboratories with Ethics clearance gained on many prior occasions.
- Capillary blood samples will be collected from the earlobe which requires a small incision with a sterilised lancet. This procedure may provide a momentary feeling of pain or discomfort, however this is only brief and no side effects are present.
- Finally, venous blood will be collected from the antecubital vein which requires a small incision with a sterilised needle. This procedure may provide a momentary feeling of pain or discomfort, however this is only brief and no side effects are present.
- This data will be published in international peer-reviewed journals; however no individual participant information or results will be included.

Time Commitments:
Each testing session will take approximately two hours, with post-exercise measures at 2 and 24 h only taking approximately half an hour each. These sessions will be organised for a time that is convenient for both subject and investigator. Participants will also be required to complete a food and activity diary prior to testing and replicate this during each testing session. The completion of this diary will require a small amount of time to fill in and may be a possible burden to the participant. Experimental conditions will be separated by at least 14 days. However, if for some reason, the participant can not finish all testing requirements, they are free to withdraw from the project at any time without penalty or discriminatory treatment.

Note: Charles Sturt University's Ethics in Human Research Committee has approved this project. If you have any complaints or reservations about the ethical conduct of this project, you may contact the Committee through the executive officer:
The Executive Officer
Ethics in Human Research Committee
Academic Secretariat
Charles Sturt University
Private Mail Bag 29
Bathurst, NSW, 2795.

Tel: (02) 6338 4628
APPENDIX B

Photographs
**Eight (8)** Double-leg bounds for maximum distance completed along the synthetic running track every 6th rotation during the exercise protocol in Study Two
APPENDIX C

Conference Presentations
CONFERENCES PRESENTATIONS

The Effects of Cold Water Immersion on the Recovery of Exercise Performance following Prolonged Intermittent-Sprint Exercise in Warm Conditions

Monique King and Rob Duffield

Presented at the Exercise and Sports Science Australia (ESSA) conference on the Gold Coast, AUSTRALIA 2009

Introduction: This study investigated cold water immersion (CWI) as a post-exercise recovery strategy for physiological and performance responses following intermittent-sprint exercise in hot environmental conditions.

Methods: Following Ethics clearance and Informed Consent, 10 male team-sport athletes performed a familiarisation session and two testing sessions, consisting of a 60-min intermittent-sprint protocol in the heat. Performance was assessed via voluntary force (VF) and activation (VA) of the right quadriceps pre- and post exercise and during recovery (immediately, 2 and 24h). Measures of core temperature (Tc), heart rate (HR), blood markers of damage, muscle soreness (MS) and RPE were measured throughout exercise and recovery. A 20-min recovery of either CWI or control (CONT) was completed following exercise, consisting of immersion in cold water (8.9±0.9°C) to the iliac crest or passive rest in the warm environment. A two-way ANOVA and Cohen’s d effect sizes were used to determine differences between conditions.

Results: Both VF and VA were significantly improved in CWI post-recovery and tended to be higher 2h-post compared to CONT, without any difference at 24h. Tc was significantly reduced following CWI, with the rate of Tc reduction greater in CWI compared to CONT. Blood markers of damage did not differ between conditions; however, perceived MS was reduced following CWI.

Conclusions: CWI appears to be effective in assisting the short-term recovery of force following exercise in the heat. Accordingly, this acute improvement in force may relate to the effective reduction of the endogenous thermal stress and ensuing increased activation of exercised musculature.
Cold Water Immersion Recovery following Simulated, Collision-Based, Intermittent-Sprint Team-Sport Exercise

Monique Pointon and Rob Duffield

To be presented at the Exercise and Sports Science Australia (ESSA) conference on the Gold Coast, AUSTRALIA 2012

Purpose: Many team-sports including rugby league/union and Australian Football involve intermittent bouts of high intensity activity, separated by bouts of low intensity activity and consist of regular collisions between opposing players throughout the course of training and/or match-play. As the combative, intense nature and repeated blunt force trauma associated with such sports may result in micro-damage to skeletal muscle and post-exercise muscle soreness, interventions such as cold water immersion (CWI), aimed at improving post-exercise recovery, have become increasingly popular. Despite this, there is a paucity of research examining the effect of CWI following high-intensity, physical collision, team-sports. Therefore, this investigation examined the effects of CWI on physiological, performance and perceptual recovery following simulated collision-based team-sport exercise.

Methods: Ten male rugby athletes performed three sessions of a 2x30-min intermittent-sprint protocol (ISE) with either tackling (T) or no tackling (CONT), followed by a 20-min CWI intervention (TCWI) or passive recovery (TCONT and CONT) in a randomized order. The ISE consisted of a 15-m sprint every minute separated by self-paced bouts of hard-running, jogging and walking for the remainder of the minute. Every 6th rotation, participants performed 5x10-m runs, receiving a shoulder-led tackle to the lower-body on each effort. Sprint time and distance covered during the ISE were recorded, with voluntary (MVC) and evoked neuromuscular function (VA), electromyogram (RMS), ratings of perceived muscle soreness (MS) and capillary and venous blood markers for metabolites and muscle damage, measured pre- and post-exercise, and immediately post-recovery, 2-h and 24-h post-recovery.

Results: Total distance covered during exercise was significantly greater in CONT (P=0.01), without differences between TCONT and TCWI (P>0.05). CWI increased MVC, VA and RMS immediately post-recovery (P<0.05). M-wave amplitude and peak twitch was increased post-recovery and 2-h post-recovery, respectively in TCWI (P<0.05). Whilst CWI had no effect on the change in blood markers for muscle damage (P>0.05), lactate was significantly reduced post-recovery compared to CONT and TCONT (P=0.04). CWI also resulted in reduced MS 2-h post-recovery compared to CONT and TCONT (P<0.05).

Conclusion: The introduction of body-contact reduces exercise performance, while the use of CWI results in a faster recovery of MVC, VA and RMS. CWI recovery also ameliorates recovery of muscle contractile properties and perceptions of soreness following collision-based exercise.
Cold Water Immersion Recovery Following Intermittent-Sprint Exercise in the Heat

Monique Pointon, Rob Duffield, Jack Cannon and Frank Marino

Presented at the annual congress of the European College of Sports Science Liverpool, UNITED KINGDOM 2011

Introduction: Implementation of cold water immersion (CWI) recovery has become increasingly popular in many professional sports, particularly following exercise in hot environmental conditions. To date, mechanisms for an ergogenic effect of CWI recovery following high-intensity, intermittent-sprint exercise in the heat remain equivocal. As such, this study examined the effects of CWI on recovery of performance, physiological and neuromuscular function following simulated team-sport exercise in the heat. This investigation aimed to examine possible mechanisms responsible for previously reported beneficial effects of CWI recovery following exercise in the heat.

Methods: Ten male team-sport athletes performed two sessions of a 2x30-min intermittent-sprint protocol (ISE) in 32°C and 52% humidity, followed by a 20-min CWI recovery intervention or passive recovery (CONT) in a randomized, crossover design. The ISE involved a 15-m sprint every minute separated by bouts of hard-running, jogging and walking. Maximal voluntary isometric force (MVC) and voluntary activation (VA), ratings of perceived muscle soreness (MS) and blood markers for muscle damage (creatine kinase (CK), aspartate aminotransferase (AST) and c-reactive protein (CRP)) were measured pre- and post-exercise, and immediately post-recovery, 2-h and 24-h post-recovery. Physiological measures of core temperature (Tcore), heart rate (HR), lactate (La⁻) and perceptions of exertion, thermal strain and thirst were also recorded throughout exercise and at the aforementioned time points.

Results: Exercise in the heat resulted in a reduction in MVC and VA, with an increase in Tcore, La⁻, CK, AST and CRP (P<0.05). CWI enhanced the post-exercise rate of reduction in Tcore (1.43±0.36°C CONT v 1.66±0.48°C CWI), HR and MS, whilst increasing post-recovery MVC and VA (P<0.05). A large trend for increased activation and peak twitch force of the right knee extensor was evident immediately and 2h post CWI recovery (P=0.09-20; d=0.8). In contrast, 24-h post-recovery MVC and activation were significantly higher in CONT compared to CWI (P=0.05).

Conclusion: Following exercise in the heat, CWI accelerated the reduction in physiological and cardiovascular load and improved acute recovery of MVC. With an increase in Pt, VA and blunted rise in AST following CWI, improvements in acute MVC were potentially due to a complex interplay of peripheral and centrally-mediated mechanisms. However, despite improved acute recovery of MVC, CWI resulted in an attenuated MVC 24 h post-recovery. While positive acute effects of CWI recovery were present post-exercise, prolonged use of CWI recovery warrants further investigation to fully elucidate the effects of CWI recovery on muscle repair and adaptation.